

Letter to the editor

Adjunctive citalopram is effective on hallucinations and depersonalization symptoms: a case report

The treatment of depressive symptoms in patients affected by schizophrenia is often a concern for clinicians [2] due to potential interaction, in terms of safety and efficacy. Citalopram seems to be a safe SSRI as adjunctive treatment to Olanzapine because of the lack of interactions. We report a serendipitous finding showing that the adjunction of Citalopram to Olanzapine, led to disappearance of residual hallucinations and depersonalization symptoms in a few weeks.

Mr A is a 27-year-old male, living in the community, with a 4-year history of schizophrenia. He was treated with low doses of Olanzapine (10 mg/daily) since 2000 because of an intolerance to dose increments (weight gain and mydriasis). The persistence of sporadic hallucinations (commenting voices) and depersonalization symptoms (when he walked alone he felt his self leaving the body) was well tolerated by the patient. The social functioning was satisfactory: he had a protected job and was involved in a comprehensive rehabilitation programme.

In the month of September he begun to experience insomnia, anxiety, weakness, depression and helplessness. A benzodiazepine was initially prescribed, but the worsening of depressed mood and the occurrence of biological symptoms such as psychomotor retardation and attention deficit, needed a prompt interruption of benzodiazepine and the start of Citalopram at 20 mg/daily. Between the fourth and sixth week of treatment depressive symptoms markedly improved, and surprisingly, the disappearance of hallucinations and depersonalization symptoms was also observed.

The onset of sexual dysfunction urged a reduction of Citalopram to 10 mg/daily without any interference on the above mentioned symptoms.

To our knowledge this is the first report suggesting that Citalopram may improve the antipsychotic efficacy of Olanzapine. Two speculative and cautious interpretations are possible.

1. A possible increase of Olanzapine serum level, due to the inhibition of Citalopram on Cytochrome P450-1A2, may be involved. The time course of psychotic symptoms disappearance is compatible with a progressive rise of Olanzapine plasma level. It still remains puzzling why none of the most common side effects of Olanzapine occurred.
2. A pharmacodynamic interaction might have occurred by modulating the neural circuitry involved in mood regulation. Citalopram may have beneficially interacted with dysfunctional areas involved in psychotic symptom generation, like the posterior inferior temporal cortex, where an increase of rCBF is reported in experiments of depressed mood provocation [1].

References

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