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The Effect of Glucocorticosteroids on the Osseointegration of Dental Implants: Pathogenetic Mechanisms and Clinical Consequences

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Abstract: Glucocorticosteroids (GCS) have a complex effect on bone metabolism, theoretically creating risks for dental implant osseointegration through glucocorticoid-induced osteoporosis (GIOP). Aim: to analyze the pathogenetic mechanisms of GCS effect on bone remodeling around dental implants and assess clinical consequences. Materials and methods: an analytical literature review was conducted in PubMed, Scopus, and eLibrary databases (2018–2025) using keywords: glucocorticoids, osseointegration, dental implants, osteoporosis. Results: pathogenetic mechanisms (suppressed osteogenesis, increased resorption, inhibited angiogenesis) create serious theoretical risks. However, clinical studies demonstrate that with careful treatment planning (two-stage protocol, adequate integration period) GCS intake does not preclude high success rates (up to 99 % osseointegration). Conclusions: long-term GCS therapy is not an absolute contraindication for dental implantation. Risk minimization strategies include bone metabolism assessment, pharmacological correction, osteoinductive implant surfaces, and careful monitoring.

Keywords: glucocorticosteroids, osseointegration, dental implants, bone remodeling, glucocorticoid-induced osteoporosis

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Влияние глюкокортикостероидов на остеоинтеграцию дентальных имплантатов: патогенетические механизмы и клинические последствия

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Аннотация: Глюкокортикостероиды (ГКС) оказывают комплексное влияние на костный метаболизм, создавая теоретические риски для остеоинтеграции дентальных имплантатов через развитие глюкокортикоид-индуцированного остеопороза (ГИОП). Цель: проанализировать патогенетические механизмы воздействия ГКС на репаративную регенерацию костной ткани в области дентальных имплантатов и оценить клинические последствия. Материалы и методы: проведен аналитический обзор литературы в базах PubMed, Scopus и eLibrary за 2018–2025 гг. по ключевым словам: глюкокортикоиды, остеоинтеграция, дентальные имплантаты, остеопороз. Результаты: патогенетические механизмы (подавление остеогенеза, усиление резорбции, угнетение ангиогенеза) создают серьезные теоретические риски. Однако клинические исследования демонстрируют, что при тщательном планировании лечения (двухэтапный протокол, адекватный период интеграции) прием ГКС не является непреодолимым препятствием для достижения высоких показателей успеха (до 99 % остеоинтеграции). Заключение: длительная терапия ГКС не является абсолютным противопоказанием к дентальной имплантации. Стратегии минимизации рисков включают оценку костного метаболизма, фармакологическую коррекцию, применение имплантатов с остеоиндуктивными поверхностными модификациями и тщательный мониторинг.

Ключевые слова: глюкокортикостероиды, остеоинтеграция, дентальные имплантаты, костное ремоделирование, глюкокортикоид-индуцированный остеопороз

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Introduction

Osseointegration, defined as a direct structural and functional connection between living bone and the surface of a functionally loaded implant, is a fundamental biological process that determines the long-term success of dental implantation [Fazliu et al., 2024; Mohammadi et al., 2023]. This dynamic process is regulated by a complex interaction of bone cells, growth factors and signaling pathways that control bone remodeling [Chotiyarnwong, McCloskey, 2020; Fazliu et al., 2024]. Long-term or high-dose use of glucocorticosteroids (GCS), despite their undeniable therapeutic value in the treatment of a wide range of autoimmune, inflammatory and other systemic diseases, is associated with a serious side effect – bone metabolism disorders and the development of glucocorticoid-induced osteoporosis (GIOP) [Fazliu et al., 2024; Chotiyarnwong, McCloskey, 2020; Cho, Sung, 2021]. These violations theoretically create risks for osseointegration of dental implants. It should be noted that the key signaling pathways regulating osteogenesis and angiogenesis (such as Wnt/β-catenin, BMP/Smad, and NF-κB), which are targeted by GCS, continue to be actively studied as potential targets for controlling osseointegration [Al Subaie, Emami, 2024]. Recent advancements

in dental implantology have emphasized the importance of understanding how systemic medications affect the bone-implant interface [D'Ambrosio et al., 2023]. The purpose of this analytical review is to systematize modern data on the key pathogenetic mechanisms by which GCS influence the process of osseointegration, and to objectively assess the clinical consequences of their use based on the results of current experimental and clinical studies [Petsinis et al., 2017; Chotiyarnwong, McCloskey, 2020; Fazliu et al., 2024; Li et al., 2025].

Objects and methods of research

An analytical review of the literature was conducted to systematize data on the effect of glucocorticosteroids (GCS) on the osseointegration of dental implants. The search for scientific publications was carried out in the PubMed, Scopus and eLibrary databases using the keywords: glucocorticoids, osseointegration, dental implants, osteoporosis, glucocorticoid-induced osteoporosis (GIOP), in Russian and English, for the period from 2009 to 2025.

The review included original *in vivo* and *in vitro* experimental studies, clinical observations, retrospective analyses, as well as systematic and narrative reviews containing data on bone remodeling, osseointegration and survival of dental implants in patients receiving GCS. Publications without specific quantitative or structural data, duplicate reviews, as well as individual abstracts and brief communications were not included in the review.

The analysis was carried out with an assessment of the described pathogenetic mechanisms of action of GCS, histological and histomorphometric characteristics of bone tissue, radiological indicators (including micro-CT data), as well as clinical outcomes regarding the integration and survival of implants.

The results and their discussion

Pathogenetic mechanisms of GCS action on bone tissue. Pathogenesis studies clearly demonstrate that glucocorticosteroids have a profound inhibitory effect on processes critical for osseointegration. The central link of this effect is: GCS inhibit the proliferation and differentiation of osteoprogenitor cells, while stimulating apoptosis of mature osteoblasts and osteocytes, which leads to a sharp decrease in bone formation and bone matrix synthesis [Chotiyarnwong, McCloskey, 2020; Fazliu et al., 2024; Lee et al., 2021]. Additional pathogenesis studies [Krasivina et al., 2019; Compston et al., 2017] clarify that GCS inhibit osteoblast differentiation via activation of PPAR-γ2, KLF15 и C/EBPα, redirecting mesenchymal stem cells to the adipocyte lineage. In parallel, an increase in sclerostin, a secreted glycoprotein with a C-terminal cysteine knot-like domain and a sequence similar to the DAN family of bone morphogenetic protein antagonists, suppresses the Wnt/β-catenin pathway, which is critical for osteogenesis [Yu et al., 2022]. The biphasic nature of GIOP has been confirmed: trabecular bone loses 10–20 % of its mass during the first 6 months of therapy, then the process slows down to 2 %/year [Compston et al., 2017].

In parallel, GCS exert complex, often dysregulated effects on osteoclasts, ultimately promoting increased bone resorption, particularly in trabecular bone, through modulation of the RANKL/RANK/OPG system and other pathways [Chotiyarnwong, McCloskey, 2020; Fazliu et al., 2024]. The resulting imbalance between decreased bone formation and preserved or increased resorption underlies the development of GIOP. An important negative factor is also the suppression of angiogenesis by GCS due to a decrease in the expression of VEGF and other proangiogenic factors, which worsens the vascularization of the implantation zone and slows down the processes of reparation and osteogenesis [Chotiyarnwong, McCloskey, 2020].

Features of glucocorticoid-induced osteoporosis in dental implantation. According to [Krasivina et al., 2019], in glucocorticoid-induced osteoporosis, pathogenetic changes worsen with age and have a number of features that distinguish it from postmenopausal osteoporosis. This variant of the disease is characterized by a more pronounced suppression of osteoblastic activity with a simultaneous increase in osteoclastic resorption, which leads to an accelerated decrease in bone mass. Trabecular bone is predominantly affected in the early stages, which is accompanied by a rapid decrease in its strength.

Glucocorticoids disrupt the formation of bone matrix by suppressing the synthesis of type I collagen and other structural proteins by osteoblasts. Additionally, steroid myopathy and a decrease in muscle mass develop, which increases the risk of falls and fractures, as well as metabolic and vascular disorders, including insulin resistance and angiopathy, which aggravate bone fragility.

Additional domestic studies also confirm that GCS cause rapid and significant changes in bone metabolism already in the first months of administration, with accelerated loss of bone mineral density and an increased risk of fractures, which requires early administration of preventive measures, including calcium preparations, vitamin D and osteotropic agents [Krasivina et al., 2019; Kulakov et al., 2019; Brudyan et al., 2023]. These pathological changes directly affect the potential for successful osseointegration in dental implantology [Ma et al., 2024].

Experimental data and correction strategies. Experimental data on HYOP models generally confirm a significant deterioration in bone remodeling around implants [Chotiyarnwong and McCloskey, 2020; Li et al., 2025]. Studies on the correction of these disorders have revealed promising strategies. Eldecalcitol (ED-71), a vitamin D analogue, has been shown to attenuate dexamethasone-induced osteoblast and osteoclast dysfunction by increasing sirtuin 1 (SIRT1) levels, which significantly improved the osseointegration of titanium implants in rats with GIOP; inhibition of SIRT1 completely blocked this beneficial effect [Kou et al., 2023; Li et al., 2025].

Another approach is to modify the surface of the implant. Studies demonstrate that the immobilization of osteoinductive factors on the titanium surface demonstrates a pronounced potential for stimulating osteogenesis in vitro and in vivo, which can compensate for the systemic effects of GCS [Li et al., 2025]. Similar approaches to accelerating osseointegration have been proposed in domestic studies, where the use of bioactive bonite coating of titanium implants ensured the complete formation of the "implant-bone" complex within 4 months, reducing the time to the orthopedic stage of treatment [Brudyan et al., 2023]. Another Russian study also reports positive pathomorphological changes in the bone tissue around dental implants when their osseointegration is stimulated [Guzov et al., 2021].

Clinical outcomes of dental implantation in patients receiving GCS. Despite the presented experimental data on the suppressive effect of GCS on osseointegration and bone regeneration, clinical data on the effect of GCS on implant survival do not demonstrate a critical negative effect. Thus, a retrospective clinical study including 31 patients (105 implants) who had been receiving GCS for a long time for various systemic diseases (rheumatoid arthritis, asthma, systemic lupus erythematosus, etc.) showed exceptionally high results: 99 % successful osseointegration when installing implants according to the classic two-stage protocol without bone grafting. There were no radiographic signs of resorption at the stage of disclosure and 99 % survival of implants during observation after loading for 71 months. At the same time, the authors emphasize the absence of a significant negative effect of GCS on osseointegration and 3-year survival in their cohort [Petsinis et al., 2017].

These findings are consistent with a recent meta-analysis, which showed that the presence of systemic osteoporosis (including GIOP) does not significantly affect dental implant survival and marginal bone loss, although it may slightly increase the risk of early complications [Lee et al., 2025]. Modern protocols for dental implantation in compromised bone emphasize the importance of adequate primary stability and modified healing periods. Recent studies on drilling techniques have shown that optimized surgical protocols can significantly improve primary stability of zirconia implants in low-density bone, which is particularly relevant for patients with GIOP [Sagheb et al., 2025]. The critical role of mesenchymal stem cells in bone regeneration and osseointegration is increasingly recognized, with recent research highlighting their potential as therapeutic targets to enhance implant integration, particularly in compromised conditions [Ma et al., 2023].

Domestic authors also emphasize that the quality of osseointegration largely depends on bone density, the shape and design of the implant, its surface microstructure, as well as compliance with loading protocols, especially in patients with osteoporosis. Thus, in low-density bone (Lekholm & Zarb class D4), the risk of implant failure is significantly higher due to poor primary stability and reduced bone-to-implant contact. This results in a reported failure rate of 30–40 %, which is 3–4 times greater

than the 5–10 % risk observed in dense bone (classes D1–D2). It has been established that cylindrical implants in osteoporotic bone provide less primary stability than conical ones, and premature loading can lead to bone resorption and loss of fixation [Kulakov et al., 2019; Brudyan et al., 2023].

Discussion

The presented results emphasize the complex and ambiguous nature of the influence of glucocorticosteroids on the osseointegration of dental implants. On the one hand, convincingly proven pathogenetic mechanisms – suppression of osteoblastogenesis and osteoblast function, dysregulation of osteoclastogenesis and resorption, inhibition of angiogenesis – create serious theoretically substantiated prerequisites for disruption of bone formation processes around the implant and an increased risk of failure, especially against the background of developed GIOP [Chotiyarnwong, McCloskey, 2020; Fazliu et al., 2024]. Experimental data on GIOP models confirm these negative effects [Chotiyarnwong, McCloskey, 2020; Li et al., 2025].

However, data from large-scale umbrella reviews summarizing the results of clinical studies indicate that GCS use per se does not demonstrate a clear and statistically significant reduction in osseointegration rates, highlighting the complexity and compensability of these pathogenetic mechanisms in clinical practice [Di Antonio et al., 2023]. On the other hand, a clinical retrospective study with long-term follow-up demonstrates that with careful treatment planning (use of a proven two-stage surgical protocol, ensuring an adequate period of osseointegration before loading) and in cases that do not require complex bone grafting procedures, the use of GCS for systemic diseases is not an insurmountable obstacle to achieving implantation success rates typical for healthy people (99 % osseointegration and survival) [Petsinis et al., 2017]. This indicates that the negative systemic effects of GCS can be compensated by the optimal choice of surgical tactics and perioperative patient management protocol.

When choosing implants, priority is given to conical structures with a rough surface (Ra 1-3 μm), which provide greater primary stability in low-density bone [Romanos et al., 2014; Xu et al., 2015], and modern surface modifications, which accelerate fibronectin adhesion and osteogenic differentiation [Li et al., 2025]. Advanced drilling protocols and implant design modifications continue to evolve, with recent evidence demonstrating that specific surgical techniques can enhance primary stability even in compromised bone conditions [Romanos et al., 2014; Sagheb et al., 2025].

Key risk minimization strategies emerging from the analysis include: careful preoperative assessment of the patient's bone metabolism, including diagnosis of possible GIOP; preference for a two-stage implantation protocol with adequate integration time; consideration of pharmacological correction of bone remodeling abnormalities (e.g., vitamin D supplements such as ED-71 [Rabczak et al., 2025], although their use in humans requires further clinical studies); and use of implants with osteoinductive surface modifications, which can locally stimulate osteogenesis and potentially mitigate systemic adverse effects [Li et al., 2025].

In addition to standard approaches to bone metabolism correction, the analyzed literature also describes strategies for optimizing osseointegration in osteoporosis, including the use of drugs that improve bone mineral density and quality before implantation, and hormonal correction in postmenopausal women [Krasivina et al., 2019; Brudyan et al., 2023]. Despite encouraging clinical data on the survival of DIs with GCS [Petsinis et al., 2017], experimental indications of a possible negative effect of GCS на долгосрочное костное ремоделирование вокруг имплантата нельзя игнорировать [Li et al., 2025]. This emphasizes the need for mandatory careful long-term monitoring of patients receiving GCS, even after successful initial osseointegration.

When prescribing GCS, hydrocortisone is preferable: its effect on BMD is 10–15 % weaker than that of prednisolone due to a smaller free fraction and physiological half-life, which is supported by studies showing a better bone safety profile [Guarnotta et al., 2024]. Dual-release formulations (e.g., Chronocort®) are promising, improving cortisol-binding capacity and demonstrating an 11.5 % increase in BMD after 2 years [Hasenmajer et al., 2023]. Further prospective clinical studies with

large sample sizes and long-term follow-up are needed to more accurately stratify the risk and optimize management protocols for such patients.

Conclusion

Glucocorticosteroids have a multi-component negative effect on the key biological processes underlying the osseointegration of dental implants, mainly through the suppression of osteogenesis, increased resorption and impaired angiogenesis, which is associated with the development of GIOP. However, current clinical data indicate that with adequate treatment planning, use of a two-stage surgical protocol and no need for complex bone grafting, long-term GCS therapy for systemic diseases is not an absolute contraindication to dental implantation and allows achieving high success rates and implant survival in the long term. Risk minimization strategies include assessment of bone metabolism, consideration of pharmacological correction (e.g. SIRT1 activators) and use of implants with osteoinductive surface modifications. A promising direction is also the development of combined methods for stimulating osseointegration, including both pharmacological and physiotherapeutic effects. A prerequisite for success is careful long-term monitoring of bone tissue around the implant in this category of patients and individual selection of a GCS drug (hydrocortisone > prednisolone).

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