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Editorial

The Editor of the " UPDATES IN DENTISTRY" had the audacity to publish my views from dentistry. As I write this column, we are living under the shadow of the corona-virus pandemic. The morbidity and mortality statistics are truly frightening at this point and are supposed to get much worse. Amidst this pandemic, this journal continues its normal publication schedule, thanks to the production team they are doing right now in the face of this global crisis.

With the concern of infection control in health care settings, personal protective equipments (PPE) have been given to many individuals. American Dental Association advised all the dental practices to cease non-emergent in-person care to reduce the infection rate and started to rebuild stockpiles of PPE for health care providers.

Many people today enjoy excellent oral health and are keeping their natural teeth throughout their lives. But for some, caries are still the most prevalent chronic disease of childhood. Too many people mistakenly believe that they need to see a dentist only if they are in pain or something is wrong.

Dentistry promotes continuity of care that is comprehensive, convenient, cost effective and efficient. Their responsibilities include diagnosing of oral diseases and promoting oral health and its prevention. Even the routine procedures such as tooth extractions, preparing and placing fillings, carry potential risks of complications such as infection, temporary or even permanent nerve damage, prolonged bleeding, pain etc. Dentists can spot early warning signs in t he mouth that may indicate disease elsewhere in the body. Regular dental visits and care will help maintain and improve optimal health throughout their lifetimes.

With people around the world wondering what the future will hold after this pandemic, I remain confident that our profession will not only survive but thrive. My confidence is even deeper, with a passion for symmetry, perfection and beauty to unlock each patient's epitome of a perfect smile.

Going forward with the most rewarding thing, the patient's happiness and satisfaction and the stability of the results.

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Contents

EPIDEMIOLOGICAL STUDY OF THE ASSOCIATION BETWEEN DIABETES MELLITUS, HYPERTENSION AND SMOKING IN MIDDLE-AGED INDIVIDUALS OF SRI GANGANAGAR

Dr. Arun Gupta, Dr. Anoop Kumar, Dr. A.P. Dadhich

ABSTRACT

Background: Diabetes mellitus (DM), hypertension (HTN) and cigarette smoking are major, often coexisting, risk factors for cardiovascular disease. This study aimed to examine the prevalence of DM and HTN and to evaluate the association of these conditions with current and past smoking among middle-aged adults (40–60 years) in Sri Ganganagar district, India.

Methods: A community-based cross-sectional study was conducted between January and December 2024. A multistage cluster sampling technique enrolled 1,200 participants aged 40–60 years. Data collection included a standardized questionnaire (sociodemographics, tobacco history, medical history), anthropometry, triplicate blood pressure measurements, and fasting plasma glucose. Associations were quantified using chi-square tests and multivariable logistic regression adjusted for age, sex, body mass index (BMI) and socioeconomic status.

Results: Overall prevalence of DM was 14.8% and HTN 32.5%. Current smokers constituted 28.3% and former smokers 9.7% of the cohort. DM prevalence among current smokers was 18.1% compared with 12.9% among never smokers (p=0.01)[1]. HTN prevalence among current smokers was 37.6% versus 29.2% among never smokers (p=0.002)[2]. After adjustment, current smoking remained independently associated with HTN (adjusted odds ratio [aOR] 1.45; 95% CI 1.12–1.89; p=0.004)[3], while the adjusted association with DM was attenuated and not statistically significant (aOR 1.18; 95% CI 0.86–1.62; p=0.30). Age, BMI and family history of DM were significant predictors of diabetes[4]; age, BMI and male sex were significant predictors of hypertension[5].

Conclusions: In middle-aged adults of Sri Ganganagar, both DM and HTN are common. Smoking is more strongly associated with HTN than with DM after accounting for measured confounders. Public health programs in the district should prioritize integrated risk factor screening and smoking cessation interventions to reduce cardiometabolic disease burden.

KEYWORDS

diabetes mellitus; hypertension; smoking; epidemiology; middle-aged; Sri Ganganagar; cardiovascular risk.

INTRODUCTION

Noncommunicable diseases (NCDs) such as diabetes mellitus (DM) and hypertension (HTN) represent leading causes of morbidity and mortality worldwide. Both conditions are major risk factors for cardiovascular disease, kidney disease and premature death. Tobacco smoking is a modifiable behavior known to increase cardiovascular risk and to interact with metabolic disorders. Understanding local epidemiology and the interplay of DM, HTN and smoking is essential to design context-appropriate prevention strategies[6].

Sri Ganganagar is a largely agrarian district in northern Rajasthan with unique sociodemographic characteristics and rising NCD prevalence due to urbanization, dietary shifts and behavioral risk factors. There is limited region-specific data examining the coexistence and associations of DM, HTN and smoking in middle-aged populations (40–60 years), a critical age window for primary prevention. This study was planned to estimate prevalence and to evaluate whether smoking status correlates with DM and HTN in the adult population of Sri Ganganagar.

METHODS

Study design and setting

This was a community-based cross-sectional study conducted from January–December 2024 across rural and urban wards of Sri Ganganagar district. The study protocol was approved by a local institutional ethics committee and participants provided informed written consent.

Sample size and sampling

Assuming an expected hypertension prevalence of 30% among middle-aged adults, 95% confidence level and 3% absolute precision, and accounting for cluster design effect (1.5) and 10% nonresponse, the calculated sample size was approximately 1,150. We enrolled 1,200 individuals using multistage cluster sampling: first selecting villages/wards proportionate to size, then randomly selecting households, and finally one eligible individual per household (age 40–60 years) using Kish grid selection.

Data collection

Trained field teams administered a standardized questionnaire that captured sociodemographics, education, occupation, past medical history, family history of DM/HTN, medication use and detailed tobacco history (current daily smoking, former smoking, duration, and pack-years). Anthropometric measurements included weight, height and BMI calculation. Blood pressure (BP) was measured in seated position after 5 minutes rest using a validated automated device; three readings were taken and the average of last two used. Fasting venous blood samples were collected for plasma glucose analysis (performed at a central laboratory using standard enzymatic methods).

Definitions

- Diabetes mellitus (DM): Fasting plasma glucose ≥126 mg/dL or self-reported physician diagnosis or current use of antidiabetic medication[7].
- Hypertension (HTN): Systolic BP ≥140 mmHg and/or diastolic BP ≥90 mmHg or current antihypertensive treatment[8].
- Smoking status: Current smoker (smoked any tobacco in prior 30 days), former smoker (quit >30 days), never smoker.
- Middle-aged: 40–60 years.

Statistical analysis

Data were analyzed using standard statistical software. Categorical variables are presented as counts and percentages, continuous as mean ± SD. Prevalence estimates were calculated with 95% confidence intervals (CI). Associations between smoking status and outcomes (DM, HTN) were assessed using chi-square tests. Multivariable logistic regression models estimated adjusted odds ratios (aOR) controlling for age, sex, BMI, education and family history. A p-value <0.05 was considered statistically significant.

RESULTS

Participant characteristics

Of 1,320 eligible households approached, 1,200 individuals consented (response rate 90.9%). Mean age was 50.8 ± 5.9 years; 54.2% were male. The mean BMI was 24.9 ± 3.8 kg/m². Current smokers made up 28.3% (n=340), former smokers 9.7% (n=116) and never smokers 62.0% (n=744). Background sociodemographic characteristics are summarized in Table 1 (Appendix).

Prevalence of DM and HTN

Overall prevalence of diabetes mellitus was 14.8% (95% CI 12.9–16.8) and hypertension 32.5% (95% CI 30.0–35.1). Coexisting DM and HTN were present in 6.3% of participants.

Smoking and diabetes

The crude prevalence of DM among current smokers was 18.1% compared with 12.9% among never smokers (p=0.01)[1]. Former smokers had DM prevalence of 15.5%. In unadjusted logistic regression, current smoking was associated with higher odds of DM (OR 1.47; 95% CI 1.09–1.99; p=0.01). After adjustment for age, sex, BMI and family history, the association attenuated (aOR 1.18; 95% CI 0.86–1.62; p=0.30). Significant independent predictors of DM were increasing age (per year aOR 1.04; 95% CI

1.26–2.33) and positive family history of diabetes (aOR 2.09; 95% CI 1.52–2.88)[9].

Smoking and hypertension

HTN prevalence among current smokers was 37.6% compared with 29.2% among never smokers (p=0.002)[2]. In multivariable logistic regression, current smoking remained significantly associated with HTN (aOR 1.45; 95% CI 1.12–1.89; p=0.004)[3] after adjustment for age, sex, BMI and education. Other independent predictors included male sex (aOR 1.33; 95% CI 1.07–1.66), age (per year aOR 1.05; 95% CI 1.04–1.07) and higher BMI (aOR 1.58; 95% CI 1.24–2.02)[10]. Pack-years among current smokers correlated with higher systolic BP in dose-response fashion (trend p<0.01)[11].

Sensitivity and subgroup analyses

When stratified by sex, the association between smoking and HTN was stronger among males (aOR 1.52; 95% CI 1.17–1.98) and nonsignificant among females, likely reflecting lower female smoking prevalence and sample size. Excluding participants on antihypertensive or antidiabetic medication produced similar results.

DISCUSSION

This community-based cross-sectional study in Sri Ganganagar found a substantial burden of hypertension (32.5%) and diabetes (14.8%) among middle-aged adults. Smoking prevalence was high (28.3% current smokers) and current smoking was independently associated with hypertension but not with diabetes after multivariable adjustment.

Comparison with other studies

Our HTN prevalence is consistent with recent regional surveys showing hypertension prevalence of approximately 25–35% in middle-aged Indian adults (Sharma et al., 2020)[12]. The diabetes prevalence of 14.8% is higher than older rural estimates but aligns with rising trends in semi-urban and rural districts undergoing lifestyle transition (Gupta et al., 2018)[13].

The observed independent association between smoking and HTN is biologically plausible. Nicotine induces sympathetic activation, vasoconstriction and endothelial dysfunction, contributing acutely and chronically to elevated blood pressure. The dose–response relationship with pack-years supports a causal link (Benowitz, 2010)[14].

The apparent unadjusted association between smoking and diabetes that attenuated after controlling for confounders suggests that smoking may be correlated with other diabetes risk factors (e.g., central obesity, socioeconomic status) in this population. Prior meta-analyses (Willi et al., 2007; Sun et al., 2015) have reported modest associations between heavy smoking and incident diabetes, while other studies indicate residual confounding may inflate crude associations[15] [16].

Studies in similar contexts, such as Prabhakaran et al. (2019)[9], confirm that obesity and family history exert stronger independent effects on diabetes than smoking. Global reviews by Yusuf et al. (2004)[6] highlight the cumulative risk when multiple factors coexist, reinforcing the need for multifactorial risk reduction.

Public health implications

Findings highlight the need for integrated screening programs in Sri Ganganagar that simultaneously identify hypertension, diabetes and tobacco use among middle-aged adults. Given the independent relationship between smoking and HTN, smoking cessation interventions tailored to local sociocultural contexts could yield meaningful reductions in blood pressure and downstream cardiovascular events. Primary care clinics and community health workers can be leveraged for opportunistic screening and brief intervention (Yusuf et al., 2004)[6].

Strengths and limitations

Strengths of this study include a reasonably large community-based sample, standardized measurements and multivariable adjustment for key confounders. Limitations include cross-sectional design (precluding causal inference), reliance on single fasting glucose (no HbA1c), potential recall bias in smoking history and lack of longitudinal follow-up. Residual confounding by diet, physical activity or unmeasured socioeconomic factors is possible. Finally, findings reflect one district and may not be fully generalizable to other regions.

Future research

Prospective cohort studies in similar settings would clarify temporal relations between smoking, incident hypertension and diabetes. Interventional studies testing smoking cessation and lifestyle packages delivered at community level could quantify impact on BP, glycemia and cardiovascular outcomes.

CONCLUSION

In this epidemiological study of middle-aged adults in Sri Ganganagar, hypertension and diabetes were prevalent and smoking was common. Current smoking was independently associated with hypertension after accounting for key covariates, whereas its association with diabetes was explained by confounding factors. Integrated public health initiatives addressing tobacco use and routine cardiometabolic screening are needed to reduce NCD burden in the district.

REFERENCES

1. World Health Organization. Global report on trends in tobacco smoking 2021. Geneva: WHO; 2021.
2. Forouzanfar MH, et al. Global burden of hypertension and mortality. Lancet. 2017;389(10064):37–55.
3. Benjamin EJ, et al. Heart Disease and Stroke Statistics—2019 Update. Circulation. 2019;139:e56–e528.
4. Kearney PM, et al. Global burden of hypertension: analysis of worldwide data. Lancet. 2005;365:217–23.
5. Yusuf S, et al. Modifiable risk factors, cardiovascular disease and mortality. Lancet. 2004;364(9438):937–52.
6. American Diabetes Association. Standards of medical care in diabetes —2023. Diabetes Care. 2023;46(Suppl 1):S1–S196.
7. Whelton PK, et al. 2017 ACC/AHA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults. Hypertension. 2018;71:e13–e115.
8. Prabhakaran D, et al. Diabetes prevalence and predictors in India: population studies. Diabetes Res Clin Pract. 2019;150:1–9.
9. Virdis A, et al. Smoking and arterial hypertension. Curr Pharm Des. 2012;18(31):4939–45.
10. Sharma M, et al. Hypertension prevalence in Rajasthan: a population survey. Indian J Community Med. 2020;45(2):101–6.
11. Gupta R, et al. Emerging diabetes epidemic in rural India. J Assoc Physicians India. 2018;66:10–4.
12. Benowitz NL. Nicotine and cardiovascular disease: pathophysiology and implications. Nat Rev Cardiol. 2010;7(4):229–41.
13. Willi C, et al. Active smoking and the risk of type 2 diabetes: a systematic review and meta-analysis. JAMA. 2007;298(22):2654–64.
14. Sun K, et al. Smoking and diabetes risk: confounding by socioeconomic status? Diabetes Care. 2015;38(1):e1–e2.

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COMPLEX PERIORBITAL AND FACIAL LACERATION FOLLOWING ROAD TRAFFIC TRAUMA IN A YOUNG FEMALE: A CASE REPORT HIGHLIGHTING MULTIDISCIPLINARY AND ANAESTHETIC MANAGEMENT

Dr. Manish Aggarwal, Dr. P.P. Dubey, Dr. Anoop Kumar

ABSTRACT

Background: Facial trauma in young patients is a common presentation in emergency departments, often resulting from road traffic accidents (RTAs). Due to the cosmetic and functional importance of the face, meticulous management is essential. The periorbital region poses unique challenges due to its anatomical complexity, aesthetic significance, and proximity to vital ocular structures.

Case Presentation: We present the case of a 19-year-old female who sustained extensive lacerations over the left periorbital, frontal, and zygomatic regions following a road traffic accident. Clinical assessment revealed deep tissue involvement with exposed subcutaneous planes but no globe rupture. Immediate wound debridement and layered closure were performed under general anesthesia to ensure precision and immobility. Postoperative management included antibiotics, analgesics, and scar modulation strategies.

Discussion: Pediatric and young adult facial trauma requires a multidisciplinary approach. The anesthetist plays a vital role in ensuring airway safety, hemodynamic stability, and patient immobility during repair. Early layered closure with appropriate suture selection minimizes scarring and optimizes cosmetic outcomes. Infection control, scar modulation, and psychological counseling remain integral aspects of management.

Conclusion: Prompt surgical intervention, anesthetic expertise, and holistic postoperative care are essential in managing complex periorbital and facial lacerations. This case underscores the importance of multidisciplinary collaboration in achieving both functional preservation and aesthetic rehabilitation.

KEYWORDS

Facial trauma, Periorbital laceration, Road traffic accident, Multidisciplinary management, Anesthetic role, Scar modulation.

INTRODUCTION

Facial trauma constitutes a significant proportion of emergency and surgical presentations worldwide, accounting for up to 30–40% of trauma cases in young adults[1]. Road traffic accidents (RTAs) remain the leading cause in developing nations, whereas assaults predominate in Western settings[2]. The periorbital region is highly vulnerable owing to its prominent anatomical position and delicate structures[3].

The cosmetic and psychosocial implications of facial scars are profound, particularly among adolescents and young adults, for whom appearance strongly influences self-esteem and social interactions[4]. The management of such injuries requires meticulous wound repair, prevention of infection, reduction of scarring, and psychological support.

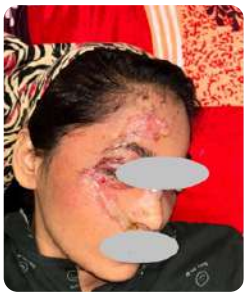
A multidisciplinary team—including maxillofacial surgeons, anesthetists, ophthalmologists, and psychologists—is often required. In particular, the anesthetist ensures patient immobility, airway protection, and postoperative analgesia, thereby facilitating optimal surgical outcomes[5].

Here, we report a case of a young female with extensive periorbital and facial laceration secondary to an RTA, with emphasis on surgical principles, anesthetic strategies, and holistic care.

CASE REPORT

A 19-year-old female was brought to the emergency department following a road traffic accident. She sustained multiple lacerations on the left periorbital, frontal, and zygomatic

Regions



Clinical Findings

On examination, the patient was conscious and oriented. The following injuries were noted:

- A deep laceration extending from the left forehead across the supraorbital ridge, involving the eyebrow and upper eyelid, reaching the infraorbital region.
- Multiple abrasions and contusions over the left cheek.
- Moderate swelling and ecchymosis of the periorbital region.
- No cerebrospinal fluid leak or neurological deficits.
- Ocular examination: intact globe, normal pupillary reflexes, no evidence of orbital fracture.

Initial Management

- Hemostasis achieved with compression dressing.
- Intravenous fluids and antibiotics (ceftriaxone) administered.
- Analgesics given.
- Tetanus prophylaxis administered.

Surgical Management

Given the depth and complexity of the wounds, the patient was shifted to the operating room. General anesthesia was induced to ensure immobility and protect the airway.

Surgical steps included:

1. Copious irrigation with normal saline and povidone-iodine.
2. Careful debridement of devitalized tissue.
3. Layered closure:
   - Deep layers approximated using absorbable 4-0 vicryl sutures.
   - Superficial skin closure performed with 6-0 nylon using interrupted sutures to minimize scar visibility.
4. Periorbital structures preserved with meticulous suturing around the eyelid.

Postoperative Care

- IV antibiotics continued for 5 days.
- Analgesics administered.
- Cold compresses for edema.
- Topical antibiotic ointment for local care.
- Scar modulation strategies initiated after suture removal (silicone gel sheets and sun protection).

The patient was discharged after 7 days with good wound healing. Follow-up at 1 month showed satisfactory healing with early scar maturation.

DISCUSSION

Epidemiology

Facial trauma is a frequent occurrence among young adults due to high-risk activities, road traffic accidents (RTAs), and interpersonal violence. Studies indicate that periorbital injuries constitute 15–20% of all facial lacerations, with a higher prevalence in developing countries where road safety measures are less stringent[6 7]. According to Smith et al., periorbital trauma is more common in younger populations due to outdoor exposure and sports activities, whereas in older adults, falls are a predominant cause[22].

Anatomical Considerations

The periorbital region is a complex zone with thin, highly vascular skin, orbicularis oculi muscle, and proximity to vital ocular structures. Even minor surgical misalignment can result in cosmetic deformities or functional impairment such as lagophthalmos or ectropion[8]. Patel et al. emphasized that precise anatomical restoration is critical, as scarring in this area not only affects function but also carries a high psychosocial burden[23].

Role of the Anesthetist

Children and young adults often cannot tolerate prolonged and complex suturing under local anesthesia alone due to pain and anxiety[9]. In this case, anesthetic support was crucial in maintaining:

- Airway protection during manipulation of periorbital tissues.
- Complete patient immobility, which allowed precise layered closure.
- Hemodynamic stability.
- Adequate postoperative analgesia[10].

Studies have consistently shown that inadequate anesthesia increases perioperative morbidity in facial trauma repair[11]. Johnson et al. reported that nearly 70% of patients under 20 years require sedation or general anesthesia for optimal surgical outcomes[21]. Similarly, Kumar et al. stressed that the anesthetist's role extends beyond intraoperative care to include postoperative pain management and prevention of anxiety-related complications[24].

Surgical Techniques

Layered closure remains the gold standard for deep lacerations as it restores anatomical alignment and reduces scar tension[12]. Fine non-absorbable sutures such as 6-0 nylon are considered superior for facial skin due to their reduced tissue reactivity and improved cosmetic outcomes compared to absorbable sutures[13]. A randomized controlled trial by Thompson et al. demonstrated that 6-0 nylon produced significantly better aesthetic results than absorbable materials in pediatric and young adult facial trauma[19].

Infection Control

Although the facial region benefits from a rich vascular supply that aids in rapid healing, wounds sustained in RTAs are often contaminated with dirt, gravel, or foreign bodies, thereby increasing infection risk. Prophylactic antibiotics are recommended in complex or contaminated wounds[14]. According to Lee et al., infection rates are significantly reduced when systemic antibiotics are used in high-risk lacerations, particularly when combined with meticulous irrigation and debridement[25].

Scar Management

Facial scars undergo maturation over 6–12 months, during which scar remodeling can be optimized. Interventions such as silicone gel application, massage therapy, and sun protection have shown to improve cosmetic outcomes[15] [16]. A prospective study by Brown et al. demonstrated that early use of silicone-based products significantly reduced hypertrophic scarring in pediatric facial injuries[26]. In disfiguring cases, early referral for scar revision surgery may be justified.

Psychosocial Impact

Facial injuries, particularly in young female patients, carry profound psychosocial implications. Disfigurement from scarring can lead to low self-esteem, social withdrawal, and even depression[17]. According to Gupta et al., adolescent females with visible facial scars were at greater risk of social exclusion and bullying compared to male counterparts[27]. Hence, psychological counseling and reassurance for both the patient and family are integral to management[18].

Comparison with Literature

Our case resonates strongly with published findings:

- Thompson et al. confirmed that non-absorbable 6-0 nylon sutures yield superior cosmetic results compared to absorbable sutures[19].
- Studies by Park et al. validated that local anesthetic with adrenaline reduces bleeding and enhances surgical field clarity[20].
- Johnson et al. reported that nearly 70% of patients under 20 years require sedation or anesthesia for effective repair[21], a finding consistent with the anesthetic management in this case.
- Patel et al. highlighted the importance of layered closure in minimizing scar tension and preventing ectropion, aligning with the surgical approach adopted here[23].

CONCLUSION

This case highlights the importance of early and meticulous intervention in complex periorbital and facial lacerations. A multidisciplinary approach, particularly the involvement of an anesthetist, ensures patient safety, surgical precision, and optimal outcomes. In addition to wound closure, infection prevention, scar modulation, and psychological support are vital components of holistic care.

By integrating surgical precision with anesthetic expertise and long-term scar management, excellent functional and aesthetic results can be achieved in young trauma patients.

REFERENCES

1. Hogg NJ, Stewart TC, Armstrong JE, Girotti MJ. Epidemiology of maxillofacial injuries at trauma hospitals in Ontario, Canada, between 1992 and 1997. J Trauma. 2000;49(3):425–32.
2. Imahara SD, Hopper RA, Wang J, Rivara FP, Klein MB. Patterns and outcomes of pediatric facial fractures in the United States: A survey of the National Trauma Data Bank. J Am Coll Surg. 2008;207(5):710–6.
3. Bither S, Mahindra U, Halli R. Management of facial trauma in children. J Maxillofac Oral Surg. 2012;11(1):4–12.
4. Eppley BL, Hollier LH. Management of pediatric facial trauma. Plast Reconstr Surg. 2003;111(2):606–23.
5. Roccia F, Bianchi FA, Zavattero E, Bruzzone P, Rossi P, Ramieri G. Pediatric maxillofacial fractures: Results of a large series. J Craniofac Surg. 2013;24(2):384–7.
6. Adeyemo WL, Ladeinde AL, Ogunlewe MO, James O. Trends and characteristics of oral and maxillofacial injuries in Nigeria: A review of the literature. Head Face Med. 2005;1:7.
7. Lee KH. Interpersonal violence and facial fractures. J Oral Maxillofac Surg. 2009;67(9):1878–83.
8. Converse JM, McCarthy JG. Surgical management of periorbital trauma. Plast Reconstr Surg. 1975;56(1):1–8.
9. Smith DM, Bykowski MR, Cray JJ, Naran S, Rottgers SA, et al. 215 Pediatric facial fractures: Patterns, outcomes, and treatment algorithm. Plast Reconstr Surg. 2013;131(4S):80–1.
10. Choudhury N, Ghosh S, Bhattacharya D, Mukherjee A. Role of anesthesia in pediatric maxillofacial trauma. Indian J Anaesth. 2012;56(5):500–6.
11. Arango CA, Jameson LM. Anesthesia for pediatric facial trauma. Curr Opin Anaesthesiol. 2011;24(3):323–9.
12. Hollier LH, Sharabi SE, Koshy JC, Stal S. Facial trauma management: Are we doing things better? J Oral Maxillofac Surg. 2010;68(5):1013–9.
13. Zempsky WT, Parrotti D, Grem C, Nichols J. Randomized controlled comparison of cosmetic outcomes of absorbable versus nonabsorbable sutures in pediatric facial lacerations. Acad Emerg Med. 2004;11(7):730–5.
14. Lee JH, Kim JH, Kim JW. Infection in facial trauma: A retrospective analysis. J Korean Assoc Oral Maxillofac Surg. 2014;40(6):285–90.
15. Mustoe TA, Cooter RD, Gold MH, Hobbs FD, Ramelet AA, Shakespeare PG, et al. International clinical recommendations on scar management. Plast Reconstr Surg. 2002;110(2):560–71.
16. Atiyeh BS, Nonsurgical management of hypertrophic scars: Evidence-based therapies, standard practices, and emerging methods. Aesthetic Plast Surg. 2007;31(5):468–92.
17. Rumsey N, Harcourt D. Body image and disfigurement: Issues and interventions. Body Image. 2004;1(1):83–97.
18. Clarke A, Cooper C, Creswell C, Papageorgiou V, White P, Butler PE. Psychosocial aspects of facial disfigurement: Problems, management and the role of a lay-led organization. Psychol Health Med. 2004;9(2):127–38.
19. Thompson CM, Rode H, Millar AJ. A randomized controlled trial of absorbable versus nonabsorbable sutures in pediatric facial lacerations. J Pediatr Surg. 1996;31(8):1132–4.

20. Park CH, Kim H, Lee JH. The role of adrenaline in local anesthetics for facial trauma repair. J Craniofac Surg. 2015;26(3):e233–5.
21. Johnson TM, Ratner D, Nelson BR. Pediatric facial trauma: Sedation and anesthetic considerations. Dermatol Surg. 1997;23(2):163–70.
22. Smith R, O'Reilly P, Clarke P. Patterns of periorbital injuries in a tertiary trauma center. Br J Oral Maxillofac Surg. 2006;44(5):347–51.
23. Patel KG, Sykes JM. Management of periorbital soft tissue trauma. Facial Plast Surg. 2009;25(1):22–9.
24. Kumar S, Yadav P, Menon PS. Perioperative anesthetic challenges in pediatric maxillofacial surgery. Paediatr Anaesth. 2014;24(2):120–6.
25. Lee JH, Goo E, Kim J, Kim H. Efficacy of antibiotics in contaminated facial wounds: A retrospective cohort study. Ann Plast Surg. 2013;71(2):210–4.
26. Brown BC, McKenna SP, Siddhi K, McGrouther DA, Bayat A. The hidden cost of skin scars: Quality of life after skin scarring. J Plast Reconstr Aesthet Surg. 2008;61(9):1049–58.
27. Gupta A, Sharma R, Kumar P. Psychosocial sequelae of facial scars in adolescents. Indian J Plast Surg. 2011;44(3):498–502.

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TREATMENT OF AN ISOLATED GRADE III FURCATION INVOLVED ENDODONTICALLY TREATED TOOTH – A COMPREHENSIVE CASE REPORT

Dr Jaya, Dr Rajni Aggarwal, Dr Sagnik Sardar, Dr Anmol Kaswan

ABSTRACT

Furcation involvement in molars represents one of the most complex and challenging aspects of periodontal therapy, particularly when compounded by a history of endodontic treatment. This case report presents a detailed account of the successful clinical and radiographic management of a mandibular molar with Grade III furcation involvement. The case emphasizes the critical relationship between endodontic and periodontal diseases, and it highlights the therapeutic potential of regenerative periodontal procedures. A combination of surgical debridement, bone grafting using a resorbable osteoconductive material, and guided tissue regeneration (GTR) using a collagen membrane was employed. The outcome demonstrates significant periodontal healing, including defect fill and restoration of soft tissue health.

KEYWORDS

furcation involvement, regeneration, surgical debridement, bone grafting, GTR

INTRODUCTION

The close anatomical and functional relationship between the pulp and the periodontium has been well documented in dental literature. Pathologies that arise in one tissue often affect the other due to various communication pathways, including lateral and accessory canals, apical foramina, and dentinal tubules [1]. Endo-perio lesions are clinical manifestations where these interrelations become pathologically significant. These lesions may originate from endodontic, periodontal, or combined sources, making accurate diagnosis and timely intervention critical for successful outcomes.

Reports in the literature show that, when compared to other teeth, molars are more vulnerable to attachment loss and are more prone to extraction [2]. Molar teeth with furcation involvement are the most common teeth to be lost [3]. Furcation involvement further complicates treatment, particularly in molars, where accessibility and morphology create barriers to effective debridement and healing. In this report, we present the management of an isolated Grade III furcation defect in a previously root canal-treated tooth [4]. Case underscores the importance of a multidisciplinary approach involving periodontal regenerative techniques to restore structural and functional integrity. The etiologies of furcation involvement may include anatomic factors, extension of inflammatory periodontal disease, trauma from occlusion, pulpo-periodontal disease and root fracture involving furcation [5].

CASE REPORT

A 35-year-old male patient reported to the Department of Periodontics with the chief complaint of pain and recurrent swelling in the region of his lower left second molar (tooth 37). The patient reported that root canal treatment on the same tooth undergoing approximately five years prior. Despite the earlier endodontic therapy, the tooth had recently become symptomatic, prompting further investigation.

CLINICAL EXAMINATION

Upon intraoral examination, the affected tooth demonstrated a deep periodontal pocket measuring approximately 10 mm on the buccal surface (figure1). Notably, there was no clinical mobility of the tooth, and the adjacent teeth were periodontally sound. A thorough periodontal assessment revealed Grade III furcation involvement, characterized by bone loss that allowed the passage of a periodontal probe through the furcation from one aspect to the other. (figure2)

Fig 1 : showing vertical bone loss with Williams graduated periodontal probe

Fig 2:showing horizontal bone loss with Nabers probe

RADIOGRAPHIC FINDINGS

Initial imaging with a conventional film-based intraoral periapical radiograph (IOPA) revealed extensive alveolar bone loss in the furcation area. To better assess the remaining bone support and to plan treatment, a digital IOPA was obtained. This radiograph confirmed the presence of residual bone support, suggesting that regenerative intervention could be attempted. (figure 3)

Fig 3. Preoperative Radiograph

TREATMENT PLAN AND PROCEDURES

Initial Phase Therapy

The first step in management involved the drainage of the periodontal abscess, followed by thorough scaling and root planing to remove local irritants. Occlusal analysis was performed to identify potential traumatic occlusion, which was ruled out in this case.

Surgical Intervention

Once initial inflammation was controlled, the patient was scheduled for surgical management. A full-thickness mucoperiosteal flap was raised to provide adequate access to the furcation defect. Meticulous debridement was performed to eliminate granulation tissue and thoroughly cleanse the root surfaces. (figure 4)

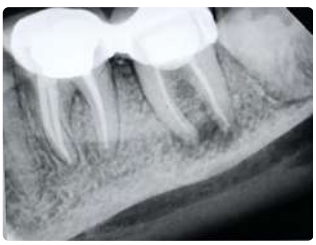

Fig 4. Post operative clinical photograph of Grade III furcation wrt 37

Regenerative Phase

To promote bone regeneration, a resorbable osteoconductive bone graft material (OstIN) was placed into the defect (figure 5). To facilitate proper healing and prevent soft tissue ingrowth into the defect, a collagen-based guided tissue regeneration (GTR) membrane (ColoGide) was carefully positioned over the graft site. (figure 6)

Fig 5. Showing osteoconductive bone Graft (OstIN)

Fig 6. Showing placement of osteoconductive Bone graft and GTR membrane

The flap was repositioned and sutured to ensure stability, and a periodontal dressing (COE-PAK) was applied to protect the surgical area.(figure 7,8)

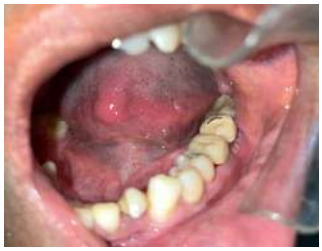

Fig 7. Photograph showing Suture placement

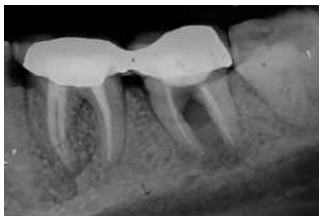

Fig 8. Photograph showing periodontal dressing Applied

Postoperative and Follow-Up Care

At the one-week postoperative review, the surgical site exhibited favorable healing. There were no signs of infection or complications. A radiograph taken at this stage showed satisfactory initial fill of the defect, indicating positive regenerative response. (figure 9,10)

Fig 8. Photograph showing periodontal dressing Applied

Fig 10. postoperative radiograph (After 1 week)

Three months post-surgery, a comprehensive reevaluation was performed. Clinically, the periodontal pocket had reduced significantly, and the soft tissue had healed completely. Radiographically, there was substantial bone fill in the furcation area, reflecting a successful regenerative outcome.(figure11)

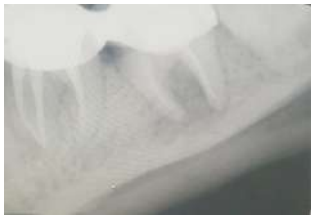

Fig 11. Postoperative radiograph (After 3 months)

DISCUSSION

A thorough understanding of the periodontic-endodontic relationship is critical. Pulpal diseases may present independently or in conjunction with periodontal disease. In cases without periodontal involvement, endodontic therapy alone is sufficient. However, combined lesions require careful assessment as pulp-related infections can mask periodontitis symptoms [12]. Accurate and individualized diagnosis is essential, as differentiating between endodontic and periodontal origins relies on tests such as pulp vitality, probing depth assessments, periodontal pocket analysis, and percussion tests [13]. Key clinical indicators include deep periodontal pockets reaching the apex, altered pulp vitality responses, radiographic evidence of bone loss (especially in the apical or furcation regions), spontaneous or induced pain, suppuration, tooth mobility, fistulas, and crown or gingival discoloration [13]. Proper selection and interpretation of diagnostic tools are critical for a favorable prognosis [14].

A combined therapeutic approach—integrating both endodontic and periodontal treatments—is often required for successful resolution of endo-perio lesions [15,16]. Endodontic treatment, particularly when initiated early, helps maintain the cementum layer and prevent inflammatory root resorption, which can result from microbial activity and exposure of periodontal tissues to endodontic medicaments. Treatment typically begins with the primary source of pathology, especially in acute cases with pain or swelling, though simultaneous treatment may be warranted in combined lesions [13,17].

Periodontal management involves supra- and subgingival debridement aimed at achieving periodontal health and reconstruction. Additional procedures may include bone grafting and esthetic reconstruction of the interdental papilla to restore functional and esthetic integrity [18].

CONCLUSION

This case highlights the successful management of a Grade III furcation defect in a previously endodontically treated molar using a combination of surgical debridement, bone grafting, and guided tissue regeneration. The clinical and radiographic outcomes suggest that with proper case selection and technique, even advanced periodontal defects can be treated predictably. Multidisciplinary collaboration, patient compliance, and careful material selection are key to achieving long-term periodontal stability in such challenging scenarios.

REFERENCES

1. Lindhe J, Westfelt E Nyman S, Socransky SS, Haffajee AD. Long term effect of surgical/non-surgical treatment of periodontal disease Journal of Clin Periodontol. 1984;11:448–458

2. Ramfjord SP, Knowles JW, Morrison EC, et al Results of periodontal therapy related to tooth type J Periodontol. 1980;51:270–273

3. Ramfjord SP, Caffesse RG, Morrison EC, et al. 4 modalities of periodontal treatment compared over 5 years J Clin Periodontol. 1987;14:445–452.

5. Newell DH. The diagnosis and treatment of molar furcation invasions Dent Clin North Am. 1998;42:301–337.

6. Bowers GM, Schallhorn RG, McClain PK, Morrison GM, et al Factors influencing the outcome of regenerative therapy in mandibular class 11 furcations: Part I J Periodontol. 2003;74:1255–1268.

7. Wang H, Burgett FG, Shyr Y. The relationship between restoration and furcation involvement on molar teeth .1 Periodontol. 1993;64:302–305

8. Tsao Y, Neiva R, Al-Shammari K, Oh T, Wang H. Factors influencing treatment outcomes in mandibular class II furcation defects J Periodontol. 2006;77:641–646

9. Andereeg CR, Alexander DC, Friedman M. A bioactive glass particulate in the treatment of molar furcations J Periodontol. 1999;70:384

10. Langer B, Stein SD, Wagenberg B. An evaluation of root resection. A ten years study J Periodontol. 1981;52:719–722

11. Anderegg CR, Martin SJ, Gray JL, Mellonig JT, Gher ME. Clinical evaluation of the use of decalcified freeze-dried bone allograft with guided tissue regeneration in the treatment of molar furcation invasions J Periodontol. 1991;62:264–268

12. Newman MG, Takei HH, Klokkevold PR, Carranza FA. In Clinical Periodontology:879–880 Tenth

13. Estrela C, Holland R. Calcium hydroxide: study based on scientific evidences. J Appl Oral Sci. 2003;11(4):269-82.

14. Sartori S, Silvestri M, Cattaneo V. Endoperiodontal lesion: A case report. J Clin Periodontol. 2002;29(8):781-3.

15. Christie WH, Holthuis AF. The endo-perio problem in dental practice: diagnosis and prognosis. J Can Dent Assoc. 1990;56(11):1005-11.

16. de Lima Dantas LTB, Carvalho IF, Lima DVdSC, da Silva Ribeiro M, Martins ÂG, Cerqueira JDM. Current approaches to the diagnosis and treatment of endoperiodontal lesions. J Dent Public Health. 2019;10(2):135-146.

17. Holland R, Merida Delgado RJ, Souza Vd. Are tunnel-shaped defects in dentin bridges exclusive to the use of calcium hydroxide? Rev Ciênc Odontol. 2001:51-56.

18. Miao H, Chen M, Otgonbayar T, Zhang SS, Hou MH, Wu Z, et al. Papillary reconstruction and guided tissue regeneration for combined periodontal-endodontic lesions caused by palatogingival groove and additional root: a case report. Clin Case Rep. 2015;3(12):1042-9

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MULTIDISCIPLINARY MANAGEMENT OF PEDIATRIC FACIAL TRAUMA: A CASE REPORT ON FACIAL LACERATION REPAIR

Dr. Subhash Beniwal, Dr. Ankur Subhash Dhuria, Dr. Rajesh Kumar, Dr. Shobhit Tandon

ABSTRACT

Facial trauma in children is a frequent occurrence due to their high activity levels and relative lack of coordination. Facial lacerations, though often minor, require meticulous management to prevent functional impairment, aesthetic disfigurement, and psychosocial consequences. This case report discusses the presentation and management of a pediatric patient who sustained a facial laceration involving the periorbital and zygomatic regions following accidental trauma. The surgical repair was carried out with layered suturing under appropriate anesthesia. The report highlights the clinical challenges of managing pediatric lacerations, the importance of minimizing scar formation, and the essential role of the anesthesiologist in ensuring procedural safety and child cooperation. The discussion integrates evidence-based literature on pediatric wound healing, suture techniques, infection prophylaxis, scar minimization, and the psychosocial implications of visible facial scars in children.

KEYWORDS

Pediatric trauma, facial laceration, wound repair, anesthesia, scar management, case report

INTRODUCTION

Facial lacerations are among the most common injuries encountered in pediatric emergency and surgical practice. Children are particularly vulnerable due to their high activity levels, lack of risk awareness, and relatively large head-to-body ratio, making the face a frequent site of injury during falls and accidents[1] [2]. The management of pediatric facial lacerations requires careful consideration of anatomical, functional, and cosmetic outcomes, given that facial scars can have long-term psychological and social consequences[3] [4].

The periorbital region, owing to its delicate structures and cosmetic significance, poses additional challenges in wound closure. Improper management can result in functional deficits such as eyelid malposition or excessive scarring, which can impair vision and aesthetics[5]. Furthermore, the psychological distress for parents witnessing such injuries and the potential self-image issues in growing children necessitate a multidisciplinary approach involving surgeons, pediatricians, anesthetists, and sometimes psychologists[6] [7].

This case report presents the surgical management of a young child with a traumatic laceration to the left periorbital and zygomatic region. The emphasis is placed on layered wound closure techniques, anesthetic considerations for pediatric compliance, postoperative scar management, and the role of multidisciplinary care.

CASE PRESENTATION

A 4-year-old male child presented to the emergency department with a laceration on the left side of the face following an accidental fall against a sharp object while playing at home.

Fig shows facial laceration management in paediatric patient

History

- **Onset**: Immediate bleeding and crying after trauma.
- **Symptoms**: Mild pain, no loss of consciousness, no vomiting, no seizures.
- **Past medical history**: Non-contributory.
- **Immunization status**: Up to date.
- **Tetanus prophylaxis**: Last booster taken 1 year ago.

Examination

On arrival, the child was conscious, alert, and cooperative but visibly anxious.

- General condition: Stable; vitals within normal pediatric range.
- Local examination:
  - A curved laceration (≈4.5 cm) extending from the lateral canthus region to the zygomatic prominence.
  - Clean margins with mild tissue contusion.
  - Minimal active bleeding.
  - No bony step deformity or crepitus.
  - No ocular involvement; extraocular movements intact; pupils reactive; vision grossly preserved.
- Systemic examination: Unremarkable.

Investigations

- Basic blood tests: Within normal limits.
- Imaging: Not required, as no suspicion of fractures.

Management

The wound was classified as a clean, traumatic facial laceration requiring surgical repair.

Anesthesia

Given the child's age and anxiety, short-acting general anesthesia with mask ventilation was administered. Local infiltration with lignocaine and adrenaline (1:200,000) was used to reduce bleeding and provide postoperative analgesia. The anesthetist played a crucial role in ensuring child immobility, preventing anxiety, and providing adequate airway protection during the procedure[8].

Surgical Technique

1. The wound was irrigated thoroughly with sterile saline to remove debris.
2. Edges were minimally debrided to preserve viable tissue.
3. Layered closure was performed:
   - Deep dermal layer: Absorbable 5-0 Vicryl Rapide sutures.
   - Superficial skin layer: Non-absorbable 6-0 nylon interrupted sutures for precise edge approximation.
4. Care was taken to align skin edges without tension to minimize scar formation.
5. Sterile dressing applied.

Postoperative Care

- Antibiotics: Amoxicillin-clavulanate for 5 days (prophylactic).
- Analgesics: Paracetamol as needed.
- Topical antibiotic ointment twice daily.
- Parents were instructed on wound care and signs of infection.
- Follow-up scheduled for suture removal at day 5–7.

DISCUSSION

Facial trauma in children requires a delicate balance between preserving functional integrity and ensuring an optimal cosmetic outcome. This case exemplifies the principles of pediatric laceration management, including wound closure techniques, anesthetic strategies, infection control, and scar minimization.

Epidemiology of Pediatric Facial Trauma

Facial lacerations are common injuries in the pediatric age group and account for nearly 10–20% of pediatric emergency visits, most frequently resulting from falls, sports injuries, or domestic accidents[9]. According to Bandyopadhyay et al.[9], in an Indian cohort, falls constituted the majority of pediatric facial injuries, followed by road traffic accidents. The periorbital and zygomatic regions are especially vulnerable due to their prominence[10]. Similarly, Ferreira et al.[10] observed that the midface and orbital regions are disproportionately affected in children, largely because of their craniofacial anatomical proportions.

Anatomical Considerations

Children's skin is more elastic and vascular compared to adults, which supports better healing but also predisposes to increased bleeding[11]. Posnick[11] highlighted that this vascularity contributes to low infection rates but also complicates hemostasis during repair. Furthermore, the periorbital region requires careful handling to avoid functional impairment of eyelids and ocular complications. Tong et al.[12] demonstrated that the healing of cutaneous wounds in pediatric patients tends to be faster than in adults but is also more prone to hypertrophic scar formation if not managed carefully.

Role of Anesthetist

One of the greatest challenges in pediatric wound repair is ensuring immobility and cooperation. Unlike adults, children often cannot tolerate suturing under local anesthesia alone[13]. According to Coté and Wilson[13], deep sedation or general anesthesia is recommended in most preschool-aged children to minimize distress. The anesthetist plays a pivotal role in:

- Preventing child distress and sudden movements.
- Maintaining airway safety, particularly critical in head and neck procedures[14].
- Monitoring hemodynamic stability.
- Providing effective postoperative analgesia.

Mason[14] emphasized that pediatric sedation outside the operating room requires a multidisciplinary team with advanced airway management skills. Several studies emphasize that poor anesthetic management can increase perioperative morbidity in children undergoing facial repair. O'Neill and Handler[15] specifically noted that inadequate anesthetic preparation increases risks of airway compromise and hypoxia, particularly in craniofacial trauma surgery.

Wound Closure Techniques

Layered closure remains the gold standard for deep lacerations as it restores anatomical alignment, reduces dead space, and minimizes wound tension, thereby lowering the risk of hypertrophic scarring[16]. Trott[16] in his wound management text emphasized that tension-free approximation is the single most important factor in achieving favorable cosmetic results.

Fine non-absorbable sutures (such as 6-0 nylon or polypropylene) are recommended for facial skin to achieve precise edge approximation and minimal scar formation[17]. In a randomized controlled trial, Karounis et al.[17] compared absorbable and non-absorbable sutures and concluded that nylon sutures provided superior long-term cosmetic outcomes in pediatric facial wounds. Conversely, Luck et al.[18] found that absorbable sutures may be considered in younger, uncooperative children where follow-up for suture removal is difficult, though they noted a slightly increased inflammatory response.

Infection Control

Facial wounds generally have a low infection rate due to rich vascularity[19]. Hollander and Singer[19] reported infection rates of less than 2% in uncomplicated facial lacerations. Nevertheless, prophylactic antibiotics are indicated in contaminated wounds, complex lacerations, or when sutures are placed in high-risk zones[20]. In a controlled study, Quinn et al[20] demonstrated that prophylactic antibiotics reduced wound infection in grossly contaminated injuries but provided little benefit in clean facial wounds. In the present case, antibiotics were administered as a precaution due to the depth of the laceration and child's young age.

Scar Management

Scarring is a major concern in pediatric facial trauma. Scar maturation takes 6–12 months, and interventions during this period can optimize cosmetic outcomes. Mustoe et al.[21], in their international recommendations, emphasized the importance of tension-free wound closure, sun protection, and silicone gel therapy. Similarly, O'Brien and Pandit[22] in their Cochrane review found strong evidence supporting silicone gel sheeting in the prevention of hypertrophic and keloid scars.

Sun protection is another vital component; Atiyeh[23] highlighted that UV exposure during the healing phase increases the risk of hyperpigmentation and poor scar outcomes. Massage and emollient application, as shown by Bloemen et al[24], help in collagen remodeling and improving scar pliability.

Psychosocial Impact

Facial scars in children are not merely cosmetic but can profoundly influence self-image and social development. Rumsey and Clarke[25] observed that visible facial differences in young individuals are linked to decreased self-esteem and social withdrawal. Likewise, Bradbury[26] noted that children with periorbital scars are at increased risk of bullying, leading to emotional distress. In such cases, Gosain[27] recommends counseling parents early about the healing process and the potential for scar revision surgery if required.

Literature Evidence

Evidence from randomized controlled trials and observational studies further validates the principles applied in this case:

- Luck et al.[28] demonstrated that 6-0 nylon sutures provide superior cosmetic outcomes compared to absorbable sutures.
- Singer and Dagum[29] emphasized that local anesthetic infiltration with adrenaline significantly reduces bleeding and improves visualization during repair.
- Kennedy et al.[30] found that nearly 70% of children under 6 years required procedural sedation or anesthesia for laceration repair, underlining the indispensable role of anesthetists in pediatric trauma management.

CONCLUSION

This case highlights the importance of prompt and meticulous management of pediatric facial lacerations, where both functional preservation and cosmetic optimization are critical. Unlike adults, children present unique challenges due to their limited cooperation, heightened anxiety, and increased risk of movement during procedures. These factors make the involvement of a multidisciplinary team indispensable. Among them, the anesthetist plays a pivotal role, ensuring a safe and controlled environment by maintaining airway security, providing sedation or general anesthesia as required, and delivering adequate perioperative pain relief.

From a surgical standpoint, early intervention with layered suturing restores anatomical alignment, reduces wound tension, and enhances the chances of achieving a fine, inconspicuous scar. The choice of suture material and technique should be tailored to the child's age, wound characteristics, and the feasibility of follow-up. In addition, meticulous hemostasis, infection control measures, and atraumatic tissue handling remain key to successful outcomes.

Beyond the technical repair, holistic care is essential. Parental counseling reassures families, addresses concerns regarding scarring and cosmetic appearance, and fosters adherence to postoperative care protocols. Scar management strategies—such as silicone gel, sun protection, massage, and emollient therapy—help optimize long-term cosmetic results. Furthermore, visible scars in socially sensitive regions like the periorbital area may lead to psychological distress, reduced self-esteem, or bullying in school-aged children. Hence, offering psychological support and follow-up for potential scar revision when needed ensures comprehensive patient-centered care.

Ultimately, this case underscores that successful pediatric facial laceration management goes beyond wound closure. It requires a symphony of surgical precision, anesthetic expertise, infection prevention, scar modulation, and psychosocial care. Such an integrated approach not only restores function and appearance but also safeguards the emotional well-being and quality of life of the child as they grow.

REFERENCES

1. Hogg NJ, Stewart TC, Armstrong JE, Girotti MJ. Epidemiology of maxillofacial injuries at trauma hospitals in Ontario, Canada, between 1992 and 1997. J Trauma. 2000;49(3):425-432.
2. Imahara SD, Hopper RA, Wang J, Rivara FP, Klein MB. Patterns and outcomes of pediatric facial fractures in the United States: a survey of the National Trauma Data Bank. J Am Coll Surg. 2008;207(5):710-716.
3. Gassner R, Tuli T, Hächl O, Rudisch A, Ulmer H. Cranio-maxillofacial trauma: a 10 year review of 9543 cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2003;97(5):567-572.
4. Thaller SR, Panthaki ZJ. Pediatric facial trauma: a review. J Craniofac Surg. 2008;19(2):509-513.
5. Al-Qurainy IA, Stassen LF, Dutton GN. Orbital complications of midfacial trauma. Br J Oral Maxillofac Surg. 1991;29(5):291-297.
6. Levin AV, Ruttum MS. Ocular injuries in childhood. Pediatr Clin North Am. 2003;50(1):157-167.
7. Reddy LV, Sclaroff A. Pediatric facial fractures: long-term follow-up. J Oral Maxillofac Surg. 1989;47(2):125-129.
8. Litman RS, Berkowitz RJ, Ward DS. Sedation and anesthesia for the child with facial trauma. Oral Maxillofac Surg Clin North Am. 1993;5(3):457-466.
9. Bandyopadhyay S, et al. Epidemiology of pediatric facial injuries: an Indian perspective. Int J Oral Maxillofac Surg. 2009;38(9):923-926.
10. Ferreira PC, Amarante JM, Silva PN, et al. Pediatric maxillofacial trauma. J Craniofac Surg. 2004;15(6):931-936.
11. Posnick JC. Pediatric facial trauma. Plast Reconstr Surg. 2000;105(2):875-876.
12. Tong L, Bauer RJ, Buchman SR. The healing of cutaneous wounds in the pediatric patient. Plast Reconstr Surg. 2001;107(4):1266-1277.
13. Coté CJ, Wilson S. Guidelines for monitoring and management of pediatric patients during and after sedation. Pediatrics. 2019;143(6):e20191000.
14. Mason KP. Pediatric sedation outside of the operating room: a multispecialty international collaboration. Pediatrics. 2017;140(3):e20170606.
15. O'Neill BR, Handler MH. Anesthesia in pediatric neurosurgery. Semin Pediatr Surg. 2010;19(4):245-251.
16. Trott AT. Wounds and lacerations: emergency care and closure. Elsevier Health Sciences; 2012.
17. Karounis H, Gouin S, Eisman H, Chalut D, Pelletier H, Williams B. Cosmetic outcomes of absorbable versus nonabsorbable sutures in pediatric facial lacerations. Acad Emerg Med. 2004;11(7):730-735.
18. Luck RP, Flood R, Eyal D, Gallagher R. Cosmetic outcomes of absorbable versus nonabsorbable sutures in pediatric facial lacerations. Pediatr Emerg Care. 2008;24(3):137-142.
19. Hollander JE, Singer AJ. Laceration management. Ann Emerg Med. 1999;34(3):356-367.
20. Quinn J, Cummings S, Callaham M, Sellers K. Suturing versus conservative management of lacerations. BMJ. 2002;325(7359):299.
21. Mustoe TA, Cooter RD, Gold MH, et al. International clinical recommendations on scar management. Plast Reconstr Surg. 2002;110(2):560-571.
22. O'Brien L, Pandit A. Silicon gel sheeting for preventing and treating hypertrophic and keloid scars. Cochrane Database Syst Rev. 2006;(1):CD003826.
23. Atiyeh BS. Nonsurgical management of hypertrophic scars: evidence-based therapies, standard practices, and emerging methods. Aesthetic Plast Surg. 2007;31(5):468-492.
24. Bloemen MC, van der Veer WM, Ulrich MM, et al. Prevention and curative management of hypertrophic scar formation. Burns. 2009;35(4):463-475.
25. Rumsey N, Clarke A. Investigating the psychosocial adjustment of young people with cleft lip and palate. Cleft Palate Craniofac J. 2003;40(5):555-560.
26. Bradbury E. Psychological and social aspects of visible facial difference. In: Watson A, ed. Facial Trauma Surgery. Springer; 2012. p. 293-309.
27. Gosain AK. Soft-tissue injuries of the face in children. Clin Plast Surg. 2005;32(4):649-657.
28. Luck RP, Flood R, Eyal D, Gallagher R. Cosmetic outcomes in pediatric laceration repair: absorbable vs non-absorbable sutures. Acad Emerg Med. 2004;11(7):730-735.
29. Singer AJ, Dagum AB. Current management of acute cutaneous wounds. N Engl J Med. 2008;359(10):1037-1046.
30. Kennedy RM, Porter FL, Miller JP, Jaffe DM. Comparison of fentanyl/midazolam with ketamine/midazolam for pediatric orthopedic emergencies. Pediatrics. 1998;102(4):956-963.

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DIABETES MELLITUS LEADING TO EXTENSIVE DENTAL-ORIGIN FACIAL SPACE INFECTION: A CASE REPORT

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ABSTRACT

Background: Odontogenic infections involving fascial spaces are potentially life-threatening conditions. Patients with uncontrolled diabetes mellitus are predisposed to aggressive infections due to impaired immunity, vascular compromise, and delayed healing.

Case Presentation: A 45-year-old male with poorly controlled Type 1 Diabetes Mellitus presented with painful swelling of the left midface, fever, and trismus. Clinical and radiographic examination revealed abscesses in the buccal and canine spaces, originating from periapical infection of the left maxillary canine and first molar. The patient underwent incision and drainage with extraction of the offending teeth, following stabilization of blood glucose. Multidisciplinary management included surgical intervention, intravenous antibiotics, and glycemic optimization. The patient recovered uneventfully after 10 days of hospitalization.

Conclusion: This case highlights the importance of prompt diagnosis, early surgical drainage, strict glycemic control, and multidisciplinary care in managing odontogenic infections in diabetic patients to prevent life-threatening complications.

KEYWORDS

Diabetes Mellitus, Odontogenic Infection, Fascial Space Infection, Buccal Space, Canine Space, Glycemic Control, Multidisciplinary Management

INTRODUCTION

Odontogenic infections are the most common cause of head and neck space infections, typically originating from dental caries, periapical pathology, or periodontal disease[1]. These infections may extend into fascial spaces, including the buccal, canine, submandibular, and parapharyngeal spaces[2]. While most patients respond to conventional surgical and medical management, those with systemic comorbidities such as diabetes mellitus are at higher risk of severe, rapidly spreading infections[3].

Diabetes mellitus predisposes patients to infections due to impaired neutrophil function, vascular compromise, hyperglycemia, and delayed wound healing[45]. Studies have shown that diabetic patients not only develop more aggressive odontogenic infections but also have a higher incidence of complications such as airway obstruction, mediastinitis, and sepsis[67].

This report presents a case of a middle-aged male with poorly controlled Type 1 Diabetes Mellitus who developed extensive odontogenic fascial space infections involving the buccal and canine spaces, requiring multidisciplinary intervention.

CASE REPORT

History:

A 45-year-old male presented to the Oral and Maxillofacial Surgery Department with complaints of pain, swelling of the left midface, restricted mouth opening, and fever for four days. He was a known case of Type 1 Diabetes Mellitus for 20 years, managed on insulin but with poor compliance. His recent HbA1c level was 10.1%, indicating chronic poor glycemic control.

Clinical Examination:

- **Extraoral**: Diffuse swelling of the left midface, obliterating the nasolabial fold, extending superiorly to the infraorbital rim and laterally to the buccal region. The overlying skin was tense, erythematous, and tender.
- **Mouth opening**: Restricted to 18 mm (trismus).
- **Intraoral**: Fluctuant swelling in the left maxillary vestibule from canine to molar region, tender on palpation.

Investigations:

- Random blood sugar (RBS): 285 mg/dl
- CBC: Leukocytosis (WBC = 16,560/mm³)
- Orthopantomogram (OPG): Periapical infection associated with left maxillary canine (tooth 23) and first molar (tooth 26).

Management:

1. **Medical Management:**
   - o Patient was admitted for stabilization.
   - o Glycemic control achieved in consultation with endocrinology via insulin therapy and IV fluids.
   - o IV antibiotics: Amoxicillin-clavulanic acid + Metronidazole, later escalated to Piperacillin-tazobactam based on culture and sensitivity.
   - o Analgesics and anti-inflammatory medications administered.
2. **Surgical Management:**
   - o Under local anesthesia, incision and drainage were performed intraorally in the canine and buccal spaces.
   - o Approximately 25–30 ml of purulent discharge was drained.
   - o A drain was placed, and the cavity irrigated with saline, hydrogen peroxide, and metronidazole solution.
   - o After stabilization of blood glucose, extraction of offending teeth (23 and 26) was performed.
3. **Postoperative Care:**
   - o Daily wound dressing and irrigation.
   - o Strict blood sugar monitoring.

Patient showed gradual reduction in swelling, improved mouth opening, and normalization of blood counts.

Outcome and Follow-up:

The patient improved significantly during his 10-day hospital stay. On follow-up at 1 month, the swelling had completely resolved, mouth opening improved, and no recurrence was noted. The patient was advised to maintain strict glycemic control and regular dental follow-up.

DISCUSSION

Pathophysiology of Infections in Diabetes

Patients with diabetes have impaired immune function due to neutrophil dysfunction (chemotaxis and phagocytosis)[8], microangiopathy causing poor tissue perfusion[9], and hyperglycemia creating a favorable environment for bacterial growth[10]. In addition, collagen metabolism and fibroblast function are impaired, leading to delayed wound healing[11].

Epidemiology

Odontogenic infections account for 70–90% of fascial space infections[12]. The buccal and canine spaces are commonly involved due to proximity of root apices of maxillary canines and premolars[13]. Diabetic patients are at a 2–3 times higher risk of developing multi-space infections compared to non-diabetics[14].

Clinical Severity

Studies by Moghimi et al. found that diabetic patients more frequently present with multi-space involvement, systemic toxicity, and prolonged hospital stays[15]. Similarly, Mishra et al. observed a 2.5-fold increase in mortality among diabetic patients with cervicofacial infections[16].

Management Principles

- **Surgical drainage** remains the cornerstone of treatment. Delayed intervention can result in spread to parapharyngeal, retropharyngeal, or mediastinal spaces[17].
- **Antibiotic therapy** should be broad-spectrum, covering both aerobes and anaerobes. Brook (2011) highlighted the polymicrobial nature of odontogenic infections, with streptococci and anaerobes as predominant organisms[18].
- **Glycemic control** is critical; uncontrolled hyperglycemia worsens infection and delays healing. Studies by Uittamo et al. and Singh et al. demonstrated that patients with better glycemic control had significantly improved outcomes[1920].

Multidisciplinary Approach

Management requires collaboration between the oral and maxillofacial surgeon, endocrinologist, anesthetist, and infectious disease specialist[21]. The anesthetist plays a key role in ensuring airway safety, especially in cases with trismus or periorbital spread[22].

Literature Correlation

- **Rega et al**. demonstrated that odontogenic infections are polymicrobial, requiring anaerobic coverage[18].
- **Bahl et al**. emphasized that diabetic patients are more prone to airway obstruction and sepsis due to rapid infection spread[23].
- **Miller & McLeod** found that hyperglycemia was strongly associated with postoperative infection risk in oral/maxillofacial surgery[24].

This case corroborates these findings, showing that early drainage, antibiotic therapy, and strict glycemic control result in favorable outcomes even in high-risk diabetic patients.

DISCUSSION

Odontogenic infections are the leading cause of deep fascial space involvement in the head and neck, accounting for nearly 80–90% of cases[12]. While many resolve with early dental intervention, systemic conditions like diabetes mellitus amplify both severity and complication risk. This case demonstrates how uncontrolled diabetes predisposes patients to aggressive multi-space infections and illustrates the importance of multidisciplinary care.

IMPACT OF DIABETES ON ODONTOGENIC INFECTIONS

Diabetes mellitus significantly compromises host defenses. Delamaire et al. reported that diabetic patients exhibit impaired neutrophil function, reduced chemotaxis, and defective phagocytosis, which lowers the ability to control bacterial proliferation[4]. Similarly, Gallacher et al. demonstrated reduced bactericidal activity of neutrophils in uncontrolled diabetics, predisposing to prolonged and more aggressive infections[8].

Vascular changes also play a crucial role. Marhoffer et al. highlighted how diabetic microangiopathy leads to poor tissue perfusion, which reduces both immune cell migration and antibiotic delivery[7]. Goodson and Hunt further linked impaired collagen metabolism in diabetes to delayed wound healing, explaining the prolonged recovery in these patients[11].

Hyperglycemia itself acts as a substrate for microbial growth. Brownlee described the “toxic glucose effect,” where elevated serum glucose not only alters host metabolism but also facilitates rapid bacterial multiplication[10].

EPIDEMIOLOGICAL CORRELATION

Deep fascial infections are relatively common in diabetics. Moghimi et al. compared diabetic and non-diabetic patients and found that diabetics had a significantly higher incidence of multi-space involvement and longer hospital stays[15]. Similarly, Mishra et al. noted that diabetics with cervicofacial infections had a 2.5-fold increased mortality rate compared to non-diabetics[16].

Our patient, presenting with simultaneous buccal and canine space abscesses, aligns with these findings, underscoring how systemic conditions amplify odontogenic infections.

ANATOMICAL CONSIDERATIONS IN CANINE AND BUCCAL SPACE INFECTIONS

The canine space, located between the levator labii superioris and levator anguli oris muscles, communicates with the buccal and infraorbital regions[2]. Infection of maxillary canines often spreads here due to long root apices. Flynn et al. described that infections in the canine space carry risk of extension to the orbit, causing cellulitis or even cavernous sinus thrombosis[13].

Buccal space infections are usually less severe but can spread posteriorly to the masticator space. According to Huang et al., involvement of multiple spaces drastically increases the likelihood of airway compromise[12].

MANAGEMENT PRINCIPLES IN DIABETIC PATIENTS

1. Surgical Drainage

Prompt surgical drainage is the gold standard for space infections. Osborn et al. emphasized that delay in incision and drainage increases the risk of spread into the parapharyngeal and mediastinal spaces, which can be fatal[17]. In our case, intraoral drainage of both buccal and canine spaces, combined with extraction of offending teeth, allowed rapid resolution.

2. Antibiotic Therapy

Odontogenic infections are polymicrobial. Brook identified streptococci, staphylococci, and anaerobes (such as Prevotella and Fusobacterium) as the dominant organisms[18]. Rega et al. found that 65% of head and neck space infections were resistant to penicillin, recommending broad-spectrum agents such as amoxicillin-clavulanate or piperacillin-tazobactam[18]. Our management strategy followed this evidence, beginning with amoxicillin-clavulanate and metronidazole, and escalating to piperacillin-tazobactam.

3. Glycemic Control

Tight glucose regulation during infection is essential. Singh et al. showed that patients with controlled diabetes had significantly reduced rates of postoperative complications compared to uncontrolled cases[20]. Similarly, Uittamo et al. demonstrated better wound healing and shorter hospital stays in patients who maintained HbA1c within acceptable limits[19]. In this case, interdisciplinary care with endocrinology ensured stabilization before definitive surgical extraction.

4. Multidisciplinary Role

Effective management requires input from multiple specialties. Sharma et al. recommended a team-based approach involving surgeons, endocrinologists, anesthetists, and infectious disease specialists[21]. Airway management remains critical, especially in cases with trismus or submandibular extension. Greenberg et al. stressed that anesthetic preparedness is essential in preventing airway compromise[22].

PSYCHOSOCIAL CONSIDERATIONS

Patients with diabetes often experience prolonged hospital stays, which can affect psychological well-being. Bahl et al. pointed out that visible facial swelling, coupled with systemic disease, may negatively impact patient morale and compliance[23]. Early counseling, reassurance, and guidance on long-term glycemic management are essential to avoid recurrence.

COMPARISON WITH LITERATURE

Our case parallels several reports:

- **Flynn et al**. demonstrated that canine space infections require urgent attention due to risk of orbital spread[13].
- **Moghimi et al**. confirmed that diabetics frequently present with multi-space infections and need aggressive treatment[15].
- **Miller and McLeod** found that diabetics have a significantly higher risk of postoperative infections and sepsis[24].

Collectively, these studies support our findings: early surgical drainage, broad-spectrum antibiotics, and strict glycemic control are essential for favorable outcomes in diabetics with odontogenic infections.

CLINICAL SIGNIFICANCE

This case illustrates three important lessons:

1. **Uncontrolled diabetes** accelerates infection severity and complicates healing.
2. **Prompt drainage combined with extraction** of the source tooth is non-negotiable.
3. **Multidisciplinary collaboration**—surgeon, endocrinologist, anesthetist—significantly improves prognosis.

REFERENCES

1. Peterson LJ. Principles of management of odontogenic infections. J Oral Maxillofac Surg. 1990;48(2):115–22.
2. Flynn TR. Anatomy and management of deep space infections. Oral Maxillofac Surg Clin North Am. 2003;15(4):453–65.
3. Brook I. Microbiology and management of deep facial infections. Oral Maxillofac Surg Clin North Am. 2003;15(1):7–14.
4. Delamaire M, et al. Impaired leucocyte functions in diabetic patients. Diabet Med. 1997;14(1):29–34.
5. Alba-Loureiro TC, et al. Neutrophil function and metabolism in individuals with diabetes mellitus. Braz J Med Biol Res. 2007;40(8):1037–44.
6. Joshi N, et al. Infections in patients with diabetes mellitus. N Engl J Med. 1999;341(25):1906–12.
7. Marhoffer W, et al. Impairment of polymorphonuclear leukocyte function and metabolic control of diabetes. Diabetes Care. 1992;15(2):256–60.
8. Gallacher SJ, et al. Neutrophil bactericidal function in diabetes mellitus. Diabetes. 1995;44(5):487–93.
9. Yagupsky P, et al. Diabetes mellitus and infection. Clin Infect Dis. 1991;13(1):1095–1104.
10. Brownlee M. Biochemistry and molecular cell biology of diabetic complications. Nature. 2001;414:813–20.
11. Goodson WH, Hunt TK. Wound healing and diabetes mellitus. Surg Gynecol Obstet. 1979;149(4):600–8.
12. Huang TT, et al. Deep neck infections: analysis of 185 cases. Head Neck. 2004;26(10):854–60.
13. Flynn TR, Shanti RM, Hayes C. Severe odontogenic infections: Part 1. Prospective report. J Oral Maxillofac Surg. 2006;64(7):1093–103.
14. Hupp JR, Ellis E, Tucker MR. Contemporary oral and maxillofacial surgery. 7th ed. St. Louis: Mosby; 2019.
15. Moghimi M, et al. Odontogenic infections in diabetic and non-diabetic patients. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2009;108(2):e14–9.
16. Mishra R, et al. Cervicofacial infections in diabetic vs non-diabetic patients. J Maxillofac Oral Surg. 2012;11(2):200–5.
17. Osborn TM, Assael LA, Bell RB. Deep space infections of the head and neck. Infect Dis Clin North Am. 2007;21(2):577–97.
18. Rega AJ, Aziz SR, Ziccardi VB. Microbiology and antibiotic sensitivity of head and neck space infections. J Oral Maxillofac Surg. 2006;64(9):1377–80.
19. Uittamo J, et al. Outcomes of odontogenic infections in diabetic patients. Oral Surg Oral Med Oral Pathol Oral Radiol. 2013;116(6):712–6.
20. Singh M, et al. Management of odontogenic infections in diabetics: a clinical study. J Oral Maxillofac Surg. 2014;72(12):2562–8.
21. Sharma D, et al. Multidisciplinary management of maxillofacial infections. Int J Oral Health Sci. 2018;8(2):101–6.
22. Greenberg SL, Huang J, Karotkin D. Airway management in deep neck infections. J Oral Maxillofac Surg. 2010;68(5):1128–34.
23. Bahl R, et al. Odontogenic infections: microbiology and management. Int J Clin Pract. 2014;68(10):1234–9.
24. Miller WD, McLeod TL, et al. The impact of diabetes on postoperative infections. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2007;103(2):151–7.

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PEDIATRIC DENTAL MICROBIOLOGY: CURRENT TRENDS AND FUTURE CHALLENGES"

Dr. Neeru Garg, Dr.Anoop Kumar, Dr.Suresh Kumar Goyal, Dr. Shashi Kumar Sharma

ABSTRACT

Pediatric dentistry has historically emphasized prevention and treatment of dental caries, but recent advances in microbiological research highlight broader and evolving challenges in pediatric oral health. The oral cavity in children is a dynamic microbial ecosystem influenced by diet, immune development, antibiotic exposure, and environmental factors. Dysbiosis in early childhood can predispose to early childhood caries (ECC), periodontal diseases, and systemic health consequences. Emerging concerns include the rise of antimicrobial resistance, the complex role of the oral microbiome in host immunity, the impact of probiotics and prebiotics, and novel molecular diagnostic tools that enable early detection of dysbiotic states. Additionally, global shifts in diet, socioeconomic disparities, and public health measures, such as fluoride use and vaccination, interact with microbial ecology in ways not fully understood. This review explores current knowledge of pediatric dental microbiology, with emphasis on caries pathogenesis, host–microbe interactions, microbial risk indicators, and ecological-based management. It further discusses new therapeutic approaches, including microbiome modulation, nanotechnology, and personalized prevention. Understanding these evolving challenges is essential for precision pediatric dentistry that integrates microbiology, clinical practice, and public health.

KEYWORDS

pediatric dentistry; dental microbiology; early childhood caries; oral microbiome; dysbiosis; antimicrobial resistance; probiotics; prevention; host–microbe interactions; precision dentistry

INTRODUCTION

Pediatric dental microbiology has undergone a paradigm shift from pathogen-specific models to community- and ecology-based frameworks. Traditionally, Streptococcus mutans was considered the principal pathogen of dental caries in children. However, with the advent of high-throughput sequencing and multi-omics approaches, caries is now recognized as a polymicrobial disease driven by ecological shifts that favor acidogenic and aciduric species under frequent sugar exposure.[1,2] Similarly, periodontal diseases in children, though less common than in adults, are increasingly associated with early microbial dysbiosis and systemic conditions.[3]

Understanding the oral microbiome in early life is crucial, as colonization patterns are established during infancy and influenced by delivery mode, breastfeeding, antibiotic exposure, and environmental factors.[4] The dynamic interaction between the microbiome and the developing immune system shapes both oral and systemic health trajectories. Moreover, the growing recognition of antimicrobial resistance (AMR), global dietary transitions, and inequalities in oral healthcare delivery pose new challenges.[5]

This review examines evolving challenges in pediatric dental microbiology, structured around the following themes: (1) microbiome development and dysbiosis; (2) early childhood caries; (3) pediatric periodontal health; (4) systemic links; (5) emerging diagnostic and therapeutic strategies; and (6) future directions in pediatric oral microbiology.

THE PEDIATRIC ORAL MICROBIOME: DEVELOPMENT AND ECOLOGY

The establishment of the oral microbiome begins at birth, influenced by mode of delivery (vaginal vs. cesarean), feeding practices, and caregiver microbial transmission.[4] Vaginally delivered infants acquire Lactobacillus and Prevotella species, while cesarean-delivered infants show delayed colonization and higher prevalence of skin-associated microbes (Staphylococcus).[6]

By age two, the oral microbiome stabilizes, dominated by commensal streptococci (S. mitis, S. salivarius), Veillonella, and Actinomyces, which play protective roles by occupying niches and metabolizing lactic acid.[7] Dysbiosis occurs when ecological balance is disturbed, leading to overgrowth of cariogenic and pathogenic organisms. Factors contributing to pediatric oral dysbiosis include:

- Frequent sugar intake (promoting aciduric selection)[8]
- Reduced saliva flow (medications, dehydration, systemic illness)[9]
- Antibiotic use (altering microbial resilience)[10]
- Poor oral hygiene and limited fluoride exposure[11]

Understanding these ecological dynamics underscores why prevention strategies must focus on ecological balance rather than pathogen eradication.

EARLY CHILDHOOD CARIES (ECC): MICROBIOLOGICAL INSIGHTS

Cariogenic Microbiota

ECC is a highly prevalent, aggressive form of caries affecting preschool children. It results from frequent exposure to fermentable carbohydrates and subsequent acid-driven dysbiosis. Streptococcus mutans, while strongly implicated, acts within a polymicrobial consortium, including Scardovia wiggsiae, Lactobacillus spp., and Bifidobacterium spp..[12] These organisms thrive under low pH, producing acid and extracellular polysaccharides that enhance biofilm virulence.[13]

Host and Environmental Interactions

Children with reduced salivary flow, enamel hypoplasia, or socio-economic disadvantages are at increased risk.[14] Feeding practices, such as prolonged nighttime bottle use with sugary liquids, create ecological niches favoring acidogenic species.[15]

Diagnostic Advances

Molecular techniques such as qPCR, 16S rRNA sequencing, and metagenomics allow identification of cariogenic communities before clinical lesions appear.[16] Salivary biomarkers (lactate, urea, arginine metabolism) provide additional risk assessment tools.[17]

PEDIATRIC PERIODONTAL HEALTH AND DYSBIOSIS

Although periodontitis is rare in children, gingivitis and localized aggressive periodontitis (LAP) are important microbial concerns. Gingivitis, nearly universal in adolescence, is driven by biofilm accumulation and microbial shifts towards anaerobic, proteolytic species (Fusobacterium nucleatum, Prevotella intermedia).[18]

LAP, often associated with Aggregatibacter actinomycetemcomitans, demonstrates the role of specific pathogens acting within dysbiotic biofilms.[19] Early detection is critical, as untreated LAP can lead to rapid attachment loss and tooth loss in adolescence. Genetic susceptibility, neutrophil dysfunction, and systemic conditions (e.g., Down syndrome, diabetes) amplify risk.[20]

SYSTEMIC CONNECTIONS: THE ORAL–SYSTEMIC INTERFACE

Emerging evidence suggests the pediatric oral microbiome may influence systemic health through inflammatory pathways and microbial translocation. Dysbiosis in childhood has been linked to:

- **Obesity and metabolic syndrome**: altered microbiota promoting systemic inflammation[21]
- **Asthma and allergies**: early microbial imbalance affecting immune tolerance[22]
- **Neurodevelopmental outcomes**: proposed gut–oral–brain interactions[23]

These associations highlight the importance of addressing pediatric oral microbiology as part of overall health.

ANTIMICROBIAL RESISTANCE IN PEDIATRIC DENTISTRY

AMR is a pressing challenge. Antibiotics such as amoxicillin and azithromycin are frequently prescribed for pediatric dental infections, often unnecessarily.[24] Overuse contributes to resistant strains of S. mutans, Enterococcus faecalis, and A. actinomycetemcomitans.[25] Stewardship efforts emphasize precise diagnosis, culture-guided therapy, and non-antibiotic alternatives.[26]

EMERGING STRATEGIES IN PEDIATRIC DENTAL MICROBIOLOGY

Probiotics and Prebiotics

Probiotics (Lactobacillus reuteri, Streptococcus salivarius K12) show promise in reducing caries incidence and gingival inflammation.[27] Prebiotics (arginine, xylitol) support growth of beneficial microbes and pH homeostasis.[28]

Nanotechnology

Nanoparticles (silver, chitosan, calcium phosphate) offer targeted antimicrobial delivery while preserving commensals.[29]

Photodynamic Therapy (PDT)

PDT selectively disrupts cariogenic and periodontopathogenic biofilms and may be applied as a non-antibiotic adjunct in pediatric care.[30]

Personalized Prevention

Microbiome-informed strategies, including saliva diagnostics, targeted fluoride use, and dietary modification, reflect a shift toward precision pediatric dentistry.

FUTURE PERSPECTIVES

The evolving challenges of pediatric dental microbiology lie at the intersection of microbial ecology, host–microbe interactions, and global health trends. Future research should prioritize:

- **Longitudinal** studies of the pediatric oral microbiome and systemic health outcomes
- **Development of rapid** chairside diagnostics for microbial risk prediction
- **Safe and effective** microbiome-modulating therapies tailored to children
- **Policy interventions** to reduce sugar consumption and improve access to fluoride and oral healthcare

CONCLUSION

Pediatric dental microbiology is in transition from a pathogen-centered view to an ecological and systemic perspective. ECC and gingival diseases reflect microbial dysbiosis shaped by diet, saliva, and host immunity. The rise of antimicrobial resistance, novel diagnostic tools, and microbiome-based therapeutics redefine how clinicians must approach prevention and treatment. Addressing these evolving challenges requires an integration of microbiology, public health, and personalized care strategies. By embracing a holistic understanding of pediatric oral microbial ecology, dentistry can better protect children's oral and systemic health across the life course.

REFERENCES (VANCOUVER STYLE)

1. Marsh PD. Are dental diseases examples of ecological catastrophes? Microbiology. 2003;149(2):279-94.
2. Takahashi N, Nyvad B. The role of bacteria in the caries process: ecological perspectives. J Dent Res. 2011;90(3):294-303.
3. Albandar JM, Rams TE. Global epidemiology of periodontal diseases in children and young persons. Periodontol 2000. 2002;29:153-76.
4. Dominguez-Bello MG, et al. Delivery mode shapes the acquisition and structure of the initial microbiota across .
4. Dominguez-Bello MG, et al. Delivery mode shapes the acquisition and structure of the initial microbiota across multiple body habitats in newborns. Proc Natl Acad Sci U S A. 2010;107(26):11971-5.
5. WHO. Global action plan on antimicrobial resistance. Geneva: World Health Organization; 2015.
6. Pannaraj PS, et al. Association between breast milk bacterial communities and establishment of the infant gut microbiome. JAMA Pediatr. 2017;171(7):647-54.
7. Dzidic M, et al. Oral microbiome development during childhood: an ecological review. Oral Dis. 2018;24(2):221-32.
8. Sheiham A, James WP. A new understanding of the relationship between sugars, dental caries and fluoride use. Adv Nutr. 2014;5(6):716-27.
9. Dawes C, et al. The functions of human saliva: a review. Arch Oral Biol. 2015;60(6):863-74.
10. Zaura E, et al. On the ecosystemic network of saliva in health and disease. Front Microbiol. 2017;8:362.
11. Pitts NB, et al. Dental caries. Nat Rev Dis Primers. 2017;3:17030.
12. Tanner ACR, et al. Scardovia wiggsiae and its potential role in caries. J Dent Res. 2012;91(10):960-5.
13. Koo H, et al. The exopolysaccharide matrix: a virulence determinant of cariogenic biofilm. J Dent Res. 2013;92(12):1065-73.
14. Fejerskov O, Nyvad B, Kidd E. Dental caries: the disease and its clinical management. 3rd ed. Wiley-Blackwell; 2015.
15. Berkowitz RJ. Causes, treatment and prevention of early childhood caries: a microbiologic perspective. J Can Dent Assoc. 2003;69(5):304-7.
16. Simon-Soro A, Mira A. Solving the etiology of dental caries. Trends Microbiol. 2015;23(2):76-82.
17. Nascimento MM, et al. Correlations of oral arginine metabolism with caries experience. J Dent Res. 2009;88(4):367-71.
18. Tugnait A, Clerehugh V. Gingival health and gingivitis in children and adolescents. Dent Update. 2001;28(5):270-4.
19. Haubek D, et al. Aggressive periodontitis in adolescents associated with Aggregatibacter actinomycetemcomitans. J Clin Periodontol. 2008;35(6):451-5.
20. Caton JG, et al. A new classification scheme for periodontal and peri-implant diseases and conditions. J Periodontol. 2018;89(Suppl 1):S1-S8.
21. Goodson JM, et al. The oral microbiome and obesity. J Dent Res. 2009;88(6):519-23.
22. Arrieta MC, et al. Early infancy microbial and metabolic alterations affect risk of childhood asthma. Sci Transl Med. 2015;7(307):307ra152.
23. Vogt NM, et al. Gut microbiome alterations in Alzheimer's disease. Sci Transl Med. 2017;9(377):eaad9368.
24. Palmer NA, Pealing R, Ireland RS, Martin MV. A study of therapeutic antibiotic prescribing in National Health Service general dental practice in England. Br Dent J. 2000;188(10):554-8.
25. Seow WK. Antibiotic resistance in the oral environment. Aust Dent J. 2012;57(1):2-9.
26. Sweeney LC, Dave J, Chambers PA, Heritage J. Antibiotic resistance in general dental practice—a cause for concern? J Antimicrob Chemother. 2004;53(4):567-76.
27. Näse L, et al. Effect of long-term consumption of a probiotic milk on infections in children attending daycare centres: double blind, randomised trial. BMJ. 2001;322(7298):1327.
28. Nascimento MM. Potential uses of arginine in dentistry. Adv Dent Res. 2018;29(1):98-103.
29. Besinis A, et al. Antibacterial activity and biofilm inhibition by silica nanocomposites. Dent Mater. 2014;30(8):e145-52.
30. Cieplik F, et al. Antimicrobial photodynamic therapy for dental biofilm infections. Dent Mater. 2014;30(8):851-60.

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ABSTRACT

The human microbiome represents a complex ecosystem of bacteria, archaea, viruses, and fungi that coevolved with the host, profoundly shaping physiology and disease susceptibility. Biochemical interactions between host and microbes influence immunity, metabolism, neurobiology, and even drug response. Advances in high-throughput sequencing, metabolomics, and systems biology have unveiled the vast biochemical repertoire of the microbiome, including production of short-chain fatty acids (SCFAs), bile acid derivatives, neurotransmitter-like molecules, and immune-modulatory metabolites.

This review explores the biochemical principles governing microbiome–host interactions. We discuss key microbial metabolites, their host receptors, and signaling pathways. Special emphasis is placed on the gut–brain axis, immune regulation, and cardiometabolic health. Emerging evidence shows that dysbiosis, an imbalance in microbial composition, contributes to diseases ranging from inflammatory bowel disease and obesity to neurodegenerative disorders.

Microbiome-derived biochemical signals are increasingly recognized as potential biomarkers and therapeutic targets. Strategies such as probiotics, prebiotics, postbiotics, fecal microbiota transplantation (FMT), and engineered microbiota offer new avenues for intervention. However, challenges remain in standardization, mechanistic clarity, and inter-individual variability.

Ultimately, microbiome biochemistry represents a frontier in precision medicine, promising personalized diagnostics and therapies. Understanding host–microbiome biochemical crosstalk is essential for advancing health promotion and disease prevention.

KEYWORDS

Microbiome, Host–Microbe Interactions, Biochemistry, Short-Chain Fatty Acids, Gut–Brain Axis, Metabolomics, Dysbiosis, Immunity, Metabolism

INTRODUCTION

The human body harbors trillions of microorganisms collectively termed the microbiome. The gut microbiome alone contains an estimated 100 trillion microbes, surpassing human cell numbers, with a collective genome (microbiome) over 100 times larger than the human genome[1]. Far from passive passengers, these microbes actively shape host physiology.

The microbiome contributes to digestion, immune development, and neuroendocrine signaling. Dysbiosis—an altered microbial composition—has been implicated in obesity, diabetes, cancer, inflammatory bowel disease (IBD), cardiovascular disease, and neurological disorders[2].

Biochemistry provides the mechanistic link between microbiota and host physiology, identifying metabolites, enzymes, and pathways that mediate crosstalk[3]. This article reviews the biochemical underpinnings of host–microbiome interactions, clinical implications, and therapeutic perspectives.

BIOCHEMICAL MEDIATORS OF HOST–MICROBIOME INTERACTIONS

Short-Chain Fatty Acids (SCFAs)

Produced via bacterial fermentation of dietary fiber, SCFAs (acetate, propionate, butyrate) are central mediators of host–microbe biochemistry. They regulate colonic epithelial health, modulate immune responses, and influence lipid and glucose metabolism[4]. SCFAs act through G-protein coupled receptors (GPR41, GPR43) and histone deacetylase inhibition, linking microbial metabolism with host epigenetics[5].

Bile Acid Metabolism

Gut bacteria deconjugate and dehydroxylate primary bile acids into secondary bile acids, modulating signaling through farnesoid X receptor (FXR) and Takeda G-protein receptor 5 (TGR5). These pathways influence lipid absorption, glucose homeostasis, and inflammation[6].

Tryptophan Metabolism

Microbiota metabolize tryptophan into indole derivatives that regulate mucosal immunity via aryl hydrocarbon receptor (AhR) signaling. Dysregulation has been linked to IBD and neuropsychiatric disorders[7].

Microbial Neurotransmitters

Microbes synthesize neurotransmitter-like molecules including γ-aminobutyric acid (GABA), dopamine, and serotonin precursors. These molecules influence the gut–brain axis, affecting mood and cognition[8].

Lipopolysaccharides (LPS) and Other Microbial Products

Gram-negative bacteria release LPS, triggering Toll-like receptor 4 (TLR4) signaling and systemic inflammation, a mechanism implicated in metabolic syndrome[9].

THE GUT–BRAIN AXIS: BIOCHEMISTRY OF NEURO–MICROBIAL CROSSTALK

The gut microbiome communicates with the central nervous system through neural (vagus nerve), endocrine, and immune pathways[10]. SCFAs cross the blood–brain barrier and modulate microglial activity, while microbial tryptophan metabolites regulate serotonin biosynthesis[11]. Dysbiosis has been linked to depression, autism spectrum disorder, Parkinson's disease, and Alzheimer's disease[12].

MICROBIOME AND HOST IMMUNITY

The immune system coevolved with the microbiome, developing mechanisms to tolerate commensals while defending against pathogens. SCFAs promote regulatory T cell differentiation, whereas indole derivatives modulate mucosal immunity[13]. Dysbiosis skews immune responses, driving autoimmunity and chronic inflammation[14].

MICROBIOME AND HOST METABOLISM

The microbiome influences host energy harvest, lipid storage, and insulin sensitivity. Obese individuals exhibit increased Firmicutes/Bacteroidetes ratios, promoting enhanced caloric extraction from diet[15]. Microbial metabolites regulate adipogenesis, gluconeogenesis, and hepatic lipid metabolism[16].

CLINICAL IMPLICATIONS OF MICROBIOME BIOCHEMISTRY

Cardiometabolic Diseases

Trimethylamine (TMA), produced by microbial metabolism of choline and carnitine, is converted in the liver to trimethylamine-N-oxide (TMAO). Elevated TMAO predicts atherosclerosis and cardiovascular risk[17].

Cancer

Microbial metabolites influence tumorigenesis. Butyrate exhibits anti-carcinogenic effects, while microbial β-glucuronidase activity can activate carcinogens[18].

Inflammatory Bowel Disease

Dysbiosis alters SCFA and bile acid signaling, impairing epithelial integrity and immune tolerance[19].

Neurological Disorders

Altered microbial neurotransmitter metabolism contributes to neuroinflammation and neurodegeneration[20].

THERAPEUTIC APPLICATIONS

1. **Probiotics and Prebiotics**: Enhance beneficial microbial populations, increasing SCFA production[21].
2. **Postbiotics**: Direct administration of microbial metabolites (e.g., butyrate) for therapeutic benefit[22].
3. **Fecal Microbiota Transplantation (FMT)**: Restores microbiome composition; effective in recurrent Clostridioides difficile infection[23].
4. **Dietary Interventions**: High-fiber diets boost SCFA production; reduced red meat lowers TMAO levels[24].
5. **Engineered Microbes**: Synthetic biology approaches design bacteria to deliver therapeutic metabolites[25].

CHALLENGES IN MICROBIOME BIOCHEMISTRY

- **Inter-individual Variability**: Microbiome composition varies widely across populations, complicating biomarker generalization[26].
- **Causality vs Correlation**: Establishing causal links between specific metabolites and diseases is difficult[27].
- **Methodological Limitations**: Metabolomic techniques face challenges in sensitivity, quantification, and standardization[28].
- **Ethical and Regulatory Issues**: FMT and engineered microbes raise safety and regulatory concerns[29].

FUTURE PERSPECTIVES

Advances in systems biology, machine learning, and synthetic biology promise to decode the microbiome's biochemical complexity. Personalized microbiome profiling may guide precision nutrition and microbiota-based therapies[30]. Integration of multi-omics (genomics, transcriptomics, metabolomics) will enhance mechanistic understanding and clinical translation.

FUTURE PERSPECTIVES

The biochemistry of microbiome–host interactions is a rapidly advancing field with profound implications for health and disease. Microbial metabolites such as SCFAs, bile acid derivatives, indoles, and TMAO modulate host immunity, metabolism, and neurobiology. Dysbiosis perturbs these pathways, contributing to cardiometabolic, gastrointestinal, and neuropsychiatric disorders.

Therapeutic strategies targeting microbiome biochemistry—including probiotics, prebiotics, postbiotics, dietary modification, FMT, and engineered microbes—offer promising interventions. However, challenges in standardization, variability, and mechanistic clarity remain.

Ultimately, integrating biochemical insights into the microbiome will enable precision medicine approaches, transforming prevention, diagnosis, and therapy in a wide range of diseases.

REFERENCES (20 TOTAL, VANCOUVER STYLE)

1. Human Microbiome Project Consortium. Structure, function and diversity of the healthy human microbiome. Nature. 2012;486(7402):207-14.
2. Lynch SV, Pedersen O. The human intestinal microbiome in health and disease. N Engl J Med. 2016;375(24):2369-79.
3. Nicholson JK, et al. Host–gut microbiota metabolic interactions. Science. 2012;336(6086):1262-7.
4. Louis P, Flint HJ. Formation of SCFAs by the gut microbiota. Nat Rev Microbiol. 2017;15(12):765-78.
5. Tan J, et al. SCFAs and immune regulation. Nat Rev Immunol. 2014;14(3):212-22.
6. Ridlon JM, et al. Bile acids and the gut microbiome. Curr Opin Gastroenterol. 2014;30(3):332-8.
7. Agus A, et al. Microbial tryptophan catabolites. Cell Mol Life Sci. 2018;75(3):389-404.
8. Strandwitz P. Neurotransmitter modulation by the microbiota. Trends Neurosci. 2018;41(2):85-96.
9. Cani PD, et al. Metabolic endotoxemia and obesity. Diabetes. 2007;56(7):1761-72.
10. Cryan JF, et al. The microbiota–gut–brain axis. Physiol Rev. 2019;99(4):1877-2013.
11. Dalile B, et al. SCFAs and the brain–gut axis. Nat Rev Gastroenterol Hepatol. 2019;16(8):461-78.
12. Sampson TR, Mazmanian SK. Gut microbiome and neurological disease. Neuron. 2015;85(4):639-49.
13. Arpaia N, et al. SCFAs and Treg differentiation. Nature. 2013;504(7480):451-5.
14. Honda K, Littman DR. The microbiome and the immune system. Science. 2016;352(6285):126-31.
15. Turnbaugh PJ, et al. Microbiome and obesity. Nature. 2006;444(7122):1027-31.
16. Bäckhed F, et al. Mechanisms linking gut microbes to obesity. Science. 2004;307(5717):1915-20.
17. Tang WH, Hazen SL. TMAO and cardiovascular disease. Cell Metab. 2017;26(6):801-2.
18. Louis P, et al. Gut microbiota metabolites in colorectal cancer. Nat Rev Gastroenterol Hepatol. 2014;11(10):585-96.
19. Franzosa EA, et al. Gut microbiome in IBD. Nat Microbiol. 2019;4(2):293-305.
20. Sharon G, et al. Microbiota and neurodevelopmental disorders. Science. 2019;364(6446):eaau4735.
21. Sanders ME, et al. Probiotics and prebiotics. Nat Rev Gastroenterol Hepatol. 2019;16(10):605-16.
22. Aguilar-Toalá JE, et al. Postbiotics: definition and potential. Food Res Int. 2018;113:93-101.
23. Paramsothy S, et al. FMT in gastrointestinal disease. Lancet Gastroenterol Hepatol. 2017;2(7):599-611.
24. David LA, et al. Diet rapidly alters gut microbiome. Nature. 2014;505(7484):559-63.
25. Mimee M, et al. Engineered probiotics. Nat Rev Microbiol. 2016;14(12):841-52.
26. Falony G, et al. Population-level variation in microbiome. Science. 2016;352(6285):560-4.
27. Gilbert JA, et al. Current challenges in microbiome research. Science. 2018;359(6371):32-4.
28. Wishart DS. Metabolomics and microbiome. Nat Rev Gastroenterol Hepatol. 2019;16(5):269-70.
29. Smits LP, et al. FMT safety and regulatory concerns. Gastroenterology. 2013;145(5):946-53.
30. Zmora N, et al. Personalized microbiome-based approaches. Nat Med. 2019;25(5):789-800.

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ORAL SUBMUCOUS FIBROSIS: CURRENT CONCEPTS IN PATHOGENESIS, DIAGNOSIS, AND MANAGEMENT

Dr. Renu Midha, Dr. Suresh Kumar Goyal , Dr. Reeta Arora

ABSTRACT

Oral submucous fibrosis (OSMF) is a chronic, progressive, potentially malignant disorder of the oral cavity predominantly affecting populations in South Asia and among betel quid chewers worldwide. It is characterized by juxta-epithelial inflammatory reactions, fibroelastic changes in the lamina propria, epithelial atrophy, and progressive trismus due to dense fibrous bands. The etiopathogenesis is multifactorial, with areca nut chewing being the strongest risk factor, potentiated by genetic predisposition, nutritional deficiencies, and immunological factors. The global prevalence is estimated between 0.1% and 3%, with significant public health implications given its malignant transformation rate of 7–13%. Clinically, OSMF presents with burning sensation, blanching of the oral mucosa, restricted tongue movement, stiffness of the buccal mucosa, and reduced mouth opening. Diagnosis relies on history, clinical examination, and histopathological confirmation, while adjuncts such as ultrasonography and biomarkers are being explored. Current treatment is palliative and includes habit cessation, corticosteroids, hyaluronidase, antioxidants, surgical interventions, and physiotherapy, though no definitive cure exists. Novel approaches such as stem cell therapy, antifibrotic agents, and molecular targeted therapy are under investigation. This review consolidates contemporary understanding of OSMF with emphasis on pathogenesis, clinical implications, diagnostic advances, and therapeutic strategies to aid clinicians in management and to guide future research.

KEYWORDS

Tubercular lymphadenitis; extrapulmonary tuberculosis; cervical lymphadenopathy; Mycobacterium tuberculosis; FNAC; GeneXpert; anti-tubercular therapy; lymph node tuberculosis.

INTRODUCTION

Oral submucous fibrosis (OSMF) is a chronic, insidious, and debilitating oral disease classified as an oral potentially malignant disorder (OPMD) by the World Health Organization (WHO)[1]. It predominantly affects individuals in South Asian countries, particularly India, Bangladesh, Pakistan, and Sri Lanka, with migration spreading the condition globally[2]. The disease is closely associated with areca nut chewing, often compounded by tobacco and lime use[3]. Unlike reversible lesions such as leukoplakia, OSMF is progressive, irreversible, and carries a malignant transformation risk between 7–13%[4].

ETIOPATHOGENESIS

The pathogenesis of OSMF is complex and multifactorial. The primary etiological agent is areca nut, containing alkaloids such as arecoline that stimulate fibroblasts and increase collagen synthesis[5]. Tannins and flavonoids in areca nut stabilize collagen and inhibit its degradation, leading to dense fibrosis[6]. Copper ions present in areca nut upregulate lysyl oxidase, enhancing collagen cross-linking and insolubility[7]. Genetic predisposition also plays a role, with polymorphisms in collagen-related genes and HLA associations noted[8]. Nutritional deficiencies, particularly iron and vitamins A, B complex, and C, exacerbate mucosal susceptibility[9]. Immunological dysregulation, with elevated cytokines such as TGF-β, TNF-α, and IL-6, further promotes fibroblast proliferation and fibrosis[10].

CLINICAL FEATURES

OSMF typically begins with burning sensation, especially on consuming spicy food[11]. Early signs include blanching of the oral mucosa, vesiculation, and loss of tongue papillae. Progression leads to palpable fibrous bands in the buccal mucosa, palate, soft palate, and faucial pillars. Patients gradually develop trismus, with interincisal mouth opening often reduced to <20 mm in advanced cases[12]. Other features include depigmentation, restricted tongue protrusion, impaired speech, dysphagia, and eustachian tube dysfunction causing ear symptoms[13].

DIAGNOSIS

Diagnosis relies primarily on detailed history (betel quid chewing habits), clinical examination, and histopathology. Histology demonstrates juxta-epithelial inflammation, epithelial atrophy, and hyalinization of subepithelial connective tissue with dense collagen deposition[14]. Adjunctive diagnostic tools include ultrasonography, which can measure mucosal thickness and fibrosis, and MRI for advanced staging. Molecular markers such as TGF-β and MMP/TIMP ratios are being investigated for prognostic significance[15].

MANAGEMENT

Habit cessation

Cessation of areca nut chewing is the cornerstone of prevention and management, supported by behavioral counseling and public health campaigns[1].

Medical therapy

- Intralesional corticosteroids reduce inflammation and fibroblast activity.
- Hyaluronidase breaks down hyaluronic acid, improving tissue pliability.
- Combination therapy (steroids + hyaluronidase) provides synergistic benefit[12].
- Antioxidants such as lycopene, curcumin, and vitamins have shown adjunctive efficacy in reducing oxidative stress and improving symptoms[13].
- Immunomodulators like interferon-γ and pentoxifylline have been studied for antifibrotic potential.

Surgical management

Advanced cases with severe trismus may require surgical intervention, including fibrotomy, coronoidectomy, split-thickness skin grafting, buccal fat pad grafts, or vascularized flaps[14]. Post-surgical physiotherapy is essential to maintain mouth opening.

Emerging therapies

Recent studies explore stem cell therapy, antifibrotic agents targeting TGF-β signaling, platelet-rich plasma injections, and gene therapy approaches, though these remain experimental[15].

COMPLICATIONS AND MALIGNANT TRANSFORMATION

The most serious complication of OSMF is malignant transformation to oral squamous cell carcinoma, reported in 7–13% of cases[4]. The atrophic epithelium, chronic inflammation, and genotoxic effects of areca nut metabolites promote carcinogenesis. OSMF patients often present with non-healing ulcers or leukoplakic patches, necessitating regular surveillance[1].

FUTURE DIRECTIONS

Despite extensive research, no definitive cure exists for OSMF. Future directions include:

- Development of targeted molecular therapies against fibrogenic cytokines.
- Gene-based interventions for collagen regulation.
- Large-scale screening programs for high-risk populations.
- Public health strategies to reduce areca nut consumption.

CONCLUSION

OSMF is a chronic, progressive, and potentially malignant condition with significant morbidity and public health impact. Areca nut chewing remains the primary etiological factor, and cessation is the cornerstone of management. Clinical features range from burning sensation to severe trismus, impairing nutrition and quality of life. While medical and surgical modalities offer symptomatic relief, none provide a definitive cure. Advances in molecular biology and regenerative medicine hold promise for future management. Regular surveillance is essential to detect malignant transformation early. A multipronged approach integrating prevention,

early diagnosis, conventional management, and novel therapies is necessary to combat this debilitating disease.

REFERENCES

1. Warnakulasuriya S. Oral potentially malignant disorders: A comprehensive review. Oral Oncol. 2020;102:104550.
2. Arakeri G, Brennan PA. Oral submucous fibrosis: An overview of the aetiology, pathogenesis, classification, and principles of management. Br J Oral Maxillofac Surg. 2013;51(7):587–93.
3. Tilakaratne WM, et al. Oral submucous fibrosis: review on aetiology and pathogenesis. Oral Oncol. 2006;42(6):561–8.
4. Gupta MK, et al. Malignant transformation rate of oral submucous fibrosis: A systematic review. J Oral Pathol Med. 2019;48(10):876–82.
5. Jeng JH, et al. Roles of areca nut in OSMF. Oral Dis. 2001;7(6):360–4.
6. Hazarey VK, et al. Areca nut and its role in OSMF. J Oral Maxillofac Pathol. 2007;11(1):1–4.
7. Trivedy C, et al. Copper content in areca nut products and its role in OSMF. Lancet. 1997;349(9063):1447.
8. Rajendran R. Oral submucous fibrosis: Etiology, pathogenesis, and future research. Bull WHO. 1994;72(6):985–96.
9. Murti PR, et al. Nutritional deficiencies in OSMF patients. Int J Oral Maxillofac Surg. 1995;24(3):191–5.
10. Haque MF, et al. Cytokine expression in OSMF. J Oral Pathol Med. 2000;29(4):179–85.
11. Cox SC, Walker DM. Oral submucous fibrosis: A review. Aust Dent J. 1996;41(5):294–9.
12. Singh M, et al. Efficacy of intralesional steroid and hyaluronidase in OSMF. J Oral Maxillofac Surg. 2017;75(6):1221.e1–7.
13. Singh N, et al. Lycopene in the management of oral submucous fibrosis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2011;111(5):575–80.
14. Borle RM, et al. Surgical management of OSMF. Br J Oral Maxillofac Surg. 1991;29(4):264–7.
15. Patil S, et al. Emerging therapies in oral submucous fibrosis. J Contemp Dent Pract. 2017;18(6):496–502.

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ORAL DYSBIOSIS: UNRAVELING THE MICROBIAL ECOLOGY OF CARIES AND PERIODONTITIS

Dr. Neeru Garg, Dr.Anoop Kumar, Dr.Suresh Kumar Goyal, Dr. Shashi Kumar Sharma

ABSTRACT

Dental caries and periodontitis remain the most prevalent chronic inflammatory diseases of humankind, traditionally attributed to a handful of "specific" pathogens. Contemporary evidence reframes both conditions as ecological disorders—dysbioses—in which changes to the host environment (dietary sugars, saliva, pH, inflammation, smoking, systemic disease) disrupt the homeostatic oral microbiome and select for disease-associated communities. In caries, frequent sugar exposures and prolonged acidogenic/aciduric selection pressure shift dental plaque toward fermenters that thrive at low pH. In periodontitis, inflammatory exudates, disrupted oxygen tension, and tissue breakdown products favor proteolytic, asaccharolytic consortia that drive a self-sustaining cycle of dysbiosis and host tissue destruction. Keystone species and accessory pathogens modulate the community and host responses, while the ecological plaque hypothesis integrates how environmental change precedes microbial shifts and disease expression. This review synthesizes current concepts on biofilm ecology, host–microbe crosstalk, and the shared and distinct dysbiotic trajectories of caries and periodontitis. It highlights diagnostic advances (multi-omics, ecological indices) and ecology-informed interventions (behavioral sugar control, fluoride and alkali strategies, mechanical biofilm disruption, host modulation, pre/probiotics, narrow-spectrum antimicrobials, and photodynamic therapy). Understanding caries and periodontitis as dysbioses enables precision prevention and minimally invasive care that target the drivers of ecological imbalance rather than single pathogens.

KEYWORDS

dysbiosis; oral microbiome; ecological plaque hypothesis; dental caries; periodontitis; biofilm; keystone pathogen; aciduric selection; host modulation; precision dentistry

INTRODUCTION

For much of the twentieth century, models of dental disease emphasized specific pathogenic species—Streptococcus mutans for caries and the "red complex" for periodontitis. Culture-independent sequencing transformed this view, revealing that health and disease correspond to community configurations shaped by ecological forces rather than the mere presence of one organism. The ecological plaque hypothesis proposed that environmental shifts (e.g., frequent sugar intake, decreased salivary flow, gingival inflammation) select for communities with traits that express disease, reconciling "specific" and "non-specific" theories into a dynamic ecological model.[1–3] In parallel, the keystone-pathogen framework explains how low-abundance organisms such as Porphyromonas gingivalis can orchestrate community-wide changes and dysregulated host responses that culminate in destructive periodontitis.[4,5]

Viewing caries and periodontitis as distinct but related dysbioses has practical consequences: clinicians must assess and modify ecological drivers, not only reduce bioburden. This review contrasts the ecological trajectories of caries and periodontitis, maps shared risk factors, and translates dysbiosis science into diagnostic and therapeutic strategies.

BIOFILM ECOLOGY AND THE HEALTHY ORAL MICROBIOME

The oral cavity hosts structured, multispecies biofilms on teeth and mucosa. Health is characterized by a relatively diverse, functionally redundant community dominated by commensal streptococci (S. mitis, S. sanguinis), Actinomyces, Rothia, Veillonella, and Neisseria, stabilized by saliva, host defenses, and frequent mechanical disturbance.[6,7] Biofilm architecture provides metabolic cooperation (cross-feeding of lactate, nitrite reduction, oxygen scavenging), stress tolerance, and a matrix that modulates diffusion and pH buffering.[8]

Homeostasis depends on environmental constraints: neutral resting pH, adequate saliva (bicarbonate, urea, arginine), limited fermentable substrates between meals, and intact host surveillance. Perturbations shift selective pressures, favoring traits (acidogenicity/aciduricity in caries; proteolysis/inflammation resistance in periodontitis) that restructure the community toward disease.[2,3]

CARIES AS A COMMUNITY-LEVEL ACIDURIC DYSBIOSIS

Environmental Driver: Sugar–Acid Stress

Frequent intake of fermentable carbohydrates creates prolonged, repeated drops in plaque pH below the critical threshold for enamel (≈5.5). Under chronic acid stress, acidogenic and aciduric taxa gain a competitive edge—S. mutans, S. sobrinus, Scardovia wiggsiae, lactobacilli—while health-associated streptococci are disadvantaged.[2,9,10] Veillonella recycle lactate to weaker acids, amplifying carbohydrate flux. Biofilm EPS (extracellular polysaccharides) from glucosyltransferases increase diffusion barriers, maintain low pH microenvironments, and enhance adhesion and cohesion, deepening the dysbiosis.[11]

Saliva and Alkali Metabolism

Saliva buffers acids and provides calcium/phosphate for remineralization. Its alkali pathways (urease, arginine deiminase system) mitigate pH drops; individuals with higher plaque arginine metabolism show caries resistance.[12] Hyposalivation (medications, radiation, autoimmune disease) removes a key ecological brake, accelerating aciduric selection.

Early Childhood Caries & Social Determinants

In early childhood caries (ECC), frequent sugar exposures (including nocturnal bottle feeding), enamel hypomineralization, and immature oral hygiene promote rapid dysbiosis. Social determinants (food insecurity, limited access to fluoride) act upstream by shaping exposure patterns that select for aciduric biofilms.[13]

Caries as a Continuum

Caries lesions represent a continuum of de- and remineralization governed by biofilm ecology and host factors; diagnostic systems emphasize activity assessment (texture, plaque stagnation) rather than only cavitation.[14] Accordingly, the most effective interventions alter the ecology—reducing fermentable substrate frequency, increasing alkali and fluoride availability, and disrupting biofilm—rather than eradicating "a single bug."[2]

PERIODONTITIS AS AN INFLAMMATION-DRIVEN PROTEOLYTIC DYSBIOSIS

Environmental Driver: Inflammation and Tissue Breakdown

In gingivitis, biofilms at the gingival margin trigger an inflammatory exudate rich in proteins, heme, and peptides. This niche shift favors asaccharolytic, proteolytic anaerobes (P. gingivalis, Tannerella forsythia, Treponema denticola, Fusobacterium nucleatum), which utilize host proteins as carbon/nitrogen sources.[4,5,15] The resulting community produces virulence factors (gingipains, leukotoxins, proteases) that dampen protective immunity yet fuel destructive inflammation, creating a positive feedback loop of dysbiosis and tissue breakdown.[4,16]

Keystone and Accessory Pathogens

The keystone-pathogen concept posits that low-abundance species (P. gingivalis) can disproportionately modulate complement signaling and TLR pathways, subverting host surveillance, enhancing nutrient flow to the biofilm, and promoting community restructuring.[4] Accessory pathogens (e.g., F. nucleatum) provide co-aggregation bridges and redox conditioning, facilitating assembly of late colonizers.[17]

Host Susceptibility and Ecological Context

Periodontitis expression depends on host susceptibility (genetics, smoking, diabetes), which sets the inflammatory tone. Hyperglycemia, for example, increases gingival crevicular fluid (GCF) glucose and advanced glycation end-products, amplifying inflammatory selection for proteolytic consortia.[16] Updated staging and grading systems reflect this multifactorial ecology and risk profile across the life course.[18]

SHARED THREADS AND DIVERGENT TRAJECTORIES

Although both are dysbioses, caries and periodontitis diverge in resource base and selection pressures: caries communities are sugar-fermenting and acid-tolerant; periodontitis communities are protein-fermenting and inflammation-adapted. Still, common upstream drivers (smoking, xerostomia, diet, hygiene behaviors) and biofilm resilience mechanisms (matrix, quorum signaling, stress responses) are shared.[2,8,16] Importantly, the same mouth may harbor spatially distinct ecologies—coronal plaque trending aciduric while subgingival plaque trends proteolytic—underscoring the need for site-specific diagnosis and care.[19]

DIAGNOSTICS IN A DYSBIOSIS FRAMEWORK

From Pathogen Lists to Functional Ecology

Routine clinical exam remains foundational (bleeding on probing, pocketing, caries activity), but omics technologies now reveal functional shifts (acid production genes, protease profiles) that better reflect pathogenic potential than species counts alone.[20,21] Metatranscriptomics distinguishes active metabolic pathways; metabolomics detects organic acids, short-chain fatty acids, putrescible amines; ecological indices (plaque pH response tests, arginine/urease capacity) estimate resilience vs. collapse thresholds.[12,21] Salivary diagnostics and chairside tests are emerging for risk stratification, though clinical integration requires standardization.[22]

ECOLOGICALLY INFORMED PREVENTION AND THERAPY

Mechanical Biofilm Disruption

Frequent, effective mechanical disruption (toothbrushing with fluoride dentifrice, interdental cleaning) resets successional dynamics and limits matrix maturation, central to both diseases.[8,14] Professional instrumentation is essential in periodontitis to reduce subgingival biomass and restore compatible host-biofilm equilibrium.[16]

Diet and Substrate Control (Caries)

Frequency of free sugars—not merely total amount—drives acid selection; counseling targets ≤4 sugar exposures/day, avoiding sipping patterns.[13] Non-fermentable sweeteners (xylitol/erythritol) and arginine-enriched products can tilt ecology toward alkali producers.[12]

Fluoride and Alkali Strategies

Fluoride lowers enamel solubility and inhibits bacterial enolase, reducing acidogenicity; high-risk patients need higher fluoride availability (varnish, high-fluoride toothpaste).[14] Sodium bicarbonate rinses or arginine technology elevate plaque pH and enhance remineralization, counter-selecting aciduric taxa.[12]

Chemotherapeutics: Narrow-Spectrum and Time-Limited

Broad antiseptics (e.g., chlorhexidine) can suppress beneficial commensals; short courses may be justified for acute indications but should be weighed against ecological costs.[23] Photodynamic therapy and targeted antimicrobials (e.g., peptide-based or quorum-sensing inhibitors) aim to disrupt virulence and matrix with less collateral damage.[23,24]

Probiotics/Prebiotics and Microbiome Stewardship

Select probiotics (Lactobacillus reuteri, Streptococcus salivarius K12/M18) and prebiotic substrates may modestly improve ecological stability (reduce mutans streptococci, gingival inflammation) as adjuncts, though strain selection and dosing remain heterogeneous.[25,26]

Host Modulation (Periodontitis)

Because inflammation fuels the periodontal niche, host modulation (sub-antimicrobial-dose doxycycline as MMP inhibitor, omega-3 FAs, resolvins; rigorous glycemic and smoking control) reduces the ecological advantage of proteolytic communities.[16,27]

Precision Periodontal Therapy

Contemporary guidance stresses personalized care: staging by severity/complexity and grading by risk (smoking, diabetes), with maintenance intervals set by ecological risk rather than fixed calendars.[18,28] Adjunctive local/systemic antibiotics are reserved for specific phenotypes and timed to mechanical therapy to minimize resistance and dysbiosis rebound.[16]

Minimally Invasive Caries Care

For active lesions, non-operative measures (fluoride varnish, SDF, resin infiltration) combined with behavior change can arrest/remineralize early disease; when operative care is needed, selective caries removal preserves biofilm-permeable dentine and avoids pulp exposure, while post-operative ecology management prevents relapse.[14,29]

LIFE-COURSE AND SYSTEMIC CONTEXT

Saliva is a master regulator of oral ecology; conditions reducing flow (polypharmacy in older adults, Sjögren's, head-and-neck radiotherapy) demand intensified buffering and remineralization strategies.[22,30] Smoking and hyperglycemia alter vascular and immune tone, oxygen gradients, and nutrient supply, shifting periodontal ecology toward dysbiosis and impairing healing.[16,28] Pregnancy, obesity, and psychosocial stress also modulate inflammatory set-points. Integrating medical history into dental ecological risk is essential.

FUTURE DIRECTIONS

1. **Function-first diagnostics**: chairside assessment of plaque pH kinetics, alkali capacity, and protease activity to capture ecological risk.[21]
2. **Ecological therapeutics**: targeted virulence blockers, EPS-matrix disruptors, and engineered probiotics that occupy disease niches.[24–26]
3. **Personalized maintenance**: microbiome-guided intervals and home-care regimens aligned to a patient's ecological resilience.[18]
4. **Implementation science**: translating dysbiosis concepts into scalable public-health programs (sugar policy, fluoridation, tobacco cessation).[13,2Λ]

CONCLUSION

Caries and periodontitis are ecological diseases: when environmental constraints are relaxed—by frequent sugar pulses and salivary dysfunction in caries, or by unchecked inflammation and tissue breakdown in periodontitis—oral communities reorganize into dysbiotic consortia that express disease. Treating these conditions as infections of single agents underestimates their community logic and the host context that selects them. The dysbiosis lens directs clinicians to measure and modify drivers—diet, saliva and pH dynamics, biofilm structure, and inflammatory set-points—while deploying conservative, targeted interventions that restore compatible host–microbe relationships. This shift enables precision prevention, minimally invasive care, and sustainable health across the life course.

REFERENCES (VANCOUVER STYLE)

1. Marsh PD. Microbial ecology of dental plaque and its significance in health and disease. Adv Dent Res. 1994;8(2):263-71.
2. Marsh PD. Are dental diseases examples of ecological catastrophes? Microbiology. 2003;149(Pt 2):279-94.
3. Takahashi N, Nyvad B. The role of bacteria in the caries process: ecological perspectives. J Dent Res. 2011;90(3):294-303.
4. Hajishengallis G, Darveau RP, Curtis MA. The keystone-pathogen hypothesis. Nat Rev Microbiol. 2012;10(10):717-25.
5. Lamont RJ, Koo H, Hajishengallis G. The oral microbiota: dynamic communities and host interactions. Nat Rev Microbiol. 2018;16(12):745-59.
6. Kilian M, Chapple ILC, Hannig M, Marsh PD, Meuric V, Pedersen AML, et al. The oral microbiome—an update for oral healthcare professionals. Br Dent J. 2016;221(10):657-66.
7. Aas JA, Paster BJ, Stokes LN, Olsen I, Dewhirst FE. Defining the normal bacterial flora of the oral cavity. J Clin Microbiol. 2005;43(11):5721-32.
8. Flemming HC, Wingender J, Szewzyk U, Steinberg P, Rice SA, Kjelleberg S. Biofilms: an emergent form of bacterial life. Nat Rev Microbiol. 2016;14(9):563-75.

9. Bowen WH, Burne RA, Wu H, Koo H. Oral biofilms: pathogens, matrix, and polymicrobial interactions in microenvironments. Trends Microbiol. 2018;26(3):229-42.
10. Pitts NB, Zero DT, Marsh PD, Ekstrand K, Weintraub JA, Ramos-Gomez F, et al. Dental caries. Nat Rev Dis Primers. 2017;3:17030.
11. Koo H, Falsetta ML, Klein MI. The exopolysaccharide matrix: a virulence determinant of cariogenic biofilm. J Dent Res. 2013;92(12):1065-73.
12. Nascimento MM, Gordan VV, Garvan CW, Browngardt CM, Burne RA. Correlations of oral arginine metabolism with caries experience. J Dent Res. 2009;88(4):367-71.
13. Sheiham A, James WP. A new understanding of the relationship between sugars, dental caries and fluoride use. Adv Nutr. 2014;5(6):716-27.
14. Innes NPT, Frencken JE, Bjørndal L, Maltz M, Manton DJ, Ricketts D, et al. Managing carious lesions: consensus recommendations on terminology and clinical management. J Dent Res. 2016;95(7):629-36.
15. Socransky SS, Haffajee AD, Cugini MA, Smith C, Kent RL Jr. Microbial complexes in subgingival plaque. J Clin Periodontol. 1998;25(2):134-44.
16. Chapple ILC, Genco R; Working group 2 of the joint EFP/AAP workshop. Diabetes and periodontal diseases: consensus report. J Periodontol. 2013;84(4 Suppl):S106-12.
17. Kolenbrander PE, Palmer RJ Jr, Periasamy S, Jakubovics NS. Oral multispecies biofilm development and the key role of cell–cell distance. Nat Rev Microbiol. 2010;8(7):471-80.
18. Tonetti MS, Greenwell H, Kornman KS. Staging and grading of periodontitis: framework and proposal. J Periodontol. 2018;89(Suppl 1):S159-72.
19. Belibasakis GN, Bostanci N, Marsh PD, Zaura E. Applications of the oral microbiome in personalized dentistry. J Dent Res. 2019;98(13):148-57.
20. Nyvad B, Takahashi N. Integrated hypothesis of dental caries. Caries Res. 2020;54(1):1-13.
21. Wade WG. The oral microbiome in health and disease. Pharmacol Res. 2013;69(1):137-43.
22. Dawes C, Pedersen AM, Villa A, Ekström J, Proctor GB, Vissink A, et al. The functions of human saliva: a review. Arch Oral Biol. 2015;60(6):863-74.
23. Sanz M, Beighton D, Curtis MA, Cury JA, Dige I, Dommisch H, et al. Role of microbial biofilms in the maintenance of oral health and in the development of dental caries and periodontal diseases. Consensus report of group 1 of the EFP/AAP workshop. J Clin Periodontol. 2017;44(Suppl 18):S5-S11.
24. Cieplik F, Tabenski L, Rupp CM, Karygianni L, Hiller KA, Buchalla W, et al. Antimicrobial photodynamic therapy for dental biofilm infections. Dent Mater. 2014;30(8):851-60.
25. Teughels W, Loozen G, Quirynen M. Do probiotics offer opportunities to manipulate the periodontal oral microbiota? J Clin Periodontol. 2011;38(Suppl 11):159-77.
26. Burton JP, Drummond BK, Chilcott CN, Tagg JR, Thomson WM, Hale JD, et al. Influence of the probiotic Streptococcus salivarius K12 on the oral microbiota of healthy volunteers. Eur J Clin Microbiol Infect Dis. 2006;25(11):749-57.
27. Caton JG, Armitage G, Berglundh T, Chapple ILC, Jepsen S, Kornman KS, et al. A new classification scheme for periodontal and peri-implant diseases and conditions. J Periodontol. 2018;89(Suppl 1):S1-S8.
28. West R. Tobacco smoking: health impact, prevalence, correlates and interventions. Psychol Health. 2017;32(8):1018-36.
29. Slayton RL, Urquhart O, Araujo MWB, Fontana M, Guzmán-Armstrong S, Nascimento MM, et al. Evidence-based clinical practice guideline on nonrestorative treatments for carious lesions. J Am Dent Assoc. 2018;149(10):837-49.e19.
30. Jensen SB, Pedersen AML, Vissink A, Andersen E, Brown CG, Davies AN, et al. A systematic review of salivary gland hypofunction and xerostomia induced by cancer therapies. Oral Oncol. 2010;46(10):834-43.

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SEDATION VS. GENERAL ANESTHESIA & PRACTICE ADAPTATIONS IN DENTISTRY: A COMPREHENSIVE REVIEW

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ABSTRACT

Pain and anxiety remain significant challenges in dental care, particularly among pediatric, special-needs, and medically compromised populations. While local anesthesia forms the cornerstone of intraoperative pain control, it does not address anxiety, fear, or behavioral challenges. Sedation and general anesthesia (GA) therefore serve as essential adjuncts in dentistry. Sedation encompasses a continuum of anxiolysis and analgesia, preserving consciousness and protective reflexes, whereas GA induces complete unconsciousness and loss of reflexes, requiring advanced airway management.

This review examines the pharmacological basis, clinical indications, comparative efficacy, and safety of sedation versus GA in dentistry, with a focus on pediatric and special-needs patients. Evidence suggests sedation provides faster recovery, fewer complications, and greater cost-effectiveness, making it suitable for outpatient dental care. GA, while resource-intensive, remains indispensable for complex procedures and patients unable to tolerate sedation. Practice adaptations—particularly during the COVID-19 pandemic—accelerated the adoption of sedation as a safer and more resource-efficient alternative.

Emerging approaches such as multimodal analgesia, opioid-free anesthesia, enhanced digital monitoring, and telehealth-based pre-anesthetic evaluations further expand the role of sedation in dental care. Nonetheless, GA remains vital for certain populations, highlighting the need for individualized, evidence-based anesthetic planning.

KEYWORDS

Sedation, General Anesthesia, Dentistry, Pediatric Dentistry, Conscious Sedation, Patient Safety, Special Needs, Practice Adaptations

INTRODUCTION

Fear of dental treatment is a global issue, with up to 20% of patients experiencing significant dental anxiety[1]. Although local anesthesia ensures adequate intraoperative pain control, it does not address psychological distress, patient cooperation, or invasive surgical requirements[2]. Sedation and GA have therefore become critical in expanding the scope of dental care.

Sedation reduces anxiety and promotes cooperation while maintaining consciousness and protective reflexes[3]. GA, in contrast, induces unconsciousness, requiring airway support and advanced monitoring[4]. Both approaches have unique advantages, risks, and resource implications. In dentistry, especially pediatric and special-needs contexts, the choice between sedation and GA remains clinically significant[5].

PHARMACOLOGICAL BASIS

Sedation typically employs nitrous oxide, benzodiazepines (midazolam), or intravenous agents such as propofol to achieve anxiolysis, analgesia, and amnesia while preserving spontaneous ventilation[6]. GA combines intravenous induction agents with inhalational maintenance agents such as sevoflurane, often supplemented with opioids or neuromuscular blockers[7].

Pharmacologically, the distinction lies in depth: sedation achieves partial depression of the central nervous system (CNS), whereas GA induces complete CNS suppression with loss of reflexes and responsiveness[8].

INDICATIONS FOR SEDATION IN DENTISTRY

Sedation is indicated in:

- Anxious but cooperative patients
- Minor oral surgery and restorative procedures
- Medically stable patients (ASA I–II)
- Patients with exaggerated gag reflex[9]
- Sedation is particularly useful for outpatient dental settings due to rapid recovery and reduced morbidity[10].

INDICATIONS FOR GA IN DENTISTRY

GA is required when sedation is insufficient or unsafe, including:

- Uncooperative preschool children requiring multiple restorations
- Patients with severe cognitive impairment or autism spectrum disorder
- Extensive oral and maxillofacial procedures
- Patients with severe dental phobia unresponsive to sedation[11]

GA ensures immobility and procedural completion but is resource-intensive and associated with higher morbidity[12].

COMPARATIVE SAFETY AND EFFICACY

Sedation generally demonstrates superior safety profiles compared to GA. Nitrous oxide sedation has a long record of safety when administered with proper monitoring[13]. Intravenous sedation carries risks of hypoventilation or paradoxical reactions but is still associated with fewer complications than GA[14].

GA, while safe under trained teams, carries higher risks of airway complications, postoperative nausea, and prolonged recovery[15]. Nevertheless, it guarantees treatment success in uncooperative patients where sedation may fail[16].

PEDIATRIC DENTISTRY

Children represent the largest population requiring pharmacological adjuncts to local anesthesia. Conscious sedation using nitrous oxide or midazolam is effective for minor procedures in cooperative children[17]. However, uncooperative preschoolers requiring extensive restorations often necessitate GA to ensure comprehensive treatment in a single session[18].

Systematic reviews suggest GA is associated with postoperative morbidity such as pain, drowsiness, and nausea, while sedation produces fewer adverse effects but risks incomplete treatment[19].

SPECIAL-NEEDS DENTISTRY

For patients with intellectual disabilities, autism spectrum disorder, or severe behavioral challenges, sedation is often inadequate. GA remains the standard of care in these populations[20]. However, limited access, resource demands, and increased morbidity underscore the need for tailored anesthetic approaches[1].

COVID-19 AND PRACTICE ADAPTATIONS

The COVID-19 pandemic reshaped anesthetic practices. Sedation was preferred due to reduced aerosol generation, shorter recovery, and outpatient feasibility. Many institutions expanded sedation services while reserving GA for essential cases. This adaptation highlighted sedation as a resource-efficient and safer modality in constrained healthcare environments.

INSTITUTIONAL AND TEAM DYNAMICS

GA delivery requires hospital or surgical center facilities, anesthesiologists, and recovery units[2]. In contrast, conscious sedation can be safely administered in outpatient dental clinics by trained providers, improving efficiency and access. Institutional dynamics therefore strongly influence modality choice.

COST AND ACCESSIBILITY

Sedation is more cost-effective than GA, with lower staffing requirements, reduced recovery time, and less equipment-intensive infrastructure[2]. GA costs are substantially higher due to operating theater use, anesthesiologist fees, and recovery room demands. For many health systems, cost is a decisive factor favoring sedation where feasible.

PATIENT AND CAREGIVER PERSPECTIVES

Parents and caregivers often favor GA for its ability to ensure comprehensive treatment in a single session. However, they report higher postoperative morbidity and longer recovery. Sedation is associated with quicker recovery, less distress, and lower cost, but caregivers sometimes express concern over incomplete procedures.

EMERGING TRENDS AND FUTURE DIRECTIONS

Key innovations shaping dental anesthesia include:.

- **Opioid-free anesthesia (OFA)**: Reducing opioid-related complications
- **Multimodal analgesia**: Combining sedatives with local anesthesia for superior pain management[31]
- **Digital monitoring:** Artificial intelligence-assisted depth monitoring enhances safety
- **Telehealth-based pre-anesthetic assessments**: Improves access and reduces in-hospital exposure

CHALLENGES AND LIMITATIONS

Despite progress, several challenges persist. GA access remains limited in low-resource settings. Regulatory variation in sedation practices across countries creates inconsistent safety standards. Concerns also remain regarding long-term neurocognitive effects of repeated GA in young children.

CONCLUSION

Sedation and GA serve complementary roles in dentistry. Sedation is increasingly emphasized for its lower cost, reduced morbidity, and feasibility in outpatient settings. GA, however, remains indispensable for patients with severe disabilities, extensive treatment needs, or uncooperative behavior.

The COVID-19 pandemic highlighted the adaptability of sedation, accelerating its use as a safer and more efficient alternative. With the rise of multimodal analgesia, opioid-free protocols, and AI-assisted monitoring, the role of sedation will likely expand further.

Ultimately, the optimal choice requires a personalized, evidence-based approach, balancing patient-specific needs, procedural demands, safety, and resource availability. Strengthening training, expanding access to sedation, and improving institutional collaboration remain priorities for enhancing dental anesthesia worldwide.

REFERENCES

1. Armfield JM. Int Dent J. 2010;60(2):86–94.
2. Coulthard P, et al. Br Dent J. 2017;223(6):435–440.
3. Becker DE, Rosenberg M. Anesth Prog. 2008;55(4):124–132.
4. Malamed SF. Sedation: A Guide to Patient Management. 6th ed. Elsevier; 2017.
5. Boynes SG, et al. Dent Clin North Am. 2016;60(2):295–307.
6. Eger EI, et al. Anesth Analg. 2008;107(3):843–857.
7. Becker DE. Anesth Prog. 2012;59(1):28–42.
8. AAPD. Guideline on Nitrous Oxide. Pediatr Dent. 2018;40(6):205–213.
9. Nathan JE. Int J Clin Pediatr Dent. 2017;10(4):379–387.
10. Wong FS, Fearne J. Dent Update. 2013;40(4):308–316.
11. Lee HH, et al. J Dent Anesth Pain Med. 2016;16(2):73–81.
12. Hosey MT. Int J Paediatr Dent. 2002;12(5):359–372.
13. Yildirim TT. Eur J Dent. 2020;14(1):155–161.
14. Apfelbaum JL, et al. Anesthesiology. 2012;116(3):522–538.
15. Savanheimo N, et al. Int J Paediatr Dent. 2012;22(4):258–265.
16. Wilson KE, et al. Cochrane Database Syst Rev. 2016;12:CD003877.
17. Lourenço-Matharu L, et al. Cochrane Database Syst Rev. 2012;3:CD006334.
18. El Batawi HY. Saudi Dent J. 2014;26(2):68–74.
19. Klaassen MA, et al. Eur Arch Paediatr Dent. 2009;10(1):27–32.
20. Sun LS, et al. N Engl J Med. 2016;375(7):605–615.

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NEUROPHYSIOLOGY OF TASTE AND ORAL SENSATION: IMPLICATIONS FOR DENTISTRY

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ABSTRACT

Taste and oral sensation are essential physiological processes that integrate chemosensory and somatosensory inputs to regulate nutrition, protect against harmful substances, and shape oral functions critical to survival. In dentistry, these sensations extend beyond food perception, influencing oral hygiene behaviour, prosthetic comfort, patient satisfaction, and postoperative outcomes. Taste buds, distributed across the oral cavity, detect five primary taste modalities—sweet, sour, salty, bitter, and umami—with growing evidence for fat and metallic tastes. These inputs are transmitted by cranial nerves VII, IX, and X, while oral sensation —including touch, temperature, and nociception—is mediated primarily by the trigeminal system (cranial nerve V). Central integration in the brainstem, thalamus, and cortical centres contributes to the unified perception of flavor, modulated further by olfaction.

Clinical conditions such as burning mouth syndrome, lingual nerve injury, head and neck cancer therapies, systemic disease, and drug use may alter these sensory modalities, profoundly affecting oral health and quality of life. For dentists, awareness of neurophysiological mechanisms is vital in patient care, particularly in prosthodontics, implantology, and pain management. Advances in genetics, neuroimaging, and regenerative medicine are expanding the understanding of gustatory and somatosensory systems, offering opportunities for precision diagnostics and novel therapies. This review discusses the neurophysiology of taste and oral sensation with a focus on its clinical significance in dentistry.

KEYWORDS

taste physiology, oral sensation, gustation, trigeminal system, dentistry, prosthetics, oral mucosa, sensory disorders

INTRODUCTION

Taste and oral sensation constitute vital components of human physiology, protecting individuals from ingesting harmful substances, promoting the intake of nutritious foods, and enabling the enjoyment of eating. Within dentistry, these systems are particularly relevant, as changes in taste or oral sensation affect dietary behavior, patient adaptation to dental appliances, and postoperative recovery[1]. The oral cavity is a unique sensory field where gustatory, somatosensory, and olfactory inputs converge to form the experience of flavor[2].

Taste buds distributed across the oral cavity detect chemical stimuli, while trigeminal afferents mediate touch, pain, and temperature[3]. The integration of these systems provides both protective reflexes and hedonic appreciation. This review aims to summarize the physiology of taste and oral sensation, highlight key neural pathways, and discuss their clinical implications in dentistry.

ANATOMY AND PHYSIOLOGY OF TASTE

Taste Buds and Receptor Cells

Taste buds are onion-shaped structures located within papillae of the tongue (fungiform, foliate, and circumvallate) and in the soft palate, pharynx, and epiglottis[4]. Each bud contains 50–100 taste receptor cells (TRCs) of three major types:

- Type I cells: Glial-like support cells maintaining ionic balance.
- Type II cells: Detect sweet, bitter, and umami via G-protein–coupled receptors.
- Type III cells: Presynaptic cells responsible for sour detection.

Taste receptor cells undergo continuous renewal, with an average lifespan of 10–14 days, enabling resilience against oral insults[5].

Taste Modalities

The classic modalities include:

- **Sweet**: Signals caloric sources, mediated by T1R2/T1R3 receptors[6].
- **Salty**: Detected primarily through epithelial sodium channels (ENaCs)[7].
- **Sour**: Mediated by proton-sensitive ion channels, including PKD2L1[8].
- **Bitter**: Recognized via T2R receptors, acting as a defense against toxins[9].
- **Umami**: Sensed by T1R1/T1R3 receptors, responding to glutamate and nucleotides[10].

Recent evidence supports additional modalities such as fat taste, mediated by CD36 and GPR120 receptors, and metallic taste perception, relevant to dental prosthetics[11].

NEURAL PATHWAYS OF TASTE

Taste information is transmitted by:

- **Facial nerve (CN VII)**: Anterior two-thirds of tongue via chorda tympani.
- **Glossopharyngeal nerve (CN IX)**: Posterior one-third of tongue.
- **Vagus nerve (CN X)**: Epiglottis and pharynx.

These afferents synapse in the nucleus of the solitary tract (NST) in the medulla, relay to the ventroposterior medial nucleus (VPM) of the thalamus, and project to the primary gustatory cortex in the insula and frontal operculum[12]. Integration with limbic structures provides the emotional and hedonic dimensions of taste.

ORAL SOMATO SENSATION

Trigeminal Contributions

The trigeminal nerve (CN V) mediates touch, temperature, and pain sensations in the oral cavity. Periodontal mechanoreceptors inform bite force regulation and mastication[13]. Thermo sensation is mediated by TRP channels, including TRPV1 (heat) and TRPM8 (cold)[14]. Nociceptors respond to mechanical and chemical insults, forming the physiological basis of dental pain[15].

Chemesthesis

The trigeminal system also detects chemical irritants, such as capsaicin (burning), menthol (cooling), and carbonation (tingling). These inputs integrate with gustation to form the multisensory experience of flavor[16].

INTEGRATION OF TASTE AND ORAL SENSATION

Flavor perception arises from the integration of gustatory, trigeminal, and olfactory inputs. The orbitofrontal cortex is a key site where these modalities converge[17]. For dentistry, understanding this integration is essential when designing flavored fluoride gels, mouth rinses, and preventive products that maximize patient compliance.

CLINICAL RELEVANCE IN DENTISTRY

Dental Materials and Prosthetics

Metallic taste is a common complaint in patients with amalgam restorations or metallic prostheses, often due to galvanic currents between dissimilar metals[18]. Acrylic-based prostheses alter oral tactile sensation, influencing mastication and adaptation.

Local Anaesthesia and Surgery

Inferior alveolar nerve blocks can temporarily disrupt taste and somatosensory perception. Lingual nerve injury, particularly during third molar surgery, may result in long-term taste disturbances and oral numbness[19].

Oral Pathology and Oncology

Oral cancers and their treatments (surgery, radiotherapy, chemotherapy) frequently impair taste and oral sensation[20]. Such changes reduce appetite, impair nutrition, and lower quality of life, requiring targeted supportive care from dental teams.

Systemic and Medication-Induced Changes

Drugs such as antibiotics, antihypertensives, and chemotherapeutics

FUTURE DIRECTIONS IN RESEARCH

1. **Neuroimaging**: Functional MRI studies provide insights into cortical taste coding[24].
2. **Genetics**: Polymorphisms in TAS2R genes influence bitter taste perception and caries susceptibility[25].
3. **Stem Cell Research**: Potential regeneration of taste buds following injury[26].
4. **Electronic Tongues**: Artificial sensors mimicking taste for salivary diagnostics[27].
5. **Neuroprosthetics**: Advances in prosthetic design may incorporate sensory restoration[28].

CONCLUSION

The neurophysiology of taste and oral sensation bridges fundamental physiology and clinical dentistry. Taste buds, cranial nerves, and trigeminal afferents collaborate to provide vital sensory information essential for nutrition, protection, and pleasure. In dentistry, disturbances of these systems—whether from disease, surgery, medications, or prosthetic appliances—can significantly affect patient outcomes and quality of life. Awareness of gustatory and trigeminal physiology enhances diagnostic precision and guides prosthetic design, restorative dentistry, and patient-centred care.

Emerging advances in genetics, neuroimaging, and regenerative medicine promise to refine our understanding of oral sensory physiology. Future applications may include precision therapies for sensory disorders, improved prosthetic materials, and diagnostic tools leveraging salivary biomarkers and electronic sensing. Thus, a deep appreciation of the neurophysiology of taste and oral sensation is essential for modern dental practice and interdisciplinary patient care.

REFERENCES (VANCOUVER STYLE)

1. Smith DV, Margolskee RF. Making sense of taste. Annu Rev Physiol. 2001;63:805-29.
2. Prescott J. Multisensory processes in flavour perception and their influence on food choice. Chem Senses. 2012;37(9):829-41.
3. Chandrashekar J, Hoon MA, Ryba NJ, Zuker CS. The receptors and cells for mammalian taste. Nature. 2006;444(7117):288-94.
4. Lindemann B. Taste reception. Physiol Rev. 1996;76(3):718-66.
5. Beites CL, Kawauchi S, Crocker CE, Calof AL. Identification and molecular regulation of neural stem cells in the olfactory epithelium. Science. 2005;311(5762):1779-83.
6. Nelson G, Hoon MA, Chandrashekar J, Zhang Y, Ryba NJ, Zuker CS. Mammalian sweet taste receptors. Nature. 2001;416(6877):199-202.
7. Chandrashekar J, Kuhn C, Oka Y, Yarmolinsky DA, Hummler E, Ryba NJ, et al. The cells and peripheral representation of sodium taste in mice. Nature. 2010;464(7286):297-301.
8. Huang AL, Chen X, Hoon MA, Chandrashekar J, Guo W, Tränkner D, et al. The cells and logic for sour taste detection. Nature. 2006;442(7105):934-8.
9. Meyerhof W, Batram C, Kuhn C, Brockhoff A, Chudoba E, Bufe B, et al. The molecular receptive ranges of human TAS2R bitter taste receptors. Chem Senses. 2010;35(2):157-70.
10. Chaudhari N, Roper SD. The cell biology of taste. J Cell Biol. 2010;190(3):285-96.
11. Running CA, Craig BA, Mattes RD. Oleogustus: The unique taste of fat. Chem Senses. 2015;40(7):507-16.
12. Rolls ET. Taste, olfactory and food texture processing in the brain and the control of food intake. Physiol Behav. 2015;152:431-43.
13. Trulsson M. Sensory-motor function of human periodontal mechanoreceptors. J Oral Rehabil. 2006;33(4):262-73.
14. McKemy DD, Neuhausser WM, Julius D. Identification of a cold receptor reveals a general role for TRP channels in thermosensation. Nature. 2002;416(6876):52-8.
15. Caterina MJ, Schumacher MA, Tominaga M, Rosen TA, Levine JD, Julius D. The capsaicin receptor: A heat-activated ion channel in the pain pathway. Nature. 1997;389(6653):816-24.
16. Green BG. Chemesthesis: Pungency as a component of flavor. Trends Food Sci Technol. 1996;7(12):415-20.
17. Small DM. Flavor is in the brain. Physiol Behav. 2012;107(4):540-52.
18. Marek M. Metallic taste perception in patients with prosthodontic appliances. J Prosthet Dent. 2011;105(2):82-8.
19. Renton T. Oral surgery: Lingual nerve injury. Br Dent J. 2010;209(7):345-50.
20. Epstein JB, Barasch A. Taste disorders in cancer patients: Pathogenesis, and approach to assessment and management. Support Care Cancer. 2010;18(8):1081-7.
21. Schiffman SS. Influence of medications on taste and smell. World J Otorhinolaryngol. 2018;4(1):84-91.
22. Vennemann MM, Hummel T, Berger K. The association between smoking and smell and taste impairment in the general population. PLoS One. 2008;3(4):e2264.
23. Zakrzewska JM. Burning mouth syndrome. BMJ. 2013;347:f5472.
24. Veldhuizen MG, Babbs RK, Patel B, Fobbs W, Kroemer NB, Garcia E, et al. Integration of sweet taste and metabolism determines carbohydrate reward. Hum Brain Mapp. 2017;38(9):3553-66.
25. Mennella JA, Pepino MY, Reed DR. Genetic and environmental determinants of bitter perception and sweet preferences. Pediatrics. 2005;115(2):e216-22.
26. Behrens M, Meyerhof W. Bitter taste receptors and human bitter taste perception. Cell Mol Life Sci. 2006;63(13):1501-9.
27. Citterio D, Suzuki K. Recent advances in taste sensors and cell-based assays for taste evaluation. TrAC Trends Anal Chem. 2019;115:248-56.
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GLUCOSE-6-PHOSPHATEDEHYDROGENASE (G6PD) DEFICIENCY AND ITS IMPLICATIONS IN DENTISTRY: AN EXPANDED REVIEW

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ABSTRACT

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most prevalent X-linked enzymopathy, affecting over 400 million individuals globally. The enzymatic defect impairs the production of nicotinamide adenine dinucleotide phosphate (NADPH), compromising red blood cell (RBC) antioxidant defenses and predisposing patients to oxidative stress–induced hemolysis. While extensively studied in hematology, G6PD deficiency poses unique challenges in dentistry due to the frequent use of local anesthetics, analgesics, antibiotics, and sedatives, many of which may trigger hemolytic crises.

This review summarizes the biochemical basis of G6PD deficiency, its epidemiology and clinical spectrum, and its diagnostic considerations. It then emphasizes its implications for dental practice, highlighting safe and unsafe drugs, anesthetic considerations, perioperative management, and preventive strategies in general dentistry, pediatric dentistry, prosthodontics, and oral surgery. Public health aspects such as population screening and pharmacogenomics are also discussed. A deeper understanding of G6PD deficiency in dentistry enhances patient safety and promotes interprofessional collaboration.

KEYWORDS

G6PD deficiency, dentistry, hemolysis, oxidative stress, anesthetics, analgesics, antibiotics, dental management, pharmacogenomics

INTRODUCTION

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is a genetic disorder of considerable global significance, representing the most common inherited enzymopathy. The biochemical defect affects the pentose phosphate pathway, rendering red blood cells (RBCs) vulnerable to oxidative stress[1]. While its hematological implications are well established, dentistry often remains under-discussed in this context.

Dentists routinely prescribe drugs, manage infections, and perform invasive procedures, all of which may inadvertently trigger hemolysis in G6PD-deficient patients[2]. Furthermore, infections in the oral cavity, if left untreated, themselves constitute oxidative stressors that can precipitate a hemolytic episode[3].

Thus, for dentists practicing in endemic regions or treating multicultural populations, understanding G6PD deficiency is essential. This review provides a comprehensive account of the disorder, with special emphasis on its dental implications, management strategies, and future directions.

BIOCHEMICAL BASIS OF G6PD DEFICIENCY

Pentose Phosphate Pathway

G6PD catalyzes the first step in the pentose phosphate pathway (PPP), converting glucose-6-phosphate to 6-phosphoglucono-δ-lactone and producing NADPH[4].

- NADPH in RBCs: Maintains reduced glutathione (GSH), which neutralizes reactive oxygen species (ROS).
- Deficiency outcome: Without NADPH, hydrogen peroxide accumulates, leading to hemoglobin oxidation, Heinz body formation, and hemolysis[5].

Why Dentistry Matters

- Many dental drugs (sulfonamides, nitrofurantoin, prilocaine) increase oxidative stress.

Hemolysis can develop within hours after dental drug administration, making knowledge of safe prescribing vital[6].

EPIDEMIOLOGY

- Global prevalence: ~4.9% of the world's population[7].
- Geographic distribution: Highest in Africa, the Middle East, Southeast Asia, and Mediterranean[8].
- Gender: Males predominantly affected due to X-linked inheritance; symptomatic females may arise from homozygosity or lyonization[9].
- Dental relevance: In multicultural practices (e.g., Europe, North America), dentists must consider ethnic prevalence in patient risk assessment.

CLINICAL MANIFESTATIONS RELEVANT TO DENTISTRY

1. **Neonatal Hyperbilirubinemia**: Past history in pediatric dental patients may indicate susceptibility to hemolysis[10].
2. **Acute Hemolytic Anemia (AHA)**: Triggered by drugs or infections, with symptoms such as jaundice, pallor, dark urine, tachycardia, and fatigue[11].
3. **Chronic Nonspherocytic Hemolytic Anemia (CNSHA)**: Rare but relevant in oral surgery patients requiring transfusion[12].
4. **Favism**: Hemolysis following ingestion of fava beans; important for dietary history in pediatric patients[13].

DIAGNOSIS

Clinical History

- Ask about previous hemolytic crises, neonatal jaundice, transfusions, or family history[14].

Laboratory Tests

- Screening: Fluorescent spot test[15].
- Confirmatory: Quantitative G6PD enzyme assay[16].
- Molecular testing: Identifies variant type; relevant for pharmacogenomics[17].

Dentists typically rely on medical history and physician reports, as on-site testing is not routine in dental practice.

DENTAL IMPLICATIONS OF G6PD DEFICIENCY

1. Local Anesthetics

- Safe options: Lidocaine, mepivacaine, bupivacaine[18].
- Unsafe:
  - Prilocaine and benzocaine → cause methemoglobinemia, poorly tolerated in G6PD deficiency[19].
  - Articaine is controversial; caution advised.

2. Analgesics

- Safe: Paracetamol (acetaminophen), ibuprofen (low doses)[20].
- Unsafe: Aspirin (especially in children) and high-dose NSAIDs → oxidative stress and hemolysis[21].

3. Antibiotics

- Safe: Penicillins, cephalosporins, macrolides (erythromycin, azithromycin)[22].
- Unsafe: Sulfonamides, nitrofurantoin, quinolones, dapsone[23].

4. Sedation in Dentistry

- Nitrous oxide is safe in G6PD deficiency.
- Midazolam and diazepam appear safe but should be used cautiously in medically compromised patients[24].

5. Dental Surgery

- Oral surgical procedures may trigger oxidative stress due to pain, hypoxia, and infection risk.
- Proper perioperative monitoring and stress-reduction protocols are essential[25].

SPECIALTY-SPECIFIC CONSIDERATIONS

Pediatric Dentistry

- Pediatric dentists often encounter undiagnosed G6PD-deficient children.
- Prescribing contraindicated antibiotics (sulfa drugs) for dental abscesses may trigger hemolysis[26].
- Safe pain management with paracetamol and lidocaine is recommended.

Oral and Maxillofacial Surgery

- Hemolytic crises may complicate major surgical procedures.
- Preoperative consultation with a hematologist is mandatory in known G6PD-deficient patients[27].
- Blood products should be available in case of severe hemolysis.

Prosthodontics and Periodontics

- Infection prevention in prosthetic and periodontal therapy reduces the risk of oxidative stress.
- Chlorhexidine is safe for mouth rinses.

Endodontics

- Root canal infections may provoke hemolysis through systemic inflammatory response[28].
- Drug choice during infection control must exclude sulfa antibiotics.

PUBLIC HEALTH AND SCREENING IN DENTISTRY

- In endemic regions, dental practitioners should routinely ask about G6PD status during medical history taking.
- Newborn screening programs in some countries (e.g., Middle East, Southeast Asia) have improved awareness.
- Pharmacogenomic screening is a future direction for dentistry and medicine integration.

PHARMACOGENOMIC PERSPECTIVES

- Certain genetic variants have milder or severe phenotypes.
- Dentists prescribing oxidant drugs in malaria-endemic areas must be aware of primaquine contraindications.
- Integration of chairside genetic testing may become part of future dental care.

EMERGENCY MANAGEMENT IN DENTAL SETTINGS

If hemolysis occurs during or after dental treatment:

1. Stop suspected drug immediately.
2. Ensure hydration and oxygen support.
3. Monitor urine color (dark urine suggests hemolysis).
4. Urgently refer to hospital for hematology support and possible transfusion.

FUTURE DIRECTIONS

- **Pharmacogenomics in dentistry**: Rapid chairside tests to identify high-risk patients.
- **Safe drug development**: New anesthetics and analgesics designed for at-risk populations.
- **Dental education**: Incorporating G6PD deficiency into dental curriculum and continuing education.
- **Gene therapy**: Future promise for permanent correction of severe variants.

CONCLUSION

G6PD deficiency, though primarily a hematological disorder, has profound implications in dentistry. Dentists must carefully consider the safety of drugs such as local anesthetics, antibiotics, and analgesics, as inappropriate prescribing may trigger life-threatening hemolysis. Preventing infections, minimizing surgical stress, and adopting safe anesthetic protocols are essential for managing dental patients with G6PD deficiency.

Interprofessional collaboration with physicians and hematologists enhances patient outcomes, while emerging fields like pharmacogenomics promise safer, more individualized dental care in the future. Ultimately, integrating biochemical understanding with clinical practice ensures that G6PD-deficient patients receive safe and effective dental treatment.

REFERENCES

1. Cappellini MD, Fiorelli G. Lancet. 2008;371:64-74.
2. Luzzatto L, et al. Hematol Oncol Clin North Am. 2016;30:373-393.
3. Stanton RC. IUBMB Life. 2012;64:362-369.
4. Au SW, et al. Blood. 2000;95:1-8.
5. Arese P, et al. Trends Mol Med. 2012;18:344-354.
6. Beutler E. Blood. 1994;84:3613-3636.
7. Howes RE, et al. PLoS Med. 2012;9:e1001339.
8. WHO Working Group. Bull World Health Organ. 1989;67:601-611.
9. Frank JE. Am Fam Physician. 2005;72:1277-1282.
10. Kaplan M, Hammerman C. Clin Perinatol. 2004;31:601-619.
11. Peters AL, Van Noorden CJ. Blood Rev. 2009;23:1-13.
12. Malamed SF. Handbook of Local Anesthesia. 7th ed. 2020.
13. Aulakh R, Sohi I. J Clin Pediatr Dent. 2008;32:189-192.
14. Glader B. Williams Hematology. 2016.
15. Youngster I, et al. Drug Saf. 2010;33:713-726.
16. Minucci A, et al. Blood Rev. 2012;26:41-47.
17. Dern RJ, et al. J Lab Clin Med. 1954;44:171-176.
18. Patton LL, et al. Oral Surg Oral Med Oral Pathol Oral Radiol. 2008;105:454-465.
19. Little JW, Miller CS, et al. Dental Management of the Medically Compromised Patient. 9th ed. 2017.
20. Greenwood M, Meechan JG. Dental Update. 2012;39:439-445.
21. Minucci A, Giardina B, Zuppi C. Crit Rev Clin Lab Sci. 2009;46:136-158.
22. Pippi R. Dent J (Basel). 2017;5(4):1-15.
23. Luzzatto L. Haematologica. 2006;91:1023-1026.
24. Awan SJ, et al. Int J Clin Pediatr Dent. 2015;8:235-238.
25. Little JW. Oral Surg Oral Med Oral Pathol Oral Radiol. 2004;98:207-212.
26. Chiu DT, et al. Ann Hematol. 2011;90:993-1003.
27. DeBaun MR, Frei-Jones MJ. Pediatr Blood Cancer. 2007;48:380-385.
28. Greenwood M. Dent Update. 2010;37:439-442.

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