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Organic brain dysfunction in late-onset depression

Medical comorbidity is common in late-onset depression. Some studies suggest the presence of mild cognitive impairment in up to 60% of patients with late-onset depression; this constitutes a major diagnostic problem in geriatric psychiatry. In response to the study of neurological findings in late-onset depression by Baldwin *et al* (2005) we performed an abstract review of Medline publications using the search term LATE ONSET DEPRESSION to identify the possible aetiological factors behind the increased occurrence of neurological signs in late-onset depression. We identified 93 citations published between 1975 and 2005, of which 75 titles were relevant. After reading all citations we found 63 abstracts discussing different aspects of late-onset depression which we have included in the review. The main findings are outlined here briefly.

Although early-onset and late-onset depression are similar phenotypically, there is a possible difference in aetiology. Vascular comorbidity, including an increased prevalence of hypertension, is common in late-onset depression. There is much clinical and biological overlap between late-onset depression and dementia, sometimes the former being the prodrome of the latter. There are at least a dozen studies showing some structural, functional and electrophysiological links between late-onset depression and Alzheimer's disease. There were observations that late-onset depression is not a prodrome for any particular type of dementia but the majority of patients who develop dementia will acquire Alzheimer's disease or vascular dementia, as they are the most common forms. From several studies an association with genetic factors or apolipoprotein E could not be established for late-onset depression.

There are a number of structural or vascular factors identified mainly through imaging studies. Region-specific decreases in grey matter (decreased volume of frontal and temporal lobes), ventricular enlargement, sulcal widening and decreased volume of hippocampus and caudate nucleus were reported in more than one study. Deep white matter lesions and increased evidence of vascular events were also found in late-onset depression. Functional imaging studies showed an association of impairment of regional cerebral blood flow in the left anterior temporal and left anterior frontal regions associated with late-onset depression. There is evidence of more frequent electroencephalographic changes in late-onset depression compared with early-onset depression. Moreover, a few studies examining psychological factors concluded that there is less association between life events and late-onset depression than early-onset depression.

These findings stress the importance of thorough physical examination in late-onset depression, as recommended by Baldwin *et al* (2005). In the absence of clear guidelines for neuroimaging in psychiatry, a detailed physical examination is necessary for the identification of the patient group in which more expensive and invasive investigations are indicated.

Baldwin, R., Jeffries, S., Jackson, A., *et al* (2005) Neurological findings in late-onset depressive disorder: comparison of individuals with and without depression. *British Journal of Psychiatry*, **186**, 308–313.

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Recurrence of post-partum and non-post-partum psychosis

The report by Robertson *et al* (2005) on the rates of recurrence of post-partum and non-post-partum psychosis in women who have experienced a previous episode of post-partum psychosis teaches us something new about the prognosis for these women. The risk of developing a subsequent non-puerperal episode is increased in women who have a family history of mental illness and is non-significantly increased for women with a personal history of illness prior to the puerperal episode. Robertson *et al* (2005) report rates of relapse following subsequent deliveries of 57%. They did not report the effects, if any, of treatment in preventing further puerperal episodes. Prophylactic treatment was only alluded to in the discussion, where, following a listing of the side-effects associated with lithium and other mood stabilisers, it was stated that treatment should only be instituted following a 'very careful weighing up of risks and benefits'. This apparently negative emphasis may be unintentional but is unfortunate for two reasons. First, although there are few studies in this area, the rates of recurrence of post-partum psychosis vary widely and have been as high as 90% (Kendell *et al*, 1987). It is very probable that these recurrence rates vary according to whether women are actively managed with prophylactic medication. Second, clinical observations of the benefits of lithium prophylaxis in post-partum psychosis are supported by some published reports which suggest that lithium prevents recurrence in up to 90% of cases (Stewart *et al*, 1991; Cohen *et al*, 1995).

The relatively low rates of recurrence of puerperal psychosis reported by Robertson *et al* (2005) may partly result from the now common practice of treating women prophylactically with mood-stabilising medication. For perinatal psychiatrists, the risk–benefit weighting of treatment with mood stabiliser *v.* no treatment in the puerperium for women who have had a prior episode of post-partum psychosis falls down very convincingly on the side of active treatment.

Cohen, L. S., Sichel, D. A., Robertson, L. M., *et al* (1995) Postpartum prophylaxis for women with bipolar disorder. *American Journal of Psychiatry*, **152**, 1641–1645.

Kendell, R. E., Chalmers, J. C. & Platz, C. (1987) Epidemiology of puerperal psychoses. *British Journal of Psychiatry*, **150**, 662–673.

Robertson, E., Jones, I., Haque, S., *et al* (2005) Risk of puerperal and non-puerperal recurrence of illness following bipolar affective puerperal (post-partum) psychosis. *British Journal of Psychiatry*, **186**, 258–259.

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