Insomnia May Blunt Response to Antidepressants

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DENVER — Chronic insomnia in depressed elderly patients confers roughly a ninefold increased risk of continued depression after as much as 12 months of antidepressant therapy, Wilfred Pigeon, Ph.D., reported at the annual meeting of the Associated Professional Sleep Societies.

"These findings suggest that chronic insomnia blunts treatment response in patients in this intervention study. This means, therefore, that chronic insomnia is not only a precipitating factor, as has been shown by a dozen or so studies, but that it now can also be considered a perpetuating factor, at least in this elderly sample.

As such, it represents a modifiable risk factor for new-onset, recurrent, and, now, ongoing depression," said Dr. Pigeon, who is assistant director of the sleep laboratory at the University of Rochester (N.Y.).

He reported on 1,221 patients aged 65 years or older with major depressive disorder—60% of them women—who participated in Project IMPACT (Improving Mood Promoting Access to Cognitive Treatment).

Participants in this primary care practice–based trial were randomized to either usual care or a stepped-care intervention involving antidepressant medication and/or counseling delivered by their primary care physician, who worked in collaboration with a Project IMPACT case manager. A total of 1,801 elderly depressed patients enrolled in the study; however, those with dysthymia only were excluded from this analysis.

Overall, 206 patients had persistent insomnia both at baseline and after 6 months, whereas 644 had transient insomnia—meaning that they had insomnia as defined by their score on the three sleep-related items on the symptom checklist (SCL) at one of these time points. The remainder did not have insomnia at either time point.

Remission of major depression was defined as a score lower than 0.5 on the 17 relevant items of the SCL, exclusive of the three items used to assess for insomnia.

With this criterion, 38% of those in the no-insomnia group had achieved remission at the 6-month mark, compared with 26% of those who had transient insomnia and just 5.8% of those who had chronic insomnia.

After controlling for patient age, gender, intervention arm, use of antidepressants at 6 months, baseline depression severity, and the number of comorbid chronic illnesses, transient insomnia was associated with a 2.0-fold increased risk of no remission at 6 months. Persistent insomnia carried a 12.1-fold increased risk, Dr. Pigeon said.

A similar pattern was observed at the 12-month mark. Of patients with chronic insomnia, 4.6% (odds ratio of 9.2) experienced remission and 17.9% had at least a 50% clinical improvement of their depression.

In a multivariate regression analysis accounting for numerous variables, insomnia severity emerged as the only significant contributor to depression severity at 6 months.

At 12 months, baseline depression severity joined severity of insomnia as a significant predictor of depression severity, although insomnia severity was a more potent contributor.

These Project IMPACT findings raise a clinically important question: Could targeted treatment for insomnia increase the likelihood or speed of response to antidepressant therapy?

"Could it even perhaps serve as prophylaxis against comorbid disorders other than depression?" Dr. Pigeon asked.

He noted that insomnia has been shown in various studies to be an independent risk factor for several disorders—including hypertension, insulin resistance, anxiety disorders, and colds and other viral infections—as well as for depression.

The studies showing insomnia to be a risk factor for either new-onset or relapsing depression go back as far as 15 years ago, with odds ratios of 2-6.

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