

Letter to the Editor

Asenapine at low doses as a treatment for psychotic anxiety

We read with special interest an original article by Ene H.M. et al. (1) published in his magazine recently in which it was suggested that asenapine may have anxiolytic properties, and thus clinical trials are recommended to examine these effects.

Asenapine is a second-generation antipsychotic characterised by a multireceptorial action profile as it has affinity for dopamine (D2, D3 and D4), serotonin $(5HT_{2A}, 5HT_{2B}, 5HT_{2C}, 5HT_{6})$ and 5HT₇) and adrenergic receptors (α a1A, α 2A, α 2B and α 2C), but with no affinity for muscarinic receptor (2).

Published scientific evidence shows that both 5HT₆ receptor antagonism and antagonism of the 5HT₇ receptor can offer benefits of cognition (3) and that 5HT₇ antagonism may be beneficial for the management of anxiety and mood (4), although more extensive studies are required (1,5) to confirm it.

We present our clinical experience with a 22-year-old, single, childless woman, the oldest of two sisters, who was administered asenapine as an anxiolytic.

The patient had no somatic personal history. Her psychiatric family history revealed two paternal aunts diagnosed with paranoid schizophrenia, one of them with mental retardation. Her mother did not have a clear diagnosis, but had symptoms classified under the neurotic spectrum. She was the product of a normal pregnancy and childbirth, and crossed development milestones normally. The patient was a poor student, and left school without completing ESO 4th.

She was diagnosed with paranoid schizophrenia at age 16, when she presented a first psychotic episode with great distress and high productivity: she suffered from delusions and injuries with significant emotional and behavioural impact. In the first months, treatment was begun with risperidone 6 mg/day with no clear clinical improvement but with extrapyramidal side effects so that a month later the treatment was changed to paliperidone 9 mg/day. Over the next 2 years, she showed gradual improvement, but the evolution of the patient was

towards social isolation, with neglect of self-care and daily routines, and a deleterious substance consumption of up to 21 of coke/day and 40 cigarettes/day. This was accompanied by subjective complaints of drowsiness, so that treatment was changed to aripiprazole 15 mg/day.

Since then, the patient has been under our treatment in the Mental Health Therapeutic Community. Here, despite the reduction in the consumption of harmful substances, episodes of intense distress were evident in addition to firstand second-phase insomnia, and therefore we added asenapine 5 mg/day to the treatment. Since then there has been significant clinical improvement in the patient, as the anxiety episodes were associated with the regularisation of the sleep-wake rhythm. The DASS 21 (Depression Anxiety Stress Scales) score for anxiety was 19 points (extremely severe anxiety) before the introduction of asenapine 5 mg/day and 5 points (mild anxiety) 2 weeks after the introduction of asenapine.

Therefore, we consider it noteworthy that even in generally recommended antipsychotic monotherapy there are certain molecules that can be useful in the treatment of other associated symptoms.

The potential anxiolytic effect of asenapine with demonstrated potential antipsychotic efficacy without risk of abuse (as occurs with benzodiazepines) makes it an option in the therapeutic management of psychotic anxiety.

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References

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