

Citalopram withdrawal symptoms

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I would like to describe the appearance of withdrawal symptoms after discontinuation of the SSRI citalopram. No similar reports were discovered after a search of MEDLINE. The SSRIs paroxetine, sertraline, fluvoxamine, and fluoxetine may cause a discontinuation syndrome presenting with dizziness, paresthesia, lethargy, nausea, lowered mood, anxiety, agitation, vivid dreams, insomnia, headache, and irritability [2, 4]. Dizziness and paresthesia are usually prominent symptoms. Hallmark features include emergence of new symptoms soon after discontinuation, and generally mild and short-lived symptoms rapidly reversed by reintroduction of the SSRI [4]. This is more common with short half-life SSRIs. A similar discontinuation syndrome has also been reported with venlafaxine [1]. SSRI discontinuation syndrome may be caused by sudden decrease of synaptic serotonin [2].

A 30-year-old man with panic and major depressive disorder had been in remission for one year with citalopram 20 mg/d, valproate 600 mg/d, and alprazolam 3 mg/d. Because of the long remission citalopram discontinuation was decided. It was slowly tapered over 3 weeks (15 mg/d for 1 week, 10 mg/d for 1 week, and 5 mg/d for 1 week). The day after the last dose he noted anxiety and irritability. Furthermore, he noted brief "bursts" of dizziness, lasting for a few seconds and occurring many times a day. He had never experienced this kind of dizziness before. He denied the presence of the other symptoms reported during SSRI discontinuation. Panic and depression did not recur. After 1 week symptoms disappeared spontaneously.

Timing (appearance soon after discontinuation, disappearance in a week), and presence of typical SSRI discontinuation symptoms (dizziness, anxiety, irritability) suggest a link with citalopram and a discontinuation syndrome. Symptoms appeared despite slow tapering. Its short half-life (about 36 hours) may be predisposing. "Bursts" of dizziness of the kind noted by this patient were also reported with venlafaxine [1] and fluoxetine [3] discontinuation. Based on the absence of previous reports, citalopram may be less likely than other SSRIs to cause a discontinuation syndrome.

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ECT followed by clozapine treatment in acute situations of schizophrenia

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Clozapine has proved to be effective for treatment-resistant or intolerant (to standard neuroleptic agents) schizophrenic patients [5] but is not used as a standard in acute schizophrenia. Recently, two studies reported the use of electroconvulsive therapy (ECT) prior to treatment with clozapine [2] and the combined use of ECT and clozapine [4]. This therapeutic association improved severely ill patients and stabilised them. We report a case study with an initial ECT treatment followed by clozapine (up to 500 mg/d) treatment.

Mr G, a 22-year-old, black, West Indian man with an 8-year history of schizophrenia, was hospitalised for an acute psychotic episode. He had never received any psychotropic treatment.

While he was experiencing auditory hallucinations that he attributed to God, he jumped through a window which led to an open broken leg; an additional bone infection occurred. Clinically, at the time of admission to a surgical department, he met DSM-IV criteria for schizophrenia with catatonic features. He was aggressive toward himself and others. The patient refused any medical care due to his delusional concerns, the patient did not care for his broken infected leg and refused any surgical treatment, so at that time the only outcome seemed to be an amputation, soon or later. Three successive drug regimens were tried: first an association of haloperidol (20 mg/d) and chlorpromazine (300 mg/d), then zuclopenthixol IM, 300 mg, every 3 days, during 4 weeks; this medication was replaced by fluphenazine (up to 450 mg/day, per os) with no further improvement for another 4-week period. Side effects were hypertonemia and cogwheel. At this time the patient thus received a high dose antipsychotic for 10 weeks with three different compounds

without any effect. The patient was considered as resistant and a good indication for clozapine. However, as he refused to take tablets, ECT was prescribed.

We then carried out nine bilateral ECT treatments during a 3-week period. The subsequent improvement in aggressiveness and acceptance of treatment allowed commencement of clozapine administration. The dosage was increased, progressively, up to 500 mg/d. Delusions and interpersonal contact improved dramatically and improvement was stable over time. At that time, the patient agreed to surgical treatment and his leg could be spared whereas orthopaedic surgeons considered that amputation was necessary, before ECT and clozapine treatments.

In this particular case, ECT improved unremitting psychotic agitation and aggressiveness, but did not clear hallucinations and delusions completely. However, it permitted eventual clozapine efficacy in a "two-step" treatment with a good clinical stabilisation and more safety for the patient

and the nurses. The benefits of ECT in schizophrenia seem interesting in some chronic situations but even more in acute relapses of this chronic disease [1, 3]. The use of clozapine in treatment-resistant patients with a high risk of violent behaviour is typically a clinical situation when ECT is a safe and effective association.

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