

Early Cerebral Vasospasm and Cerebral Infarct Following Surgery for Craniopharyngioma - Two Case Reports

Venkatesan Sanjeevi¹, Roopesh Kumar²

Abstract

Background: Delayed Cerebral vasospasm can occur following surgical resection of Craniopharyngioma in sellar and suprasellar subarachnoid cisterns. Early cerebral vasospasm following Craniopharyngioma surgery without subarachnoid hemorrhage is very rare; the pathophysiology is poorly understood and can cause significant post operative morbidity and mortality.

Case description: A 3 years old female presented with recurrent suprasellar, underwent re-exploration and excision of the tumor. She had Diabetes Insipidus post operatively, managed with Tab Minirin and subsequently had neurological deterioration with dense left sided hemiplegia. CT brain showed hypodensity in Right fronto temporo parietal region with mass effect and midline shift, suggestive of cerebral infarction, underwent Right fronto temporo parietal decompressive craniectomy. She improved clinically and neurologically. On two months followup her muscle power improved to 4/5 in both left upper and lower limb, underwent cranioplasty. Another case, a 37 years old male presented with blurring of vision, MRI brain showed sellar suprasellar Craniopharyngioma compressing the optic chiasma. Patient underwent near-total excision, post operatively he had diabetes insipidus, generalized tonic-clonic seizure and dense hemiplegia on right side, MRI brain showed infarct in Left Ganglio capsular region and in the left middle cerebral artery territory and subsequent MRI showed increase in the size of infarct with mass effect and midline shift, an emergency decompressive Craniectomy was performed. During intra-op after durotomy brain was bulging and non-pulsatile. Neurological status did not improved inspite of the surgery and developed severe brain stem dysfunction.

Conclusion: Early cerebral vasospasm and cerebral infarct following Craniopharyngioma surgery is very rare. Early diagnosis, aggressive management of Diabetes Insipidus and vasospasm, avoiding spillage of the cystic components of Craniopharyngioma and minimal handling of tumor part close to the hypothalamus as well as leaving behind the capsule close to the hypothalamus during Craniopharyngioma surgery plays an important role in reducing morbidity and mortality.

Keywords: Craniopharyngioma; Early vasospasm; Cerebral infarct; Diabetes Insipidus.

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Introduction

Delayed Cerebral vasospasm can occur following

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surgical resection of Craniopharyngioma in sellar and suprasellar subarachnoid cisterns. Early vasospasm following Craniopharyngioma surgery without subarachnoid haemorrhage is very rare; the pathophysiology is poorly understood and can cause significant post operative morbidity and mortality. Early diagnosis, aggressive treatment of vasospasm and DI, avoiding spillage of cystic Craniopharyngioma contents and minimal handling of tumor close to hypothalamus will improve the outcome. We are reporting 2 cases of early cerebral vasospasm following Craniopharyngioma surgery.

Case Report 1

A 3 years old female presented with recurrent suprasellar Craniopharyngioma. MRI brain showed solid and cystic component compressing the bilateral optic tract, optic chiasma and brainstem (Figure 1). Patient underwent Re- exploration and excision of tumor, leaving a small tumor below the optic chiasma on right side as it was adherent (Figure 2). Per operative period was uneventful, patient was extubated after surgery and shifted to Intensive care unit for neuro observation. On POD-0 she had Diabetes Insipidus, started on Tab Minirin. On POD 1 she had neurological deterioration with dense left sided hemiplegia. CT brain showed hypodensity in Right fronto temporo parietal region suggestive of cerebral infarction with mass effect and midline shift (Figure 3). An emergency Right fronto temporo parietal decompressive craniectomy was performed and managed in Intensive care unit with pediatric intensivists and endocrinologist. She improved clinically and muscle power on left side improved 3/5 in lower limb and 2/5 in upper limb. On two months followup her muscle power improved to 4/5 in both left upper and lower limb, underwent cranioplasty.

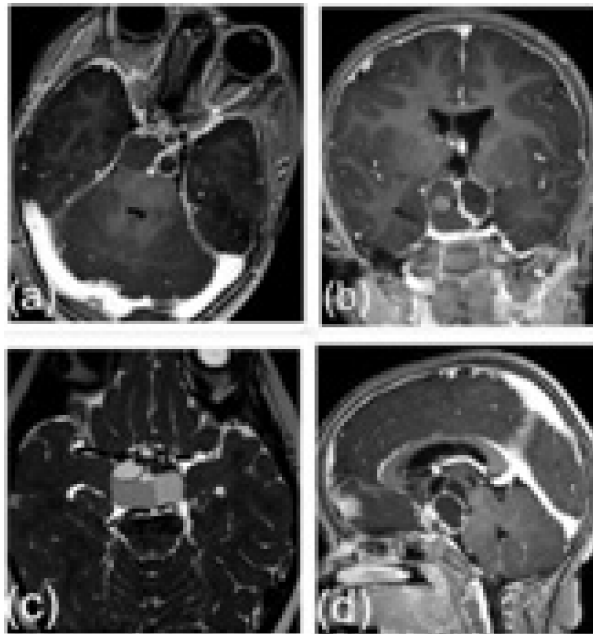


Figure 1: (a) (b) T1WI MRI brain with contrast shows Craniopharyngioma with multiple solid and cystic components (c) T2WI shows Craniopharyngioma adherent to the vessels (d) T1WI MRI brain with contrast sagittal section shows suprasellar Craniopharyngioma compressing the optic chiasma.

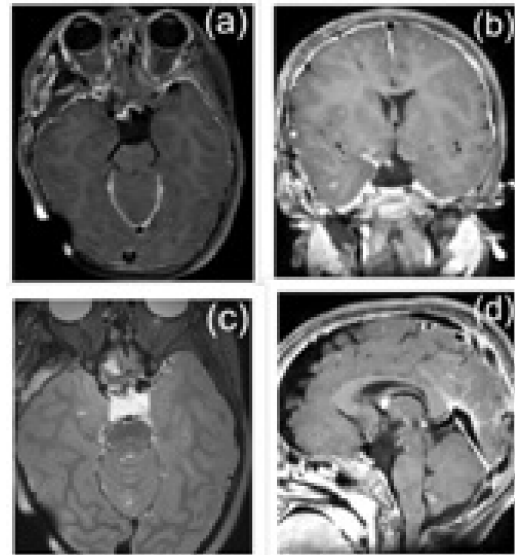


Figure 2: (a) (b) T1WI MRI brain shows near total excision of Craniopharyngioma. (c) T2WI MRI brain shows small residual solid component adherent to right optic chiasma. (d) T1WI MRI brain sagittal section shows optic chiasma freed from the Craniopharyngioma.

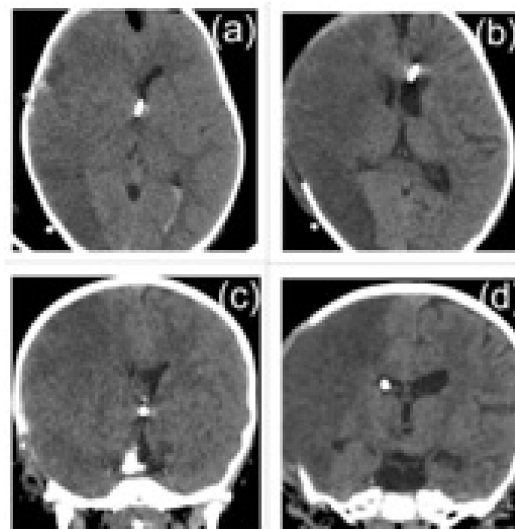


Figure 3: (a) (c) CT brain shows hypodensity seen in right fronto temporo parietal region with mass effect. (b) (d) CT brain shows post right fronto temporo parietal decompressive craniectomy status.

Case Report 2

A 37 years old adult male presented with blurring of vision, MRI brain showed sellar suprasellar Craniopharyngioma compressing the optic chiasma (Figure 4). Patient underwent Right

pterional craniotomy and excision of the tumor. Postoperatively he had diabetes insipidus and was managed with intravascular fluids and vasopressin infusion. On POD - 0 he had generalized tonic-clonic seizure and dense hemiplegia on right side. MRI brain showed infarct in Left Ganglio capsular region and in the left middle cerebral artery territory (figure 5). Patient was started on nor epinephrine infusion to target Mean arterial pressure around 100 and cerebral perfusion pressure around 120. On POD-1 MRI brain was repeated, showed increase in the size of infarct with mass effect and midline shift with uncal herniation. Emergency decompressive craniectomy was performed (Figure 5). During intra-op after durotomy brain was bulging and non-pulsatile. Neurological status of the patient not improved inspite aggressive treatment and developed severe brain stem dysfunction and expired on POD - 4. In this case, vasospasm occurred in a remote vessel, contralateral to the side of surgery.

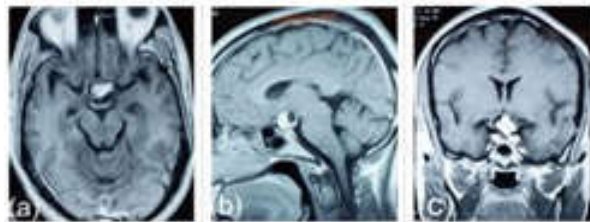


Figure 4: (a) (b) (c) MRI brain shows Craniopharyngioma with solid and small cystic component.

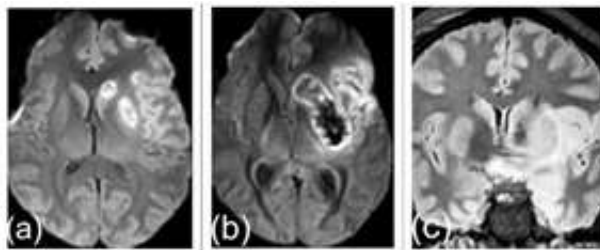


Figure 5: (a) Early post-operative MRI brain shows minimal infarction seen in left ganglio capsular region.

(b) (c) On POD-1 MRI brain shows increase in size of the infarction with mass effect and midline shift.

Discussion

Delayed Cerebral vasospasm following surgical intervention in sellar and suprasellar subarachnoid cisterns is common and can cause significant post operative morbidity and mortality.¹ Early cerebral vasospasm and infarct without SAH is rarely seen in Craniopharyngioma surgery, not reported in literature so far and the pathophysiology is poorly

understood. Various possible causes for vasospasm after surgery includes, direct mechanical damages to arterial walls, deposition of blood in basal cistern, meningitis, hypothalamic damage or release of some chemical substance during tumour removal.^{2,3,4,5} In majority of the reported cases of vasospasm after resection of Craniopharyngioma, the tumor was located in the suprasellar region invading or compressing hypothalamus. Disinhibition or stimulation of the median eminence may cause cerebral vasospasm. Other causes could be related to increased reactivity to noradrenaline; serotonin or prostaglandin release following surgery along with impairment of endothelial derived relaxant factor. In an experimental study by Kamal et al., Craniopharyngioma fluid was shown to cause vasospasm of femoral vessels in rat and its spillage during surgery can be the cause of vascular complications after surgery for Craniopharyngioma.^{6,7,8} In another study Wilson et al, vasospasm was found after injection of hypothalamic extract in a dog model.⁹ In Case 1 in addition to the vasospasm, sudden uncontrolled diabetes insipidus causing severe osmotic dehydration and could have contributed to the cerebral infarction. In Case 2, the pathophysiology could not be understood completely as vasospasm occurred in contralateral side of surgery and also it was a solid tumor without cyst. Aggressive management of vasospasm and DI will decrease the morbidity and mortality. While doing surgery, avoid spillage of the cystic components of Craniopharyngioma and minimal handling of tumor part close to the hypothalamus as well as leaving behind the capsule close to the hypothalamus. Strict input/output chart, serial serum electrolytes monitoring, neurological assessment and intensive care provided by the intensivists and endocrinologist will improve the outcomes after Craniopharyngioma surgery. The diagnosis should be made as early as possible before developing complete infarct. CT brain perfusion study helps in finding tissues at risk of vasospasm, followed by cerebral angiography. Intra operative papaverine and hypertensive therapy can prevent post operative vasospasm. Intra arterial papaverine and balloon angioplasty used for the treatment of proven vasospasm.^{10,11}

Conclusion

Early cerebral vasospasm and cerebral infarct following Craniopharyngioma surgery is very rare, early diagnosis, aggressive management of DI and

vasospasm and team of intensivists, endocrinologist and neurosurgeons plays an important role in reducing morbidity and mortality. Avoid spillage of the cystic components of Craniopharyngioma and minimal handling of tumor part close to the hypothalamus as well as leaving behind the capsule close to the hypothalamus during Craniopharyngioma surgery. To best of our knowledge, our cases are being the first reported early cerebral vasospasm and cerebral infarct following Craniopharyngioma surgery.

Abbreviations and Acronyms

SAH: Subarachnoid hemorrhage

MRI: Magnetic resonance imaging

DI: Diabetes insipidus

POD: Post operative day

CT: Computed tomography

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