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Exudative Catarrhal Otitis Media: Modern Approaches To Diagnosis And Management

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Abstract
Exudative catarrhal otitis media (ECOM) represents an inflammatory process within the tympanic cavity, characterized by the accumulation of serous or mucous effusion in the absence of pronounced signs of acute inflammation. The condition is one of the most common causes of hearing impairment in preschool- and early school-aged children and frequently develops with minimal symptoms, ultimately leading to persistent morphological changes of the middle ear. This article provides up-to-date information on the pathogenesis, classification, clinical features, diagnostic methods, and therapeutic approaches to ECOM, including medical treatment, endoscopic techniques, and preventive strategies.
Keywords: otitis media with effusion; exudative catarrhal otitis media; Eustachian tube dysfunction; tympanometry; conductive hearing loss; pediatric otolaryngology; PRP/PRF therapy; myringotomy; tympanostomy tubes; adenoid hypertrophy; middle ear effusion.

Introduction

Exudative catarrhal otitis media is among the most prevalent ENT disorders in children. According to various studies, its incidence reaches 60–85% in the 3–7-year-old age group. The condition has significant clinical relevance, as prolonged ECOM can lead to persistent conductive hearing loss, impaired speech and psychomotor development, and an increased risk of progression to adhesive otitis media or chronic forms of the disease.

The primary pathogenetic mechanism is dysfunction of the Eustachian tube, resulting in impaired ventilation of the middle ear, negative pressure formation, and effusion accumulation. The disease often follows a latent course, necessitating a high level of clinical vigilance among primary care physicians and the use of objective diagnostic methods.

Pathogenesis of Exudative Catarrhal Otitis Media

The pathogenesis of exudative catarrhal otitis media is a complex, multilevel process driven by the combined influence of anatomical features, functional disturbances, immunological responses, and inflammatory changes in the nasopharynx and middle ear. The condition develops against the background of persistent impairment of middle-ear ventilation, leading to alterations in intracavitary pressure, transudation of serous fluid, and progressive accumulation of viscous mucous secretions. These mechanisms are especially pronounced in children due to the structural and physiological characteristics of their Eustachian tube and mucosal tissues.

The key pathogenetic factor is impaired ventilatory function of the Eustachian tube. Anatomical features typical of childhood—namely, a wider, shorter, and more horizontally oriented Eustachian tube—predispose to frequent dysfunction even with minimal inflammatory or allergic changes in the nasopharynx. Even mild mucosal edema in the tubal area compromises aeration of the tympanic cavity, leading to marked negative pressure and retraction of the tympanic membrane. Under conditions of reduced intratympanic pressure, plasma begins to transude through the capillaries of the middle-ear mucosa, initiating fluid accumulation.

Inflammatory pathology of the nasopharynx plays a significant role in the development

of ECOM. In children, adenoidal hypertrophy, chronic adenoiditis, recurrent upper-respiratory infections, and allergic rhinitis create a persistent inflammatory environment characterized by mucosal edema in the tubal zone and mechanical obstruction of air passage through the Eustachian tube. As a result, the mucosa of the tympanic cavity begins actively secreting serous-mucous exudate, and due to impaired drainage, this fluid gradually accumulates, altering the physicochemical environment of the middle ear.

Changes in the properties of the secretion represent another critical mechanism. Under chronic inflammation, secretory epithelial cells increase the production of mucin—a high-molecular-weight glycoprotein responsible for the viscosity and density of the effusion. Simultaneously, mucociliary clearance is suppressed: ciliary activity decreases, the epithelial layer thickens, and the overall ability to evacuate secretions diminishes. Consequently, dense, adhesive mucous effusion accumulates in the tympanic cavity, a condition often referred to as “glue ear,” reflecting its morphological nature and its impact on ossicular mobility.

Allergic factors significantly influence the course of exudative otitis. A substantial proportion of children exhibit atopic predisposition manifested by hyperreactivity of the nasopharyngeal mucosa. Mediators such as histamine, leukotrienes, and prostaglandins contribute to persistent edema and chronic Eustachian tube dysfunction. Allergic inflammation also enhances mucus production and promotes hyperplasia of glandular structures, leading to the formation of thick, cell-rich effusion. Therefore, children with allergic rhinitis or bronchial asthma often experience prolonged or recurrent ECOM.

Immune characteristics of early childhood also contribute to the persistence of the disease. Immaturity of local immune defenses, insufficient production of secretory IgA, reduced macrophage activity, and incomplete barrier function of mucosal tissues promote prolonged inflammation. Viral and bacterial toxins further impair Eustachian tube function and alter secretion properties, reinforcing the pathological cycle: dysfunction → effusion → hypoventilation → progression of otitis.

Classification

The classification of exudative otitis media reflects the sequential evolution of pathological changes in the middle ear. Four major stages are distinguished, each with characteristic clinical and pathogenetic features that determine management strategies.

1. Catarrhal (initial) stage.

This stage is marked by persistent Eustachian tube dysfunction and negative pressure in the tympanic cavity. Retraction and decreased mobility of the tympanic membrane are observed. Effusion is absent or minimal. Symptoms are often mild or absent.

2. Exudative stage.

Serous fluid begins to accumulate due to transudation and impaired mucociliary clearance. The secretion remains relatively thin, causing mild hearing loss. Parents typically first notice reduced responsiveness to speech.

3. Mucoid stage.

Under chronic inflammation, mucin production increases, and the effusion becomes thick and adhesive (“glue ear”). Evacuation becomes nearly impossible. Conductive hearing loss becomes significant.

4. Fibrous stage.

Persistent effusion induces structural remodeling, including fibrosis, scar formation, thickening of the mucosa, and development of adhesive otitis. The tympanic membrane becomes immobile, and conservative therapy becomes less

effective, often requiring surgical intervention.

Clinical Presentation

Clinical manifestations of exudative otitis media are highly variable, but a predominantly silent, minimally symptomatic course is characteristic. The absence of pain and acute inflammation often leads to delayed diagnosis.

The most typical symptom is progressive hearing loss, manifested by frequent requests to repeat speech, reduced response to quiet sounds, or increased television volume.

A feeling of ear fullness is common but not universal. Older children and adults may experience autophony due to reduced tympanic membrane mobility. Some patients report a sensation of fluid movement when tilting the head, indicating early-stage mobile effusion.

Among school-aged children, hearing impairment may cause reduced academic performance, attention difficulties, and fatigue. In younger children, ECOM may lead to delayed speech development, as adequate auditory perception is vital for speech acquisition. Infants often have minimal or nonspecific symptoms, contributing to late detection during routine examinations.

Diagnosis

The diagnosis of exudative catarrhal otitis media (ECOM) is based on a comprehensive combination of clinical, otoscopic, and instrumental methods that allow an objective assessment of the condition of the tympanic cavity, Eustachian tube function, and the degree of hearing impairment. Since the disease often follows a latent course and is not accompanied by pronounced inflammatory manifestations, the use of objective diagnostic tools is essential for early effusion detection and accurate staging of the pathological process.

In clinical practice, otoscopy plays a key role, providing visual assessment of the tympanic membrane and external auditory canal. Typical signs of ECOM include retraction of the tympanic membrane due to negative pressure in the tympanic cavity and changes in its coloration—ranging from pale yellow to bluish—reflecting the presence of transudate or mucoid effusion. In some cases, fluid levels or air bubbles may be observed, which shift with head movement and serve as pathognomonic indicators of effusion. Pneumatic otoscopy offers additional information by assessing tympanic membrane mobility, which is significantly reduced or absent in the presence of fluid.

To evaluate the nasopharynx and Eustachian tube more precisely, endoscopy is widely used. This method enables visualization of the tubal orifice, assessment of adenoid hypertrophy, mucosal condition, and signs of inflammation or edema in the tubal region. Children with ECOM frequently exhibit a combination of adenoiditis and inferior turbinate hypertrophy, contributing to chronic Eustachian tube dysfunction. Endoscopy allows clinicians to identify anatomical obstructions affecting middle-ear ventilation and determine the need for surgical correction such as adenoidectomy.

The primary instrumental method with high sensitivity and specificity is tympanometry (impedance audiometry). This technique assesses the compliance of the tympanic membrane and pressure within the middle ear. ECOM most commonly presents with a type B tympanogram, characterized by a flat curve indicating the presence of effusion that completely restricts ossicular and tympanic membrane mobility. In some cases, a type C tympanogram may be recorded, reflecting significant negative pressure and an early stage of Eustachian tube dysfunction preceding effusion accumulation. Impedance audiometry is

complemented by acoustic reflex testing, with absent reflexes serving as an additional indicator of conductive pathology. Audiometric testing is an essential component of diagnostics, providing quantitative assessment of hearing loss. ECOM typically manifests as mild to moderate conductive hearing loss (Grade I–II), with an increased air–bone gap and preserved bone conduction thresholds. For young children who cannot undergo tonal audiometry, objective methods such as otoacoustic emissions or acoustic reflex measurements are employed to detect subtle functional impairments.

Additional imaging methods are used when chronic structural alterations are suspected. Ultrasound assessment of the tympanic membrane and tympanic cavity, though less common, may help evaluate membrane thickness and effusion characteristics. Computed tomography (CT) of the temporal bones is indicated in long-standing or recurrent ECOM to identify tympanosclerosis, fibrotic changes, ossicular anomalies, or other structural defects. CT is particularly valuable when planning surgical intervention or assessing potential comorbid conditions.

Treatment, Complications, and Prevention

The therapeutic strategy for exudative catarrhal otitis media aims to restore Eustachian tube ventilation and drainage, reduce local inflammatory changes, and promote effusion clearance from the tympanic cavity. Current clinical guidelines emphasize a comprehensive, stepwise approach that takes into account both middle-ear pathology and associated nasopharyngeal conditions.

Conservative treatment focuses on managing inflammatory diseases of the upper respiratory tract. Treatment of adenoiditis, rhinitis, and rhinosinusitis reduces nasopharyngeal mucosal edema and restores patency of the Eustachian tube orifice. In patients with allergic rhinitis, topical intranasal corticosteroids are included to reduce inflammation and normalize mucociliary clearance. Mucolytic and secretolytic agents help thin the viscous effusion and facilitate drainage. Mechanical restoration of Eustachian tube patency may involve Politzer inflation or transnasal catheterization. Physiotherapeutic techniques such as ultraviolet irradiation and laser therapy applied to the tubal area may also be beneficial. Importantly, systemic antibiotics are not routinely indicated, as ECOM is not an acute suppurative process.

In recent years, bioregenerative approaches based on autologous plasma-derived products have gained popularity. Application of PRP/PRF to the nasopharynx and Eustachian tube region is considered a potential method for stimulating mucosal regeneration, enhancing microcirculation, and improving drainage function. Clinical observations demonstrate favorable outcomes with fibrin matrices, particularly in patients with chronic or recurrent forms of the disease.

If conservative therapy fails within 2–3 months, surgical intervention becomes necessary. The most common procedures are myringotomy to facilitate effusion drainage and tympanostomy, involving placement of a ventilation tube to ensure prolonged aeration of the tympanic cavity. In cases of significant adenoid hypertrophy, adenoidectomy is essential, as adenoidal obstruction is a major contributor to chronic Eustachian tube dysfunction and recurrence of ECOM.

Prolonged ECOM carries an increased risk of complications. The most significant are persistent conductive hearing loss, adhesive otitis media, and formation of tympanosclerotic foci that irreversibly reduce tympanic membrane and ossicular mobility. In young children, prolonged hearing impairment may lead to delayed

speech and psychomotor development. Chronic, relapsing ECOM also predisposes patients to long-term structural changes within the middle ear.

Preventive measures focus on timely correction of nasopharyngeal diseases and elimination of risk factors. Regular treatment of chronic rhinitis, sinusitis, and adenoiditis reduces the frequency of Eustachian tube dysfunction. Timely adenoidectomy in cases of marked adenoidal hypertrophy prevents persistent hearing loss and recurrent ECOM. Additional preventive strategies include vaccination against pneumococcal infection, minimizing exposure to tobacco smoke, ensuring adequate breastfeeding and proper feeding position in infants, and comprehensive management of allergic diseases. These measures reduce both primary incidence and risk of chronic progression.

Conclusion

Exudative catarrhal otitis media represents a significant medical and social concern, particularly in pediatric populations. Early diagnosis and timely management are essential to prevent permanent hearing impairment and improve quality of life. The application of objective diagnostic methods, individualized treatment strategies, and modern minimally invasive technologies substantially enhances therapeutic success. Promising directions include the development of endoscopic surgical techniques, bioregenerative approaches, and novel methods aimed at improving Eustachian tube function.

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