REVIEW ARTICLE

CONTENTS

Complications of immediate-loading dental implants: Current insights into etiology and pathogenesis

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ABSTRACT

Aim: To analyze the scientific literature on the etiology and pathogenic mechanisms underlying complications arising from dental implantation under immediate loading protocols and to outline promising approaches to their prevention and management.

Materials and Methods: A review of the scientific literature addressing the complex mechanisms underlying potential complications of dental implantation under immediate loading protocols was conducted.

Conclusions: The literature suggests that the primary causes of early dental implant failure include compromised mechanical stability during the critical phase of bone remodeling, inadequate seal control, and violation of the biological width. Soft tissue thickness deficiency (thin biotype) or the absence of keratinized mucosa triggers pathological bone destruction, which, in turn, facilitates infection and inflammation, further exacerbating destructive changes. These mechanisms are interrelated and mutually reinforce one another.

The findings underscore the need to develop protocols for guided implant therapy in high-risk patients, to implement clear algorithms for objective monitoring of implant stability at all stages of treatment, and to identify the optimal sealing material. These measures have the potential to improve approaches to the fixation of prosthetic restorations.

KEY WORDS: dental implantation, biomechanical, biological, infectious inflammatory processes, immediate loading

Wiad Lek. 2026;79(4):877-883. doi: 10.36740/WLek/219915 DOI

INTRODUCTION

In recent decades, alongside the two-stage protocol, immediate loading with simultaneous healing abutment placement has been increasingly adopted in dental implantology, driven by the need to minimize rehabilitation time and to optimize aesthetic outcomes [1, 2]. At the same time, inadequate dental care results in dysfunction of the stomatognathic system, disorders of the digestive and respiratory systems, and the development of aesthetic defects, ultimately leading to social and psychological maladaptation in patients. Consequently, the steadily increasing number of forensic medical examinations related to dental service quality, together with the associated litigation risk, contributes to stress and burnout among dentists [3]. Despite the high success rates of dental implant osseointegration, early and delayed complications remain a significant concern [4]. Unlike the delayed protocol, in which the implant is isolated from external forces during the osseointegration phase, immediate loading transforms the surgical site into a dynamic system, where even minimal disturbance to equilibrium can lead to adverse outcomes [5].

AIM

To analyze the scientific literature on the etiology and pathogenesis of complications arising from dental implantation under immediate loading protocols and to examine key aspects of their prevention and management.

MATERIALS AND METHODS

A review of the scientific literature on the etiology and pathogenesis of complications arising from dental implantation under immediate loading protocols was conducted. Selected studies from the 1980s and 1990s

were included to provide historical context regarding past successes and failures, while the literature published between 2000 and 2024 was analyzed for insights into the complex mechanisms underlying potential complications.

REVIEW

According to the literature, these causes can be classified as *biomechanical* (loss of primary stability), *biological* (impaired osseointegration and altered biological width), and *infectious-inflammatory* (peri-implantitis) [6].

Biomechanical factors associated with implant failure under immediate loading. Primary (mechanical) stability is achieved solely through the macromechanical retention of the implant threads within the trabecular bone and cortical plate and is maximal at the time of placement. Secondary (biological) stability arises from new bone formation on the titanium surface. Between these phases, a transitional period known as the "stability dip" occurs, typically between the third and fifth postoperative weeks [7].

Compression of bone tissue required to achieve high insertion torque (35–45 Ncm) leads to local ischemia and osteocyte micronecrosis at the bone–implant interface [8]. In response, a remodeling process is initiated, during which osteoclasts resorb the traumatized bone tissue supporting the dental implant. During the interval between extensive resorption of intact bone tissue and the presence of newly formed, insufficiently mineralized bone, the overall stability of the system reaches its minimum. At this stage, masticatory forces during immediate loading are transmitted to the weakened remodeling zone. Between the third and fourth postoperative weeks, the risk of implant loss is highest, as mechanical support has been lost, and biological stability has not yet developed [9]. Excessive compression (over 60–70 Ncm) can lead to pathological bone resorption by compressing the Haversian canal vessels [2]. When the pressure on the implant bed walls exceeds capillary blood pressure (approximately 30–40 mmHg), ischemia occurs, and if it persists, osteocytes die from hypoxia [10]. This is particularly critical in cortical bone (D1 type) due to its low vascularity; therefore, adherence to a thread-tapping protocol or the use of implants with active, knife-edge threads, rather than compressive threads, in dense bone is an essential preventive measure [11]. Indeed, surgical complications under immediate loading correlate with bone type. In D3 and D4 bone (predominantly in the posterior maxilla), achieving the required insertion torque is challenging due to low trabecular density, which provides inadequate rigidity for fixation. Consequently, the "stability dip" is more pronounced and prolonged in these patients. The incidence of early implant failure under immediate loading in D4 bone is three to four times higher than in D1/D2 bone [2].

In immediate implantation into an extraction socket, the most common error is positioning the implant in the center of the socket rather than toward the palatal side. This often results in negative outcomes, as the buccal bone wall may be less than 2.0 mm thick or entirely absent. The buccal bone plate is a tooth-dependent structure that undergoes resorption after tooth extraction. Consequently, if an implant is positioned too far buccally, it will inevitably become exposed during the remodeling process. To achieve a stable and esthetic outcome, buccal bone thickness should be at least 2.0 mm [12].

During this period, micromotion amplitude is critical. Micromotions within the 30–50 μm range are within physiological limits and may even stimulate osteogenesis via the piezoelectric effect. However, maintaining this range in clinical practice is extremely challenging, particularly when removable prosthetic restorations or single crowns are used [13]. A micromotion threshold of 100–150 μm is generally accepted as the upper limit for successful dental implant osseointegration [14]. If micromotion within the socket exceeds 150 μm under occlusal loading, neoangiogenesis is disrupted, and fibrin fibers, which serve as a matrix for osteogenic cell migration, are ruptured. Under these conditions, multipotent mesenchymal cells differentiate into fibroblasts rather than osteoblasts, forming a connective tissue capsule. Clinically, this presents as implant mobility without signs of purulent inflammation (aseptic fibrous integration) 6–8 weeks postoperatively [15].

Regardless of the protocol, dental implant placement elicits a foreign body reaction: osseointegration represents a protective host response in which the body attempts to isolate the titanium implant by forming a bony barrier ("shielding off") around it [16]. In successful cases, an immunological equilibrium is achieved; however, under immediate loading, this equilibrium is only relative. Overall, mechanical stress and micromotion activate the pro-inflammatory M1 macrophage phenotype. Rather than transitioning to the reparative M2 phenotype (which promotes osteoblast activity), these cells continue to secrete pro-inflammatory cytokines, thereby sustaining chronic inflammation. Consequently, peri-implantitis is not regarded as a distinct disease but rather as a disruption of the foreign body equilibrium that progresses to implant rejection [17].

Osteotomy site preparation generates frictional heat. Heating the bone to 47 °C for 1 minute induces irreversible protein denaturation (particularly alkaline phosphatase), vascular coagulation, and osteocyte necrosis [18]. The risk of thermal necrosis increases when using guided surgical templates, as temperatures in the apical region of the implant osteotomy can reach 50–55 °C, thereby inducing protein denaturation [19]. Thermal shock inhibits the expression of heat shock protein 70 (HSP70), leading to bone necrosis around the implant. By the fourth to fifth postoperative week, as the body begins to sequester necrotic tissue, the implant loses stability and fails. Under immediate loading, this process is accelerated by micromotion [8].

The direction of loading is critical. Axial (vertical) forces are well tolerated by the bone and dental implant, promoting bone compaction. In contrast, horizontal (lateral) forces arising from premature contacts or steep cusp inclines of temporary crowns generate destructive bending moments in the crestal region of the implant. Under immediate loading, the cortical plate at the implant neck experiences the highest stress. If this area is thin (<1 mm) or damaged during tooth extraction, crater-like bone resorption may occur. Marginal bone loss of 1–2 mm compromises esthetics and reduces bone–implant contact, thereby reducing the system's overall load-bearing capacity [13].

Impaired osseointegration and biological width violation. A fundamental challenge of any two-piece implant system is the inability to achieve a hermetic seal between the implant and abutment, as microgaps inevitably form at the interface. The size of these microgaps can range from 1 to 49 µm, depending on the connection type (flat-to-flat or conical) and the precision of casting or milling of the prosthetic components [20]. Occlusal forces applied to the healing abutment induce elastic deformation of the abutment screw and micromotion at the implant–abutment interface. This phenomenon is referred to as the micro-pumping effect [21]. Compared to flat-to-flat connections, conical connections demonstrate significantly greater mechanical stability and resistance to microleakage. Owing to the cold-welding effect and frictional fixation, Morse taper connections minimize abutment micromotion, thereby reducing the micro-pumping effect [22]. In cases of subcrestal placement of implants with flat-to-flat connections, bone resorption continues until the implant platform is exposed or the bone level is stabilized 1.5–2.0 mm below the implant–abutment interface. This results in the formation of a crater-like defect that serves as a niche for plaque accumulation and further progression of peri-implantitis [23]. The concept of platform switching, involving the use of an abutment with a smaller diameter than the implant platform, shifts the inflammatory cell infiltrate horizontally toward the implant's central axis and has been shown to significantly reduce vertical bone loss [24]. However, even with platform-switched conical connections, effective bacterial sealing cannot be guaranteed, particularly under immediate loading, when achieving final screw torque is limited by the risk of implant rotation. Under these conditions, the internal chamber of the implant becomes an ideal anaerobic incubator for the most virulent "red complex" pathogens [25]. Hollow spaces within the implant have been shown to harbor up to 10^8 colony-forming units (CFUs) of bacteria [26].

Continuous release of bacterial endotoxins through the microgap induces a chronic inflammatory response in the surrounding tissues. The inflammatory infiltrate, in turn, serves as a source of pro-inflammatory cytokines – interleukin-6 (IL-6), interleukin-1 beta (IL-1β), and tumor necrosis factor alpha (TNF-α) – which stimulate the expression of receptor activator of nuclear factor kappa-B ligand (RANKL) on osteoblasts [27]. Osteoclastogenesis is subsequently activated, with osteoclasts resorbing bone tissue to remove it from the site of infection, thereby establishing the biologic width. Vitamin D exerts anti-inflammatory effects by suppressing the production of IL-1, IL-6, and TNF-α, reducing RANKL expression, and inhibiting osteoclastogenesis. In patients with vitamin D deficiency (<20 ng/mL), the risk of early implant loss increases by 140% [28].

The transmucosal healing abutment is placed into a fresh post-extraction socket or a prepared surgical site. A persistent clinical challenge remains the transition zone between the natural gingival attachment and the peri-implant soft tissue interface. In natural dentition, collagen fibers insert perpendicularly into the root cementum, whereas the titanium surface induces the formation of parallel-oriented fibers [29]. This "biological cuff" is characterized by a reduced fibroblast count and inadequate vascularization. Around zirconia abutments, a structure more closely resembling the natural tooth is formed, with a portion of the collagen fibers oriented perpendicularly [30]. The use of temporary plastic crowns or standard titanium healing abutments with microrough surfaces during immediate loading carries specific risks. Since fibroblast adhesion to polished titanium is reduced, surface roughness promotes biofilm accumulation, occurring as early as 30 minutes after surgery, and bacterial invasion can trigger an inflammatory response, leading to marginal bone resorption and gingival recession [31]. By positioning

the implant platform deep subgingivally (typical in the esthetic zone), complete removal of excess cement becomes virtually impossible. Residual cement acts both as a rough surface promoting biofilm formation and as a toxic agent, eliciting a pronounced inflammatory response when in contact with connective tissue [32]. Therefore, the current gold standard for prevention is the use of screw-retained restorations or customized abutments, in which the cementation margin is positioned at the gingival level.

The role of soft tissue thickness. A vertical gingival thickness of 2.0 mm or less triggers inevitable bone resorption of approximately 1.5 mm, regardless of the implant platform position (supracrestal or epicrestal) [33]. Evidence suggests that thin soft tissues fail to provide adequate vascular supply to the interface region and that peri-implant mucosal ischemia leads to marginal necrosis of the flap, wound dehiscence, and exposure of the cover screw or healing abutment. Compromised vascularization leads to an ineffective immune response, facilitating early pathogen colonization of the implant surface even before the healing process is complete [34]. The temporary crown serves as a matrix for soft-tissue contouring. A concave abutment design allows for increased connective tissue volume, whereas a convex emergence profile results in tissue compression, thinning, and eventual recession [35]. The absence of keratinized mucosa (<2 mm) significantly correlates with higher plaque indices, bleeding on probing, and patient discomfort during oral hygiene procedures [36].

Infectious inflammatory complications (peri-implantitis). Early complications under immediate loading result from a breakdown in the immune system's tolerance to the titanium implant, with bacteria acting merely as triggers, while tissue destruction is primarily mediated by the host's own cells. Bone resorption is an immune-mediated process, and the success of implantation depends on whether the titanium surface is first colonized by host cells (fibroblasts/osteoblasts) or by bacteria [37]. As early as 2 weeks after implantation, the implant environment is colonized by *P. gingivalis* and T. *forsythia*, even in patients with clinically healthy periodontal tissues. The "red complex", including *Porphyromonas gingivalis*, *Tannerella forsythia,* and *Treponema denticola*, is of particular concern. *P. gingivalis* acts as a keystone pathogen; using its enzymes, it does not merely degrade tissues but suppresses the immune system, specifically the complement system, thereby facilitating the unimpeded proliferation of other bacterial species. Furthermore, *P. gingivalis* can invade epithelial cells, thereby evading the effects of antibiotics [25]. Given that the average size of oral pathogens (such as *P. gingivalis* and *T. denticola*) ranges from 0.5 to 1.0 μm, while their toxins and pro-inflammatory mediators are measured in nanometers, even a minimal microgap creates favorable conditions for bidirectional exchange of fluids and bacteria between the internal environment of the implant and the surrounding tissues. Fungal infection represents a critical component of the pathological process that threatens the integrity of the implant-supporting bone. Modern clinical *Candida* strains, isolated from patients with oral diseases, exhibit high resistance to conventional antifungal agents. This complicates the management of peri-implant infectious complications and necessitates the development of novel antimycotics [38].

DISCUSSION

An analysis of the literature on the biomechanical causes of complications in dental implantation performed according to immediate loading protocols indicates that there are no clear data on the correlation between radiographic bone density and its metabolic status, a consideration essential for predicting a "stability dip." Therefore, the development of an index integrating computed tomography parameters with blood biochemical markers, specifically serum alkaline phosphatase and osteocalcin levels, appears promising. Such an approach would facilitate the identification of patients with predominant bone resorption relative to bone formation, thereby justifying their exclusion from the standard loading protocol.

According to the literature, microgaps at the implant–abutment interface represent a persistent source of bacterial leakage, which is further exacerbated by the micro-pumping effect during chewing. Therefore, the development of a protocol for intraoperative sealing of the implant's internal cavity is warranted. However, the optimal choice of sealing material remains unresolved. Accordingly, comparative clinical and microbiological studies are needed to assess the efficacy of silicone or antiseptic-loaded matrices in both sealing the implant chamber and reducing the load of "red complex" periodontal pathogens in the peri-implant sulcus.

Bone loss results from the immune response to foreign bodies and biofilms, leading to osteoimmunology imbalance regulated by the RANKL–OPG (osteoprotegerin) system. Therefore, factors such as hypovitaminosis, vitamin D deficiency, and metabolic disorders that may compromise osseointegration should be considered. Moreover, the potential use of agents that inhibit proinflammatory cytokine

synthesis and reduce osteoclast activity in the early postoperative period warrants further investigation.

According to current evidence, soft tissue thickness deficiency is critical for marginal bone stability. Conventional augmentation techniques are often traumatic and may increase the risk of infection. The use of platelet-rich fibrin (PRF) membranes requires further investigation to assess the effects of growth factors on the rate of socket healing and the formation of a "biological cuff" around the abutment.

CONCLUSIONS

An analysis of the literature indicates that complications in dental implantation under immediate loading protocols are multifactorial, with biomechanical, biological, and infectious inflammatory factors acting as interrelated pathogenetic links that frequently overlap and exacerbate tissue disintegration. Accounting for these factors can facilitate the development of protocols that incorporate an individualized approach at various stages of implantation.

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This work was conducted within the research program of the Department of Surgical Dentistry, Ivano-Frankivsk National Medical University: "Comprehensive Surgical Preparation Methods for Patients Undergoing Prosthetic Rehabilitation Using Osteoplasty and Implantation Techniques, Considering Inflammatory and Dystrophic Processes in the Maxillofacial Region" (0121U113110).

CONFLICT OF INTEREST

The Authors declare no conflict of interest

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RECEIVED: 18.09.2025
ACCEPTED: 13.01.2026

CREATIVE COMMONS 4.0