

*Depression  
and  
Cardiac Disorders*

*By*

***Dr. (Prof). P. C. Shastri***

- HON PSYCHIATRIST - B.Y.L.NAIR HOSPITAL (RTD.)
- PROFESSOR OF PSYCHIATRY - T. N. MEDICAL COLLEGE (RTD.)

PRESIDENT -

- INDIAN PSYCHIATRIC SOCIETY (WEST ZONE) 1995-96.
- INTEGRATED SENSATIONS INDIA (ISI) 1996-99
- THE RESEARCH SOCIETY FOR THE CARE, TREATMENT AND TRAINING OF CHILDREN IN NEED OF SPECIAL CARE –1999-2001

CHAIRMAN -

- SMT. MOTIBAI THACKERSEY INSTITUTE FOR RESEARCH IN THE FIELD OF MENTAL RETARDATION , 1975 - 2001.
- CHILD PSYCHIATRY SECTION - INDIAN PSYCHIATRIC SOCIETY, 1995-2001

The interplay among personality, life events, and illness may generate a variety of psychopathological changes that may be manifest in the cardiac patient or as cardiac symptoms in the otherwise healthy patient. Sick role issues, for example, may complicate a return to normal functioning for the recovered patient whose dependency needs, family dynamics, or financial interests, are served by protracted disability. Patients with cardiac illness can present with anniversary reaction, experiencing symptoms similar to those experienced by a deceased, same sex patient or other important persons on the occasion of an annual celebration or remembrance. The heart's symbolic significance also renders it vulnerable as a site for symptoms that are Psycho-dynamically derived.

Whereas a 4% to 5% current prevalence rate of MDD exists in community sample, Symptoms of depression are found in 12% to 36% of patients with general medical conditions. The rate of depression may be higher in patients with specific medical condition. MDD is identified as independent condition and calls for specific treatment when it occurs in the presence of general medical condition.

The Depression guideline panel included four possible relationships between depression and general medical condition.

1. Depression is biologically caused by the general medical condition.
2. An individual who carries a genetic vulnerability to MDD manifests the onset of depression, triggered by the general medical condition .
3. Depression is psychologically caused by the general medical condition.
4. No causal relationship exists between the general medical condition and the mood disorder.

The first two cases warrant initial treatment directed at the general medical disorder. Treatment is advocated for persistent depression on stabilization of the general medical condition : When the general medical condition causes depression, specific treatment for the former condition is optimized; Psychiatric management, education and anti-depression medication are administered to treat the depression. Cases in which the two conditions are not etiologically related, appropriate treatment is indicated for each disorder.

Depression is significantly associated with increased morbidity and mortality in myocardial infarction patients as well as in patients having coronary artery disease without myocardial infarction. The full assessment and treatment of MDD in patients, with coronary artery disease are indicated. Prevalence estimate of MDD myocardial infarction patients range from 40 to 65%. In 15 months period, patients 55 yrs or older who had mood disorder evidenced mortality rate four times higher than expected, and coronary heart disease or stroke accounted for 63% of deaths. Depression may promote poor adherence to cardiac rehabilitation and worse outcome. During the first year of recovery, more social problems have been observed in infarction patients with moderate to severe depression than in non-depressed infarction patients.

Major depression is a common comorbidity associated with ischemic heart disease (IHD). There is growing evidence that Psychological stress in general and depression in particular predispose to cardiovascular diseases. Persons who have mental stress during daily life are at twice the risk of myocardial infarction and patients with post myocardial infarction depression have higher mortality rates than non-depressed control. These data suggest a psychophysiologic mechanism underlying of depressed patient, to IHD. Clinical studies have demonstrated that depression is associated with a much higher risk of both Cardiovascular morbidity and mortality, which could be caused by platelets

activation. Physicians should maintain a heightened level of clinical suspicion for depression and depressive disorder in persons with IHD, particularly those individuals who are recovering from an acute ischemic event, such as myocardial infarction. Furthermore, depression may complicate the recovery of IHD, but in most cases depression can be effectively treated with anti depressant agents. (Am. Heart .J. 2000: 140: 563-9: )

The association between depression and IHD has been studied in epidemiological and observational studies, elevated total and cardiovascular mortality rates were found among patients treated for major depression. The exact mechanism underlying this relation and benefits of depression treatment on cardiovascular morbidity and mortality have not been established whether depression increases the risk of cardiovascular disease or cardiovascular disease itself increase the incidence of depression is not clear. However, numerous studies have demonstrated that depression adversely affects the cause and prognosis of cardiovascular disease.

Depression in the presence of other medical illness is associated with additive effects on functioning. Social dysfunction is twice as high in persons with advanced coronary artery disease and depression as in person with either condition alone. Untreated depression can complicate recovery from cardiac events.

Depression symptoms after acute MI have been reported in 60% of patients. The prevalence of major depression in patients after MI has been estimated to be as high as 20%. Even in patients who have not had an MI but have angiographically proven CAD, the prevalence of depression is 18%.

Depression has been identified as trigger for both nonfatal and fatal MI. Depression also has 50% greater risk of fatal IHD.

Frasure Smith et al have reported – 4 fold increase in Risk of fatal IHD.

Major depression is the greatest predictor of cardiac complications, accounting for more than double risk of developing an adverse cardiac complication.

There is a considerable and compelling body of evidence demonstrating that depression and co-morbid IHD lead to an increased risk of death, regardless of which illness occurred first. The underlying pathophysiologic characteristics and the effects of antidepressant treatment on mortality rate are subjects of ongoing clinical investigation.

### **Potential mechanism explaining increase in mortality rate in depressed patients after MI :**

A1 Decreased Vagal and increased Sympathetic tone in depressed person.

B1 More susceptible to cardiac arrhythmias.

C1 Reduce heart rate variability.

D1 Emotional distress.

E1 Abnormality in platelet reactivity.

## **Treatment of Depression A MUST As :**

- Depressed patients after MI take longer time to return to work and have greater stress and psychosocial impairment.
- Depressed patients with MI are more likely to drop out of exercise programme than non depressed patients.
- Depressed smokers are 40% less likely to stop smoking than patients who non depressed smokers
- Depressed patients with CAD, are less likely to comply with low dose aspirin than non-depressed patient.

Depression has been shown to worsen the prognosis of the persons with IHD and may be viewed as a risk factor similar to hypercholesterolemia

As with cholesterol, the evidence for effect on risk appears to be proportional. The effect of treatment is defined for cholesterol (lowering low-density lipoproteins decrease the risk of MI). However, we do not know whether lowering the rates of depression actually results in improved survival or diminished cardiovascular risk. The impact of non-pharmacological and pharmacological treatment on risk is currently under investigation. Clinicians eagerly await the result for the ENRICH study, which should help to ascertain whether psychosocial intervention for the treatment of depression is beneficial. Pharmacologic therapy for the treatment of depression is valuable, but the effect on outcome is as yet unknown.

## **TWO WAY CONNECTIONS**

Cardiovascular disease frequently coexists with psychiatric disorders. We have become increasingly aware that the co-morbidity of psychiatric and cardiovascular disease is attributable not only to the co-occurrence of two

independent illness, but also to the development of cardiac disease as a complication of emotional or psychiatric problems and, conversely, the development of psychiatric disorders as complications of cardiovascular disease. The relationships between heart disease and mental disorder, stress, and emotional distress exemplify the complexity of psychosomatic medicine, comprising both somatopsychic and psychosomatic effects.

## **Depression**

Several lines of investigation have pointed to the importance of depression as risk factor for the development or progression coronary artery disease. Epidemiological studies, such as the Stinson Country study, showed a higher than expected cardiovascular disease mortality in depressed individual in the community. The excess cardiovascular mortality was sufficient to cause an increase in total mortality. No other cause of death, except suicide and accidents, was increased in depressed subjects compared with non depressed subjects. Avery and Winokur identified causes of mortality in patients hospitalized for treatment of depression. They found higher mortality in patients who were judged to have received inadequate treatment or to have been treatment non-responders. The main cause of increased mortality in this group was cardiovascular disease. Booth-Kewley and Friedman, in a meta-analysis of psychological factors in coronary disease, conclude that the relative risk of incidence of coronary artery disease associated with depression is approximately 2:1.

In the Cardiac Arrhythmia Pilot Study, a test of antiarrhythmic drug therapy that included bio-behavioral measures in its baseline data set, depression was an independent predictor of post-mortality. Similarly, Ladwig et al found an increase in 6-month mortality and decreased return to work in MI patients who were depressed in the first month after MI. Five hundred sixty male survivors of acute MI were studied at 2 to 3 weeks postinfarct. Depression was not significantly

related to infarct size, history of angina pectoris, occurrence of late potential, or age, but was related to the presence of dyspnea and recurrent MI. At 6-month follow up there were 12 cardiac deaths and 17 arrhythmic events. Post MI depression was associated with increased risk for these events in univariate analysis. In a multivariate logistic regression examining other variables with univariate association with the outcomes, including recurrent MI, late potential, dyspnea, arrhythmia on Holter monitoring, and age, major depressive disorder was of marginal significance ( $P < 0.07$ ), but severity of depression was significantly associated with risk of death ( $P < 0.001$ ).

Frasure –Smith et al in a landmark study demonstrated that even after controlling for other prognostic risk factors major depressive disorder after MI substantially increased the risk mortality after 6 to 18 months. They studied 222 postinfarction patients at the Montreal Heart Institute. Patients were interviewed approximately 10 days after their infarction to establish the presence of Diagnostic and Statistical Manual III-R major depressive disorder according to modified criteria. (The usual criteria of one week's duration of symptoms and functional impairment were eliminated.) Patients also completed the Beck Depression Inventory and other rating scales. In the 35 depressed patients, the mortality rate was 15% at 6 months compared with 3% in the non-depressed patients. After adjusting for other risk factors, such as previous infarction, signs of left ventricular failure, and age, they estimated the relative risk of death associated with depression as almost 4:1. At 18 months follow up, the presence during the first 10 days after MI of elevated depression symptoms, defined as a Beck Depression Inventory score of 10 or more, was associated with a nearly eight fold increase in mortality. Strikingly, the combination of depression and frequent premature ventricular contractions on a 24-hour electrocardiogram recording obtained soon after the index MI was the most powerful predictor of death. Neither depression alone nor premature ventricular contractions alone was associated with increased mortality, whereas mortality in patients with both depression and premature ventricular contractions was nearly 80%.

Intervention trials are currently underway to determine whether Pharmacologic or psychosocial interventions can affect the course of depression and subsequent cardiovascular mortality in post-MI patients. Although no results are yet available from these trials, their potential impact on standard assessment and care for MI patients is clearly enormous.

## **Conclusion**

Depression is a common problem in patients with heart disease. Several studies of patients with acute MI have reported a prevalence rate of major depressive illness of approximately 20% in the post-MI period.

Depression is associated with reduced return to work and quality of life, as well as increase mortality, as described previously. Appropriate treatment for depression in heart disease patients is therefore important in light of its high prevalence and negative effects.