

# Lipomatous Meningioma Associated With Cerebral Vascular Malformation

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A unique epileptic patient with intracranial neoplasm is reported in which a meningioma with lipomatous and osseous components was found associated with cerebral arteriovenous malformation at the same location in the right frontal lobe. The development of the leptomeningeal neoplasm may have been influenced by the underlying vascular anomaly. The cerebral arteriovenous malformation and altered hemodynamics also could have induced the sudden onset of clinical seizures, hemiparesis, and unconsciousness.

**KEY WORDS:** meningioma, lipoma, arteriovenous malformation

## INTRODUCTION

Rare examples of two completely different intracranial neoplasms coexisting in the same locus have been reported [1-3]. Most examples of tumors that are contiguous in the central nervous system (CNS) are cerebral glioma and meningioma [4-6]. The case described here is an unusual lesion of concurrent benign leptomeningeal neoplasm and cerebral arteriovenous malformation found in frontal lobe. The meningioma contained many mesenchymal tissue varieties, including arachnoidal cap cells, fat cells, and osteocytes within the same mass. This complex is possibly explicable on the basis of common origin from ectomesenchymal properties of neural crest [7]. The relationship between leptomeningeal neoplasm and cerebral vascular malformation is also discussed.

## CASE REPORT

A 69-year-old woman was hospitalized because of chest pain, left-sided focal seizures, and left hemiparesis. She had a long history of chronic pulmonary disease, hypertension, and diabetes mellitus. Physical and neurological examination disclosed blood pressure of 160/90 mmHg and, surprisingly, papilledema. Her mental status in hospital rapidly declined and she became unresponsive shortly after admission. Constant clonic jerking of the left leg was noted but there was no spontaneous, purposeful movement of the left extremity, and left Babinski sign was present. Cranial nerves were bilaterally intact.

Routine blood and urine examinations were unremarkable. Electrocardiogram revealed evidence of an old myocardial infarction in the inferior left ventricular wall. Cerebral computerized axial tomograms (CAT) demonstrated a large, low-density mass in the region of the right frontal lobe with surrounding tissue edema (Fig. 1A). Contrast administration increased the staining around the rim of the mass medially and posteriorly (Fig. 1B). The frontal horn of the right lateral ventricle was posteriorly displaced and a moderate right-to-left midline shift affected the lateral ventricles and anterior aspect of the falx. Carotid angiograms confirmed this right frontal mass revealing prominent vascularity beneath the mass.

At craniotomy, encapsulated, friable, yellow tissue was found overlying the superior and middle frontal gyri. It appeared to arise from the region of the arachnoidal villi in the parasagittal region and invaginated the right frontal pole. The lesion was entirely resected after frozen section revealed its benign nature. Prominent vascularity of the underlying cerebral cortex was observed at surgery and that portion of cortical tissue was resected. The patient was well when seen 1 year after craniotomy, although her medical problems persisted. The cerebral CAT revealed no evidence of neoplastic recurrence.

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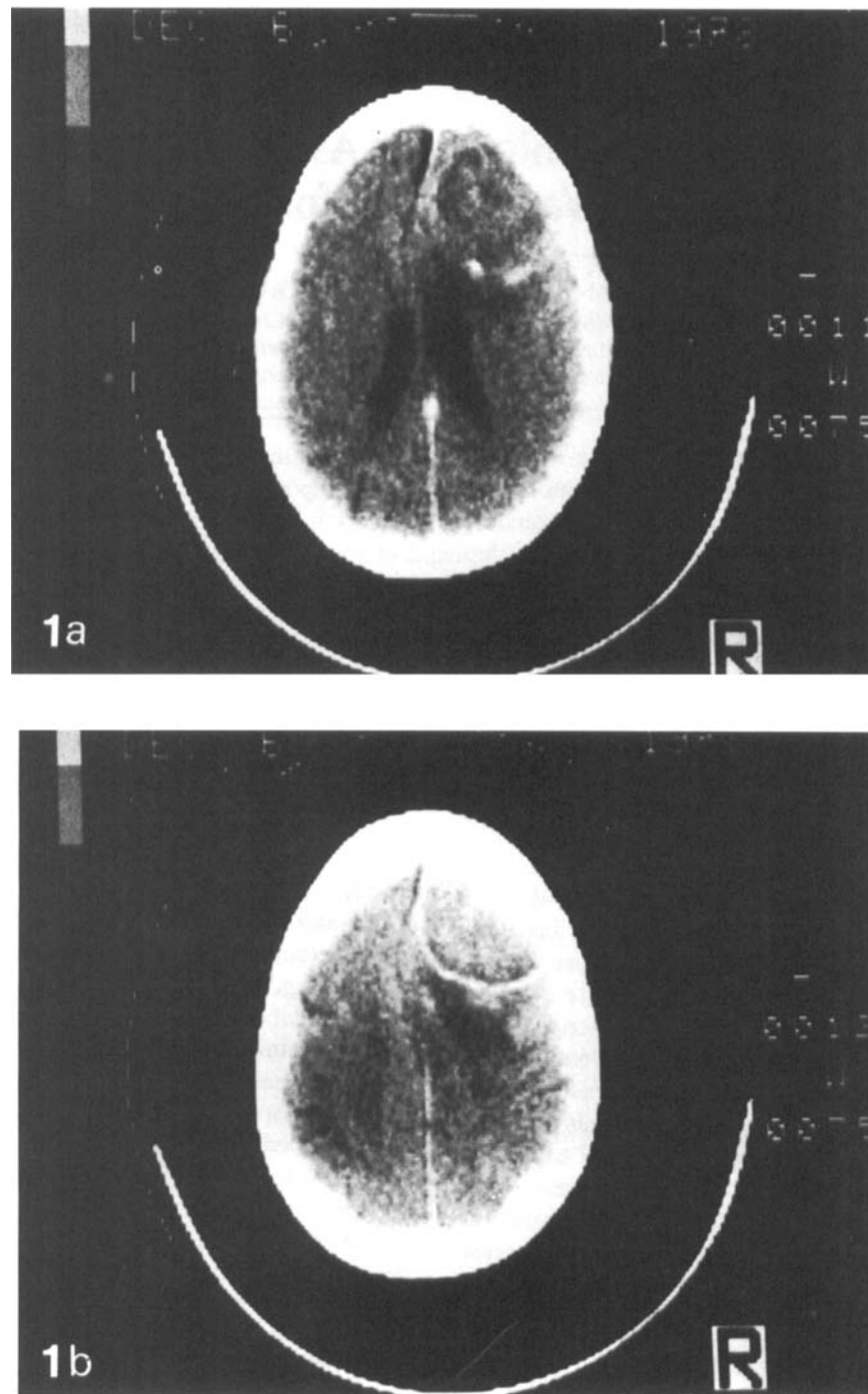


Fig. 1. CAT scans: (a) A well circumscribed mass with focal calcification around the rim of the mass in the region of the right frontal lobe causing distortion of the right lateral ventricle. (b) Perfusion enhancement demonstrating the staining around the lesion.

The specimen consisted of multiple segments of friable tissue weighing 5 gm. Microscopically, the mass was composed of several mesenchymal tissues one of which was well differentiated adipose tissue (Fig. 2). These mature fat cells contained a "hole" in the nucleus ("Lochkern") characteristic of true fatty tissue. Reticular and collagenous fibers interlaced through the fatty tissue.

Focal calcium deposits and bone were also noted. Polygonal cells with ill-defined borders and bland uniform nuclei typical of meningioma were interspersed in the fatty tissue (Fig. 3). Neither mitotic figures nor pleomorphism were seen. Invasion of underlying brain was not found beneath the neoplasm. However, clusters of numerous thick-walled arteries and veins of various cali-

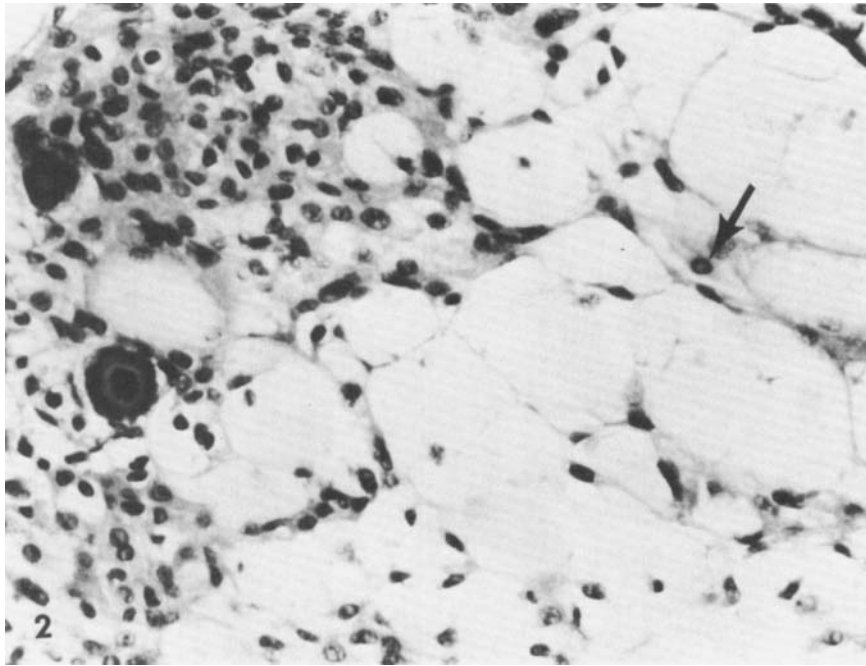


Fig. 2. Lipomatous meningioma showing admixture of meningocytes and adipose tissue cells. Note intranuclear inclusion (arrow). Hematoxylin and eosin ( $\times 250$ ).

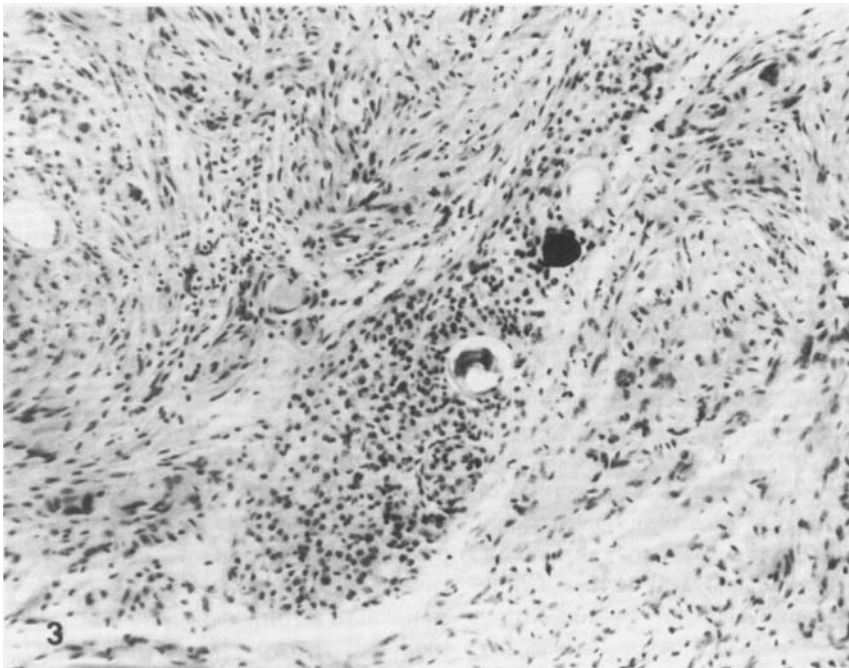


Fig. 3. Focus of meningioma with psammoma bodies. Hematoxylin and eosin ( $\times 120$ ).

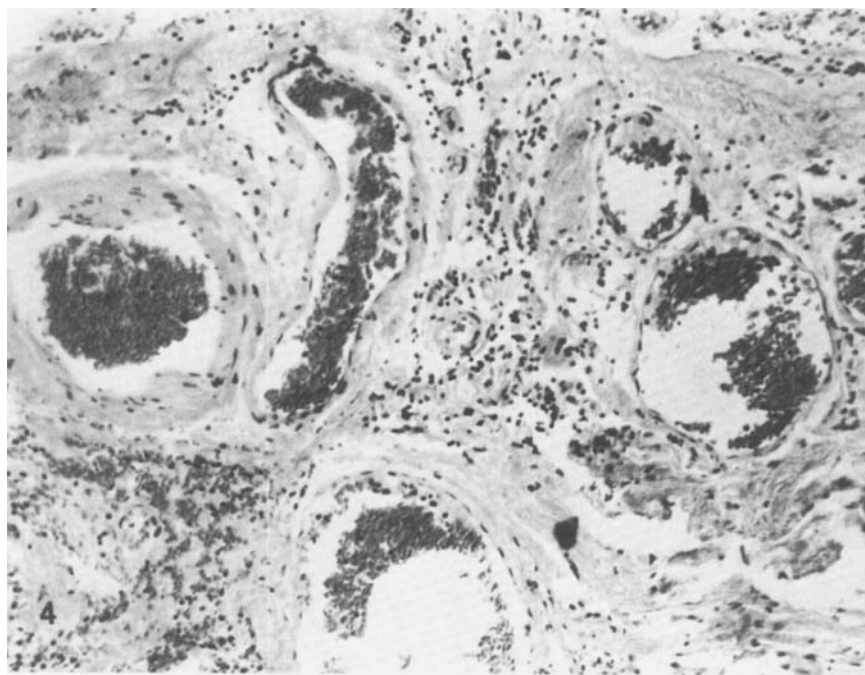


Fig. 4. Clusters of arteries and veins representing arteriovenous malformation in frontal cortex beneath the meningioma. Hematoxylin and eosin ( $\times 48$ ).

bers as well as focal calcification were present in the underlying cerebral cortex producing a typical arteriovenous malformation (Fig. 4). Elastic tissue stains revealed a wavy, elastic lamina in these arteries. Recent hemorrhages and hemosiderin-laden macrophages were also present. There were no meningocytes in any portion of the tissue containing the anomalous vascular channels.

### DISCUSSION

The parasagittal location and attachment of this tumor to the leptomeninges and the syncytial appearance of neoplastic cells in association with psammoma bodies are all diagnostic features of a meningioma. The lesion reported here, however, is unusual in that it contained so much adipose tissue and bone. Additionally, it rested on a separate mass of arteriovenous malformation. Such a lesion is at least uncommon and we are unaware of its previous description.

Intracranial meningioma is, of course, a common neoplasm and accounts for 15% of all primary intracranial neoplasms [8]. Occasionally adipose tissue and bone are present within the leptomeninges as well as in a meningioma [9–10]. Although some confusion existed earlier with regard to various mesenchymal tissue such as fat, cartilage, and bone in meningioma, there seems to be general agreement that such tissue varieties are exemplifying the multipotentiality of their common origin from ectomesenchymal properties of the neural crest. Cells of

the crest may differentiate into many cell types including Schwann cells, meningocytes, and mesenchymal cells [7]. It is, therefore, not surprising to encounter bone and lipomatous tissue in leptomeningeal neoplasm as observed in the present meningioma.

Arteriovenous malformation may comele with other neoplasms, including neurilemmoma and meningioma [11–13]. But in the present instance the arteriovenous malformation was distinctly separated from the overlying leptomeningeal mass. The contiguity of these two different lesions might suggest that the growth of one tumor could have been influenced by the presence of the other, or the two lesions could have a coincidental origin. Cushing and Eisenhardt [14] stated that leptomeningeal tumors may be related to chronic irritation of arachnoid cells by whatever process. Cerebral neoplasm is certainly a source of irritation and hence might also stimulate lesions in the leptomeninges. Gass and van Wagenen [4] described a case of oligodendroglioma in a 56-year-old woman that occurred in relation to an overlying small superficial meningioma. Tanaka et al [6] have published details of a case in which meningioma occurred 23 years after partial removal of an oligodendroglioma at the same site. Kasantikul and Brown [12] have reported two examples of cerebral meningioangiomas in which focal arachnoidal hyperplasia as well as a fully developed independent meningioma have been found in association with underlying cerebral lesions. These data suggest that certain

local cerebral proliferating masses could be factors influencing growth of meningeal neoplasms. Based on the congenital nature of vascular malformation and the fact that the arteriovenous malformation tends to present clinically in younger persons, it is reasonable to suggest that the existence of cerebral angioma in our case could have been present for a lengthy period of time prior to development of the meningeal tumor [8].

The clinical manifestation of sudden onset of focal seizures and hemiparesis in the present case is reasonably related to the anatomic location of the mass. Seizures may be the first indication of meningioma of the convex surface of cerebrum. It is, however, difficult to assume that meningeal tumor alone could produce sudden onset of both seizures and hemiparesis as well as rapid alteration of mental status. The presence of vascular lesion in the underlying cerebrum, on the other hand, could provide a kinetic mechanism for the evocation of such symptoms. Focal rupture of these vascular anomalies may, of course, produce immediate onset of such clinical features. The presence of erythrocytes and hemosiderin pigment in the surrounding tissue indicates that indeed there was recent local cerebral hemorrhage in our case that points to a relation to the arteriovenous malformation.

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