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Review Article

Anatomy and physiology of the palatine tonsils, adenoids, and lingual tonsils

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Abstract *Objective:* This review aims to discuss the basic anatomy and physiology of the palatine and pharyngeal tonsils, with reference to how this foundational understanding may affect patient management and surgical procedures in these regions of the upper airway.
Methods: A literature search was performed using PubMed and Google Scholar using the MeSH terms tonsils, adenoids, anatomy, physiology, and adenotonsillectomy. Primary sources were excluded if they were abstracts only, non-English language, or non-human studies. Thirty-five sources were included in this review.
Results and conclusions: The pharyngeal and palatine tonsils are compact yet physiologically complex mucosa-associated lymphoid tissues that make up a portion of Waldeyer's ring. As part of the mucosal immune system, these structures function in exogenous antigen sampling and stimulation of immune responses. Aberrant immune activation and/or regulation can lead to a myriad of pathologies, with adenotonsillar hypertrophy, chronic tonsillitis/adenoiditis, and recurrent otitis media among the most commonly encountered conditions by otolaryngologists. While the pathophysiology of these conditions is still incompletely understood, current evidence and future investigations may reveal patterns amenable to targeted medical management. When medical management fails, tonsillectomy and/or adenoidectomy may be indicated for patient care. Though routine procedures, the execution of tonsil and/or adenoid removal requires a thorough understanding of the anatomy of these lymphoepithelial organs so as to minimize the risk for rare serious complications that can occur.

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Introduction

Tonsillectomy and adenoidectomy are among the most common surgical procedures in the United States and around the world.[1] Historically, tonsil surgery was limited to partial excision for fear of removing healthy tissue.[2] Credit for the first complete tonsillectomy is given to Celsus in the 1st century AD.[3] Over time, as popularity grew for the procedure, partial excision continued to be considered the safest and best method for excision. By the 19th century, superior lighting, ability to control hemostasis, and anesthetic techniques made complete excision possible.

Today, surveys indicate that complete excision via electrocautery is the preferred method for excision around the world thought many other techniques have been carried out successfully.[4] An understanding of the anatomy and physiology of these mucosa-associated lymphoid tissues is paramount to understanding their diseased states as well as implications for surgical extirpation.

The palatine tonsils, adenoids, tubal tonsils, and lingual tonsils are lymphoepithelial tissues that make up the components of Waldeyer's ring, named after the German anatomist Heinrich Wilhelm Gottfried von Waldeyer-Hartz. These entities are together a part of the mucosal immune system. Their primary function is to participate in the secondary immune system by immunologically sampling antigens and local pathogens.[5] They are strategically located at the junction of the respiratory and digestive tracts to be able to serve as a site for continued lymphoid stimulation. The purpose of this review is to describe the anatomy and physiology of Waldeyer's ring and how it pertains to surgical planning and subsequent patient management.[5]

Discussion

Waldeyer's ring consists of non-contiguous mucosal-associated lymphoid tissues (MALT) arranged in a circumferential configuration in the nasopharynx and oropharynx.[6] Positioned at the common entry of the respiratory and alimentary tracts, Waldeyer's ring plays a unique role in screening and filtering exogenous antigens, as well as in the initiation and maintenance of immune responses. Waldeyer's ring includes the following four structures: (1) the pharyngeal tonsil, located at the midline along the roof and posterior wall of the nasopharynx; (2) the tubal tonsils (also known as Gerlach tonsils), located on either lateral nasopharyngeal wall immediately posterior to the Eustachian tube orifice and in close association with the torus tubarius; (3) the palatine tonsils, located along each lateral oropharyngeal wall between the anterior and posterior tonsillar pillars; (4) the lingual tonsils, located along the base of tongue and contiguous with the palatine tonsils at the glossotonsillar sulcus.[6,7] As the palatine tonsils and pharyngeal tonsil are of greatest interest to the pediatric otolaryngologist in clinical practice, the remainder of this review will focus on these two components of Waldeyer's ring.

Surgical anatomy of the palatine tonsil

The palatine tonsil is a lymphoid structure housed within the tonsillar fossa, which is bordered anteriorly and posteriorly by mucosal folds (commonly referred to as the anterior and posterior tonsil pillars) comprising the palatoglossus and palatopharyngeus muscles, respectively. The superior constrictor muscle forms the lateral border of the tonsillar fossa. Deep to this muscle is a layer of loose connective tissue and the buccopharyngeal fascia, which is the final boundary between the tonsil and the parapharyngeal space.[7]

Numerous branches of the external carotid artery system penetrate through the superior pharyngeal constrictor muscle to perfuse the tonsil. The inferior pole is supplied primarily by the tonsillar artery, a branch of the facial artery. Additional blood supply to the inferior pole comes from the dorsal lingual branches of the lingual artery. The ascending palatine artery, another branch of the facial artery, divides distally into two branches, one of which also supplies the palatine tonsil. The superior pole of the palatine tonsil is supplied primarily by tonsillar branches of the ascending pharyngeal artery and by the descending palatine artery, a branch of the internal maxillary artery. Venous drainage occurs via the paratonsillar vein, which eventually joins the pharyngeal venous plexus and the common facial vein. Lymphatic drainage basins include the jugulodigastric and, occasionally, retropharyngeal lymph nodes. Sensory input to the palatine tonsil and tonsillar fossa is carried by the lesser palatine nerve from the second division of the trigeminal nerve (V2) and by tonsillar branches from the glossopharyngeal nerve. Because the glossopharyngeal nerve also provides sensation to the middle ear via the tympanic nerve branch, patients with tonsillar disease or recent tonsillar procedures often complain of referred otalgia.[7,8]

When performing procedures involving the palatine tonsil, such as tonsillectomy or drainage of peritonsillar abscesses, providers must remain cognizant of important nearby neurovascular structures. The internal carotid artery runs through the post-styloid parapharyngeal space. While infrequent, injury can occur, particularly in cases of tortuous or medialized carotid arteries.[9] The artery typically lies 2.5 cm posterolateral to the tonsillar fossa in adults,[7] but can lie within 1.5 cm of the fossa in children under 11 kg.[9]

Perhaps more frequently applicable to clinical practice is the position of the glossopharyngeal nerve. This nerve

travels deep to the stylopharyngeus muscle and then traverses between the superior and middle pharyngeal constrictor muscles before giving off a lingual branch, which enters the tongue base near its junction with the palatoglossus muscle and supplies taste sensation to the posterior 1/3 of the tongue.[7] Cadaveric dissections have shown that the lingual branch can pass within as little as 5.5 mm of the posteroinferior border of the tonsillar fossa and may be tightly adherent to the tonsillar fossae in up to 1/5 cadaveric specimens.[10,11] Due to this anatomical relationship, the nerve is susceptible to injury during tonsillectomy, as implicated in reports of dysgeusia following this procedure.[11–15]

Surgical anatomy of the pharyngeal tonsil

The pharyngeal tonsil (termed "adenoid") is an aggregate of lymphoid tissue lying in the midline along the roof and posterior wall of the nasopharynx, at the level of the sphenoid and occipital bones. The pharyngobasilar fascia lies deep to the inferior edge of the pharyngeal tonsil, and then continues more inferiorly as the pharyngeal constrictor muscles. Though uncommon, Grisel's syndrome, or non-traumatic atlanto-axial subluxation, has been observed following adenoidectomy. Laxity of the ligaments surrounding this cervical joint is thought to occur as a result of inflammation, which may occur from aggressive adenoidectomy and/or hyperextension in a patient with anatomical risk factors.[16]

The pharyngeal tonsil's blood supply includes the ascending pharyngeal artery, along with contributions from a branch of the ascending palatine artery, the tonsillar branch of the facial artery, the pharyngeal branch of the internal maxillary artery, and the artery of the pterygoid canal (a branch of the maxillary artery or in some cases the internal carotid artery). Venous drainage passes from the external pharyngeal venous plexuses to the paratonsillar veins, and eventually to the facial or internal jugular veins. Lymphatic drainage is to the retropharyngeal and pharyngomaxillary lymph nodes.[17]

Physiology of the palatine & pharyngeal tonsil

To better understand the physiology of the palatine and pharyngeal tonsils, a brief embryologic review is necessary. The medial epithelial surface of the tonsil forms from the second branchial pouch, as solid epithelial cores invaginate into the surrounding mesenchyme. These cores eventually canalize and form crypts. Around week 16–17 of embryological development, lymphocytes and lymphoid stem cells invade the deeper lamina propria and begin to form follicles and what will eventually become germinal centers. As these lymphoid elements grow, the deepest layers of the lamina propria eventually coalesce into a thin membrane that forms the tonsillar capsule. More superficial connective tissue fibers, primarily consisting of type III collagen, form septae that traverse between the crypts and become continuous with the deeper capsule.[18,19]

In postnatal life, the branching crypts, totaling approximately 10–30 per tonsil, give the tonsils a "pitted" appearance on their medial free edge. The crypts resemble tubular diverticula and have a fibrovascular core surrounded by lymphoid tissue and the epithelial surface is comprised of non-keratinized stratified squamous epithelium along the medial (luminal) surface of the tonsil. The crypts themselves are lined by a non-uniform distribution of stratified squamous epithelium and reticulated crypt epithelium. This latter epithelium, also referred to as lymphoepithelium, is similar to Peyer's patches found in the gastrointestinal tract. Reticulated epithelium is less orderly than stratified squamous epithelium and contains both epithelial and non-epithelial cells, particularly lymphoid cells. This epithelial layer can be quite thin and even lack a basement membrane in some regions. With lymphocytes and dendritic cells just deep to the epithelial surface, this histologic arrangement allows for rapid transport and presentation of exogenous antigens to the lymphoid cells for efficient initiation of an immune response. Furthermore, the invaginated structure of the tonsillar crypts significantly increases the total surface area that can participate in this process of antigen sampling and can facilitate direct trapping of foreign material entering the oropharynx. This is of particular importance because the palatine tonsils do not have an afferent lymphatic network as other lymphoid organs like lymph nodes and the spleen.[18,20]

The pharyngeal tonsil has some similarities to the palatine tonsils in gross and histologic appearance. The free surface of the pharyngeal tonsil is characterized by mucosal folds that project anteriorly and laterally, with a much smaller number of crypts as compared to the palatine tonsils. Histologically, the pharyngeal tonsil is composed primarily of pseudostratified ciliated columnar epithelium, with lymphoid follicles, fewer in number than the palatine tonsil, arranged throughout the mucosal folds. Superiorly, a capsule separates the pharyngeal tonsil from the periosteum of the sphenoid and bony occiput; and connective tissue septa extend from this capsule into the tissue of the pharyngeal tonsil, separating it into 4–6 segments.[18,20]

A complete discussion of the immunologic function of the palatine and pharyngeal tonsils is beyond the scope of this review and is detailed elsewhere.[20–23] Briefly, exogenous antigens are "sampled" through an incompletely understood process thought to involve M (membrane)-cells, also found in Peyer's patches, whose structure facilitates antigen uptake from the naso-/oropharynx. Once these antigens cross the epithelium of the luminal tonsillar surface, they are processed by antigen-presenting cells (APCs), such as dendritic cells and macrophages, and then presented to T cells and B cells in the neighboring extrafollicular region. If the antigen has been encountered previously, a secondary immune response is stimulated via T-cell proliferation and/or secondary antibody production by B cells.[21] If the encountered antigen is novel and successfully recognized by a helper T cell, activation, proliferation, and differentiation into a T-cell population specific to this antigen ensues, as long as appropriate co-stimulatory signals are present. These T cells stimulate naive B cells, which then travel to nearby follicles and differentiate into antigen-specific plasma cells and memory B cells, eventually forming a germinal center. These cells can then leave their respective lymphoid structures via high endothelial

venules and travel to other mucosal sites, such as the nasal mucosa, where they receive further signals to terminally differentiate into specific immunoglobulin-producing cells, with predominant production of IgG and IgA. Immunoglobulins are directly secreted by the pharyngeal tonsil and extravasate between palatine tonsil epithelial cells to reach their respective tonsillar surfaces for immune surveillance, preventing antigen attachment to host tissues and/or stimulating immune-mediated destruction.[6,22,23]

Equally important to this immunologic response is apoptosis, or programmed cell death. This process helps to maintain homeostasis as the tonsils continually encounter new antigens, and also serves to eliminate autoreactive or non-specific immune cells within the organs. When functioning appropriately, apoptosis will minimize the risk for pathologies such as autoimmune disease, pathologic lymphoid organ hyperplasia, and/or decreased immune function due to decreased lymphocyte immunocompetence.[24]

Literature has questioned a possible downside to adenoidectomy with or without tonsillectomy in the long term effects of a patient's immune function. One population cohort study out of Denmark reported an increased risk of allergies, respiratory and/or infectious diseases later in life after undergoing adenoidectomy/adenotonsillectomy.[25] A study by Jalali et al[26] found a decrease in CD10 in pediatric patients after a tonsillectomy and theorized a decreased B cell and antibody production in postoperative patients. Other groups have suggested a temporary decrease in immune function during the immediate postoperative period with an eventual return to normal function. The idea of altered immunity after adenoidectomy/adenotonsillectomy is a difficult question to address due to the multitude of factors that play into one's immune system and long term health.

Clinical implications of tonsillar physiology

The immunologic function of the palatine and pharyngeal tonsils leads to their rapid growth during the early years of life. The exact mechanism of growth is not completely understood but is thought to occur as exogenous antigen presentation catalyzes germinal center development, lymphoid hyperplasia, and expansion of the tonsillar parenchyma. The pharyngeal and palatine tonsils typically reach their maximum size by age 6 and puberty, respectively. After this time, involution occurs via increased fibrous tissue production and eventually fatty atrophy, usually by 8–10 years of age and adulthood, respectively.[16]

As the palatine and pharyngeal tonsils undergo their initial rapid growth phase through antigen sampling, some individuals develop pathologic immune-related processes, such as chronic adenoiditis or tonsillitis, recurrent otitis media, rhinosinusitis, and even allergic disease.[27] Other individuals develop complications associated with anatomic obstruction of the oropharynx and nasopharynx, such as sleep-disordered breathing/obstructive sleep apnea (OSA) from adenotonsillar hypertrophy and nasal obstruction, rhinosinusitis, and recurrent otitis media from adenoid hypertrophy and choanal and/or Eustachian tube obstruction.

The mechanisms underlying these pathologies, particularly adenotonsillar hypertrophy, are not fully understood, with theories involving genetic predisposition, infections, environmental exposures, and aberrant immune responses.[24,28] Given the immunologic function of Waldeyer's ring, a number of groups have focused their attention on investigating immunologic phenomena that may underlie these processes. While the pharyngeal and palatine tonsils can protect from foreign pathogens, current evidence suggests that tonsillitis or adenoiditis occurs when foreign antigens escape immune defenses, become trapped in crypts, and proliferate before the immune system can mount a sufficient response.[21,22] In cases of recurrent infection, current evidence suggests that changes in the reticulated epithelium can impair efficient antigen uptake, while immaturity of antigen-presenting cells can lead to decreased capability in effectively activating the immune response, sometimes referred to as local immunosuppression.[22,29] This finding has been further supported by Chen et al, whose group showed that in tonsillar specimens from patients with recurrent tonsillitis, cellular senescence was observed in the epithelium (predominantly among macrophages) and interfollicular area (predominantly among T-cells).[28] In comparison, cellular senescence predominated in the germinal centers (predominantly among macrophages) and interfollicular area in patients with tonsillar hypertrophy. Cellular senescence suggests an impaired immune response that can lead to impaired phagocytosis and pathogen killing, as well overgrowth of follicles due to lymphoid cell accumulation.[28]

In addition to impaired cell function, biofilms are felt to promote pathogen survival and to further impair the immune function of the pharyngeal and palatine tonsils.[30,31] Though the most prevalent inflammatory cytokines and bacterial organisms associated with specific pathologies is quite variable within the literature, most agree that, regardless of the specific bacterial organisms, a polymicrobial microbiome occurs in the setting of chronic infectious processes.[32–37]

The current indications for tonsillectomy and adenoidectomy are outside the scope of this article, but these indications, as well as general management of pediatric patients with the above clinical pathologies, can often be found in clinical practice guidelines published by national otolaryngology societies. Though these procedures are brief and routinely performed by otolaryngologists, they can carry significant risks based on the underlying anatomy of the relevant lymphoid organs, as outlined above. Providers should have a strong foundational understanding of this anatomy when performing these procedures. A basic understanding of the physiology of Waldeyer's ring will also help providers to better understand and counsel their patients on medical management of adenotonsillar pathologies, which is often required prior to surgical intervention, as well as to approach the care of patients who remain refractory to standard care. In light of ongoing efforts to better understand many of these disease processes and the

rapid evolution of therapeutics in the current era of medicine, an understanding of adenotonsillar physiology will facilitate critical analysis of improved and/or targeted medical therapies that may arise in the future and become relevant to patient management.

Conclusions

Positioned at the junction of the respiratory and digestive systems, the lymphoid tissue of Waldeyer's ring is positioned to serve as a primary antigen sampling point. The compact anatomy and local physiology of this tissue are important to understand for any surgeon operating on this anatomic area. Further understanding of factors causing adenotonsillar hypertrophy and chronic adenotonsillitis are yet to be elucidated, but remain important in further understanding disease processes and surgical implications.

Declaration of competing of interest

None.

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