Review Article

Functional Endoscopic Sinus Surgery: Indications and Complications in the Ophthalmic Field

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Abstract

Functional Endoscopic Sinus Surgery (FESS) is a highly sophisticated type of surgery, which has revolutionized the surgical management of chronic sinus diseases. In the ophthalmic field, FESS plays a crucial role in the management of a few conditions, but not without risks. Ophthalmic complications associated with FESS are well documented. They mainly occur due to the shared common anatomic areas between ophthalmology and otolaryngology. Ophthalmic complications can vary in severity from very trivial cases such as localized hematoma collection, which is not very problematic to very devastating cases, such as optic nerve damage, which can lead to complete blindness. In order to minimize such complications, safety measures need to be considered prio to the surgery, these include; precise knowledge of detailed anatomy, the operating surgeon's ability to interpret precisely the para nasal sinus CT scan and experienced procedural surgical skills.

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Introduction

Historical Perspective

Endoscopic sinus surgery for the treatment of acute and chronic sinusitis was well established by the first third of this century.[1] It was based on the commendable anatomical studies of Zuckerkandl,[2] Onodi [3] and Grünwald. Transnasal endoscopic sinus surgery was introduced in the mid 1980s. The term FESS was coined by Kennedy.[4] Endoscopic orbital decompression was first described by Kennedy[5] and Michel[6] in the early 1990s. Over the years, enhanced visualization of key anatomic landmarks such as the region of the orbital apex, which is a critical area of decompression in optic neuropathy, has made endoscopic surgery a versatile tool.

Functional Endoscopic Sinus Surgery (FESS) and Instrumentation

The concept of FESS is the removal of tissue obstructing the Osteo Metal Complex (OMC) and the facilitation of drainage while conserving the normal non-obstructing anatomy and mucous membrane. The rigid fiberoptic nasal telescope provides superb intra-operative visualization of the OMC, allowing the surgery to be focused precisely on the key areas. The image can be projected onto a television monitor through a small camera attached to the eyepiece of the endoscope. Microdebriders remove the pathologic tissue while preserving normal mucosa.[7] Over the past 20 years, endoscopic sinus surgery has been widely used as a safe and effective treatment for Para Nasal Sinus (PNS) disorders. Powered instrumentation and stereotactic image-guided surgery have improved efficiency and safety of this procedure. Endoscopic approaches to benign tumors of the nose, sinuses, anterior cranial fossa and the orbit are now becoming widely established. The combination of suction with powered dissection has revolutionized endoscopic sinus surgery.[7] However, the potential for complications [8] has shadowed the procedure. Endoscopic sinus surgery presented a series of complications in the late 1980s and early 1990s.[9] However, new technology of instrumentation has produced undisputable advances. The powered cutting instrument is thought to be safe around the skull base and lamina papyracea because it cannot grasp the intact bone, however, this sense of safety is undermined by the fact that the instrument can grasp and cut free edges of the bone.[10]

The field of FESS was not only limited to the domain of Otolaryngology. Its indications in ophthalmology, orbit endoscopy in particular, became clearer but often at a great risk. The ophthalmic indications for FESS include orbital decompression of thyroid orbitopathy,[5] lacrimal obstruction,[11] Optic Nerve (ON) decompression, traumatic loss of vision, and pituitary tumor surgery. [12]

The relationship between ophthalmology and otolaryngology has been exploited in conditions such as silent sinus syndrome, lacrimal duct problems, optic nerve decompression, and orbital decompression, drainage of subperiosteal abscess, orbital trauma, tumor surgery, and complications of endoscopic sinus surgery.

Clinical Anatomy

The Sinonasal tract and orbit are contiguous structures. As rightly mentioned by Chastain and Sindwani,[13] practical limits between the fields of otolaryngology and ophthalmology have produced an area of "no man's land" in which otolaryngologists feel as uneasy in the orbit as ophthalmologists do in the nose.

Developmentally, until the age of four years, the maxillary sinus expands laterally to the infraorbital canal. Yet, at the age of eight years, pneumatization extendes laterally to the infraorbital canal. It is extremely important for all FESS surgeons and ophthalmologists to remember the following important relations of the orbit to the cranium:

1. The orbital apex and the lateral orbit are related to the middle cranial fossa.
2. Superiorly, the orbit is related to anterior cranial fossa.
3. Inferior orbital fissure communicates with the middle cranial fossa.

During anterior to posterior dissection in FESS, the following ocular clinical anatomy should be considered to avoid iatrogenic complications:

1. Laterally, the optic nerve may lie in intimate contact with the posterior ethmoid cells (cells of Onodi) and sphenoid sinus.[9]
2. In cases where it is necessary to open the sphenoid sinus, entry should be as far medially and inferiorly as possible, to decrease the risk of injury to the optic nerve and internal carotid artery.

Surgical Aspects

Endoscopic sinus surgery under local and topical anesthesia has presented some safety concerns in the sense that any periorbital injury would evoke a painful response from the patient and any injury of the orbital hematoma would result in visual loss to the patient. Thus No such advantages are possible under general anesthesia.

The extent to which the surgeon enlarges the ostium of inferior turbinate is important. Excessive anterior extension risks damage to the nasolacrimal duct and should be avoided. The patients should be instructed not to blow their nose during first 48 hours after surgery because there is a risk of surgical emphysema of the orbit and sometimes face and cranial cavity.

In the presence of ethmoiditis with orbital edema or subperiosteal abscess located lateral to the lamina papyracea, it is better to use the endonasal approach in FESS.

During FESS an intact muco-periosteum lined cavity should be preserved in the areas of the medial orbital wall, skull base and the frontal recess to avoid orbital complications. Use of powered instruments during FESS requires great care and a wide knowledge of the anatomy related to the orbit. It has been shown that these instruments can cause suction and or lead to the exposure of the periorbital fat.[14]

In acute orbital conditions (< 3 weeks), the orbital fat is dirty due to diffuse edema and / or hemorrhage, while in chronic stages it is dirty due to scarring.

Radiology in FESS

The PNS and orbit are areas of high contrasting densities with air, fat and soft tissue, which increase the accuracy of the examination. Axial views are better for delineation of medial rectus muscle and the orbital abscess, while coronal cuts are useful for orbital and sinus anatomy. Anatomical variations of the orbit and PNS can occur not only among individuals but also within the same individual between two sides. In the context of FESS and its associated neuro-ophthalmic complications, all FESS surgeons should be well versed with the following variations in clinical anatomy:

1. Dehiscence of the Lamina Papyracea (LP) or developmental anomalies of the medial orbital wall.[15]
2. The ON may be directly exposed within the sphenoid sinus.[16]
3. Presence of Onodi cells (pneumatized posterior ethmoidal cells).

Radiological studies of the orbital and PNS structures prior to surgery can fully appreciate such anatomical variations.[17] CT scans perfectly display any orbital and intracranial complications. It has been found to correlate with surgical findings in only 84% of the orbital complications of sinus involvement.[18] Magnetic Resonance Imaging (MRI) is better for fungal infections of the PNS and orbit. Careful review of coronal and axial CT scans before surgery facilitates the approach to the sphenoid sinus.

Ophthalmic Indications for FESS

Giving full details of the surgical steps is beyond the scope of this article; however, important surgical steps corroborating with the clinical anatomy, complications and prevention have been briefly touched.

Advances in fiberoptic technology, endoscopic techniques and instrumentation have expanded applications of endoscopic surgery from inflammatory, infectious and neoplastic diseases of the PNS to include disorders of the orbit and cranium.[19] The risks of injuries to the ocular structures associated with FESS depend on:

1. Surgeon's experience
2. Extent and severity of sinonasal disease
3. History of previous sinus surgery
4. Intraoperative view of the anatomical structures of the PNS and orbit[20]

1. Orbital Apex Lesions

Such lesions often present a diagnostic challenge. Besides CT,[21] MRI and clinical findings, tissue histological diagnosis is often necessary.[22] Several approaches to the orbital apex have been described.[23] CT-guided fine needle aspiration biopsy,[21] has several pitfalls for example; the exact cytological diagnosis cannot be obtained, the posterior apex lesions are often difficult to aspirate and there is always a risk of injury to the optic nerve and contents of the superior orbital fissure. With endoscopes, it is possible to approach the orbital apex transnasally for tissue biopsy or decompression of the orbit or optic nerve.

2. Orbital Decompression in Dysthyroid Orbitopathy

Proptosis is usually associated with thyrotoxicosis, diffuse toxic goiter, and rarely Hashimoto's thyroiditis. However, it can also manifest in hypothyroid and euthyroid patients after radioactive iodine therapy. Endomysial fibroblasts produce mucopolysachrides which contribute to the inflammatory process, ultimately leading to degeneration of extraocular muscles followed by fat replacement.[24] Surgery is the mainstay of therapy for treatment of acute sight-threatening Grave's disease. It is the most direct and effective way to treat orbital apex syndrome. Ocular recession from endoscopic decompression alone ranges from 2mm to 12 mm (average 3.5mm). Additional concurrent lateral decompression to endoscopic procedure provides extra 2mm of globe recession.[25]

The goals of the orbital surgery are:

1. To expand the orbital confinements, thereby, reducing intra-orbital pressure, and relieving the optic nerve compression.
2. Eliminate corneal exposure.
3. Take care of cosmetic disfigurement [26]

General guidelines for endoscopic orbital decompression in Grave's orbitopathy:

1. Only that portion of the floor should be removed that lies medial to the infraorbital nerve.
2. Postoperative diplopia can be avoided or reduced if a 10mm-wide sling of fascia overlying the medial rectus muscle is preserved during orbital decompression.
3. Do not remove bone in the region of frontal recess, or the herniated fat may obstruct drainage of the frontal sinus.

Several external approaches for decompression of the orbital walls have been described in the literature.[27-28] Decompression of the medial orbital wall and the floor using an external ethmoidectomy incision is most commonly used in spite of the fact that this approach provides limited access to the orbital apex. Endoscopic surgical technique allows excellent visualization of the landmarks and full decompression of the medial orbital wall which may be extended as far as the optic canal.[5] The thicker sphenoidal bone overlying the optic nerve may also be removed more safely using a drill. The inferior wall can be decompressed up to the infra-orbital nerve via a wide middle meatal antrostomy. The endoscopic approach avoids scarring and carries a much smaller risk for the nasolacrimal system and infra-orbital nerve. In Graves's orbitopathy, it is preferred to perform orbital decompression during a chronic phase. The incidence of improvement following endoscopic orbital decompression for Grave's orbitopathy ranges from 22% to 89%.[1] Postoperative deterioration of visual acuity occurs in less than 5% of patients. [25]

3. Orbital Abscess

Sinusitis continues to be the most common cause of orbital inflammation and infection, especially in children. The causes of vision loss in orbital infections include optic neuritis, traction on the optic nerve or retinal artery thrombosis. Residual visual sequel tend to occur more in patients who had a visual acuity of 20/60 or less at the time of starting treatment or those who did not undergo, or had delayed surgery.[29] Subperiosteal abscesses are usually on the medial wall of the orbit between the periosteum and the LP. Lateral displacement of MR muscle by at least 2mm is diagnostic of a rim- enhancing subperiosteal abscess. [30]

Surgical indications include:

1. Deterioration in visual acuity.
2. Relative Afferent Pupillary Defect (RAPD).
3. Continuing fever after 36 hours of medical treatment.
4. Clinical deterioration after 48 hours.
5. No improvement after 72 hours of medical treatment.[31]

An intraorbital abscess with a high intraorbital pressure should undergo a wide orbital wall decompression, in addition to periorbital incision and drainage of the abscess. Posterior ethmoidectomy is done if there is extension of the abscess towards orbital apex. This also facilitates a wide exposure and decompression of the medial orbital wall, especially if orbital pressure remains elevated. This step is more indicated for intra-orbital abscesses, and rarely for subperiosteal abscesses. One should be careful not to violate periorbita in the case of subperiosteal abscess.

Extra-conal intraorbital abscesses are better dealt with using a 30-degree telescope after incision of the periorbita. Intraconal abscesses need a combined approach, as well as an active participation of an ophthalmologist.

Computer Aided Surgery (CAS) has gained momentum during the past two decades. Though there is paucity of literature on its application in the aspect of the management of orbital abscesses,[32] the objective is to find a significant technique in dealing with tumors and mucoceles of the sinonasal cavities involving the orbit.

The endonasal endoscopic approach for subperiosteal abscesses involves intranasal ethmoidectomy and removing portions of the LP,[33] allowing the pus to drain into the middle meatus and the nasal cavity. A combined external transcaruncular and transnasal approach has also been used.

Advantages of endoscopic surgey in orbital abscesses are:

1. Unsurpassed and magnified view of the medial orbital wall.
2. Lateral and "round the corners" view with angled telescopes.
3. Simultaneous dealing of sinonasal cavities.
4. No facial incisions.

The endoscopic technique has a similar success rate as traditional open approaches, but with a shorter hospital stay and less postoperative edema.[34]

4. Orbital Fractures

Amongst many injuries of head and neck, orbital floor Blow Out Fractures (OBFs) are technically easiest to treat endoscopically because the maxillary sinus provides an optical cavity to work in. Visualization of the middle and posterior third of the orbital fracture is challenging with conventional approaches because of postero-superior angulation of the orbital floor, relative to antero-inferior orbital rim. Endoscopy guided orbital fracture repair has become popular in terms of better visualization, illumination and reduction of herniation of orbital soft tissues. Due to a wide variation in orbital wall anatomy based on gender and ethnicity, there may be "no safe" area of dissection to guarantee "no damage" to the posterior structures of the orbit.[33] Deterrence in exploring the posterior dissection may lead to residual entrapped tissue or improper implant positioning, leading to enophthalmos. The endoscope is a vital tool to overcome such limitations.

4.1 Indications for endoscopic repair of OBFs:

These indications remain same as for traditional repair, namely:

1. Isolated orbital floor injuries with EOM entrapment.
2. Enophthamos.
3. More than 50% disruption of the orbital floor.

Trapdoor and medial OBFs are the best candidates for endoscopic surgery. Large dissections lateral to the infraorbital nerve may lead to postoperative paresthesia. Complex (two-walled) fractures should not be managed endoscopically.

For small orbital floor fractures the use of endoscope via gingivo-buccal incision and a maxillary sinus approach allow visualization of suspected trapdoor fractures of inferior orbital floor with entrapped inferior rectus or inferior oblique muscles. This approach is ideal for cases not requiring implants.[35] Alternative endoscopic approaches to the floor for larger fractures include:

1. Transantral approach described by Persons and Wong.[36]
2. Sublabial (Caldwell-Luc) incision by Strong.[37]
3. Combination of endoscopic orbital floor fracture repair and balloon catheter technique by Ikeda and colleagues.[38]

Endoscopy guided surgery is very helpful in cases of delayed fracture repair or secondary repair with tissue scarring. Isolated medial orbital wall fractures are more common, more severe and more frequently the cause of enophthalmos than inferior orbital floor fractures.[39] The endonasal endoscopic approach in these cases has shown promising results with less incidence and less severity of enophthalmos, and improved or resolved diplopia.[40] Conventional current transconjunctival and subciliary hidden incisions provide a wide exposure for visualization and implant placement; however, orbital fat proplapse makes it difficult to see posterior orbital shelf. Besides, postoperative lower eyelid malposition is known to occur in 1.2% to 4.2% of patients.[41] Hence the endoscopic transmaxillary approach eliminates the potential for such limitations and complications.

4.2 Advantages related to orbital blow out fractures and endoscopy

1. Confirmation of implant placements.
2. Conservative mucosal dissection.
3. Ensuring almost no "left over" of orbital floor bone fragments.
4. Intraoperative assessment of orbital floor disruption and zygomatico-maxillary complex fracture.

4.3 Complications related to orbital blow out fractures and endoscopy

The complications related to orbital blow out fractures and endoscopy are the same as for open repair except the eyelid injury; these include diplopia, enophthalmos and blindness. The incidence of enophthalmos is less with endoscopic surgery because of its ability to visualize the posterior bony shelf and confirmed implant position. The incidence and severity of iatrogenic V_2 paresthesia is much less with endoscopic approach compared to open one.

5. Endoscopic Dacryocystorhinostomy (EDCR)

The concept of intranasal DCR is not new. The approach was introduced by Caldwell,[42] almost one hundred years ago but failed due to difficulties in visualization. In 1974 Jokinen and Karza[43] revived the endonasal approach for the lacrimal system. New operating microscopes and endoscopic telescopes,[44] provided better illumination and magnification for transnasal lacrimal surgery. EDCR allows drainage of an obstructed lacrimal sac without the need for a skin incision. The fiberoptic light is passed through the canaliculi to identify the lacrimal sac. The medial wall of the canal is removed by a drill, curette or laser.[45] The use of drills rather than

laser is recommended because drills can create a wide exposure of both sac and the duct without heating the surrounding bone at the edge of the opening.[46] Built in suction at the resection site increases visibility and maneuverability while irrigation minimizes bone heating. Anterior and posterior flaps of the sac mucosa can be rotated over the bony edges and screwed in place with a small bipolar weld. The past decade has seen a tremendous increase in the use of endoscopic surgery for the correction of primary and recurrent lacrimal obstructions.[47] Key points for localization of the lacrimal sac include the point of insertion of root of the middle turbinate on the lateral nasal wall, and the maxillary line. The lacrimal sac is located lateral to the maxillary line at its superior aspect. It may be handy to introduce a 20-gauge vitreoretinal fiberoptic endoilluminator into the superior or inferior canaliculus after punctal dilatation. This facilitates visualization and identification of the anatomical structures like lacrimal sac by transillumination. The endoilluminator is advanced gently until a hard stop that signifies the lacrimal bone medial to the lacrimal sac.

In revision cases of EDCR, it is important to create a generous bony rhinostomy extending from "above the middle turbinate attachment" to the level of "midpoint of the maxillary line". In revision procedures many anatomic structures like nasal mucosa, medial wall of the lacrimal sac, the lateral portion of the lacrimal sac containing internal punctum, and orbital soft tissue, are incorporated into a zone of cicatrial tissue that occludes the ostium. Any vigorous avulsion of this cicatrix can injure the common canaliculus and medial canthal tendon. It is therefore, recommended that the surgeon closely observe the medial commissure while gentle traction is placed on the tissue at the rhinostomy site. Reports have confirmed the value of transnasal DCR as a highly successful alternative to external DCR in children with persistent distal nasolacrimal system obstruction.[48-49] Earlier removal of silicone tubing (3-6 weeks) is recommended in children to decrease the incidence of DCR failure due to granuloma formation at the nasolacrimal fistula site.[50]

5.1 Advantages of EDCR over external DCR

1. Absence of facial incision.
2. Preservation of integrity of orbicularis oculi muscle and medial palpebral ligament which form the functional lacrimal pump mechanism.
3. Simultaneous correction of any intranasal pathologic conditions which may contribute to EDCR failure.

5.2 Indications for primary EDCR

1. Tearing and infection associated with primary acquired NLD obstruction.
2. NLD obstruction secondary to inflammatory and infiltrative disorders.
3. Lacrimal duct injuries associated with sinus surgery and facial trauma.
4. Atypical dacryostenosis.

5.3 Contraindications for EDCR

1. Suspected neoplasm involving lacrimal outflow system.
2. Relative contraindications include large diverticulum lateral to lacrimal sac, common canalicular stenosis and large lacrimal system stones.

5.4 Complications of EDCR

1. Formation of synechiae between lateral nasal wall and middle turbinate or nasal septum.
2. Lacrimal sump syndrome (accumulation of lacrimal debri in inferior portion of the lacrimal sac due to gravity, and its inadequate removal.
3. Bleeding.

5.5 Predicaments of successful EDCR

1. Adequate rhinostomy at a proper place.
2. Sufficient bone removal inferior to the level of sac-duct junction.
3. Due respect to the nasal-mucosal structures.

6. Endoscopically Assisted Conjunctivodacryocystorhinostomy (ECDCR)

In CDCR, a fistula is created from medial commissural conjunctiva into the nasal cavity. Pyrex glass tube (Jones tube) is placed in the fistula in most cases. Leaving the posterior half of the caruncle in place protects the medial bulbar conjunctiva from inflammation due to contact with Jones tube orifice. The success rate of ECDCR is 60% to 99%, compared to 80% to 90% in external CDCR.[51]

6.1 Indications of ECDCR

1. Canalicular agenesis.
2. Canalicular obstruction.
3. Common Canalicular obstruction.
4. Lacrimal pump dysfunction e.g. facial nerve palsy

6.2 Advantages of ECDCR over external CDCR

1. Less operative time.
2. Less blood loss.
3. Higher primary success rate due to better visualization of intranasal landmarks.
4. Successful confirmation of the tube placement by endoscopy.[51]

6.3 Complications of ECDCR

1. Tube migration, which may be internal or external. Internal migration leads to closure of conjunctiva over the tube orifice and epistaxis; while external migration leads to conjunctival and corneal inflammation.
2. Pyogenic granuloma

7. Optic Nerve Injury and FESS

Traumatic optic neuropathy may be direct or indirect. Direct traumatic optic neuropathy results from penetrating injuries where the intra-orbital portion of the optic nerve (ON) is generally injured. Indirect traumatic optic neuropathy is due to blunt head trauma with or without associated fractures of the orbital canal. The ON is at risk of injury within the sphenoid sinus, especially if there is a thin piece of bone or mucosa, separating the nerve from the sinus cavity.[52]

If the ON is transected, MRI does not show any cerebrospinal fluid (CSF) surrounding the ON. The leaked CSF in the inferior part of the orbit wall gives a diffuse high signal. The aerated posterior ethmoidal cells (cells of Odoni) when present, put the ON at a greater risk of injury during ethmoidectomy. Odoni cells falsely suggest to the surgeon a more posterior location of the ON than its actual location.

Dilatation of the pupil on operating table may indicate a direct injury to the ON. The cause may either be ocular ischemia or damage to the pupillomotor nerves. Severe orbital hemorrhage with secondary compressive optic neuropathy may take few hours to cause RAPD. It is advised that all patients in the recovery room should have pupillary examination done.

Optic Nerve Decompression

Trauma is the most common cause of optic neuropathy that demands its decompression. It is best performed using a drill.[53] Traumatic visual loss is the most common indication for optic nerve decompression. Decompression is not indicated in direct traumatic optic neuropathy resulting from penetrating injuries, where intra-orbital portion of the ON is injured. Decompression is also not indicated if there is complete disruption of the ON; however, in edema or hematoma of the ON, decompression may be considered. No clear benefit has been found for either corticosteroid therapy or the surgical optic canal decompression. Based on international optic nerve trauma study and literature review, the treatment is determined on individual patient basis.[54]

The cause of blindness can be due to compressive optic neuropathy resulting from hemorrhage within the bony optic canal or within the meningeal sheaths of the nerve. In case visual loss does not improve with steroids, the bone may be removed from the optic canal with a diamond blur under excellent visualization of 180 degrees or more.

ON decompression in pseudotumor cerebri and ischemic optic neuropathy is usually limited to the medial and inferior portions of the bony optic canal.

Endoscopic orbital apex decompression involves removal of medial and portion of the orbital floor.[25]

Endoscopic ON decompression appears to be most successful in patients with non-traumatic compressive optic neuropathy like benign tumors and inflammatory lesions. The length of optic canal to be decompressed depends upon its indication e.g. the size and location of the neoplasm causing compressive optic neuropathy. In traumatic optic neuropathy and dysthyroid orbitopathy, removal of 10mm of bone posterior to the sphenoid sinus is usually adequate.[55] Improvement following ON decompression may be immediate (due to relief of mechanical conduction block) or late (weeks to months), due to remyelination and consequent normal efferent conduction.[56]

It is mandatory to check visual acuity, orbital appearance and ocular motility after FESS.

Endoscopic ON decompression preserves the olfaction and ensures better visualization, early recovery, and no scarring. While thinning the medial wall of the optic canal with a drill, care must be taken to prevent contact with the carotid artery prominence which is situated just infero-posterior to the ON. Care should also be taken to avoid thermal damage to the ON as a result of heat generation during drilling.

8. FESS and Extraocular Muscles (EOM)

Detachment of the EOMs from the globe and there retraction into the posterior orbit can occur secondary to trauma or as a surgical complication. The muscle(s) may be ruptured or transected as a result of the injury.[57-58] It is possible that a small defect in the orbital wall (not detected by CT) could allow a powered cutting instrument to aspirate orbital fat and or EOM into the sinus without entering the orbit.[8] Loss of a rectus muscle may also occur as a complication of strabismus surgery,[58] retinal detachment surgery,[59] orbital surgery,[8] or paranasal sinus surgery.[59]

The clinical signs of a lost rectus muscle include a large unexpected under correction or overcorrection post strabismus surgery, mild exophthalmos, widening of the interpalpebral fissure, marked limitation of ductions in the field of action of the lost muscle and abnormal head position.

Traditionally, patients with hyphema and EOM entrapment are extremely difficult to manage because they require globe retraction, which in itself increases the risk of raised IOP, secondary hemorrhage and visual loss. The transmaxillary endoscopic

approach offers an excellent solution without any need for globe retraction. Also conventional non-endoscopic surgeries are often delayed increasing the chances of permanent damage to the EOM due to strangulation.

The most commonly lost EOM from any cause is the Medial Rectus (MR).[57] Other muscles reported to be injured during FESS are inferior rectus and superior oblique.[60-61] The injury to the superior oblique occurs due to direct drawing of the orbital periosteum in the area of the muscle, into the suction port of the drill, producing traction on the trochlea. Strabismus surgery should be preceded by orbital exploration and release of entrapped tissues, if possible. Surgical retrieval of the lost MR muscle may be quite difficult because the muscle has no fascial attachments to oblique muscles. When MR muscle is lost, it tends to recoil into the orbit, posterior to where the recti muscles penetrate the Tenon's capsule. MR lies close to the LP. The injury may damage the muscle itself, its nerve and vascular supply or combination. The injury to the MR muscle presents clinically as a gross divergent squint and almost absent adduction. Delayed recovery of the adduction explains the nerve injury and the time taken to regenerate.

Plager and Parks[59] have reported retrieving only one of ten lost MR muscles. They advise against trying to retrieve a lost MR muscle as the search may result in fat adherence syndrome.

The transnasal endoscopic approach to retrieve a lost MR muscle involves infracturing of middle turbinate, removing the uncinate process, ethmoidectomy, removing LP to the level of anterior and posterior ethmoidal arteries and incising the periosteum after exposing the periorbita.

The medial orbitotomy route for external approach involves a modified Lynch incision along the inferior margin of the eyebrow extending down halfway between the inner canthus and the anterior aspect of the nasal bone. The anterior ethmoidal artery should be coagulated and divided. The periorbita is incised and dissection carried into the fat until MR is identified and grasped with forceps through the Lynch incision. The muscle is held with the Alligator tooth forceps through the sub-tenon incision and reattached to the globe with 6-0 polyglactin.[62] Absence of MR muscle may require medial transposition of vertical recti but not without a risk of anterior segment ischemia. Any muscle when injured by the powered dissection is seldom surgically repairable. The rotating cutting jaws churn a portion of the muscle, fraying the remaining ends, which are not possible to approximate with perfect anatomical landmarks. In such cases a fused vision in primary gaze may be accepted as a surgical success. The new powered endoscopic sinus devices reduce the risk of hemorrhage but have a greater potential for damaging EOMs if used by inexperienced surgeons or misdirected.[63]

If on imaging there is lack of evidence of structural damage to the orbital contents and EOMs but clinically there is an obvious ocular motility dysfunction, it may be an indication of a vascular or nerve injury to the muscle. For management of multiple muscle injuries, multipositionl MRI can be used to demonstrate muscle contractility in various gazes and determine if the transected muscle is functioning, and suitable for primary repair. CT imaging is not indicated here due to radiation exposure hazard. Also CT may not detect edema of the muscle in acute or subacute stages. Iris angiography is useful to avoid anterior segment ischemia.

Guidelines for treating muscle injuries during FESS.[60]

1. In cases where the involved muscle is intact, but paretic due to contusion injury or the injury is to its nerve, no immediate surgical intervention is recommended. The antagonistic rectus muscle should be injected with botulinum toxin while awaiting recovery of the paretic muscle to avoid its contracture.
2. In cases with transection injury, if the remaining posterior segment of the MR muscle is longer than 20mm and is functional, muscle recovery via an anterior orbital approach should be attempted.
3. In cases of muscle destruction, muscle transposition procedures may be helpful and the potentially hazardous posterior orbital explorations avoided.

Complications of FESS

The orbit is at risk of complications during FESS because of the following important clinical anatomical relations with the PNS and skull base:

1. Orbit is the lateral margin of the ethmoid area.
2. LP is a thin bone and can be fractured easily resulting in fat herniation, intra-orbital bleeding and EOM damage.
3. Posteriorly, ON is at risk as it lies in a more medial plane, and closer to the lateral wall of the posterior ethmoid cells (Onodi cells) and sphenoid sinus.
4. Superiorly the ethmoid artery is at risk of transaction.
5. Lacrimal duct is susceptible to damage because it lies just anterior to the uncinate process.

Orbital involvement in endoscopic sinus surgery occurs in 0.5% [64] to 3% of all procedures, and represents 16% to 50% of all complications. Most common risks encountered in endoscopic sinus surgery include bleeding, infection, injury to the eye and its adnexa, cerebrospinal fluid leak, anosmia etc. Most orbital complications occur when the suction cutting tip of this incredible powered instrument is inadvertently misdirected into the orbit or intracranially. Many minor orbital injuries which would have been trivial with conventional instruments turn into major complications

when powered dissection or suction is used. Any injury or dehiscence of the LP and consequent exposure of periorbital or orbital fat starts the sequence of events for orbital complications. If surgery is not stopped at this stage, the suction forces at the tip of the instrument can draw in exposed tissue, and then severed by the cutting, rotational jaws. This can include the medial rectus muscle and the ON as well. The presence of fat or periorbita in the surgical field can be confirmed by gentle ballotment of the eye and endoscopic observation at the site of the injury.[65]

The stages of surgery during which the orbit may be at the greatest risk of breach include ethmoidectomy or any ethmoid sinus surgery,[66] and uncinectomy for the medial orbital wall, and maxillary antrostomy plus sinusotomy for the inferior orbit. This is so because the LP, especially at the extremes of age, is very thin, and may be incomplete in some patients. The LP is especially at risk when approached from the nasal space through a diseased sinus. Anatomical variations of the sinus,[15] and lack of tactile feedback from mechanized systems add to the risk of orbital complications.

Overall the orbital complications of FESS can be divided into minor and major complications. Minor complications include; periorbital ecchymosis, orbital emphysema, transient diplopia, edema and formation of lipogranuloma.[67]

Major complications include; Extra-ocular muscle injury, persistent diplopia, nasolacrimal duct injury, orbital hemorrhage or hematoma, orbital foreign body, optic nerve injury, blindness, subperiosteal abscess, abscess of the orbital tissue, orbital cellulitis, cavernous sinus thrombosis, enophthalmos, injuries to the vascular and nervous structures of the orbit,[68-69] and orbital emphysema leading to blindness.[70] Open globe injury to the eye during endoscopic surgery, probably secondary to perforation of the LP, and entrance into the orbit and the globe by the electrocautery tip has been reported by Castellarin et al.[71] The injury resolved spontaneously without any surgical intervention. The clinical sequel of orbital injuries can range from pain, diplopia to blindness.

1. Orbital Hematoma

Orbital hematoma is an ophthalmic emergency, because an intraorbital bleed can rapidly produce an orbital compartment syndrome (visual loss, external ophthalmoplegia, tense orbit, central retinal artery occlusion) with permanent injury to the optic nerve if ischemia persists more than 90 minutes. The source of bleeding may be injured lamina papyracea, periorbita, extraocular muscles, or traction on the orbital fat resulting in avulsion of an orbital vessel. Orbital hemorrhage may be rapid from arterial bleeding (most likely source is the anterior ethmoidal artery, which can be easily encountered posterior to the frontal recess) or slow due to venous ooze.[72] Typically this artery is located within the skull base; however, it can project from the skull base. When injured, the artery retracts into the orbit causing rapid hemorrhage into a confined space, resulting in orbital hematoma. This complication is less likely to occur with the posterior ethmoidal artery because of its location near anterior roof of the sphenoid, and it's less accessibility to the instruments. If bleeding arteries cannot be approached endoscopically, external incisions may be necessary to ligate them manually. Following FESS, CT orbits is indicated to assess the status of the globe.

Early signs of an orbital hematoma include preseptal edema, ecchymosis, orbital proptosis and raised IOP. Bleeding in the field of surgery makes it very difficult to distinguish between important anatomical landmarks like mucosa, periorbita and orbital fat. Orbital hemorrhage is usually intraorbital, but subperiosteal orbital hematoma has been reported.[73]

Fundus examination is an important part of FESS when assessment of the blood flow to the optic nerve head is indicated. In normal blood flow, digital pressure on the globe raises orbital pressure above the diastolic pressure so that retinal arteries pulsate. Orbital hemorrhage may raise the orbital pressure to this level so that vessels flash spontaneously. If the orbital pressure rises above systolic pressure, as can happen in severe intraorbital hemorrhage from anterior ethmoidal artery bleeding, the arteries will close so that no pulsations can be seen. If this episode lasts over two hours, two phenomena can occur:

1. Boxcarring (stagnant flow creating intra-arterial clots) of the retinal blood vessels.
2. Cherry red spot, which if persists for 90 to 120 minutes, can lead to irreversible retinal ischemia.

Management

Not all patients with orbital hemorrhage require surgical intervention; however, the role of an ophthalmologist in such cases is undisputable. If the IOP is <30mmHg, the vision is normal and the optic nerve head circulation is adequate, the patient can be observed; however, if IOP is high, eye massage, intravenous dexamethasone and topical beta-blockers should be given. Anterior chamber paracentesis, hyperosmotic agents generally are not useful.[74] Lateral canthotomy with inferior cantholysis should be done if IOP in anesthetized patient is more than 40mmHg or if a conscious patient complains of severe retrobulbar pain associated with signs like Marcus-Gunn pupil and cherry-red macula. A 10 to 20 mm incision from lateral commissure through the lateral canthus instantly reduces IOP by approximately 14mmHg. [75] Releasing the lateral canthal tendon[76] from its attachment to Whitnall's tubercle reduces IOP by an additional

19mmHg. Although frequently advocated, lateral canthotomy and cantholysis may be insufficient to treat a major orbital hemorrhage. In such cases subciliary incision, transorbital decompression with fenestration of the periorbita ensuring fat prolapse and evacuation of hematoma may be required. Transcaruncular orbitotomy can identify ethmoid arterial bleed and pave way for medial wall decompression.[77] Additional decompression of the orbital floor and lateral orbital wall is also possible. As a matter of fact, it is rare that an ophthalmologist will be immediately available in operation theatre (unless everything is planned), and if fortunate to have one, he or she must be experienced in orbital surgery. Also not all otolaryngologists are likely to be familiar with newer transcaruncular approaches for orbital decompression. Under these circumstances it is desired that the operating otolaryngologist should be familiar with primary treatment for orbital hemorrhage like canthotomy and catholysis.

2. Diplopia

15% to 63% of postoperative FESS patients report new onset diplopia or worsening of pre-existing symptoms of diplopia.[25,6] Diplopia is due to change in the vector of pull of EOMs. Decompressive surgery rarely alleviates pre-existing diplopia, and those who develop diplopia after decompression surgery often need strabismus surgery.

Guidelines to decrease or avoid diploia after FESS:

1. Strut preservation of inferomedial bone between decompressed floor and medial wall.[78]
2. Maintenance of the facial sling in the region of MR muscle.[79]
3. Balanced decompression (concurrent medial and lateral wall).[54]

However, techniques designed to limit diplopia also limit the extent of decompression, and postoperative diplopia is often accepted as a concession to improve visual acuity and other more serious complications.

3. Epiphora

The incidence of this complication after FESS ranges from 0.3% to 1.7%. [80] It is more likely to occur if maxillary antrostomy is extended too far anterior with transection of the nasolacrimal duct, hence EDCR is the remedy. Surgery on frontal sinus may damage lacrimal sac, whereas uncinectomy or middle meatal antrotomy may injure the nasolacrimal duct within the lacrimal canal.

4. Complications Related to Optic Nerve Sheath Decompression

These may include damage to the optic nerve fibers, ophthalmic artery, CSF leakage, meningitis etc. clear risks and absence of the data to suggest benefits of sheath decompression do not recommend this procedure in general.

5. Neuro-opthalmic Complications

Anisocoria and accomodation palsy have been reported after endoscopic surgery.[81] These probably occur due to perineural edema caused by intra-maxillary manipulation around LP and / or damage to the parasympathetic fibers within the oculomotor nerve or ciliary ganglion.[20] Spread of local anesthetic agent can also cause anisocoria lasting for few hours. [82] There is a favorable response to oral corticosteroids. Image aided neuro-ophthalmic procedures have made the procedure safer and improved the prognosis. A detailed review of the intraoperative use of computer aided surgery can be found from the American academy of otolaryngology at the given website.[83]

Following are the general guidelines for intraoperative prevention of complications:

1. Thickness, contour and presence of infraorbital or supraorbital structures should be identified.
2. Anterior ethmoidal artery is a critical structure to identify in order to avoid intra-operative bleeding. Coronal CT images show a bony nipple at the junction of the medial rectus and superior oblique muscles to identify a useful landmark for the location of this artery.
3. Identification of sphenoethmoidal cells (Onodi), which occur in 8% to 14% of the general population before FESS is critical. Mistaking Onodi cells for the sphenoid sinus can lead to incomplete dissection and place the optic nerve and the orbit at risk.
4. Both optic nerve and carotid artery form an indentation in the lateral wall of the sphenoid sinus. This can be unilateral or bilateral. 5%-7% of these have dehiscent bone which exposes these two vital structures to the intraoperative injury. Preoperative imaging in the axial plane reveals excellent detail of the sphenoid sinus and its relationship with these two structures, thus avoiding iatrogenic complications.
5. IV anesthesia, relative hypotension and relative bradycardia minimize intraoperative blood loss.
6. Topical decongestants, prothrombotic agents and bipolar cautery should be available.
7. Inspection of periorbita and periorbital fat if LP is violated. If periorbita is not injured and there are no signs of orbital injury, surgery can proceed. If periorbita is cut, and orbital fat is exposed, intraocular pressure measurement and forced duction test should be performed.
8. Blind cautery of the periorbital fat should be avoided to prevent injury to the EOMs and the ON. Bipolar electrocautery works well where bleeding does not involve the orbit itself.

9. It is wise to keep the eyes uncovered during endoscopic surgery so that surgery can be stopped immediately if there is any indication of orbital swelling, afferent pupillary defect or eyelid bruising.
10. Do not use nasal packing over the exposed orbital apex to avoid pressure on the ON.

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