Neurosurgical complications after intranasal ethmoidectomy

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Abstract

Intranasal ethmoidectomy is a common otolaryngological procedure. Despite the potential for serious intracranial complications, there is a paucity of reports describing the neurosurgical complications of the procedure. Two patients with intracranial complications of intranasal ethmoidectomy, and the relevant medical literature, are reviewed. The anatomy of the ethmoid air cells and their relation to the intracranial cavity are described. The importance of definitive, emergent repair with attention to the potential for vascular injury is discussed.

Intranasal ethmoidectomy is a common otolaryngological procedure performed for the treatment of chronic ethmoiditis with or without polyposis, for acute complicated ethmoiditis, and for biopsy of suspected ethmoid sinus tumours. The procedure offers certain advantages over external and intraoral approaches, because it avoids the incisions and complications of these approaches and improves visualisation of the ethmoid air cells.[1] However, in the early twentieth century Mosher described intranasal ethmoidectomy as one of "the most dangerous and blindest of all surgical operations".[2] Despite the potential for intracranial complications during ethmoidectomy, there is a paucity of reports describing the neurosurgical complications of this procedure.

The two cases described were referred to the neurosurgical service of the Dartmouth-Hitchcock Medical Center and illustrate serious intracranial complications of intranasal ethmoidectomy. These cases prompted a review of the medical literature and suggested the adoption of a more aggressive treatment strategy for treating patients with these problems.

Case 1

A 70 year old white man had numerous nasal operations for treatment of allergic rhinitis, chronic sinusitis and recurrent nasal polyps. A transnasal ethmoidectomy/sphenoidectomy was complicated by perforation of the roofs of the ethmoid and right sphenoid sinuses. The patient did not awaken readily from surgery, and on the second postoperative day remained obtunded and became febrile. Skull CT revealed an 8 × 10 mm deficit in the roof of the ethmoid sinus. A CT scan demonstrated a

right frontal lobe haematoma with blood in the suprasellar cisterns and interhemispheric fissure. Neurosurgical consultation was obtained. A ventricular drain was placed and antibiotics were started.

One week after surgery, the patient was transferred to our institution with a clinical picture of florid meningitis. A CT scan showed increasing pneumocephalus with a large collection of air in the right lateral ventricle. A right frontal craniotomy with fascia lata graft repair of the basal skull defect was carried out. Cerebrospinal fluid cultures grew *Candida albicans*; the patient received a full course of intravenous and intrathecal amphotericin B plus broad spectrum antibacterial parenteral antibiotics. Although his meningitis resolved, the patient made little neurological improvement and was transferred to a nursing home.

He was admitted two months later for the treatment of an extradural tension pneumocephalus.[3] A right external ethmoidectomy, a substitute dural graft to the posterior ethmoid and sphenoid sinuses, and aspiration of extradural air through an existing burr hole were performed. He also required ventriculoperitoneal shunting for hydrocephalus. One year after surgery the patient is at home, alert, conversant, and intermittently confused with a flat affect.

Case 2

A 65 year old white woman with a history of chronic sinusitis and numerous rhinological procedures had a transnasal ethmoidectomy at another institution. Excessive bleeding and transient hypotension were noted at surgery. Postoperatively, the patient was difficult to arouse, and a CT scan demonstrated a large cribriform plate defect, intracranial air frontally and temporally, and intraventricular haemorrhage involving both lateral ventricles and the third ventricle. The patient was transferred to the neurosurgical service at the Dartmouth-Hitchcock Medical Center for further evaluation. A left frontal craniotomy was carried out as an emergency repair of the anterior fossa defect. Life-threatening bleeding was noted when some of the nasal packing from the floor of the anterior fossa was removed. The left frontal pole was removed and aneurysm clips were placed on the left fronto-polar artery. A fascia lata/muscle graft was used to obliterate the basal skull defect. Postoperatively, the patient developed cerebrospinal rhinorrhoea and required revision of the anterior fossa repair; ultimately

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Medial aspect of ethmoid roof

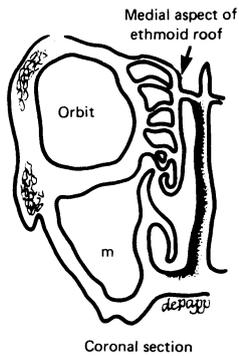Orbit

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Coronal section

Figure 1 Coronal drawing of ethmoid air cells showing their relation to cribriform plate.

she required a ventriculoperitoneal shunt. One month after this last operation the patient was awake, alert, following commands, and demonstrating a frontal lobe affect.

Discussion

The ethmoid bone is a complex bone that has both intracranial and intranasal components (fig 1). Intracranially, the crista galli forms a midline strut anchoring the falx cerebri. The cribriform plate is perforated by the olfactory nerves and the anterior ethmoidal arteries and abuts the crista galli laterally. The cribriform plate is the most dependent portion of the anterior cranial fossa. The fovea ethmoidalis is the solid bone that ascends approximately 3–4 mm laterally from the cribriform plate and forms the roof of the ethmoid air cells laterally and posteriorly. Medially and anteriorly, the roof of the air cells is formed by the cribriform plate. The perpendicular plate of the ethmoid is the inferior midline extension of the crista galli and contributes to the bony midline septum.

The air cells are pyramidal in shape with their bases situated posteriorly. Their medial wall forms the lateral wall of the nasal cavity. They are 4–5 cm in length, 2–3 cm in height, and 0·5–1·5 cm in width from front to back.[4] At birth, they are the most fully developed air cells; hence children usually present with ethmoiditis when suffering complications of sinusitis.[5] It is believed that embryologically the other paranasal sinuses are extensions of the ethmoid air cells. The frontal and maxillary sinuses arise from the anterior ethmoid cells and the sphenoid sinus emanates from the posterior cells. The ethmoid air cells are separated from the orbit laterally by the thin

lamina papyracea. The air cells are usually divided into two groups based on their drainage pattern: an anterior group and a posterior group. The anterior cells drain under the cover of the middle turbinate, and the posterior cells drain under the superior turbinate. Both the superior and middle turbinates are components of the ethmoid bone.

The middle turbinate is the landmark for intranasal ethmoidectomy. Anteriorly, it is continuous with the ethmoid roof and attaches between the cribriform plate and fovea ethmoidalis. Therefore, dissection lateral to the middle turbinate prevents violation of the cribriform plate. Posteriorly, the middle turbinate has no attachment to the anterior cranial fossa. The middle turbinate is frequently pneumatised and involved in disease. This may require its removal, but every effort to preserve the middle turbinate landmarks is made by experienced otolaryngologists. This preservation is crucial, as most of the afflicted patients require further nasal procedures, and scarring can obscure normal landmarks. Both of the patients described above had previous nasal procedures.

Two other anatomical areas are potential sources of complication. The lamina papyracea is a thin slip of bone separating the anterior ethmoid cells from the orbit. Perforation of this bone can result in serious orbital trauma including medial rectus injury. The posterior ethmoid cells, in their most posterior extent are within 1 mm of the optic nerve as it travels through the sphenoid bone. There have been reports of the optic nerve travelling through a completely pneumatised sphenoid bone.[4] The optic nerve is then anatomically inside the posterior ethmoid air cell labyrinth.

Because of the anatomical relationships of the ethmoid bone, intranasal ethmoidectomy carries with it a potential for serious intracranial complications. The scepticism about the approach was allayed by Freedman and Kern in 1979, when they reviewed 1000 consecutive cases from 1957–72 with an overall complication rate of 2·8% without mortality or blindness.[6] The two most serious complications were meningitis and cerebrospinal rhinorrhea. In 1982, Taylor et al reviewed 326 procedures, with a complication rate of 2·5% with no blindness, meningitis, or deaths.[7] Violation of the cribriform plate with subsequent development of cerebrospinal fluid leak and infection are well recognised complications[2 6–10]; however, the potential for intracranial vascular trauma is less well elucidated. Sachdev et al[11] reported two cases of subarachnoid haemorrhage after ethmoidectomy, one occurring secondary to a traumatic cerebral aneurysm.

In reviewing the literature we discovered two cases of carotid-cavernous fistuli after ethmoidectomy and sphenoidectomy.[2 12] Maniglia et al[2] provide the only report of serious complications of ethmoidectomy: three cases of optic nerve damage resulting in total blindness; two cases of loss of ocular motility, one case of cerebrospinal fluid leak leading to recurrent meningitis and epidural abscess, one

Frontal sinus

Corpus callosum
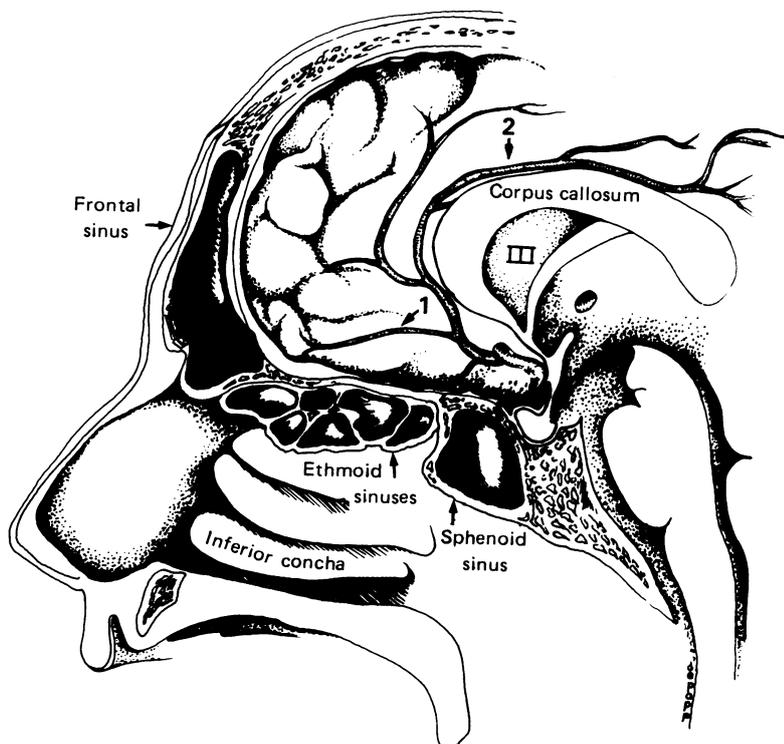
Ethmoid sinuses

Inferior concha

Sphenoid sinus

Figure 2 Parasagittal drawing of relation of ethmoid air cells to intracranial cavity and their proximity to intracranial vessels: 1) fronto-polar artery, and 2) pericallosal artery.

carotid-cavernous fistula, and one case of anterior cranial fossa damage resulting in death. Similar to the two cases we describe, the complications in these cases represent life-threatening conditions associated with this procedure. The larger series delineate few complications, most notably cerebrospinal rhinorrhea, which is treated conservatively or with an external dural graft repair. Both our patients presented with large basal skull defects amenable only to intracranial repair. These should be considered emergent cases, and repair of the basal skull defect should be done definitively and not temporised by drainage. The potential for vascular injury should be recognised, and preoperative angiography should be considered before repair (fig 2).

The intracranial complications of this very common otolaryngological procedure need to be recognised by the neurosurgeons. Definitive repair should be done with attention to the potential for vascular injury.

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