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EDITORIAL

How to avoid complications in skull base surgery?

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Over the past ten years, advances in skull base surgery approaches have broadened the indications of traditional resection limits. Stammberger[1] was one of the pioneers who performed a complete endonasal endoscopic excision of an anterior skull base tumor. Since then, more complex and challenging skull base lesions have been treated through an endoscopic approach.

The advantages of the endoscopic approach include a better visualization of the parasellar and clivus regions, the possibility of access to intracranial structures such as the frontal lobe, decreased perioperative morbidity, wide surgical field exposure and improved cosmetic results compared with traditional open trans-facial or craniofacial approaches.

The complex anatomy and the heterogeneity of the lesions, as well as the close proximity to crucial neurovascular structures, make this a very challenging area to treat. Some more common complications include bleeding, infections, cerebral stroke, endocrine complications, CSF leaks, meningitis, direct neurovascular injuries, early or delayed postoperative epistaxis, severe frontal headaches, visual changes, tension pneumocephalus, or death.

Minimizing risks and thinking about strategies to manage complications preoperatively help create better outcomes for the surgeon and the patient. To achieve the best results, optimal management of skull base pathologies requires a multidisciplinary approach with ENT surgeons, neurosurgeons and plastic surgeons and careful surgical planning including preoperative work-up, prophylaxis and postoperative monitoring.

It is much easier to avoid complications if you and the team are prepared for them. Thereby, the cornerstone of preoperative assessment is to review the radiologic images, which can alert the surgeon to potential surgical pitfalls: low-lying cribriform plate (Keros type 3), dehiscence of the carotid or optic canal, anatomic variants such as sphenoethmoidal (Onodi) cell or undeveloped frontal sinus. Also, anterior and posterior ethmoidal arteries are important anatomic structures to be identified, especially in revision surgery where protective bone in these regions may have been removed. Moreover, gadolinium-enhanced images are valuable in identifying dural enhancement and parenchymal extension[2].

The preoperative assessment should focus on the history of blood disorders and includes three basic elements: vascular compartment, platelets and coagulation factors[3]. Congenital and acquired coagulopathies can appear from any of these components (Osler-Weber-Rendu syndrome, thrombocytopenia, idiopathic thrombocytopenic purpura, hemophilia, von Willebrand's disease and medication such as warfarin and heparin).

Also, identifying the patients in treatment with oral anticoagulants and/or antiplatelet agents is essential. Current guidelines recommend stopping both clopidogrel and aspirin for 7-10 days before the surgery. Warfarin should be stopped 5 days prior to the surgery. On top of that, most studies recommend an international normalized ratio (INR) value of less than 1.5. In cases with moderate or high thromboembolic risk, anticoagulant treatment with unfractionated heparin is recom-

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mended for three days before surgery[3].

In cases of vascular tumors, preoperative embolization reduces the risk of haemorrhage and improves the visualization of the surgical field. It is recommended that embolization be performed within 24 to 72 hours before surgery. Embolic agents used are polyvinyl alcohol, ethyl vinyl alcohol copolymer (EVOH), liquid n-butyl cyanoacrylate (n-BCA), trans acryl microspheres or micro coils. An important feature that must be taken into account is the possibility of agent migration through non-target arterial vessels leading to catastrophic complications such as blindness, stroke or death[3].

Additionally, preoperative corticosteroids could be helpful due to their anti-inflammatory and antiedematous effects[4].

Patient positioning (reverse Trendelenburg) is another important factor that reduces venous return improving haemostasis during surgery. Recent studies demonstrated that head elevation reduces mean arterial pressure in the elevated part by about 2 mmHg for each 2.5 cm above the cardiac level.

Additionally, hypotension anaesthesia is the reduction of systolic blood pressure to 80-90 mmHg using propofol (infusion or inhaled) and remifentanil, thus decreasing the risk of bleeding by 30%[3].

In cases of intraoperative bleeding during endoscopic skull base surgery, topical vasoconstrictors, local anaesthetic with vasoconstrictor injection, haemostatic biomaterials, topical antifibrinolytics, or gelatin-thrombin matrix can be used.

The reconstruction of the skull base is essential to prevent cerebrospinal fluid leaks (CSF) and to prevent the risk of meningitis or pneumocephalus. The incidence of CSF leaks is approximately 5%[5].

CSF leaks can occur anywhere along the skull base: the junction of the anterior ethmoidal artery and middle turbinate along the anterior ethmoid roof (the thinnest and most susceptible to injury area), the cribriform plate, the posterior ethmoid roof, the roof of the sphenoid sinus or the posterior wall of the frontal sinus. It occurs by injuring the skull base during surgery or by excessive intraoperative bleeding predisposing to surgical disorientation. In these cases, it is recommended to end the surgical procedure and not proceed further. The majority of cases can be successfully repaired through an endoscopic approach at the end of the skull base procedure. Nasoseptal flap is useful in covering most of the skull base defects. Artificial dura, fascia lata are also valuable materials to use for reconstruction of the skull base. Depending on the severity, CSF leaks can be managed conservatively with bed rest. Additionally, lumbar drains might reduce intracranial pressure.

Intraoperative leaks during ESS are usually small (<1 cm) and typically repaired with a single layer consisting of a free intranasal mucosal graft harvested from the septum or turbinate. Larger defects require two layers, using septal bone or cartilage placed on the intracranial side. Then, the mucosal graft is stabilized and protected with topical haemostatic agents (Gelfoam or Surgicel) and followed by absorbable packing material and non-absorbable packing. The patients are advised to sneeze with mouth open, to avoid nose blowing, bearing down and constipation.

The use of prophylactic antibiotics in skull base surgery is not clearly defined. The presence of nasal microbiome and the close proximity to the intracranial cavity increases the risk of infection. However, in the absence of a CSF leak, the risk of meningitis is minimal. In the case of a CSF leak, the risk of meningitis increases by up to 30%, and the incidence of infection increases with the duration of the CSF leak[2]. Thus, preventing CSF leaks is essential to avoid infectious complications.

The incidence of cranial nerve injuries is 2.5% for transient and 1.8% for permanent neurologic deficits[6]. In patients with sellar lesions, close attention must be paid to the neurovascular structures within the cavernous sinus (oculomotor nerve – CN III, maxillary nerve – CN V2, abducens nerve – CN VI and the sympathetic plexus around the internal carotid artery). Pituitary macroadenomas are frequently associated with visual field impairments (46-75%) and impaired visual acuity (14-44%) caused by direct optical nerve/chiasmal compression. Lamina papyracea penetration, the use of instrumentation within the orbital apex, the manipulation within the superolateral sphenoid sinus, if the optic canal is dehiscent, or an unrecognized Onodi cell during a posterior ethmoidectomy can represent a risk for optic nerve injury with an increased risk of blindness.

If an injury of the optic nerve is suspected, treatment includes high-dose systemic steroids and regular assessment of visual acuity. A postoperative scan is mandatory. If the visual acuity does not improve in 24-72 hours, optic nerve decompression is required[7].

Internal carotid artery (ICA) injury is one of the most catastrophic complications that can occur during endoscopic skull base surgery, with an incidence of 0.016-1%[8]. It can occur in cases of hyperpneumatized sphenoid sinus which extends too far laterally, if the carotid wall is penetrated or if the dissection was performed along the lateral sphenoid wall. To avoid ICA injury, the surgeon should

always enlarge the opening in an infero-medial direction, should not use instrumentation within the sinus cavity and not remove the intersphenoid septum. Recent studies showed that the micro-Doppler ultrasound and neuronavigation might decrease the rates of ICA injuries[2].

Complication avoidance depends on preoperative awareness of potential surgical pitfalls, meticulous surgical technique, proper knowledge of sinonasal landmarks and adequate intraoperative haemostasis.

Anticipation and appropriate preparedness for surgery are essential to limiting complications. When complications do occur, prompt recognition and proper management usually result in good patient outcomes.

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