Necrotising sialometaplasia — A deceptive ulcer of the palate: A case report

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INTRODUCTION
Necrotizing sialometaplasia is a benign self limiting and rare inflammatory disease of the major and minor salivary glands usually the latter. It was first described as a distinct entity by Abrams et al. in 1973 and approximately 300 cases have been reported in the world literature.[1] It mimics epithelial malignancy histopathologically and may lead to radical surgery if misdiagnosed.

It characteristically involves the palate in 80% of the cases, involvement of other sites such as the retromolar pad, gingiva, lip, tongue, cheek, larynx, and trachea have also been reported.[2,3] The incidence in the major salivary glands is approximately 10%. It can occur anytime from 17 to 80 years of age, the mean age being 50 years in men and 36 years in women. The sex predilection tends toward the male population with a ratio of 2:1.[4] The exact etiology is not yet ascertained but local ischemia and many other local factors may be probable reasons.[5]

CASE REPORT
A 31-year-old male reported to a private clinic with the chief complaint of ulcer in the palate of one week duration. The patient was a chronic alcoholic and smoker. He gave a history of gradually enlarging bilateral swelling in the hard palate, which ulcerated later on. He presented with ulcers on the posterior hard palate on either side of the mid palatine suture, the right ulcer measured 0.5 × 1.0 cm and the left ulcer measured 0.5 × 0.5 cm. Both the ulcers exhibited an indurated base with erythematous irregular borders [Figure 1]. It was non-tender on palpation. A provisional diagnosis of necrotizing sialometaplasia was given with mucoepidermoid carcinoma and squamous cell carcinoma as differential diagnoses.

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ABSTRACT
Necrotising sialometaplasia is a rare, locally destructive inflammatory condition of the salivary glands. It gains importance in that it mimics a malignant process both clinically and microscopically. A case of necrotizing sialometaplasia manifesting as bilateral synchronous ulcers in the palate is discussed with emphasis on appropriate diagnosis.

Key words: Bilateral, indurated base, necrotising sialometaplasia, palate, salivary glands
An incisional biopsy was performed and was sent for histopathological examination. On histopathological examination, a hyperkeratinized stratified squamous epithelium with pseudo-epitheliomatous hyperplasia was evident [Figure 2]. Few areas exhibited ulceration [Figure 3]. The connective tissue exhibited areas of coagulative necrosis of salivary gland acini with mucin pooling [Figure 4] and areas of squamous metaplasia of the ducts [Figure 5]. A reactive inflammatory process in a richly vascularized stroma was evident [Figure 6]. A diagnosis of necrotizing sialometaplasia was made. The patient was placed under periodic follow-up, the ulcers healed uneventfully.

DISCUSSION

Necrotizing sialometaplasia is a self-limited inflammatory process, usually manifesting as a painful ulcer with indurated base histopathologically mimicking an epithelial malignancy. The most widely accepted theory propounds ischemia as the main etiological agent for necrotizing sialometaplasia. Denture wear, dental injections, blunt force trauma, habits like alcohol and tobacco use as well as upper respiratory infections have also been proposed to be predisposing factors. The case being discussed is a chronic alcohol and tobacco user.

In most cases necrotizing sialometaplasia usually occurs spontaneously with initial symptoms like fever, chills, malaise, and swelling. The case being discussed had a history of gradually increasing bilateral swelling, which subsequently ulcerated. Most of the cases occurred at the hard palate with two-thirds of the lesion being unilateral and few being bilateral, synchronous, or metachronous. The case being discussed presented with bilateral synchronous ulcerations of the hard palate in the posterior region. The presentation as an ulcer with indurated base simulated a malignant process. Kogh et al. reported a case of unusual necrotising sialometaplasia, which presented as a palatal swelling bilaterally accompanied with complete anesthesia of the palate. Routine hemogram of our case was well within the normal parameters and the test for syphilis was non-reactive.
Hematoxylin and eosin staining remains the gold standard for diagnosis. A detailed case history and a correct histopathologic delineation is necessary in order to avoid misdiagnosis. Anneroth and Hansen described the histopathogenesis of necrotising sialometaplasia by proposing five histological stages: Infarction, sequestration, ulceration, repair, and healing. Histological features range from lobular necrosis, sequestration of necrotic acini, ulceration, pseudo epitheliomatous hyperplasia of adjacent epithelium, squamous metaplasia of ductal epithelium and inflammatory change. The case being discussed exhibits pseudo epitheliomatous hyperplasia of the epithelium, ulceration in few areas, coagulation necrosis of the acini with mucin pooling in few areas, squamous metaplasia of the ductal epithelium, and chronic inflammatory infiltrate. Both squamous cell carcinoma and mucoepidermoid carcinoma are infiltrative tumors and in contrast to necrotising sialometaplasia will have cellular atypia and abnormal mitoses.

**CONCLUSION**

Simple exclusion of the differential diagnoses does not readily give the diagnosis of necrotising sialometaplasia but it is rather a combined set of features, the clinical as well as the histopathologic with the absence of dysplastic cytologic features, which clinches the diagnosis of necrotising sialometaplasia. An apt application of knowledge leading to appropriate diagnosis thereby avoiding mutilating surgery upholds the dictum *primum non noncerum*.

**REFERENCES**


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