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Journal of Psychosomatic Research 67 (2009) 285–287

Journal of Psychosomatic Research

Broken heart: Broken mind

The increased frequency of sudden cardiac death after earthquakes and bombings has long proposed that emotional distress plays a role in acute coronary syndromes and arrhythmias. Since the 1960s, several controlled, prospective, epidemiological studies have reported emotional distress as a major risk factor for the onset and worsening of coronary heart disease (CHD) [1]. The Interheart study, a case control trial of around 29,000 participants in 52 countries, has found that psychosocial risk factors, including stress, depression, and low generalized locus of control, are responsible for 32.5% of the population attributable risk for myocardial infarction (MI). This is independent of, and only slightly less than, the population attributable risk for lifetime smoking (35.7%) and greater than that for hypertension (17.9%) or obesity (20.0%) [2]. Also, depression has repeatedly been found in many studies to predict early-onset CHD; increased post-MI mortality (1.5–5.07 times risk); and increased cardiac symptoms such as chest pain and fatigue [3].

Based on the abovementioned data which provides compelling evidence that depression is associated with a worse outcome in CHD population, there is a need to examine mediators that explain this association. A number of behavioral and biological factors were implicated including lack of exercise, diabetic dyscontrol, smoking, medication noncompliance, HPA axis dysregulation, platelet aggregation, vascular inflammation, endothelial dysfunction, decreased heart rate variability, autonomic instability, and hypertension. One of the clinical mediators is severity and the treatment-resistant quality of depression. Recently, a follow-up analysis of the SADHART trial found patients who failed to recover from depression after a CHD event or scored greater than 18 on the Hamilton Depression Rating (HAM-D) scale in the first 2 weeks post MI to have doubled the mortality rate compared to those who return to psychological health even 7 years after the initial cardiac event [4].

Furthermore, researchers studied different depressive symptoms and psychosocial constructs as a predictive factor of hypertension and CHD. Chida and Steptoe [5], in their major meta-analysis of 80,000 individuals, demonstrated that anger and hostility were associated with a 19% increase in CHD events in healthy individuals especially men and a 24% increase in risk among those with pre-existing CHD. Interestingly, in a sample of women with suspected myocardial ischemia, somatic but not cognitive/affective depressive symptoms were associated with an increased risk of cardiovascular-related mortality and events [6]. In a prospective study of 616 population-based sample of initially normotensive, middle-aged men from eastern Finland, it was revealed that men reporting high levels of hopelessness at baseline were three times more likely to become hypertensive in the intervening 4 years than men who were not hopeless [7]. In this issue of the *Journal of Psychosomatic Research*, Stern [8] tried to explore whether individual depressive symptoms might predict the incidence of hypertension in a cohort of 240 initially normotensive Mexican-American and European-American elders. The authors found that only helplessness significantly predicted incident hypertension, reassessed a mean of 7.0 years later, independent of other demographic and social factors [8].

The prevalence rates of depression in CHF samples range from 24% to 42% and are independently associated with a poor prognosis [9]. Furthermore, a sample of 14,000 patients, mostly with diagnosis of CHD and no previous diagnosis of depression or heart failure (HF), were prospectively followed for roughly 6 years. Post-coronary artery disease depression in this sample was linked to four times higher HF risk, regardless of whether the depression is treated with antidepressants [10]. Although HF severity and disability may impact the onset and maintenance of depressive symptoms, they do not fully account for the symptoms, suggesting that nondisease psychosocial factors may contribute to the high incidence of depressive symptoms. Individuals who use problem-solving and social support-seeking coping strategies have fewer depressive symptoms, whereas individuals who use more escape-avoidance coping (e.g., wishful thinking) have more depressive symptoms [11]. In this issue, Trivedi et al. [12] did a cross-sectional study on 222 stable HF patients and found BDI $\geq$10 to be associated with greater likelihood of behavioral disengagement, denial, mental disengagement, venting and pessimism, and lower perceived social support. Their results raise the possibility that interventions designed to improve coping may reduce depressive symptoms [12].

0022-3999/09/$ – see front matter.
doi:10.1016/j.jpsychores.2009.07.012

Unfortunately, depression in the cardiac population is underdiagnosed and undertreated. Currently, the American Academy of Family Medicine recommends that patients having an MI should be screened for depression using a standardized depression symptom checklist at regular intervals during the post-MI period, including during hospitalization [13]. In the literature, different screening instruments have been evaluated in depressed cardiac population with various results. Patient's Health Questionnaire, Hospital Anxiety and Depression Scale, and The Ketterer Stress Symptom Frequency Checklist showed the best results in cardiac depressed patients [14]. Denial is a major barrier against detection of emotional suffering especially in a type A personality male cardiac population. For that reason, independent spousal/friend ratings are a powerful tool to overcome this denial. In one study, all three scales of the self-report version of the Ketterer Stress Symptom Frequency Checklist–Revised (KSSFCR) "AIAI" (aggravation, irritation, anger, and impatience), depression, and anxiety were associated with both a positive family history and early age at initial diagnosis (AAID) of CHD [15]. In the same study, a series of regression models were used to demonstrate that the KSSFCR scales may plausibly account for 22–32% of the variance in the relationship between a positive family history and AAID. Because of previously documented denial in males, the analyses were repeated in a subgroup of males for whom spouse/friend KSSFCRs were obtained. Spouse/friend-reported AIAI was related to both early family history and AAID, and could have accounted for 68% of the common variance.

Several studies indicated that the Beck Depression Inventory (BDI) does not perform uniformly in different clinical and nonclinical populations. In this issue of the journal, Forkmann et al. [16] assessed 126 cardiac inpatients using both BDIt (total scores) and BDIc/e (cognitive and emotional and excluding somatic depressive symptoms). They concluded that the BDI cannot be recommended as a formal screening instrument in cardiac inpatients since cut-off scores were not of sufficiently high sensitivity and specificity. However, the shorter BDIc/e could be used as an alternative to BDIt, which may be confounded in physically ill patients [16].

CHD and cerebrovascular accident share many epidemiological, pathophysiological, as well as psychosocial correlates. Anxiety and depression are common after a stroke. Prevalence of anxiety ranged between 22% and 25%, and depression between 24% and 30% [17]. The progression of recovery following a stroke can be altered by treating depression, which has been shown to improve recovery in activities of daily living and cognitive impairment and to decrease mortality [18]. However, post-stroke depression and anxiety are often not adequately detected or treated. Therefore, there is a need for valid and reliable screening instruments in order to identify vulnerable patients. Beck Depression Inventory, Hospital Anxiety and Depression Scale, and SCL-90, as self-report questionnaires, and the Montgomery and Åsberg Depression Rating Scale (MADRS) and Hamilton Depression Rating Scale, as clinician-rated scales, are all accepted methods for the assessment of depression and anxiety in stroke patients [19]. However, studies fail to agree which screening instrument is superior.

Both HADS and MADRS focus more on the psychological symptoms of depression, which partially minimize the confounding impact of symptoms of physical illness being ill-judged as suggestive of depression. Although there is no agreement on the diagnostic cut-off score for both HADS and MADRS, generally, most studies in stroke patients reveal that lower diagnostic cut-offs would be more suitable in this population. In this issue, Sagen et al. [20] assessed 104 stroke patients, consecutively admitted to a stroke unit, with HADS and MADRS 4 months after stroke. Anxiety occurred in 23% of patients, and depression (including major depression, minor depression, and dysthymia) occurred in 19%. For anxiety, the optimal screening cut-off was 4 for HADS-A and 6 for HADS-total; for depression, optimal cut-offs were 4 for HADS-D, 11 for HADS-total, and 8 for MADRS. At cut-offs commonly used in clinical practice for depression screening (HADS-D: 8; MADRS: 12), the MADRS performed marginally better than the HADS. Overall, MADRS and HADS-D perform acceptably as screening instruments for depression, and HADS-A for anxiety after stroke. However, lower HADS cut-offs than recommended for the general population should be considered for stroke patients [20].

As our population ages and cardiovascular and cerebrovascular events increase, clinicians should be vigilant in monitoring psychiatric symptoms that occur post such events and be aware of the contribution of psychological constructs to mortality and morbidity following these events. We hope that this issue of the journal will further our understanding of such complex relationship.

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