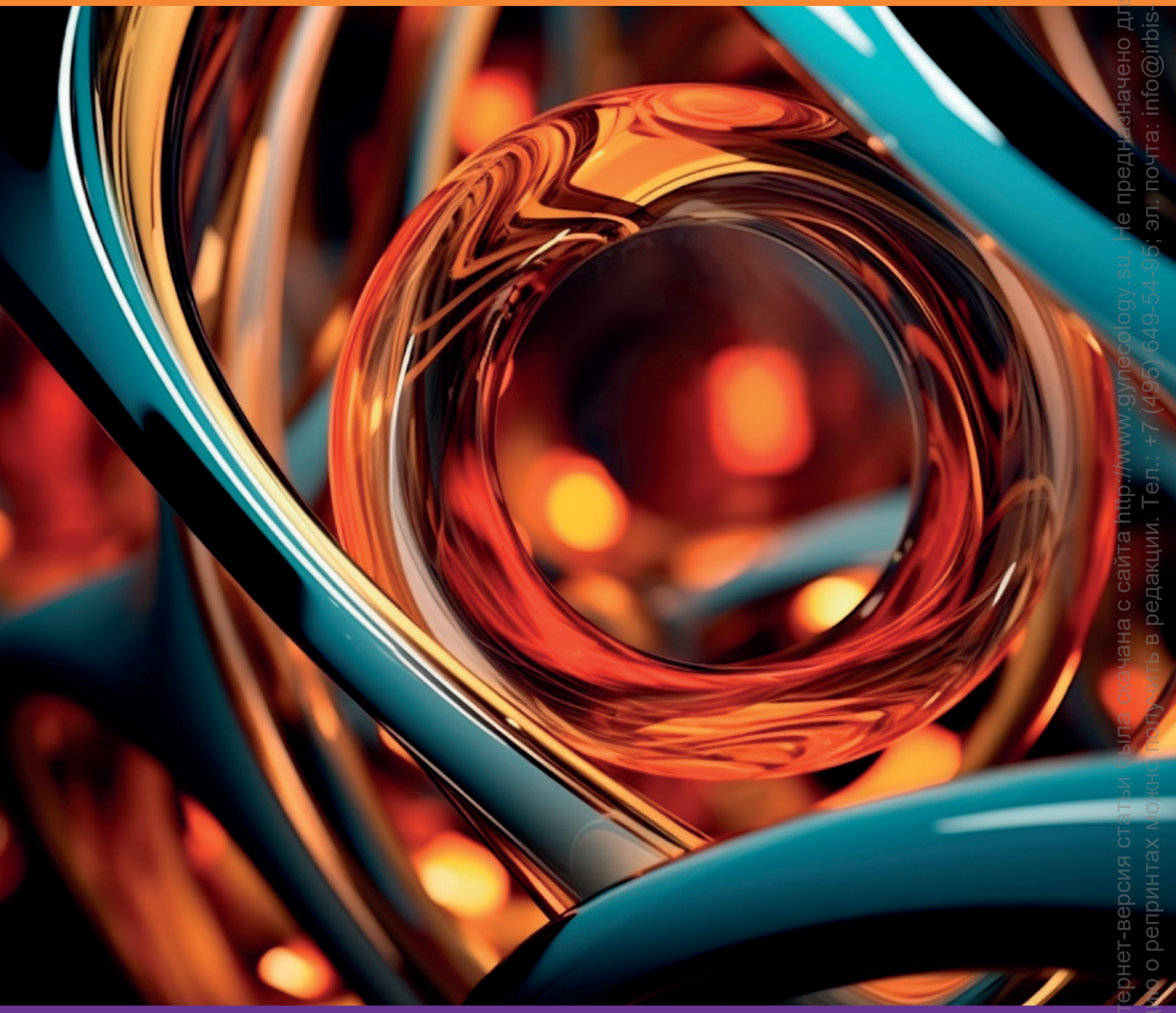
ISSN 2313-7347 (print)
ISSN 2500-3194 (online)

АКУШЕРСТВО ГИНЕКОЛОГИЯ РЕПРОДУКЦИЯ

Включен в перечень ведущих рецензируемых журналов и изданий ВАК

2025 • том 19 • № 6



OBSTETRICS, GYNECOLOGY AND REPRODUCTION

2025 Vol. 19 No 6

https://gynecology.su

ISSN 2313-7347 (print)
ISSN 2500-3194 (online)

https://doi.org/10.17749/2313-7347/ob.gyn.rep.2025.678

Menopause, postmenopausal osteoporosis, and dental implantation: the role of menopausal hormone therapy

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Abstract

Here, we summarize current evidence on the impact of postmenopausal osteoporosis, menopause-related hormonal changes, and hormone therapy on dental implant outcomes. Epidemiology and pathogenesis of bone alterations are reviewed, with particular emphasis on the role of estrogen, progesterone, calcitonin, growth hormone, and insulin-like growth factor-1 (IGF-1) deficiency. Special attention is paid to the effects of menopausal hormone therapy and bioidentical forms of estradiol and progesterone on osteoporosis course and the effectiveness of dental implantation. The analysis highlights the risks of implant loss and the opportunities of interdisciplinary approach in dentistry and endocrinology to optimize implant osseointegration in postmenopausal women.

Keywords: postmenopausal osteoporosis, dental implantation, menopausal hormone therapy, bioidentical sex hormones, antiresorptive therapy, osteoanabolic therapy, alveolar bone

For citation: Medvedeva E.V., Ivanov N.V., Vesnina A.F., Khromova E.A., Yusupova Sh.K. Menopause, postmenopausal osteoporosis, and dental implantation: the role of menopausal hormone therapy. *Akusherstvo, Ginekologia i Reprodukcia = Obstetrics, Gynecology and Reproduction.* 2025;19(6):966–975. (In Russ.). https://doi.org/10.17749/2313-7347/ob.gyn.rep.2025.678.

Менопауза, постменопаузальный остеопороз и дентальная имплантация: роль менопаузальной гормональной терапии

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Obstetrics, Gynecology and Reproduction

Резюме

В статье обобщены современные данные о влиянии постменопаузального остеопороза, гормональных изменений в менопаузе и гормональной терапии на исходы дентальной имплантации. Рассмотрены эпидемиология, патогенез костных изменений, роль дефицита эстрогенов, прогестерона, кальцитонина, гормона роста и инсулиноподобного фактора роста. Особое внимание уделено влиянию менопаузальной гормональной терапии, биоидентичных форм эстрадиола и прогестерона на течение остеопороза и эффективность дентальной имплантации. Представлен анализ рисков потери дентальных имплантатов и возможности междисциплинарного подхода в стоматологии и эндокринологии для оптимизации остеоинтеграции имплантатов у пациенток в постменопаузе.

Ключевые слова: постменопаузальный остеопороз, дентальная имплантация, менопаузальная гормональная терапия, биоидентичные половые гормоны, антирезорбтивная терапия, остеоанаболическая терапия, альвеолярная кость

Для цитирования: Медведева Е.В., Иванов Н.В., Веснина А.Ф., Хромова Е.А., Юсупова Ш.К. Менопауза, постменопаузальный остеопороз и дентальная имплантация: роль менопаузальной гормональной терапии. *Акушерство, Гинекология и Репродукция.* 2025;19(6):966–975. https://doi.org/10.17749/2313-7347/ob.gyn.rep.2025.678.

Highlights

What is already known about this subject?

- Postmenopausal osteoporosis is associated with an increased risk of dental implant failure. Hypoestrogenism, secondary hyperparathyroidism, and insulin-like growth factor-1 (IGF-1) deficiency impair alveolar bone microarchitecture. Antiresorptive therapy with bisphosphonates and denosumab increases bone density but is associated with the risk of jaw osteonecrosis.

What are the new findings?

- The effects of bioidentical forms of estradiol and progesterone on dental implant osseointegration are discussed.
- Data on the role of calcitonin, follicle-stimulating hormone, and systemic inflammation in predicting implant outcomes are presented.
- A concept of combined use of bioidentical estrogens and progestogens as a part of menopausal hormone therapy in postmenopausal women planning dental implantation is proposed.

How might it impact on clinical practice in the foreseeable future?

- It may allow for individualized planning of implant preparation in postmenopausal women; justify the rationale for preliminary hormonal correction in women planning dental implantation; expands interdisciplinary collaboration between dentists, endocrinologists, and gynecologists.

Основные моменты

Что уже известно об этой теме?

- Постменопаузальный остеопороз связан с повышенным риском неудач дентальной имплантации. Гипоэстрогения, вторичный гиперпаратиреоз и дефицит инсулиноподобного фактора роста (IGF-1) ухудшают микроархитектуру альвеолярной кости. Антирезорбтивная терапия бисфосфонатами и деносумабом повышает плотность кости, но сопряжена с риском остеонекроза челюсти.

Что нового дает статья?

- Рассмотрено влияние биоидентичных форм эстрадиола и прогестерона на остеоинтеграцию дентальных имплантатов.
- Представлены данные о роли кальцитонина, фолликулостимулирующего гормона и системного воспаления в прогнозе эффективности имплантации.
- Предложена концепция комбинированного применения биоидентичных эстрогенов и прогестагенов в составе менопаузальной гормональной терапии у женщин в постменопаузе, планирующих дентальную имплантацию.

Как это может повлиять на клиническую практику в обозримом будущем?

- Позволит индивидуализировать план подготовки имплантации у женщин в постменопаузе; обосновывает целесообразность предварительной гормональной коррекции у женщин, планирующих дентальную имплантацию; расширяет междисциплинарное взаимодействие врачей-стоматологов, эндокринологов и гинекологов.

Введение / Introduction

Menopause is a natural stage in a woman's life, characterized by the cessation of menstruation due to depletion of the ovarian follicular reserve. The average age of menopause onset in Europe and Russia is 49–51 years [1, 2]. Postmenopause is accompanied by a sharp decline in estrogen and progesterone secretion, as well as changes in the levels of other hormones including parathyroid hormone, calcitonin, growth hormone, and insulin-like growth factor-1 (IGF-1) which lead to impaired bone metabolism and the development of postmenopausal osteoporosis [3–5].

Postmenopausal osteoporosis (PMO) is one of the leading risk factors for fractures and significantly contributes to reduced quality of life in older women. In dental practice, however, it has additional clinical relevance – reduced bone mineral density (BMD) worsens conditions for dental implantation, increases the risk of peri-implantitis, and accelerates alveolar ridge resorption [6, 7].

With increasing life expectancy and the rising proportion of women over 50, the number of postmenopausal patients requiring implant-supported prosthetic treatment continues to grow [8]. This requires a more thorough interdisciplinary approach involving prosthodontists, maxillofacial surgeons, endocrinologists, and gynecologists.

Osteoporosis epidemiology / Эпидемиология остеопороза

According to the International Osteoporosis Foundation (IOF), osteoporosis is diagnosed in one out of three women over the age of 50, and osteopenia is found in nearly half of women in this age group [9]. Russian epidemiological studies demonstrate that the prevalence of postmenopausal osteoporosis among women of postmenopausal age ranges from 34–36 %, while osteopenia is observed in 43–46 % [10].

Loss of bone mass in postmenopause affects not only the axial skeleton but also the alveolar bone of the jaws. In the first 10 years after menopause, the reduction in alveolar ridge density may reach 25–30 %, particularly in patients with severe osteoporosis [11]. This directly influences the prognosis of dental implantation – primary stability is reduced, osseointegration is delayed, and the risk of marginal bone resorption increases.

Pathogenesis of bone changes in postmenopause / Патогенез костных изменений в постменопаузе

A literature review demonstrates a relationship between osteoporosis and alveolar bone status under conditions of inflammatory oral diseases. Systemic reductions in bone mineral density increase the vulnerability of alveolar bone to inflammatory resorption, characteristic of periodontitis. Menopausal osteoporosis contributes to accelerated trabecular breakdown, cortical layer thinning, and reduced regenerative potential of jawbone tissue. These changes increase susceptibility to peri-implantitis and amplify the rate of marginal bone loss around implants. Shared pathogenetic mechanisms – inflammation, hormonal disturbances, and metabolic shifts connect systemic osteoporosis with local oral processes and create a basis for implant-related complications [12]. The pathogenesis of postmenopausal osteoporosis is associated with the combined effects of hormonal, metabolic, and inflammatory factors (**Fig. 1**).

Estradiol deficiency / Дефицит эстрадиола

Estradiol plays a key role in regulating bone remodeling through modulation of the RANK/RANKL/OPG system (nuclear factor activator/nuclear factor receptor activator/osteoprotegerin). It inhibits osteoclast differentiation and stimulates the production of osteoprotegerin, which blocks the interaction between RANKL and RANK [13].

Estradiol deficiency leads to increased RANKL expression and reduced osteoprotegerin levels, accelerating bone resorption. Estrogens also promote angiogenesis in bone tissue; their decline impairs microcirculation and nutritional supply to alveolar bone, further raising the risk of resorption and complications during dental implantation. Large-scale studies confirm that women receiving estrogens-based menopausal hormone therapy have higher bone mineral density and lower fracture rates [14].

Progesterone deficiency / Дефицит прогестерона

Progesterone is also an important regulator of bone turnover that stimulates osteoblasts, activates type I collagen synthesis, and promotes osteoid mineralization. After menopause, progesterone deficiency leads to decreased osteoblast activity and reduced new bone formation [15].

Clinical observations indicate that combined estrogen–progestin therapy results in a greater increase in spinal bone mineral density compared with estrogen monotherapy [14], thereby confirming that progesterone deficiency independently worsens postmenopausal bone

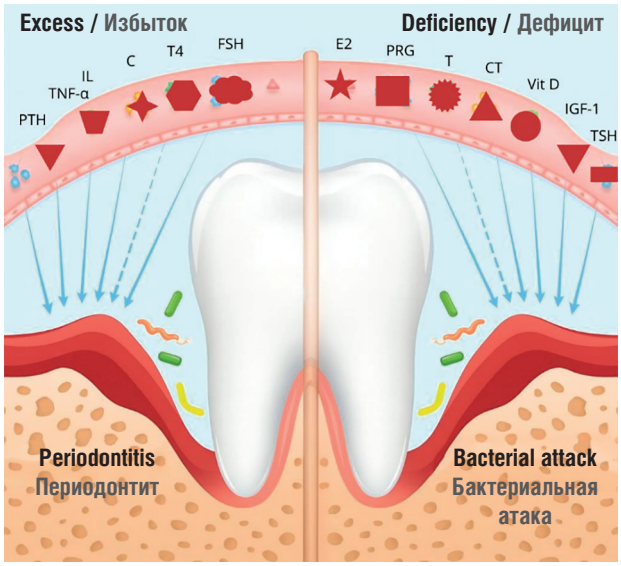

Figure 1. Factors influencing jawbone tissue in postmenopausal women [12].

Note: *PTH – parathyroid hormone; TNF-α – tumor necrosis factor-α; IL – interleukin; C – cortisol; T4 – thyroxine; FSH – follicle-stimulating hormone; E2 – estradiol; PRG – progesterone; T – testosterone; CT – calcitonin; Vit D – vitamin D; IGF-1 – insulin-like growth factor-1; TSH – thyroid-stimulating hormone.*

Рисунок 1. Факторы, влияющие на состояние костной ткани челюстей в постменопаузе [12].

Примечание: *PTH – паратгормон; TNF-α – фактор некроза опухоли-α; IL – интерлейкин-6; C – кортизол; T4 – тироксин; FSH – фолликулостимулирующий гормон; E2 – эстрадиол; PRG – прогестерон; T – тестостерон; CT – кальцитонин; Vit D – витамин D; IGF-1 – инсулиноподобный фактор роста-1; TSH – тиреотропный гормон.*

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alterations and that restoring progesterone levels exerts a protective effect on bone tissue, including alveolar bone.

Follicle-stimulating hormone excess / Избыток фолликулостимулирующего гормона

Upon menopause, follicle-stimulating hormone (FSH) level rises sharply. Previously, FSH was viewed only as a marker of ovarian failure, but current evidence shows that it directly influences bone metabolism [16]. Experimental studies demonstrate that FSH directly stimulates osteoclast differentiation and activity, increasing bone resorption independently of estrogen levels [17]. Thus, the hypergonadotropic state in postmenopause *per se* contributes to accelerated bone loss. In alveolar bone, elevated FSH can enhance localized resorption and may be associated with more profound cervical bone loss around implants.

Elevated parathyroid hormone / Повышенный паратгормон

Calcium deficiency and estrogen depletion in postmenopause lead to secondary hyperparathyroidism. Chronically elevated parathyroid hormone stimulates osteoclast differentiation, increases resorption, and accelerates bone loss, particularly in metabolically active bones such as alveolar bone [18]. This results in increased bone turnover, decreased local density at implant sites, and a higher risk of marginal resorption.

Calcitonin deficiency / Дефицит кальцитонина

Calcitonin, secreted by thyroid parafollicular cells, is an endogenous inhibitor of osteoclast activity. It normally reduces bone resorption by suppressing osteoclastogenesis and reducing osteoclast lifespan [19]. In postmenopause, calcitonin levels decline, removing this inhibitory effect and accelerating cortical thinning. For alveolar bone, reduced calcitonin is particularly detrimental, as it promotes marginal resorption and increases peri-implantitis risk.

Somatotropin and insulin-like growth factor-1 deficiency / Дефицит гормона роста и инсулиноподобного фактора роста

Growth hormone (GH) and IGF-1 are major regulators of bone anabolism. GH stimulates osteoblast proliferation, while IGF-1 enhances collagen synthesis and osteoid mineralization [20, 21]. Their decline in postmenopause reduces bone formation and slows reparative processes. Low IGF-1 level is associated with higher fracture risk and impaired healing after surgery.

Thyrotropin deficiency and thyroxine excess / Дефицит тиреотропного гормона и избыток тироксина

Thyroid hormones significantly influence bone metabolism. Thyroid-stimulating hormone (TSH) directly inhibits osteoclastogenesis and has a protective effect on bone tissue. When its levels decrease, this inhibitory effect is lost, leading to increased resorption [22]. Conversely, excess blood thyroxine (T4) accelerates bone turnover, increases osteoclast activity, and reduces the density of both trabecular and cortical bone. In postmenopausal women, even subclinical hyperthyroidism is associated with an increased fractures risk, and for alveolar bone, this manifests itself as accelerated marginal resorption and poorer conditions for implant osseointegration.

Testosterone deficiency / Дефицит тестостерона

Although testosterone is considered a key male hormone, it is also important for women, and its decline in postmenopause exacerbates osteoporotic changes. Testosterone can directly as well as indirectly through aromatization to estradiol stimulate osteoblastic activity and increase type I collagen synthesis. Androgen deficiency in women leads to decreased bone anabolism and lowered cortical bone density [23–25]. In the maxillofacial region, it manifests as decreased alveolar bone strength and delayed recovery after surgery.

Cortisol excess / Избыток кортизола

Glucocorticoids exert a strong catabolic bone-related effect. Chronic hypercortisolism including subclinical states (e.g., Cushing's syndrome or prolonged glucocorticoid therapy) suppresses osteoblast function, increases osteocyte apoptosis, and impairs bone matrix formation. Cortisol also elevates RANKL expression and enhances bone resorption [26].

In postmenopause, hypercortisolism is especially detrimental because it acts synergistically with estrogen deficiency, accelerating loss of bone mass, including alveolar bone.

Catecholamines (adrenaline and norepinephrine) excess / Избыток катехоламинов (адреналин и норадреналин)

Activation of the sympathetic nervous system plays an important role in regulating bone metabolism. Adrenaline and norepinephrine act through β_2-adrenergic receptors on osteoblasts, suppressing their activity and stimulating RANKL expression. This leads to enhanced osteoclastogenesis and accelerated bone resorption [27].

In postmenopausal women, increased sympathetic activity associated with stress and metabolic disturbances further exacerbates the loss of mineral density and worsens bone quality in the jaws.

Vitamin D deficiency / Дефицит витамина D

Vitamin D is a key regulator of calcium–phosphorus metabolism and bone mass maintenance. Its deficiency leads to impaired intestinal calcium absorption,

secondary hyperparathyroidism, and increased bone resorption [28].

Vitamin D deficiency is particularly common in postmenopausal women and is associated with lower bone density, increased fracture risk, and greater marginal resorption of alveolar bone. Low vitamin D levels also impair healing after dental implantation and increase the likelihood of peri-implantitis.

Sex hormone-binding globulin / Глобулин, связывающий половые гормоны

Sex hormone-binding globulin (SHBG) level increases in postmenopause, which lowers estradiol and testosterone bioavailability even when their absolute serum concentrations remain within normal range. This exacerbates the deficiency of sex steroids for bone tissue. High SHBG levels are associated with a higher risk of hip and vertebral fractures in older women [29]. For dental practice, increased SHBG implies reduced regenerative capacity of bone and poorer implant osseointegration.

Systemic inflammation / Системное воспаление

Hypoestrogenism in postmenopause is accompanied by activation of pro-inflammatory cytokines, including interleukin (IL) IL-1β, IL-6 and tumor necrosis factor-alpha (TNF-α). These mediators promote RANKL expression on osteoblasts and stromal cells, enhancing osteoclast differentiation and disrupting bone remodeling balance [30].

As a result, both trabecular and cortical bone loss accelerates. IL-6 and TNF-α additionally impair osteoblast function, reducing collagen synthesis and osteoid mineralization. Clinical studies show that women with elevated IL-6 levels experience faster BMD decline regardless of age or body mass index (BMI). In the maxillofacial region, systemic inflammation impairs microcirculation and reduces bone regenerative potential of bone after surgical procedures, negatively affecting implant osseointegration.

Local inflammation / Local inflammation

Postmenopausal osteoporosis is frequently accompanied by inflammatory oral diseases, primarily periodontitis. Reduced bone mineral density renders alveolar bone more sensitive to local inflammatory mediators. Studies show that women with PMO exhibit more pronounced alveolar ridge resorption in periodontitis compared with women with normal BMD [12]. Inflammatory infiltrates in gingival and peri-implant tissues stimulate cytokine expression (IL-1β, TNF-α), matrix metalloproteinases, and prostaglandins, accelerating collagen matrix degradation and bone destruction around teeth and implants. Alveolar bone, being highly metabolically active, resorbs faster than axial skeletal bone. This explains the higher incidence of implant complications – primarily marginal resorption and peri-implantitis in postmenopausal women. Deficiencies in estradiol, progesterone, androgens, calcitonin, vitamin D, IGF-1, and TSH level, as well as excess FSH, parathyroid hormone, thyroxine, cortisol, TNF-α, IL-1β, and IL-6 lead to increased alveolar bone resorption. Additional negative influences include medications, bacterial load, xerostomia, and inflammation in periodontal disease, collectively creating unfavorable conditions for dental implant osseointegration (**Fig. 1**) [12].

Characteristics of alveolar bone in postmenopause / Особенности альвеолярной кости в постменопаузе

Alveolar bone has a higher metabolic turnover than overall skeletal system and responds faster to hormonal changes. This explains why postmenopausal alterations in the jaws often precede systemic declines in BMD detected by densitometry [31].

In PMO, the following changes are typically observed:
- cortical layer thinning;
- reduced number and thickness of trabeculae;
- enlarged inter-trabecular spaces;
- impaired microcirculation.

These alterations decrease the osteointegration potential and require modification of surgical implant placement techniques.

Impact of pharmacological methods for osteoporosis prevention and treatment on dental implant outcome / Влияние фармакологических методов профилактики и лечения остеопороза на исходы дентальной имплантации

Postmenopausal osteoporosis affects all stages of osseointegration spanning from primary stability to the long-term maintenance of the dental implant. Under conditions of reduced bone density, the bone-to-implant contact decreases, and the risks of micromovements as well as fibrous integration increase [6, 32]. Systemic inflammation typical to hypoestrogenism further worsens conditions by activating osteoclasts and increasing resorption around the implant neck [18]. Clinically, this may manifest as accelerated marginal bone loss during the first 12 months post-implantation [33].

Antiresorptive therapy: a dual role / Антирезорбтивная терапия: двойственная роль

Antiresorptive medications (bisphosphonates, denosumab) are the standard of care in treating postmenopausal osteoporosis [4, 10, 34]. On one hand, they increase bone mineral density and may improve long-

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term implant stability; on the other hand, long-term use is associated with the risk of medication-related osteonecrosis of the jaw [35].

The risk of osteonecrosis is particularly high for intravenous bisphosphonates and in the presence of contributing factors such as traumatic oral surgery, therapy duration > 4 years, and concurrent use of glucocorticoids or anti-angiogenic drugs [36].

Osteoanabolic therapy / Остеоанаболическая терапия

Treating postmenopausal osteoporosis is crucial for optimizing dental implant outcomes. Antiresorptive medications reliably increase BMD and reduce fracture risk, but long-term use is connected with drug-associated jaw osteonecrosis, especially following oral surgery [12].

By contrast, osteoanabolic therapy with teriparatide is considered promising for improving osseointegration. Clinical studies show that short-term teriparatide therapy improves bone–implant contact and enhances primary stability [37].

Teriparatide (rhPTH 1–34), a recombinant fragment of parathyroid hormone (first 34 amino acids) with anabolic effects when administered intermittently, is the first osteoanabolic medication approved for the treatment of postmenopausal osteoporosis.

Mechanisms of action include:

- stimulation of osteoblast proliferation and differentiation;
- enhanced type I collagen synthesis;
- increased alkaline phosphatase activity;
- improved osteoid mineralization;
- upregulation of IGF-1, further enhancing bone formation.

Teriparatide enhances bone regeneration in the maxilla in osteoporotic women and accelerates osseointegration of dental implants [38]. A pilot study showed that a short 6-week teriparatide course following implant placement increased the bone-to-implant contact area and improved implant stability [37].

Thus, teriparatide is the most extensively studied osteoanabolic therapy in dentistry and may improve implant outcomes in women with postmenopausal osteoporosis.

Abaloparatide is a synthetic analog of parathyroid hormone-related peptide (PTHrP 1–34) developed as an alternative to teriparatide. Its mechanism is similar but offers several advantages: more selective binding to the PTH receptor in its “anabolic conformation”; strong anabolic effect with less stimulated resorption.

In the ACTIVE trial, abaloparatide vs. placebo increased lumbar spine BMD by 11 % over 18 months and reduced new vertebral fractures by 86 % versus placebo [39].

The VERO trial found comparable efficacy between abaloparatide and teriparatide, with abaloparatide related to fewer cases of hypercalcemia [40].

Although direct implantology studies are limited, experimental data show that abaloparatide enhances osteoblast proliferation and bone matrix formation around implants, suggesting potential relevance in dentistry.

Romosozumab is a monoclonal antibody that inhibits sclerostin, an endogenous inhibitor of the Wnt/β-catenin signaling pathway, which is central to osteoblast activity.

Uniquely, romosozumab combines two mechanisms: stimulation of osteoanabolic activity (activation of osteoblasts); suppression of bone resorption (reduction of RANKL expression).

In the FRAME trial, 12 month-romosozumab treatment increased lumbar spine and hip BMD by 13 % and 6 %, respectively, significantly exceeding antiresorptive therapy effects [41].

In the ARCH trial, romosozumab reduced new vertebral fractures incidence by 73 % compared with alendronate [42].

For dental implantology, romosozumab is particularly promising because it simultaneously enhances bone formation and reduces resorption potentially lowering marginal bone loss around implants and improving long-term stability.

Impact of menopausal hormone therapy on osteoporosis and the effectiveness of dental implantation / Влияние менопаузальной гормональной терапии на остеопороз и эффективность дентальной имплантации

Menopausal hormone therapy (MHT) with estrogens and progesterone has been shown to retard bone loss, reduce fracture risk, and improve bone microarchitecture [1, 4, 12]. In alveolar bone, this translates into increased mineralization, thicker trabeculae, and improved vascularization.

Clinical studies show that women receiving MHT exhibit significantly less marginal bone loss around implants compared with controls during 5-year follow-up [43].

Use of bioidentical 17β-estradiol (gel) and micronized progesterone (gel or capsules) maintains stable hormone levels without significant impact on liver metabolism or coagulation [44].

Estradiol increases osteoprotegerin expression, reduces RANKL, and improves microcirculation, that is critical for early osseointegration [12]. Progesterone stimulates osteoblasts, enhances mineralization, and increases mechanical strength of newly formed bone [13].

In postmenopausal implant patients, combining estradiol and progesterone may improve primary stability and reduce early implant loss, although large-scale randomized trials are needed for conclusive evidence [44].

A systematic review of the role of reproductive hormones in bone metabolism has shown that bone remodeling is regulated not only by estrogens and

androgens. Follicle-stimulating hormone, luteinizing hormone (LH), prolactin, inhibin, activin, and several other peptide factors also play crucial roles. In postmenopause, FSH levels increase and directly stimulate osteoclastic activity, accelerating bone resorption. At the same time, the decline in IGF-1 and growth hormone limits *de novo* bone formation and slows reparative processes. Disrupting the balance among such hormones lead to trabecular bone loss, impaired microcirculation, and increased fragility of the alveolar ridge. Thus, the spectrum of endocrine changes in menopause extends far beyond estrogen deficiency and encompasses multifactorial influences on bone, collectively determining the risk of osteoporotic changes [16].

Current evidence suggests that optimizing hormonal status in postmenopause particularly through the use of bioidentical forms of estradiol and progesterone may improve dental implant outcomes by:

1. increasing bone mineral density and maintaining microarchitectural integrity;
2. improving microcirculation in the implant site;
3. dampening systemic inflammation;
4. supporting osteoblastic activity.

However, definitive conclusions require large-scale multicenter randomized clinical trials with sufficient follow-up duration (≥ 5 years) and standardized methods for evaluating dental implant osseointegration.

A meta-analysis of randomized clinical trials demonstrated that combined estrogen–progestin therapy exerts a more pronounced effect on bone mineral density than estrogen monotherapy. The analysis assessed more than 1,000 postmenopausal women and compared changes in spinal BMD across various MHT regimens. It was found that adding a progestin to estrogen provided a significant additional increase in bone mass – on average +0.68 % per year. This effect was observed across different therapeutic regimens, including low-dose estrogen therapy. The findings confirm the synergistic action of the two hormones: estrogens reduce bone resorption, while progestins stimulate osteoblast activity and osteoid mineralization. This combined mechanism helps preserve trabecular structure and reduces the risk of cortical bone resorption [14].

Conclusion / Заключение

Postmenopausal osteoporosis is a significant risk factor for dental implant failure due to complex alterations in bone metabolism, microcirculation, and inflammatory status. Menopausal hormone therapy using bioidentical forms of estrogens and progestogens, when appropriately selected and used in the absence of contraindications, may serve as an effective adjunct in osteoporosis prevention and management for women planning dental implantation. In select cases, combining MHT with osteoanabolic therapy may further enhance the effectiveness of dental treatment.

An interdisciplinary approach involving an endocrinologist, gynecologist, and dentist is essential for individualized treatment planning, hormonal profile optimization, and selection of the optimal timing for implantation.

ARTICLE INFORMATION	ИНФОРМАЦИЯ О СТАТЬЕ
Received: 31.08.2025. **Revision received:** 29.09.2025. **Accepted:** 21.10.2025. **Published online:** 27.10.2025.	**Поступила:** 31.08.2025. **В доработанном виде:** 29.09.2025. **Принята к печати:** 21.10.2025. **Опубликована онлайн:** 27.10.2025.
Author's contribution	**Вклад авторов**
All authors contributed equally to the article.	Все авторы внесли равный вклад в написание и подготовку рукописи.
All authors have read and approved the final version of the manuscript.	Все авторы прочитали и утвердили окончательный вариант рукописи.
Conflict of interests	**Конфликт интересов**
The authors declare no conflict of interest.	Авторы заявляют об отсутствии конфликта интересов.
Funding	**Финансирование**
The study did not receive any external funding.	Исследование не имело спонсорской поддержки.
Publisher's note	**Комментарий издателя**
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