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Pediatric Kimura's disease: Case Report & Review of Literature

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Abstract

Kimura's disease (KD), also known as eosinophilic hyperplastic lymphogranuloma is a rare benign chronic inflammatory disorder first described by Kimm and Szeto in 1937, with further details provided by Kimura in 1948. It typically presents in young Asian males as painless unilateral subcutaneous masses in the head and neck, particularly post-auricular, affecting lymph nodes, soft tissues, and salivary glands, often mimicking neoplastic conditions. Diagnosis relies on blood and tissue eosinophilia, raised IgE levels and histological examination showing eosinophilic infiltration and lymphocyte proliferation. The aetiology is uncertain but may involve hypersensitivity or immune responses. Early diagnosis of Kimura's disease may spare the patient from unnecessary invasive diagnostic procedure. We describe a case of Kimura's disease in a 13-year-old male presenting with Right post auricular swelling and also provide a brief review of the literature focusing on its clinical presentation, diagnostic challenges, optimal management strategies, and primary histopathologic differential diagnosis.

Keywords: Kimura disease, eosinophilia, IgE

Introduction

Kimura's disease (KD), also known as eosinophilic hyperplastic lymphogranuloma, is a rare chronic inflammatory disorder affecting the lymphoproliferative tissue [1]. The condition primarily affects young Asian males in their early 30s [1 -3] but there is an increasing focus on children [4] in recent years. It usually presents as subcutaneous enlargements in the head and neck region especially in the post-auricular area [3] including major salivary glands and lymph nodes.[5] It can be locally aggressive. Early recognition of KD is essential to prevent unnecessary treatment and further investigation into its differential diagnosis as they are frequently misdiagnosed as neoplasms.[6] KD first described by Kimm and Szeto in 1937, as a unique inflammatory lesion in head and neck region associated with regional lymphadenopathy, peripheral blood eosinophilia and elevated serum IgE levels [7]. Kimura et al in 1948 elaborated on this self-limited allergic or autoimmune process to establish the pathogenesis of KD in their paper titled "Unusual Granulation Combined with Hyperplastic Changes of Lymphatic Tissue "and termed it as eosinophilic hyperplastic granuloma [8]. Diagnosis of KD can be challenging. Biopsy or Surgical excision of the affected swelling allows the greatest diagnostic accuracy by histopathological analysis and has been the main stay of treatment.

Case Report

13 years old male child presented with right sided post auricular painless (Figure 1 A) swelling since childhood. There were no head and neck infection or any other ENT complaints. There was history of mild pruritis and itching over the lower extremities. There were no other constitutional symptoms such as fever, weight loss, and night sweats. The overlying skin of swelling was normal. On ENT examination Bilateral tympanic membranes in the ears were intact. There was non-tender swelling measuring 2-3 cms in size, over the mastoid process. Swelling was non compressible, mobile side to side. Swelling did not reduce on medication. There were few skin rashes over lower extremities on account of pruritis.

On Investigation of Routine CBC showed Hb 11.5 gm%, TLC 6,800/mm3, Eosinophills7.7% (Normal 1-6%), PT 15.6 (Normal 10-14) and INR 1.2 (Normal 0.8-1.2) . Viral markers for HBsAg, HCV, Anti-HIV Ab and Anti HCV Ab were non-reactive.

Serum Immunoglobulin IGE levels were 2,394.0 IU/ml (Normal < or = 200 IU/ml).

Urine Routine Examination was Negative for proteinuria.

Ultrasound Neck and Post auricular region showed both lobes of thyroid and Isthmus, both parotid and submandibular salivary glands normal in shape, size and echogenicity with no obvious focal lesion noted. Vessels of neck were seen normally on both sides. Multiple enlarged lymph nodes were seen in bilateral Level II, III, V cervical station and Right post auricular region with preserved fatty hila. Largest in Right cervical station Level II measuring 1.6x0.8 cm, in Left cervical station Level measuring 2.1x0.9 cm and in Rt post auricular Region measuring 1.5x0.6 cm. The diagnosis of Bilateral Cervical lymphadenopathy was given on ultrasonography.

Fine Needle Aspiration cytology of Post auricular Region was reported as features suggestive of chronic granulomatous inflammation. Patient was admitted later for surgical removal and excision of Post auricular swelling under local anesthesia (Figure 1 B). The excised tissue was sent for histopathological examination.

Gross Examination

Specimen received consisted of two greyish brown to greyish white irregular soft tissue pieces. The larger was measuring 2 x 1.2 x 0.3 cm and the smaller was measuring 1.8 x 1.0 x 0.3cm. The Cut surface of both was solid greyish white and firm. (Figure 1 C)

Microscopic examination (Figure 2 a-f)

The Sections showed lymph node with perinodal fat and muscle. The subcapsular sinus was obliterated by enlarged follicles with peripheral concentric layer of lymphocytes and prominent hyperplastic hyalinized vessels in the interfollicular stroma. Also present were focal collection of eosinophils infiltrating these follicles and in interfollicular areas with folliculolysis.

Zeil Nelson stain (ZN) for Acid fast bacilli was negative.

Diagnosis of lymphoproliferative disease with possibility of 1. Castleman's Disease - hyaline vascular type 2. Kimuras Disease was given.

Immune histochemistry for IgE, CD21, CD35 and Factor VIII related antigen was advised for confirmation of the disease.

Immunohistochemistry for CD21 showed immunoreactivity in well-organized meshwork pattern with in germinal centers of secondary lymphoid follicles and Factor VIII related antigen was immunoreactive in large vessels in para cortical areas. No intra follicular positivity seen. Immuno profile features ruled out a diagnosis of Castleman's disease. Hence, the diagnosis of Kimuras ds was made due to peripheral eosinophilia, markedly elevated serum IgE levels and on IHC for CD21 And factor VIII related antigen.

Discussion

Kimura's Disease is a benign rare, chronic inflammatory disorder which was first described in 1937 by Kimm and Szeto in Chinese literature as "eosinophilic hyperplastic lymphogranuloma" [7]. Kimura et al. in 1948 reported it in Japanese literature titled "Unusual granulation combined with hyperplastic changes of lymphatic tissue'' after this, entity became widely known as Kimura's disease.[8]. The condition is most common in young men of Asian descent in 2nd and 3rd decades of life [6], but there is

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an increasing focus on pediatric patients [4] in recent years. In the present case report the patient was a 13 years old male child. KD presents with painless subcutaneous nodules in the head and neck region often unilateral and frequently associated with regional lymphadenopathy usually preauricular, axillary and inguinal with or without involvement of parotid and submandibular glands. Sometimes bilateral involvement is also seen. Unusual sites being auricle, scalp, orbit, nerves and spermatic cord. It is a benign condition with self -limiting course [9]. In our case, child presented with asymptomatic, non-tender Bilateral cervical and right post auricular swelling since childhood. Patient had elevated eosinophils (7.7%) as well as elevated serum IgE levels (2,394 .0 IU/ml) (Normal < or = 200 IU/ml).

The etiology and pathogenesis of KD remain uncertain. Although it might be a self-limited allergic or auto immune response triggered by an unknown persistent antigenic stimulus. Some researchers have suggested that persistent antigen stimulation following arthropod bites and allergic reactions triggered by parasitic infections such as fungi [10, 11] may influence alterations in immune regulation, potentially contributing to the onset of KD. In our case, the acid fast staining for tuberculous bacilli was negative and absence of any constitutional symptoms were suggestive of non-infective pathogenesis of KD. Presently, it is widely accepted that T cells play a crucial modulatory role in eosinophil development, with type 1 and type 2 T helper cells originating from T cells (Th1 and Th2) being the primary factors associated with excessive eosinophil and IgE production through Interleukins (IL 4 &IL5) that is seen in KD. [12], Thus an increase in Th2 cells and related cytokines may explain the tissue and peripheral eosinophilia and increased serum IgE production. IgE mediated immune reaction in mast cell recruitment, activation, and degradation that is believed to be the root of Kimura's disease, also predisposes the patient to allergic conditions like asthma, chronic urticaria, pruritus and rhinitis [12] , may explain common symptoms seen in KD, including skin rashes and pruritus [2]. In our case patient showed elevated eosinophils (7.7%) as well as elevated serum IgE levels (2,394 .0 IU/ml) (Normal < or = 200 IU/ml). The child had symptoms of mild pruritus and skin rashes over the lower extremities.

Proteinuria has been reported in 12–16% of patients with KD, with 60–78% of these patients developing nephrotic syndrome [13,14]. In our case, urine routine examination was negative for proteinuria. In recent years, an increasing number of scholars have found that KD is often accompanied by kidney disease. Kimura's cases with kidney disease can exhibit various pathological changes upon biopsy. These changes include minimal change disease of the kidneys, mesangial proliferative glomerulonephritis, membranous glomerulopathy [15-17], and focal segmental glomerulosclerosis [16].

Imaging studies are not specific but can help in staging the extent and progression of the disease as well as the lymph node involvement. In a study by Mai Yet al, ten patients who underwent ultrasound examination showed masses located in enlarged lymph nodes and parotid glands mainly appeared as round or oval echo. In addition, thickened cortex and reduced or absent medulla were exhibited in the lymph nodes. The masses in the soft tissue exhibited as hypoechoic or mixed echo, with blurred boundaries [4]. In a previous review of ultrasound reports from 58 patients with KD, the mass presented as ill-defined, solid, and heterogeneous echogenic lesions, and blood flow signals could be found partially. Enlarged hilum of lymph node was seen in all involved lymph nodes [18].

In our case, Multiple enlarged lymph nodes were seen in bilateral level II, III & V cervical station and right posterior auricular region with preserved fatty hila, the largest measuring in right II cervical station measuring 1.6 x 0.8 cm, in left II cervical station measuring 2.1 x 0.9cm and in right post auricular region measuring about 1.5x 0. 6cm. Impression of Bilateral cervical lymphadenopathy was given. Thyroid and salivary glands were reported normal on ultrasonography.

Hyui et al provided an overview of constant, frequent and rare characteristics of lymph nodes in Kimura's disease which include preserved nodal architecture, follicular hyperplasia with reactive germinal centers, eosinophilic infiltrate and proliferation of postcapillary venules in the interfollicular areas. Also other frequent findings include proteinaceous deposits in germinal centres , polykaryocytes and rare giant cells (Warthin Finkeldey type giant cells), eosinophills in germinal centers or small eosinophilic granuloma, folliculolysis, eosinophilic microabssceses, and

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perivenular sclerosis of variable degree is often observed. Nodal architecture is largely preserved in most cases, however capsular fibrosis with subcapsular sinus obliteration and perinodal soft tissue involvement is frequently present.[3,19] Immunohistochemical staining with IgE shows a characteristic reticular staining pattern of germinal centers [6]

In our case, on histopathological examination of lymph nodes showed similar features. Some predominant features were obliteration of subcapsular sinuses by enlarged follicles with peripheral concentric layer of lymphocytes and prominent hyperplastic hyalinized vessels in the inter follicular stroma. Additionally, focal collection of eosinophils infiltrating these follicles and in interfollicular areas withfolliculolysis was present. Immunohistochemistry for CD21 showed immunoreactivity in well-organized meshwork pattern within germinal centers for secondary lymphoid follicles and factor VIII related antigen was immunoreactive in large vessels in para cortical areas. No intra follicular positivity seen. So, the diagnosis of KD in our case was through the clinical picture, histological examination of excised tissue and immunohistochemistry.

The diagnosis of KD is not easy and differential diagnosis includes angiolymphoid hyperplasia with eosinophilia(ALHE), Langherhan's cell histiocytosis , Hodgkin's lymphoma, Nodular T Follicular Helper (TFH) Cell Lymphoma (nTFHL), Kaposis sarcoma Castleman's disease, reactive follicular hyperplasia, tuberculosis, parasitic lymphadenitis, and many more [3,20] . ALHE being the closest differential. Differentiating KD from (ALHE) requires a strict analysis of clinical and histologic features because the diseases are similar and were once thought to be the same disorder [9,21]. Current evidence strongly suggests that kimura's disease and then disease known to dermatologists as Angio lymphoid hyperplasia with eosinophilia are different entities. The term ALHE persisted in the literature and in usage by pathologists to denote epithelioid hemangioma (EH) [22]. Both diseases usually present with soft tissue masses in the head and neck region with prolonged and indolent clinical course but in Angiolymphoid Hyperplasia with Eosinophilia, the lesions are mostly dermal or subcutaneous and not often in lymph nodes, which is a common location for Kimura's lesions. In both cases, histologic examination of the lesion shows lymphocyte infiltration, numerous eosinophils, germinal center formation, and proliferative blood vessels. But most important, the vascular proliferation in Angiolymphoid Hyperplasia form aggregates or lobules comprising plump endothelial cells with epithelioid or histioid change demonstrating nuclei of varied size and shape , abundant clear to eosinophilic vacuolated cytoplasm, imparting hobnail appearance and hemosiderin deposits [9,22] Also, the endothelial lining usually is more than one cell thick. These changes are not seen in KD. These histological changes suggest that Angiolymphoid Hyperplasia with Eosinophilia is neoplastic in origin, whereas KD is an immune-mediated disorder [23].FOSB nuclear positivity in the endothelial cells of ALHE has been reported [24], likely due to oncogenic FOS rearrangements [25,26].

Other neoplastic entities characterized by an eosinophilic infiltrate on histology can occur in the head and neck, to include classic Hodgkin lymphoma (CHL) and Langerhans cell histiocytosis (LCH). There salient features discussed in table 1 along with some other pathologies.

Many other pathologies pose differential diagnosis besides lymphoma which are lymphadenitis and lymphnode hyperplasia of various causes. Pathologists commonly utilize antibodies such as CD3, CD5, CD20, PAX5, CD21, IgD, and Ki67 for this purpose. The primary considerations for differential diagnosis of lymphadenitis typically encompass tuberculous lymphadenitis, cat scratch disease (CSD), KikuchiFujimoto disease (KFD), and parasitic lymphadenitis[6].

Specific etiologies of follicular hyperplasia, such as Castleman disease show histologically large follicles with regressed germinal centres surrounded by concentric rings of mantle zone. The follicle show marked vascular proliferation and hyalinization of their abnormal germinal centers: they have been confused with Hassall's corpuscles and splenic white pulp . Many of the large cells with vesicular nuclei present in the hyaline center are follicular denritic cells as evidenced by their strong immunoreactivity for CD 21 and CD35.There is a tight concentric layering of lymphocytes at the periphery of the follicles (corresponding to mantle zone) resulting in onion skin appearance. The Interfollicular stroma also show

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prominent ,with numerous hyperplastic vessels of post capillary venules type and admixture of plasma cells ,and KPI positive plasmacytoid monocytes [27] and eosinophilic microabscesses are absent .Immunohistochemically there is polyclonal immunoglobulin production by plasma cells and large numbers of suppressor T cells are found in interfollicular areas. Strong positivity for factor VIII related antigen is seen in endothelium of interfollicular vessels but only weak and focal reaction for this marker is found in the hyalinized vessels **lo**cated in the center of the follicles [28**].** In our case, Immunohistochemistry for CD21 showed immunoreactivity in well-organized meshwork pattern with in germinal centres of secondary lymphoid follicles and factor VIII related antigen was immunoreactive in large vessels in para cortical areas . No intra follicular positivity seen**.** Immunoprofile features ruled out a diagnosis of Castleman's disease**.**

Other specific etiologies of follicular hyperplasia, such as, toxoplasmosis, syphilis, and HIV-associated lymphadenopathy, have well-characterized features [29]. These diseases are not likely to be in the differential diagnosis of KD as they lack the other classic features of KD

Other possible conditions to consider when diagnosing KD affecting the parotid gland include Mikulic'z disease , infective parotitis, Sjogren's syndrome and different types of salivary gland tumors.[30].

The main stay of treatment is excision of masses with post-surgery administration of glucocorticoids ,biologics, radiotherapy and immunosuppressive agents. Adjuvant therapy with surgical excision is preferred in patients with the following conditions: a tumor greater than or equal to 3 cm in size, symptom duration longer than or equal to 5 years, peripheral blood eosinophilia greater than or equal to 20%, or serum IgE greater than or equal to 10000 IU/ml to achieve the effective control of local recurrence.[31]

Conclusion

Kimura's disease is a unique inflammatory condition that poses diagnostic and therapeutic challenges due to its rarity and overlapping features with other disorders. A thorough understanding of its clinical, histologic, and immunologic characteristics is essential for accurate diagnosis and management. While the prognosis remains favourable, knowledge of KD will put the physicians in a better position to diagnose and treat the disease that may enhance patient outcomes.

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Table 1. Kimura disease and common differential diagnosis

Entity	Eosinophilia & Serum IgE	Gross lesion	Histological features	Immohisto chemistry
Kimura disease	Usually seen	Solitary lesions are mostly In the deep subcutaneous tissues, frequently associated with Regional lymphadenopathy and salivary gland involvement	Follicular hyperplasia, eosinophils, eosinophilic folliculolysis and microabscesses, postcapillary venule proliferation, proteinaceous deposits, Warthin-Finkeldy polykaryocytes	IgE reticular staining within germinal center
ALHE	Rare peripheral eosinophilia	Small, superficial dermal papulonodules, frequently erythematous, accompanied by bleeding and pruritus. Regional lymphadenopathy is rare	Vascular proliferation with hobnailed endothelial cells, eosinophills	+ Staining CD31& Factor VIII related antigen in endothelial cells FOSB nuclear expression in endothelial cells
Castleman disease	Absent	Lymphadenopathy or mass with secondary symptoms related to mass Localized or systemic progressive ds with lymphadenopathy of multiple sites	Regressed germinal centres surrounded by concentric rings of mantle zone (onion skin appearance) . Interfollicular areas show increased vascular proliferation and plasmacytoid dendritic cells; eosinophilic microabscesses are absent	Strong immunoreactivity for CD 21 & CD35 by concentric rings of follicular dendritic cells
Langherans Cell histiocytosis	Eosinophils are increased	Unifocal or multifocal, cutaneous lesions and lymphadenopathy in	Effacement of lymph node architecture, histiocytes with “coffee	CD1a+, S100+, and CD207 (langerin)+ in histocytes

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		head/neck, often in children	bean like" grooved nuclei, eosinophils	
Hodgkin lymphoma. Eosinophil	Uncommon & rarely seen	Nodal, supradiaphragmatic masses, B symptoms	Effacement of lymph node architecture, Hodgkin-Reed-Sternberg (HRS) cells, eosinophils	CD15+, CD30+, weak PAX5 + in HRS cell
Nodal T Follicular Helper Cell Lymphoma, Follicular Type	Eosinophills often present	Generalized lymphadenopathy, advanced-stage disease	Effacement of lymph node architecture, follicular pattern, intermediate-sized lymphocytes, eosinophils	CD3+, CD4+, strong PD1+,CD10+ and other T follicular helper cell marker
Reactive hyperplasia	variable	Generalised Lymphadenopathy, variable systemic symptoms	Variable histology (follicular hyperplasia, paracortical/interfollicular hyperplasia, sinus histiocytosis), variable eosinophils	Polyclonal B- and T-cells, no specific markers
Tuberculous lymphadenitis	Not seen	Cervical Lymphadenopathy ,large multinodular mass with draining sinus communicating with skin(scrofuloderma)	Multiple small epithelioid granulomas ,caseous necrosis,langhan's giant cells ,epithelioid cells and lymphocytes	ZN stain for acid fast bacilli + and other Ancillary tests - PCR and CB -NAAT

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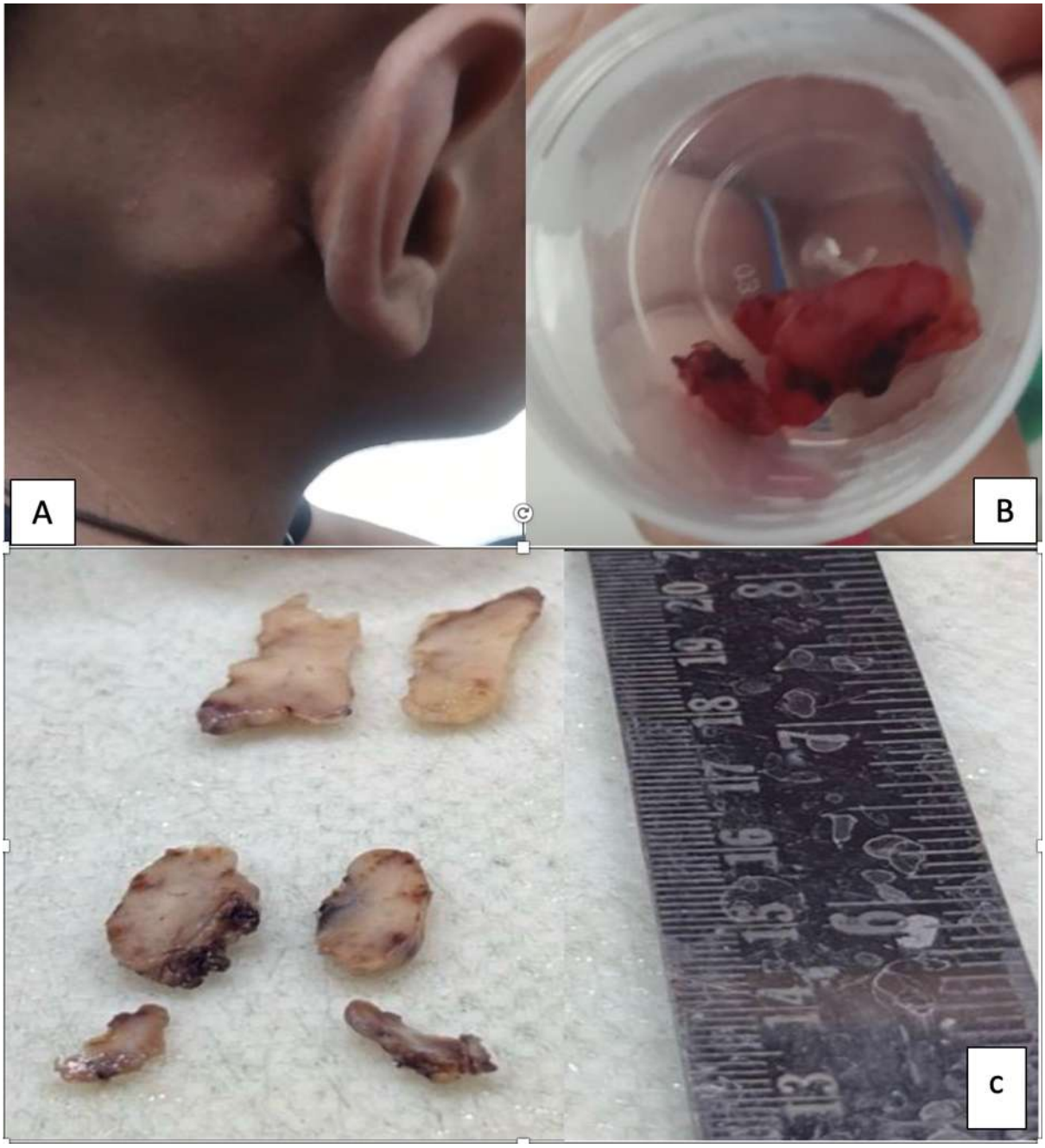


Figure 1: A. Postauricular swelling B. Excised lymph node C. Gross specimen- cut section solid greyish white and firm

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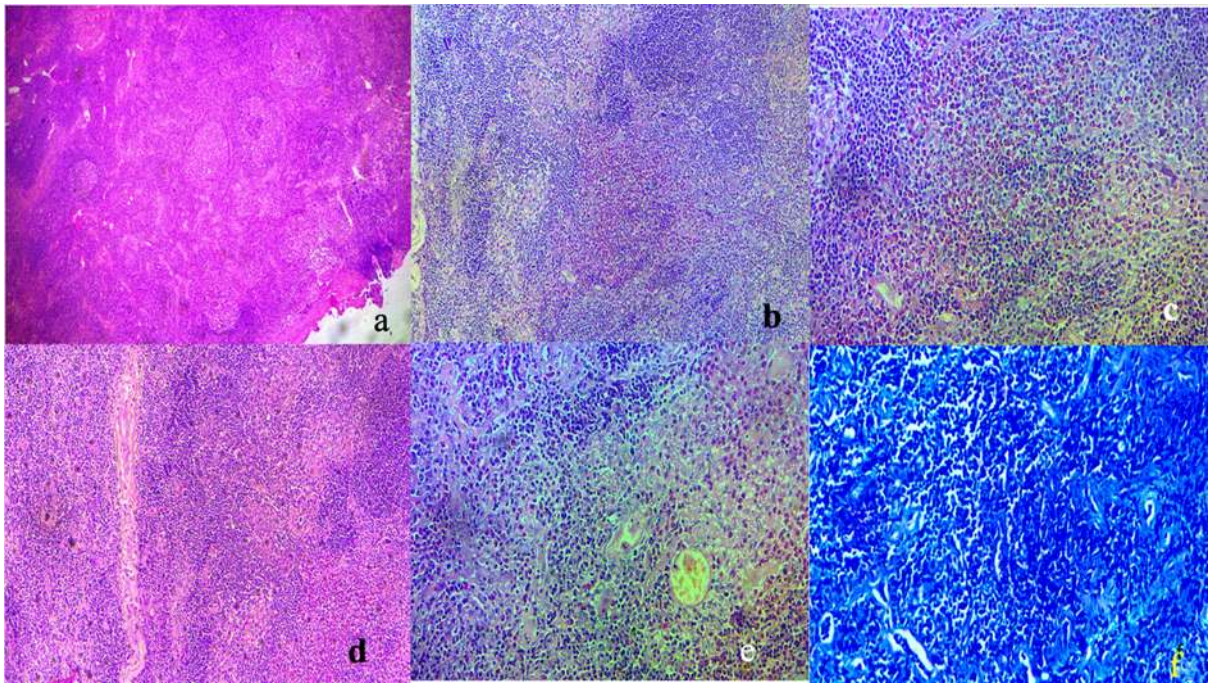


Figure 2: Histo-pathological sections a. Hyperplastic Lymphoid follicle with germinal centers (H&E 10X) b. Diffuse interfollicular eosinophilic infiltrates with eosinophilic microabscess (H&E 20X) c. High power showing prominent eosinophils with folliculolysis (H&E 40X) d. Interfollicular areas with increased vascular proliferation (H&E 20X) e. Endothelial cell proliferation with capillary lumen containing RBCs in the paracortical region (H&E 40X) f. ZN stain Negative for acid fast bacilli (100X)

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