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Letter to the editor

Severe extrapyramidal symptoms with fluvoxamine despite neuroleptics withdrawal

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Extrapyramidal symptoms (EPS) have been reported with selective serotonin reuptake inhibitors (SSRI) used alone (Coulter and Pillans, 1995). When EPS occur with a concomitant neuroleptic treatment, the role of SSRI may be more controversial.

Mr C, a 45-year-old man with a diagnosis of recurrent major depression and a past history of myocardial infarction, was admitted during a recurrent episode of the melancholic type. Before his admission, he was started on fluvoxamine at the dose of 50 mg daily for 1 week in addition to his usual cardiovascular regimen, acebutolol 400 mg/day and pravastatine 1 tab/day. A physical examination did not detect EPS. Fluvoxamine was increased to 150 mg tid and associated levopromazine 100 mg/day. Ten days later, due to psychotic symptoms, amisulpiride 600 mg/day was added. The symptomatology worsened during the next 10 days to a stuporous state with rigidity, tremor and akinesia. Deglutition difficulties were marked; levopromazine and amisulpiride were then discontinued but not fluvoxamine, which was increased to 300 mg/day. Two days later, Mr C experienced rigidity, neurovegetative signs and elevated temperature (102 °F) with dysphagia. The chest X-ray showed a right basal opacity. Mr C was then transferred to the Pneumology Department with the diagnosis of inhalation pneumonia. Infectious outcome was favourable with an antibiotic regimen. Nevertheless, persistent EPS with subsequent impending food inhalation prevented us from removing the nasogastric feeding tube. Fluvoxamine was discontinued after 8 days of unchanged EPS that dramatically resolved in 48 h and totally disappeared in 5 days.

This report illustrates a life-threatening distressing condition that appeared with the association fluvoxamine–neuroleptics and persisted in spite of neuroleptic withdrawal, until fluvoxamine was discontinued. Deglutition disorders, which we consider as EPS in this case, raised the risk of inhalation pneumonia and maintained dehydration.

Fluvoxamine like other SSRI inhibits some of the hepatic Cytochrome P450 isozymes (Crewe et al, 1992). A pharmacological hypothesis about the clinical data could be that there may have been a marked increase in the plasma concentration of levopromazine and amisulpiride. Both of these drugs may have persisted in plasma and brain tissue of the patient. We should have measured the neuroleptic plasma concentrations to be sure that the dramatic reduction of clinical symptoms was not coincident to a reduction in the plasma concentration; however, since EPS remained unchanged with no decrement in symptom severity until fluvoxamine withdrawal, then a decrease in 48 h, it is consistent with direct fluvoxamine contribution to EPS in this case. This is in agreement with some published case reports and a recent review (Wils, 1992; Arya, 1994).

The potential severity of EPS justifies paying attention to patients experiencing severe major depressive disorders who receive SSRI-neuroleptic associations. In this case, neuroleptic withdrawal was not sufficient to reduce EPS. Plasma neuroleptic measures may be helpful to determine whether fluvoxamine itself or increasing and persisting concentration of neuroleptics are responsible for observed EPS.

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