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Case Report

Ear,Nose and Throat Section

Vocal Cord Amyloidosis in a Middle-aged Female: A Rare Clinical Entity

PRAGATI SHIKHA[1], SAGAR GAURKAR[2], PRASAD DESHMUKH[3], JASLEEN KAUR[4], ANJORI RAUT[5]

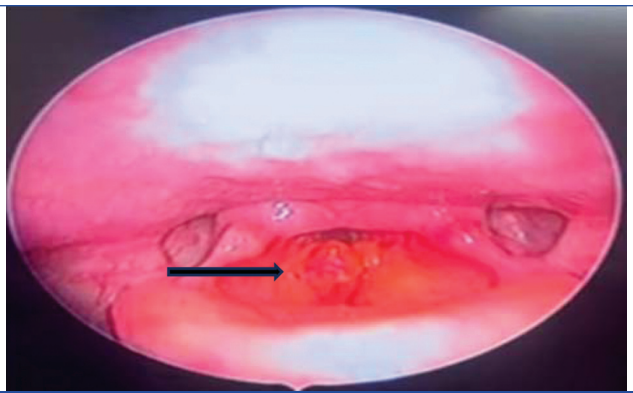
ABSTRACT

Primary laryngeal amyloidosis is a rare disorder, accounting for approximately 1% of all benign laryngeal lesions. The authors, here, report the case of a 55-year-old female who presented to the Otorhinolaryngology Department with complaints of change in voice for the past 2-3 years. The patient described a gradual onset of hoarseness, strained quality, and fatigue of voice. Additionally, she experienced quivering, effortful phonation, and a relatively high pitch. Video-directed laryngoscopy revealed a polypoid or polypoid-like, reddish growth over the true vocal cords. A Contrast-Enhanced Computed Tomography (CECT) scan of the neck showed features suggestive of glottic and subglottic stenosis. The patient subsequently underwent microlaryngoscopy under general anaesthesia, and a unilateral excisional biopsy was taken from the lesion. Histopathological examination of a 20× section demonstrated Congo red positivity under polarised light, confirming the presence of amyloid deposits. A corresponding Haematoxylin and Eosin (H&E)-stained section (20×) showed proliferation of fibroblasts arranged in fascicles with areas of hyaline degeneration. Postoperatively, the patient was advised speech therapy, and at three weeks of follow-up, significant improvement in voice quality was noted. The present case highlights the rarity of primary laryngeal amyloidosis and underscores the importance of considering this diagnosis in patients presenting with long-standing hoarseness of voice. A multidisciplinary approach, including surgical excision, postoperative speech therapy, and long-term follow-up, is essential for achieving favourable outcomes and reducing recurrence rates.

Keywords: Congo red, Fascicles, Fibroblasts, Laryngeal amyloidosis, Speech therapy

CASE REPORT

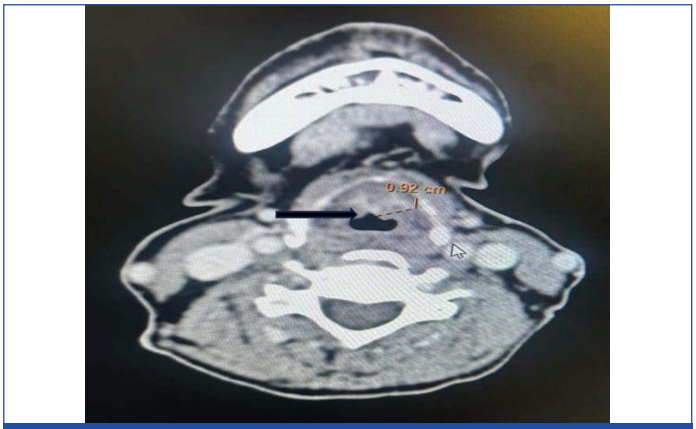
A 55-year-old non-smoker, non-diabetic, and non-hypertensive female presented to the Otorhinolaryngology Department with complaints of change in voice for the past 2-3 years. The patient was apparently asymptomatic prior to this period and reported a gradual onset of hoarseness, strained voice quality, and voice fatigue. She also complained of a quivering voice during speech, effortful phonation, and a relatively high pitch. Additionally, she reported occasional breathlessness on exertion, with no associated dysphagia or odynophagia. There was no history of voice overuse, laryngeal trauma, or significant family history. On physical examination, no neck swelling or palpable mass was observed. The clinician counselled the patient regarding the procedure and obtained informed consent. Video-directed laryngoscopy revealed a non-ulcerative, smooth, nodular lesion in the supraglottic region involving the bilateral aryepiglottic folds, covering the false vocal cords and partially obscuring the true vocal cords [Table/Fig-1]. The bilateral true vocal cords were equal and mobile.



[Table/Fig-1]: Shows non-ulceroproliferative smooth growth over bilateral aryepiglottic folds and false vocal cords.

A comprehensive systemic evaluation was conducted to rule out systemic amyloidosis and multiple myeloma. An abdominal fat pad biopsy was negative for amyloid deposition. Urine analysis was negative for Bence-Jones proteins. Routine laboratory investigations, including complete blood count, liver function tests, renal function tests, and serum protein electrophoresis, were within normal limits. Based on these findings, the patient was diagnosed with primary laryngeal amyloidosis.

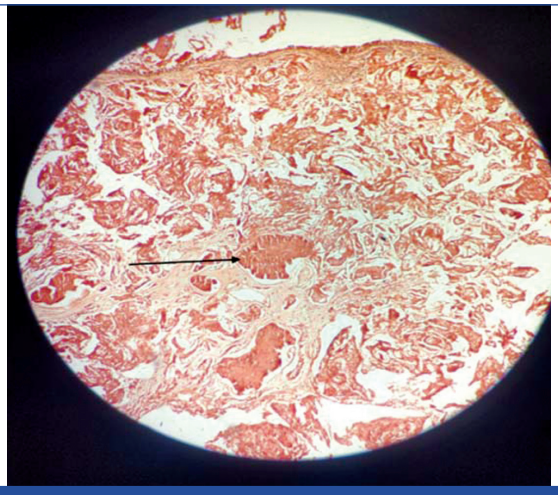
A CECT scan of the neck demonstrated circumferential thickening of the vocal cords and subglottic region, predominantly posteriorly, causing luminal narrowing (Anteroposterior×Transverse: 9×5 mm), suggestive of glottic and subglottic stenosis [Table/Fig-2].



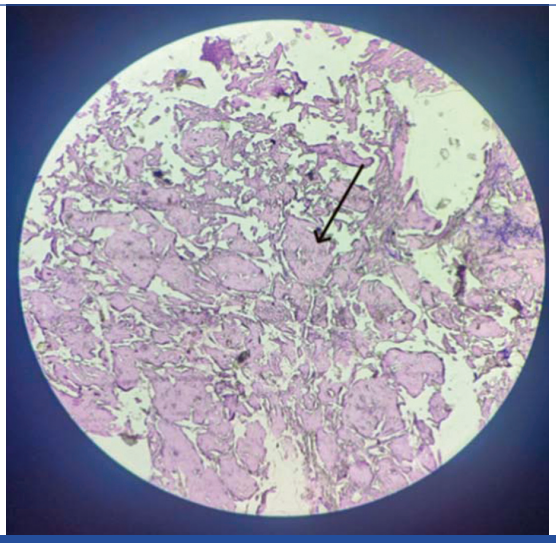
[Table/Fig-2]: CECT neck - axial view showing circumferential thickening in the vocal cords (black arrow) of around 0.92 cm.

The patient underwent microlaryngoscopy under general anaesthesia, during which a unilateral excisional biopsy was obtained. Gross examination revealed a smooth-surfaced mass. Histopathological analysis of a 20x magnification Congo red-stained section showed characteristic apple-green birefringence under polarised light, confirming amyloid deposition [Table/Fig-3]. The H&E-stained section (20×) revealed fibroblast proliferation arranged in fascicles with hyaline degeneration [Table/Fig-4]. No evidence of malignancy was identified.

Post microlaryngoscopy with excisional biopsy, speech therapy was advised. At three weeks of follow-up, the patient demonstrated improvement in voice quality.



[Table/Fig-3]: Congo red staining was positive (black arrow) (20X).



[Table/Fig-4]: H&E stain shows proliferation of fibroblasts arranged in fascicles with hyaline degeneration (black arrow) (20X).

DISCUSSION

Amyloidosis is a disorder characterised by the extracellular accumulation of proteinaceous material in tissues. The exact aetiology remains unclear. While amyloidosis can involve multiple organs, the kidneys, bronchus, and larynx are among the most commonly affected sites [1]. Although the microscopic appearance of amyloid deposits is uniform, amyloidosis represents a heterogeneous group of diseases. More than 20 biochemical variants have been identified, with immunoglobulin light-chain {Amyloid Light-chain (AL)} and Amyloid Associated (AA) types being the most common [2].

According to the literature, the true vocal cords are the most frequently involved site in head and neck amyloidosis. In descending order of frequency, laryngeal involvement typically affects the ventricle, vestibular folds, vocal folds, epiglottis, aryepiglottic folds, and subglottis. Involvement of the subglottic region and false vocal cords is relatively uncommon [3]. In primary laryngeal amyloidosis, the true vocal cords are most commonly affected [4]. In contrast, amyloid deposition confined to the false vocal cords is extremely rare. This makes the present case an unusual clinical presentation, highlighting the importance of considering amyloidosis as a differential diagnosis in patients presenting with atypical laryngeal lesions.

Laryngeal amyloid lesions are typically described as well-defined, submucosal soft-tissue masses with minimal or no contrast enhancement. In some cases, they may present as circumferential thickening of the glottic or subglottic soft tissues or as more diffuse intralaryngeal infiltration, suggesting a deeper submucosal component [5].

A definitive diagnosis of amyloidosis requires histopathological confirmation through tissue biopsy and demonstration of amyloid using appropriate staining techniques, such as Congo red staining [6]. Although laryngoscopy can identify visible lesions, Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) are valuable in assessing the extent of disease involvement. On CT, amyloidosis typically appears as diffuse laryngeal soft-tissue thickening with increased density. On MRI, amyloid deposits demonstrate signal intensity similar to that of skeletal muscle, which is a useful distinguishing feature, as neoplastic lesions generally do not exhibit this pattern [5].

The primary treatment modality for laryngeal amyloidosis is surgical excision. Recent studies indicate that Carbon dioxide (CO_2) laser excision of amyloid deposits in the larynx and trachea is the preferred treatment approach owing to its precision and minimal tissue damage [7]. Yiotakis I et al., reported a similar case of primary laryngeal amyloidosis involving the aryepiglottic fold in a 23-year-old female who presented with hoarseness and dysphagia. The lesion was excised using a CO_2 laser, followed by speech therapy, which was recommended as the treatment of choice [8].

Mesolella M et al., presented a case series of five female patients with localised laryngeal amyloidosis without systemic involvement. All patients were successfully treated using microlaryngoscopy with CO_2 laser or cold instruments. The authors recommended a structured follow-up protocol consisting of monthly examinations for the first six months, bimonthly follow-up until the end of the second year, six-monthly reviews for the subsequent two years, and annual follow-up thereafter. They also advised a minimum of 10 years of laryngological follow-up following CO_2 laser excision due to the risk of recurrence [9]. Long-term, individualised surveillance remains essential, as recurrence is not uncommon, even after complete surgical excision [7].

CONCLUSION(S)

The present case report highlights the rare and unique presentation of primary laryngeal amyloidosis, emphasising the importance of including this condition in the differential diagnosis of patients presenting with progressive voice changes. The diagnosis was confirmed through a combination of clinical evaluation, laryngoscopy, imaging, and histopathological examination. The patient was successfully managed using a multidisciplinary approach, incorporating surgical excision and post-operative speech therapy, underscoring the importance of comprehensive management and long-term follow-up.

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PARTICULARS OF CONTRIBUTORS:
1. Junior Resident, Department of Otorhinolaryngology and Head and Neck Surgery, Jawahar Lal Nehru Medical College, Wardha, Maharashtra, India.
2. Professor, Department of Otorhinolaryngology and Head and Neck Surgery, Jawahar Lal Nehru Medical College, Wardha, Maharashtra, India.
3. Professor, Department of Otorhinolaryngology and Head and Neck Surgery, Jawahar Lal Nehru Medical College, Wardha, Maharashtra, India.
4. Senior Resident, Department of Otorhinolaryngology and Head and Neck Surgery, Jawahar Lal Nehru Medical College, Wardha, Maharashtra, India.
5. Junior Resident, Department of Paediatric and Preventive Dentistry, Sharad Pawar Dental College, Wardha, Maharashtra, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Pragati Shikha,
Junior Resident, Department of Otorhinolaryngology and Head and Neck Surgery, Jawahar Lal Nehru Medical College, Datta Meghe Institute of Higher Education and Research, Wardha, Maharashtra, India.
E-mail: pragati.shikha2103@gmail.com

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