CLINICAL AND MORPHOLOGICAL PARALLELS IN THE ETIOPATHOGENESIS OF SECRETORY OTITIS MEDIA

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Abstract. Introduction. According to global statistics, secretory otitis media (SOM) ranks first among middle ear infections. This pathology is believed to be characteristic of the pediatric population; however, recent global statistics indicate an increase in the percentage of SOM cases among the adult population. Information about adult-onset SOM varies, with no consensus on the causes of its occurrence. Diverse clinical presentation of the condition, often unsatisfactory treatment outcomes, and a high percentage of recurrences have necessitated a more in-depth study of the etiological factors associated with adult-onset SOM.

The objective of the research was to determine clinical and morphological parallels in the etiopathogenesis of adult-onset SOM as well as to establish the relationship between the clinical, otoscopic, and endoscopic presentations and pathomorphological changes in the tubal tonsils in these patients.

Materials and Methods. Sixty-eight (38 men and 30 women) patients with SOM at the age of 18 to 65 years were examined. Fifty-three patients were diagnosed with unilateral pathological process, while in 15 individuals, bilateral SOM was observed. All the patients underwent a comprehensive assessment of their complaints, collection of their medical history, and a full ear, nose, and throat examination using conventional methods, optical techniques, video endoscopy of the nasopharynx and tubal tonsils, and otomicroscopy. In 15 patients, to exclude a malignant process, tissue samples from the tubal tonsils were taken for pathological examination.

Results. One of the causes of adult-onset SOM could be pathomorphological changes in the tubal tonsils, specifically their inflammation, hypertrophy, and in some cases, tumours. The clinical course and stages of SOM development depend on the duration of auditory tube obstruction. Inflammation of the tubal tonsils usually results in catarrhal and serous SOM, while hypertrophy or hyperplasia of the tubal tonsils is observed in patients with mucous SOM.

Conclusions. Pathomorphological changes in the tubal tonsils have been established as one of the reasons for developing SOM in adults. Both the clinical presentation of SOM and the stage of its progression depends on the type of pathomorphological changes, their localization (upper or lower pole), and the duration of auditory tube obstruction. A prolonged, recurrent, and treatment-resistant course of SOM may be associated with hypertrophy of the tubal tonsil or tumours. Clinical and morphological parallels identified by us will enable timely utilization of effective etiopathogenetic treatment of SOM and prevent the occurrence of complications.

Keywords: Secretory Otitis Media; Tubal Tonsils; Pathomorphology of the Tubal Tonsils; Lymphoid Tissue; Acute Respiratory Viral Infections; Optical Endoscopy; Otomicroscopy

Introduction

According to global statistics, secretory otitis media (SOM) ranks first among middle ear infections [1-5].

The clinical course and presentation of SOM are determined by the presence of the etiological and pathogenetic factors specific to this condition. SOM is a multifactorial disease; however, the majority of specialists consider impaired Eustachian tube functions, specifically ventilation and drainage, as its primary cause [6-9]. Nasal and sinus disorders, including those of viral etiology, are implicated as the main etiological factors for developing Eustachian tube dysfunction. Other contributing factors include deviated septum, chronic sinusitis, allergic reactions, adenoid hypertrophy, as well as immune and hormonal disorders [1,2,10-13].

This pathology is believed to be characteristic of the pediatric population [9,10,12,14-18]. However, recent global statistics indicate an increase in the percentage of adult-onset SOM cases [3-5,19,20].

A lot of research works are devoted to SOM in children. They demonstrate a consensus on the etiology and pathogenesis of the condition, with diagnostic criteria being established and the main approaches to the treatment and rehabilitation of patients being determined [2-5,10,12,14,16,21]. Information about adult-onset SOM varies, with no consensus on the causes of its occurrence. In the literature, there are occasional data regarding a potential influence of the tubal tonsils on Eustachian tube dysfunction resulting in SOM.

Diverse clinical presentation of the condition, often unsatisfactory treatment outcomes, and a high percentage of recurrences have necessitated a more in-depth study of the etiological factors associated with SOM in adults.

The objective of the research was to determine clinical and morphological parallels in the etiopathogen-
thesis of adult-onset SOM as well as to establish the relationship between the clinical, otoscopic, and endoscopic presentations and pathomorphological changes in the tubal tonsils in these patients.

Materials and Methods

The examination of patients was carried out at the Department of Otolaryngology with the Course of Neck and Head Surgery, Ivano-Frankivsk National Medical University (IFNMU), Ivano-Frankivsk, Ukraine.

The study included 68 patients with SOM at the age of 18 to 65 years who were divided into two groups. Group I comprised 38 (23 men and 15 women) subjects; the average age - 32.93±4.1 years; disease duration - from 10-14 days to 1 month. Group II included 30 (18 men and 12 women) individuals; the average age - 38.21±4.8 years; disease duration - more than 1 month. Fifty-three patients were diagnosed with unilateral pathological process, while in 15 individuals, bilateral SOM was observed.

Patients were assigned to groups after a comprehensive assessment of their complaints, collection of their medical history, and a full ear, nose, and throat (ENT) examination using conventional methods and optical techniques.

The nose and nasopharynx were examined by means of a video endoscopy system Karl Storz (Germany) with a 0- and 30-degree, 2.7-mm- and 4.0-mm-diameter endoscope. The structure of the nasal and nasopharyngeal cavities was studied in detail, with special attention given to the state of the tubal tonsils. Otomicroscopy was performed using an operating microscope OPMI 1-FC (Carl Zeiss, Germany) with magnification of 6x and 12x. In cases of suspected tumour of the tubal tonsil, biopsy was performed under endoscopic guidance after administering local anesthesia. The obtained samples were fixed in a 10% neutral formalin solution (pH - 7.0) for 24 hours. Tissue samples were then rinsed well under running water for 10-20 min and dehydrated in an alcohol series with ascending concentrations, followed by chloroform and a mixture of chloroform and paraffin (1:1). Paraffin blocks were then prepared. Histological sections with a sickness of 5-6 μm were generated using a rotary microtome and stained with hematoxylin and eosin. The obtained histological specimens were examined under a light microscope MC 300 (Micros, Austria) and captured with a digital camera Toup Tek Photonics AMAO75, the TouView v. 3 software (Educational and Scientific Laboratory of Morphological Analysis, the Department of Human Anatomy, IFNMU, Ivano-Frankivsk, Ukraine).

Results

All patients presented with complaints of ear congestion, a feeling of fluid and pressure in the affected ear, tinnitus, hearing loss, autophony, and fluctuating hearing.

From the medical history, it has been established that the main causes of SOM, as reported by patients, were colds and infectious diseases, specifically acute respiratory infection and acute respiratory viral infection.

Patients with acute suppurative otitis media, nasopharyngeal neoplasms, chronic rhinosinusitis, and allergic rhinitis were not included in the study.

SOM was diagnosed based on the patient’s complaints (ear congestion, hearing loss, ear or head noises, ear fullness), medical history data with a focus on the duration of the condition, prior treatment and its effectiveness, otoscopy findings as well as the results of tuning fork tests, tympanometry, pure-tone threshold audiometry, endoscopic examination of the nasal and nasopharyngeal cavities with a detailed assessment of the state of the tubal tonsils, and computed tomography (CT) of the nasopharynx and temporal bones.

During otomicroscopy, in patients with serous SOM (Group I), the tympanic membrane exhibited colors ranging from gray-white, pinkish to cyanotic with pronounced vascular injection. The air-fluid levels or bubbles were observed behind the tympanic membrane, which changed with changes in body position. In some patients, the tympanic membrane appeared thickened, retracted in the upper portions and convex in the posterior ones, indirectly indicating the accumulation of exudate in the tympanic cavity. The light cone was deformed; the short process of the malleus protruded into the lumen of the external auditory canal (Fig. 1a, b).

Fig. 1. Otomicroscopy image showing the tympanic membrane of a 31-year-old male patient (a) and a 29-year-old female patient (b) with serous secretory otitis media.
In patients with mucus SOM (Group II), otomicroscopy showed the tympanic membrane being retracted in the unstretched portion, convex in the lower quadrants, immobile, thickened, and yellowish indicating the presence of thick, viscous secretion within the tympanic cavity, limiting the movement of the auditory ossicles (Fig. 2a, b).

Endoscopy of the nasopharynx showed a hyperemic, infiltrated, and enlarged tubal tonsil behind the auditory tube in the fossa of Rosenmüller in patients of Group I; in some cases, it exhibited a granular surface and accumulation of mucus (Fig. 3a, b).

In most patients with mucus SOM (Group II), an enlarged, thickened, and granular tubal tonsil with surface infiltration and accumulation of thick mucus was observed (Fig. 4a). It is worth noting that in hypertrophy of the lower tubal tonsil portions, the course of the disease was prolonged and clinically mildly expressed. Hypertrophy of the upper tubal tonsil portions, especially extending onto the torus tubarius, resulted in the obstruction of the pharyngeal orifice of the auditory tube, subsequently disrupting its ventilation and drainage functions (Fig. 4b).

The material for pathomorphological examination was obtained during the removal of hypertrophied portions of the tubal tonsils, where conservative therapy was ineffective, as well as from patients with suspected malignant processes (6 patients of Group I and 9 patients of Group II). In patients of Group I, signs of inflammation of lymphoid tissue of the tubal tonsils were found (Fig. 5).

In the clinical presentation of tubal tonsil inflammation, lymphoid tissue of the tubal tonsils was covered by the ciliated pseudostratified columnar epithelium with a height of 56.48±2.61 μm. In certain areas, the surface epithelium was infiltrated by lymphocytes migrating from lymphoid tissue, showing mild signs of hyperplasia. The lymphoid follicles appeared small. Isolated specimens exhibited sclerotic changes in the form of delicate fibrous structures between lymphoid tissue and follicles. In patients of Group II, hypertrophy signs were found (Fig. 6).

In the clinical presentation of tubal tonsil hypertrophy, pathomorphological changes in the tubal tonsils were characterized by remodeling in the superficial prismatic epithelium and lymphoid tissue. The hypertrophied tubal tonsils exhibited an uneven surface with depressions. The prismatic epithelium underwent metaplasia into a non-keratinized stratified squamous epithelium, occasionally with degenerative and dystrophic changes and infiltration by lymphocytes and neutrophils (Fig. 6a). The height of the metaplastic epithelium was 85.31±7.78 μm. It should be noted that metaplasia of the surface epithelium serves as a morphological marker of chronic inflammation. Almost all specimens displayed areas of connective tissue proliferation with visualization of coarse fibrous structures and focal infiltrations (Fig. 6a, b). In these same patients, blood vessels with pronounced signs of vascular wall sclerosis were frequently identified (Fig. 6b). Lymphoid tissue was characterized by hyperplastic secondary follicles packed closely together with light centers and surrounded by a lymphoid cuff (Fig. 6d). Malignant tumours were diagnosed in five (4 women and 1 man) patients (Fig. 7). To confirm the histopathological diagnosis, the specimens were subjected to immunohistochemical investigation.

The tubal tonsil suspected of having tumour was covered with metaplastic non-keratinized stratified squamous epithelium with a height of 126.8±52.6 μm. Epithelial cells were densely arranged with eosinophilic cytoplasm. The cell nuclei were enlarged, round to oval, with distinct nucleoli. In most cases, the epithelium covering the tubal tonsil exhibited pronounced degenerative and dystrophic changes in the form of perinuclear cytoplasmic clearing.

Patients diagnosed with tubal tonsil tumours were excluded from the study. Patients with a verified diagnosis continued their treatment at the Ivano-Frankivsk Regional Oncology Dispensary.

**Discussion**

Our research analysis has confirmed numerous literature data that SOM is a multifactorial disease. The pathogenesis of its development is multifaceted. Some etiological and pathogenetic factors are interrelated, which complicates the course of the disease. Identifying new pathogenetic links of SOM will allow for achieving better functional outcomes, reducing recurrence rates, and improving patient quality of life.

According to our research, one of the causes of adult-onset SOM could be pathomorphological changes in the tubal tonsils, specifically their inflammation, hypertrophy, and in some cases, malignant processes. The clinical course and stages of SOM development depend on the duration of auditory tube obstruction. Inflammation of the tubal tonsils usually results in catarrhal and serous SOM, while hypertrophy or hyperplasia of the tubal tonsils is observed in patients with mucous SOM. Considering pathomorphological changes in the tubal tonsils in the etiopathogenesis of SOM will enable timely application of effective treatment, thereby allowing for preventing complications, particularly the formation of scars, atelectasis, and myringosclerosis, which in turn can lead to hearing loss and social isolation.

**Conclusions**

Thus, based on our conducted research, clinical and morphological parallels have been established in the etiopathogenesis of adult-onset SOM.

1. Pathomorphological changes in the tubal tonsils have been established as one of the reasons for developing SOM in adults.

2. Both the clinical presentation of SOM and the stage of its progression depends on the type of pathomo-
Fig. 2. Otomicroscopy image showing the tympanic membrane of a 42-year-old male patient (a) and a 34-year-old male patient (b) with mucous secretory otitis media.

Fig. 3. Endoscopy image showing the tubal tonsil of a 42-year-old male patient (a) and a 34-year-old female patient (b) with serous secretory otitis media.

Fig. 4. Endoscopy image showing the tubal tonsil of a 42-year-old male patient (a) and a 34-year-old male patient (b) with mucous secretory otitis media.
Fig. 5. Inflammation of the tubal tonsil in a 26-year-old male patient. Hematoxylin and eosin staining. a) 10x10 magnification; b) 10x40 magnification. 1 - surface respiratory epithelium infiltrated by lymphocytes migrating from lymphoid tissue; 2 - lymphoid follicle; 3 - parafollicular lymphoid tissue; 4 - red blood cell deposit; 5 - red blood cells outside the blood flow, hemorrhages.

Fig. 6. Hypertrophy of the tubal tonsil in a 48-year-old male patient. Hematoxylin and eosin staining. a) 10x40 magnification; b) 10x10 magnification; c) 10x40 magnification; d) 10x10 magnification. 1 - metaplasia of the surface epithelium; 2 - sclerotic areas; 3 - lymphoid tissue; 4 - perivascular sclerosis; 5 - hyperplasia of parafollicular lymphocytes; 6 - a group of hyperplastic secondary follicles packed closely together and surrounded by a lymphoid cuff.
3. A prolonged, recurrent, and treatment-resistant course of SOM may be associated with hypertrophy of the tubal tonsil or tumours.

4. Clinical and morphological parallels identified by us will enable timely utilization of effective etiopathogenetic treatment of SOM and prevent the occurrence of complications.

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