

of transverse myelitis. No systemic manifestations of SLE appeared during a 4-year follow-up, the residual spastic tetraparesis appeared unchanged and MG was in remission without treatment.

A wide variety of autoimmune diseases have been reported in patients with MG [2]. The association between MG and MS is not considered frequent: in many reports of the literature the diagnosis of MG according to the present criteria appears uncertain [3] and, similarly, the diagnosis of MS was clinically established without the support of laboratory findings. The diagnosis of MG and MS in this young female was made according to widely accepted diagnostic criteria.

Interestingly, this patient developed, after thymectomy, relapsing transverse myelitis that always affected the same cervical level. During these episodes, we found serological features which were strongly suggestive of a collagen vascular disease. Among the various pathogenic mechanisms that underlie recurrent transverse myelitis, the preexisting MS may be considered a possible candidate. However, the evidence of serological markers of SLE during the relapses of myelitis strengthen the hypothesis of an SLE-related relapsing myelitis. Recurrent transverse myelitis has been reported as an isolated manifestation of SLE [4] and an association between MG and recurrent transverse myelitis with laboratory markers of SLE has been described [5]. The frequency of SLE in MG patients appears higher than expected, and SLE often develops after thymectomy [6, 7], perhaps favored by the loss of T suppressor cells that follows thymectomy [8]. Also in our patient a decrease in T suppressor cells was detected during each recurrence of transverse myelitis. In this case the genetic background probably played a role in the combination of autoimmune diseases [9]: HLA typing disclosed the contemporaneous presence of B8, DR2 and DR3 HLA antigens which seem to have increased the risk of concurrent autoimmune conditions and thymectomy would appear to be a triggering factor.

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Acute Inflammatory CNS Disease after MDMA ('Ecstasy')

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The amphetamine derivative (+/-)3,4-methylenedioxymethamphetamine (MDMA, 'ecstasy', 'Adam') (fig. 1) has emerged as a popular drug of abuse over the last two decades. MDMA produces a mixture of central stimulant and psychedelic effects, many of which are mediated by the neurotransmitters dopamine and serotonin [1]. There has been a recent increase in case reports on serious side effects such as psychiatric disorders (panic attacks, depression, paranoid psychoses, flashbacks, catatonic stupor), neurologic diseases (seizures, intracerebral haemorrhage, sinus venous thrombosis, cerebral infarction) and complications outside the nervous system (disseminated intravascular coagulation, fulminant hyperthermia, rhabdomyolysis, acute renal failure, gastro-intestinal haemorrhage, acute hepatitis, impaired liver function, aplastic anaemia) [2-6]. We present a case of acute inflammatory cerebral disease associated with heavy MDMA misuse.

A 22-year-old male presented with one generalized seizure after a 5-day history of fever ($>39^{\circ}\text{C}$) and gradual reduction of vigilance and orientation. Due to increasing respiratory failure mechanical respiration was initiated. Routine laboratory evaluations revealed signs of inflammation with elevated leucocyte count ($25,100/\mu\text{l}$, 63% segmented) and C-reactive protein (117 mg/l). Magnetic resonance imaging (MRI) showed in the T_2 -weighted scans disseminated, hyperintense white matter lesions in all parts of the brain (fig. 2) with decreased signal intensity and minor gadolinium enhancement in T_1 -weighted scans. CSF analysis yielded mononuclear pleocytosis ($60/\mu\text{l}$; 74% lymphocytes, 14% granulocytes, 12% monocytes), elevated protein (901 mg/l) and normal lactate content. Soluble intercellular adhesion molecule 1 (sICAM-1) was detected in CSF and serum with an index of 2.8, indicating an intrathecal synthesis [7]. Electroencephalography revealed an unspecific diffuse alteration. Culture examinations for bacteria and fungi, serologic tests for neurotropic bacteria, fungi and viruses, screening for common vasculitis parameters including rectal biopsy, ultrasound sonography of cerebral arteries and echocardiography were all negative. After 7 days of treatment (high dose prednisolone, amoxicilline/gentamycin, mannitol 15%, analgesia) the patient gained consciousness. After another 3 days of slight confusion he totally recovered.

After his recovery the patient and his girlfriend reported that he had taken orally an unknown, but extensive dose of ecstasy in the evening prior to the acute disease onset. He had practised an abuse of this drug in lower amount (once or twice a week) for half a year before. The consumption of other drugs was denied. Since we got acquainted with this fact too late, we were not capable of detecting MDMA or any other drug in the patient's body fluids now, 14 days after disease onset. Two weeks after disease onset and after clinical recovery a second CSF analysis was performed (leucocyte count $10/\mu\text{l}$, protein 320 mg/l). MRI revealed resolution of lesions to a high degree, although some were still visible.

Here we report a case of an acute febrile condition with inflammatory CNS involvement following an excessive abuse of MDMA. With regard to MRI and CSF findings either acute disseminated encephalomyelitis (ADEM) or immune-mediated vasculitis may be

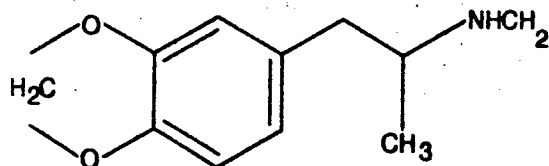
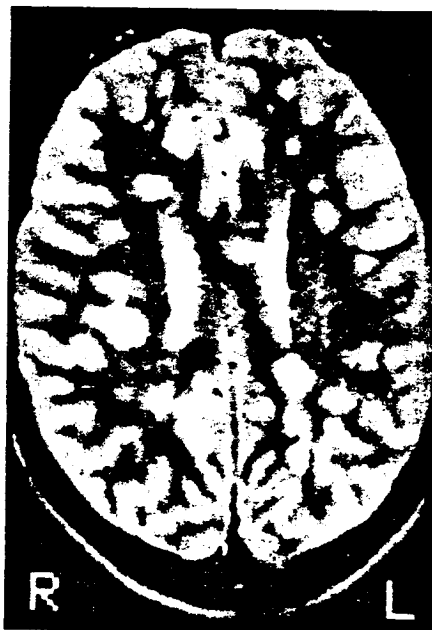


Fig. 1. Structure of the ring-substituted amphetamine derivative (+/-)-3, 4-methylenedioxymethamphetamine (MDMA, 'ecstasy').

Fig. 2. MRI scans of the 22 year old patient, showing with T₂ technique minated white matter lesions.



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considered. However, due to the lack of a recent infectious disease or vaccination and the short disease duration with excellent outcome EM seems rather unlikely. The lesions still visible on second MRI scans may have been definite infarctions that had been surrounded by edema on the first series of scans. sICAM was synthesized intracranially but is an unspecific indicator of inflammation and has never been investigated in cerebral vasculitis so far.

We were not able to exactly define the trigger of this disease. Yet, the patient had a history of extensive MDMA abuse prior to the acute disease onset. There is a strong temporal correlation between drug intake and disease onset. Moreover, some of the symptoms presented by the patient are known side effects of MDMA (fever, seizures, psychiatric disturbances) [3]. Because ecstasy is known to be a 'dirty drug' and frequently combined with other drugs, it is equivocal whether the syndrome was due to ecstasy alone or to additional substances.

We suggest that the above described acute inflammatory CNS disease was triggered by MDMA, possibly initiating a special form of hypersensitivity vasculitis. Cerebral vasculitis is a rare complication of amphetamine intake, but has never been histopathologically confirmed in ecstasy abuse so far [8, 9]. MDMA may either serve as a trigger or induce delayed type hypersensitivity (DTH) via serotonin release and consecutive activation of DTH effector T cells [10].

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