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Head and neck amyloidosis – report of five cases

Amyloidoza w rejonie głowy i szyi – opis serii przypadków

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ABSTRACT:

Background: Amyloidosis is a group of diseases caused by the extracellular accumulation of insoluble fibers called amyloid in the tissues and organs. They have a secondary beta-sheet structure, which makes them resistant to proteolysis. In histological examination amyloid deposits stain with Congo red and show an apple-green birefringence in polarized light. Amyloid deposits disturb the function of organs and cause clinical symptoms. Their formation or accumulation in the system may be acquired or inherited. Due to the location of amyloid deposits we distinguish systemic and localized amyloidosis with the formation of tumors (usually from light chains).

Case reports: 5 cases of amyloidosis in the head and neck region are presented in this paper. The locations of the amyloid deposits were as follows: larynx, nasopharynx, sublingual and submandibular gland and the tongue. The initial clinical presentation correlated with location of amyloid tumour in our patients. Two patients had history of local recurrence of the disease. Surgical resection and histopathological examination were performed. Sections stained with Congo red confirmed the diagnosis of amyloidosis. Three patients had potential conditions predisposing to amyloidosis: previous radiotherapy, chronic inflammation due to hepatitis C virus infection and graft versus host disease.

Conclusion: Amyloidosis should be considered as the cause of symptoms in pathologies of the head and neck region. The diagnosis requires a histopathological examination. The systemic form of the disease must be ruled out in all patients with head and neck amyloidosis. In localized amyloidosis the surgical resection of the lesions is the procedure of choice, however the organ's functionality should be taken into account.

KEYWORDS: amyloidosis, amyloidosis of the nasopharynx, head and neck amyloidosis, larynx amyloidosis, localized amyloidosis, salivary gland amyloidosis, tongue amyloidosis

STRESZCZENIE:

Podstawy naukowe: Amyloidoza, czyli skrobawica, to grupa chorób, których wspólną cechą jest pozakomórkowe gromadzenie się w tkankach i narządach nierozpuszczalnych białek o budowie włókienkowej, zwanych amyloidem. Mają one drugorzędową strukturę β-kartki, co czyni je opornymi na proteolizę. W skrawkach histologicznych złogi amyloidu wybarwiają się czerwienią Kongo i wykazują zielone świecenie w świetle spolaryzowanym. Depozyty amyloidu zaburzają funkcje narządów i powodują objawy kliniczne. Ich powstawanie lub gromadzenie się w ustroju może być stanem nabytym lub dziedzicznym. Ze względu na lokalizację złogów amyloidu rozróżniamy: (1) amyloidozę układową, czyli uogólnioną, oraz (2) miejscową z tworzeniem guzów (najczęściej z łańcuchów lekkich immunoglobulin).

Opisy przypadków: W niniejszej pracy opisano 5 przypadków amyloidozy w regionie głowy i szyi. Zmiany zlokalizowane były w obrębie: krtani, nosogardła, ślinianki podjęzykowej i podżuchwowej oraz języka. Pacjenci zgłaszali objawy typowe dla procesu rozrostowego obejmującego wymienione narządy. U dwóch osób choroba miała charakter nawrotowy. Zmiany usunięto operacyjnie. Na podstawie badania histopatologicznego pobranych tkanek i charakterystycznego barwienie czerwienią Kongo i Saturna, rozpoznano obecność złogów amyloidu. U trzech pacjentów występowały stany predysponujące do wystąpienia amyloidozy: uprzednia radioterapia, przewlekły stan zapalny związany z zakażeniem wirusem zapalenia wątroby typu C i choroba przeszczep przeciwko gospodarzowi. U chorego z makroglosją rozpoznano postać uogólnioną amyloidozy.

Podsumowanie: Amyloidoza może imitować objawy zmian rozrostowych w rejonie głowy i szyi. Jej rozpoznanie wymaga badania histopatologicznego. U każdego chorego z miejscową amyloidozą musi zostać wykluczona postać uogólniona. W przypadku postaci miejscowej, resekcja chirurgiczna zmian jest postępowaniem z wyboru. Należy wziąć pod uwagę zachowanie funkcjonalności narządu.

SŁOWA KLUCZOWE: amyloidoza, amyloidoza głowy i szyi, amyloidoza języka, amyloidoza krtani, amyloidoza miejscowa, amyloidoza nosogardła, amyloidoza ślinianek

INTODUCTON

Amyloidosis is an idiopathic disease caused by accumulation of insoluble protein fibers referred to as amyloid. The disease may be localized or systemic, congenital or acquired, while most cases are associated with chronic inflammations and hematological disorders.

Several types of amyloidosis can be distinguished on the basis of the type of proteins comprising the amyloid fibers and the clinical presentation of the disease. Amyloidosis often accompanies chronic inflammations, most frequently non-specific enterocolitis, rheumatoid arthritis, juvenile idiopathic arthritis, ankylosing spondylitis, bronchiectasis, Sjögren's syndrome, systemic lupus erythematosus, tuberculosis and Reiter's syndrome [1]. In primary amyloidosis, deposits are composed of immunoglobulin light chains (AL). Secondary, or reactive, amyloidosis is observed in the course of chronic inflammation. In this case, amyloid fibers are derived from acute phase proteins. Long-term hemodialytic treatment leads to accumulation of β2-microglobulin (Aβ2M) while familial amyloidosis is usually caused by mutations of transthyretin-encoding genes.

It is difficult to assess the prevalence of amyloidosis. Estimated prevalence rate is 5–13 cases per million per year [2]. According to the estimates, localized disease accounts for 9 to 15% of all cases [2]. In about 19% of cases, amyloid deposits are located in the head and neck region [3].

Symptoms suggestive of localized amyloidosis may be observed in generalized disease, thus necessitating comprehensive diagnostics of patients due to the serious prognostic implications.

Upon electron microscopy examination, amyloid deposits present as non-branched fibers with β-sheet structure. Basic H&E staining reveals amyloid deposits as amorphous, extracellular masses of pale pink color. Microscopic image may only provide grounds for suspicion of amyloidosis as it is not disease-specific. The gold standard in the diagnosis of amyloidosis is Congo red staining (a histochemical staining assay). In the assay, amyloid fibers are stained red. Since the same color is also attained e.g. by collagen fibers, the specimens have to be examined under polarized light. In such cases, actual amyloid deposits demonstrate apple-green birefringence i.e. appear to be colored with different shades of green or yellow [4].

Within the head and neck region, cases of amyloidosis were reported within the nasal cavity, paranasal sinuses, nasopharynx, oropharynx, tonsils, oral cavity, tongue, trachea, bronchi, and larynx. The most common symptom of amyloidosis within the head and neck region is macroglossia; its prevalence is estimated at 15–20% of cases of primary light-chain amyloidosis. Larynx is the second most common location of amyloid deposits. Vestibular folds are affected in 55% of cases of laryngeal amyloidosis, followed by laryngeal ventricle in 36% of cases, subglottic space in 36% of cases, vocal folds in 27% of cases, aryepiglottic folds in 23% of cases, and anterior commissure of the larynx in 14% of cases [5]. Amyloidosis accounts for ca. 0.5–1% of benign laryngeal lesions. In contrast to glottic location, laryngeal involvement is usually associated with local deposition of AL and rarely accompanies the generalized disease. The most common symptom of laryngeal amyloidosis upon patient presentation is dysphonia; acute airway obstruction is rare. Amyloid deposition may also lead to diffuse thickening of vocal folds. Small lesions may present as smooth, non-ulcerated nodules ca. 1.5 cm in diameter. Diagnosis can be established on the basis of direct laryngoscopy, biopsy, and histopathological examination [6]. Available imaging modalities lack specific features warranting the suspicion of amyloidosis. In computed tomography (CT) scans, amyloid deposits have been reported as soft tissue densities whereas in magnetic resonance scans, amyloid-affected tissues are isointense with skeletal muscles [7].

Laryngeal involvement is usually reported in adult patients, mainly in the 5th decade of life; some exceptional pediatric cases were also observed. Male to female prevalence ratio is 3:1 [8].

Salivary gland amyloidosis is very rare and may be observed as either localized or systemic disease. Usually, it is characterized by smooth or lobular gland enlargement [9].

CASE REPORTS

Patient 1 Laryngeal amyloidosis

A 69-year-old male patient was hospitalized four times at the Department of Otolaryngology of the Medical University of Warsaw in years 2013 to 2018 due to recurrent hypertrophic lesions within the larynx and the resulting hoarse voice. During the first stay, directoscopy and subsequent histopathological examination of laryngeal specimens revealed low-grade epithelial dysplasia and amyloid deposits present within the perivascular parenchyma. The patient was followed up in an outpatient setting; videostroboscopic examinations revealed periodic recurrence of hypertrophic lesions, primarily affecting vestibular folds, leading to three subsequent hospitalizations at the Department of Otolaryngology with microsurgical laser procedures being performed in the patient's larynx (Fig. 1.). Mucosal hypertrophy of both vestibular folds and laryngeal ventricules, smooth and soft to the touch, non-ulcerated, was observed intraoperatively; the tissue was with a fat appearance but more solid in cross-section. Each time, histopathological examination of specimens from hypertrophic laryngeal mucosa revealed the presence of amyloid deposits without signs of dysplasia. The patient give the history ofćńarterial hypertension and was a non-smoker. He was consulted by specialists in internal medicine and hematology to exclude the generalized form of the disease. For the last year, the patient has been followed up with no recurrence of laryngeal lesions being observed in that time.

Patient 2 Sublingual salivary gland amyloidosis

A 43-year-old patient was admitted to the Department of Otolaryngology of the Medical University of Warsaw for oral cavity tumor management. The patient had noticed the lesion six months before presentation; she also reported tongue parestesis. Physical examination revealed a well-defined tumor of the oral cavity floor at the site of the left sublingual salivary gland; the tumor was 1.5 x 2 cm in size and was not tender upon palpation. The patient had

been previously treated for hepatitis C. She had been diagnosed with hypothyroidism in the course of the Hashimoto disease and was receiving hormone replacement therapy. Ultrasound scanning revealed a well-defined, hypoechogenic, heterogeneous oral floor lesion sized 3.3 x 1.5 cm, with no vascular enhancement being observed in the power Doppler mode. Submandibular salivary glands were unremarkable and cervical lymph nodes were not enlarged (Fig. 2.). Surgery performed from transoral approach revealed an encapsulated, solid tumor located laterally to the submandibular salivary gland outflow duct and adjacent to the internal mandibular surface, on the mylohyoid muscle, with no adhesions to the surrounding structures. Sublingual salivary gland tissue was identified posteriorly to the tumor. The lesion was dissected in its entirety along with the adjacent salivary gland tissue, with the continuity of the outflow duct being maintained intact. Histopathological examination revealed amyloid deposits present within the salivary gland and the adjacent fibrotic tissue as well as within the blood vessel walls, as confirmed by Congo red staining. Healing was uncomplicated. For a year, the patient has been followed up without signs of local recurrence. Systemic amyloidosis was excluded.

Patient 3 Amyloidosis of the tongue and submandibular salivary gland

A 59-year-old male patient was admitted to the Department of Otolaryngology of the Medical University of Warsaw with a suspected proliferative lesion of the tongue. More than 14 years before, the patient had received an allogeneic hematopoietic stem cell transplantation due to chronic myeloid leukemia. After the transplant, the patient was diagnosed with chronic graft versus host disease and was followed up at the Department of Hematology, Oncology, and Internal Medicine of the Medical University of Warsaw without signs of recurrent leukemia. The patient declared to have been experiencing recurrent erosions and ulcers as well as dry mouth sensation within the oral cavity. In the recent three months, he observed a deterioration in tongue mobility along with the accompanying dysphagia and dysarthria. The patient also reported to have experienced subfebrile temperatures and significant body weight loss.

Physical examination revealed enlargement of the entire tongue, increased solidity upon palpation, and impaired tongue mobility. In addition, signs of fungal infections of the oral cavity were detected. Numerous enlarged lymph nodes were palpable on both sides of the neck, their diameters up to 2 cm. Submandibular salivary glands were bilaterally enlarged and solid. Laboratory analyses revealed polyclonal hypergammaglobulinemia of the IgG class, hypoalbuminemia, and slightly elevated C-reactive protein (CRP) levels.

A magnetic resonance imaging (MRI) scan of the neck revealed entirely enlarged, cauliflower-like tongue with an infiltration subject to pathological enhancement upon contrast administration; the infiltrate involved mainly the base of the tongue and oral cavity floor muscles, namely both genioglossus and geniohyoid muscles. The infiltrate extended to the lateral wall of the oropharynx on the right and the parapharyngeal space. Enlarged cervical lymph nodes were also visualized on the right, their diameters not exceeding 2 cm (Fig. 3.).

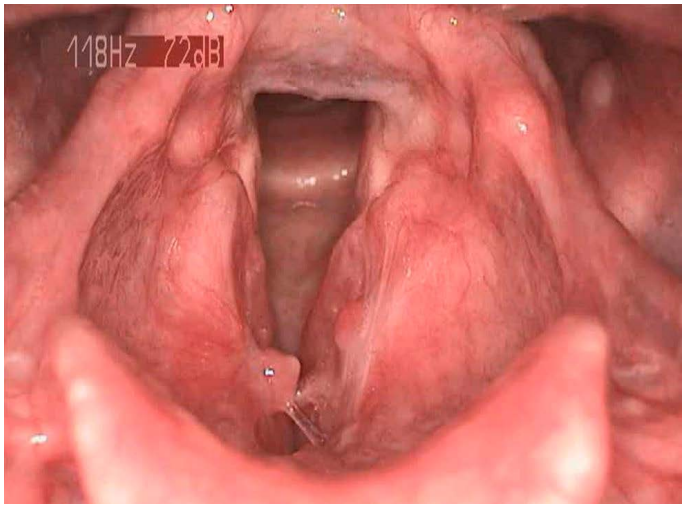

Fig. 1. Videolaryngostroboscopic examination of the larynx in Patient 1 reveals bilateral hypertrophy of vestibular folds and laryngeal ventricules.

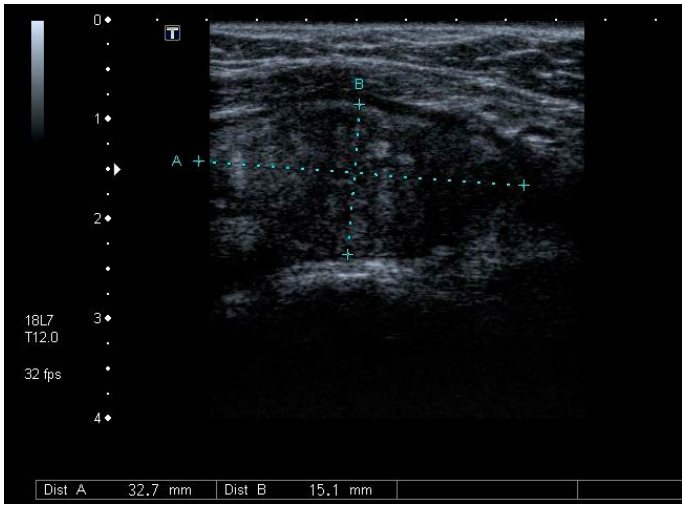

Fig. 2. Ultrasound scan of the left submandibular region in Patient 2 reveals well-defined, hypoechogenic, non-homogeneous lesion sized 33 x 15 mm at the oral cavity floor.

Directoscopy was performed to reveal an infiltrate involving the base of the tongue, mainly on the right, reaching beyond the medial line. Ulceration was observed at the oral cavity floor, on the right. Specimens of the tongue and oral cavity floor were collected for histopathological examination. Right submandibular salivary gland was removed and its posterior pole was in contact with the infiltrate involving suprahyoid muscles. Tracheostomy and gastrostomy were performed. Histopathological examination of the oral cavity floor, tongue, and submandibular salivary gland lesion using Congo red and Saturn red staining revealed greenish gleam under polarized light corresponding to amyloid deposits within the tissues of all structures (Fig. 4.). The patient was referred to the Department of Hematology where systemic amyloidosis was confirmed and appropriate treatment was initiated.

Patient 4 Laryngeal amyloidosis

A 57-year-old patient with a history of radiation therapy performed three years earlier due to laryngeal cancer was admitted to the Department of Otolaryngology due to the suspected tumor recurrence.

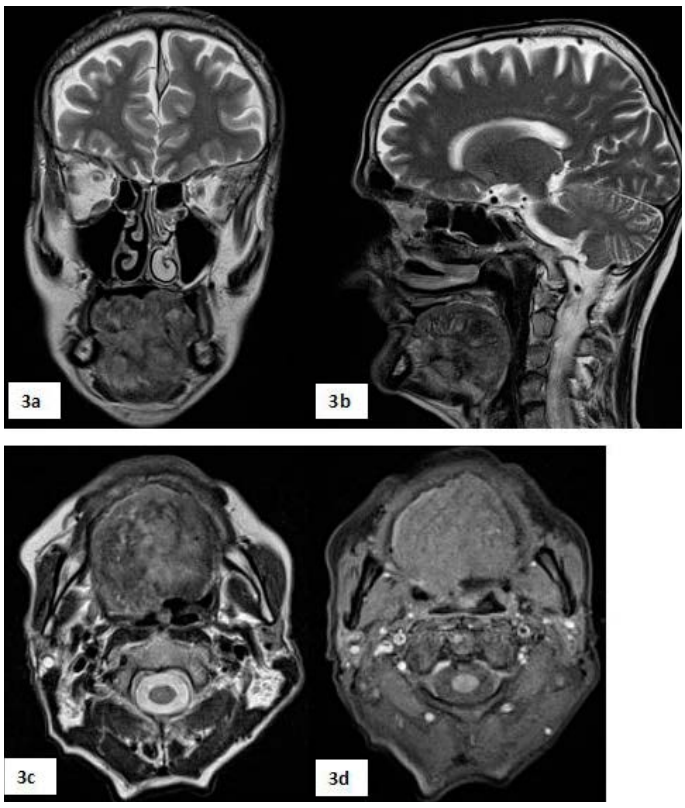

Fig. 3. Magnetic resonance scan of the neck in Patient 3 reveals entirely enlarged, cauliflower-like tongue: frontal (3a), sagittal (3b), and transverse (3c) projections; contrast administration revealed pathological enhancement of the entire tongue and the lateral pharyngeal wall (3d).

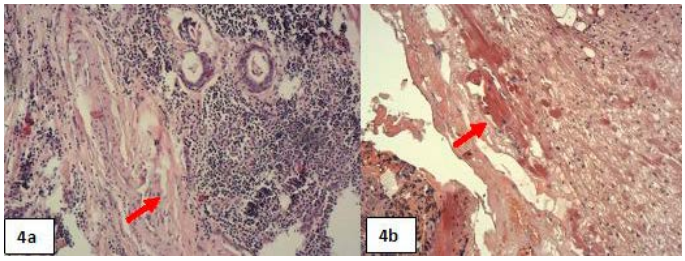

Fig. 4. Histological examination of submandibular salivary gland in Patient 3: 4a. Hematoxylin and eosin staining (pink-stained amyloid deposits marked with an arrow), 4b. Congo red staining (red-stained amyloid deposits marked with an arrow).

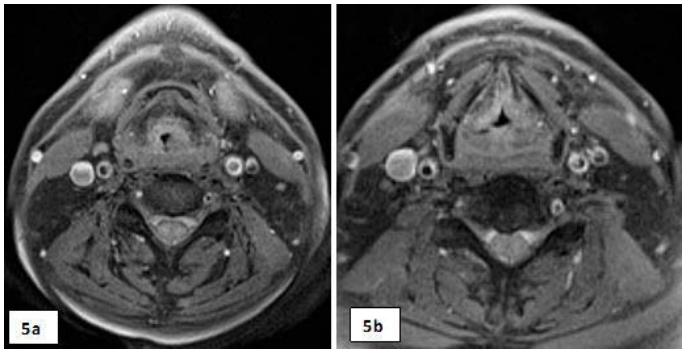

Fig. 5. Magnetic resonance scan of the neck in patient 4 reveals thickening of laryngeal structures at the levels of ary-epiglottic folds (5a) and vestibular folds (5b), homogeneously enhanced following contrast administration.

Otolaryngological examination of the larynx revealed epiglottic swelling and hypertrophic lesions of vestibular folds. MRI scan revealed generalized thickening of soft laryngeal structures starting from ary-epiglottic folds to the glottic level with laryngeal lumen narrowed down to ca. 5 mm in transverse cross-section at the vestibular folds level. Pathological laryngeal tissues enhanced homogeneously following contrast administration. Progression of lesions was observed compared to an examination performed one year before (Fig. 5.).

Directoscopy was performed under general anesthesia to reveal swollen epiglottic structures including epiglottis, vestibular folds, arytenoids, as well as hypertrophic base of the tongue. Vestibular folds were partially removed and specimens were collected for histopathological examination from the tongue base. Histopathological examination of laryngeal specimens revealed generalized inflammation and amyloid deposits. Systematic amyloidosis was excluded.

Patient 5 Nasopharyngeal amyloidosis

A 61-year-old patient was admitted to the Department of Otolaryngology due to suspected recurrence of nasopharyngeal tumor as suggested by follow-up MRI scan. Examination revealed a well-defined, contrast-enhanced lesion sized 3.2 x 2.2 cm, adjacent to the nasopharyngeal roof, penetrating into the left parapharyngeal space and reaching the internal carotid artery. Patient reported no complaints other then difficulties in nasal breathing. Medical history as reported by the patient included a resection of nasophayngeal tumor on the left six years before. Histopathological examination of tumor reveled amyloidosis (AL and transthyretin deposits). The patient had been diagnosed at the Department of Hematology, where lymphoproliferative process or systemic amyloidosis were excluded. Patient was diagnosed with euthyreotic nodular goiter and arterial hypertension. With 45° endoscopic monitoring, soft palate was pulled away and the tumor was excised from the nasopharynx and parapharyngeal space. Histopathological examination confirmed the presence of amyloid deposits within the excised tumor mass.

DISCUSSION

Amyloidosis is a group of diseases caused by extracellular accumulation of insoluble protein fibers with a secondary β-sheet structure which disturb the function of affected organs [10]. The disease is usually diagnosed between the fourth and the sixth decade of life. Localized disease is observed in a younger population compared to the generalized disorder [11]. About 20% of diagnosed cases of amyloidosis involve the head and neck area [4].

The study describes 5 cases of amyloidosis within the head and neck region. The lesions were located within the larynx, oropharynx, sublingual salivary gland, submandibular salivary gland, and the tongue. Patients reported symptoms typical for proliferative processes within the aforementioned organs. In two of these cases, the disease was of recurrent nature. Three patients had conditions which predisposed them to amyloidosis such as history of radiation therapy, chronic inflammation related to hepatitis C, and graft versus host disease. Systemic amyloidosis was diagnosed in a patient with macroglossia.

Local amyloidosis within the larynx accounts for less than 1% of benign tumors within that organ [8]. Amyloidosis of the larynx is usually due to a localized form of the disorder and rarely accompanies the systemic form [12]. Regardless of the statistical considerations, generalized amyloidosis should be excluded in all cases of localized disease. Methods used in the diagnosis of generalized amyloidosis include aspiration biopsy of the abdominal wall adipose tissue (sensitivity 54–82%), biopsy of rectal mucosa (sensitivity 69–97%), biopsy of labial mucosa with tiny salivary glands (sensitivity 80–100%) or, rarely, bone marrow biopsy [13]. In localized amyloidosis, endoscopic resection of lesions is the procedure of choice; however, open surgeries may be needed in advanced disease [14]. Radiation therapy is controversial and should be considered only in nonresectable lesions [12]. The prognosis of laryngeal amyloidosis is very good; however, patients should be followed up for 5–7 years due to the possibility of disease recurrence [15].

Deposition of amyloid within the parotid gland is very rare and only several cases have been reported to date. Usually, salivary gland amyloidosis is associated with systemic form of the disease. Usually, deposits are accumulated in superficial regions of the parotid gland with no resultant impairment of facial nerve function. Amyloidosis of salivary glands may also lead to Sjögren's syndrome [16, 17]. Localized nasopharyngeal amyloidosis is very rare and accounts for about 3% of lesions within the head and neck region [17]. Relevant cases were described in as few as 15 articles in worldwide literature, including the case presented herein [2]. Symptoms are not disease-specific. As in the case of other pathologies developing within the nasopharynx, patients report impaired nasal patency, impaired hearing, recurrent otitis media and Eustachian tube dysfunction. Resection of lesions is the treatment of choice. No evidence is available for potential tendency to progression and generalization of untreated localized disease. In patients with high surgery-related risk, monitoring of localized amyloidosis and follow-up on the disease development is recommended since despite their benign character, amyloid deposits lead to an enlargement of the affected organ. Resection of localized amyloidosis should be taken into consideration in all patients [18].

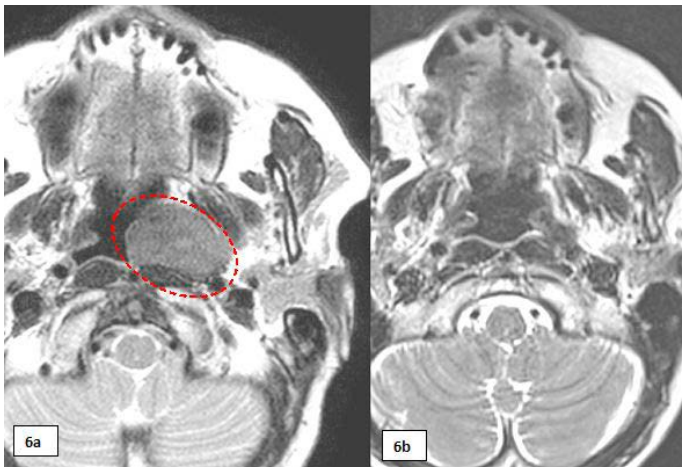

Fig. 6. Magnetic resonance (MRI) scan acquired in Patient 5 reveals nasopharyngeal tumor on the left – encircled with a dashed line (6a); whereas follow-up MRI scan one year after the procedure reveals no signs of amyloidosis recurrence.

SUMMARY

Localized amyloidosis within the head and neck region is a very rare disorder characterized by slow progression and usually affecting the larynx, salivary gland, and laryngeal lymphatic tissues. Disease symptoms and imaging studies are non-specific. Diagnosis may be determined on the basis of histopathological examination. Generalized disease must be ruled out in every patient with localized amyloidosis by a multidisciplinary team of physicians. Surgical resection of localized lesions with the maintenance of affected organ function is the management of choice. It is recommended that patients are followed up for at least 7 years due to the risk of recurrence.

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