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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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HEMANGIOMA OF THE FACE: A CASE REPORT

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

ABSTRACT

Hemangiomas are the most common benign vascular tumors, resulting from an abnormal proliferation of endothelial cells. They usually present in infancy with rapid growth followed by slow involution; however, some lesions persist into adolescence and adulthood, causing cosmetic and functional morbidity. Hemangiomas of the face are particularly significant because of their visibility, potential complications, and psychosocial impact. Here, we present the case of a 22-year-old male with a facial hemangioma involving the upper lip, nasolabial fold, and infraorbital region. The lesion was present since childhood and gradually enlarged, producing facial asymmetry and emotional distress. Clinical examination revealed a reddish, soft, compressible, non-pulsatile lesion, while contrast-enhanced MRI confirmed the diagnosis of hemangioma. The patient was treated with staged sclerotherapy and surgical excision followed by flap reconstruction, yielding a satisfactory cosmetic and functional outcome. This case emphasizes the importance of a multidisciplinary approach in the diagnosis and management of hemangiomas, integrating clinical evaluation, advanced imaging, and individualized therapeutic planning. A detailed review of the literature is also presented, covering the classification, pathophysiology, diagnostic tools, therapeutic modalities, and prognosis of hemangiomas, highlighting the need for early intervention to minimize complications and optimize aesthetic results.

KEYWORDS

Hemangioma; vascular tumor; facial lesion; sclerotherapy; surgical excision

INTRODUCTION

Hemangiomas are benign vascular tumors caused by proliferation of endothelial cells. They are distinct from vascular malformations, which result from structural anomalies of blood vessels without endothelial proliferation[1]. Hemangiomas are the most common vascular tumors of infancy, affecting nearly 5–10% of infants[2]. Most hemangiomas appear within the first weeks of life, undergo a rapid proliferative phase, and later regress spontaneously over several years[3].

Despite their benign nature, hemangiomas located in cosmetically and functionally significant areas, such as the face, oral cavity, or airway, may cause serious complications[4]. Facial hemangiomas are associated with ulceration, bleeding, visual disturbances, airway obstruction, speech impairment, and psychological distress[5]. Adult patients with persistent or untreated hemangiomas often present with deformity and a significant impact on quality of life[6].

The management of hemangiomas has evolved considerably. While most cases can be managed conservatively, problematic lesions require active intervention. The advent of propranolol as a first-line systemic therapy has revolutionized treatment in children[7]. In adults, surgical excision, sclerotherapy, embolization, and laser therapy remain the mainstay[8]. Here, we present a case of facial hemangioma in an adult male and review the literature regarding its pathogenesis, classification, diagnosis, and management.

CASE PRESENTATION

A 22-year-old male presented with a large reddish swelling on the right side of the face involving the upper lip, extending into the nasolabial fold and infraorbital region. The lesion was first noted in early childhood, and although initially small, it progressively increased in size over the years. The patient reported occasional pain and swelling after minor trauma but denied spontaneous bleeding or ulceration. His primary concern was cosmetic disfigurement and social embarrassment.

Outcome: Postoperative recovery was uneventful. The patient showed significant improvement in appearance and reported improved self-confidence and social interactions.

Fig 1 – Hemangioma on face

DISCUSSION

Hemangiomas are characterized by clonal proliferation of endothelial cells and represent a paradigm of postnatal vascular neoplasia rather than a simple malformation. Molecular studies implicate dysregulated angiogenesis with overexpression of pro-angiogenic factors such as vascular endothelial growth factor (VEGF) and basic fibroblast growth factor (bFGF), as well as altered signaling through the VEGF receptors and downstream pathways that promote endothelial cell proliferation and survival[6,10]. Boscolo and Bischoff discuss the role of vasculogenesis and progenitor endothelial cells in infantile hemangioma development, supporting a model in which circulating endothelial progenitor cells home to the lesion and expand under pro-angiogenic cues[6]. North et al. and others have shown that GLUT-1 expression is highly specific for infantile hemangiomas and helps distinguish them histologically from congenital vascular malformations and other vascular tumours, a point underscored in multiple clinicopathologic series[5,11]. Comparative reviews (Drolet et al., Léauté-Labrèze et al.) synthesize these molecular data and note that the balance between angiogenic stimulation and mechanisms of involution (apoptosis, reduced proliferation, remodeling) determines clinical behavior[1,2].

CLASSIFICATION — CONCORDANCE ACROSS STUDIES

The commonly used classification schemes (infantile vs congenital; superficial/deep/mixed; involuting patterns such as RICH/NICH) are grounded in both clinical course and histopathology[7,12–14]. Mulliken and Glowacki's endothelial-based taxonomy remains widely cited for clinical decision-making, while later epidemiologic and imaging studies (Metry, Chang) validate that morphology and depth predict complications and response to therapy[7,8,9]. For example, deep or mixed hemangiomas are more likely to require intervention and are less likely to undergo complete involution than small superficial lesions[9].

CLINICAL FEATURES — WHAT OTHER SERIES REPORT

Large facial hemangiomas, as in our patient, are repeatedly identified in the literature as high-risk for functional complications (ocular, airway, feeding) and psychosocial sequelae[3,4]. Enjolras et al. and Haggstrom et al. reported that so-called "alarming" hemangiomas (those with ulceration, bleeding, or proximity to critical structures) have a higher rate of complications and often need early referral to multidisciplinary teams[3,13]. Prospective cohorts likewise emphasize the impact of lesion location on outcomes and recommend early assessment for periorbital, perioral, and airway-adjacent lesions[9,13].

DIAGNOSIS — IMAGING AND COMPARATIVE FINDINGS

Clinical assessment remains primary, but multiple imaging modalities provide complementary information. Doppler ultrasound is widely used as the first-line imaging because it is noninvasive, bedside-available, and effective at demonstrating low-flow versus high-flow patterns and lesion extent; Dubois and colleagues document characteristic sonographic features that aid differentiation from vascular malformations[11]. MRI is considered the gold standard for mapping deep extent, involvement of muscle or bone, and for surgical planning; classic T2 hyperintensity and contrast enhancement patterns are well described in radiologic series and correlate with histologic

MANAGEMENT — EVIDENCE FROM COMPARATIVE REPORTS

Therapeutic approaches are tailored to lesion characteristics and patient age. The paradigm shift initiated by Léauté-Labrèze et al. established oral propranolol as a highly effective first-line therapy for problematic infantile hemangiomas, showing rapid softening and involution in many cases and changing management algorithms worldwide[14]. For adult or non-responsive lesions, interventional options such as sclerotherapy (bleomycin, ethanol, sodium tetradecyl sulfate) and embolization are supported by case series and comparative reviews (Muir et al., Burrows), demonstrating lesion shrinkage and reduction in intraoperative bleeding risk[15],[12].

Surgical excision remains important for residual, fibrotic, or disfiguring lesions in older patients—Buckmiller et al. emphasize that staged excision after preoperative volume reduction (sclerotherapy/embolization) optimizes cosmetic results and minimizes blood loss[10]. Laser therapy (pulsed dye) has robust evidence for superficial lesions and ulceration management, as described in dermatologic series. Multidisciplinary reports concur that combined modalities (e.g., sclerotherapy followed by surgical resection) often yield the best balance of efficacy and aesthetics for complex facial hemangiomas[3],[15].

PROGNOSIS AND PSYCHOSOCIAL CONSIDERATIONS

Large facial hemangiomas often leave residual contour deformity despite involution. Several observational studies highlight the long-term psychosocial impact of visible vascular lesions in adolescents and adults, advocating for early psychosocial support and reconstructive planning alongside medical therapy[3],[9]. Recurrence after excision is uncommon when resection is complete, but incomplete resection or residual nidus after sclerotherapy can lead to regrowth, a caveat noted across surgical series[15],[10].

HOW OUR CASE ALIGNS WITH THE LITERATURE

Large facial hemangiomas often leave residual contour deformity despite involution. Several observational studies highlight the long-term psychosocial impact of visible vascular lesions in adolescents and adults, advocating for early psychosocial support and reconstructive planning alongside medical therapy[3],[9]. Recurrence after excision is uncommon when resection is complete, but incomplete resection or residual nidus after sclerotherapy can lead to regrowth, a caveat noted across surgical series[15],[10].

MANAGEMENT

Management strategies include:

1. Observation: Small, uncomplicated infantile hemangiomas often regress spontaneously.
2. Medical therapy:
   - Propranolol has become first-line therapy in infantile hemangiomas.
   - Corticosteroids and interferon-α were used previously but are now rarely employed.
3. Interventional therapy:
   - Sclerotherapy with agents such as ethanol, bleomycin, or sodium tetradecyl sulfate induces endothelial damage and fibrosis.
   - Embolization can reduce intraoperative bleeding in large lesions.
4. Surgical excision: Indicated in persistent, symptomatic, or disfiguring lesions, particularly in adults.
5. Laser therapy: Pulsed dye laser is effective for superficial cutaneous hemangiomas.

In the present case, combined sclerotherapy and surgical excision achieved a good balance between lesion removal and aesthetic restoration.

Fig 2. Post operative view after 3 months

Outcome: Postoperative recovery was uneventful. The patient showed significant improvement in appearance and reported improved self-confidence and social interactions.

PROGNOSIS

The prognosis of hemangiomas depends on size, location, and treatment modality. Small superficial lesions often involute completely, while deep or large lesions require intervention. Early diagnosis and timely treatment improve outcomes and reduce psychosocial burden.

CONCLUSION

Facial hemangiomas, though benign, can cause significant morbidity due to cosmetic disfigurement, functional impairment, and psychosocial stress. A multidisciplinary approach is essential for optimal management. Clinical examination, supported by advanced imaging, remains the cornerstone of diagnosis. Treatment must be individualized, ranging from observation to sclerotherapy, embolization, or surgical excision. In this case, staged therapy combining sclerotherapy and surgery provided excellent functional and cosmetic results. Early recognition and intervention are critical in preventing complications and restoring quality of life.

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A Case Report

CLINICAL MANAGEMENT OF A SIEBERT CLASS III MANDIBULAR RIDGE DEFECT WITH A FIXED-REMOVABLE HYBRID PROSTHESIS- A CASE REPORT

Dr. Shalini Chauhan, Dr. Komal Shah, Dr. Nidhi Shree, Dr. Pranesh Rattan Trehan

ABSTRACT

Managing cases with multiple missing teeth and associated ridge defects presents a significant challenge in prosthodontics. Conventional fixed partial dentures may not be suitable due to long edentulous spans or insufficient ridge support, while removable partial dentures may lack adequate retention and patient comfort. The fixed-removable prosthesis, known as the Andrews Bridge System, offers a practical solution by combining the benefits of both fixed and removable components. The fixed segment consists of porcelain-fused-to-metal crowns joined by a bar cemented onto prepared abutment teeth. The removable portion comprises prosthetic teeth set in an acrylic base, retained over the bar using a metal or plastic sleeve track mechanism. This design provides enhanced aesthetics, retention, and stability with minimal palatal or lingual extension. It also allows for easy removal, maintenance, and hygiene access. Indications for this system include long-span edentulous areas, ridge defects, cleft palate cases, and patients who are intolerant to conventional removable dentures. This article presents a clinical case of a patient with multiple missing lower anterior teeth and a Seibert's Class III ridge defect, successfully managed with the Andrews Bridge System. The prosthesis restored functional efficiency, aesthetics, and patient confidence with a conservative and versatile approach.

KEYWORDS

Andrews Bridge system, Bar-Sleeve Retentive Design, Partial Denture Esthetics, Combination Prosthodontics.

INTRODUCTION

Removable partial dentures (RPDs) are commonly used to replace multiple missing teeth, restoring both function and aesthetics. However, many patients prefer fixed prostheses due to their superior function, appearance, and psychological comfort.[1]

In cases where a fixed prosthesis is not suitable—such as when multiple teeth are missing, the remaining teeth are periodontally compromised, or there are ridge defects—a combination of fixed and removable components may be the optimal solution.[2]

The fixed-removable system, known as the Andrews Bridge System, was introduced by Dr. James Andrews in 1965 to address such complex cases. This design involves a fixed porcelain-fused-to-metal (PFM) bridge anchored to abutment teeth, with a removable acrylic segment replacing the missing ridge and teeth. The removable part slides over a cast bar using a sleeve or track mechanism, allowing for improved retention, stability, and esthetic tooth arrangement with minimal extension.[3]

CASE PRESENTATION

A 37-year-old female patient reported to the Department of Prosthodontics and Crown & Bridge with the chief complaint of missing lower front teeth and dissatisfaction with her smile. She had previously used a removable partial denture replacing teeth 31, 32, 41, and 42, which she discontinued due to discomfort and poor retention. The patient expressed a strong preference for a fixed solution but declined implant therapy due to financial constraints. Clinical examination revealed a partially edentulous mandibular anterior region (31, 32, 41, and 42) with ridge resorption. A fixed prosthesis was present in the maxillary anterior region (11, 12, 21, and 22). An orthopantomograph (OPG) confirmed a Seibert Class III ridge defect in the mandibular anterior area. Teeth 33 and 43 were found to be suitable as abutments.

TREATMENT PLANNING

Considering the patient's aesthetic concerns, financial limitations, and need for hygiene-friendly prosthesis, a fixed-removable prosthesis based on the Andrews Bridge design was planned.

The treatment involved PFM retainers on 33 and 43 connected by a bar, supporting a removable acrylic pontic segment replacing the missing anterior teeth.

Intentional root canal treatment was performed on 33 and 43, followed by tooth preparation for PFM crowns. Final impressions were recorded using the double-mix single-step technique with polyvinyl siloxane (3M ESPE Express, India). Master casts were poured using Type IV dental stone (Neelkanth, India). Wax patterns for the retainers were fabricated, and a prefabricated plastic bar (Bredent, UK) was positioned parallel to the ridge, maintaining a 2–3 mm space for hygiene access.

The metal framework was cast using cobalt-chromium alloy (Wirobond C, Sweden), and a metal try-in was completed. Shade selection was done with the VITA shade guide. A wax-up of the anterior teeth was evaluated for esthetics and phonetics. The ceramic build-up of the fixed PFM component was completed and cemented using glass ionomer luting cement (3M Ketac, India).

The removable acrylic segment was fabricated using self-cure resin (DPI, India). The undercut beneath the bar was blocked out, and a clip was picked up into the denture with self-cure acrylic resin. The final prosthesis was finished, polished, and delivered after assessing esthetics, phonetics, retention, and comfort.

Post-operative instructions included guidance on insertion and removal of the prosthesis, use of an interdental brush for cleaning under the bar, and maintenance of oral hygiene. The patient was advised on the importance of regular follow-ups to ensure long-term success.

DISCUSSION

Rehabilitating multiple missing teeth in the presence of severe ridge resorption, especially in the anterior mandible with a Seibert Class III defect, poses a significant prosthodontic challenge. Traditional removable partial dentures are often used in such scenarios due to their ability to replace both teeth and lost alveolar tissue. However, these prostheses are frequently associated with reduced retention, limited stability, and poor patient comfort, leading many patients to prefer fixed options for better function and aesthetics.[4]

Fixed prostheses alone are typically contraindicated in cases with long edentulous spans or deficient ridge form due to compromised support and poor long-term prognosis. In such cases, the Andrews Bridge System serves as a practical and conservative alternative, combining the strength of fixed restorations with the adaptability of a removable segment.[5]

This system offers numerous advantages, including improved aesthetics, ease of maintenance, better hygiene access, and enhanced phonetics. The removable segment allows patients to clean the area beneath the bar efficiently, reducing plaque accumulation and soft tissue irritation. The bar and clip design ensures stability while also functioning as a stress breaker, minimizing excessive forces on the abutment teeth.[6]

Preiskel has noted additional benefits, including reduced prosthesis bulk, minimal wear of retention components, and the ease of fabricating duplicate removable sections using transfer sleeves. The Andrews Bridge System is also available in two forms: bar-supported by natural abutments or bone-anchored via implants. In this case, the use of a bar supported by natural teeth was preferred due to the patient's financial constraints and the absence of sufficient bone for implant placement.[6]

In this case, the use of the Andrews Bridge System allowed for successful prosthetic rehabilitation with excellent patient satisfaction. It provided a stable, retentive, and esthetic solution while maintaining accessibility for hygiene and minimizing soft tissue trauma. This treatment modality remains a valuable option in cases where other prosthetic approaches are limited by anatomical or financial constraints.

SUMMARY

The Andrews Bridge System proved to be an effective prosthetic solution for managing multiple missing anterior teeth associated with a ridge defect in a patient unwilling to undergo surgical intervention. By combining the advantages of both fixed and removable prostheses, it successfully restored esthetics, function, and patient comfort. This design also allowed for ease of hygiene maintenance and adaptability in future adjustments. The case highlights the importance of careful treatment planning and patient-centered care in selecting prosthetic options that meet both clinical and personal needs.

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Figure 1: Tooth Preparation 33, 43

Figure 2: Metal trial with bar attached parallel to the ridge

Figure 3: Cementation of PFM prosthesis and fixed segment of Andrew's Bridge

Figure 4: Post-operative view of Andrew's Bridge replacing 31, 32, 41, 42 with bar and sleeve attachment.

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COMPREHENSIVE MANAGEMENT OF SOFT TISSUE FACIAL TRAUMA IN A YOUNG FEMALE PATIENT: A CASE REPORT

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

ABSTRACT

Background: Facial trauma is a common emergency presentation, with both functional and psychosocial consequences. It may involve soft tissue, skeletal components, or a combination of both. Because the face is highly vascular and cosmetically significant, prompt diagnosis and meticulous management are essential to optimize outcomes.

Case Presentation: A 25-year-old female presented with a right supraorbital laceration and associated periorbital swelling after blunt trauma. The wound measured approximately 4–5 cm and was accompanied by facial abrasions. Clinical and radiological evaluations confirmed the absence of underlying fractures or intracranial injuries. Emergency management included wound cleansing, hemostasis, tetanus prophylaxis, and systemic antibiotics. Definitive treatment involved layered suturing under local anesthesia along relaxed skin tension lines (RSTL).

Figure 1(a) Before Treatment

Figure 1 (b)- After Treatment

Outcome: Wound healing was uneventful. At one-month follow-up, the patient showed satisfactory cosmetic and functional recovery without complications.

Conclusion: This case highlights the importance of systematic evaluation, immediate wound care, appropriate closure techniques, and follow-up in managing facial trauma. A multidisciplinary approach further enhances outcomes, reducing long-term scarring and psychological impact.

KEYWORDS

facial trauma; soft tissue laceration; supraorbital injury; wound closure; cosmetic outcome; case report

INTRODUCTION

Facial trauma accounts for a substantial proportion of trauma-related emergency visits, with an incidence ranging from 15% to 40% of all trauma cases worldwide[1]. The etiology varies geographically; in developing countries, road traffic accidents (RTAs) remain the leading cause, while assaults and sports injuries predominate in developed nations[2]. The 20–40 year age group is most affected, reflecting increased exposure to outdoor activities, occupational hazards, and interpersonal conflicts[3].

Facial injuries may be classified into:

1. Soft tissue trauma – abrasions, contusions, lacerations, avulsions.
2. Skeletal injuries – nasal fractures, zygomaticomaxillary complex (ZMC) fractures, mandibular fractures, orbital wall injuries.
3. Combined injuries involving soft tissue and bone[4].

Soft tissue injuries, though often overlooked compared to fractures, are of critical importance because they directly influence aesthetic outcomes. Even a small scar in the facial region may lead to psychological distress, reduced self-esteem, and social impairment[5]. Thus, precise anatomical repair following plastic surgical principles is essential.

Here, we report a case of isolated supraorbital soft tissue trauma in a young female patient. The case emphasizes emergency wound management, layered closure techniques, and postoperative scar care.

CASE PRESENTATION

Patient Profile

A 25-year-old woman presented to the emergency department of a tertiary care hospital after sustaining blunt trauma to the right side of her face following a fall.

History

- Chief complaint: Profuse bleeding from a cut over the right forehead near the eyebrow, associated with swelling and bruising.
- History of present illness: Trauma occurred 2 hours prior to admission. No history of unconsciousness, seizures, vomiting, nasal bleed, or visual disturbance.
- Past medical history: No history of bleeding disorders, anticoagulant use, diabetes, or hypertension. Immunization status up to date.
- Family/social history: Non-smoker, occasional alcohol use.

Examination

- General condition: Patient conscious, alert, oriented.
- Vitals: Blood pressure 112/74 mmHg, pulse 88/min, respiratory rate 16/min, afebrile.
- Local examination:
  - A linear laceration (~4.5 cm) extending obliquely across the right supraorbital ridge.
  - Margins irregular with active bleeding.
  - Associated periorbital edema and ecchymosis.
  - Multiple superficial abrasions on the right cheek.
  - No step deformity, crepitus, or infraorbital hypoesthesia.
- Ophthalmic evaluation: Extraocular movements intact, pupils equal and reactive, no subconjunctival hemorrhage, globe intact.
- Systemic examination: Normal.

Investigations

- Laboratory tests: CBC, coagulation profile, and blood glucose within normal limits.
- Imaging:

Non-contrast CT (NCCT) of head and face – ruled out fractures of frontal bone, orbital roof, or zygomatic complex. No intracranial hemorrhage noted[13].

MANAGEMENT

Initial Emergency Care

- Wound irrigated with 500 ml of normal saline followed by diluted povidone-iodine.
- Hemostasis achieved via direct pressure and temporary clamping of small bleeders.
- Tetanus toxoid (0.5 ml IM) administered.
- Intravenous ceftriaxone (1 g) given as prophylactic antibiotic[6].
- Intravenous diclofenac (75 mg) for analgesia.

Definitive Surgical Management

Performed under local anesthesia (2% lignocaine with adrenaline).

- Wound margins carefully debrided to remove devitalized tissue.
- Layered closure technique:
  - Subcutaneous tissues approximated using absorbable 4-0 vicryl sutures.
  - Skin closed with non-absorbable 5-0 prolene in interrupted fashion.
  - Closure aligned along RSTL for optimal scar concealment[7].
- Sterile dressing applied.

Postoperative Care

- Oral antibiotics continued for 5 days.
- Topical mupirocin ointment applied twice daily.
- Sutures removed on postoperative day 6.
- Patient educated regarding wound hygiene, scar massage, and use of sunscreen.

OUTCOME AND FOLLOW-UP

1. Short-term: Uneventful recovery with no wound infection or dehiscence.
2. At 1 month: Mild hyperpigmented scar visible, but cosmetically acceptable. No functional deficit in eyelid movement or vision.
3. Scar management: Advised silicone gel application for 8 weeks and regular massage.

DISCUSSION

Epidemiology of Facial Trauma

Multiple studies confirm that young males remain the most affected group due to occupational risks, violence, and RTAs. However, a study by Boffano et al. (2015) showed an increasing trend in female facial trauma cases, largely due to changing social roles and increased participation in outdoor activities[8]. Regional variations exist—RTAs dominate in Asia and Africa, while assault-related injuries are more frequent in Europe and North America[9],[10].

Role of Imaging

While clinical examination remains essential, CT imaging is universally considered the gold standard. Holmes et al. (2001) highlighted that plain radiographs often miss orbital, nasal, and zygomatic fractures, whereas CT provides detailed multiplanar imaging, crucial for surgical planning[13]. A recent review by Kellman & Goyal (2017) also emphasized that 3D reconstruction CT further improves accuracy and facilitates communication with patients regarding surgical needs[12].

Principles of Soft Tissue Repair

Soft tissue injuries demand precise repair. Manson & Crawley (1989) emphasized that early repair within the first 6–8 hours significantly reduces infection and improves cosmetic outcomes[1]. Borges (1984) introduced the principle of Relaxed Skin Tension Lines (RSTL), which has become the gold standard in minimizing scar visibility[7]. A more recent review by Hollier et al. (2010) added that meticulous layered closure and tension-free suturing reduce hypertrophic scar formation[11].

Scar Formation and Management

Scar outcome is influenced by wound location, patient age, skin type, and closure technique. Mustoe et al. (2002) proposed international recommendations for scar management, advocating for silicone gel, pressure therapy, and intralesional steroids in resistant scars[15]. Newer modalities such as fractional CO_2 laser therapy and platelet-rich plasma (PRP) have been reported to improve scar remodeling.

Psychosocial Impact

The psychosocial burden of facial trauma cannot be underestimated. Lee (2012) highlighted that even minor facial scars may significantly impair self-image and social interaction, particularly in young female patients[5]. A qualitative study by Strong & Sykes (1998) emphasized the importance of psychological counseling alongside surgical repair for holistic rehabilitation[16].

Multidisciplinary Approach

Complex trauma often necessitates coordinated care. The EURMAT study (2015) involving 7 European trauma centers concluded that maxillofacial surgeons, ophthalmologists, neurosurgeons, and psychologists must collaborate for optimal results[8]. Recent advances include virtual surgical planning (VSP), which improves preoperative planning in complex fractures.

CONCLUSION

This case illustrates successful management of isolated supraorbital soft tissue trauma in a young female. The outcome was favorable due to early wound cleaning, anatomical layered closure, prophylactic antibiotics, and follow-up scar care. Facial trauma management must balance life-saving priorities with cosmetic considerations, as neglecting either may compromise long-term outcomes. A multidisciplinary, patient-centered approach ensures optimal recovery and minimizes psychological burden.

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MULTISYSTEM FACIAL TRAUMA FOLLOWING ROAD TRAFFIC ACCIDENT – A CLINICAL AND SURGICAL PERSPECTIVE

Dr.Manish Aggarwal, Dr. Mahendra Marothi , Dr. Reeta Arora, Dr. Bhorika Aggarwal

ABSTRACT

Background: Road traffic accidents (RTAs) remain a major public health concern, accounting for significant morbidity and mortality worldwide. Among the injuries sustained, facial trauma is particularly devastating due to its impact on vital functions such as vision, mastication, and airway integrity, in addition to cosmetic and psychosocial implications.

Case Presentation: We present a 32-year-old male involved in a high-speed motorcycle accident, brought unconscious to the emergency department with extensive facial lacerations, mandibular fracture, and cervical involvement. The patient was managed according to Advanced Trauma Life Support (ATLS) principles, with early airway protection, hemodynamic stabilization, and definitive surgical repair. Multidisciplinary care involving maxillofacial surgeons, anesthesiologists, and critical care physicians ensured functional and aesthetic recovery.

Discussion: This case emphasizes the importance of prioritizing airway management in facial trauma, supported by CT-based imaging for precise diagnosis, layered closure of soft tissues, and staged fixation of skeletal injuries. Scar management, infection prophylaxis, and psychological rehabilitation are equally crucial. Global literature highlights RTAs as the leading cause of maxillofacial injuries in developing nations, in contrast to interpersonal violence in developed countries.

Conclusion: RTAs demand a structured, multidisciplinary approach. Early stabilization, meticulous repair, and long-term psychosocial support are critical to achieving optimal outcomes.

Keywords: Road traffic accident, facial trauma, airway management, mandibular fracture, multidisciplinary approach

INTRODUCTION

Road traffic accidents (RTAs) are a leading cause of trauma-related deaths and disabilities worldwide, accounting for over 1.3 million deaths annually according to the World Health Organization (WHO)[1]. In low- and middle-income countries, the burden is disproportionately high due to inadequate road safety measures, limited enforcement of traffic laws, and lack of pre-hospital care[2].

Facial trauma following RTAs presents unique challenges due to the complex anatomy of the face, its vascularity, and its aesthetic importance. Injuries may involve soft tissues, skeletal components, or both. These can compromise the airway, vision, and craniofacial function while also leading to significant long-term psychosocial effects[3].

This case report details the evaluation and management of a young male with severe facial trauma sustained in an RTA, highlighting the importance of a multidisciplinary and staged approach.

CASE PRESENTATION

History and Pre-hospital Details

A 32-year-old male was involved in a high-speed motorcycle collision without helmet protection. He was found unconscious at the site with active bleeding from multiple facial wounds and was brought to the emergency department within 45 minutes.

Clinical Examination

On arrival:

- **General condition**: GCS 9/15, HR 120/min, BP 90/60 mmHg, SpO_2 88% (room air).
- **Airway**: Compromised due to orofacial bleeding and swelling. **Immediate** orotracheal intubation was performed.
- **Breathing**: Bilateral air entry present; ventilation initiated.
- **Circulation**: IV fluids and 2 units of PRBC transfused.
- **Neurological status:** Pupils equal and reactive.
- **Exposure**: No other systemic injuries detected.

Figure (a) showing pre-operative view

Figure (b) showing immediately after suturing

Local Findings:

- Deep lacerations involving periorbital, malar, mandibular, and cervical regions.
- Left auricular cartilage exposed with partial avulsion.
- Crepitus over left mandibular body suggesting fracture.
- Periorbital edema and subconjunctival hemorrhage, but globe intact.

Investigations:

- **Blood work**: Hb 9.8 g/dL, leukocytosis 14,200/µL.
- **CT Face**: Comminuted fracture of mandibular body and parasymphysis, Intact midfacial skeleton, no intracranial bleed.
- **Ophthalmology consult**: No ocular rupture or visual deficit.

MANAGEMENT

Emergency Phase

- **Airway**: Secured with orotracheal intubation.
- **Hemorrhage control**: Direct pressure and electrocautery.
- **Prophylaxis**: IV ceftriaxone, metronidazole, tetanus toxoid.
- **Resuscitation**: IV fluids, blood transfusion, analgesia.

Definitive Surgical Management

- Wound irrigation and debridement using saline and povidone-iodine.
- Layered closure respecting relaxed skin tension lines (RSTL).
- Auricular repair with cartilage approximation.
- **Mandibular fracture fixation**: Open reduction and internal fixation (ORIF) with titanium plates was performed once the patient was hemodynamically stable (day 10).

Postoperative Care

- ICU monitoring for 72 hours.
- Antibiotics continued for 7 days.
- Sutures removed on day 7.
- Scar management initiated with silicone gel.
- Psychological counseling for trauma-related anxiety.

OUTCOME AND FOLLOW-UP

- Patient extubated on day 5, stable thereafter.
- Primary wound healing achieved with acceptable cosmetic outcome.
- Functional occlusion restored post-ORIF.
- At 6 weeks: Minimal scarring, no visual or auditory complications.
- Patient continued scar care and physiotherapy for mandibular movement.

DISCUSSION

Epidemiology of RTA-related Trauma

Road traffic accidents (RTAs) remain the leading cause of maxillofacial trauma in developing countries, accounting for up to 60% of facial fractures[4].

In contrast, interpersonal violence and assaults dominate in Western countries, as reported by Haug et al.[5]. Several multicenter studies, including the EURMAT project, confirm that young men in their 20s and 30s remain the most vulnerable group, largely due to occupational exposure, high-risk driving, and lack of helmet/seatbelt compliance[6]. However, recent literature also indicates an increasing incidence of female trauma victims, reflecting their rising participation in the workforce and outdoor activities.

Airway and Resuscitation

The airway remains the top priority in the management of maxillofacial injuries. Bleeding, edema, and bony disruption can compromise patency. Hollier et al. emphasized that early intubation significantly reduces morbidity and mortality, while delayed airway control increases the risk of hypoxia and aspiration[7]. In more complex cases, especially with panfacial trauma, surgical airway procedures such as cricothyroidotomy or tracheostomy may be required. Studies also highlight that an airway-first approach, followed by circulation and neurological assessment, conforms to ATLS guidelines, ensuring survival.

Role of Imaging

CT imaging with 3D reconstruction is considered the gold standard in maxillofacial trauma assessment. Kellman & Goyal confirmed its superiority over plain radiographs in detecting orbital, nasal, and zygomatic fractures[8]. Furthermore, 3D CT reconstruction aids in surgical planning, enabling more precise reduction and fixation of complex fractures. Recent advances include virtual surgical planning (VSP), which has been shown to improve operative accuracy and patient outcomes by providing computer-assisted models for fracture realignment.

Soft Tissue Management

Soft tissue repair remains crucial for both function and aesthetics. The guiding principles include:
·Hemostasis to prevent hematoma and secondary infection.
·Minimal debridement to preserve viable tissue, as aggressive excision worsens cosmetic outcomes.
·Layered closure along relaxed skin tension lines (RSTL) to camouflage scars[9].
Hollier et al. further emphasized the value of early repair within 6–8 hours to reduce infection risk and improve wound healing. In cases with tissue loss, local or regional flap coverage may be necessary to restore form and contour.

Skeletal Trauma

Mandibular fractures are the most frequent post-RTA injuries due to the mandible's prominence. ORIF with titanium plates remains the gold standard, restoring stability and occlusion[10]. However, recent literature highlights the importance of timing: fixation should be performed once the patient is hemodynamically stable. In severely comminuted fractures, staged approaches or external fixation may be required. The role of resorbable fixation systems has also been explored, though titanium remains more widely accepted due to its strength and reliability.

Infection Control

Although the face has a rich vascular supply that promotes healing, infection remains a concern, especially in contaminated wounds. Cummings & Del Beccaro demonstrated that prophylactic broad-spectrum antibiotics significantly reduce wound infection risk[11]. For mandibular fractures involving intraoral communication, antibiotic prophylaxis is considered essential. Moreover, proper wound irrigation and early debridement are equally critical preventive strategies.

Scar Formation and Psychosocial Impact

Despite optimal surgical repair, scars are often inevitable. Mustoe et al. published international recommendations on scar management, emphasizing silicone gel application, scar massage, and photoprotection as first-line interventions[12]. In resistant cases, intralesional corticosteroid injections, pressure therapy, or laser resurfacing can improve scar appearance. Beyond physical healing, psychosocial consequences—including anxiety, depression, and reduced social interaction—are frequently reported. Strong & Sykes highlighted that facial scars carry a greater psychological burden than scars on other body parts, necessitating early counseling and reassurance[13].

Multidisciplinary Approach

Optimal management of facial trauma requires a multidisciplinary team approach. Emergency physicians secure airway and resuscitation, maxillofacial and plastic surgeons address skeletal and soft tissue injuries, ophthalmologists assess orbital involvement, and psychologists provide emotional rehabilitation. The EURMAT study confirmed that centers with multidisciplinary trauma teams achieve better functional and aesthetic outcomes[14]. Moreover, advances such as virtual surgical planning and 3D printing of patient-specific implants highlight the importance of integrating technology into modern multidisciplinary trauma care.

CONCLUSION

This case demonstrates that road traffic accidents remain a leading cause of severe facial trauma in developing nations. Early airway management, hemodynamic stabilization, meticulous layered closure, staged skeletal fixation, and long-term scar and psychological care are key to successful outcomes. A structured multidisciplinary approach ensures comprehensive rehabilitation for patients with devastating facial injuries.

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FUNCTIONAL AND ESTHETIC REHABILITATION OF DECAYED PRIMARY ANTERIOR TEETH USING PREFABRICATED POST AND STRIP CROWNS

Dr. Mahima Singh, Dr. Suruchi Juneja Sukhija, Dr. Thoihenbi Oinam, Dr. Rachna Kaswan

ABSTRACT

Restoring primary teeth for both function and aesthetics, especially in the anterior region, poses a significant challenge. Severely decayed primary incisors often lack sufficient structure for retaining full-coronal esthetic restorations. Various techniques, including intracanal post placement, have been explored to enhance retention in pulpectomy treated teeth. Among these, glass fiber-reinforced posts stand out due to their strength, biocompatibility, and esthetic compatibility. These posts provide internal support, improving resistance to dislodgement and fracture. Strip crowns remain a popular esthetic choice due to their natural appearance, ease of application, and affordability, making them widely used in pediatric dentistry. When combined with intracanal reinforcement, strip crowns offer a more durable and visually appealing restoration. This integrated approach not only enhances clinical outcomes but also ensures the retention of primary teeth until natural exfoliation. Maintaining these teeth supports essential functions like speech, mastication, and arch space preservation for permanent successors, thereby playing a crucial role in a child's oral development. This case report describes challenging task of a Case of early childhood caries patients with mutilated maxillary incisors restored with a strip form composite restorations. This case report presents the complex management of a patient with early childhood caries, featuring severely damaged maxillary incisors that were restored using strip-form composite restorations.

KEYWORDS

Primary teeth, dental caries, composite dental resin

INTRODUCTION

Dental caries is still one of the most prevalent chronic disorders in children, even though it is mostly avoidable. The first primary molars and upper front teeth are usually affected by early childhood caries. The pulp of the upper front teeth is frequently impacted, making them the most seriously affected. In extreme situations, the disorder may result in the complete loss of the tooth's crown.

Restoring esthetics in primary anteriors is challenging due to small size, thin enamel, pulp proximity, limited bonding area, child cooperation, and cost. Severe decay can affect speech, chewing, appearance, and may cause malocclusion. Therefore, timely restoration is essential to maintain primary teeth until exfoliation.[1,2] Treatment decisions are influenced by the child's age and behavior for effective long-term outcomes.[3]

Restorative options for severely decayed teeth include prefabricated crowns, resin composites, biological restorations, and post-and-core techniques. In advanced cases, endodontic therapy with intracanal posts—made from metal, composite, or biological materials—is needed. Fiber-reinforced posts are increasingly favored over metal posts due to their translucency, crown support, and ease of use. This case report discusses a three-year-old with severely decayed maxillary anteriors restored using glass fiber-reinforced composite resin posts (GFRC), a modern fiber post system.[4,5]

This case report presents the complex management of a patient with early childhood caries, featuring severely damaged maxillary incisors that were restored using strip crowns.

CASE REPORT

A three-year-old boy was brought to the Department of Pedodontics and Preventive Dentistry, Surendera Dental College and Research Institute, with a complaint of decayed upper front teeth for the past 4–5 months.

Clinical examination showed complete primary dentition, with no associated pain or swelling. Teeth 61 and 62 had extensive caries, while 51 and 52 were grossly decayed. Deep caries was also noted on 75. Radiographs confirmed pulp involvement in 52, 51, 61, and 62 with adequate root length. A treatment plan was made to perform pulpectomy, followed by placement of glass fiber-reinforced composite posts and composite buildups for 51 and 52, and restoration of all four incisors with strip crowns. The parents were informed about the procedure, its benefits, limitations, alternatives, and possible outcomes without treatment.

Fig 1(a) Preoperative photograph wrt 52,51,61,62

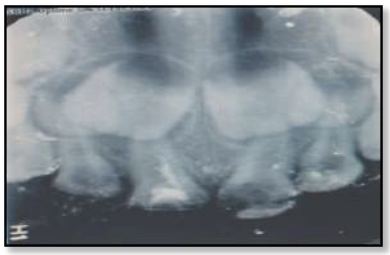

Fig 1(b) Preoperative radiograph wrt 52,51,61,62

An infraorbital nerve block with palatal infiltration was given for 51, 52, 61, and 62. Gross caries removal was performed using a No. 330 round bur, and pulp chambers were accessed. Working length was determined with a No. 10 K-file and pulp extirpation done using a No. 15 H-file. Root canals were prepared with irrigation using 2.5% NaOCl and saline, then dried with paper points. Metapex was placed using a reamer under isolation using cotton rolls and saliva ejectors [Figure 2].

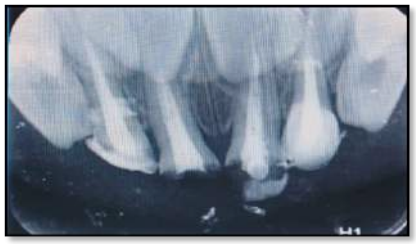

Figure 2: IOPA showing obturation done with metapex wrt 52,51,61,62

One week later, post space in 51 and 52 was prepared by removing 4 mm of Metapex using H-file and 1 mm of GIC was placed as a barrier. A prefabricated fiber post matching canal diameter was selected. The post space was cleaned, etched with 37% phosphoric acid for 15 seconds, rinsed, and dried. A bonding agent was applied and cured for 20 seconds. Flowable composite was placed into the canal, and the glass fiber post was inserted and cured for 60 seconds [Figure 3(a)]. The post was trimmed, leaving 2 mm coronally exposed. Enamel was etched, bonded, and cured. Core build-up was completed using flowable composite and light-cured. Final restoration of 51, 52, 61, and 62 was done using hybrid composite [Figure 3 (b)].

Figure 3(a): Post placement wrt 52,51

Figure 3(b): Core buildup wrt 52,51,61,62

Strip crowns were selected to match tooth size (Figure 4). The composite core was etched with 37% phosphoric acid for 15 seconds, rinsed, and gently dried. A bonding agent was applied, air-thinned, and light-cured for 10 seconds. Composite-filled strip crowns were placed with gentle pressure for proper adaptation. Excess was removed before curing. Each crown was light-cured from buccal, lingual, and incisal sides for 40 seconds. Once set, the celluloid forms were peeled off, and any excess was finished and polished with burs and discs. Occlusion was adjusted, and final polishing ensured a natural look. (Figure 5)

Figure 4: Placement of strip crowns wrt 52,51,61,62 to check appropriate size

Figure 5: Postoperative image of strip crowns wrt 52,51,61,62

DISCUSSION

In today's cosmetically aware and modern society, well-aligned and properly contoured white teeth are seen as a benchmark of beauty. They are not only perceived as aesthetically pleasing but also reflect good nutrition, self-esteem, personal hygiene, and socioeconomic standing.[6]

Strip crowns are among the most technique-sensitive restorations and can be challenging to place. However, bonded resin composite strip crowns are considered one of the most esthetic options for restoring severely decayed primary incisors. The choice and success of this treatment depend on several factors, including the clinician's preference, parental esthetic expectations, the child's behavior, and the ability to control moisture and bleeding during the procedure.[7]

The ultimate goal of restoring severely decayed anterior primary teeth is to allow the patient to retain these teeth and allow their natural exfoliation without any pulpal complications, which may ultimately result in damage to the permanent successors.[8]

This study reported 100% retention rate of strip crowns till the normal exfoliation occurred due to the use of glass fiber-reinforced posts which provides enhanced retention in severely decayed primary anterior teeth where minimal coronal structure remains, allowing successful placement of strip crowns and restoring both function and esthetics.

In a prospective clinical study with a one-year follow-up, Judd and colleagues (1990) reported a 100% retention rate for composite resin strip crowns in a sample of 92 primary teeth. Similarly, Grosso et al. (1987) and Mendes et al. (2004) described successful outcomes in a case using a short composite resin post within the pulpal chamber of anterior teeth that had undergone pulpectomy.[9,10,11]

In modern pediatric dentistry, an array of aesthetic treatment modalities has been developed to effectively address dental caries and traumatic injuries in the primary dentition, aiming not only to restore function but also to meet the growing esthetic expectations of patients and their caregivers.[12]

CONCLUSION

Retention of primary teeth remains a key challenge in pediatric dentistry. Restoring decayed incisors supports a child's overall well-being. Glass fiber-reinforced post and core systems offer

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RECENT CONCEPTS IN PROSTAGLANDINS: BIOLOGY, TOOLS, AND THERAPEUTIC FRONTIERS

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ABSTRACT

Prostaglandins (PGs) are bioactive lipid mediators derived from arachidonic acid that regulate inflammation, vascular tone, renal function, immunity, reproduction, and tissue repair. Since their discovery, PGs have been recognized as essential signaling molecules but also as culprits in pain, fever, cancer progression, and cardiovascular disease. Historically, their pharmacological manipulation relied heavily on nonsteroidal anti-inflammatory drugs (NSAIDs), which broadly inhibit cyclooxygenase (COX) enzymes. Despite their efficacy, NSAIDs are associated with gastrointestinal bleeding, renal toxicity, and cardiovascular risks, highlighting the need for more selective interventions.

Recent research has advanced beyond COX blockade into more precise strategies, including selective inhibition of microsomal prostaglandin E synthase-1 (mPGES-1), modulation of prostaglandin transporters such as SLCO2A1 (PGT), and receptor-level targeting, particularly of EP4 in cancer immunotherapy. Concurrently, the integration of lipidomics, advanced synthetic chemistry, and receptor-biased pharmacology is reshaping how we measure, interpret, and exploit prostaglandin biology. This review synthesizes the recent conceptual advances in prostaglandin research, highlighting how the field is shifting toward precision modulation with promising therapeutic implications.

KEYWORDS

Prostaglandins; Cyclooxygenase; mPGES-1; Prostaglandin transporters; EP4 receptor; Lipidomics; Targeted therapy

INTRODUCTION

Prostaglandins belong to the eicosanoid family of signalling molecules, derived from arachidonic acid through enzymatic action of cyclooxygenases (COX-1 and COX-2). From their origin, PGs undergo conversion into distinct molecules (PGE_2, PGD_2, $PGF_2\alpha$, PGI_2, and thromboxane A_2), each with specialized receptors and functions.

Physiologically, prostaglandins maintain gastric mucosal integrity, regulate renal perfusion, mediate uterine contractions during labour, and modulate platelet aggregation. Pathologically, they contribute to inflammatory cascades, tumor immune evasion, neurodegenerative disorders, and cardiovascular dysfunction. The dual nature of prostaglandins makes them both indispensable regulators of homeostasis and drivers of disease.

For decades, therapeutic intervention centered on NSAIDs that inhibit COX enzymes, thereby reducing prostaglandin synthesis. Although effective, this strategy is non-selective: protective prostanoids are suppressed alongside harmful ones, leading to adverse events. COX-2 selective inhibitors were developed to mitigate gastrointestinal risks but introduced cardiovascular complications. These limitations shifted the field toward more precise, node-specific, and receptor-targeted approaches.

1. From COX Inhibition to Node-Specific Targeting: The Case of mPGES-1

The identification of microsomal prostaglandin E synthase-1 (mPGES-1) as a downstream enzyme converting PGH_2 into PGE_2 has opened opportunities for selective intervention. Unlike COX inhibitors, mPGES-1 inhibition spares the synthesis of other prostanoids, including prostacyclin (PGI_2), which is cardioprotective.

Preclinical studies have shown that pharmacological inhibition or genetic deletion of mPGES-1 leads to significant suppression of inflammation, fever, and pain responses without the thrombotic risk associated with COX-2 inhibitors[1,2]. Furthermore, mPGES-1 inhibition may restore a favorable prostanoid balance, reducing excessive PGE_2 while maintaining or even enhancing PGI_2 levels[4].

Clinically, vipoglanstat has progressed to phase 2 trials, where it demonstrated reduction of inflammatory biomarkers with acceptable safety[3]. This represents the first major step toward translating selective PGE_2 suppression into human therapy. Importantly, unlike NSAIDs, mPGES-1 inhibitors could allow tissue- and context-specific suppression of pathological PGE_2 without systemic prostanoid depletion.

Thus, mPGES-1 represents a paradigm shift from broad COX blockade toward pathway-precise anti-inflammatory therapy, and ongoing trials will determine whether this strategy can become a safer alternative to NSAIDs

2. Transport and Homeostasis: The Role of SLCO2A1 (PGT)

The regulation of prostaglandin signalling extends beyond synthesis and degradation into the realm of transport biology. The prostaglandin transporter (PGT), encoded by SLCO2A1, controls the uptake and clearance of extracellular PGs, particularly PGE_2.

Defective SLCO2A1 function has been implicated in rare conditions such as chronic enteropathy associated with SLCO2A1 (CEAS) and pachydermoperiostosis (PHO)[5]. Recent genetic discoveries have identified novel splice variants, expanding the mutation spectrum and enabling earlier diagnosis and genetic counselling.

At a physiological level, PGT regulates prostaglandin availability in tissues such as the lung, kidney, placenta, and gastrointestinal tract[5]. In wound biology, overexpression of PGT reduces extracellular PGE_2, delaying wound closure—especially in diabetic ulcers. This suggests that PGT inhibition could serve as a therapeutic approach to promote tissue repair[6].

Environmental factors can also modulate transporter activity. Cigarette smoke exposure suppresses SLCO2A1 expression via AHR signaling, lowering prostaglandin clearance and potentially exacerbating inflammatory lung pathology[7]. These findings emphasize the dynamic interplay between genetics, environment, and transporter biology in shaping prostaglandin activity.

Therapeutically, targeting PGT opens avenues for localized enhancement or suppression of prostaglandin action, which could offer advantages over systemic COX inhibition.

3. Tumor Immunology and EP Receptor Precision

The tumor microenvironment (TME) is enriched in PGE_2, which fosters immunosuppression through engagement of EP receptors. Of the four receptors (EP1–EP4), EP4 has emerged as a critical mediator of immune evasion.

PGE_2–EP4 signaling reprograms myeloid-derived suppressor cells, reduces dendritic cell antigen presentation, and dampens cytotoxic T cell activity[8]. This has positioned EP4 as a target for immuno-oncology.

Clinical translation is underway. Vorbipiprant, a selective EP4 antagonist, is currently tested in combination with PD-1 inhibitors. Early-phase trials demonstrate safety, tolerability, and encouraging antitumor activity, supporting the concept of PGE_2 as an immune checkpoint[9]. Similarly, grapiprant (RMX1002) has completed phase I trials as another EP4-directed therapy[10].

Preclinical research is exploring next-generation antagonists with biased signaling properties. Shikonin, for instance, acts as a dual G-protein/β-arrestin modulator of EP4, offering potential to fine-tune downstream pathways while minimizing off-target effects[11].

Beyond oncology, EP4 modulation may have applications in chronic infections, autoimmunity, and cardiovascular inflammation, where immune regulation is central. Thus, EP receptor pharmacology illustrates how prostaglandin biology is evolving into targeted immunomodulation. Strip crowns were selected to match tooth size (Figure 4). The composite core was etched with 37% phosphoric acid for 15 seconds, rinsed, and gently dried. A bonding agent was applied, air-thinned, and light-cured for 10 seconds. Composite-filled strip crowns were placed with gentle pressure for proper adaptation. Excess was removed before curing. Each crown was light-cured

from buccal, lingual, and incisal sides for 40 seconds. Once set, the celluloid forms were peeled off, and any excess was finished and polished with burs and discs. Occlusion was adjusted, and final polishing ensured a natural look. (Figure 5)

4. Beyond Inflammation: Ophthalmology and Vascular Medicine
Prostaglandins retain major roles outside immunology.

- Ophthalmology: $PGF_2\alpha$ analogs such as latanoprost, bimatoprost, tafluprost, and travoprost are first-line in glaucoma therapy. They reduce intraocular pressure by enhancing uveoscleral outflow. Recent innovations include nitric oxide-donating prostaglandins and preservative-free formulations, which improve tolerability and compliance[12].
- Vascular Medicine: Intravenous prostaglandins (e.g., iloprost, alprostadil) are used to treat peripheral vascular disease, pulmonary hypertension, and digital ulcers in systemic sclerosis. A recent meta-analysis confirmed short-term efficacy in systemic sclerosis–related Raynaud's phenomenon, though larger controlled studies are needed to establish long-term benefits[13].

These examples underscore the clinical versatility of prostaglandins, bridging inflammation, vascular biology, and ocular physiology.

5. Lipidomics and Analytical Advances

A major revolution in prostaglandin science has been the advent of lipidomics—the high-throughput quantification of lipid mediators by mass spectrometry. Lipidomics enables simultaneous tracking of multiple prostanoids and oxylipins, revealing complex network dynamics.

In kidney disease, lipidomic profiling shows that PGE_2 pathway activation correlates with albuminuria during glomerular hyperfiltration, highlighting prostaglandins as potential biomarkers of renal injury[14]. In sepsis, lipidomic analysis has uncovered widespread disturbances in oxylipin metabolism, which may help predict outcomes in critically ill patients.

Standardization of methods is key. Recent guidelines provide detailed protocols for sample preparation, chromatographic separation, and data interpretation, improving reproducibility across laboratories[15].

Meanwhile, synthetic chemistry has achieved breakthroughs in prostaglandin synthesis. Traditional routes required 15–20 steps with challenging stereocontrol. New chemoenzymatic strategies reduce synthesis to just 5–7 steps, enabling gram-scale production and facilitating drug discovery[16].

Together, lipidomics and synthetic chemistry empower researchers to map prostaglandin biology at systems scale and generate tools for translational research.

6. Rethinking Safety: Preserving Beneficial Prostanoids

The NSAID story highlighted that prostaglandins are not uniformly detrimental. While excess PGE_2 drives inflammation and cancer progression, PGI_2 protects against thrombosis, and PGD_2 regulates sleep and allergy. Broad suppression, as seen with NSAIDs, thus disrupts beneficial functions.

Modern strategies seek to preserve protective prostanoids while reducing harmful ones. mPGES-1 inhibition exemplifies this, as it suppresses PGE_2 but spares PGI_2^1,4. Transporter-based modulation may allow tissue-specific adjustments, enhancing wound healing without systemic effects[6,7].

This nuanced view reframes prostaglandin pharmacology from "block everything" to "rebalance selectively", aligning with the principles of precision medicine.

7. Future Directions

Three key trajectories are shaping prostaglandin research:

- Node-Specific Anti-Inflammatories: Continued clinical evaluation of mPGES-1 inhibitors, with pharmacodynamic biomarkers such as urinary PGE-M to track efficacy.
- Immuno-Oncology Combinations: Integration of EP4 antagonists with immune checkpoint inhibitors, myeloid-targeting therapies, and radiotherapy.
- Systems Pharmacology: Application of lipidomics, transporter activity assays, and genetic stratification (SLCO2A1 mutations) to tailor prostaglandin modulation to disease and patient context.

If successful, these approaches could transform prostaglandins from nonspecific drug targets into a platform for personalized therapy across inflammatory, oncologic, vascular, and metabolic diseases.

CONCLUSION

Prostaglandins are ancient mediators newly reborn as precision targets. Recent research highlights how selective modulation—whether through mPGES-1 inhibitors, EP receptor antagonists, transporter regulation, or lipidomic-guided strategies—can retain the benefits of prostanoids while mitigating their risks. Advances in synthetic chemistry and analytical lipidomics provide the technical foundation for this transformation.

The field is moving away from broad suppression and toward strategic rebalancing of prostanoid networks, marking a conceptual evolution in pharmacology. With ongoing clinical trials and translational research, prostaglandins are poised to become a cornerstone of precision medicine in the coming decade.

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SUZETRIGINE (VX-548): A NOVEL NON-OPIOID NAV1.8 INHIBITOR FOR THE MANAGEMENT OF MODERATE-TO-SEVERE PAIN

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ABSTRACT

Opioid overuse and safety liabilities have driven an urgent search for effective, non-addictive analgesics. Suzetrigine (VX-548), a first-in-class, peripherally acting small-molecule inhibitor of the voltage-gated sodium channel NaV1.8, represents a new therapeutic strategy targeting nociceptor excitability rather than central opioid receptors. NaV1.8 is highly enriched in peripheral pain-sensing neurons and critical for action-potential propagation in inflammatory and neuropathic states, enabling selective analgesia while minimizing central nervous system effects. In January 2025 the U.S. Food and Drug Administration (FDA) approved suzetrigine (brand name JOURNAVX™) for adults with moderate-to-severe acute pain, based on phase 3 trials in abdominoplasty and bunionectomy demonstrating superior analgesia over placebo and broadly comparable pain reduction to hydrocodone/acetaminophen, with generally mild-to-moderate adverse events. The approved regimen begins with a 100-mg loading dose (two 50-mg tablets) on an empty stomach, followed by 50 mg every 12 hours; grapefruit products should be avoided and strong CYP3A inhibitors are contraindicated. This narrative review summarizes the mechanistic rationale, pivotal clinical evidence, safety and drug–drug interaction profile, regulatory status and dosing, potential clinical role, and remaining uncertainties for suzetrigine as an opioid-sparing option in acute pain management.[1–4]

KEYWORDS

Suzetrigine; VX-548; NaV1.8 inhibitor; acute pain management; non-opioid analgesic; sodium channel blocker; JOURNAVX; postoperative pain; opioid-sparing strategies; FDA approval.

INTRODUCTION

Acute pain remains one of the most common reasons for healthcare encounters and medication use. For decades, clinicians have relied on opioids for moderate-to-severe pain despite well-recognized risks including respiratory depression, constipation, nausea, misuse, and opioid use disorder (OUD). Progress in non-opioid pharmacology has been incremental, dominated by NSAIDs and acetaminophen. A mechanistically distinct approach —selectively dampening peripheral nociceptor excitability—has long been attractive but elusive. Voltage-gated sodium (NaV) channels underlie action potential initiation and conduction; among these, NaV1.8 (SCN10A) is tetrodotoxin-resistant and primarily expressed in small-diameter dorsal root ganglion neurons that convey nociceptive input. Genetic and pharmacologic studies have validated NaV1.8 as a pain target in inflammatory and neuropathic models.[2–4]

VX-548 (suzetrigine) was designed to potently and selectively inhibit NaV1.8, reducing peripheral pain-signal propagation without engaging central opioid pathways. Early human studies suggested clinically relevant analgesia and a favorable tolerability profile. In 2025, the FDA approved suzetrigine for the short-term treatment of moderate-to-severe acute pain in adults—the first new analgesic class for acute pain in over two decades—potentially reshaping multimodal analgesia strategies.[1]

MECHANISM OF ACTION AND PHARMACOLOGIC RATIONALE

NaV1.8 contributes a major fraction of the inward sodium current in nociceptors, particularly at lower temperatures and during inflammatory sensitization, and mediates repetitive firing required for sustained nociceptive transmission. Because NaV1.8 expression is largely limited to peripheral sensory neurons, selective inhibition is predicted to reduce pain signaling while sparing central synaptic function and cardiac/brain NaV isoforms (e.g., NaV1.5, NaV1.1), potentially improving safety. The broader NaV family includes targets with unacceptable off-target risks; therefore, selectivity is critical. Preclinical and translational off-target risks; therefore, selectivity is critical. Preclinical and translational evidence—antisense knockdown, small-molecule blockers, and human genetics—has linked NaV1.8 activity to thermal and mechanical hyperalgesia, strengthening the mechanistic basis for clinical testing.[2–4]

CLINICAL DEVELOPMENT

Early proof-of-concept

A pivotal randomized, double-blind, placebo- and active-controlled program evaluated VX-548 in acute post-surgical pain (abdominoplasty, bunionectomy). In the New England Journal of Medicine report, higher-dose VX-548 reduced the time-weighted summed pain intensity difference (SPID) over 48 hours versus placebo, with adverse events predominantly mild-to-moderate. These phase 2/2b data established clinical activity and dose–response, supporting phase 3.[5–6]

Phase 3 results

Vertex's phase 3 program comprised two large, similarly designed trials in abdominoplasty and bunionectomy, each with placebo and hydrocodone/acetaminophen (HB/APAP) comparators. Top-line disclosures reported significant superiority to placebo on primary pain endpoints and clinically meaningful pain reductions, with a faster onset than placebo. Comparative efficacy to HB/APAP was broadly similar on overall pain relief, though onset appeared slower than HB/APAP in bunionectomy. An independent comparative-effectiveness review (ICER, December 2024 draft) concluded that evidence versus placebo is robust, and that versus opioids or NSAIDs is "promising but inconclusive," given overlapping confidence intervals, rescue-medication handling, and the relatively low opioid dose used in trials.[7–8]

Regulatory approval (2025)

On January 30, 2025, the FDA approved suzetrigine (JOURNAVX™) tablets for adults with moderate-to-severe acute pain. The approval reflects a new mechanism of analgesia and follows Breakthrough Therapy, Fast Track, and Priority Review designations. Efficacy was demonstrated in two randomized, double-blind trials with access to ibuprofen rescue; safety was informed by pooled controlled data (n≈874) and an open-label study spanning various acute pain conditions (n≈256). Common adverse reactions included pruritus, muscle spasms, elevated creatine phosphokinase, and rash.[9]

Dosing, Administration, and Practical Use

According to the FDA-approved labeling, patients take a 100-mg loading dose (two 50-mg tablets) on an empty stomach (≥1 hour before or 2 hours after food). Starting 12 hours later, the maintenance dose is 50 mg orally every 12 hours, with or without food. Use the shortest duration consistent with treatment goals; suzetrigine has not been studied beyond 14 days. Grapefruit products should be avoided. Strong CYP3A inhibitors (e.g., clarithromycin, itraconazole) are contraindicated; coadministration with moderate CYP3A inhibitors or moderate hepatic impairment requires dosage modification (transitioning to once-daily dosing after the fourth dose).[10–11]

These practical considerations allow suzetrigine to slot into peri-operative order sets and discharge prescriptions as part of multimodal regimens (e.g., acetaminophen, scheduled NSAID where appropriate, regional anesthesia), with the potential to reduce or replace opioid rescue in selected patients.[8]

Safety Profile and Drug–Drug Interactions

Across controlled trials, adverse events were predominantly mild to moderate; nausea was less common than with HB/APAP in at least one surgery model, an advantage for post-operative recovery. The label highlights pruritus, muscle spasms, CPK elevations, and rash as the most common reactions. From a mechanistic perspective, peripheral NaV selectivity aims to minimize central nervous system and cardiac effects; nevertheless, ICER flagged theoretical risks (e.g., arrhythmia) as areas for ongoing pharmacovigilance given NaV channel biology. Real-world data will be important to clarify these risks and to define any renal safety signals hinted at in limited datasets.[8–9]

Suzetrigine is metabolized via CYP3A pathways; thus, strong CYP3A inhibitors are contraindicated, and grapefruit should be avoided. Guidance is

provided for moderate CYP3A inhibitors and hepatic impairment. Clinicians should review concomitant medications carefully (e.g., certain antifungals, macrolides, HIV protease inhibitors). No boxed warning for misuse, addiction, or respiratory depression applies, reflecting its non-opioid mechanism.[10]

How Does Suzetrigine Compare to Opioids and NSAIDs?

The core clinical question is not whether suzetrigine "works"—it does versus placebo—but how it fits relative to established therapies. In the pivotal program, overall pain reduction was similar to hydrocodone/acetaminophen at doses lower than some routine clinical regimens; onset of relief may be slower in certain models (bunionectomy), while nausea appears less frequent. Network meta-analyses attempting broader cross-trial comparisons with higher-dose opioids and NSAIDs show overlapping confidence intervals, limiting firm rank-ordering. Reading the totality of evidence, suzetrigine appears to offer meaningful analgesia comparable to commonly used combinations for many patients, with a differentiated safety/abuse profile. This supports its use in multimodal regimens and as an opioid-sparing or opioid-replacing option, particularly in patients at risk for opioid-related harms.[8]

Health-System and Societal Considerations

ICER's draft cost-effectiveness modeling (using a placeholder one-week course price) suggested suzetrigine could be cost-saving versus HB/APAP when OUD risks from short opioid courses are considered over the long term—though results depend heavily on uncertain real-world OUD incidence after brief opioid exposure. Post-approval pricing reports indicate a wholesale price around US$15.50 per 50-mg tablet in early coverage analyses; payers will shape access through formulary placement and prior authorization. From a public-health perspective, an effective non-opioid class could reduce the need for opioids in routine post-operative care, potentially lowering exposure in high-risk populations.[8],[12–13]

Place in Therapy: Who May Benefit Most?

Peri-operative/post-procedural pain: For moderate-to-severe pain after procedures such as abdominoplasty, bunionectomy, hernia repair, or orthopedic day surgery, suzetrigine can be initiated with a loading dose and maintained every 12 hours for several days. It can substitute for or reduce opioids where NSAIDs are contraindicated (e.g., history of GI bleeding, renal risk) or poorly tolerated, and where acetaminophen alone is insufficient.[9]

Patients with opioid risk factors or intolerance: Individuals with prior substance use disorder, obstructive sleep apnea, obesity hypoventilation, or troublesome opioid adverse effects (nausea/vomiting, constipation, pruritus) may particularly benefit from an opioid-sparing plan featuring suzetrigine.[9]

Settings emphasizing early mobilization and enhanced recovery: Lower nausea burden and lack of respiratory depression risk may support earlier ambulation and discharge, aligning with ERAS protocols when combined with regional anesthesia and non-opioids. (Direct ERAS data with suzetrigine are not yet available.)[8]

Practical Implementation Tips

1. **Embed in order sets**: Include the labeled loading dose timing (empty stomach) and maintenance schedule; specify avoidance of grapefruit and contraindication with strong CYP3A inhibitors.
2. **Plan rescue**: Maintain access to acetaminophen and/or NSAIDs as appropriate; some pivotal participants used ibuprofen rescue, which mirrors real-world multimodal care.
3. **Educate patients**: Emphasize that suzetrigine is non-opioid, taken on a schedule (not PRN initially), and that some patients may experience pruritus or muscle spasms.
4. **Review concomitants**: Screen for CYP3A interactions and hepatic impairment to adjust dosing or avoid therapy.[9],[10]

Limitations and Unanswered Questions

Comparative efficacy head-to-head: While trials included an HB/APAP arm, broader active-comparator data against higher-dose opioids, IV options, and various NSAIDs remain indirect; network analyses are hypothesis-generating due to cross-trial variability.

Onset and functional outcomes: Onset may vary by pain model; further studies could clarify time to meaningful relief and impacts on sleep, mobilization, and opioid-rescue consumption.

Generalizability: Most pivotal data come from two surgical models; evidence across diverse acute pain settings (e.g., trauma, dental surgery, cesarean section) will refine external validity.

Long-term safety: Although studied up to 14 days, real-world pharmacovigilance will be essential to identify rare adverse events, confirm the absence of pro-arrhythmic signals, and define any renal considerations.

Chronic pain: Early signals in neuropathic conditions have been mixed; to date, the approval is limited to acute pain, and the role in chronic pain remains investigational.[8],[14]

CONCLUSION

Suzetrigine inaugurates a long-anticipated class of peripherally selective analgesics that modulate nociceptor excitability by inhibiting NaV1.8. Its FDA approval for moderate-to-severe acute pain provides clinicians with a mechanistically novel option that can meaningfully reduce pain while avoiding opioid-specific risks. The labeled regimen is straightforward, the safety profile to date is acceptable, and the drug integrates naturally into multimodal strategies. Key next steps include expanding comparative-effectiveness evidence across procedures, tracking real-world safety, optimizing patient selection, and evaluating health-system impact on opioid exposure. As data accumulate, suzetrigine could become a cornerstone in modern, opioid-sparing acute pain care[1]

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RECENT CONCEPTS IN AVASCULAR NECROSIS (OSTEONECROSIS) OF THE FEMORAL HEAD

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ABSTRACT

Avascular necrosis (AVN), or osteonecrosis, is a progressive ischemic condition marked by bone cell death due to impaired blood flow, leading to femoral head collapse and secondary osteoarthritis. It arises from both traumatic vascular injury and non-traumatic causes such as corticosteroid use, alcohol, coagulation disorders, dyslipidemia, and genetic factors. Early diagnosis is crucial, as interventions are most effective before subchondral fracture; MRI remains the gold standard, with ARCO (2019) and ACR (2022) guidelines confirming that radiographs plus MRI are generally sufficient for staging. Lesion size and location strongly predict collapse, while novel approaches like radiomics, lipid biomarkers, and genetic profiling are under study for individualized risk assessment. Management centers on hip preservation in early disease and arthroplasty in advanced stages. Core decompression (CD) remains standard, increasingly combined with biologic augmentation such as bone marrow aspirate concentrate (BMAC) or mesenchymal stem cells, which show promise in delaying progression. Adjuncts like bisphosphonates, statins, anticoagulants, protected weight-bearing, and shock wave therapy may add benefit if started early. Emerging strategies include stromal- and adipocyte-targeted therapies and predictive nomograms, while future directions call for standardized cell therapies, validated biomarkers, and consensus on conservative care.

KEYWORDS

avascular necrosis; osteonecrosis; femoral head; core decompression; bone marrow aspirate concentrate

INTRODUCTION

Avascular necrosis (AVN) of the femoral head is a disabling condition that predominantly affects young to middle-aged adults and can culminate in early hip osteoarthritis if not recognized and treated before collapse. Etiologies include traumatic vascular disruption (e.g., femoral neck fracture, dislocation) and non-traumatic pathways such as corticosteroid exposure, alcohol use, coagulation and lipid metabolism disorders, and systemic diseases; incidence estimates in the United States reach 10–20k new cases annually [1]. Contemporary work reframes AVN as a common ischemic endpoint precipitated by diverse upstream hits that converge on marrow fat hypertrophy, microvascular failure, and impaired bone remodeling .

IMAGING, STAGING, AND RISK PREDICTION

Imaging

MRI is the most sensitive modality for early AVN and is recommended for both symptomatic and contralateral hip screening in high-risk contexts . The double-line sign on T2-weighted sequences and subchondral low-signal rim on T1/T2 are classic, while contrast/perfusion MRI can help distinguish AVN from bone-marrow edema syndrome and subchondral insufficiency fractures [5]. While CT can delineate subchondral fracture and collapse, contemporary evidence suggests that routine CT is not required for diagnosis/staging when ARCO 2019 criteria are applied—plain radiographs plus MRI suffice for most patients . Beyond MRI, 18F-FDG or 18F-NaF PET/CT may show higher sensitivity or prognostic utility in selected cases, with higher SUVmax associated with impending collapse, though this is not yet standard of care [3].

Staging

The Association Research Circulation Osseous (ARCO) 2019 revision standardized a four-stage system: Stage I (X-ray normal; MRI or bone scan positive), Stage II (X-ray abnormal without subchondral fracture), Stage III (subchondral/necrotic zone fracture; IIIA ≤2 mm depression; IIIB >2 mm), and Stage IV (osteoarthritis) [6]. The ACR Appropriateness Criteria (2022, revised) reinforce MRI's centrality in characterization and in distinguishing key mimics[7].

Predicting Collapse

Lesion size and location are the dominant predictors of collapse and should drive treatment selection [6]. The Japanese Investigation Committee (JIC) and modified Kerboul angle quantify necrotic extent; small lesions may remain stable without intervention, whereas large weight-bearing dome lesions frequently progress[8]. Recent nomogram work integrates ARCO stage, lesion metrics, and patient factors to predict 2-year collapse risk and support shared decision-making [9]. Radiomics and lipid biomarkers show promise for steroid-induced AVN but need external validation [4].

CONSERVATIVE AND ADJUNCTIVE THERAPIES

Conservative measures aim to alleviate pain, reduce mechanical stress, and potentially slow progression in pre-collapse disease—best as adjuncts or temporizing strategies [6,10]. Systematic reviews suggest that protected weight-bearing can offer short-term symptomatic relief and may delay collapse in select early lesions, but effects are heterogeneous and highly stage-dependent[11]. Pharmacologic options include bisphosphonates—observational data and long-term cohorts indicate they may decrease pain and delay collapse/arthroplasty in early stages when started before subchondral fracture [3]. Other agents (statins in high-dose steroid recipients; anticoagulation for documented thrombophilia) may be considered case-by-case [1,6]. Physical modalities such as extracorporeal shock wave therapy (ESWT) have mixed evidence; they may improve pain/function but rarely change structural endpoints without combined surgical measures [6,12]. Across these modalities, careful patient selection and early institution are key, and none replaces structural restoration when the necrotic segment is large or fracture has occurred [6,12].

CORE DECOMPRESSION AND BIOLOGIC AUGMENTATION

Core decompression (CD) reduces intraosseous pressure and promotes revascularization; it remains the workhorse for symptomatic Stage I–II lesions with small-to-moderate necrotic areas [1,13]. Over the last decade, biologic augmentation using bone marrow aspirate concentrate (BMAC) or concentrated autologous bone marrow–derived MSCs has become a leading strategy to enhance outcomes. Comparative and prospective series—some with 5–10-year horizons—report improved radiographic progression-free survival and delayed conversion to total hip arthroplasty (THA) when CD is combined with concentrated marrow cells, particularly in early stages and smaller lesions[13,14]. Cellular quality (CD34+ counts/CFU-F) may correlate with outcomes, underscoring the need for standardized harvest and processing protocols [2].

Technique refinements include multiple small-diameter channels, use of expandable reamers to target the necrotic zone, and adjunctive bone grafting (autograft or synthetic) to support the subchondral plate [13].Ongoing prospective multi-center trials are evaluating standardized BMAC augmentation in early AVN, which should clarify patient selection and effect sizes [9].

JOINT-PRESERVING STRUCTURAL PROCEDURES

When necrotic lesions are larger but pre-collapse or minimally collapsed (ARCO IIB–IIIA), surgeons may combine CD/biologics with structural grafting to buttress the subchondral plate, or consider rotational osteotomies that offload the necrotic segment from the weight-bearing dome[11]. Hip-preserving strategy selection hinges on lesion size/location and patient age/activity demands; recent reviews synthesize algorithms indicating that small, medially located lesions fare best with CD±biologics, while larger superolateral lesions often require structural support or progress to arthroplasty [11,14].

ARTHROPLASTY

For ARCO IIIB–IV disease (substantial collapse, acetabular involvement), total hip arthroplasty provides reliable pain relief and function. Modern implants yield outcomes approaching those for primary osteoarthritis, although younger age at implantation raises concerns about lifetime revision risk. The strategic goal is not to defer THA at all costs but to preserve the

native hip when structurally feasible and to convert to arthroplasty once collapse/mechanical incongruity negates preservation benefits [1].

SPECIAL CONTEXTS: STEROID-INDUCED AND POST-COVID CASES

Steroid-induced AVN remains common; predictive models based on lipid profiles and radiomics may help identify high-risk individuals for surveillance imaging [4]. Post-COVID steroid exposure has renewed attention to early detection; cohorts suggest that bisphosphonate-based regimens instituted pre-collapse can limit progression in carefully selected patients [3].

PRACTICAL, STAGE-BASED APPROACH (SYNTHESIS OF RECENT CONCEPTS)

1. **High-risk patient, hip pain or incidental MRI changes** → Obtain radiographs and MRI; stage with ARCO 2019; measure lesion size/location (JIC / modified Kerboul)[1,6,7,9,15]
2. **Small, medial early lesions (ARCO I–II, low Kerboul angle)** → Consider observation with risk-modifying measures (protected weight-bearing, control lipids/alcohol/steroids) ± pharmacologic adjuncts; close MRI follow-up [15]
3. **Symptomatic early lesions (especially lateral dome involvement)** → Core decompression; discuss biologic augmentation (BMAC/MSC) given growing evidence of improved survivorship, while noting heterogeneity and need for standardized protocols [2,5,13,14]
4. **Large pre-collapse or minimal collapse (II–IIIa)** → CD plus structural grafting/osteotomy in select anatomies; ensure shared decision-making using nomograms and patient factors [11]
5. **Established collapse or secondary OA (IIIb–IV)** → Proceed to THA, as joint preservation is unlikely to succeed .

FUTURE DIRECTIONS

Priorities include

1. multicenter randomized trials of CD+cell therapies with standardized harvest/dose metrics;
2. external validation of radiomic/biomarker models across etiologies;
3. mechanistic studies on adipocyte–MSC crosstalk and targeted therapies; and
4. consensus on conservative care bundles and timing relative to surgical intervention[4,6,11].

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Review Article

THE BRACHIAL PLEXUS: ANATOMY, VARIATIONS, AND CLINICAL SIGNIFICANCE

Dr. Thakur Shailesh Kumar Singh, Dr. P.P. Dubey, Dr. Beena Arora, Dr. Harish Mehta

ABSTRACT

The brachial plexus is a complex network of nerves formed by the anterior rami of spinal nerves C5–T1, responsible for the motor and sensory innervation of the upper limb. It plays a crucial role in upper limb function, and its detailed understanding is essential for anatomists, clinicians, and surgeons alike. The plexus is organized into roots, trunks, divisions, cords, and branches, ultimately giving rise to the major peripheral nerves of the upper limb, including the musculocutaneous, median, ulnar, radial, and axillary nerves. Development occurs during embryogenesis through axonal growth guided by molecular signals, with variations frequently observed in formation and branching patterns. Clinical significance is vast, as brachial plexus injuries may occur due to trauma, obstetric complications, tumors, or iatrogenic causes, leading to severe functional deficits. Advances in imaging, including MRI and high-resolution ultrasound, aid in diagnosis, while electrophysiological studies provide insight into lesion severity. Treatment ranges from conservative management to microsurgical nerve repair, grafting, or transfer. This review highlights the detailed anatomy, embryology, common variations, and clinical aspects of the brachial plexus, emphasizing its diagnostic and surgical importance. Early recognition and multidisciplinary management remain critical in optimizing outcomes for patients with brachial plexus pathologies.

KEYWORDS

Brachial plexus; peripheral nerves; cervical roots; nerve injury; Erb's palsy; Klumpke's palsy

INTRODUCTION

The brachial plexus is a major peripheral nerve network formed by the ventral rami of the lower cervical and first thoracic spinal nerves (C5–T1)[1]. It provides the sensory and motor supply to the upper limb, shoulder girdle, and portions of the thorax. Owing to its anatomical complexity and variations, the brachial plexus is a frequent subject of clinical interest, particularly in relation to trauma, obstetric complications, and surgical interventions[2]. A thorough understanding of its anatomy and clinical correlations is vital for medical students, surgeons, anesthesiologists, and neurologists[3].

ANATOMY

The brachial plexus is conventionally divided into five parts: roots, trunks, divisions, cords, and terminal branches.

- **Roots**: Derived from C5–T1 anterior rami. Occasionally contributions arise from C4 or T2 (prefixed and postfixed plexus)[4].
- **Trunks**: Roots merge to form upper (C5–C6), middle (C7), and lower (C8–T1) trunks.
- **Divisions**: Each trunk splits into anterior and posterior divisions, supplying flexor and extensor compartments, respectively.
- **Cords**: Named relative to the axillary artery—lateral, medial, and posterior.
- **Branches**: Terminal branches include musculocutaneous, axillary, radial, median, and ulnar nerves. Several supraclavicular and infraclavicular branches supply the shoulder, chest wall, and scapular muscles[5].

EMBRYOLOGY

The brachial plexus develops during the fourth to sixth weeks of intrauterine life. Neural crest cells migrate to form spinal ganglia, and motor axons extend distally to connect with developing limb buds[6]. Molecular guidance cues such as netrins, semaphorins, and neurotrophins orchestrate axonal pathfinding[7]. Variations in embryonic signaling explain many of the anatomical differences observed in adults.

ANATOMICAL VARIATIONS

Numerous variations are documented in the formation and branching patterns of the plexus. Common anomalies include:

- Prefixed (C4–C8) and postfixed (C6–T2) plexuses[8].
- Variations in musculocutaneous–median nerve communications.
- Absence or duplication of specific branches[9].
- Variable origins of long thoracic and dorsal scapular nerves.

These variations have profound implications during surgical procedures and anesthetic blocks.

CLINICAL CORRELATIONS

Obstetric injuries

Brachial plexus birth palsy occurs during difficult deliveries due to traction on the plexus. Erb's palsy (C5–C6 injury) results in an adducted, medially rotated arm, while Klumpke's palsy (C8–T1 injury) leads to claw hand and Horner's syndrome in severe cases[10].

Traumatic injuries

High-energy trauma, such as road traffic accidents, may cause avulsion, rupture, or stretch injuries. Root avulsions are the most severe, often requiring nerve transfers[11].

Neoplastic and inflammatory lesions

Tumors such as Pancoast tumors, metastatic disease, or inflammatory neuropathies can compress or infiltrate the plexus[12].

Iatrogenic injuries

Brachial plexus block anesthesia, central venous catheterization, and surgical procedures may inadvertently injure the plexus if anatomical landmarks are not respected[13].

IMAGING AND DIAGNOSTICS

High-resolution ultrasonography provides dynamic visualization of plexus anatomy and pathology[14]. MRI is the gold standard for evaluating root avulsions, tumors, and inflammatory lesions. Electrophysiological studies, including nerve conduction studies and electromyography, help differentiate preganglionic from postganglionic lesions[15].

MANAGEMENT

Management strategies depend on etiology and severity:

- **Conservative therapy**: Physiotherapy and splinting in mild or recovering injuries.
- **Pharmacological therapy**: Neuropathic pain management with antiepileptics and antidepressants.
- **Surgical interventions**: Nerve grafts, transfers (e.g., spinal accessory to suprascapular nerve), and microsurgical repairs for avulsion injuries[11].
- **Rehabilitation**: Long-term physiotherapy and occupational therapy are essential for functional recovery.

CONCLUSION

The brachial plexus represents one of the most intricate nerve networks in the human body. Its complex anatomy and frequent variations make it clinically significant in trauma, obstetrics, anesthesia, and oncology. Early recognition of plexus pathology, aided by imaging and electrophysiology, can significantly improve management outcomes. Advances in microsurgical techniques and regenerative medicine continue to offer hope for patients with devastating brachial plexus injuries. A strong foundation in its anatomy and clinical relevance remains indispensable for medical professionals.

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TUBERCULAR LYMPHADENITIS: CLINICAL PERSPECTIVES, DIAGNOSTIC CHALLENGES, AND MANAGEMENT

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ABSTRACT

Tubercular lymphadenitis (TBLN) is the most common form of extrapulmonary tuberculosis (EPTB), accounting for up to 30–40% of EPTB cases worldwide. It primarily involves cervical lymph nodes but may also affect axillary, inguinal, mesenteric, and mediastinal nodes. TBLN arises from hematogenous or lymphatic spread of Mycobacterium tuberculosis from a primary pulmonary focus, though many patients lack active lung disease. The disease typically affects young adults and women in endemic regions. Clinically, it presents with gradually enlarging, firm, painless lymph nodes that may become matted, suppurate, or form sinuses if untreated. Diagnosis requires a combination of clinical suspicion, fine-needle aspiration cytology (FNAC), Ziehl–Neelsen staining, mycobacterial culture, histopathology, and molecular assays such as GeneXpert, which simultaneously detects tuberculosis and rifampicin resistance. Imaging modalities like ultrasound and CT help in staging and monitoring. The mainstay of treatment is anti-tubercular therapy (ATT) under WHO-recommended regimens; surgery is reserved for diagnostic dilemmas or residual abscesses. The prognosis is generally favorable, but drug resistance, paradoxical reactions, and recurrence complicate management. This review summarizes current understanding of epidemiology, pathogenesis, clinical features, diagnostic strategies, and management of tubercular lymphadenitis, highlighting the need for early detection, compliance to therapy, and integration of molecular tools for improved outcomes.

KEYWORDS

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

INTRODUCTION

Tuberculosis (TB) remains one of the leading infectious diseases globally, with approximately 10.6 million new cases and 1.3 million deaths reported in 2022 according to the WHO[1]. While pulmonary TB predominates, extrapulmonary tuberculosis (EPTB) constitutes 15–20% of cases in immunocompetent individuals and up to 50% in HIV-positive patients[2]. Among EPTB forms, tubercular lymphadenitis (TBLN) is the most common, particularly in endemic countries such as India, Bangladesh, and African nations[3].

EPIDEMIOLOGY

TBLN accounts for 30–40% of EPTB presentations[4]. Cervical nodes are most frequently involved, giving rise to the term "scrofula" historically[5]. The disease commonly affects young adults and shows a female predominance in several studies[6]. Risk factors include HIV infection, malnutrition, diabetes mellitus, immunosuppression, and close contact with active TB cases[7].

ETIOPATHOGENESIS

The pathogenesis involves lymphatic or hematogenous dissemination of M. tuberculosis bacilli from a primary pulmonary focus, which may be latent or clinically silent[8]. The bacilli trigger granulomatous inflammation characterized by epithelioid histiocytes, Langhans giant cells, and caseating necrosis[9]. The host immune response, particularly T-cell mediated immunity and cytokines like IFN-γ and TNF-α, plays a pivotal role in disease progression or containment[10].

CLINICAL FEATURES

Patients usually present with:

- Gradual, painless enlargement of lymph nodes, most often cervical (posterior triangle)[11].
- Nodes are discrete initially but later become matted due to periadenitis.
- Advanced cases may develop liquefaction, abscess formation, and sinus tracts discharging pus.

(2022, revised) reinforce MRI's centrality in characterization and in distinguishing key mimics[7].

Predicting Collapse

Lesion size and location are the dominant predictors of collapse and should drive treatment selection [6]. The Japanese Investigation Committee (JIC) and modified Kerboul angle quantify necrotic extent; small lesions may remain stable without intervention, whereas large weight-bearing dome lesions frequently progress[8]. Recent nomogram work integrates ARCO stage, lesion metrics, and patient factors to predict 2-year collapse risk and support shared decision-making [9]. Radiomics and lipid biomarkers show promise for steroid-induced AVN but need external validation [4].

CONSERVATIVE AND ADJUNCTIVE THERAPIES

Conservative measures aim to alleviate pain, reduce mechanical stress, and potentially slow progression in pre-collapse disease—best as adjuncts or temporizing strategies [6,10]. Systematic reviews suggest that protected weight-bearing can offer short-term symptomatic relief and may delay collapse in select early lesions, but effects are heterogeneous and highly stage-dependent[11]. Pharmacologic options include bisphosphonates—observational data and long-term cohorts indicate they may decrease pain and delay collapse/arthroplasty in early stages when started before subchondral fracture [3]. Other agents (statins in high-dose steroid recipients; anticoagulation for documented thrombophilia) may be considered case-by-case [1,6]. Physical modalities such as extracorporeal shock wave therapy (ESWT) have mixed evidence; they may improve pain/function but rarely change structural endpoints without combined surgical measures [6,12]. Across these modalities, careful patient selection and early institution are key, and none replaces structural restoration when the necrotic segment is large or fracture has occurred [6,12].

DIAGNOSIS

Clinical evaluation

Persistent, painless lymphadenopathy with systemic symptoms in endemic areas strongly suggests TBLN, though differential diagnoses must be considered.

Fine-needle aspiration cytology (FNAC)

FNAC is minimally invasive and shows granulomas with necrosis and giant cells. Ziehl–Neelsen (ZN) staining detects acid-fast bacilli in ~40–60% of cases[13].

Culture and molecular methods

Culture on Lowenstein-Jensen medium is the gold standard but time-consuming. GeneXpert/CBNAAT provides rapid detection and rifampicin resistance profiling[14].

Histopathology

Excisional biopsy remains the diagnostic gold standard in equivocal cases, revealing caseating granulomatous inflammation[15].

Imaging

Ultrasonography demonstrates hypoechoic nodes with intranodal necrosis and matting, while CT/MRI help assess deep-seated nodes and complications.

DIFFERENTIAL DIAGNOSIS

- Non-tubercular mycobacterial infections
- Lymphomas
- Metastatic lymphadenopathy
- Sarcoidosis
- Chronic pyogenic lymphadenitis[11]

native hip when structurally feasible and to convert to arthroplasty once collapse/mechanical incongruity negates preservation benefits [1].

MANAGEMENT

Medical therapy

The cornerstone of treatment is anti-tubercular therapy (ATT) following national and WHO guidelines. Standard regimens include a 6-month course (2 months of isoniazid, rifampicin, pyrazinamide, ethambutol; followed by 4 months of isoniazid and rifampicin)[12]. Treatment duration may be extended to 9 months in complicated or drug-resistant cases.

Surgical intervention

Surgery is generally limited to diagnostic biopsy, drainage of cold abscesses, or excision of residual nodes causing functional or cosmetic issues[13].

Special considerations

- **HIV-coinfection** requires concurrent antiretroviral therapy.
- **Drug resistance** must be promptly identified using GeneXpert or line probe assays.
- **Paradoxical reactions** may occur, presenting as worsening lymphadenopathy during treatment; these usually resolve without change in therapy but may require corticosteroids[14].

COMPLICATIONS AND PROGNOSIS

Complications include sinus formation, scarring, fistulae, and rarely systemic dissemination. With appropriate ATT, prognosis is excellent, though relapse occurs in 3–13% of cases[15]. Drug resistance and poor compliance remain significant challenges.

CONCLUSION

Tubercular lymphadenitis remains the most prevalent extrapulmonary manifestation of tuberculosis. Early recognition, accurate diagnosis through FNAC and molecular tools, and adherence to WHO-recommended ATT are critical for cure. Surgical interventions are supportive but not primary. Ongoing challenges include HIV coinfection, drug resistance, and treatment adherence. Future directions lie in developing rapid diagnostics, shorter treatment regimens, and strategies to address multidrug-resistant TB. A multidisciplinary approach integrating clinicians, microbiologists, and public health systems is essential to reduce the burden of this age-old disease.

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DIABETES PREVENTION VIA LIFESTYLE INTERVENTIONS: A COMPREHENSIVE REVIEW

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ABSTRACT

Type 2 diabetes mellitus (T2DM) has emerged as one of the most pressing public health challenges worldwide, with its prevalence increasing at alarming rates in both developed and developing countries. The disease is strongly associated with obesity, sedentary behavior, unhealthy diets, stress, and poor sleep quality. Although pharmacological interventions can delay or manage the onset of diabetes, lifestyle modifications remain the most effective, sustainable, and cost-efficient preventive approach. This review explores the current understanding of lifestyle interventions—including dietary modification, physical activity, weight management, sleep optimization, and stress reduction—and their role in preventing the onset of T2DM. Landmark clinical trials such as the Diabetes Prevention Program (DPP), Finnish Diabetes Prevention Study (DPS), and PREDIMED-Plus have provided robust evidence supporting the long-term efficacy of lifestyle-based prevention. The review further discusses mechanistic pathways linking lifestyle choices with glucose metabolism, insulin sensitivity, and inflammation. Public health strategies, such as community-based interventions and digital health platforms, are also considered, highlighting the need for multidisciplinary approaches. Preventive strategies require tailoring to sociocultural contexts and individual risk profiles to ensure adherence and long-term benefits. With the global rise in diabetes-related morbidity, mortality, and economic burden, prioritizing lifestyle interventions represents an urgent imperative for health systems worldwide.

KEYWORDS

Type 2 Diabetes Mellitus, Lifestyle Intervention, Diet, Physical Activity, Weight Loss, Prevention, Public Health

INTRODUCTION

Diabetes mellitus is a chronic metabolic disorder characterized by hyperglycemia due to impaired insulin secretion, insulin resistance, or both. Type 2 diabetes mellitus (T2DM) accounts for more than 90% of cases globally and is linked to rising rates of obesity, sedentary lifestyles, and unhealthy dietary habits[1]. According to the International Diabetes Federation (IDF), the global prevalence of diabetes was estimated at 537 million adults in 2021 and is projected to reach 783 million by 2045[2]. India and China alone contribute a large share of this burden, underscoring the global health emergency.

Preventing T2DM is of paramount importance, given its long-term complications—including cardiovascular disease, nephropathy, neuropathy, and retinopathy—that significantly impair quality of life and increase healthcare costs[3]. Pharmacological agents such as metformin and newer drugs (e.g., GLP-1 receptor agonists, SGLT2 inhibitors) have demonstrated efficacy in delaying diabetes onset, but their long-term use is limited by cost, adherence, and potential side effects[4]. Lifestyle interventions, by contrast, have consistently shown superior efficacy in reducing the risk of T2DM in high-risk individuals, with sustained benefits long after active interventions have ceased[5].

This review discusses the role of lifestyle modifications in T2DM prevention, with emphasis on dietary strategies, physical activity, weight loss, sleep hygiene, and stress management. Evidence from landmark studies and meta-analyses is summarized, followed by discussion of public health implications and future directions.

PATHOPHYSIOLOGY OF DIABETES AND ROLE OF LIFESTYLE FACTORS

The pathophysiology of T2DM involves a complex interplay of genetic, environmental, and behavioral factors. Insulin resistance in skeletal muscle, adipose tissue, and the liver is central to disease development, often accompanied by β-cell dysfunction[6]. Lifestyle factors—such as excess caloric intake, physical inactivity, chronic stress, and inadequate sleep—contribute to these pathophysiological processes.

- **Dietary excess** promotes adiposity, lipotoxicity, and chronic inflammation, impairing insulin signaling[7].
- **Sedentary lifestyle** reduces glucose uptake in muscle, decreases mitochondrial efficiency, and promotes insulin resistance[8].
- **Sleep deprivation** increases cortisol secretion and sympathetic nervous system activity, worsening insulin sensitivity[9].
- **Chronic stress** elevates inflammatory cytokines and dysregulates the hypothalamic–pituitary–adrenal axis, further aggravating glucose dysregulation[10].

Thus, targeting these modifiable lifestyle factors offers a rational, evidence-based approach for diabetes prevention.

DIETARY INTERVENTIONS

Caloric Restriction and Balanced Nutrition

Energy restriction has consistently been shown to improve insulin sensitivity and reduce diabetes risk. The Diabetes Prevention Program (DPP) demonstrated that intensive lifestyle modification targeting a 7% reduction in body weight reduced diabetes incidence by 58% over 3 years[11].

Mediterranean Diet

The Mediterranean diet—rich in olive oil, nuts, fruits, vegetables, legumes, and fish—has shown strong protective effects. The PREDIMED-Plus trial reported a 31% reduction in diabetes incidence with a calorie-controlled Mediterranean diet combined with behavioral interventions[12].

Low-Carbohydrate and Low-Glycemic Index Diets

Low-glycemic index diets slow glucose absorption and reduce postprandial hyperglycemia, improving insulin sensitivity[13]. Low-carbohydrate diets also demonstrate favorable short-term outcomes, though long-term sustainability remains debated[14].

Plant-Based Diets

Vegetarian and vegan diets, particularly those emphasizing whole grains, legumes, fruits, and vegetables, are associated with lower risk of diabetes[15]. High fiber intake enhances satiety, improves gut microbiota diversity, and reduces glucose excursions[16].

PHYSICAL ACTIVITY AND EXERCISE

Physical activity improves glucose uptake, increases muscle insulin sensitivity, and reduces adiposity. The American Diabetes Association recommends at least 150 minutes of moderate-intensity aerobic exercise weekly, combined with resistance training[17].

- **Aerobic exercise** (e.g., brisk walking, cycling, swimming) enhances cardiorespiratory fitness and glucose utilization.
- **Resistance training** improves lean body mass, basal metabolic rate, and long-term glycemic control[18].
- **High-intensity interval training (HIIT)** has emerged as an efficient strategy, with evidence suggesting significant improvements in insulin sensitivity and weight reduction in shorter timeframes[19].

The Finnish Diabetes Prevention Study found that participants engaging in ≥4 hours of moderate physical activity per week experienced a 63% risk reduction in diabetes onset[20].

WEIGHT MANAGEMENT AND OBESITY CONTROL

Obesity is the strongest modifiable risk factor for T2DM. Weight loss of even 5–10% has been shown to significantly reduce diabetes risk[21]. Sustained weight management requires a combination of dietary change, increased activity, and behavioral strategies.

Bariatric surgery has demonstrated dramatic reductions in diabetes incidence among severely obese individuals, with remission rates up to 75% in some studies[22]. However, surgery is not scalable for population-level prevention, reinforcing the importance of lifestyle interventions.

SLEEP AND STRESS MANAGEMENT

Inadequate sleep and chronic stress are often overlooked but important determinants of diabetes risk. Short sleep duration (<6 hours per night) increases risk of T2DM by 28% in meta-analyses[23]. Mind–body interventions such as yoga, mindfulness meditation, and cognitive behavioral therapy reduce stress and cortisol levels, thereby improving insulin sensitivity[24].

EVIDENCE FROM LANDMARK TRIALS

Inadequate sleep and chronic stress are often overlooked but important determinants of diabetes risk. Short sleep duration (<6 hours per night) increases risk of T2DM by 28% in meta-analyses[23]. Mind–body interventions such as yoga, mindfulness meditation, and cognitive behavioral therapy reduce stress and cortisol levels, thereby improving insulin sensitivity[24].

EVIDENCE FROM LANDMARK TRIALS

Several clinical trials provide strong evidence:

- **Diabetes Prevention Program (DPP, USA)**: Lifestyle modification reduced T2DM incidence by 58%, compared with 31% for metformin over 3 years[11].
- **Finnish Diabetes Prevention Study (DPS)**: Achieved 58% risk reduction with diet and exercise changes[20].
- **PREDIMED-Plus Trial (Spain)**: Mediterranean diet plus behavioral interventions reduced diabetes risk by 31%[12].
- **Da Qing Study (China)**: Long-term follow-up showed 43% risk reduction after 20 years[25].

These findings establish lifestyle modification as the gold standard for diabetes prevention across diverse populations.

PUBLIC HEALTH IMPLICATIONS

The global diabetes epidemic necessitates population-level strategies. Workplace wellness programs, community-based health promotion, and digital health platforms can increase reach and adherence[26]. Policies to reduce consumption of sugar-sweetened beverages and promote active transport also show promise[27].

Digital tools—such as smartphone applications, telemedicine, and wearable sensors—can provide personalized feedback and support, enhancing long-term adherence[28].

FUTURE DIRECTIONS

Emerging evidence suggests that personalized nutrition—guided by genetic, epigenetic, and microbiome profiles—may further enhance the effectiveness of lifestyle interventions[29]. Integrating digital health technologies, artificial intelligence, and community-level engagement can accelerate translation into practice[30].

CONCLUSION

Lifestyle interventions represent the cornerstone of type 2 diabetes prevention. Decades of research, including large randomized controlled trials and long-term cohort studies, have consistently demonstrated that modifying diet, increasing physical activity, achieving and maintaining weight loss, optimizing sleep, and managing stress can significantly reduce diabetes incidence across diverse populations and ethnic groups. Unlike pharmacological therapies, which primarily target isolated pathways and often carry side effects or adherence challenges, lifestyle changes address the root causes of diabetes by reversing insulin resistance, reducing systemic inflammation, and promoting metabolic resilience.

The evidence is clear: even modest improvements—such as losing 5–7% of body weight, walking briskly for 30 minutes daily, or adopting a Mediterranean-style diet—yield profound reductions in diabetes risk. Moreover, the benefits of lifestyle change extend beyond glycemic control, improving cardiovascular health, reducing cancer risk, and enhancing overall quality of life. Importantly, these strategies are cost-effective and scalable, making them particularly valuable in low- and middle-income countries where the diabetes epidemic is growing most rapidly.

Despite this strong evidence, significant barriers remain. Modern lifestyles are increasingly sedentary, driven by urbanization, technology use, and unhealthy food environments. Structural determinants—including socioeconomic status, cultural norms, and healthcare access—further complicate prevention efforts. To achieve meaningful impact, individual interventions must be embedded within broader public health frameworks that promote healthy environments, regulate unhealthy food marketing, expand access to recreational spaces, and incentivize preventive care.

Looking forward, the future of diabetes prevention lies in integration and personalization. Combining traditional lifestyle approaches with advances in digital health, artificial intelligence, and precision nutrition may allow for tailored interventions that enhance adherence and long-term sustainability. Community engagement, workplace wellness programs, and school-based education will be essential to instill healthy habits from an early age. In parallel, healthcare systems must shift from reactive, treatment-focused models toward proactive prevention-oriented care.

In summary, preventing type 2 diabetes through lifestyle modification is not only possible but also imperative. With global diabetes prevalence projected to continue rising, prioritizing lifestyle-based prevention strategies offers the most effective, sustainable, and holistic solution. By empowering individuals, reshaping environments, and leveraging technological innovation, the global health community has an unprecedented opportunity to curb the diabetes epidemic and secure a healthier future for generations to come.

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LIPIDOMICS AND CARDIOVASCULAR RISK PREDICTION: EMERGING INSIGHTS

Dr. Rahul Goyal, Dr. K.K. Arora, Dr. Anoop Kumar, Dr Jaya Tanwar

ABSTRACT

Cardiovascular disease (CVD) remains the leading cause of morbidity and mortality worldwide despite advances in diagnosis, treatment, and prevention. Traditional lipid markers such as total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides have long been used in risk prediction. However, these markers fail to fully capture the complex lipid biology underlying atherosclerosis and other cardiovascular disorders. Lipidomics, an advanced branch of metabolomics, enables comprehensive profiling of lipid species, offering deeper insights into disease mechanisms and personalized risk stratification.

Lipidomics utilizes mass spectrometry (MS) and nuclear magnetic resonance (NMR) spectroscopy to characterize thousands of lipid species, including phospholipids, sphingolipids, glycerolipids, and sterols. Recent studies demonstrate that specific lipid species, such as ceramides, lysophosphatidylcholines, and oxidized phospholipids, strongly correlate with incident CVD events, independent of conventional risk factors. Ceramide-based risk scores, for example, have shown superior predictive power compared to LDL-C alone.

This article reviews the principles of lipidomics, methodological advances, clinical evidence linking lipid species to cardiovascular outcomes, and integration of lipidomic biomarkers into predictive algorithms. Furthermore, we discuss challenges including standardization, cost, bioinformatics complexity, and translation into clinical practice. Emerging data suggest lipidomics has the potential to refine CVD risk stratification, identify therapeutic targets, and monitor treatment response in a personalized medicine framework.

KEYWORDS

Lipidomics, Cardiovascular Disease, Risk Prediction, Biomarkers, Ceramides

INTRODUCTION

Cardiovascular disease (CVD) accounts for nearly 18 million deaths annually, making it the leading cause of global mortality[1]. Despite the widespread use of lipid measurements such as LDL-C, HDL-C, and triglycerides in risk assessment, many individuals experiencing myocardial infarction or stroke exhibit normal lipid profiles[2]. This highlights the need for more precise biomarkers.
Lipidomics, a subfield of metabolomics, systematically studies the full complement of lipids in biological systems. With the aid of mass spectrometry and computational analytics, lipidomics enables the identification and quantification of thousands of lipid species[3]. By unraveling lipid metabolism in health and disease, lipidomics offers a new dimension for cardiovascular risk prediction.

TRADITIONAL LIPID MARKERS AND THEIR LIMITATIONS

For decades, LDL-C has been considered the primary therapeutic target for CVD prevention. Clinical trials consistently demonstrate that LDL-C lowering reduces CVD events[4]. HDL-C has also been viewed as cardioprotective, although recent evidence challenges its causal role[5].

However, reliance on these parameters has limitations. Many patients with well-controlled LDL-C still suffer adverse cardiovascular events, while others with elevated LDL-C remain free of disease[6]. This discrepancy underscores the need to move beyond traditional lipid panels.

PRINCIPLES OF LIPIDOMICS

Lipidomics aims to provide a comprehensive picture of lipid species and their interactions with proteins, metabolites, and genes. It encompasses:

- **Structural lipidomics**: identification of lipid classes (e.g., phospholipids, sphingolipids).
- **Functional lipidomics**: analysis of bioactive lipids in signaling pathways.
- **Clinical lipidomics**: translation into diagnostics and prognostics[7].

Key techniques include liquid chromatography–mass spectrometry (LC-MS), shotgun lipidomics, and NMR spectroscopy[8]. Advanced informatics pipelines facilitate integration of lipidomic data with clinical phenotypes.

LIPID CLASSES RELEVANT TO CARDIOVASCULAR DISEASE

Ceramides

Ceramides are sphingolipid metabolites involved in apoptosis, insulin resistance, and inflammation. Elevated plasma ceramide levels predict major adverse cardiovascular events (MACE), independent of LDL-C[9]. Ceramide-based risk scores (CERT1, CERT2) outperform conventional lipid measures in stratifying high-risk individuals[10].

Lysophosphatidylcholines (LPCs)

LPCs are generated during lipoprotein oxidation and promote endothelial dysfunction and inflammation[11]. Elevated LPCs have been associated with coronary artery disease and unstable plaques[12].

Sphingomyelins

Sphingomyelins influence membrane integrity and lipoprotein metabolism. Certain sphingomyelin species correlate with subclinical atherosclerosis and adverse events[13].

Oxidized Phospholipids (OxPLs)

OxPLs bound to apolipoprotein B particles are pro-inflammatory and pro-atherogenic. Elevated OxPLs predict coronary artery disease progression and events[14].

Triglyceride-rich Lipoproteins and Lipid Subfractions

Lipidomic profiling reveals heterogeneity among triglyceride-rich particles, with specific species conferring higher risk than others[15].

CLINICAL EVIDENCE LINKING LIPIDOMICS TO CVD

Population Studies

Large-scale cohort studies, including the FINRISK and Framingham cohorts, demonstrate that ceramides and sphingolipids improve prediction of myocardial infarction beyond traditional markers[16].

Risk Scores

Ceramide risk scores (CERT1 and CERT2) have been validated in multiple cohorts and incorporated into clinical practice in some European centers[17].

Therapeutic Monitoring

Lipidomic signatures can monitor response to statins, PCSK9 inhibitors, and dietary interventions[18]. For instance, reductions in ceramides correlate with statin-mediated risk reduction, suggesting utility as a therapeutic biomarker[19].

INTEGRATION INTO RISK PREDICTION MODELS

Current risk calculators (e.g., Framingham Risk Score, ASCVD Risk Estimator) rely on age, blood pressure, smoking, and basic lipids. Incorporating lipidomic markers significantly improves discrimination and reclassification[20]. Machine learning approaches further enhance predictive accuracy by integrating multi-omic data.

PRACTICE ADAPTATIONS AND CHALLENGES

Standardization

Heterogeneity in sample handling, lipid extraction, and MS techniques hampers reproducibility. Efforts are underway to establish international guidelines for lipidomic workflows.

Cost and Accessibility

Lipidomic profiling remains expensive and technologically demanding, limiting clinical adoption. Advances in automation and high-throughput MS may improve cost-effectiveness.

Data Complexity

Lipidomic datasets are vast and require advanced bioinformatics. Integration with genomics and proteomics adds further complexity.

Translation to Clinical Use

While ceramide scores are already in limited use, broader adoption requires validation across diverse populations, standardization, and demonstration of cost-effectiveness.

FUTURE DIRECTIONS

1. **Multi-omic Integration**: Combining lipidomics with genomics and proteomics for holistic risk assessment.
2. **Point-of-Care Lipidomics**: Development of rapid, bedside assays for ceramides and other lipids.
3. **Therapeutic Targeting**: Identifying lipid-modifying interventions beyond LDL-C, such as ceramide synthase inhibitors.
4. **Personalized Medicine**: Tailoring interventions based on individual lipidomic signatures.

CONCLUSION

Lipidomics represents a transformative advance in cardiovascular risk prediction. By moving beyond traditional lipid parameters, lipidomics identifies novel biomarkers such as ceramides, LPCs, and OxPLs that provide superior prognostic information. Emerging risk scores demonstrate the clinical potential of lipidomics to improve patient stratification and therapeutic monitoring.

Nonetheless, challenges remain in terms of standardization, accessibility, and clinical translation. Future research integrating lipidomics with genomics, proteomics, and advanced analytics promises to deliver precision cardiovascular medicine. Ultimately, lipidomics is poised to refine prevention, diagnosis, and treatment of cardiovascular disease, reducing the global burden of morbidity and mortality.

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* Proposed