

SELLAR CAVERNOUS HEMANGIOMA WITH INTERNAL CAROTID ARTERY ENCASEMENT

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ABSTRACT

Sellar and parasellar cavernous hemangiomas are rare vascular malformations that often mimic more common lesions such as meningiomas or pituitary macroadenomas. We report a 60-year-old hypertensive male who presented with subacute left-sided weakness, dysphasia, and subtle cranial nerve deficits. MRI showed a large lobulated sellar and parasellar mass with T1 isointensity, marked T2 hyperintensity, centripetal post-contrast filling, and complete encasement of the cavernous segment of the left internal carotid artery, without luminal thrombosis. The lesion was initially treated with neuronavigation-guided pterional craniotomy and partial resection, and the diagnosis of cavernous hemangioma was made retrospectively after correlation with the dynamic enhancement pattern. We compare this case with published series regarding demographics, internal carotid artery encasement, imaging characteristics, and outcomes with surgery versus stereotactic radiosurgery. Accurate preoperative recognition of the characteristic imaging pattern is crucial because it may shift management towards primary radiosurgical treatment, which is associated with high rates of tumor control and lower cranial nerve morbidity.

Keywords: Cavernous sinus hemangioma; sellar mass; internal carotid artery encasement; dynamic contrast-enhanced MRI; stereotactic radiosurgery

Introduction

Sellar and parasellar masses have a broad differential diagnosis that includes pituitary adenomas, meningiomas, craniopharyngiomas, schwannomas, and vascular lesions. Cavernous sinus cavernous hemangiomas (CSCHs) are rare benign vascular malformations that account for only a small fraction of cavernous sinus lesions and show a marked female predominance, with most patients presenting in the fourth to sixth decades of life.^{1,2} These lesions are composed of slow flow dilated vascular channels and are frequently mistaken for more common extra axial tumors because of their vivid contrast enhancement and intimate relationship with the cavernous sinus and internal carotid artery.¹⁻³ On MRI, CSCHs are typically iso or hypointense on T1 weighted images, markedly hyperintense on T2 weighted

images, and demonstrate a characteristic pattern of peripheral nodular enhancement with gradual centripetal filling on dynamic post contrast sequences, which helps distinguish them from meningiomas and pituitary macroadenomas.^{2,3} Diffusion weighted imaging and, in select cases, labeled red blood cell scintigraphy provide additional support for the diagnosis.^{2,4,5}

Historically, large CSCHs were treated with open microsurgical resection, but this approach is often associated with substantial intraoperative blood loss, incomplete excision, and cranial nerve morbidity, especially when the lesion encases the cavernous internal carotid artery.^{5,6} Stereotactic radiosurgery has emerged as a safe and effective alternative that achieves high rates of volume reduction and symptom improvement with

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low permanent morbidity.^{1,7-9}

We report a large sellar and parasellar cavernous hemangioma with complete encasement of the cavernous internal carotid artery in an older hypertensive male. We compare the clinical, radiologic, and therapeutic features of this case with published literature and highlight practical implications for diagnosis and management in a resource limited setting.

Case Presentation

Clinical History

A 60 year old man with long standing hypertension presented with one month of progressive left sided weakness, slurred speech, and dysphasia. Neurological examination revealed left upper motor neuron facial weakness, impaired fine motor movements of the left hand, and subtle cranial nerve deficits. There was no obvious visual field defect or proptosis.

There was no history of head trauma, neurosurgery, chronic sinusitis, or endocrine symptoms. An initial working diagnosis of subacute ischemic stroke with a possible underlying mass lesion was considered.

Imaging Findings

MRI brain with contrast demonstrated a large lobulated sellar and left parasellar lesion measuring approximately 58 x 40 x 44 mm. The mass was isointense to gray matter on T1 weighted images and markedly hyperintense on T2 weighted images.

Post contrast T1 weighted images showed avid heterogeneous enhancement with peripheral nodular enhancement, centripetal filling, and progressive delayed contrast uptake. A dural tail was seen along the adjacent dura.

The lesion completely encased the cavernous segment of the left internal carotid artery and displaced the supraclinoid ICA, while preserving luminal opacification and flow. It abutted the contralateral supraclinoid ICA without evidence of thrombosis or significant stenosis. CT angiography confirmed encasement of the cavernous ICA, involvement of the left cavernous sinus, and preserved arterial opacification.

Additional MRI findings included acute infarcts in the right corona radiata and centrum semiovale, Fazekas grade III chronic microvascular ischemic changes, chronic lacunar infarcts, and multiple supra and infratentorial

microhemorrhages, consistent with hypertensive small vessel disease. Motion artefact partially limited detailed evaluation.

Postoperative MRI demonstrated residual enhancing tissue along the left ICA, blood products in the surgical bed, and a small left parietal subdural collection.

Discussion

Cavernous sinus cavernous hemangiomas (CSCHs) are rare benign vascular malformations of the cavernous sinus microcirculation, accounting for a small proportion of cavernous sinus occupying lesions.¹ In a pooled analysis of 338 published cases, CSCHs represented approximately 2 to 3 percent of all cavernous sinus lesions and showed a marked female predominance, usually presenting in the fourth to sixth decades of life.^{1,2}

Our patient is a 60 year old male, which places him at the older end of the reported spectrum and within the minority male subgroup. Noblett et al. described two male patients with CSCH encasing the cavernous internal carotid artery and highlighted that such cases can present later and pose particular surgical challenges.⁵

The most common presenting symptoms reported in the literature include headache, orbital pain, visual disturbance, and cranial nerve III, IV, V, or VI palsies due to mass effect within the cavernous sinus.^{1,2,5} In Osunronbi et al., cranial nerve palsy and visual symptoms were the dominant complaints.¹

In contrast, our patient presented primarily with dysphasia and hemiparesis, which are better explained by concomitant acute infarcts and severe chronic microvascular ischemic disease rather than direct mass effect from the parasellar lesion. Although CSCH has been reported in patients with other neurological comorbidities, the combination of a large cavernous hemangioma and extensive hypertensive microangiopathy with acute infarction is less frequently emphasised in prior series.^{1,2}

Large and giant CSCHs can extend into the sella, middle cranial fossa, and suprasellar cistern.^{3,6} The lesion in our case measured nearly 6 cm in maximal dimension, comparable to the largest tumors in surgical series. Goel et al. reported 45 surgically treated CSCHs with an average age of 34 years. In that series, encasement

of the cavernous internal carotid artery was common and made complete microsurgical resection difficult. [6] Noblett et al. similarly described lesions that fully encased, but did not significantly narrow, the cavernous ICA, which is a key imaging clue.⁵ Our case reproduces this pattern. The lesion completely encased the cavernous ICA and displaced the supraclinoid ICA, yet luminal opacification was preserved on CT angiography and MR angiography. This configuration is typical of CSCH, which tends to envelop the artery rather than constrict it, and helps distinguish it from meningioma or cavernous sinus thrombosis.^{4,5}

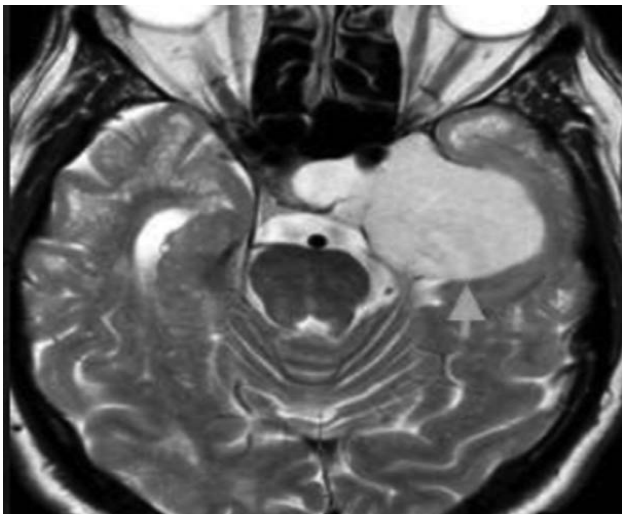


Figure 1a: Axial T2-weighted MRI shows a lobulated hyperintense lesion in the left parasellar region, originating from the cavernous sinus and extending into the middle cranial fossa (gray arrow).

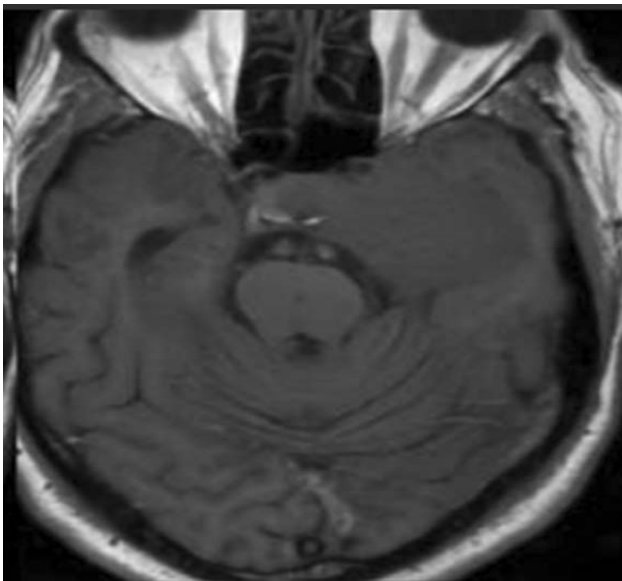


Figure 1b: Axial T1-weighted MRI demonstrates the lesion as hypointense relative to brain parenchyma.

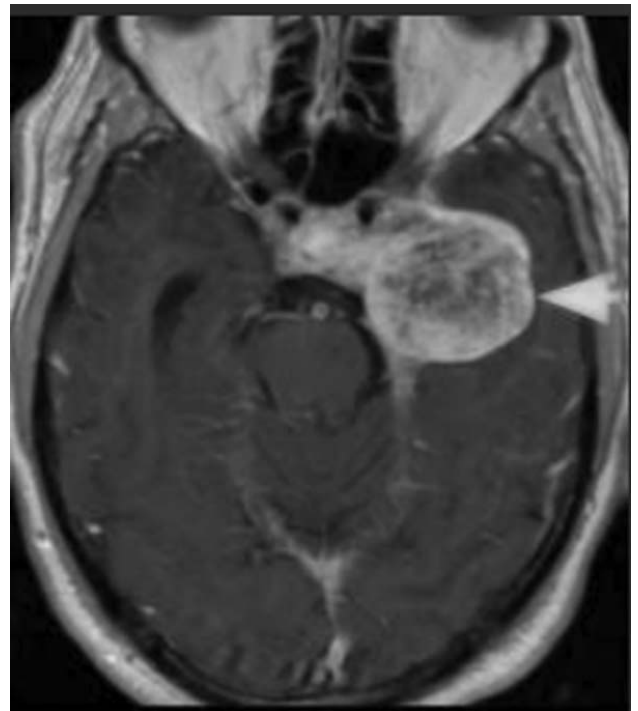


Figure 1c: Post-contrast T1-weighted image shows peripheral nodular enhancement with centripetal filling.



Figure 1d: Delayed post-contrast imaging reveals progressive central enhancement with encasement of the cavernous segment of the left ICA (gray arrow).

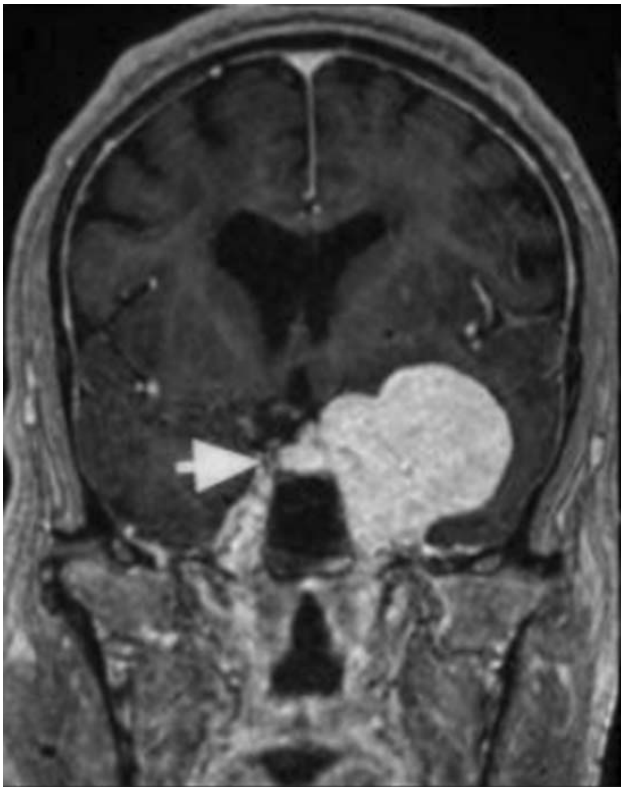


Figure 1e: Coronal post-contrast T1-weighted image demonstrates normal enhancement of the pituitary gland (gray arrow).



Figure 2a: Axial non-contrast CT of the head reveals a hyperdense lesion in the left parasellar region.



Figure 1f: MR angiography depicts superior-medial displacement of the left supraclinoid ICA and MCA due to the lesion.



Figure 2b: Arterial phase CT demonstrates subtle peripheral enhancement.



Figure 2c: Venous phase CT demonstrates progressive nodular peripheral enhancement, characteristic of cavernous hemangioma.

Imaging characteristics and differential diagnosis

Several imaging variables are characteristic of cavernous sinus cavernous hemangiomas:

- **Signal on conventional MRI:** CSCHs are usually iso or hypointense on T1 weighted images and markedly hyperintense on T2 weighted images, often more homogeneous than meningiomas.^{2,3} This pattern was present in our patient.
- **Dynamic contrast enhancement:** Dynamic contrast enhanced MRI often demonstrates peripheral nodular enhancement in the early phase, followed by gradual centripetal filling and intense homogeneous enhancement on delayed images.³ In the series by Jinhu et al., 95 percent of CSCHs showed heterogeneous early enhancement with progressive filling, which proved highly helpful for diagnosis.³ Our case shows the same centripetal delayed enhancement.
- **Diffusion weighted imaging:** Mahajan et al. showed that CSCHs typically demonstrate facilitated diffusion with higher apparent diffusion coefficient values compared with meningiomas and schwannomas, which tend

to be more cellular and show relative restriction.² When available, diffusion imaging can therefore support the diagnosis.

- **Functional imaging:** Labeled red blood cell blood pool scintigraphy can show progressive pooling of tracer within the lesion, which corroborates the vascular nature of CSCH and has been used in diagnostically challenging cases.^{4,5}

Pituitary macroadenomas usually arise from the pituitary gland, remodel rather than envelop the cavernous ICA, and often show more rapid and relatively homogeneous enhancement. Sellar meningiomas may exhibit a dural tail, but they are usually less T2 hyperintense and are more likely to indent or narrow the ICA.²⁻⁴ In our case, a dural tail was present and initially contributed to the impression of meningioma. However, the marked T2 hyperintensity and centripetal delayed enhancement pattern were more typical of cavernous hemangioma and, in retrospect, could have suggested the diagnosis preoperatively.

Historically, large CSCHs were treated with open microsurgical resection. Early series reported substantial intraoperative blood loss and a significant risk of new cranial nerve deficits when attempting gross total resection, particularly in tumors that encased the ICA and surrounded multiple cranial nerves.^{5,6}

In the 45 patient series by Goel et al., radical resection could often be achieved, but with considerable operative complexity and non trivial morbidity.⁶ Noblett et al. emphasized that microsurgical resection of ICA encasing CSCH in male patients was particularly challenging, often requiring careful debulking and still carrying risk of neurological injury.⁵

Our patient underwent neuronavigation guided pterional craniotomy with an attempt at maximal safe resection. Intraoperatively the lesion was highly vascular and soft, and complete excision was not possible because of the intimate relationship with the cavernous ICA. Residual tumor remained along the artery, which is consistent with the difficulties described in the surgical literature for large ICA encasing CSCHs.^{5,6}

Over the last two decades, stereotactic radiosurgery has become the preferred definitive treatment for typical CSCH in many centers.^{1,7-9} Yamamoto et al. reported a seven institute Japanese series of 30 patients treated with Gamma Knife radiosurgery. Tumor volume reduction

was seen in essentially all patients, with substantial shrinkage and improvement or stabilisation of cranial nerve deficits, and very low permanent morbidity.⁸ Lee et al. conducted an international multicenter study of 31 patients and confirmed that Gamma Knife radiosurgery resulted in more than 50 percent volume reduction in all patients at 6 months, with durable long term control and minimal new cranial neuropathies.⁷ Cho et al. studied temporal volume change after Gamma Knife in 26 CSCHs and demonstrated rapid early shrinkage followed by gradual additional reduction, supporting radiosurgery as an effective stand alone treatment even for relatively large lesions.⁹ These radiosurgical outcomes compare favourably with the higher complication rates and incomplete resections reported in surgical series for large CSCHs that encase the ICA.⁶⁻⁹ When the imaging pattern is typical and radiosurgery is available, many authors now recommend stereotactic radiosurgery as primary therapy, reserving open surgery for decompression or diagnostic uncertainty.^{1,7,8}

Learning points in a resource limited setting

This case highlights several important points that are particularly relevant in low and middle income settings:

1. Pattern recognition is critical. Even without advanced perfusion imaging, the combination of marked T2 hyperintensity, centripetal delayed enhancement, and complete ICA encasement without luminal narrowing should strongly suggest CSCH.²⁻⁴

2. Dynamic contrast and diffusion imaging add diagnostic confidence. When available, these sequences can help differentiate CSCH from meningioma or pituitary macroadenoma and may justify avoiding high risk attempts at gross total resection.^{2,3}

3. Management should integrate commodities and local resources. In older hypertensive patients with extensive small vessel disease, the risk of perioperative complications from open surgery is higher. Where Gamma Knife or linear accelerator based radiosurgery is accessible, typical CSCH with ICA encasement is often better managed with primary radiosurgery.⁷⁻⁹

In our patient, the initial diagnostic uncertainty and concern for other pathologies led to a surgical approach.

Retrospective review of the imaging, informed by the literature, suggests that early recognition of the characteristic pattern might have supported a radiosurgical first strategy, potentially reducing surgical risk.

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