



Epiglottic Collapse in Obstructive Sleep Apnea

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Along with the development of diagnostic techniques, many studies have been conducted to find the anatomical causes of obstructive sleep apnea (OSA). The velum, oropharynx, tongue base, and epiglottis have been widely considered to be the common obstruction sites. However, the role of the epiglottis in sleep apnea is poorly understood compared to the other anatomical sites. The epiglottis causes OSA either alone or simultaneously with other obstruction sites. We have here reviewed the epidemiology, pathophysiology, diagnosis, and treatment of epiglottic collapse in patients with OSA based on the literature published to date. **Sleep Med Res 2021;12(1):15-19**

Key Words Obstructive sleep apnea, Epiglottis, Sleep apnea syndromes.

INTRODUCTION

Obstructive sleep apnea (OSA) is the periodic decrease or cessation of airflow during sleep due to an upper respiratory tract obstruction. OSA is a relatively common disorder with a prevalence of 1–2% of the total population, but with a higher incidence in middle age of 4% in men and 2% in women [1]. Untreated OSA is associated with an increased risk of cardiovascular disease, cognitive impairment, metabolic disease, and mortality [2].

First line treatments include continuous positive airway pressure (CPAP) but this approach has a poor long-term compliance of 46–85% and patients often refuse to use it initially [3]. Secondary OSA treatments can include surgery and oral devices. Surgery has proven to be an effective option for reducing cardiovascular morbidity and mortality [4]. Obstruction may occur in all sites of the upper respiratory tract in OSA cases, usually in the soft palate, tongue base and epiglottis. Identifying the obstruction sites and determining the appropriate surgical method increase the success rate of treatments [5].

Drug induced sleep endoscopy (DISE) is also now being used as a new diagnostic tool for selecting therapeutic strategies for OSA [6]. DISE has improved our understanding of the many factors involved in this disorder, one of which is epiglottic collapse. The role of the epiglottis in OSA has been underestimated over the past few decades, but its importance in OSA has emerged and the interests in treatments that target this region have grown in recent years. In our present article, we review the current literature on the epidemiology, pathophysiology, diagnosis, and treatment of epiglottic collapse in OSA patients.

EPIDEMIOLOGY

Depending on its direction and extent, epiglottic collapse is divided into a partial or complete anteroposterior (A-P) or lateral collapse. Most instances of epiglottic collapse in OSA are a complete A-P collapse, with a rate ranging from 16.8–65.4%. This is followed by a partial A-P col-

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lapse, with a frequency ranging from 3.8–26.1%. The earliest studies of epiglottic collapse involved an analysis of the hypopharynx and larynx using a flexible endoscope when the patient was awake. According to a study by Catalfumo et al. [7] involving an awake endoscopy on 104 persistent OSA patients who received uvulopalatopharyngoplasty (UPPP), 11.5% of the cases had an abnormally positioned epiglottis.

As DISE became popular, many studies were conducted to identify the frequent areas of upper respiratory tract obstruction using this method. The reported prevalence of epiglottic obstruction in OSA patients has varied from study to study, ranging from 12.5–73.5%. In a 2015 report by Lan et al. [8], the authors noted 12.5% and 26.6% of their series of 64 OSA patients had a partial and complete A-P collapse, respectively. These authors found no cases of partial lateral epiglottic collapse in their cohort, but 2 patients (3.1%) showed a complete lateral collapse. Koutsourelakis et al. [9] reported that among 49 OSA patients assessed using DISE prior to upper respiratory surgery, 36 cases (73.5%) experienced some degree of epiglottic collapse. In the responder group in that report, i.e., cases with a post-operative apnoea-hypopnoea index (AHI) of less than 10 that was at least a 50% decrease from the baseline, 43.4% of the patients had a complete A-P collapse, 26.1% had a partial A-P collapse and 4.3% had a lateral epiglottic collapse. In the non-responder group, 65.4% of the patients experienced a complete A-P collapse, 3.8% had a partial A-P collapse, and 3.7% had a lateral collapse. A recent study using DISE of 324 patients with both non-apneic snoring and OSA reported a floppy epiglottis (FE) in 60 of the subject patients (18.5%) [10]. In the position dependent group in that report, 19.4% of the patients had a complete A-P collapse and in the non-position dependent group, 16.8% of patients had a complete A-P collapse [10]. Notably, however, no significant correlation was found by the authors between the presence of epiglottic collapse and the presence of position dependence.

Several studies have also been conducted using DISE to find remaining obstructions in OSA patients who have not responded to CPAP or surgery. In one retrospective study of 30 cases of failed CPAP with an AHI of less than 5, 11 patients (37%) showed a persistent A-P collapse, including a collapse at the velum, tongue base, or epiglottis level and a multilevel collapse, and eight patients (27%) in that study had a FE [11]. Kim et al. [12] evaluated their DISE findings in patients with persistent OSA after UPPP surgery. Of the 50 patients included in that study series with persistent OSA, 22 (44%) cases had partial or complete epiglottic collapse. In summarizing the studies mentioned above, it appears that the prevalence of epiglottic collapse in OSA patients who fail airway surgery ranges from 44–72.9%.

Isolated epiglottic collapse refers to an A-P collapse other than a lateral or secondary epiglottic collapse of a multilevel area. Few studies to date, however, have investigated this type of collapse. Golz et al. [13] found that 48 out of the 187 OSA patients they examined using nasopharyngoscopy during sleep had retrodis-

placement of the epiglottis. Of these cases, 27 (14.4%) had an isolated epiglottic collapse, whereas 21 had an airway collapse at other sites. Woodson [14] conducted DISE in 117 OSA patients and reported a 16% rate of isolated obstruction from a ptotic epiglottis. Twelve out of 334 OSA cases (3.5%) were identified using DISE as an isolated epiglottic collapse group in a prior retrospective analysis of 334 consecutive patients with socially disturbing snoring or symptoms of OSA [15]. In summary, therefore, 3.5% to 14.4% of OSA patients show an isolated epiglottic collapse.

Attempts have been made on other studies to identify the obstruction sites in OSA using methods other than DISE or an awake endoscope. In one such report, 48 OSA patients were examined using videofluoroscopy, and epiglottic collapse was observed in 14 cases (29.2%) [16].

PATHOPHYSIOLOGY

The underlying pathophysiology of epiglottic collapse is still not well understood. In the current literature, several mechanisms have been proposed to explain this phenomenon including 1) secondary to anterior-posterior collapse of the tongue base that pushes the epiglottis backwards; 2) a complete isolated anterior-posterior epiglottic collapse, also known as the trapdoor phenomenon; and 3) lateral collapse of the epiglottis due to underdevelopment of the epiglottis itself [10]. These are further discussed below.

Secondary to an Anterior-Posterior Collapse of the Tongue Base

When a bulky tongue base pushes it backwards, the epiglottis blocks the upper airway. Macroglossia, hypotonia, retrognathia, or lingual tonsil hypertrophy can cause the collapse of the tongue base and epiglottis.

Trapdoor Phenomenon

The trapdoor phenomenon, or FE, refers to a collapsible epiglottis that blocks the airway [17]. The reason for the airway obstruction in this instance is a prolapse of the epiglottis into the posterior pharyngeal wall during strong inspiration. The cause of this phenomenon is unknown, but several possible hypotheses can be considered. First, involuntary swallowing during sleep can cause an epiglottic prolapse in some people. Alternatively, some patients with a weakened glossoepiglottic ligament are more prone to an epiglottis collapse than the general population. An unanswered question in this regard is whether an epiglottis of a particular shape is more prone to collapse. The relationship between the shape of the epiglottis and its potential collapse is still controversial. In a prior case-control study involving 11 isolated epiglottic collapse cases and 44 controls, there were no differences found in the epiglottic shape or curvature between the two groups [15]. In another study, however, in which an esophagogastro-

odendoscopy was performed in more than 50 patients, the authors identified a positive association between a concave posterior surface of the epiglottis and the degree of airway collapse, and consequently the severity of OSA [17].

Underdevelopment of the Epiglottis

Laryngomalacia is the most common cause of stridor in infants. In these patients, the epiglottis can easily block the upper airway and cause apnea. Verkest et al. [18] reported a 77% prevalence of OSA in children with laryngomalacia. The proposed mechanisms to date include 1) a delayed maturation or “hypotonia” of the supporting cartilaginous structures of the larynx; 2) redundant soft tissue in the supraglottis; and 3) a foreshortened or aryepiglottic fold [19-21]. Although laryngomalacia resolves with the growth and development of a child, acquired laryngomalacia in adults has also been reported. Adult laryngomalacia is broadly classified into idiopathic [13,17] or postoperative and post-traumatic secondary cases [19]. The mechanism of idiopathic adult laryngomalacia has not been fully elucidated.

DIAGNOSIS

Friedman staging, cephalometry, CT/MR, Müller’s maneuver using a flexible endoscope, and intrapharyngeal pressure manometry are the most widely used diagnostic tools to identify obstruction sites in patients with OSA. The Müller’s maneuver using a flexible endoscope has the advantage of being simple, economical, minimally invasive, and capable of checking the dynamic upper respiratory tract. It is, thus, a commonly used approach in OSA patients to identify the obstruction sites. The Müller’s maneuver method has the disadvantage, however, of low reproducibility and range of interpretations among different examiners. Moreover, all of the tests mentioned above are limited by their inability to accurately represent the obstruction that occurs during sleep because they are conducted while the patient is awake [20,21].

Since Croft and Pringle [22] first described “sleep nasoendoscopy during sedation” in 1991, DISE has been widely used to determine the exact site of the upper airway collapse in patients with OSA. The biggest advantage of DISE is that it allows the direct observation of the obstruction in the upper respiratory tract during sleep [5,6]. This technique has, thus, proven to be a useful method of assessing the location, severity, and pattern of airway obstruction. The velum, oropharynx, tongue base, epiglottis (VOTE) classification is a standardized method for evaluating DISE results and shows excellent intra- and inter-rater reliability [6]. DISE also has several drawbacks, however. First, it is conducted under conditions of pharmacologically induced sleep, not natural sleep. Second, DISE mainly reflects stage 2 sleep i.e., not the entire stages of sleep. Third, it is difficult to simultaneously evaluate the oropharynx and tongue base with DISE. How-

ever, DISE is currently the gold standard tool for assessing OSA.

Videofluoroscopy is a method in which the patient is placed in a supine position to perform induced sleep, and the lateral side is then photographed via fluoroscopy to identify the obstruction site. This method also has some limitations, however. First, it measures only the lateral side and only anterior or posterior obstructions can be checked. Second, it is performed during induced sleep only, as is the case with DISE. However, it is a valuable non-invasive method of observing the entire upper airway tract [16].

Azarbarzin et al. [23] conducted a trial in 2017 to detect distinct airflow patterns in patients with epiglottic collapse. In that study, epiglottic collapse was characterized by a rapid fall in the inspiratory flow, more variable inspiratory and expiratory flow, and a reduced tidal volume. The usefulness of these flow characteristics has not yet been verified, but this test can be performed concurrently with polysomnography during natural sleep, and may thus become widely used in the future.

TREATMENT

CPAP

Interest in epiglottic collapse in OSA has been increasing in recent years, and a variety of therapeutic options have been attempted to resolve this condition. CPAP, the standard treatment method for OSA, has also been attempted, but has shown limited efficacy [15]. The role of CPAP is to stent the upper airway through the application of continuous positive pressure. However, in the case of primary epiglottis collapse, CPAP can exacerbate the airway obstruction by pushing the epiglottis down into the laryngeal inlet [24,25]. There are also some case reports that CPAP failure is related to epiglottic collapse [26].

In a study published by Shimohata et al. [27], the authors reported their results for 17 patients displaying multiple system atrophy during DISE, 12 of whom showed a FE. None of the patients with severe FE (n = 3) improved with CPAP treatment. In patients with mild FE (n = 9), CPAP caused more severe FE during inspiration, reducing the oxygen saturation levels in 2 cases and improved airway obstruction in the remaining 7 patients [25,27]. There have been some case reports of infants with laryngomalacia treated with CPAP [28]. Except for a few instances, however, CPAP generally aggravates the epiglottic collapse, making it problematic to use as a therapeutic method in OSA.

Positional Therapy and Oral Devices

There are several reports on the effects of positional therapy and the use of oral devices to treat an epiglottic collapse. A previous case-control study of 224 patients by Kim et al. [29] compared the clinical characteristics of cases with and without an epiglottic collapse (epi group vs. non-epi group). The epi group cases in that report showed a significantly lower body mass index and OSA severity. In addition, the epi cases were expected

to respond well to the effects of oral devices or positional therapy. Kent et al. [30] reported on the outcomes in 11 out of 35 patients with epiglottic obstruction after oral appliance therapy. Seven of these patients showed persistent epiglottic obstruction, whereas the symptoms improved in the remaining four cases. According to findings of Sung et al. [15], of 11 patients with isolated epiglottic collapse in their study series, 9 cases (81.8%) could be resolved with a mouth open and jaw thrust maneuver, which simulated the use of an oral appliance. Six cases in that study (54.4%) were resolved by turning their heads to mimic the lateral sleeping posture.

Vonk et al. [10] reported from their study that only 4 out of 60 patients with a FE had a persistence of this condition when performing a lateral head rotation. A FE was found in only one subject when the patient was tilted to the side of the head and torso. The FE still existed in 10 of these patients after applying the jaw thrust maneuver. As such, positional therapy and oral devices have been reported to have some effect in alleviating epiglottic collapse, but the research is still limited in this regard and further validation is needed.

Epiglottis Surgery

Partial epiglottectomy

A partial epiglottectomy is a useful treatment for a trapdoor epiglottis and laryngomalacia. The procedure can be performed in various ways, and the epiglottis is excised using a laser or monopolar coagulator. Catalfumo et al. [7] described their use of an endoscopic CO₂ laser partial epiglottectomy. The results of that study indicated that a partial epiglottectomy can increase the cure rate of patients with OSA by 10–15%. In 2001, a partial epiglottectomy using monopolar diathermy was described by Oluwasanmi and Mal [31] who reported four cases treated in this way because of snoring and/or OSA due partly or wholly to a FE.

Golz et al. [13] published the results of a laser partial epiglottectomy in 2000 as a treatment for OSA and laryngomalacia in which a U-shaped wedge is excised in the suprahoid portion of the epiglottis. Polysomnography performed postoperatively in adult OSA patients showed statistically significant improvement in 85% of cases with this therapy. In all of the cases of laryngomalacia treated with this method, the stridor was permanently stopped after surgery with complete cessation of the apneic episodes.

Excessive resection of the epiglottis can lead to aspiration, and insufficient resection runs the risk of being ineffective against OSA. It is difficult to properly determine the volume of epiglottis to cut without causing postoperative sequelae. Bartolomeo et al. [32] described their experiences with a 25-year-old man with laryngomalacia managed using CO₂ laser V-shape partial epiglottectomy. The authors stated that this treatment minimizes the risk of false passage, while ensuring permanent respiratory airflow through the epiglottic V during epiglottic movement.

Epiglottis stiffening operation

The epiglottis stiffening operation was first described in 2019 by Salamanca et al. [24]. In this procedure, the lower half of the lingual side of the epiglottis is cauterized in the area between the lateral glosso-epiglottic folds using suction cautery. This induces stiffening and scar retraction of tissues as a result of secondary healing. There were no patients with complications such as dysphagia or aspiration dysphonia during a follow-up period of between 3 and 15 months.

Tongue Base Advancement

Hyoid suspension (hyothyroidopexy)

Hyoid suspension represents another surgical option for the treatment of epiglottic collapse. Kezirian [33] reported that none of their patients with a suspended hyoid developed epiglottic collapse. According to a prior meta-analysis by Song et al. [34], isolated hyoid surgery reduces the severity of OSA and improves sleepiness in adults. In that review, hyothyroidopexy was reported to reduce AHI by the greatest amount (50.7%), followed by hyoid myotomy with suspension (a 38.3% reduction in AHI) and hyoid expansion (a 7.1% reduction in AHI). Askar et al. [35] performed a voice and swallowing assessment in 17 patients who had undergone hyothyroidopexy. Their comparative analysis of the pre- and post-operative results of voice and swallowing measures revealed a non-significant difference. Hyoid suspension is, thus, a theoretically possible surgical option in OSA, but there are no studies that have proven its direct effect on epiglottis collapse.

Genioglossus advancement

The genioglossus muscle is the most important muscle for maintaining airway patency during sleep. Genioglossus advancement, first introduced in 1984 by Riley et al. [36], increases the muscle tension of the tongue and limits posterior collapse during sleep. Several studies have demonstrated that genioglossus advancement combined with an UPPP achieves an improvement in OSA patients. Complications from this surgery may include hematoma, injury to the genioglossus muscle, and abnormal sensation of the mandible. There is a possibility also that an epiglottic collapse will improve as the tongue base advances, but there is no current research on this.

CONCLUSION

Traditionally, the velum and oropharynx have been considered the main sites of obstruction in OSA patients. As diagnostic tools such as DISE have become popular for upper airway tract examinations during sleep, the critical role of the epiglottis in OSA has been revealed. The prevalence of epiglottic collapse in patients with OSA is likely to be higher than previously described. If there is residual obstruction even after CPAP or

UPPP surgery, an upper respiratory reassessment should be performed to determine the presence and type of epiglottic collapse. Personalized treatments for OSA are, thus, warranted, based on the type of collapse.

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None.

Conflicts of Interest

The authors have no financial conflicts of interest.

Authors' Contribution

Conceptualization: Chung YS. Data curation: all authors. Supervision: Chung YS. Writing—original draft: Bae MR. Writing—review & editing: Chung YS.

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