Annals of General Psychiatry

BioMed Central

Poster presentation

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Mania associated with ziprasidone initiation

Nikolas Nikolaidis*, Vasilis Bozikas, Kostas Latoudis, Grigoris Kounenis, Kleopatra Savvidou and Kostas Fokas

Address: 2nd Department of Psychiatry, Aristotle University of, Greece
* Corresponding author

from International Society on Brain and Behaviour: 2nd International Congress on Brain and Behaviour
Thessaloniki, Greece. 17–20 November 2005

Published: 28 February 2006

Annals of General Psychiatry 2006, **5**(Suppl 1):S271 doi:10.1186/1744-859X-5-S1-S271

Case Report

Ziprasidone is effective for the acute and maintenance treatment of schizophrenia, as well as for the treatment of acute mania. It generally has a benign adverse effects profile. However, cases of mania associated with the initiation of treatment with ziprasidone have been reported. In the present study we present three cases of hypomania and mania associated with the initiation of ziprasidone administration.

Case 1

Mr A. is a 20 year-old man with schizophrenia, paranoid type, who received ziprasidone after five weeks of unsuccessful treatment with olanzapine up to 30 mg/day. After ten days, when the dose of ziprasidone had been gradually titrated up to 160 mg/day, he presented hyperthymia, irritability, psychomotor agitation, pressure of speech, grandiose delusions, decreased need for sleep, and sexual indiscretion (public masturbation). After an episode of physical assault against two other patients, Mr A. was restrained and ziprasidone was replaced with haloperidol IM and then per os. Two weeks later, the patient exhibited significant improvement of his behaviour, but persecutory and grandiose delusions, as well as auditory hallucinations, remained unchanged.

Case 2

Mr B. is a 47 year-old man with schizophrenia, paranoid type, who after a short period of treatment with haloperidol IM received ziprasidone (80 mg/day). After four days, expansive mood, delusions of grandiosity and pressure of speech were added to his psychopathology. Finally, about three weeks after ziprasidone initiation, the patient left the hospital without permission. Eight days later, he was brought back to hospital and haloperidol per os was administered, resulting in sufficient control of his symptoms 10 days later.

Case 3

Mr C. is a 31 year-old man with schizophrenia, undifferentiated type, in whom risperidone (8 mg/day) was replaced with ziprasidone (80 mg/day). Three days later, he manifested elated mood accompanied by an increase in goal-directed activity, which later progressed to severe agitation, sexual indiscretions, and unrestrained money waste. Simultaneously, the patient displayed decreased need for sleep and grandiose delusions. Two months after ziprasidone initiation, its dose was raised to 160 mg/day, but fifteen days later no improvement was observed. Finally, ziprasidone was replaced with olanzapine (15 mg/day), with which his symptoms were effectively controlled after 3 days.

There is evidence that atypical antipsychotics, speciffically risperidone, olanzapine and zipresidone, induce (hypo) manic switches. Manic symptoms caused by olanzapine and risperidone are probably related to their 5HT2/D2 receptor occupancy. Ziprasidone is not only a potent serotonin (5HT2A) and dopamine (D2) receptor antagonist, but also an antagonist of 5HT1D and 5HT1A receptors, as well as of norepinephrine and serotonin reuptake pumps. Perhaps the tricyclic-like antidepressant pharmacological profile of the drug, which is comparable to that of amitriptyline and imipramine, might additionally explain why in some patients it precipitates mania.