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Production of Otitis Media Mucin and Mucous Cell Metaplasia

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Abstract

Otitis medium (OM) with mucoid effusion is a form of OM that usually progresses to chronic OM in young children. It is characterised by mucous cell metaplasia/hyperplasia in the middle ear cleft and thick fluid accumulation in the middle ear canal. The development of OM with mucoid effusion is influenced by a number of variables, particularly problems with mucin synthesis brought on by middle ear bacterial infection and Eustachian tube dysfunction. By examining cellular and molecular processes such mucin formation and mucous cell differentiation in the middle ear mucosa with OM, we will analyse several aspects of this condition in this review. Infectious diseases, factors that cause the formation of mucin, and pertinent signalling pathways will also be covered.

Mucous cell metaplasia, which causes mucous hyper secretion and the condition to persist, is a major problem in otitis media. The molecular pathways behind mucous cell metaplasia in otitis media are not well understood, yet. Atonal homolog 1 (Atoh1), a basic helix-loop-helix (bHLH) transcription factor, has been demonstrated to be crucial for the differentiation of intestinal goblet cells in numerous studies of intestinal epithelial homeostasis. On the other hand, it has been suggested that the "Ets" transcription factor family member SAM-pointed domain-containing Ets transcription factor (SPDEF), causes asthma or lung viral illnesses to cause mucous cell metaplasia. Recent research have shown the relationship between these variables, proving that Spdef works downstream of Atoh1. Due to the fact that the pulmonary and middle ear epithelia both come from the same respiratory tract, we could use the advantages of these results to further our understanding of otitis media. When it comes to treating otitis media with mucous cell metaplasia, which is usually referred to as "intractable" in clinical settings, Atoh1 and SPDEF may be the best therapeutic targets.

Keywords: Otitis media with effusion; Tympanostomy tube; Ventilation tube; Grommet; Child; Lacrimal gland; Nasolacrimal ducts; Mucin; Cancer; Inflammation; Ear; External; Middle

Introduction

Middle ear, tracheal, digestive, and reproductive system mucus discharges commonly contain mucin glycoproteins. A Protective inherent defence mechanism the production of mucus trap invasive microorganism for later elimination by the mucociliary clearance system while also lubricating and protecting the epithelium. However, excessive mucin inhibits the mucociliary clearance mechanism in chronic infections, causing mucus to build up and the mucus-lined epithelial tracts to perform poorly [1]. MUC5AC mucin, one of the 24 mucin genes known so far, has been demonstrated to be crucial in the development of upper respiratory tract infections, including otitis media (OM). Overproduction of mucus and excessive middle ear irritation are characteristics of OM. Increased mucus effusion into the tympanic cavity of the ear in OM patients limits the movement of the eardrum and middle ear bones and causes hearing issues [2]. It has been demonstrated that the degree of hearing loss is correlated with a higher mucin concentration in the middle ear effusion. Although the host's intrinsic defence mechanism against middle ear infections includes the production of mucin, too much mucin can compromise mucociliary clearance and cause conductive hearing loss. As a result, mucin expression needs to be strictly regulated [3].

In the middle ear mucosa, otitis media (OM) is characterised by the formation of mucins. Every time there is inflammation in the middle ear cavity, there are a lot of mucous cells (also known as "goblet cells") in the infected middle ear mucosa. An ENT clinical practitioner would typically observe mucus and pus in the middle ear cavity and/or the external canal if the ear drum is perforated in a typical instance. Otolaryngologists frequently notice a build-up of sticky mucus or slimy substance, resembling a rubble band, in the middle ear cavity in a typical instance of chronic OM. Similar circumstances can be observed in chronic mastoiditis [4].

An extensive spectrum of pharmacological actions, including antioxidant, anticancer, anti-inflammatory, antibacterial, and ant diabetic activities, are said to be present in the yellow pigment curcumin, which is extracted from the Curcuma longa rhizome. Due to curcumin's lack of dose-limiting toxicity, prolonged use is possible with little side effects. The United States Food and Drug Administration has given curcumin its "generally regarded as safe" (GRAS) designation. Despite having pleiotropic benefits on a wide range of illnesses, curcumin's use is severely constrained by its low absorption. In a mouse model of OM, we recently discovered that curcumin has an inhibitory effect on NTHi-induced neutrophil recruitment. Curcumin's impact on controlling MUC5AC mucin, a significant cause of OM disease, has not yet been determined [5].

In this study, we show that curcumin reduces middle ear epithelial cells' ability to produce MUC5AC when NTHi is presenting both in vitro and in vivo. Through the suppression of p38 MAPK and the stimulation of the negative regulator MKP-1, curcumin reduced MUC5AC expression. As a result, by inhibiting the overproduction of MUC5AC mucin, our findings supports the claim that curcumin

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has anti-inflammatory properties and can be used to treat NTHi-induced OM. Growing evidence points to the significance of biofilms in otolaryngologic infections. The majority of the research on this topic now focuses on in vitro studies, with the majority of the issues concerning medical implants [6]. Numerous recent publications have demonstrated the occurrence of biofilms on the tonsils' and adenoids' mucosal surfaces. Additionally, biofilm has been shown in otitis media with effusion, direct middle ear mucosa biopsy specimens, and a nonhuman primate model of chronic otitis media. Standard culture methods almost never pick up biofilms because they cannot explain their intricate, three-dimensional characteristics. The ability to detect and identify bacteria has been made possible by molecular diagnostics based on nucleic acid upon amplification methods, and different imaging technologies have enabled researchers with insights into the significance of biofilms in human infections. A technology with high resolution that offers ultrastructure investigation of biofilms is scanning electron microscopy (SEM). Our goal is to investigate if cholesteatomas or chronic otitis media in patients are associated with biofilm [7].

Materials and Methods

We solicited participation from patients undergoing surgical procedures for our study. The Eskisehir Osmangazi University Faculty of Medicine's ethical committee gave the study the thumbs up. The tissue samples were taken from 34 patients receiving normal surgical care at the Eskisehir Osmangazi University Medical Faculty between October 2011 and May 2012. There were 18 men and 16 women among these patients. Chronic suppurative otitis media (CSOM) (30 specimens), chronic non suppurative otitis media (CNSOM) (33 specimens), and chronic otitis media with cholesteatoma were the three categories into which the chronic otitis media (COM) patients were categorised (39 specimens) [8]. The middle ear mucosa, mastoid tissue, and ossicle of the patients in each group were also taken for tissue samples. The middle ear mucosa was also categorised as normal, hypertrophic, or granulated tissue with accompanying mucosa throughout the procedure. Only when the debridement of the tissue was required during the surgical procedure was tissue extracted. A biofilm formation assessment was performed on each eroded ossicle that couldn't be repaired.

A generic classification algorithm is composed of two steps: the extraction of numerical features intended to distinguish across classes and the subsequent classification using these features. To lessen the impact of image artefacts, we include a pre-processing step before feature extraction in the otitis media classifier [9].

As a prefixation phase, the tissue samples were immediately immersed in 2.5% glutaraldehyde for 24 hours at 4°C (prepared in 0.1 M phosphate buffer, pH 7.4). They were then post fixed with 1% osmium tetroxide for 1 hour at room temperature, rinsed twice with 0.1 M phosphate buffer (pH 7.4), and finally rinsed with distilled water. The specimens were then dehydrated for 30 minutes with absolute alcohol after 15 minutes each with progressive percentages of ethyl alcohol (30%, 50%, 70%, 90%, and 96%). The critical point drier Polaron CPD 7501 Critical Point Dryer was used to dry the specimen (VG. Microtech, East Sussex, UK). Carbon conductive paint was employed for mounting, and Polaron SC7620 Sputter Coater was used to coat the specimens in gold. Finally, a JEOL scanning electron microscope was used to analyse each specimen (JEOL JSM-5600LV). Each sample's various parts were methodically examined. The presence of three elements—bacteria-sized and -shaped particles, an amorphous substance that is consistent with glycocalyx surrounding the bacteria, and surface binding-was required for a sample to be classified as having a biofilm [10].

In samples that tested positive for biofilm, scanning electron microscopy showed that the distribution of bacterial micro colonies was not uniform across the tissue surface. Occasionally, in samples that at low magnifications looked to be negative, a biofilm was discovered as the magnification was increased. In certain regions, extracellular material was seen connecting the bacteria. As the magnification grew, however, samples that first looked to be positive also revealed a rough surface structure of the tissue or erythrocytes, indicating that the sample was biofilm negative.

The dependent variable was otitis media with effusion, whereas the independent variable was laryngopharyngeal reflux. Calculating the reflux symptom index (RSI) and reflux finding score allowed researchers to determine the laryngopharyngeal reflux variable (RFS). When RSI > 13 and RFS > 7, laryngopharyngeal reflux was deemed to be present. Non-LPR patients are defined as subjects having RSI scores of less than 13 and RFS scores of less than 7 [11]. Using ear complaints, pneumatic otoscopy, and tympanometry tests, otitis media with effusion was identified. The diagnosis of otitis media with effusion was made if one or more of the following ear complaints were present: intermittent mild ear pain, fullness in the ear, tinnitus, and hearing loss in one or both ears; and minimal or obstructed tympanic membrane movement as determined by otoscopy pneumatic and/or tympanogram B performed during tympanometry investigation. In order to obtain the prevalence ratio, the prevalence of OME in the LPR group was divided by that of OME in the non-LPR group [12].

Discussion

The middle ear mucosa had much more biofilms than the mastoid and ossicle samples, according to the findings of our study. This is probably because it is situated close to the external auditory canal. We also found that biofilm growth was less frequent in the ossicle samples. The ossicles are known to hang suspended within the tympanic cavity and to have a generally subpar immune response. In fact, because of this, we anticipated that this area would have a higher biofilm rate. Ossicles, on the other hand, were the areas in our study where the least biofilm was found. On the ossicle surfaces, we did not notice a disturbance caused by infection. This ailment could also aid in preventing [13].

The granulated tissue may develop as a result of bacterially driven middle ear inflammation or as a subsequent reaction to microbial biofilm adherence to alloplastic materials including tympanostomy tubes and partial or total ossicular replacement prostheses. Recurrent infections or hypertrophy, according to Chole and Faddis, may increase the likelihood that the germs are hidden from the host's defences. In addition, numerous, occasionally resistant bacteria are regarded to be the primary cause of hypertrophy. In our investigation, we also found that tissue samples that were hypertrophic and granulated had greater biofilm rates than samples of normal mucosa [14].

The absence of a control group is a drawback of the current investigation. The ethical challenge of obtaining tissue from a suitable control group is that it should be made up of tissue from age-matched control people who have never experienced an infection of the upper airways. Therefore, it was not possible to include controls in our study. We found certain issues with employing SEM, despite the fact that it has been frequently utilised by researchers to identify and describe biofilms. For instance, it was challenging to survey the entire specimen for the presence of biofilms, despite the fact that our sample size was too small. Occasionally, these areas could not be thoroughly studied due to the rugged topographic structure of the surface or the crypts. more recent methods, such confocal laser scanning microscopy [15].

Conclusions

Studies of pulmonary mucous cell metaplasia and intestinal epithelial homeostasis have made tremendous strides during the past ten years. In the earlier investigations, Atoh1 was identified as a factor that caused goblet cells to differentiate, whereas in the later studies, SPDEF was identified as a factor that caused mucous cell metaplasia. Now that we understand mucous cells at the molecular level, we can use this knowledge to treat otitis media. The response of mucous cell metaplasia to infections is extremely rudimentary. Initially, this response involves secreting additional mucins and their chaperones in order to defend the mucociliary system. These released compounds are intended to lubricate the respiratory tract's lumen and expel dangerous items. However, ciliated cells suffer as a result of mucous cell metaplasia's disruption of the efficient mucociliary transport mechanism.

Finally, our findings are consistent with the concept that biofilms play a role in CSOM, cholesteatoma, and, to a lesser extent, CNSOM. Topical or systemic antimicrobials must be used carefully in this case. Surgery is the initial option, and during surgery, normal tissue must be carefully separated from hypertrophic tissue. After the operation, there are several potential causes for failure. Remaining biofilms may be a factor in the surgery's failure if the tissue that has the capacity to host them, such as granulated tissue, cannot be cleansed thoroughly.

Acknowledgement

None

Conflict of Interest

None

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