Physostigmine Reversal of Psilocybin Intoxication

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Recently we observed a case of acute intoxication caused by eating mushrooms containing psilocybin, an anti-serotonergic drug known to cause hallucinations. This case of proven psilocybin intoxication is presented as an anticholinergic syndrome. Rapid reversal of all symptoms was obtained by the intravenous administration of physostigmine.

REPORT OF A CASE

A 21-year-old man, with no previous psychiatric drug abuse history, was taken to the hospital after eating at least 30 mushrooms in a discotheque. The patient was very excited and anxious; he vomited on arrival at the hospital. He was conscious and his auditory perception was enhanced. Several times every minute he experienced visual hallucinations, which were characterized by the 'flashback' phenomenon, referring back repeatedly to a single specific traumatic incident in his past, associated with the suicide of a girlfriend. Between these hallucinations he was very anxious and restless but could not be addressed. His face was extremely red and his skin was very warm and dry. The blood pressure was 170/115 mmHg, heart rate 120/min, and the rectal temperature of 37.8° C. Most striking were the widely dilated pupils, which were unreactive to light. He complained of difficulty in micturition. The vomitus contained the remains of the mushrooms. Laboratory values were: Hb 16.9 g/dl, Ht 49%, leukocytes 12.2 x 10^9/ l, m³, sodium 140 mEq/l, potassium 3.38 mEq/l, creatinine 1 mg/dl, glucose 90.8 mg/dl, and serum osmolality 296 mosmol/l.

The clinical picture being very suspicious of a belladonna intoxication, 0.4 mg physostigmine was given very slowly intravenously over a period of 5 minutes. Five minutes later, the interval between the hallucinations became longer, lasting several minutes. After 25 minutes no more hallucinations were observed; the patient was still apprehensive that the hallucinations returned and he complained of thirst. After 40 minutes he was quiet and relaxed; arterial blood pressure was 125/80 mmHg and the pulse rate 80/min. A polynuria of one liter in 15 minutes occurred. The pupils were still dilated, but reactive to light. The patient was observed for 36 hours; no periods with hallucinations, excitement, or anxiety reappeared. Additional physostigmine was not required. The pupil diameter returned to normal within a few hours. The mushrooms and urine from the patient were sent for biochemical analysis. The mushrooms were identified as Psilocybe semilanceata; they contained psilocybin. The urine, tested by the methods of thin layer chromatography and ultraviolet spectrophotometry, was found to contain psilocin. Atropine and scopolamine were absent.

DISCUSSION

Mushrooms have probably been used as hallucinogenic agents for many centuries by the Indians of Mexico and other parts of Central America (Psilocybe mexicana Heim). Psilocybe semilanceata is a very common fungus in many parts of the world, growing in North and South America, Australia, England, France, Scandanavia, and also in the Netherlands. The principle active substance of these hallucinogenic mushrooms is psilocybin.1,2 Symptoms caused by an intoxication of psilocybin often are defined as sympathomimetic.3,4 The recommended therapy is chlorpromazine and diazepam.5,6 Despite this therapy, patients have symptoms of intoxication lasting from 96 to 120 hours; Benjamin even described a case in which panic attacks persisted for three months after eating mushrooms containing psilocybin.7

In contrast to the literature, we believe that the symptoms caused by psilocybin intoxication, were not due to a central stimulation of the sympathetic nervous system, but possibly due to a direct or indirect blockade of the central and peripheral parasympathetic nervous system. One of the first signs of sympathetic stimulation is excessive sweating, whereas the skin of our patient was warm and dry. Because the signs and symptoms of this psilocybin intoxication were identical to those of atropine or scopolamine intoxication, the patient was successfully treated with intravenous physostigmine. Further investigations obviously will be required to further clarify the mechanism of psilocybin intoxication and its reversal by physostigmine.

REFERENCES