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Citalopram-induced decreased libido

Sir: Sexual dysfunction is a frequent but under-recognised side-effect of treatment with antidepressant drugs. It causes distress, impairs quality of life and reduces compliance with treatment. We report a case of citalopram-induced decreased libido which improved on discontinuation of the medication.

A 35-year-old man was referred with major depression of moderate severity. Despite his depression his sexual function was unimpaired. He was commenced on citalopram 20 mg daily and his affective symptoms improved by the third week of treatment. However, 10 days after commencing citalopram he had a complete loss of interest in sex. He subsequently discontinued his medication at the end of the fourth week, after which his libido returned within seven days. He refused to resume citalopram or any other antidepressant medication for fear of further loss of libido. Because of the complete loss of libido, the effects on other aspects of sexual function could not be assessed, nor did we have an opportunity for re-challenge.

Narango *et al* (1987) have reported decreased libido in subjects with alcohol problems treated with citalopram, and Nyth & Gottfries (1990) reported decreased libido induced by citalopram in elderly subjects. Citalopram is the most selective of the serotonin reuptake inhibitors. The decreased libido observed in this patient might be explained by the fact that sexual motivation and performance are inversely related to synaptic serotonin concentration (Ahlenius *et al*, 1989). The need to enquire about sexual function before initiation of antidepressant drugs and at subsequent follow-up cannot be over-emphasised.

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Clozapine treatment, eosinophilia and agranulocytosis

Sir: Amital *et al* (1997) report that initial eosinophilia in a 37-year-old man beginning clozapine treatment was followed eight weeks later by agranulocytosis and suggest a possible association.

The incidence they quote of clozapine-induced eosinophilia of 0.2–1.0% is based on spontaneous adverse event reporting and is almost certainly an underestimate. Amital *et al* refer to a paper by Banov *et al* (1993) in which an overall incidence of 14.4% is reported in a group of 118 patients. Gerlach *et al* (1989) have reported a survey of 354 patients starting clozapine treatment and undergoing full blood count with differential every week for the first 18 weeks of treatment. This group represents 30% of the total clozapine-treated population in Denmark between 1985 and 1987. Forty-one per cent of patients receiving clozapine as monotherapy had eosinophilia at some point. This is in close agreement with a rate of eosinophilia of 46% reported in 65 patients during up to eight weeks' clozapine treatment in a clinical trial by Claghorn *et al* (1987) in the USA. Hummer *et al* (1994) found an even higher incidence of 62% in a group of 68 Austrian patients. It is, however, surprising that, in a survey of the first 602 French patients to receive clozapine, an incidence of only 4.3% was found (Pere *et al*, 1992), despite weekly full blood count monitoring during the first 18 weeks.

The incidence of eosinophilia in clozapine-treated patients is, however, probably considerably higher than that of agranulocytosis. Thus, even if an association could be proven, the great majority of patients in whom a transient eosinophilia has been noted will not go on to develop an agranulocytosis; and so eosinophilia is of virtually no clinical utility in predicting clozapine-induced agranulocytosis.

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Clozapine monotherapy and ketoacidosis

Sir: I wish to report an unusual side-effect of clozapine.

A 50-year old man with a 15-year history of DSM–IV schizophrenia was admitted to hospital with a view to a change in his medication regime. The patient had been treated with a range of neuroleptic medication with little effect on his delusions and increasingly hostile behaviour. Ten days before admission the patient received flupenthixol decanoate 80 mg in depot form. He was also taking chlorpromazine 150 mg every six hours, and procyclidine 5 mg every 12 hours. On admission routine blood chemistry was normal.

He was commenced on a reducing programme of his oral medication which was stopped three days after admission. Clozapine was commenced on the fourth day of admission at a dose of 25 mg daily. The dose was gradually increased over seven days to 100 mg in the morning and 200 mg in the evening.

The patient began to complain of lethargy and thirst on the tenth day after admission and shortly after developed chest pain and dyspnoea. Laboratory investigations revealed a serum blood glucose level of 23.5 mmol/L. Blood gasses revealed a pH of 7.091 and a P_{CO_2} of 8.5 mmHg. A hyperkalaemia (consistent with a diagnosis of a ketoacidosis) of 4.9 was potentially life-threatening.

Clozapine treatment was immediately suspended following confirmation with the Clozapine Patient Monitoring Service (CPMS) that at least one previous case of hyperglycaemia and ketoacidosis had been reported in the literature (Kostakoglu *et al*,