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Case Report



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A Case Series on Surgical Management of Glotto-Supraglottic Amyloidosis

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Abstract

Benign glottosupraglottic mass is rare. Amyloidosis remains an important pathology that has to be kept in mind while managing such lesions. The ideal management still remains a challenge for the modern otolaryngologist, with many cases recurring due to associated risks of anterior glottic webs and subglottic stenosis. In this article, we emphasise two cases with glotto-supraglottic amyloidosis where patients had presented with hoarseness of voice and were further evaluated according to the standard protocol and underwent ML scopy + laser excision, and histopathology showed amorphous eosinophilic infiltrate suggestive of laryngeal amyloidosis. They were further followed up and found to have a good phonatory outcome with no complications.

Keywords: Glottic Mass; Glottic Amyloidosis; Laryngeal Amyloidosis; CO2 Laser

Introduction

The stress of phonation is more on true vocal cords, and as a result, they are more commonly affected with nodules, polyps, and cysts. This leads to limited literature on benign false cord neoplasms. False cords, however, are also a part of vocal articulation during glottal stops and pressed phonation [1]. They have also been linked to laryngeal lumen closure during swallowing, coughing, gagging, etc. [2]. False cords also play a role in lubricating the true cords [3]. As such, it is important to recognise benign lesions affecting both cords. Here, we present two cases of glottic-supraglottic mass that had different clinical appearances but were histopathologically confirmed as amyloidosis.

Primary laryngeal amyloidosis is a rare disease. It causes significant dysphonia with a notable risk of airway

obstruction and subsequent development of stridor. It also imposes a treatment challenge as there is no known definite cure to date. Glottic amyloidosis has very little literature, and as such, it is important to highlight its management. Here, we present two cases of glottic amyloidosis that had different clinical appearances but were histopathologically confirmed as amyloidosis.

Case Reports

Case 1

A 74-year-old female patient came with complaints of a change in voice for 40 years and a cough for 1 month. She did not have difficulty in swallowing or breathing. She had undergone 4 vocal cord surgeries to date, done every 10 years or so. She had a history of hypertension, diabetes mellitus,

and hyperlipidaemia. Video laryngoscopy showed a left glottic and supraglottic mass, obscuring the anterior glottis. Bilateral vocal cords were mobile. A CT scan of the larynx with MRI correlation showed an ill-defined hypoenhancing soft tissue density lesion involving the left anterior 1/3rd of the vocal cord and the anterior commissure, measuring 1.0*1.4*0.8 cm. Area of calcification noted within. No subglottic or supraglottic extension noted. No cartilage involvement was noted (Figure 1).



Microlaryngoscopy revealed a left glottosupraglottic mass. CO2 laser excision was done. The frozen section showed amyloidosis. Histopathology showed stratified squamous epithelium with subepithelial acellular amorphous eosinophilic infiltrate seen in stroma. Congo red stain showed birefringence, suggestive of amyloidosis. The patient had an uneventful postoperative recovery. Rheumatology and haematology referral was done. Systemic amyloidosis was ruled out.

Case 2

A 37-year-old female patient presented with complaints of change in voice for 2 years. She was an advocate by profession.

She was a K/C/O Hodgkin's Lymphoma and had undergone Chemoradiotherapy 15 years back. Videolaryngostroboscopy showed a left-sided false cord mass. CT revealed an illdefined, hypodense area measuring 1*0.5 cm in the left false cord with asymmetrical compression of the left ventricle. Obliteration of the left paraglottic fat stripe noted. Microlaryngoscopy revealed a fleshy left glottosupraglottic mass. CO2 laser excision was done. Histopathology revealed extracellular amorphous eosinophilic material suggestive of amyloid. Rheumatology and haematology referral was done. Systemic amyloidosis was ruled out.She was then followed up and found to be symptom free.



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Figure 3: Histopathology image showing apple green birefringence.



Figure 4: CT larynx protocol showing the mass.

Discussion

Amyloidosis is an autoimmune disease characterised by the deposition of protein aggregates in different tissues. This elicits a chronic inflammatory reaction involving the lymphocytes and the plasma cells [4]. Amyloidosis can be systemic or localised. In systemic, the precursor protein is produced in the liver or the bone marrow and released into circulation and deposited as amyloid fibrils elsewhere in the body [5]. Although laryngeal amyloidosis can be a manifestation of systemic amyloidosis, it is usually a localised disease [6]. Laryngeal amyloidosis comprises less than 1% of benign laryngeal masses [7]. Males have a higher preponderance to females with a 3:1 ratio. It can occur at any decade of life, but peak incidence is at the fifth decade [8]. The ventricles, false cords, true cords, epiglottis, aryepiglottic folds, and subglottis are affected in decreasing order [9]. Dysphonia, especially progressive dysphonia, is the most common presentation. Dyspnoea and stridor are seen with larger lesions. Videolaryngostroboscopy usually shows an oedematous glottic structure. It can also present as protruding, smooth, submucosal nodules [10]. The lesions may be yellow, orange, or grey-coloured [11]. Biopsy specimens are seen as a homogenous, acellular, eosinophilic, extra-cellular infiltrate [12]. It stains Congo red positive with apple-green birefringence, which is its confirmatory test [13].

CT findings can be non-specific, showcasing a homogeneous, well-defined submucosal mass, but it may help demonstrate punctate calcifications, while MRI may show enhancement and altered signal characteristics [14]. Surgery is the mainstay of treatment, but recurrences are common. Low-dose radiotherapy at 45 Grey can be considered. There is no cure for amyloidosis to date.

Microlaryngoscopy and laser excision resulted in disease clearance in both cases. Precautions must be taken to maintain a good phonatory outcome. These include taking care not to traumatise the lamina propria and free edges of the vocal fold. Trauma to the opposite cord in the anterior commissure has to be avoided. Both patients were pleased with their voice outcome. Serial videolaryngostroboscopies were done. There were no recurrences till the time of publication. Systemic amyloidosis was ruled out.

Conclusion

Amyloidosis has to be kept in mind when managing glotto-supraglottic masses. A videolaryngostroboscopic examination as well as radiological assessment, where necessary, has to be done. Confirmatory diagnosis is via histopathology.

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