Acute Dystonic Reaction to Ecstasy

To the Editor:

Ecstasy (3,4-methylenedioxymethamphetamine) is a recreational drug widely used in Europe and the United States. Intake of ecstasy frequently causes trismus or bruxism, muscle stiffness, and ataxia, and may also cause psychiatric side effects such as paranoia, depression, or psychosis (1). Occasional reports describe more severe ecstasy-related adverse effects, including convulsions, collapse, hyperthermia, disseminated intravascular coagulation, hypotension, and hepatotoxicity (1). In the patient, described here consumption of ecstasy led to an unusual neurological complication.

A 22-year-old man, who had no personal or family history of neurological or systemic disease, but who admitted being an occasional cocaine user, took two tablets of ecstasy. Several months had elapsed since his last intake of cocaine, and he had never taken ecstasy before. Approximately 11 h after ingesting ecstasy, he awoke from a normal night's sleep with a feeling of restlessness, accompanied by difficulty in turning his head and mild dysphagia. Neurological examination on the same day showed spasmodic torticollis to the right with laterocollis and a coarse action tremor of the right upper limb. These manifestations responded promptly to intramuscular anticholinergic treatment (biperidene lactate, 10 mg). The tremor and dysphagia disappeared gradually within 4 days, the torticollis within 11 days. When treatment was interrupted 19 days after the onset of symptoms, the patient complained only of discomfort on turning his head to the left, a symptom that disappeared completely within 2 weeks. Eight months after taking ecstasy, he remained symptom-free.

Our patient's clinical picture resembles an acute dystonic reaction to antidopaminergic neuroleptic drugs. The effectiveness of the symptomatic anticholinergic treatment also suggests a basal ganglia dysfunction. Interestingly, his disorder had a longer recovery course than that usually seen in patients with neuroleptic-induced dystonic reactions. The long time course might depend not upon pharmacokinetic mechanisms but on underlying changes in receptor sensitivity. Ecstasy stimulates serotonin release (2), and serotonergic pathways innervate the substantia nigra and globus pallidus (3), the two major output structures of the basal ganglia. Although ecstasy, like other amphetamine derivatives, exerts major effects on serotonin release, it also influences dopaminergic systems. In the case reported here, the fact that the patient had consumed cocaine—a well-known dopamine stimulant (4)—without complaining of adverse effects makes it unlikely that stimulation of the dopaminergic system precipitated his acute ecstasy-related dystonic reaction. Unusual dystonic reactions of this kind, presumably occur, as do neuroleptic-induced reactions, only in predisposed individuals (5). Alternatively, ecstasy-related acute dystonia might go unreported because drug users tend to underestimate self-limiting complications. Whatever the explanation, acute dystonia induced by ecstasy hardly surprises, considering that another psychostimulant, cocaine, can precipitate or even induce hyperkinetic movement disorders (6). This case report suggests that young adults with acute-onset dystonia should be questioned about possible ecstasy intake.

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Diabetes Mellitus Presenting as Paroxysmal Kinesigenic Dystonic Choreoathetosis

Paroxysmal kinesigenic dystonic choreoathetosis (PKC) is a disorder that is characterized by attacks of chorea, dystonia, or tonic posturing lasting <5 min and triggered by sudden movements or startle (1). These episodes can occur up to 100 times a day and can be preceded by paresthesias or vague sensory phenomenon. Some patients have pain associated with the dystonia. Both familial and sporadic cases can occur. Sensorium is not altered during the episodes. Patients are asymptomatic between attacks (2). PKC has been reported in patients with thalamic infarcts (3), multiple sclerosis (4), progressive supranuclear palsy (5), basal ganglia calcifications (6), thyrotoxicosis (7), and hypoglycemia (8). Nonketotic hyperglycemia can cause seizures, visual hallucinations, focal neurologic signs, disturbance of con-