

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

Abstract

Artery of Percheron (AOP) is a rare anatomical variant in which a single perforating artery arising from the P1 segment of the posterior cerebral artery supplies paramedian thalamic and rostral midbrain. The occlusion of AOP produces bilateral thalamic ischemia 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 (EES); 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. A 52-year-old female underwent an extended endoscopic endonasal approach with intraoperative neurophysiological monitoring. In the postoperative period, she presented with a decreased level of consciousness and bilateral mydriasis. Magnetic resonance imaging showed rostral midbrain and paramedian thalamic ischemia congruent with AOP infarction. AOP infarction may be associated with extended EES when treating lesions with retrosellar extension. Every effort should be made to preserve the small perforating arteries. Intraoperative neurophysiological monitoring of the motor and sensory pathways may not detect damage to the AOP.

Keywords: Artery of Percheron, endoscopic approach, pituitary, skull base, thalamic infarct

Introduction

The 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.^[1,2]

In their case series, Romero *et al.*^[3] reported an arterial injury rate of 0.5% (0.125% for the internal carotid artery [ICA]). Gardner *et al.*^[4] found an ICA lesion rate of 0.3%. In the systematic review performed by Chin *et al.*,^[2] 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.^[3] Pathophysiology and associated clinical and radiological characteristics are discussed.

For this research, no funding was received.

All authors certify that they have no financial interest or nonfinancial interest in the subject matter or materials discussed in this manuscript. An Institutional Review Board (IRB) has approved the present study.

Case Report

A 52-year-old female patient presented to the emergency department complaining of long-standing headache associated with diplopia. The patient presented the physical features of acromegaly and left sixth cranial nerve palsy. Blood tests showed elevated insulin-like growth factor-1 and prolactin levels.

How to cite this article: Pereira MR, Hernández FM, Cortés CA. Artery of percheron and endoscopic endonasal surgery: Case report and review of the literature. *Asian J Neurosurg* 2020;15:777-80.

Submitted: 23-Dec-2019

Revised: 05-Feb-2020

Accepted: 25-Feb-2020

Published: 28-Aug-2020

**Marta Rico Pereira,
Fernando Muñoz
Hernández,
Carlos Asencio
Cortés**

*Department of Neurosurgery,
Sant Pau Hospital, Barcelona,
Spain*

Address for correspondence:

*Marta Rico Pereira, MD,
Department of Neurosurgery,
Sant Pau Hospital, Barcelona,
Spain.
E-mail: mricop@santpau.cat*

Access this article online

Website: www.asianjns.org

DOI: 10.4103/ajns.AJNS_370_19

Quick Response Code:



This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: WKHLRPMedknow_reprints@wolterskluwer.com

Brain computed tomography (CT) scan showed a lesion with suprasellar extension and also to the posterior fossa behind the dorsum sellae and posterior clinoid processes. Pituitary magnetic resonance imaging (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 1a-c].

The 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.

Nonreactive bilateral mydriasis was evidenced after the completion of surgery. Brain CT scan revealed subarachnoid hemorrhage in perimesencephalic cisterns [Figure 2]. No hydrocephalus was observed. Sedation was slowly withdrawn. The patient presented minimal left brachial flexion and 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 diffusion-weighted imaging (DWI)/apparent diffusion coefficient sequences in rostral midbrain and paramedian thalami, suggesting acute ischemia congruent with AOP infarction [Figure 3a-c]. MR angiography showed normal flow signal and normal morphology of circle of Willis and main intracranial arteries. The right posterior communicating artery (PCOM) was visible and prominent.

After the 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 center.

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

AOP is an unusual anatomical variant^[5,6] in which a single perforating artery of the P1 segment of the PCA supplies

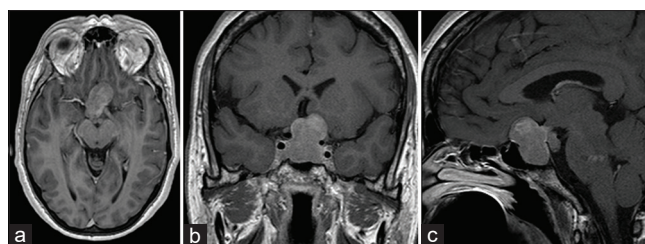


Figure 1: Axial (a), coronal (b), and sagittal (c) T1 with gadolinium magnetic resonance imaging, showing the tumor with suprasellar and retroclival extension. Displacement and compression of the chiasm and left optic nerve, as well as contact with basilar artery and P1 segments are observed



Figure 2: Axial computed tomography scan showing postoperative subarachnoid hemorrhage in the basal and inter-peduncular cisterns

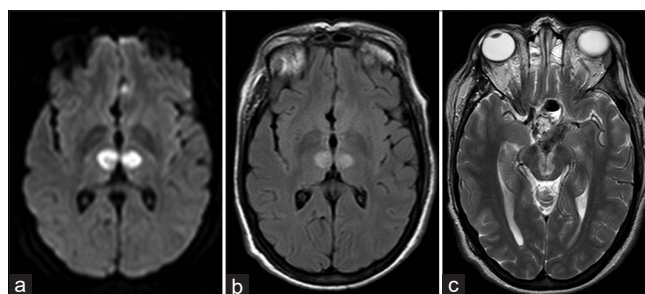


Figure 3: Postoperative axial diffusion-weighted imaging (a), fluid attenuated inversion recovery (FLAIR) (b) and T2 (c) magnetic resonance imaging. Hypersignal is observed in the bilateral medial thalamus and rostral midbrain, indicative of ischemia

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 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, the involvement of AOP can cause

bilateral paramedian thalamic ischemia, with or without midbrain ischemia.

Clinical presentation of AOP occlusion^[7-19] is variable due to interindividual differences in thalamus anatomy and function, as well as to the variability in its vascularization. However, the presentation mainly includes vertical oculomotor palsy, memory involvement, and alterations in the level of consciousness. If the entire midbrain is affected, this leads to mesencephalothalamic or thalamopeduncular syndrome, which can include other oculomotor disorders and the involvement of long motor pathways.^[9,11-13]

AOP infarction can be diagnosed by brain MRI^[6,14,15,20] 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.*^[6] identified four ischemic patterns of AOP infarction. In their 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.*^[16] 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 tumor capsule. The patient awoke with a low level of consciousness and bilateral ptosis. On brain CT, they observed subarachnoid hemorrhage 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 *et al.* to explain bilateral thalamic infarction. We believe that AOP 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 the 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 hemorrhage. 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 the endonasal approach.

First, if the tumor 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 the 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 AOP infarction associated with extended EES. 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 these small vessels.

Ethics approval

An IRB has approved the present study.

Patient consent

The patient has consented to the submission of the case report for submission to the journal.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Ammirati M, Wei L, Ciric I. Short-term outcome of endoscopic versus microscopic pituitary adenoma surgery: A systematic review and meta-analysis. *J Neurol Neurosurg Psychiatry* 2013;84:843-9.
2. Chin OY, Ghosh R, Fang CH, Baredes S, Liu JK, Eloy JA. Internal carotid artery injury in endoscopic endonasal surgery: A systematic review. *Laryngoscope* 2016;126:582-90.
3. Romero AD, Lal Gangadharan J, Bander ED, Gobin YP, Anand VK, Schwartz TH. Managing arterial injury in endoscopic skull base surgery: Case series and review of the literature. *Oper Neurosurg (Hagerstown)* 2017;13:138-49.

4. Rowan NR, Turner MT, Valappil B, Fernandez-Miranda JC, Wang EW, Gardner PA, *et al.* Injury of the carotid artery during endoscopic endonasal surgery: Surveys of skull base surgeons. *J Neurol Surg B Skull Base* 2018;79:302-8.
5. Garcia-Grimshaw MA, Peschard-Franco M, Gutierrez-Manjarrez F. Bilateral thalamic ischemic stroke second. *Coreus* 2018;10:E2676.
6. Lazzaro NA, Wright B, Castillo M, Fischbein NJ, Glastonbury CM, Hildenbrand PG, *et al.* Artery of Percheron infarction: Imaging patterns and clinical spectrum. *AJNR Am J Neuroradiol* 2010;31:1283-9.
7. Sankar T, Souster J, Steinke DE. Bilateral thalamic infarction following transsphenoidal surgery. *Can J Neurol Sci* 2008;35:522-5.
8. Thurtell MJ, Halmagyi GM. Complete ophthalmoplegia: An unusual sign of bilateral paramedian midbrain-thalamic infarction. *Stroke* 2008;39:1355-7.
9. Raut TP, Baheti G, Hinduja A, Makhija P, Ansari K, Sharma V, *et al.* Sudden onset altered sensorium: Artery of Percheron infarct. *J Neurol Neurosci* 2017;8:212.
10. Kumral E, Evyapan D, Balkir K, Kutluhan S. Bilateral thalamic infarction. Clinical, etiological and MRI correlates. *Acta Neurol Scand* 2001;103:35-42.
11. Reilly M, Connolly S, Stack J, Martin EA, Hutchinson M. Bilateral paramedian thalamic infarction: A distinct but poorly recognized stroke syndrome. *Q J Med* 1992;82:63-70.
12. Rangel-Castilla L, Gasco J, Thompson B, Salinas P. Bilateral paramedian thalamic and mesencephalic infarcts after basilar tip aneurysm coiling: Role of the artery of Percheron. *Neurocirugia (Astur)* 2009;20:288-93.
13. Roitberg BZ, Tuccar E, Alp MS. Bilateral paramedian thalamic infarct in the presence of an unpaired thalamic perforating artery. *Acta Neurochir (Wien)* 2002;144:301-4.
14. Matheus MG, Castillo M. Imaging of acute bilateral paramedian thalamic and mesencephalic infarcts. *AJNR Am J Neuroradiol* 2003;24:2005-8.
15. Graff-Radford NR, Damasio H, Yamada T, Eslinger PJ, Damasio AR. Nonhaemorrhagic thalamic infarction. Clinical, neuropsychological and electrophysiological findings in four anatomical groups defined by computerized tomography. *Brain* 1985;108(Pt 2):485-516.
16. Aryan S, Thakar S, Hegde AS. Artery of Percheron infarction after endoscopic pituitary surgery. *Acta Neurochir (Wien)* 2016;158:1973-5.
17. Arauz A, Patiño-Rodríguez HM, Vargas-González JC, Arguelles-Morales N, Silos H, Ruiz-Franco A, *et al.* Clinical spectrum of artery of Percheron infarct: Clinical-radiological correlations. *J Stroke Cerebrovasc Dis* 2014;23:1083-8.
18. Khanni JL, Casale JA, Koek AY, Espinosa Del Pozo PH, Espinosa PS. Artery of Percheron infarct: An acute diagnostic challenge with a spectrum of clinical presentations. *Cureus* 2018;10:e3276.
19. Lepore FE, Gulli V, Miller DC. Neuro-ophthalmological findings with neuropathological correlation in bilateral thalamic-mesencephalic infarction. *J Clin Neuroophthalmol* 1985;5:224-8.
20. Raphaeli G, Liberman A, Gomori JM, Steiner I. Acute bilateral paramedian thalamic infarcts after occlusion of the artery of Percheron. *Neurology* 2006;66:E7.