

## **Necrotizing Sialometaplasia Of The Palate: A Case Report**

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**Abstract :** Necrotizing sialometaplasia is a benign, self-limiting and rare inflammatory disease which, on clinical and histological examination, mimics malignant neoplasms. Necrotizing sialometaplasia is an uncommon inflammatory condition that affects salivary glands. The complete self-healing of the lesions occurred in 3 weeks. Since this entity presents clinical and histopathological findings resembling either mucoepidermoid carcinoma or squamous carcinoma diagnostic failure may terminate in needless mutilating surgery.

**Keywords:** Minor salivary glands, Necrotizing sialometaplasia, Oral ulcer, Palate.

### **Introduction**

Necrotizing sialometaplasia is an inflammatory condition first described by Abrams et al. in 1973. The condition most frequently involves minor salivary glands in the hard palate region<sup>[1]</sup> and can also occur in other anatomical locations, soft palate, lip, retromolar area, tongue, mucobuccal fold, tonsillar fossa, parotid, sub-lingual, submandibular, nasal cavity, incisive canal, maxillary sinus and larynx. <sup>[2]</sup> It clinically presents as a painful ulcerated lesion, however the symptoms and clinical appearance may vary. The lesions are usually unilateral but bilateral lesions occur in as many as 20% of cases. <sup>[3]</sup> We report a case of a 40 year old male patient corresponding to the clinical and histopathological features of necrotisingsialometaplasia in the palatal region.

### **Case report**

A 40 -year-old male patient reported to the department of oral medicine and radiology with a chief complaint of pain in the hard palate since 10 days which was sudden in onset, severe, intermittent, radiating in nature aggravates on taking food and does not have any history of taking medication. He also gave a history of swelling since 10 days that had developed into ulcer after the first three days. There was no history of trauma, paresthesia and fever. The past medical history was non contributory. The intraoral examination revealed two deep ulcers (3x1 cm and 1x1 cm), on hard palate (Fig. 1). The edges of the ulcers were elevated but not indurated, and the base was covered with necrotic slough. Based on the history and clinical examination a provisional diagnosis of suppurative osteomyelitis was made for which

differential diagnosis of deep fungal infection, mucoepidermoid carcinoma, squamous cell carcinoma were considered. The panoramic radiograph and occlusal radiographs were taken which revealed mild periodontitis. (Fig 2 and Fig 3) His haematological findings were within the normal limits except raised erythrocyte sedimentation rate. The culture test was negative which ruled out fungal infection. An incisional biopsy was done under local anaesthesia and the histological section revealed areas of necrosis and inflammatory granulation tissue along with scattered lymphoid cells. There was diffuse mixed inflammatory infiltrate consisting of neutrophils, plasma cells, macrophages and extensive squamous metaplasia of the salivary ducts and acini. (Fig 4) The symptomatic treatment was given to the patient and the lesion healed within the period of 6 weeks.

## Discussion

Necrotizing sialometaplasia is a rare inflammatory necrotizing reactive process that can involve minor and major salivary glands. The exact cause of necrotizing sialometaplasia is not known, but ischemia of local blood supply in the salivary gland lobules is the most accepted theory. The causes of the ischemia include local trauma, local anesthesia, ill-fitting dentures, smoking, alcohol consumption, radiation, allergies, upper respiratory tract infection, intubation, surgical procedures involving the area and chronic vomiting.<sup>6,7</sup> Brannon observed a mean age incidence of 46 years, in a series of 69 cases and a predominance of 2:1 male to female and 5:1 white to African-Americans.<sup>2</sup>

Clinically, NS most often presents as a deep-seated ulcer, however, few cases manifest as non-ulcerated swelling or mass.<sup>8</sup> Abrams<sup>1</sup> proposed the histopathological criteria of necrotizing sialometaplasia: necrosis of acinar cells of seromucinous glands; squamous metaplasia of salivary ductal epithelial and acini; pseudoepitheliomatous hyperplasia of the epithelium lining the gland; mucous release; inflammatory response associated with granulation tissue in or around the glands; intact lobular architecture; and histologically benign nuclear morphology, although normal mitoses can sometimes be observed. Brannon<sup>2</sup> et al. described the predominance of coagulative necrosis of acini in early lesions and of squamous metaplasia and reactive fibrosis in later lesions. Anneroth and Hansen proposed five histologic stages in the development of necrotizing sialometaplasia: infarction, sequestration, ulceration, repair, and healing. They emphasized that these stages could overlap and would be dependent upon the extent and severity of damage.<sup>9</sup> The differential diagnosis of necrotizing sialometaplasia should consider other ulcerous and erosive lesions, including those of traumatic or inflammatory/infectious aetiology, e.g., dental fissures, major aphthae, tuberculosis, tertiary syphilis or deep fungal infection in patients with AIDS or under immunosuppressive treatment and those of cancerous origin, e.g., squamous cell carcinoma, mucoepidermoid carcinoma, adenoid carcinoma and leukaemia.<sup>9-12</sup> The management of necrotizing sialometaplasia includes symptomatic treatment and the lesions healed within two to three months.

## Conclusion

The significance of this lesion is that as its clinical appearance mimics other lesions particularly malignant neoplasms; therefore proper diagnosis by investigations is necessary for the management of the patient.

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## Figures



Fig1: intraoral picture showing ulcerated lesion on the hard palate.

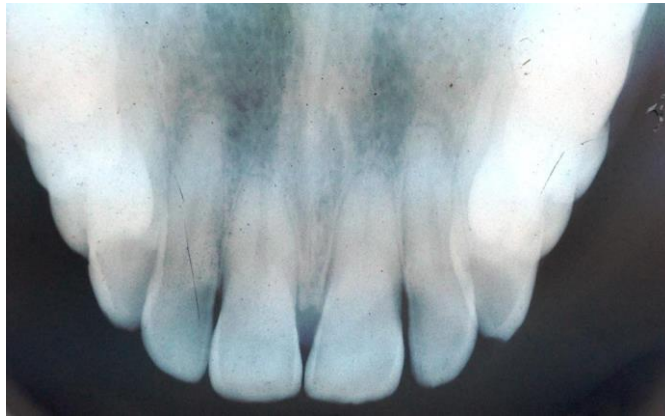


Fig 2: Occlusal radiograph of the patient



Fig 3: Panoramic radiograph of the patient

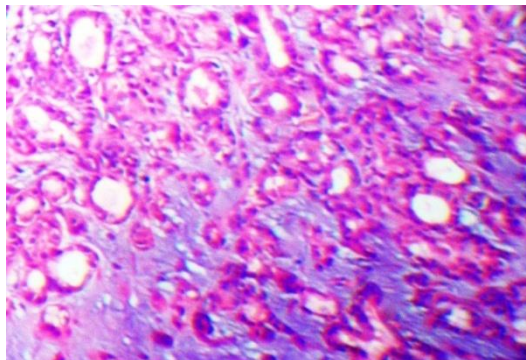


Fig 4: Haematoxylin and eosin stained section showing mixed inflammatory infiltrate.