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Case Report

Adenoid Hypertrophy in Adults: An Underdiagnosed Entity?

Abstract

Adenoid enlargement is uncommon in adults. We studied the varied presentation of adenoid hypertrophy in two adults. A 45 year old male who presented with nasal obstruction and snoring, and a 35 year old male who presented with non resolving chronic otitis media. In both the patients, adenoid hypertrophy was diagnosed by nasal endoscopy and confirmed by CT scan. Adenoidectomy in these patients resulted in symptomatic improvement. Therefore, adenoid hypertrophy should also be considered as a cause or contributing factor in nasal obstruction and related pathologies in adults.

Introduction

The adenoids (or pharyngeal tonsil) is a condensation of lymphoid tissue in the posterosuperior wall of the nasopharynx. It forms a part of Waldeyer's ring of lymphoid tissue at the portal of entry of the upper respiratory tract [1]. In early childhood it is the first site for immunological contact of inhaled allergens. Adenoids are present at birth, show physiological enlargement upto 6 years of age, atrophy at puberty and almost completely disappear by 20 years of age [2]. Clinical symptoms are common in young age due to small volume of nasopharynx and the increased frequency of upper respiratory tract infections. It appears to have an important role in the development of immunological memory in younger children [3]. Adenoid hypertrophy was considered uncommon in adults as examination of the nasopharynx by posterior rhinoscopy was inadequate. Many cases were misdiagnosed and accordingly maltreated [4]. However, in the current clinical practice, with the advent of nasal endoscopes and improved imaging modalities, adenoidal tissue is not uncommonly found in adults. Insipite of these developments, there is lack of data regarding adenoid hypertrophy in adults. We studied the varied presentation of two cases of adult adenoid hypertrophy who presented with nasal obstruction and snoring; and non-resolving chronic otitis media.

Case 1

A 45 year old male presented to the ENT OPD, Lok Nayak Hospital, with complaints of nasal obstruction, snoring and repeated awakening at night due to apnea spells. On detailed history, it was found that patient was a chronic smoker for past 25 years. On examination, nasal endoscopy revealed a pale lobulated mass in the nasopharynx (Figure 1). Contrast enhanced CT scan showed a non-enhancing mass in the nasopharynx, extending upto bilateral nasal cavity, suggestive of adenoid hypertrophy (Figure 2). Nasal endoscopic biopsy was done which was suggestive of lymphoid hyperplasia. The patient was managed conservatively for 3 months with steroid nasal spray. However, there was no improvement in the symptoms and patient was planned for surgery. Transnasal endoscopic powered adenoidectomy was done under general anesthesia. Post-operative biopsy report was suggestive of adenoid hyperplasia. Adenoidectomy resulted in resolution of symptoms and immediate improvement in sleep. Topical steroid nasal spray was continued for one year postoperatively. After one year follow up, the patient was asymptomatic. There was no residual tissue in the nasopharynx or any recurrence.

Case 2

A 35 year old male presented with bilateral chronic otitis media with mucosal disease. Even after tympanoplasty with cortical mastoidectomy, patient had persistent ear discharge. There was only temporary relief of symptoms and an underlying

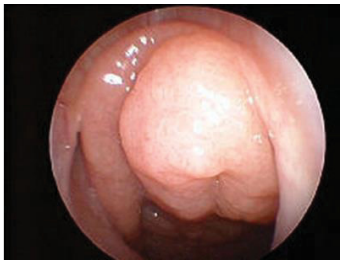

Figure 1: Nasal endoscopy showing enlarged adenoids.

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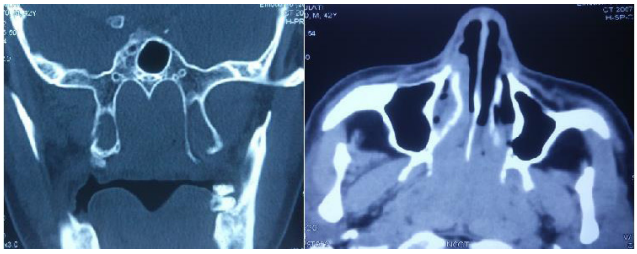

Figure 2: CT scan: coronal and axial views showing enlarged adenoids.

pathology was sought for. Nasal endoscopy revealed a lobulated mass in the nasopharynx. Contrast enhanced CT scan indicated a non-enhancing mass in the nasopharynx. Nasal endoscopic biopsy was done and histopathology report was suggestive of hypertrophied lymphoid tissue. Transnasal endoscopic powered adenoidectomy was done under general anesthesia. After a few months of surgery, the ear remained dry. Successful revision tympanoplasty was performed after 6 months. After one year follow up the patient was asymptomatic. Following surgery, there were no progress in the adenoid hyperplasia and related symptoms.

Discussion

Adenoid is condensation of lymphoid tissue in the posterosuperior part of the nasopharynx. Santorini described the nasopharyngeal lymphoid aggregate or 'Lushka tonsil' in 1724. The term adenoid was coined by Wilhelm in 1870, to what he described as nasopharyngeal vegetations [5]. Along with palatine tonsils, lingual tonsils, tubal tonsils and the lateral pharyngeal bands, it forms the inner Waldeyer's ring. Adenoids protect the body from airborne infections and produce antibodies providing immunity. They have an important role in the development of 'immunological memory' in young children [3].

Adenoids are present at birth, show physiological enlargement upto 6 years of age, atrophy at puberty and almost completely disappear by 20 years of age [2]. Although adenoid tissue undergoes regression towards the adolescent period, but adenoid hypertrophy is also seen in the asymptomatic adult population. Adenoid enlargement was considered uncommon in adults as examination of the nasopharynx by posterior rhinoscopy was inadequate and hence, many cases of adenoid hypertrophy in adults were misdiagnosed and accordingly maltreated [4]. However, with the routine use of nasal endoscopes in the OPD and increasing availability of imaging modalities like CT scans, adenoid hypertrophy is increasingly being diagnosed in adults. In our cases, adenoid hypertrophy was diagnosed with the help of nasal endoscopy and confirmed by CT scan.

Although the exact cause of adenoid hypertrophy in adults is not known, but various aetiopathogenetic mechanisms have been proposed. The persistence of childhood adenoids could be due to chronic inflammation [4] or re-proliferation of regressed adenoid tissue in response to infection or irritants [6]. Finkelstein et al. [7], reported the presence of obstructed adenoids in 30% of heavy smokers. Our first case was a chronic smoker as well. Adenoid hypertrophy caused by viruses in adults with compromised immunity, especially those receiving organ transplantation and those with HIV, is a well-known phenomenon [8]. However, this has been excluded as a reason for adenoid hypertrophy in our study.

There are various clinical features that can be associated with adenoid hypertrophy. Patients can have nasal obstruction, which may result in oral breathing, recurrent nasal infections and hypo nasal speech [4]. Higher percentage of children with adenoid hypertrophy have been reported to suffer from snoring compared to adults [2]. It can also be associated with varied ear symptoms due to Eustachian tube blockage, such as, otitis media with effusion, retraction, non-healing chronic otitis media, as seen in our second case. Kamel et al. [4], studied 35 adult patients with adenoidal hypertrophy. Five patients had bilateral secretory otitis media (OME), 18 had nasal obstruction and four presented with snoring. Following adenoidectomy, four of the five patients with OME improved, while one required subsequent ventilation tube insertion. Biopsy from all 35 patients were benign. The authors also noted histological differences in the adenoidal tissue between adults and children, in that adult adenoidal tissue was either smooth or irregular and did not show crypts or vertical furrows. A study conducted by Yildrim et al. [2], in 2008 showed etiopathological characteristics of adult and childhood adenoid hypertrophy (40 adults and 23 children). Both adenoid hypertrophy forms were similar in terms of symptomatology and associated inflammations. However, there were significant differences in otitis media rate, with effusion and dullness, and retraction in the tympanic membrane, both more prevalent in childhood adenoid hypertrophy. Adult adenoid hypertrophy was associated with nasal septum deviation in 25% of patients. These results underline the importance of considering adenoid hypertrophy as a cause or contributing factor in nasal obstruction and related pathologies in adults and support the theory that it represents a long standing inflammatory process rather than being a novel clinical entity.

There is scarcity of data regarding incidence and etiological factors responsible for adenoid hypertrophy in adults. Kapusuz [9], reported the prevalence of adenoid hypertrophy in adults as 26.28% (139 out of 525 patients) in Turkey, Hamad [10], 17% (24 out of 140 patients) and Rout [5], 21% (21 out of 100 patients). In studies where they have been compared to normal sized adenoids, a chronic infection with Hemophilus influenza, normal bacteria of the upper respiratory tract, has been identified. There may be some adenoidal enlargement occurring with chronic allergic states. In adult adenoid hyperplasia, malignancies of B type blood cells (lymphoma palsmacytoma) or HIV must be considered [5]. In a series by Rout et al. [5], allergy was associated with 30% of the adult adenoid hypertrophy. HIV infection was associated with 3.3% of the cases and non-Hodgkin's and other sinonasal malignancy was associated with 3.3% cases each.

Adenoid hypertrophy can be reduced by long term steroid nasal spray. Surgery is indicated in those individuals who don't respond to medical management. In a study by Demirhan

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et al, [11], in 2010, showed that in 76% of patients with adenoid hypertrophy, surgery was eliminated with fluticasone proprionate nasal spray. However, if the adenoid re-enlarges and recreates the symptoms, surgery would be necessary, as in our cases.

The adenoid enlargement in adults is not uncommon, and often underestimated in adults with nasal obstruction and should always be borne in mind as a cause for otology or rhinology symptoms, as seen in both our patients. However, more studies are required to find out the incidence of adenoid hypertrophy in adults and its relation with smoking and allergy.

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