

in women, suggesting an oestrogen withdrawal hypothesis in these disorders (Deuchar & Brockington, 1998). Oestrogen release into the peripheral blood is influenced by secretion of the pituitary hormone gonadorelin. We describe a psychotic disorder associated with administration of a long-acting luteinising hormone-releasing hormone agonist (LALHRHA) triptorelin.

Mrs A., 32 years old with two children, had been receiving monthly treatment for 2 months with triptorelin (intramuscular depot formulation: 3 mg/4 weeks) for suspected endometriosis. A few days after the second injection, she experienced persecutory delusions, agitation, and auditory hallucinations, which resolved within two months under neuroleptic medication and were immediately followed by a major depressive episode. This episode fulfilled DSM-IV (American Psychiatric Association, 1994) criteria for schizoaffective disorder. There was no personal history of psychotic disorder (mild baby blues followed the birth of the first child) and no familial history of psychiatric disorder. One year after this episode, she became pregnant. There were no psychiatric symptoms during the pregnancy. Five days after the delivery she presented in a psychotic state clinically similar to the previous one.

Peripheral oestrogen modulates dopaminergic transmission (Fink *et al.*, 1996). The oestrogen decrease has been hypothesised as being a causative factor in puerperal and premenstrual psychoses in predisposed individuals (Deuchar & Brockington, 1988). LALHRHAs administered in a depot formulation induce a paradoxical desensitisation of pituitary gonadotrophin secretion, resulting in a reduction in secretion of luteinising hormone and follicle stimulating hormone, and consequently a reduction in oestrogen secretion within one week (Broekmans *et al.*, 1992).

As observed in puerperal and premenstrual psychoses, the decrease in oestrogen levels induced by the administration of LALHRHAs is consistent with a modification in the brain oestrogen environment, and is simultaneously associated with the occurrence of acute psychosis. The occurrence of both puerperal psychosis (suggesting an individual predisposition) and LALHRHA-associated psychosis in the same individual suggests a common aetiology. The puerperal and the LALHRHA-associated psychosis showed clinical and hormonal similarities, suggesting that a hormonal mechanism (the delivery-induced and the LALHRHA-

induced oestrogen decrease) might be aetiologically relevant in this patient.

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Manic episode due to gabapentin treatment

Sir: Short & Cooke (1995) reported on a case of hypomania associated with gabapentin treatment. In a comment on this report Hauck & Bhaumik (1995) felt that alternating psychosis might be the cause (though electroencephalogram (EEG) was lacking). We report on a 35-year-old woman with a manic episode under gabapentin treatment in whom alternating psychosis as a possible causal factor could be excluded. She suffered from complex partial and tonic-clonic seizures for at least three years. The magnetic resonance imaging scan revealed glio-neuronal hamartia both in the left hippocampus and the right amygdala. A gabapentin monotherapy (3200 mg/day) was achieved five months before admission. Seizure frequency and EEG (within normal range) remained unchanged, however, the patient suffered from labile mood around menstruation and reported a slightly elevated mood in general.

Elevated mood and increased activity with brief episodes of depression over two days led to the patient's admission to hospital. Speech was slightly accelerated. The initial score on the Bech-Rafaelson Mania scale (BRMAS; Bech *et al.*, 1978) was 23. Neurological examination and EEG remained normal. Epileptic seizures had not occurred before admission. She did not have a personal or a family history of psychiatric disorders. Psychiatric symptoms

disappeared within five days after discontinuation of gabapentin (BRMAS=5). At that time an anticonvulsant and anti-manic treatment with lorazepam (2 mg/day) and oxcarbazepine (1200 mg/day) was established.

Psychic reactions to gabapentin seem to be rare. Only two cases of hypomanic episodes have been reported (Wong *et al.*, 1997). Moreover, there is evidence that gabapentin has therapeutic effects in affective psychosis (Young *et al.*, 1997). In the case reported here alternating or postictal hypomanic psychosis could be excluded. No other factors possibly contributing to interictal psychosis were detected. Thus, we feel that treatment with gabapentin is related to the patient's manic episode, even more so since she remained without any psychic abnormalities in a two-year follow-up. This may be in line with clinical observations on mood-elevating effects of gabapentin.

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Polydactyly and functional psychosis

Sir: I wish to comment on the report by Cardno *et al.* (1998), which described five cases of co-occurrence of polydactyly and functional psychosis. I would like to report on two patients with polydactyly who also presented with mood disorders.

A 48-year-old married male had post-axial polydactyly on the right hand. There was no family history of polydactyly. His daughter had schizophrenia. At the age of 35, he developed elated mood, grandiose delusions and other features suggesting a diagnosis of mania. The episode was successfully treated with neuroleptics for six months. He developed an episode of

major depression after a symptom-free period of eight months without any medication. The patient was put on imipramine and lithium. One year later, he still takes lithium and is symptom-free.

The second patient was a 46-year-old male with post-axial polydactyly on the left hand. Out of three brothers, one also had polydactyly on the right hand and also mood disorder (bipolar, currently on lithium and carbamazepine prophylaxis). At age 36, he developed elated mood, increased psychomotor activity and grandiose delusions. The episode was treated for about seven months. He developed major depression after a symptom-free period of nine

months and was prescribed fluoxetine and lithium. After six months, he is receiving only lithium and is symptom-free.

The association of schizophrenia and subtle neurodevelopmental abnormalities (Murray, 1994) and an excess of minor physical anomalies (O'Collaghan *et al*, 1991), probably caused during foetal development, has been reported but there are still no reports of co-occurrence of polydactyly and mood disorders. In the two cases reported the daughter of one of the patients had a history of schizophrenia and the other patient's brother had a history of polydactyly and bipolar mood disorder. More detailed studies of the association

between neurodevelopmental abnormalities and functional psychosis are warranted.

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One hundred years ago

Psychiatry as a specialty

An utterance from Prof. Von Jaksch always compels respectful attention [sic]. At the German Congress for Internal Medicine held at Wiesbaden not long ago, that eminent clinician expressed the opinion that children's diseases, dermatology, *even neurology* (italics ours) should be taught, except when material is very plentiful, by the professor of clinical medicine. Only one specialty must the student go to the specialist for, namely, mental diseases. He

declared the importance of this specialty to have become so clear in recent years, and the experience required for its proper teaching to be of such a special character, while a knowledge of it is indispensable, that a special professorship in mental diseases is always needed, and, moreover, that the study of this specialty should be made obligatory in every medical school. The JOURNAL welcomes the emphatic pronouncement. It is especially grateful for the opinion that "even neurology" has a lesser claim to rank as a specialty than

psychiatry. Not that alienists have any quarrel with or even a grudge against their sister specialists. There are not wanting, however, the signs of a desire on the part of neurologists to extend the scope of their domain so as to include mental diseases, while it has always seemed to us that the greater should include the less.

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