

1-1-2006

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Recommended Citation

TUFAN, ZELİHA KOÇAK; BULUT, CEMAL; KINIKLI, SAMİ; IRMAK, HASAN; YILMAZ, GÜL R.; and DEMİRÖZ, ALİ PEKCAN (2006) "A Case Report of Ecstasy-induced Acute Hepatic Failure," *Turkish Journal of Medical Sciences*: Vol. 36: No. 5, Article 10. Available at: <https://journals.tubitak.gov.tr/medical/vol36/iss5/10>

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A Case Report of Ecstasy-induced Acute Hepatic Failure

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A Case Report of Ecstasy-induced Acute Hepatic Failure

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Abstract: 3,4-Methylenedioxyamphetamine (MDMA), also known as "ecstasy", is a synthetic, psychoactive drug chemically similar to the stimulant methamphetamine and the hallucinogen mescaline. It can cause cognitive, physical, psychological and neurological side effects. Ecstasy-induced acute renal failure, liver damage and brain edema are reported in the literature. We report an MDMA-induced toxic hepatitis case which was thought to have viral origin.

Key Words: Ecstasy, toxic hepatitis, 3,4-methylenedioxyamphetamine, MDMA

Ecstasy'e Bağlı Bir Toksik Hepatit Olgusu

Özet: Sentetik amfetamin derivativesi olan 3,4-metilenendioksümetamfetamin (MDMA) veya "Ecstasy" psikodelik ve stimulan etkileri olan bir ilaçtır. Psikolojik ve fiziksel yan etkilere yol açabilir. MDMA'ya bağlı karaciğer ve böbrek yetmezliği, beyin ödemi gelişen vakaların sayısı son yıllarda giderek artmaktadır. Bu çalışmada akut viral hepatit ön tanısı ile yatırılan ancak MDMA'ya bağlı toksik hepatit tanısı ile tedavi edilen bir olgu sunulmaktadır.

Anahtar Sözcükler: Ecstasy, toksik hepatit, 3,4-Methylenedioxyamphetamine, MDMA

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Introduction

3,4-Methylenedioxyamphetamine (MDMA), so-called "ecstasy", is a psychoactive amphetamine-derived drug. Street names for MDMA other than ecstasy include Adam, XTC, hug, beans, and love drug. It was used in past centuries for psychotherapy. It is popular among adolescents and young adults in the nightclub scene and during weekend-long dance parties known as raves, and has spread beyond the rave and nightclub scene to a variety of urban, suburban and rural areas. MDMA can cause psychological side effects like confusion, depression, sleep problems, drug craving, and severe anxiety (1). The physical side effects include increases in heart rate and blood pressure, a special risk for people with circulatory problems or heart disease, and other symptoms such as muscle tension, involuntary teeth clenching, nausea, blurred vision, syncope, and chills or sweating. In high doses, MDMA can interfere with the body's ability to regulate temperature. This can lead to a sharp increase in body temperature, resulting in liver, kidney, and cardiovascular system failure (1). Animal experiments have shown a serotonergic destruction in the brain caused by MDMA. It is also known that MDMA can increase the activity levels of serotonin, dopamine and norepinephrine, leading to cognitive and memorial disorders among long-term MDMA users (2,3). Ecstasy-induced acute renal failure, liver failure and brain edema are reported in the literature (4-6). We report a patient with MDMA-related toxic hepatitis, thought to be of viral origin, who was admitted to our clinic for investigation of the jaundice etiology.

Received: March 08, 2006
Accepted: October 09, 2006

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Case Report

A 29-year-old woman presented to our outpatient department with fever, nausea, fatigue, and complete jaundiced appearance, including her eyes. The patient was thought to have viral hepatitis and was admitted to our clinic to determine the etiology. On physical examination, vital signs including body temperature were normal. The sclera,

oral mucosa and skin were icteric and a scar from cholecystectomy operation (2 years before) was noted; other findings were normal. Her laboratory findings were as follows: Hb: 12.7 g/dl, Htc: 37%, WBC: $8,800 \times 10^3/\mu\text{L}$, Plt: $147,000 \times 10^3/\mu\text{L}$, ESR: 2 mm/h, glucose: 167 mg/dl, total bilirubin: 24 mg/dl, direct bilirubin: 15.67 mg/dl, SGPT: 1164 U/L, SGOT: 1468 U/L, total protein: 6.38 g/dl, albumin: 3.66 g/dl, alkaline phosphatase: 293 U/L, GGT: 276 U/L, PTT: 19.6 seconds, INR: 1.82, and aPTT: 42.1 seconds. Her viral hepatitis markers were: HBs Ag (-), Anti-HBs (-), Anti-HBc IgM (-), HBe Ag (-), Anti-HAV IgM (-), and Anti-HCV (-). Anti-HIV 1/2 and TORCH group antibodies (IgM and IgG) were all negative. No antibody in the serum for leptospirosis was established by using micro-agglutination test (MAT) for serology. Anti-nuclear antibody (ANA), anti-mitochondrial antibody (AMA), smooth muscle antibody, leukocyte common antigen, IgM and IgG, copper and ceruloplasmin levels in urine and serum were checked to investigate autoimmune hepatitis and Wilson's disease, but all were within normal ranges. On ultrasound evaluation, the liver was of normal dimensions but the parenchyma was minimally hypoechoic, granular and heterogenic. No cholestasis or common bile duct stone was seen. Abdominal computerized tomography (CT) revealed normal findings. The patient repeatedly denied drug usage when questioned. She was given supportive therapy and vitamin K. One week later it was learned from one of her friends that she had been using ecstasy for more than one year. The patient eventually also admitted that she and her husband had been using ecstasy for a long period but she refused to provide details of how frequently. Her liver enzymes returned to normal levels and she was discharged from the hospital, but failed to return for her follow-up visits in the outpatient department.

Discussion

Although ecstasy is known as a safe drug among the young population, there are reports of fatal complications. Signs of acute toxicity most often seen are fulminant hyperthermia, hyperexcitatory states, acute renal failure and hyponatremia (7,8). The effects of MDMA on the liver vary from acute hepatitis to fulminant liver failure. Liver necrosis was found on autopsies of patients who died because of MDMA-related liver failure

(9). While the mechanism of liver failure due to MDMA is still unknown, the possible effects of MDMA are effects on body temperature regulation, toxic effects of ecstasy tablet ingredients and for some people the genetic tendency of toxic effects of amphetamine and its derivatives. However, effects of dose and addiction duration and risk factors for MDMA-related liver damage are not clear. In one study, ecstasy was found to be the second factor responsible for liver damage after viral etiology among those under 25 years of age (5).

Greene et al. (4) reported a series of seven patients who ingested ecstasy in a nightclub and presented with varying degrees of MDMA toxicity. Three of them had presented with features of severe MDMA toxicity. One died within an hour of hospital admission, another died four days later after developing fulminant hepatic failure, and the third recovered after 12 days in intensive care. Lange-Brock et al. (10) reported a 17-year-old female patient who had developed acute liver failure after regular use of ecstasy over a six-month period. She made a full recovery following liver transplantation.

Ellis et al. (6) reported eight cases of ecstasy-related acute liver damage. Two patients had presented after collapse within six hours of ecstasy ingestion with hyperthermia, hypotension, convulsions, and subsequent disseminated intravascular coagulation with rhabdomyolysis together with biochemical evidence of severe hepatic damage. One patient had recovered and the other with evidence of hyperacute liver failure was transplanted but subsequently died. Four patients had presented with acute liver failure without hyperthermia. One died before a donor organ became available, and two died within one month post-transplantation of overwhelming sepsis. Two patients presented with abdominal pain and jaundice and recovered over a period of three weeks.

In our case, the clinical and laboratory findings resembled cholestasis. All etiologic factors including other drugs which can cause a similar clinical presentation were investigated, but nothing other than ecstasy was determined as a cause. In conclusion, ecstasy and other drugs have to be considered as causes of non-viral hepatitis and should be questioned carefully during anamnesis. Efforts should be given to better educating the young population regarding the dangers of ecstasy use.

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