DOI: http://dx.doi.org/10.22122/arya.v15i4.1834 Published by Vesnu Publications

Correlation between air pollution and hospitalization due to myocardial infarction

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Original Article

Abstract

BACKGROUND: Air pollution is associated with increased risk of cardiovascular disease (CVD). This study aims to evaluate the correlation between air pollutants and hospitalization due to myocardial infarction (MI) as part of "correlation of air pollution with hospitalization and mortality of CVDs and respiratory diseases (CAPACITY) study".

METHODS: This case-crossover study analyzed the data of 319 patients who were admitted with diagnosis of ST-elevation MI (STEMI) or non-ST-elevation MI (NSTEMI) in three main hospitals of Isfahan, Iran. The data of airborne pollutants including particulate matter < 10 µm (PM_{10}), particulate matter < 2.5 µm ($PM_{2.5}$), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), carbon monoxide (CO), and ozone (O_3) as well as climatic indices (temperature, wind speed, and humidity) at 24 hours, 48 hours, and one week before admission were extracted from CAPACITY study. The conditional logistic regression method was used to evaluate the correlation between air pollutants and MI hospitalization.

RESULTS: 319 patients with mean age of 63.15 ± 28.14 years, including 238 men (74.6%), and 207 patients with STEMI (64.8%) were recruited. The risk of hospitalization significantly increased in patients with STEMI and 10-unit increment in $PM_{2.5}$ at 48 hours before admission [odds ratio (OR) = 3.70, 95% confidence interval (CI): 1.69-7.69]. Although, majority of air pollutants had positive association with hospitalization in patients with NSTEMI, they were not statistically significant.

CONCLUSION: This study showed significant association between elevated $PM_{2.5}$ at 48 hours before admission and hospitalization of patients with STEMI. This finding can warn policymakers to design better care services for patients at risk of acute MI during the times of increased air pollution.

Keywords: Air Pollution, Myocardial Infarction, Hospitalization, Airborne Particulate Matter

Date of submission: 22 July 2018, *Date of acceptance:* 01 Apr. 2019

Introduction

Recently, cardiovascular diseases (CVDs) are introduced as the major cause of death and disability around the world.[1] Different individual and environmental risk factors are associated with

How to cite this article: Davoodabadi Z, Soleimani A, Pourmoghaddas A, Hosseini SM, Jafari-Koshki T, Rahimi M, et al. **Correlation between air pollution and hospitalization due to myocardial infarction.** ARYA Atheroscler 2019; 15(4): 161-7.

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CVDs, while air pollution is currently recognized as the most common environmental risk factor for them.[2] Based on the World Health Organization (WHO) report, seven million premature deaths are attributed to air pollution each year, including 2.4 million deaths due to heart diseases and 1.4 million deaths due to stroke. More than four million deaths are assigned to ambient air pollution.[3]

The relationships between air pollutants and ischemic heart disease (IHD) have been demonstrated in numerous studies.[4,5] Longitudinal studies, particularly in developed countries, have highlighted the long-term effects of pollutants on the incidence of these diseases.[6-8] Time-series and case-crossover studies, on the other hand, have emphasized the short-term impacts of pollutants, especially suspended particles (2.5 μ).[9-11]

A total of 3245 persons/year per 100000 age-standardized disability-adjusted life year (DALY) in Iran is attributed to IHD and it is known as the major cause of mortality in the country.[12] Air pollution is an important risk factors for IHD. Using global models such as WHO's AirQ, multiple researches were conducted to evaluate the actual effect of air pollutant on specific diseases in Iran.[13,14] Other ecological surveys evaluated the effect of air pollution on acute coronary syndrome (ACS) in Iran.[15]

The correlation of air pollution with hospitalization and mortality of CVDs and respiratory diseases study (CAPACITY study) aimed to evaluate the correlation between air pollution and hospital admission or death from heart and lung diseases in Isfahan, Iran.[16] As part of the CAPACITY study, the present study was conducted to evaluate the relation between air pollutants and hospital admission due to ST-elevation myocardial infarction (STEMI) or non-ST-elevation myocardial infarction (NSTEMI).

Materials and Methods

This case-crossover (each case was considered as its own control) study was conducted in the framework of the CAPACITY study. The CAPACITY study was a multicenter well-defined research performed from March 2010 to March 2012. The data of all inhabitants of Isfahan either admitted in 15 hospitals of Isfahan or died with the definite diagnosis of cardiovascular or respiratory disease were collected. The disease was diagnosed based on the International Classification of Diseases-10th revision (ICD-10). The data of air pollutants were obtained from Isfahan Department of Environment (DOE). Time-series and case-crossover design were the two methods conducted for this study. In this study, the data of three main hospitals from 15 medical centers were gathered. More details about CAPACITY study have been presented elsewhere.[16]

This study extracted data related to CAPACITY participants who were hospitalized for myocardial infarction (MI) in three main hospitals of Isfahan (Chamran, Noor, and Al-Zahra Hospitals). The patients' file numbers were used to retrieve their records from hospital archives and collect additional information regarding their hospitalization status. The basic demographic data (age and gender), presence of diabetes mellitus (DM), hypertension (HTN), and current aspirin usage were recorded. The changes in ST segment during the hospital stay, laboratory data namely "troponin, urea, creatinine, and hemoglobin" levels at the admission time, left ventricular ejection fraction (LVEF) in echocardiography, the angiography results (if any), and patient's conditions at discharge were obtained from the files. The administration of fibrinolytic drugs for patients with STEMI was evaluated and recorded. Patients with incomplete records were excluded from the study.

Information about air pollutants namely carbon monoxide (CO), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), particulate matter smaller than 2.5 μm ($PM_{2.5}$) and smaller than 10 μm (PM_{10}), and ozone (O_3) were obtained from the CAPACITY data and presented as mean daily concentrations. In CAPACITY study, raw data were collected from six fixed pollution-monitoring stations supervised by Isfahan DOE. Hourly concentrations of pollutants were measured and recorded in Excel files by these stations. The mean concentration of each pollutant was calculated every day of the study span for the all regions of Isfahan. In addition, in order to consider the effects of climatic variables, the file containing mean daily temperature, humidity, and wind speed values was extracted. Data about air pollutants and climatic variables were recorded in current study at 24 hours, 48 hours, and 1 week before admission.

Quantitative variables were presented as mean ± standard deviation (SD) and were compared by independent sample t-test. Categorical data were expressed as frequency and percentage and chi-square test and Fisher's exact test were performed whenever was necessary. Patients were categorized in two groups of STEMI and NSTEMI based on clinical diagnosis. Crude conditional logistic regression model was used to evaluate the association between levels of air pollutants in the time points of 24 hours, 48 hours, and one week

Table 1. Patients' basic characteristics and the mean values of air pollutants and climatic variables during the study period

Patients' basic characteristics	Value	Air pollutants and climatic variables	Value (Mean ± SD)
Male gender [n (%)]	238 (74.60)	O_3 (ppb)	26.98 ± 13.17
History of DM [n (%)]	89 (27.80)	NO_2 (ppb)	43.76 ± 23.24
History of HTN [n (%)]	111 (34.70)	PM_{10} (µg/m^3)	126.18 ± 50.83
History of aspirin intake [n (%)]	59 (18.49)	SO_2 (ppb)	42.58 ± 32.91
ECG changes during admission [n (%)]	209 (65.50)	CO (ppb)	3.77 ± 2.00
Fibrinolytic drugs intake [n (%)]	149 (46.70)	$PM_{2.5}$ (µg/m^3)	53.91 ± 21.43
MI with ST-elevation [n (%)]	207 (64.89)	Temperature (°F)	57.70 ± 18.97
Living at clearance time [n (%)]	288 (90.28)	Dew point (%)	28.16 ± 8.66
Age (year) (mean ± SD)	63.15 ± 28.14	Wind speed (mile/h)	4.86 ± 2.13

DM: Diabetes mellitus; HTN: Hypertension; ECG: Electrocardiography; MI: Myocardial infarction; Ppb: Particle per billion; O_3: Ozone; NO_2: Nitrogen dioxide; PM_{10}: Particulate matter < 10 µm; SO_2: Sulfur dioxide; CO: Carbon monoxide; $PM_{2.5}$: Particulate matter < 2.5 µm; SD: Standard deviation

before admission and hospitalization for MI in the two groups. Adjusted models considering the confounding impacts of temperature, dew point, and wind speed were also performed in both groups. The results were expressed in form of odds ratio (OR) and 95% confidence interval (CI). All ORs were presented for each 10-unit increase in air pollutants. Statistical analysis was done with Stata software (version 9, Stata Corporation, College Station, TX, USA). Statistical significance was assessed at the level of 0.050.

Results

A total of 319 patients with MI with mean age of 63.15 ± 28.14 years, including 238 men (74.60%) and 207 (64.8%) with ST-elevation were recruited in this study. Table 1 summarizes the basic characteristics of study participants and mean daily concentrations of pollutants as well as daily temperature, dew point, and wind speed during the study period.

Tables 2 and 3 present the results of conditional logistic regression on the impact of each 10-unit increment in different air pollutants in association with the risk of hospitalization separately in patients with STEMI and NSTEMI, respectively.

As can be seen in both groups, majority of air pollutants showed direct association with risk of hospitalization; however, only the $PM_{2.5}$ levels at 48 hours before admission increased significantly the risk of hospitalization for STEMI both in crude and adjusted models, in which each 10-unit increment in $PM_{2.5}$ levels increased the hospitalization risk about 3.70 times (95% CI: 1.69-7.69 from adjusted model).

Table 2. The relationship between 10-unit increase in air pollutants before admission and the risk of hospitalization in patients with ST-elevation myocardial infarction (STEMI)

Time of exposure to pollutants	Pollutants	Crude model			Adjusted model*		
		OR	95% CI	P	OR	95% CI	P
24 hours before hospitalization	$PM_{2.5}$	1.30	(0.96-1.75)	0.088	1.37	(0.97-1.96)	0.077
	PM_{10}	1.02	(0.97-1.06)	0.475	1.03	(0.97-1.07)	0.301
	SO_2	1.05	(0.89-1.25)	0.519	1.06	(0.90-1.27)	0.456
	CO	3.03	(0.31-33.33)	0.341	2.86	(0.29-33.33)	0.371
	NO_2	1.09	(0.93-1.28)	0.297	1.07	(0.90-1.27)	0.445
	O_3	1.10	(0.81-1.50)	0.533	1.14	(0.83-1.55)	0.423
48 hours before hospitalization	$PM_{2.5}$	3.03	(1.56-6.25)	0.001	3.70	(1.69-7.69)	0.001
	PM_{10}	1.01	(0.97-1.06)	0.511	1.02	(0.97-1.06)	0.483
	SO_2	0.97	(0.85-1.09)	0.609	0.97	(0.85-1.09)	0.609
	CO	1.02	(0.85-1.22)	0.836	1.02	(0.85-1.23)	0.825
	NO_2	1.03	(0.89-1.17)	0.727	1.02	(0.88-1.17)	0.668
	O_3	0.96	(0.76-1.22)	0.718	0.93	(0.72-1.19)	0.531
1 week before hospitalization	$PM_{2.5}$	1.12	(0.82-1.52)	0.493	1.04	(0.73-1.49)	0.813
	PM_{10}	1.01	(0.97-1.05)	0.585	1.02	(0.98-1.06)	0.353
	SO_2	0.96	(0.87-1.07)	0.404	0.95	(0.86-1.05)	0.375
	CO	1.02	(0.92-1.12)	0.816	1.01	(0.90-1.12)	0.917
	NO_2	1.04	(0.93-1.16)	0.458	1.03	(0.92-1.16)	0.642
	O_3	0.86	(0.71-1.04)	0.105	0.89	(0.72-1.05)	0.150

* Adjusted for wind speed, temperature, and dew point; P-values resulted from conditional logistic regression

$PM_{2.5}$: Particulate matter < 2.5 µm; PM_{10}: Particulate matter < 10 µm; SO_2: Sulfur dioxide; CO: Carbon monoxide; NO_2: Nitrogen dioxide; O_3: Ozone; OR: Odds ratio; CI: Confidence interval

Table 3. The relationship between 10-unit increases in air pollutants before admission and the risk of hospitalization in patients with non-ST-elevation myocardial infarction (NSTEMI)

Time of exposure to pollutants	Pollutants	Crude model			Adjusted model*		
		OR	95% CI	P	OR	95% CI	P
24 hours before hospitalization	$PM_{2.5}$	1.30	(0.46-3.70)	0.620	7.69	(0.17-333.33)	0.290
	PM_{10}	1.02	(0.93-1.13)	0.579	1.03	(0.92-1.14)	0.550
	SO_2	1.10	(0.83-1.46)	0.480	1.12	(0.83-1.50)	0.438
	CO	1.00	(0.06-15.98)	> 0.999	1.50	(0.08-27.30)	0.760
	NO_2	1.11	(0.78-1.58)	0.529	1.20	(0.81-1.77)	0.340
	O_3	0.93	(0.45-1.93)	0.845	0.92	(0.44-1.95)	0.840
48 hours before hospitalization	$PM_{2.5}$	5.00	(0.69-50.00)	0.110	1.43	(0.11-16.66)	0.830
	PM_{10}	1.01	(0.93-1.08)	0.890	1.01	(0.93-1.08)	0.888
	SO_2	1.01	(0.99-1.04)	0.110	1.01	(0.99-1.04)	0.130
	CO	1.03	(0.78-1.37)	0.800	1.02	(0.77-1.36)	0.801
	NO_2	1.31	(0.93-1.83)	0.110	1.34	(0.93-1.92)	0.101
	O_3	0.64	(0.37-1.10)	0.102	0.64	(0.37-1.10)	0.120
1 week before hospitalization	$PM_{2.5}$	5.00	(0.69-50.00)	0.500	1.43	(0.11-16.66)	0.830
	PM_{10}	1.01	(0.93-1.08)	0.890	1.01	(0.93-1.08)	0.887
	SO_2	1.01	(0.99-1.04)	0.110	1.01	(0.99-1.04)	0.130
	CO	1.03	(0.78-1.37)	0.800	1.02	(0.77-1.36)	0.803
	NO_2	1.31	(0.93-1.83)	0.110	1.34	(0.93-1.92)	0.103
	O_3	0.64	(0.37-1.10)	0.103	0.64	(0.37-1.10)	0.120

* Adjusted for wind speed, temperature, and dew point; P-values resulted from conditional logistic regression
$PM_{2.5}$: Particulate matter < 2.5 μm, PM_{10}: Particulate matter < 10 μm; SO_2: Sulfur dioxide; CO: Carbon monoxide; NO_2: Nitrogen dioxide; O_3: Ozone; OR: Odds ratio; CI: Confidence interval

Discussion

The current study investigated the association of levels of different air pollutants with hospitalization in patients with STEMI and NSTEMI in Isfahan. In this study, $PM_{2.5}$ concentrations at 48 hours before admission were significantly related with hospitalization in patients with STEMI. In spite of direct associations between the majority of other different air pollutants with hospitalization in studied time points, they did not show any significant relation. Some studies had proposed a direct significant association between the level of air pollutants, except for O_3, and MI incidence.[17] Multiple studies have proposed significant relation between some air pollutants and the admission with diagnosis of STEMI or NSTEMI, while others have rejected the presence of such association. A study in Belgium used a crossover model to evaluate 11428 patients with records in the STEMI registration system during 2009-2013. It found the incidence of STEMI to have significant positive correlations with elevated levels of $PM_{2.5}$, PM_{10}, and NO_2 at 24 hours before MI. Elevations in PM_{10} were more strongly related with STEMI in patients over 74 years of age. In the case of NO_2, however, patients below 54 years of age were at greater risk. This study found the strongest relationship between NO_2 and STEMI.[18] Another study on 673 patients with MI detected the strongest significant association between $PM_{2.5}$ and STEMI just one hour before the onset of STEMI. The relationships between STEMI and PM_{10} levels at 3, 12, and 24 hours before MI were not significant. In addition, no relationship was observed between NSTEMI and exposure to $PM_{2.5}$. Moreover, in patients with a history of HTN, the effect of PM was more prominent. The only method that was used in this study for evaluation of $PM_{2.5}$ effect was case-crossover approach.[19]

Another case-crossover study in the United Kingdom (UK) assessed the association between airborne contaminants and STEMI and NSTEMI. It investigated nearly 523000 patients admitted during 2003-2010. The database of Myocardial Ischaemia National Audit Project (MINAP) in England was used. Air pollutants exposure was evaluated immediately and two days before the onset of the acute cardiac event. The results showed no links between pollutants and STEMI. Contrary to the two studies discussed above, there was a significant relationship between maximum hourly NO_2 concentration per day and the incidence of NSTEMI. This correlation persisted after adjustments for O_3 and $PM_{2.5}$.[20]

In another study, the relationship between hospitalization due to STEMI and air pollutants was investigated using a time-series model in Tabriz, Iran. The results showed STEMI to be significantly

related with maximum hourly NO_2 concentration on the day of admission, and mean 24-hour CO concentration on the pre-admission day. The relationship between 24-hour CO and STEMI was stronger than that between STEMI and NO_2. The study reported no relationships between STEMI and PM, SO_2, and O_3 concentrations.[21]

A time-series study in Spain also revealed associations between hospitalization due to STEMI and increased $PM_{2.5}$, PM_{10}, and NO_2 concentrations.[22] A two-year case-crossover study on 106000 patients with STEMI and 12719 patients with NSTEMI in 26 cities of China indicated an association between increased $PM_{2.5}$ concentration before the onset of the MI and higher risk of STEMI. The incidence of STEMI had significant relationships with $PM_{2.5}$ levels zero to five days before the incidence of the condition. There was, however, no link between $PM_{2.5}$ and NSTEMI.[23]

A recent study on 208 Iranian patients used a case-crossover model to explore the relationship between STEMI and airborne contaminants. According to the results, STEMI was significantly associated with PM_{10} and $PM_{2.5}$. In addition, higher age, DM, and multi vessel involvement had stronger relationships with PM concentration.[24] Evidently, most studies have identified PM as the pollutant associated with the occurrence of STEMI. Furthermore, the majority of previous studies, except for a few,[20] have rejected the presence of significant relationships between pollutants and NSTEMI. Additionally, various studies with different models have used different exposure times. Our study also highlighted a relationship between PM concentrations 48 hours before hospital admission and hospitalization due to STEMI.

Several mechanisms, including coagulation, inflammation, vascular dysfunction, and autonomic dysfunction are involved in ACS (e.g., MI). All these mechanisms lead to thrombosis, binding of circulating platelets to each other, and vessel wall damage. An acute plaque rupture occurs in STEMI which is absent in NSTEMI. In the meantime, endogenous thrombolysis has a critical role in the clot autolysis and preventing complete vascular obstruction. Indeed, the balance between thrombosis and thrombolysis results in the occurrence of STEMI and/or NSTEMI.[25,26] Air pollutants, especially $PM_{2.5}$, appear to increase platelet and fibrinogen activities, stimulate plaque formation, and decrease endogenous thrombolysis.[27,28] This can justify the observed relationship between pollutants and STEMI.

An important limitation in our study was lack of data on patient complaints, clinical demonstrations, and paraclinical outcomes in the health information system at the time of admission. However, other studies, particularly in developed countries, did not encounter such a limitation, because all precise information of patients is available in a data registration system that can facilitate the implementation of extensive studies at a lower cost and time. The other limitation of our survey was the quite small number of study population that leads to relatively unreliable conclusion.

Conclusion

This survey outlined a significant correlation between $PM_{2.5}$ and the increased risk of STEMI. Although the majority of air pollutants showed a direct association with increased risk of hospitalization for STEMI and NSTEMI, none of the observed associations, more likely due to low sample size and particularly few patients in studied subgroups, were statistically significant. These findings can encourage policymakers to design policies for pollutant reduction. It also emphasizes the need for providing better care services on days with elevated air pollution levels and the following days for patients with higher risk of acute MI.

Acknowledgments

We appreciate the assistance and invaluable support of the director and members of Isfahan Cardiovascular Research Institute, Isfahan Provincial Governor Office and its Crisis Management Office, and Isfahan DOE. This manuscript has been written based on a cardiology residency dissertation (code: 396491) approved by Department of Cardiology, School of Medicine, Isfahan University of Medical Sciences.

Conflict of Interests

Authors have no conflict of interests.

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