we can conclusively state that patients with bipolar disorder and schizophrenia have comparable risks for metabolic syndrome.

Finally, we agree with Bartoli et al. (1) that further research should assess the relative contribution to metabolic syndrome of not only different psychiatric diagnoses, but also other components such as genetics, medication use, and health behaviors. To allow such analyses, these highly relevant moderating and mediating variables should be assessed more uniformly in studies reporting on metabolic syndrome frequencies in patients with specific mental disorders.

References

1. Bartoli F, Carrà G, Crocamo C, Carretta D, Clerici M: Bipolar disorder, schizophrenia, and metabolic syndrome (letter). Am J Psychiatry 2013; 170:927–928
2. Vancampfort D, Vansteelandt K, Correll CU, Mitchell AJ, De Herdt A, Sienaert P, Probst M, De Hert M: Metabolic syndrome and metabolic abnormalities in bipolar disorder: a meta-analysis of prevalence rates and moderators. Am J Psychiatry 2013; 170: 265–274
3. Mitchell AJ, Vancampfort D, Sweers K, van Winkel R, Yu W, De Hert M: Prevalence of metabolic syndrome and metabolic abnormalities in schizophrenia and related disorders: a systematic review and meta-analysis. Schizophr Bull 2013; 39:306–318
4. Mitchell AJ, Vancampfort D, De Herdt A, Yu W, De Hert M: Is the prevalence of metabolic syndrome and metabolic abnormalities increased in early schizophrenia? a comparative meta-analysis of first episode, untreated and treated patients. Schizophr Bull 2013; 39:295–305

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Antepartum Depression: Treatment With Computer-Assisted Cognitive-Behavioral Therapy

TO THE EDITOR: Depression affects 7%–12% of pregnant women (1), and left untreated, it is associated with adverse outcomes including preterm birth and lower birth weight (2). Many pregnant women do not pursue treatment because of practical barriers (e.g., lack of time or child care) or fear of adversely affecting fetal health with antidepressant use (3). Computer-assisted cognitive-behavioral therapy (CBT), with demonstrated efficacy in nonpregnant depressed samples (4), uses a web-based multimedia program integrated with abbreviated CBT to reduce the scheduling burden on the patient and to circumvent the risks of antidepressant use. We report here on a patient with a major depressive episode during pregnancy who underwent computer-assisted CBT.

"Ms. B," a 29-year-old woman, gravida 1, para 0, with two previous depressive episodes, presented at 18 weeks gestation with depressive symptoms including low mood, anhedonia, hypersomnia, low energy, and guilt. A diagnosis of recurrent major depressive disorder was confirmed using the Structured Clinical Interview for DSM-IV. Ms. B had discontinued sertraline (100 mg) 8 months earlier for pregnancy planning. Computer-assisted CBT comprised eight therapy sessions over 6 weeks with a clinical psychologist (3.75 hours of direct contact), with between-session web-based modules ("Good Days Ahead"; Empower Interactive, San Francisco) (5). Therapy sessions included collaborative agenda setting, reviewing skill implementation, and previewing web-based content. Web-based modules included video vignettes, psychoeducational content, and exercises (e.g., automatic thought records). Ms. B had good adherence and experienced remission. She exhibited a 94% reduction in her Hamilton Depression Rating Scale scores from the pretreatment assessment (score, 17) to the posttreatment assessment (score, 1). Her Beck Depression Inventory scores (pretreatment score, 26; posttreatment score, 2) and Edinburgh Postnatal Depression Scale scores (pretreatment score, 17; posttreatment score, 5) also decreased markedly. Her Global Assessment of Functioning score improved from 55 to 90. Subjective report indicated that Ms. B found computer-assisted CBT acceptable, and she expressed satisfaction in avoiding antidepressant use during pregnancy.

Computer-assisted CBT represents a short-term accessible treatment for antepartum depression. Adapting technologies to patient populations facing practical barriers is critical to improving mental health care accessibility. Few randomized controlled trials have assessed CBT for antepartum depression (6), and none have assessed computer-assisted CBT in this population. This case report suggests the need for larger-scale research on the effectiveness of computer-assisted CBT for antepartum depression.

References

1. Bennett HA, Einarson A, Taddio A, Koren G, Einarson TR: Prevalence of depression during pregnancy: systematic review. Obstet Gynecol 2004; 103:698–709
2. Grote NK, Bridge JA, Gavin AR, Melville JL, Iyengar S, Katon WJ: A meta-analysis of depression during pregnancy and the risk of preterm birth, low birth weight, and intrauterine growth restriction. Arch Gen Psychiatry 2010; 67:1012–1024
3. Petersen I, Gilbert RE, Evans SJ, Man SL, Nazareth I: Pregnancy as a major determinant for discontinuation of antidepressants: an analysis of data from the Health Improvement Network. J Clin Psychiatry 2011; 72:979–985
4. Wright JH, Wright AS, Albano AM, Basco MR, Goldsmith LJ, Raffield T, Otto MW: Computer-assisted cognitive therapy for depression: maintaining efficacy while reducing therapist time. Am J Psychiatry 2005; 162:1158–1164
5. Wright JH, Wright AS, Beck AT: Good Days Ahead: The Multimedia Program for Cognitive Therapy. Louisville, Mindstreet, 2004
6. Sockol LE, Epperson CN, Barber JP: A meta-analysis of treatments for perinatal depression. Clin Psychol Rev 2011; 31: 839–849

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Clozapine-Induced Obsessive-Compulsive Symptoms in Bipolar Disorder

TO THE EDITOR: Clozapine remains the best option for the large proportion of patients with treatment-resistant schizophrenia. Other data support its use for treatment-resistant bipolar disorder. Despite these therapeutic advantages, clozapine is underutilized because of the risk of serious complications (e.g., agranulocytocis) and more common side effects (e.g., sialorrhea). It has been recently reported that 38.9% of patients receiving clozapine therapy report past-week obsessive-compulsive symptoms (1). We document two cases of a clear temporal association between obsessive-compulsive symptoms and clozapine.

"Mr. A," a 52-year-old man with a diagnosis of bipolar I disorder, had been hospitalized for mania with psychosis at age 34 and had shown partial response to various antipsychotics and mood-stabilizers. At age 38 he took clozapine, 600 mg/day, and experienced full symptom remission and a return to good psychosocial functioning. He developed disabling checking behaviors 2 years later, which were relieved with sertraline, 200 mg/day. At age 50, the patient insisted on discontinuing clozapine and sertraline. At 100 mg/day of clozapine, his obsessive-compulsive symptoms completely resolved. However, within 2 months, the patient developed a full manic episode requiring hospitalization. He was stabilized on clozapine, 300 mg/day, and discharged, but within 4 months, the checking behaviors returned and sertraline was restarted, bringing partial relief. A year later, he again insisted on reducing the clozapine dosage, and the cycle of obsessive-compulsive symptom remission, mania, response to clozapine, re-emergence of obsessive-compulsive symptoms, and improvement with sertraline was observed.

"Mr. B," a 42-year-old man with a diagnosis of bipolar I disorder, was hospitalized several times for mania with psychosis and showed partial response to pharmacotherapy. At age 28, he started clozapine and achieved symptom resolution. Within 6 months, he developed disabling checking behaviors, which responded minimally to serotonin reuptake inhibitors. When he first presented at our clinic, the patient was taking 200 mg/day of clozapine, with severe checking behaviors but without mania. Clozapine was tapered over 6 months with cross-titration of quetiapine. Obsessive-compulsive symptoms dramatically improved with 100 mg/day of clozapine, but the patient experienced mania. This eventually resolved with 1,200 mg/day of quetiapine. One year after clozapine discontinuation, the patient's obsessive-compulsive symptoms remitted and psychosocial functioning improved.

Both cases illustrate clear temporal relationships between clozapine and the development of obsessive-compulsive symptoms in patients with no history of the condition. In Mr. A, over 15 years we witnessed the late appearance of obsessive-compulsive symptoms, with complete resolution after discontinuation of clozapine and rapid recurrence of the full syndrome upon restarting the drug. This on-off-on cycle was repeated within a year. The case of Mr. B illustrates the potential severity of clozapine-induced obsessive-compulsive symptoms and the marked improvement with discontinuation. Both cases involve bipolar disorder, suggesting that obsessive-compulsive symptoms with clozapine occur independently of schizophrenia. This finding is potentially clinically relevant and, to our knowledge, previously unreported, although a lack of clozapine blood levels is a limitation of our interpretive ability. Clozapine is a choice for treatment-resistant bipolar disorder, but obsessive-compulsive symptoms can significantly complicate therapy.

Reference

1. Scheltema Beduin AA, Swets M, Machielsen M, Korver N; Genetic Risk and Outcome of Psychosis Investigators: Obsessive-compulsive symptoms in patients with schizophrenia: a naturalistic cross-sectional study comparing treatment with clozapine, olanzapine, risperidone, and no antipsychotics in 543 patients. J Clin Psychiatry 2012; 73:1395–1402

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