

Artery of Percheron and Endoscopic Endonasal Surgery: A Case Report and Review of the Literature

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Abstract

Introduction: Artery of Percheron (AOP) is a rare anatomical variant in which a single perforating artery arising from P1 segment of the posterior cerebral artery supplies paramedian thalami and rostral midbrain. Occlusion of AOP produces bilateral thalamic ischaemia and may be a rare complication in relation to an extended endoscopic endonasal approach. We report the case of a patient who developed AOP damage during endoscopic endonasal surgery; to our knowledge, this complication has been previously reported only in one case, in relation to a second surgery. We also review the anatomical variants in thalamic vascularization and the factors that may be involved in this complication.

Case report: A 52-year-old female underwent an extended endoscopic endonasal approach with intraoperative neurophysiological monitoring. In the postoperative period she presented with decreased level of consciousness and bilateral mydriasis. MRI showed rostral midbrain and paramedian thalami ischaemia congruent with AOP infarction.

Conclusion: Artery of Percheron infarction may be associated with extended endoscopic endonasal surgery when treating lesions with retrosellar extension. Every effort should be made to preserve the small perforating arteries. Intraoperative neurophysiological monitoring of motor and sensory pathways may not detect damage to the artery of Percheron.

Keywords: Artery of percheron; Thalamic infarct; Endoscopic approach; Skull base; Pituitary

Abbreviations: AOP: Artery of Percheron; MRI: Magnetic Resonance Imaging; ICA: Internal Carotid Artery; EES: Endoscopic Endonasal Surgery; IGF-1: Insulin-Like Growth Factor; PCA: Posterior Cerebral Artery; GCS: Glasgow Coma Score; PCOM: Posterior Communicating Artery

Introduction

Endoscopic endonasal approach to the skull base has become increasingly common in recent years, with the consequent decrease in the microscopic transsphenoidal surgery. Postoperative outcomes are good and global morbidity and mortality rates are often lower comparing to transsphenoidal surgery. Endonasal surgery allows for broader exposure when an expanded approach is utilized, although some authors have reported a higher rate of vascular complications when using an extended approach [1-3]. In their case series, Romero et al. [4] reported an arterial injury rate of 0.5% (0.125% for the Internal Carotid Artery [ICA]). Gardner and colleagues [5] found an ICA lesion rate of 0.3%. In the systematic review performed by Chin et al. [5] the ICA lesion rate with this technique range from 0.2 to 1%. We describe the case of a patient who underwent Endoscopic Endonasal Surgery (EES) for an invasive pituitary adenoma, with unexpected damage to the Artery of Percheron (AOP) during the surgery, causing rostral midbrain and bilateral thalamic infarction. This is a very rare complication, with only one previous case reported in the literature, related to a second surgery. Pathophysiology and associated clinical and radiological characteristics are discussed.

Case Report

A 52-year-old female patient presented to the emergency department complaining of long-standing headache associated with diplopia. The patient presented physical features of acromegaly and left sixth cranial nerve palsy. Blood tests showed elevated IGF-1 and prolactin levels. Brain CT scan showed a lesion with suprasellar extension and also to the posterior fossa behind the dorsum sellae and posterior

clinoid processes. Pituitary MRI confirmed the sellar lesion with slight homogeneous contrast enhancement. Suprasellar component produced chiasm and left optic nerve distortion. The lesion also contacted with basilar artery and the P1 segments of both Posterior Cerebral Arteries (PCA), as well as the right cavernous sinus (Figure 1). The

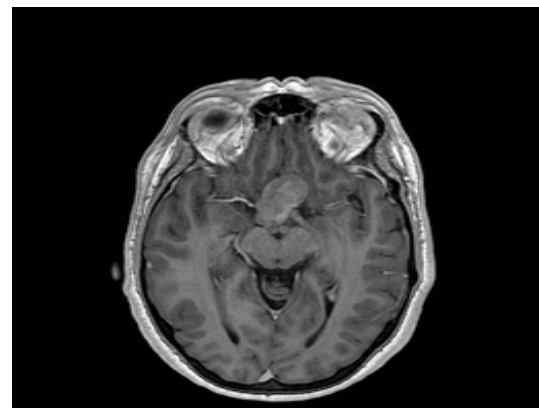


Figure 1: Axial, coronal and sagittal, T1 with gadolinium MRI, showing the tumor with suprasellar and retroclival extensión; Displacement and compression of the chiasm and left optic nerve, as well as contact with basilar artery and P1 segments are observed.

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patient underwent an expanded endoscopic endonasal approach with intraoperative neurophysiological monitoring. The clivus, sella turcica, tuberculum sellae, and part of the sphenoid planum were drilled. The sellar component and the suprasellar portion were resected. Curved ring curettes were used to access and resect the retroclival region and the area posterior to the dorsum sellae, although excision was probably subtotal due to the lack of direct visualization. There were no incidents during the surgery.

Motor and somatosensory evoked potentials remained stable at all times. Intraoperative bleeding was minimal and hemodynamic stability was maintained throughout the operation, with systolic blood pressure between 90 and 120 mmHg during the surgery. Non-reactive bilateral mydriasis was evidenced after completion of surgery. Brain CT scan revealed subarachnoid haemorrhage in perimesencephalic cisterns (Figure 2). No hydrocephalus was observed. Sedation was slowly withdrawn. The patient presented minimal left brachial flexion, preserved corneal reflexes with persistence of nonreactive mydriasis. Given the patient's neurological status, we decided to maintain sedation with intracranial pressure monitoring using a parenchymal sensor.

An MRI showed signal restriction in DWI/ADC sequences in rostral midbrain and paramedian thalami, suggesting acute ischaemia congruent with AOP infarction (Figure 3). MR angiography showed normal flow signal and normal morphology of circle of Willis and

main intracranial arteries. The right posterior communicating artery was visible and prominent. After withdrawal of sedation, the patient presented a Glasgow Coma Scale of 7 points, with bilateral third cranial nerve palsy. Tracheostomy and percutaneous gastrostomy were performed, and the patient was transferred to an intensive rehabilitation centre. One year after surgery, the patient presented the following neurological sequelae: Third cranial nerve palsy requiring blepharoplasty, spastic tetraparesis and neuropsychological disorders with impulsivity and emotional lability.

Discussion

Artery of Percheron is an unusual anatomical variant [6-10] in which a single perforating artery of the P1 segment of the PCA supplies blood to medial thalami and rostral midbrain bilaterally. It was first described by the French neurologist Gerard Percheron in 1973. Thalamus and midbrain vascularization is highly complex, with arterial afferents arising from the PCA and the Posterior Communicating Artery (PCOM) through perforating branches. Vascular supply to the thalami can be divided into four territories: Anterior, paramedian, inferolateral and posterior. Thalamic paramedian arteries are also responsible for vascularization to some regions of the brainstem, including interpeduncular nucleus, decussation of the superior cerebellar peduncles, medial red nucleus, nuclei of third and fourth cranial nerves and the anterior periaqueductal gray matter. Given this distribution, involvement of AOP can cause bilateral paramedian thalamic ischaemia, with or without midbrain ischaemia [11-14].

Clinical presentation of AOP occlusion is variable due to interindividual differences in thalamus anatomy and function, as well as to variability in its vascularization. However, the presentation mainly includes vertical oculomotor palsy, memory involvement and alterations in level of consciousness. If the entire midbrain is affected, this leads to mesencephalothalamic or thalamus-peduncular syndrome, which can include other oculomotor disorders and involvement of long motor pathways [15-20]. AOP infarction can be diagnosed by brain MRI with visualization of the infarction at initial stages, showing hypersignal in medial thalamus bilaterally, with or without mesencephalic involvement. The optimal sequence for early detection is DWI. By contrast, the AOP is rarely visualized by conventional arteriography due to its small size.

Based on a study of 37 patients, Lazzaro et al. [10] identified four ischemic patterns of AOP infarction. In that study, 43% of patients presented bilateral paramedian thalamic involvement with midbrain, 38% presented bilateral paramedian thalamic involvement without midbrain, 14% bilateral paramedian thalamic with anterior thalamus and midbrain, and 5% showed bilateral paramedian thalamic involvement with anterior thalamus without midbrain. Aryan et al. [3] reported the other case of AOP infarction after EES. Those authors described the case of a patient who underwent a second surgery for pituitary adenoma with suprasellar extension and chiasmatic compression. During the operation, they observed an episode of hypertension and bradycardia that resolved spontaneously. They also observed minor bleeding from the posterior part of the tumour capsule. The patient awoke with a low level of consciousness and bilateral ptosis. On brain CT, they observed subarachnoid haemorrhage in basal cisterns and the MRI showed thalamic infarction. Postoperatively, the patient showed progressive improvement. At 6 weeks, she obeyed orders, but still presented hemiparesis.

We agree with the mechanisms proposed by Aryan and colleagues to explain bilateral thalamic infarction. We believe that AOP

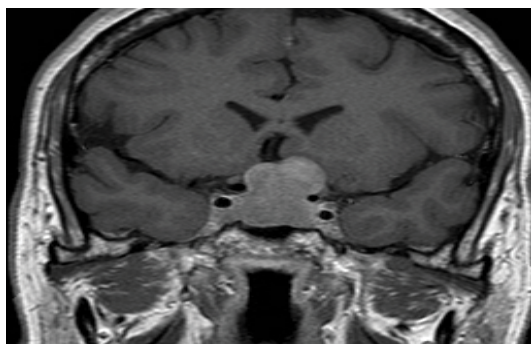


Figure 2: Axial CT scan, showing post-operative subarachnoid hemorrhage in the basal and inter-peduncular cisterns.

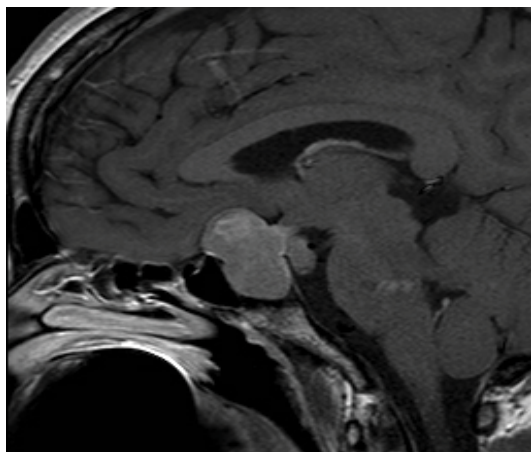


Figure 3: Post-operative axial DWI, FLAIR and T2, MRI; Hypersignal is observed in the bilateral medial thalamus and rostral midbrain, indicative of ischemia.

involvement may occur for two reasons in the context of an endonasal endoscopic approach with expansion to the sellar region, as in our case. The first and most likely cause is involvement of the AOP in the region of the basal cisterns during resection. In our case, visualization of the area without resection of the sellar dorsum was limited. As a result, the use of ring curettes without precise visual control could have resulted in inadvertent damage to the AOP. However, due to the small size of this artery, the amount of bleeding in our patient was unremarkable. In addition, in this case, intraoperative neurophysiological monitoring did not warn us of the presence of a parenchymal lesion. The second potential cause of the infarction could have been vascular irritation with vasospasm secondary to postoperative subarachnoid haemorrhage. We papaverine during surgery and systolic blood pressures were maintained throughout between 90 and 120 mmHg to ensure sufficient irrigation.

Based on our experience, we believe that the following factors should be considered when attempting to treat suprasellar and retrosellar lesion by endonasal approach. First, if the tumour cannot be adequately visualized, then resection should not be attempted, even in the case of soft tumors that would otherwise be considered easily resectable. Second, ring curettes may inadvertently damage small caliber vessels. Thus, these instruments should not be used without optimal visual control. Third, in case of retrosellar extension, dorsum sellae resection may be considered with dislocation of the pituitary gland (if the gland is intact). Finally, intraoperative neurophysiological monitoring may not detect thalamic lesions.

Conclusion

The present report describes a case of artery of Percheron infarction associated with extended endoscopic endonasal surgery. Pituitary adenomas or other lesions with retrosellar extension may involve small perforating arteries of the thalamus and brainstem; for this reason, every effort should be made to try to preserve these vascular structures during surgery. Direct visualization can prevent damage to small perforating arteries. However, it is important to keep in mind that intraoperative neurophysiological monitoring of motor and sensory pathways may not detect intraoperative damage to this small vessels.

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