Neurogenic Bladder and Chronic Urinary Retention Associated with MDMA Abuse

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ABSTRACT

Introduction: The use of 3,4-methylenedioxymethamphetamine (MDMA, known as “ecstasy”), a synthetic amphetamine and “club drug,” has been associated with acute, transient urinary retention. We report a case of neurogenic bladder and chronic urinary retention associated with MDMA abuse.

Case report: A 21-year-old male presented to the emergency department (ED) because he had abdominal pain and difficulty urinating. He had experienced difficulty in initiating urination over the past 1.5 months, with periods of 24 to 36 hours between voids and large volumes of urine. The patient had a chronic pattern of MDMA use, taking 4 tablets/day for 3 months. Two weeks before coming to the ED, he had been admitted to an inpatient drug rehabilitation center. During the time since that admission, the patient had visited EDs repeatedly for insertion and removal of Foley catheters to relieve the urinary retention until he could be admitted to a urologic service. Cystometrogram was abnormal, finding no sensation of bladder fullness after instillation of 350 mL of saline and inability to generate a voluntary voiding pressure. Cystoscopy revealed no outlet obstruction. The findings were consistent with neurogenic bladder. The patient was given prescriptions for bethanecol and phenazopyridine, and told to continue a 10-day course of sulfamethoxazole/trimethoprim for urinary tract infection. He was discharged with a Foley catheter in place. Symptoms of urinary retention persisted at 1-year follow-up, despite self-catheterization and complete cessation of MDMA use.

Conclusion: Chronic MDMA use may lead to neurogenic bladder and chronic urinary retention.

INTRODUCTION

3,4-Methylenedioxymethamphetamine (MDMA, known as “ecstasy”) is a synthetic amphetamine analog that has been used as an appetite suppressant. In the 1970s, it was prescribed by psychiatrists for use during psychotherapy and marriage counseling for its empathogenic effects [1–3]. Its use as a “club drug” has become particularly common among adolescents and young adults in the setting of raves and dance parties to enhance social intimacy and sensory stimulation. A 2003 survey conducted by the U.S. Department of Health and Human Services found that 2.1 million individuals aged 12 and older

had used ecstasy during the preceding year, with approximately 1% of young adults aged 18 to 25 having used it within the past 30 days [4].

MDMA ingestion increases the release of serotonin, dopamine, and norepinephrine from presynaptic neurons and delays their metabolism by inhibiting monoamine oxidase. Adverse effects include hyperthermia, hyponatremia, and syndrome of inappropriate antidiuretic hormone secretion (SIADH). Several cases of transient urinary retention following MDMA ingestion have been reported in the medical literature [5–7]. We report a case of long-term urinary retention in a young patient following prolonged MDMA use.

Acknowledgment: The manuscript was copyedited by Linda J. Kesselring, MS, ELS, the technical editor/writer in the Department of Emergency Medicine, University of Maryland School of Medicine.

Keywords: methylenedioxymethamphetamine, ecstasy (drug), neurogenic bladder, urinary retention

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CASE REPORT

A 21-year-old male presented to the emergency department (ED) for abdominal pain and difficulty urinating. He had been experiencing intermittent difficulty in initiating urination over the past 1.5 months, noting periods of 24 to 36 hours between voids, with large volumes of urine during infrequent voids. Although he had voided a large amount of urine in the ED lavatory prior to being evaluated, he reported his most recent previous void to be approximately 19 hours prior to ED presentation. He denied any recent dysuria, hematuria, or urethral discharge, and denied any history of urinary retention, dysuria, renal insufficiency, prostatic hypertrophy, or recurrent urinary tract infections. His medical history included bipolar disorder, asthma, and hiatal hernia (repaired). Medications included paroxetine hydrochloride, albuterol, fluticasone propionate, and aripiprazole. Social history was pertinent for cigarette smoking, occasional alcohol consumption, and chronic ecstasy use (4 tablets per day for more than 1 year). The patient reported no use of ecstasy or other illicit substances for the past 1.5 months; he had been in an inpatient drug rehabilitation program during this time. Review of symptoms was positive for chronic occipital headaches, poor memory, slowed speech, difficulty with word finding, constipation, and increased thirst over the past 2 months.

Physical examination revealed a thin, white male in mild distress secondary to suprapubic discomfort, which had been relieved significantly by urination. Initial vital signs were as follows: temperature, 97.7° F (36.5° C); heart rate, 72 bpm; blood pressure, 136/74 mm Hg; respiratory rate, 18 breaths/min; and pulse oximetry, 98% on room air. Pupils were 2 mm in diameter, symmetric, and reactive to light. Mucous membranes were moist, and skin turgor was normal. Cardiovascular examination was normal, and lungs were clear to auscultation bilaterally. The abdomen was soft and nondistended, with normoactive bowel sounds. Genitourinary examination demonstrated no significant abnormalities, including no costovertebral angle tenderness, urethral discharge, hernias, phimosis, or testicular tenderness or masses. The bladder was nonpalpable, and rectal exam demonstrated no evidence of prostatitis or prostatic hypertrophy. Neurological examination revealed an alert young man with no focal deficits. Cranial nerves and cerebellar function were intact, there were no paresthesias, weakness or paralysis. He had a flat affect but otherwise had no other findings neurologically.

Laboratory evaluation revealed a normal complete blood count with no evidence of leukocytosis. The basic metabolic profile revealed the following: Na, 142 mmol/L; Cl, 107 mmol/L; BUN, 5.36 mmol/L (18 mg/dL); creatinine, 70.8 µmol/L (0.8 mg/dL). Urinalysis was normal, and a urine drug screen was negative for amphetamine, benzodiazepine, cannabinoid, cocaine metabolite, opiate, and phencyclidine. Urine electrolyte concentrations were as follows: Na, 30 mmol/L; K, 6 mmol/L; Cl, 32 mmol/L. Computed tomography scan of the brain demonstrated no intracranial abnormalities.

A Foley catheter was placed and attached to a leg-bag reservoir, leading to output of approximately 100 mL of clear yellow

urine over the subsequent 3 hours. The patient was referred for outpatient urology follow-up. Eight days later, he presented to a different ED, complaining of difficulty urinating. He had removed the original urinary catheter during the intervening week and required placement of a second Foley catheter for urinary retention during this second ED visit. Two days later, he presented to the ED again, with complaints of urinary retention, dysuria, and urethral discharge, having once again removed his urinary catheter. At this time, a third Foley catheter was placed. The patient was started on sulfamethoxazole/trimethoprim for a urinary tract infection and was admitted for inpatient urology evaluation. Subsequent cystometrogram was grossly abnormal, with no sensation of bladder fullness demonstrated over 350 mL of infused saline solution, and inability to generate a voluntary voiding pressure. Cystoscopy revealed some evidence of prostatitis, but no significant outlet obstruction. The findings were consistent with neurogenic bladder, 8 weeks after the patient's last dose of MDMA. He had remained in a drug treatment program throughout the course of these ED visits. The patient was initiated on bethanechol chloride, 50 mg by mouth every 6 hours, and phenazopyridine hydrochloride, 200 mg by mouth every 8 hours, as well as continued on sulfamethoxazole/trimethoprim. He was discharged with a Foley catheter in place and instructed to follow up with urology in 2 weeks.

CONCLUSION

Normal filling and voiding of the bladder is influenced by a complex interaction of autonomic and somatic neuronal control. Successful micturition requires coordination of detrusor muscle contraction with relaxation of the urinary sphincter. MDMA increases the release of endogenous norepinephrine stores, exerting a sympathomimetic effect on the bladder, which can lead to urinary retention. Effects are typically acute and transient, precipitated by recent MDMA ingestion, and resolve 24 to 48 hours of onset [5–7].

Long-term or permanent urinary retention associated with MDMA or other amphetamine abuse has not been previously reported in the literature. Although the cause of this patient's urinary retention can not be unequivocally attributed to his history of MDMA abuse, other potentially causal influences and comorbidities, such as spina bifida, are absent. This case report is limited by the fact that the only method of confirmation of exposure is the patient's self-reported history of MDMA abuse and subsequent admission to a drug treatment facility for the same. It is also limited since "ecstasy" pills often contain adulterants such as ephedrine and methamphetamine or may not contain MDMA at all, as seen with some "herbal" ecstasy pills which contain 1-benzylpiperazine (BZP). BZP has caused urinary retention, though no reports have confirmed that it causes chronic neurogenic bladder [8].

Of particular interest is the persistence of urinary retention despite the patient's reported cessation of MDMA ingestion more than 1 month prior to presentation, supported by a negative

urine drug screen, and continued participation in a drug-treatment program. The mechanism of MDMA abuse leading to chronic urinary retention is not clear. Possibilities include chronic upregulation of sympathomimetic influence on the urinary bladder, inhibition of parasympathetic control, persistent urinary sphincter contraction, and detrusor muscle dysfunction. This case suggests that chronic urinary retention may be a rare but debilitating complication of long-standing MDMA abuse.

The authors have no potential financial conflicts of interest to report.

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