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EDITED BY KIRIAKOS XENITIDIS

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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Stewart, D. E., Klompenhouwer, J. L., Kendell, R. E., *et al* (1991) Prophylactic lithium in puerperal psychosis. The experience of three centres. *British Journal of Psychiatry*, **158**, 393–397.

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Authors' reply: We agree with Dr O'Keane regarding the severity and potentially devastating consequences of post-partum psychosis in women with a history of bipolar disorder and assure her that any negative emphasis she detected in our brief comments regarding prophylactic treatment were indeed unintended. The brief report format did not allow us to discuss this aspect of management at length but we have taken up this issue more fully in our recent editorial (Jones & Craddock, 2005).

We would, however, defend our contention that the decision to commence mood-stabilising (or indeed any) medication in women of child-bearing years should follow a 'very careful weighing up of risks and benefits'. Any medication should be started assuming that the women may become pregnant and future pregnancy and contraception should be actively discussed at the earliest possible opportunity.

We would also argue that the evidence base for the use of prophylaxis in women with bipolar illness in the post-partum period is not as robust as would be ideal. As Dr O'Keane has outlined, the literature does support the use of lithium in this context, although the retrospective (and partially overlapping) studies differed in when lithium was commenced – important as there may be practical problems in achieving therapeutic levels quickly following delivery and the onset of puerperal psychosis is typically in the few days following delivery. In our series of 101 women with post-partum psychosis more than half had an onset on days 1–3 with over a fifth on the first post-partum day (further details available from the authors on request). With regard to other mood stabilisers, there are few data in the literature. A recently published study demonstrated no efficacy for sodium valproate (Wisner *et al*, 2004) and, despite anecdotal reports of the benefit of typical or atypical antipsychotic medication as prophylaxis, there are no data regarding their use in this context.

Finally, it is our experience that women have strong views on the acceptability of taking medication during pregnancy and while breast-feeding. This may account for the fact that out of the 54 women in our study who went on to have a further pregnancy, only six took prophylactic medication in the puerperium (lithium or haloperidol). Although only two went on to have a recurrence of puerperal psychosis, the numbers are clearly too small to draw conclusions regarding the efficacy of prophylaxis.

This is an area, therefore, in which management decisions are not straightforward but the frequency and severity of post-partum episodes in women with bipolar disorder must weigh heavily in the risk–benefit analysis. What is needed, we can all agree, is further research to provide empirical data on which clinicians, women, and their families can base these difficult decisions.

Jones, I. & Craddock, N. (2005) Bipolar disorder and childbirth: the importance of recognising risk. *British Journal of Psychiatry*, **186**, 453–454.

Wisner, K. L., Hanusa, B. H., Peindl, K. S., *et al* (2004) Prevention of postpartum episodes in women with bipolar disorder. *Biological Psychiatry*, **56**, 592–596.

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Value of measuring suicide intent

The paper by Harriss *et al* (2005) addresses the very relevant issue of measuring suicide intent in the evaluation of future suicide risk. Measuring suicide intent is more useful than measuring the lethality of the attempts (i.e. the degree of danger to life resulting from self-injurious behaviour; Beck *et al*, 1975). Assessing the intent can be particularly useful in situations where there is no correlation between the expected and actual outcome of the method used as may happen in those with a low level of literacy. Accuracy of expectations about the likelihood of dying moderates the relationship between suicide intent and medical lethality (Brown *et al*, 2004).

Identifying a cut-off to differentiate between high-intent and low-intent attempts is very difficult. Median scores on the Suicide Intent Scale (SIS) were used by Harriss *et al* (2005) to categorise high-intent and low-intent attempts. Their results showed that women with high intent repeat suicide attempts whereas men with low intent tend to do so. Since there was a gender difference in the median values, the cut-off score used for males (10) was higher than that used for females (8). By virtue of using separate cut-off scores, men were classified as having low intent even if they had similar scores on the SIS to women in the high-intent group, possibly affecting the repetition rates. Quantifying and classifying suicide intent have been approached in different ways by various researchers. Baca-Garcia *et al* (2004) studied the characteristics which influence emergency psychiatrists in decisions to hospitalise after a suicide attempt, and found that a cut-off of 11 on the SIS correctly classified 72% of participants. However the authors clearly acknowledge the advantages of using an extensive clinical checklist over an instrument such as the SIS. Although the SIS was not originally designed to predict repetition of self-harm, it may be possible to identify similar cut-off points to predict the likelihood of repetition of suicide attempts when used with other known risk factors. For any risk assessment to be clinically meaningful it should be based on a composite index which takes into account various factors, including the level of suicide intent, the severity of depression, the degree of hopelessness, the impact of life events and the lethality of the attempt.

Baca-Garcia, E., Diaz-Sastre, C., Resa, E. G., *et al* (2004) Variables associated with hospitalization decisions by emergency psychiatrists after a patient's suicide attempt. *Psychiatric Services*, **55**, 792–797.

Beck, A. T., Beck, R. & Kovacs, M. (1975) Classification of suicidal behaviors: I. Quantifying intent and medical lethality. *American Journal of Psychiatry*, **132**, 285–287.

Brown, G. K., Henriques, G. R., Sosdjan, D., *et al* (2004) Suicide intent and accurate expectations of lethality: predictors of medical lethality of suicide attempts. *Journal of Consulting and Clinical Psychology*, **72**, 1170–1174.

Harriss, L., Hawton, K. & Zahl, D. (2005) Value of measuring suicidal intent in the assessment of people attending hospital following self-poisoning or self-injury. *British Journal of Psychiatry*, **186**, 60–66.

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Free will and volition

Although I agree with Professor Henderson (2005) that we should acknowledge that

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