Prevention and Management of Bleeding During Endoscopic Approaches to Skull Base Pathologies

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Prevention and Management of Bleeding During Endoscopic Approaches to Skull Base Pathologies

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none

BACKGROUND
The rate of serious permanent morbidity and mortality with endonasal approaches has declined secondary to increased knowledge of the pertinent anatomy, advanced neuroimaging and navigation techniques, better surgical instruments, and improved exposure and reconstruction strategies.1-3 Although rare, vascular injury remains a potentially serious complication. However, with limited systematically-collected and reported data, the exact incidence rate of vascular injuries is difficult to determine. In terms of arterial injuries, the incidence based on reported series likely ranges from 0.3%-9% (Table 1),4-11 with higher rates most commonly associated with chordomas and chondrosarcomas involving the clivus. Venous injury is comparatively less severe and easier to manage. As a result, there is a comparatively lower impetus to publish epidemiological data and management strategies for these injuries. The consequences of arterial injury include fatal hemorrhage, vessel occlusion or thromboembolism causing infarction, development of a pseudoaneurysm (PA), carotid-cavernous fistula (CCF), subarachnoid hemorrhage (SAH), and vasospasm.6-7,9 Surgical expertise and detailed knowledge of the neurovascular anatomy is critical to the avoidance and management of vascular injuries.

Avoidance of vascular Injury
Pertinent vascular anatomy
The dominant venous structures in skull base surgery are the cavernous sinuses (CS) flanking the sellar region and the basilar venous plexus on the dorsal surface of the clivus.12 The CS on either side are connected through the superior and inferior inter-cavernous sinuses; these need to be identified during drilling of the sellar bone and managed during opening of the dura. The internal carotid arteries (ICAs) coursing within the CS are the most vulnerable major arteries in the approach toward the sellar/parasellar regions. The distance between the cavernous carotids is on average 23mm (Range 12-30mm),13-15 though rarely this may be as small as 4mm.16 The parasellar ICA may potentially be devoid of sphenoid bone coverage in up to 4% of the population.16 This defect may not be readily identified on pre-operative imaging and its potential presence must be kept in mind during drilling of the sellar bone or using monopolar cautery in this area. In approximately 25% of the population the ICAs penetrate the medial wall of the cavernous sinus and directly contact the gland, potentially hindering the ability to develop a surgical plane during tumor dissection.17 Sellar neoplasms may displace the cavernous carotids laterally or encase the vessels altogether, increasing the risk of intraoperative hemorrhage from the ICA or its branches.
Adjuncts for management of hemorrhage

Preoperative
Imaging can help elucidate the relationship between critical neurovascular structures and the pathology. Balloon test occlusion (BTO) with neuro-monitoring may be indicated in cases wherein the potential for vascular injury is high (e.g. extensive vascular encasement by the pathology). Embolization of potentially vascular tumors may be necessary to minimize intraoperative bleeding.

Intraoperative
Neuro-monitoring can be critical during the management of intraoperative vascular complications, through either indicating the neurological impact of active hemorrhage or the consequences of over-packing the site of hemorrhage during hemostasis. For example, over-packing of ICA bleeding can result in the compression of cranial nerves and even loss of anterograde blood flow, both of which can be detected with intraoperative neuro-monitoring.

Using computerized surgical navigation and micro-doppler ultrasonography, location of the ICA can be confirmed prior to surgical drilling or incising the dura. In addition, the evolution of surgical tool design has contributed to minimizing the risk of vascular injury during dural opening as well; examples of these include the low-profile angled blades or scissors which direct cutting force away from the intradural surface.2,17 Intradurally, sharp extra-capsular dissection of arachnoid planes and central debulking of tumors (to avoid vessel avulsion) are important strategies. If efforts in developing a plane between the tumor and a surrounding neurovascular structure are not successful, a subtotal resection may be advisable.

Management of intraoperative bleeding

General: Regardless of the degree of bleeding, it is essential that the endoscope is not withdrawn from the surgical site. A pitfall is to over-pack the repair, which may result in carotid occlusion; neuro-monitoring should be assessed during such maneuvers to prevent irreversible ischemia.

Venous
A nuisance, but rarely life threatening. Total intravenous anesthesia does not increase intracranial pressure, which is helpful for minimizing venous bleeding. Although CS bleeding can be brisk, it is usually easily managed with head elevation and gentle packing with Surgicel or FloSeal.

Arterial: The majority of ICA injuries are small and can be immediately controlled with rudimentary hemostatic methods (e.g. Gelfoam and the application of pressure with a cottonoid patty). Small lacerations can sometimes be definitively repaired with bipolar coagulation and packing with Surgicel. Larger ICA injuries during endonasal surgery are much more difficult to manage. A number of packing materials and techniques have been described, including gelfoam, fibrin glue, muslin gauze, and a crushed muscle patch.18-20 Larger lacerations can be managed with a myriad of strategies such as utilizing a two-layered muslin gauze patch that is reinforced with a fat graft or collagen sponge.21,27 Direct repair or reinforcement of the laceration may not be feasible and vessel sacrifice may be necessary. An immediate postoperative angiogram is critical to evaluate the vessel repair and to rule out early postoperative pseudoaneurysm formation.

Pseudoaneurysms (PAs): The rate of postoperative ICA PA formation is highest with direct vessel injury.22 PA rupture, typically within days to weeks from diagnosis, may result in SAH, CCF, life-threatening epistaxis, or a nidus for distal thromboembolism/infarction.23,24 Even with a normal immediate postoperative angiogram, a repeat angiogram should be repeated within 7-10 days, particularly when suspicion for ICA injury is high. If open craniotomy to sacrifice the vessel is undertaken, an angiogram is necessary for assessing collateral circulation and the feasibility of an extracranial-intracranial bypass.21 Endovascular management is an alternative and includes complete ICA occlusion, coiling the PA, or vessel reconstruction with stent-assisted coiling. Coiling alone is often not successful and there is a possibility for dissection or thromboembolic events.25 Endovascular stenting may be a more feasible option.26

Figure 2
Although perforating branches of the ICA are small and injury to them can easily be controlled, their supply of critical structures makes the neurological consequence of sacrificing these vessels potentially dire.
**CASE REPORT**

A 51-year-old male presented with a three-month history of progressive visual loss. Formal visual field testing and optical coherence tomography (OCT) revealed a chiasmal compression pattern. Contrast MRI showed a large meningioma growing along the planum sphenoidale, sella, and eroding into the sphenoid sinus (Figure 3A-D). An endoscopic endonasal approach for tumor resection was performed. Intraoperatively, a small amount of arterial bleeding was observed while aspirating tumor in the vicinity of the left ICA. The bleeding was controlled with FloSeal and the application of pressure with a cottontoid. As adequate tumor debulking had already been achieved, the procedure was stopped as a precaution due to the concern of possible ICA injury.

Immediate postoperative examination showed improved visual field exam and no other changes neurologically. MRI showed significant tumor debulking and decompression of the optic chiasm. Early in the post-operative course MRA, CTA, and an angiogram revealed normal intracranial vasculature.

Two weeks postoperatively the patient was readmitted with the acute onset of severe epistaxis, which was temporarily controlled with nasal packing. Emergent angiogram revealed a left cavernous ICA PA (Figure 4A-B). Balloon test occlusion showed no venous filling delay and the patient passed a hypotensive challenge. Several options were considered, including ICA sacrifice, bypass, and coiling of the PA. Ultimately, the PA was coiled. Although the epistaxis completely resolved, dissection and stenosis of the left ICA resulted in intermittent hemiparesis and impaired language. Imaging-confirmed left ICA dissection and embolic phenomena (Figure 5A). The decision was thus made to occlude the parent vessel with coils delivered through an endovascular approach (Figure 5B).

On immediate postoperative exam, the patient exhibited no residual weakness. The residual meningioma was subsequently treated with fractionated stereotactic radiotherapy and the patient remains stable for over five years since initial surgery.

**CONCLUDING KEY POINTS**

1. Endonasal anatomical and technical expertise is necessary to avoid and manage vascular injuries
2. Cavernous sinus meningiomas may not have a plane separating them from the ICA
3. If ICA injury suspected, keep looking!
4. ICA pseudo-aneurysms are best treated by vessel occlusion and early bypass when indicated
REFERENCES


