Review Article

Palatal Perforation, a Morbid Clinical Entity in the Oral Cavity

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Abstract

The palate is a favorable site of the oral cavity for soft tissue lesions. Palatal perforation is an uncommon clinical condition. It may result from inherited or acquired factors. The acquired causes include infections, malignancy, and certain drug abuse. There are different factors such as diseases process or effects of certain drugs can affect the blood supply leading to tissue ischemia and lesions in the palate. Chronic necrosis of the epithelium lining the palate may destroy the bone to leave a bony sequester that eventually results in palatal perforation. The two most typical symptoms of an oronasal fistula are speech difficulties and nasal regurgitation. For histological study, palatal lesions should be biopsied. It is necessary to get the proper cultures and stains for fungus, bacteria, and mycobacteria. Depending on the underlying etiology, palatal perforations have different treatment options and prognoses. The treatment of palatal perforation and prognosis vary depending on the cause. This review focuses on details of palatal perforation including its etiopathology, classification, clinical features, and its current management.

Keywords: Cocaine abuse, oronasal fistula, palatal perforation, palate, tertiary syphilis

Introduction

Palatal perforation is an uncommon condition due to chronic necrosis of the palatal bone which results in communication between the oral and nasal cavities.[1] There are several causes for resulting in palatal perforations, and they range from infective to noninfective etiologies. The perforation of the palate can also be classified as congenital or acquired as per the etiological origin. Diagnosing palatal perforation is facilitated by thoroughly documenting the patient's history and conducting an assessment focused on factors such as living in areas where fungal infections are common, past substance abuse, and potential risk factors for acquired immunodeficiency.[2] Depending on the underlying etiology, palatal perforations have different treatment options and prognoses. There is specific management of the palatal perforation which includes sealing the defect or repairing the defect. Obturators remain a simple and effective option for managing palatal perforation. Obturators are also successful methods for managing masticatory and speech problems.[3] Surgical repair is also an important option for managing palatal perforation. Closing the oronasal fistula has involved a wide range of surgical techniques, from tiny local flaps to microvascular tissue transplants.[3] This review aims to summarize data related to etiopathology, classifications, clinical features, diagnosis, and current treatment options of palatal perforation.

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Methods of Literature Search

Research articles regarding palatal perforation were searched via multiple approaches. Literature reviews were conducted for considering scientific papers for word palatal perforation. A search strategy following the Preferred Reporting Items for Systematic Reviews and Meta-analyses for narrative review guidelines was devised. This strategy focused on identifying abstracts of published articles, while additional research articles were identified manually through citation tracking. Randomized controlled trials, observational studies, comparative studies, case series, and case reports were assessed for eligibility in this review. A total of 48 articles (20 case reports, 12 case series, and 16 original articles) were identified across various databases, with 42 of them meeting the inclusion criteria and being included in the review [Figure 1]. This article reviews the details of the palatal perforation. This review does not require ethical approval.

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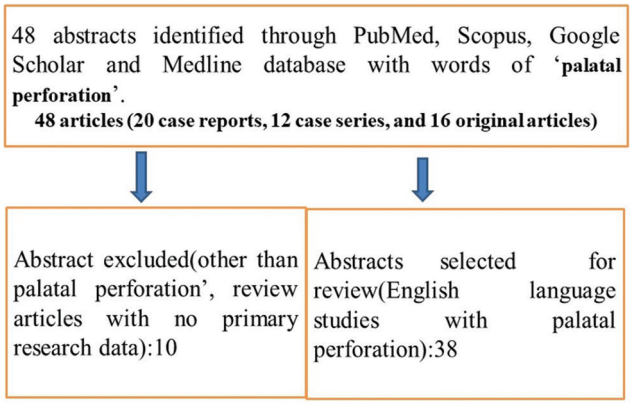

Figure 1: Method of literature search

Etiopathology

The palate benefits from a robust blood supply facilitated by the greater and lesser palatal arteries. This vascular network likely plays a crucial role in wound healing and metabolic support by delivering oxygen and nutrients to the area.[4] The palatal tissues are firmly adherent to the underlying bone and it is relatively dense. Clinical conditions that compromise the blood supply, through vasoconstriction, deprive the tissue of its required sustenance leading to necrosis of the overlying epithelium. The smooth muscle contraction within the wall of the arteries may cause transient ischemia of the structures distally with tissue necrosis.[5] Palatal perforations can arise from a range of causes, including both infectious and noninfectious pathologies. Notably, intranasal drug abuse is increasingly common among younger individuals. Studies suggest that nasal insufflation of substances such as cocaine, heroin, other opioids, stimulants, benzodiazepines, and diet pills may result in palatal perforations.[6] The local presentation of cocaine abuse depends on its route of administration. Snorting often results in isolated perforation of the nasal septum but also can lead to hard palate perforation.[7] Cocaine is one of the psychoactive drugs used worldwide, being extracted from the leaves of *Erythroxylum coca* plant.[7] Its abusive use can aggravate different consequences in the human body including manifestations in the oral cavity. Cocaine abuse can cause midpalatal perforations. The vasoconstrictive and caustic effect of this drug can result in direct irritation and ischemia of the nasal and palatine mucosa, leading to palatal perforation and oronasal fistula secondary to maxillary bone destruction.[8] Rhinoliths are calcified masses present in the nasal cavity, leading to erosion of the palatal bone and causing palatal perforation.[9] The longstanding presence of rhinoliths can cause ulceration of the nasal mucosa and bone destruction at the floor of the nasal cavity.[9] Infective causes for resulting palatal perforation include leprosy, tertiary syphilis, tuberculosis, rhinoscleroma, naso-oral blastomycosis, leishmaniasis, actinomycosis, histoplasmosis, coccidioidomycosis, and diphtheria.[10] Granulomatous lesions such as those seen in tuberculosis often manifest as chronic, singular, nonhealing ulcers characterized by irregular, indurated margins that are undermined, with necrotic bases.[11] Palatal involvement can result in ulcerations, granuloma formation, or perforation, with a higher incidence typically seen in the hard palate. Patients with tertiary syphilis commonly present with destructive gummatous lesions, which can cause substantial damage to the orofacial regions. Tissue destruction leading to perforation is more prevalent in individuals with strong immune responses and is thought to be associated with hypersensitivity to acid-fast bacilli.[12] The most common oral sign of mucormycosis is ulceration of the palatal region, which occurs from necrosis due to invasion of a palatal vessel.[13] Extension of the mucormycosis from the sinuses into the oral cavity causes painful, black necrotic ulcerations in the hard palate. This lesion is usually large and deep, resulting in denudation of the underlying bone.[14] Invasive oral aspergillosis is usually higher among immunosuppressive patients presenting with oral ulcers. The infection of the maxillary sinus can be complicated by direct invasion of the fungus into the palate, with necrosis and erosion of the palate leading to an oronasal fistula.[15] Iatrogenic causes of palatal perforation including certain surgical procedures such as maxillectomy, tooth extraction, and septal surgery can result in palatal perforation. Some autoimmune disorders manifest palatal perforations. These autoimmune disorders include lupus erythematosus, sarcoidosis, Crohn's disease, and Wegner's granulomatosis. Lupus, a common autoimmune condition, presents with oral cavity manifestations.[16] The affected region of the palate typically starts with erythematous areas, sometimes accompanied by white patches. The boundaries of the lesion often lack sharp demarcation but frequently exhibit the development of a narrow zone of keratinization. In addition, there is a propensity for bleeding, ulceration, and eventual perforation.[17] Lupus, an autoimmune condition, can affect the oral cavity. The affected region in the oral cavity includes the palate which shows erythematous areas with or without white patches. The neoplasms of the palate like NK cell lymphoma present with soft palate or posterior hard palate swelling followed by perforation.[18] The lesion advanced to deep necrotic ulceration in the midline of the palate, resulting in the destruction of palatal tissues and the formation of an oronasal fistula.

Classifications of Palatal Perforation

Palatal perforation may be classified on the basis of the size of perforation into small (<2 mm), medium (3–5 mm), or large (>5 mm).[19] As per the location of the palatal perforation, the oronasal fistula can be anterior fistula, midpalatal fistula, fistula at the junction of the soft palate, and hard palate and soft palate fistula. Perforation at the hard palate and soft palate is the most common location for the development of the oronasal fistula.[20]

Clinical Presentations

The roof of the oral cavity is formed by the palate which separates it from the nasal cavity. The palatal lesions are often

presented in the form of ulcers and severe form as perforation. The perforation of the palate [Figure 2] makes communication between the nasal cavities and the oral cavity. The site of the perforation may be at the hard palate, soft palate, or the junction hard and soft palate.[21] The patient presents pain at the site of the palatal ulcer. The patient with palatal perforation may complain of a painful sensation from the palate when the patient eats spicy food which may cause associated weight loss. Patient of palatal perforation complaints of nasal regurgitation of foods and slurring of speech. Patients of palatal ulcers with tuberculosis are often associated with cough, low-grade fever, chills, night sweats, and weight loss.[12] The differential diagnosis of palatal perforation includes tertiary syphilis, leprosy, mechanical trauma, intranasal cocaine abuse, malignancies, Wegner's granulomatosis, and midline nonhealing granuloma [Table 1].[22]

Diagnosis

Numerous disorders can lead to a palatal perforation that is clinically indistinguishable, presenting a challenging diagnostic situation. Diagnosis typically relies on histopathological examination and immunohistochemistry.[23] Imaging studies such as computed tomography (CT) scans or magnetic resonance imaging (MRI) scan are performed to assess palatal lesions. Diagnosis of infective lesions is mainly based on clinical, histopathological, and microbiological studies. It is very vital to diagnose the lesions causing the palatal perforation early with the help of clinical and laboratory investigations. The diagnosis of infective etiology is mainly based on clinical, histopathological, and microbiological studies. Biopsy from the margin of the palatal perforation is usually done to rule out granulomatous inflammation and neoplasms. Antinuclear antibodies and antineutrophil cytoplasmic antibodies are advised to rule out autoimmune causes. Polymerase chain reaction can be suggested for mycobacterial infection.[24] Chest X-rays and even contrast-enhanced CT of the chest are done to rule out pulmonary pathology. VDRL test is helpful to rule out syphilis. Most of the palatal perforations of oronasal fistulas are located in the hard palate or at the junction of the hard and soft palate. Oral and nasal endoscopy are helpful to identify the site and extension of the palatal perforation. Endoscopic examination may reveal the presence of crusts at the site of the palatal perforation. Cone-beam CT is helpful to visualize the bony defect of the palate.[25] CT scans often confirm the endoscopic features and show perforation of the palate and nasal septum and destruction of associated destruction of turbinates. MRI reveals areas of abnormal signal in the palatal perforation such as hypointensity of mucosal and submucosal tissue and nonhomogeneous enhancement.[26] The diagnosis of palatal perforation may be delayed particularly in the elderly population with underlying cognitive impairment or dementia, where clinical presentations may remain unnoticed for a longer period.

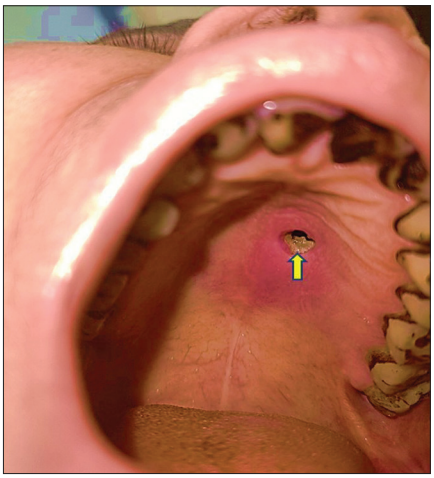

Figure 2: Palatal perforation (yellow arrow)

Table 1: Differential diagnosis of palatal perforation

Etiological group	Examples
Infections	Tuberculosis, tertiary syphilis, mucormycosis, actinomycosis, diphtheria, and typhoid
Collagen vascular disease	Wegener's granulomatosis
Trauma	
Tumor	Carcinoma, lymphoma, and melanoma
Granulomatosis	Sarcoidosis
Idiopathic	Midline lethal granuloma
Other	Cocaine abuse

Treatment

A multidisciplinary approach is needed in palatal perforation to manage the defect along with to prevent malnutrition, aspiration, or impaired speech. The treatment options vary on the basis of disease extent and exact location of the perforation of the palate.[27] Early detection of exact pathogens in infective etiology plays a crucial role in antimicrobial agents.[28]

Surgical repair

The defect in the palate should be sealed using a two-layer approach, ensuring that both layers consist of adequately vascularized tissue and the suturing is done without creating tension. In addition, some sources suggest employing a three-layer closure method for palatal perforations.[29] The intermediate layer may be cartilage, bone, and acellular dermal matrix used during the repair of the palatal defect to close the oronasal fistula. A small local flap can be used to close the small palatal perforation. A flap is created by turning over tissue from the mucoperiosteal layer on the unaffected side to form the nasal lining. Another flap is rotated from the opposite mucoperiosteal layer to construct the oral layer.[30] The required rotation flap is frequently larger than anticipated. Closure of the palatal defect with a single layer may be possible in highly specific instances. In such cases, a larger turnover flap is harvested and tucked beneath the opposite mucoperiosteal layer using a double-breasting technique.[30] Nevertheless, there is a significant recurrence rate associated

with single-layer closure. Initially described as a cheek flap for primary palatoplasty in cases of a short palate, this technique proves beneficial when the fistula is positioned close to the junction of the hard and soft palates, especially in instances where the cleft is wide. The buccal mucosal flap, a flap based posteriorly with its base situated near the retromolar trigone, serves this purpose. The flap's distal portion can be harvested up to a point slightly short of the commissure of the oral cavity, with the only consideration during flap harvest being the preservation of the papilla of the parotid duct. In children, a small flap is typically harvested, whereas in young adults, a wider flap can be prepared. If necessary, the flap can be harvested from both the oral and nasal layers. Incorporating a portion of the buccinator muscle into the flap enhances its strength and reliability, rendering it a buccinator myomucosal flap. The application of a tongue flap typically involves a two-stage procedure. When executed correctly, this flap is considered safe and well-tolerated in the pediatric age group. It is recommended for larger palatal perforations where there is a considerable tissue deficit at the mucoperiosteal layer of the palate. The positioning of this flap can be anterior or posterior depending on the location of the defect. To ensure its vascularity, the average thickness of the myomucosal flap should not be <6 mm.[30] Ensuring a thorough repair of the nasal layer is crucial for achieving a successful outcome. The donor area is typically closed primarily, minimizing the risk of any residual defect in the tongue or speech problems. Complications associated with the tongue flap include hemorrhage and spontaneous detachment from the palate. To enhance the success rate of the flap outcome, a prefabricated flap retainer may be utilized.[31] Some surgeons tried with free radial forearm flap to repair palatal perforation where local tissue is not available.[32] The composition of the flap may be fasciocutaneous, adipocutaneous, or adipofascial depending on the surgeon's choice. Turbinate flap from the nasal cavity has been reported for closure of the palatal perforation.[33] The temporoparietal galeal flap is also an important flap for the closure of the palatal perforation.[34] The facial artery myomucosal flap was outlined for use in repairing larger oronasal fistulas, particularly those extending from the anterior palate to the midpalatal region.[35] One study showed the use of acellular dermal matrix for primary palatoplasty to prevent oronasal fistula.[34]

Nonsurgical closure

Palatal perforation can be closed by a plate made by an orthodontist. This is done when there are several unsuccessful surgical attempts or the patient is not interested in undergoing the surgical procedure. The prosthetic obturator is helpful to avoid nasal reflux and facilitates swallowing and speech performance.[36] The sole contraindication to the obturator is the patient's tolerance, as in some instances, the size required to effectively seal the defect may induce nausea.[37] An alternative temporary solution involves placing a silicone button to close the palatal perforation, which can improve nasal airflow and reduce the progression of local necrosis.[38]

Conclusion

Palatal perforations can occur due to several causes. The etiology of palatal perforation includes both infectious and noninfectious causes. Patients exhibiting palatal perforation should undergo a comprehensive evaluation to explore various potential causes. The diagnosis of palatal perforation is done by complete history taking and evaluation with a history of previous drug use and other risk factors for acquired immunodeficiency. Palatal lesions should undergo biopsy for histopathological examination and culture testing for bacteria, mycobacteria, and fungi using appropriate stains and cultures. Early diagnosis of the lesions responsible for palatal perforation is crucial, aided by clinical and laboratory investigations. Management of palatal perforation aims to eradicate the underlying cause through appropriate treatment, followed by reconstruction of the defect.

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Conflicts of interest

There are no conflicts of interest.

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