Depression in the physically ill

(Index words: depression, antidepressants, continuing medical education, physically ill, management)

Summary

The risk of depression is increased in many medical and surgical conditions. Specific serotonin reuptake inhibitors are generally recommended as first line treatment in the physically ill. Care must be taken in prescribing antidepressants in the physically ill, because of side-effects, hepatic and renal impairment, and potential for drug interactions.

Introduction

Major depression occurs in 5-10% of primary care patients and 10-14% of medical inpatients [1]. The risk of depression is increased in coronary artery disease, cerebrovascular accidents, neurological disorders, malignancies and diabetes. In the physically ill biological symptoms of depression – anorexia, weight loss, sleep disturbance and lethargy may be caused by the illness itself making the diagnosis of depression difficult. However, the diagnostic criteria are the same as for those without physical illness. Presence of other symptoms such as loss of interest, hopelessness, reduced self-esteem, guilt and suicidal ideas help in the diagnosis.

Tricyclic antidepressants (TCA) and selective serotonin reuptake inhibitors (SSRIs) are both effective in the treatment of depression in the physically ill. However, tricyclics, because of side-effects, are contraindicated in many conditions and SSRIs are generally recommended as first line treatment in the physically ill. There are little data regarding the use of newer antidepressants such as serotonin noradrenaline reuptake inhibitors (venlafaxine and duloxetine) and bupropion. Treatment may need to commence at lower doses due to increased sensitivity to side-effects in the elderly, in those with renal and hepatic dysfunction, and where there is risk of drug interactions.

Cardiovascular disease

Depression is a risk factor for both the development and the worsening of coronary heart disease. About 15-20% of patients with acute myocardial infarction experience a major depressive disorder. Depression increases all-cause mortality and cardiac mortality in those with ischaemic heart disease (IHD). Pathophysiological mechanisms responsible for this increase include increased platelet activation and aggregation, autonomic dysfunction causing elevated heart rate, low heart rate variability, exaggerated heart rate responses to physical stressors, high variability in ventricular repolarisation, and low baroreceptor sensitivity.

Tricyclic antidepressants and SSRIs are effective in the treatment of depression in patients with IHD. However, TCAs are known to affect heart rate variability, and cause orthostatic hypotension and conduction delays, and are best avoided in patients with IHD.

The SSRIs are relatively safe and effective in the treatment of depression in patients with IHD. There is evidence that antiplatelet effects of SSRIs may reduce the risk of ischaemic events. The SSRIs interfere with serotonin accumulation in platelets and normalises elevated indices of platelet activation and aggregation in patients with depression and IHD. Unlike the tricyclic antidepressants they do not cause orthostatic hypotension or arrhythmias. Antidepressants other than TCA and SSRIs have not been studied in detail in depressed patients with cardiovascular disease and their safety is unknown. Venlafaxine may cause hypertension and regular blood pressure monitoring is recommended.

Stroke

About one-third of patients develop depression after stroke. Peak prevalence is between 6 months and 2 years after stroke. Post-stroke depression (PSD) has significant effect on rehabilitation, motor recovery, activities of daily living, social life, and mortality [2].

Diverse mechanisms may be involved in the aetiology of PSD. One hypothesis postulates a deficiency of serotonin at central receptor sites. Cerebral lesions, aphasia, dependence in activities of daily living and social isolation too contribute to its development.

Diagnosis of PSD is made difficult by the unreliability of biological symptoms and the presence of cognitive and communicative defects caused by stroke. Use of special scales developed for use in stroke patients such as the structured assessment for depression in brain damaged individuals (SADBD) and the post-stroke depression rating scale (PSDRS) help in diagnosis. The Beck depression inventory (BDI) is useful for assessment of PSD because of its relatively low reliance on biological symptoms.

Establishment of a regular sleep pattern, a healthy diet, exercise and social support help in the treatment of depression. Both TCAs and SSRIs are effective in the treatment of PSD but SSRIs may be preferable because of the lower side-effect risk. Tricyclics cause orthostatic hypotension which can lead to falls, constipation and urinary retention which may be troublesome. TCAs are not recommended for use in patients with co-morbid IHD.