Lateral Radiography of Nasopharynx vs Rigid Nasal Endoscopy" for Adenoid Hypertrophy - The utility reiterated

First Author	Dr. Apeksha Raj [Post Graduate III year]
Second Author	Dr .SBV.Chandrasekhar [Associate professor] Mobile No : 9908029872
Third Author	Dr. Tanvi Gadde [Senior Resident]
Fourth Author	Dr. S. Indira Devi [Professor & HOD] Mobile No: 9959360123
Fifth Author	Dr.N.Venkatrama Reddy [Professor]
Name of Corresponding Author	Dr .P. Ramakrishnaiah [professor] Mobile No: 9966791501

Subject : ENT [OTO RHINO LARYNGOLOGY]

Full Address for Hard Copy With Name :

Dr. P.Ramakrishniah ,Professor ,Department of ENT ,Bhaskar Medical college , Yenkapally , Moinabad Mandal , Ranga Reddy District, Telangana

Mobile No. **9966791501**

Abstract

Aim : To correlate adenoid hypertrophy clinico-radiological findings with rigid diagnostic nasal endoscopic findings

Patients and Methods: A cross-sectional study conducted over 18 months to evaluate the correlation between clinical symptoms, radiological imaging, and endoscopic findings in patients diagnosed with adenoid hypertrophy (AH). 80 patients between the ages of 5 and 50 years, presenting with symptoms such as nasal obstruction, snoring, mouth breathing, recurrent upper respiratory tract infections (URTIs), hearing loss associated with otitis media with effusion (OME), and adenoid facies, were enrolled. Patients with congenital anomalies, previous surgeries, craniofacial syndromes, or other interfering nasopharyngeal conditions were excluded.

After obtaining ethical clearance and informed consent, a structured survey proforma was used to collect comprehensive demographic and clinical data, including medical, personal, and family history. A complete general physical and systemic examination was followed by an ENT assessment. Clinical evaluation involved the Brodsky-Moore-staneiwich grading for tonsillar size. Radiological assessment of adenoid size was done using the lateral view X-ray of the nasopharynx and graded using the Fujikoshi (Fujioka) method based on the adenoid/nasopharynx (A/N) ratio. Endoscopic evaluation with a rigid naso-pharyngo-scope assessed the degree of choanal obstruction and was graded using the Clemens grading system.

All patients underwent baseline laboratory investigations including hematological, biochemical, serological, and imaging tests to ensure safe management. Every subject initially received conservative medical management comprising intranasal mometasone spray and oral antihistamines. Surgical intervention was considered in cases where symptoms persisted or were severe, with 90% undergoing conventional adeno-tonsillectomy and 10% undergoing coblation-assisted procedures.

- **Results:** The study highlights that adenoid hypertrophy predominantly affects school-aged children, with a mean age of 9.83 years, and is more commonly observed among males and individuals from lower-middle socioeconomic backgrounds. Snoring and mouth breathing were the most frequent clinical complaints, reflecting the typical symptom profile of AH. Radiological and endoscopic evaluations both revealed a predominance of

moderate-grade hypertrophy, with a strong positive correlation ($r = 0.818$, $p < 0.001$) between the two modalities. This indicates that although endoscopy remains the gold standard, lateral X-ray continues to be a practical and reliable tool in settings where advanced endoscopic facilities are not readily available even in this era of generation -z children.

The study also identified significant co-existing conditions, such as tonsillar hypertrophy, deviated nasal septum, and inferior turbinate hypertrophy, underscoring the multifactorial nature of upper airway obstruction in these patients.

Universal initial treatment with intranasal corticosteroids and antihistamines proved to be a rational and effective first-line approach. However, surgical intervention remained necessary in the majority of cases, with conventional adenotonsillectomy being the preferred method.

- **Conclusion:** X-ray Nasopharynx is of importance in the diagnosis of Adenoid Hypertrophy ,especially in children in whom performing endoscopy is a tedious task and proves handy for the decision making . Inspite of better diagnostic tools like CT PNS and MRI , X-Ray Nasopharynx lateral view for soft tissues stands the test of time , being economical ,easy to perform, rapid ,limited radiation and scientific for a decision making in Adenoid Hypertrophy.
- **Key words:** Adenoid Hypertrophy ,Rigid Nasopharyngoscopy ,Diagnostic Nasal Endoscopy,Adenoidectomy

INTRODUCTION

Adenoids, also known as nasopharyngeal tonsils, are normal lymphoid tissues present on the junction of roof and posterior wall of the nasopharynx, forming a part of Waldeyer's ring at the entry of the upper respiratory tract. Adenoids are the first site of immunological contact for inhaled antigens in early childhood. They produce B cells, which give rise to IgG and IgA plasma cells. They provide natural acquired immunity in early childhood and appear to have an important role in developing an 'immunological memory' in younger children [1].

Adenoids become evident by 6 months to 1 year of life, increase rapidly in size during the first 6–8 years of life, and generally atrophy by 15 years of age in most children [2]. The growth of adenoid tissues is not in agreement with the growth of the bony nasopharynx, leading to nasal obstructive symptoms of adenoid hypertrophy (AH).[3.4]

Adenoid hypertrophy is related to an increased size of the adenoids, which can occur with or without an acute or chronic infection of the adenoids. This condition is more common in children, and the prevalence has been estimated at 34.5%.[5] It has been demonstrated that adenoid hypertrophy is also seen in the normal adult population and may cause nasal obstruction. Adenoid enlargement is uncommon in adults, and because examination of the nasopharynx by indirect posterior-rhinoscopy is inadequate, many cases of enlarged

adenoid in adults are misdiagnosed and accordingly mistreated. The presence of lymphoid hyperplasia in the adult nasopharynx, including the persistence of childhood adenoids, is associated with chronic inflammation. Regressed adenoidal tissue may re-proliferate in response to infections and irritants.[6]

Adenoid hypertrophy can occur because of infectious and non-infectious etiologies. Infectious causes of adenoid hypertrophy include both viral and bacterial pathogens. Viral pathogens include adenovirus, coronavirus, Epstein-Barr virus (EBV), herpes simplex virus, and para-influenza virus.[7] Many aerobic bacterial species have been implicated in contributing to infectious adenoid hypertrophy, including Streptococcus species, Staphylococci, Neisseria, Corynebacterium, and Mycoplasma pneumonia.[8] *Fuso-bacterium* and *Prevotella* have also been identified as anaerobic organisms involved in infectious adenoid hypertrophy. [9] Multiple non-infectious causes of adenoid hypertrophy have also been suggested, including gastro-esophageal reflux, allergies, and exposure to cigarette smoke. In adults, adenoid hypertrophy can also be a sign of a more serious condition such as HIV infection, lymphoma, or malignancy.[10-11]

The increase in size of the germinal centers of the lymphoid tissue and lymphoid follicles is the pathological and anatomical basis of hypertrophy. A vicious cycle consisting of inflammation, hypertrophy and/or hyperplasia, retention of secretions, and recurrent inflammation is assumed to be the underlying cause. Inflammatory cytokines, including **Interleukin-1 (IL-1), Interleukin-6 (IL-6),** and **Tumor Necrosis Factor-alpha (TNF-α),** are released, promoting tissue proliferation and vascular permeability. Additionally, **Vascular Endothelial Growth Factor (VEGF)** contributes to angiogenesis, further

exacerbating tissue enlargement. Allergies or other types of antigen exposure may also play a role, where **IgE-mediated hypersensitivity** leads to persistent eosinophilic inflammation, intensifying the hypertrophic process. The combined effects of immune overactivation, persistent inflammation, cytokine release, and environmental influences result in the pathological enlargement of the adenoids. [12]

Nasal obstruction by hypertrophic adenoid tissue can cause the patient to complain of rhinorrhea, difficulty breathing through the nose, chronic cough, post-nasal drip, snoring, and/or sleep-disordered breathing in children. If the nasal obstruction is significant, the patient can suffer from sinusitis as a result and may complain of facial pain or pressure. Obstruction of the Eustachian tube can lead to symptoms consistent with Eustachian tube dysfunction, such as muffled hearing, otalgia, crackling or popping sounds in the ear, and/or recurrent middle ear infections.

On physical exam, the patient with adenoid hypertrophy will often breathe through the mouth, have a hypo-nasal character to the voice, and may have the facial characteristics known as adenoid facies. Adenoid facies is commonly known as “long face syndrome” as it’s characterized by a long, lean face with a slightly open mouth. Individuals with adenoid facies typically have an arched palate, underdeveloped upper jaw bones, a short upper lip, elevated nostrils, and dental crowding of the front teeth. These facial features are the result of mouth breathing from chronic nasal obstruction and can be seen in young children whose bone structure is still developing. [13-14]

Figure 1- Adenoid facies

Appropriate diagnostics play a crucial role in determining the most effective approach for managing adenoid hypertrophy. The decision to undergo adenoidectomy should be made cautiously, as the impact of post-adenoidectomy on the immune system remains a topic of debate. Clinical judgment for recommending adenoidectomy should be based on a combination of clinical presentation, physical examination, and supporting diagnostic tests.[15-16] A thorough clinical assessment, including history taking and physical examination, can be quantified using questionnaires, clinical scores, and nasal obstruction evaluations.[17] However, objective investigations are essential to accurately determine the degree of adenoid hypertrophy. Evaluating adenoid size in the nasopharynx is challenging due to limited visibility, making it necessary to use reliable assessment methods. Several diagnostic tools are available, including posterior rhinoscopy, acoustic rhinometry, rhinomanometry, lateral cervical X-ray, and nasopharyngoscopy. Among these, nasopharyngoscopy and radiological examinations are the most commonly used modalities in clinical practice [18].

Radiological examinations have been widely used to assess adenoid size due to their simplicity, ease of performance, and reproducibility. However, several factors can limit their effectiveness, including anatomical distortions caused by patient movement, particularly in children, breathing-related motion artifacts, radiation exposure, and the interpretation of two-dimensional images that may not fully reflect the actual condition of the adenoids.[19] Another method for evaluating adenoid hypertrophy is nasopharyngoscopy, considered the gold standard for direct visualization of adenoid size. Despite its advantages, nasopharyngoscopy is not available in all healthcare facilities. Additionally, its effectiveness is subjective, relying heavily on the examiner's skill, and it can be challenging to perform due to the uncooperative behavior often exhibited by pediatric patients.[20-21]

Each diagnostic modality for adenoid examination comes with its own set of advantages and limitations. Therefore, there is a need to develop a clinically based diagnostic approach for adenoid hypertrophy, complemented by objective supplementary examinations. This study aimed to explore the correlation between the clinical presentation of adenoid hypertrophy and the findings from radiological and nasopharyngoscopy examinations. The results of this study can serve as a practical guide for general practitioners in primary care to determine whether a patient requires referral to a higher-level health facility. Additionally, it provides guidance for Otorhinolaryngology-Head and Neck Surgery (ORL-HNS) specialists in deciding on adenoidectomy based on clinical, radiological, or nasopharyngoscopy findings, ultimately contributing to better health outcomes and improved quality of medical decision.

The adenoid is one of the important lymphoid tissues of the Waldeyer ring and one of the body's first line of immune defense and effector organs in both mucosal-type and systemic-type adaptive immunity.[22] In general, it attains its maximum size between 3 and 7 years and then regresses. However, because of various etiologies, such as upper respiratory tract infection, allergic episodes, and others, it becomes hypertrophied, and this enlargement in its size may lead to certain consequences, such as nasal obstruction, snoring and mouth breathing, sleep disturbance, Eustachian tube obstruction, otitis media, failure to thrive, and maxillofacial growth anomalies, in young children.[23]

Pereira et al [5] conducted a systematic review, wherein a total of 5248 patients were included. Seventeen studies were included in the meta-analysis showing an AH prevalence of 49.70% (confidence interval (CI): 39.92 to 59.50).

Adenoid hypertrophy is an obstructive condition due to enlarged adenoids. This can occur with or without an acute or chronic infection of the adenoids. AH is often underestimated in adults with nasal obstruction and is often misdiagnosed as a nasal or sinus pathology. Adenoids are physiologically enlarged in size from birth up to the age of 6 years, and after that they gradually regress in size to disappear completely at nearly 16 years of age, but they may persist into adult life. Cowan in 1982 described the persistence of adenoid tissues in the non-pediatric age group.[37] Theobald in 1948 and Heffner in 1987 also reported marked hyperplasia of adenoid tissues in adults [38-39]. A study conducted by Rout et al [40] showed that the most frequently involved age group in adults is 16- 25

years, and adult males are more commonly involved than females, maybe due to more exposure to outdoor pollutants.

Etiology

- **Infectious Causes**: The adenoids play a crucial role in the immune system by trapping pathogens that enter through the nose and mouth. As a result, they are susceptible to infections, which can lead to their enlargement.[8]
- **Viral Infections:** Common viruses, such as the Epstein–Barr virus, adenovirus, coronavirus, coxsackievirus, cytomegalovirus (CMV), herpes simplex virus, para-influenza virus, and rhinovirus, can infect the adenoids, causing inflammation and subsequent hypertrophy.

- **Bacterial Infections:** Bacterial pathogens like alpha-, beta-, and gamma-hemolytic Streptococcus species, H. Influenzae, Staphylococcus aureus,N. gonorrhoeae, C. diphtheriae, C. pneumoniae, and M. pneumoniae.

- Chronic or recurrent infections can result in persistent inflammation and enlargement of the adenoidal tissue.

- **Immune System Factors:** Alterations in immune responses are believed to contribute significantly to adenoid hypertrophy.[12]
- **Cytokine Production:** Studies have shown that children with adenoid hypertrophy exhibit elevated levels of pro-inflammatory cytokines, such as interferon-γ (IFN-γ), interleukin-1 (IL-1), IL-10, and tumor necrosis factor-α (TNF-α). These cytokines can promote inflammation and tissue growth within the adenoids.
- **Immune Reactions:** Aberrant immune reactions, possibly due to environmental exposures or allergens, may lead to chronic inflammation and subsequent hypertrophy of the adenoidal tissue.

- **Genetic Factors:** Genetic predisposition plays a role in the development of adenoid hypertrophy.

- **Family History:** A positive family history of adenoid hypertrophy suggests a hereditary component. Specific genetic variations, such as polymorphisms in genes encoding Toll-like receptors (TLR2 and TLR4) and SCGB1D4,

  have been associated with an increased susceptibility to this condition. [41]
- **Environmental Exposures:** Certain environmental factors have been linked to

the development of adenoid hypertrophy.

- **Passive Smoking:** Exposure to secondhand smoke has been identified as a risk factor, potentially due to its irritant effects on the respiratory mucosa, leading to chronic inflammation.[42]

- **Air Pollution:** Irritants present in polluted air can cause chronic inflammation of the adenoidal tissue, contributing to its enlargement. [40]
- **Gastro-esophageal Reflux Disease (GERD):** GERD has been implicated as a potential cause of adenoid hypertrophy, especially in infants and young children. The reflux of gastric contents into the nasopharynx can lead to inflammation and subsequent enlargement of the adenoids.[43]
- **Allergic Reactions:** Allergies can cause chronic inflammation of the adenoids, leading to their hypertrophy. Children with allergic rhinitis, for example, may experience persistent nasal inflammation that contributes to adenoidal enlargement. [44]
- **Hormonal Factors:** Hormonal influences on lymphoid tissue growth have been suggested, although the exact mechanisms remain unclear. It is hypothesized that hormonal changes during growth phases in children may contribute to adenoid enlargement. [12]

Pathogenesis of AH

The clinical and anatomic basis of hypertrophy is the enlargement of the germinal centers of lymphoid tissue and lymphoid follicles. The fundamental reason is thought to be a vicious cycle of inflammation, hypertrophy, and/or hyperplasia, secretory retention, and

recurrent inflammation

Experimental studies summarized in a review by Kuper et al show that the adenoid plays the earliest immunological role in the NALT system.[25] The immunological functions of the adenoids lead to their rapid growth during the early years of life. During childhood, its shape and size alter significantly, where dynamic growth is seen between ages 3 and 6 years, which may be related to relatively slower growth of the nasopharyngeal cavity. After the age of 6, the growth of the adenoid becomes inhibited under normal conditions, while the nasopharyngeal cavity grows and widens the respiratory tract. Later in life, the lymphoid tissue undergoes involution (due to the fibrous tissue expansion and fatty atrophy), so that in most adults, the adenoid appears in a residual form, but it never completely disappears.

Macroscopic changes accompany microscopic and functional changes therein. These adenoids are most immunologically active between 4 and 10 years of age in humans, and their involution begins after puberty. As a result, the B cell population decreases and the ratio of T cells to B cells increases. When the adenoids become infected, inflammation of the crypts leads to the inactivation of immunologically active cells, where the ability to transport antigens decreases, which in turn leads to metaplasia into a multilayered squamous epithelium. Such changes lead to inefficient antigen uptake and impaired cell functions.

AH is most likely associated with immune reactions, hormonal factors, or genetic factors. Among immune disturbances, for example, up regulation of interleukin (IL)-32 in adenoid

tissue might have a potential effect on AH progression through stimulation of pro inflammatory cytokines production as well as pyroptosis in human nasal epithelial cells mediated by NOD1/2/TLR4/NLRP3 pathway (nucleotide-binding oligomerization domain-containing protein/toll-like receptor/nucleotide-binding oligomerization domain, leucine-rich repeat and pyrin domain-containing proteins). [45]

Moreover, elevated levels of proinflammatory cytokines such as high-sensitivity C-reactive protein, IL-1 and IL-10, interferon-γ (IFN-γ), TNF-α (tumor necrosis factor α), as well as intercellular adhesion molecule-1 in children with AH have been observed. Genetic factors include, among others, polymorphisms in genes coding: SCGB1D4 (IFN-γ stimulated cytokine regulating chemotaxis of immune cells), TLR2 and TLR4 (play a key role in the regulation of the immune system through recognition of molecular patterns commonly found on pathogens (pathogen-associated molecular patterns).[12]

Normally, there is a well-established balance between the natural flora of the adenoid and the immune response, however, this can be disturbed by recurrent viral and bacterial infections and colonisation by pathogens. The biofilm theory in adenoid hypertrophy proposes that the presence of bacterial biofilms on the surface of hypertrophied adenoid tissue contributes significantly to chronic inflammation and recurrent infections, particularly in children. Biofilms are structured communities of bacteria encased in a self-produced polymeric matrix that adhere to surfaces, including the mucosa of the adenoids. These biofilms enable bacteria to evade the host immune response and resist antibiotics,

making infections more persistent and difficult to treat.[46] Frequent infections or allergies in the upper respiratory tract often give rise to hypertrophic processes. A vicious cycle consisting of inflammation, hypertrophy and/or hyperplasia, retention of secretions, and recurrent inflammation is assumed to be the underlying cause.

Clinical presentation:

- Some patients can develop an abnormal immune response, which in turn leads to chronic inflammation of the adenoid, which may then spread to adjacent areas of the mucosa, for example, of the nose, sinuses, and middle ear.
- Chronic adenoiditis develops in cases when foreign antigens elude immune response, become trapped in the crypts, and proliferate before an effective immune response can be activated.
- Symptoms accompanying are a chronic runny nose, nasal obstruction, snoring, an altered breathing pattern from nasal to oral, and bad breath.
- Adenoids causing airway obstruction can lead to: Obstructive sleep apnea syndrome (OSAS), Chronic sinusitis, OME, Formation of malocclusions and developmental anomalies in the craniofacial area, Speech disturbances and articulation errors, Disorders in physical and intellectual development.

Diagnosis:

Clinical Evaluation:

Adenoid hypertrophy is an obstructive condition, with its symptomatology depending on the obstructed structure. Nasal obstruction by hypertrophic adenoid tissue can cause the patient to complain of rhinorrhea, difficulty breathing through the nose, chronic cough, post-nasal drip, snoring, and/or sleep-disordered breathing in children. If the nasal obstruction is significant, the patient can suffer from sinusitis as a result and may complain of facial pain or pressure. Obstruction of the Eustachian tube can lead to symptoms consistent with Eustachian tube dysfunction, such as muffled hearing, otalgia, crackling or popping sounds in the ear, and/or recurrent middle ear infections.

The primary focus of visual inspection should be on the presence of adenoid facies. In a typical patient with adenoid facies, the mouth is permanently open and the tip of the tongue is visible. In addition, eczema is often present at the entrance to the nose. A specialist medical examination should include rhinoscopy (if possible and tolerated), an inspection of the nasopharynx (using a flexible endoscope, if possible), an evaluation of the palatine tonsils, an assessment of lymph nodes for enlargement, and bilateral oto-microscopy. Malocclusion, dental malposition, and a high palate can be indicative of adenoid hyperplasia. Palpation of the hard and soft palate should be performed before surgery to identify a submucous cleft, if present.

The Brodsky-Moore-Stanbury grading system classifies tonsillar hypertrophy based on the percentage of the oropharyngeal airway occupied by the tonsils, as observed during clinical examination.

- Grade 0 indicates that the tonsils are entirely within the tonsillar fossa, usually seen in patients who have had a tonsillectomy.
- Grade 1+ means the tonsils occupy less than 25% of the transverse oropharyngeal space.
- Grade 2+ describes tonsils that fill less than 50% of the oropharyngeal width.
- Grade 3+ corresponds to tonsils that occupy less than 75% of the oropharyngeal space.
- Grade 4+ is the most severe, where the tonsils occupy more than 75% of the oropharyngeal space and may even be touching at the midline (often referred to as "kissing tonsils").

This grading system is simple, clinically useful, and helps in decision-making regarding the need for surgical intervention, particularly in patients presenting with obstructive symptoms or recurrent infections.

Radiological Assessment

Radiographic evaluation plays a crucial role in diagnosing adenoid hypertrophy. Various imaging techniques, including lateral neck X-rays, computed tomography (CT) scans, and magnetic resonance imaging (MRI), provide valuable insights.

- **Lateral Neck X-ray:** This is one of the most commonly used imaging techniques for adenoid hypertrophy. The patient is positioned laterally with the head slightly extended while an X-ray beam is directed at the nasopharynx. The image helps measure the adenoid-to-airway ratio and provides an estimate of the adenoid size and its impact on airway obstruction. (Figure 5) The advantage of the lateral neck X-ray is its accessibility, affordability, and quick execution. However, it only provides a two-dimensional view and may not be as accurate as endoscopy in assessing airway obstruction.[47-48]

  Adenoid-to-nasopharyngeal ratio (ANR) (Figure 5): Adenoid depth (AD) thickness will be measured by drawing a perpendicular line from a line drawn along the straight part of the anterior margin of basi-occiput to the most convex part of the adenoid pad. Nasopharyngeal depth (ND) will be calculated by drawing another line between the spheno-occipital synchondrosis and to postero-superior edge of the hard palate. The adenoid-to-nasopharyngeal ratio (ANR) will then be calculated by dividing AD by ND.[49] The value will then be documented in percentage by multiplying by 100. Based on the ANR, the subjects were then categorized into 4 groups;

  - Grade 1-0–25%
  - Grade 2- 25–50%,
  - Grade 3-50–75%
  - Grade 4-75–100%

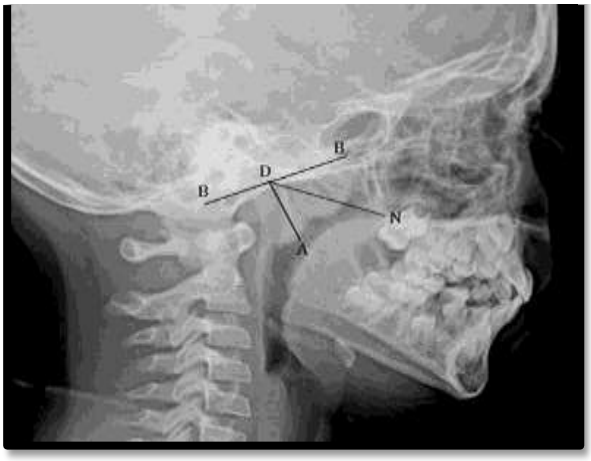

Figure 2: Plain radiograph of nasopharynx lateral view (LNX) showing how adenoidal—nasopharyngeal ratio will be calculated

Fujikoshi (Fujioka) Grading System (based on A/N ratio):

- Grade 1 (Mild hypertrophy): A/N ratio < 0.25
- Grade 2 (Moderate hypertrophy): A/N ratio 0.25 – 0.50
- Grade 3 (Severe hypertrophy): A/N ratio 0.50 – 0.75
- Grade 4 (Very severe hypertrophy): A/N ratio > 0.75

- **Computed Tomography (CT) Scan:** CT scans provide detailed imaging of the nasopharyngeal region, making them useful in cases where anatomical abnormalities or masses need evaluation. The patient lies supine on the CT table, and a non-contrast scan is performed to assess adenoid size and airway obstruction. The images are then reconstructed for a better anatomical understanding. The advantage of CT scans is their superior imaging capability compared to X-rays. However, they involve higher radiation exposure and are not commonly used as a first-line diagnostic tool. CBCT can accurately identify clinically relevant adenoid hypertrophy with 88% sensitivity and 93% specificity.[50] (Figure-6)

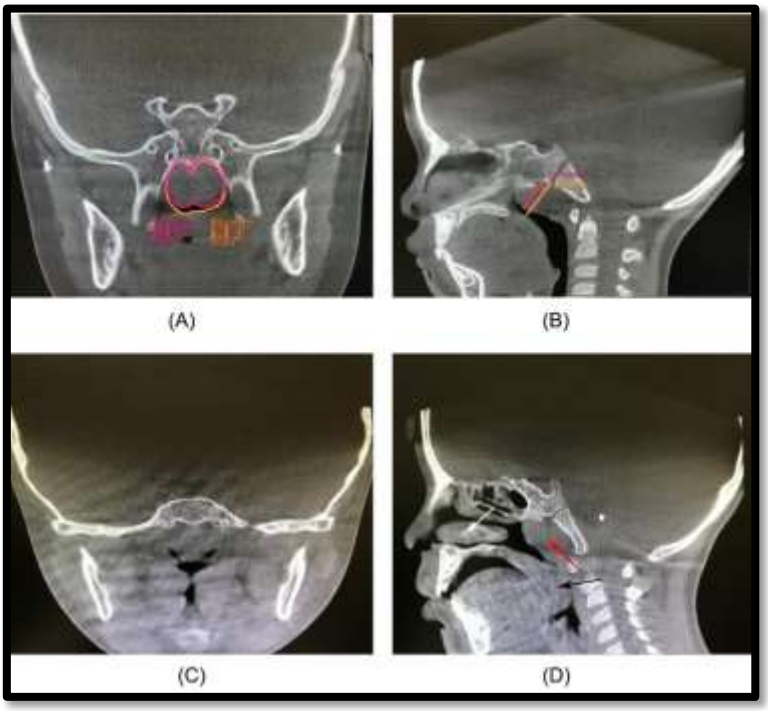

Figure 3: Coronal scan and sagittal reconstruction of CBCT.

(A) Coronal scan with hypertrophic adenoids seen at the posterior nostril, the software can automatically calculate the cross-sectional area (adenoids in the red circle, posterior nostril in the yellow circle). (B) Sagittal reconstruction calculating A/N (red is the thickness of adenoids, yellow is the width of the nasopharyngeal cavity at the most convex part of the adenoids) values. (C) Coronal scan showing hypertrophic tonsils in the oropharynx (black arrows). (D) Sagittal reconstruction showing hypertrophic inferior turbinates (white arrows), hypertrophied adenoids (red arrows), hypertrophied tonsils (black arrows), and airway compression and obstruction.

- **Magnetic Resonance Imaging (MRI):** MRI provides high-resolution images of the nasopharyngeal region and is particularly useful in complex or recurrent cases requiring detailed anatomical studies.(Figure 7) The patient is positioned supine inside the MRI scanner, and a non-contrast scan of the nasopharyngeal region is performed. MRI offers excellent soft tissue contrast and is radiation-free, making it a safer alternative for pediatric patients. However, it is more expensive, time-consuming, and may require sedation in young children due to motion artifacts.[51]

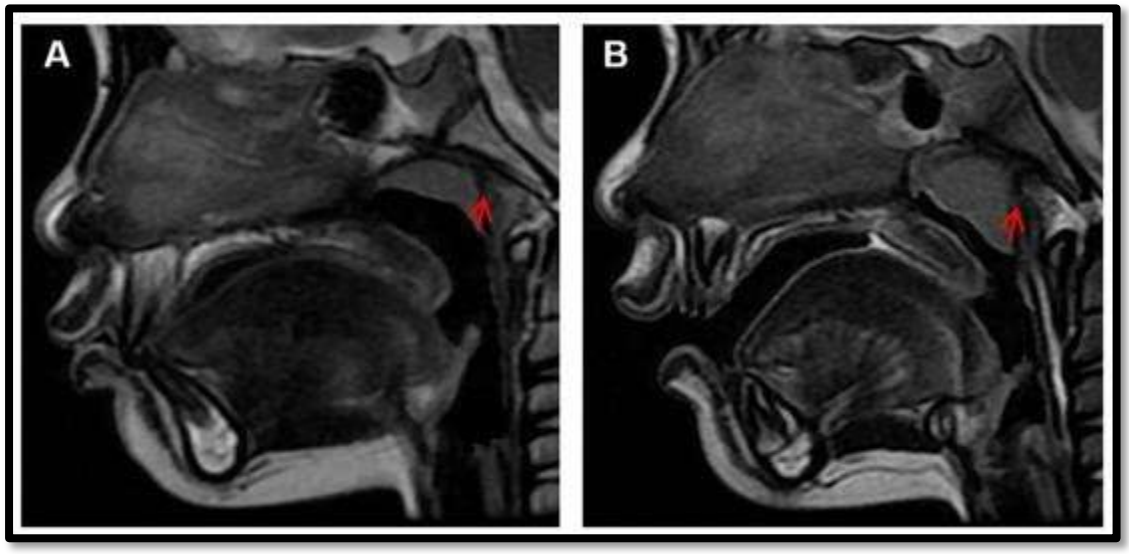

Figure 4: MRI Images of normal and hypertrophic adenoids.

(A) The red arrow indicates a normal adenoid; (B) The red arrow indicates a hypertrophic adenoid.

Endoscopic examination:

Nasal endoscopy and fiber optic nasopharyngoscopy are effective tools for direct visualization of the adenoids. Nasal endoscopy performed using a flexible or rigid endoscope allows for an accurate assessment of adenoid size and airway obstruction. Fibre optic nasopharyngoscopy provides a more detailed evaluation of the nasopharyngeal region and can be performed in an outpatient setting with minimal discomfort.

Naso-pharyngoscopy: This procedure involves the insertion of a flexible fibre optic or rigid endoscope through the nasal cavity to visualize the nasopharyngeal area directly. The patient is typically seated upright, and topical anesthesia may be applied to minimize discomfort. The endoscope is carefully maneuvered to assess the adenoid tissue, its size, and any associated airway obstruction. The primary advantage of nasopharyngoscopy is its ability to provide real-time, high-resolution imaging of the adenoid tissue. It allows

direct assessment of adenoid hypertrophy, the degree of airway obstruction, and the presence of any associated conditions like nasal polyps or chronic inflammation. The procedure is quick, does not involve radiation exposure, and provides immediate results.[52]

CLEMANS Endoscopic grading:

Based on the examination, grade I is scored when fibre optic endoscopy imaging revealed adenoid tissue occupying only the upper segment of the nasopharyngeal cavity (≤ 25%) with almost completely free choanal openings. Grade II is scored if the adenoid tissue is confined to the upper half (25-50%) of the nasopharyngeal cavity with sufficiently pervious choana and perfect visualization of the tube ostium. Grade III obstruction is given if adenoid tissue occupies about 75% of the nasopharynx with partial involvement of the tube ostium and considerable obstruction of choanal openings. In grade IV cases, the adenoid tissue reaches the lower choanal border without allowing visualization of the tube ostium (> 75%). (Figure 8) [53]

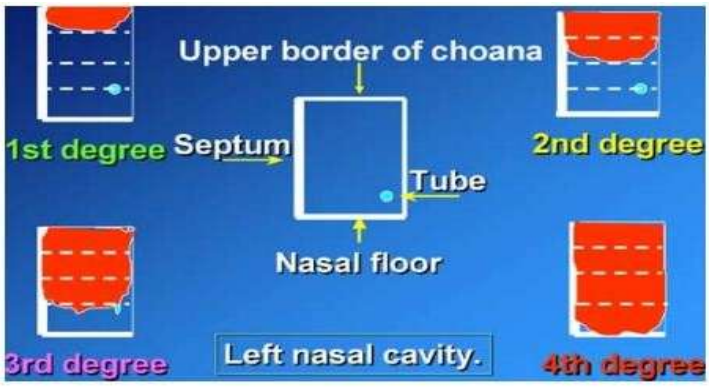

Figure 5: Endoscopic grading of adenoid hypertrophy

AIM AND OBJECTIVES OF THE STUDY

Aim of the study:

- To correlate adenoid hypertrophy with clinico-radiological findings and rigid diagnostic nasal endoscopic findings.

Objectives of the study:

- To clinically examine patients presenting with symptoms of adenoid hypertrophy
- To formulate the correct method of capturing X-ray Nasopharynx lateral view for soft tissues to diagnose adenoid hypertrophy
- To perform Rigid Diagnostic Nasal Endoscopy in patients with adenoid hypertrophy
- To correlate clinico-radiological findings with rigid diagnostic nasal endoscopy findings

PATIENTS AND METHODS

STUDY SETTING:

- The study was conducted in the Department of Otorhinolaryngology, Bhaskar Medical and General Hospital, Yenkapally.

STUDY POPULATION:

- All patients reporting to the Otorhinolaryngology OPD presenting with complaints of adenoid hypertrophy.

STUDY DESIGN: It is a cross-sectional study

STUDY DURATION: 18 months

SAMPLE SIZE: 80 cases
INCLUSION CRITERIA:

- Patients in the age group of 5 to 50 years with symptoms of Adenoid Hypertrophy, like:

  Nasal Obstruction, Nasal Discharge, Snoring & Mouth Breathing,Recurrent URTI, Unilateral & Bilateral hearing loss associated with OME, Adenoid facies

Exclusion Criteria

- Patients with congenital anomalies
- Patients with a history of recurrence after surgery
- Patients with concomitant or pre-existing craniofacial abnormalities or syndromes that could affect adenoid evaluation.
- Patients with other nasopharyngeal or upper airway pathologies that could confound the assessment of adenoid hypertrophy.
- Patients who had received medical treatment (e.g., intranasal corticosteroids) or interventions (e.g., adenoidectomy) before the assessment.

METHODOLOGY:

After obtaining permission from the institutional ethics committee, the study was conducted in the Department of Otorhinolaryngology, Bhaskar Medical College and General Hospital. Informed consent will be taken from the patient and their attendants.

A survey sheet was used to obtain the following information:

1. Demographic details: age, gender, occupation, socioeconomic status, and address.
2. Chief complaint with a history of present illness (onset, progression, radiating symptoms, aggravating and relieving factors, and associated symptoms)
3. Past medical history of Hypertension/ Diabetes mellitus/ Tuberculosis /Thyroid disorder/ Coronary artery disease/ Cerebrovascular accidents/ Epilepsy/ Asthma/ Drugs. Any previous medical or surgical treatment will be noted.
4. Family history
5. Personal history: appetite/ diet/ digestion/ bowel and bladder habits/ sleep/ addiction to smoking or alcohol or tobacco (frequency, intensity, and duration were obtained).
6. General examination
    - Patient conscious, coherent, cooperative
    - Built/ Nourishment
    - Pallor, Icterus, Cyanosis, Clubbing
    - Generalized lymphadenopathy
    - Edema
7. Vitals:
    - Temperature

- Pulse pressure
- Blood pressure
- Respiratory rate

8. Systemic examination: Cardiovascular, Respiratory, Gastrointestinal, Musculoskeletal system, Neurological, and Genitourinary systems were evaluated and any abnormality was noted.
9. Nasopharynx evaluation:

- Clinical grading: assessed the patients' symptoms (e.g., nasal obstruction, mouth breathing, snoring) and conducted a physical examination (e.g., inspection of the nasal cavity, oropharynx, and adenoid pad). The Brodsky-Moore-Staneiwich grading system classifies tonsillar hypertrophy based on the percentage of the oropharyngeal airway occupied by the tonsils, as observed during clinical examination.
  - Grade 0 indicates that the tonsils are entirely within the tonsillar fossa, usually seen in patients who have had a tonsillectomy.
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  - Grade 4+ is the most severe, where the tonsils occupy more than 75% of the oropharyngeal space and may even be touching at the midline (often

referred to as "kissing tonsils").

- Radiological assessments were performed using appropriate imaging modalities, such as X-ray or computed tomography (CT) scans. The radiological measurements included adenoid size, volume, and/or specific grading systems. Fujikoshi's grading system (often referred to as the Fujikoshi or Fujioka method) is a radiographic technique commonly used to assess adenoid hypertrophy using lateral nasopharyngeal X-rays. Fujikoshi (Fujioka) Grading System (based on A/N ratio):
  - Grade 1 (Mild hypertrophy): A/N ratio < 0.25
  - Grade 2 (Moderate hypertrophy): A/N ratio 0.25 – 0.50
  - Grade 3 (Severe hypertrophy): A/N ratio 0.50 – 0.75
  - Grade 4 (Very severe hypertrophy): A/N ratio > 0.75
- Endoscopic examinations were conducted using a flexible or rigid endoscope. The endoscopic findings included visual assessment of adenoid size, appearance (e.g., hypertrophic, edematous), and associated findings (e.g., secretions, obstructive patterns). The Clemens endoscopic grading system is utilized for assessing adenoid hypertrophy via nasopharyngoscopy. This grading system categorizes adenoid size based on the percentage of choanal obstruction observed during endoscopic examination:
  - Grade 1: 0–25% obstruction
  - Grade 2: 26–50% obstruction
  - Grade 3: 51–75% obstruction
  - Grade 4: 76–100% obstruction

10. Laboratory investigations: the following investigations were conducted for patient before the treatment; ECG, CBP with ESR, CRP, Complete Urine Examination, FBS/PLBS, HbA1C, serum electrolytes, serum creatinine, blood urea, LFT, bleeding time, clotting time, blood grouping, Rh typing, Chest X-ray, serological tests for HIV, HBsAg, and HCV.

Statistical Analysis:

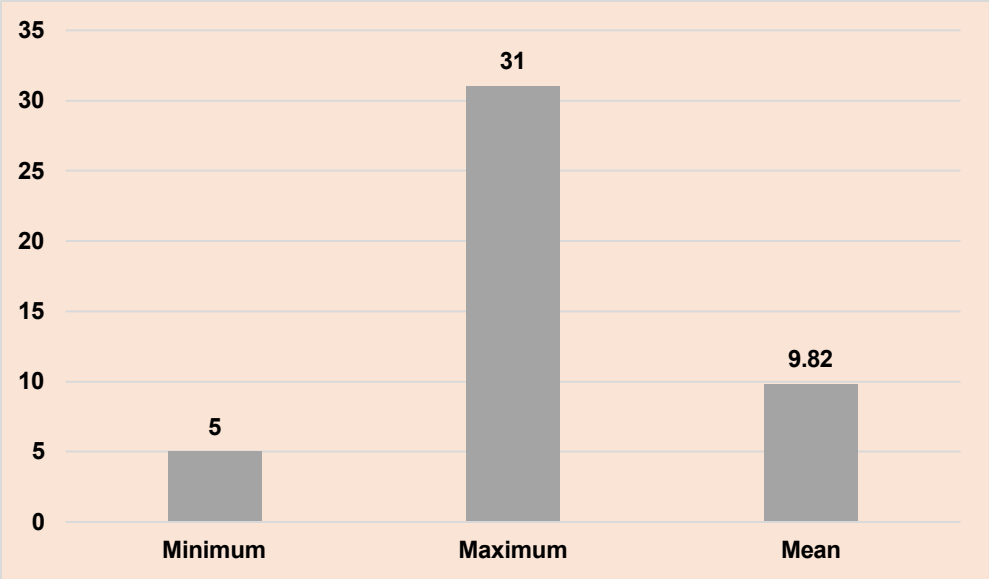
Statistical analysis was performed with IBM SPSS Statistics 22.0 (IBM Corp., Armonk, NY, USA). The fit of the data to a normal distribution was analyzed with the Shapiro–Wilk test. The descriptive data were provided in the form of mean and SD, while the categorical data were presented in numbers and percentages. The correlation between clinical, radiological, and endoscopic findings was done using the Spearman Correlation test. A p-value of ≤0.05 is considered statistically significant.

OBSERVATION AND RESULTS

Table 1: Descriptive data of age

	N	Minimum	Maximum	Mean	SD
age	80	5.00	31.00	9.8250	3.81461

Table 1: Descriptive data of age



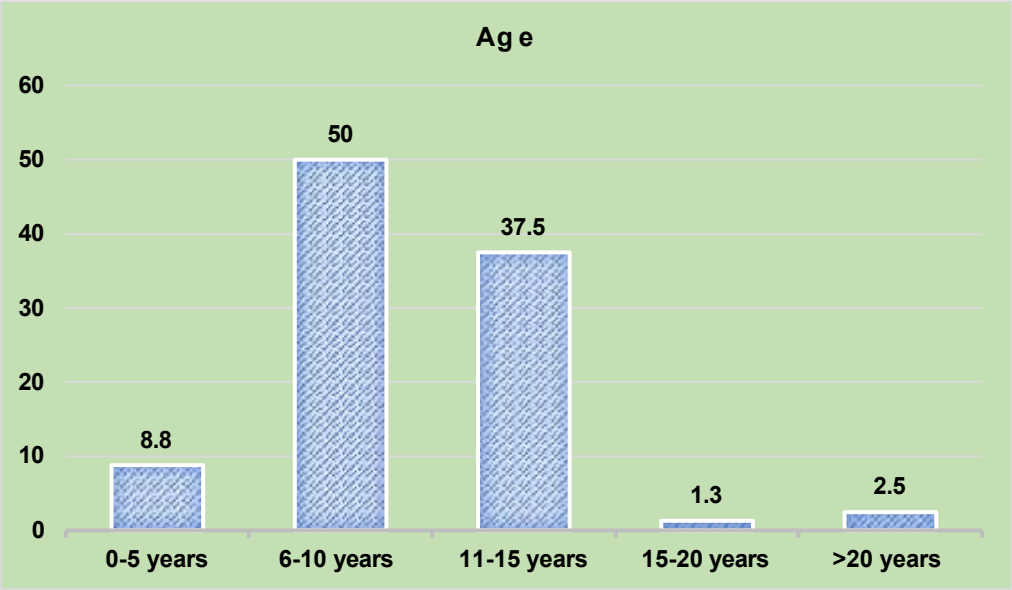
Inference:

Table and Graph 1 provide descriptive statistics for the age of the study population. The mean age was 9.83±3.81 years, with the youngest participant being 5 and the oldest 31. This indicates that the study predominantly involved children but included a few adult cases as well.

Table 2: Distribution based on age

Age	n	%
0-5 years	7	8.8
6-10 years	40	50.0
11-15 years	30	37.5
15-20 years	1	1.3
>20 years	2	2.5

Graph 2: Distribution based on age



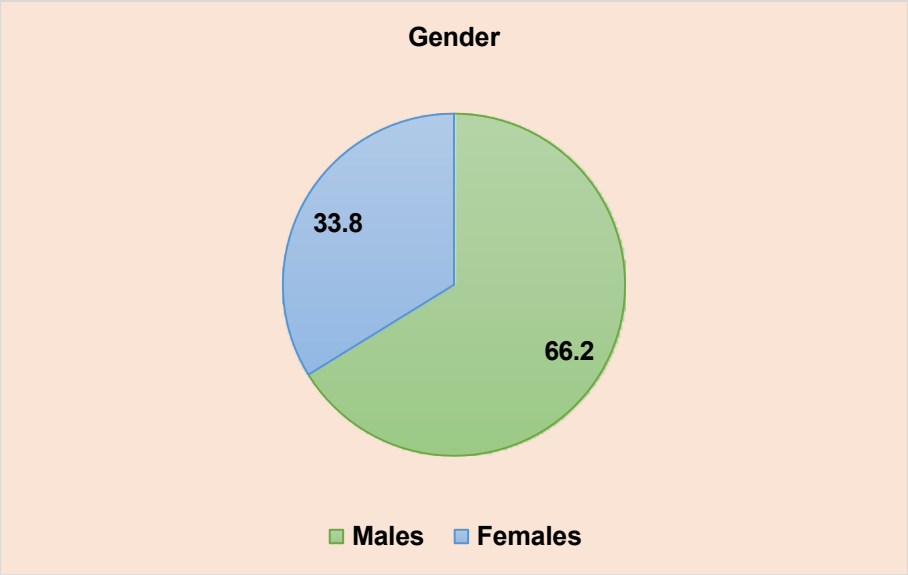
Inference:

Table and Graph 2 represent the distribution of subjects based on age. Most of the patients (50.0%) were aged between 6–10 years, followed by 37.5% in the 11–15 years group. A smaller proportion fell into the 0–5 years category (8.8%), while very few were aged 15–20 years (1.3%) or above 20 years (2.5%). This confirms that adenoid hypertrophy symptoms were most commonly observed in school-aged children.

Table 3: Distribution based on Gender

Gender	n	%
Males	53	66.3
Females	27	33.8

Graph 3: Distribution based on Gender



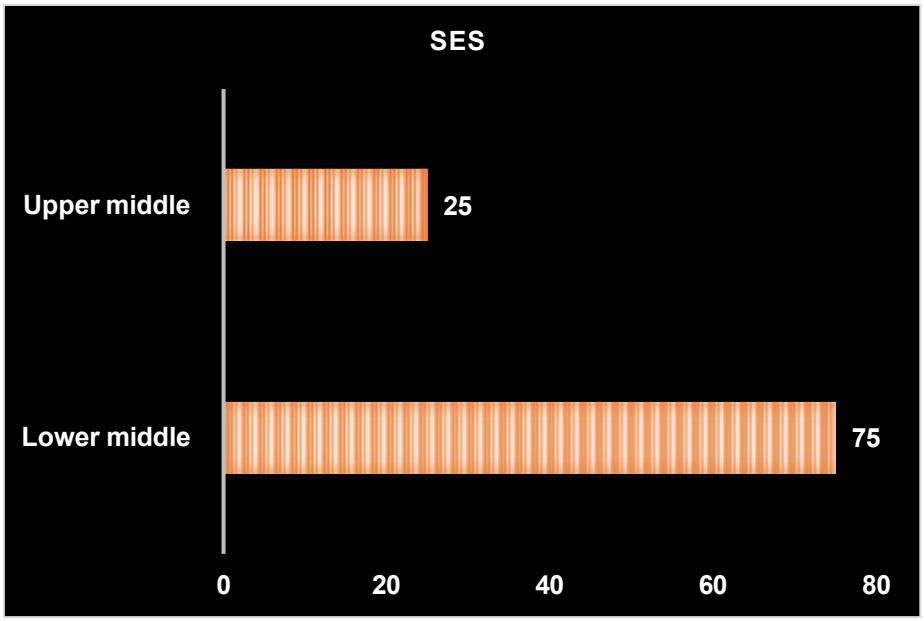
Inference:

Table and Graph 3 represent the distribution of subjects based on gender. The study showed a male predominance, with 66.3% of the patients being male, while 33.8% were female. This suggests that males may be more frequently affected or brought for evaluation of adenoid-related symptoms.

Table 4: Distribution based on SES [socio economic status]

SES	n	%
Lower middle	60	75.0
Upper middle	20	25.0

Graph 4: Distribution based on Socio economic status [SES]



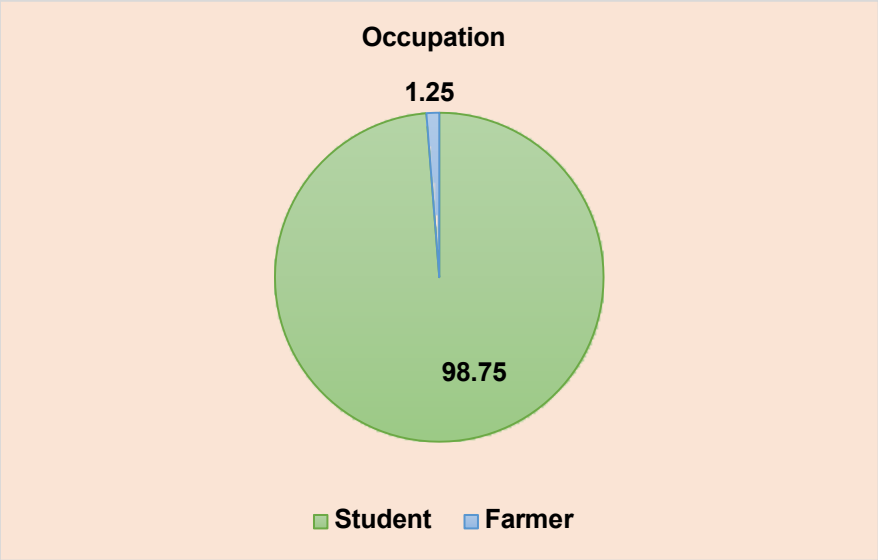
Inference:

Table and Graph 4 represent the distribution of subjects based on the SES. A significant proportion of the subjects belonged to the lower-middle socio-economic class (75.0%), while 25.0% were from the upper-middle class. This could reflect accessibility patterns to the hospital or health-seeking behaviour in specific socio-economic groups.

Table 5: Distribution based on occupation

Occupation	n	%
Student	79	98.75
Farmer	1	1.25

Graph 5: Distribution based on occupation



Inference:

Table and Graph 5 represent the distribution of subjects based on occupation. Nearly all the subjects (98.75%) were students, which aligns with the predominantly paediatric age group. Only one participant (1.25%) was a farmer, likely one of the adult subjects.

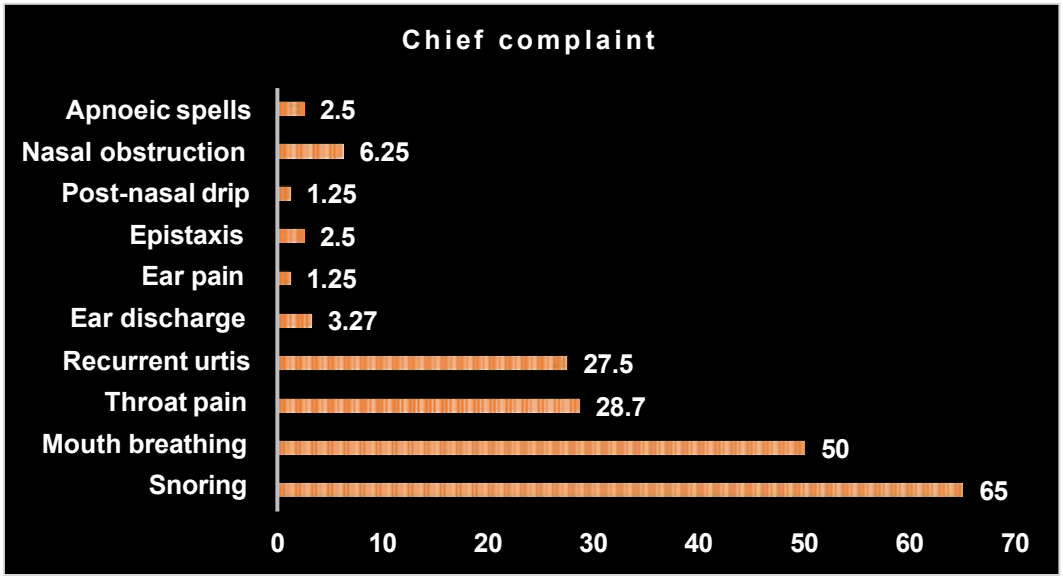
Table 6: Distribution based on the chief complaint

Chief compliant	n	%
Snoring	52	65
Mouth breathing	40	50
Throat pain	23	28.7
Recurrent urtis	22	27.5

Ear discharge	3	3.27
Ear pain	1	1.25

Epistaxis	2	2.5
Post-nasal drip	1	1.25
Nasal obstruction	5	6.25
Apnoeic spells	2	2.5

Graph 6: Distribution based on the chief complaint



Inference:

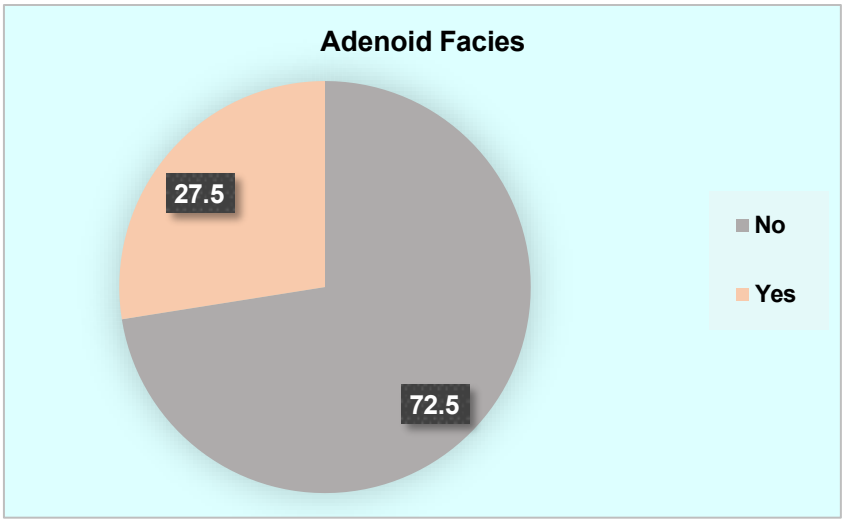
Table and Graph 6 present the chief complaint reported by subjects. Snoring (65.0%) and mouth breathing (50.0%) were the most commonly reported symptoms, consistent with typical presentations of adenoid hypertrophy. Other complaints included throat pain (28.7%), recurrent upper respiratory tract infections (27.5%), and less frequently ear-related symptoms and nasal obstruction.

Table 7: Distribution of subjects based on the adenoid facies

Adenoid facies	n	%

No	58	72.5
Yes	22	27.5

Graph 7: Distribution of subjects based on the adenoid facies



Inference:

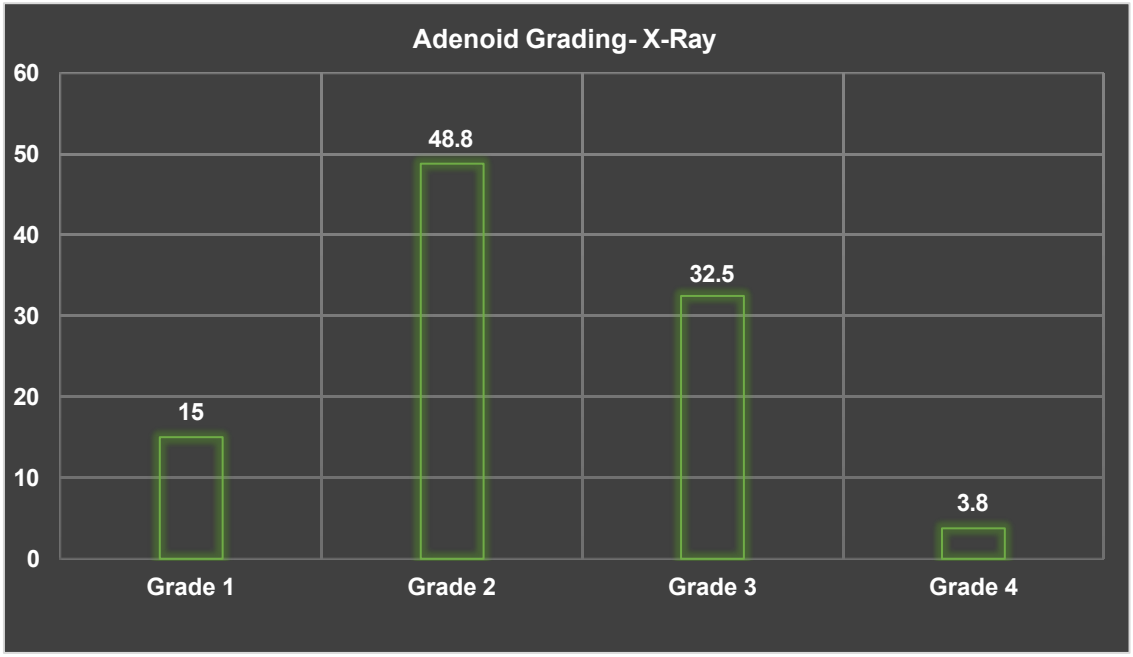
Table and Graph 7 represent the distribution of subjects based on adenoid facies. Adenoid facies were observed in 27.5% of the subjects, while 72.5% did not exhibit such features. This shows that classical facial features of chronic adenoid hypertrophy are absent in all affected individuals.

Table 8: Grading of adenoids in X-Ray

X-Ray Grading	n	%
Grade 1	12	15.0

Grade 2	39	48.8
Grade 3	26	32.5
Grade 4	3	3.8

Graph 8: Grading of adenoids in X-Ray



Inference:

X-ray findings showed that 48.8% of patients had Grade 2 adenoid hypertrophy. Grade 3 was seen in 32.5%, while Grade 1 and Grade 4 were observed in 15.0% and 3.8%, respectively. This reflects that most children had moderate enlargement on radiology.

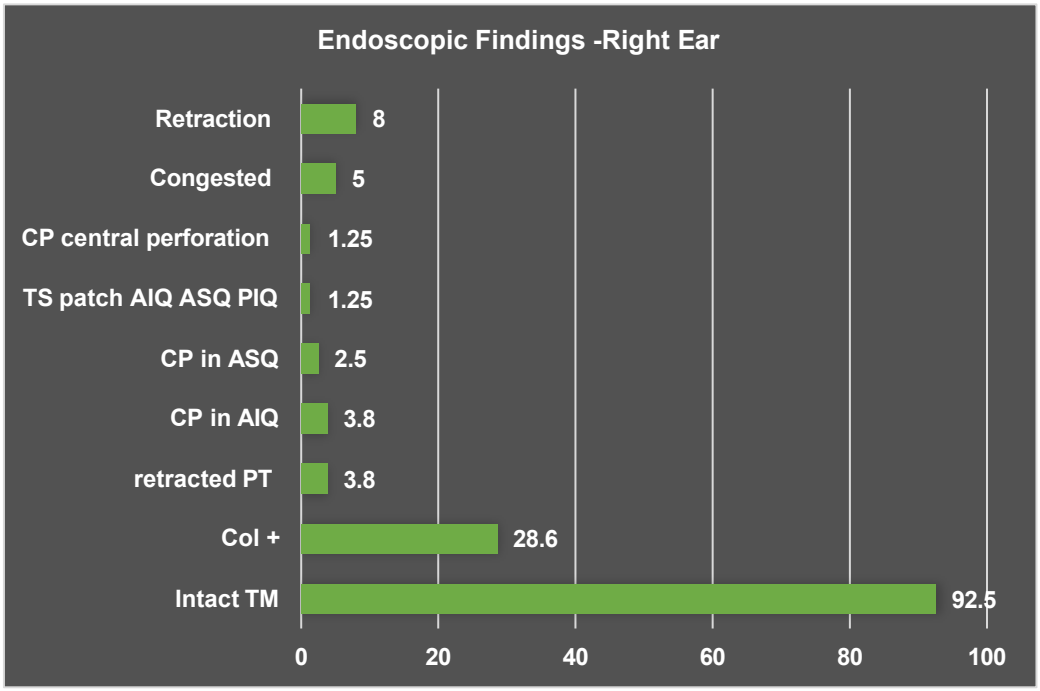
Table 9: Distribution based on the endoscopic findings of the Right ear

Right ear	n	%

Intact TM	74	92.5
Col +	23	28.6
retracted PT	3	3.8
CP in AIQ	3	3.8

CP in ASQ	2	2.5
TS patch AIQ ASQ PIQ	1	1.25
CP central perforation	1	1.25
Congested	4	5
Retraction	10	8

Graph 9: Distribution based on the endoscopic findings of the Right ear



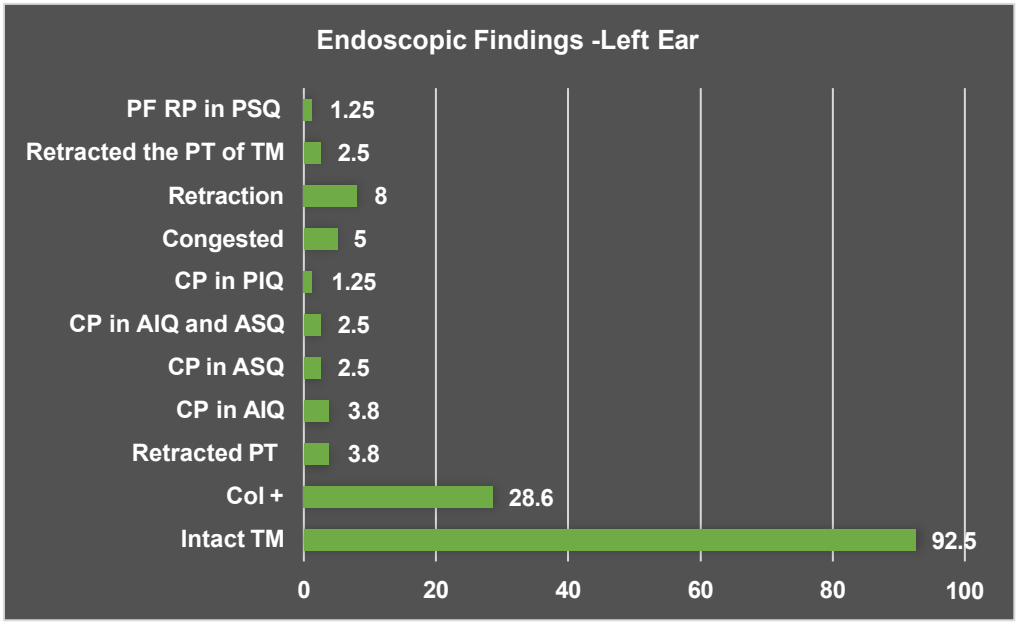
Inference:

Endoscopy of the right ear showed that the tympanic membrane was intact in 92.5% of cases. Other common findings included congestion (5.0%) and retraction (8.0%). Some showed positive cold oto-scopy signs, central perforations, or other localized retraction findings, indicating varying middle ear involvement.

Table 10: Distribution based on the endoscopic findings of the Left ear

Left ear	n	%
Intact TM	74	92.5
Col +	23	28.6
Retracted PT	3	3.8
CP in AIQ	3	3.8
CP in ASQ	2	2.5
CP in AIQ and ASQ	2	2.5
CP in PIQ	1	1.25
Congested	4	5
Retraction	10	8
Retracted the PT of TM	2	2.5
PF RP in PSQ	1	1.25

Graph 10: Distribution based on the endoscopic findings of the Left ear



Inference:

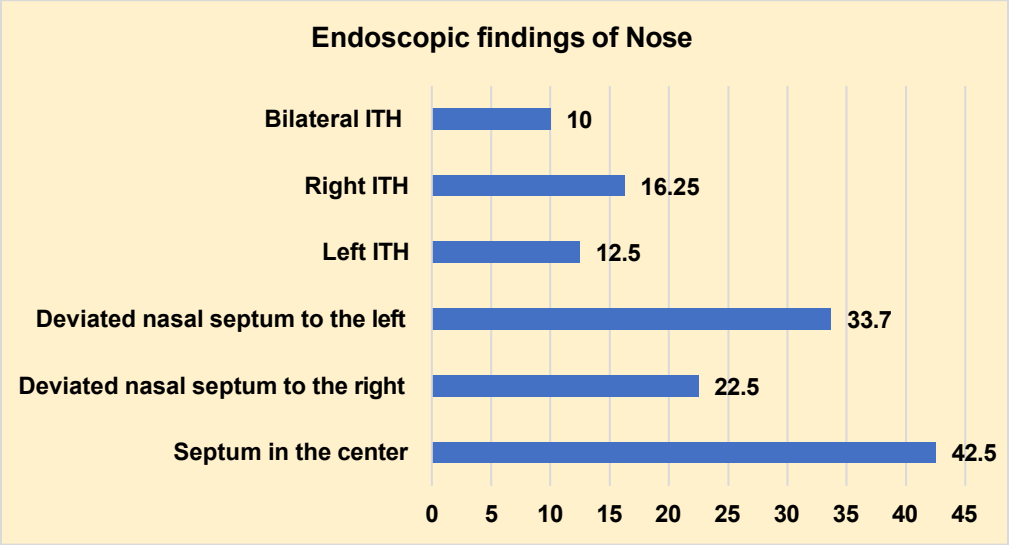
Similar to the right ear, 92.5% had intact tympanic membranes. Retraction was present in 8.0% of cases. Various degrees and locations of central perforations and congestion were also noted, again pointing to ear involvement in some patients with adenoid

hypertrophy.

Table 11: Distribution based on the endoscopic findings of the Nose

Nose	n	%
Septum in the centre	34	42.5
Deviated nasal septum to the right	18	22.5
Deviated nasal septum to the left	27	33.7
Left ITH	10	12.5
Right ITH	13	16.25
Bilateral ITH	8	10

Graph 11: Distribution based on the endoscopic findings of the Nose



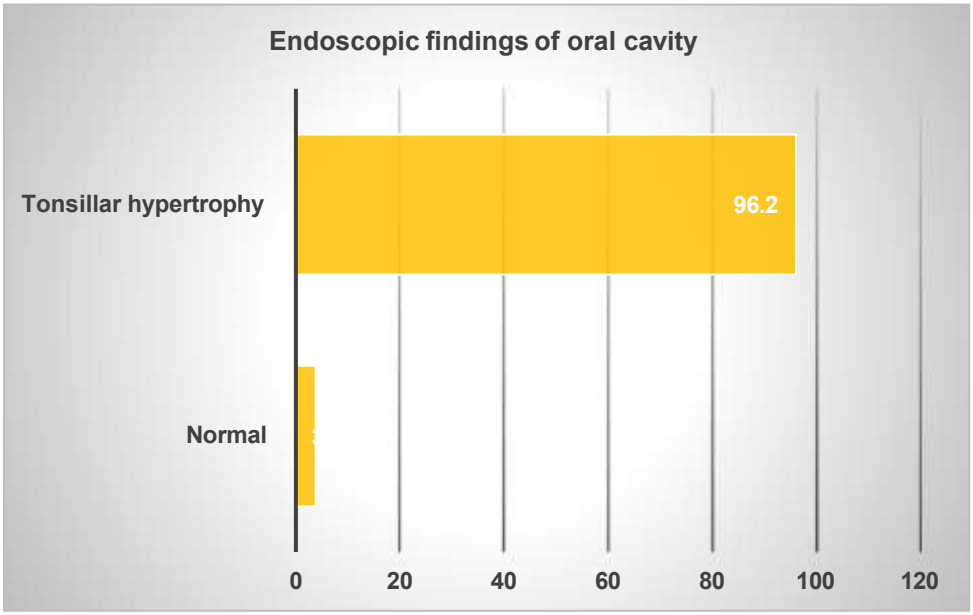
Inference:

The nasal septum was centrally located in 42.5% of cases. Deviations were seen in 22.5% to the right and 33.7% to the left. Inferior turbinate hypertrophy (ITH) was also present — 12.5% had left ITH, 16.25% had right ITH, and 10.0% had bilateral ITH. These structural variations could contribute to or result from chronic nasal obstruction.

Table 12: Distribution based on the endoscopic findings of the oral cavity

Oral cavity	n	%
Normal	3	3.8
Tonsillar hypertrophy	77	96.2

Graph 12: Distribution based on the endoscopic findings of the oral cavity



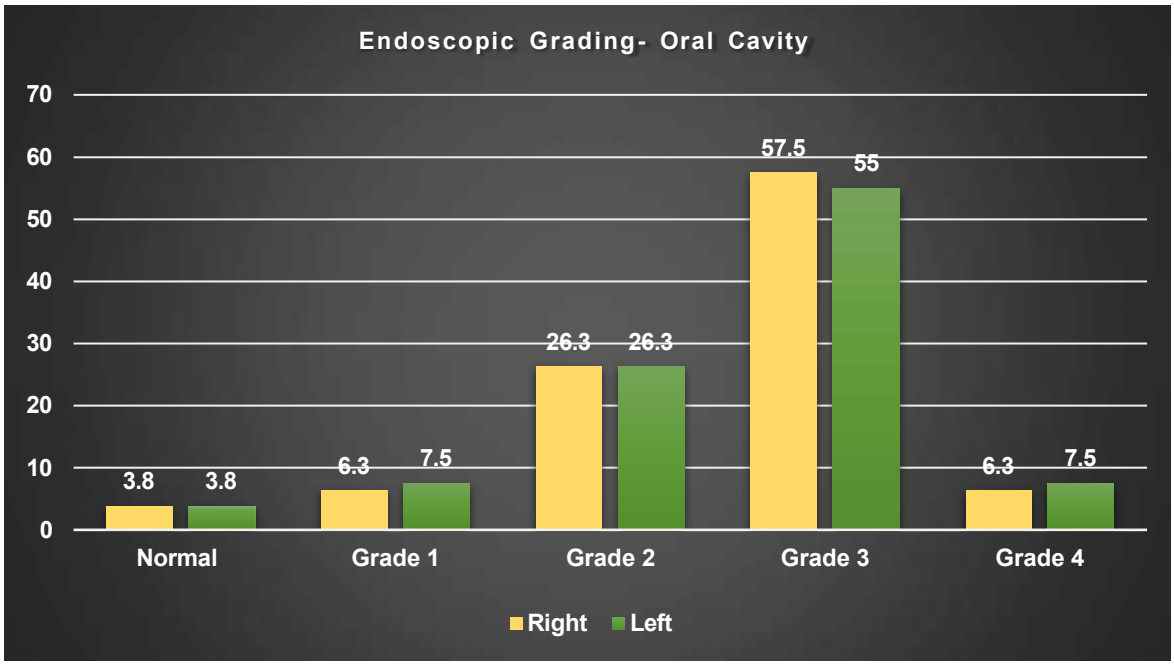
Inference:

Most subjects had tonsillar hypertrophy (96.2%), and only 3.8% had a normal throat. This shows a strong association between adenoid hypertrophy and tonsillar enlargement.

Table 13: Distribution based on the grading of endoscopic findings of the oral cavity

Oral Cavity- Grade	Right		Left	
	n	%	n	%
Normal	3	3.8	3	3.8
Grade 1	5	6.3	6	7.5
Grade 2	21	26.3	21	26.3
Grade 3	46	57.5	44	55
Grade 4	5	6.3	6	7.5

Graph 13: Distribution based on the grading of endoscopic findings of the oral cavity



Inference:

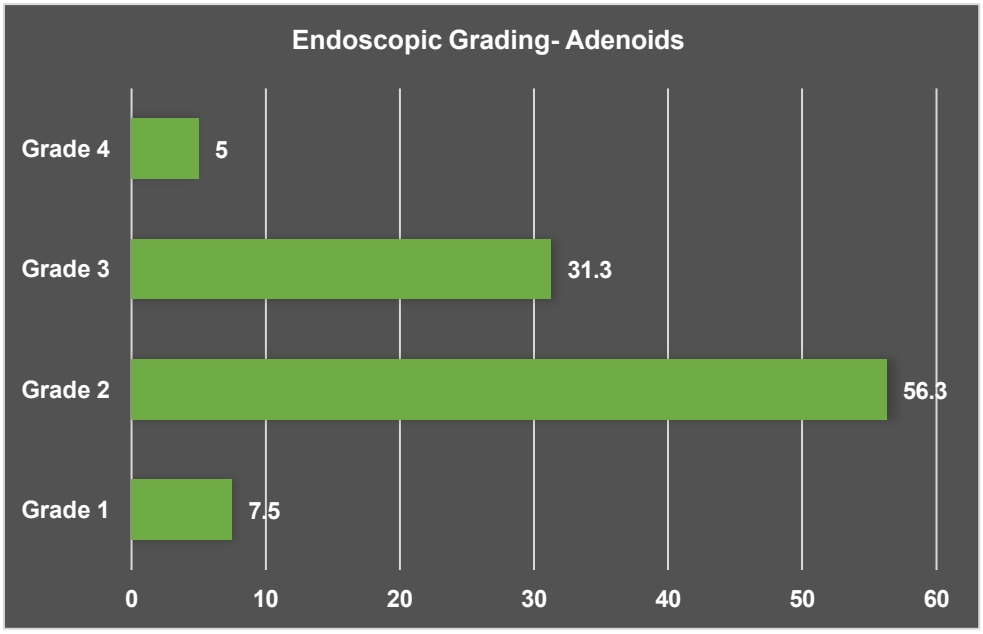
On endoscopic grading, Grade 3 tonsillar enlargement was the most frequent finding on both sides (57.5% right, 55.0% left), followed by Grade 2 (26.3%). Grade 4 and Grade 1

were less common, and normal tonsils were seen only in 3.8% of patients. This indicates that most subjects had moderate to severe tonsillar hypertrophy.

Table 14: Distribution based on the grading of endoscopic findings of the adenoids

Adenoids	n	%
Grade 1	6	7.5
Grade 2	45	56.3
Grade 3	25	31.3
Grade 4	4	5

Graph 14: Distribution based on the grading of endoscopic findings of the adenoids



Inference:

Endoscopic evaluation of adenoids showed Grade 2 hypertrophy in 56.3% and Grade 3 in 31.3%. Grade 1 and Grade 4 were found in 7.5% and 5.0%, respectively. This supports the predominance of moderate adenoid enlargement based on direct visualization.

Table 15: Correlation between the X-ray and endoscopic findings of adenoids

Correlation		Endoscopic n (%)				
		Grade 1	Grade 2	Grade 3	Grade 4	Total
X-Ray n(%)	Grade 1	6(100)	6(13.3)	0(0)	0(0)	12
	Grade 2	0(0)	35(77.8)	4(16)	0(0)	39
	Grade 3	0(0)	4(8.9)	21(84)	1(25)	26
	Grade 4	0(0)	0(0)	0(0)	3(75)	3
	Total	6	45	25	4	80
Correlation r value		0.818				
P value		**<0.001***				

Spearman Correlation test; p≤0.05 considered statistically significant

Inference:

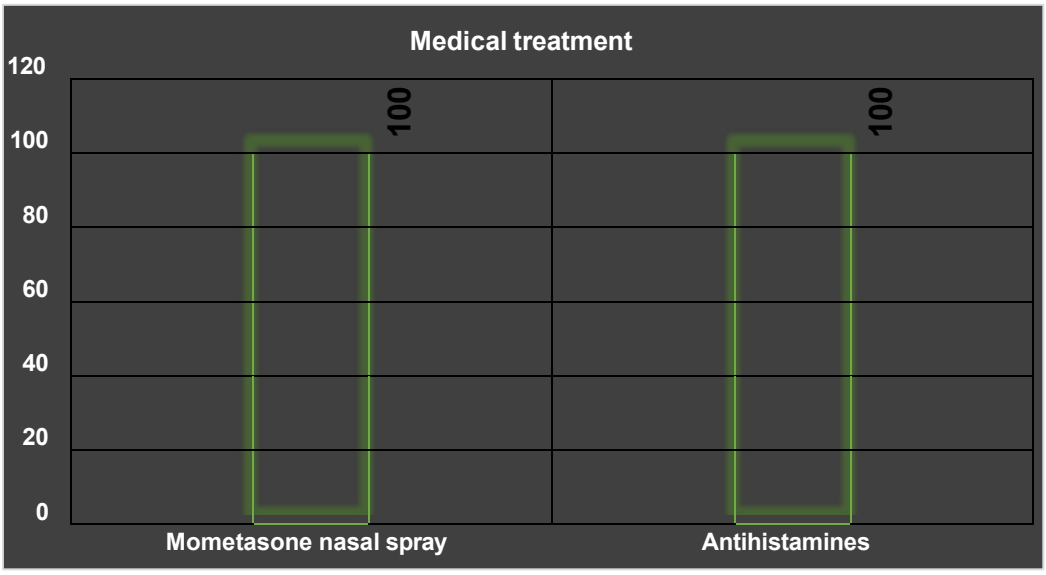
Notably, 100% of cases graded as Grade 1 on X-ray were also confirmed as Grade 1 endoscopically, 77.8% graded as Grade 2 matched with endoscopic Grade 2, and 84% of X-ray Grade 3 cases matched with endoscopic Grade 3. There was a consistent and progressive increase in endoscopic grading corresponding to increasing radiographic grades.

A strong correlation was found between radiological and endoscopic grading of adenoid hypertrophy, with a correlation coefficient (r) of 0.818 ($p<0.001$). This indicates a high level of agreement between radiological and endoscopic methods in grading adenoid hypertrophy, reinforcing the reliability of X-ray assessment, especially in settings where endoscopy may not be feasible.

Table 16: Distribution based on the medical treatment

Medical treatment	n	%
Mometasone nasal spray	80	100
Antihistamines	80	100

Graph 15: Distribution based on the medical treatment



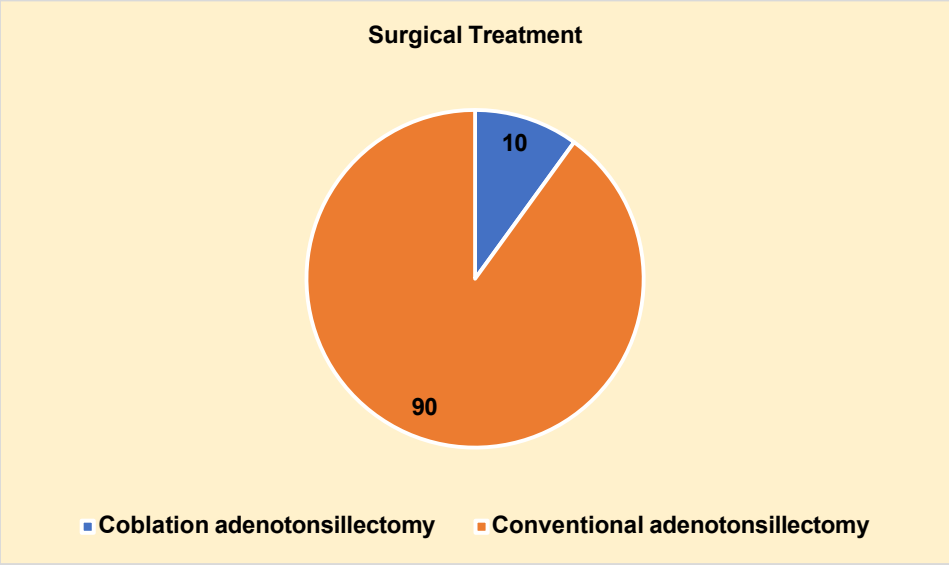
Inference:

All patients (100%) received medical therapy, including mometasone nasal spray and antihistamines. This indicates that conservative management was universally applied as the initial approach.

Table 17: Distribution based on the Surgical treatment

Surgical treatment	n	%
Coblation adenotonsillectomy	8	10
Conventional adenotonsillectomy	72	90

Graph 16: Distribution based on the Surgical treatment



Inference:

Among the patients, 90.0% underwent conventional adenotonsillectomy, while 10.0% were treated using coblation-assisted surgery. This shows a preference for conventional methods in this setting, possibly due to cost or availability.

DISCUSSION

Adenoidal hypertrophy (AH) and its measurement by clinical examination, imaging techniques, and endoscopic evaluation have been reported. Clinical examination with nasal obstruction is notoriously unreliable. Anterior rhinoscopy may be normal or show increased secretions, hypertrophy, or congestion of the inferior turbinates, while in some children, posterior rhinoscopy may identify large adenoids. The imaging techniques of the nasopharynx to visualise enlarged adenoids, still in use today, are X-rays. A lateral radiograph gives a measure of the absolute size of the adenoids and also an assessment of their relation to the size of the airway.[19] X-rays in the diagnosis of enlarged adenoids had been less popular by the turn of the last century with the advent of flexible nasopharyngoscopy, which is now considered to be the gold standard.

With the introduction of flexible fibre optic scope, examination of the nasal cavity and nasopharynx was made possible. Croft et al [65] used flexible endoscopy to assess the airway of sleep-associated upper airway obstruction in infants and young children. Prospective studies of the nasal cavity and nasopharynx in children using the fibre optic scopy have shown the size of the adenoid tissue to correlate with the results of tympanometry and radiography, as well as with the complaint of nasal obstruction and

snoring.[66] The latter test, however, has the disadvantage of being an invasive procedure. However, there is a paucity of literature; thus, the present study aimed to explore the correlation between the clinical presentation of adenoid hypertrophy and the findings from radiological and nasopharyngoscopy examinations.

In the current study, the mean age of the subjects was observed to be 9.83 ± 3.81 years, and a range spanning from 5 to 31 years, aligns with established patterns of AH prevalence. Predominantly affecting children, AH typically manifests in the pediatric population, with the adenoids reaching their maximum size between 5 and 7 years of age and a relative decrease in post-nasal space, in addition to the high incidence of upper respiratory tract infection (viral or bacterial) due to low immunity during the childhood period. Subsequently, these lymphoid tissues usually undergo involution during adolescence, often regressing by the age of 16. [40] This progression is well-documented in oto-laryngological literature, underscoring the commonality of AH in younger individuals

A systematic review and meta-analysis reported an AH prevalence of approximately 34.46% among children and adolescents, reinforcing the notion that AH is predominantly a paediatric condition.[5] The inclusion of adult cases in our study, although less common, is noteworthy. While AH is rare in adults due to the typical involution of adenoidal tissue, it can persist or recur, leading to clinical symptoms. Studies have documented adult cases of AH, often attributing persistence to factors such as chronic infections, allergic reactions, or environmental exposures. For instance, a study conducted in Turkey found a 26.28% prevalence of AH in adults undergoing para-nasal sinus computed tomography,

highlighting that AH can be a significant contributor to nasal obstruction in the adult population.[67]

Furthermore, research from India has reported that AH accounted for 21% of adult nasal obstruction cases, emphasizing the clinical relevance of considering AH in differential diagnoses for adults presenting with such symptoms. These findings suggest that while AH is predominantly a pediatric condition, its presence in adults, though less frequent, is clinically significant and warrants attention.[40] The present study's age distribution corroborates existing literature, affirming that AH primarily affects the pediatric population but can also be present in adults. This underscores the importance of considering AH in both children and adults presenting with relevant clinical symptoms, ensuring comprehensive evaluation and appropriate management across age groups.

The present study's observation of a male predominance in AH, with 66.3% of patients being male and 33.8% female, aligns with findings from several previous studies. For instance, a study by Dogru et al. reported that 68% of children with AH alone and 72% with AH combined with chronic otitis media with effusion were male, indicating a significant male predominance in AH cases.[68] Similarly, a retrospective analysis of pediatric adenoid surgeries in Sweden from 2004 to 2013 found that 60% of the 40,829 children who underwent adenoid surgery were boys, further supporting the trend of higher AH prevalence in males.[69] Chandrasekhar SBV et al[57] also observed AH in majority of males (58.3%) in comparison to females (41.6%). In contrast, Sarma and Khaund reported a higher prevalence of AH among females.[58]

The reasons behind this gender disparity are not entirely understood, but several hypotheses have been proposed. One suggestion is that males may have a higher susceptibility to upper respiratory tract infections during early childhood, which can contribute to lymphoid tissue hypertrophy, including the adenoids. Additionally, behavioral factors such as increased outdoor activity in boys might lead to greater exposure to environmental allergens and pollutants, potentially exacerbating adenoidal enlargement. However, it's important to note that not all studies have found a significant gender difference in AH prevalence. For example, a study by Evcimik et al[10] reported no significant difference in AH incidence between male and female children with allergic diseases.

The current study's finding that 75% of AH cases were from the lower-middle socioeconomic class, with 25% from the upper-middle class, aligns with existing literature highlighting a higher prevalence of AH among children from lower socioeconomic backgrounds. In a study conducted in Yozgat province, Turkey, researchers found that children from families with lower socioeconomic status had significantly higher rates of adeno tonsillectomy operations. This suggests a correlation between lower socioeconomic status and increased incidence of AH requiring surgical intervention.[70] Higher exposure to environmental pollutants, inadequate nutrition, and limited access to healthcare services, all of which can contribute to the development and persistence of AH.

The present study found that snoring (65.0%) and mouth breathing (50.0%) were the most commonly reported symptoms among patients with adenoid hypertrophy (AH), which is consistent with the typical clinical presentation of this condition. These findings are supported by several previous studies. For instance, Moideen et al. reported nasal obstruction followed by snoring as the most prevalent symptoms in their study population.[49] Similarly, Adegbiji et al[71] observed snoring in 87.0% of their patients, making it the most frequent symptom. Shetty et al[72] also identified snoring (36.0%) as a common presenting feature in children with AH. In another study by Yildirim et al[73], snoring was the predominant symptom reported by 52.2% of children and 20.0% of adults diagnosed with AH. Adedeji et al[3] reported an even higher association, with snoring present in 97.8% and nasal discharge in 96.7% of patients. Chandrasekhar SBV et al[57] reported nasal block (50%) and recurrent sore throat (21%) as the main chief complaints. Taken together, this consistent evidence across multiple studies underscores the clinical importance of snoring and mouth breathing as reliable indicators of adenoid hypertrophy.

In the present study, adenoid facies were observed in 27.5% of subjects with adenoid hypertrophy (AH), while 72.5% did not exhibit such features. In contrast to our study, a higher prevalence of adenoid facies (40%) was reported by Chandrasekhar et al [57]. However, the presence of adenoid facies is not a definitive indicator of AH. Studies have shown that while there is an association between AH and certain craniofacial patterns, not all individuals with AH develop the classical features of adenoid facies. For instance, a cross-sectional study assessing the prevalence of AH among 12-year-old children found that although there was a relationship between AH and certain skeletal patterns, the

manifestation of adenoid facies was not consistent across all cases.[74] Alam et al observed adenoid facies in 14% of the children with AH.[75] Zhang et al stated in their review that adenoid facies is a result of the long-term vicious cycle of mouth breathing, adenoid hypertrophy, and atypical craniofacial development.[76]

In the present study, lateral neck radiographs demonstrated that 48.8% of patients had Grade 2 adenoid hypertrophy and 32.5% had Grade 3, indicating that the majority exhibited moderate enlargement on radiological assessment. These findings are in line with those reported by Sarma and Khaund, who observed Grade 2 hypertrophy in 32% and Grade 3 in 48% of their study population, reinforcing the predominance of moderate to severe hypertrophy on X-ray.[58] Similarly, Pathak et al. found that 42% of children had moderate adenoid enlargement, while 38% had small adenoids and 20% had large adenoids.[77]

Chandrasekhar SBV et al [57] study observed that most of the X-ray naso-pharynx showed grade I and grade II, accounting for 40% each. Supporting this, Jyothirmai et al [17] reported that 51% of children had Grade 3 adenoids and 6.7% had Grade 1, further confirming the utility of lateral radiographs in detecting varying degrees of adenoid hypertrophy. Collectively, these findings underscore the consistency of radiographic grading patterns across different populations and emphasize the value of lateral neck X-rays as a reliable screening tool in the evaluation of adenoid hypertrophy.

On a positive note, endoscopic examination of the ear revealed that 90% of the present study subjects had an intact tympanic membrane. However, various degrees and locations of central perforations and congestion were noted, indicating ear involvement in some AH patients. AH is known to contribute to Eustachian tube dysfunction (ETD) by mechanically obstructing the nasopharyngeal orifice of the Eustachian tube, leading to impaired ventilation of the middle ear. This dysfunction can result in negative middle ear pressure, effusion, and, over time, TM changes such as retraction, thinning, or perforation. A study by Bhat et al. found that 36% of patients with AH had asymptomatic otitis media with effusion (OME), highlighting the silent nature of middle ear pathology in this population. [78]

In the present study, endoscopic examination of the nose in AH subjects revealed deviated nasal septum in 22.5% (right) and 33.7% (left), and 10-16% had Inferior turbinate hypertrophy (ITH). These structural variations could contribute to or result from chronic nasal obstruction. A 39.5% prevalence of DNS and 13% of ITH was reported by Rawat et al.[6] Rasheedi et al observed DNS in 11.3% of adult subjects with AH.[79] In a study undertaken by Khan et al[80], the most common rhinological conditions present were ethmoidal nasal polyps (34.89%), followed by DNS (26.17%), and inferior turbinate hypertrophy (24.83%). In a study by Thimmappa et al[81], DNS (79.0%) was the most common finding, while inferior turbinate hypertrophy was seen in 12.0% of patients. DNS coexisted with 25.0% of patients with adenoid hypertrophy in a study conducted by Yieldirim et al [73]. Developmental nasal septum deviation may indirectly cause low-grade chronic inflammation of the adenoids, interfering with their physiological regression.

In the present study, endoscopic examination of the oral cavity revealed that 96.2% of subjects with adenoid hypertrophy (AH) also exhibited tonsillar hypertrophy, highlighting a strong association between these two conditions. Zhao et al. reported that over 80% of children seeking orthodontic treatment had either adenoid or tonsillar hypertrophy, emphasizing the high prevalence of these conditions in pediatric populations.[82] The co-occurrence of AH and tonsillar hypertrophy is clinically significant, as it can exacerbate upper airway obstruction, leading to complications such as obstructive sleep apnea (OSA). Research indicates that adeno-tonsillar hypertrophy is a major determinant of OSA in children

In the present study, endoscopic evaluation of adenoids showed Grade 2 hypertrophy in 56.3% and Grade 3 in 31.3%. Bhise et al[83] reported Grade I adenoid hypertrophy in 59.4%, and Grade 2 in 26.6%. In a study by Thimmappa et al [81], grade I adenoid hypertrophy was observed in 26 cases (26%); grade II in 48 cases (48%); grade III in 20 cases (20%); and grade IV in 6 cases (6%). In contrast to the present study, most of the participants had grade I adenoid hypertrophy in the study undertaken by Kapusuz et al [67]. Results of the present study contrast with those of the study conducted by Saeed et al[84], where grade IV adenoid hypertrophy was observed in 67.0% of patients, while only 6.0% of patients had grade I adenoid hypertrophy. Also, in Chandrasekhar et al[57] study, the majority of the subjects had grade 1 (28.3%) and grade II (46.6%) AH in DNE examination.

The present study demonstrated a strong positive correlation ($r = 0.818$, $p < 0.001$) between radiological and endoscopic grading of adenoid hypertrophy. A progressive agreement was noted across different grades, with 100% of X-ray Grade 1, 77.8% of Grade 2, and 84% of Grade 3 cases corresponding to their respective endoscopic grades. This emphasizes a substantial level of agreement between the two modalities, especially in the more apparent and advanced grades of hypertrophy. Similar observations have been reported in several previous studies, reinforcing the reliability of endoscopy and radiography as complementary diagnostic tools.

Pathak et al[77] found good agreement between the X-ray and endoscopic methods, with the sensitivity and specificity of X-ray being 79.41% and 75%, respectively, while nasal endoscopy showed even higher sensitivity (87.10%), though with slightly lower specificity (63.16%). Jyothirmai et al[17] also reported highly significant correlations between endoscopic and clinical grading ($p = 0.0006$), as well as between radiological and endoscopic grading ($p = 0.0003$), and a significant correlation between clinical and radiological grading ($p = 0.04$). These findings further support the clinical value of combining endoscopic and radiological assessments. Similarly, Dawood and Khammas[23] found Grade 3 adenoid hypertrophy to be the most common in all modalities. The agreement between flexible and perioperative rigid nasal endoscopy versus radiology was statistically significant ($p = 0.0001$), and the comparison between the two endoscopic techniques revealed no significant difference, indicating the reliability and consistency of nasal endoscopy.

Several other studies also reported strong correlations. Jain et al[85] found a highly significant correlation between radiological and endoscopic grading ($p = 0.001$), and Ehab Yaseen et al[86] confirmed similar findings in their study. Kindermann et al[87] reported a sensitivity of 92% and specificity of 71% for nasal flexible fiberoptic endoscopy, while Dixit et al[88] observed a 62% agreement between X-ray and endoscopy. In the study by Peedikakaal et al [63], a significant correlation ($p = 0.006$) was observed, particularly for Grade III and IV adenoids, although cases with Grade II hypertrophy on endoscopy often appeared as Grade III on radiographs. Mlynarek et al[89] also demonstrated that the percentage of airway obstruction assessed by lateral neck X-ray highly correlated with findings on fiber-optic rhinoscopy, reinforcing the diagnostic relevance of radiographs when used alongside endoscopy.

These consistent findings across various studies can be attributed to several factors. Nasal endoscopy offers a direct, three-dimensional visualization of the adenoids, allowing precise grading. Meanwhile, radiographs, though limited to two-dimensional imaging, can still reliably reflect the degree of airway obstruction when properly standardized. The anatomical location and growth pattern of adenoids, often posterior and midline, are typically well-visualized by both methods, explaining the high degree of correlation observed in most studies.

However, some studies have reported discrepancies between radiographic and endoscopic findings, particularly in cases of severe hypertrophy. For instance, Sarma and Khaund[58] found that 32% of patients exhibited Grade IV hypertrophy on nasal

endoscopy, whereas only 12% showed the same on radiographs. Similarly, Souza and Hennemann [90] reported that 27% of patients with no radiographic signs of airway obstruction had severe hypertrophy as seen on fiberoptic nasoscopy. Lourenco et al [91] also noted that children with small adenoids on X-ray were often found to have moderate or large adenoids on endoscopy. These differences highlight the tendency of lateral X-rays to underestimate adenoid size in certain cases.

Several technical and anatomical factors contribute to such discrepancies. X-ray imaging provides a two-dimensional view and is more susceptible to positional variations, respiratory motion, and lack of standardization. In contrast, endoscopy offers a direct and dynamic visualization of the adenoid tissue, capturing details of lateral or anterior growth that might be missed on radiographs. Additionally, radiographic assessments may not accurately reflect functional obstruction, especially in cases where hypertrophy is asymmetrical or extends in directions not easily visualized in lateral views.

Despite these limitations, lateral X-rays remain valuable as a screening and supplementary diagnostic tool. As emphasized by Gill et al[92], although nasal endoscopy is emerging as the gold standard for diagnosing adenoid hypertrophy, the lateral X-ray of the nasopharynx continues to serve as a reliable method. When used together, these diagnostic modalities provide a comprehensive evaluation, aiding in accurate diagnosis and optimal patient management.

The universal application of conservative management using mometasone nasal spray and antihistamines in our study reflects a strategic approach to initially manage adenoid

hypertrophy non-surgically. Intranasal corticosteroids, particularly mometasone furoate, have demonstrated efficacy in reducing adenoid size and alleviating associated symptoms. For instance, a study by Monga et a[93] reported significant improvements in nasal obstruction, reduction in adenoid size, and reduced number of adenoidectomies required following an 8-week course of mometasone furoate nasal spray. Similarly, a randomized controlled trial by Yilmaz et al[94] found that adolescents treated with mometasone furoate experienced notable symptom relief, although changes in adenoid size were not statistically significant. The addition of antihistamines addresses the allergic component often associated with adenoid hypertrophy, potentially enhancing the overall therapeutic effect. This combined medical therapy serves as a viable first-line treatment, potentially reducing the need for surgical intervention in select patient populations.

In our study, 90% of patients underwent conventional adenotonsillectomy, while 10% received coblation-assisted surgery. This distribution reflects the prevailing preference for traditional techniques, likely due to factors such as cost-effectiveness, equipment availability, and surgeon familiarity. However, coblation-assisted adenotonsillectomy has been associated with several perioperative advantages. A systematic review and meta-analysis by Ahmad et al[95] reported that coblation tonsillectomy resulted in less postoperative pain, reduced intraoperative blood loss, and shorter operative times compared to cold dissection techniques. Similarly, a randomized controlled trial by Vyas et al[96] found less postoperative pain and earlier return to normal activities. Despite these benefits, concerns regarding higher costs and the need for specialized equipment may limit the widespread adoption of coblation techniques.

The present study acknowledges certain limitations; firstly, a small sample size and a single-centre study may limit the generalisability of the findings. There may be superimposition of anatomic structures and inter-observer variations in interpreting the X-rays, also, the positional changes and respiratory movements may also affect the X-ray images.

CONCLUSION

The study highlights that adenoid hypertrophy predominantly affects school-aged children, with a mean age of 9.83 years, and is more commonly observed among males and individuals from lower-middle socioeconomic backgrounds. Snoring and mouth breathing were the most frequent clinical complaints, reflecting the typical symptom profile of AH. Radiological and endoscopic evaluations both revealed a predominance of moderate-grade hypertrophy, with a strong positive correlation ($r = 0.818$, $p < 0.001$) between the two modalities. This indicates that although endoscopy remains the gold standard, lateral X-ray continues to be a practical and reliable tool in settings where advanced endoscopic facilities are not readily available.

The study also identified significant co-existing conditions, such as tonsillar hypertrophy, deviated nasal septum, and inferior turbinate hypertrophy, underscoring the multifactorial nature of upper airway obstruction in these patients. Universal initial treatment with intranasal corticosteroids and antihistamines proved to be a rational and effective first-line approach. However, surgical intervention remained necessary in the majority of

cases, with conventional adeno-tonsillectomy being the preferred method. Despite certain

limitations, such as small sample size and single-center design, this study reinforces the diagnostic and therapeutic value of combining clinical evaluation with radiological and endoscopic findings in the comprehensive management of adenoid hypertrophy.

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