



## CASE REPORT

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# TRAUMATIC ULCERATIVE GRANULOMA WITH STROMAL EOSINOPHILIA (TUGSE): A CLINICAL CASE REPORT

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**Abstract.** *Traumatic ulcerative granuloma with stromal eosinophilia (TUGSE) is a rare, benign, and self-limiting oral lesion characterized by chronic ulceration and a distinctive eosinophil-rich inflammatory infiltrate. This case report presents a 45-year-old male patient with a chief complaint of a growing and mildly painful wound on the center of his tongue, which had been present for five weeks. History did not reveal any specific intra-oral injuries, but the patient's work is related to skydiving. Clinical examination revealed an oval lesion with dense edges on the dorsal part of the tongue, covered with a fibrinous coating, measuring about 1.5 cm in diameter, slightly painful on palpation. A decision was made to carry out an excisional biopsy, which was performed under local anesthesia, and sent for histopathological investigation. The morphological finding corresponded to the diagnosis, and the healing process went smoothly. TUGSE occurs worldwide across a broad age range, with peaks in infancy and middle age, and a slight male predominance. Its etiology is often linked to localized trauma, though other factors like viral infections and toxic agents may contribute. Histopathologically, it is characterized by eosinophil-rich granulation tissue with deep inflammatory infiltration, sometimes mimicking malignancies. Clinically, it presents as a slow-progressing ulcer, often mistaken for squamous cell carcinoma, with the tongue being the most common site. Treatment focuses on trauma removal, conservative management, and in persistent cases, surgical intervention, with a generally favorable prognosis. Overall, TUGSE poses diagnostic challenges, emphasizing the need for accurate clinical and histological diagnosis to ensure appropriate management.*

**Key words:** TUGSE, tongue, epidemiology, diagnosis, treatment, case report

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## INTRODUCTION

Traumatic ulcerative granuloma with stromal eosinophilia (TUGSE) is a rare, benign, and self-limiting oral lesion characterized by chronic ulceration and a distinctive eosinophil-rich inflammatory infiltrate [1-3]. First described by Popoff in 1956 and later observed as Riga-Fede disease in infants, it has since been recognized across all age groups, with peak incidences in infants (teething-related trauma) and adults aged 30-50 years [1, 2, 4]. Clinically, TUGSE presents as a solitary, painful ulcer with erythematous margins, often mimicking malignancies like squamous cell carcinoma or infectious processes such as syphilis [1, 5, 6]. While trauma is the primary etiological factor – typically from sharp teeth, dental malocclusion, or ill-fitting prostheses – non-traumatic cases suggest potential contributions from viral or toxic agents [2, 4].

Histopathologically, TUGSE features ulceration with fibrinopurulent exudate, dense stromal infiltration by eosinophils, lymphocytes, and large mononuclear cells, and variable CD30+ lymphocytes, raising diagnostic challenges due to overlaps with lymphoproliferative disorders [2, 5, 6]. Despite its benign nature, TUGSE warrants histological confirmation via biopsy to exclude malignancy, as clinical features alone are often insufficient for diagnosis [1, 4, 6].

This paper aims to delve into the epidemiology, clinical presentation, diagnostic features, and treatment approaches of TUGSE, with the presented case report of a young patient with TUGSE on the dorsal part of the tongue highlighting the diagnosis and management of this benign condition.

## CLINICAL CASE DESCRIPTION

This case report presents a 45-year-old male patient who was admitted to the Department of Maxillofacial Surgery of the "Sveti Panteleimon" Hospital in Plovdiv with a chief complaint of wound on the center of his tongue that he claimed had been present for five weeks. The patient reported that the wound was growing and began being mildly painful. His history did not reveal any specific intraoral injuries, but the patient work is related to skydiving and he often receives injuries to the face and upper/lower extremities. Clinical examination revealed an oval lesion with dense edges on the dorsal part of the tongue, covered with a fibrinous coating, measuring about 1.5 cm in diameter, and being slightly painful on palpation (Figure 1). A decision was made to carry out an excisional biopsy, which was performed under local anesthesia. The biopsy was sent for histopathological investigation.

The macroscopic examination showed that in the resection material from the tongue, a nodular, soft on section, grayish-whitish, superficial ulcerated lesion was found, measuring 1.5 cm x 2 cm. The lesion was well demarcated from the surrounding tissue of the tongue, without forming a capsule.

The healing process went smoothly, and the patient was followed-up after 10 days. On this postoperative check-up, the healing of the surgical site was satisfactory (Figure 3). There was no reported recurrence.

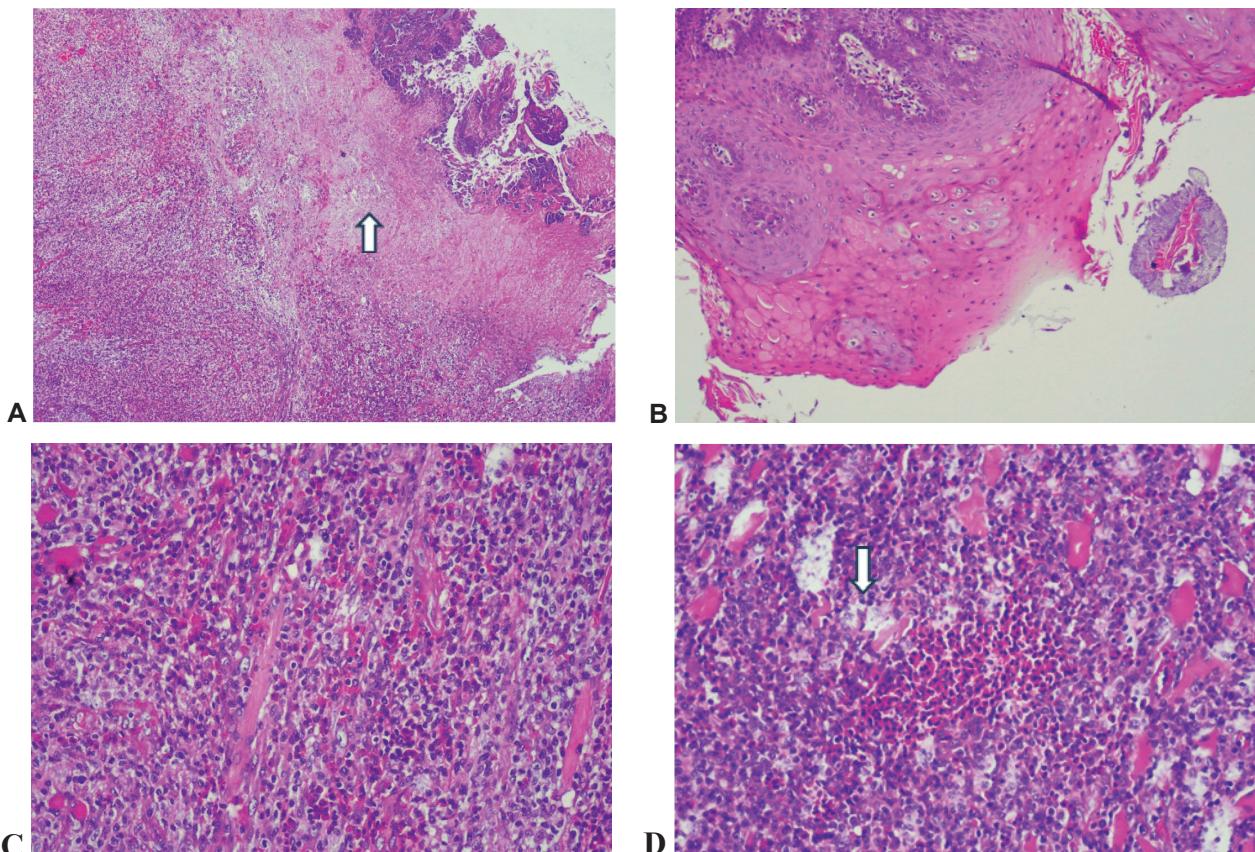


**Fig. 1.** Clinical image showing an oval lesion with dense edges on the dorsal part of the tongue, covered with a fibrinous coating



**Fig. 2.** Clinical image showing the healing of the surgical site after the excisional biopsy

The histological examination was performed on routine paraffin sections stained with Hematoxylin/Eosin. An ulcerative lesion with superficial fibrinoid necrosis with bacterial colonies, leukocytes and a productive, inflammatory process at the bottom of the ulcer, represented by lymphocytes, fibrocytes, macrophages and massive eosinophils with areas of eosinophilic abscesses, was found. Strong proliferation of vessels from maturing granulation tissue was observed. In the vicinity of the lesion, the epithelium showed pronounced hyperkeratosis, parakeratosis, acanthosis and papillomatosis. The morphological finding corresponded to Traumatic Ulcerative Granuloma with Stromal Eosinophilia (TUGSE) (Figure 3).



**Fig. 3.** Histopathological findings. **A)** Ulcerative lesion with superficial fibrinoid necrosis with bacterial colonies, (standard magnification 4x); **B)** Epithelium of the tongue in the vicinity with pronounced hyperkeratosis, parakeratosis, acanthosis and papillomatosis (standard magnification 10x); **C)** Productive, inflammatory process at the bottom of the ulcer represented by lymphocytes, fibrocytes, macrophages and massive eosinophils (standard magnification 20x); **D)** Eosinophilic abscesses (standard magnification 40x)

## DISCUSSION

TUGSE is a rare, benign, and self-limiting oral lesion characterized by chronic ulceration and a distinctive eosinophil-rich inflammatory infiltrate [1-3].

The epidemiology of TUGSE is not fully studied. There is no specific geographic distribution reported, as it is a rare condition that can occur worldwide. Demographically speaking, it can occur across a wide age range, from as young as 8 years to as old as 80 years, with the mean age being approximately 49 years [7]. The highest incidence is observed in the fifth and sixth decades of life, with a notable peak in the fourth to seventh decades [7-9]. There is also a reported peak in the first two years of life, often associated with teething [10]. There is a slight male predominance, with a male-to-female ratio of approximately 1.6:1 [8]. However, some studies suggest a minimal female predominance [9].

TUGSE is considered a rare condition, and due to its benign nature and potential for misdiagnosis, exact incidence and prevalence figures are not well-doc-

umented. It is often underreported and/or misdiagnosed due to its resemblance to malignancies like squamous cell carcinoma, which contributes to the lack of comprehensive epidemiological data [1, 4, 8].

The condition is often associated with localized trauma, although the exact mechanisms remain unclear. More than 50% of cases may not have an identifiable traumatic cause [9], but it had been reported that factors, like injuries caused by dental issues, such as missing or malposed teeth, or partial dentures, can contribute to its development [8]. Other factors like viral infections and toxic agents may also contribute to its development [1].

Histopathologically, TUGSE is characterized by eosinophil-rich granulation tissue. [1, 11]. Surface ulceration with loss of epithelium is a hallmark, exposing underlying connective tissue stroma [1, 12, 13]. The stroma shows irregular, dense collagen fibers mixed with proliferating endothelial cells and blood vessels, often accompanied by focal necrosis [12]. Muscle infiltration is common, with inflammatory cells penetrating deep into submucosal and skeletal muscle lay-

ers, causing muscle fiber degeneration [2, 12, 14]. It presents as a polymorphic infiltrate dominated by eosinophils, alongside T-lymphocytes, B-lymphocytes, histiocytes, plasma cells, and macrophages [2, 12, 13]. Large atypical mononuclear cells with epitheliotropism (infiltration into epithelial layers) are observed in some cases [12]. Eosinophil density varies; while prominent in most cases, some lesions lack overwhelming eosinophilia [1, 14].

Key immunohistochemical features are the CD30+ cells (activated lymphocytes) – detected in 40-70% of cases, suggesting overlap with lymphoproliferative disorders [1]. Clonal T-cell receptor gene rearrangements have been reported, indicating potential monoclonality in recurrent lesions [1, 11]. The pathogenesis of TUGSE can be explained with the fact, that chronic trauma initiates ulceration, allowing microbial or toxin entry, triggering an exaggerated immune response involving mast cells and eosinophils [13, 14]. Eosinophils release mediators like histamines, perpetuating inflammation and tissue damage [13].

Clinically, TUGSE must be distinguished from squamous cell carcinoma, Langerhans cell histiocytosis, angiolymphoid hyperplasia, and infections like syphilis or tuberculosis [1, 12-14]. Histological mimics include atypical histiocytic granuloma and lymphomatoid papulosis due to similarities in inflammatory patterns [1, 13, 14].

TUGSE presents as a chronic, slow-progressive lesion with rolled, elevated, and indurated margins, often mimicking squamous cell carcinoma [1, 8]. It can be asymptomatic or painful, with lesions typically appearing as solitary ulcers or submucosal masses [9]. The tongue is the most common site for TUGSE, particularly the left posterolateral surface [1]. Other sites include the buccal mucosa, vestibular mucosa, gingiva, lip, mucobuccal fold, retromolar area, and palate [5, 8]. Treatment focuses on addressing underlying trauma and promoting healing through conservative or surgical approaches. TUGSE's benign nature and responsiveness to trauma removal make conservative management the first-line approach, reserving surgery for refractory cases [2, 15, 17, 18].

Conservative management includes eliminating the trauma sources (removing ill-fitting dentures, sharp teeth, or other irritants). This is critical, as TUGSE often resolves spontaneously after trauma cessation [1, 15, 16]. Topical steroids such as 0.1% triamcinolone acetonide mouthwash (3-4 times daily) or intraläsional steroid injections can also be used to reduce inflammation and accelerate healing [1, 15, 17, 19].

In addition, topical analgesic gels (e.g., benzocaine) can also be applied for pain relief [13].

Surgical methods include biopsy, since it may trigger healing by disrupting the ulcer and improving blood flow [2, 16, 20]. Excision is recommended for persistent lesions or when malignancy is suspected, because it can provide rapid healing and histopathological confirmation [2, 15]. In addition, electrocoagulation or cryotherapy (liquid nitrogen) can be used for large ulcers [1, 15], and systemic steroids (e.g., prednisone) can also be prescribed in severe cases [17].

Prognosis is generally favorable with most cases resolving within weeks to months, with low recurrence if trauma is eliminated [1, 3]. Follow-up is usually mandatory for at least 2 years to monitor recurrence or malignancy [1, 21].

## CONCLUSIONS

Overall, Traumatic Ulcerative Granuloma with Stromal Eosinophilia (TUGSE) presents significant diagnostic challenges due to its clinical and histopathological resemblance to malignant conditions, particularly squamous cell carcinoma and lymphoma. This similarity underscores the critical importance of a thorough clinical evaluation supported by histopathological examination to achieve an accurate diagnosis. Given its association with chronic mechanical trauma, a detailed patient history and identification of potential etiologic factors are essential for effective management. While TUGSE is generally self-limiting, persistent cases may require intervention, including biopsy for confirmation and removal of underlying traumatic factors. Enhanced awareness among clinicians and oral surgeons can help prevent unnecessary aggressive treatments and ensure appropriate, conservative management. Further research is warranted to elucidate the precise pathogenesis of TUGSE and optimize treatment strategies.

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