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A 13-year-old boy, with normal birth and developmental history, and no personal or family history of neuropsychiatric disorders, developed episodic behavioural abnormalities for a period of 10 months. The episodes, lasting a few minutes, were characterized by sudden onset of alteration in consciousness and aimless running. There were also episodes of recurrent hiccoughs with abdominal discomfort and jerky movements of the hip. He was diagnosed as having epilepsy and commenced on sodium valproate (300 mg/day). An electroencephalogram (EEG) showed generalized spike-wave complexes, but his CT scan was unremarkable. After 4 months, due to poor improvement, sodium valproate was changed to carbamazepine (400 mg/day); after a further 6 months, carbamazepine was changed to oxcarbamazepine (300 mg/day) also due to a lack of efficacy. At this juncture, he was also commenced on citalopram (20 mg/day) with a suspicion that some of the abnormal behaviours could be pseudoseizures. Although his seizures gradually decreased with this regimen, he developed a new set of behavioural oddities characterized by excessive speech, reckless money spending, wandering tendency and middle insomnia. Psychiatric examination revealed hyperactivity, overabundant speech, euphoric affect and inflated self-esteem and confidence; his Young's mania rating scale score was 13 (total score = 60; generally considered cut-off of acute mania = 12). Hence, with the diagnosis of epilepsy (partial seizures) and citalopram-induced mania, he was commenced on olanzapine 5 mg/day along with ongoing oxcarbamazepine 300 mg/day; the citalopram was discontinued. At the time of follow-up examination 1 month later, he was free of manic symptoms. Because a repeat EEG revealed bifronto-central spikes and sharp waves, with predominance over left side, the dose of oxcarbamazepine was increased to 450 mg/day.

Citalopram seems to be the most likely reason for the manic episode given that the patient had no bipolar diathesis as evidenced by either personal or family history of an affective illness. Likewise, the affective episode could not be conceptualized as either postictal or interictal psychiatric morbidity because the patient's seizures were well controlled before the emergence of such symptoms and because studies document that interictal psychosis usually appears several years after the onset of epilepsy [6]. While it is unknown whether organic factors might amplify the risk of manic switch, the fact that this patient had neither bipolar disorder before citalopram therapy nor family predisposition supports previous reports of citalopram-induced manic symptoms in patients with no known bipolar diathesis [4,5]. Another intriguing issue is the onset of manic symptoms while the patient was on a mood-stabilizing antiepileptic agent, oxcarbamazepine. The findings of this report suggest the need for careful citalopram dose titration in adolescents with epilepsy.

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Serotonergic symptoms in neonates exposed to SSRIs during pregnancy

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A recently published study [1] describes clinically significant symptoms of serotonergic over-stimulation in 20 neonates whose mothers took either fluoxetine or citalopram in late pregnancy, compared to 20 matched control infants. These effects were no longer present at 2 weeks. It is crucially important to note that only neonates whose mothers took fluoxetine were affected, and citalopram-exposed infants did *not* differ from controls. The authors do not sufficiently emphasize this point, and imply that the findings apply to SSRIs in general. Fluoxetine has an extremely long half-life, and the authors seem to omit this from their interpretation of the findings.

This is essentially a pilot study and requires replication with a larger sample size. There are already papers warning about fluoxetine in late pregnancy and suggesting a 15–30% dose reduction 2 weeks before delivery in view of the reduced capacity of the neonatal liver to metabolize and clear it. This, of course, poses the risk of relapse of depression in the mother.

I believe, on the basis of this study clinicians should *not* cease prescribing SSRIs during pregnancy *nor* cease or reduce dosage prior to delivery. Rather the message from this study is to favour SSRIs having shorter half-lives for treatment of pregnant women. There is no

evidence from this or other studies that such treatment endangers the infant, and the benefits in terms of effective treatment of depression will usually outweigh any risk.

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Generic services and early psychosis

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I read with interest the paper by Yung *et al*. [1] which concluded that generic services may not provide optimum treatment for early psychosis patients and that treatment in this setting may not be cost-effective. This conclusion was based on, as the authors acknowledge, a limited comparison with a specialized service, EPPIC. By necessity this comparison was unable to adequately control for a variety of clinical and demographic variables that are likely to influence outcomes. In addition, the mean age of patients in the generic service was 27 years. EPPIC specifically targets patients in the youth age range 15–25 years and would not provide a service to a considerable number, if not the majority, of patients receiving treatment in the generic service, substantially limiting the value of this comparison. Differences in the duration of untreated psychosis (DUP) between the groups would not explain the difference in age of presentation; in contrast it is possible that the inclusion of an older treatment group in the generic service could explain the difference in DUP and possibly some of the less favourable outcome in patients treated in the generic service. In other words, the generic service may have been treating a greater number of poor prognosis patients who due to the nature of their symptoms (for example greater negative and cognitive symptoms) may have presented at an older age, with a longer DUP and with less treatment-responsive symptoms requiring a longer duration of hospital treatment. Patients of this sort in the catchment area of the specialist service may not present until too old to access the service.

As importantly for the implications of this work for service development, was the clinical context in which these patients were managed. As described in the paper, at the time of the audit, there were no specialized programs within the service for the treatment of these patients. There are numerous models in which these important patients can be managed. The paper compared an 'undeveloped' generic service to a specialized streamed early psychosis service. Another alternative is the use of specialized programs within generic services. These can take a variety of forms including secondary consultation and education programs, selected clinicians within certain teams developing a specific treatment focus or the development of specific dedicated clinical programs. A comparison of specialized early psychosis programs with early psychosis programs within generic mental health services may provide interesting results. For example, a study of a community-based program for early psychosis patients conducted in the late 1990s found a low rate of hospital treatment and a comparably low DUP [unpublished data, 1998; 2]. This program was based in a generic mental health service and involved the development of special treatment skills within a generic community mental health team. Planned prospective comparisons of various models of treatment within generic services are urgently required before conclusions can be drawn as to the clinical and health economic appropriateness of the management of early psychosis patients within these services.

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