Myocardial infarction and depression: A review article

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Abstract

BACKGROUND: Depressive symptoms are common among post myocardial infarction (MI) patients and may cause negative impacts on cardiac prognosis. Depression is observed in 35-45% of MI patients. While depression is an independent risk factor for MI, post-MI depression has been shown to be a risk factor for mortality, morbidity, and decreased quality of life in patients. The link between depression and MI is bidirectional in which behavioral and biological mechanisms have been proposed to be involved. The combination of these mechanisms is likely to involve in increasing the risk of mortality. Epidemiological studies have shown the link between depression and increased risk for development of cardiovascular disease, MI, and cardiac mortality. The adverse impact of depression on prognosis of heart disease is preventable with the right treatment. A number of therapeutic approaches including cardiac rehabilitation, social support, cognitive behavioral therapy, and antidepressants have been suggested for post-MI depression. However, due to their adverse effects, tricyclic antidepressants are recommended to be avoided for treating post-MI depression. On the other hand, administering selective serotonin reuptake inhibitors (SSRIs) shortly after MI would lessen their major side effects.

Keywords: Myocardial Infarction, Depression, Mortality, Treatment of Depression, Behavioral Mechanisms, Biological Mechanisms.

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Introduction

Incidence of depressive symptoms after myocardial infarction (MI) is a very common psychological problem with negative influences on prognosis of cardiac disease.[1] Increasing evidence shows that depression can be an independent risk factor for MI and its associated mortality and complications. It can also decrease quality of life in patients with MI.[2,3] Therefore, preventing depression through noticing the psychological status of patients with MI can in turn inhibit its negative impacts on disease process after MI. Such prevention requires adequate knowledge and understanding about predisposing and revealing factors of depression in these patients.[4,5] Reports indicate that depression during hospitalization occurs in more than 45% of patients following the incidence of MI.[6] Furthermore, the overall assessment of various studies shows that major depression (MD) happens in 15-30% of cases while mild depression or depressive symptoms are seen in approximately 20% of patients with MI. Therefore, it can be stated that different forms of depression have been reported in 35-50% of patients with MI.[7-9]

Depression

Mood disorders include a wide range of psychological disorders whose clinical image is mostly formed by mood disturbances.[10,11] Natural, high and/or depressed moods are among the wide spectrum of different moods a normal individual experiences. While healthy people are able to control their emotional and mood status, subjects with mood disorders feel not to have control over their moods.[10] As the most prevalent mood disorder,[11] depression involves sadness, depressed mood, apathy, and inability in enjoying.[10] Although depression seems to have a high prevalence in Iran, exact statistics are not available. In addition to the incidence of depression at community level, depression is frequently seen in patients who refer to all health centers including various specialized clinics and public hospitals.[12] A common survey by the World Health Organization and the World Bank has illustrated that depressive disorders are among the top 10 leading causes of inability and disability in the world.[13] If not properly treated, depression can cause physical and emotional disability, early mortality, decreased efficiency, and family problems.[14] Furthermore, great attention has

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been recently paid to economic costs and negative consequences of depression.[14] Considering the high prevalence and debilitating nature of depressive disorders and the high costs they impose on the individuals and society, they should receive special attention in the field of mental health. Studying and reviewing the etiology, symptomatology, and treatment of various forms of depressive disorders in different populations such as cardiac patients are definitely of high importance.

Depressive Signs and Symptoms: Despite the diversity and extent of depressive signs and symptoms, depression has four main categories of symptoms including mood symptoms such as sadness, nostalgia, and shock, cognitive symptoms such as extreme pessimism and hopelessness toward future, motivational symptoms such as mental and motion slowness and inability in decision-making, and somatic symptoms such as appetite loss, decreased libido, weight loss, lack of energy, and bodily pains. However, diagnosis of depression does not require the simultaneous existence of all symptoms. In fact, higher number and intensity of symptoms would result in a more confident judgment of depression.[15]

Natural Depression and Depressive Disorder: There is a very fine line between natural depression and depressive disorder and the same depressive symptoms are experienced in both problems. However, clinical depression is different from natural depression regarding the number, intensity, and duration of symptoms. Therefore, persistent number, intensity, and duration of depressive symptoms would indicate the incidence of depressive disorder. In summary, in depressive disorders, the range, intensity, and duration of symptoms are high enough to disrupt and impair an individual's functioning.[16] In addition, in depressive disorder, depressed mood has a certain qualification which distinguishes it from the natural feelings of sadness or grief. Moreover, patients would describe depressive disorder as a type of psychological excruciating pain.[10]

Depressive disorders have been categorized into different groups based on the clinical image, symptom duration, and etiology. The diagnostic and statistical manual of mental disorders fourth edition text version (DSM-IV-TR) has generally categorized depressive disorders into three major depressive disorders, dysthymic disorder, and depressive disorder not otherwise specified. Some of these categories have been further divided into subgroups.[10]

Depression in Individuals with Somatic Diseases: Although often neglected, depression is a comorbid disorder commonly experienced by patients with somatic diseases. The prevalence of depression in general population has been estimated as 6-25%.[16] However, its incidence is higher in individuals with somatic or physical diseases and particularly among hospitalized patients. The prevalence of this disorder is different among various somatic diseases and has been reported between 5-50%.[17] Some previous studies which used screening methods such as Beck's Depression Inventory reported the prevalence rate of depression among somatic patients as 5-66%.[18] It is stated that averagely one-third of somatic patients will suffer from depression.[7] Moreover, more than 46% of elderly patients with acute diseases suffer from depression.[19]

Since treatment of depressive disorders in physical patients can prevent further subsequent problems, they need to be well diagnosed and assessed.[18] Ignoring depression as a comorbidity of somatic diseases can cause adverse consequences and complications. Depression in somatic patients has frequently been reported to have negative impacts on disease consequences. Negative consequences of depression in somatic patients include increased disability, exacerbation of treatment and prognosis, increased duration of diseases, increased level of health care costs, increased functional difficulties, increased rate of mortality and comorbidities, decreased quality of life, and decreased treatment acceptance.[20] Despite the alarming evidence about the consequences of depression accompanied by physical diseases, it has not been widely considered. Therefore, depression is not usually diagnosed in somatic patients.[21] Although despite the high prevalence of this disorder among somatic patients, it is well treatable, it mostly remains untreated.[22]

Depression following MI: Results of a study demonstrated that depression following MI is not only a passing and temporary response to a stressful event, but patients may suffer from depression even long after MI.[1] Many studies have shown that depressive symptoms following MI are an independent risk factor for mortality of patients.[3,23] The outcomes of depression are thus comparable to those of left ventricular functioning and insufficient medical care.[7,24] Gross mortality risk (without controlling other risk factors) 6 months after MI in patients with depressive symptoms are almost 6 times greater than those without depressive symptoms. This high risk of mortality can still be seen 18 months after MI.[25] Even after controlling the effects of other main predictors of MI complications and mortality such as left ventricular ejection fraction (LVEF), Killip class, age, and history of MI, the impact of depression following MI remains as an independent predictor of long-term mortality.[23,25]

In addition, findings of a study have shown high risk of mortality not only in patients diagnosed as depressed based on DSM-IV, but also in individuals with symptoms of depression who cannot be diagnosed as depressed according to DSM-IV.[25] Recent studies have indicated that even the incidence of depressive symptoms following MI can have a negative impact on prognosis of cardiac disease. The association between mild symptoms of depression and increased risk of mortality following MI is a new finding which emphasizes the need to consider depression following MI.[24] In fact, mild symptoms of depression indicate levels of negative stress and emotions experienced by a patient.[25]

Depression is a major and important predictor of disability,[26] low quality of life,[27] and delayed return to work[28] following the MI. Depression following the MI is correlated with irregular participation in cardiac rehabilitation programs[23] and the incidence of high-risk behaviors.[5] Furthermore, depression following the MI may cause decreased acceptance of treatment by the patient and thus exacerbate the MI treatment.[29] In summary, many studies have emphasized that regardless of MI features and its cardiac outcomes, depression is an independent and strong risk factor for mortality following MI. The correlation between depression and mortality risk factor has been reported to be a graded relationship. Thus, more severe depression would result in a higher mortality rate. Increased mortality risk among depressed MI patients has been suggested between 4 months to 10 years following MI.[30]

Prevalence of Depression following MI: Depression has been reported in a significant percentage of patients with MI.[24] The prevalence of depression following the MI varies in different populations and using different tools.[31] Most studies have reviewed the prevalence of depression following MI during primary hospitalization and at a number of time intervals after discharge. However, depression has been defined differently in various studies. In fact, the results would be different if depression is assessed by one scale rather than through clinical interview and considering diagnostic criteria.[31] Some patients suffer from depression during the initial hospitalization. Therefore, some studies have reviewed the continuation of depression process and its frequency (prevalence) after discharge. Such studies have considered various time intervals ranging from a month and to longer periods after discharge. In review articles and literature concerning the prevalence of depression following MI, full texts and abstracts of more than 30 articles during 1986-2004 were available. While certain articles reported the prevalence of depression during hospitalization and its outcomes during the first 6 months after MI, some other researchers reported the prevalence of depression and its outcomes until 12 months later.[7] As indicated earlier, various studies have reported the prevalence of (major and mild) depression as in patients with MI 35-50%.[7,31]

Depression and Risk of Cardiovascular Diseases: Suffering from cardiovascular diseases (CVDs) is the result of interactions between many factors. The role of some of these factors such as hypercholesterolemia, smoking, hypertension, diabetes, age, genetic factors, and gender has been well identified. Modification and control of these classic risk factors currently constitute the principles of CVD control recommendations and programs.[32] Moreover, epidemiological studies have shown the correlation between clinical depression or depressive symptoms and increased risk of subsequent CVD, MI, and cardiac death.[33] Likewise, prognostic studies have frequently emphasized this relationship. In a study, 1190 medical male students were followed-up for 40 years. The follow-up indicated that during this time, 12% of these individuals suffered from depression. The history of depression in turn doubled the risk of CVD or MI.[34] In another study in the U.S., 4492 subjects with no proved coronary heart disease were evaluated for 6 years. The results of this study showed that even after controlling other coronary risk factors, depressive symptoms had a significant and independent relationship with CVD and mortality.[35]

Potential Mechanism of the Relationship between Depression following MI and its Adverse Outcomes: No known mechanism has ever been able to completely explain the correlation between depression following MI and increased rate of mortality.[36] However, so many potential mechanisms have been suggested that can be divided into two overall categories of behavioral mechanisms and biological mechanisms. A combination of these two categories would probably cause increased risk of mortality in patients with MI.

1. ***Behavioral Mechanisms***: Depression is able to exacerbate other risk factors of MI. It may increase the consumption of tobacco, alcohol, and high-fat foods, lead to wrong lifestyle, and increase some high-risk behaviors.[37] In addition, depression is a major and important predictor of low quality of life in the year following MI.[27] Depression can make the treatment of MI treatment more difficult by reducing motivation to participate in cardiac rehabilitation and therapeutic programs,[36] increasing high-risk behaviors,[38] and reducing treatment acceptance by patients.[5,29]

2. *Biological Mechanisms*: In addition to behavioral outcomes of depression, some physiological mechanisms should exist between psychological depressive symptoms and mortality of patients with MI. Some of these mechanisms are arrhythmia, homeostasis, and inflammation.

Studies have shown that arrhythmia is one of the mechanisms that explain the correlation between psychological factors such as depressive symptoms and sudden cardiac death.[39] Comparing depressed and non-depressed patients with cardiac disease who have been matched in terms of age and sex showed that depressed cardiac patients suffered from decreased heart rate variability (HRV).[40] Decreased HRV indicates abnormal sympathetic tone to heart with or without low input (abnormal) parasympathetic system. This situation can be an intermediate mechanism between depression following MI and increased risk of cardiac death. Patients with depression following MI have a higher rate of premature ventricular contractions (PVC) compared to non-depressed cardiac patients.[41] Reviewing patients with MI showed a direct correlation between PVC and depression during the first 10 hours after the incidence of MI.[42] The results of studies on human and animal samples which connected the cardiac events with psychological factors showed the mechanism of arrhythmia to be highly important in sudden cardiac death.[43] The fundamental premise in the abovementioned studies is that vulnerable myocardium following MI, acute ischemia, and emotional arousal can simply cause ventricular arrhythmia.[42]

Although some findings have indicated a correlation between PVC and increased rate of mortality following MI, even controlling arrhythmia cannot decrease the mortality rate in depressed patients.[44] Results of a study illustrated that preventing or treating depression is more necessary than controlling arrhythmia to improve and increase longevity in patients with PVC.[43,44]

Homeostasis is another potential mechanism in establishing a correlation between depression and adverse outcomes following MI. Evidence has indicated increased platelet activity in depressed patients. Neurobiological basis of depression has a very close association with changes in serotonin receptors and transmission routes. Platelet receptors, including 5-hydroxytryptamine-2 (5HT2), in depressed patients would have some changes that can cause increased rate of platelet activity.[45]

Inflammation is one of the other potential mechanisms that have been suggested to justify the association between depression and adverse effects of MI. The immune system can play a role in progression of MI symptoms and its clinical manifestations. On the other hand, the role of psychological factors as risk factors of MI has been confirmed. Psychological factors may influence on the progression of MI symptoms through psychoneuroimmunologic mechanisms.[33] The onset and exacerbation of atherosclerosis are associated with the severity of vascular inflammation which is identified with increased level of inflammatory mediators such as interleukin 1 (IL1), interleukin 6 (IL6), tumor necrosis factor alpha (TNF-α), and C-reactive protein (CRP). CRP has been particularly introduced as a significant predictor of increased risk. In individuals without coronary disease, an increased CRP level can predict higher risk of acute coronary syndrome. In patients with a history of acute MI, higher level of CRP is correlated with poor prognosis, particularly in males. Therefore, adverse coronary outcomes and depression can be connected together through pathways related to CRP and other inflammatory indicators.[45]

Depression can cause changes in the immune system. Immunological correlates of depression include increased peripheral blood leukocytes (especially neutrophils and monocytes), decreased number of lymphocytes, increased concentration of cytokines in the blood (IL-6 and TNF-α), deceased functional indicators, and increased antibodies for viruses such as cytomegalovirus.[33] In addition, high activity of corticotropin-releasing hormone (CRH) is well seen in depression. These mechanisms can cause processes involved in changing atherosclerotic plaques from stable to unstable status. They would therefore be the potential factors in establishing the predicting role of depression in coronary syndromes and prognosis of coronary diseases in MI patients.[46]

Some other mechanisms including increased sympathetic activity are also effective on establishing a relationship between depression and MI complications. Depression is correlated with increased activity of sympathetic nervous system. Recent evidence has shown that depressed cardiac patients have changes in heart rate speed which indicates a possible relation of depression with changes in balance between sympathetic and parasympathetic systems.[47] The activity of the sympathetic nervous system not only is correlated with increased myocardial ischemia and arrhythmia, but may also increase blood pressure, insulin resistance, and infection vulnerability. Furthermore, depression is correlated with two adverse outcomes and impacts following MI, i.e. omega-3 fatty acid

deficiency and increased rate of homocysteine, which seem to exacerbate coronary disease.[48]

Therefore, many behavioral (particularly reduced attention to medical recommendations), neurohormonal, immunological, and coagulatory mechanisms have been suggested as ways through which depression can have serious and risky outcomes for patients with MI.[6,9,41] In general, it appears that these mechanisms, particularly increased coagulability or arrhythmia, can directly cause increased mortality in depressed coronary patients.

Treating Depression Following MI: Although depression has adverse consequences for patients with MI, its negative effects on prognosis of coronary disease and physical status of patients are potentially preventable.[49] Depression in normal patients (except those with coronary disease) is often treated acceptably. In spite of the less clear success rate of treatment in coronary patients compared to other individuals, depressive disorder in coronary patients is controllable, too.[23,41,49]

Nevertheless, previous studies have shown that less than 20% of patients with depression following MI underwent depression treatment. In other words, despite the lethal and serious effects of depression on prognosis of cardiac disease and physical status of patients, depressive symptoms following MI are ignored in 80% of patients.[50] Therefore, it is obvious that diagnosis of depression in such patients, particularly hospitalized patients with high risk for the incidence of depressive symptoms following MI, is of extremely high importance in order to prevent from or implement subsequent therapeutic actions. Preventive measures, as well as therapeutic actions for depression following MI can well reduce the mortality risk, increase the acceptance of therapeutic methods and rehabilitation, promote health level and quality of life, prevent from the incidence of high-risk behaviors, and ultimately prevent negative consequences of depression in coronary disease course.[51,52]

Many therapeutic methods including cardiac rehabilitation, social support, cognitive behavioral therapy (CRT), and antidepressants have been used for depression following MI. However, until recently, there was no approved evidence suggesting the efficacy and safety of antidepressants in cardiac patients. More attention has lately been given to the effects of antidepressants on subsequent cardiac complications and mortality of patients with MI. Although selective serotonin reuptake inhibitors (SSRIs) are safer than tricyclic antidepressants,[49] antidepressants can have adverse impacts on cardiac performance.[49,53]

Findings of studies concerning the efficacy of SSRIs in patients with depression following MI are limited. Roose et al. showed paroxetine and nortriptyline to be effective in reducing depression in elderly patients with coronary disease. They also found fluoxetine to be less effective than nortriptyline in elderly cardiac patients with severe depression.[54] Tricyclic antidepressants cause increased heart rate, slow interventricular cardiac conduction, decreased orthostatic hypotension, and result in the incidence of PVC in depressed patients with cardiac or non-cardiac diseases.

In a study in 2000, the appropriateness of fluoxetine in depressed patients with MI was reviewed. The results showed that fluoxetine was effective in reducing hostility particularly in patients with mild depression. Moreover, it was an appropriate drug to treat depressed patients following MI whose depression had begun three months following the incidence of MI.[55]

In a large study, the complications and effectiveness of pharmaceutical and non-pharmaceutical treatments for depression following MI were evaluated. It compared the effects of a psychosocial intervention with cognitive behavioral treatment along with SSRIs with conventional treatment on cardiac outcomes of three groups of patients (total number = 2481) during 1996-2001 in 8 clinic centers in the U.S. These three groups included a group of patients with depression following MI, a group of patients with low perceived social support following MI, and a third group including patients with both complications. These patients were followed-up for 29 months. The results indicated that in terms of mortality, there was no significant difference between the groups who received conventional treatment and those who underwent pharmaceutical and non-pharmaceutical interventions. Furthermore, in this study, there was no significant difference between mere depression improvement, low perceived social support, and the combination of the two problems. However, the results showed that therapeutic methods have been effective on improvement of psychosocial problems (depression and perceived social support) in patients. Although the recovery resulted from psychosocial intervention was lower than expected compared to conventional treatment, the intervention improved depression and social isolation.[56]

A controlled randomized clinical trial was conducted on MI patients with major depressive disorder in 40 cardiac and psychiatry outpatient clinics in the U.S., Europe, Canada, and Australia during 1997-2001. It aimed to compare the effects of

sertraline with placebo. It evaluated changes in baseline LVEF, cardiac complications, adverse coronary heart events, Hamilton Scale scores, and overall clinical assessment of depression. The results showed that sertraline had no significant impact on LVEF and various cardiac indicators of patients. However, adverse cardiac outcomes occurred in 14.5% of the patients treated with sertraline while the rate was 22.4% in the placebo group. The overall clinical assessment of depression and Hamilton Scale scores suggested the significant efficacy of sertraline compared to placebo. Generally, this study indicated SSRI antidepressants, e.g. sertraline, as more effective drugs in treatment of post-MI depression among patients without any other threatening diseases. Therefore, psychological interventions and treatments with SSRIs were concluded to successfully reduce depressive symptoms in MI patients.[44]

Although many antidepressants are considered to treat patients following MI, most tricyclic antidepressants are avoided due to their effects on increasing heart rate, causing orthostatic hypotension, slow interventricular cardiac conduction, and vulnerability to ventricular arrhythmias. On the other hand, SSRI antidepressants would not have serious side effects if prescribed immediately after MI.

Conclusion

In general, it should be noted that although depression in patients with somatic diseases is experienced as comorbidity, it is ignored in many cases. Considering the high prevalence of post-MI depressive symptoms and their negative effects on prognosis of cardiac disease, special attention must be paid to this psychological problem in MI patients. The results obtained from reviewing the previous articles concerning post-MI depression need to be taken into account. We found depressive symptoms to continue long after MI and to be independent risk factors for mortality among patients. Identifying the main mechanisms through which depression influences the incidence of MI complications is necessary in determining prognosis and treatment of such patients. Accordingly, identifying MI patients with depression and also those at the risk of depression is undoubtedly a quite important subject in clinical practice. The relationship between depression and MI is a reciprocal relationship. Epidemiological studies have shown the correlation between clinical depression or depressive symptoms and increased risk of subsequent coronary heart diseases, MI, and cardiac death.

Fortunately, negative impacts of depression on prognosis of coronary disease and somatic status of patients are potentially preventable. Despite the fact that success rate of depression treatment in cardiac patients is not that obvious compared to other individuals, a combination of pharmaceutical and non-pharmaceutical treatments can also control depressive disorder in cardiac patients.

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Conflict of Interests

Authors have no conflict of interests.

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