


Intracranial Intradural Vascular Injury during Endoscopic Endonasal Transsphenoidal Surgery: A Case Report and Literature Review

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Abstract

Background Transsphenoidal surgery (TSS) is a procedure for sellar or midline masses in the skull base. Among the reported complications are iatrogenic vascular injuries; that are rare, yet they carry devastating outcomes, with an incidence of injury between 0.34 and 2.6%. The cavernous internal carotid artery is the most commonly injured. However, intradural arterial injuries are much less reported with challenging management. We report a rare incident of intradural arterial injury during TSS, and we compared our management to the summarized few cases reported in the literature.

Case Report We report a 43-year-old female who had a recurrent planum sphenoidal meningioma. She underwent trans-nasal transsphenoidal endoscopic resection that was complicated with intraoperative bleeding due to an injury to the anterior communicating artery that was challenging to control, resulted in a bilateral loss of flow in A1 segments of anterior cerebral artery and required endovascular management. The patient had a good recovery postoperatively without the typical picture of ACA syndrome.

Conclusion Intradural arterial injury is exceedingly rare in TSS, with no clear standard of care for the management. Collateral blood supply allows definitive management with minimal morbidity. Identifying the risk factors beforehand, as well as performing such cases in a well-resourced center, are crucial elements of safety.

Keywords

- ▶ vascular injury
- ▶ endoscopic endonasal approach
- ▶ endovascular
- ▶ intradural
- ▶ complication

Introduction

Transsphenoidal surgery (TSS) has become widely accepted as the preferred approach not only for pituitary adenomas but also for other sellar or midline masses in the skull base.^{1–5} Complications of this approach are well documented, including cerebrospinal fluid (CSF) leak, diabetes insipidus, and infection.^{6–9} Among which are vascular complications that are rare and yet can lead to a fatal sequel, with an incidence of

injury ranging between 0.34 and 2.6%.^{10–13} The cavernous part of the internal carotid artery (ICA) is found to be the most commonly injured artery in TSS.^{12,13} However, intradural arterial injury is exceedingly rare, which makes the management difficult.¹⁴ Intradural vascular injuries that have been reported during TSS include posterior cerebral artery (PCA), posterior communicating (P-comm) artery, ophthalmic artery, anterior communicating (A-comm) artery, and anterior cerebral artery (ACA).^{11,15–20} Also, indirect vascular complications

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(embolus and stroke) in TSS are reported.^{10,13} In this paper, we report a case that had an injury to the ACA, resulting in bilateral ACA occlusion during endoscopic TSS for planum sphenoidale meningioma and will discuss these injuries and their management and outcomes.

Case Report

A 43-year-old female presented with a 1-year history of severe progressive decline in visual acuity. She underwent a left frontal craniotomy 8 years prior, for a subtotal resection of a large planum sphenoidal meningioma World Health Organization grade I (►Fig. 1A–D). Neurological examination revealed cranial nerve functions within normal, apart from marked bilateral decreased visual acuity. Her motor, sensory, and coordination examination were unremarkable. The ophthalmologic assessment revealed significant impairment of visual

acuity with only preserved light perception bilaterally; light reflexes and extraocular muscles were within normal.

Computed tomography (CT) of the brain showed left post-surgical frontal encephalomalacia with recurrent meningioma; there is marked underlying hyperostosis causing bilateral narrowing of optic canals (►Fig. 1E). Magnetic resonance imaging (MRI) scan demonstrated a recurrent enhancing mass measuring 5.4×4 cm extending posteriorly into pituitary fossa and sphenoid and ethmoid sinuses inferiorly, with marked optic nerves and chiasm compression; there is marked increase in hyperostosis compared with her first MRI scan. The recurrent tumor enhanced homogeneously, as well as part of the hyperostosis, following intravenous contrast administration, and the ACA, A-comm complex was adherent to the tumor (►Fig. 1F–J).

The patient underwent endoscopic endonasal trans-sphenoidal surgery with navigation guidance. Surgery started

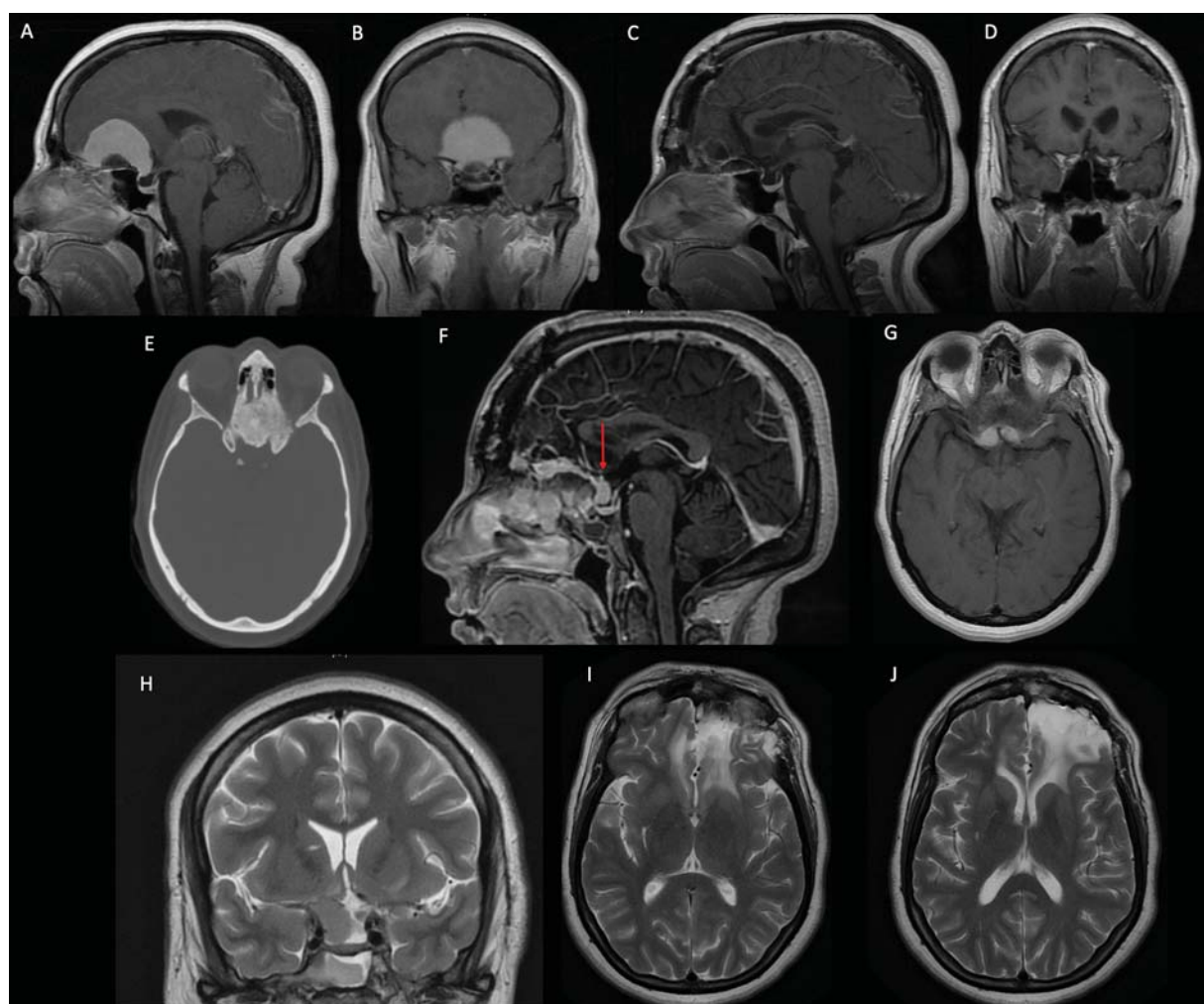


Fig. 1 (A, B) Axial and sagittal views of the brain MRI images (T1 with contrast) at first presentation 8 years ago. (C, D) Sagittal and coronal views of the brain MRI images (T1 with contrast) after first craniotomy performed 8 years ago. (E) Axial bone window computed tomography of the brain demonstrating hyperostosis of sphenoid and ethmoid bones, causing narrowing of both optic canals. (F, G) Sagittal and axial views of the brain MRI images (T1 with contrast) at current presentation demonstrating recurrent meningioma along planum sphenoidale with focal extension into the upper anterior portion of pituitary fossa. Note the proximity of the ACA-ACOM junction to the posterior border of the tumor (red arrow in sagittal view). Note the associated encephalomalacia, mainly involving the left frontal lobe from the previous craniotomy. (H, I, J) Coronal and axial views of the brain MRI images (T2) demonstrating the relation of the lesion to the right ICA-ACA arteries and the preexisting preoperative encephalomalacia, mainly involving the left frontal lobe. ACA, anterior cerebral artery; ACOM, anterior communicating artery; ICA, internal carotid artery; MRI, magnetic resonance imaging.

with bilateral maxillary antrostomy, ethmoidectomy, sphenoidotomy, and nasoseptal flap elevation. There was part of the soft tumor in the ethmoid sinuses which was removed, bilateral optic nerve bone decompression was performed, and the hyperostotic skull base was drilled. The dura was then opened, and piecemeal removal of the tumor was performed. However, during dissection around the superior part of the tumor capsule, marked intracranial bleeding was encountered from what appeared to be from the A-comm artery. The bleeding source was challenging to visualize to apply an aneurysm clip and was controlled after packing with Gelfoam and Tachosil materials. The patient required the initiation of massive transfusion protocol due to significant blood loss (3 L). Then surgery was concluded with skull base reconstruction in multiple layers, including the application of fat and fascia layers and nasoseptal flap followed by nasal packing. However, upon transferring the patient from

the operative table, she started to bleed again from the nose. Then she was put back, and packing was removed, and skull-base corridor was reopened to identify the source that was discovered to be intradural coming from the A-comm artery area; the anatomy and extent of vascular injury was difficult to visualize, and was hardly stabilized after mounting pressure with hemostatic agents pulling the ACA-ACOM (anterior communicating) complex down against the planum with Penfield number 4 instrument.

After that, the neuro intervention team was emergently involved, and the patient was shifted to the angiogram suite with the packing and instrument in-place. The cerebral angiogram showed no flow in both A1 segments of ACA without extravasation or vasospasm; thus, bilateral A1 segments of ACAs were occluded to prevent recurrent hemorrhage and postoperative blood pressure (BP) augmentation using a coil on right and onyx on the left side (→Fig. 2A–F).

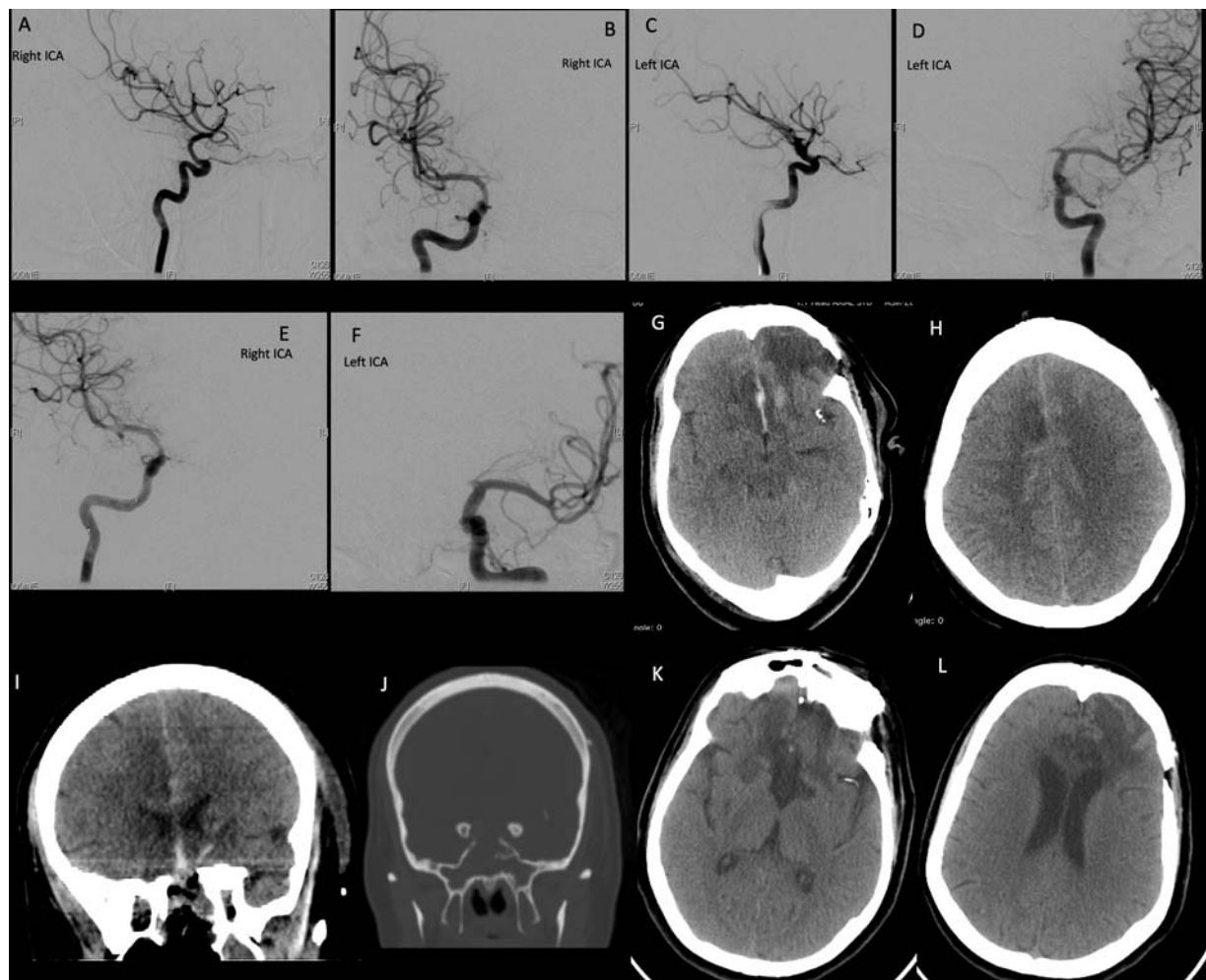


Fig. 2 (A, B) Lateral and anterior–posterior views of conventional angiography images of the right ICA that was performed immediately after the intraoperative incident of arterial injury that was temporarily stabilized, demonstrating the loss of flow in the first segment (A1) of right anterior cerebral artery before coiling. (C, D) Lateral and anterior–posterior views of conventional angiography images of the left ICA that also demonstrate similar description with loss of flow in A1. (E, F) Anterior–posterior views of conventional angiography images of the right and left ICA, demonstrating successful coiling on the right side and onyx embolization on the left side. (G, H, I) Immediate postoperative axial and coronal views of the brain CT showing small hematoma at the surgical bed, subarachnoid hemorrhage, and small intraventricular extension, with only a tiny area of ischemic changes in the right paramedian frontal lobe. (J) Coronal bone window view of the brain CT obtained postoperatively demonstrating the decompression of the optic. (K, L) axial views of the brain CT obtained 4 days postoperatively (endoscopic endonasal surgery), showing the persistent stable right paramedian ischemic changes. CT, computed tomography; ICA, internal carotid artery.

Afterward, the patient was brought back to the operative theater for redo construction of the skull base.

Immediate postoperative CT scan revealed a small hematoma at the surgical bed, subarachnoid hemorrhage, and small intraventricular extension, with only a tiny area of ischemic changes in the right paramedian frontal lobe (►Fig. 2G–J). The patient was transferred to the intensive care unit (ICU) for monitoring, and BP augmentation above 160 mm Hg systolic was ensued to induce collateral supply. She was extubated after 24 hours of surgery and remained hemodynamically stable. Her Glasgow coma score was 14/15 (due to confusion) and sustained right arm weakness of motor grade 1/5, and bilateral lower limb weakness with a power of 2/5 motor grade. CT scan revealed small areas of ischemic changes noted in left frontoparietal lobes and the anterior paramedian portion of the right frontal lobe (►Fig. 2K, L).

The patient was discharged from ICU after 2 weeks and required 6 months of daily care with physiotherapy and occupational therapy. She was then discharged to a rehabilitation center for further care. Upon discharge, she had normal lower limb power on the left side. However, on the right side, it was 3/5, and she became able to walk with assistance. Regarding vision in comparison to preoperative assessment, ophthalmological

review reported no changes. MRI scan at 6-month follow-up demonstrated small residual lesion and stable findings regarding the ischemic changes (►Fig. 3).

Discussion

TSS is a relatively safe procedure compared with the open approach and has a less invasive approach and postoperative hospital-stay, with a mortality rate of less than 1%.¹⁶ Complications rate in this surgery is estimated at around 6.3%, accounting mainly for CSF leak and diabetes insipidus.^{6–8,10} Vascular complications during TSS are most feared and associated with limited visualization for its control intraoperatively with an incidence ranging between 0.34 and 2.6%.^{10–13} The majority of publications that concern vascular complications in TSS were accounted for ICA, as its injury is the most common in this approach.¹² However, iatrogenic intradural arterial injuries are rare and reported cases in the literature are associated with variable morphology and management, with no clear standard of care (►Table 1).^{11,15–20} It is also worth noting that iatrogenic vascular complications in TSS are not necessarily to be observed intraoperatively, as in pseudoaneurysm or stenosis.^{19,21}

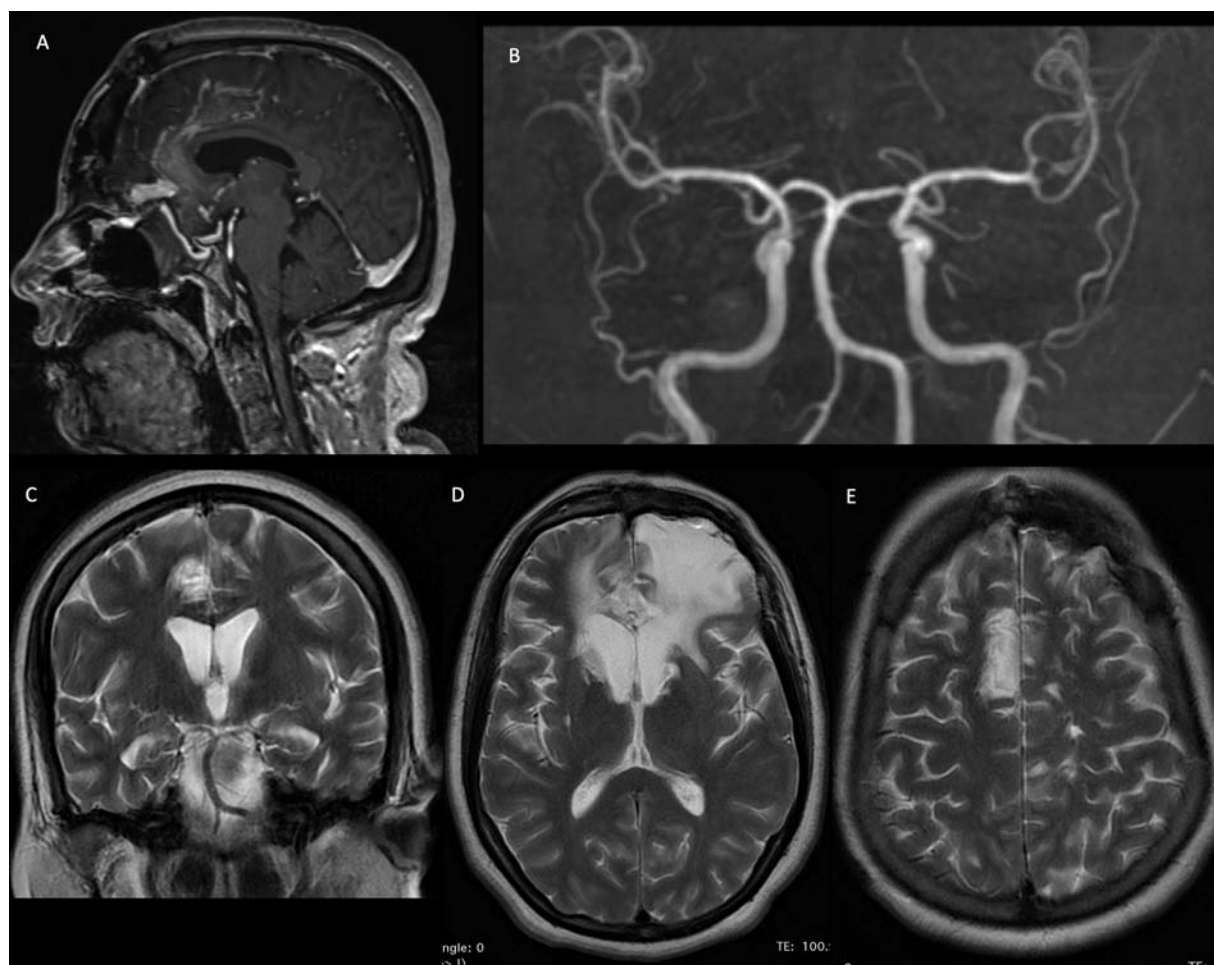


Fig. 3 (A) Sagittal MRI brain images (T1 with contrast) 6-month postoperatively (endoscopic endonasal surgery) demonstrating the small residual tumor and adequate reconstruction of the skull base floor. (B) Magnetic resonance angiography of the brain demonstrating persistent loss of flow in bilateral A1 segments. (C, D, E) Coronal and axial views of the brain MRI images (T2), demonstrating persistent right frontal lobe paramedian infarction with no interval ischemic changes. MRI, magnetic resonance imaging.

Table 1 Summary of the reported cases of intradural vascular injuries during transsphenoidal surgery

Author (year)	Age/gender	Diagnosis	Injured artery	CT findings	Angiographic findings	Management	Follow-up	Complication	Reported risk factors
Ciceri et al ¹⁵ (2006)	56/F	Pituitary adenoma	PCA	SAH	Saccular pseudoaneurysm	Parent artery occlusion	1 day	dead	NA
Kassam et al ¹⁶ (2011)	NA	Olfactory groove meningioma	Frontopolar artery	NA	Pseudoaneurysm	Endovascular treatment	NA	Permanent right lower hemiparesis and cognitive deficit	NA
Cinar et al ¹⁷ (2013)	60/M	Pituitary tumor	PCA	SAH	Pseudoaneurysm	Parent artery occlusion with coils	NA	Good	NA
Cinar et al ¹⁷ (2013)	60/F	NA	Basilar trunk	NA	Pseudoaneurysm	Aneurysm occlusion (stent-assisted coiling)	NA	Good	NA
Cinar et al ¹⁷ (2013)	37/F	NA	ACA	SAH	Pseudoaneurysm and ipsilateral ICA occlusion	Parent artery occlusion with coils	NA	Hemiparesis	NA
Rodríguez-Hernández et al ¹⁸ (2013)	27/F	Pituitary adenoma	PCA	IVH, hydrocephalus, thalamic infarction	Pseudoaneurysm	Direct PCA-SCA bypass with proximal and distal clipping, trapping the pseudoaneurysm	3 months	Mild left hemiparesis 4/5	NA
Lee et al ¹⁹ (2015)	32/F	Pituitary adenoma	PCA	SAH, IVH, hydrocephalus	Pseudoaneurysm	Observation resulted in nonvisible aneurysm in 3 weeks	3 years	none	NA
Romero et al ¹¹ (2017)	40/M	Pituitary adenoma	A-Comm A	SAH	pseudoaneurysm	A-comm artery occlusion with coil	6 months	none	NA
Romero et al ¹¹ (2017)	69/F	Tuberculum sella meningioma	ACA	NA	NA	Intraoperative Weck clip occlusion	9 months	none	Encasement of left supraclinoid ICA, and bilateral ACA (A1)
Romero et al ¹¹ (2017)	53/M	Pituitary adenoma	Ophthalmic artery	No hemorrhage	Discontinued follow	Arterial occlusions with coiling	3 months	none	NA
Morinaga et al ²⁰ (2019)	68/M	Pituitary adenoma	P-Comm A	SAH	Pseudoaneurysm	Two-staged stent-assisted coil embolization	10 months	Hydrocephalus- shunted	Redo surgery for tumor recurrence
Current case	43/F	Planum sphenoidal meningioma	A-Comm A	SAH, IVH	Occluded first segment of anterior cerebral artery	Endovascular bilateral A1 occlusion	8 months	Right side weakness 3/5, and diabetes insipidus	Redo surgery for tumor recurrence

Abbreviations: ACA, anterior cerebral artery; A-Comm A, anterior communicating artery; CT, computed tomography; F, female; ICA, internal carotid artery; IVH, intraventricular hemorrhage; M, male; NA, not available; PCA, posterior cerebral artery; P-Comm A, posterior communicating artery; SAH, subarachnoid hemorrhage; SCA, superior cerebellar artery.

We report a case that had an ACA complex injury in the form of ACOM artery injury that resulted in bilateral ACA occlusion during a redo TSS. Other intradural arterial injuries described include PCA, A-comm artery, P-comm artery, ophthalmic, and artery. Injuries are not necessarily recognized intraoperatively, and sometimes only associated with minor controllable bleeds. These cases are usually related to postoperative acute deficits or failure to wean off the intubation.^{18,19} Most of these injuries occur due to excessive manipulation or dissection that can avulse a perforator vessel adhered to the tumor and result in vascular wall compromise that is not well visualized in such a narrow operative corridor.¹⁸ In these cases, pseudoaneurysms are usually evident in CT angiography or a conventional angiogram (► **Table 1**).

In cases where intraoperative arterial injury with excessive bleeding is recognized, trials to control the bleeding intraoperatively using glue, cauterization, or muscle flap for tamponade are necessary to allow at least temporary control, allowing patients hemodynamic stability and subsequent transfer to the angiogram suite, as demonstrated in our case after quite challenging recurrent bleeding. There is a single case, however, reported by Romero et al that described intraoperative direct control of the parent artery (ACA) with weck clips occlusion after the failure of bipolar cauterization and muscle flap implantation.¹¹

The rule of angiographic management after encountering intradural iatrogenic arterial injury is crucial, and the majority of reported cases ultimately required occlusion or sacrifice of the parent artery, especially when angiography shows adequate compensation from cross-circulation.^{11,15,17} Similar to most ICA injury cases during TSS, that was ultimately managed with endovascular parent artery occlusion.^{17,22} In one case reported by Romero et al, multiple trials to preserve the ACA had failed and the patient ultimately required occlusion of the parent artery. Furthermore, the patient experienced multiple dissecting aneurysms in the ICA.¹¹ A recent case reported by Morinaga et al described a P-comm artery aneurysm after TSS, which was managed with coiling and subsequently followed by a second stage stenting because of incomplete obliteration of the aneurysm, and parent artery preservation was successful.²⁰ Parent artery preservation is recently becoming possible in certain arterial injury cases first described in ICA injuries.²³ Rodriguez-Hernandez et al report another novel technique to preserve blood supply; he described microsurgical arterial bypass and pseudoaneurysm entrapment as an option in a case of iatrogenic PCA injury that resulted in a satisfactory outcome.¹⁸

Postoperatively, we targeted a systolic BP of above 160 mm Hg to reduce the ischemic penumbra. Endovascular arterial obliteration allowed us to safely augment the BP in the ICU, which we believe significantly affected the outcome in our case by minimizing ischemic insults. This practice of BP augmentation is also used in acute ischemia patients and demonstrated positive results. Neurological improvement was observed in 33% of patients who had an ischemic stroke and managed with BP augmentation having a systolic BP of 130 to 180 mm Hg as a threshold. Furthermore, stroke patients who are admitted with systolic BP either above or

below the range mentioned above (130–180 mm Hg) had the worst outcomes.^{24–26}

Our patient had small bilateral areas of infarction in ACA territory more evident on the right side demonstrated in postoperative CT and did not develop the full picture of ACA syndrome contrary to what we expected as we did bilateral occlusion of the ACA. She improved over time with extensive rehabilitation until she became minimally dependent, which suggests that collateral blood supply could compensate ACA occlusion to a significant extent. Previously, reported ACA injuries during TSS were all unilateral, with our case being unique in its bilateral involvement. When arteries are adherent or encased by tumor tissue, the risk of injury increases, other predisposing factors for iatrogenic vascular injury also include less surgical experience, prior TSS, intraoperative tract deviation, and radiation therapy exposure.^{13,21,27}

The learning points out of this case is to studying the images before the surgery to appreciate the proximity or involvement of the artery to the targeted tumor. If the tumor indeed was adherent to an artery and endoscopic approach would not be optimal to have sufficient exposure with optimal visualization, one should consider craniotomy over the endoscopic endonasal approach, especially in a redo surgery or cases that have risk factors predisposing arterial injury. We operated on this patient in a center with a high level of care and resources that allowed us to access the angiogram facility in excellent timing. However, it is worth noting that such rare complications if occurred in a center with lower resources that outcome would be remarkably different.

Conclusion

Intradural arterial injury during TSS surgery is an exceedingly rare entity, with no clear standard of care to manage such cases. Collateral blood supply allows definitive management of vascular injuries with minimal morbidity, a concept to which our case illustrated significant support. Postoperative BP augmentation, along with comprehensive rehabilitation, is an essential factor for recovery. Identifying the risk factors beforehand, as well as performing such cases in a well-resourced center, are crucial elements in the management of these cases.

Conflict of Interest

None declared.

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