

TRAUMATIC ULCERATIVE GRANULOMA WITH STROMAL EOSINOPHILIA: SINISTER APPEARING INNOCUOUS LESION

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ABSTRACT

Trauma within the oral cavity is one of the common incidence of occurrence which can be an inducer as well as propagator for wide range of oral lesion of diverse course. A unique combination of formation of granulomatous lesion with reactive eosinophilic stromal infiltration in response of trauma is "traumatic ulcerative granuloma with stromal eosinophilia (TUGSE)". It is a reactive, infrequent, self-limiting condition of oral mucosa. The commonly involved site and the clinical presentation might click to suspect it as a lesion of oral cancer. Clinical history and examination guide through the diagnosis whereas histopathology forms the basis of the diagnosis.

KEY WORDS

Eosinophil, Granuloma, Oral mucosa, Traumatic



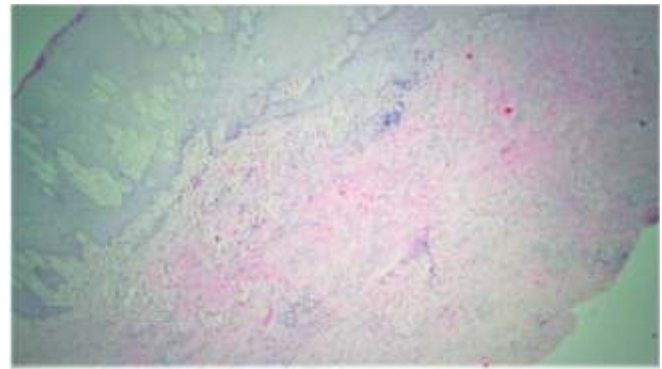
INTRODUCTION

Traumatic ulcerative granuloma with stromal eosinophilia (TUGSE) is a self-limiting distinctive entity also known as traumatic eosinophilic granuloma and traumatic granuloma.^{1,2} It is an infrequent, benign oral disease that affects the commonly affects lateral or ventral surface of the tongue¹ along with buccal and vestibular mucosa, palatal mucosa, retromolar area, gingiva, and the floor of the mouth.³ It is most often caused by trauma, such as a sharp or broken tooth or restorative material, but it may also be caused by accidents or self-inflicted injuries.^{1,4} TUGSE appears as an ulcer with elevated and indurated margins with a yellowish fibrinous base and rapid growth of the lesion could lead to over diagnosis of this lesion as oral squamous cell carcinoma (OSCC).⁵

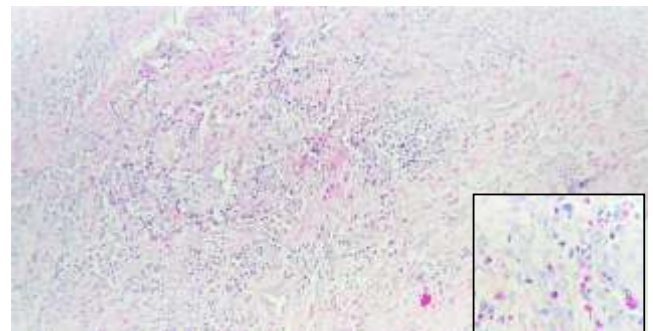
Here we report an uncommon case of TUGSE highlighting the significance of clinico-pathological correlation and timely management of the case.

CASE REPORT

A 61 years old female presented with a single, erythematous, soft tissue mass in lower back region of jaw since four weeks. The lesion extended in the lingual aspect of mandible from 44 to 47 region, measuring 10 x 10 mm² in maximum dimension and tender on palpation without any relevant medical and personal history. The mass was ulcerated, slow growing, progressive in nature and associated with mild pain which lasted for few minutes and relieved itself. Root stump with sharp edges in relation to 44, 45 and 47 were present, causing trauma to the lesion site. Excisional biopsy of the lesion along with extraction of root stumps 44, 45 and 47 was done. A clinical diagnosis of oral fibroma was made. The two bits of specimens were received in 10% neutral buffered formalin and sent for routine histopathological processing. The processed tissues were embedded, sectioned into 3 µm thick section and stained with hematoxylin and eosin. The section revealed hyperplastic parakeratotic stratified squamous epithelium with long, elongated rete ridges and core of connective tissue entrapped within the epithelium. Underlying connective tissue was loosely fibrocellular with fibroblast, inflammatory cells infiltrates mainly of mononuclear cell type along with increased vascularity. The deeper area within the connective tissue shows dense eosinophilic infiltration showing bilobed nuclei and intense eosinophilic granules within the cytoplasm adjacent to the area resembling granulation tissue. (Microphotograph 1 and 2) The histopathological diagnosis after clinical review was concluded as traumatic ulcerative granuloma with stromal eosinophilia. The patient was recalled after a week for evaluation which showed uneventful healing.



Microphotograph 1: Hyperplastic Parakeratinized stratified squamous epithelium with acanthosis and long elongated rete ridges (H&E, 10X)



Microphotograph 2: Deep in the connective tissue collection of eosinophils and lymphocytes are evident (H&E, 10X); Inset: Eosinophils, eosinophilic cytoplasmic granules with bilobed nuclei (H&E, 40X)

DISCUSSION

Traumatic ulcerative granuloma with stromal eosinophilia is an uncommon lesion characterized by solitary ulceration of the oral mucosa.¹ Riga first defined this lesion clinically in 1881, and Fede first described it histologically in 1890. Shapiro and Juhlin recognised it as a distinct entity in 1970, but it was only in 1983 that Elzy coined the word TUGSE.⁶ Since TUGSE is not widely recorded in the literature, the exact frequency is uncertain, but it is thought to be fairly frequent.¹ Men and women are affected almost equally,^{1,7} with a slight female predominance.⁷ It can be reported at any age, but it is most frequently discovered in the fifth decade of life.^{1,2}

The pathogenesis of TUGSE is uncertain; however, trauma induced ulceration, allows microorganisms, toxins, and foreign particles to enter the surrounding tissue. The mast cell-eosinophil reaction, which recruits eosinophils cause more damage by exacerbating inflammation locally, is one of the considered pathogenesis of the lesion.⁸ These lesions are almost ulcerated but some have been described as submucosal masses.⁸ It is usually manifested as a single ulcer varying in size from a few millimetres to more than 6 cm in diameter, with an average size of 2 cm². Purulence may be present, and the ulcer with a base of white fibrinous necrotic debris. The margins of the ulcer are frequently elevated and indurated, and in the early stages the circumambient

mucosa has an inflammatory red halo. In older lesions, the peripheral erythema is often absent.¹The symptoms range from slightly tender to excruciatingly painful lesion. Ulcers can last for weeks or months, and in some cases, years.^{1,9}

Clinical diagnosis alone of TUGSE is challenging, so histopathology with clinical correlation is the must to confirm the diagnosis. An ulcerated epithelium with a polymorphic inflammatory cell infiltrate with a significant predominance of eosinophils spreading from the superficial epithelium to the deep layers of muscle tissue is the quintessential histopathological feature of TUGSE.¹⁰

In our case 61 year female have lesion due to sharp teeth and, histopathological findings revealed granulation tissue along with eosinophils uniformly distributed across the connective tissue confirming the diagnosis. The precise function of eosinophils in TUGSE has yet to be determined. They may contribute to mucosal degeneration by secreting inflammatory cytokines like tumour necrosis factor. Similarly, eosinophils' inability to synthesise transforming growth factors may explain TUGSE's delayed healing.¹¹

Clinical appearance of TUGSE mimics malignancies such as squamous cell carcinoma, lymphoma, and salivary gland tumours, making differential diagnosis difficult. Even the histopathological differential diagnosis may include many lesions marked by infiltration of eosinophil within the connective tissue; such as Langerhans cell disease, Angiolymphoid Hyperplasia with Eosinophilia (ALHE),

Kimura disease, some forms of lymphomas, allergic reactions, and parasitic diseases.^{12,13}

Therefore practitioners much be judgemental while enlisting the differential diagnosis of condition like TUGSE, where it might represent from reactive to benign to malignant lesions.

CONCLUSION

TUGSE is a benign oral mucosal lesion of unknown pathogenesis. The clinical and histopathological characteristics alone of TUGSE can cause difficulty of diagnosis due to the large number of pathologies with conflicting clinical and histopathological differential diagnosis. Therefore a proper clinico-pathological correlation is paramount in cases like TUGSE, for proper management of the patients.

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PATIENT CONSENT

The written consent was obtained from patient.

CONFLICT OF INTEREST

None

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