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Case report

Ecstasy and dementia in a young subject

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Summary – We report a cerebrovascular accident after Ecstasy (MDMA) intake which induced a frontal dementia in a young man.

dementia / brain, injury / Ecstasy

CASE REPORT

Cerebrovascular injury was induced by drug abuse in a young subject. Sudden occurrence of frontal dementia in a 20-year-old man was induced by oral intake of Ecstasy (3,4-methylenedioxymetaamphetamine [MDMA]). The suddenness and magnitude of the lesions were astonishing and the social management of the young patient was worth studying.

This 20-year-old man, who occasionally used various substances, exhibited damage to the entirety of his frontal lobes (left and right) and his right temporal lobe after taking Ecstasy.

Immediately after drug intake, psychomotor agitation with epileptoid signs occurred and the patient fell into a coma. He needed respiratory support for 5 days. When he awakened from the coma, he exhibited a dissociation syndrome with delirium and visual hallucinations. That pharmacopsychosis was treated with neuroleptics (haloperidol and cyamemazine). After 20 days of this treatment, a frontal syndrome occurred and persisted after treatment withdrawal. The patient was totally amnesic of his past and could not remember new facts. He presented with a number of gesticular and verbal stereotypes. He was socially and sexually uninhibited, manifested hyperphagy and coprolalia and was considered to be socially very dangerous.

Electroencephalograph could not be performed because of the patient's agitation.

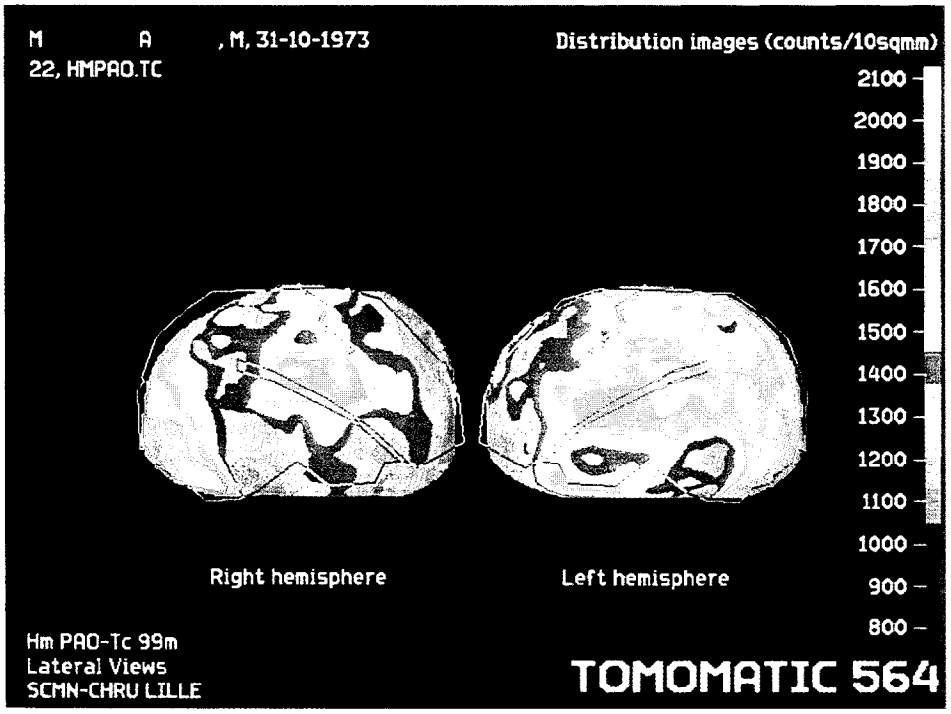
Brain magnetic resonance imaging (MRI) revealed cortical and deep global atrophic encephalopathy, more marked in the temporal region but with preserved hippocampus and major ventricular dilation.

Brain blood flow was measured 2 months after the end of coma, which revealed deeply altered brain profusion, bilaterally localized in the prefrontal lobes, the right anterior pole and associated posterior cortex of the right hemisphere (fig 1).

Brain angiography was also performed at the same time. No vascular abnormality was noted except a thinness of distal arteries in the right and left frontal regions and the right temporal region, with no impact on brain tissue; no circulatory restriction was observed. Biological workups were normal.

CONCLUSION

The reality of cerebrovascular accidents secondary to drug intake has only recently been known. In most cases these include hemorrhagic or ischemic lesions which are small in volume. These lesions may result from short transient ischemia affecting the brain arteries and induce somatic of mental deficiency which can remain unnoticed because of the subject's unconsciousness. These deficiencies may include loss of hearing, ringing or buzzing in the ear and visual disorders resulting from damaged micro-arteries (Swanson et al, 1991). In this case, however, the lesions were very large. It appeared that Ecstasy was responsible for these types of lesions. The patient fell into a coma within 1–2 min after taking the drug. The blood



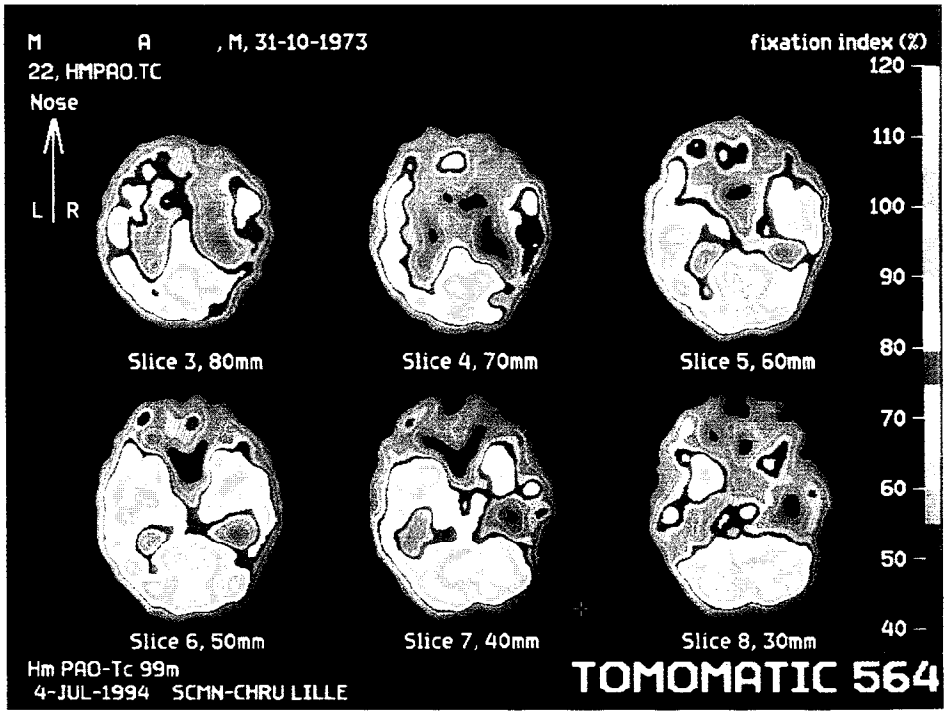


Fig 1. Cerebral debimetry, with Hm PAO-Tc 99m, in a 20-year-old subject after intake per os of Ecstasy.

alcohol content, however, should be taken into account. When high, it can induce vasoconstriction (Hillbom, 1987; Camargo, 1989; Gorelick, 1989; Mathew and Wilson, 1991) in the same manner as products released by a lit cigarette, of which nicotine is only one (Mathew and Wilson, 1991), and a combination of other products or substances. Other than opioid agonists, all addictive substances can induce vasospasm and/or vasoconstriction (Gorelick, 1990).

Mixing alcohol with other psychotropic substances is an old and frequent practice. Drug mixtures appear to be just as dangerous as drugs taken alone. However, the recently introduced new chemically engineered drugs like Ecstasy deserve to be paid special attention, either used alone or mixed with other substances. Users of these novel substances need rigorous neurological and psychiatric clinical assessment, and brain MRI combined with cerebral blood flowmetry are crucial if there is coma.

Cumulative potentiation effects are present under habitual use of alcohol, nicotine, cocaine, caffeine, and amphetamines. Cerebrovascular accidents are one of the risks incurred. Subjects with cerebrovascular disorders are more at risk than the general population. The use of addictive substances, either mixed or as single products, constitutes an epidemic risk among adolescents and young adults that cannot be ignored by neurologists and psychiatrists.

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