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ARYA Atherosclerosis

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Serial Issue: 71 *Volume 15, Issue 3, May 2019* *Print ISSN: 1735-3955*
Online ISSN: 2251-6638

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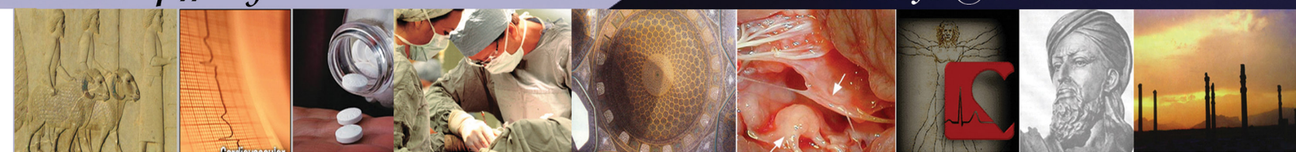

ARYA Atherosclerosis

Official Journal of the Isfahan Cardiovascular Research Institute, Isfahan University of Medical Sciences

Owner: Isfahan University of Medical Sciences
Email: publications@mui.ac.ir
Publisher: Vesnu Publications
Tel/fax: +98 31 32224335, +98 31 32224382
http://farapub.com Email: farapublications@gmail.com

Circulation: 500
Distribution: International
Language: English
Interval: Bimonthly
Print ISSN: 1735-3955, **Online ISSN:** 2251-6638

Address: ARYA Journal Office, Shahid Rahmani Alley, Moshtagh 3rd St, Isfahan Cardiovascular Research Institute, Isfahan, Iran
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DOI: http://dx.doi.org/10.22122/arya.v15i3.1765 Published by Vesnu Publications

Right ventricular dysfunction and associated factors in patients after coronary artery bypass grafting

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Original Article

Abstract

BACKGROUND: Coronary artery bypass grafting (CABG) surgery is widely accepted as a revascularization method for coronary artery disease (CAD). Despite survival benefit and improvement in quality of life, CABG may impose major morbidities and significant complications. Right ventricle (RV) dysfunction is an important complication that may affect patient's longevity and functional capacity. The aim of this study was to evaluate the relationship between RV dysfunction and some invisible parameters like inferior vena cava (IVC) size with physical capacity.

METHODS: In this prospective study, 61 eligible CABG candidates were enrolled and RV function was assessed by echocardiographic parameters before CABG and one week and six months after the procedure, using tricuspid annular plane systolic excursion (TAPSE), Tei Index (TI), peak systolic movement (Sm) (cm/s), and IVC size. Functional capacity was assessed by six-minute walk test (6-MWT) 6 months after CABG.

RESULTS: 58 patients who did not have any perioperative RV dysfunction were remained until the end of study; mean age was 58.2 ± 7.9 years with 68.9% being men, and 3 patients died after CABG. Preoperatively, septal motion, RV indices, and IVC size were normal in all patients. The frequency of RV dysfunction according to abnormal TAPSE index, TI, and peak Sm one week after surgery was 81.0%, 79.0%, and 62.0%, respectively, and 6 months after surgery was 49.0%, 49.0%, and 37.0%, respectively. Mean walked distance in 6-MWT was significantly less in patients with RV dysfunction, older age, and higher number of involved vessels (P < 0.001).

CONCLUSION: The significant reduction in RV function and impairment of exercise capacity after CABG in this study suggests cardiologists to pay more attention to RV assessment in follow-up visits of patients undergoing GABG.

Keywords: Coronary Artery Bypass Surgery, Right Ventricle, Inferior Vena Cava

Date of submission: 18 Mar. 2018, *Date of acceptance:* 27 Mar. 2019

Introduction

Cardiovascular diseases (CVDs) are the leading cause of mortality in most countries with increasing prevalence around the world,[1] mostly coronary heart disease (CHD).[2] Coronary artery bypass grafting (CABG) is one of two main revascularization strategies.[3]

How to cite this article: Chinikar M, Rafiee M, Aghajankhah M, Gholipour M, Hasandokht T, Imantalab V, et al. **Right ventricular dysfunction and associated factors in patients after coronary artery bypass grafting.** ARYA Atheroscler 2019; 15(3): 99-105.

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This procedure is widely used for many patients around the world[4] and has significant post-surgical complications that may end in death or serious morbidity.[5] One of the most important complications of CABG is right ventricle (RV) dysfunction.[6] RV function has been considered a significant determinant of post-CABG outcome and is associated with high mortality and morbidity.[7] Therefore, diagnosis and management of RV dysfunction after CABG is crucial for maintenance of hemodynamic stability and organ function in early post-operation period and prognostic for later phase.[8]

Although depressed RV function is a recognized echocardiographic finding after CABG, the mechanism and clinical significance of this phenomenon is poorly understood.[9] Cardiopulmonary bypass (CPB), perioperative myocardial ischemia, intra-operative cardiac damage, cardioplegia, and pericardial disruption or adhesion are the most proposed mechanisms.[10,11] RV dysfunction in early and late post-operation periods after CABG has been reported by many investigators and in addition it has been emphasized that RV dysfunction lasts at least 12-18 months after the procedure.[12,13] Some reports have proposed that RV dysfunction recovers after 6 months[14] and long-term RV dysfunction is mainly associated with preoperative RV dysfunction, rather than CABG.[15] Moreover, some researchers have suggested that it does not affect the exercise performance and is not clinically significant.[16]

Depressed RV function significantly affects cardiac re-hospitalization and patients' mortality[17] and morbidity. Determination of RV dysfunction after CABG and its predictors is clinically important. Controversy in published studies necessitates evaluating this underestimated derangement after CABG. The current study aimed to determine the frequency of patients with RV dysfunction within one week and 6 months after CABG and effective factors.

Materials and Methods

In this prospective study, 61 eligible CABG candidates at Dr. Heshmat Hospital in Rasht, Iran, were enrolled by simple sampling method. Patients who did not have these factors were included in the study: suffering recent myocardial infarction (MI), previous CABG, atrial fibrillation (AF), significant valvular heart disease (VHD), pulmonary hypertension (PH), history of severe pulmonary disease, pulmonary embolism (PE), left bundle branch block (LBBB) in electrocardiogram (ECG), abnormal septal motion(ASM) in echocardiography, periprocedural MI, RV dysfunction before surgery according to echocardiographic findings [tricuspid annular plane systolic excursion (TAPSE) < 1.6 cm, Tei Index (TI) > 55%, peak systolic movement (Sm) < 10 cm/s, inferior vena cava (IVC) size > 21 mm],[18] and poor acoustic window. The study was approved by the Ethics Committee of Guilan University of Medical Sciences, Rasht, Iran (IR.GUMS.REC.1394.86).

MI associated with CABG is defined by increased cardiac biomarker levels to 5 times the 99th percentile of the reference range during the first 72 hours after surgery, in addition to one of the following criteria: 1) new pathologic Q waves or new LBBB, 2) new graft or coronary artery occlusion, confirmed by angiographic examination, or 3) new loss of viable myocardium or regional wall motion abnormality, confirmed by imaging.[19]

Demographic characteristics, including age, gender, diabetes mellitus (DM), hypertension (HTN), smoking, and body mass index (BMI) were recorded. Patients were diagnosed as DM, when fasting blood sugar (FBS) was ≥ 126 mg/dl or random blood sugar was > 200 mg/dl with symptoms, and were diagnosed as hypertensive, when systolic and diastolic blood pressures were greater than 140 mmHg and 90 mmHg, respectively, or when they were on antihypertensive medication. Patients were considered overweight according to BMI > 25 kg/m^2 and were considered obese when BMI was > 30 kg/m^2. The number of grafts, right coronary artery (RCA) and RV branch lesions and their revascularization, number of diseased arteries, left ventricular ejection fraction (LVEF), pump time, hypothermia, cardioplegia, and pericardial closure were recorded, as well.

Two-dimensional (2D) echocardiography and tissue Doppler imaging (TDI) were performed for all patients before CABG, one week, and six months after the procedure (with Vivid 3 device, GE healthcare model, Germany) by an expert cardiologist. The function of RV was assessed by TAPSE, peak Sm velocity, and TI, based on the protocol explained in previous studies.[20] In addition, IVC size and respiratory variation were measured by M-mode method in subcostal view. ASM was visually speculated by M-mode method from parasternal and apical views. Functional capacity was assessed by the six-minute walk test (6-MWT) 6 months after surgery.

All patients underwent general anesthesia with similar protocol, including induction of anesthesia

with 3 mg/kg/h propofol and 0.5 to 1.0 mg/kg/min remifentanil infusion. CABG with CPB was performed using ascending aortic cannulation and a two-stage venous cannulation in the right atrium. Antegrade cold cardioplegia and moderate hypothermia (approximately 32 °C) were used in all patients and total revascularization was done. In 17 patients, partial pericardial closure was performed.

Normal distribution assumption was checked by skewness and Kurtosis criteria (lower than 1) and Kolmogorov-Smirnov test (K-S test). Continuous and categorical variables were expressed as mean and standard deviation (SD) and frequency and percentage, respectively. McNemar's test was used for comparing the frequency of RV dysfunction between first and second period. Paired t-test was used to compare the mean of RV function indices before surgery and one week after surgery and also one week and 6 months after surgery. Logistic regression test was used to assess the influence of the independent variables on RV dysfunction. To compare the mean distance paced in 6-MWT in patients with and without RV dysfunction based on three indices, independent t-test was used. Linear regression analysis was used to determine the effective factors on 6-MWT. Repeated measures analysis of variance (ANOVA) was used to determine the effect of time for different variables. Data were analyzed using SPSS software (version 19, SPSS Inc., Chicago, IL, USA). P-values < 0.050 were considered statistically significant.

Results

Among 61 patients undergoing CABG who participated in this study, 3 patients were excluded from the study due to cerebrovascular accident (CVA), bleeding after surgery, and inferior wall rupture due to MI. All three indices of RV function, IVC size, and SM were normal before surgery. Baseline characteristics of patients are presented in table 1.

In the first follow-up after surgery (one week after surgery), the frequency of patients with abnormal TAPSE index, TI, and peak Sm were 81.0%, 79.3%, and 62.0%, respectively. The size of IVC and SM was normal in all patients.

Table 1. Baseline demographic characteristics of patients (n = 61)

Characteristic	Amount [n (%)]
Male gender	42 (68.9)
DM	28 (45.9)
HTN	41 (67.2)
Smoking	18 (29.5)
Angiographic result	
SVD	3 (5.0)
2VD	11 (18.3)
3VD	46 (76.7)
Graft number	
1	1 (1.7)
2	7 (11.7)
3	29 (48.3)
4	21 (35.0)
5	2 (3.3)
RCA disease	54 (88.5)
RV branch disease	14 (23.0)
RCA graft	44 (73.3)
Pericardial closure	17 (27.9)
	Mean ± SD
Age (year)	58.2 ± 7.9
LVEF (%)	38.2 ± 22.7
Pump time (minutes)	60.3 ± 27.4

DM: Diabetes mellitus; HTN: Hypertension; SVD: Single vessel disease; 2VD: Two vessel disease; 3VD: Three vessel disease; RCA: Right coronary artery; RV: Right ventricle; SD: Standard deviation; LVEF: Left ventricular ejection fraction

In the second follow-up (6 months after surgery), the number of cases with RV dysfunction decreased based on all three indices: TAPSE < 1.6 cm was observed in 49.0% of patients, TI > 55% in 49.0%, and peak Sm < 10 cm/s in 37.0% of patients. The size of IVC and SM was reported normal in all patients (Table 2).

Table 2. Frequency of right ventricle (RV) dysfunction according to echocardiographic indices at baseline, one week, and 6 months after surgery

Variable	Frequency [n (%)]		
	Baseline (n = 61)	One week after CABG (n = 60)*	6 months after CABG (n = 59)**
Abnormal TAPSE	0	48 (80.0)	28 (47.4)
Abnormal peak systolic movement	0	36 (58.0)	21 (35.5)
Abnormal Tei index	0	46 (76.6)	28 (47.4)
Abnormal IVC	0	0	0
Abnormal septal motion	0	60 (100)	59 (100)

* One patient died one week after surgery; ** One patient lost to follow up

CABG: Coronary artery bypass grafting; TAPSE: Tricuspid annular plane systolic excursion; IVC: Inferior vena cava

Table 3. Comparison of the mean of right ventricle (RV) function indices during the study period

Variable	Amount				
	Before		One week after CABG		6 months after CABG
	Mean ± SD	P*	Mean ± SD	P**	Mean ± SD
TAPSE (cm)	2.50 ± 0.30	0.001	1.30 ± 0.20	0.001	1.50 ± 0.20
Peak systolic movement (cm/s)	13.60 ± 2.20	0.001	9.10 ± 1.30	0.001	9.90 ± 1.50
Tei index	30.70 ± 7.50	0.001	64.90 ± 11.80	0.002	57.30 ± 15.10
IVC size (cm)	1.11 ± 0.21	0.002	1.16 ± 0.25	0.001	1.19 ± 0.23

* Comparison of the mean of right ventricle (RV) function indices between before surgery and one week after surgery using paired t-test; ** Comparison of the mean of RV function indices between one week after surgery and 6 months after surgery using paired t-test

CABG: Coronary artery bypass grafting; SD: Standard deviation; TAPSE: Tricuspid annular plane systolic excursion; IVC: Inferior vena cava

Paired comparison was conducted to compare the mean RV function indices before surgery and one week after surgery and also one week and six months after surgery, using paired t-test. As shown in table 3, the mean of TAPSE significantly decreased from 2.25 ± 0.30 cm before surgery to 1.30 ± 0.20 cm one week after surgery (P < 0.001), and then significantly increased to 1.50 ± 0.20 cm six months after surgery (P < 0.001).

The mean TI increased from 30.7 ± 7.5 percent before surgery to 64.9 ± 11.8 percent one week after surgery (P < 0.001) and decreased to 57.3 ± 15.1 percent six months after surgery (P < 0.002). The mean of peak Sm index decreased from 13.6 ± 2.2 cm/s to 9.1 ± 1.3 cm/s one week after surgery (P < 0.001) and then significantly increased to 9.1 ± 1.3 cm/s six months after surgery (P < 0.001). The result of repeated measures ANOVA showed that the mean of RV function indices was significantly different during the time of study: TAPSE (P = 0.001), TI (P = 0.001), peak Sm (P = 0.001), and IVC (P = 0.001).

To determine the effective factors on RV dysfunction after 6 months, we conducted logistic regression analysis. Independent variables were demographic variables, angiography results, DM, and HTN. Dependent variables were RV function indices as binominal (normal or abnormal). Logistic regression analysis for TAPSE index showed that only age was significant effective factor [odds ratio (OR) = 0.88, 95% confidence interval (CI): 0.78-0.98, P = 0.020]. For TI, two factors including age and DM were effective [(OR = 0.87, 95% CI: 0.70-0.97, P = 0.010) and (OR = 6.70, 95% CI: 0.45-14.30, P = 0.030), respectively]. There were no significant effective factors on peak Sm index in 6 months after surgery. IVC size was normal during study period but the mean of IVC size significantly increased. Hence, we used linear regression analysis to determine effective factors on IVC size in second follow-up. The result of linear regression analysis showed that only history of HTN was significant effective factor (b = 0.1, β = 0.2, 95% CI: 0.01-0.28, P = 0.040).

6-MWT was conducted for all study patients 6 month after surgery. The mean of 6-MWT result was 326 ± 50 m (minimum = 255 m, maximum = 450 m). As shown in table 4, mean distance paced in 6-MWT in patients with RV dysfunction according to RV function indices was significantly lower than those without RV dysfunction (P < 0.001).

Table 4. Comparison of mean distance paced in six-minute walk test (6-MWT) in study patients with/without right ventricle (RV) dysfunction based on three indices

Variable	Mean ± SD	P*
TAPSE < 1.6 (cm)	298.89 ± 42.80	0.001
TAPSE > 1.6 (cm)	351.21 ± 46.30	
Tei index > 55%	301.67 ± 43.10	0.001
Tei index < 55%	348.62 ± 48.80	
Peak systolic movement < 10 (cm/s)	296.43 ± 37.60	0.001
Peak systolic movement > 10 (cm/s)	343.71 ± 50.90	

* Independent t-test

SD: Standard deviation; TAPSE: Tricuspid annular plane systolic excursion

Based on linear regression analysis, age (b = -3.1, β = -0.5, P = 0.001), number of involved vessels (b = -33.5, β = -0.3, P = 0.006), graft number (b = -20.3, β = -0.3, P = 0.010), RV dysfunction based on TAPSE six months after surgery (b = 52.3, β = 0.5, P = 0.001), peak Sm six months after surgery (b = 47.2, β = 0.4, P = 0.010), and TI six months after surgery (b = 46.9, β = 0.3, P = 0.001) were significant effective factors on 6-MWT. Results of multiple regression analysis are shown in table 5. As showed in this table, older age, higher number of involved vessels, and severity of RV dysfunction according to TABSE, TI, and peak Sm deteriorated 6-MWT.

Table 5. Predictive factors for six-minute walk test (6MWT) by multiple linear regression analysis

Variable	b	SE	95% CI	P*
Age	-2.9	0.67	-4.30-1.60	0.001
Number of involved vessels	-34.9	8.80	-52.60-17.90	0.001
TI	-1.3	0.33	-1.90-0.63	0.001

* Multiple regression analysis
TI: Tei index; SE: Standard error; CI: Confidence interval

Discussion

This study evaluated changes in RV function and IVC size after CABG that have been considered by some investigators. The findings indicated that in patients with normal RV indices preoperatively, one week (abnormal TAPSE 81.0%, TI 79.0%, and peak Sm 62.0%) and 6 months after surgery (abnormal TAPSE 49.0%, TI 49.0%, and peak Sm 37.0%), all indices showed significant decrement. Mean distance paced in 6-MWT in patients was significantly less in patients with RV dysfunction, older age, multivessel disease, and higher graft number.

Similarly, previous studies evaluated the echocardiographic indices of RV dysfunction in patients after CABG, but the results were controversial. RV function had significant influence on patients' outcome in several studies.[21] Ojaghi et al. evaluated echocardiographic indices in 30 Iranian patients and have reported reduced TAPSE and peak Sm velocity one week after CABG that remained after one month, while TI increased significantly.[20] In our study, RV dysfunction remained even up to 6 months postoperatively. Alam et al. have also evaluated RV function by tricuspid annular velocity and demonstrated reduced function 1 and 3 month(s) after CABG.[13] Hedman et al. demonstrated that RV function remained depressed one year after CABG and suggested permanent changes in the majority of patients after CABG,[16] findings that are contrary to our results. Alam et al.[13] reported that RV dysfunction partially recovered after 1 year, and Pegg et al. reported complete recovery after 6 months.[14]

One noticeable observation in our experience was the distance paced in 6-MWT that decreased in patients with RV dysfunction, while Hedman et al. reported improved exercise performance 3 months after CABG, despite decreased RV function.[16] These discrepancies in the results of the studies can be attributable to the differences in demographic characteristics of patients, as older age and other parameters as higher number of involved vessels and grafts that had deleterious effect on 6-MWT result.

There is an increased risk of coronary artery disease (CAD) in subjects with risk factors including HTN, obesity, dyslipidemia, and smoking.[22,23] We considered these risks in the present study and the results showed that patients undergoing CABG had a high prevalence of HTN and DM and were smoker and overweight (67.2%, 45.9%, 29.5%, and 68.0%, respectively). More analysis proved that two of the indices (TAPSE and peak Sm) were not associated with major risk factors, while TI one week after surgery was more impaired in patients with DM.

Off-pump coronary artery bypass (CAB) represents an acceptable alternative to conventional on-pump surgery[24] and has been reported to be associated with reduced oxidative stress[25] and less pro-inflammatory cytokine expression compared to conventional CABG.[26] On cellular level, systemic inflammatory response due to the release of pro-inflammatory cytokines is a recognized feature of CPB.[27] Diller et al. reported a non-significant trend that off-pump surgery might be associated with less RV damage.[12] Larger studies may be required to investigate whether off-pump CABG is associated with less RV damage compared to on-pump surgery.

The present study had several strengths, including assessment of all three indices of 2D echocardiography, IVC size, and exercise test together that could give a bright view to researchers and physicians to speculate the effects of CABG on all RV parameters. Limitation of this study is lack of control group. Moreover, although the sample size was calculated based on previous studies, only 58 patients completed the 6-month follow-up who were selected from one center that decreased the credibility of the results. Multicenter studies with larger sample size can add to our knowledge.

In conclusion, there was a significant reduction in RV function, increase in IVC size, and impairment of exercise test in patients with RV dysfunction six months after CABG. Peak Sm index one week after surgery was associated with its value prior to surgery and number of diseased arteries, and TI one week after surgery was affected by history of DM and TI before surgery and was more impaired in older patients, while deteriorated 6-MWT was observed in patients with RV dysfunction, older age, higher numbers of involved vessels, and higher grafts.

Conclusion

The significant reduction in RV function and impairment of exercise capacity after CABG in this

study suggest cardiologists to pay more attention to RV assessment in follow-up visits of patients undergoing GABG.

Acknowledgments

We wish to thank the Dr. Heshmat Hospital registry system for cooperation and also Vice Chancellor for Research of Guilan University of Medical Sciences for financial support (Research number: 94040214).

Conflict of Interests

Authors have no conflict of interests.

References

1. Jemal A, Ward E, Hao Y, Thun M. Trends in the leading causes of death in the United States, 1970-2002. JAMA 2005; 294(10): 1255-9.
2. Jousilahti P, Laatikainen T, Salomaa V, Pietila A, Vartiainen E, Puska P. 40-year CHD mortality trends and the role of risk factors in mortality decline: the North Karelia project experience. Global heart. 2016; 11(2): 207-12.
3. Wijns W, Kolh P, Danchin N, Di Mario C, Falk V, Folliguet T, et al. Guidelines on myocardial revascularization. Eur Heart J 2010; 31(20): 2501-55.
4. Li Z, Kravitz RL, Marcin JP, Romano PS, Rocke DM, Denton TA, et al. Survival enhancing indications for coronary artery bypass graft surgery in California. BMC Health Serv Res 2008; 8: 257.
5. Passaloglou IT, Sabashnikov A, Zeriouh M, Reutter S, Fatullayev J, Choi YH, et al. Rapid diagnostics and treatment of early complications after CABG surgery: A life saver. Heart Surg Forum 2013; 16(6): E346-E350.
6. Rosner A, Avenarius D, Malm S, Iqbal A, Schirmer H, Bijnens B, et al. Changes in right ventricular shape and deformation following coronary artery bypass surgery-insights from echocardiography with strain rate and magnetic resonance imaging. Echocardiography 2015; 32(12): 1809-20.
7. Pouleur AC, Rousseau MF, Ahn SA, Amzulescu M, Demeure F, de Meester C, et al. Right ventricular systolic dysfunction assessed by cardiac magnetic resonance is a strong predictor of cardiovascular death after coronary bypass grafting. Ann Thorac Surg 2016; 101(6): 2176-84.
8. Itagaki S, Hosseinian L, Varghese R. Right ventricular failure after cardiac surgery: Management strategies. Semin Thorac Cardiovasc Surg 2012; 24(3): 188-94.
9. Siddiqui MM, Jalal A, Sherwani M, Ahmad MZ. Right ventricular dysfunction after coronary artery bypass grafting is a reality of unknown cause and significance. Heart Surg Forum 2012; 15(4): E185-E188.
10. Daly RC, Chandrasekaran K, Cavarocchi NC, Tajik AJ, Schaff HV. Ischemia of the interventricular septum. A mechanism of right ventricular failure during mechanical left ventricular assist. J Thorac Cardiovasc Surg 1992; 103(6): 1186-91.
11. Joshi SB, Salah AK, Mendoza DD, Goldstein SA, Fuisz AR, Lindsay J. Mechanism of paradoxical ventricular septal motion after coronary artery bypass grafting. Am J Cardiol 2009; 103(2): 212-5.
12. Diller GP, Wasan BS, Kyriacou A, Patel N, Casula RP, Athanasiou T, et al. Effect of coronary artery bypass surgery on myocardial function as assessed by tissue Doppler echocardiography. Eur J Cardiothorac Surg 2008; 34(5): 995-9.
13. Alam M, Hedman A, Nordlander R, Samad B. Right ventricular function before and after an uncomplicated coronary artery bypass graft as assessed by pulsed wave Doppler tissue imaging of the tricuspid annulus. Am Heart J 2003; 146(3): 520-6.
14. Pegg TJ, Selvanayagam JB, Karamitsos TD, Arnold RJ, Francis JM, Neubauer S, et al. Effects of off-pump versus on-pump coronary artery bypass grafting on early and late right ventricular function. Circulation 2008; 117(17): 2202-10.
15. Joshi SB, Roswell RO, Salah AK, Zeman PR, Corso PJ, Lindsay J, et al. Right ventricular function after coronary artery bypass graft surgery a magnetic resonance imaging study. Cardiovasc Revasc Med 2010; 11(2): 98-100.
16. Hedman A, Alam M, Zuber E, Nordlander R, Samad BA. Decreased right ventricular function after coronary artery bypass grafting and its relation to exercise capacity: A tricuspid annular motion-based study. J Am Soc Echocardiogr 2004; 17(2): 126-31.
17. Lella LK, Sales VL, Goldsmith Y, Chan J, Iskandir M, Gulkarov I, et al. Reduced right ventricular function predicts long-term cardiac re-hospitalization after cardiac surgery. PLoS One 2015; 10(7): e0132808.
18. Mann DL, Zipes DP, Libby P, Bonow RO, Braunwald E. Braunwald's heart disease: A textbook of cardiovascular medicine. Philadelphia, PA: Elsevier/Saunders; 2015.
19. Thygesen K, Alpert JS, White HD. Universal definition of myocardial infarction. Eur Heart J 2007; 28(20): 2525-38.
20. Ojaghi Z, Moaref A, Nouhi F, Maleki M, Mohebi A. Evaluation of right ventricular function after coronary artery bypass grafting. Iran Heart J 2007; 8(1): 13-9.
21. Maslow AD, Regan MM, Panzica P, Heindel S, Mashikian J, Comunale ME. Precardiopulmonary bypass right ventricular function is associated with poor outcome after coronary artery bypass grafting in patients with severe left ventricular systolic dysfunction. Anesth Analg 2002; 95(6): 1507-18, table.

22. Voelkel NF, Quaife RA, Leinwand LA, Barst RJ, McGoon MD, Meldrum DR, et al. Right ventricular function and failure: Report of a National Heart, Lung, and Blood Institute working group on cellular and molecular mechanisms of right heart failure. Circulation 2006; 114(17): 1883-91.
23. Ryden L, Standl E, Bartnik M, Van den Berghe G, Betteridge J, de Boer MJ, et al. Guidelines on diabetes, pre-diabetes, and cardiovascular diseases: Executive summary. The Task Force on Diabetes and Cardiovascular Diseases of the European Society of Cardiology (ESC) and of the European Association for the Study of Diabetes (EASD). Eur Heart J 2007; 28(1): 88-136.
24. El-Hamamsy I, Cartier R, Demers P, Bouchard D, Pellerin M. Long-term results after systematic off-pump coronary artery bypass graft surgery in 1000 consecutive patients. Circulation 2006; 114(1 Suppl): I486-I491.
25. Gonenc A, Hacisevki A, Bakkaloglu B, Soyagir A, Torun M, Karagoz H, et al. Oxidative stress is decreased in off-pump versus on-pump coronary artery surgery. J Biochem Mol Biol 2006; 39(4): 377-82.
26. Gasz B, Lenard L, Racz B, Benko L, Borsiczky B, Cserepes B, et al. Effect of cardiopulmonary bypass on cytokine network and myocardial cytokine production. Clin Cardiol 2006; 29(7): 311-5.
27. Davidson MJ. Can the off-pump coronary artery bypass debate shed light on postoperative right heart dysfunction? Circulation 2008; 117(17): 2181-3.

DOI: http://dx.doi.org/10.22122/arya.v15i3.1843 Published by Vesnu Publications

Association between ambient air pollution and hospitalization caused by atrial fibrillation

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Original Article

Abstract

BACKGROUND: Many studies have shown the worst effects of air pollution on cardiovascular diseases (CVDs). Present study focused on the relationship between atrial fibrillation (AF), as one of the common arrhythmias, and air pollutants in Isfahan, Iran, an industrial city in the Middle East.

METHODS: A case-crossover design was used to explore the associations between air pollution and AF hospitalized patients with ventricular response (VR) > 90 beats per minute (bpm) (fast response) and those with VR ≤ 90 bpm. All patients' records were extracted from their hospital files. Air pollutants data including particulate matter less than 10 μ (PM_{10}), $PM_{2.5}$, carbon monoxide (CO), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and ozone (O_3) were obtained from the Correlation of Air Pollution with Hospitalization and Mortality of Cardiovascular and Respiratory Diseases (CAPACITY) study. Conditional logistic regression test was used to measure the relationship between pollutants and hospitalization due to AF.

RESULTS: Records of 369 patients, including 173 men (46.9%) who were hospitalized for AF during the study period and had complete data were extracted. Although a positive but not statistically significant relationship was shown between 10-unit increases in all pollutants (except PM_{10}) and the hospitalization due to AF in patients with rapid VR (RVR), the only significant relationship was observed in case of NO_2 [odds ratio (OR) = 1.26, 95% confidence interval (CI) = 1.0-2.1, P = 0.031].

CONCLUSION: This study showed positive significant relationships between NO_2 and the hospitalization due to AF in patients with RVR. NO_2 is a greenhouse gas whose levels are expected to increase due to global environmental changes. Therefore, relevant strategies should be adopted to decrease its levels, especially in industrial cities like Isfahan.

Keywords: Air Pollution, Atrial Fibrillation, Nitrogen Dioxide

Date of submission: 07 Aug. 2018, ***Date of acceptance:*** 10 Mar. 2019

Introduction

Recent studies by the World Health Organization (WHO) have shown that more than 90% of the world's population is exposed to air pollution. Ambient air pollution was responsible for about

How to cite this article: Saifipour A, Azhari A, Pourmoghaddas A, Hosseini SM, Jafari-Koshki T, Rahimi M, et al. **Association between ambient air pollution and hospitalization caused by atrial fibrillation.** ARYA Atheroscler 2019; 15(3): 106-12.

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four million deaths, mainly due to cardiovascular diseases (CVDs) in 2017. In addition to an increase in death rate due to CVD, air pollution is associated with increased complications and duration of hospitalization in patients suffering from heart problems. Hypertension (HTN),[1-3] cardiac arrhythmias,[4,5] heart failure (HF) deterioration, and acute ischemic and atherosclerotic cardiovascular complications are among the short-term consequences of air pollution.[6] Exposure to airborne contaminants causes a variety of cardiac arrhythmias, including atrial fibrillation (AF).[7] Independent relationships have been reported between death due to AF and concentrations of contaminants on the day before death.[8] Cardiac autonomic dysfunction caused by air pollution leads to various complications including AF.[9] In addition, air pollution can cause inflammation, oxidative stress, changes in atrial pressure, and ultimately AF.[10]

AF is the most widespread treatable cardiac arrhythmia.[11] It is associated with increased risk of stroke,[12] myocardial infarction (MI),[13] HF,[14] dementia,[15] and chronic kidney disease (CKD).[15] This prevalent arrhythmia doubles the risk of mortality following acute coronary disease and stroke.[16,17] There is an association between the incidence of AF and age, i.e., the prevalence of this arrhythmia varies from 1% in people younger than 60 years to 9% in those over 80 years of age. In Iran, the prevalence of AF in individuals over 70 years of age has been reported as high as 6%.[18] The prevalence of this disorder may even reach 14% in patients with stroke.[19]

The prevalence of AF in Iranians over 50 years of age has been estimated as 3%.[20,21] Considering the prevalence of AF and its potential irreversible complications, identification of its risk factors, determination of risk factor exposure, and planning of risk factor control policies can be a priority in the management of irrelevant diseases. This study investigated the relationship between AF and different air pollutants as part of a study on "The Correlation of Air Pollution with Hospitalization and Mortality of Cardiovascular and Respiratory Diseases (CAPACITY)" conducted in Isfahan, Iran, from March 20, 2010 to March 20, 2012.[22]

Increased ambient air pollution was associated with increased risk of episodes of rapid ventricular response (RVR) and AF, and we analyzed this association using a case–crossover design.

Materials and Methods

This case-crossover study was part of the CAPACITY research which evaluated the relationship between hospitalization and death from cardiovascular and respiratory diseases and airborne contaminants during March 20, 2010 to March 20, 2012. Data about all cases of hospitalization and/or death due to cardiovascular and respiratory diseases in Isfahan City during the mentioned period were collected from the hospitals and cemetery of Isfahan. Data on the concentrations of gaseous and non-gaseous pollutants including particulate matter < 10 μ and < 2.5 μ (PM_{10} and $PM_{2.5}$, respectively), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), carbon monoxide (CO), and ozone (O_3) were collected from fixed air monitoring stations in the city of Isfahan. The CAPACITY study used two time-series and case-crossover models to assess the relationships between cardiovascular and respiratory diseases and air pollutants.[22]

The present study extracted the names and characteristics of all patients who were diagnosed with AF [I48 in the International Classification of Diseases, 10th revision (ICD-10)] and admitted to Isfahan hospitals during March 20, 2010 to March 20, 2012 from the CAPACITY study. The causes of hospitalization were determined by the hospital physician but were re-examined in this study. The patients' additional information, including symptoms at the time of admission, history of diabetes, and history of HTN, along with ejection fraction (EF) in the echocardiography performed during hospitalization, were extracted from the relevant hospitals. Patients with incomplete data were excluded from the study.

Air pollutant data including PM_{10}, $PM_{2.5}$, CO, SO_2, NO_2, and O_3, as well as weather information, including temperature, humidity, and wind speed were collected from the CAPACITY study (obtained from the Department of Environment, Isfahan Province). This information was collected from six fixed air monitoring stations installed in different areas of Isfahan recording the hourly concentrations of each pollutant. The CAPACITY study collected data on temperature, humidity, and wind speed from Isfahan Weather Forecast Organization and the National Oceanic and Atmospheric Administration (NOAA) National Environmental Satellite, Data, and Information Service (http://www.nesdis.noaa.gov). All data were evaluated in the CAPACITY study and the outliers and missing points were determined. The average daily values of weather pollutants were merged into a single file.

A case-crossover design allowed for investigation of the acute effects of exposure to air

pollution. The subject exposure before the time of the event (case period) was compared with the distribution of exposure estimated from separate control periods and the matched sets were analyzed using conditional logistic regression. Thus, each patient served as his or her own control.

Case periods were defined by the time prior to onset of each confirmed arrhythmic event. Daily pollution concentrations and weather conditions were then matched to the case and control time periods for analysis.

The effect of 10-unit increase of air pollutants on hospitalization with heart rate more than 90 beats per minute (bpm) was determined using conditional logistic regression and crude and adjusted odds ratios (ORs) by temperature (˚f), dew point (˚f), and wind speed (mile/h).

The patients were divided in two groups: VR rate > 90 bpm and ≤ 90 bpm. Qualitative variables were expressed as number and percentage and for comparison between them, chi-square test or Fisher's exact test were used. Quantitative variables were expressed as mean ± standard deviation (SD) and compared with independent t-test or Mann-Whitney U test (if normality assumption was not hold). Kolmogorov-Smirnov test (K-S test) was used to determine whether normality assumption existed.

Statistical analysis was conducted using Stata software (version 9, Stata Corporation, College Station, TX, USA). P-value less than 0.050 was considered statistically significant.

Results

A total of 451 patients with AF were hospitalized in seven educational hospitals affiliated to Isfahan University of Medical Sciences, Isfahan City, during the study period. However, only 369 patients, including 173 men (46.9%), had complete data and were entered into the study. The patients' mean age was 66.3 ± 15.9 years. The patients visited the hospital mainly due to chest pain (n = 61, 16.0%), dyspnea (n = 41, 10.7%), dyspnea on exertion (DOE) (n = 81, 21.2%), dizziness (n = 19, 5.0%), and tachycardia (n = 17, 4.5%). The frequency of these complaints did not differ significantly between the two groups. Table 1 shows the history of diseases, symptoms, and results of echocardiography in all patients and the two groups.

Table 1. Demographic characteristics of hospital admissions for atrial fibrillation (AF) in Isfahan, Iran, 2010-2012

Variable	All subjects (n = 369)	Subjects with PR ≤ 90 bpm (n = 108, 29.3%)	Subjects with PR > 90 bpm (n = 261, 70.7%)	P
	Total	PR ≤ 90	PR > 90	
Age (year)	66.31 ± 15.88	65.55 ± 15.06	66.56 ± 16.24	0.657**
SBP (mmHg)	130.30 ± 27.78	129.17 ± 29.69	130.58 ± 27.25	0.693**
DBP (mmHg)	78.90 ± 13.42	77.41 ± 13.75	79.43 ± 13.32	0.227†
Number of pulses (bpm)*	117.80 ± 33.05	75.78 ± 51.50	130.91 ± 26.19	< 0.001**
EF value	50.70 ± 15.10	48.69 ± 15.27	51.27 ± 15.06	0.278**
Hb concentration	13.41 ± 2.60	13.16 ± 2.92	13.48 ± 2.53	0.443**
Creatinine (mg/dl)	1.22 ± 0.49	1.20 ± 0.40	1.23 ± 0.51	0.560†
Arterial oxygen saturation	90.71 ± 8.76	88.75 ± 5.12	90.90 ± 9.16	0.221†
History of VHD	87 (23.6)	23 (21.3)	64 (24.5)	0.539*
Medicines received in the hospital				
- Beta blocker	105 (28.5)	27 (25.0)	78 (29.9)	0.596*
- Statin	53 (14.4)	17 (15.7)	36 (13.8)	0.118*
- Warfarin	67 (18.2)	22 (20.4)	45 (17.2)	0.056*
Male gender	173 (46.9)	39 (36.1)	134 (51.3)	0.581*
History of diabetes	86 (23.3)	23 (21.3)	63 (24.1)	0.472*
History of HTN	148 (40.1)	33 (30.5)	115 (44.1)	0.567*
Status (death)	13 (3.5)	2 (1.8)	11 (4.2)	0.529*

Values are presented as mean ± standard deviation (SD) or median [interquartile range (IQR)] for quantitative variables and number and percentage for qualitative variables.

* Chi-square or Fisher's exact test were used where appropriate.

** Two independent samples t-test was used.

† Mann-Whitney U test was used.

PR: Pulse rate; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; EF: Ejection fraction; Hb: Hemoglobin; Bpm: Beats per minute; VHD: Valvular heart disease; HTN: Hypertension

Table 2. The mean daily concentrations of pollutants in Isfahan, Iran, during the study period (March 20, 2010 to March 20, 2012)

Variable	Mean ± SD	Minimum	Maximum
O_3 (ppb)	29.09 ± 15.18	3.00	101.0
CO (ppm)	3.93 ± 2.46	0.00	16.0
SO_2 (ppb)	41.33 ± 35.04	0.00	147.5
NO_2 (ppb)	40.53 ± 23.11	0.01	128.0
PM_{10} (µg/m^3)	129.86 ± 58.85	36.40	528.3
$PM_{2.5}$ (µg/m^3)	54.14 ± 29.61	8.00	296.1
Temperature (˚f)	55.77 ± 18.58	27.80	92.2
Dew point (˚f)	27.42 ± 9.23	7.30	46.6
Wind speed (mile/h)	4.82 ± 2.52	0.80	15.5

Ppb: Particle per billion; Ppm: Part per million; O_3: Ozone; CO: Monoxide carbon; SO_2: Sulfur dioxide; NO_2: Nitrogen dioxide; PM: Particulate matter; SD: Standard deviation

As seen in table 1, the two groups (VR ≤ 90 and VR > 90 bmp) had no significant differences except in the number of pulses. This difference was expected due to the classification of patients based on pulse rate (PR). Table 2 presents the mean concentrations of pollutants and weather factors during the study period.

Table 3 presents the details of the effect of air pollution on hospitalization due to AF using a crude and adjusted model for temperature (˚f), dew point (%), and wind speed (mile/h).

As seen, the only significant relationship was observed between a 10-unit increase in NO_2 and the hospitalization due to AF. However, 10-unit increases in other pollutants, except PM_{10}, had positive but not statistically significant associations with the hospitalization due to AF and RVR (VR > 90 bpm).

Discussion

The direct association between NO_2 concentration and rate of hospitalization due to AF was the only significant relationship in this study. Although increments in other pollutants increased the frequency of hospitalization due to AF, these relationships were not significant. Various studies have investigated the effects of pollutants on the incidence of cardiac arrhythmias, including AF, around the world. A cohort study followed patients with implantable cardioverter-defibrillator (ICD) for three years and found 798 cases of ventricular fibrillation (VF). The incidence of these cases was related with $PM_{2.5}$ and O_3.[5] Another cohort study reported a significant relationship between ventricular arrhythmias and SO_2 concentration. It evaluated 56 patients and a total of 139 VF events and found increased NO_2 24 hours before the incidence of arrhythmia. However, this increase had no significant associations with arrhythmias.[23]

A cohort study extracted electrocardiographic (ECG) changes from the records of patients with ICD and identified significant relationships between the incidence of AF and exposure to PM, CO, and NO_2, especially two hours before AF. Overall, 124 AF events occurred in individuals who had no previous AF records and the incidence of the arrhythmia was strongly associated with the mentioned contaminants, especially NO_2.[24]

Table 3. The effects of a 10-unit increase in pollutants at 24 hours before the onset of atrial fibrillation (AF) in patients with rapid ventricular response (RVR)

Pollutants	Crude			Adjusted*		
	OR	95% CI	P	OR	95% CI	P
$PM_{2.5}$	1.017	0.950-1.089	0.612	1.019	0.951-1.092	0.579
PM_{10}	0.992	0.930-1.059	0.830	0.994	0.931-1.062	0.879
SO_2	1.017	0.849-1.218	0.854	1.014	0.846-1.215	0.878
CO**	1.064	0.864-1.310	0.557	1.062	0.862-1.309	0.570
NO_2	1.260	1.024-1.551	0.029	1.259	1.020-1.553	0.031
O_3	1.082	0.763-1.533	0.657	1.083	0.760-1.544	0.655

* Adjusted for temperature (˚f), dew point(%), and wind speed (mile/h)

** For CO effect, 1-unit increase was conducted

OR: Odds ratio; CI: Confidence interval; PM: Particulate matter; SO_2: Sulfur dioxide; CO: Monoxide carbon; NO_2: Nitrogen dioxide; O_3: Ozone

A two-year time-series study on emergency room (ER) visits in Brazil revealed associations between arrhythmias and air contaminants. The study designed a model based on generalized linear Poisson regression with seasonal adjustment to investigate the relationship between contaminants and the incidence of arrhythmias. The results showed that atrial arrhythmias, including flutter and fibrillation, and other arrhythmias were associated with increases in CO, NO_2, and PM_{10} concentrations within the quartiles.[25]

A 14-year time-series study in Rome, Italy, investigated the relationship between the frequency of patients with AF referring to ER and air pollutants (PM_{10}, $PM_{2.5}$, NO_2) at different time intervals. Models based on two pollutants and adjusted for temperature, humidity, individual characteristics, and consumed drugs were also developed. During the study, nearly 80000 people were admitted to ERs due to AF. The study showed an association between hospitalization due to AF and exposure to PM 24 hours before the incidence of arrhythmia. Hospitalization due to AF had the strongest relationships with PM in men over 75 years of age and with NO_2 in women over 75 years of age. It was also associated with exposure to NO_2, and more strongly with $PM_{2.5}$ 24 hours before the incidence in all ages.[26]

In a large case-crossover study in the United Kingdom (UK), patient registration programs, e.g., the Myocardial Ischaemia National Audit Project (MINAP), were used to extract the data of around 2 million patients admitted to ERs due to CVD during 2003-2008. Data about the mean daily concentrations of CO, PM_{10}, $PM_{2.5}$, SO_2, and NO_2, as well as the maximum 8-hour concentrations of O_3 per day, were extracted from air monitoring stations with minimum distance from each individual patient. The designed model was examined based on different time intervals and up to four days before the event. The strongest relationship was observed between NO_2 levels and hospitalization due to different cardiovascular causes, including AF. In addition, the hospitalization of patients after non-ST-segment elevation MI (NSTEMI) had significant relationships with SO_2 and NO_2 during the four-day period before the incidence. The effects of NO_2 were more profound in subjects over 70 years old and also in women.[27]

NO_2 is produced from diesel fuel and secondary reactions of NO with O_3. It is usually related with other pollutants and can exacerbate their effects. NO_2 per se can cause autonomic system dysfunction and increase the risk of AF.[26,28]

It should be borne in mind that although AF is a major risk factor for stroke and MI, it is often asymptomatic and undetectable. Consequently, the treatment may not be initiated until a complication occurs. Therefore, based on the results of this study, it is preferable to control the risk factors of AF, e.g., airborne contaminants, especially NO_2. Furthermore, in addition to pollutant reduction policies, screening methods to diagnose and treat AF should be adopted in the country. A combination of these measures and control strategies, especially on polluted days, will reduce the hospitalization due to both AF and its complications.

Another important point is the rapid change in the AF process and the development of symptoms, due to which studies on the effects of pollutants on the hospitalization due to AF should consider the few hours before hospitalization as the exposure time.[24] Based on the results of other studies, obtaining reliable results regarding the effects of environmental factors, such as pollutants, on the incidence of AF requires larger research, cohort studies, using large databases such as health information system (HIS) and Iranian electronic health record (SEPAS) in Iran, updating the available databases, and recording patients' full information (e.g., history of hospitalization, diseases, and medication use) and changes in their home and work addresses.

Another finding of this study was that the mean annual concentrations of NO_2, PM_{10}, $PM_{2.5}$, and SO_2 within the study period were almost twice the Iranian and global standards. High levels of pollutants can be attributed to the presence of various industries using fossil fuels, as well as the increased number of vehicles, in particular diesel cars, followed by elevated use of fossil fuels in the city of Isfahan. Moreover, due to sanctions in 2011, Iran could not import gasoline and had to use the gasoline produced in the country which did not have Euro 4 standard according to some sources. It is, hence, critical to pay attention to high levels of pollutants and develop relevant measures to control them.

Another notable result of this study was the high frequency of women hospitalized for AF in Isfahan. Similar to our findings, Habibzadeh et al. studied patients referred to a first-level health center and showed a higher frequency of women among the 463 50-80-year-old patients with AF.[20] In contrast, Schnabel et al. reviewed a 50-year record of patients admitted for AF and reported a higher incidence of this arrhythmia in men.[29] In the Gulf Survey of

Atrial Fibrillation Events (Gulf SAFE) on 2043 patients with AF visiting ERs, men had a higher overall frequency, but the frequency of women was higher in patients with comorbidities and resistant AF.[30] Accordingly, it seems necessary to assess gender distribution of Iranian patients with AF, especially those at a high risk of complications.

Conclusion

The greater frequency of women among patients with AF, higher concentrations of pollutants compared to global standards, and a relatively strong significant relationship between NO_2, as an important pollutant, and the hospitalization due to AF or development of its symptoms were the most important findings of this study. Therefore, relevant strategies should be adopted to identify patients and examine the reasons for the higher rate of hospitalization due to AF in women. It is also essential to develop effective measures to decrease the levels of pollutants (especially NO_2) and precursor pollutants (e.g., O_3). Timely AF control methods should also be used before the development of complications on highly-polluted days. In addition, due to the possibility of quick effects of pollutants on the hospitalization due to AF, further studies are required to design models assessing very short exposure times before the hospitalization due to AF symptoms. Models should also consider combinations of pollutants in order to study the impacts of precursor pollutants on other contaminants.

Acknowledgments

The authors wish to express their gratitude to the Isfahan Cardiovascular Research Institute, the Disaster Management Office, Provincial Governor's Office, and Department of Environment of Isfahan for their support. This study has been derived from a residence of cardiology dissertation approved by School of Medicine, Isfahan University of Medical Sciences.

Conflict of Interests

Authors have no conflict of interests.

References

1. Zanobetti A, Canner MJ, Stone PH, Schwartz J, Sher D, Eagan-Bengston E, et al. Ambient pollution and blood pressure in cardiac rehabilitation patients. Circulation 2004; 110(15): 2184-9.
2. Dvonch JT, Kannan S, Schulz AJ, Keeler GJ, Mentz G, House J, et al. Acute effects of ambient particulate matter on blood pressure: Differential effects across urban communities. Hypertension 2009; 53(5): 853-9.
3. Urch B, Silverman F, Corey P, Brook JR, Lukic KZ, Rajagopalan S, et al. Acute blood pressure responses in healthy adults during controlled air pollution exposures. Environ Health Perspect 2005; 113(8): 1052-5.
4. Peters A, Liu E, Verrier RL, Schwartz J, Gold DR, Mittleman M, et al. Air pollution and incidence of cardiac arrhythmia. Epidemiology 2000; 11(1): 11-7.
5. Rich DQ, Schwartz J, Mittleman MA, Link M, Luttmann-Gibson H, Catalano PJ, et al. Association of short-term ambient air pollution concentrations and ventricular arrhythmias. Am J Epidemiol 2005; 161(12): 1123-32.
6. Franchini M, Mannucci PM. Air pollution and cardiovascular disease. Thromb Res 2012; 129(3): 230-4.
7. Hoek G, Brunekreef B, Fischer P, van Wijnen J. The association between air pollution and heart failure, arrhythmia, embolism, thrombosis, and other cardiovascular causes of death in a time series study. Epidemiology 2001; 12(3): 355-7.
8. Dominici F, McDermott A, Daniels M, Zeger SL, Samet JM. Mortality among residents of 90 cities. In: Dominici F, McDermott A, Daniels M, Zeger SL, Samet JM, Editors. Revised analyses of time-series studies of air pollution and health. Boston, MA: Health Effects Institute; 2003. p. 9-24.
9. Link MS, Dockery DW. Air pollution and the triggering of cardiac arrhythmias. Curr Opin Cardiol 2010; 25(1): 16-22.
10. Aviles RJ, Martin DO, Apperson-Hansen C, Houghtaling PL, Rautaharju P, Kronmal RA, et al. Inflammation as a risk factor for atrial fibrillation. Circulation 2003; 108(24): 3006-10.
11. Piccini JP, Hammill BG, Sinner MF, Jensen PN, Hernandez AF, Heckbert SR, et al. Incidence and prevalence of atrial fibrillation and associated mortality among Medicare beneficiaries, 1993-2007. Circ Cardiovasc Qual Outcomes 2012; 5(1): 85-93.
12. Lip GY, Nieuwlaat R, Pisters R, Lane DA, Crijns HJ. Refining clinical risk stratification for predicting stroke and thromboembolism in atrial fibrillation using a novel risk factor-based approach: the euro heart survey on atrial fibrillation. Chest 2010; 137(2): 263-72.
13. Soliman EZ, Safford MM, Muntner P, Khodneva Y, Dawood FZ, Zakai NA, et al. Atrial fibrillation and the risk of myocardial infarction. JAMA Intern Med 2014; 174(1): 107-14.
14. Miyasaka Y, Barnes ME, Gersh BJ, Cha SS, Bailey KR, Abhayaratna W, et al. Incidence and mortality risk of congestive heart failure in atrial fibrillation patients: A community-based study over two decades. Eur Heart J 2006; 27(8): 936-41.
15. Kwok CS, Loke YK, Hale R, Potter JF, Myint PK.

Atrial fibrillation and incidence of dementia: A systematic review and meta-analysis. Neurology 2011; 76(10): 914-22.
16. Stewart S, Hart CL, Hole DJ, McMurray JJ. A population-based study of the long-term risks associated with atrial fibrillation: 20-year follow-up of the Renfrew/Paisley study. Am J Med 2002; 113(5): 359-64.
17. Miyasaka Y, Barnes ME, Bailey KR, Cha SS, Gersh BJ, Seward JB, et al. Mortality trends in patients diagnosed with first atrial fibrillation: A 21-year community-based study. J Am Coll Cardiol 2007; 49(9): 986-92.
18. Krijthe BP, Kunst A, Benjamin EJ, Lip GY, Franco OH, Hofman A, et al. Projections on the number of individuals with atrial fibrillation in the European Union, from 2000 to 2060. Eur Heart J 2013; 34(35): 2746-51.
19. Behrouz R, Powers CJ. Epidemiology of classical risk factors in stroke patients in the Middle East. Eur J Neurol 2016; 23(2): 262-9.
20. Habibzadeh F, Yadollahie M, Roshanipoor M, Haghighi AB. Prevalence of atrial fibrillation in a primary health care centre in Fars Province, Islamic Republic of Iran. East Mediterr Health J 2004; 10(1-2): 147-51.
21. Ahmadi A, Etemad K, Khaledifar A. Risk factors for heart failure in a cohort of patients with newly diagnosed myocardial infarction: A matched, case-control study in Iran. Epidemiol Health 2016; 38: e2016019.
22. Rabiei K, Hosseini SM, Sadeghi E, Jafari-Koshki T, Rahimi M, Shishehforoush M, et al. Air pollution and cardiovascular and respiratory disease: Rationale and methodology of CAPACITY study. ARYA Atheroscler 2017; 13(6): 264-73.
23. Rich DQ, Kim MH, Turner JR, Mittleman MA, Schwartz J, Catalano PJ, et al. Association of ventricular arrhythmias detected by implantable cardioverter defibrillator and ambient air pollutants in the St Louis, Missouri metropolitan area. Occup Environ Med 2006; 63(9): 591-6.
24. Link MS, Luttmann-Gibson H, Schwartz J, Mittleman MA, Wessler B, Gold DR, et al. Acute exposure to air pollution triggers atrial fibrillation. J Am Coll Cardiol 2013; 62(9): 816-25.
25. Santos UP, Terra-Filho M, Lin CA, Pereira LA, Vieira TC, Saldiva PH, et al. Cardiac arrhythmia emergency room visits and environmental air pollution in Sao Paulo, Brazil. J Epidemiol Community Health 2008; 62(3): 267-72.
26. Solimini AG, Renzi M. Association between air pollution and emergency room visits for atrial fibrillation. Int J Environ Res Public Health 2017; 14(6).
27. Milojevic A, Wilkinson P, Armstrong B, Bhaskaran K, Smeeth L, Hajat S. Short-term effects of air pollution on a range of cardiovascular events in England and Wales: Case-crossover analysis of the MINAP database, hospital admissions and mortality. Heart 2014; 100(14): 1093-8.
28. Brook RD, Rajagopalan S, Pope CA 3rd, Brook JR, Bhatnagar A, Diez-Roux AV, et al. Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. Circulation 2010; 121(21): 2331-78.
29. Schnabel RB, Yin X, Gona P, Larson MG, Beiser AS, McManus DD, et al. 50 year trends in atrial fibrillation prevalence, incidence, risk factors, and mortality in theFramingham Heart Study: A cohort study. Lancet. 2015; 386(9989): 154-62.
30. Shehab A, Zubaid M, Bhagavathula AS, Rashed WA, Alsheikh-Ali AA, AlMahmeed W, et al. Sex differences in management and outcomes of patients with atrial fibrillation in the Middle East: Gulf survey of atrial fibrillation events (Gulf SAFE). PLoS One 2017; 12(5): e0175405.

DOI: http://dx.doi.org/10.22122/arya.v15i3.1839 Published by Vesnu Publications

Validity and reliability of the Iranian version of the Cardiac Exercise Self-Efficacy Scale

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Original Article

Abstract

BACKGROUND: The assessment of exercise self-efficacy in patients with cardiovascular disease (CVD) is necessary to conduct tailored interventions. The aim of the current study was to validate the Iranian version of the Cardiac Exercise Self-efficacy Scale (CESE) for patients with CVD.

METHODS: To develop the Iranian version of the CESE scale, a forward and back translation procedure was followed. Data were collected from 260 patients with CVD who were admitted to Imam Ali Cardiovascular Hospital, Iran, using convenience sampling. Psychometric properties of the scale including validity (face and content validity, discriminant, concurrent, convergent, divergent, and construct validity) and reliability (internal consistency, and test-retest reliability) were assessed. SPSS software was used for statistical analysis.

RESULTS: The questionnaire had a good face and content validity and reliability, with Cronbach's alpha of 0.87 and intraclass correlation coefficient (ICC) of 0.42. The questionnaire discriminated well between subgroups according to their medical conditions and the "health transition" item in the Short-Form Health Survey (SF-36). There was a significant correlation between CESE and the physical components of the SF-36 ($P < 0.001$). In addition, a strong to moderate significant correlation was found between the CESE and the Exercise Self-efficacy Scale (ESES) ($r = 0.77$; $P < 0.01$) and between CESE and the Hospital Anxiety and Depression Scale (HADS) total ($r = -0.45$; $P < 0.001$). The exploratory factor analysis (EFA) identified a four-factor structure model, explaining 71.02% of the observed variance.

CONCLUSION: The Persian version of the CESE is a valid and reliable instrument for the evaluation of CVD patients' exercise self-efficacy level in performing regular exercise behaviors.

Keywords: Exercise, Self-Efficacy, Cardiovascular Diseases, Validity, Reliability, Iran

Date of submission: 29 July 2018, ***Date of acceptance:*** 13 Mar. 2019

Introduction

Cardiovascular disease (CVD) is the leading cause of death worldwide, accounting for 50% of deaths in all developed countries. Although the incidence rate of CVD in less developed countries like Iran is lower than in Western countries, it is increasing due to their modernization and shift toward Western lifestyle.[1,2]

Physical activity seems to play an important role in quality of life (QOL),[3] but in patients with CVD there are several factors that may potentially limit a physically active lifestyle, including reduced aerobic exercise capacity and impaired muscle endurance.[4] The reasons for patients' inactivity included cardiac limitations, respiratory causes, and inappropriate advice regarding exercise. In addition to clinical issues, some psychological and cognitive factors affect patients' physical activity.[5,6]

Self-efficacy is a cognitive factor influencing health and QOL. Perceived self-efficacy is defined as beliefs about one's abilities to perform or maintain a behavior such as compliance to exercise.[7-9] In fact, self-efficacy, as a component of social cognitive theory (SCT), is the beliefs of personal efficacy assigned to the acquisition of knowledge on which training is founded.[10] A high level of self-efficacy is associated with more exercise in patients with CVD. In addition, evidence has been provided for the effectiveness of interventions for improving self-efficacy on controlling cardiovascular risk factors.[11]

How to cite this article: Rajati F, Rajati M. **Validity and reliability of the Iranian version of the Cardiac Exercise Self-Efficacy Scale.** ARYA Atheroscler 2019; 15(3): 113-22.

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Thus, the evaluation of exercise self-efficacy in patients with CVD will assist health care professionals in the provision of tailored interventions for the improvement of a patient's confidence in performing exercise[12] and health behaviors.[13] Some exercise self-efficacy scales have been developed to assess exercise self-efficacy in Iranian[14] and other languages.[15,16] Nevertheless, to our knowledge, there is a lack of valid disease-specific questionnaire to evaluate exercise self-efficacy in Persian-speaking patients with CVD. The Cardiac Exercise Self-Efficacy Scale (CESE) is a valid and reliable questionnaire developed as a cardiac-specific exercise self-efficacy scale. The reliability and validity of the CESE have been well established in a rehabilitation setting.[17] Therefore, the present study was conducted to translate the CESE into Persian and to evaluate the psychometric properties of the CESE among Persian-speaking patients with CVD.

Materials and Methods

This study used a methodological design to translate the CESE from English into Persian and to evaluate the validity and reliability of the translated scale in a sample of Iranian patients with CVD. For the validation and adaptation of the questionnaire, the number of participants should be at least 5-10 times of the number of items of an instrument for determining the factor structure.[18] For this purpose, we recruited 260 patients with CVD admitted to Imam Ali Cardiovascular Hospital, Iran, from May to September 2016, using convenience sampling method. Intercultural adaptation was performed according to the study by Aksayan et al.[19]

Procedures

Translation and back translation: Translation and blind back translation was used to translate the CESE from English into Persian according to the guideline provided by Beaton et al.[20] A bilingual physiotherapist, a health professional, fluent in English, and one translator translated the CESE separately. The three produced versions were cross-examined and compared by the authors and divergences were modified. Thus, the consolidated forward translation version was produced. Then, a panel of committee members including a cardiologist, health educator, and sport medicine physician evaluated the initial version for content equivalence. The expert panel raised some queries for the translators. The discrepancies were modified and resolved by consensus, resulting in the two primary Persian versions of the CESE.

A Persian-English translator and a native-English-speaking translator, who were not familiar with the original version, translated the two primary Persian versions of CESE back into English independently. Following this, each version was cross-examined to produce the back translation version. For content comparison, a bilingual expert panel compared the content of each item in the back-translated version with the corresponding item in the original English version.

The pre-final version of the Persian questionnaire was examined using a pilot study to assess the face validity of the questions. A convenient sample of 35 Iranian patients with CVD admitted to Imam Ali Cardiovascular Hospital participated in this phase. They were asked to respond to the questionnaire and express any problems in understanding to indicate items difficult to understand, offensive, or confusing. Finally, after several revisions based on the results of the pilot study and expert panel opinions, the final version was finalized.

We used face-to-face interviews for data collection to avoid selection bias related to illiterate participants and to reduce the number of non-respondents. All participants were interviewed by a trained interviewer individually. To assess test-retest reliability, 30 patients completed the CESE over 2 weeks. The clinical data, including history of disease, comorbidities, and drug use, were obtained from the patients' medical records.

Ethical approval for the study was obtained from Kermanshah University of Medical Sciences, Iran, (Registration code: 95145). All patients were asked to complete the consent form.

The questionnaires

Demographic questionnaire: To assess the demographic characteristics and disease-specific characteristics of participants, we developed a 12-item instrument. The open and structured items of this instrument include age, gender, birth date, education level, job, weight, height, smoking duration and cessation duration, positive history of psychiatric disorders, pulmonary, neurologic, digestive, kidney, skeletal, and endocrinology diseases, and history of hyperglycemia, hyperlipidemia, hypertension, obesity, and CVD.

The Cardiac Exercise Self-Efficacy scale: The CESE is a 16-item instrument originally developed by Hickey et al. to assess confidence in performing exercise in cardiac patients.[17] Each item is rated on a 5-point Likert-type scale, ranging from 1 (very little confidence) to 5 (highest confidence). Individuals

were asked to assess their confidence level in behaviors such as fitting exercise into a busy day, warming up before exercise, and cooling down after exercise. Cronbach's alpha for the CESE instrument was 0.97 at baseline. In addition, test-retest reliability was estimated at 0.87 using intraclass correlation coefficient (ICC) in a sample of cardiac rehabilitation participants. Known-groups validity was documented in a sample of marathon runners, and this sample reported significantly ($P < 0.010$) higher self-efficacy scores than the participants receiving cardiac rehabilitation.[17]

Exercise Self-Efficacy Scale: The Exercise Self-Efficacy Scale (ESES) is an 18-item scale developed by Bandura (1997) to assess exercise behavior in patients with diabetes mellitus (DM).[10] Participants were asked to rate their confidence in getting themselves to perform regular exercise (3 or more times per week). The score scale ranged from 0 (cannot do at all) to 10 (highly certain I can do). The Cronbach's alpha coefficient of the original version of ESES was 0.89, demonstrating a high internal consistency.[15,21] The Persian version of the ESES is well documented and its validity and reliability are acceptable, the Cronbach's alpha for the whole scale has been reported as 0.92.[14]

Hospital Anxiety and Depression Scale: Zigmond and Snaith originally developed the Hospital Anxiety and Depression Scale (HADS) to measure anxiety and depression in the setting of a hospital medical outpatient clinic.[22] This 14-item self-report scale consists of the two subscales of anxiety and depression, each including 7 items. The completion of the scale requires 2–5 minutes. Each item is scored within the range of 0-3; this means that a person can score between 0 and 21 in each of the anxiety or depression subscales. Cut-off scores are available for quantification. A score of 0–7 shows normal status, and 8–10 suggests probable depressive and anxiety symptoms. Scores of 11–21 indicate a clinical case of depression or anxiety.[22] The HADS scale has been validated in many languages and settings.[23-30] The Persian version of the HADS is available and its validity and reliability is acceptable, Cronbach's alpha for anxiety and depression subscales was reported as 0.78 and 0.86, respectively. Validity was determined using known-groups comparison analysis, which showed satisfactory results. Furthermore, the results of convergent validity showed that the Pearson correlation coefficient varied from 0.47 to 0.83 for the anxiety subscale and from 0.48 to 0.86 for the depression subscale.[31]

Short Form-36 Health Survey Scale: The Short Form-36 Health Survey Scale (SF-36) is a well-known generic scale of health status consisting of 36 items with the 8 domains of physical functioning, mental health, social functioning, vitality, and role limitations due to emotional problems, role limitations due to physical problems, bodily pain, and general health. Question 2 shows the variation in health condition over the past year, which is not scored and is named the "health transition item".[32] The subscales are scored from 0 to 100, with higher scores indicating better health status. We used the Persian version of the SF-36.

The validation of the Iranian version of the SF-36 is well documented with its Cronbach's alpha ranging from 0.77 to 0.90. Validity was assessed using the known-groups and convergent analysis. In addition, known-groups analysis indicated that the SF-36 discriminated well between sub-groups of individuals who differed in sex and age.[33]

Descriptive statistics were calculated for all variables. Data are presented as mean ± standard deviation (Mean ± SD) for quantitative variables, and frequency of occurrence or percentage for categorical variables. To investigate the normality of distribution of the interval variables, Kolmogorov–Smirnov (K-S) test was used. In addition, floor and ceiling effects were also examined. All statistical analyses were conducted at the significance level of 5% using SPSS software (version 20, IBM Corporation, Armonk, NY, USA).

Psychometric analysis

Validity

Face and Content Validity: The face and content validity of CESE were investigated quantitatively and qualitatively by the related experts. We asked 10 related experts to examine the validity of each item of CESE quantitatively and qualitatively. Content validity ratio (CVR) and content validity index (CVI) were assessed according to the Lawshe method.[34] The validity of each question was evaluated by adding the number of experts who had scored the question as 3 or 4, divided by the total number of experts, using a four-point scale. CVI value for the total set of items was computed using the sum of the "3" and "4" scores (relevancy) percentage from each expert divided by the total number of experts.[35] A CVI score of higher than 0.80 was considered as acceptable.[36] CVR scores were calculated to determine the necessity of each item. A CVR score of equal to or higher than 0.62 was considered a good content validity by 10 experts based on the Lawshe table.[37]

Floor and ceiling effect: The floor and ceiling values, respectively, indicate the percentage of the patients who obtained the lowest and highest scores in CESE subscales separately. When more than 15% of the participants obtain the lowest or highest possible scores, floor or ceiling effects are considered present, respectively.[38]

Construct Validity: The construct validity of the CESE was determined using exploratory factor analysis (EFA). Factor structure of the Persian version of the CESE was assessed using EFA and utilizing principal component analysis and viramax rotation. Factor loadings of higher than 0.40 were considered as illustrative of a significant relationship between item and scale. Two primary tests were conducted to assess data fit, the Bartlett test to evaluate the factorability of items and the Kaiser-Meyer-Olkin (KMO) test to measure sampling adequacy.

Concurrent validity: There is a possible correlation between QOL and self-efficacy in cardiac patients. So, concurrent validity was assessed by measuring the association of the PSES and the SF-36 scores.

Convergent and divergent validity: To assess convergent validity, we assumed that there are strong relationships between Iranian versions of the CESE with ESES. Thus, we evaluated the correlation between the mean scores of the CESE and ESES. To examine divergent validity, we assumed that subjects with higher self-efficacy experienced lower levels of anxiety and depression. Thus, we compared the mean score of the Persian version of the CESE with total HADS score and the score of its subscales. We used the Pearson correlation to quantify concurrent, convergent, and discriminant validity. A correlation coefficient of greater than 0.3 between the relevant scales was considered as acceptable.[39]

Discriminant validity: We conducted known-groups analysis to determine how well the Iranian version of the CESE discriminates between subcategories of patients who differed in terms of medical condition. To achieve this, patients with CVDs were divided into subcategories including patients who were undergoing coronary artery bypass surgery (CABG), patients with and without heart failure, patients with and without obesity [Body mass index(BMI ≥ 30)], and patients with and without dyslipidemia. Dyslipidemia was defined as low-density lipoprotein (LDL) cholesterol ≥ 160 mg/dl, high-density lipoprotein (HDL) cholesterol < 40 mg/dl, total cholesterol ≥ 240 mg/dl, triglycerides ≥ 200 mg/dl, and/or positive history of relevant medication intake.[40] To compare the mean total CESE scores between each subgroup of patients, we conducted independent t-test. We also assessed the discriminative power of CESE between three groups of patients according to the "health transition" item (item 2 of SF-36), indicating the level of variation in general health over the previous year. This item rates patients based on a 5-point scale ranging from "much better now than one year ago" to "much worse now than one year ago". We re-categorized patients according to this item into three subgroups including "not changed," "improved", and "deteriorated" status. Accordingly, we used ANOVA with Tukey's post hoc comparisons to evaluate mean differences of total CESE scores between the abovementioned subgroups of patients in terms of health transition item.

Reliability

Internal consistency: The internal consistency of the CESE was calculated using Cronbach's alpha and item-total correlation. Values ≥ 0.70 were accounted satisfactory.[41] A total correlation of an item ≥ 0.4 was considered acceptable.[42]

Test-retest: Reliability of the CESE was evaluated in a random sample of patients with CVDs, who did not participate in any rehabilitation or intervention program, over a 14-day period by performing ICC analysis (the two-way random model). The same interviewer conducted the test and retest interviews.

ICC values between 0.4 and 0.75 were considered as fair to good, and values higher than 0.75 were considered as excellent. The Bland–Altman method was used to compare scale scores between the test and retest procedures.[43].

Results

Participant demographics: Eligibility screening was conducted on 260 patients with coronary heart disease (CHD) in the current study. All of these patients (100%) completed the whole questionnaire and were included in the final data analysis. The mean age of the participants was 48.90 ± 13.77 years. The majority of the patients were men (n = 141; 54.2%). In total, 60% of them had a secondary education. Approximately, 29.2% of patients (n = 76) had undergone CABG, 50% of whom (n = 38) had undergone CABG more than 1 year ago. Moreover, 78 (30%) patients had had a myocardial infarction (MI) 1-72 months ago. More than one-third of patients underwent coronary stent placement. The majority of the participants had comorbidities including hypertension (80%) and dyslipidemia (68.46%). In addition, more than one-third of the patients had a respiratory disease and DM. The clinical and sociodemographic characteristics of the participants are provided in table 1.

Table 1. Clinical and sociodemographic characteristic of the patients (n = 260)

Variables	n (%)
Sex (male)	141 (54.23)
Smoking (yes)	132 (52.30)
Level of education	
Illiterate	14 (5.38)
pre-diploma and high school diploma	90 (34.61)
Bachelor's degree	152 (58.46)
Master's and Doctoral degree	4 (1.53)
CAD subgroup	
CABG	76 (29.23)
MI	78 (30.00)
Coronary stent placement	94 (36.15)
Other diagnosis	12 (21.66)
Comorbidity	
Hypertension	220 (80.00)
Dyslipidemia	178 (68.46)
Respiratory disease	114 (43.80)
Diabetes	106 (40.80)
CNS disease	52 (20.00)
Musculoskeletal disease	40 (15.40)
Renal disease	16 (6.20)
Mental disease	8 (3.07)
Gastrointestinal disease	8 (3.07)
Anti-diabetic treatment (insulin, gemfibrozil, and Glibenclamide)	54 (22.50)
Cardiovascular drugs	
Antiplatelet	122 (46.92)
Antilipid	120 (46.20)
B-blocker	162 (62.30)
ACEI	142 (54.60)
ARB	74 (28.50)
Anti-coagulant	12 (4.60)

CAD: Coronary artery disease; CABG: Coronary Artery Bypass Surgery; MI: Myocardial infarction; CNS: Central nervous system disease; ACEI: Angiotensin-converting-enzyme inhibitors; ARB: Angiotensin receptor blocker

The descriptive statistics of the Cardiac Exercise Self-Efficacy Scale: The K-S test was used to assess the normality of each CESE item. The results showed that data were normally distributed. The mean total score of the CESE was (2.62 ± 0.7).

Floor and ceiling effects: No participant scored the highest score for the total CESE, thus providing no ceiling effects of the total scale. Only 1.5% of participants obtained the lowest scores, indicating no/low floor effects for total CESE (Table 2).

Validity

Face and content validity: Based on the responses of the 10 experts, minor changes in the translated items of the final CESE indicated good face validity for the scale. The scale CVI was 0.82 and the item CVIs ranged from 0.76 to 0.88, while the scale CVR was 0.88, and the item CVRs ranged from 0.73 to 0.89, indicating the good content validity of the CESE.

Table 2. Assessment of floor and ceiling effect in the Iranian version of the Cardiac Exercise Self-Efficacy Scale (CESE)

Scale and subscales	Floor effect [n (%)]	Ceiling effect [n (%)]
CESE for knowledge	6 (2.3)	14 (5.4)
CESE for Overcoming Barriers	64 (24.6)	4 (1.5)
CESE for Time Management	18 (6.9)	10 (3.8)
CESE for Recovery	50 (19.2)	0 (0)
Total	4 (1.5)	0 (0)

CESE: the Cardiac Exercise Self-Efficacy Scale

Concurrent validity: To determine concurrent validity, we calculated the correlation between CESE and SF-36. Therefore, the two questionnaires were completed by patients who accepted to fill out both questionnaires. In total, 98 participants completed the CESE and the SF-36 at the same time. Correlations between the CESE scale and SF-36 domains were calculated. These results illustrate that the correlation between the two instruments was acceptable. Physical domains of SF-36 including physical functioning (r = 0.54), physical role functioning (r = 0.39), bodily pain (r = 0.39), general health perceptions (r = 0.59), and physical component summary (0.49) significantly correlated with the total score of the CESE (P < 0.001). Emotional role functioning and social role functioning were also two mental dimensions that significantly correlated with total CESE (r = 0.33 and r = 0.47, respectively). However, the correlation between the physical component of SF-36 and CESE was 0.41, while the correlation between the mental component of SF-36 and CESE was 0.34.

Convergent/divergent validity: The mean ESES score was 39.62 ± 16.43. There was a strong correlation between the CESE and ESES (r = 0.77; P < 0.010), providing a good convergent validity of the CESE.

The mean score of the anxiety and depression subscales of the HADS were 7.63 ± 3.12 and 8.60 ± 2.98, respectively. There was a significant and negative correlation between the mean CESE score and HADS total and subscales scores (P < 0.001). The Pearson coefficients between CESE, and the anxiety and depression subscales and total HADS score were -0.45, -0.57, and -0.57, respectively, indicating a high divergent validity of the CESE.

Discriminant validity: Known-groups comparison analysis showed significant differences in the total CESE score between patients who had experienced MI and patients who had undergone CABG. Patients who had undergone CABG had significantly lower mean total CESE score than those who had not undergone CABG (2.21 ± 0.64 vs. 2.80 ± 0.65; P < 0.001). Similarly, patients with MI had significantly lower total CESE score than those without this clinical diagnosis (2.21 ± 0.66 vs. 2.80 ± 0.66; P < 0.001). Obese patients had lower CESE score than patients who had a BMI of lower than 30 (2.26 ± 0.57 vs. 2.7 ± 0.69; P < 0.001). In addition, patients with dyslipidemia reported significantly lower mean CESE scores than those without dyslipidemia (2.44 ± 0.61 vs. 3.04 ± 0.69; P < 0.001).

In addition, the mean score of CESE was only significantly different between patients who were categorized as "deteriorated" and "improved" (2.17 vs. 2.89; P < 0.050) and between "deteriorated" and "not changed" subgroups (2.17 vs. 2.53; P < 0.050) in terms of the health transition item (Figure 1).

Construct validity: The result of Bartlett's test of sphericity was significant (P < 0.001), indicating the appropriateness of data to perform factor analysis. The KMO value of sampling adequacy was 0.85, more than the recommended index of 0.60.[44]

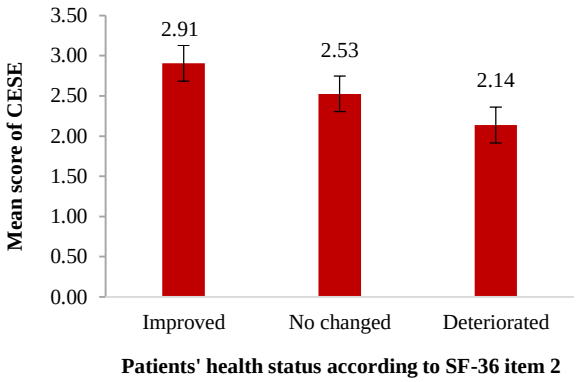

Figure 1. The mean score of the Cardiac Exercise Self-Efficacy Scale (CESE) according to health status

Viramax rotation was applied for factor analysis. The result showed that the Persian version of the CESE had four factors. As shown in table 3, factor 1, including items 1, 2, 3, 5, 6, 8, 11, 12, and 15, is related to knowledge of the exercise for cardiac patients, thus we called this factor "cardiac exercise self-efficacy in knowledge".

Table 3. The factor structure of the Persian version of the Cardiac Exercise Self-Efficacy Scale by principal components factor analysis

Item	Questions	Factor 1 CESE for Knowledge	Factor 2 CESE for Overcoming Barriers	Factor 3 CESE for Time Management	Factor 4 CESE for Recovery
CESE1	"Warming up" before exercise	0.535		0.585	
CESE2	Exercising without getting chest pain	0.556		0.512	
CESE3	Knowing when I have exercised too much and need to stop	0.831			
CESE4	Exercising when it is inconvenient				0.567
CESE5	Knowing what my heart rate should be before and after exercise	0.867			
CESE6	"Cooling down" after exercise	0.746			
CESE7	Fitting exercise into a busy day			0.782	
CESE8	Enduring strenuous exercise	0.532	0.547		
CESE9	Knowing what exercise is healthy for me		0.875		
CESE10	Knowing when I can increase my exercise level		0.880		
CESE11	Enduring moderate exercise	0.862			
CESE12	Taking my heart rate before and after exercise	0.805			
CESE13	Resuming my pre-hospital level of activity				0.825
CESE14	Enduring light exercise				0.746
CESE15	Exercising for at least twenty minutes three times each week	0.486		0.543	
CESE16	Exercising at home by myself		0.537		
% variance		41.650	11.210	10.62	7.540
Eigenvalue		6.660	1.790	1.700	1.200

CESE: Cardiac Exercise Self-Efficacy Scale
The values under 0.45 was removed from the table.

In addition, factor 2, including items 8, 9, 10, and 16, is related to "cardiac exercise self-efficacy for overcoming barriers". Factor 3, including items 1, 15, and 7, is related to "cardiac exercise self-efficacy in time management". Factor 4, including items 4, 14, and 13, is related to "cardiac exercise self-efficacy for recovery". However, items 1, 2, and 15 were loaded on both factors 1 and 3 (Table 3).

Reliability: Cronbach's alpha for the total score of CESE was 0.87. The correlations between the subscales of CESE and total CESE are presented in table 4. The correlation coefficients between each subscale and the total CESE score ranged from 0.28 (recovery) to 0.92 (knowledge). In test-retest analysis, the ICCs for the total CESE was 0.42 ($P < 0.001$). ICC for each item ranged from 0.51 to 0.74 with a median equal to 0.61, which were within the range of acceptable values.

Table 4. Correlations of total Cardiac Exercise Self-Efficacy Scale (CESE) score with each subscale

CESE subscales	Correlation with total CESE*
Knowledge	0.92
Overcoming Barriers	0.70
Time Management	0.80
Recovery	0.28

* Correlation is significant at the 0.01 level (2-tailed).
CESE: Cardiac Exercise Self-Efficacy Scale

In the Bland-Altman analysis, difference between the test and the retest scores plotted against the mean results (Figure 2). None of the measurements was out of the ± 2 SD range. The Bland-Altman analysis showed low bias (-0.88) and limits of agreement (-8.5 to 6.7).

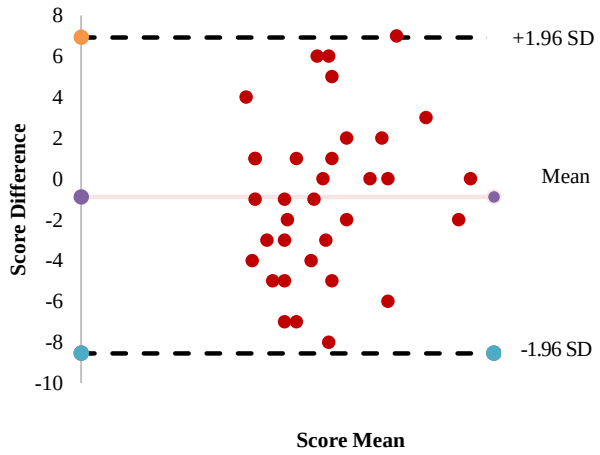

Figure 2. Bland-Altman graphic representation of the test and retest
The horizontal purple line shows the mean bias of -0.88 ± 3.90.

Discussion

This study aimed to evaluate the psychometric properties of the Iranian version of the CESE in patients with CVD. The results of our study showed that the Persian version of the CESE has good validity (i.e., high level of construct validity, concurrent validity, convergent/divergent validity, discriminant validity, and construct validity) and reliability (i.e., a medium ICC and high Cronbach's alpha).[37] CVR and CVI were not reported in the original version of the questionnaire.[17] This study was the first to examine the floor and ceiling effect of the CESE. The CESE has a low floor and no ceiling effect for the total scale, indicating acceptable difficulty in the items for this population. However, such an evaluation has not been implemented in the original version.[17] All items were responded, meaning that all items in the Persian version of the CESE were clear and applicable for patients. However, when we consider individual items, some patients did not respond in a similar range and had a variation, especially for item 13. This item asks the patients to rate their confidence in resuming their pre-hospital level of activity. An explanation for this matter might be that some of the patients were hospitalized more than 1 year ago. Therefore, they had forgotten their level of activity before hospitalization and this may be the cause of their delay in responding.

The concurrent validity of the CESE was approved in our study, in which significant and moderate correlations were found between the CESE scale and all physical subscales of the SF-36. These correlations were significant in only two mental subscales including social role functioning and emotional role functioning. It has been generally accepted that such a moderate correlation is desirable.[38] This is also consistent with the results of some studies indicating a strong correlation between self-efficacy and functional status, exercise behavior, QOL, and social support in patients with CVD.[45-47]

Additionally, the discriminant validity of the CESE was confirmed; significant differences were found in the CESE score between the patients with and without CABG, and MI. Moreover, 2 comorbidities were negatively associated with patients' level of exercise self-efficacy. In an alternative approach, to examine discriminant validity, we identified individuals with "improved", "not changed," and "deteriorated" health status according to their response to the health transition item in the SF-36. The CESE could discriminate between groups that were categorized as

"deteriorated" and those categorized as either "not changed" or "improved" in their health status.

In addition, the CESE demonstrated good convergent validity with significantly strong correlation with the ESES. Noroozi et al. validated the Persian version of Bandura's ESES in patients with DM.[14] Bandura's ESES has also been validated in an Australian cardiac rehabilitation setting.[15] Regarding the strong correlation between ESES and CESE, it seems that two items are similar in their concepts. However, the CESE is a cardiac-specific instrument compared to the ESES. Thus, we expected the CESE to be a better tool than the ESES for the measurement of exercise self-efficacy among patients with CVD.[48] The CESE also demonstrated good divergent validity with significantly negative correlations with the total score of the HADS, and its anxiety and depression subscales. This is also consistent with some studies indicating a significant negative relationship between physical activity and emotional problems such as depression and anxiety among patients with coronary artery disease (CAD).[49-51]

An exploratory principal component factor analysis identified a 4-factor structure model, explaining 71.02% of the observed variance (knowledge: 41.65, overcoming barriers: 11.21, time management: 10.62, and recovery: 7.54). Each item had a factor loading of 0.45 or higher, which was considered acceptable.[52] Furthermore, 3 items were loaded on 2 factors. Since the factor loadings were approximately equivalent in magnitude for these items on both factors, they could be categorized under both of them. Therefore, item 1 ("warming up before exercise"), item 2 ("exercising without getting chest pain"), and item 15 ("exercising for at least 20 minutes 3 times each week") were categorized under both subscales 1 and 3 because the three items could evaluate both knowledge and time management of patients. However, these results were inconsistent with the original version of the CESE that is one-dimensional. This may be due to the difference between the settings of the studies and the inclusion of all general CAD patients in the original study, which was conducted in the cardiac rehabilitation setting. To our knowledge, no further studies have been conducted on the validation of the CESE, and therefore, no well-documented resource is available with which to compare our results. In the study of Dong et al.,[53] in which the psychometric properties of the ESES were evaluated in Chinese-speaking stroke patients, a two-factor model was reported, which was also inconsistent with the original version of Kim et al.[54] The difference between models reported in these languages might be because patients' perceptions of their confidence in performing designated exercise behaviors can often differ according to different culture and social contexts.

In the present study, the internal consistency of the CESE was confirmed with a Cronbach's alpha of 0.87 for the total scale. In addition, test-retest reliability was assessed to evaluate the stability of the CESE over 2 weeks. Moreover, test-retest agreement in the Bland–Altman plot was evaluated. The ICC was lower than we expected (ICC: 0.42; $P < 0.001$). This may be due to some patients' participation in rehabilitation programs and management by rehabilitation staff during the 2 interval weeks of test-retest. Hence, some patients did not have similar health care conditions during this period. However, the Bland-Altman plot approved the reliability of the questionnaire. This finding was in agreement with those reported in the validation of the ESES.[15]

Limitations: This study had four limitations. First, the study participants were recruited from one hospital in Western Iran, which may affect the generalizability of the findings to a wider population in Iran. Second, Although the explanatory factor analysis results indicated that the four-factor structure was perfect, this method is a data-driven method for exploring the factor structure and it should not be used to confirm factor structure.[41] Therefore, further studies are required to confirm the factor structures of the CESE reported in this paper using confirmatory factor analysis. Third, the value of ICC was low, which is probably due to improving patients' medical condition. Forth, our study was a cross-sectional study; thus, it does not allow the assessment of the responsiveness of the Persian version of the CESE to some patients' clinical variation over time. Therefore, it is recommended that future studies assess these changes using longitudinal data.

Conclusion

The study results confirmed that the Persian version of the ESES is a valid and reliable instrument for the assessment of CVD patients' confidence level in performing regular exercise behaviors. Healthcare professionals can use it in Persian-speaking patients with CVD to conduct psychological interventions to improve their self-efficacy and persistence in exercise behaviors.

Acknowledgments

The authors express their sincere gratitude for the cooperation of patients and staff of Imam Ali Hospital. This study was approved by the research ethics committee of Kermanshah University of Medical Sciences (Registration number: 95145).

Conflict of Interests

Authors have no conflict of interests.

References

1. Murray CJ, Lopez AD. Alternative projections of mortality and disability by cause 1990-2020: Global Burden of Disease Study. Lancet 1997; 349(9064): 1498-504.
2. Rezaeian M, Dehdarinejad A, Esmaili Nadimi A, Tabatabaie S. Geographical epidemiology of deaths due to cardiovascular diseases in counties of kerman province. Iran J Epidemiol 2008; 4(1): 35-41. [In Persian].
3. Pucci GC, Rech CR, Fermino RC, Reis RS. Association between physical activity and quality of life in adults. Rev Saude Publica 2012; 46(1): 166-79.
4. Kroonstrom LA, Johansson L, Zetterstrom AK, Dellborg M, Eriksson P, Cider A. Muscle function in adults with congenital heart disease. Int J Cardiol 2014; 170(3): 358-63.
5. Sandberg C, Thilen U, Wadell K, Johansson B. Adults with complex congenital heart disease have impaired skeletal muscle function and reduced confidence in performing exercise training. Eur J Prev Cardiol 2015; 22(12): 1523-30.
6. Sandberg C, Engstrom KG, Dellborg M, Thilen U, Wadell K, Johansson B. The level of physical exercise is associated with self-reported health status (EQ-5D) in adults with congenital heart disease. Eur J Prev Cardiol 2015; 22(2): 240-8.
7. Craig A, Tran Y, Siddall P, Wijesuriya N, Lovas J, Bartrop R, et al. Developing a model of associations between chronic pain, depressive mood, chronic fatigue, and self-efficacy in people with spinal cord injury. J Pain 2013; 14(9): 911-20.
8. Middleton J, Tran Y, Craig A. Relationship between quality of life and self-efficacy in persons with spinal cord injuries. Arch Phys Med Rehabil 2007; 88(12): 1643-8.
9. Middleton JW, Tate RL, Geraghty TJ. Self-efficacy and spinal cord injury: Psychometric properties of a new scale. Rehabilitation Psychology 2003; 48(4): 281-8.
10. Bandura A. Self-efficacy: The exercise of control. New York, NY: W H Freeman; 1997.
11. Sol BG, van der Graaf Y, van Petersen R, Visseren FL. The effect of self-efficacy on cardiovascular lifestyle. Eur J Cardiovasc Nurs 2011; 10(3): 180-6.
12. Scholz U, Sniehotta FF, Schwarzer R. Predicting physical exercise in cardiac rehabilitation: The role of phase-specific self-efficacy beliefs. J Sport Exerc Psychol 2005; 27(2): 135-51.
13. Rajati F, Kamali K, Parvizy S. Public health customers' experiences of health accessibility: A phenomenological study. Iran J Epidemiol 2011; 7(2): 17-24. [In Persian].
14. Noroozi A, Ghofranipour F, Heydarnia AR, Nabipour I, Tahmasebi R, Tavafian S. The Iranian version of the exercise self-efficacy scale (ESES): Factor structure, internal consistency and construct validity. Health Education Journal 2011; 70(1): 21-31. [In Persian].
15. Everett B, Salamonson Y, Davidson PM. Bandura's exercise self-efficacy scale: Validation in an Australian cardiac rehabilitation setting. Int J Nurs Stud 2009; 46(6): 824-9.
16. Resnick B, Jenkins LS. Testing the reliability and validity of the self-efficacy for exercise scale. Nurs Res 2000; 49(3): 154-9.
17. Hickey ML, Owen SV, Froman RD. Instrument development: Cardiac diet and exercise self-efficacy. Nurs Res 1992; 41(6): 347-51.
18. Tinsley HE, Tinsley DJ. Uses of factor analysis in counseling psychology research. J Couns Psychol 1987; 34(4): 414-24.
19. Aksayan S, Guzum S. A Guideline for intercultural scale adaptation: Stages of scale adaptation and language adaptation. Hemsirelik Arastirma Gelistirme Dergisi 2002; 4: 9-14. [In Turkish].
20. Beaton DE, Bombardier C, Guillemin F, Ferraz MB. Guidelines for the process of cross-cultural adaptation of self-report measures. Spine (Phila Pa 1976) 2000; 25(24): 3186-91.
21. Darawad MW, Hamdan-Mansour AM, Khalil AA, Arabiat D, Samarkandi OA, Alhussami M. Exercise self-efficacy scale: Validation of the arabic version among jordanians with chronic diseases. Clin Nurs Res 2018; 27(7): 890-906.
22. Zigmond AS, Snaith RP. The hospital anxiety and depression scale. Acta Psychiatr Scand 1983; 67(6): 361-70.
23. Lisspers J, Nygren A, Soderman E. Hospital anxiety and depression scale (HAD): Some psychometric data for a Swedish sample. Acta Psychiatr Scand 1997; 96(4): 281-6.
24. Spinhoven P, Ormel J, Sloekers PP, Kempen GI, Speckens AE, Van Hemert AM. A validation study of the hospital anxiety and depression scale (HADS) in different groups of Dutch subjects. Psychol Med 1997; 27(2): 363-70.
25. Bocerean C, Dupret E. A validation study of the hospital anxiety and depression scale (HADS) in a large sample of French employees. BMC Psychiatry 2014; 14: 354.

26. Herrero MJ, Blanch J, Peri JM, De Pablo J, Pintor L, Bulbena A. A validation study of the hospital anxiety and depression scale (HADS) in a Spanish population. Gen Hosp Psychiatry 2003; 25(4): 277-83.

27. Pais-Ribeiro J, Silva I, Ferreira T, Martins A, Meneses R, Baltar M. Validation study of a Portuguese version of the hospital anxiety and depression scale. Psychol Health Med 2007; 12(2): 225-35.

28. Muszbek K, Szekely A, Balogh EM, Molnar M, Rohanszky M, Ruzsa A, et al. Validation of the hungarian translation of hospital anxiety and depression scale. Qual Life Res 2006; 15(4): 761-6.

29. Miklavcic IV, Snoj Z, Mlakar J, Pregelj P. Validation of the Slovenian version of Hospital Anxiety and Depression Scale in female cancer patients. Psychiatr Danub 2008; 20(2): 148-52.

30. Terkawi AS, Tsang S, AlKahtani GJ, Al-Mousa SH, Al MS, AlZoraigi US, et al. Development and validation of Arabic version of the hospital anxiety and depression scale. Saudi J Anaesth 2017; 11(Suppl 1): S11-S18.

31. Montazeri A, Vahdaninia M, Ebrahimi M, Jarvandi S. The Hospital Anxiety and Depression Scale (HADS): Translation and validation study of the Iranian version. Health Qual Life Outcomes 2003; 1: 14.

32. Ware JE Jr, Sherbourne CD. The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. Med Care 1992; 30(6): 473-83.

33. Montazeri A, Goshtasebi A, Vahdaninia M, Gandek B. The short form health survey (SF-36): translation and validation study of the Iranian version. Qual Life Res 2005; 14(3): 875-82.

34. Lawshe CH. A quantitative approach to content validity. Pers Psychol 1975; 28(4): 563-75.

35. DiIorio CK. Measurement in Health Behavior: Methods for Research and Evaluation. Hoboken, NJ: John Wiley & Sons; 2006.

36. Polit DF, Beck CT, Owen SV. Is the CVI an acceptable indicator of content validity? Appraisal and recommendations. Res Nurs Health 2007; 30(4): 459-67.

37. Ayre C, Scally AJ. Critical values for Lawshe's content validity ratio: Revisiting the original methods of calculation. Meas Eval Couns Dev 2014; 47(1): 79-86.

38. Terwee CB, Bot SD, de Boer MR, van der Windt DA, Knol DL, Dekker J, et al. Quality criteria were proposed for measurement properties of health status questionnaires. J Clin Epidemiol 2007; 60(1): 34-42.

39. Portney LG, Watkins MP. Foundations of clinical research: Applications to practice. Upper Saddle River, NJ: Prentice Hall Health; 2000.

40. Turgeon RD, Anderson TJ, Grégoire J, Pearson GJ. 2016 Guidelines for the management of dyslipidemia and the prevention of cardiovascular disease in adults by pharmacists. Can Pharm J (Ott) 2017; 150(4): 243-50.

41. Nunnally JC, Bernstein IH. Psychometric theory. New York, NY: McGraw-Hill; 1994.

42. National Research Council, Mathematical sciences education board. Measuring what counts: A conceptual guide for mathematics assessment. Washington DC: National Academies Press; 1993.

43. Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. Lancet 1986; 1(8476): 307-10.

44. Tabachnick BG, Fidell LS. Using multivariate statistics. Boston, MA: Allyn and Bacon; 2001.

45. Bay A, Sandberg C, Thilen U, Wadell K, Johansson B. Exercise self-efficacy in adults with congenital heart disease. Int J Cardiol Heart Vasc 2018; 18: 7-11.

46. Lapier TK, Cleary K, Kidd J. Exercise self-efficacy, habitual physical activity, and fear of falling in patients with coronary heart disease. Cardiopulm Phys Ther J 2009; 20(4): 5-11.

47. Pashaei T, Shojaeizadeh D, Rahimi Foroushani A, Ghazitabatabae M, Moeeni M, Rajati F, et al. Effectiveness of relapse prevention cognitive-behavioral model in opioid-dependent patients participating in the methadone maintenance treatment in Iran. Iran J Public Health 2013; 42(8): 896-902.

48. Zhang X, Zhan Y, Liu J, Chai S, Xu L, Lei M, et al. Chinese translation and psychometric testing of the cardiac self-efficacy scale in patients with coronary heart disease in mainland China. Health Qual Life Outcomes 2018; 16(1): 43.

49. Strohle A. Physical activity, exercise, depression and anxiety disorders. J Neural Transm (Vienna) 2009; 116(6): 777-84.

50. Petruzzello SJ, Landers DM, Hatfield BD, Kubitz KA, Salazar W. A meta-analysis on the anxiety-reducing effects of acute and chronic exercise. Outcomes and mechanisms. Sports Med 1991; 11(3): 143-82.

51. Bonnet F, Irving K, Terra JL, Nony P, Berthezene F, Moulin P. Anxiety and depression are associated with unhealthy lifestyle in patients at risk of cardiovascular disease. Atherosclerosis 2005; 178(2): 339-44.

52. Mulaik SA. Foundations of factor analysis. Boca Raton, FL: CRC Press; 2009.

53. Dong X, Liu Y, Wang A, Wang M. The psychometric properties of Chines version of SC Exercise Self-Efficacy Scale in patients with stroke. Patient Prefer Adherence 2016; 10: 1235-41.

54. Kim NH, Seo JA, Cho H, Seo JH, Yu JH, Yoo HJ, et al. Risk of the development of diabetes and cardiovascular disease in metabolically healthy obese people: The Korean genome and epidemiology study. Medicine (Baltimore) 2016; 95(15): e3384.

DOI: http://dx.doi.org/10.22122/arya.v15i3.1588 Published by Vesnu Publications

The effect of educational and encouragement interventions on anthropometric characteristics, obestatin and adiponectin levels

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Original Article

Abstract

BACKGROUND: Lifestyle modification is the most important strategy for control of obesity and overweight. Obestatin and adiponectin are the biomarkers of obesity. Thus, this study was performed to examine the effect of educational and encouragement interventions and lifestyle modifications on obesity anthropometric as well as obestatin and adiponectin levels.

METHODS: This semi-experimental study was conducted on a subsample of TABASSOM study. Participants were 41 overweight and obese children and adolescents aged 6-18 years old and 45 overweight and obese adults aged 19-65 years old. Anthropometric characteristics including height, weight, waist and hip circumferences, and body fat percentage (BFP) were measured at the first and after one year at the end of study. We implemented some educational and encouragement interventions regarding dietary modification and physical activity during the study. Obestatin and adiponectin levels were measured at the first and end of study by enzyme-linked immunosorbent assay (ELISA) method.

RESULTS: The study did not show significant effect on anthropometric characteristics such as body mass index (BMI) and waist circumference (WC). BFP decreased significantly in boys, total children and adolescent group, and waist-to-hip ratio (WHR) decreased significantly only in adolescent boys after 1 year ($P < 0.050$).

CONCLUSION: Educational and encouraging interventions and lifestyle modifications could lead to decrease of body WHR and BFP in adolescent boys. This is helpful in controlling the increasing rate of obesity.

Keywords: Obesity, Overweight, Adults, Children, Adolescents, Obestatin, Adiponectin

Date of submission: 21 Feb. 2018, ***Date of acceptance:*** 30 Jan. 2019

Introduction

Obesity is a major cause of non-communicable diseases (NCDs). Studies have shown that weight gain contributes to the incidence of several different diseases, including hypertension (HTN), diabetes, cardiovascular diseases (CVDs), and cancer.[1,2] In Iran, the prevalence of overweight and obesity is 50.5% in men, 60.4% in women, 18.6% in boys, and 21.4% in girls.[3,4] The World Health Organization (WHO) is seeking through its plans to stop the increasing trend of obesity and overweight in the world by 2025.[5]

Prevention and treatment are mainly focused on lifestyle modification, including the modification of nutrition and increasing physical exercise.[5] Making changes in the living environment and developing guidelines for a healthy lifestyle are major strategies for the treatment of obesity and overweight.[6]

How to cite this article: Sajjadi F, Mohammadifard N, Maghroun M, Shirani F, Karimi S, Taheri M, et al. **The effect of educational and encouragement interventions on anthropometric characteristics, obestatin and adiponectin levels.** ARYA Atheroscler 2019; 15(3): 123-29.

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Regaining weight and behavioral change after interventions also comprise a major challenge in the treatment of obesity. Methods need to be devised that help control weight for a longer time and permanently, if possible.

The interventions performed within the Isfahan Healthy Heart Program (IHHP), which is a community-based interventional program conducted in Isfahan, Iran,[7,8] were found to have helped modify certain lifestyle-related factors to an extent.[9,10]

Various biological and psychological factors contribute to gaining back weight after weight loss.[11] Evidence suggests that some treatments for obesity fail in the long term due to the compensatory decrease in metabolism and thus lead to further weight gain.[12] The hormones affecting hunger and satiety play an influential role in this phenomenon.[13] Obestatin is a gastric hormone that affects the stimulation of appetite and thereby energy balances in the body.[14] This hormone stimulates the appetite, regulates weight, and opposes ghrelin.[15] Adiponectin, which is secreted from fat tissues, also affects blood glucose regulation and fatty acid catabolism, such that it improves the body's response to insulin by increasing metabolism.[16] In a study to determine the relationship between obestatin and serum insulin levels, body mass index (BMI), waist circumference (WC), and obesity ratios were significantly higher in obese children, while the obestatin level was significantly lower in these subjects.[17]

Different methods of weight loss are effective in the treatment of obesity, including diet, physical exercise, and surgery, and studies have demonstrated the separate effects of these methods on ghrelin, leptin, adiponectin, resistin, and insulin both in adults and children.[18] Few studies have examined that how training individuals to choose their preferred method of weight loss affects their weight goals and what long-term consequences ensue these interventions.[19,20] This study seeks to answer the question of whether or not giving training to individuals about the different methods of weight loss and allowing them to freely choose their preferred method help modify these biomarkers and whether the changes in these biomarkers correlate with long-term weight loss or further weight gain. This study was conducted to examine the effect of educational and encouragement interventions on obesity indicators and biomarkers including adiponectin and obestatin levels in overweight and obese subjects.

Materials and Methods

This quasi-experimental study was conducted on February 2012 until March 2013 with a before-after design on a subgroup of people who had participated in the TABASSOM study. In TABASSOM study, overweight and obese volunteers willing to participate in the "lose weight and win" competition were invited through a public announcement through the Islamic Republic of Iran Broadcasting (IRIB) and in the schools.[11] The participants included 86 persons that were divided into two groups: 41 children and adolescents (aged 6-18 years) and 45 adults (aged 19-65 years). The inclusion criteria consisted of residing in Isfahan City and having a BMI of 25 or higher for the adults and 85th percentile or higher for the children and adolescents.[21] Participants should have no systemic illness, liver and renal disease, and endocrine and mental disorders. Those who did not practice other methods of weight loss, including pharmacotherapy and surgery, were also allocated. We excluded non-compliant subjects from the study. Only 4 children were excluded due to unwillingness to continue. The study was approved by the Ethics Committee of Isfahan Cardiovascular Research Institute (ICRI). Written informed consents were obtained from all participants. Trained health professionals carried out detailed interviews at study baseline to obtain information about participants' socioeconomic and demographic characteristics.[22]

In order to implement a comprehensive interventional program, educational and encouraging interventions were applied in the course of one year.

Educational interventions: Educational materials (e.g., pamphlets, CDs, and multimedia programs) were used to explain weight control strategies and inappropriate weight loss methods (e.g., particular medical treatments and their side effects). The participants were also provided with a pamphlet about healthy diet, a CD about physical activity, and follow-up cards. They were asked to keep the cards during the follow-up sessions (6 and 12 months later). Furthermore, after coordination with the Culture Department of Isfahan Municipality, extensive community-based interventions regarding obesity and obesity prevention were provided in 17 cultural centers. Five educational pamphlets were also distributed in schools of Isfahan with the help of Isfahan Municipality's Committee for Citizenship Culture. Several educational posters were also prepared and installed on billboards in crowded areas of the city.

Encouraging interventions: After six months, the participants with at least 10% weight loss were included in a competition. They were reevaluated after another six months. Twenty individuals who showed weight loss or no weight gain after the second period were randomly rewarded. The anthropometric indices, obestatin and adiponectin levels, were re-evaluated after one year.

Participants' weight and height were measured with no shoes on and in light clothing using a seca scale with a precision of 0.5 kg and a plastic meter with a precision of 0.5 cm. The BMI was calculated using the equation 'weight in kilograms divided by the square of the height in meters'. The waist and hip circumferences were measured over light clothing at a standing position using a plastic meter, and the WC was measured at the center of the lower rib and the top of the hip, and the hip circumference at the femur.[21] The waist-to-hip ratio (WHR) was also calculated. The body fat percentage (BFP) was measured with a body composition analyzer (Ironman InnerScan Body Composition Monitor from Tanita).

Participants' adiponectin and obestatin levels were measured using the enzyme-linked immunosorbent assay (ELISA) method and with a buffer kit and an Eastbiopharm elisa kit (Hangzhou Eastbiopharm Co., USA) at baseline and at the end of the project, i.e., one year later.[23]

All the information collected from the questionnaires and the initial demographic information obtained from the candidates were entered into a computer using Epi Info software. Once thoroughly analyzed and managed, the data were reported as mean and standard deviation (SD), and the non-normally distributed data were reported as median and interquartile range, and all this data were then tabulated.

The paired sample t-test was used to compare the data before and after the intervention, and Wilcoxon's nonparametric test (the Wilcoxon signed-rank test) was used to compare the non-normally distributed data. The independent sample t-test was used to examine the percentage of changes between the two genders, and Mann-Whitney's nonparametric U test was used for the non-normally distributed data. Data were analyzed in SPSS software (version 15, SPSS Inc., Chicago, IL, USA). The level of statistical significance was set at $P < 0.050$.

Results

Table 1 indicates the mean demographic and anthropometric data of adults and children and adolescents based on gender. The mean weight, BMI, WHR, and BFP differed significantly between the two genders in adults and WHR in children and adolescents ($P < 0.050$).

Comparison of the median obestatin level before and after the intervention in the adults and children and adolescents by gender has been shown in table 2. Although the obestatin level increased among the women and decreased among the men, the difference observed was not statistically significant ($P > 0.050$). There was no significant obestatin reduction in children and adolescents ($P > 0.050$).

The level of adiponectin had a non-significant change in adults and children and adolescents ($P > 0.050$) (Table 3).

Table 4 shows the mean of BMI, WC, WHR, and BFP before and after the intervention in adults and children and adolescents by gender. A significant difference was shown between mean of WHR and BFP in boys ($P = 0.040$ and $P = 0.008$, respectively) and BFP in total children and adolescents ($P = 0.006$), but no significant difference was observed in adults. The other indices did not show significant differences before and after the intervention in either of the age groups ($P > 0.050$).

Table 1. Basic characteristics of study participants

Characteristics	Adults				Children and adolescents			
	Women	Men	P	Total	Girls	Boys	P	Total
Number	22	23		45	18	23		41
	Mean ± SD	Mean ± SD		Mean ± SD	Mean ± SD	Mean ± SD		Mean ± SD
Age (year)	40.60 ± 9.80	45.20 ± 10.80	0.160	43.05 ± 10.50	12.50 ± 3.30	12.60 ± 2.90	0.870	12.56 ± 3.06
Weight (kg)	79.10 ± 11.80	86.60 ± 9.50	0.030	82.90 ± 11.20	60.81 ± 16.52	64.29 ± 20.02	0.550	62.76 ± 18.43
BMI (kg/m^2)	31.63 ± 4.30	28.84 ± 2.46	0.010	30.20 ± 3.72	26.30 ± 4.24	26.89 ± 4.59	0.670	26.63 ± 4.40
WC (cm)	96.07 ± 9.78	99.60 ± 15.00	0.140	97.88 ± 7.83	81.28 ± 8.71	86.96 ± 11.52	0.090	84.46 ± 10.65
WHR	0.89 ± 0.08	0.95 ± 0.04	0.001	0.92 ± 0.07	0.84 ± 0.06	0.91 ± 0.04	< 0.001	0.88 ± 0.06
BFP	35.95 ± 5.14	26.40 ± 4.23	< 0.001	31.07 ± 6.70	34.95 ± 4.56	30.34 ± 7.83	0.050	32.20 ± 6.86

BMI: Body mass index; WC: Waist circumference; WHR: Waist-to-hip ratio; BFP: Body fat percentage; SD: Standard deviation

Table 2. Comparing the median of obestatin level before and after the intervention in two groups of adults and children and adolescents based on sex

Obestatin (ng/ml)	Before intervention	After intervention	P
	Median (25^{th}-75^{th})	Median (25^{th}-75^{th})	
Adults			
Women	6.03 (5.24-6.20)	6.12 (5.43-6.86)	0.350
Men	5.58 (4.76-6.44)	5.43 (5.01-5.88)	0.130
Total	5.93 (5.03-6.24)	5.76 (5.11-6.62)	0.200
Children and adolescents			
Girls	0.91 (0.75-1.08)	0.93 (0.80-1.00)	0.570
Boys	3.77 (2.51-8.29)	3.85 (3.08-6.20)	0.320
Total	2.13 (0.94-4.08)	2.86 (2.67-4.52)	0.130

Discussion

The results showed that the education and encouragement interventions did not have a significant effect on anthropometric factors in either of the age groups; however, they did lead to a significant reduction in the WHR and BFP in the boy participants; furthermore, no changes were observed in adiponectin and obestatin levels in either age group.

It has been suggested that adiponectin, which is a peptide derived from fat tissues, is inversely correlated with obesity;[24,25] so that the reduced changes in serum adiponectin largely contribute to obesity-related complications and problems.[26,27] Studies also show that when adiponectin decreases, the body reserves fat in risky regions, such as the muscular tissues, and the fat percentage then increases in these regions.[28] However, no significant reductions were observed in anthropometric indicators in this study, and adiponectin also did not change significantly.

Evidence suggests that when the adiponectin level is high, the body protects itself from the shortage of energy through reserving fat in the fat cells. This fat tissue is first added to the layer beneath the skin, and when adiponectin decreases, the body continues to reserve more fat in certain critical regions, which can lead to inflammation and increased risk of heart disease.[29,30]

Few studies have examined the obestatin peptide and produced contradictory results about plasma obestatin level. A study conducted in 2008 on overweight children and adolescents showed a significantly higher plasma obestatin level in the overweight group compared to the study group with normal weight. That study found that weight loss increased obestatin in the overweight group.[19] Ghanbari-Niaki et al. reported similar findings, only with this difference that the level of obestatin increased significantly after weight loss in their study; while, before their weight loss, the overweight children had lower levels of obestatin compared to the group of children with normal weight.[31]

The present study found that obestatin levels did not change significantly in the adults or children and adolescents; however, the WHR and BFP changed significantly in the boy participants. Obesity and overweight were found to decrease the obestatin level.[32] In a study by Beasley et al., overweight and obese people had lower levels of obestatin compared to people with normal weight.[32]

Table 3. Comparing the mean of adiponectin level before and after the intervention in two groups of adults and children and adolescents based on sex

Adiponectin (ng/ml)	Before intervention	After intervention	P
	Mean ± SD	Mean ± SD	
Adults			
Women	33.10 ± 19.65	31.62 ± 18.11	0.340
Men	29.73 ± 21.60	26.23 ± 19.28	0.280
Total	31.40 ± 20.50	28.88 ± 18.86	0.240
Children and adolescents			
Girls	24.65 ± 12.81	22.22 ± 11.52	0.310
Boys	25.85 ± 13.92	29.73 ± 14.39	0.240
Total	25.32 ± 13.30	25.85 ± 13.11	0.760

SD: Standard deviation

Table 4. Comparing the mean anthropometric characteristics before and after the intervention in two groups of adults and children and adolescents based on sex

Variable	Before intervention	After intervention	P
	Mean ± SD	Mean ± SD	
Adults			
BMI (kg/m^2)			
Women	31.63 ± 4.30	31.43 ± 3.69	0.410
Men	28.84 ± 2.46	28.43 ± 3.29	0.330
Total	30.20 ± 3.72	29.95 ± 3.71	0.180
WC (cm)			
Women	96.07 ± 9.78	96.09 ± 8.97	0.820
Men	99.61 ± 5.00	99.49 ± 8.60	0.630
Total	97.88 ± 7.83	97.75 ± 3.71	0.520
WHR			
Women	0.89 ± 0.08	0.88 ± 0.08	0.730
Men	0.95 ± 0.04	0.96 ± 0.04	0.710
Total	0.92 ± 0.07	0.92 ± 0.07	0.600
BFP			
Women	35.95 ± 5.14	35.77 ± 4.74	0.580
Men	26.40 ± 4.23	26.68 ± 7.84	0.500
Total	31.07 ± 6.70	31.06 ± 8.54	0.840
Children and adolescent			
BMI (kg/m^2)			
Girls	26.30 ± 4.24	26.21 ± 3.90	0.740
Boys	26.89 ± 4.59	26.74 ± 5.03	0.620
Total	26.63 ± 4.40	26.55 ± 4.56	0.540
WC (cm)			
Girls	81.28 ± 8.71	81.16 ± 8.71	0.580
Boys	86.96 ± 11.52	86.43 ± 14.7	0.310
Total	84.46 ± 10.65	84.38 ± 12.60	0.380
WHR			
Girls	0.84 ± 0.06	0.85 ± 0.06	0.530
Boys	0.91 ± 0.04	0.89 ± 0.06	0.040
Total	0.88 ± 0.06	0.87 ± 0.06	0.210
BFP			
Girls	34.59 ± 4.56	33.83 ± 4.99	0.330
Boys	30.34 ± 7.83	26.87 ± 9.07	0.008
Total	32.20 ± 6.16	29.93 ± 8.25	0.006

BMI: Body mass index; WC: Waist circumference; WHR: Waist-to-hip ratio; BFP: Body fat percentage; SD: Standard deviation

In the study by Lippl et al., no significant differences were observed in the obestatin level between the overweight and the thin children; however, the level of obestatin increased significantly in the group of overweight children after their weight loss with diet, but the change was not significant in overweight children who did not lose much weight.[33] Overall, obestatin appears to inhibit overeating and is associated with proven appetite-suppressing effects and thus, plays a key role in weight control.[33] Obestatin and adiponectin can be used as markers of the success of interventions and also to monitor treatments.[34] Obesity is an independent risk factor in adulthood that is closely related to health factors.[35,36] Interventions are necessary for the control, reduction, or at least stabilization of obesity. The results of this study revealed that instructional and incentive interventions cannot reduce overweight and obesity, but can prevent weight gain and control its progressive trend, especially in children and adolescents, who are a more sensitive age group, since the modification of their weight ultimately leads to lower rates of obesity in the society. In a study conducted by Sahota et al. in the United Kingdom for examining the effects of an intervention program on the obesity, the subjects' BMI did not change significantly after the intervention (which is in line with the present findings), but some of their dietary habits were

slightly modified.[37] Studies show that exercise can probably affect obestatin levels.[38] The intervention performed in this study targeted increased physical activity and improved diets through educational materials, CDs, and offering prizes; however, the results showed that even these strategies could not really increase the frequency of physical activity.

Conclusion

The interventions performed did not significantly affect the anthropometric factors in either age group examined in the study; however, they did lead to a significant reduction in the WHR and BFP in boy participants. Moreover, these interventions did not significantly affect serum adiponectin and obestatin levels. Although the instructional and incentive interventions given did not significantly reduce obesity, they are still regarded as helpful due to their inhibitory effect on the increasing trend of obesity. Further long-term comprehensive interventions performed with better facilities are suggested for achieving a maintenance lifestyle modification to decrease obesity indices and increase adiponectin and obestatin levels.

Acknowledgments

Here it is necessary to thank the management of the development of research, evaluation of research, and coordination of research centers that approved the implementation of research project (number 191124) in Isfahan University of Medical Sciences.

Conflict of Interests

Authors have no conflict of interests.

References

1. Calza S, Decarli A, Ferraroni M. Obesity and prevalence of chronic diseases in the 1999-2000 Italian National Health Survey. BMC Public Health 2008; 8: 140.
2. Guh DP, Zhang W, Bansback N, Amarsi Z, Birmingham CL, Anis AH. The incidence of co-morbidities related to obesity and overweight: A systematic review and meta-analysis. BMC Public Health 2009; 9: 88.
3. Sarrafzadegan N, Kelishadi R, Sadri G, Malekafzali H, Pourmoghaddas M, Heidari K, et al. Outcomes of a comprehensive healthy lifestyle program on cardiometabolic risk factors in a developing country: the Isfahan Healthy Heart Program. Arch Iran Med 2013; 16(1): 4-11.
4. Kelishadi R, Mohammadifard N, Sarrazadegan N, Nouri F, Pashmi R, Bahonar A, et al. The effects of a comprehensive community trial on cardiometabolic risk factors in adolescents: Isfahan Healthy Heart Program. ARYA Atheroscler 2012; 7(4): 184-90.
5. World Health Organization. Commission on Ending Childhood Obesity [Online]. [cited 2017]; Available from: URL; https://www.who.int/end-childhood-obesity/en
6. Gao Y, Griffiths S, Chan EY. Community-based interventions to reduce overweight and obesity in China: A systematic review of the Chinese and English literature. J Public Health (Oxf) 2008; 30(4): 436-48.
7. Sarrafzadegan N, Kelishadi R, Siadat ZD, Esmaillzadeh A, Solhpour A, Shirani S, et al. Obesity and cardiometabolic risk factors in a representative population of Iranian adolescents and adults in comparison to a Western population: The Isfahan Healthy Heart Programme. Public Health Nutr 2010; 13(3): 314-23.
8. Sarraf-Zadegan N, Sadri G, Malek Afzali H, Baghaei M, Mohammadi Fard N, Shahrokhi S, et al. Isfahan Healthy Heart Programme: A comprehensive integrated community-based programme for cardiovascular disease prevention and control. Design, methods and initial experience. Acta Cardiol 2003; 58(4): 309-20.
9. Sarrafzadegan N, Kelishadi R, Esmaillzadeh A, Mohammadifard N, Rabiei K, Roohafza H, et al. Do lifestyle interventions work in developing countries? Findings from the Isfahan Healthy Heart Program in the Islamic Republic of Iran. Bull World Health Organ 2009; 87(1): 39-50.
10. Mohammadifard N, Kelishadi R, Safavi M, Sarrafzadegan N, Sajadi F, Sadri GH, et al. Effect of a community-based intervention on nutritional behaviour in a developing country setting: The Isfahan Healthy Heart Programme. Public Health Nutr 2009; 12(9): 1422-30.
11. Abolhassani S, Irani MD, Sarrafzadegan N, Rabiei K, Shahrokhi S, Pourmoghaddas Z, et al. Barriers and facilitators of weight management in overweight and obese people: Qualitative findings of TABASSOM project. Iran J Nurs Midwifery Res 2012; 17(3): 205-10.
12. Abete I, Parra D, Crujeiras AB, Goyenechea E, Martinez JA. Specific insulin sensitivity and leptin responses to a nutritional treatment of obesity via a combination of energy restriction and fatty fish intake. J Hum Nutr Diet 2008; 21(6): 591-600.
13. Seoane LM, Al-Massadi O, Caminos JE, Tovar SA, Dieguez C, Casanueva FF. Sensory stimuli directly acting at the central nervous system regulate gastric ghrelin secretion. An ex vivo organ culture study. Endocrinology 2007; 148(8): 3998-4006.
14. Lee MJ, Fried SK. Integration of hormonal and nutrient signals that regulate leptin synthesis and

secretion. Am J Physiol Endocrinol Metab 2009; 296(6): E1230-E1238.
15. Zhang JV, Ren PG, Avsian-Kretchmer O, Luo CW, Rauch R, Klein C, et al. Obestatin, a peptide encoded by the ghrelin gene, opposes ghrelin's effects on food intake. Science 2005; 310(5750): 996-9.
16. Meier U, Gressner AM. Endocrine regulation of energy metabolism: Review of pathobiochemical and clinical chemical aspects of leptin, ghrelin, adiponectin, and resistin. Clin Chem 2004; 50(9): 1511-25.
17. Zhang MY, Li F, Wang JP. Correlation analysis of serum obestatin expression with insulin resistance in childhood obesity. Genet Mol Res 2017; 16(2).
18. Goodson JM, Kantarci A, Hartman ML, Denis GV, Stephens D, Hasturk H, et al. Metabolic disease risk in children by salivary biomarker analysis. PLoS One 2014; 9(6): e98799.
19. Reinehr T, de Sousa G, Roth CL. Obestatin and ghrelin levels in obese children and adolescents before and after reduction of overweight. Clin Endocrinol (Oxf) 2008; 68(2): 304-10.
20. Zou CC, Liang L, Wang CL, Fu JF, Zhao ZY. The change in ghrelin and obestatin levels in obese children after weight reduction. Acta Paediatr 2009; 98(1): 159-65.
21. NHLBI Obesity Education Initiative. The Practical Guide Identification, Evaluation, and Treatment of Overweight and Obesity in Adults [Online]. [cited 2000]; Available from: URL: https://www.nhlbi.nih.gov/files/docs/guidelines/prctgd_c.pdf
22. Golshiri P, Yarmohammadi P, Sarrafzadegan N, Shahrokhi S, Yazadani M, Pourmoghaddas M. Developing and validating questionnaires to assess knowledge, attitude, and performance toward obesity among Iranian adults and adolescents: Tabassom study. ARYA Atheroscler 2012; 7(Special Issue): S119-S124.
23. World Health Organization, Pan American Health Organization. Maintenance manual for laboratory equipment. 2nd ed. Geneva, Switzerland: World Health Organization; 2008.
24. Kishida K, Funahashi T, Shimomura I. Adiponectin as a routine clinical biomarker. Best Pract Res Clin Endocrinol Metab 2014; 28(1): 119-30.
25. Kishida K, Funahashi T, Matsuzawa Y, Shimomura I. Visceral adiposity as a target for the management of the metabolic syndrome. Ann Med 2012; 44(3): 233-41.
26. Goldstein BJ, Scalia R. Adiponectin: A novel adipokine linking adipocytes and vascular function. J Clin Endocrinol Metab 2004; 89(6): 2563-8.
27. Smith J, Al-Amri M, Sniderman A, Cianflone K. Leptin and adiponectin in relation to body fat percentage, waist to hip ratio and the apoB/apoA1 ratio in Asian Indian and Caucasian men and women. Nutr Metab (Lond) 2006; 3: 18.
28. Kamada Y, Matsumoto H, Tamura S, Fukushima J, Kiso S, Fukui K, et al. Hypoadiponectinemia accelerates hepatic tumor formation in a nonalcoholic steatohepatitis mouse model. J Hepatol 2007; 47(4): 556-64.
29. Esposito K, Pontillo A, Di Palo C, Giugliano G, Masella M, Marfella R, et al. Effect of weight loss and lifestyle changes on vascular inflammatory markers in obese women: A randomized trial. JAMA 2003; 289(14): 1799-804.
30. Coughlin CC, Finck BN, Eagon JC, Halpin VJ, Magkos F, Mohammed BS, et al. Effect of marked weight loss on adiponectin gene expression and plasma concentrations. Obesity (Silver Spring) 2007; 15(3): 640-5.
31. Ghanbari-Niaki A, Saghebjoo M, Rahbarizadeh F, Hedayati M, Rajabi H. A single circuit-resistance exercise has no effect on plasma obestatin levels in female college students. Peptides 2008; 29(3): 487-90.
32. Beasley JM, Ange BA, Anderson CA, Miller Iii ER, Holbrook JT, Appel LJ. Characteristics associated with fasting appetite hormones (obestatin, ghrelin, and leptin). Obesity (Silver Spring) 2009; 17(2): 349-54.
33. Lippl F, Erdmann J, Lichter N, Tholl S, Wagenpfeil S, Adam O, et al. Relation of plasma obestatin levels to bmi, gender, age and insulin. Horm Metab Res 2008; 40(11): 806-12.
34. Hirose H, Yamamoto Y, Seino-Yoshihara Y, Kawabe H, Saito I. Serum high-molecular-weight adiponectin as a marker for the evaluation and care of subjects with metabolic syndrome and related disorders. J Atheroscler Thromb 2010; 17(12): 1201-11.
35. Salmon J, Ball K, Crawford D, Booth M, Telford A, Hume C, et al. Reducing sedentary behaviour and increasing physical activity among 10-year-old children: Overview and process evaluation of the 'Switch-Play' intervention. Health Promot Int 2005; 20(1): 7-17.
36. Sarrafzadegan N, Rabiei K, Nouri F, Mohammadifard N, Moattar F, Roohafza H, et al. Parental perceptions of weight status of their children. ARYA Atheroscler 2013; 9(1): 61-9.
37. Sahota P, Rudolf MC, Dixey R, Hill AJ, Barth JH, Cade J. Randomised controlled trial of primary school based intervention to reduce risk factors for obesity. BMJ 2001; 323(7320): 1029-32.
38. United States, Department of Health and Human Services. Healthy People 2010: Understanding and Improving Health. Burlington, MA: Jones and Bartlett Publishers; 2000.

DOI: http://dx.doi.org/10.22122/arya.v15i3.1688 Published by Vesnu Publications

Tricuspid annular plane systolic excursion is correlated with poor outcome in surgery for rheumatic heart valvular disease

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Original Article

Abstract

BACKGROUND: Right ventricular (RV) function is a major determinant of clinical outcome, but its function indices have not been studied well in surgery for rheumatic valvular heart disease. The aim of this study was to determine the correlation of tricuspid annular plane systolic excursion (TAPSE) with outcome of rheumatic heart valve surgery.

METHODS: A prospective comparative study was conducted including 100 eligible patients who were divided into two groups based on RV function as assessed by TAPSE measured by two-dimensional (2D) echocardiography preoperatively. Those with TAPSE less than 15 mm were included in group 1 and those with TAPSE of 15 or more were included in group 2.

RESULTS: 50 patients were included in group 1 and 50 patients in group 2. Mean age of the patients was 56.78 ± 15.21 years in group 1 and 54.46 ± 15.03 years in group 2 (P = 0.444). 34 (34%) patients underwent aortic valve replacement (AVR), 35 (35%) underwent both aortic and mitral valves replacement, and 31 (31%) ones had mitral valve replacement (MVR). A significant difference was found between the duration of ventilation (5.15 ± 2.80 hours in group 1 vs. 3.72 ± 2.71 hours in group 2, P = 0.001), postoperative inotropic requirement [more than 24 hours in 18 (36%) patients in group 1 vs. 7 (14%) patients in group 2, P = 0.003], total intensive care unit (ICU) stay (8.92 ± 3.62 days in group 1 vs. 5.20 ± 2.06 days in group 2, P = 0.001), and mortality [7 (14%) in group 1 vs. 2 (4%) in group 2, P = 0.038].

CONCLUSION: TAPSE less than 15 mm in patients undergoing surgical correction for rheumatic valvular heart disease leads to poor outcomes. These patients need special attention perioperatively.

Keywords: Rheumatic Heart Diseases, Right Ventricle, Cardiac Surgery

Date of submission: 10 Oct. 2017, ***Date of acceptance:*** 07 Feb. 2019

Introduction

Rheumatic heart disease (RHD) is still a public health issue in underdeveloped countries especially Indo-Pak subcontinent and Africa. It has been estimated recently that worldwide 15.6 million people have RHD and there are 470000 new cases of rheumatic fever and 233000 deaths attributable to rheumatic fever or RHD each year.[1] The severe forms of the disease will ultimately require surgical intervention in the form of repair or replacement of the affected valve.

Various parameters of surgical outcome have been studied in these patients. The function of right ventricle (RV) affects outcome in valvular heart diseases. Pinzani et al. reported that the presence of RV failure led to an increase in mortality from 5% to 11% in the perioperative period and from 8% to 22% during follow up.[2] However, the knowledge about indices of dysfunction of the RV and surgical outcome is limited.[3]

How to cite this article: Khan I, Shahbaz A, Iqbal M, Khan AR, Riaz W, Sayyed MS, et al. **Tricuspid annular plane systolic excursion is correlated with poor outcome in surgery for rheumatic heart valvular disease.** ARYA Atheroscler 2019; 15(3): 130-5.

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Variables of RV function have not yet been included in large-scale risk stratification models like Parsonnet score (PS) and EuroECORE. Variables studied in various studies are enlarged RV, tricuspid regurgitation (TR), pericardial effusion, and myocardial performance index (Tei index) as well as fractional area change (FAC).[4] Tricuspid annular plane systolic excursion (TAPSE) is a simple feasible marker of RV dysfunction (RVD). It is a valuable prognostic marker in various cardiac diseases including heart failure (HF). It is also a valuable marker of measuring the function of RV.[5] A rise in pulmonary hypertension (PH), secondary to aortic or mitral valve disease, leads to dilatation of the RV and decreased function. This eventually leads to enlargement of the tricuspid annulus (TA) and TR.

Measurement of TAPSE involves the displacement of tricuspid valve (TV) ring in the longitudinal direction of the RV during systole using an M-mode ultrasound technique through an apical four-chamber view. It has low dependence on the ultrasound image quality and requires no specific ultrasound equipment and analysis software for its measurement.

Our study investigates the usefulness of TAPSE as a tool for outcome measures in surgery for rheumatic valvular disease. A better understanding of the predictors of outcome can lead to changes in operative and postoperative strategies.

Materials and Methods

A prospective comparative study was conducted at the Department of Cardiac Surgery, Punjab Institute of Cardiology, Lahore, Pakistan, from August 2015 to August 2016. The study protocol was approved by the ethical review board and individual consent from the patients was waivered. A total of 100 consecutive patients who underwent surgery for rheumatic valvular disease, both mitral and aortic, were included in the study through purposive sampling. Patients who had an additional procedure, were operated in emergency, and those with atrial fibrillation (AF) and redo procedures were excluded from the study. All the patients underwent valve replacement with either mechanical or bioprosthetic valves. The choice of the type of valve prosthesis was left to the operating surgeon. All the surgeries were performed by the two senior authors, Ahmad Shahbaz and Waseem Riaz.

Patients were divided into two groups based on the function of the RV as assessed by TAPSE measured by two-dimensional (2D) echocardiography preoperatively. TAPSE was measured through an apical four-chamber view; placing the cursor of the M-mode through the junction of the TV plane and RV free wall, and the difference in the displacement of RV base during systole and diastole was noted. Those with TAPSE less than 15 mm were included in group 1 and those with TAPSE of 15 mm or more were included in group 2. All the important demographic variables and important echocardiographic parameters were recorded on preformed proformas. The two groups were followed up till discharge. The in-hospital outcomes were recorded and compared statistically.

Data were analysed using SPSS software (version 17, SPSS Inc., Chicago, IL, USA). Continuous variables were summarized as mean ± standard deviation (SD) and categorical variables were summarized as frequency and percentage.

Differences in baseline demographic characteristics, and echocardiographic and operative data between patients in both groups were assessed using t-test for continuous data and chi-square test for categorical data. Fisher's exact test was used for categorical variables where the frequencies were small. A two-tailed P-value less than 0.050 was considered significant.

Preoperative and major postoperative outcome variables were recorded and data were analysed using SPSS software. Shapiro-Wilk test was used to ascertain the normality of the data because of the relatively small number of patients.

Results

50 patients were included in group 1 and 50 patients in group 2. Mean age of the patients was 56.78 ± 15.21 years in group 1 and 54.46 ± 15.03 years in group 2. 34 (34%) patients underwent aortic valve replacement (AVR), 35 (35%) patients underwent both aortic and mitral valves replacement, and 31 (31%) patients underwent mitral valve replacement (MVR). Groups 1 and 2 had similar demographic and clinical variables (Table 1). Patients presenting with New York Heart Association (NYHA) class IV dyspnoea were 12 (24%) in group 1 and 9 (18%) in group 2. Overall, the P-value for various degrees of NYHA class in both groups was 0.687.

Preoperative echocardiographic data of patients in both groups is shown in table 2. The left ventricular ejection fraction (LVEF) was 56.32 ± 6.90% in group 1 and 53.98 ± 9.52% in group 2 (P = 0.162). Preoperative RV size was 25.71 ± 7.44 mm in group 1 and 26.10 ± 5.02 mm in group 2 (P = 0.759).

Table 1. Demographic variables of the two study groups

Variable		Groups		P
		Group 1 (TAPSE < 15) (n = 50)	Group 2 (TAPSE ≥ 15) (n = 50)	
Gender [n (%)]	Male	20 (40)	27 (54)	0.228
	Female	30 (60)	23 (46)	
NYHA class [n (%)]	I	9 (18)	8 (16)	0.687
	II	29 (58)	33 (66)	
	III	12 (24)	9 (18)	
Hypertension [n (%)]		4 (9)	3 (6)	> 0.999
Diabetes [n (%)]		17 (34)	9 (18)	0.110
Weight (kg) (mean ± SD)		62.00 ± 10.31	57.66 ± 9.70	0.032
BSA (m^2) (mean ± SD)		4.90 ± 2.30	2.30 ± 5.40	0.002
Preoperative creatinine (mg/dl) (mean ± SD)		0.85 ± 0.21	0.81 ± 0.24	0.377
Age (year) (mean ± SD)		56.78 ± 15.21	54.46 ± 15.03	0.444
Height (cm) (mean ± SD)		156.09 ± 19.30	154.12 ± 20.30	0.620

P-value less than 0.050 was considered significant
TAPSE: Tricuspid annular plane systolic excursion; NYHA: New York Heart Association; BSA: Body surface area; SD: Standard deviation

Preoperative pulmonary arterial pressure was significantly high in group 1 (38.00 ± 13.44 mmHg) compared to group 2 (32.90 ± 12.30 mmHg) (P = 0.051).

Mechanical valves were used in 37 (74%) of the group 1 patients and 42 (84%) of the group 2 patients. Bioprosthetic valves were used in 13 (26%) of the group 1 patients and 8 (16%) of the group 2 patients (Table 3). No significant difference was noted between the groups with respect to the type of prosthetic valves used (P = 0.210). The important intraoperative variables like cardiopulmonary bypass (CPB) time and cross-clamp time were non-significant between the two groups (P = 0.809 and P = 0.890, respectively). Among the major postoperative outcome variables, duration of ventilation was 5.15 ± 2.80 hours in group 1 vs. 3.72 ± 2.71 hours in group 2 (P = 0.001), postoperative inotropic requirement for more than 24 hours was 18 (36%) in group 1 vs. 7 (14%) in group 2 (P = 0.003), total intensive care unit (ICU) stay was 8.92 ± 3.62 days in group 1 vs. 5.20 ± 2.06 days in group 2 (P = 0.001) and mortality was 7 (14%) in group 1 vs. 2 (4%) in group 2 (Table 3).

Discussion

RVD carries a poor prognosis in patients undergoing open heart surgery. RVD particularly is a strong predictor of poor outcome in patients undergoing surgery for rheumatic valvular disease.[3]

Table 2. Preoperative echocardiographic variables

Variable		Groups		P
		Group 1 (TAPSE < 15) (n = 50)	Group 2 (TAPSE ≥ 15) (n = 50)	
Preoperative TR [n (%)]	Mild	18 (36)	22 (44)	0.230
	Moderate	17 (34)	15 (30)	
	Severe	15 (30)	13 (26)	
Preoperative RV size (mm) (mean ± SD)		25.71 ± 7.44	26.10 ± 5.02	0.759
Preoperative LVIDD (mm) (mean ± SD)		51.46 ± 4.20	49.32 ± 4.27	0.143
Preoperative LVIDS (mm) (mean ± SD)		36.87 ± 12.39	35.72 ± 11.93	0.637
Preoperative LVPWD (mm) (mean ± SD)		9.14 ± 2.56	9.43 ± 2.06	0.534
Pulmonary arterial pressure (mmHg) (mean ± SD)		38.00 ± 13.44	32.90 ± 12.30	0.051
LVEF (%) (mean ± SD)		56.32 ± 6.90	53.98 ± 9.52	0.162

P-value less than 0.050 was considered significant
TAPSE: Tricuspid annular plane systolic excursion; TR: Tricuspid regurgitation; RV: Right ventricle; LVIDD: Left ventricular internal diastolic dimension; LVIDS: Left ventricular internal dimension in systole; LVPWD: Left ventricular posterior wall dimension; LVEF: Left ventricular ejection fraction; SD: Standard deviation

Table 3. Intraoperative data and postoperative outcome variables

Variable		Groups		P
		Group 1 (TAPSE < 15) (n = 50)	Group 2 (TAPSE ≥ 15) (n = 50)	
Operation type [n (%)]	AVR	12 (24)	22 (44)	0.107
	DVR	20 (40)	15 (30)	
	MVR	18 (36)	13 (30)	
Type of valve used [n (%)]	Mechanical	37 (74)	42 (84)	0.210
	Bioprosthetic	13 (26)	8 (16)	
Postoperative inotropic requirement > 24 hours [n (%)]		18 (36)	7 (14)	0.003
Mortality [n (%)]		7 (14)	2 (4)	0.038
Cross-clamp time (minute) (mean ± SD)		70.85 ± 24.79	71.57 ± 27.30	0.890
Bypass time (minute) (mean ± SD)		107.89 ± 38.80	106.00 ± 39.57	0.809
Duration of ventilation (hour) (mean ± SD)		5.17 ± 2.80	3.72 ± 2.71	0.001
Total ICU stay (day) (mean ± SD)		8.92 ± 3.62	5.20 ± 2.06	0.001
Hospital stay (day) (mean ± SD)		11.72 ± 4.12	6.27 ± 3.46	0.001

P-value less than 0.050 was considered significant

TAPSE: Tricuspid annular plane systolic excursion; AVR: Aortic valve replacement; DVR: Double (both aortic and mitral) valve replacement; MVR: Mitral valve replacement; ICU: Intensive care unit; SD: Standard deviation

These patients present either a pressure overload or a volume overload scenario to the RV. In case of mitral stenosis (MS) and mitral regurgitation (MR), it is the PH that causes a pressure overload in the RV; while in TR, it is the volume overload that causes RVD. The RV adapts better to volume overload than pressure overload. RHD mostly presents a pressure overload context in the form of PH. A moderate to severe rise in pulmonary pressure leads to RV dilatation and in the long run to failure.[6]

Because of the complex three-dimensional (3D) geometry of the RV, identifying and accurately classifying its function can be difficult. The current markers are well studied for end stage disease only.[7]

Although our understanding of RV function is increasing with the advent of newer modalities like tissue Doppler imaging (TDI) and speckle tracking echocardiography (STE), most of these modalities, however, require good experience. 3D echocardiography has been widely studied as a method to assess RV volume and is used to evaluate RV function.[8] However, it also has limitations like limited acoustic windows for imaging because of an intricate 3D anatomical structure of the RV. Cardiovascular magnetic resonance (CMR) imaging has been useful for anatomical assessment of the RV. In a study by van Wolferen et al., the predictors of 1-year survival in patients with PH were RV volume, RV myocardial mass, and stroke index measured by CMR.[9]

TAPSE is a well-known echocardiographic parameter. It measures the apex to base shortening of RV during systole. The transverse diameter of RV is much smaller compared to LV. But the surface-to-volume ratio is much larger for the RV than LV. Therefore, smaller changes in the transverse dimension will cause large increase in ejection. For this reason, to achieve a normal ejection, the change in dimension will have to be in another dimension. Thus, changes in transverse dimensions did not correlate with RVEF, while TAPSE did in a study by Kaul et al.[10] TAPSE is simple to measure and it has low dependence on ultrasound image quality. It has been studied well in the context of HF and other conditions like pulmonary embolectomy.[11] However, TAPSE as a predictor for poor outcome in rheumatic valve surgery has not been studied well.

Various values or cut-off values for TAPSE have been studied by different investigators. Schmid et al. used 18 mm cut-off value.[11] Tamborini et al. suggested 17 mm value.[12] On the other hand, lower values have also been used in many studies. Foale et al. defined mild RVD as TAPSE Of 13-15 mm.[13] Horton et al. stated a TAPSE more than 15 mm as normal.[14] For this reason, we used 15 mm as the cut-off value for defining RVD.

Kjaergaard et al. demonstrated a worse median term outcome in terms of mortality for patients with decreased TAPSE in patients with HF.[15] Similar results were presented by Forfia et al. in patients with PH.[16] Samad et al. reported a significantly increased mortality in patient with TAPSE of less than 15 mm.[17] Our study also showed an increased early mortality when TAPSE was decreased in patients with RHD after surgery. The relation of TAPSE with worse outcome in

RHD was also demonstrated by Pande et al.[3] They noted that RVD could still exist in valvular patients even in the absence of significant PH. This can be attributed to ventricular interdependence and RV ischemia. This might be the reason why PH was non-significant between the two groups in our study.

RVD is associated with an increased ventilation time, more blood transfusions, and a longer stay in the ICU.[17]Our study also showed an increased postoperative ventilation time and ICU stay in patients with TAPSE less than 15 mm. Sun et al.[18] found no difference in outcome in terms of hospital stay. This is likely due to the variety of surgical pathologies in our patients compared to patients with only TV pathology in the study by Sun et al.[18]

This single centre experience with a comparatively small sample size certainly has limitations. We did not present any long-term follow up. It will certainly be interesting to see the long-term follow up of these patients dichotomized only based on TAPSE. Moreover, the small number of postoperative events did not allow the development of a regression model to see independent predictors of outcome and comparison with TAPSE. Moreover, bias cannot be ruled out in the reporting of echocardiographies as they were performed by different echocardiographers from our cardiology department.

Conclusion

TAPSE less than 15 mm in patients undergoing surgery for rheumatic valvular disease leads to poor outcomes. These patients need special attention perioperatively. TAPSE can be used to identify these patients early and so, can lead to changes in management strategies for a better outcome. Large-scale studies are needed for further understanding of the predictive value of TAPSE in rheumatic valvular disease.

Acknowledgments

We wish to thank Dr. Farrukh Maqbool for his insights on the echocardiographic data. This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflict of Interests

Authors have no conflict of interests.

References

1. Carapetis JR, Steer AC, Mulholland EK, Weber M. The global burden of group A streptococcal diseases. Lancet Infect Dis 2005; 5(11): 685-94.
2. Pinzani A, de Gevigney G, Pinzani V, Ninet J, Milon H, Delahaye JP. Pre- and postoperative right cardiac insufficiency in patients with mitral or mitral-aortic valve diseases. Arch Mal Coeur Vaiss 1993; 86(1): 27-34.
3. Pande S, Agarwal SK, Dhir U, Chaudhary A, Kumar S, Agarwal V. Pulmonary arterial hypertension in rheumatic mitral stenosis: Does it affect right ventricular function and outcome after mitral valve replacement? Interact Cardiovasc Thorac Surg 2009; 9(3): 421-5.
4. McLaughlin VV, Archer SL, Badesch DB, Barst RJ, Farber HW, Lindner JR, et al. ACCF/AHA 2009 expert consensus document on pulmonary hypertension a report of the American College of Cardiology Foundation Task Force on Expert Consensus Documents and the American Heart Association developed in collaboration with the American College of Chest Physicians; American Thoracic Society, Inc.; and the Pulmonary Hypertension Association. J Am Coll Cardiol 2009; 53(17): 1573-619.
5. Mohammed SF, Hussain I, AbouEzzeddine OF, Takahama H, Kwon SH, Forfia P, et al. Right ventricular function in heart failure with preserved ejection fraction: A community-based study. Circulation 2014; 130(25): 2310-20.
6. Haddad F, Doyle R, Murphy DJ, Hunt SA. Right ventricular function in cardiovascular disease, part II: Pathophysiology, clinical importance, and management of right ventricular failure. Circulation 2008; 117(13): 1717-31.
7. Simon MA, Pinsky MR. Right ventricular dysfunction and failure in chronic pressure overload. Cardiol Res Pract 2011; 2011: 568095.
8. Schindera ST, Mehwald PS, Sahn DJ, Kececioglu D. Accuracy of real-time three-dimensional echocardiography for quantifying right ventricular volume: Static and pulsatile flow studies in an anatomic in vitro model. J Ultrasound Med 2002; 21(10): 1069-75.
9. van Wolferen SA, Marcus JT, Boonstra A, Marques KM, Bronzwaer JG, Spreeuwenberg MD, et al. Prognostic value of right ventricular mass, volume, and function in idiopathic pulmonary arterial hypertension. Eur Heart J 2007; 28(10): 1250-7.
10. Kaul S, Tei C, Hopkins JM, Shah PM. Assessment of right ventricular function using two-dimensional echocardiography. Am Heart J 1984; 107(3): 526-31.
11. Schmid E, Hilberath JN, Blumenstock G, Shekar PS, Kling S, Shernan SK, et al. Tricuspid annular plane systolic excursion (TAPSE) predicts poor outcome in patients undergoing acute pulmonary embolectomy. Heart Lung Vessel 2015; 7(2): 151-8.

12. Tamborini G, Pepi M, Galli CA, Maltagliati A, Celeste F, Muratori M, et al. Feasibility and accuracy of a routine echocardiographic assessment of right ventricular function. Int J Cardiol 2007; 115(1): 86-9.
13. Foale R, Nihoyannopoulos P, McKenna W, Kleinebenne A, Nadazdin A, Rowland E, et al. Echocardiographic measurement of the normal adult right ventricle. Br Heart J 1986; 56(1): 33-44.
14. Horton KD, Meece RW, Hill JC. Assessment of the right ventricle by echocardiography: A primer for cardiac sonographers. J Am Soc Echocardiogr 2009; 22(7): 776-92.
15. Kjaergaard J, Akkan D, Iversen KK, Kober L, Torp-Pedersen C, Hassager C. Right ventricular dysfunction as an independent predictor of short- and long-term mortality in patients with heart failure. Eur J Heart Fail 2007; 9(6-7): 610-6.
16. Forfia PR, Fisher MR, Mathai SC, Housten-Harris T, Hemnes AR, Borlaug BA, et al. Tricuspid annular displacement predicts survival in pulmonary hypertension. Am J Respir Crit Care Med 2006; 174(9): 1034-41.
17. Samad BA, Alam M, Jensen-Urstad K. Prognostic impact of right ventricular involvement as assessed by tricuspid annular motion in patients with acute myocardial infarction. Am J Cardiol 2002; 90(7): 778-81.
18. Sun X, Ellis J, Kanda L, Corso PJ. The role of right ventricular function in mitral valve surgery. Heart Surg Forum 2013; 16(3): E170-E176.

DOI: http://dx.doi.org/10.22122/arya.v15i3.1303 Published by Vesnu Publications

Macronutrient intake and physical activity levels in individuals with and without metabolic syndrome: An observational study in an urban population

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Short Communication

Abstract

BACKGROUND: We aimed to compare dietary macronutrient intake and physical activity level (PAL) between community-based samples of Iranian adults with metabolic syndrome (MetS+) and without metabolic syndrome (MetS-).

METHODS: This cross-sectional study was conducted among 3800 men and women aged 35-65 years. The International Diabetes Federation (IDF) criteria were used to define MetS. A 24-hour recall was used to evaluate dietary intake. The James and Schofield human energy requirements equations were used to calculate PAL and questions were categorized into time spent on activities during work (including housework), during non-work time, and in bed.

RESULTS: The mean ± standard deviation (SD)age of the MetS+ and MetS- subjects was, respectively, 48.8 ± 7.8 years (521 men and 1178 women) and 47.6 ± 7.5 years (714 men and 1222 women) (P = 0.930). The mean energy intake was higher in the MetS+ men compared with MetS- men (1977.4 ± 26.6 vs. 1812.7 ± 21.7 Kcal; P < 0.001). Crude and energy-adjusted intake from total fat was lower in MetS+ women compared with MetS- women (both P ≤ 0.010). PALs were lower in MetS+ compared with MetS- participants (P < 0.001). After adjusting for confounders, no significant association was observed between the intake of individual macronutrients and MetS. In contrast, PAL was inversely associated with the incidence of MetS [OR = 0.34 (95% CI: 0.17-0.57); P < 0.001].

CONCLUSION: In the current study, there was an inverse relationship between PAL and the risk of MetS, but no association between individual dietary macronutrients intake and the incidence of MetS.

Keywords: Nutritional Assessment, Basal Metabolic Rate, Physical Activity

Date of submission: 22 Oct. 2018, ***Date of acceptance:*** 13 Jan. 2019

Introduction

Metabolic syndrome (MetS) as defined by the International Diabetes Federation (IDF) criteria is characterized by waist circumference ≥ 80 and ≥ 94 in women and men, respectively, and 2 or more of the following criteria: increased blood pressure, impaired glucose tolerance, hypertriglyceridemia,

How to cite this article: Motamed S, Mazidi M, Safarian M, Ghayour-Mobarhan M Moohebati M, Ebrahimi M, et al. **Macronutrient intake and physical activity levels in individuals with and without metabolic syndrome: An observational study in an urban population.** ARYA Atheroscler 2019; 15(3): 136-45.

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and low concentrations of serum high-density lipoprotein (HDL) cholesterol.[1] MetS is an important risk factor for mortality and morbidity associated with several chronic conditions including cardiovascular disease (CVD), diabetes mellitus (DM), and some cancers.[2-6] In American adults, the prevalence of MetS has been reported as 34%.[7] The number of Iranian women with MetS (55%) is higher compared to men (30.1%).[8]

Several factors are known to contribute to the etiology of MetS.

Several studies have showed that lifestyle factors such as an inappropriate diet and physical activity level (PAL) have a key role in the development of MetS.[2,9] An energy restricted diet including a suitable distribution of macronutrients coupled with moderate PAL is associated with an improvement in several risk factors of MetS and it has been shown that it delays the beginning of complications.[10]

A macronutrient diet evidently has an important effect on obesity via affecting several metabolic processes, appetite, and thermogenesis.[11] Additionally, macronutrient oxidation and total energy expenditure in obese subjects can be affected by changes in the content of the diet.[11]

Some studies have reported that a high carbohydrate intake is associated with low levels of serum high-density lipoprotein cholesterol (HDL-C) and increased plasma triacylglycerol concentration.[12,13] Moreover, Kim et al. suggested that both the quantity and quality of carbohydrate intake have a positive relationship with the risk of MetS in women, although this relationship was dependent on body mass index (BMI).[14] Additionally, there are several studies that support a relationship between a high-fat diet (HFD) and the presence of obesity; this may be due to hyperphagia,[15,16] or a reduction in sensitivity to satiety hormones (e.g., cholecystokinin).[17] A HFD is also more likely to result in a positive energy balance.[18] HFD-related postprandial insulin resistance (IR) is importantly mediated by impairment of parasympathetic-dependent insulin action, which is associated with adiposity.[19] A sedentary lifestyle with a low PAL also plays a vital role in the constellation of risk factors associated with MetS.[20,21] Adequate leisure-time physical activity is important in preventing MetS phenotypes.[20] Exercise reduces abdominal adiposity, improves insulin action and HDL-C levels, and reduces the risk of type 2 diabetes even without weight reduction.[22,23]

Despite the fact that the association between several aspects of diet and components of MetS (obesity, high blood pressure, dyslipidemia, and glucose intolerance) has been investigated, few observational studies that have evaluated the association of macronutrient intake and physical activity levels with the presence of MetS. Furthermore, the results of the limited data that have been published are not conclusive and cannot be extrapolated to the Iranian population. Therefore, the aim of this study was to assess the relationship between macronutrient intake and physical activity levels, and the presence of MetS in order to provide evidences for lifestyle modification as an important factor related to MetS.

Materials and Methods

A population of 3800 subjects aged 35-65 years was selected from Mashhad Stroke and Heart Atherosclerotic Disorders (MASHAD: 2010-2020) Study, Mashhad, Iran, using stratified-cluster method.[24-27] The following subjects were excluded: pregnant women, individuals with CVD, diabetes mellitus, or other metabolic disease, and those taking dietary supplements. The definition of MetS was based on the definition of the IDF.[1] Each participant provided a written informed consent form, and the study was approved by the ethics committee of Mashhad University of Medical Sciences, Iran.

Anthropometric parameters (weight, height, and waist circumference) were measured using standard protocols on which detailed information is presented in previous studies.[24-27] Blood pressure (BP) was measured twice 30 minutes apart in the seated position in a participant at rest for at least 15 minutes using a standard mercury sphygmomanometer calibrated according to the Iranian Institute of Standards and Industrial Research. BMI was calculated as weight (kg) divided by height squared (m^2).

More information on measurements is provided elsewhere.[24-27] A full fasting lipid profile, comprising total cholesterol, triglycerides, HDL-C, and low-density lipoprotein cholesterol (LDL-C), was determined for each participant. Serum lipid and fasting blood sugar (FBS) concentrations were measured using enzymatic methods.

A 24-hour recall questionnaire was used to collect information on food and drink consumed over the last 24 hours.[2] To assess the nutritional intake of the subjects, Dietplan6 software (Forestfield Software Ltd., UK) was used to analyze macronutrient and micronutrient intake. The total energy intake and the values of crude and energy-

adjusted intake of all macronutrients were reported in this study. Macronutrients were considered as a percentage of total caloric intake.

PAL was evaluated by a physical activity questionnaire. PAL was calculated as the total energy expenditure (TEE) as a ratio of the basal metabolic rate (BMR) over a 24-hour period.[2] The questions on physical activity were divided into time spent on activities during work, during non-work time, and in bed.[28] Physical activity level at work was scored using an ascending scale of intensity.[29] The sum of all scores constitutes the overall PAL. The participants were divided into quartiles within total PAL.

SPSS software (version 16.0, SPSS Inc., Chicago, IL, USA) was used to analyze the data. The histogram and Kolmogorov-Smirnov test were applied to check the normal distribution of continuous variables. Categorical variables were compared using chi-square test. Continuous data with normal distribution were presented as mean and standard deviation (SD) or median and interquartile range in the case of skewed distribution, and categorical data were presented as frequency or percentage (%). Nutrient intake adjustment for total energy intake was done via residual method.[30] Energy-adjusted nutrient intakes were obtained as the residuals from the regression model in which absolute nutrient intake and total energy intake were considered as the dependent and independent variable, respectively.[30] Qualitative variables were compared using chi-square test. The normally distributed data (data were presented as mean ± SD) of the participants with and without MetS were compared using Student's t-test. For variables with non-normal distribution (data was presented as the median and interquartile range) Mann-Whitney test was applied.

Logistic regression was applied to calculate the odds ratios (ORs) and 95% confidence intervals (CIs) for MetS (lowest quintile considered as that reported by Freire et al.[30]). Models were corrected for sex, age, smoking, PAL, total energy, BMI, and past medical history. Trend analysis was performed by assigning the median intake for each nutrient to individuals, then, considering it as a continuous variable in logistic regression.[30] A two-sided $P \leq 0.050$ was used to characterize significant results.

Results

Of the 3800 (aged 35 to 65 years) participants in the final analytic sample, 1699 (44%) had MetS. The prevalence of MetS was 42% (n = 521) in men and 49% (n = 1178) in women (P = 0.001). MetS+ participants were older than MetS- participants, but age differences reached significance only in women (48.7 vs. 46.9 years; P < 0.001). Systolic BP (SBP), diastolic BP (DBP), waist girth, blood glucose, total cholesterol and triglycerides, uric acid, and C-reactive protein (CRP) (all P < 0.001) were significantly higher and HDL cholesterol was significantly lower (P < 0.001) in both men and women with MetS compared with those without MetS. Furthermore, MetS+ men had higher BMI than their MetS- counterparts (128.9 vs. 118.8 kg/m^2; P < 0.001), while the MetS+ women group had higher LDL cholesterol (126.4 vs. 123.5 mg/dl; P = 0.040) and higher prevalence of smokers (21% vs. 14%; P < 0.001) compared with the MetS- women group (Table 1). Significant gender × MetS interactions were apparent for smoking, and HDL and LDL cholesterol.

The mean values of total energy intake and crude intake of all macronutrients were higher in men with MetS than in those without MetS (all P < 0.001). However, there were no differences in energy-adjusted intake of macronutrients between the two groups (P > 0.050). Surprisingly, the crude intake of total fat (P < 0.010), saturated fatty acid (P < 0.050), and monounsaturated fatty acid (P < 0.010) was higher in women without MetS. Energy-adjusted intake of total fat was significantly different between MetS+ and MetS- subgroups in women (P = 0.010). Gender × MetS interactions were significant for crude intake of total fat, and crude and total energy-adjusted intake of poly-unsaturated fatty acid.

Once correction (sex, age, PAL, smoking, past medical history, energy intake, and BMI) was carried out, it appears that the likelihood of having MetS was no longer significant for the intake of macronutrients (P > 0.050 for all) (Table 2). The ORs of MetS decreased across quartiles of PAL with the top quartile of PAL being associated with an OR of 0.34 (95% CI: 0.17-0.57) relative to the lowest quartile (Table 3).

Discussion

PALs were lower in the subjects with MetS compared subjects without MetS. However, adjusted total macronutrient intake had no significant association with the presence of MetS.

MetS+ subjects had higher levels of serum high sensitivity C-reactive protein (hs-CRP) and uric acid concentration.

Table 1. Demographic, cardiometabolic factors, and daily intake characteristics of subjects with and without metabolic syndrome

Characteristic	Total			Men			Women			Gender* MetS interaction P
	MetS+ (n = 1699)	MetS− (n = 1936)	P	MetS+ (n = 521)	MetS− (n = 714)	P	MetS+ (n = 1178)	MetS− (n = 1222)	P	
Education level			0.750			0.625			0.382	0.732
< 1 year (%)	299 (17.60)	292 (15.08)		40 (7.0)	49 (6.8)		259 (21.9)	243 (19.9)		
1-8 years (%)	822 (48.30)	897 (46.30)		218 (41.8)	288 (40.3)		604 (51.4)	609 (49.9)		
> 8 years (%)	578 (34.00)	747 (38.50)		263 (50.4)	377 (52.9)		315 (26.7)	370 (30.2)		
Current cigarette smoking			0.440			0.396			< 0.001	< 0.050
Yes (%)	389 (22.80)	387 (20.00)		139 (28.0)	209 (24.0)		250 (21.0)	178 (14.0)		
Physical activity level (%)			< 0.001			< 0.010			< 0.010	0.192
Sedentary (%)	1319 (77.63)	1118 (57.70)		415 (80)	416 (58.2)		904 (76.0)	702 (58.0)		
Low activity (%)	289 (17.01)	561 (28.97)		73 (14.0)	164 (23.2)		216 (18.0)	397 (33.0)		
Active (%)	68 (4.00)	196 (10.10)		21 (4.1)	88 (12.4)		47 (3.0)	108 (9.0)		
Very active (%)	20 (1.10)	59 (3.00)		10 (1.9)	44 (6.2)		10 (0.8)	15 (1.2)		
Age	48.79 ± 7.93	47.66 ± 7.65	< 0.001	49.10 ± 7.6	48.90 ± 8.00	0.694	48.70 ± 8.06	46.90 ± 7.30	< 0.001	0.939
Weight (kg)	75.97 ± 12.35	68.40 ± 12.35	< 0.001	83.40 ± 11.0	72.70 ± 30.01	< 0.001	72.60 ± 11.40	66.41 ± 11.90	0.901	0.321
Waist circumference (cm)	100.72 ± 9.78	91.06 ± 12.10	< 0.001	101.80 ± 6.9	89.50 ± 10.80	< 0.001	100.20 ± 10.70	91.90 ± 12.60	< 0.001	0.462
Body mass index (kg/m2)	29.77 ± 4.13	26.70 ± 4.40	< 0.001	28.80 ± 3.3	25.50 ± 3.20	< 0.001	30.10 ± 4.30	27.52 ± 4.66	0.811	0.553
Systolic blood pressure (mm Hg)	128.98 ± 19.43	116.13 ± 15.25	< 0.001	128.90 ± 17.1	118.80 ± 15.50	< 0.001	128.90 ± 20.30	114.50 ± 14.80	< 0.001	0.762
Diastolic blood pressure (mm Hg)	83.95 ± 11.71	76.23 ± 10.21	< 0.001	85.20 ± 10.5	78.40 ± 10.20	< 0.001	83.30 ± 12.10	74.90 ± 9.90	< 0.001	0.144
Blood glucose (mg/dl)	88.10 ± 25.10	80.08 ± 14.95	< 0.001	87.92 ± 22.7	81.13 ± 15.27	< 0.001	88.17 ± 23.90	79.40± 4.70	< 0.001	0.545
Cholesterol (mg/dl)	198.57 ± 38.77	188.38 ± 37.50	< 0.001	190.60 ± 37.8	184.50 ± 37.90	0.005	200.60 ± 38.70	190.60 ± 37.30	< 0.001	0.366
High-density lipoprotein cholesterol (mg/dl)	37.96 ± 7.56	44.77 ± 10.24	< 0.001	34.96 ± 6.4	40.39 ± 8.66	< 0.001	40.66 ± 7.33	47.30 ± 10.20	< 0.001	< 0.050
Low-density lipoprotein cholesterol (mg/dl)	123.93 ± 34.29	122.10 ± 32.09	0.110	118.40 ± 32.7	119.70 ± 31.80	0.514	126.39 ± 34.70	123.50 ± 32.10	0.042	< 0.050
Triglyceride (mg/dl)	185.42 ± 92.67	112.52 ± 58.45	< 0.001	179.0 (IQR 144-236)	109.0 (IQR 79-150)	< 0.001	166.0 (IQR 119-210)	97.0 (IQR 73-125)	< 0.001	0.474
Uric acid (mg/dl)	4.92 ± 2.17	4.42 ± 1.25	< 0.001	5.62 ± 1.3	5.17 ± 1.20	< 0.001	4.61 ± 2.30	3.9±1.02	< 0.001	0.072
High sensitive C-reactive protein (mg/l)	1.8 (IQR 1.12-3.47)	1.3 (IQR 0.83-2.52)	0.110	1.4 (IQR 1.01-2.7)	1.3 (IQR 0.8-2.3)	< 0.001	2.1 (IQR 1.2-3.8)	1.3 (IQR 0.9-2.6)	< 0.001	< 0.050
Energy (kcal)	1651.65 ± 586.67	1644.06 ± 560.53	0.590	1977.40 ± 26.6	1812.70 ± 21.70	< 0.001	1465.30± 13.40	1473.00 ± 13.80	0.632	0.122
Protein										
Crude intake (g)	60.41 ± 24.74	59.51 ± 24.70	0.270	71.40 ± 1.1	65.90 ± 0.90	< 0.001	55.40 ± 0.65	55.70 ± 0.70	0.802	0.469
Total energy adjusted (g)	59.72 ± 15.44	59.06 ± 16.16	0.210	59.90 ± 0.8	59.90 ± 0.60	0.994	59.60 ± 0.40	58.50 ± 0.40	0.703	0.338

Table 1. Demographic, cardiometabolic factors, and daily intake characteristics of subjects with and without metabolic syndrome (continue)

Characteristic	Total			Men			Women			Gender* MetS interaction P
	MetS+ (n = 1699)	MetS− (n = 1936)	P	MetS+ (n = 521)	MetS− (n = 714)	P	MetS+ (n = 1178)	MetS− (n = 1222)	P	
Total energy (%)	14.79 ± 3.76	14.58 ± 3.93	0.110	13.60 ± 0.1	13.7 ± 0.1	0.740	13.80 ± 0.10	13.50 ± 0.10	0.021	0.229
Carbohydrate										
Crude intake (g)	232.31 ± 96.36	230.25 ± 94.53	0.510	282.07 ± 4.6	258.80 ± 3.70	< 0.001	210.10 ± 2.41	213.60 ± 2.50	0.312	0.322
Total energy adjusted (g)	230.65 ± 39.75	229.73 ± 41.40	0.490	231.30 ± 2.1	232.90 ± 1.60	0.544	230.30 ± 1.03	228.00 ± 1.10	0.213	0.265
Total energy (%)	55.93 ± 9.83	55.57 ± 10.23	0.270	55.30 ± 0.4	55.30 ± 0.30	0.768	54.20 ± 0.20	53.50 ± 0.20	0.604	0.140
Total fat										
Crude intake (g)	59.99±25.07	60.42 ± 24.58	0.600	70.50 ± 1.1	64.30 ± 0.90	< 0.001	55.20 ± 0.60	58.03 ± 0.60	0.004	< 0.050
Total energy adjusted (g)	59.06±15.28	59.74 ± 16.14	0.190	58.60 ± 0.7	58.04 ± 0.60	0.118	59.20 ± 0.40	60.60 ± 0.40	0.012	0.124
Total energy (%)	32.85±8.57	33.39 ± 8.96	0.060	31.10 ± 0.3	31.00 ± 0.30	0.108	32.00 ± 0.20	33.00 ± 0.20	0.009	0.262
Saturated fatty acid										
Crude intake (g)	16.20±8.27	16.49 ± 8.00	0.270	18.90 ± 0.4	17.90 ± 0.30	0.039	14.90 ± 0.20	15.60 ± 0.20	0.041	0.233
Total energy adjusted (g)	16.62±6.42	16.98 ± 6.41	0.080	16.50 ± 0.3	16.90 ± 0.20	0.188	16.60 ± 0.10	16.90 ± 0.10	0.202	0.882
Monounsaturated fatty acid										
Crude intake (g)	16.30±7.39	16.29 ± 7.41	0.970	16.20 ± 0.3	15.90 ± 0.20	< 0.001	14.50 ± 0.20	14.70 ± 0.20	0.042	0.124
Total energy adjusted (g)	16.03±4.97	16.09 ± 5.46	0.720	16.24 ± 0.3	15.98 ± 0.20	0.114	15.90 ± 0.10	16.10 ± 0.10	0.165	0.951
Poly unsaturated fatty acid										
Crude intake (g)	20.72±10.88	20.64 ± 10.69	0.820	23.90 ± 0.5	21.20 ± 0.30	< 0.001	18.80 ± 0.30	19.30 ± 0.30	0.015	< 0.050
Total energy adjusted (g)	20.48±8.59	20.50 ± 8.81	0.960	20.10 ± 0.4	19.20 ± 0.30	0.074	20.60 ± 0.20	21.20 ± 0.20	0.085	< 0.050
Cholesterol										
Crude intake (g)	204.74 ± 179.76	207.09 ± 177.56	0.690	252.30 ± 9.9	236.40 ± 7.10	0.181	183.60 ± 4.30	189.80 ± 4.70	0.342	0.786
Total energy adjusted (g)	203.10 ± 169.06	206.19 ± 169.08	0.580	217.90 ± 9.5	218.60 ± 6.90	0.955	196.60 ± 4.10	198.70 ± 4.50	0.729	0.816

* P < 0.050 is significant. Values are expressed as mean ± SD for normally distribution data and median with interquartile range of non-normally distributed data. The Student's t-test is used for comparison of normally distributed data and Mann-Whitney test for comparison of non-normally distribution data between men with and without MetS and women with and without MetS.

MetS: Metabolic syndrome; MetS+: with metabolic syndrome; MetS−: without metabolic syndrome; IQR: Interquartile range; SD: Standard deviation

Macronutrient intakes are reported as a percentage of the total energy.

Table 2. Odds ratios of metabolic syndrome across quintiles of energy-adjusted macronutrients intake

Nutrients	Quintiles of intake					P
	Q1	Q2	Q3	Q4	Q5	
Protein						0.182
Median intake (g/d)	41.1	52.2	58.9	66.0	78.20	
Range of intake	< 47.7	47.8-55.7	55.7-62.1	62.1-70.5	> 70.5	
OR	1	0.79 (0.64-0.99)	0.96 (0.77-1.02)	0.92 (0.74-1.15)	0.85 (0.68-1.06)	
Carbohydrate						0.666
Median intake (g/d)	181.2	211.5	230.2	248.5	279.08	
Range of intake	< 199.7	199.8-221.7	221.8-238.8	238.9-260.4	> 260.5	
OR	1	0.78 (0.62-0.97)	1.05 (0.84-1.31)	0.97 (0.78-1.21)	0.94 (0.75-1.17)	
Fat						0.376
Median intake (g/d)	40.1	52.0	58.9	66.5	78.30	
Range of intake	< 47.0	47.0-55.5	55.5-62.8	62.8-71.5	> 71.5	
OR	1	1.24 (0.99-1.55)	1.10 (0.88-1.37)	1.04 (0.84-1.30)	0.97 (0.78-1.23)	
Saturated fatty acid						0.092
Median intake (g/d)	10.0	13.5	16.0	18. 9	24.70	
Range of intake	< 12.0	12.0-14.8	14.8-17.3	17.3-21.0	> 21.00	
OR	1	1.05 (0.84-1.31)	1.06 (0.85-1.32)	0.89 (0.71-1.11)	0.88 (0.70-1.09)	
Monounsaturated fatty acid						0.545
Median intake (g/d)	9.9	13.6	16.0	23.2	26.20	
Range of intake	< 12.2	12.2-14.9	14.9-17.1	17.1-45.9	> 45.90	
OR	1	1.21 (0.97-1.52)	1.20 (0.96-1.49)	1.22 (0.98-1.53)	1.07 (0.86-1.33)	
Polyunsaturated fatty acid						0.958
Median intake (g/d)	9.9	16.1	20.2	24.2	31.30	
Range of intake	< 13.3	13.3-18.4	18.4-22.0	22.0-27.0	> 27.00	
OR	1	1.10 (0.88-1.37)	0.98 (0.79-1.22)	1.06 (0.85-1.32)	1.02 (0.82-1.27)	
Niacin						0.889
Median intake (g/d)	57.1	113.7	158.5	232.8	391.20	
Range of intake	< 89.8	89.8-135.6	135.7-186.1	186.2-312.3	> 312.50	
OR	1	0.98 (0.79-1.23)	0.98 (0.78-1.22)	0.86 (0.69-1.07)	1.04 (0.84-1.30)	

Data are expressed as median intake, range of intake, and OR (95% CI) and are adjusted for sex, age, physical activity level, smoking, past medical history, energy intake, and BMI.
ORs: Odds ratios; CI: Confidence interval; BMI: Body mass index

Chen et al. found a significant positive relationship between serum concentration of hs-CRP and the occurrence of MetS.[31] Furthermore, Konishi et al. reviewed the association between hs-CRP and MetS in epidemiological studies and reported that serum hs–CRP, as a main component of MetS, is generally increased in obese, hypertensive, and diabetic subjects as well as in subjects with low HDL-cholesterol and high triglyceride.[32] Oda and Kawai showed that hs-CRP could be considered as an inflammatory component of MetS in Japanese individuals.[33]

In agreement with our results, several other studies found that serum uric acid was associated with MetS and its related risk factors.[34-42] Zhang et al. suggested that uric acid levels may be an independent risk marker for obesity, hypertension, and dyslipidemia, as the main components of MetS.[43] Another study showed an association between hyperuricemia and increased risk of MetS.[34]

Table 3. Odds ratios of metabolic syndrome across quartiles of physical activity level

Physical activity	Quartiles of PAL				P
	Q1	Q2	Q3	Q4	
PAL					< 0.001
Median	1.22	1.47	1.69	2.06	
Range	(0.00-1.40)	(1.40-1.59)	(1.60-1.89)	(1.90-36.95)	
OR	1	0.64 (0.50-0.81)	0.45 (0.29-0.66)	0.34 (0.17-0.67)	

Data are expressed as median intake, range of intake, and OR (95% CI) and are adjusted for sex, age, smoking, past medical history, energy intake, and BMI.
ORs: Odds ratios; CI: Confidence interval; BMI: Body mass index; PAL: Physical activity level

The results of a cross-sectional study among Korean Urban Rural Elderly (KURE) individuals suggested a positive relationship between increased serum concentration of uric acid and the presence of MetS.[36] In a longitudinal study, a high level of uric acid has been shown to be related to a higher risk of MetS in men after 10 years of follow-up.[44]

A significant inverse association was detected between physical activity and the presence of MetS. This is consistent with the findings of two previous studies that reported regular physical exercise reduces overall body adipose tissue in general, and abdominal adiposity in particular, in obese and overweight subjects.[45,46] Therefore, as a result of reduced adipose tissue, other components of MetS would be affected.[45] Guinhouya et al. reviewed cross-sectional, prospective, cohort, and intervention studies examining the effect of physical activity on MetS, its components, and IR.[46] They found that higher PAL was consistently associated with an improved metabolic profile and a reduced risk for MetS and IR, which was in agreement with the current study findings.[46] A significant association has been reported for the highest duration of time watching television or playing screen-related games, and sedentary time with MetS.[47]

The negative association between physical activity and the risk of MetS can be attributed to the response of the adipocytes cell surface receptors to various factors including secretion of catecholamines during exercise which in turn mobilize fat deposits,[2] and attenuate IR as the most important components of MetS. Physical activity has been reported to improve the markers of endothelial dysfunction in adolescents with MetS.[48] Furthermore, exercise can help to maintain a higher lean body mass (LBM) and a higher resting energy expenditure (REE) associated with reduced age-related changes including obesity caused by lowered REE and LBM or inflammation which has occurred mainly as a result of obesity.[2]

Adjusted total macronutrient intake had no significant association with the presence of MetS. Brunner et al.[49] and Dalle Grave et al.[50] have shown that dietary carbohydrate intake is unrelated to the risk of MetS. Likewise, Ludwig et al. reported that the dietary intake of fat, carbohydrate, and protein had conflicting associations with the risk factors of CVD.[51] In addition, a meta-analysis of prospective epidemiological cohort studies evaluating the association of saturated fat with CVD concluded that there is no significant evidence for the association between dietary saturated fat and an increased risk of CVD.[52] However, one study has previously shown that a high total carbohydrate intake may be associated with elevated concentrations of plasma lipids and fasting glucose levels, and may result in the MetS in some individuals.[53] Lopez et al. observed the adverse effects of saturated fatty acid (SFA) on the risk of CVD and glycemic control.[54] The differences between the current findings and the results of previous studies could be attributed to the differences in study design, definitions of MetS, sample size, population group, and the method of dietary assessment.

The lack of consistency of the result of the present study with other studies regarding the association between macronutrient intake and MetS might be the result of the greater impact of dietary intake in earlier phases of life.[52-55] According to the Barker hypothesis, maternal nutrition and fetal and infant growth have an important impact on the risk of CVD in adults.[56] Additionally, modification in the dietary intake of the participants by previous medical recommendation could be considered as another reason for the lack of significant differences of the adjusted macronutrient intake between subjects with and without MetS and lack of relationship between nutritional factors and MetS.

Overall, the cross-sectional design of the present study should be taken into account when interpreting the results. Since 24-hour dietary recall methodology, which is based on self-recall, was used in the present study, its inadequate precision especially among obese subjects or women should be considered as a limitation. Scagliusi et al. suggested that a greater BMI, social desirability score, and body dissatisfaction score and lower income are associated with systematic underreporting.[57] Therefore, underreporting of energy intake might be an obstacle in the investigation of obese subjects.[58]

In the present study, the association between nutrient intake and MetS was investigated among a large sample of adults, which was a representative sample. Therefore, the results of the present study could reflect the general population of Iran. Additionally, we controlled for a number of confounding factors including age, sex, smoking, family history of chronic diseases, as well as total energy intake. However, there are some potential limitations, which need to be considered. The first limitation was the cross-sectional design of the study that does not allow the determination of cause-effect relationships. Second, assessing energy and nutrient

intake in this study was based on a single 24-hour dietary recall. In spite of adjusting several potential confounding factors, recall bias and unresponsive bias potentially affected the result of risk factor analysis. Moreover, it may have been better if 3 days of 24-hour dietary recall were analyzed.

Conclusion

An inverse relationship was found between PAL and the risk of MetS, but adjusted dietary macronutrients intake had no significant association with the presence of MetS. Serum concentrations of hs-CRP and uric acid were significantly higher in subjects with MetS.

Acknowledgments

Mashhad University of Medical Sciences has provided the financial support for this study (85134). We are particularly grateful to the patients and their family members who volunteered to participate in this study.

Conflict of Interests

Authors have no conflict of interests.

References

1. Alberti KG, Zimmet P, Shaw J. The metabolic syndrome-a new worldwide definition. Lancet 2005; 366(9491): 1059-62.
2. Mahan LK, Escott-Stump S, Raymond JL, Krause MV. Krause's food & the nutrition care process. Philadelphia, PA: Elsevier Health Sciences; 2012.
3. Ford ES. Risks for all-cause mortality, cardiovascular disease, and diabetes associated with the metabolic syndrome: A summary of the evidence. Diabetes Care 2005; 28(7): 1769-78.
4. Cobbold JF, Anstee QM, Taylor-Robinson SD. The importance of fatty liver disease in clinical practice. Proc Nutr Soc 2010; 69(4): 518-27.
5. Hamilton MT, Hamilton DG, Zderic TW. Role of low energy expenditure and sitting in obesity, metabolic syndrome, type 2 diabetes, and cardiovascular disease. Diabetes 2007; 56(11): 2655-67.
6. Mottillo S, Filion KB, Genest J, Joseph L, Pilote L, Poirier P, et al. The metabolic syndrome and cardiovascular risk a systematic review and meta-analysis. J Am Coll Cardiol 2010; 56(14): 1113-32.
7. Mozumdar A, Liguori G. Persistent increase of prevalence of metabolic syndrome among U.S. adults: NHANES III to NHANES 1999-2006. Diabetes Care 2011; 34(1): 216-9.
8. Azimi-Nezhad M, Herbeth B, Siest G, Dade S, Ndiaye NC, Esmaily H, et al. High prevalence of metabolic syndrome in Iran in comparison with France: What are the components that explain this? Metab Syndr Relat Disord 2012; 10(3): 181-8.
9. Lira FS, Pimentel GD, Santos RV, Oyama LM, Damaso AR, Oller do Nascimento CM, et al. Exercise training improves sleep pattern and metabolic profile in elderly people in a time-dependent manner. Lipids Health Dis 2011; 10: 1-6.
10. Magkos F, Yannakoulia M, Chan JL, Mantzoros CS. Management of the metabolic syndrome and type 2 diabetes through lifestyle modification. Annu Rev Nutr 2009; 29: 223-56.
11. Lopez-Fontana CM, Sanchez-Villegas A, Martinez-Gonzalez MA, Martinez JA. Daily physical activity and macronutrient distribution of low-calorie diets jointly affect body fat reduction in obese women. Appl Physiol Nutr Metab 2009; 34(4): 595-602.
12. Choi H, Song S, Kim J, Chung J, Yoon J, Paik HY, et al. High carbohydrate intake was inversely associated with high-density lipoprotein cholesterol among Korean adults. Nutr Res 2012; 32(2): 100-6.
13. Steemburgo T, Dall'Alba V, Gross JL, Azevedo MJ. Dietary factors and metabolic syndrome. Arq Bras Endocrinol Metabol 2007; 51(9): 1425-33.
14. Kim K, Yun SH, Choi BY, Kim MK. Cross-sectional relationship between dietary carbohydrate, glycaemic index, glycaemic load and risk of the metabolic syndrome in a Korean population. Br J Nutr 2008; 100(3): 576-84.
15. Warwick ZS, Schiffman SS. Role of dietary fat in calorie intake and weight gain. Neurosci Biobehav Rev 1992; 16(4): 585-96.
16. Warwick ZS, Synowski SJ, Rice KD, Smart AB. Independent effects of diet palatability and fat content on bout size and daily intake in rats. Physiol Behav 2003; 80(2-3): 253-8.
17. Savastano DM, Covasa M. Adaptation to a high-fat diet leads to hyperphagia and diminished sensitivity to cholecystokinin in rats. J Nutr 2005; 135(8): 1953-9.
18. Yudell BE. Interactive effects of macronutrients on obesity and metabolic syndrome development [Thesis]. Champaign, IL: University of Illinois at Urbana-Champaign; 2009.
19. Afonso RA, Lautt WW, Schafer J, Legare DJ, Oliveira AG, Macedo MP. High-fat diet results in postprandial insulin resistance that involves parasympathetic dysfunction. Br J Nutr 2010; 104(10): 1450-9.
20. Das M, Pal S, Ghosh A. Interaction of physical activity level and metabolic syndrome among the adult Asian Indians living in Calcutta, India. J Nutr Health Aging 2012; 16(6): 539-43.
21. Schrauwen P. Physical activity and diabetes: Current considerations. Appl Physiol Nutr Metab 2007; 32(3): 535-6.
22. Laaksonen DE, Lindstrom J, Lakka TA, Eriksson JG, Niskanen L, Wikstrom K, et al. Physical

activity in the prevention of type 2 diabetes: The Finnish diabetes prevention study. Diabetes 2005; 54(1): 158-65.
23. Franz MJ, VanWormer JJ, Crain AL, Boucher JL, Histon T, Caplan W, et al. Weight-loss outcomes: A systematic review and meta-analysis of weight-loss clinical trials with a minimum 1-year follow-up. J Am Diet Assoc 2007; 107(10): 1755-67.
24. Ghayour-Mobarhan M, Moohebati M, Esmaily H, Ebrahimi M, Parizadeh SM, Heidari-Bakavoli AR, et al. Mashhad stroke and heart atherosclerotic disorder (MASHAD) study: Design, baseline characteristics and 10-year cardiovascular risk estimation. Int J Public Health 2015; 60(5): 561-72.
25. Khayyatzadeh SS, Moohebati M, Mazidi M, Avan A, Tayefi M, Parizadeh SM, et al. Nutrient patterns and their relationship to metabolic syndrome in Iranian adults. Eur J Clin Invest 2016; 46(10): 840-52.
26. Mazidi M, Heidari-Bakavoli A, Khayyatzadeh SS, Azarpazhooh MR, Nematy M, Safarian M, et al. Serum hs-CRP varies with dietary cholesterol, but not dietary fatty acid intake in individuals free of any history of cardiovascular disease. Eur J Clin Nutr 2016; 70(12): 1454-7.
27. Mazidi M, Nematy M, Heidari-Bakavoli AR, Namadchian Z, Ghayour-Mobarhan M, Ferns GA. The relationship between dietary intake and other cardiovascular risk factors with blood pressure in individuals without a history of a cardiovascular event: Evidence based study with 5670 subjects. Diabetes Metab Syndr 2017; 11(Suppl 1): S65-S71.
28. James WP, Schofield E. human energy requirements a manual for planners and nutritionists. Oxford, UK: Oxford Medical Publications; 1990.
29. Daryani A, Berglund L, Andersson A, Kocturk T, Becker W, Vessby B. Risk factors for coronary heart disease among immigrant women from Iran and Turkey, compared to women of Swedish ethnicity. Ethn Dis 2005; 15(2): 213-20.
30. Freire RD, Cardoso MA, Gimeno SG, Ferreira SR. Dietary fat is associated with metabolic syndrome in Japanese Brazilians. Diabetes Care 2005; 28(7): 1779-85.
31. Chen LX, Zhang SD, Zhu LL, Sun M. Association of metabolic syndrome with serum interleukin-10 and high sensitive C reactive protein(hs-CRP) in old men. Zhong Nan Da Xue Xue Bao Yi Xue Ban 2008; 33(10): 970-4.
32. Konishi M, Watanabe K, Okada K. Relation of hs-CRP and metabolic syndrome in epidemiological study. Nihon Rinsho 2006; 64(Suppl 9): 530-5.
33. Oda E, Kawai R. Comparison between high-sensitivity C-reactive protein (hs-CRP) and white blood cell count (WBC) as an inflammatory component of metabolic syndrome in Japanese. Intern Med 2010; 49(2): 117-24.
34. Abbasian M, Ebrahimi H, Delvarianzadeh M, Norouzi P, Fazli M. Association between serum uric acid (SUA) levels and metabolic syndrome (MetS) components in personnel of Shahroud University of Medical Sciences. Diabetes Metab Syndr 2016; 10(3): 132-6.
35. Bagheri B, Zargari M, Meshkini F, Dinarvand K, Mokhberi V, Azizi S, et al. Uric acid and coronary artery disease, two sides of a single coin: A determinant of antioxidant system or a factor in metabolic syndrome. J Clin Diagn Res 2016; 10(2): OC27-OC31.
36. Choi H, Kim HC, Song BM, Park JH, Lee JM, Yoon DL, et al. Serum uric acid concentration and metabolic syndrome among elderly Koreans: The Korean Urban Rural Elderly (KURE) study. Arch Gerontol Geriatr 2016; 64: 51-8.
37. Kawada T. Cross-sectional and longitudinal study on the association between serum uric acid and metabolic syndrome. Clin Chim Acta 2016; 455: 201.
38. Obeidat AA, Ahmad MN, Haddad FH, Azzeh FS. Leptin and uric acid as predictors of metabolic syndrome in jordanian adults. Nutr Res Pract 2016; 10(4): 411-7.
39. Safiri S, Qorbani M, Heshmat R, Tajbakhsh R, Eslami Shahr Babaki A, Djalalinia S, et al. Association of Serum Uric Acid With Cardiometabolic Risk Factors and Metabolic Syndrome in Iranian Adolescents: The CASPIAN-III Study. Iran J Kidney Dis 2016; 10(3): 126-34.
40. Sah SK, Khatiwada S, Pandey S, Kc R, Das BK, Baral N, et al. Association of high-sensitivity C-reactive protein and uric acid with the metabolic syndrome components. Springerplus 2016; 5: 269.
41. Yu TY, Jee JH, Bae JC, Jin SM, Baek JH, Lee MK, et al. Serum uric acid: A strong and independent predictor of metabolic syndrome after adjusting for body composition. Metabolism 2016; 65(4): 432-40.
42. Zurlo A, Veronese N, Giantin V, Maselli M, Zambon S, Maggi S, et al. High serum uric acid levels increase the risk of metabolic syndrome in elderly women: The PRO.V.A study. Nutr Metab Cardiovasc Dis 2016; 26(1): 27-35.
43. Zhang Q, Zhang C, Song X, Lin H, Zhang D, Meng W, et al. A longitudinal cohort based association study between uric acid level and metabolic syndrome in Chinese Han urban male population. BMC Public Health 2012; 12: 419.
44. Sun HL, Pei D, Lue KH, Chen YL. Uric Acid Levels Can Predict Metabolic Syndrome and Hypertension in Adolescents: A 10-Year Longitudinal Study. PLoS One 2015; 10(11): e0143786.
45. Gonzalez Calvo G, Hernandez Sanchez S, Pozo Rosado P, Garcia Lopez D. Positive effects of physical exercise on reducing the relationship between subcutaneous abdominal fat and morbility

risk. Nutr Hosp 2011; 26(4): 685-91.
46. Guinhouya BC, Samouda H, Zitouni D, Vilhelm C, Hubert H. Evidence of the influence of physical activity on the metabolic syndrome and/or on insulin resistance in pediatric populations: A systematic review. Int J Pediatr Obes 2011; 6(5-6): 361-88.
47. Saleh D, Janssen I. Interrelationships among sedentary time, sleep duration, and the metabolic syndrome in adults. BMC Public Health 2014; 14: 666.
48. Camarillo-Romero E, Dominguez-Garcia MV, Amaya-Chavez A, Camarillo-Romero MS, Talavera-Pina J, Huitron-Bravo G, et al. Effects of a physical activity program on markers of endothelial dysfunction, oxidative stress, and metabolic status in adolescents with metabolic syndrome. ISRN Endocrinol 2012; 2012: 970629.
49. Brunner EJ, Wunsch H, Marmot MG. What is an optimal diet? Relationship of macronutrient intake to obesity, glucose tolerance, lipoprotein cholesterol levels and the metabolic syndrome in the Whitehall II study. Int J Obes Relat Metab Disord 2001; 25(1): 45-53.
50. Dalle Grave R, Calugi S, Centis E, Marzocchi R, El Ghoch M, Marchesini G. Lifestyle modification in the management of the metabolic syndrome: Achievements and challenges. Diabetes Metab Syndr Obes 2010; 3: 373-85.
51. Ludwig DS, Pereira MA, Kroenke CH, Hilner JE, Van Horn L, Slattery ML, et al. Dietary fiber, weight gain, and cardiovascular disease risk factors in young adults. JAMA 1999; 282(16): 1539-46.
52. Siri-Tarino PW, Sun Q, Hu FB, Krauss RM. Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease. Am J Clin Nutr 2010; 91(3): 535-46.
53. Westman EC, Volek JS, Feinman RD. Carbohydrate restriction is effective in improving atherogenic dyslipidemia even in the absence of weight loss. Am J Clin Nutr 2006; 84(6): 1549.
54. Lopez S, Bermudez B, Ortega A, Varela LM, Pacheco YM, Villar J, et al. Effects of meals rich in either monounsaturated or saturated fat on lipid concentrations and on insulin secretion and action in subjects with high fasting triglyceride concentrations. Am J Clin Nutr 2011; 93(3): 494-9.
55. Volek JS, Feinman RD. Carbohydrate restriction improves the features of Metabolic Syndrome. Metabolic Syndrome may be defined by the response to carbohydrate restriction. Nutr Metab (Lond) 2005; 2: 31.
56. Barker DJ. The intrauterine environment and adult cardiovascular disease. Ciba Found Symp 1991; 156: 3-10.
57. Scagliusi FB, Ferriolli E, Pfrimer K, Laureano C, Cunha CS, Gualano B, et al. Characteristics of women who frequently under report their energy intake: A doubly labelled water study. Eur J Clin Nutr 2009; 63(10): 1192-9.
58. Scagliusi FB, Ferriolli E, Pfrimer K, Laureano C, Cunha CS, Gualano B, et al. Underreporting of energy intake in Brazilian women varies according to dietary assessment: A cross-sectional study using doubly labeled water. J Am Diet Assoc 2008; 108(12): 2031-40.

DOI: http://dx.doi.org/10.22122/arya.v15i3.1902 Published by Vesnu Publications

Sharing the power through promoting heart health literacy: A participatory action research in Iran

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Special Report

Abstract

BACKGROUND: Low health literacy can act as a barrier to effective disease self-management. The study aimed to promote heart health literacy in Iranian society.

METHODS: This study was conducted as a participatory action research (PAR) based on Zuber-Skerritt Model to design and implement a program for promoting heart health literacy in Iranian society. Participants were selected among adults with heart diseases and their family members, as well as their health care providers in Chamran Hospital, Isfahan Heart Friends association and researchers, and Isfahan Cardiovascular Research Institute, Isfahan, Iran. Data collection was conducted using interviews. Content analysis was used to analyze the data to promote heart health literacy. Promoting of heart health literacy was implemented in different levels in Isfahan from March 2017 to October 2017. The effect of the program was evaluated based on interviews, feedback, and focus groups at the individual level.

RESULTS: Finally, at the World Heart Week, a healthy heart campaign was formed with the slogan "Share the power". At the end of this program, participants experienced significant empowerment during the project to promote heart health literacy. The three main themes indicating this feeling of empowerment were “Being worried about the hearts of others”, “Sensitization to the care of the heart”, and “General understanding of heart health”.

CONCLUSION: PAR can be an effective way to promote heart health literacy in Iranian society. It integrates the voices of the marginalized group promoting heart health literacy in Iranian society.

Keywords: Heart, Health Literacy, Action Research, Iran

Date of submission: 01 Nov. 2018, ***Date of acceptance:*** 24 Mar. 2019

Introduction

Currently, chronic diseases, especially atherosclerosis, high blood pressure, and its consequences are considered to be the most important health problems in underdeveloped countries, as well as developed countries.[1,2] It is anticipated that by 2030, 23 million deaths will occur annually due to cardiovascular disease (CVD) worldwide.[3] In Iran, official statistics from the Ministry of Health and Medical Education show that 33.0%-39.3% of deaths in the country are due to CVD.

How to cite this article: Akbari M, Mousavi MS, Shirani F, Massoumi G, Shafeie M, Niknejad R, et al. **Sharing the power through promoting heart health literacy: A participatory action research in Iran.** ARYA Atheroscler 2019; 15(3): 146-51.

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CVDs are the first cause of death with 39.3% of all deaths, of which 19.5% are related to heart attacks, 9.3% are due to stroke, 3.1% are due to hypertension (HTN), and the rest are related to other CVDs. According to a study conducted by Akbari Sari et al., to estimate 50 invasive procedures in Iran, there are 75 coronary artery bypass grafting (CABG) and 61 angioplasty per 100000 of population in Iran.[4] Also, in a study in Kerman Province, Iran, the annual mortality rate due to these diseases in the population over 40 years of age was estimated 14 per 1000 people and the number of patients requiring outpatient care was estimated approximately as 1935000 people.[5]

In this regard, hospitals as the second and third level prevention centers should be able to serve proportional to the growth of these diseases. One of the important issues in prevention is improving the health literacy of individuals and their ability in the first to third levels of prevention.[6] Health literacy means cognitive-social skills that determine the motivation and ability of individuals to achieve, understand, and use information in a way that maintains and improves their health.[7] People with good health literacy will surely have better health. Today, the key role of individuals as the main factor in managing their health has been emphasized.[8]

The need for education and information provision to improve the ability of maintaining health and improving the health literacy of patients is essential. In a study in Australia conducted by mixed method through interviewing, a focused group of policymakers showed that in the first level of prevention of CVD, information provision, and integration of caring for patients with complex needs and experiencing many illnesses had better results for patients.[9] However, another study has emphasized on barriers of education for cardiac patients.[10] Some other evidence has supported such a view. For example, a study about surveillance of cardiovascular health literacy level showed that about 60% of respondents were unaware of the symptoms of a heart attack, and 20% knew only two or four signs. Mean knowledge, attitude, and practice were 79.3, 74.3, and 48.0 percent, respectively. About 44% of the respondents had insufficient knowledge and less than 20% had adequate knowledge. The attitude and practice also, were 15.9% and 13.9%, respectively. This study showed that the level of health literacy in a low-income country was low even among patients with CVD.[11] Awad and Al-Nafisi also reported that 60% of people did not know about their heart disease, and symptoms such as shortness of breath or chest pain were signs that approximately 48% to 50% of people knew about. Most people considered the important role of the drug in controlling the disease and did not know the importance of observing other cases. This study showed that individuals' knowledge about CVD and preventive behaviors was inadequate and wider educational programs and interventions were needed.[12] Some studies in Iran also examined the level of health literacy at the community level and obtained results indicating that the level of health literacy in the community was moderate or borderline.[13-15]

According to studies and researchers' experiences, it seems that current programs in country hospitals often focus on care and treatment in the hospital environment, and follow-up and community-based dimensions are less covered. Based on these studies, collaborative qualitative research that helps people's ability can be useful in improving the level of health literacy in different fields. Therefore, the present study aimed to empower people through improving the level of health literacy in the field of CVDs in collaboration with Shahid Chamran Hospital and Isfahan Cardiovascular Research Institute, Isfahan, Iran.

Materials and Methods

The present study was a participatory action research (PAR) by Zuber-Skerritt method aimed to improve the heart health literacy in 4 consecutive phases including planning phase, action phase, observation phase, and reflection phase. PAR is one of the interdisciplinary research methods in which researchers carry out interdisciplinary research through exploratory and interventional interventions, with precise observation and continuous reflection of outcomes. Action research is a systematic exploration that collects and generates information about a specific problem and, while making changes in the system, leads to system reform.

The collaborative nature of the research project required individuals to be willing to participate in the study and consider themselves as part of the research team. Therefore, after forming the research team, the necessary planning for improving the heart health literacy was carried out in the community. First, the planning session with the relevant specialists for translation and provision of educational content and a joint educational conference on the contents of the necessary materials and providing education to the community with the method of playing the roles

and speeches between providers and recipients of services were held and the healthy heart friends campaign was organized with the participation of enthusiasts and specialists through the organization of the healthy heart association and virtual social networks. During the implementation of the campaign and providing sociologist education, observation and evaluation of the problems of the program was done from the viewpoint of the participants in the program. In the next stage, the reflection of the effects of the campaign was examined in the framework of a qualitative health literacy interview with the covered society. Participants in different phases were different in terms of composition, diversity, and the characteristics of entering the study. So that, some people willing to participate in the study, who were able to express their experiences and opinions, were invited to interview. In order to enrich the information and gain a wider perspective, based on purposive sampling, participants from different age, gender, and education level were recruited. Regarding the diverse spectrum of participant samples in this research, the research environment included the Cardiology Research Institute of Isfahan University of Medical Sciences, Isfahan, and public places (parks and main squares in Isfahan) were used for the access to the individuals without CVD. In the sampling of reflection stage, 20 individuals without CVD who participated in the healthy heart campaign during the study were selected purposefully. The number of samples during the study was obtained according to the amount of information and continued until the data saturation was completed.[16]

All participants received written information and provided written informed consent. Data were collected anonymously. The study was approved by the Isfahan Cardiovascular Research Center.

In order to collect data in the reflection phase, a deep semi-structured interview method was used through questionnaires.[16] The interviews began with the question "What is your understanding and experience of heart health?", "Can you tell me what risks threatens your heart?", and "What are the strategies to prevent heart health?". The interviews were conducted in a dedicated room that only interviewer and interviewee were present. The duration of each interview was 30-90 minutes. Recorded interviews were immediately followed by each word after the completion of each interview and were transcribed verbatim. After the interview, the analysis was performed and then the next interview was carried out, and so the work continued until data saturation.[17] Two additional interviews were conducted to ensure data saturation, but no new data were obtained. At the end of each interview, participants received a gift worth $15.

The analysis team read all the transcripts and analyzed the data. For the analysis of the qualitative data, qualitative content analysis (Graneheim and Lundman[18]) was used. The study used inductive method to find the different dimensions and variables affecting the promoting of heart health literacy in Iranian society. In general, the content analysis process in this study was described in the sequential steps: determining the content of the analysis, defining the unit of analysis and initial coding, classifying the codes under the classes, forming sub-categories from these classes, and forming the main categories of the sub-categories. Data encoding was done independently, which was discussed for coordination. Managing and analyzing the data was done using MAXQDA 10 software (VERBI Software, Berlin, Germany).

Conducting interviews continued until reaching saturation, and selecting the sample was purposive, paying attention to the maximum diversity in the participants regarding age, sex, and education level. Different ratios of participants were considered to provide validity. In addition, peer and member check were used to determine the validity and appropriateness. To confirm the transferability of the findings, the position of the participants was fully and extensively described and details on the methodology and background information were provided to judge and evaluate others about the findings. Moreover, conducting open interviews and expressing the story of the experiences of participants, reflexivity and long-term presence, maintaining the documentation and accurate transmission, and maintaining the impartiality in delivering the results were considered as indicators of trustworthiness in a qualitative study.[19]

We used peer debriefing by three expert colleagues in qualitative research to assure the credibility of data; also we applied member checking of the findings by participants. To assure dependability of results, 4 participants read the transcription and categories.

Results

As indicated in the Methods Section, this study was carried out in four phases of planning, action,

observation, and reflection, the results of four stages being presented as follows:

The result of the analysis of the documents/notes and experiences of the research team and other participants/providers during the planning process was indicated coherently after determining the main problem. At this stage, all of the participants considered improving the level of health literacy as one of the main problems of the Iranian community and planning began accordingly. In 7 three-hour focus groups, the research team looked at strategies for improving the heart health in the community. In the first focus group, the research team consisted of representatives of the Heart Friends Association, the supervisor of the Isfahan Heart Hospital, three cardiologists, the head of the Heart and Cardiology Center of Isfahan, and three heart researchers. The outcome of this meeting was to plan how to proceed through a storm of thought.

For example, one of the first programs to increase heart health literacy was to train specialist staff to train self-care of healthy heart in the community. For this reason, the nurse preparation workshop was held for six hours. In this workshop, which its scientific secretary was a faculty member with experience of expanding the role of nurses in the education in chronic non-communicable diseases (NCDs) and its lecturers were professors, nurses, doctors, nutritionists, and experienced psychologists, important topics and educational content for general education and improving health literacy in the form of the role of educator were presented and topics such as "education of the importance of HTN", "education of healthy nutrition", "education of the importance of exercise and physical activity", and "education of the importance of drug consuming" were presented to the community and the role of educator in the education of reducing stress and quitting smoking with practical examples was educated to the nurses who were members of the healthy heart campaign as well as a number of audiences of the community (Heart Friends Association).

After education of the trainers, at the same time as the World Heart Week, healthy heart campaign was held with the presence of trainers from nursing and pediatric groups in the main squares of Isfahan. In this campaign, cardiologists, heart nurses, heart friends, recovered heart patients, and a number of medical students who were involved in the workshop mentioned above and were skilled in this field, sensitized people to the prevention of heart disease and heart health education. In addition to the education and screening of the healthy heart campaign, five hike meetings with the motto of the World Heart Federation, i.e., "Share the power", were held in different places of Isfahan City. In this screening, high blood pressure along with providing the necessary training to the community was assessed. Many questions were asked from people to sensitize them. Some of these questions were: "Do you know what the heart is doing?", "Do you know what the fuel is for your heart?", "Do you know how long your heart works?", and "Do you know what to do to improve your heart's power?". After sensitizing, individual training was done. Speeches were held in the squares of the city. The question and answer in this campaign intensified. After organizing the workshop and setting up the campaign, the number of healthy heart friends and the participation of the healthcare team, especially nurses, increased, and the increased skills of service providers and educators on how to train the heart health literacy was the result of the campaign.

The three main themes indicating this feeling of empowerment are "Being worried about the hearts of others", "Sensitization to the care of the heart", and "General understanding of heart health".

"I have not been so worried about my heart so far, I just realized that I had to change my lifestyle, I had to tell family members how important it is to do something and not to have heart problems" (Participant 5).

Most of the participants in the campaign, after knowing the condition of the heart and being aware of heart disease and its solutions, became sensitive to their and others' hearts and began to train and sensitize the others as cascades.

One participant (a businessman) stated: "I really did not know that heart is so important, I thought heart disease is related to the old age, but now I found that the narrowing of the arteries begins from the very beginning" (Participant 14).

Another participant also stated: "I am experiencing a lot of stress, now I realize that I have to get stress away from myself. I quit smoking today, promising to remember the things I learned" (Participant 19).

Discussion

The three main themes indicating the feeling of empowerment are "Being worried about the hearts of others", "Sensitization to the care of the heart", and "General understanding of heart health".

In the context of "Sensitization to the care of the heart", the present study showed that the sensitivity of individuals to CVD was improved during and after exercise. Several studies consistent with the present study showed that the sensitivity and health literacy of the heart patients was low.[20,21] Ghisi et al. reported that low health literacy led to the majority of these patients being re-admitted.[21] Training to improve the level of health literacy of patients with coronary artery disease (CAD) is essential and increases the knowledge, commitment, and the tendency to adhere to healthy lifestyle.[22]

The experiences of Ehrenthal et al. in a study suggest the usefulness of education for increasing health literacy, so that the learners' grades after the lecture were significantly higher and increased more for people with lower base scores.[23] Therefore, in line with the above study, it could be said that qualitative statements and narratives from an interview with the audience of educational programs in this study confirmed the results of quantitative studies related to health literacy.

Despite efforts to raise awareness among American women about the risk of heart disease, only about half of all women (56%) were aware of heart disease as the number one killer in the United States (US); among dark-skinned women, it was even less than one. This increasing of awareness should not only occur at the patient level, but also at the level of policymakers and care-providers. In order to achieve the goals of reducing heart disease among all people, brave and creative approaches are needed. By sharing experiences and what is learnt, people can add new healthy heart behaviors to their routine lifestyle. These relationships create a stable and professional career.[24]

Other topics in this study were concerns about the health of others. Although based on the slogan of World Health Organization (WHO), providing the health for all is responsibility of the community, the responsibility of the people of the community for their own health and for others should not be forgotten. In this regard, consistent with the current study, the study by Thompson et al. showed that individual counseling as well as community engagement was one of the most effective preventive learning styles among people in a rural area in Australia.[9]

General understanding of heart health was another theme obtained in this study. Communication at community level enhances interpersonal interaction and acts as a strong strategy for facilitating the use of subjective knowledge by learners. Several studies have shown that observance of the principles of health literacy improves the comprehension of the subject and changes the behavior in the community.[25-27]

Therefore, according to interviews, the study results showed that the main aim, i.e., to raise the level of health literacy in the community, was achieved. Hence, the researchers continued this campaign on a weekly basis with the participation of other hospitals in Isfahan.

The limitations of the present study were: samples were selected from a small section of the population, and this qualitative study provided a limited perspective on the results of interventions.

Conclusion

The advent of health literacy is useful for providing powerful theoretical guidance and practical strategies. Health literacy, in coordination with other determinants of health, has an increasing perception of the factors affecting health promotion in the individual, organizational, and social environments. Since the combination of collaborative approaches and social science theories can be effective in promoting interventions and health literacy levels in the community, researchers suggest that a study be conducted with this aim in the future. Also, designed health communications are often overly public, so it is suggested that a study in coordination with the abilities, preferences, and living conditions of learners should be designed and implemented.

Acknowledgments

The researchers are grateful to the chair management, nursing management, educational deputy, research deputy, and faculty members of the Heart Department of Shahid Chamran Educational, Research, and Therapeutics Hospital and Cardiovascular Research Institution in Isfahan.

This study was sponsored by the Isfahan Cardiovascular Research Center. The authors are thankful to the Vice-Chancellor and the participants.

Conflict of Interests

Authors have no conflict of interests.

References

1. Joshi MD, Ayah R, Njau EK, Wanjiru R, Kayima JK, Njeru EK, et al. Prevalence of hypertension and associated cardiovascular risk factors in an urban

slum in Nairobi, Kenya: A population-based survey. BMC Public Health 2014; 14: 1177.
2. Sarrafzadegan N, Rabiei K, Alavi M, Abedi H, Zarfeshani S. How can the results of a qualitative process evaluation be applied in management, improvement and modification of a preventive community trial? The IHHP Study. Arch Public Health 2011; 69(1): 9.
3. Siegel R, Ma J, Zou Z, Jemal A. Cancer statistics, 2014. CA Cancer J Clin 2014; 64(1): 9-29.
4. Akbari Sari A, Babashahy S, Olyaeimanesh A, Rashidian A. Estimating the frequency and rate of first 50 common types of invasive procedures in Iran healthcare system. Iran J Public Health 2012; 41(10): 60-4.
5. Talebizadeh N, Haghdoust AA, Mirzazadeh A. An epidemiological model (markov chain) of cardiovascular disease in Iran. Payesh Health Monit 2009; 8(2): 163-70. [In Persian].
6. Lonsdale C, Sanders T, Cohen KE, Parker P, Noetel M, Hartwig T, et al. Scaling-up an efficacious school-based physical activity intervention: Study protocol for the 'Internet-based Professional Learning to help teachers support Activity in Youth' (iPLAY) cluster randomized controlled trial and scale-up implementation evaluation. BMC Public Health 2016; 16(1): 873.
7. Hersh L, Salzman B, Snyderman D. Health literacy in primary care practice. Am Fam Physician 2015; 92(2): 118-24.
8. Yavuz H, Simsek Z, Akbaba M. Health-risk behaviors in agriculture and related factors, southeastern Anatolian region of Turkey. J Agromedicine 2014; 19(4): 364-72.
9. Thompson SC, Haynes E, Woods JA, Bessarab DC, Dimer LA, Wood MM, et al. Improving cardiovascular outcomes among Aboriginal Australians: Lessons from research for primary care. SAGE Open Med 2016; 4: 2050312116681224.
10. Alavi M, Irajpour A, Giles T, Rabiei K, Sarrafzadegan N. Barriers to education in cardiac rehabilitation within an Iranian society: A qualitative descriptive study. Contemp Nurse 2013; 44(2): 204-14.
11. Vaidya A, Aryal UR, Krettek A. Cardiovascular health knowledge, attitude and practice/behaviour in an urbanising community of Nepal: A population-based cross-sectional study from Jhaukhel-Duwakot Health Demographic Surveillance Site. BMJ Open 2013; 3(10): e002976.
12. Awad A, Al-Nafisi H. Public knowledge of cardiovascular disease and its risk factors in Kuwait: A cross-sectional survey. BMC Public Health 2014; 14: 1131.
13. Miri A, Ghanbari MA, Najafi A. The relationship between health literacy and the recovery rate of cardiovascular patients after bypass surgery. Journal of Health Literacy 2016; 1(2): 83-91. [In Persian].
14. Saatchi M, Panahi M, Ashraf Mozafari A, Sahebkar M, Azarpakan A, Baigi V, et al. Health literacy and its associated factors: A population-based study, Hormuz Island. Iran J Epidemiol 2017; 13(2): 136-44. [In Persian].
15. Rezaee Esfahrood Z, Haerian Ardekani A, Rahmanian M, Ghaffari Targhi M. A survey on health literacy of referred diabetic patients to Yazd diabetes research center. Toloo e Behdasht 2016; 15(3): 176-86. [In Persian].
16. Holloway I, Galvin K. Qualitative research in nursing and healthcare. Hoboken, NJ: John Wiley & Sons; 2016.
17. Malterud K, Siersma VD, Guassora AD. Sample size in qualitative interview studies: Guided by information power. Qual Health Res 2016; 26(13): 1753-60.
18. Graneheim UH, Lundman B. Qualitative content analysis in nursing research: Concepts, procedures and measures to achieve trustworthiness. Nurse Educ Today 2004; 24(2): 105-12.
19. Denzin NK, Lincoln YS. The SAGE handbook of qualitative research. Thousand Oaks, CA: SAGE; 2011.
20. Cajita MI, Cajita TR, Han HR. Health literacy and heart failure: A systematic review. J Cardiovasc Nurs 2016; 31(2): 121-30.
21. Ghisi GLM, Chaves GSDS, Britto RR, Oh P. Health literacy and coronary artery disease: A systematic review. Patient Educ Couns 2018; 101(2): 177-84.
22. Tawalbeh LI, Ahmad MM. The effect of cardiac education on knowledge and adherence to healthy lifestyle. Clin Nurs Res 2014; 23(3): 245-58.
23. Ehrenthal DB, Haynes SG, Martin KE, Hitch JA, Addo SF, O'Neill E, et al. Evaluation of the Heart Truth professional education campaign on provider knowledge of women and heart disease. Womens Health Issues 2013; 23(2): e87-e93.
24. Arslanian-Engoren C. Building partnerships to reduce heart disease in women. Heart Lung 2016; 45(3): 161.
25. Zite NB, Wallace LS. Use of a low-literacy informed consent form to improve women's understanding of tubal sterilization: A randomized controlled trial. Obstet Gynecol 2011; 117(5): 1160-6.
26. Wolf MS, Davis TC, Curtis LM, Webb JA, Bailey SC, Shrank WH, et al. Effect of standardized, patient-centered label instructions to improve comprehension of prescription drug use. Med Care 2011; 49(1): 96-100.
27. Arcia A, Suero-Tejeda N, Bales ME, Merrill JA, Yoon S, Woollen J, et al. Sometimes more is more: Iterative participatory design of infographics for engagement of community members with varying levels of health literacy. J Am Med Inform Assoc 2016; 23(1): 174-83.

DOI: http://dx.doi.org/10.22122/arya.v15i3.1959 Published by Vesnu Publications

Sub-obstacles related to long distance and lack of easy access to outpatient cardiac rehabilitation services

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Letter to Editor

Date of submission: 27 Jan. 2019, *Date of acceptance:* 20 Mar. 2019

Dear Editor

Cardiac rehabilitation programs (CRs), as one of the third-level prevention services, plays a significant role in improving the quality of life of patients, and reducing sudden deaths .[1] In recent years, the importance of participating in various forms of CRs has been well highlighted.[2-4] However, the rate of participation in Iran is less than 15%, and almost half of the patients will not be able to complete the programs.[5,6]

This problem is due to several barriers, such as the lack of awareness of the benefits of CRs, illiteracy, aging, anxiety, depression, lack of insurance coverage, and long distances and lack of easy access.[5,6] Although the long distance is considered as one of the obstacles to complete CRs, the indirect effects of this problem appear to have a more negative effect on the continuity of treatment. Distance-related effects include increased costs of metropolitan transportation, disruptions to the normal routine of daily life, need for a family member's permanent help and assistance, lack of sleep, low intake and delays in meals, and lack of individual healthcare facilities in CR centers.

In most CR centers, training classes and aerobic exercise begin at 8.30 am. The patients who live in adjoining cities and remote areas are facing numerous challenges for timely attendance. They usually wake up very early, and do not eat enough breakfast. This causes lack of sleep, sleepiness, and loss of blood sugar in patients, and it mostly causes them to be nervous and agitated. The patients' entourages, who are generally young people, are experiencing occupational, economic, and family problems because of the length of sessions (26-40 sessions of 3 hours). The patients are disturbed by the observation of this condition. Patients, especially those who suffer from financial problems, have to bear additional cost of transport.[7] At the end of each session, the patients return to their place of residence immediately, and these travels will shift their meals. Meanwhile, because of the limitations of physical space, some CR centers do not have enough health facilities that cause the patients to suffer until they reach home.

These factors, all of which are effective in not registering and not adhering to CRs, are directly related to long distance and lack of easy access to these centers. Despite the numerous patients in Iran and the vastness of the country's territory, there are currently only 16 CR centers in 11 cities.[8] This challenge makes it difficult for patients to access these services. Hence, we recommend that short and accessible delivery formats, such as hybrid and home-centered programs, be used alongside traditional hospital-based programs.[2] In this case, patients with cardiovascular diseases are more likely to participate in CRs and complete the programs.

Acknowledgments

We appreciate the Clinical Research Development Center of Imam Reza Hospital, Kermanshah University of Medical Sciences, Kermanshah, Iran.

Conflict of Interests

Authors have no conflict of interests.

References

1. Lee JY, Ahn JM, Park DW, Kang SJ, Kim YH, Lee SW, et al. Impact of exercise-based cardiac rehabilitation on long-term clinical outcomes in patients with left main coronary artery stenosis. Eur J Prev Cardiol 2016; 23(17): 1804-13.

How to cite this article: Heydarpour B, Ahmadi M, Komasi S. **Sub-obstacles related to long distance and lack of easy access to outpatient cardiac rehabilitation services.** ARYA Atheroscler 2019; 15(3): 152-3.

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2. Saeidi M, Soroush A, Komasi S, Singh P. A hybrid cardiac rehabilitation is as effective as a hospital-based program in reducing chest pain intensity and discomfort. Korean J Pain 2017; 30(4): 265-71.
3. Komasi S, Saeidi M. Case formulation and comprehensive cardiac rehabilitation programs tailored to the unique risk factors and consequences profile. ARYA Atheroscler 2018; 14(6): 276-7.
4. Komasi S, Soroush A, Saeidi M. Off-center cardiac rehabilitation focused on extended emotional relationship and common health gains. ARYA Atheroscler 2018; 14(1): 44-5.
5. Moradi B, Maleki M, Esmaeilzadeh M, Abkenar HB. Physician-related factors affecting cardiac rehabilitation referral. J Tehran Heart Cent 2011; 6(4): 187-92.
6. Heydarpour B, Saeidi M, Ezzati P, Soroush A, Komasi S. Sociodemographic predictors in failure to complete outpatient cardiac rehabilitation. Ann Rehabil Med 2015; 39(6): 863-71.
7. Shanmugasegaram S, Oh P, Reid RD, McCumber T, Grace SL. Cardiac rehabilitation barriers by rurality and socioeconomic status: A cross-sectional study. Int J Equity Health 2013; 12: 72.
8. Komasi S, Saeidi M. Hybrid cardiac rehabilitation as an alternative to common hospital-based cardiac rehabilitation in Iran: An appropriate model for the Iranian health system limitations, culture, and patients. Res Cardiovasc Med 2017; 6(2): e39367.