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Clinical Notes

Carotid Artery Obstruction Following LSD Capsule Ingestion

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A wide variety of acute and chronic psychiatric symptoms have been seen in patients taking lysergic acid diethylamide (LSD).[1] We report a case of particular interest because the patient developed focal neurologic signs.

Patient Summary

A 14-year-old boy with no previous history of epilepsy, cardiovascular or neurological disease, or head trauma was hospitalized because of a short generalized seizure characterized by eye rolling, clonic and tonic movements, and loss of consciousness. For one month prior to admission, his mother had noted episodes of elation and drowsiness. Family history was unremarkable. A history obtained from four of his companions revealed that the patient had taken four LSD capsules shortly before the seizure. No other capsules were found.

Physical examination revealed a well-developed boy, mildly lethargic and restless. The blood pressure was 102/70 mm Hg; pulse rate, 88 beats per minute and regular; respiratory rate, 16 per minute; and temperature 100.6 F (38.1 C). There were no signs of trauma or nuchal rigidity. Positive physical findings were confined to the neurological system. The patient was unable to respond to spoken words and exhibited jerking movements of the extremities. The cranial nerves were intact, and there was no localized muscle weakness. The deep tendon reflexes were hyperactive and symmetrical, and the plantar responses were flexor.

Laboratory Work.—Laboratory values were as follows: Hemoglobin, 16.2 gm/100 ml; hematocrit, 47%; white blood cell count, 10,500/cu mm, with 67% neutrophils, 32% lymphocytes, and 1% eosinophils; urine, normal; erythrocyte sedimentation rate, 14 mm/hr; fasting blood glucose, 102 mg/100 ml; blood urea nitrogen, 9 mg/100 ml; and serologic test for syphilis, negative. Cerebrospinal fluid (CSF) protein concentration was 51 mg/100 ml; glucose, 82 mg/100 ml; and the CSF was clear and without cells. Electrolyte levels, chest x-ray film, electrocardiogram, skull x-ray films, and brain scan were normal.

Hospital Course.—On the second hospital day, the plantar responses became extensor, and generalized spasticity was present. The spastic tendencies and Babinski signs disappeared on the third day, but the latter returned on the left side on the following day. On the fifth hospital day, the patient developed a left hemiplegia, left homonymous hemianopsia, and conjugate deviation of the eyes to the right. A right carotid arteriogram performed on the seventh day revealed progressive narrowing of the internal carotid artery from its origin in the neck to the carotid siphon with a total obstruction just before its bifurcation into the middle and anterior cerebral arteries. A thin ophthalmic artery was the only branch to fill with contrast media. On the following day, the patient was taken from the hospital by his parents against medical advice. At that time, the left hemiplegia was still present, but the extraocular movements were normal, and the patient was alert.

Comment

LSD, usually consumed through the impregnation of sugar cubes and blotters, has begun to appear in capsule form. When an individual chews a sugar cube or blotter, there is a potential autoregulation of the dose because he may stop when the hallucinogenic effect is achieved. Ingestion of these capsules, on the other hand, allows for the irrevocable delivery of an unknown amount of LSD and possibly myriad contaminants.

A cerebrovascular accident with focal neurologic signs has not been previously described in association with LSD ingestion. The carotid artery obstruction had an arteriographic appearance (Figure) compatible with arteriospasm. It is unlikely that vasoconstriction resulted from the introduction of a needle and contrast media into the common carotid artery, because both the common and external carotid arteries were of normal caliber and the branches of the latter vessel filled promptly and completely.

Internal carotid artery obstruction, an unexpected finding in a 14-year-old, previously healthy boy, might lead one to consider a vasculitis such as polyarteritis or cranial arteritis, but the normal sedimentation rate and lack of multisystem complaints largely refute this diagnosis. Vasospasm secondary to a seizure would be expected to cause obstruction in the major vessels of the neck rather than in the intracranial portion of the internal carotid artery.

Although we know of no previous reports of severe arteriospasm or oc-

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Carotid arteriogram. Left, *Common and external carotid arteries are patent, and branches of latter fill before the internal carotid artery.* Right, *Internal carotid artery is narrow, and its filling is delayed. There is a total obstruction of the distal end* (arrow).

clusion following LSD consumption, one can postulate a possible etiologic relationship. Like its close congener, methysergide, LSD is an amine derivative of the ergot alkaloids, which are potent vasoconstrictors.[2] The amine derivatives are thought to have a much smaller effect on vasomotor tone than the parent compounds, but methysergide has been clearly implicated as a vasospastic agent. Reversible peripheral arterial insufficiency,[3-10] intestinal ischemia,[11] and angina pectoris[3] have been described in young, previously healthy patients taking methysergide for prophylaxis against vascular headaches.

Since the pharmacologic actions of methysergide are shared by LSD,[2] it is reasonable to presume that LSD might also be responsible for arterial constriction in patients with hypersensitive vessels. Furthermore, in view of the findings in our patient, one might postulate that arteriospasm may be involved in the pathogenesis of some LSD-related mental aberrations.

Summary

After the ingestion of capsules said to contain LSD, a 14-year-old boy developed focal neurologic changes associated with obstruction of an internal carotid artery. Physicians should be alert to probable serious vasospastic effects of some encapsulated, illicit drugs. There may be an etiologic relationship between LSD and arterial obstruction.

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