

## Bilateral MCA Infarct Following Resection of Giant Olfactory Groove Meningioma: A Rare Complication

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**Abstract:** There are various complications which have been described following excision of giant olfactory groove meningiomas and most of them have some pathophysiological basis and logic explaining their occurrence. Similarly, vascular insult to bilateral anterior cerebral arteries (ACA) could occur during surgery owing to its close proximity to the tumor. However, a 52 year old male who underwent complete resection of giant olfactory groove meningioma developed bilateral hemiparesis (right>left) 14 hours postoperatively following an episode of hypotension and was found to have bilateral MCA infarct on CT scan. Various investigations were done to clarify what must have gone wrong. In this case report, we discuss the possible underlying patho-physiological mechanisms for this peculiar and rare complication and try to find whether it is possible to predict its occurrence in future cases.

**Keywords:** Giant olfactory groove Meningioma, Complications, Bilateral MCA Infarct

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### I. Introduction

There has been tremendous progress in investigations and management of intracranial tumors over past few decades making resection of intracranial tumors relatively safer. Despite these advances, surprising complications do occur which leave us wondering about the possible cause and whether we can ever reliably predict them preoperatively. We present an unusual postoperative complication of bilateral MCA infarct developing after resection of olfactory groove meningioma and would discuss the pathophysiological basis for the same.

### II. Case Report

A 52 year old male presented with complaints of headache and memory disturbances for 6 months duration. On examination, he was conscious and coherent. His visual acuity was finger counting at one metre in left eye and normal in right eye. There were visual field defects in the form of scotomas involving both eyes. Imaging findings were consistent with olfactory groove meningioma of size 8x7x6 cm (336cm<sup>3</sup>) with minimal perilesional edema and intense contrast enhancement (fig 1). Bifrontal craniotomy was performed in supine position with head neutral and slightly extended. Dura was opened horizontally on either side after ligating and dividing the anterior most (juxta crista galli) part of superior sagittal sinus. Brain was moderately tense. Tumor was seen by gently elevating the frontal lobes away from the base of the anterior cranial fossa. Initially its attachment in the region of olfactory groove was coagulated with bipolar diathermy and divided. After intra tumoral decompression while dissecting from the frontal lobes in the gliotic plane, suddenly brain swelling was noticed. Anaesthetist noted bradycardia but there was no hypotension. Injection atropine and mannitol were administered. At the same time, durotomy was extended over the frontal lobes bilaterally and bilateral partial temporal craniectomy was performed. Brain again became lax and started pulsating within 10 minutes. Tumor capsule was excised and simpson grade 2 excision was achieved. No vessel was sacrificed during the procedure. Following tumor resection, bilateral ICA bifurcation, A1, A2 along with anterior communicating artery (ACOM) and bilateral M1 were well visualized. Atherosclerosis of left internal carotid artery was noted. Bilateral vasospastic narrowed ACAs were pushed posteriorly and optic chiasm was pushed posterosuperiorly and towards the right side. At the end of the procedure, brain was pulsating well. Bone flap was replaced and loosely anchored. Blood loss during the procedure was 350ml only.

Postoperatively, after 4 hours, patient was moving all four limbs and was irritable. He was kept on elective ventilation postoperatively. Patient developed hypotension after 7 hours of surgery for which inotropic support was started. After 14 hours, left hemiparesis was noticed and ct scan head showed infarct in bilateral MCA territories (left>right) with mild effacement of both the lateral ventricles but the basal cisterns were preserved. Postoperative changes and pneumocephalus were seen in the frontal polar regions (fig 2). Patient was again taken up for the surgery and bilateral frontotemporal craniectomy along with removal of anterior parietal bone on left side was performed as a preventive measure. Patient continued to be drowsy along with

bilateral hemiparesis. Carotid colour doppler and cardiac 2 d echo were normal. Retrospective analysis of the preoperative MRI scans revealed a doubtful narrowing of proximal segment of left MCA as compared to its distal part (fig 3). Patient was gradually weaned off inotropic support and ventilator and tracheostomy was done on POD (postoperative day) 7. Brain was found to be bulging through the craniectomy defect on POD 40 and repeat CT scan was done which revealed hydrocephalus (fig 4). The CSF analysis was suggestive of meningitis and lumbar drain was placed. Injectable antibiotics were continued based on CSF culture and sensitivity report. cranial CT angiogram done 4 months following surgery revealed non visualisation of initial part of left MCA and filling of distal part by collateral circulation suggesting chronic compression (narrowing) by tumor of left MCA (fig 5). After 4 months of surgery, patient continued to be persistent vegetative state and condition remained same till the report of this case (9 months).

### **III. Discussion**

Olfactory groove meningioma comprises 9.8% of adult meningiomas and usually attains big size by the time they present because of the location of the lesion. Surgery is the treatment of choice. Various postoperative complications mentioned are worsening of mental function due to frontal lobe retraction, anterior cerebral artery injury, visual loss, cerebrospinal fluid leak, infection and postoperative seizures. Postoperative bilateral MCA infarct without overt intra operative vascular insult is a surprising complication and has not been described in the literature.

Although the most logical explanation for such an event is intraoperative vascular insult, however, in that case anterior cerebral arteries should have been affected as they are present in relation to the tumor. Contrary to this, bilateral middle cerebral arteries were involved in present case. In absence of overt vascular damage, this doesn't seem to be the underlying primary cause.

Intraoperatively, atherosclerosis of the left ICA was noted and there was narrowing of initial part of left M1 on MRI. Atherosclerotic plaque in the region of left MCA may have ruptured into the lumen causing obliteration of the artery. The ruptured plaque fragments would have got dislodged and were carried away into opposite circulation by the flow of blood across the ACOA. As they could not go towards the vasospastic narrow A2's, they might have lodged into some of the branches of right MCA. This could explain the bigger infarct size in left MCA territory compared to right.

Another possible explanation for this complication is the development of vasospasm developing following tumor resection. There are random case reports and only a few case series which highlight this point. Most commonly, this phenomenon is described after resection of pituitary adenoma<sup>1</sup> and epidermoid cyst<sup>2</sup>. Less frequently it is reported following resection of other tumours such as meningioma<sup>3</sup>, acoustic neuroma<sup>3</sup>, esthesioneuroblastoma<sup>4</sup>, pilocytic astrocytoma<sup>5</sup>. The exact pathogenesis of this peculiar phenomenon is unclear and appears to be multi factorial. Various hypotheses have been proposed to this. Vasospasm developing after subarachnoid hemorrhage has been shown by Fisher to be related to the amount of blood in the subarachnoid cisterns<sup>6</sup>. Similarly, blood entering into the subarachnoid cisterns during tumor surgery may be the cause of vasospasm. Bejjani et al found out that tumors which are more vascular are more likely to have vasospasm<sup>3</sup>. Other causes proposed by them are manipulation of major vessels of basal cisterns during resection, preoperative encasement of vessel by tumor and vessel narrowing. Narrow vessel leads to decreased vessel reserve in case of hypotension as seen in the present case. Direct mechanical irritation of the smooth muscle cells or the vasa nervosum may be the cause of vasospasm in case of encased arteries. Contrary to this, in present case A1, ACOA and A2 bilaterally were handled but they did not show (except bilateral per operative A2 vasospasm) any signs of insult or infarct.

According to Aoki et al tumor location and surgical approach may contribute to the development of the vasospasm<sup>7</sup>. Similarly, Mohammad Almubaslat et al suggested that blood may collect in a particular subarachnoid cistern or around major vessels depending on specific pattern of intraoperative bleeding<sup>4</sup>. This may be a reason for vasospasm occurring in one particular vessel territory. However in the present case, there was no major blood collection around the ICA bifurcation bilaterally.

Therefore, it is suggested by some authors that in cases where heavy intra operative bleeding was encountered, it is better to obtain a postoperative CT scan to look for subarachnoid blood and to repeat trans cranial doppler in cases where blood is found. Also in cases of unexplained delayed neurological deficit following cranial base tumor surgery, prompt angiography is indicated<sup>3</sup>. Angiography could not be done immediately in present case as patient was found to have established infarct with mass effect in the postoperative ct scan done for the right hemiparesis and emergency decompressive surgery had to be done for the same.

### **IV. Conclusions**

Bilateral MCA infarct developing after resection of giant olfactory groove meningioma surgery is a rare and serious complication with unclear pathophysiologic mechanisms. It may not be entirely possible to

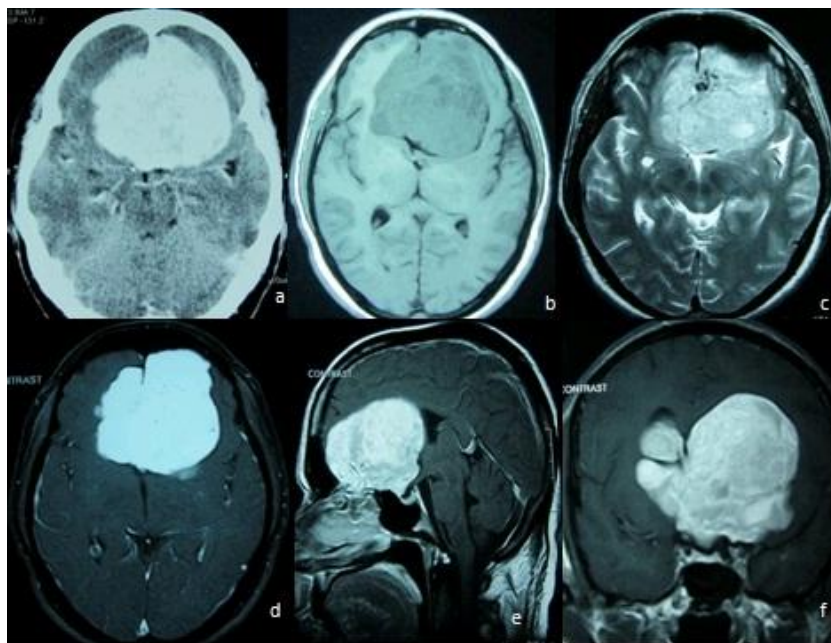
predict its occurrence except sometimes, when angiography has been done preoperatively in high risk cases such as aged patients having atherosclerotic blood vessels and arterial vascular tree.

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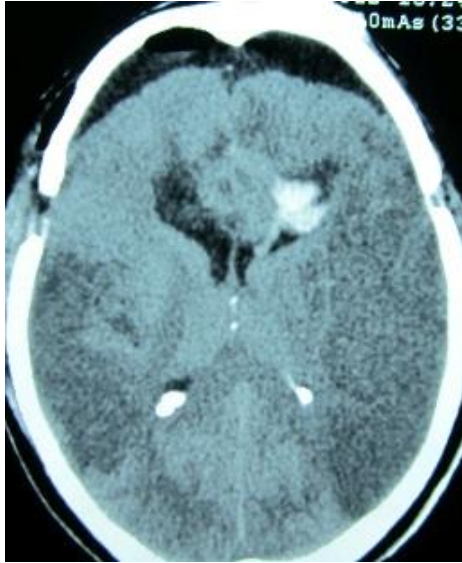
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### Legends

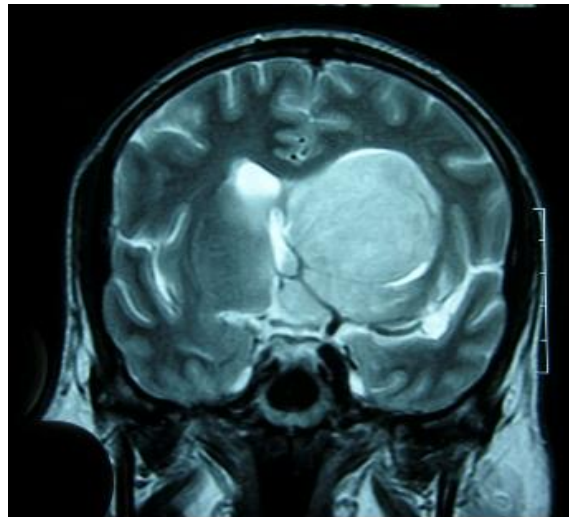
**Figure 1:** CECT Brain (1a) Showing An Intensely Enhancing Extraaxial Lesion Of Size 8x7x6 Cm Present In The Basifrontal Region And Extending Into Suprasellar Area And Causing Mass Effect On Surrounding Brain Structures. MRI Brain Showing The Lesion Was Iso To Hypointense On T1WI (1b), Hyperintense On T2WI (1c) Arising From The Region Of Olfactory Groove More To The Left Side Without Significant Perilesional Edema. It Showed Heterogenous Contrast Enhancement With Evidence Of Dural Tail (1d,E,F).



**Figure 2:** CT Scan Done On Post Operative Day 1 Showed Infarct In Bilateral MCA Territories (Left>Right) With Mild Effacement Of Bilateral Lateral Ventricles. Postoperative Changes And Pneumocephalus Were Seen In The Frontal Polar Region.



**Figure 3:** T2WI Coronal Sections (Preoperative MRI Scan) Showing Doubtful Narrowing Of Proximal Segment Of Left MCA.



**Figure 4:** CT Scan Head Done On POD 40 Showing Communicating Hydrocephalus And Brain Bulging Through The Craniectomy Defect.



**Figure 5:** CT Angiogram Of Brain Reveals Non Visualisation Of Initial Part Of Left MCA And Filling Of Distal Part By Collateral Circulation.

