Case report

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Necrotizing sialometaplasia of the palate: incidence, etiological factors, clinical features and treatment

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Abstract

Necrotizing sialometaplasia (NS) is a very rare benign formation of the hard palate, of a locally destructive and inflammatory nature, commonly affecting minor salivary glands. Due to NS clinical signs and location, this pathological lesion closely resembles certain carcinomas, such as a mucoepidermoid carcinoma or squamous cell carcinoma, and may therefore result in misdiagnosis and inappropriate courses of treatment.

Aim. The aim of this publication is to summarize and analyze the etiological factors leading to incidence of Necrotizing sialometaplasia, its prevalence in the population, methods of diagnosis, differentiation from other diseases with similar clinical manifestations and its treatment.

Materials and methods. For the purpose of this research, 45 dental literature sources have been reviewed, dated from the first described cases to the present days, published in English. This publication also presents a clinical case of Necrotizing sialometaplasia on the hard palate in the mouth.

Conclusion. Understanding the etiological factors for the occurrence of Necrotizing sialometaplasia, along with its clinical and histological features, can aid in the accurate diagnosis and treatment approach, thus avoiding unnecessary radical and invasive procedures.

Keywords: sialometaplasia, ischemia, self-limiting disease, minor salivary glands

Introduction

Necrotizing sialometaplasia (NS) is a very rare benign formation of the hard palate, of a locally destructive and inflammatory nature, commonly affecting the minor seromucinous salivary glands. It is localized mainly in the middle and posterior sections of the hard palate, and in two thirds of the cases it tends to be unilateral. The first reports describing this type of pathological finding were documented by Abrams et al. in 1973 [1]. Later, Dunlap and Barker also shared data gathered from clinical cases consistent with necrotizing sialometaplasia [2]. Necrotizing sialometaplasia presents as a self-limiting crater-shaped lesion marked by protruding and slightly reddened edges, but it can also appear as a painful or mildly painful swelling or erythema of the palate [3, 4], which is not responsive to local treatment and persists for some time until it resolves.

Aim

The purpose of this publication is to review, summarize and analyze existing research on the etiological factors leading to NS incidence, to track its features throughout the clinical course, to explore the methods of diagnosis and differentiation from other diseases with similar clinical symptoms such as neoplastic processes and chronic inflammatory diseases.

Materials and Methods

For the purpose of this research, 45 dental literature sources have been reviewed, dated from the first described cases to the present days, published in English.
The present paper reviews and summarizes the factors involved in the occurrence of Necrotizing sialometaplasia particularly on the palate among other locations, and its inherent histological features distinguishing it from certain malignant tumors. NS clinical signs and main methods of diagnosis and treatment are also discussed.

Results

Necrotizing sialometaplasia (NS) is a single rapidly growing crater-like lesion most commonly located in the middle and posterior regions of the hard palate. This pathological finding presents as a lesion, well demarcated from the surrounding tissues, with raised edges, sometimes reddish, having a gray-black base, and appears spontaneously, usually re-epithelializing within 3 - 12 weeks without any special treatment. It is most often found on the hard palate, but other locations have also been reported including the soft palate, upper and lower lip, tongue, mucobuccal fold, tonsillar fossa [5], the floor of the mouth [6, 7] and even the nasal cavity, the maxillary sinus and larynx [5, 8, 9]. NS clinical features, its spontaneous appearance and persistent secondary healing can lead to diagnostic and treatment errors since NS lesions closely resemble neoplastic and chronic inflammatory processes. Misidentifying a neoplastic process as necrotizing sialometaplasia and leaving it untreated could be fatal for the patient.

A case report

During a clinical examination of a palatal ulcer at the Faculty of Dental Medicine, Medical University of Varna, Bulgaria, we encountered a case of Necrotizing sialometaplasia of the palate in a 47-year-old female patient.

The finding revealed a single well-demarcated, mildly painful lesion located between the middle and posterior third of the palate, to the left of the midline, matching the described clinical features. The lesion, measuring 17 mm by 12 mm, was well-defined with a crater-like shape, a base covered in gray necrotic tissue, and a raised surface compared to the surrounding tissue. Palpation revealed no tenderness, and no perforation of the palatine bone was observed. Antibacterial therapy (Amoxicillin) was prescribed for seven days to rule out bacterial infection and a biopsy date was scheduled. The patient was advised to maintain meticulous oral hygiene, rinse twice daily with a chlorhexidine solution, and to cease smoking. One week after the prescribed therapy, the lesion was found to have decreased in both size and depth compared to its initial presentation. The patient missed the scheduled biopsy appointment, but a visit for a different dental treatment a month later revealed no palatal pathology. Although a histological examination was not performed to confirm the detected pathological lesion, the clinical features and spontaneous recovery suggested that it was Necrotizing sialometaplasia of a minor salivary gland of the palate.

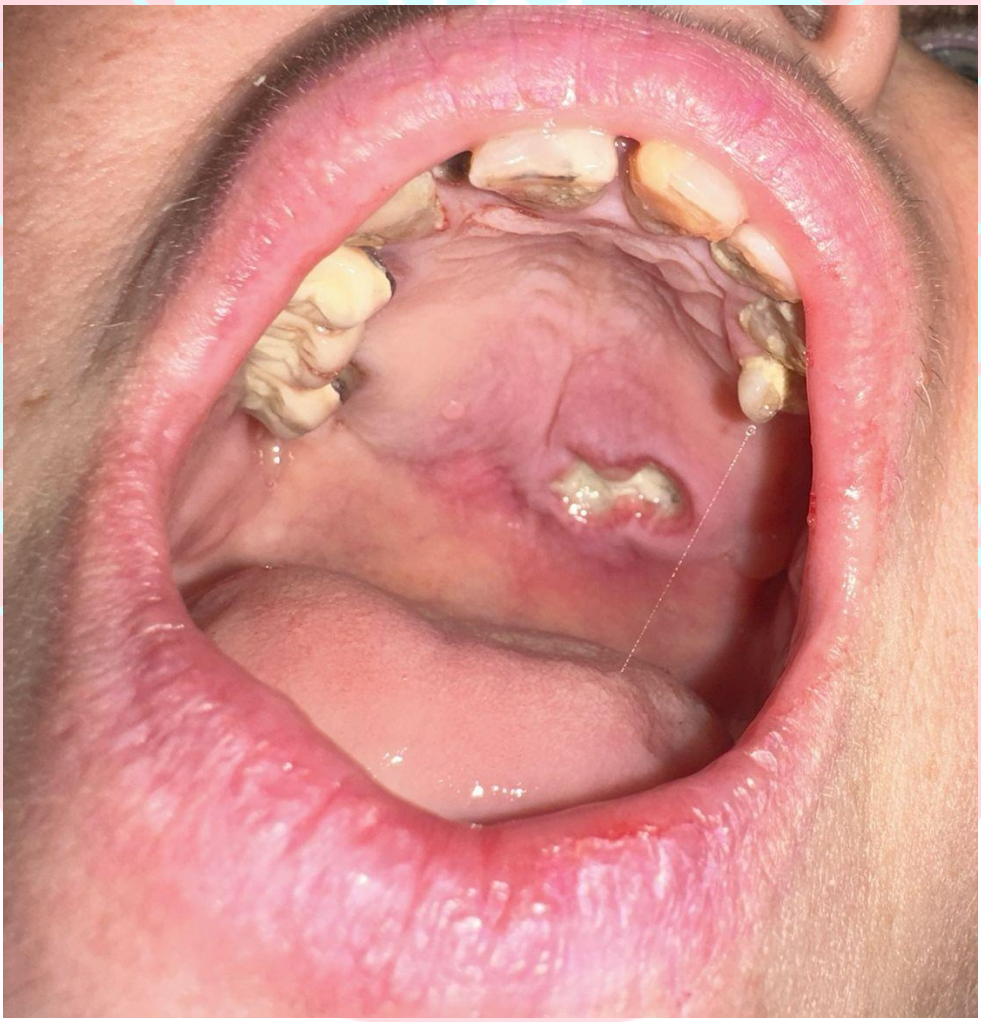

Fig1. Intraoral view during first clinical examination

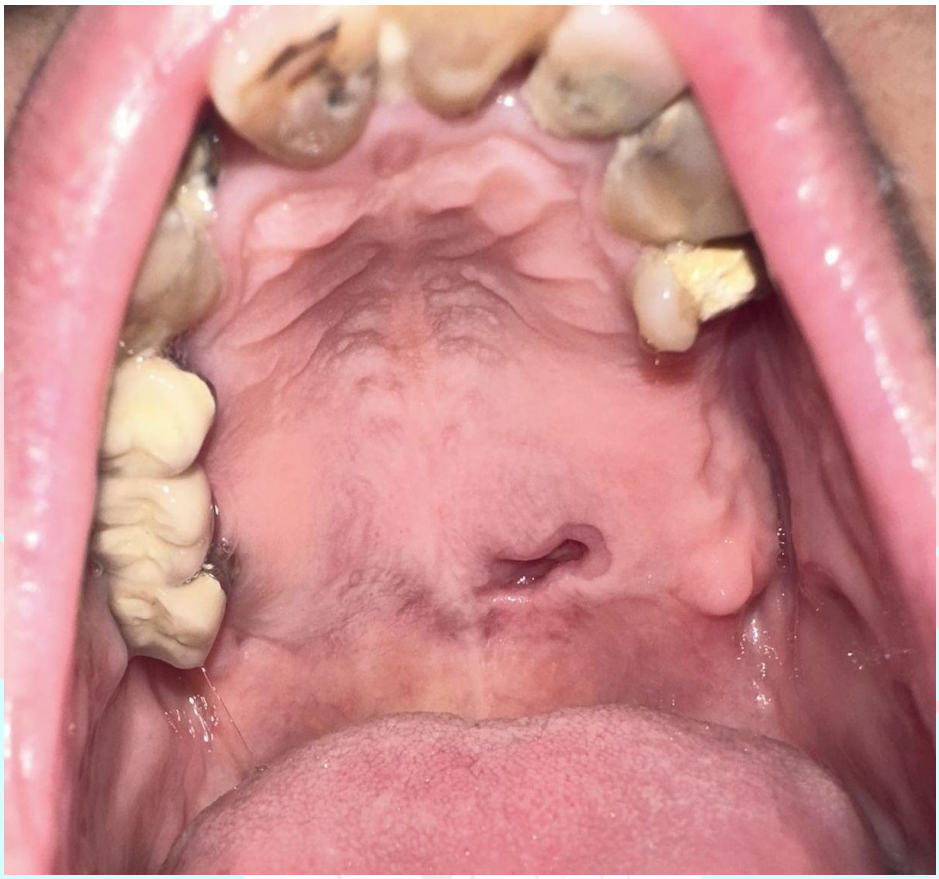

Fig. 2 Intraoral view one week after prescribed therapy

Discussion

Necrotizing sialometaplasia (NS) is a rare benign formation that most commonly affects minor salivary glands on the palate, but it can also occur in other locations where these glands are present. Unilateral localization is characteristic in 71% of cases, while bilateral manifestations are observed in approximately 12% of affected patients [5]. The etiology and pathogenesis of Necrotizing sialometaplasia have not yet been definitively elucidated, despite being the subject of numerous scientific studies and publications. It is assumed that necrosis occurs due to prolonged ischemia, with various physical, mechanical, and chemical factors, as well as biological effects on blood vessels, playing significant roles in this process. Due to the destructive and inflammatory characteristics of this pathological lesion, it is assumed to be associated with harmful habits such as smoking and frequent alcohol consumption [10], as well as upper respiratory tract infections, traumatic damage to the palatal mucosa, surgical procedures, local anesthesia [5,11], and intubation [12, 13].

The use of corrigents to prolong anesthesia and to reduce bleeding may lead to ischemia. Infiltrating anesthetic into an area lacking submucosa, like the hard palate, necessitates applying greater pressure during administration. Experimental studies have shown a direct correlation between repeated applications of local anesthesia to the palate and histological changes in the mucosa that resemble those seen in Necrotizing sialometaplasia [14]. Other predisposing factors include mechanical trauma during intubation,

extubation, or prolonged anesthesia [15]. Using uncomfortable maxillary prostheses can negatively impact the mucosa of the hard palate and potentially lead to necrotizing sialometaplasia [5, 16].

Some diseases and conditions such as bulimia and gastroesophageal reflux are also indicated as a possible cause for the occurrence of Necrotizing sialometaplasia. In cases of bulimia, the repeated insertion of fingers into the mouth to induce vomiting creates conditions conducive to chronic trauma of the palatal mucous membrane, subsequently exposing it to the corrosive effects of stomach contents. Low pH irritates soft tissues and damages tooth structures. This principle underlying the occurrence of Necrotizing sialometaplasia has been documented in a number of publications [15, 17, 18, 19].

Smoking affects the soft tissues in the oral cavity directly and reduces blood flow to the oral mucosa. For this reason, this harmful habit is also regarded as a contributing factor to the development of palatal sialometaplasia [15]. According to a study, the risk of developing Necrotizing sialometaplasia in smokers is estimated to be around 10% [20].

Frequent use of alcohol, especially spirits, can also be a factor in the occurrence of necrotizing sialometaplasia of the minor salivary glands. In most cases, however, it is combined with other harmful habits such as smoking and cocaine use [5, 21, 22].

Certain diseases and conditions associated with an increased risk of thromboembolism may be associated with NS development, such as pancreatic cancer, where a propensity for deep venous thromboembolism due to the cytokines secreted by the tumor has been described [23].

The use of narcotic substances such as cocaine [2, 21], along with local radiotherapy [11], are also identified as causes of Necrotizing sialometaplasia of the palate. Allergic reactions have also been implicated in Necrotizing sialometaplasia of the palate. These factors impact the blood vessels, leading to prolonged ischemia, which can subsequently result in necrosis [5]. Furthermore, some systemic diseases, such as sickle cell disease, Raynaud's disease, Buerger's disease, related to blood supply disorders, have also been associated with NS, confirming ischemic necrosis as the main etiological factor [1, 5, 24]. Another hypothesis links the appearance of this pathological entity to vessel thrombosis. However, there is limited microscopic evidence of this, with only one described case of vasculitis [18, 25]. Nevertheless, the relatively rare incidence of this pathological lesion in the oral mucosa among the large proportion of patients with harmful habits cannot be fully explained, nor can the trigger mechanism for its development be identified.

In terms of incidence, Necrotizing sialometaplasia is more commonly found in males, predominantly in the fourth or fifth decade of life, with Caucasians being more susceptible than African-Americans [5]. On average, 0.03% of all oral biopsies yield evidence of Necrotizing sialometaplasia [26]. Although NS is a rare inflammatory non-recurrent necrotizing lesion, there have been cases where it reappeared in contralateral sites some time after its initial occurrence [24].

According to histological characteristics, NS lesions exhibit regular lobular architectonic structure of the salivary gland, although certain areas feature coagulative necrosis of the mucinous acini [1]. Microscopic features reveal pseudoepithelomatous hyperplasia of the covering epithelium, and squamous metaplasia of ducts and acini with cells exhibiting uniform nucleus with occasional normal mitosis without cellular atypia and mixed inflammatory reaction composed of plasma cells, neutrophils, a great amount of eosinophils and macrophages [1, 27, 28]. Pseudoepithelomatous hyperplasia of the epithelium and metaplasia have historically posed diagnostic challenges due to their strong resemblance to squamous cell carcinoma of epithelial origin. A number of studies have provided evidence of diagnostic difficulties, some of which have resulted in large and unnecessarily radical resections [5, 29, 30]. However, subsequent histological analyses have identified additional features, such as the presence of residual lamina in some metaplastic nests, which are not observed in mucoepidermoid carcinoma and squamous cell carcinoma [31]. Some authors propose that Necrotizing sialometaplasia is caused by immunological or allergic factors, as evidenced by the accumulation of a large number of eosinophilic granulocytes in the lesion [32]. Data indicate a correlation

between the persistence of the lesion and its histological characteristics. For instance, coagulative necrosis is more common in fresh lesions, whereas fibrosis and squamous metaplasia are more dominant features in older lesions [5, 28]. Variations in the histological sections observed microscopically are attributable to the stage at which the biopsy is taken. Based on these characteristics, Anneroth and Hansen have identified several stages in the development of necrotizing sialometaplasia: infarction, sequestration, ulceration, repair, and healing [10].

Werning et al. describe Subacute necrotizing sialadenitis (SANS) as a nonulcerated but painful firm swelling of the posterior hard palate, that persists for a week and then resolves [33]. Some authors believe it should be classified as a distinct nosological entity [34, 35, 36, 37].

Because there is no ulceration as seen in the most typical versions of NS, and healing occurs more quickly, typically within 1 to 3 weeks [35]. The reason why ulcers do not form has not been established, but some suggest it is likely due to an inflammatory reaction associated with thrombosis or the involvement of smaller blood vessels, leading to a limitation of ischemia [25]. Histopathologically, it fully corresponds to classic Necrotizing sialometaplasia of minor salivary glands [34].

When a definitive diagnosis of NS cannot be made, immunohistochemical methods can be employed to demonstrate the lack of immunoreactivity for p53, low reactivity for MIB1 (KI-67), and the presence of calponin-positive myoepithelial cells [17, 38].

From a clinical point of view, NS of the palate is a rapidly developing, well-defined crater-like lesion, with slightly raised edges, measuring 1 - 3 cm in diameter. It is typically located lateral to the midline of the palate as a single unilateral lesion; however, according to literature, it can also be bilateral in 20% of reported cases [5]. Subjective complaints most often include a moderately to slightly painful wound, a feeling of discomfort, or a painful swelling. Some patients report altered sensitivity or acute pain preceding mucosal changes, likely due to vascular disorders and subsequent ischemia and necrosis [17, 28, 39].

Additional symptoms include a painless wound on the palate and impaired or absent sensitivity along the course of the n. palatinus major, preceding the appearance of edema and subsequent ulceration [28].

Diagnosis is based on the patient's history, particularly the appearance of a relatively rapidly formed crater-like wound on the palate, which may be painful or slightly painful and occurs without preliminary signs. Imaging studies, such as conventional X-ray examinations, CT, CBCT, MRI scans need to be appointed to visualize the condition of the palatine bone. In NS, the bone remains intact since ischemic necrosis affects only soft tissues, while in some diseases (carcinomas and syphilis) with a less favorable prognosis there is a disruption of the bone’s integrity. As previously mentioned, five histological stages can be identified (infarction, sequestration, ulceration, repair, and healing), depending on the time of biopsy since the onset of the lesion. Consequently, in biopsies performed some time after the onset of NS, the necrosis required for diagnosis may not be detected, with pseudoepitheliomatous hyperplasia being predominantly observed instead [15].The presence of necrosis is not always considered an indication for histological examination and may not be prominently represented in the lesion itself, depending on the stage of the lesion. Therefore, we recommend waiting before performing a biopsy, especially if radiological findings indicate that the integrity of the underlying palatal bone has not been disturbed [15]. Such lesions should be monitored to exclude the possibility of a neoplastic process.

Given the clinical characteristics and location on the palate, the differential diagnosis of Necrotizing sialometaplasia should include mucoepidermoid carcinoma, squamous cell carcinoma, adenoid cystic carcinoma and other neoplasms. Unlike NS, these conditions typically have a longer development period, do not spontaneously resolve, and are often painless or slightly painful. Other pathological lesions to be differentiated from NS include bacterial infections, dental fissures, major aphthae, and chronic inflammatory diseases such as syphilis, tuberculosis, or granulomatous processes localized on the hard palate [40].

The differential diagnosis should also include lesions that develop relatively quickly and can cause ulceration of the palate, such as Wegener's granulomatosis. Wegener's disease is a rare systemic autoimmune condition that results in vasculitis of the small blood vessels in the upper respiratory tract and is characterized by necrotizing granulomas. Other diseases that may resemble NS include zygomycosis, lymphoma, palatal perforations due to prolonged cocaine use, tertiary syphilis, and others.

Accurate diagnosis becomes more challenging when necrotizing sialometaplasia occurs in atypical locations, such as the lining of the cheek [41].

When histological findings are consistent with NS but there is suspicion of neoplastic processes, it is recommended to use specific markers such as MIB-1 (Ki-67), p53, calciponin and pattern cytokeratin expression [15, 17, 42].

Elevated values of Ki-67 and p53, for example, are indicators of malignancy [17]. Literature also describes cases where NS and adenoid cystic carcinoma co-occur and are observed histologically within the same lesion [43]. Misdiagnosing a neoplastic process as necrotizing sialometaplasia of a minor salivary gland could have fatal consequences for the patient's prognosis.

Complete healing through secondary epithelialization, without specific treatment, is usually observed after about 3 - 12 weeks [5, 28]. Recurrence is uncommon, though several cases have been documented [24, 44]. There is no established protocol for active treatment based on literature, although corticosteroids and antibiotics, such as triamcinolone (3 x 100 mg injections at weekly intervals), have been administered, likely due to the inflammatory component. Despite their use, no significant difference in the healing process has been observed [28]. Typically, the lesion fills with granulation tissue and fully epithelizes within about three months, although in most cases, healing may occur earlier [45].

Conclusion

Necrotizing sialometaplasia is defined as a rare, self-limiting, crater-like benign lesion that typically resolves within 3 - 12 weeks, followed by complete epithelialization. During this period, mainly supportive and symptomatic treatments are applied. For this reason, mandatory diagnostic biopsy and surgical excision are rarely required for NS, especially in the absence of radiological evidence of palatine bone involvement. Understanding the etiological causes, along with the clinical and histological characteristics of necrotizing sialometaplasia, can aid in proper diagnosis and optimize the healing approach.

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