

Delayed cerebral vasospasm following surgery for craniopharyngioma

Sir,

Vasospasm is commonly seen following subarachnoid bleed due to aneurysmal rupture. Less commonly, it is encountered after head trauma and resection of brain tumors.^[1] We describe a case of delayed symptomatic vasospasm after surgery for craniopharyngioma, which was proved by Computed Tomogram (CT)-Angiography. The possible causes have been discussed and the need to keep this possibility in mind has been reiterated so that therapy can be started early or timely neuroradiological intervention can be undertaken.

A 14-year-old male presented with growth retardation and progressive visual loss for 6 months. He also had features of raised intracranial pressure for 1 month. Radiology revealed a large suprasellar mass with retrosellar extension with speckled calcification. Lesion had both cystic and solid component [Figure 1]. Hormonal work up had shown reduced growth hormones, gonadotropins and modestly raised prolactin. The thyroid hormones and cortisol were normal. After a ventriculo-peritoneal shunt, he was operated through right trans-sylvian route. Gross total excision was achieved and there was inadvertent partial injury to the stalk. The arachnoid plane was followed during surgery and there was no damage to the intracranial vessels including the perforators. As the vessels appeared normal, intraoperatively, papaverine was not instilled.

The patient was conscious, alert in the immediate postoperative period, maintaining a Glasgow Coma Scale (GCS) of 15/15, with no motor deficit, but with the exception of diabetes insipidus that could be managed easily with subcutaneous vasopressin. Immediate postoperative scan showed gross total excision with small amount of operative site hematoma and surrounding subarachnoid hemorrhage [Figure 2 upper row]. On the fifth postoperative day, patient's neurological status deteriorated and he became irritable, with a GCS of 13/15 (E3V4M6). The electrolytes were within normal limits. Meningitis was ruled out by lumbar puncture. The Cerebrospinal fluid (CSF) showed less than 100 cells with CSF sugar two-thirds of blood sugar. The dose of steroids, however, was stepped up (already on perioperative) on a prophylactic basis, keeping a possibility of chemical meningitis. The thyroid level on 6th postoperative day was normal. The serum cortisol levels were not obtained as the patient was already on perioperative steroids. The CT scan, done on 6th postoperative day showed a small infarct in the right caudate nucleus with no other noticeable finding [Figure 2]. The cause of this infarct was suspected to be perforator involvement secondary to surgery, and basal vessel spasm was not suspected. The thyroid hormonal levels were found to be low on 14th postoperative day for which he was started on oral thyroxine. The growth hormone and gonadotropins were not done in the postoperative period and he was administered depot preparations of these, as the preoperative values were low.

On the seventh day, he developed left hemiparesis, grade 2/5 along with persisting drowsiness. Noncontrast CT Head done at this moment revealed evolving left middle cerebral artery (MCA) territory infarct. CT angiography done on day 7 showed focal spasm of the distal Internal carotid artery (ICA), proximal anterior

and middle cerebral arteries [Figure 2]. In retrospect, we realized that the CT Angiogram should have been done on 6th postoperative day when the infarct was small and timely intervention (intraarterial Papaverine or Balloon Angioplasty) could have been done. Besides, early CT

Perfusion was not performed which could have helped us to determine the brain tissue at risk. Patient was started on oral nimodipine (60 mg 4th hourly). Triple H therapy (hemodilution, hypervolemia, and hypertension) was instituted. Repeat CT, 48 hours later showed established infarct corresponding to ICA [Figure 2]. He showed only modest improvement in hemiparesis but continued to remain drowsy. During the Triple 'H' therapy, renal parameters and arterial blood gas (ABG) were monitored frequently as the child was on vasopressin, which increases the risk of renal and pulmonary complications. Unfortunately the transcranial Doppler was not functioning at that time. The therapy was stopped after 4 days as there was minimal clinical improvement and the CT head showed ICA territory infarct. He was discharged 2 weeks later with residual hemiparesis (grade 3/5) and preferred to sleep most of the time.

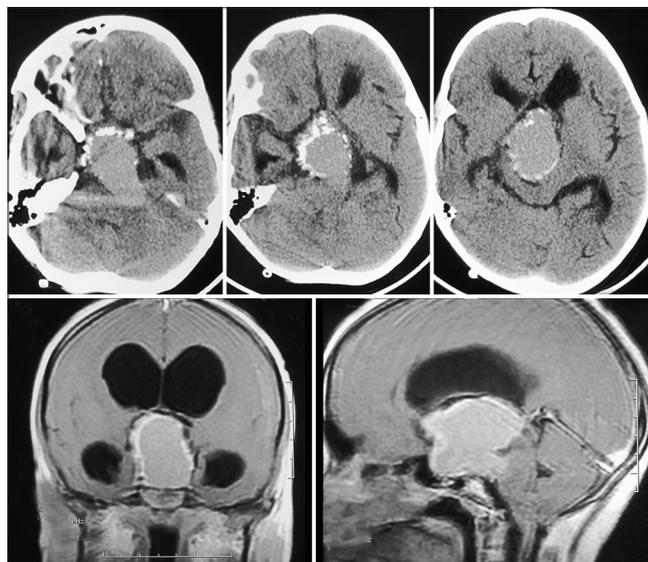


Figure 1: The upper row shows preoperative axial CT images showing speckled calcification with suprasellar and retrosellar extension. The lower row shows coronal and sagittal MRI images showing suprasellar mass

The pathogenesis of vasospasm following skull base tumor resection is speculative.^[2] Subarachnoid blood following tumor resection is considered as the main cause for delayed vasospasm. The delay is usually of about 4-7 days which overlaps with vasospasm of subarachnoid bleed following aneurysmal rupture. The blood degradation products have been implicated for the vasospasm.^[2,3] However, there was small amount

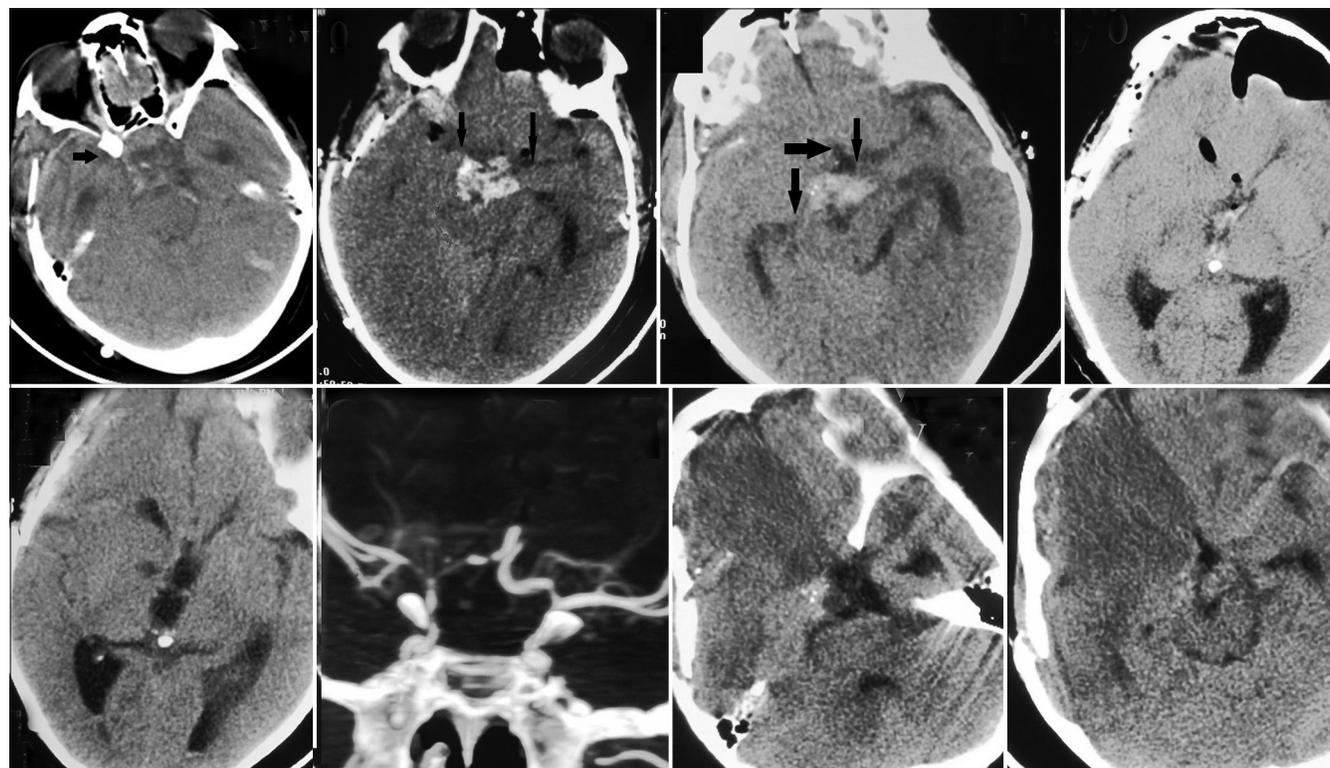


Figure 2: The upper row shows immediate postoperative axial CT images showing gross total excision with some blood at the operative site. There is no subarachnoid blood (black arrows point the carotid cistern, deep sylvian fissure, anterior interhemispheric fissure and ambient cisterns free of blood). Lower row-The day 6-CT image showing small infarct in the right lentiform nucleus. CT angiogram reconstructed image showing spasm of the right distal ICA, A1 and M1 arteries. Day 9-CT images showing well established infarct in the right cerebral hemisphere corresponding to ICA territory

of subarachnoid hemorrhage in our case. Mechanical trauma to vessels has also been thought of as a cause.^[2] However, one would expect it to occur immediately following surgery and not in a delayed fashion. Alternatively, mechanical trauma might contribute to vasospasm by releasing chemical substances from the surrounding tissue that may act in a delayed fashion.^[2]

The third cause may be due to spillage of fluid from craniopharyngioma cyst that also induces chemical meningitis.^[4] Preoperative vasospasm has been observed following spontaneous rupture of craniopharyngioma cyst.^[4] Experiments in rats have shown arterial spasm on 4th day following instillation of craniopharyngioma fluid on femoral vessels.^[5] Apart from this, hypothalamus has been postulated to play a role in vasospasm and median eminence may be involved in this phenomenon. In majority of the reported cases of vasospasm after tumor resection, the tumor was located in suprasellar region probably invading or compressing the hypothalamus. These tumors, perhaps through disinhibition or stimulation of the median eminence may predispose to vasospasm. Other causes could be increased reactivity to noradrenaline, serotonin, or prostaglandins released following surgery along with impairment of endothelial derived relaxant factor may give rise to vasospasm.

The treatment options are not different from subarachnoid hemorrhage (SAH) induced vasospasm and include triple H therapy and intraarterial papaverine.^[2] Triple H therapy, especially the hemodilution is fraught with renal and pulmonary complication and is not preferred nowadays. Balloon angioplasty can be attempted in focal vasospasm. Intraarterial nimodipine or papaverine can be instilled in case of diffuse vasospasm or segments of vessels that are not accessible to balloon angioplasty. The diagnosis should be made as early as possible, before full blown infarct is visible. The surgeon should be aware of such a possibility keeping a high index of suspicion. CT Perfusion study would help reveal the brain tissue at risk, and thereby confirming the suspicion of spasm. Angiography should be done as soon as a new deficit appears after a delay of 4-5 days of surgery, particularly if the CT Perfusion study is positive. Intraoperatively if the vessels are handled, it is preferable to instill papaverine, even if there is no obvious vasospasm. Unfortunately the diagnosis of vasospasm was not picked up early in the index case so as to institute timely hypertensive therapy or balloon angioplasty/intraarterial papaverine. We did not attempt balloon angioplasty in view of already developed infarct.

This report highlights the possibility of delayed symptomatic vasospasm following surgery for

craniopharyngioma, its timely detection and institution of treatment.

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