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Scopolamine Patch for Clozapine-Induced Sialorrhea

To the Editor: A common complication of treatment with clozapine is the development of sialorrhea. Incidence rates of hypersalivation noted in four review articles varied from zero to 80 percent, and the rate in premarket testing was 31 percent (1–4). Increased salivation may be attributable to either clozapine-induced muscarinic M4 or adrenergic agonism (1–4). Because sialorrhea is generally greater at night (3), another possible mechanism is clozapine-related alteration of the circadian rhythm.

Recommended management of sialorrhea includes dose reduction, anticholinergic agents, a clonidine patch, and ophthalmic atropine (1–5). Also, pirenzepine, a muscarinic M1 receptor antagonist, is available, and it has been occasionally used for this purpose in Europe (3,4). However, in practice most cases of sialorrhea are not adequately treated, because available treatment options have limited efficacy or unwanted side effects.

One possible alternative that, to

our knowledge, has not been previously described in the literature involves the use of a scopolamine patch. We report the case of a woman taking clozapine who had profound sialorrhea and who achieved significant relief from her symptoms with a scopolamine patch.

The patient was a 44-year-old unmarried Caucasian woman who was given a diagnosis of schizoaffective disorder at age 19 and who had been in treatment since 1997 in the department of psychiatry at the University of Connecticut Health Center. Before 1993 she had undergone multiple trials of medications, including typical and atypical antipsychotics, mood stabilizers, benzodiazepines, and clonidine, to control frequent exacerbations of symptoms, with only partial response. She continued to have residual depressive and psychotic symptoms even when her condition was stabilized. The first clozapine trial began in 1993. Her illness went into substantial remission after she began taking clozapine at a dosage of 900 mg of a day, and she avoided hospitalization for eight years.

The patient experienced sialorrhea during this time, but the condition was not socially embarrassing and did not require management. Unfortunately, clozapine treatment was complicated by the development of morbid obesity—a weight gain of 120 pounds—and hypercholesterolemia. Because of a strong family history of cardiac disease, the patient became concerned about the risk of premature cardiac-related death and decided to stop taking clozapine in August 2001. She clinically decompensated, and frequent inpatient stays were required. Neither trials of the newer atypical antipsychotics nor a course of electroconvulsive therapy significantly minimized the patient's psychotic symptoms.

The decision was made to reintroduce clozapine. Five weeks after the treatment was initiated, profound sialorrhea developed at a daily clozapine dosage of 500 mg. All attempts to decrease the dosage did not reduce hypersalivation and led to increased psychopathology. Controlled-release tolterodine at a dosage of up to 4 mg a day in divided doses, a clonidine patch in dosages of up to .3 mg every 24 hours, and benztropine did not reduce the sialorrhea. The patient then started transdermal scopolamine at a dosage of 1.5 mg every 72 hours. Sialorrhea resolved entirely within hours. The benefits have persisted for many months.

The antimuscarinic scopolamine in transdermal form represents an effective option for the treatment of clozapine-induced sialorrhea. Although it can cause the same wide array of anticholinergic side effects as benztropine and other drugs of this class, the transdermal delivery allows for a highly controlled and minimal dose. As evidenced in this case, it is well tolerated and convenient. Its therapeutic value in the treatment of hypersalivation should be further investigated in a controlled prospective study.

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Trauma and Tragedy in New York City

To the Editor: Dr. Satel contends in her editorial in the December issue (1) that posttraumatic psychopathology never really materialized among