CASE REPORT

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Cerebral Intraventricular Lipoma and Sudden Death

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ABSTRACT: A lipoma in the left lateral cerebral ventricle of a 73-year-old male is reported. This rather infrequently occurring lesion was an incidental finding in the patient's postmortem examination and probably accounted for the acute hydrocephalus that lead to his sudden death.

KEYWORDS: pathology and biology, intracranial lipoma, sudden death, intracranial lipoma

A 73-year-old white man without any known medical history was admitted to Good Samaritan Hospital in Suffern, New York for vague complaints.

The patient was disoriented and displayed an uncooperative attitude, signing himself out shortly after admission, against medical advice and before any work-up could be initiated.

He collapsed on the street some hours later and was brought back to the hospital, where he was pronounced dead on admission. A postmortem examination of the body was conducted the next day at the Office of the Medical Examiner of Rockland County, New York.

Autopsy Findings

Except for findings in the central nervous system, the results of the gross and microscopic examination of the body did not contribute to explain the patient's demise.

Toxicologic studies for carbon monoxide, ethanol and other volatiles, barbiturates, basic drugs, and heavy metals yielded negative results.

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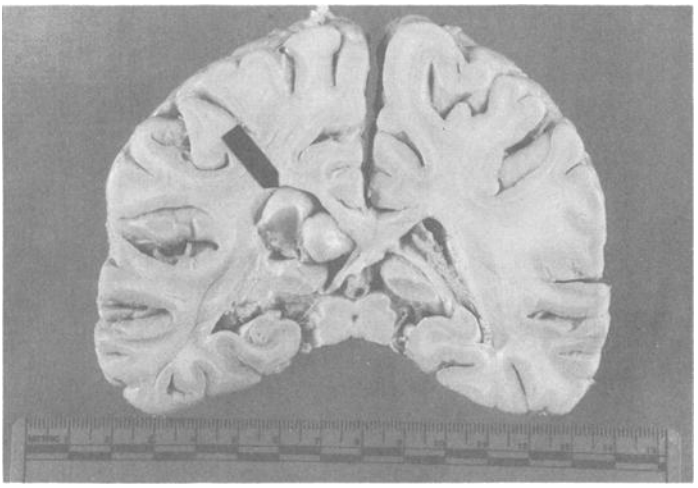

FIG. 1—*Coronal section of the brain viewed from behind, with the lipoma occupying the dilated left lateral ventricle* (arrow).

Central Nervous System

The brain with the cerebellum weighed 1350 g; a herniation on the inferior aspect of the cerebellum with molding of the tonsils against the foramen magnum was noted.

Sequential coronal sections through the supratentorial tissues revealed a fatty mass in the left lateral ventricle. This mass almost totally filled the central part of the ventricle, causing its dilatation and a shifting of the septum pellucidum to the right (Fig. 1).

The fatty mass inside the left ventricle measured 35 by 26 by 24 mm. It was solid, lobulated, well encapsulated and nonadherent to the walls. Some choroidal tissue was attached to its posterior pole (Fig. 2).

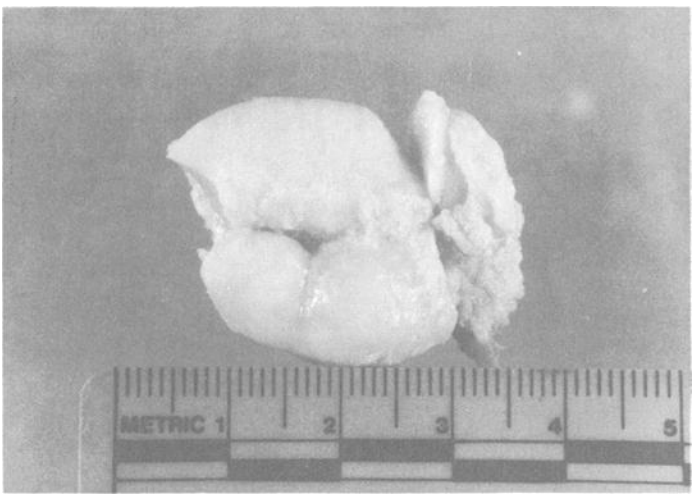

FIG. 2—*Lipoma, gross, lateral view. Note the smooth, encapsulated surface of the lesion, and the presence of choroid tissue attached to its posterior pole (to the right). The tip of the anterior pole of the lesion is missing.*

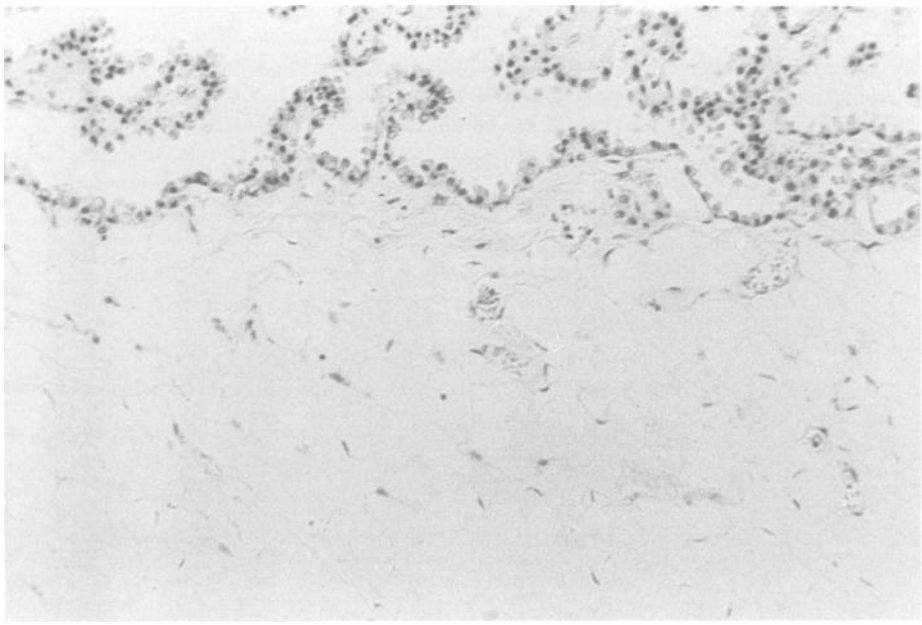

FIG. 3—*Lipoma, microscopic. The lesion is exclusively formed by mature, unremarkable fat cells. Choroidal tissue is attached to the external aspect of the thin fibrotic capsule surrounding the lesion. (×100)*

Microscopic examination showed the mass to be composed of normal, mature fat cells (Fig. 3).

Discussion

Intracranial lipomas are infrequent postmortem findings, being present, for instance, in only 0.08% of the cases in a series of 5000 unselected, consecutive autopsies [*1*].

Even in more selected neuropathologic studies such as the one by Budka, [*2*] the incidence of intracranial lipomas was low, being found in 0.45 and 0.25% in the two independent series of autopsies quoted by this author.

The use of noninvasive diagnostic techniques for the in vivo detection of these lesions produced results similar to the ones just stated, as in a series of Kazner et al., [*3*] in which, out of 3500 brain tumors studied by computerized tomography only 0.34% turned out to be lipomas.

In a third of the cases, intracranial lipomas are found adjacent to the corpus callosum, [*4*] as initially described in the literature by Rokitansky in 1856 (quoted in [*3*]).

Lipomas at this site have been identified as a possible cause of epilepsy. In fact, the association of epilepsy and lipomas impinging upon the corpus callosum seems to be recognized with increasing frequency [*5*].

The criteria used for the definition of intracranial lipomas are not very stringent. Some authors tend to include in that category of lesions hamartomas, and even malformations with agenesis of the corpus callosum or other anomalies, or both, rather than true neoplasms [*1–6*].

Contrary to those, the lesion reported here may be regarded, based on its gross and microscopic features, just as a simple, encapsulated and benign proliferation of adipose cells.

This lesion may have been growing uneventfully without interfering with the host's activity. However, upon overlapping the homolateral foramen of Monro, it may have obstructed the passage of cerebrospinal fluid through a valve mechanism. This may have led to the internal hydrocephalus and other changes noted at the postmortem examination, with compression of the vital centers in the brain stem as the terminal event.

Acknowledgment

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