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Review Article

Impact of systemic diseases on healing outcomes on facial trauma patients

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ABSTRACT

Facial trauma is considered a major public health issue. It is mainly caused by road traffic accidents, falls, and sports injuries. Facial trauma is associated with various functional and aesthetic adverse outcomes. The epidemiology of facial injuries differs globally, with variations in prevalence, causes, injury patterns, severity, and clinical outcomes. Facial trauma healing involves wound and fracture healing. Factors affecting healing include age, weight, and comorbidities. Systemic diseases are major determinants of the healing of injuries in various parts of the body. However, studies investigating their effects on facial trauma healing are scarce. This review aims to discuss how systemic diseases affect facial trauma healing. Diabetes induces some systemic alterations, including hyperglycemia and peripheral arterial disease, that can significantly delay healing. Chronic kidney disease delays healing through various mechanisms, including delayed granulation, disruption of keratinization kinetics, tissue edema, and large epithelial gaps. Diabetes also may impair fracture healing as a result of the elevated concentration of TNF-α at the fracture site and elevated osteoclasts in the diabetic callus. While the impact of calcium and vitamin D on fracture healing has been debatable. Although systemic diseases have a significant impact on trauma in various parts of the body, evidence on their impact on the healing outcomes of facial trauma specifically is still lacking. Thus, further research should focus on this specific topic.

Keywords: Facial trauma, Facial trauma healing, Wound healing, Fracture healing, Healing outcomes, Systemic diseases, Diabetes mellitus, Cardiovascular diseases

INTRODUCTION

Facial trauma is a serious health issue, with 25% of patients with severe injuries having concomitant facial injuries.[1] It has been associated with long-term negative influence on quality of life. Facial trauma is always accompanied by serious life-threatening injuries to the head and chest and may require many constructive and plastic surgeries with prolonged hospital stays.[2,3] Causes of facial trauma include road traffic accidents, falls, sport injuries, assaults, and

animal-related injuries, and industrial injuries.[3,4] The epidemiology of facial injuries differs globally, with variations in prevalence, causes, injury patterns, severity, and clinical outcomes influenced by the socio-economic status and cultural background of the population.[5,6]

Facial trauma healing can be divided into wound healing and fracture healing. Wound healing is a process through which four main phases occur: hemostasis, inflammation, proliferation, and dermal remodeling, resulting in restoration of function.[7,8] Wound healing starts with hemostasis that involves rapid contraction of blood vessels and formation of a blood clot preventing exsanguination from vascular damage.[9] Platelets play a key role in the processes of hemostasis and coagulation. Platelets and thrombin activation lead to the formation of an insoluble clot (eschar), consisting of fibrin, fibronectin, vitronectin, and thrombospondin.[10] The primary functions of the eschar are plugging the wound and preventing bleeding. Secondary functions include providing a scaffold for incoming immune cells, protecting against bacterial invasion, and harboring a reservoir of cytokines and growth factors to guide the behavior of wound cells in early repair.[11]

Fracture healing is usually initiated by secondary healing, as primary healing, which is characterized by direct bone formation, requires absolute stability to be initiated.[12] Secondary healing leads to the formation of bony bridging of the fracture gap through intramembranous and endochondral ossification.[13] Intramembranous ossification is induced by osteoblastogenesis that occurs in the periosteum. Osteoblasts directly deposit osteoid, which then mineralizes into bone.[13] Endochondral ossification includes chondrogenesis via cell differentiation, formation of a cartilaginous callus bridging the fracture, mineralization and expansion of the immature callus, and remodeling by osteoblasts and osteoclasts.[14]

The process of fracture healing typically starts with inflammation and the formation of hematoma at the fracture site, which induce the release of cytokines and different immune cells. These mechanisms activate the recruitment and migration of mesenchymal stem cells from the periosteum and endosteum to the fracture site.[14]

Multiple studies reported that various intrinsic and extrinsic factors have a significant impact on trauma healing.[12,15] Intrinsic factors include injury to the periosteum or endosteum and poor vascularization at the injury site, while extrinsic factors include systemic diseases, medications, and social habits, such as alcohol and smoking. Healing injuries can be significantly impaired by systemic diseases, including malnutrition, diabetes mellitus, and cardiovascular diseases. Nevertheless, their effect on the healing outcomes of facial trauma is unclear.

The review aims to explore current evidence focusing on the impact of systemic diseases on the healing of facial trauma, highlighting the impact on wound healing and fracture healing.

METHODS

A comprehensive literature search was conducted in Medline (via PubMed), Scopus, and Web of Science databases up to 24 July 2025. Medical Subject Headings (MeSH) and relevant free-text keywords were used to identify synonyms. Boolean operators (AND, OR) were applied to combine search terms in alignment with guidance from the Cochrane Handbook for Systematic Reviews of Interventions. Key search terms included: “facial trauma” OR “facial injury” OR “facial fracture” OR “facial wound” AND “healing” OR “wound healing” OR “fracture healing” OR “healing outcomes” AND “systemic diseases” OR “comorbidities” OR “diabetes mellitus” OR “cardiovascular diseases”. Summaries and duplicates of the found studies were exported and removed by EndNote X8. Any study that discusses the impact of systemic diseases on healing outcomes in facial trauma patients and is published in peer-reviewed journals was included. All languages are included. Full-text articles, case series, and abstracts with related topics are included. Case reports, comments, and letters were excluded.

DISCUSSION

Facial trauma: nature and types

Facial trauma is one of the most encountered traumas in radiology, even outside level 1 trauma centers. Facial trauma is mainly caused by falls, motor vehicle accidents, sports, fights, and assaults.[16] It may be distributed symmetrically or asymmetrically and may involve a single bone or multiple bones. Fractures of the midface are the most common, followed by fractures of the lower face (mandible) and the upper face (frontal bone and superior orbital rim).[16] Imaging, especially cone beam computed tomography (CBCT) and multidetector computed tomography (MDCT), plays a key role in diagnosis, classification, and treatment planning of facial trauma.[17,18] The facial skeleton comprises horizontal and vertical buttresses that absorb impact and guide reconstructive efforts (Figure 1).[23]

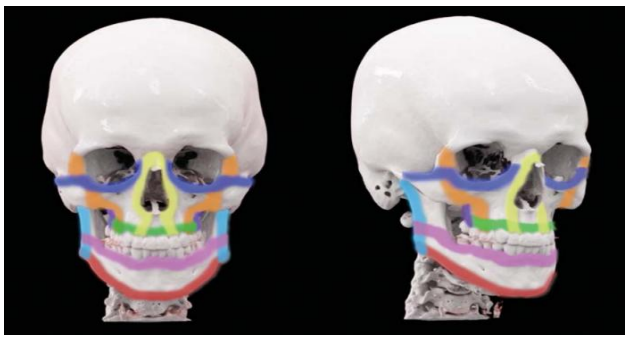

Figure 1: Vertical and horizontal buttresses of the face skeleton.[23]

Buttresses are structural zones of dense bone that absorb impact and guide reconstructive efforts. The horizontal

buttresses include the upper transverse maxillary buttress, the lower transverse maxillary buttress, the upper transverse mandibular buttress, and the lower transverse mandibular buttress, while the vertical buttresses include the medial maxillary buttress, the lateral maxillary buttress, the posterior maxillary (pterygomaxillary) buttress, and the posterior vertical (the posterior delineation of the ramus mandibulae) mandibular buttress.[17,18]

Nasal fracture is the most common isolated fracture of the face due to its protrusion. Nasal bone fracture can be diagnosed physically, and an X-ray may be performed to confirm the diagnosis. X-ray is also performed for medicolegal issues. Septal hematoma is a potential complication requiring radiologic awareness.[18,19] The mandible is a commonly fractured bone due to its location and its relative lack of support. Mandibular fracture accounts for 36–59% of maxillofacial injuries. It may occur in one location (50%), two locations (40%), or more than two locations (10%).[16] Various classifications for mandibular fractures are available, and the most common classification is based on the anatomical location of the fracture: coronoid process, condyle, body, angle, ramus, alveolar crest, and symphysis mentalis.[18,20] Condylar fractures, often displaced, are categorized as intracapsular (condylar head) or extracapsular (neck, subcondylar).[18-21]

Furthermore, facial trauma can lead to naso-orbitoethmoid fractures, zygomaticomaxillary complex fractures, Le Fort fractures, and orbital fractures. One of the most common facial traumas is dental trauma, affecting about one-third of individuals during their lifetime.[22] Typically, it presents as a fracture, luxation, or both. Dental fractures always affect tooth structure and may worsen pulp vitality, while dentoalveolar fractures involve the alveolar process and usually accompany luxation. Luxation injuries typically involve the periodontal ligament and include avulsion, subluxation, and concussion. Initial assessment of dental trauma should include periapical radiographs; however, CBCT is preferred in this type of trauma.[20-22]

Horizontal buttresses: the upper transverse mandibular buttress (pink), the upper transverse maxillary buttress (dark blue), the lower transverse maxillary buttress (green), and the lower transverse mandibular buttress (red). Vertical buttresses: the lateral maxillary buttress (orange), the medial maxillary buttress (yellow), the posterior vertical mandibular buttress (light blue), and the posterior or pterygomaxillary buttress (purple).[16]

Systemic diseases impact on wound healing

Multiple factors can affect wound healing; these factors are listed in Table 1.[16] Over a hundred alterations that occur in wound healing in diabetic patients have been identified in all phases of healing.[24] The hemostatic state of diabetes mellitus is characterized by hormonal dysregulation, impaired platelet activation, reduced neuropeptides, and irregular molecular signaling and release.[25] During the inflammatory phase, diabetes significantly affects cell migration and activation, while during the proliferative phase, it significantly affects angiogenesis, collagen deposition, granulation tissue formation, and epithelialization.[26] Additionally, matrix metalloprotease activity and collagen turnover are altered during maturation.

Systemic alterations associated with diabetes, including hyperglycemia, peripheral arterial disease, malnutrition, worsening renal function, and the high risk of reinjury associated with peripheral neuropathy, can also significantly delay healing.[25] For instance, hyperglycemia induces enzyme and protein dysfunction, affecting cell and nutrient delivery by altering basement membranes. Furthermore, vascular diseases lead to local hypoxia, compromising angiogenesis.[27] Diabetic patients have fewer, less active macrophages and increased antiangiogenic factors, impeding granulation and epithelialization.[28] Chronic kidney disease (CKD) can significantly delay healing through various mechanisms, including delayed granulation, disruption of keratinization kinetics, tissue edema, and large epithelial gaps.[25] CKD is associated with chronic inflammation, diminished angiogenesis, and cell proliferation, which also remarkably impairs wound healing.[29] Additionally, CKD-induced uremia impairs fibroblasts, hydroxyproline, and collagen formation. Although this impairment can be improved by dialysis, dialysis can cause nutrient losses, further worsening healing.[29]

Malnutrition can significantly affect healing, since protein and energy substrates contribute to all phases of healing.[30] Deficiency of nutrients, electrolytes, minerals, and vitamins can also impair the process of healing. For instance, arginine contributes to immune, endocrine, and endothelial functions essential for wound healing. Zinc, iron, and vitamin C enhance collagen stability and immune response.[15,30,31]

Furthermore, obesity and aging have a significant effect on wound healing. Obesity is associated with complications, including hematoma and seroma formation, dehiscence, infection, and pressure and venous ulceration, all of which impair wound healing.[15] Aging is associated with reduced fibroblast and collagen density and increased elastin fragmentation, delaying healing and decreasing skin strength and elasticity.[32] Notably, after the age of 40, healing time doubles.[32] Hormonal changes, particularly reduced estrogen in men, may also impair healing in older adults.[15]

Systemic infection impairs healing, as it leads to inflammation, which in turn delays the proliferative and remodeling stages. Bacteria can also destroy collagen by secreting collagenases, resulting in chronic wounds, necrosis, or sepsis. Surgical site infections increase the risks of hernia and anastomotic failure, yet prevention strategies remain inconclusive and under investigation.[33]

Table 1: Factors affecting wound healing.[16]

Category	Example
Genetic	Inborn errors of collagen synthesis: Ehlers-Danlos, Marfan syndrome, osteogenesis imperfecta, epidermolysis bullosa
Systemic	Metabolic: diabetes mellitus, chronic renal failure, nutritional: malnutrition, obesity, vitamin deficiencies, cardiovascular: chronic obstructive pulmonary disease, congestive heart failure, inflammatory/autoimmune: vasculitis, rheumatoid arthritis, systemic lupus erythematosus, scleroderma, granulomatosis with polyangiitis (previously Wegener), polyarteritis nodosa, cryoglobulinemia, Raynaud disease, pyoderma, and environmental/toxin: smoking, alcohol, drugs of abuse
Infectious	Bacterial
Post-therapy	Chemotherapy, radiation, and drugs: steroids, nonsteroidal anti-inflammatory drugs

Systemic disease impact on fracture healing

Diabetes mellitus increases the risk of fractures and impaired fracture healing.[34,35] Multiple studies reported increased risk of fractures, fracture malunion, and reoperation among diabetic patients; however, this increased risk is not explained by lower bone mineral density levels, particularly with type 2 diabetes patients.[34,36] The impairment of fracture healing in diabetes can be attributed to the elevated concentration of TNF-α at the fracture site and elevated osteoclasts in the diabetic callus.[37] Studies investigating fracture healing in diabetic rodents showed inconsistent results. Kayal et al reported elevated osteoclastogenesis and chondrocyte apoptosis in type 1 diabetic mice. These deficits decreased endochondral bone formation and enhanced cartilage loss. Notably, they were reversed by insulin administration.[38] On the other hand, Hu et al reported that increased glucose levels were associated with decreased osteoclast activity and osteoclastogenesis in type 2 diabetic rats.[39]

Mangialardi et al investigated fracture healing in diabetic humans and reported dysfunction of CD146+ pericytic cells, leading to a reduction in the cellular and vascular supply at the fracture site.[40] Notably, the utilization of an intramedullary delivery system for the local administration of insulin at the fracture site has improved fracture healing in both diabetic and nondiabetic rats.[41] Furthermore, systemic insulin and vitamin D3 treatment managed to reverse delayed fracture healing in female type 1 diabetic mice, possibly due to increased IGF-1 production in the fracture callus.[42]

The impact of calcium and vitamin D deficiencies on fracture healing has been conflicting in previous animal studies.[43,44] A previous study found that various types of diet, including one deficient in calcium, phosphorus, and vitamin D, significantly impaired fracture healing.[43] Other studies showed that calcium- and phosphorus-deficient, vitamin D-deficient, or calcium- and vitamin D-deficient diets have no effect on fracture healing.[44] Vitamin D deficiency has been associated with impaired fracture healing. A case series found that more than 80% of vitamin D-deficient patients experienced non-unions. On the other hand, a previous study found no significant differences in vitamin D levels between patients with non-unions and those with normal healing.[45,46] Furthermore, vitamin C deficiency, vitamin B6 deficiency, and hypoalbuminemia from low dietary protein intake have been associated with impaired fracture healing, even though all of them are supported by low levels of evidence.[47-49]

Future implications and recommendations

Although it has been emphasized that systemic diseases have a significant impact on healing outcomes of fractures and wounds in various parts of the body, evidence on their impact on healing outcomes of facial trauma specifically is still lacking. It is critical to conduct large-scale clinical studies that investigate the effects of systemic diseases, such as diabetes mellitus and cardiovascular diseases, on facial fractures and wound healing. These studies should focus on estimating various outcomes, including long-term function, incidence of complications, aesthetics, and time-to-union. Cellular and molecular mechanisms through which systemic diseases affect healing should also be investigated. This can be done by mechanistic research, which can develop preclinical models to address this issue. Studies should also evaluate the influence of systemic disease management on facial trauma healing outcomes. Therapies, such as regenerative medicine, local insulin application, and vitamin D supplementation, showed promise in improving healing outcomes. Future studies should also investigate possible biomarkers for impaired healing of facial trauma caused by systemic diseases. This may facilitate the evaluation of health trajectory, risk assessment, and tailored therapeutic strategy.

CONCLUSION

Systemic health plays a critical yet underrecognized role in the healing of facial trauma. Conditions such as diabetes, malnutrition, cardiovascular disease, and chronic kidney disease can significantly impair both wound and fracture healing, leading to prolonged recovery and increased complication rates. To date, no studies specifically evaluating the impact of systemic diseases on the healing outcomes of facial trauma were identified in the literature. Given the complex interplay between systemic disease and facial tissue repair, further research and multidisciplinary

approaches are essential to improve outcomes and tailor treatment strategies for affected patients.

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