Depression: the sleeping giant

Introduction

Although chest pain is a common presenting symptom in patients with anxiety and depression it is usually not due to ischaemic heart disease[1]. However, when psychiatric morbidity co-exists with ischaemic heart disease the interaction has serious consequences.

Depression and myocardial infarction

Depression occurs commonly after myocardial infarction, with 18% of patients suffering a major depressive syndrome and 27% a minor depressive syndrome[2]. The prevalence depends upon the diagnostic criteria used and upon the time of measurement following acute myocardial infarction. However, there is a consistently higher frequency of depressive disorder in patients with acute coronary syndromes than in the British population as a whole[3], in which depression affects approximately 6%[4]. Not only is depression more frequent in patients with acute coronary syndromes but it also carries an adverse prognosis in terms of recurrent clinical events, with a fivefold increase in mortality after myocardial infarction[5]. Studies have consistently shown that levels of depression are not, or are only weakly, correlated with the severity of ischaemic disease[6]. Depression is an independent risk factor for prognosis in all categories of patients with ischaemic heart disease. Hence, affective disorders increase the risk of cardiac death or non-fatal myocardial infarction in patients with a poor prognosis for other reasons, for example, in those with an ejection fraction below 30%[7] and in patients with significant ventricular arrhythmias[8]. The strongest association in terms of outcome is with major depression, but less severe forms of ‘negative affect’ would also appear to be important in determining prognosis[9].

Depression and stable coronary artery disease

There are similar data to indicate that depression is an equally frequent and important adverse prognostic factor in patients with stable coronary artery disease. Major depression occurs in 18% of patients with angiographic evidence of coronary artery disease with a history of prior myocardial infarction[10]. Amongst patients with stable symptoms who were found to have a greater than 75% stenosis in at least one coronary artery during diagnostic catheterization, those with moderate to severe depression had a 69% increased risk of death from cardiac causes and a 78% increased risk of death from any cause[11]. An intermediate increase in risk would appear to be present in patients with more mild forms of depression and with depressive personality types[12]. The adverse prognosis with depression in the presence of coronary artery disease is not only severe but is also prolonged, with depressed patients having a 72% increased risk of cardiac death compared to the non-depressed after ten years[11].

The link between physical and psychological disease

There are both behavioural and pathophysiological reasons as to why depression may adversely affect prognosis in coronary artery disease. Firstly, depressed patients are less compliant with treatment regimes, are more likely to leave cardiac rehabilitation programmes prior to completion[13] and are less likely to alter adverse risk factor profiles, such as stopping smoking[14]. Secondly, depression may in itself interfere with the doctor–patient relationship, so that physicians are less inclined to aggressive intervention in patients who appear either apathetic or who have multiple complaints[15]. Thirdly, depression is more common in patients who are socially isolated, have little social support and who have few economic resources, all factors which are also known to adversely affect prognosis following myocardial infarction[16]. The main theory relating to a physiological link between coronary artery disease and depression centres upon the potential of the autonomic system to increase the arrhythmic potential of ischaemic myocardium. Healthy depressed patients have an increased resting heart rate compared to controls and depressed patients with coronary artery disease have reduced heart rate variability compared to non-depressed cardiac patients[17]. Increases in resting heart rate and reduction in heart rate variability are both predictors of poor outcome in coronary artery disease[18]. Thus, depression may alter sympathovagal balance and increase the risk of fatal arrhythmia due to decreased vagal tone. However, an actual increase in arrhythmic death has not yet been demonstrated in depressed cardiac patients. A second
Mortality is reduced by between 20–25% following myocardial infarction. The combination of exercise and psychological support leads to a direct reduction in the level of depression and improvements in emotional well-being following myocardial infarction. Exercise alone reduces the risk of developing depression and probably improves established depression. Mortality is reduced by between 20–25% following rehabilitation programmes which provide the patient with both exercise and psychological support. However, there remains some doubt as to the efficacy of psychosocial and cognitive programmes alone to alter personality and behaviour in the absence of exercise, since the improvement in prognosis with these approaches appears to be small. It is difficult to find cost-effective and rapid treatment, acceptable to most patients, which is capable of altering chronic personality features and pervasive emotional tones such as depression.

Can pharmacological treatment of depression improve prognosis beyond the existing benefits of cardiac rehabilitation? At present, depression is rarely treated in patients with coronary artery disease. In part, this is due to the failure of physicians to identify patients at risk, with only one in four coronary patients with major depression being diagnosed but, in addition, there has been concern that established anti-depressants may induce arrhythmias, including bradycardia and supraventricular tachycardias, although the incidence would appear to be less than 0.0003%.[33] The second concern lies in the potential for drug interaction due to the metabolism of SSRIs through the cytochrome p450 system in the liver, the inhibition of the IID6 enzyme. Awareness of the importance of this problem has been heightened by the withdrawal of mibefradil (Posicor) from the market due to harmful interactions, yet little is known about the clinical impact of p450 inhibition with the use of SSRIs in cardiac patients. There is a need to establish the prognostic benefit of reducing depression with pharmacotherapy in patients following myocardial infarction before a recommendation on the use of SSRIs can be made.

Conclusion
Depression is an independent and clinically important adverse risk factor for patients with coronary artery disease. At present, physicians are poor at identifying patients with depression and infrequently initiate treatment. Depressed patients may be identified easily with self-report questionnaires and physician-directed screening tools, yet these are not routinely used. Cardiac rehabilitation schemes offer some chance of improvement following myocardial infarction, but patients with stable coronary artery disease are usually not involved with these. Results from intervention studies may require that renewed interest be shown in the mental health of patients with coronary artery disease.

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References


