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Osseointegration challenges of dental implants in dialysis-dependent chronic kidney disease patients

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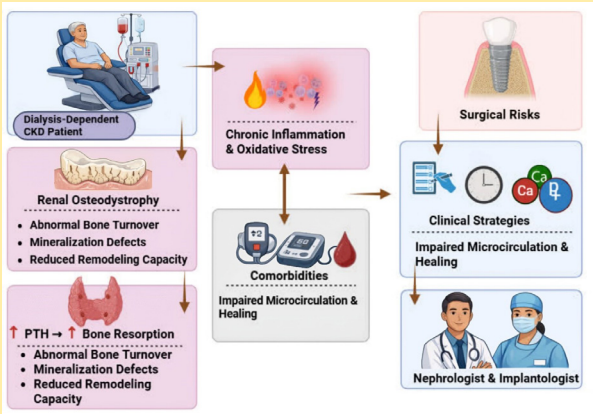
ABSTRACT



Dental implant therapy in dialysis-dependent chronic kidney disease (CKD) patients presents significant osseointegration challenges due to systemic metabolic disturbances inherent to end-stage renal disease. Renal osteodystrophy, characterized by abnormal bone turnover, mineralization defects, and secondary hyperparathyroidism, critically compromises alveolar bone quality and remodeling capacity essential for implant stability. Elevated parathyroid hormone levels induce excessive bone resorption, while hyperphosphatemia and hypocalcemia disrupt hydroxyapatite crystallization, impairing the bone-implant interface formation. Uremic toxins accumulate despite dialysis, inducing chronic inflammation and oxidative stress that suppress osteoblast activity and angiogenesis while promoting osteoclastogenesis. Additionally, frequent comorbidities, including diabetes, hypertension, and anemia further compromise microcirculation and tissue healing. Anticoagulant use during hemodialysis increases perioperative bleeding risks, potentially disrupting early clot formation and cellular migration at the surgical site. Although dental implants remain a viable rehabilitation option for edentulous CKD patients, success rates are generally lower compared to healthy populations, with higher incidences of marginal bone loss and late failures. Careful patient selection, optimized dialysis timing around surgery, stringent control of calcium-phosphate products and parathyroid hormone levels, and also extended healing periods are recommended clinical strategies. Limited long-term prospective studies specifically addressing implant outcomes in this population highlight an evidence gap requiring further investigation. Multidisciplinary coordination between nephrologists and implantologists is essential to mitigate risks. Though not an absolute contraindication, implant placement in dialysis-dependent patients demands thorough preoperative assessment of bone metabolism markers, individualized surgical protocols, and realistic patient counseling regarding potentially prolonged osseointegration timelines and guarded long-term prognosis compared to non-renal compromised individuals.

Implication for health policy/practice/research/medical education:
Achieving successful osseointegration in dialysis-dependent chronic kidney disease (CKD) patients presents a multifaceted challenge due to systemic complications affecting bone metabolism, immune function, and overall healing capacity. Careful patient selection, comprehensive pre-surgical evaluation, simplified surgical protocols, and prophylactic measures are essential. Further studies on the effects of systemic medications, the role of vitamin D supplementation, and the application of growth factors may provide additional avenues for improving dental implant outcomes in this vulnerable patient population.

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Review

Introduction

Chronic kidney disease (CKD) is a widespread health issue characterized by a gradual decline in kidney function, posing significant challenges across various aspects of patient care, including dental implant placement (1). The number of CKD patients is increasing, with most of them experiencing oral symptoms that often lead to early tooth loss. Dental implants can significantly improve the quality of life for these patients, but their compromised immune systems elevate the risk of infections (1). Patients with CKD, particularly those undergoing dialysis or kidney transplants, often exhibit profound alterations in bone metabolism. These alterations impact the homeostasis of calcium, phosphorus, vitamin D, parathyroid hormone (PTH), and fibroblast growth factor (FGF) (2). Such changes can lead to demineralization of jaw bones, reduced bone trabeculae, decreased cortical bone thickness, fibrocystic bone lesions, bone fractures, and delayed wound healing following tooth extraction (3). This makes oral health management for elderly hemodialysis patients a serious clinical concern (4). Specific challenges for dental implant osseointegration in dialysis-dependent CKD patients arise from these systemic complications (1, 3). Meanwhile, accelerated marginal bone loss within six months and reduced osseointegration have been observed in end-stage renal disease (ESRD) patients, potentially leading to early implant loss and treatment difficulties (5). Previous authors showed in ESRD patients, implants failed due to varying degrees of bone loss, although no correlation between PTH levels and primary implant stability or implant loss was found (5). Besides, new clinical guidelines have been developed to ensure safer dental implant procedures for dialysis patients with kidney failure. These guidelines emphasize that dentists should engage kidney specialists early in the treatment planning process (1). Therefore, a systematic consideration of patient history and complete blood tests are mandatory before surgery. The dental implant procedure itself should be kept as simple and non-invasive as possible. Post-operation, prophylactic antibiotics are recommended to minimize the risk of infection and complications with dialysis (6). This overview sought to consider osseointegration challenges of dental implants in dialysis-dependent CKD patients.

Search strategy

For this narrative review, we conducted a literature search across multiple databases, including PubMed, Google Scholar, the Directory of Open Access Journals (DOAJ), Web of Science, EBSCO, Scopus, and Embase, using a variety of relevant keywords like; osseointegration, vitamin D, parathyroid hormone, end-stage renal disease, fibroblast growth factor, chronic kidney disease, renal osteodystrophy and secondary hyperparathyroidism.

Osseointegration in dialysis-dependent CKD

The success of dental implants is significantly dependent on osseointegration, which is defined as the direct structural and functional connection between living bone and the implant surface. This process is crucial for implant stability, enabling both the loading of implants and their long-term clinical success. The interface between the titanium implant and the bone plays a vital role in this integration (7). Osseointegration involves an initial mechanical interlock followed by biological fixation through continuous bone growth and remodeling around the implant surface. This complex process is influenced by numerous factors affecting bone formation and maintenance at the implant interface (8). Factors influencing osseointegration include patient-related systemic conditions, surgical technique, and implant design. The quality of the implant biomaterial and the bone's healing capacity, alongside the bio-functional remodeling of peri-implant tissues, are major contributors to maintaining osseointegration and the quality of osteoreception (9). Researchers are actively focused on enhancing osseointegration by reducing bacterial adhesion while promoting the recruitment, adhesion, and proliferation of osteogenic elements like osteoblasts and fibroblasts (10).

Factors influencing dental implant osseointegration

Systemic diseases and medications can significantly influence dental implant osseointegration, survival rates, and peri-implant tissue health. Previous studies found that osteoporosis and diabetes are the most frequently investigated pathologies in this context (11). Other systemic diseases, such as neurological disorders, HIV, hypothyroidism, and cardiovascular diseases, and drugs like beta-blockers, anti-hypertensive agents or diuretics, do not appear to decrease the rate of implant osseointegration (11). However, certain medications, including proton-pump inhibitors and serotonin reuptake inhibitors, seem to negatively affect implant osseointegration. Meanwhile, chronic use of systemic medications that interfere with bone turnover and healing can impact osseointegration, potentially leading to premature implant loss (12). In fact, the mechanism of osseointegration is similar to bone remodeling and healing, making it susceptible to such interference (13). The role of vitamin D in osseointegration is also being explored, particularly in patients with systemic conditions like CKD (14). Studies have investigated whether vitamin D deficiency negatively impacts implant osseointegration and if supplementation can improve it. Results from animal studies indicated that vitamin D deficiency

can negatively affect new bone formation and bone-to-implant contact (BIC) (14). Conversely, other animal studies showed that vitamin D supplementation enhanced bone-to-implant contact and new bone formation around implants (14). Enhanced impact of vitamin D supplementation on osseointegration has been observed in subjects with diabetes mellitus, osteoporosis, and CKD (14). Other studies involving human subjects suggest that low serum vitamin D levels are associated with a higher tendency for early dental implant failure. When vitamin D was supplemented, osseointegration was successful in some cases, demonstrating a beneficial effect on bone level changes during the osseointegration process (15). While vitamin D deficiency appears to negatively affect osseointegration in animals, and supplementation seems to improve it in animals with systemic diseases, further investigation is needed to confirm these assumptions in humans (14).

Impact of CKD-MBD on skeletal microenvironment

Chronic kidney disease-mineral and bone disorder (CKD-MBD) fundamentally alters the skeletal microenvironment in ways that directly impact the cascade of cellular events necessary for successful titanium integration into alveolar bone (16). Nearly ninety percent of dialysis-dependent patients experience significant oral manifestations including periodontal disease, accelerated tooth loss, xerostomia, and mucosal alterations that often necessitate prosthetic rehabilitation, yet the very pathophysiological processes driving their renal failure simultaneously compromise the bone's capacity to heal around an implant surface (17). The challenges begin with the profound disruption of calcium-phosphate homeostasis that characterizes advanced renal disease. As glomerular filtration rate declines below fifteen milliliters per minute per 1.73 square meters, the kidneys lose their ability to excrete phosphate effectively, leading to hyperphosphatemia that triggers a cascade of hormonal responses (18). Elevated phosphate levels stimulate fibroblast growth factor 23 (FGF23) secretion from osteocytes, which normally would promote phosphaturia, but in the absence of functional nephrons, this compensatory mechanism fails while FGF23 levels continue rising exponentially. These extraordinarily elevated FGF23 concentrations, often hundreds of times above normal reference ranges in dialysis patients, exert direct inhibitory effects on osteoblast differentiation and mineralization while simultaneously suppressing renal 1-alpha-hydroxylase activity, thereby crippling the conversion of vitamin D to its active form (19). This condition creates a vicious cycle where vitamin D deficiency further impairs intestinal calcium absorption, contributing to hypocalcemia that chronically stimulates PTH secretion (19). The resulting secondary hyperparathyroidism represents perhaps the most significant direct challenge to osseointegration in this population. Persistently elevated PTH levels drive excessive osteoclastic bone resorption, creating a high-turnover bone state characterized by widened osteoid seams, disorganized collagen matrix, and under-mineralized bone tissue (20). Histomorphometric studies of iliac crest biopsies from dialysis patients reveal that up to eighty percent demonstrate some form of renal osteodystrophy, with mixed uremic osteodystrophy and osteitis fibrosa cystica representing the predominant high-turnover lesions (21). This pathological bone architecture lacks the structural integrity and predictable healing capacity required for stable implant anchorage (22). The disorganized trabecular architecture with excessive resorption cavities provides inadequate primary stability during implant placement, while the under-mineralized osteoid fails to support the mineralization front necessary for de novo bone formation along the titanium surface during the first weeks of healing (23,24). Previous studies using rodent models of CKD have demonstrated that titanium implants placed in uremic bone exhibit significantly reduced bone-to-implant contact percentages compared to healthy controls, with histological analysis revealing persistent fibrous tissue interposition rather than direct bone apposition at the interface (25-27). This impaired early osseointegration appears most pronounced during the initial inflammatory and proliferative phases of healing, suggesting some degree of late-stage integration may eventually occur, the critical window for establishing mechanical stability is compromised (16,27-29). As mentioned above, vitamin D deficiency compounds these challenges through multiple mechanisms beyond its classical role in calcium homeostasis. Nearly universal among dialysis patients due to reduced renal activation, dietary restrictions, and limited sun exposure, vitamin D insufficiency directly impairs osteoblast function and collagen synthesis while dysregulating the immune response necessary for coordinated healing (30). Vitamin D receptors on macrophages and lymphocytes modulate the inflammatory cascade following surgical trauma; since, deficiency shifts the balance toward prolonged pro-inflammatory cytokine production including elevated tumor necrosis factor-alpha and interleukin-6, which directly inhibit osteoblast differentiation while stimulating osteoclastogenesis (31). Indeed, the therapeutic administration of active vitamin D compounds in renal patients presents its own paradoxical challenges, as these agents primarily target parathyroid suppression rather than skeletal mineralization, and excessive dosing risks hypercalcemia and vascular calcification without necessarily improving bone quality at implant sites (32,33). Moreover, the accumulation of advanced

glycation end products represents another underappreciated impediment to osseointegration in this population (34,35). Uremic toxins that normally would be cleared by functioning kidneys accumulate in dialysis patients despite regular treatment sessions, with many small and middle molecules exhibiting half-lives extending beyond the interdialytic period (36). These uremic retention solutes, including indoxyl sulfate, p-cresyl sulfate, and asymmetric dimethylarginine, directly impair osteoblast proliferation and alkaline phosphatase activity while promoting oxidative stress within the bone microenvironment (37,38). In vitro studies demonstrate that physiological concentrations of these toxins reduce mineralized nodule formation by human osteoblasts by thirty to fifty percent and increase osteoblast apoptosis through mitochondrial dysfunction pathways (39). The chronic inflammatory state endemic to dialysis patients further exacerbates this cellular toxicity (40). Elevated C-reactive protein and interleukin-6 levels, present in many hemodialysis recipients due to bioincompatible dialysis membranes, recurrent vascular access procedures, and underlying comorbidities like diabetes and atherosclerosis, create a systemic milieu that favors catabolic over anabolic bone metabolism (41, 42). This inflammation-driven bone loss operates through receptor activator of nuclear factor kappa-B ligand (RANKL) up-regulation, tipping the RANKL/osteoprotegerin ratio decisively toward osteoclast activation and bone resorption (43). The consequences for dental implants manifest as accelerated marginal bone loss during the critical first six months post-placement (44), with longitudinal studies reporting average crestal bone resorption in maxilla and mandible among ESRD patients, which significantly exceeding the normal value (45,46). Additionally, anticoagulation management presents additional practical challenges during implant surgery and the immediate postoperative period (47). Most dialysis patients receive heparin during hemodialysis sessions to prevent circuit clotting, creating a transient but significant coagulopathy that complicates surgical hemostasis (48). While heparin's effects are relatively short-lived with a half-life of approximately sixty to ninety minutes (49), the timing of implant placement relative to dialysis sessions requires careful coordination to minimize bleeding complications without unnecessarily delaying necessary dental rehabilitation (1). More problematic are patients maintained on chronic oral anticoagulants like warfarin for atrial fibrillation or thromboembolic prophylaxis, which cannot be easily interrupted given their elevated cardiovascular risk profile (50). The delicate balance between maintaining therapeutic anticoagulation to prevent life-threatening thrombotic events and achieving adequate hemostasis for primary wound closure represents a genuine clinical dilemma, as persistent bleeding into the osteotomy site may mechanically disrupt the blood clot necessary for initiating the osseointegration cascade while creating a hematoma that serves as a nidus for bacterial colonization (51-54). Likewise, infection risk constitutes perhaps the most immediate threat to implant survival in dialysis-dependent patients (55). Uremia-induced neutrophil dysfunction, characterized by impaired chemotaxis, phagocytosis, and intracellular killing capacity, creates a state of functional immunosuppression despite normal or elevated white blood cell counts (56). This cellular immune defect, compounded by frequent healthcare exposures, indwelling vascular access devices, and often suboptimal nutritional status, renders these patients exceptionally vulnerable to both surgical site infections and hematogenous seeding of the implant-bone interface (57-59). Bacteremia during routine dialysis sessions occurring in patients annually due to catheter-related infections or access site contamination, which poses a particular threat during the vulnerable osseointegration period when the implant lacks a protective epithelial seal and vascular integration remains incomplete (60,61). The consequences of peri-implant infection in this population can be catastrophic, as impaired healing capacity often transforms what might be a manageable mucositis in healthy individuals into rapidly progressive peri-implantitis with extensive bone destruction and inevitable implant loss (62,63). Nutritional deficiencies further undermine the biological foundation for osseointegration (64). Protein-energy wasting affects many hemodialysis patients, manifesting as reduced serum albumin, prealbumin, and lean body mass (65). Adequate protein intake is necessary for collagen synthesis during bone healing, yet dietary protein restrictions historically imposed on renal patients, coupled with anorexia of uremia and dialysis-induced amino acid losses, frequently result in suboptimal substrate availability for tissue repair (66). Accordingly, micronutrient deficiencies including zinc, copper, and vitamin C, as the critical cofactors for collagen cross-linking and osteoblast function, further compromise the quality of newly formed bone at the implant interface (67). The cumulative effect of these nutritional deficits is bone of inferior mechanical properties with reduced tensile strength, incapable of withstanding functional occlusal loads even if initial osseointegration appears radiographically successful (68, 69). Bisphosphonate therapy, occasionally prescribed for management of renal osteodystrophy or concomitant osteoporosis, introduces another layer of complexity through its profound suppression of bone turnover (70). While high-turnover bone disease predominates in dialysis patients, some develop adynamic bone disease characterized by severely suppressed bone formation rates, particularly after aggressive parathyroid suppression with

vitamin D analogs or calcimimetics (71). The addition of bisphosphonates in this context risks inducing a state of near-complete bone metabolic arrest where the minimal remodeling necessary for adaptive bone response to implant placement becomes impossible (72). Furthermore, the renal clearance of nitrogen-containing bisphosphonates like alendronate and zoledronate is significantly impaired in CKD, leading to drug accumulation and prolonged skeletal half-life that may persist for years after discontinuation. This condition creates a persistent risk for medication-related osteonecrosis of the jaw following any dentoalveolar surgery, including implant placement or subsequent peri-implant procedures (73). The pathophysiology of medication-related osteonecrosis of the jaw in renal patients consists a triad of suppressed bone turnover preventing micro-damage repair, compromised soft tissue healing due to antiangiogenic effects, and local bacterial challenge overwhelming an already impaired immune response, as a perfect storm that can transform routine implant therapy into a source of chronic, non-healing osteonecrotic lesions (74, 75). Despite these challenges, emerging clinical evidence suggests that dental implants can achieve acceptable survival rates in carefully selected dialysis patients when managed through a meticulous approach (1).

Periodontal disorders in CKD

Patients with renal disorders often exhibit a higher prevalence of periodontal disease compared to individuals without such disorders (76). Experimental studies have demonstrated that osseointegration can be achieved in animals with induced CKD, leading to the hypothesis that dental implants can osseointegrate and remain functionally stable in patients with renal disorders (27). Histomorphometric results have shown that estrogen deficiency, when combined with CKD, synergistically impairs the bone-to-implant contact ratio and implant push-in resistance in mice (77). Several studies detected that, successful osseointegration requires the normal functioning of biological activities involved in bone remodeling, which includes both bone resorption by osteoclasts and new bone formation by osteoblasts (78). Any event altering bone repair and healing can consequently affect successful osseointegration, potentially leading to premature implant loss or peri-implant complications (79). The mere presence of a disease does not necessarily preclude dental implant therapy or significantly impact long-term outcomes, as some controlled disorders allow implant survival rates comparable to healthy individuals (80). Malnutrition, inflammation, and atherosclerosis are interconnected factors that contribute to the progression of kidney disease. Despite advancements in renal clearance techniques, morbidity and mortality rates remain high for individuals with ESRD (81). Disturbances in hematological-based indices, such as neutrophil/lymphocyte ratio, platelet/lymphocyte ratio, malnutrition-inflammation score, and systemic immune-inflammation index, can affect the outcomes for chronic hemodialysis patients (82,83). For instance, a study comparing chronic hemodialysis patients with healthy controls found significant differences in prognostic nutritional index, malnutrition-inflammation score, platelet/lymphocyte ratio, and systemic immune-inflammation index. These indices reflect underlying inflammatory and nutritional states that could indirectly influence a patient's capacity for successful osseointegration (84). Hence, the challenges in osseointegration for dialysis-dependent CKD patients highpoint the need for meticulous and individualized treatment planning (16). Though evidence suggests that osseointegration can be achieved in patients with certain bone disorders like osteoporosis and ectodermal dysplasia, the planning process requires careful consideration of the degree of tissue involvement, patient age, and skeletal development compared to systemically healthy patients. It is preferable to avoid extensive surgeries in medically compromised patients to minimize complications, sometimes necessitating alternative approaches such as salvaging existing prostheses with novel attachment systems (85,86). Recently, growth factors like platelet-derived growth factors, bone morphogenetic proteins, transforming growth factor-beta, insulin-like growth factor, vascular endothelial growth factor, platelet-rich plasma, and platelet-rich fibrin show potential in enhancing bone healing and implant stability by promoting osteoblast activity, angiogenesis, and matrix formation, thereby accelerating peri-implant bone regeneration (87). Delivery methods such as platelet concentrates, surface coatings, and biomaterial integration have demonstrated promising outcomes in this regard. These advancements could offer strategies to improve osseointegration in compromised patients (87).

Conclusion

In summary, osseointegration in dialysis-dependent CKD patients presents multifactorial challenges requiring careful clinical consideration. The primary obstacle stems from renal osteodystrophy and CKD-mineral bone disorder, which disrupt normal bone remodeling through secondary hyperparathyroidism, altered calcium-phosphorus homeostasis, and impaired osteoblast function. These metabolic disturbances compromise early-stage bone-implant integration, with studies demonstrating reduced bone-implant contact strength during initial healing phases despite eventual osseointegration potential. Additional complications include heightened infection susceptibility due to uremia-

induced immunosuppression, increased bleeding risks from anticoagulant use during hemodialysis sessions, and xerostomia promoting oral dysbiosis. Furthermore, altered drug metabolism necessitates modified antibiotic and analgesic regimens, while periodontal bone loss accelerates marginal bone resorption around implants. Nevertheless, emerging evidence suggests dental implants can achieve acceptable survival rates in this population when managed appropriately. Success hinges on interdisciplinary coordination with nephrologists to schedule procedures on non-dialysis days, optimize calcium/vitamin D status preoperatively, extend healing periods beyond standard protocols, and implement rigorous antimicrobial prophylaxis. Though, CKD does not absolutely contraindicate implant therapy, clinicians must recognize that osseointegration follows a delayed, less predictable trajectory. Future research should focus on biomaterial modifications and adjunctive therapies targeting FGF-23 pathways to enhance bone quality. Finally, with meticulous patient selection, modified surgical protocols, and vigilant long-term monitoring, dental implants remain a viable rehabilitation option for dialysis-dependent patients seeking functional oral restoration.

Conflicts of interest

The author declares that he has no competing interests.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the author utilized Perplexity to refine grammar points and language style in writing. Subsequently, the author thoroughly reviewed and edited the content as necessary, assuming full responsibility for the publication's content.

Ethical issues

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the author.

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