Reducing potential for harm

I enjoyed the review of assessing harm to self and others by Drs. Charles Scott and Phillip J. Resnick (“Assessing potential for harm: Would your patient injure himself or others?” CURRENT PSYCHIATRY, July 2009, p. 24-33). All too often mental health professionals rely on “gut instinct” and neglect evidence-based strategies when assessing for dangerousness. Generally, I believe this to be an issue of complacency rather than willful neglect or lack of training.

I would like to add a few points that I believe are key to conducting a proper assessment of suicide risk. First, schedule and document a firm follow-up appointment with the patient after evaluation. Second, I believe religious and spiritual beliefs function as a protective factor for many individuals, although this varies from person to person. And last, inquiring about the patient’s immediate future orientation (eg. “What are your plans for tomorrow?”) is crucial when conducting a comprehensive risk assessment.

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No need to soften criteria

I am concerned about the article on “soft bipolarity” and easing the diagnostic criteria for bipolar disorder (BP) II (“Soft bipolarity: How to recognize and treat bipolar II disorder,” CURRENT PSYCHIATRY, July 2009, p. 40-48).

I’ve found no issue as vexing as that of dealing with the “soft” end of the so-called “bipolar spectrum.” At that end of the spectrum, it can be very difficult to determine whether my patient’s symptoms are most properly attributed to 1 or more of several other DSM-IV conditions, most notably attention-deficit/hyperactivity disorder (ADHD), posttraumatic stress disorder (PTSD), and borderline personality disorder. Including overactivity would sweep in a multitude of patients with other diagnoses, most notably ADHD. A number of psychiatric conditions can cause at least 1 night of not sleeping. Softening the diagnostic criteria for hypomania to include only 1 night of sleeplessness would capture a number of patients who do not have BP.

In my clinical practice, I routinely encounter patients who I believe have been misdiagnosed with BP II or BP not otherwise specified by clinicians who are using “soft” criteria such as those promoted by Dr. Daniel J. Smith. These patients often have been exposed to a number of psychiatric medications that have caused adverse effects and have not lead to significant benefits. Instead of using “soft” criteria for BP, I adhere to the “hard criteria” for BP II and other conditions in the DSM-IV when making diagnoses, and I utilize evidence-based treatments for these conditions. Supporting my skepticism is the fact that patients who would meet soft BP II criteria often experience excellent responses to treatments for conditions such as PTSD or ADHD, and ultimately never require treatment for BP.

I believe there is real potential for harm to our patients in softening current criteria:

• Overdiagnosis of bipolar disorder in my experience leads to underdiagnosis and undertreatment of other psychiatric conditions.

• Diagnosis naturally leads to treatment, often with drugs that do not have good data supporting their use for BP II, as Dr. Smith states in his article.

• Medications for bipolar disorder are among the most toxic medications used in psychiatry, with serious side effects, including renal failure, weight gain, Stevens-Johnson syndrome, and hypercholesterolemia.

Exposing more patients to these treatments without clear evidence that softening the diagnostic criteria identifies those with true bipolar disorder is a frightening prospect.

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Antipsychotics and bones

Antipsychotics were not mentioned in Drs. Sarah K. Rivelli and Andrew J. Muzyk’s list of psychiatric medications that could increase the risk of osteoporosis (“Protect patients’ bones when prescribing,” Medicine in Brief, CURRENT PSYCHIATRY, June 2009, p. 23-25). Data show that hyperprolactinemia associated with antipsychotics can increase osteoporosis risk.1

Antipsychotics often are given on a long-term basis, which creates con-
cern for all patients taking these medications, especially because obtaining prolactin levels typically is not the standard of care. For medical professionals, linking hyperprolactinemia with osteoporosis may seem like common sense, as is linking hyperprolactinemia with antipsychotics, but we rarely correlate antipsychotics with osteoporosis. We should make that connection in consideration of the long-term health effects antipsychotics have on our patients.

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Reference

Drs. Rivelli and Muzyk respond

We agree with Dr. Cho about the need to be aware of deleterious effects of antipsychotics on bone density. Hyperprolactinemia from antipsychotics results from antagonism of D2 receptors on pituitary lactotroph cells. Blockade prevents dopamine stimulation, which normally inhibits prolactin release. Stimulation of serotonin-2A (5-HT2A) receptors on pituitary lactotroph cells also contributes to prolactin release. Second-generation antipsychotics (SGAs) strongly inhibit 5-HT2A receptors in the tuberoinfundibular pathway, which means these agents may have a lower risk of hyperprolactinemia compared with first-generation antipsychotics (FGAs). Osteoporosis is caused by prolonged dysregulation of the HPA axis and hypogonadism.1

Other factors—including a schizophrenia diagnosis, sedentary lifestyle, smoking, substance abuse, and malnutrition—also may contribute to osteoporosis.2 This condition may be highly prevalent and underdiagnosed in male schizophrenics.3 We would consider patients on chronic antipsychotic therapy—particularly those receiving higher doses or FGAs—at higher risk of osteoporosis.

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References