ISSN: 2687-8410
DOI: 10.33552/ACCS.2026.05.000621
Archives of Clinical Case Studies
Iris Publishers

Case Report

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Laryngeal Amylosis

Smail kharoubi*

Ent Head and Neck Surgery, university baji mokhtar, Annaba, Algeria

***Corresponding author:** Smail Kharoubi, Ent Head and Neck Surgery, university baji mokhtar, Annaba, Algeria

Received Date: November 10, 2026

Published Date: November 16, 2026

Abstract

Primary amyloidosis is a rare condition with focal involvement representing 9%-15% of those cases. Isolated laryngeal amyloidosis is a rare condition. We present an observation of a 58-year-old female patient presented dysphomia and sided pharyngeal discomfort during swallowing. Fibroscopic exam showed a pinkish-white edema isolated to the right aryepiglottic fold. CT scan usually shows thickening of the right aryepiglottic fold, with no other abnormalities. Biopsy and histologic exam (congo red stain) recognize the diagnosis of amyloidosis. Additional investigations confirmed the localized (laryngeal) nature of the disease. A literature review was conducted in conjunction with this case presentation.

Keywords: Amyloidosis; endoscopic microsurgery; condo red stain

Introduction

Amyloidosis is a group of disorders caused by extracellular deposition of insoluble fibrillar protein (amyloid) with characteristic features on microscopy, histochemistry and ultrastructural examination. Amyloidosis consists of different forms of systemic or isolated organ lesions characterized by fibrillary protein deposits in extra-cellular tissue. The cause is still considered idiopathic. Von Rokitansky first described such deposits in 1842, but it was not until 1851 that Virchow applied the term amyloid to describe this deposition [1,2]. The isolated involvement of the larynx is the most frequent form in the ENT sphere and represent 0,2 to 1,2% of all benign tumors of larynx. Burrow and Neumann in 1875 reported first case of Laryngeal amyloidosis [3]. Histologically, the presence of amyloid can be confirmed by the characteristic Congo red staining under polarized light microscopy. Treatment is surgical, consisting of lesion removal using cold instruments or a laser. Recurrence is possible, and long-term follow-up is highly recommended to detect early recurrence or systemic involvement.

Clinical Observation

A 58-year-old female patient with a history of type 2diabetes and hypertension presented with a several-month history of progressive dysphonia, associated with right-sided pharyngeal discomfort during swallowing. She had previously sought multiple consultations and received various treatments without clinical improvement. Examination of the oral cavity and oropharynx was unremarkable. Flexible endoscopy revealed pinkish-white edema isolated to the right aryepiglottic fold. The vocal cords showed no abnormalities, with preserved mobility (figure 1).

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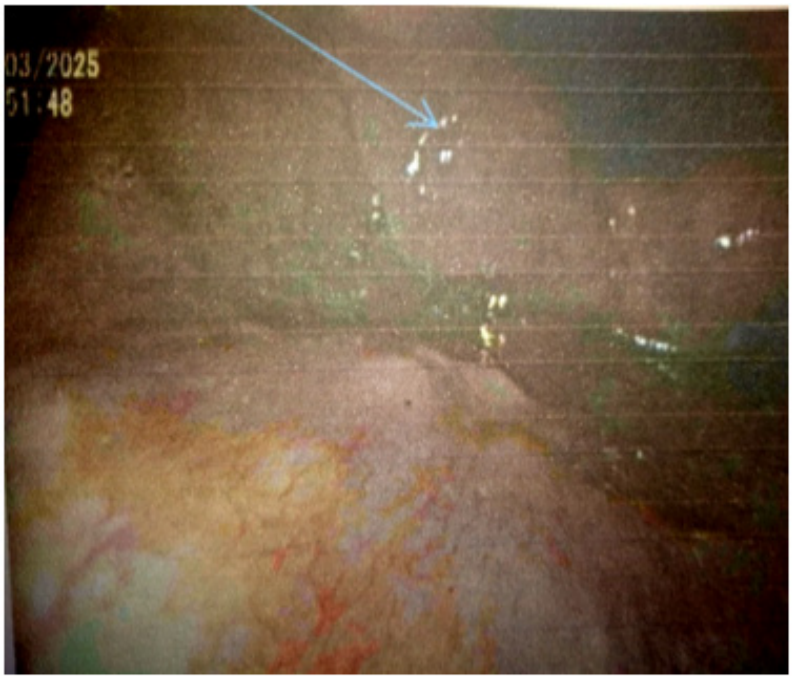


Figure 1: Endoscopic view: diffused edema of right arytenoids (blue arrow).

Cervical examination showed no regional lymphadenopathy, and the ENT examination was otherwise unremarkable. A laryngeal CT scan demonstrated thickening of the right aryepiglottic fold, without calcification and no other abnormalities in the laryngeal or cervical structures (figure 2&3).

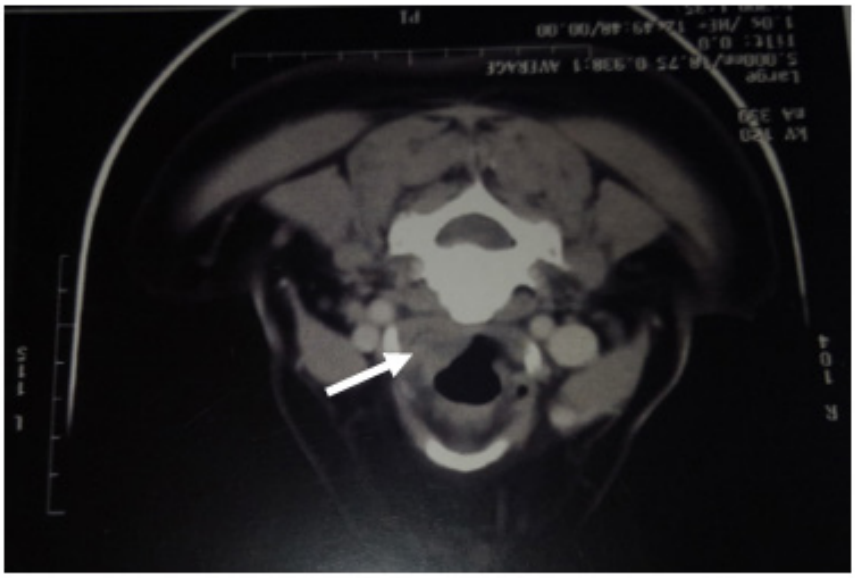


Figure 2: Cervical Axial CT scan: swelling of right arytenoids (white arrow).



Figure 3: Saggital axial CT scan: swelling of right arytenoids (white arrow).

The chest X-ray was normal, and an upper gastrointestinal endoscopy revealed unremarkable findings, with no history of gastroesophageal reflux disease (GERD) Preoperative investigations including renal and liver function tests, electrocardiography, blood glucose, and coagulation profiles were normal. Direct laryngoscopy under general anesthesia revealed a firm edema isolated to the right aryepiglottic fold. The hypopharynx and cervical esophagus appeared normal. Multiple incisional biopsies and a reduction of the edematous mucosa being performed using micro-scissors. Histopathological examination was consistent with laryngeal amyloidosis, confirmed by Congo red staining (figure 4&5).

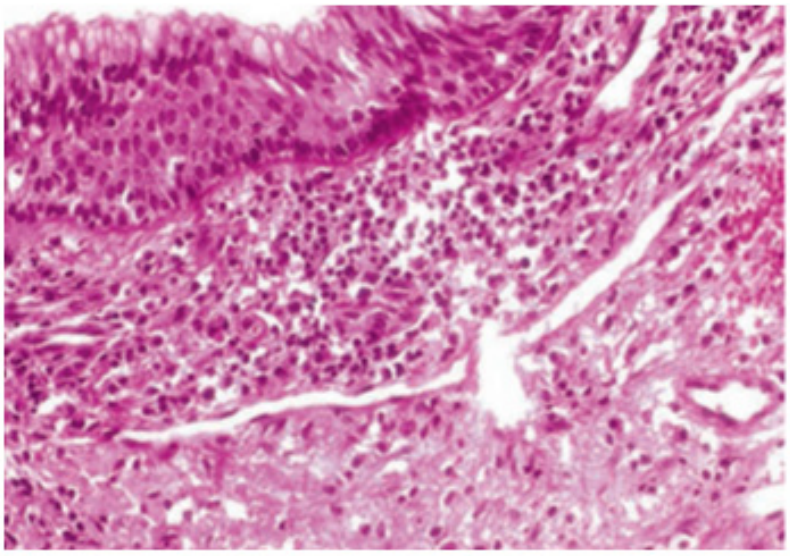


Figure 4: Histologic aspect; Subepithelial lymphoplasmacytic infiltrate near an area of amyloid deposition.

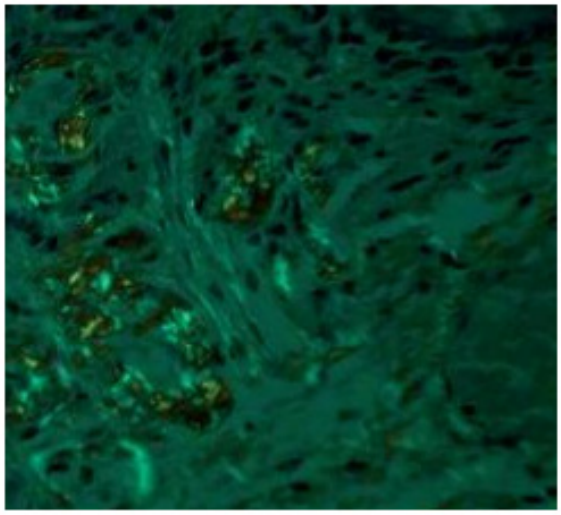


Figure 5: Amyloidosis. Congo red staining under polarized light.

Additional investigations, including serum and urine protein electrophoresis, abdominal ultrasonography, and beta2 macroglobulin testing, were performed to rule out systemic amyloidosis and plasma cell dyscrasias; all results were within normal limits. An internal medicine consultation confirmed the localized nature of the disease. At the one-month follow-up, the patient showed significant improvement in dysphonia and complete resolution of dysphagia, alongside a notable reduction in laryngeal edema. The patient's condition remains stable at ten months post-diagnosis.

Discussion

The larynx is the most common site of localized head and neck amyloidosis. Laryngeal amyloidosis as isolated, without systemic involvement. However, systemic involvement was reported in 7% of 177 patients with laryngeal amyloidosis [4]. Until 1990, only approximately 300 cases of upper airway amyloidosis had been reported in the literature [5]. Between 2000 and 2026, around 450 cases of isolated laryngeal amyloidosis were described in the literature [6]. In the pediatric population, only two cases of laryngeal amyloidosis had been reported up until 2006 by Naraghi [7], whereas ten cases were collected in the literature between 2006 and 2026 [8]. Laryngeal amyloidosis may affect individuals at the age of 11 to 80 years, with a peak incidence in the fifth decade [9]. It has higher male predilection with 3:1 male female ratio [10]. In the head and neck, localized amyloidosis is most common in the larynx. Other sites include the sali vary glands, oral cavity, pharynx, nasopharynx, sinonasal cavities, larynx, trachea, bronchi and lungs, and lymph nodes. In the larynx, disease occurred in true and false vocal cords, subglottis and supraglottis, and anterior commissure, which are the common locations in laryngeal amyloidosis [11].

Multifocal disease is present in up to 15% of patients in tracheobronchial tree, sinus, nasal and nasopharyngeal site [12]. The exact etiology of amyloidosis is still unclear. There is no definite established link with smoking, drinking, vocal abuse, or recur rent infection. Theory proposed is that amyloid light chains are produced by plasma cell clones. Another theory is its occurrence secondary to body's inability to remove light chain proteins leading to localized deposits [13]. Hoarseness of voice is the most common presentation of Laryngeal amyloidosis but sometimes patients present with dyspnea, stridor, persistent dry cough, sleeping apnea syndrome or dysphagia and hemoptysis. Flexible laryngoscopy (fibroscopy) is the initial step in the evaluation of voice or respiratory disturbances. It allows for the assessment of the larynx and cervical trachea, providing critical information on both primary organic conditions (mucosal or submucosal lesions) and functional status (vocal cord mobility). Direct laryngoscopy remains the cornerstone examination and is performed under general anesthesia.

It enables a comprehensive evaluation of the local lesion (including its site and extension) and any associated lesions (involving the pharynx, tongue, tonsils, or esophagus), while facilitating a biopsy or excisional biopsy. On laryngoscopy, the aspect is may variable. The lesion appears as a yellow, grey or red subepithelial non- ulcerated nodule or a pedunculated polypoid lesion. In some case diffuse infiltration can be noted. The appearance of small, irregular, soft proliferations instead of a firm, large deposit suggests above all the possibility of a hereditary systemic apo A-I amyloidosis rather than a localized laryngeal AL amyloidosis [14]. Vocal cord fixation is a rare phenomenon, which is mostly associated with other predisposing factors such as mediastino-pulmonary localization or neurological complications. Bilateral vocal cord involvement progressing to life-threatening respiratory failure due to upper airway obstruction has been

reported [15]. During the 1970s, the Benn hold test; which involved the intravenous injection of Congo red and the detection of color changes in laryngeal lesions was abandoned due to its low reliability and, more notably, its toxicity (such as allergic reactions, nephrotoxicity and liver deposits). It was subsequently replaced by in vivo histological testing. A detailed family history and physical examination (including the liver, spleen, and lymph nodes) are required.

The main benefit of serial imaging is the correlation with patient symptoms, identification of the extent of disease, and assisting in delineating appropriate timing for surgery [16]. On CT, the lesion typically presents as a homogeneous, well-defined submucosal soft tissue mass that may be spontaneously hyperdense, showing only mild enhancement after intravenous contrast administration [17]. Punctuate calcifications may also be observed. On MRI, the mass displays consistent signal intensity, appearing hypointense or isointense on both T1-weighted and T2-weighted images. Indeed, amyloid fibrils exhibit a signal intensity similar to that of skeletal muscle fibers across all pulse sequences [18]. Whole-body scintigraphy is considered mandatory by many authors in the evaluation of amyloidosis. However, data from various literature studies remain controversial. The primary aim of this imaging modality is to monitor disease progression or regression. Nevertheless, 18 F-FDG remains a non-specific tracer for amyloid deposits. Furthermore, 18F-FDG PET/CT can differentiate systemic amyloidosis (characterized by a lack of FDG uptake at the amyloid site) from localized amyloidosis (which exhibits increased uptake).

This imaging modality also holds clinical utility for treatment evaluation and patient follow-up [19]. Meanwhile, specific amyloid-targeting radiotracers, such as 123I-SAP (serum amyloid protein component), 99mTc-aprotinin, and 11C-PiB (Carbone-11 Pittsburgh Compound), are currently being evaluated and may offer superior diagnostic specificity in the near future [20]. Histologically, acellular, eosinophilic, amorphous materials were noted below surface epithelium. Congo red stain under polarized microscopy showed apple-green birefringence. Immunohistochemical analysis serves as an essential diagnostic tool for characterizing amyloid deposits, with particular emphasis on the following panel of molecular markers [21]; CD138 / MUM1: Immunoreactivity to these markers highlights the infiltration of plasma cells, the cellular source of the immunoglobulin light chains that aggregate into amyloid fibrils. -CD20 / CD3: These stains serve to characterize the adjacent inflammatory microenvironment by delineating the distribution of B- and T-cell lymphocytes. -Serum Amyloid P (SAP) Protein: This component typically demonstrates widespread, non-specific positivity across all lesions, reflecting its constitutive presence as a structural element in all variants of amyloid deposits. Electron microscopy revealed the characteristic linear, non-branching fibrillar amyloid materials.

In the workup of amyloidosis, conducting specific laboratory tests is a conventional procedure. This screening aims to identify systemic amyloidosis, ruling out associations with malignant hematologic diseases such as multiple myeloma, or evaluating chronic dysfunction in target organs like the kidneys and liver. The standard laboratory panel includes the erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), liver function tests, renal function studies, quantitative immunoglobulin assays, serological testing for rheumatoid arthritis, urinalysis, serum and/or urine protein electrophoresis, and Bence-Jones protein analysis. The diagnosis of amyloidosis requires tissue biopsy, with Congo red immunohistochemical staining showing a pink or red color under normal light and the classical apple-green birefringence under polarized light. A comprehensive systemic workup in amyloidosis is essential to distinguish between the localized and systemic forms of the disease. Only 15% of laryngeal amyloidosis cases have systemic involvement [22]. One primary justification for this research is the significant morbidity associated with systemic disease. Complete blood cell count, liver and renal function tests, urinary test for Bence-Jones protein, chest CT scan, electrocardiogram, echocardiogram, and abdominal ultrasound may be warranted. Also required a serological test for rheumatoid arthritis.

A rectal biopsy, a simple needle biopsy of abdominal Fator a bone marrow, lip or skin biopsy may be performed to exclude systemic involvement [23]. In addition, on biopsy, clonal plasma cells and giant cells appear to be constant in cases of localized amyloidosis in contrast to the lack of cellular infiltrates in almost every case of systemic amyloidosis. Laryngeal amyloidosis can be associated with various laryngeal diseases, notably malignant tumors. Several cases have been reported in the literature describing the coexistence of laryngeal amyloidosis with small cell carcinoma, squamous cell carcinoma, MALT lymphoma, or medullary thyroid carcinoma invading the larynx. While these lesions may be fortuitously associated, amyloidosis may also reflect a favorable immune status or a localized form of immune reaction. Repeat biopsies must be performed if the diagnosis is uncertain or inconsistent with the clinical symptoms or endoscopic findings. Furthermore, laryngeal amyloidosis can represent a mechanism of healing following treatment, such as surgery or radiotherapy. [24-26].

Laryngeal Amyloidosis in Child

The laryngeal amyloidosis in pediatric population is very rare. The youngest reported case of localized laryngeal amyloidosis is that of an 8-year-old female. The male to female ratio is 3:1 [27], Clinically, it presents with progressive hoarseness, dyspnea and sleep disordered. Nagasaka reported the first case in the literature showing the coexistence of extramedullary plasmacytoma and localized amyloidosis of the larynx in children. The author supports the concept that localized laryngeal amyloidosis may be a manifestation of low-grade B-cell neoplasms [28]. As in adults, additional investigations were recommended to rule out systemic amyloidosis. Treatment is similar to the adult protocol and consists of excision via suspension laryngoscopy using cold instruments or a CO2 laser. O'Halloran utilized a laryngofissure approach, with excision of the thyroid cartilage, to treat laryngeal amyloidosis involving both the false and true vocal cords in a 9-year-old girl [29]. Regular follow-up (including fibroscopy and imaging) is necessary

to prevent recurrence, especially within the first five years.

A wide range of diseases can mimic laryngeal amyloidosis. These include saccular cysts of the larynx, laryngocele, sarcoidosis, metastatic tumors, polyps, malignant neoplasms, and salivary gland tumors [30]. Other reported conditions comprise squamous cell carcinoma, medullary thyroid carcinoma with laryngeal invasion, chondrosarcoma, lymphoma, as well as various inflammatory disorders. Medical history, risk factors, clinical examination, and imaging (CT scans and MRI) provide an initial assessment. A laryngeal biopsy remains the definitive diagnostic procedure. Spontaneous regression has been exceptionally described. Lewis (2025) reported a notable case of laryngeal amyloidosis in an 82-year-old man, who experienced an 85% spontaneous regression of the lesion within two months [31]. Pollak, however, reported the regression of a laryngeal amyloid tumor following a simple biopsy. Conversely, Kramer and Som (1935) appeared to have stimulated the enlargement of a similar tumor via diagnostic biopsy. Galluzi presented an important study about 515 cases of laryngeal amyloidosis (majority of supraglottic region) with surgical treatment; endoscopic excision with cold or powered instruments and 28.4% of recurrence [32].

Prognosis is excellent and the long-term follow-up should be conducted for at least five to seven years, due to the possibility of late recurrence and systemic involvement [33]. Lanks recommend annual surveillance for up to 10 years, as it may have a long latency, with recurrences reported after 8–14 years from the initial diagnosis. Moreover, studies with long follow-up with amyloidosis of the larynx and trachea have reported that none of the patients developed systemic amyloidosis, B-cell lymphoma, or multiple myeloma [15]. Early diagnosis and treatment of laryngeal amyloidosis may reduce the recurrence of the disease. The treatment aims at a minimal invasive endoscopic surgery with functional organ preservation (voice and normal breathing). Treatment may vary from simple observation (small asymptomatic lesions), surgery with different modalities depending upon the extent of involvement of larynx. Surgery continues to be the predominant modality for treatment, but radiation and potentially medical approaches are on the horizon. Endoscopic excision can be done either with cold knife, CO2 LASER, Microdebrider or with Coblation wands [34].

Surgical difficulty varied depending on the laryngeal lesion, being more straightforward for localized forms and complex for multiple locations, where a multi-stage procedure may be required. Advanced cases may require open surgery with partial and/or total laryngectomies [35]. Localized amyloidosis of the larynx is usually described as a non-bleeding lesion. Chow reported a case of fatal, massive upper respiratory tract hemorrhage following surgical excision. This critical data must be carefully considered when managing the perioperative and postoperative stages of these laryngeal lesions [36]. Postoperative recovery showed minimal dysphonia with no dyspnea. Surgical complications such as granulomatosis scar tissue formation, tracheostomy, laryngotracheal stenosis, pneumothorax and concomitant malignancy were developed in 17.5% of patients [34]. In the postoperative stage, numerous protocols combine surgical resection with supportive medical treatments, such as oral corticosteroids and colchicine, to optimize outcomes.

Wadhawa found effective result with oral Bortezomib and corticosteroid after debulking diffuse laryngeal amyloidosis [37]. The conformal external beam radiotherapy (RT) for local control of progressive airway amyloidosis was performed in some cases. The protocol utilizes 20 Gy in 10 fractions over 2 weeks. Radiotherapy prevents progressive amyloid deposition [38]. Minimally invasive interventions for benign neoplastic lesions of the larynx present clinical interest in view of obtaining a radical curative outcome with minimal trauma, especially for the patients who have contraindications for general anesthesia or transoral microsurgery. Trans nasal flexible endoscopic surgery has been used in management of laryngeal amyloidosis (cold surgery with flexible forceps, diathermy snare excision, Nd:YAG laser ablation and combined techniques) [39]. There is no effective medical treatment for laryngeal amyloidosis. Several studies have evaluated the use of the immunomodulatory agents (thalidomide, bortezomib, or lenalidomide), in combination with melphalan or cyclophosphamide but without appreciable results.

Among the new treatments currently under development, anselamimab is an experimental chimeric monoclonal antibody designed for the treatment of immunoglobulin light chain (AL) amyloidosis. When administered alongside therapies targeting plasma cell dyscrasia, anselamimab has significantly reduced all-cause mortality and cardiovascular hospitalizations in patients with kappa AL amyloidosis, potentially offering a novel therapeutic option for this specific isotype [40]. Therapies such as daratumumab, pomalidomide, bortezomib, and birtamimab are currently under evaluation. Golombic utilized this approach in a patient presenting with laryngeal amyloidosis secondary to smoldering myeloma. The patient had previously undergone surgical treatment in 2006, 2008, and 2009, combined with radiotherapy. The patient subsequently received curcumin therapy at an initial dose of 1,500 mg per day, which was gradually increased to 3,600 mg per day. At the 2011 follow-up, a laryngeal endoscopy was performed, revealing no evidence of disease progression. The authors hypothesize that curcumin shares a similar mechanism of action to lenalidomide [41].

Conclusion

Laryngeal amyloidosis is a rare benign laryngeal lesion, but actually the most common site of amyloid deposition in the head, neck and respiratory tract. Dysphonia is the main symptom and the diagnosis depends on histology, with pathognomonic positive Congo red stain and apple- green birefringence under polarized light. Imaging is useful in mapping lesions, which may often be more extensive than they appear during laryngoscopy. Treatment depends on the symptoms and is most commonly surgical but minimally invasive. Long- term annual follow- up is mandatory, as recurrence is common and can have a long latency. The future management of laryngeal amyloidosis will likely reflect a paradigm shift from purely local surgical treatment (organ-preserving and

functional Laser interventions- CO2 or Diode) to the emergence of targeted and systemic medical therapies (Daratumumab for severe, recurrent localized laryngeal disease). Optimized, Minimally Invasive Surveillance Protocols is also very important and will to be performed for a specific following and relies on high-definition laryngoscopy combined with narrow-band imaging (NBI) allows for early identification of submucosal structural changes and microvascular alterations indicative of early disease relapse.

Acknowledgment

None.

Conflicts of Interest

No conflict of Interest.

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