**Carotid Artery Occlusion Following Ingestion of LSD**

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**Abstract:**

A 20-year-old white woman developed a left hemiplegia following the ingestion of LSD. Initial angiography revealed marked constriction of the internal carotid artery at the level of the siphon, which, in a few days, was converted to occlusion of the carotid artery at that level. It is suggested that LSD, an amine derivative of the ergot alkaloids, was responsible for the initial vasoconstriction and subsequent occlusion.

**Additional Key Words**

- acute hemiplegia in young adults
- lysergic acid diethylamide
- drug abuse

**Introduction**

The sudden onset of hemiplegia in a drug abuser has been well documented, usually follows parenteral drug injection (heroin, amphetamine) and has been related to both embolization and/or arteritis. The occurrence of hemiplegia following oral drug ingestion has been less often recorded. When this has occurred, it has been related to known vasoconstricting agents or to congeners of known vasoconstricting agents. There have been previous reports relating the onset of a focal neurological deficit to lysergic acid diethylamide (LSD), an amine derivative of the ergot alkaloids, which are known to be potent vasoconstricting agents. We wish to report another case in which there was a close temporal relationship between ingestion of LSD and the onset of a hemiplegia.

**Case Report**

L.B., a 20-year-old, right-handed white woman, was admitted to the Good Samaritan Hospital, West Islip, New York, on February 20, 1971, with a left hemiplegia of acute onset. There was no history of congenital or rheumatic heart disease, systemic infection, hypertension, diabetes, hyperlipidemia, migraine headaches, or head or neck trauma. There was no familial history of hypertension, diabetes, hyperlipidemia, early heart attack or stroke, or migraine headaches.

Late the previous evening she had ingested LSD orally beginning "a trip." She stated that this was the third occasion she had used LSD and that no heroin or other drugs had been used. She was not on oral contraceptive agents. On the morning of admission, she developed a severe generalized headache associated with nausea and vomiting followed by the onset of the left-sided weakness.

Examination at this time revealed a lethargic girl with slow speech but without aphasia and a left hemiplegia; the right limbs were normal; there were no sensory or visual field abnormalities. Lumbar puncture was performed and revealed normal opening and closing pressures, clear, colorless CSF without cells and with a normal protein. On the third hospital day, a right carotid angiogram was performed and this showed marked constriction of the internal carotid artery at the siphon with only a trickle of dye beyond this point (fig. 1). Left carotid injection filled the entire carotid system including the major portion of the right side. Brain scan with Technetium 99m demonstrated an area of increased uptake roughly corresponding to the distribution of the right middle cerebral artery. The patient's neurological condition remained stable and she was later transferred to Bellevue Hospital, New York. A repeat right carotid angiogram, done nine days after the first angiogram, now showed occlusion of the carotid artery at the level of the siphon, the site of the previous constriction (fig. 2). The patient's neurological condition remained unchanged.

**Comment**

In this patient, the hemiplegia was secondary to an occlusion of the internal carotid artery. The initial angiogram showed a high-grade stenosis at the level of the siphon followed, within nine days, by occlusion at this site, suggesting that the underlying mechanism was vasoconstriction (secondary to local spasm) followed by stenosis and secondary thrombosis. The age of the patient, the normal-appearing left carotid artery and remainder of the cerebral circulation and the site of the occlusion, at the level of the carotid siphon, rather than at the common carotid bifurcation, all make it unlikely that the occlusion was on an atherosclerotic basis.

**Discussion**

There have been two case reports of focal clinical defects after LSD. Sobel et al. reported a 14-year-old boy who ingested four LSD capsules shortly before developing generalized cerebral dysfunction.
Right carotid angiogram showed marked narrowing of the carotid artery at the level of the siphon. A thin trickle of dye is seen distal to the site of narrowing.

manifested by seizures and confusion followed within five days by the onset of a focal deficit: a left hemiplegia and a left homonymous hemianopia. A right carotid angiogram performed two days after this revealed constriction 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. Lignelli et al. reported a 19-year-old who, after the injection of LSD and heroin, developed a focal neurological deficit characterized by global aphasia and in whom angiography showed a diffuse arteritis with alternating areas of constriction and dilatation of intracranial vessels. Also, Rumbaugh et al. reported a 24-year-old who developed generalized cerebral dysfunction characterized by seizures and headaches following the ingestion of LSD, and in whom cerebral angiography revealed several small focal arterial occlusions presumably secondary to vasoconstriction.

LSD, lysergic acid diethylamide, is a close congener of methysergide, 1-methyl D-lysergic acid butanolamine (Sansert), and both are amine derivatives of the ergot alkaloids, potent vasoconstricting agents. The ergot alkaloids are vasoconstrictors of both the intracranial and extracranial branches of the external carotid artery. This is the basis for their use in the treatment of migraine headaches. Methysergide is thought to act by a similar mechanism. There have been several reports of peripheral vascular insufficiency with peripheral arterial occlusions secondary to the use of methysergide. While behavioral effects presumably unrelated to vasospasm have been stressed with LSD, the exaggeration of focal defects in patients with known focal lesions after the ingestion of LSD
suggests that, like its close congener, methysergide, LSD may have a vasospastic action as well.

The occurrence of a hemiplegia following the occlusion of an internal carotid artery in a young person without evidence of congenital or rheumatic heart disease, systemic arteritis, premature atherosclerosis and drug abuse, while uncommon, has been well documented. Several etiologies have been considered, but the usual relationship of the onset of the hemiplegia to either local trauma or an infection in the region of the carotid artery has led to the concept of a local arteritis as a cause of local carotid artery spasm, and vasoconstriction with subsequent occlusion. Angiography, and postmortem examination corroborate this idea. A similar mechanism of local vasoconstriction (with or without arteritis) with subsequent arterial occlusion in response to LSD is suggested as being responsible for the hemiplegia in our patient.

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References
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