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Depression follows Myocardial Infarction

Dr. Anil Kumar Batta

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ABSTRACT

Unfortunately, depression is now a well documented independent risk factor of coronary artery disease. Post-myocardial infarction (MI) patients with a clinician-diagnosed depressive disorder or self-reported depressive symptoms carry a 2.0- to 2.5-fold increased relative risk of new cardiovascular events and cardiac mortality Questions about the pathophysiologic mechanism of depression in this setting are paralleled by uncertainties about the optimal treatment of depression for patients recovering from a myocardial infarction and by a lack of knowledge about whether treating depression lowers the associated increased mortality risk. Ongoing research studies will help to determine the benefits of psychosocial interventions and of antidepressant therapy for patients soon after myocardial infarction. Although the identification of depression as a risk factor may by itself be a reason to incorporate a comprehensive psychological evaluation into the routine care of patients with myocardial infarction. This practice should certainly become standard if studies show that treating depression reduces the increased mortality risk of these patients. Treatment with selective serotonin reuptake inhibitors (SSRIs) significantly improved outcome of what one can become a major catastrophe (Jonge et al). Although non-randomized trial, this could essentially relate to intrinsic pharmacologic properties of SSRIs causing, for example, restoration of subtle platelet hyperactivity in the depressed. Clearly, before another clinical trial of depression treatment is initiated in post-MI populations, we need more information on the "cardio toxic" subtypes of depression. But the query still persists. Keeping all these chronic outbursts in mind a study was conducted on indoor and outdoor patients attending or admitted in GGS Medical College & Hospital, Faridkot. 67 MI diagnosed and treated patients attending the post MI clinics were interviewed for symptoms of depression. We investigated if there are differences in pre- and post-MI characteristics between these subtypes. Persons who are depressed and who have pre-existing cardiovascular disease have a 3.5 times greater risk of death than patients who are not depressed and have cardiovascular disease. A comparison was made between first-ever and ongoing or recurrent depression on demographic and cardiac data, personality, and depression characteristics Results: Approximately 165 percent of patients with acute myocardial infarction report experiencing symptoms of depression in a structured study. Major depression is present in 15 to 22 percent of these patients. Depression is an independent risk factor in the development of and mortality associated with cardiovascular disease in otherwise healthy persons. Cognitive-behavior therapy is the preferred psychological treatment. Selective serotonin reuptake inhibitor antidepressants are the recommended pharmacologic treatment because of the relative absence of effects on the cardiovascular system. The combination of a selective serotonin reuptake inhibitor with cognitive-behavior therapy is often the most effective treatment for depression in patients with cardiovascular disease.

1. Introduction

Depression commonly co-occurs with myocardial infarction (MI) (1-3). However, the relationship is complex and involves many unresolved questions of both theoretical and clinical importance.

* Corresponding Author : **Anil Kumar Batta**
Professor, Dep't of Medical Biochemistry,
Baba Farid Univ.of Health Sciences,
Faridkot, Punjab, India

1. How common is a history of major depression in patients who survive an MI?

2. What are the medical and psychiatric correlates of a history of major depression?

3. Does a history of major depression influence patients' medical and psychiatric evolution during the year after MI? tween them.

4. What are the correlates of depression in the hospital?

5. What is the evolution of patients depressed in the hospital?

6. What is the evolution of patients who become depressed after discharge?

7. Is it possible to predict post discharge depression?

8. Do patients who are depressed in the hospital differ in baseline characteristics from those who only become depressed post discharge?

9. Do in-hospital depressions differ in clinical characteristics from depressions that begin post discharge?

MI depression is related cardiovascular risk. There is strong evidence that individuals with depression show increased morbidity and mortality from coronary heart disease (Rugulies, 2002) but the mechanisms involved remain unclear. Individuals with a history of recurrent depression, who are otherwise healthy, show increased inflammation, platelet activation, endothelial dysfunction, and reduced heart rate variability and baroreceptor sensitivity. However, with the exception of platelet function, which improves with selective serotonin reuptake inhibitors, these anomalies are not corrected by antidepressant treatment. Furthermore, endothelial function and baroreceptor sensitivity, which can lead respectively to progression of the atherosclerotic process and to sudden cardiac death, do not improve when depressive symptoms are in remission (Broadleyet al, 2006). Thus there is no evidence that treatment of depressive symptoms post-myocardial infarction corrects these underlying pathological processes and, if it does not, cardiac outcomes disclosed by clinical trials is unlikely to show improvement irrespective of their statistical power. By analogy, although hyperglycemia characterizes diabetes, tight glucose control alone has only a modest impact on cardiovascular events. Similarly, depressive illness is characterised by acute episodes of depression, but other systemic abnormalities are present and persist between acute depressive episodes. Accordingly, it may be unreasonable to believe that treatments assessed by their influence on the affective state alone will reduce cardiovascular events.Atherosclerosis begins in childhood and becomes manifest much later in life, with myocardial infarction as a very late presentation. Similarly, depression is a lifelong disorder with onset in early adulthood. It should be noted that currently depression is not even included in cardiovascular risk tables and that individuals with depression might benefit from introduction of statins, or other preventative measures.Coronary heart disease and depression are two major public health problems and it is of concern that reports of treatments for depression failing to enhance survival post-myocardial infarction may result in less interest in studying the links be

MATERIALS AND METHODS

The study was conducted in the coronary care unit (CCU) of a large tertiary care teaching hospital after obtaining permission from the Institution's Review Board. Patients admitted to the CCU with acute myocardial infarction showing ST elevation in ECG were consecutively enrolled. Those younger than 20 years, those who were unable to comprehend and/ or react meaningfully were excluded from the study. The patients were informed about the purpose and nature of the study. They were enrolled in the study after obtaining written informed consent. Through direct questioning, the patient's subjective stress level during the 2-4 week period preceding the acute coronary event was assessed. The stress was graded as I to IV using the Subjective Stress Functional Classification (SS-FC): SS-FC I: No perceived mental stress or only basal levels, SS-FC II: More than usual but not affecting daily routine, SS-FC III: Significantly high stress affecting daily routine and SS-FC IV: Worst stress in life, occurrence of major, unexpected or life-changing events.

SS-FC I and II were considered as 'low' stress population while the higher grades were considered as 'high' stress groups. This classification of patients into different stress groups was done by direct patient interview conducted by two investigators independently within 24 hours of CCU admission. This is a novel classification of subjective stress along the familiar lines of NYHA functional classification.

In addition, patients with coronary artery disease (CAD) who presented to the cardiology outpatient department during the study period for routine follow-up were enrolled as 'controls' after obtaining informed consent.

Table: 1

Table 1: Study groups according to level of subjective stress

Stress Level	SS-FC	Acute MI patients (n= 150)	Controls (n= 150)
Low stress	I	36 (24.0)	55 (36.67)
SS-FC I and II	II	34 (22.67)	65 (43.33)
High Stress	III	57 (38.0)	22 (14.67)
SS-FC III and IV	IV	23 (15.33)	8 (5.33)

Figures in parentheses indicate percentages

Table: 2

Table 2: Subjective stress in study groups I and II

	Acute MI patients (n=150)	Controls (n= 150)	P value
Low stress SS-FC I and II	70 (46.67)	120 (80.0)	<0.001
High Stress SS-FC III and IV	80 (53.33)	30 (20.0)	

SS-FC = Subjective stress functional classification
Figures in parentheses indicate percentages

Table: 3

Table 3: Cardiovascular risk factor profile of the study groups

Risk factors	Study (acute MI) cases				Controls
	Total Cases (n=150)	Low stress SS-FC I, II (n=71)	High stress SS-FC III, IV (n=79)	P Value High Vs low stress	(n=150)
Age in mean years	53.5± 11.5	54.5±10.4	52.5±12.3	0.29*	39.0 ±16.1
Sex (% male)	95 (63.33)	49	46	0.23	94 (62.67)
Smoking (%)	64 (42.67)	34	30	0.29	27 (18.0)
Hypertension (%)	52 (34.67)	24	28	0.97	24 (16.0)
Diabetes mellitus (%)	44 (29.33)	18	26	0.4	20 (13.33)
Cholesterol (%)	25 (16.67)	8	17	0.14	15 (10.0)
Prior CAD (%)	24 (16.0)	15	9	0.16	3 (2.0)

SS-FC = Subjective stress-functional classification; *t= 1.07, d.f.= 148. Figures in parentheses indicate percentages

Figure

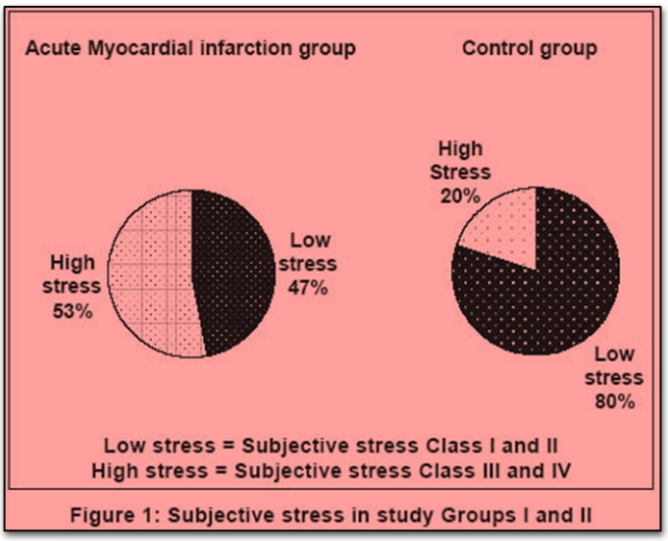

Figure 1: Subjective stress in study Groups I and II

Discussion

Patients who developed depression for the very first time after their MI ("incident post-MI depression") showed the highest risk of a new cardiovascular event. Strikingly, this risk was significantly lower and similar in patients who were depressed before the onset of MI ("non-incident post-MI depression") and in patients without post-MI depression. These findings held when controlled for MI severity, personality factors, and socioeconomic status. Strength of their protocol is that they performed a structured clinical interview to formally diagnose depression. This sound methodology supports another recent study showing that depressive symptom level at the time of, but not before, hospitalization predicted all-cause mortality at 5-year follow-up, even after adjustment for other prognostic indicators (5). Most remarkably, these findings suggest that depression onset before MI may not be causally linked with MI, supporting the most recent notion that this link is partially due to a shared genetic vulnerability (6).

It is of utmost clinical importance to find treatment options for incident post-MI depression. In order to decrease cardiac risk, such treatments must also favorably affect alterations in cardiovascular biology accompanying depression. The ENRICHD trial suggests this treatment may not be standard cognitive behavioral therapy used to treat depression in the general population (3). Also, in the SADHART (Sertraline Antidepressant Heart Attack Trial), sertraline did not reduce depressed mood more than placebo in patients with onset of a major depressive episode after they entered the hospital because of an acute coronary syndrome (7). De Jonge et al. (4) encourage fellow researchers to adopt a more individually tailored approach to treatment. They remind us that current standard diagnostic manuals of psychiatric disorders diagnose depression based on the sum of depressive symptoms adding up to a syndrome of diagnosis of depression. Diagnostic manuals exclude concerns about the etiology of depression to arrive at a descriptive type of depressive disorder that can be reliably diagnosed by different investigators across different populations and times. However, if we view post-MI depression as failure to adjust to the traumatic experience of heart attack (7), its origin is of course qualitatively different from depression developing after the loss of a loved one before MI. In the first case, a tailored psychological intervention would focus on coping with the stress of a life-threatening experience and, in the second, on supporting the mourning process.

De Jonge et al. (4) seem to have unraveled incident post-MI depression as one cardiotoxic subtype of depression. What we have not yet resolved and what ought to guide treatment recommendations in the future are the presumably numerous contributing factors to incident post-MI depression. As suggested by the authors, these are best assumed to be both biological and psychosocial in nature. We specifically feel that considering the effect of the subjective experience of an acute MI on onset of depression after MI could teach us another important lesson. Patients who perceived their heart attack as a trauma involving threatened death to which they responded with intense fear or helplessness show substantial levels of post-traumatic stress symptoms. More precisely, approximately 15% of post-MI patients suffer from clinical post-traumatic stress disorder (PTSD) related to their MI (8). The diagnosis of acute PTSD requires that for more than 1 month, patients re--experienced the MI spontaneously in thoughts or dreams, avoided cues related to the MI such as taking cardiac medication, and affirmed symptoms of hyper--arousal such as sleeplessness and irritability (9). Post-traumatic stress disorder predicted cardiovascular readmission rate in post-MI patients (10). Symptoms of PTSD substantially overlap with symptoms of depression (9). However, sole treatment of depression will only partially address PTSD-related symptoms and thus might not benefit cardiovascular health in a substantial proportion of depressed post-MI patients.

Regarding accumulated anecdotal evidence from patients who became clinically depressed after they received beta-adrenergic receptor blocking drugs, one factor that could evoke post-MI depression is beta-blocker therapy. In a prospective, carefully conducted case-control study also presented in this issue of the Journal, van Melle et al. (11) investigated whether beta-blocker use during hospitalization for MI predicts the development of depressive symptoms and disorders during the first year post-MI. Their assessment of the beta-blocker regimen before MI and during follow-up is strength of their study. They did not, however, control for previous episodes of depression, which could have prompted beta-blocker initiation before MI in patients who somatize depressive affect (e.g., in the form of palpitations and trembling) (12). In addition, their analysis considered some patients who were also part of the sample studied by de Jonge et al. (4), but we are not told whether beta-blocker use specifically affected symptom levels of incident post-MI depression at different times of follow-up.

The authors found no significant difference in any depression end point between beta-blocker users and non-users well-matched in terms of demographic factors, baseline depressive symptom level, and MI severity (11). Adjustment for potential confounders, including risk factors for cardiac disease and diseases contraindicative of beta-blocker use, maintained this observation.

Although supporting their findings, they did not discuss a previous meta-analysis of randomized trials of beta-blocker therapy in cardiovascular disease—including MI—accumulating data from over 10,000 patients followed up for at least 6 months (13). This meta-analysis showed that overall frequency of depressive symptoms was the same in the beta-blocker and placebo groups and that depressive symptom frequency did not relate differently to either lipophilic or hydrophilic beta-blocking compounds (13).

Beta-blocker therapy belongs to the current guidelines for the management of patients with acute MI and reduces mortality of post-MI patients by approximately 20% (13). In spite of this evidence, beta-blockers are underused in secondary prevention of MI even though post-MI patients with diseases contraindicative for these drugs have clear cardiovascular benefits from carefully monitored beta-blockade (14). Correctly, van Melle et al. (11) state that no population-based study is able to discount individual susceptibility to a depressogenic effect of beta-blockers. Specifically, their study cannot rule out the possibility that post-MI patients taking high-dose beta-blockade might become depressed with a delay of at least half a year after their MI, thereby calling for close monitoring of depressed mood in this particular group of patients. Nevertheless, we agree that the most important lesson to be learned from their study is to abandon the general reluctance in prescribing beta-blockers to post-MI patients who are depressed and do not have any absolute contraindication for this medication.

Conclusion

We conclude with a comment aimed at integrating important aspects of the 2 studies discussed here (4,11). Early in the course of an acute psychological trauma, therapy with propranolol recently prevented storing of traumatic memories in the brain by blocking neurotransmission (15). We feel tempted to encourage an extension of this mesmerizing avenue of research to MI populations. We hypothesize that beta-blocker therapy might similarly benefit patients who experience their heart attack as particularly traumatic, thereby perhaps decreasing onset of incident post- MI about 1 week after discharge many AMI patients experience symptoms of anxiety. However, 3–18 months after the event, AMI patients were not more anxious or depressed than the Norwegian general reference population. Psychological morbidity in AMI patients, as indicated by symptoms of anxiety and depression, seems to have declined compared with levels reported a decade ago. However, patients showing symptoms of anxiety and depression after discharge following an AMI are at risk for experiencing a persistence of the same symptoms. Assessment and treatment of anxiety and depression, and encouraging lifestyle changes after AMI, continue to be important in post-AMI care that maximizes the outcomes for AMI patients. Selective serotonin reuptake inhibitor antidepressants are the recommended pharmacologic treatment because of the relative absence of effects on the cardiovascular system. The combination of a selective serotonin reuptake inhibitor with cognitive-behavior therapy is often the most effective treatment for depression in patients with cardiovascular disease.

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