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·防治实践·

腭部坏死性唾液腺化生1例及文献回顾

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【摘要】 目的 探讨坏死性唾液腺化生的临床特征、病理表现、诊断要点及鉴别诊断，为该病的诊断与治疗提供依据。**方法** 本研究已通过单位医学伦理委员会审查批准，并获得患者知情同意。回顾1例发生于右侧软硬腭交界处的坏死性唾液腺化生患者资料，结合其临床表现、影像学及组织病理学的检查结果，综合分析其诊断过程，并对该病的相关文献进行复习回顾。**结果** 患者为24岁男性，既往有咀嚼槟榔史（半年余，2颗/d），口内检查见右侧软硬腭交界处一不规则弹坑状溃疡（3 mm × 6 mm × 5 mm），深达骨面。螺旋CT显示局部软组织缺损，周围骨质未见明显破坏。组织病理学提示黏膜及小唾液腺组织慢性炎症，未见恶性改变。最终诊断为坏死性唾液腺化生。经局部抗炎后，溃疡于就诊3周后完全愈合。文献复习结果表明，坏死性唾液腺化生是一种罕见的良性唾液腺疾病，好发于硬软腭交界处，多见于40~60岁的中年男性。其病因未明，目前普遍认为与机械刺激引发的局部缺血致唾液腺小叶梗死相关。因其临床表现近似恶性肿瘤，常被误诊。治疗以局部抗炎防腐促进愈合为主。**结论** 坏死性唾液腺化生是一种良性自限性疾病，临床表现与恶性肿瘤相似，易导致误诊。诊断需结合临床、影像及病理结果综合判断。治疗以保守对症为主。

【关键词】 口腔黏膜病；坏死性唾液腺化生；溃疡；腭部；自限性；槟榔；组织病理；口腔恶性肿瘤

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Necrotizing sialometaplasia of the palate: a case report and literature review

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【Abstract】 Objective To enhance the recognition of necrotizing sialometaplasia (NS) by elucidating its clinical, pathological characteristics and key diagnostic points, providing a basis for the diagnosis and treatment of the disease. **Methods** This study has been reviewed and approved by the Medical Ethics Committee, and informed consent has been obtained from patients. Review the data of a patient with NS occurring at the junction of the right soft and hard palate, and comprehensively analyze its diagnostic process based on its clinical manifestations, imaging, and histopathological examination results. And review the relevant literature on the disease. **Results** This study describes a 24-year-old male patient with a documented betel nut habit (2 pieces/day for >6 months), who presented with a bone-deep, irregular crateriform ulcer (3 mm × 6 mm × 5 mm) localized to the right hard-soft palate junction. Spiral CT showed a local soft tissue defect with no apparent underlying bone destruction. Histopathology demonstrated chronic inflammation

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of the mucosal and minor salivary gland tissues, with no evidence of malignancy. A final diagnosis of NS was established. The ulcer healed completely three weeks after initiation of local anti-inflammatory therapy. A literature review indicates that NS is a rare, benign salivary gland disorder, typically occurring at the hard-soft palate junction in middle-aged men (40-60 years). Its etiology remains unclear, but it is widely attributed to salivary lobe infarction following mechanical trauma-induced ischemia. Due to its clinical resemblance to malignancy, it is often misdiagnosed. Treatment entails local anti-inflammatory measures and meticulous wound care aimed at promoting mucosal healing. **Conclusion** NS is a self-limiting, benign condition that poses a significant diagnostic challenge due to its close clinical simulation of malignancy. Thus, accurate diagnosis requires a combined assessment of clinical presentation, radiological features, and pathological findings. Treatment is predicated based on a conservative strategy with an emphasis on symptomatic management.

【Key words】 oral mucosal diseases; necrotizing sialometaplasia; ulcer; palate; self-limiting; betel nut; histopathology; oral malignant tumors

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坏死性唾液腺化生(necrotizing sialometaplasia,NS)是一种较为罕见、具有自限性的良性唾液腺病变,多发生于软硬腭交界区域的唾液腺组织[1]。病变多表现为快速进展的无痛性或疼痛性溃疡或肿胀,典型者呈“弹坑样”外观,因其形态近似恶性肿瘤,常引发患者严重的恐癌心理[2]。坏死性唾液腺化生的发病机制尚不完全明确,目前普遍认为与局部缺血所致唾液腺小叶梗死密切相关[3]。尽管坏死性唾液腺化生具有自愈倾向,其临床表现和组织病理学特征却与鳞状细胞癌、黏液表皮样癌等恶性肿瘤高度相似,易导致误诊及过度治疗[4]。因此,深入开展对坏死性唾液腺化生的临床识别与病理鉴别研究,对于避免临床过度治疗、提升诊断准确性具有重要临床意义。本文拟报道1例坏死性唾液腺化生患者病例资料并进行相关文献复习,为坏死性唾液腺化生的诊断与治疗提供依据。

1　病例资料

1.1　病史

患者,男,24岁,1周前右上腭部无明显诱因“起疱”,自行戳破后形成溃疡,稍不适。患者怀疑病损恶变,遂来我院就诊。否认吸烟、饮酒史,有咀嚼槟榔史半年余(2颗/d),戒槟榔2月余。否认系统性病史及药物过敏史。本病例收集经患者知情同意及南京大学医学院附属口腔医院伦理委员会审核批准(NISH-2025NL-030)。

1.2　专科检查

右侧软硬腭交界处见一不规则弹坑状溃疡,溃疡约3 mm × 6 mm × 5 mm,深达骨面,溃疡边缘发白,质地稍韧,基底部糜烂,伴白色坏死物,触痛不明显。软腭后份黏膜发白,质地稍韧,触及纤维条索,张口度正常。腭部病损附近牙体组织未及明显缺损,未见锐利牙尖。右侧面部上颌窦区无压痛,无鼻塞(图1a)。

1.3　实验室检查

血常规、肝肾功能、血糖、凝血功能未见明显异常。人类免疫缺陷病毒、乙型肝炎病毒、丙型肝炎病毒、梅毒螺旋体检查显示阴性。

1.4　影像学检查

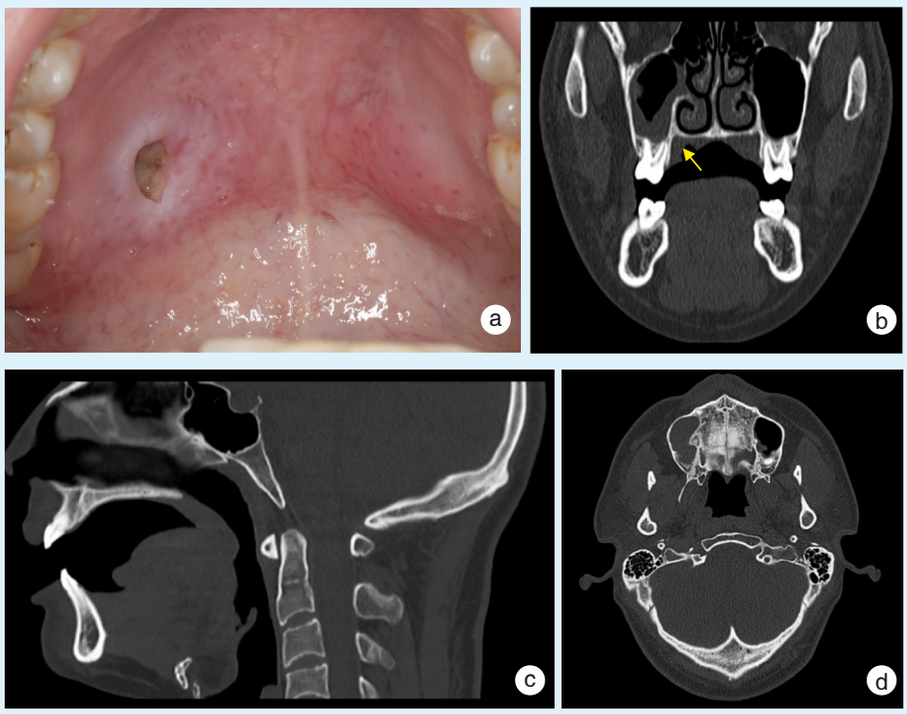
螺旋CT示:右腭部后份,相当于右上颌第一、二磨牙区腭侧可见一圆弧形软组织缺损,轴位最大切面约1.36 cm × 0.58 cm,边界影尚清,深度部分接近骨面,周围骨质未见明显吸收,缺损区前后界软组织稍增厚,右侧腭大孔及翼腭管未见明显增宽影;右上颌窦实腔内软组织影,上下颌骨骨质结构尚完整,颅底骨质结构尚完整,所示双侧颌下区及颈深上区未见明显肿大淋巴结影(图1b~1d)。

1.5　组织病理学检查

经患者知情同意,局部麻醉下切取右侧上腭溃疡病损组织,送病理检查,激光热凝止血。HE检查示:“右上腭”黏膜及小唾液腺组织轻度慢性炎症伴散在嗜酸性粒细胞浸润,灶性区上皮下裂隙形成(图2a)。建议进一步结合临床。

1.6　免疫组化检查

经患者知情同意,于本院病理科行免疫组化染色,结果显示固有层中Ki-67(散在+):见于炎症



a: a clinical intraoral photograph showing an irregular, crateriform ulcer (approximately 3 mm × 6 mm × 5 mm) at the right hard-soft palate junction, extending to bone with raised whitish margins and necrotic base. The posterior soft palate mucosa appears whitish and firm in consistency, with palpable fibrous bands. The interincisal mouth opening is within normal limits. No dental defects or sharp cusps were noted adjacent to the lesion. The right maxillary sinus area was nontender without nasal obstruction. b: coronal view. The spiral CT scan reveals a well-defined, arc-shaped soft tissue defect in the posterior right palate (as an arrow showed), adjacent to the maxillary molars, which extends to the intact bone surface without erosion. Soft tissue opacity is seen in the right maxillary sinus. c: sagittal view. No evidence of significant bone resorption is seen in the surrounding palatal region. d: axial view intact right greater palatine foramen and pterygopalatine canal; with right maxillary sinus opacity

Figure 1　Clinical manifestations and spiral CT examination of a 24-year-old male patient with necrotizing sialometaplasia of the palate

图1　24岁男性腭部坏死性唾液腺化生患者的临床表现及螺旋CT检查

细胞与腺上皮；p53(散在弱+)：符合野生型表达模式；p63(弱+)：见于导管肌上皮；p16(−)：提示该病变不是由高危型HPV感染所致；CK5/6(+)：阳性表达于导管肌上皮及鳞状化生的腺上皮(图2b~2f)。

1.7　诊断

结合临床、实验室检查、影像学检查、HE及免疫组化检查结果，最终诊断：主要诊断：右上腭坏死性唾液腺化生；次要诊断：①口腔黏膜下纤维化；②右上颌窦炎。

1.8　治疗方案

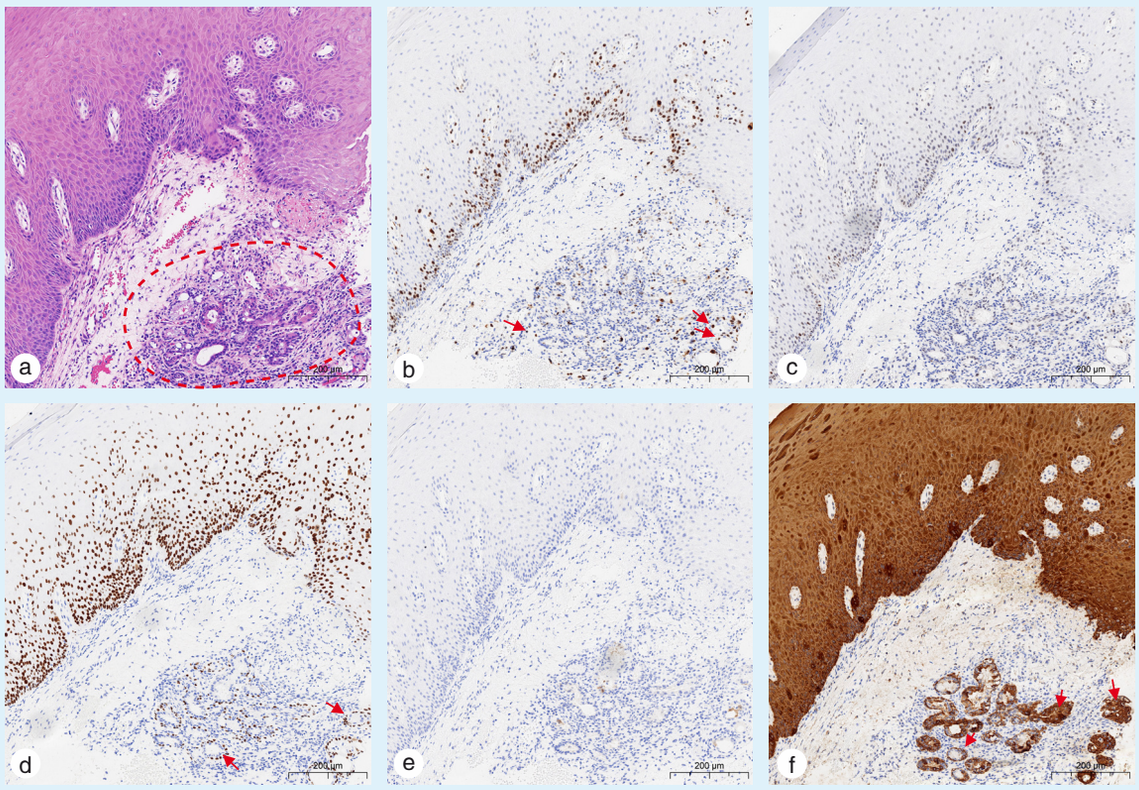
治疗以局部用药控制感染、减轻炎症并促进组织再生为主。给予葡萄糖酸氯己定含漱液，含漱每次3 min，3次/d；曲安奈德乳膏，局涂，3次/d。嘱患者清淡饮食，禁食辛辣刺激、硬、粗糙食物，保持口腔卫生，1、2、3周后复诊。

2　治疗结果

治疗1、2、3周后复诊，1周后复诊：见腭部溃疡面积因活检手术较之前变大，患者诉疼痛感基本消失。2周后复诊：溃疡面积显著缩小约50%，周围组织充血、水肿基本消退，表面可见新生上皮覆盖，基底柔软。3周后复诊：溃疡完全愈合，黏膜颜色、质地基本恢复正常，未见明显瘢痕形成。在整个治疗及随访期间，患者未诉任何不适，药物耐受性良好(图3)。

3　讨　论

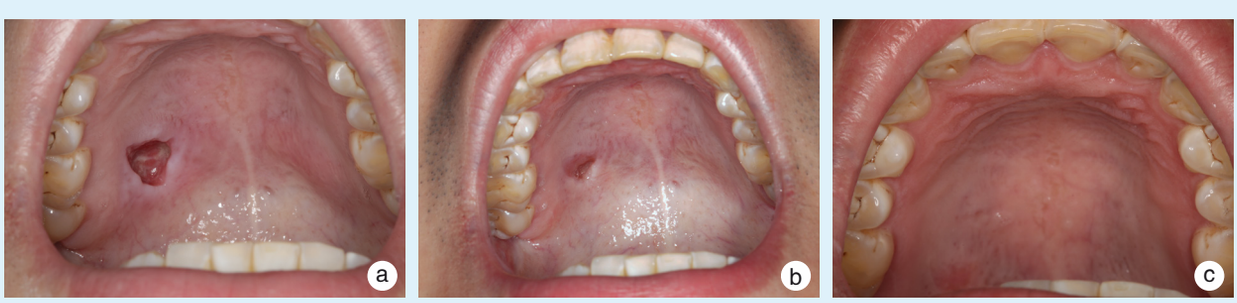
坏死性唾液腺化生是一种罕见的良性自限性唾液腺疾病，其临床及病理表现与恶性肿瘤极具相似性，常导致诊断困难[5]。本文通过1例发生于腭部的坏死性唾液腺化生病例，从多角度进行系统分析，以深化对该病的认识并指导临床实践。



a: HE staining(×200) reveals mild chronic inflammation with scattered eosinophil infiltration in the mucosa and minor salivary gland tissue of the right upper palate(as indicated within the red circle). Immunohistochemical results within the lamina propria: b: Ki-67 (×200); scattered positivity in inflammatory cells and glandular epithelium (as indicated by the arrows); c: P53 (×200); scattered weak positivity indicative of wild-type expression; d: p63 (×200); weak positivity in ductal myoepithelial cells(as indicated by the arrows); e: p16 (Negative)(×200): this finding is suggestive of an etiology independent of high-risk HPV infection; f: CK5/6 (×200); positive in ductal myoepithelium and in glandular epithelium with squamous metaplasia (as indicated by the arrows)

Figure 2　HE and immunohistochemical staining of palatal lesion tissue of a 24-year-old male patient with necrotizing sialometaplasia of the palate

图2　24岁男性腭部坏死性唾液腺化生患者上腭病损组织的HE及免疫组化染色



a: clinical manifestations after one week of treatment. The ulcer was temporarily enlarged due to the biopsy. b: clinical manifestations after two weeks of treatment. The ulcer area was reduced by approximately 50% with visible re-epithelialization. c: clinical manifestations after three weeks of treatment. The ulcer showed complete healing without scarring

Figure 3　Clinical manifestations after treatment of a 24-year-old male patient with necrotizing sialometaplasia of the palate

图3　24岁男性腭部坏死性唾液腺化生患者治疗后的临床表现

坏死性唾液腺化生于1973年由Abrams等[6]首次报道。流行病学研究表明,本病具有明显的性别倾向性,好发于男性,可发生于任何年龄段,但以40~60岁的中老年人群最为常见[7]。本病例患

者为24岁男性，与上述一般好发人群的年龄分布存在一定差异。在发病部位方面，坏死性唾液腺化生具有较典型的分布特征：约75%以上的病例发生于硬腭后份近软硬腭交界处[8]。此外，病变也可出现在其他唾液腺分布区域（如唇、颊、扁桃体等），甚至偶见于喉、鼻咽等部位[9-12]。本例病变位于腭部，符合坏死性唾液腺化生最常见的发生位置。

坏死性唾液腺化生的本质是唾液腺小叶的缺血性梗死[13]。其发病机制涉及多种因素，包括局部创伤（牙科注射、假牙佩戴）[14-15]、剧烈呕吐[16]、长期吸烟[17]、与动脉粥样硬化或血管炎相关的全身性疾病[18-19]，以及酗酒等其他因素[20]。这些因素可通过引起腭大动脉分支的痉挛、栓塞或物理性压迫，最终导致唾液腺小叶急性缺血，继而引发腺泡坏死、炎症反应及导管上皮的修复性鳞状化生[21]。Yoshimoto等[22-23]最新研究指出，缺氧会直接引发唾液腺腺泡坏死，并促使残留的肌上皮细胞通过转化生长因子-β3（transforming growth factor-β3，TGF-β3）信号通路导致导管鳞状化生，从而推动坏死性唾液腺化生的早期病变发展，这为理解坏死性唾液腺化生的病理过程提供了新的分子机制解释。本病例患者曾有咀嚼槟榔史，一方面，槟榔粗硬的纤维构成一种长期的机械刺激作用于口腔黏膜，导致黏膜更容易出现溃疡病损；另一方面，其含有的槟榔碱通过激活“活性氧-内质网应激-磷酸化细胞外调节蛋白激酶/Yes相关蛋白”信号轴，介导血管内皮细胞发生内皮—间质转化，从而转分化为成纤维样细胞并异常分泌胶原，最终导致微血管周围纤维化沉积[24]。同时，槟榔碱还能刺激上皮细胞释放转化生长因子-β1，进而促使成纤维细胞过量表达血栓反应蛋白1（thrombospondin 1，THBS1），而高表达的THBS1通过抑制内皮细胞功能与血管生成，导致黏膜血管减少，直接加剧组织缺氧[25]。此外，槟榔碱本身也可诱导内皮细胞自噬并抑制其血管形成能力[26]。而组织局部缺血是坏死性唾液腺化生发病的重要因素。因此，咀嚼槟榔可能是机械与化学刺激共同作用增加坏死性唾液腺化生的发病率。

坏死性唾液腺化生的临床表现易与恶性肿瘤混淆。典型表现为快速出现的单侧腭部黏膜深大溃疡，边界清晰，周缘隆起发红，溃疡中心凹陷覆盖伪膜，底部可触及硬结。在某些情况下，黏膜表面完好无损，病变凸起，易误判为脓肿[2]。其特征性的临床特点是“发展迅速”与“症状轻微”的不匹配：虽然溃疡面较大、形态可怖，但患者疼痛感通常不明显或仅为轻度疼痛，部分患者在溃疡出现前可有麻木或不适感[16]。病变通常在6~12周内自发愈合，这一时间特征对诊断具有重要提示意义[16]。本病例中，患者溃疡在1周内迅速发展，但无明显不适。值得关注的是，经药物局部治疗约3周后，病损即迅速愈合，较文献报道的自然病程更为缩短。

Abrams等[6]将坏死性唾液腺化生的主要组织学特征概括为以下几点：①唾液腺小叶的缺血性坏死；②尽管存在明显的坏死和炎症，但仍保留完整的小叶结构轮廓；③导管及腺泡的鳞状化生，邻近小叶内可见黏液或坏死碎片；④细胞核形态呈良性，虽可见正常核分裂象；⑤腺体内及周边可见显著的急、慢性炎症反应及肉芽组织形成。此外，免疫组化染色在疑难病例的鉴别诊断中具有重要辅助价值。坏死性唾液腺化生通常表现为良性免疫表型：Ki-67（散在+）、p53（散在弱+）、p63（弱+）[27]，而CK5/6（+）则证实了细胞的鳞状上皮来源。上述指标共同构成与鳞状细胞癌等恶性肿瘤鉴别的关键依据[28-29]。然而必须强调，免疫组化结果需始终与其他检查相结合进行综合判断，不可作为独立诊断依据。本病例虽未呈现典型的坏死性唾液腺化生组织学特征，但亦未见明确恶性证据。此现象一方面可能是该病例在病理上表现为非典型的坏死性唾液腺化生特征有关；另一方面可能与取材范围有限（切取病变边缘0.3 cm组织，深达骨面），所获组织可能主要反映周边炎症反应区。为明确诊断，建议在距病变边缘0.5~1 cm处深切取材，以确保获取更具代表性的组织进行病理评估。

在坏死性唾液腺化生诊断中，MRI和CT的核心价值不在于确诊，而在于评估病变范围、排除其他疾病、发现提示其良性本质的线索，从而避免将这一良性病变误诊为癌症而导致不必要的根治性手术[30]。坏死性唾液腺化生在CT上通常表现为软组织增厚或缺损，其深面的骨组织完全正常，无骨质破坏征象[31]。这一特点与鳞状细胞癌或黏液表皮样癌等恶性肿瘤可能伴随的骨质破坏形成明显对比[32-33]。值得注意的是，当坏死性唾液腺化生病变进展严重时，也可能导致骨质穿孔，在影像学上极易与恶性肿瘤的骨质破坏相混淆，从而显著增加临床鉴别诊断的难度[31]。本病例螺旋CT表

现支持良性病变诊断，为坏死性唾液腺化生的诊断提供了良性依据。

坏死性唾液腺化生的鉴别诊断涉及多种具有溃疡表现的良恶性疾病（表1）。首先要与良性溃疡区分。重型复发性阿弗他溃疡具有周期性发作的病史与愈后留瘢痕的特点，组织学上仅为非特异性炎症，无腺体坏死或化生[34]。结核性溃疡无自限性，组织病理学可见典型的结核结节，且胸部X线可能发现肺结核病灶，这些特征可与坏死性唾液腺化生明确区分[35]。其次需与恶性病变进行鉴别。鳞状细胞癌与坏死性唾液腺化生均可表现为腭部溃疡，但前者具有浸润性生长方式、显著的细胞异型性及病理性核分裂象，影像学常提示侵袭性骨破坏[36]。在唾液腺源性肿瘤中，黏液表皮样癌的特征性表现为由黏液细胞、表皮样细胞及中间细胞构成的异质性肿瘤巢，常伴囊腔形成[37]，而腺样囊性癌则以筛状或管状结构及显著的神经周侵犯为标志[38]。

表1　坏死性唾液腺化生与其他溃疡类疾病的鉴别要点

Table 1　Key points for differential diagnosis of necrotizing sialometaplasia and other ulcerative diseases

Disease	Age	Sites	Clinical presentation	Periodicity	Self-limiting	Histological presentation	Imaging
Necrotizing sialometaplasia	Middle-aged (40-60 years)	Junction of soft and hard palate	Large, deep ulceration, potentially extending to bone, edges may be raised, base with granulation tissue	No	Yes	Preservation of salivary lobular architecture; lobular necrosis; squamous metaplasia of acini and ducts; inflammatory cell infiltration	Possible pressure resorption of bone, no malignant bone destruction
Major recurrent aphthous ulcer	Young and middle-aged adults	Posterior oral cavity	Deep ulcer, regular and well-defined margins, heals with scarring	Yes	Yes	Non-specific inflammatory infiltrate; no epithelial dysplasia	Not applicable
Tuberculous ulcer	Young and middle-aged adults	Lip, vestibular groove, tongue	Deep ulcer, irregular shape with undermined edges, granulation tissue at base	No	No	Tuberculous granulomas with epithelioid cells, Langhans giant cells, lymphocytes, and central caseous necrosis	Chest X-ray may show pulmonary tuberculous lesions
Squamous cell carcinoma	Middle-aged and elderly	Ventral and lateral tongue, floor of mouth	Ulcerovegetative mass with indurated, rolled edges. Surface may be granular, necrotic, or cauliflower-like	No	No	Infiltrating squamous cell nests; marked cellular atypia (hyperchromasia, pleomorphism, pathological mitoses); keratin pearls	CT/MRI demonstrates invasive bone destruction and lymph node metastasis
Adenoid cystic carcinoma	Middle-aged and elderly	Palate, tongue, buccal mucosa	Round/nodular mass, infiltrative, frequent perineural spread (numbness/pain), may perforate palate	No	No	Composed of ductal and myoepithelial cells forming tubular, cribriform, or solid patterns; prominent perineural invasion	CT: bony erosion/destruction. MRI: nerve thickening (*e.g.*, enlarged greater palatine canal)
Mucoepidermoid carcinoma	All age groups	Palate, retromolar area	Painless mass; blue/purple mucosa (mucin); low-grade: cyst-like; high-grade: resembles squamous cell carcinoma	No	No	Composed of mucous cells, epidermoid cells, and intermediate cells forming tumor nests; with cystic spaces and mucus lakes lined by tumor epithelium	Low-grade: well-circumscribed and cyst-like. High-grade: infiltrative, with potential bone destruction

在坏死性唾液腺化生的治疗中临床医生的首要关键是正确诊断坏死性唾液腺化生，保护患者免受不必要的、破坏性的手术创伤。成功的治疗不依赖于复杂的手术或药物，而建立于正确的诊

断、耐心地解释和建立患者信心的基础之上。也有研究学者根据坏死性唾液腺化生的病损面积总结出治疗建议[39]：①对于直径0.5~1 cm的小型病变，应观察等待，无需积极治疗，此类病灶具备自愈能力，部分病例可在3 d内自行消退；②对于约1.5 cm的中等病变，可考虑使用光生物调制疗法，通过低强度激光刺激细胞代谢，加速伤口愈合并控制继发感染[40]；③对于3 cm×4 cm的大型或复杂病变，则需进行外科清创联合旋转带蒂腭瓣修复术，但其愈合周期较长，可达3个月[41]。

本病例提示临床医生面对极易误诊的腭部深大溃疡，在病损组织病理学特征不典型的情况下，可通过临床表现、影像学检查及免疫组化等多种手段综合评估，排除恶性可能。对于确诊为坏死性唾液腺化生的患者应采取局部对症治疗与随访观察为主的保守策略，以最小干预获最大治疗效果。但对于极少数病损面积较大的病例，手术清创后再覆盖瓣膜对加速病损愈合有极大帮助。

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