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Review Article

An overview on the role of epiglottis in obstructive sleep apnoea

Vijaya K. Paramasivan[1], Mithra S. John[2]*

[1]Department of ENT, Madras ENT Research Foundation, Chennai, Tamil Nadu, India
[2]Department of ENT, Pushpagiri Institute of Medical Sciences and Research Centre, Thiruvalla, Kerala, India

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***Correspondence:**
Dr. Mithra S. John,
E-mail: drmithrasara@gmail.com

ABSTRACT

Obstructive sleep apnoea (OSA) is common sleep disorder with its multimodal effect on health. OSA, if untreated can lead to various cardiovascular, cerebrovascular, psychological, cognitive as well as sexual morbidities. Continuous positive airway pressure (CPAP) device is considered as the gold standard in the treatment of OSA. Epiglottic collapse (EC) in OSA has evolved an important factor in the management of OSA as the CPAP may worsen the sleep apnoea. EC in itself is classified into primary or secondary, partial or complete, anteroposterior or lateral. Epiglottic collapse is considered as one of the causes for poor adherence to CPAP devices. There are newer diagnostic modalities to diagnose and distinguish primary and secondary epiglottic collapse of which interventional drug induced sleep endoscopy plays a viable and important role. CPAP still serves as a primary treatment modality for multilevel OSA with EC. Surgical treatment modalities for EC is aimed at improving the compliance of CPAP as well as for relieving the obstruction. Surgical options primarily include epiglottectomy, glossoepiglottopexy, epiglottis stiffening operations.

Keywords: Epiglottis, Obstructive sleep apnoea, Interventional DISE, CPAP, Surgeries for epiglottic collapse

INTRODUCTION

Obstructive sleep apnoea (OSA) has become a fairly common condition among the adult population with a prevalence of around 11%, with males being 14% and females around 6% in India.[1] This prevalence rate is comparable to the international rates of around 10% of which approximately 30% undergo collapse at the level of epiglottis.[2] Epiglottis, one of the unpaired cartilages of larynx has emerged as an important contributor to upper airway obstruction in OSA syndrome.[3,4] Currently OSA is a well-known entity, which if left untreated can lead to various spectrum of neurological, cardiovascular, metabolic disorders, depressed quality of life, road traffic accidents and death.[5-11] OSA is a part of sleep disordered breathing spectrum characterized by repeated episodes of partial or complete obstruction of upper airway at single or multiple levels despite breathing efforts. OSA is currently classified as mild, moderate and severe largely based on apnoea hypopnea index (AHI).[12] However other parameters routinely considered in assessing OSA severity are oxygen desaturation index, lowest oxygen saturation, daytime sleepiness and other co morbidities.[13]

Hence diagnosing and management of OSA is of utmost significance to an ENT surgeon. Earlier The surgical treatment for OSA used to be uvulopalatopharyngoplasty (UPPP). With its success rate of 24-66% in moderate to severe OSA, interest was shifted to identifying the exact site of obstruction as well as other non-anatomical causes for OSA like poor neuromuscular coordination, low arousal thresholds and high loop gain.[14-16]

Patients with epiglottic collapse alone tend to have lower BMI and AHI values compared to patients with multi-level obstruction. Generally, patients with isolated epiglottic collapse have only mild to moderate OSA when compared to OSA with multi-level obstruction.[17]

TYPES OF EPIGLOTTIC COLLAPSE

Obstruction at the level of epiglottis during OSA can be classified as partial or complete, primary or secondary. Primary epiglottic collapse is again divided into anteroposterior (AP) collapse (floppy epiglottis) and lateral collapse. Primary collapse is one where the epiglottis obstructs the laryngeal aditus independently creating a trapdoor phenomenon whereas secondary collapse is one where the tongue base pushes the epiglottis posteriorly towards posterior pharyngeal wall.

Epiglottic collapse (EC) can be isolated or a part of multi-level obstruction. Among the types of epiglottic collapse complete AP collapse is more common (16.8-65.4%) than incomplete AP collapse (3.8-26.1%).[18] Lateral collapse is much rare compared to AP collapse.

Significance of epiglottis in evaluation and management of OSA is still evolving and there is still no defined consensus regarding the same. Hence this article is aimed at providing a contemporary outlook exclusively on the role, evaluation and various treatment options; of epiglottic obstruction in OSA.

ETIOPATHOGENESIS

Epiglottis in made of elastic cartilage attached inferiorly to the inner surface of inter laminar angle of thyroid cartilage via thyroepiglottic ligament. It is a leaf shaped cartilage with free superior border. It is attached to hyoid via hyoepiglottic ligament, the medial part of which lies beneath median glossoepiglottic ligament. Lateral glossoepiglottic folds, like their median counterpart are mucosal folds that connect epiglottis to the tongue base.

Laxity of epiglottis leading to epiglottic collapse has been attributed to various factors like the shape and length of epiglottis, degeneration of hyoepiglottic ligament, variations in the position of hyoid bone with respect to epiglottis.

Shape of epiglottis

There are classically 3 shapes of epiglottis, considering the superior part of epiglottis - omega shaped, normal epiglottis with anterior convexity and flat epiglottis. In one study, omega shaped epiglottis in which the angle between the lateral halves of superior surface of epiglottis is less than 90 degrees and the normal epiglottis are less prone for collapse.[19] Flat epiglottis were more prone for collapse and trapdoor phenomenon. The flat shape of epiglottis partially recovered its anterior convexity on doing Esmarch's jaw thrust manoeuvre during DISE. This highlights the role of oral appliances in selected cases of OSA due to epiglottic collapse. There is a fourth shape suggested by the authors where the superior border is curved forward and the rest of the free epiglottis is flat. This shape, they suggest is transitory shape before becoming completely flat.

Length of epiglottis

In one study, epiglottic length more than 16.6 mm; measured using drug induced CT scanning were more prone for collapse when compared to those less than this value.[20] In the same study they evaluated the significance of the angle between the long axis of epiglottis and the vertical plane which however was found to be insignificant.

Degeneration of hyoepiglottic ligament

Hyoepiglottic ligament attaches the epiglottis to the medial and superior border of the body of hyoid. It is suggested that long standing pressure on this ligament by tongue base can lead to degeneration and lengthening of hyoepiglottic ligament thereby resulting in epiglottic collapse mostly secondary to tongue base obstruction.[18]

Position of hyoid bone

One of the significant parameter of cephalometric evaluation in OSA is hyoid to mandibular plane length (H-MP). OSA of varying severity are associated with H-MP value more than 15±3 mm.[21] Position of hyoid bone in relation to various pharyngeal structures plays a vital role in the degree of obstruction of tongue base and epiglottis. This is proven by the improvement in OSA symptoms in patients who underwent hyoid repositioning surgeries like hyothyroidopexy.

Laryngomalacia

In a subset of adult epiglottic collapse laryngomalacia has been suggested as a cause.[18] Adult laryngomalacia is mostly idiopathic, post traumatic or post-operative.[19]

DIAGNOSIS

Involvement of epiglottis alone or along with other pharyngeal structures needs to be clearly identified before initiating the management. Using an oral device like mandibular advancement device or continuous positive airway pressure device (CPAP) when epiglottis is involved won't be always beneficial, it could even worsen the condition. Hence the role of investigations in diagnosis cannot be over emphasized.

Anatomical evaluation by awake flexible NPL scope is routinely done as an OPD procedure to rule out any sites of mechanical obstructions and to perform Mueller's manoeuvre to give a clue regarding the possible site of obstruction; but it has now lost its significance owing to poor diagnostic value.

Polysomnography

Overnight polysomnography (PSG) is the gold standard in diagnosing OSA wherein type, degree of sleep apnoea, oxygen desaturation, sleep fragmentation, arousals,

respiratory efforts in addition to several other criteria are assessed. As a common thought it was not considered to distinguish between epiglottic and non epiglottic collapse. However, in a study by Azarbarzin et al, flow characteristics in airflow could be utilised in differentiating epiglottic collapse from the non epiglottic ones.[22] In this study, PSG was done along with nasopharyngoscope placed just above epiglottis to visually confirm if the flow characteristics were correlating with true epiglottic collapse. They found that when there is epiglottic collapse, there is well defined reproducible intrabreath airflow characteristics in epiglottic collapse namely, rapid changes in inspiratory flow discontinuities, increased jaggedness, fluttering of airflow and reduced tidal volume when compared to patients with non epiglottic collapse. In a subset of patients, for validation of their study, pneumotach flow and nasal pressures were measured simultaneously which showed correlation. The discontinuity indices, jaggedness and fluttering of inspiratory flow correlated with transformed nasal pressure values. This study suggests that epiglottis collapse can be reliably diagnosed in the sleep lab itself. They even suggested a "classic" snoring sound in patients with epiglottic collapse.

Radiological investigations

Various radiological investigations including lateral cephalometry, CT and MRI has been suggested, but they play only supplementary roles in diagnosis. As mentioned above, length of epiglottis may determine its collapsibility. In this study done on 36 patients with moderate to severe OSA, CT scan in awake state, and under sedation were taken from the level of orbital floor to the hyoid bone20. The length of epiglottis and epiglottic angle were measured in the awake state. Epiglottic collapse (EC) and postero inferior movement of hyoid were measure in the sedated state (dynamic CT). Mid sagittal plane was used to measure the epiglottic length from tip to the base of epiglottis. The angle of epiglottis to the vertical axis was also measured. This study found out that the role epiglottic angle in EC was statistically insignificant. In this study, a cut off value of 16.6 mm of epiglottic length had 100% sensitivity and 65.22% specificity for predicting epiglottic collapse. Again in dynamic CT, there were 2 classifications such as epiglottic collapse (EC) and non epiglottic collapse (NEC). EC is where there is antero-posterior (AP) movement of epiglottis and contact with posterior pharyngeal wall (PPW). NEC is where either there is no AP moment of epiglottis or there is AP movement with no contact with PPW. They analysed the posteroinferior movement of hyoid bone and found that it is more in EC compared to NEC; 4.8±3.7 and 3.0 ±3.7 mm respectively. The drawback of this study is that, they couldn't correlate their findings with DISE due to medical cost. However, an awake uprise flexible NPL scope was done prior to CT scanning to visualize the glottis and determine the degree of concealment of glottis by epiglottis. There was no correlation between the degree of concealment and epiglottic collapse.

Drug induced sleep endoscopy

Drug induced sleep endoscopy (DISE), introduced by Croft and Pringle provides a targeted means of diagnosing sites of obstruction in OSA as it is done in an artificially induced sedation. The sedative agents commonly used are propofol, dexmedetomidine and midazolam; given as a bolus or target controlled infusion. When dealing with an epiglottic collapse in OSA, there are certain roles for other sites of obstruction. In the Starlings resistor model, obstruction at one site can create more negative pressure downstream. So, when there is palatal obstruction, negative pressure downstream develops which; in turn can result in collapse at the level of oropharynx, tongue base and/or epiglottis. Hence when there is collapse of lateral pharyngeal wall, epiglottis or tongue base along with palatal obstruction, which is almost always universal, it implies it could be secondary to palatal involvement. In such a scenario, placing a nasopharyngeal tube (NPT) while performing DISE can actually eliminate this downstream negative pressure development; as it plays as a stent in keeping the retropalatal region open.[23] It even simulates a scenario following successful palatal surgery; to correctly diagnose a primary epiglottic collapse. When NPT was used while performing a DISE rightly known as interventional DISE; there was partial to complete reduction in downstream airway collapse in patients with multi-level obstruction.

TREATMENT

Non-surgical

Non-surgical treatment options for epiglottic collapse are CPAP, oral appliances and positional therapy.

Continuous positive airway pressure

The primary gold standard treatment for OSA of any severity is continuous positive airway pressure (CPAP) which usually results in admirable improvement in OSA symptoms as well as AHI improvement; even though the non-adherence of CPAP usage is around 38.4% for short term and 33.6% for long term.[24] However, usage of CPAP in epiglottic collapse is still controversial.

The anatomical orientation of epiglottis is such that when CPAP tries to splint the airway, a lax and large epiglottis tends to fall backward and thereby obstructing the laryngeal inlet. However, it is found that not all case of epiglottic collapse is associated with CPAP failure. In a study by Sung et al, it was found that the there was no significant difference in the value of mean CPAP pressures and residual AHI in epiglottic group and the control group.[25] This could be attributed to the fact that AP length of epiglottis is longer that the AP length of larynx. Hence when epiglottis falls backwards it leaves a triangular slit between the lateral surface of epiglottis and posterior wall of hypo pharynx through which ventilation can occur. CPAP adherence however is poor in patients with

epiglottic collapse compared to non epiglottic group. This could be attributed to choking sensation that arises when the epiglottic falls back and closes the larynx. Also, the patient with epiglottic collapse usually have lower BMI and AHI compared to patients with multi-level obstruction. Such subgroup of patients was usually associated with poor adherence to CPAP.

This does not mean that all epiglottic collapse OSA patients have CPAP failure. A large subset of patients with EC tolerate CPAP well with substantial improvement in AHI. This can be attributed to the following factors: some studies have shown that clinically significant EC is much lesser than that diagnosed through DISE; and a bilateral slit between lateral vallecular and epiglottis resulting from EC could still serve the function of an airway.

In another study, CPAP had varying effects on epiglottis-it could; alleviate a partial collapse, not affect complete collapse or worsen a partial collapse. Epiglottic collapse is not alleviated by CPAP.[26] It may even require higher CPAP pressures (more than 10 cm of H_20). However, the peak inspiratory flow and median inspiratory ventilation normalized with CPAP in all cases regardless of its effect on epiglottis.

Hence considering these study results, CPAP is still considered the first line of management in an OSA patient with EC.

Oral appliances

Mandibular advancement devices, though used rarely has been found to improve epiglottic collapse in a subset of patients with mild OSA.[27] However owing to associated temporomandibular joint arthropathies and dental malocclusions on long term use, its use should be considered only in carefully selected patients.

Positional therapy

Positional OSA is one when there is AHI more than 5/hour with 50% reduction in AHI in non-supine position when compared to supine position. In a subset of patients, epiglottic collapse seems to be dependent on the position of the head and trunk of the patient. Hence positional therapy should be tried for management of mild OSA with epiglottic collapse.[28] Trying the tennis ball technique where a spherical foam or mass is secured in the inter scapular region thereby preventing the patient from lying supine has been tried with good results but poor long-term compliance. Using sleep position trainers where vibrotactile stimuli while supine prompts the patient to assume non supine position has better compliance.

Surgical

Tongue base surgery, complete or partial epiglottectomy, glossoepiglottopexy, epiglottic stiffening operation, tongue base advancement surgeries and maxillomandibular advancement surgeries in addition to hypoglossal nerve stimulation are the various surgical options available for epiglottic collapse.

Epiglottectomy

Complete epiglottecotmy has been abandoned owing to the risk of aspiration. However, a partial epiglottectomy where in a V shaped or U shaped resection of epiglottis is done in its upper part leaving the lateral part; thereby opening the airway and reducing the risk of aspiration, has been tried with good results.[29] This can be done with monopolar cautery, CO_2 laser, coblation or trans oral robotic surgery (TORS). Post-operative complications include bleeding and breathing difficulty which need to monitored closely for a minimum period of 24 hours. Following partial epiglottectomy, the mean CPAP pressures reduces drastically resulting in improved compliance.[29] Hence partial epiglottectomy may be considered when there is multilevel obstruction with primary EC and single level obstruction at the level of epiglottis associated with poor CPAP compliance due to elevated pressure requirements.

Glossoepiglottopexy

In this technique, the mucosa over tongue base, valleculla and lingual surface of epiglottis is removed using coblation, laser or monipolar cautery.[30] Care is taken to retain 5 mm of mucosa around the rim of lingual epiglottis. This is done to retain sensation and prevent complications such as apiration. However, the perichondrium of remaining lingual epiglottis is exposed. This procedure induces synechiae between lingual surface of epiglottis and corresponding area of tongue base and; this can be reinforced by suturing the base of tongue to the epiglottis using absorbable sutures. Transoral glottoepiglottopexy as suggested by Roustan et al is a modification of Monnier's glossoepiglottopexy done for laryngomalacia in children.[31] This method provides stable airway with anatomical and physiological preservation of larynx. This procedure begins with denuding the mucosa over vallecula, tongue base and lingual surface of epiglottis. This is followed by transcutaneously securing the epiglottis to the tongue base using two suture wires passed through 16 gauge needles and secured with a loop of one of the suture wires. Complications include suture breakage, epiglottic laceration, bleeding, dysguesia and dysphagia this method of glossoepiglottopexy is even reversible when complication arise.

Epiglottic stiffening operation

Here the lingual surface of a floppy epiglottis is cauterised with monopolar cautery, laser or coblation from one lateral glossoepiglottic fold to the other including median glossoepiglottic fold exposing the perichondrium; leaving the free edge of lingual epiglottis.[32] The resulting scar formation results in a stiffened epiglottis with improvement of AHI.

Tongue base advancement

Tongue base advancement can be achieved by hyoid suspension via hyothyroidopexy or genioglossus advancement.[33] Stabilization of tongue base inevitably pulls the epiglottis forwards and indirectly prevent the epiglottic collapse. Hyothyroidopexy begins with a skin crease infra hyoid incision, exposure of hyoid bone, release of infrahyoid musculature and strap muscles division. This is followed by exposure of thyroid cartilage. Four non absorbable sutures are from around the hyoid and through the thyroid cartilage, securing the hyoid in an anterior and inferior location. This position results in stretching of hyoepiglottic ligament resulting in widening of hypo pharyngeal airway as well as stabilization of epiglottis.

Genioglossus advancement evolved over the years since its description by Riley et al. It begins with a gingivolabial sulcus incision and exposure of entire length of mandible underlying chin. A rectangular piece of the mandible involving the genial tubercle is defined and cut including the inner and outer cortex. Thus segment is pulled anteriorly and secured with a screw. This pulls the genioglossus muscle anteriorly and thereby stabilizing the epiglottis. Recent changes in this procedure include virtually delineating the genial tubercle, making lateral osteotomies upto inferior border of mandible and making an angulation of 10-20 degrees when lateral osteotomies are made so that larger width of lingual cortex is obtained.[34] Advantages of this technique are better chin aesthetics and larger area of contact between lingual and buccal cortices when the segment is advanced. Even though tongue base advancement procedures theoretically prevent collapsibility of epiglottis, there is still no research into this or proven data for the same.

Maxillomandibular advancement

Maxillomandibular advancement (MMA) involves Le fort I osteotomies of maxilla and sagittal split osteotomy of mandible which are rotated and then advanced. MMA stabilizes the lateral pharyngeal wall, velum and tongue thereby stabilizing the epiglottis too. Improvement of epiglottic collapse in patients who underwent MMA has not been completely elucidated. Studies by Liu et al shows that when multilevel obstruction with EC occur in moderate severe OSA, MMA resulted in equivocal improvement in EC which was confirmed by post-operative DISE and computational fluid dynamics airflow modeling.[35,36] Pre and post-operative DISE following MMA shows no net difference in EC more so in complete anteroposterior collapse in studies by Kastoer et al and Zhou et al.[37,38] However the net effect of MMA on epiglottic collapse can differ based on the type of collapse.[39]

Hypoglossal nerve stimulation

Hypoglossal nerve stimulation (HNS), a neuromodulatory surgical treatment for OSA, includes an implantable pulse generator (IPG) and 2 ports- one for sensing respiratory movements and another for stimulating main or medial trunk of hypoglossal nerve; unfortunately, is still not approved for use in India. Three incisions are made- first between hyoid and mandible to place the stimulating cuff around hypoglossal nerve, second on the upper border of fourth or fifth intercostal muscle to place the sensing lead on to the pleura and the third incision to place the IPG 5 cm below the clavicle medial to deltopectoral groove lateral to the nipple line; all done on the same side. The components are connected to the IPG by subcutaneous tunnelling.

The efficacy in improving EC in multilevel obstruction OSA has been studied by Xiao et al and Heiser et al with appreciable results.[40,41] When hyoid moves forward, epiglottis which is attached to it via hyoepiglottic ligament moves and opens up the lower part of upper airway. Hyoid bone moves anteriorly when there is active contraction of geniohyoid which is innervated by C1. Active hyoid suspension achieved by contraction of geniohyoid can be achieved by inclusion of C1 in the simulation cuff along with hypoglossal nerve. C1 runs along the trunk of hypoglossal nerve until it branches with some variation between individuals. Hence identification of C1 during procedure can be a challenging step. Inclusion of C1 in selective HNS results can treat a floppy epiglottis.[42]

Tongue base surgery

Tongue base; whether lymphoid hyperplasia or muscular hypertrophy has to be addressed to treat the associated secondary epiglottic collapse. Tongue base can be reduced using trans oral robotic surgery (TORS) or coblation assisted tongue base ablation methods. A wedge resection of lymphoid tissue starting from midline at the level of foramen caecum upto a depth of 1.25-1.5 cm laterally where a depth of 0.5-0.75 cm is done; in order to prevent damage of neurovascular structures in the depth of tongue base (lingual vessels and the hypoglossal nerve).[43]

DISCUSSION

The role of epiglottic collapse in the management and treatment of OSA is manifold. Isolated epiglottic collapse in patients with OSA have lower degrees of severity compared to patients with multi-level OSA with EC. EC should be suspected when patient complains of choking sensation during CPAP usage. Diagnosis of epiglottic collapse is mainly during drug induced sleep endoscopy; whether primary or secondary; partial or complete, AP or lateral.

Ideally interventional DISE should be performed when primary EC is visualized during routine DISE. Treatment of EC collapse is multivariate including a trial of CPAP. Surgical options should be considered to improve the compliance of CPAP usage as well as for primary treatment of OSA. Surgical option for primary EC mainly

incudes partial epiglotttectomy, glossoepiglottopexy and epiglottic stiffening procedures.

CONCLUSION

Mechanism and management of epiglottic collapse in OSA is not yet thoroughly elucidated till date due to multifactorial ethology of OSA itself. Detailed evaluation and management protocol must be followed to confirm epiglottic collapse. Managing the epiglottic collapse itself improves the CPAP compliance as well as AHI in a patient with multi-level obstruction. As evidenced by various studies, CPAP is still the first line of management EC even though theoretically CPAP can accentuate the EC. CPAP failure can be managed in a systematic way from positional therapy, oral appliance and to surgical management.

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