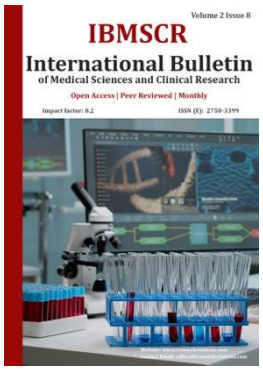

ASSESSMENT OF BONE TISSUE CONDITION DURING DENTAL IMPLANTATION IN PATIENTS WITH SOMATIC DISEASES

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Summary: The issue of dental implantation in patients with chronic somatic diseases is becoming increasingly important in modern dental practice. The increase in life expectancy, the growing prevalence of endocrine, cardiovascular, and metabolic disorders, as well as age-related changes in bone tissue, necessitate a more thorough preparation of patients for implant treatment. Particular attention is paid to the condition of bone metabolism, since disturbances in bone remodeling processes may affect implant osseointegration and the course of the postoperative period. In this regard, the study of indicators characterizing bone tissue condition, as well as factors contributing to the development of complications during dental implantation, remains highly relevant. A comprehensive assessment of the structural and functional characteristics of bone tissue makes it possible to determine preventive strategies and optimize the management of patients with concomitant somatic pathology.

Key words: dental implant, somatic pathology, somatic diseases, osteopenia, osteoporosis. markers of osteogenesis, calcium-regulating hormones, densitometry.

Introduction: Currently, somatic diseases are considered relative contraindications to dental implantation; however, in certain cases, such as decompensated diabetes mellitus and chronic systemic diseases, the risk of complications associated with implant treatment may significantly increase [8,11,12,17]. This may be explained by several well-known factors, including decreased general and local immunity, impaired microcirculation, reduced adaptive response to surgical intervention, as well as an increased risk of both local and systemic postoperative complications. Nevertheless, patients with such pathologies are frequently encountered in everyday clinical practice, accounting for approximately 7–10% of cases.

Osteoporosis is a disease affecting the skeletal system and may result from multiple etiological factors, while its prevalence increases with age [4,5,15]. According to the World Health Organization, osteoporosis currently ranks fourth among non-infectious diseases in terms of mortality, following cardiovascular diseases, oncological disorders, and diabetes mellitus.

At present, osteoporosis and osteopenia remain highly relevant due to their widespread occurrence among patients seeking dental implantation. These conditions may be associated with hormonal imbalance caused by age-related and pathological changes in the human body, as well as degenerative-dystrophic disorders. Additional contributing factors may include consanguineous marriages, pregnancy-related stress, nutritional deficiencies, occupational hazards, and adverse environmental conditions.

The introduction of modern diagnostic and therapeutic technologies, including micro-computed tomography, osteodensitometry, biochemical markers of bone metabolism, and novel pharmacological agents, allows early detection of degenerative changes in the

musculoskeletal system and timely implementation of preventive and therapeutic measures aimed at reducing the risk of impaired structural and functional properties of bone tissue and pathological fractures associated with decreased bone mass and altered bone architectonics leading to osteoporosis. Among the key scientific directions in the study of bone structural and functional disorders are ultrasound bone scanning and identification of secondary causes of osteoporosis associated with disturbances in calcium-regulating hormones and bone formation markers, which are closely related to age and concomitant diseases [9].

There is a certain risk associated with dental implantation in patients with somatic diseases, since surgical intervention may provoke exacerbation of the underlying pathology. Fear and pain experienced by the patient may contribute to myocardial ischemia, bronchospasm, and hypertensive crisis [8,11,12], while the somatic disease itself may negatively affect wound healing processes.

It is also well known that pathogenic microflora accumulated in periodontal pockets may release endotoxins into the bloodstream, which subsequently induce endothelial damage through inflammatory mediators. In patients with arterial hypertension, diabetes mellitus, and chronic obstructive pulmonary disease, where endothelial dysfunction already exists, inflammatory changes may extend beyond periodontal tissues and spread systemically, potentially resulting in infectious-toxic shock or sepsis.

It should also be noted that chronic obstructive pulmonary diseases rank third worldwide in terms of prevalence, disability, and mortality.

Every year, the number of patients with arterial hypertension increases by several hundred thousand worldwide. Consequently, the likelihood of implantologists encountering patients with a history of hypertension continues to rise annually.

For implantologists, it is important to consider that prolonged arterial hypertension and long-term antihypertensive therapy may lead to the phenomenon of vascular rarefaction, resulting in microcirculatory disturbances and vascular wall alterations that can adversely affect tissue repair processes due to a reduction in the total exchange surface of blood vessels [2]. Furthermore, multicenter evidence-based studies have demonstrated that the progression of complications such as retinopathy, nephropathy, and neuropathy depends not only on glycemic levels but also on the duration of hyperglycemia exposure [10].

Thus, for a dental implantologist, not only the established diagnosis but also the condition of adaptive and compensatory mechanisms is of major importance. Therefore, the decision regarding the feasibility and extent of dental implantation should be based not only on the diagnosis itself but also on the duration and effectiveness of the corrective treatment provided.

Objective: To identify early impairments in the structural and functional characteristics of bone tissue in patients with osteopenia and osteoporosis who are being evaluated for dental implantation, and to propose preventive recommendations for reducing the risk of progression of these impairments.

Materials and Methods: A combined clinical and laboratory examination was carried out in 88 patients with partial edentulism who presented for outpatient dental treatment, aged 43 to 64 years. The study population was predominantly female — 61 patients (69%); males numbered 27 (31%). The comorbidity profile included: hypertensive disease — 33 patients (38%), peptic ulcer disease — 7 patients (8%), and diabetes mellitus — 6 patients (7%). The control group consisted of 42 generally healthy individuals of both sexes.

The following ultrasound densitometry parameters were assessed: Speed of Sound (SOS, m/s), Broadband Ultrasound Attenuation (BUA, dB/MHz), and Stiffness Index (SI, %).

Serum levels of calcium-regulating hormones were measured in all examined patients, including: parathyroid hormone (PTH), calcitonin (CT), and calcitriol (CTr). PTH and CT were determined using the reagent kits "PTH-ELISA" (XEMA, Russia) and "Calcitonin-ELISA" (XEMA, Russia), respectively. Quantitative determination of CTr in serum was performed by enzyme-linked immunosorbent assay (ELISA) using the 1,25 Vitamin D ELISA kit (Immundiagnostik, Germany). As a marker of bone formation, serum osteocalcin (OC) was measured by ELISA using the N-MID Osteocalcin kit (Nordic Bioscience Diagnostics A/S, Canada). To assess the intensity of bone resorption, the level of deoxypyridinoline (DPD) was determined in the first morning fasting urine specimen using the Metra DPD EIA kit (Quidel Corporation, USA) by ELISA, expressed as a ratio to creatinine in the same urine sample. The calcium-to-creatinine ratio (Ca/Cr) was also calculated in the first two-hour fasting morning urine portion.

Statistical analysis was performed using Statistica 6.0 software; both parametric and non-parametric statistical methods were applied for data processing.

Results: Analysis of the structural and functional state of bone tissue in the examined patients revealed that among the 88 patients, only 29 (33%) demonstrated densitometric parameters — SI, SOS, and BUA — within the normal range for their respective age groups. The remaining 59 (67%) patients exhibited impairments in the structural and functional properties of bone tissue: osteopenia of varying severity was identified in 47 (79.7%), and osteoporosis in 12 (20.3%).

Age-stratified analysis of ultrasound densitometry data demonstrated significantly reduced SOS, SI, and BUA values across all age groups. Osteopenia was observed in 8 patients (17.1%) aged 43–49 years, in 23 patients (48.9%) aged 50–59 years, and in 16 patients (34.0%) aged 60–64 years. Osteoporosis was recorded in 3 patients (25.0%) aged 43–49 years, in 7 patients (58.3%) aged 50–59 years, and in 2 patients (16.7%) aged 60–64 years.

Analysis of ultrasound densitometry data stratified by nosological form and sex revealed that in postmenopausal women, as well as in patients with diabetes mellitus, all method parameters were significantly lower ($p < 0.001$).

The state of hormonal regulation of bone formation and resorption processes plays an important role in the pathogenesis of structural and functional bone disorders, including in patients who have undergone dental implantation. The principal calcium-regulating hormones — calcitonin, calcitriol, and parathyroid hormone — were measured in 59 patients who had undergone dental implantation.

Calcitonin suppresses the functional activity of osteoclast and osteocyte cell populations and thereby inhibits bone resorption. This component of anti-hypercalcaemic action, mediated by the direct effect of calcitonin on osteolytic activity, operates independently of bone tissue's self-regulatory mechanisms and of renal and intestinal function.

Serum calcitonin measurement revealed a marked and statistically significant reduction in the overall patient group — 3.56 ± 2.01 versus the control group value of 23.3 ± 2.01 ($p < 0.001$) — indicating predominance of bone resorption processes associated with osteoclast activation and suppression of osteoblast functional activity. In the 12 patients with osteoporosis, the calcitonin level was not only significantly lower than the control group value — 2.17 ± 1.04 versus 23.3 ± 2.01 ($p < 0.001$) — but also lower than the value in the 47 patients

with osteopenia — 3.46 ± 1.08 versus 23.3 ± 2.01 ($p < 0.001$). The degree of impairment in bone formation processes showed a distinct correlation with age, sex, and concurrent systemic pathology.

Calcitriol is a steroid hormone that regulates calcium metabolism. In intestinal cells, it induces the synthesis of Ca^{2+}-carrier proteins that facilitate the absorption of calcium ions and phosphates from the intestinal lumen into epithelial cells, and their subsequent transport from cells into the bloodstream against a concentration gradient across intestinal membranes. In the kidneys, calcitriol stimulates the reabsorption of calcium ions and phosphates. At low calcium ion concentrations, calcitriol promotes the mobilisation of calcium from bone tissue.

Serum calcitriol measurement revealed a statistically significant reduction in the overall patient group — 20.38 ± 2.75 versus the control group value of 60.8 ± 3.86 ($p < 0.001$) — indicating enhanced osteoclast activity and increased bone resorption. In the osteoporosis group, the calcitriol level was 15.23 ± 2.11, significantly lower than both the control group value (60.8 ± 3.86; $p < 0.001$) and the osteopenia group value of 21.71 ± 2.76 ($p < 0.05$), indicating marked suppression of osteoblast function and predominance of bone resorption over bone formation.

Parathyroid hormone (PTH) regulates serum calcium concentration. Its physiological action involves inhibiting bone tissue formation by influencing osteoblast and osteocyte populations, which in turn release insulin-like growth factor-1 and cytokines that stimulate osteoclast metabolism; osteoclasts secrete alkaline phosphatase and collagenase, leading to bone matrix degradation. PTH indirectly increases tubular reabsorption of calcium cations, promotes phosphate excretion by the kidneys, and enhances intestinal calcium absorption (by inducing calcitriol synthesis). The net effect of PTH is an increase in plasma calcium concentration, a decrease in bone calcium content (demineralisation of the bone matrix), and a decrease in plasma phosphate concentration.

Serum PTH measurement revealed a significantly elevated level in the overall patient group — 51.33 ± 2.43 versus the control group value of 43.37 ± 2.16 ($p < 0.01$) — indicating osteoclast activation and intensification of bone resorption. In the osteoporosis group, PTH significantly exceeded the control group value — 53.46 ± 2.17 versus 43.37 ± 2.65 ($p < 0.001$) — as well as the osteopenia group value of 42.53 ± 3.09 ($p < 0.001$). The elevated serum PTH in patients with confirmed osteoporosis indicated the systemic involvement of the hormonal profile and numerous biochemical processes in bone formation and resorption.

Among the biochemical constants reflecting bone remodelling processes and osteogenesis, the most informative are osteocalcin and deoxypyridinoline.

Osteocalcin is the most informative marker of bone formation. It is produced by osteoblasts during osteosynthesis and partly enters the bloodstream. Serum osteocalcin measurement in the overall patient group demonstrated a significant reduction — 79.11 ± 3.44 versus the control group value of 117.0 ± 4.97 ($p < 0.001$) — reflecting decreased osteoblast function and consequently insufficient bone formation. This was most pronounced in the osteoporosis group, where the osteocalcin level was 68.19 ± 5.71, significantly lower than the control group (117.0 ± 4.97; $p < 0.001$) and the osteopenia group value of 81.94 ± 3.65 ($p < 0.001$).

Deoxypyridinoline/creatinine ratio serves as a marker of bone resorption, being released into the bloodstream from bone tissue as a result of osteoclast-mediated degradation. A

statistically significant elevation in deoxypyridinoline was observed in the examined patients — 6.28 ± 0.21 versus the control group value of 4.64 ± 0.83 ($p < 0.01$) — indicating osteoclast activation and predominance of bone resorption. In the osteoporosis group, the deoxypyridinoline level was significantly higher than the control group value — 6.98 ± 0.34 ($p < 0.001$) — and significantly higher than in the osteopenia group — 6.22 ± 0.29 ($p < 0.05$).

Table 1.

Levels of Calcium-Regulating Hormones and Bone Remodelling Markers

Indicator	Control Group	Study Group (all patients)
Calcitonin	23.3 ± 2.01; p < 0.001	3.56 ± 2.01
Calcitriol	60.8 ± 3.86; p < 0.001	20.38 ± 2.75
Parathyroid hormone (PTH)	43.37 ± 2.16; p < 0.01	51.33 ± 2.43
Osteocalcin	117.0 ± 4.97; p < 0.001	79.11 ± 3.44
Deoxypyridinoline / creatinine	4.64 ± 0.83; p < 0.01	6.28 ± 0.21

Table 2.

Levels of Calcium-Regulating Hormones in Patients with Structural and Functional Bone Impairments

Indicator	Osteoporosis Group	Osteopenia Group
Calcitonin	2.17 ± 1.04; p < 0.001	3.46 ± 1.08; p < 0.001
Calcitriol	15.23 ± 2.11; p < 0.001	21.71 ± 2.76; p < 0.05
Parathyroid hormone (PTH)	53.46 ± 2.17; p < 0.01	42.53 ± 3.09; p < 0.001
Osteocalcin	68.19 ± 5.71; p < 0.001	81.94 ± 3.65; p < 0.001
Deoxypyridinoline / creatinine	6.98 ± 0.34; p < 0.001	6.22 ± 0.29; p < 0.05

Note: All p-values are given in comparison with the control group.

Conclusions: The results of this study demonstrate the presence of marked impairments in the structural and functional properties of bone tissue in patients undergoing dental implantation. These impairments are manifested by a statistically significant reduction in the principal densitometric parameters — SOS, BUA, and SI. They are most pronounced in individuals aged over 60 years, in postmenopausal women, and in patients with systemic diseases (including diabetes mellitus), and are accompanied by an imbalance of calcium-regulating hormones (decreased calcitonin and calcitriol levels, elevated parathyroid hormone) and alterations in bone remodelling markers (decreased osteocalcin, elevated deoxypyridinoline).

The bone tissue impairments identified in patients undergoing dental implantation indicate the necessity of preparatory measures prior to the surgical procedure, aimed at correcting bone remodelling disorders and restoring the strength characteristics and architecture of bone tissue, as well as the need for long-term monitoring of structural, functional, and biochemical parameters of bone metabolism at all stages of treatment.

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