REVIEW ARTICLE

The spectrum of pathological findings of tonsils in children: A clinicopathological review

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Abstract

Tonsillectomy is among the most commonly performed operations in children. Although follicular lymphoid hyperplasia is usually the main and only pathologic finding at microscopic examination, a variety of other rare but important pathologic changes may be encountered. This review aims to provide an inclusive practical resource and reference for both training and practising pathologists. It discusses the spectrum of pathologic findings, including both neoplastic and non-neoplastic conditions and provides illustrative images.

Keywords: Tonsil, tonsillectomy, children, pathology

INTRODUCTION

In an ideal system, removed tonsils – as all surgically removed specimens - would benefit from pathologic examination. However, institutions are increasingly limiting or eliminating routine microscopic examination of tonsils as a cost saving measure. Aside from losing the opportunity to document findings that may be unexpected, other downsides of this trend include depriving practicing pathologists as well as trainees in the field of pathology of a valuable experience and diagnostic skill in tonsillar pathology. In this article, we discuss the spectrum of common and uncommon pathologic findings of tonsils with illustrative images.

1. Anatomy and Histology of Tonsils

The upper respiratory tract contains a significant amount of lymphoid tissue, most of which is arranged in the Waldeyer's ring. The latter consists of the palatine tonsils, lingual tonsils and pharyngeal tonsils (also referred to as the adenoids). The normal palatine tonsils consist of lymphoid tissue arranged in follicles, covered by squamous epithelium which extends to line deep crypts that penetrate each tonsil (Fig. 1A). Tonsillar tissue also includes minor mucous salivary glands along with a rich network of blood and lymphatic vessels (Fig. 1 B & C). The lingual and pharyngeal tonsils consist of loose submucosal collections of similar lymphoid tissue; however, no crypts are present. The overlying epithelium of the pharyngeal tonsils may be of respiratory or non-keratinising squamous epithelium type. The lingual tonsils are covered by non-keratinising squamous epithelium.

In the palatine tonsils, the squamous epithelium becomes thinner and loosely textured in crypts.[1] Ultra-structural studies have shown small holes in the epithelium of crypts,[2] perhaps to facilitate maximal infiltration of lymphocytes in the context of an inflammatory reaction (Fig. 1D). The tonsillar lymphoid follicles are similar to those in lymph nodes, with the observation that marginal zones are usually obscured except in florid follicular hyperplasia. The lymphoid cells infiltrating the epithelium of the crypts appear to be of marginal zone B-cell type, while most of those in surface epithelium are of T-cell type.[1]

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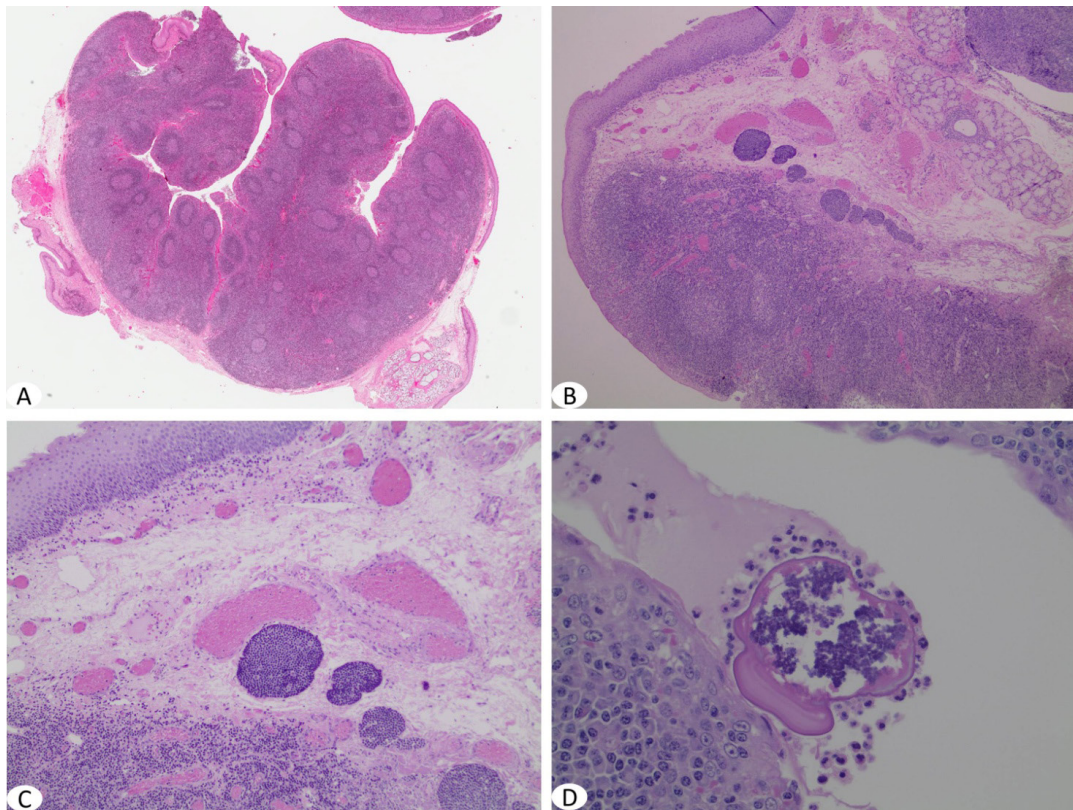
FIG. 1: Normal palatine tonsil (A), demonstrating lymphoid follicles, blood vessels, lymphatic vessels & minor salivary glands (B&C) and superficial inflammation in tonsillar crypt with neutrophils surrounding a droplet of bacteria containing saliva (D).

2. Clinical and Surgical Aspects

Tonsillectomy is among the most commonly performed operations in children, with more than half a million adenotonsillectomy procedures performed annually in the United States.[3] The two most common indications are throat infections and sleep-related breathing disorders.[3-5] The absolute indications for tonsillectomy include obstruction of the nasopharyngeal or oropharyngeal airways, interference with swallowing, clinical suspicion of malignant tumour of the tonsils, and uncontrollable haemorrhage from tonsillar blood vessels.[6] The American Academy of Otolaryngology-Head and Neck Surgery clinical practice guidelines list the following indications for adenotonsillectomy in children.[3,4]

1- Sleep disordered breathing.
2- Recurrent throat infections.
3- Dysphagia or voice quality changes related to enlarged tonsils.
4- Periodic fever, aphthous stomatitis, pharyngitis, and cervical adenitis.
5- Peritonsillar abscess in children with other indications for adenotonsillectomy.
6- Halitosis.
7- Chronic tonsillitis unresponsive to antimicrobials.
8- Tumour or haemorrhage of tonsils.
9- Paediatric autoimmune neuropsychiatric disorder associated with streptococci.
10- Chronic group-A streptococcus carrier status.

In the United States, the number of tonsillectomies has declined progressively since the 1970s.[6,7] Reports suggested that the decline was mainly in tonsillectomies performed for infectious indications, while the number of tonsillectomies performed for obstructive indications may have actually increased.[6,8,9] Most of these operations are performed as ambulatory, same-day procedures.[6,7] Tonsillectomy is performed infrequently in children younger than 3 years of age. Contraindications for tonsillectomy include 3 general categories: velopharyngeal insufficiency, haematologic disorders (anaemia and disorders of haemostasis), and active local infection.[6]

3. Pathologic Examination

Examination protocols

The approach to pathologic examination varies from one health care system to another, and may be different from one hospital to the other in the same system. Many hospitals perform histologic examination routinely on all specimens. Some use age as a criterion to identify the need for histologic study. There are published studies that attempted to include risk factors for malignancy to assist in making decisions about whether to perform histologic evaluation,[10] while some suggested clinical suspicion and specimen asymmetry as required criteria for performing a thorough histological examination.[11]

Advantages of routine microscopic examination

In an ideal system, all surgically removed specimens would benefit from pathologic examination. The advantages for routine pathologic examination include detection of incidental findings and providing tissue material for research, teaching, and other academic activities. Increasingly, institutions are limiting, or eliminating, certain specimens (including tonsils) from the list of those that qualify for routine microscopic examination. This trend is driven mainly by cost rationing.

4. Common Pathologic Findings

4.1 Follicular lymphoid hyperplasia (FLH)

Similar to pathologic findings in enlarged paediatric lymph nodes, the most common finding in removed tonsils is reactive follicular hyperplasia. Areas of active, mixed and chronic inflammation may be seen.

4.2 Active inflammation and peritonsillar abscesses

Areas of active and chronic inflammation are common and there may be areas of superficial ulceration. Peritonsillar abscesses, while rare and usually unilateral, are the most common complication of acute tonsillitis. In these cases, the acute infection of the tonsillar crypts may extend beyond the tonsillar capsule to involve the peritonsillar space, between the tonsil and the superior pharyngeal constrictor muscle (Fig. 2). If epithelial inflammation is extensive and ulcerations of squamous epithelium are prominent, the possibility of viral infections such as Epstein–Barr virus (EBV) or adenovirus
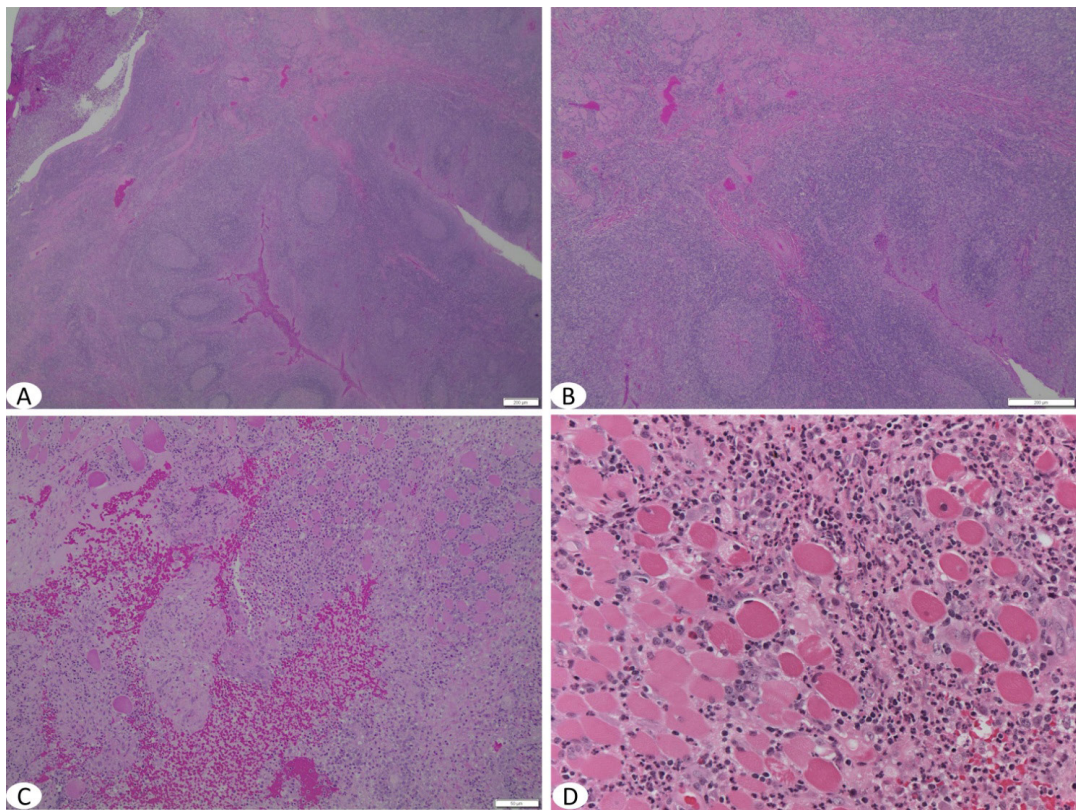

FIG. 2: An abscess deep at the base of a tonsil (A&B; scale bar = 200μm) and higher magnification showing neutrophilic infiltrates separating muscle fibers (C&D; scale bar- C = 50μm).

should be kept in mind.

4.3 Actinomyces

Actinomyces are gram positive, anaerobic, commensal bacteria, found in oral cavity, colon and vagina in humans. The incidence of Actinomyces of the palatine tonsil can be as high as 40%, as reported in a recent study.[12] It has been suggested that actinomycosis infection of the tonsil may indicate an aetiologic role for this organism in tonsillar and adenoidal hypertrophy.[13] On H&E section, the presence of actinomycosis can be recognized as aggregates of filamentous basophilic microorganisms arranged in a radial spoke-like fashion, the so called "ray-fungus" appearance of an Actinomyces colony (Fig. 3).

4.4 Keratin filled cysts

Squamous lined cysts are commonly found in the head and neck region. Dermoid and epidermoid cysts can rarely occur in the palatine tonsil. They arise from the epithelium that has been trapped in deeper tissue during embryonic period or from abnormal inclusion of cells during surgery or trauma.[14] More common are simple keratin filled cysts which vary in size from a few millimeters to more than 2 cm (Fig. 4).

5. Uncommon Pathologic Findings

5.1 Non-neoplastic
5.1.1 Viral infections

Bacterial and viral infections can cause tonsillitis. Streptococcal strains are the most common bacteria, while common viral causes of tonsillitis include adenoviruses, Epstein-Barr virus, human papilloma virus, influenza and parainfluenza viruses, and others such as enteroviruses, herpes simplex viruses. Microscopic examination of tonsils in most viral and bacterial infections shows hypertrophied and hyperplastic lymphoid follicles with excessive development of the germinative clear center as a reaction to the presence of antigens.

Certain viruses show specific histologic findings as follows:
Adenovirus
Viral intranuclear and intracytoplasmic inclusions with positive immunohistochemical
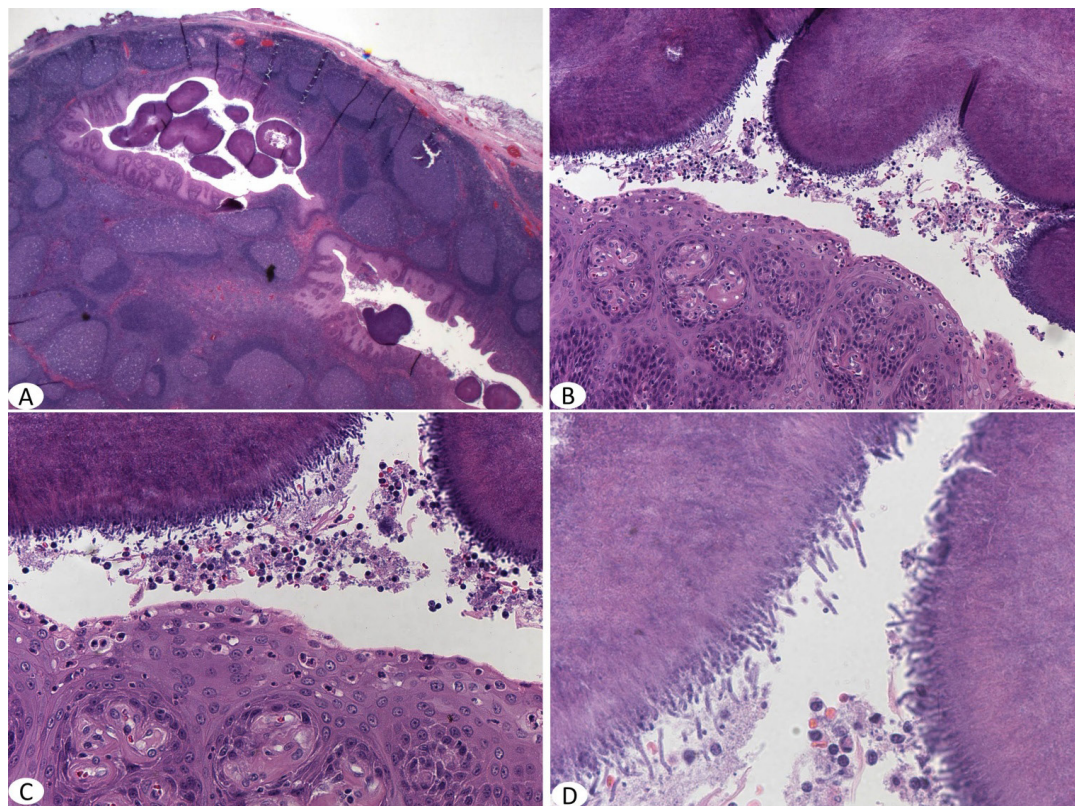
FIG. 3: Aggregates of Actinomyces bacteria in tonsillar pits (A&B) and higher magnification shows the filamentous bacteria arranged in radial spoke-like fashion (C&D).
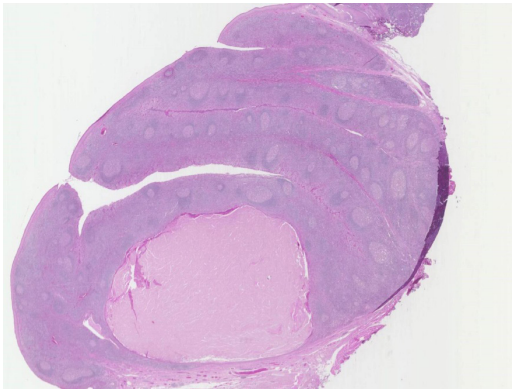
FIG. 4: A keratin cyst lined by squamous epithelium
and filled with keratin material

staining specific for adenovirus are noted. The intranuclear inclusions during late infection are surrounded by a clear halo, which may obstruct visualisation of the nuclear membrane, resulting in a smudged appearance. These "smudged" cells are classically seen in adenovirus infection. Adenovirus may persist in tonsils and adenoid as a latent infection[15] (Fig. 5).

EBV
Palatine tonsils of children can be colonised by EBV and this virus may be involved in the pathogenesis of recurrent tonsillitis and tonsillar hypertrophy. The microscopic appearence of tonsils infected with EBV is similar to histologic findings of lymph nodes in infectious mononucleosis, which range from non-specific follicular hyperplasia to proliferation of large immunoblastic cells of varying severity. Plasma cells and plasmacytoid cells are also admixed with immunoblasts and lymphocytes, giving a polymorphous appearance. Sometimes the immunoblasts form clusters, or even sheets, partially effacing the lymph node, which may be confused with lymphoma (Fig. 6).

HPV
There is little data about tonsillar HPV infection in the literature. In a study, the rate of HPV infection in non-neoplastic tonsils was 8.5% and in neoplastic tonsils was 51%.[16] HPV infection of the tonsils can show similar viral cytopathic effects seen in HPV infection of other body

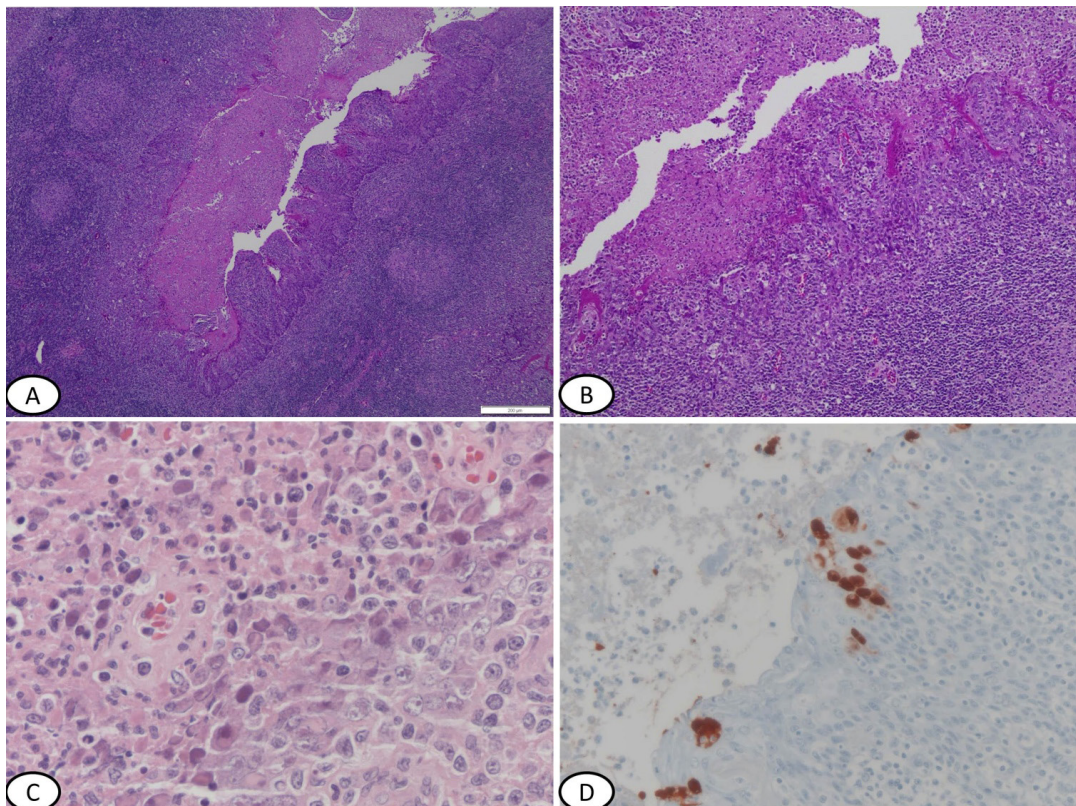
FIG. 5: Active inflammation and epithelial hyperplasia of tonsilar pit due to adenovirus infection (A&B) and
higher magnification shows the characteristic glassy nuclear inclusions in H&E (C) and that was stained
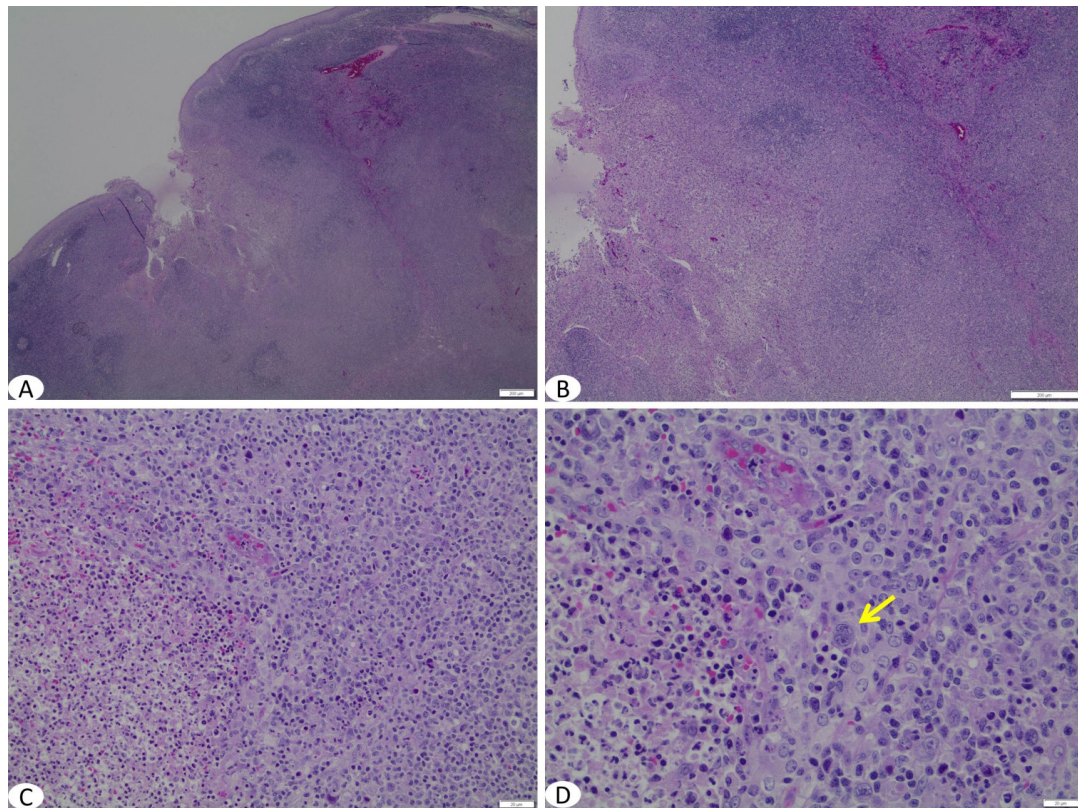with adenovirus immunoperoxidase stain (D). Scale bar = 200μm.

FIG. 6: Tonsil shows inflammation, superficial ulceration and expanded interfollicular T-cell zone typical of EBV infection (A&B; scale bar = 200µm) and at higher magnification there is mixed inflammation with atypical lymphocytes (C; scale bar = 20µm). Reactive atypical lymphoid cells may show binucleation or multinucleation with prominent nuclei that may be misconstrued as Reed-Sternberg cells (D; scale bar = 20µm) (yellow arrow).

sites (skin and cervix) in the form of extensive papillomatosis, hyperkeratosis, koilocytosis and prominent keratohayline globules (Fig. 7).

5.1.2 Granulomatous inflammation

Granulomatous inflammation of the tonsils and adenoids is uncommon.[17] The underlying disorders may be local or systemic. Tuberculosis, sarcoidosis, Crohn's disease, fungal infection and histoplasmosis have been reported to cause granulomas in the tonsils.[18-21] Granulomatous inflammation of the tonsil may occur secondary to foreign body material (like food particles) or ruptured inclusion cyst, and sometimes represents an exaggerated immune response to chronic tonsillitis. However, a careful work-up must be done to exclude any underlying systemic causes.

Tuberculosis
Primary tonsillar tuberculosis is rare and seen mostly in areas with high incidence of tuberculosis. Ziehl-Neelsen stain should be performed in the case of any tonsillar granuloma to exclude this possibility.

Cat-scratch disease
Cat-scratch disease (CSD) is a zoonotic infection caused by Bartonella henselae occurring in about 24000 patients annually in the United States. It typically presents with fever and regional lymphadenopathy and is considered as one of the most common causes of chronic lymphadenitis in paediatric population. It generally has a benign and self-limited course, though this is not always the case. Rare forms of extra-nodal involvement may occur such as involvement of the tonsils, the initial clinical features were indistinguishable from those of acute bacterial tonsillitis with jugulodigastric lymphadenopathy.[22]

Histologic examination may show focal areas of necrotising granulomatous inflammation, with extensive neutrophilic infiltrates seen in center of well developed granulomas. Early non-necrotising granulomas and multinucleated giant
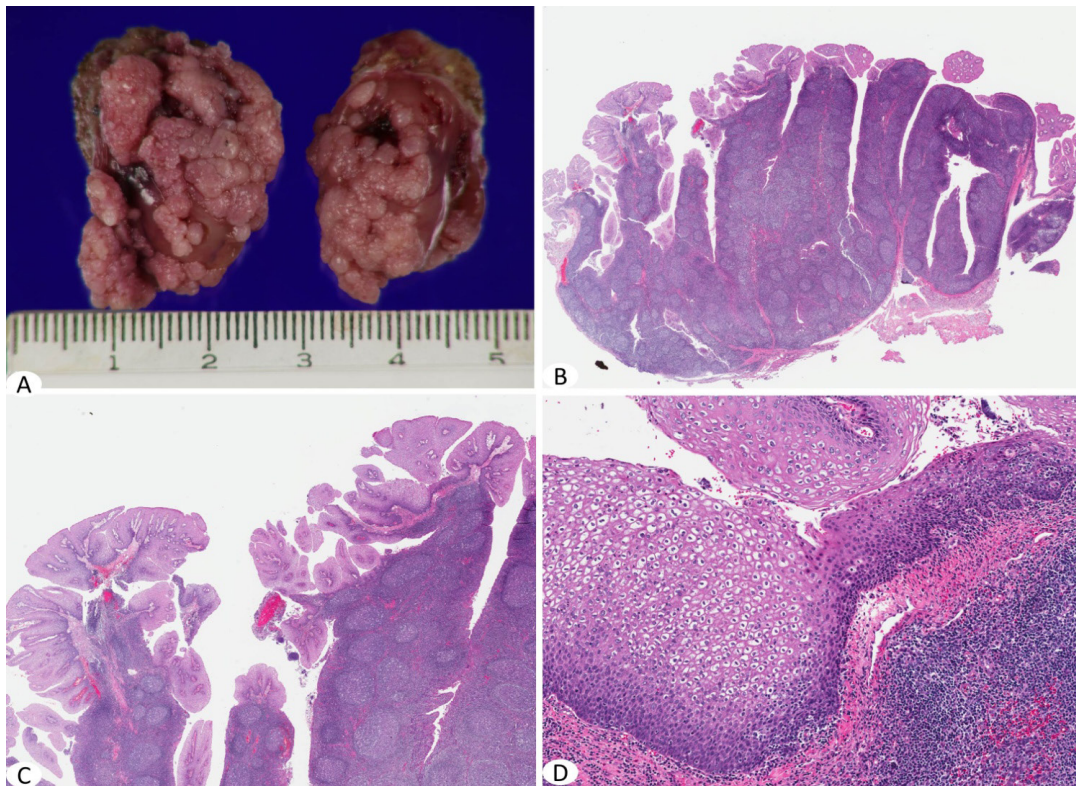
FIG. 7: Papillomatosis and hyperkeratosis of HPV infection of a tonsil (A, B and C). Higher magnification shows the characteristic koilocytosis of the squamous epithelium (D).

cells may also be seen (Fig. 8). The causative organisms are difficult to find even when Warthin-Starry stain is used; however additional ancillary techniques such as polymerase chain reaction (PCR) may be used for diagnosis[23] and are now available for paraffin embedded tissue.

Crohn's disease
A non-caseating epithelioid granuloma in the tonsil is a rare extraintestinal manifestation of Crohn's disease. The prevalence rate of oral involvement in Crohn's disease quoted in the literature ranges between 0.5 to 80.0%.[18,24] In addition to granulomatous inflammation, oral involvement can manifest as mucosal ulceration in different anatomic sites and the involvement may precede intestinal manifestation (Fig. 9).

5.1.3 Rosai Dorfman disease
Sinus histiocytosis with massive lymphadenopathy (Rosai Dorfman disease) is an uncommon benign condition characterised clinically by massive enlargement of cervical lymph nodes and histologically by lymphatic sinus dilatation due to histiocytic proliferation and mixed inflammation. The clinical course is variable. About 25% of cases have involvement of extranodal sites, especially the upper respiratory tract, salivary glands, orbit, testis and skin. Lesions at other sites may appear before the appearance of lymph nodes. Patients with extensive lung and liver involvement may not have an innocent course. There may be osseous involvement in paediatric age group. Other rare sites included solitary involvement of talus, triquetrum, sclera, conjunctiva, thymus, pleura, nasopharynx, genitourinary and central nervous system.[25]

It is a disease of unknown aetiology and the histological characteristics of the histiocytic cells are pale granular cytoplasm and round nuclei with distinct red central nucleoli. These cells also exhibit emperipolesis and are positive for S-100 and negative for CD1a (Fig. 10). [26-28]

An isolated form of Rosai Dorfman disease involving the tonsils with no associated lymphadenopathy or other organ involvement may be encountered.[29] Our literature search revealed seven reported cases of tonsillar involvement, combining those that included lymph node involvement in addition to cases of isolated tonsilar involvement of Rosai Dorfman
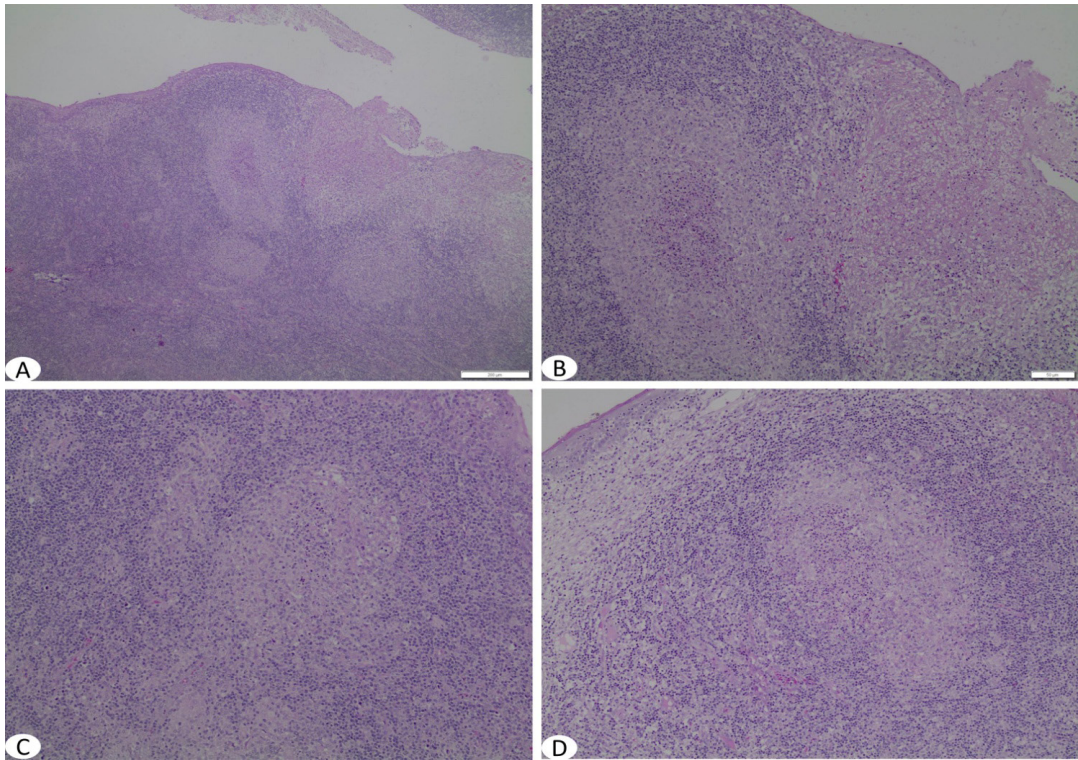
FIG. 8: Sections of a tonsil with cat-scratch infection demonstrating necrotising granulomatous inflammation (A&B; scale bar- A = 200µm, B = 50µm) with higher magnification showing neutrophils in the center of some granulomas (C&D).
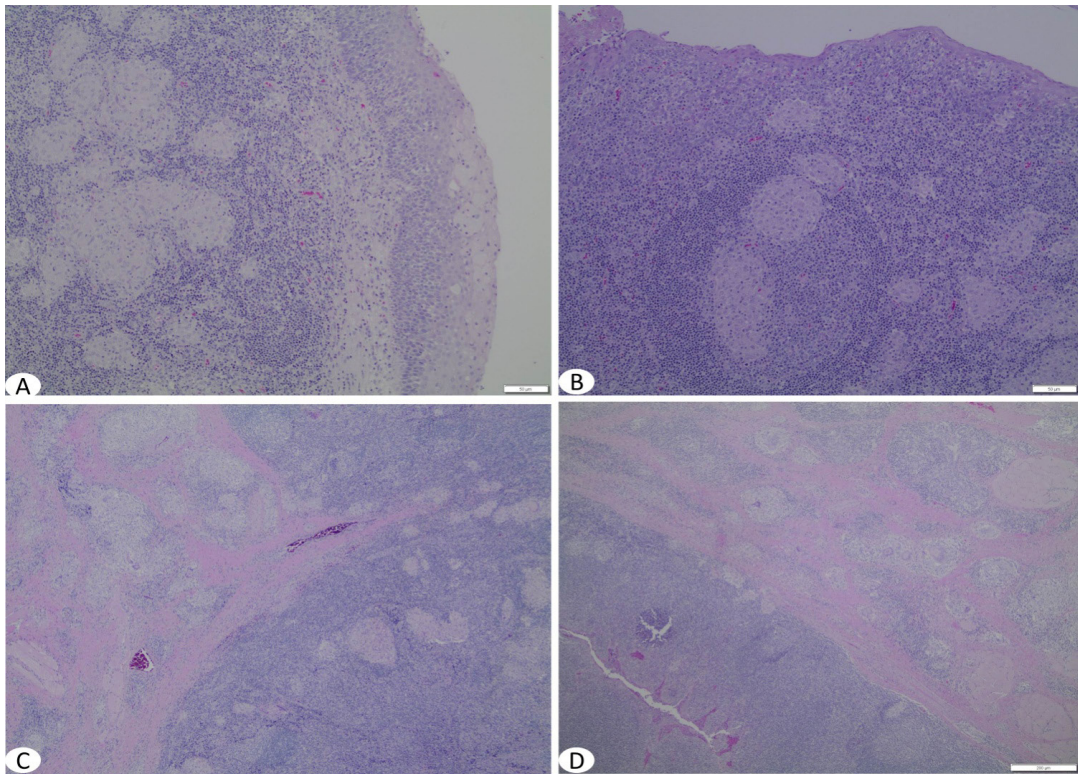

FIG. 9: Non-necrotising epithelioid granulomata in the tonsil of a child with Crohn's disease. (A&B, scale bar = 50µm; D, scale bar = 200µm)
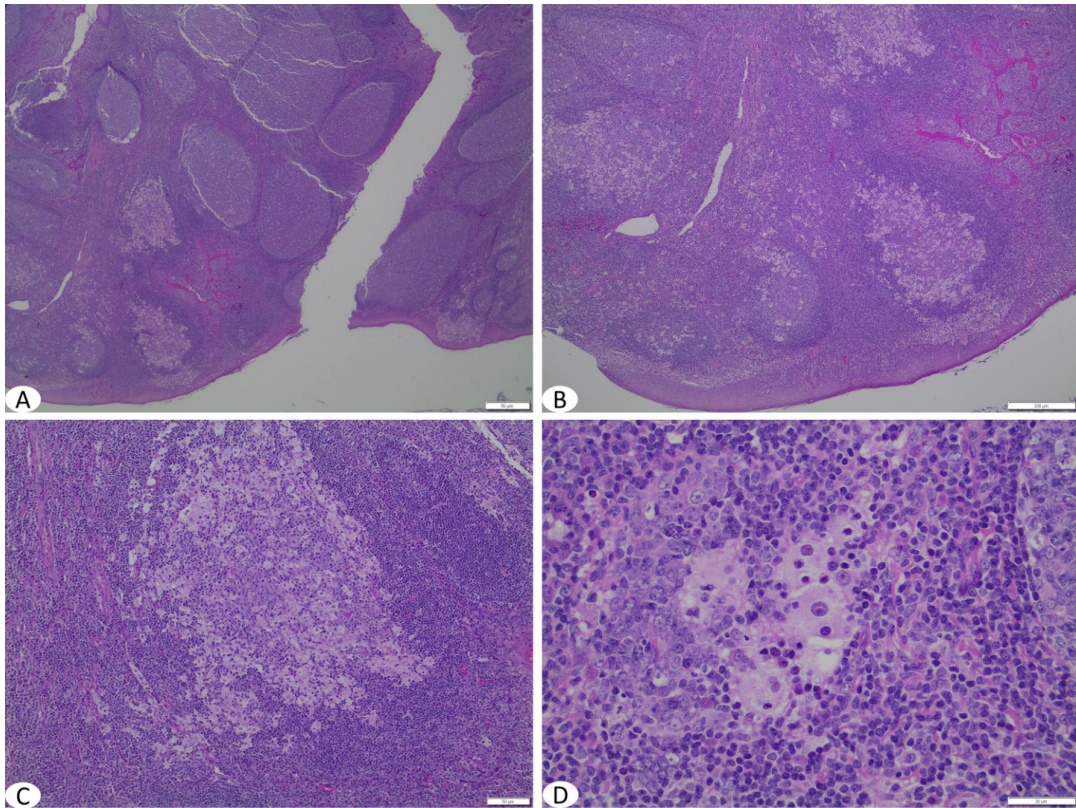
FIG. 10: Rosai-Dorfman disease of tonsil showing infiltrates of large histiocytic cells, mainly in germinal centers of lymphoid follicles (A, B & C). Higher magnification shows that individual histiocytes possess round centrally located nuclei, prominent nucleoli and exhibit abundant cytoplasm that contains lymphocytes or other inflammatory cells. (A&C, scale bar = 50μm, B, scale bar = 200μm, D, scale bar = 20μm)

disease. It involved a wide range of age, from 4 to 79 years old. Of the 5 cases which the age was recorded, three belong to paediatric age group. The male to female ratio is 5:2.[28-32]

Rosai-Dorfman's disease usually follows a self-limiting course that spontaneously resolves. However, a minority of cases may have immune dysfunction or may potentially relapse, hence it is important to diagnose and follow up these cases.

5.1.4 Storage diseases
Tangier disease
Tangier disease is a rare autosomal recessive lipid metabolism disorder caused by a mutation in the ABCA1 gene, located in chromosome 9q31. The ABCA1 gene encodes an ATP-binding cassette transporter that facilitates phospholipid and cholesterol transport. The mutation gives rise to a defect in cellular cholesterol removal, resulting in severe deficiency of plasma levels of high density lipoproteins and apolipoprotein A-1.[33] A defect in cellular cholesterol removal results in extensive accumulation of cholesterol esters in macrophages in peripheral tissues. Clinically, the pathognomonic finding, apart from an abnormal plasma lipoprotein profile, is the presence of large, hyperplastic, bright orange-yellow coloured tonsils and adenoids.[33, 34]

Microscopically, the tonsils and adenoids demonstrate a prominent accumulation of pale-staining, foamy histiocytes that contain lipid droplets and occasional crystalline material. The foamy histiocytes aggregate in clusters, often in the parafollicular areas.[34] Although there is no treatment for Tangier disease, this is an important diagnosis to recognise, as these patients have a high risk of premature coronary artery disease.[34,35]

Lipid storage disease
Lysosomal storage disease is a broad term used to describe a heterogeneous group of more than fifty inherited disorders that arise due to deficiencies or defects in lysosomal enzymes and other essential proteins that leads to the accumulation of undigested substrates. This

results in cellular dysfunction and a variety of clinical manifestations. The diseases are generally classified by the specific accumulated substrate.[36,37]

Lipid storage diseases are a subgroup of disorders with a lysosomal hydrolase deficiency that causes lysosomal accumulation of specific sphingolipid substrates. Examples include Gaucher disease, Niemann-Pick disease, Wolman disease and cholesterol ester storage disease. Abnormal storage of substrate can be found in many tissues including the reticuloendothelial system. Certain lipid storage diseases, including mucopolysaccharidoses, have been reported to present clinically as tonsillar hypertrophy.[38,39]

Histologic examination of affected tissues, including the tonsil, demonstrate aggregates and/or sheets of macrophages with round, uniform lipid inclusions that fill and distend the cytoplasm giving them a foamy appearance (Fig. 11). Although non-specific, identification and awareness of these histologic features can help support further biochemical and molecular testing that can lead to earlier diagnosis of certain storage diseases.[40,41]

5.1.5 Neoplastic
Tonsils are common site for various neoplastic lesions, both benign and malignant.

Squamous papilloma
Pedunculated squamous papillomas may arise from the soft palate, tonsil, or the epiglottis. Male to female ratio is 1:1.5 and the mean age was 33 years.[42] Study showed an established connection between HPV and the development of squamous papilloma.[43] It commonly arises from the tonsillar pillar, although it may arise from the surface of the tonsil itself. The stratified squamous lining layer in this area gives rise to this lesion. This lesion is purely an incidental finding usually, and rarely requires treatment. Histologically, tonsillar squamous papillomas are composed of well differentiated benign-appearing squamous epithelial papillae on thin fibrovascular stalks. The majority of these papillomas usually lack the viral associated koilocytic changes.

Lymphomas
Primary extranodal non-Hodgkin's lymphomas are the most common malignant tumours of the head and neck in the paediatric population.
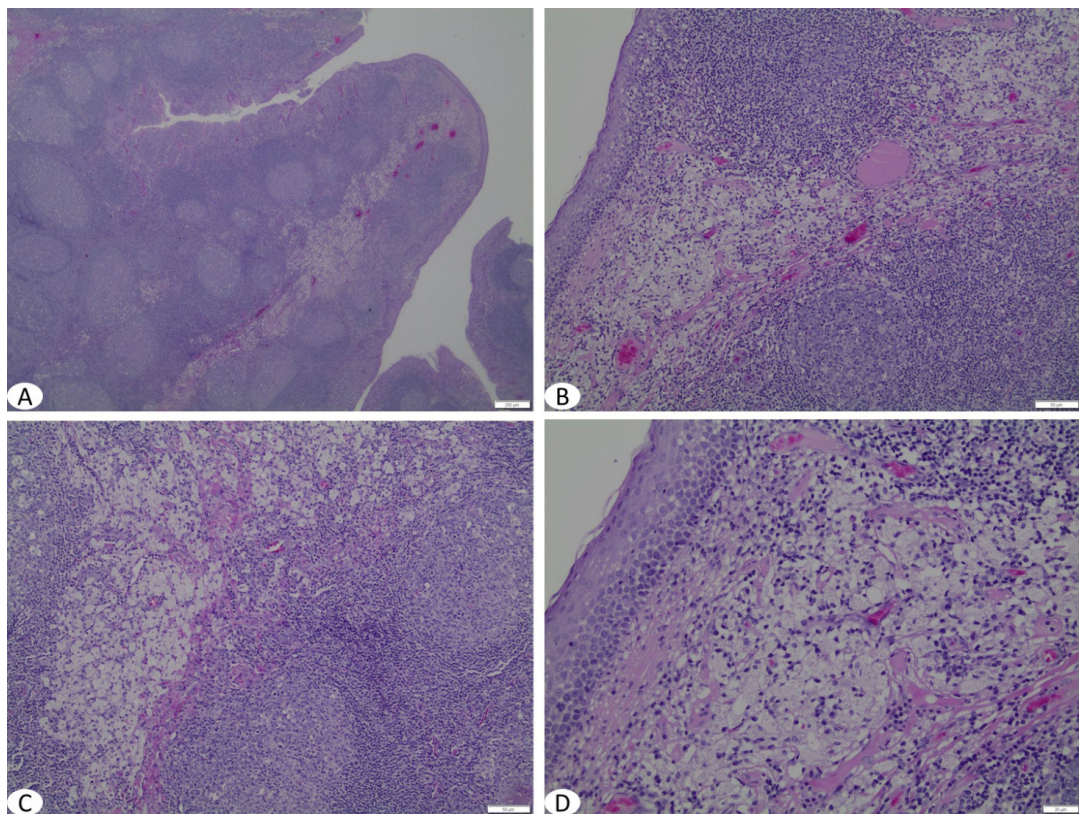

FIG. 11: Clusters of foamy histiocytes at the parafollicular and interfollicular regions of the tonsil of a child with a lipid storage disease (A, B, C &D). (Scale bar: A=200μm, B&C=50μm, D=20μm)

Primary tonsillar lymphoma comprises < 1% of head and neck cancer with non-Hodgkin lymphoma (NHL) being the most common type. Non-Hodgkin's lymphoma of the oral cavity and oropharynx accounts for 13% of all primary extranodal NHL with approximately 70% of these occurring in the tonsils.[44] The palatine tonsil is the most frequently involved site followed by palate, gingiva and tongue. Most lymphomas found in the palatine tonsils are of B-cell type.

A review of the literature showed that a majority of tonsillar lymphomas in the paediatric population were Burkitt lymphoma, with few cases of diffuse large B-cell lymphoma, follicular lymphoma and T-cell lymphoma (see Table 1). However in the adult population, diffuse large B-cell lymphoma is the most common type. Mohammadianpanah *et al*. found that in his series of 14 cases of NHL diagnosed over 10 years, that diffuse large B-cell lymphoma is the most common subtype, followed by small cell lymphoma, immunoblastic lymphoma, and anaplastic large cell lymphoma.[45] Similarly, Makepeace *et al*. also found diffuse large B-cell

lymphoma as the commonest type, occurring in 27 of the 51 cases of tonsillar lymphoma.[46]

Although the exact aetiology remains unclear, a number of predisposing factors have been identified, including human immunodeficiency virus and Epstein-Barr infection.[47]

Burkitt lymphoma
Burkitt lymphoma is an aggressive form of non-Hodgkin B-cell lymphoma mostly seen in childhood. There are three subtypes of Burkitt lymphoma: endemic (found in Africa), sporadic (found in North America and Europe) and immunodeficiency related. The endemic (African) subtype of Burkitt lymphoma is mostly seen between ages 5-7 and more than 50% of cases involve the maxilla or mandible. The sporadic (non-African) form of the disease may be seen in adults as well as children around 10-12 years of age. The sporadic form of the disease presents with abdominal, medullary or lymphatic involvement rather then head and neck involvement. Only 5% of the cases involve Waldeyer's ring as an initial presentation.

TABLE 1. Reported cases of tonsillar lymphoma in paediatric population

Authors	No of patients	Age (years)	Gender	Types of lymphoma
Amit et al. 2012 [53]	1	6	Male	Follicular lymphoma
Banthia et al. 2003 [54]	-	-	-	Burkitt lymphoma
Berkowitz et al. 1999 [55]	7	2 to 9	-	NHL
Booth et al. 2013 [56]	3	5	-	Burkitt lymphoma
		14	-	Follicular lymphoma
		15	-	DLBCL
Cianci et al. 2013 [57]	1	7	Male	Burkitt lymphoma
Dolev et al. 2008 [58]	6	Paediatric	-	-
Garavello et al. 2004 [59]	2	Paediatric	-	NHL
García-Ortega et al. 1999 [60]	-	-	-	Burkitt lymphoma
Guimarães et al. 2012 [44]	2	5	Female	DLBCL
		11	Female	DLBCL
Kraus et al. 1990 [61]	1	10	Male	Burkitt lymphoma
Meirelles et al. 1998 [62]	2	Paediatric	-	Burkitt lymphoma
Poulsen 1982 [63]	-	Paediatric	-	Burkitt lymphoma
Tewfik et al. 1996 [64]	-	Paediatric	-	Burkitt lymphoma
van Lierop et al. 2007 [65]	-	Paediatric	-	T-cell lymphoma
Williams et al. 2003 [11]	1	2	Male	Burkitt lymphoma
Zeglaoui et al. 2009 [66]	1	4	Male	NK/T-cell lymphoma

Palatine tonsil involvement is reported in 2.9% of head and neck Burkitt lymphoma.[48]

The histologic features of tonsillar involvement are similar to those in lymph nodes, showing a monotonous lymphoid proliferation composed of medium sized cells with round nuclei, finely clumped chromatin and multiple small basophilic nucleoli. The cytoplasm is deeply basophilic and usually contains lipid vacuoles. The tumour has an extremely high proliferation activity as evident by Ki-67 immunohistochemical Staining. A 'starry sky' pattern is present, which is imparted by numerous benign macrophages that ingested apoptotic tumour cells (Fig. 12).

Mucosa associated Lymphoid Tissue Lymphoma (MALT)

The gastrointestinal tract is the most frequent extra nodal location for lymphoma of mucosa-associated lymphoid tissue (MALT). MALT arising from the tonsil is rare. However if Waldeyer's ring is involved, the tonsil is the most commonly affected site. It is characterised by specific histological features and a remarkably indolent clinical course.[49,50]

The histological hallmark features of MALT are small atypical lymphoid cells with irregular nuclear membranes infiltrating lymphoid follicles and/or expanding marginal zones and infiltrating mucosa (Fig. 13). Immunophenotypically, MALT lymphomas have been reported to express CD21, CD35, CD43, CD20 and bcl-2 protein, with absence of markers such as CD10, CD5, and CD23 antigen, and the presence of light chain restriction. There is no specific marker for MALT.[51,52]

Non-haematopoietic small blue cell tumours

The tonsillar region can be the primary site for non-haematopoietic tumours of childhood, most notably rhabdomyosarcoma.[67,68] Radiologically, it may mimic peritonsillar cellulitis or abscess.[69] Histologically, it is composed of sheets of spindled, moderate to poorly differentiated cells with eosinophilic eccentric cytoplasm. A condensed layer of tumour cells beneath an intact epithelium, also known as a "cambium layer" may be seen (Fig. 14).
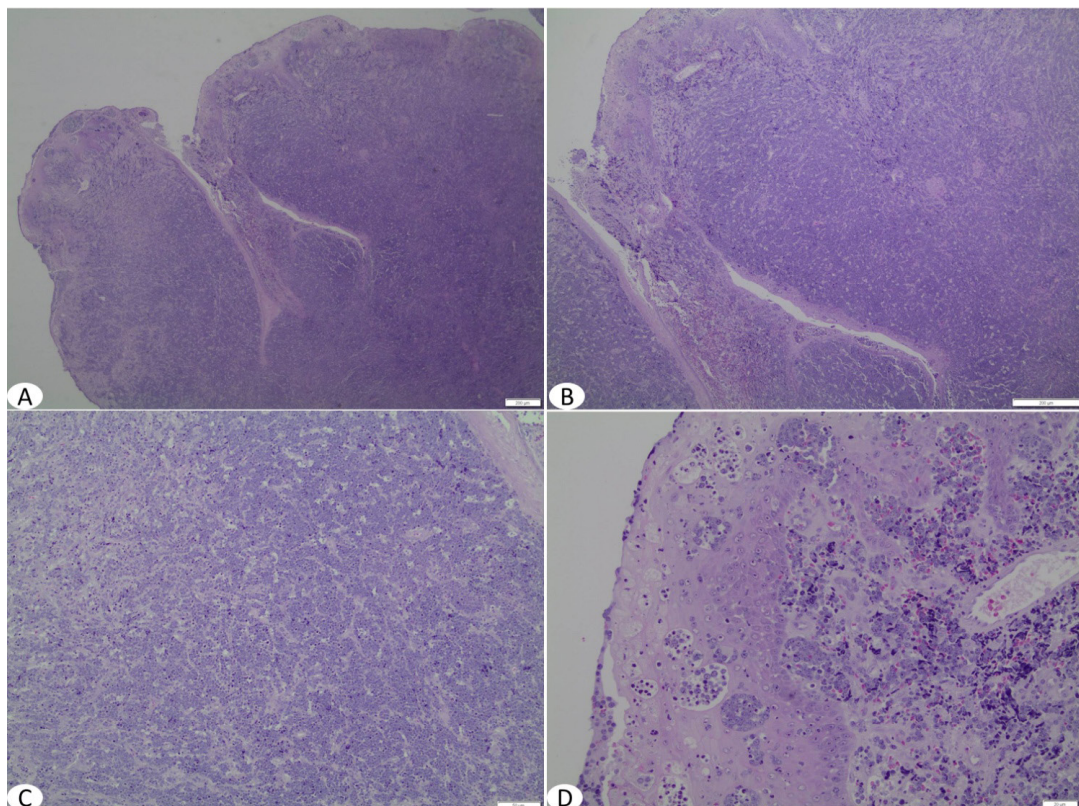

FIG. 12: A tonsil with Burkitt lymphoma showing effaced architecture (A&B, scale bar = 200μm) and an infiltration of lymphoma cells with classical "starry star" appearance (C, scale bar = 50μm). Lymphoma cells often infiltrate the epithelium forming small nests (D, scale bar = 20μm).
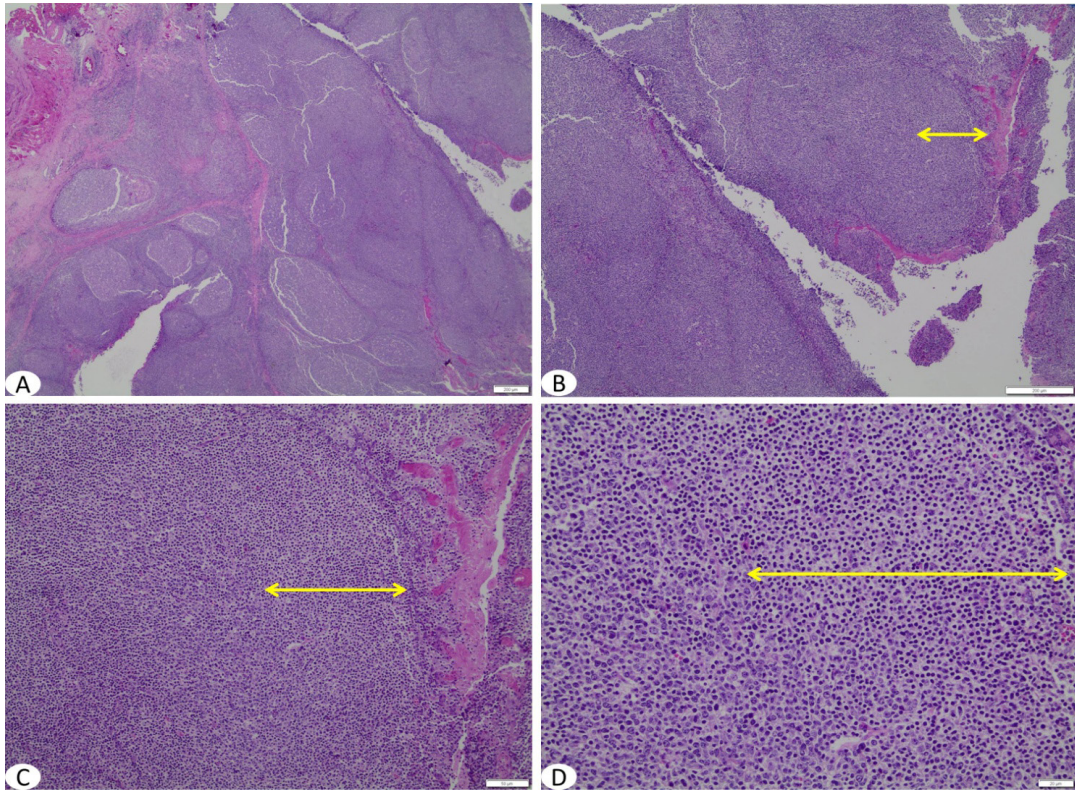
FIG. 13: Sections from a tonsil involved with MALT, showing abnormal crowded lymphoid follicles (A&B, scale bar = 200μm) with expansion of marginal zones (C&D, scale bar- C = 50μm and D = 20μm).
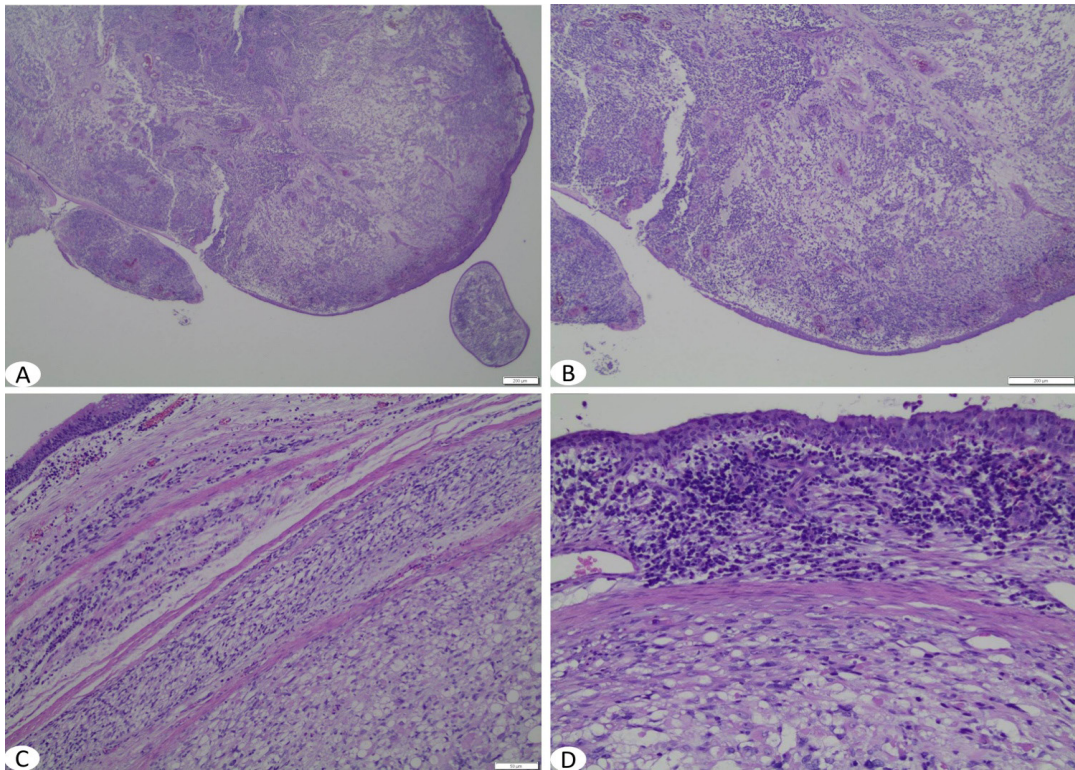

FIG. 14: Sections from a tonsil with rhabdomyosarcoma showing replacement of usual architecture by infiltrating "small blue' cell tumour (A&B, scale bar = 200μm) Higher magnification showing sheets of spindled cells with hyperchromatic nuclei and eosinophilic cytoplasm. A condensed layer of tumour cells beneath the epithelium "cambium layer" is seen (C&D, scale bar C = 50μm).

COMMENTS

In conclusion, even though most of the time, the pathologic examination of tonsils yields a reactive non-specific process, such as follicular lymphoid hyperplasia, routine microscopic examination can at times provide important clues to certain localised or systemic conditions that may have been otherwise missed. Practicing pathologists need to be aware of both the common and uncommon diseases that may be encountered in tonsils.

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