

Oral traumatic ulcer.

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Figure 1. Low-power microscopic image shows a traumatic ulcer of the lateral tongue. A hypertrophic squamous epithelium (arrows) is immediately adjacent to the ulcer, showing an abrupt transition. Note the extension of the inflammatory infiltrate into the muscle at the bottom of the photo.

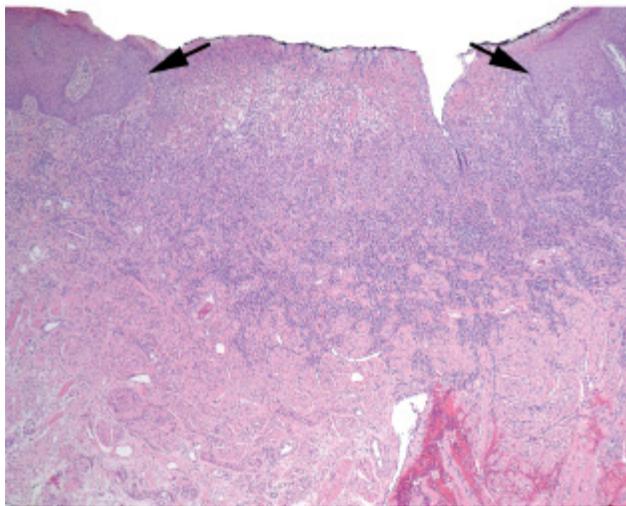
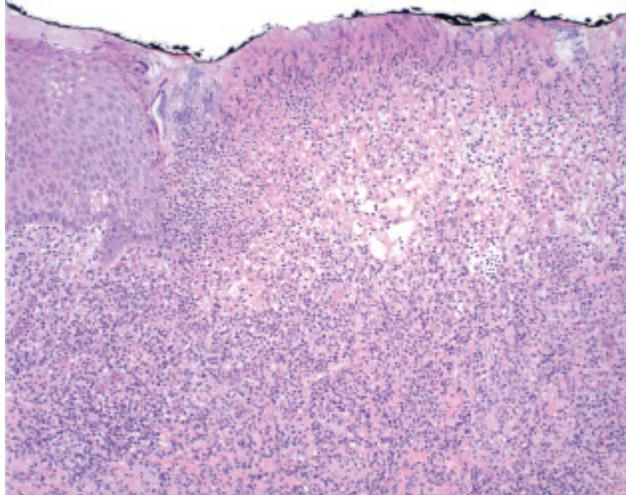


Figure 2. Intermediate-power microscopic view demonstrates the fibrinopurulent exudate and very abrupt "traumatic" edge to the epithelium. Inflammation fills the stroma.



A traumatic ulcer is a chronic traumatic ulceration of the oral mucosa that shows unique histopathologic features. Also known as *traumatic granuloma*, *eosinophilic granuloma*, and *Riga-Fede disease*, this lesion is usually caused by some sort of mechanical injury. The most common

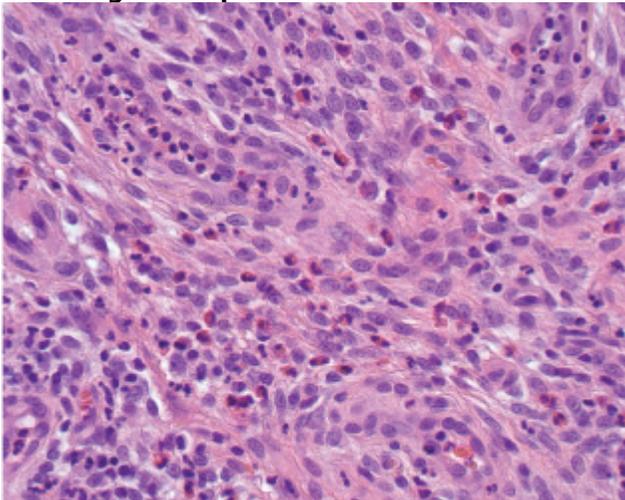
causes include accidental trauma from biting, malposed teeth, and even sharp foodstuffs. However, self-inflicted wounds caused by nocturnal clenching or tongue and lip biting, electrical and thermal injuries, hot foods or beverages, and even factitial injuries yield a similar finding. Ulceration of the ventral tongue as a result of tongue thrusting in infants with natal or neonatal teeth is referred to as *Riga-Fede disease*.

Oral traumatic ulcer is probably under-reported, but it is considered less common than aphthous stomatitis. The age at presentation is usually related to the specific cause, with a male predominance. Patients present most commonly with a painful ulcer covered by a fibrinopurulent membrane on the lateral border of the tongue, although anywhere in the oral cavity can be affected. The rim of hyperkeratosis and induration may mimic squamous cell carcinoma.

Ulcers that do not spontaneously resolve may need excision, although a biopsy may instigate resolution. Steroid injections into the lesion have been advocated by some authors. It is important to note that the source of the trauma should be removed, if possible; otherwise there is a high rate of recurrence.

Microscopically, an abrupt ulcer is covered by a very thick fibrinopurulent exudate or membrane (figure 1). The immediately adjacent epithelium will frequently show pseudoepitheliomatous hyperplasia, while the ulcer bed contains granulation tissue (figure 2). The granulation tissue shows an endothelial proliferation with a rich investment by inflammatory cells, including lymphocytes, histiocytes, neutrophils, eosinophils, and even plasma cells (figure 3). The inflammation may extend into the subjacent muscle bundles (figure 1). The muscle bundles may appear atrophic, with withered cytoplasm. Atypical histiocytes may also be seen in a few cases. Mitotic figures are usually easily identified, while necrosis tends to be limited to thermal or electrical injury cases. A number of cells may be CD30-positive, and monoclonal rearrangements have been reported, although their significance is unknown.

Figure 3. Higher-power view shows the granulation tissue with a mixed inflammatory infiltrate, including eosinophils with associated vessels.



The most common differential diagnostic consideration is with recurrent aphthous stomatitis, which tends to have superficial ulcers without the inflammatory infiltrate extending into the muscle. CD30-positive T-cell lymphoma is very uncommon, but generally has a heavier inflammatory infiltrate and more atypia.

Suggested reading

1. Baroni A, Capristo C, Rossiello L, et al. Lingual traumatic ulceration (Riga-Fede disease). *Int J Dermatol* 2006; 45 (9): 1096-7.
2. el-Mofty SK, Swanson PE, Wick MR, Miller AS. Eosinophilic ulcer of the oral mucosa. Report of 38 new cases with immunohistochemical observations. *Oral Surg Oral Med Oral Pathol* 1993; 75 (6): 716-22.
3. Hirshberg A, Armariglio N, Akrish S, et al. Traumatic ulcerative granuloma with stromal eosinophilia: A reactive lesion of the oral mucosa. *Am J Clin Pathol* 2006; 126 (4): 522-9.
4. Segura S, Romero D, Mascaró JM Jr., et al. Eosinophilic ulcer of the oral mucosa: Another histological simulator of CD30+ lymphoproliferative disorders. *Br J Dermatol* 2006; 155 (2): 460-3.

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