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Parulis: An Overview for Dental Clinicians

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Abstract: A parulis is a visible stomal opening of a fistulous tract arising from pulp necrosis and periapical infection. The pathogenesis involves a polymicrobial community dominated by Fusobacterium, Streptococcus, and Porphyromonas species, and inflammasome-driven periapical bone destruction that channels pus toward the mucosa or skin. Clinically, it ranges from a painless gingival papule to an extraoral cutaneous sinus and is frequently misdiagnosed as a different condition. Non-surgical root canal treatment resolves both abscesses and tracts. Adjunct antibiotics are reserved for spreading or refractory infections and should be culture-guided.

Keywords: Parulis, Dental Sinus Tract, Periapical Abscess, Endodontic Infection, Odontogenic Fistula, Differential Diagnosis, Antibiotic Therapy, NLRP3 Inflammasome.

Review Paper

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INTRODUCTION

A parulis, colloquially termed a gum boil, is an intraoral or extraoral stomal opening of a sinus tract that communicates with a periapical abscess [1]. Despite being one of the most visible signs of endodontic disease, it remains a frequent target for misdiagnosis, especially when pus tracks to the face or neck rather than the gingiva [2]. Morphologically similar lesions, such as pyogenic granulomas and odontogenic cysts, may further obscure the diagnosis [3]. This review concisely summarizes the pathobiology, clinical features, and management of parulis for practising dental clinicians.

Etiopathogenesis

Parulis formation begins with pulp necrosis due to caries, trauma, or periodontitis, which provides bacteria with unrestricted access to the root canal system and ultimately to the periapex [1]. The resulting infection follows the path of least resistance through the cancellous bone and soft tissue, perforating the cortical plate to create a granulation tissue-lined fistulous tract, whose surface opening constitutes the parulis [4].

The infecting flora were robustly polymicrobial. A 2024 systematic review of 21 clinical studies showed that primary apical infections are dominated by Pseudoramibacter alactolyticus, Fusobacterium, Streptococcus, Porphyromonas endodontalis, and Prevotella species, with Firmicutes being the most prevalent phylum [5]. High-throughput 16S rRNA sequencing of persistent periapical infections demonstrated that sinus tract formation is associated with a significantly distinct microbial signature enriched in Treponema, Fusobacterium, Olsenella, and Dialister compared to infections lacking a tract, implying that microbial community composition actively drives tract development [6].

Host-mediated tissue destruction further amplifies this process. Bacterial lipopolysaccharides and damage-associated molecular patterns released at the periapex activate the NLRP3 inflammasome in local macrophages, fibroblasts, and osteoclasts, triggering IL-1β and IL-18 maturation and inflammatory cell pyroptosis [7]. The downstream effects, including amplified osteoclastogenesis and suppressed osteoblast differentiation, progressively erode the alveolar cortex, widen the drainage path, and ultimately create the mucosal or cutaneous opening observed clinically [7].

Clinical Features

A parulis typically presents as a soft, erythematous, fluctuant papule on the attached gingiva near the apex of the offending tooth, frequently without pain or dental symptoms, a characteristic that delays its presentation [4]. When the infection channels through deeper tissue planes, a cutaneous sinus opens on the chin, cheek, or submandibular area; mandibular teeth are responsible for approximately 80% of these extraoral presentations [4]. Extraoral parulis is often misdiagnosed as a sebaceous cyst, furuncle, or parotid fistula by non-dental practitioners, leading to cycles of ineffective incision, antibiotic therapy, and surgical curettage [2]. Simultaneous intraoral and extraoral sinus tracts from a single tooth, although uncommon, have been documented and further complicate the recognition of the disease [1].

Management

Source elimination is the definitive treatment for this condition. For restorable teeth, non-surgical root canal treatment achieves closure of the sinus tract within weeks of obturation, with a one-year follow-up confirming complete resolution and minimal scarring, even in extraoral cases [2]. Extraction is indicated for non-restorable teeth. Surgical excision of the tract is neither required nor advisable; it regresses spontaneously once the bacterial reservoir is eliminated, and excision without dental treatment results in recurrence [4].

Adjunct antibiotics are appropriate only for spreading infections, systemic involvement, or immunocompromise. Odontogenic abscesses predominantly yielded gram-positive cocci, including Staphylococcus (38.24%) and Streptococcus (29.41%). Amoxicillin remains the empirical first choice, with linezolid demonstrating susceptibility exceeding 90% for both species. Given the documented macrolide resistance (azithromycin susceptibility as low as 46% for Staphylococcus), empirical macrolide use requires caution [8]. In refractory or recurrent cases, microbiological culture with susceptibility testing, ideally supported by molecular methods that detect significantly greater bacterial diversity than conventional culture, guides definitive antibiotic selection [8, 9].

CONCLUSION

Parulis is a readily treatable but easily mismanaged consequence of neglected pulp diseases. Accurate diagnosis relies on a structured clinical examination, pulp testing, radiographic evaluation, and sinus tract tracing. Non-surgical endodontic treatment resolves the fistula predictably, and antibiotics are adjuncts, not substitutes. Understanding the inflammatory cascade and distinct microbial ecology of sinus tract–associated infections allows dental clinicians to counsel patients effectively and select appropriate treatment.

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