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Editorial comment

Treating residual insomnia in schizophrenia: examining the options

An editorial comment to Baandrup L, Jennum P, Lublin H, et al 'Treatment options for residual insomnia in schizophrenia' (1)

In their discussion paper, Baandrup et al. (1) describe a systematic review of randomized controlled trials of treatment options for residual insomnia in patients with schizophrenia. We offer supplementary comments to the views that they have expressed.

In our study (2), melatonin was associated with statistically and clinically significant superiority over placebo in schizophrenia patients with residual insomnia. Nevertheless, even at endpoint, the melatonin patients were still substantially sleep impaired. Additionally, it appeared that the benefits of melatonin were fewer during the second week relative to the first week of treatment. Thus, melatonin may not be a practical solution for insomnia in schizophrenia.

We believe that behavioural interventions (3) are also not a practical solution. This is because, despite antipsychotic treatment, many patients with schizophrenia are challenged by residual positive, negative and/or cognitive symptoms, all of which could interfere with the ability to practice behavioural interventions.

What remains? Only drug therapy with medications that have adequate hypnotic properties. We do not advocate conventional hypnotics such as zopiclone, zolpidem and benzodiazepine drugs because of the dependence liability, and because of the risk of a disinhibition syndrome (4). We also do not advocate other sedating drugs, such as certain antihistaminics, antidepressants and anxiolytics, chiefly because there is no evidence base for their safety and efficacy for residual insomnia in schizophrenia.

We strongly believe that the simplest and most logical solution is augmentation with an adequate dose of a sedating antipsychotic, preferably one with a short half-life (e.g. immediate-release quetiapine), which carries a low risk of next-morning carryover effects. Augmentation could increase the antipsychotic efficacy of the primary drug and is preferable to switching antipsychotics in patients who are otherwise stable because there is no assurance that a new antipsychotic will be as effective as the existing one. In this context, judicious antipsychotic polypharmacy is no longer a taboo thought (5).

In the end, all treatment is based on risk-benefit analyses. The risks associated with antipsychotic augmentation (e.g. increased cost, increased adverse effect burden, poor medication adherence) should be weighed against the potential benefits (better quality of life, less physiological stress and hence less risk of destabilization) in individualized decision-making. We agree that the problem is sufficiently common and important to warrant specific interventional research.

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