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MODERN CONCEPTS OF DENTAL IMPLANTS OSSEOINTEGRATION (LITERATURE REVIEW)

Tillakhodjaeva Madina Maxirovna
Assistant at the department of Faculty Prosthetic Dentistry, Tashkent State Medical University,
Akbarova Afruza Avzalovna
Student of the Faculty of Dentistry Tashkent State Medical University

Annotation: *The rapid development of dental implantation technologies and their extensive use in restorative dentistry have stimulated growing interest in the mechanisms governing the integration of implants with bone tissue. This review was conducted to evaluate and organize available domestic and international data concerning the osseointegration of titanium dental implants.*

A review of contemporary literature indicates that modern investigative methods and interdisciplinary scientific approaches have substantially revised traditional views on implant integration. Particular attention has been given to the biological mechanisms and cellular interactions occurring at the implant–bone interface during various stages of healing. Current evidence suggests that titanium structures cannot be regarded as completely biologically inert, and that the process of osseointegration should also be examined from an immunological standpoint.

Efficient regeneration of bone tissue in maxillofacial diseases, traumatic conditions, and dental implantation procedures requires a balanced interaction between the nervous, endocrine, and immune systems that regulate

osteogenesis. These processes are influenced by multiple biologically active mediators, including serotonin, beta-endorphin, and other regulatory molecules.

Today, implant-supported rehabilitation is widely used in modern orthopedic dentistry for the treatment of patients with partial or total tooth loss. Worldwide clinical experience confirms the effectiveness of dental implants in a variety of clinical situations, including both fixed prosthetic restorations and stabilization of removable dentures, providing long-term functional stability and predictable treatment success [1,8].

Key words: *osteointegration, osteoconduction phase, alkaline phosphatase.*

In recent years, prevalence of dental implantation has led to growing scientific interest in the biological mechanisms underlying the interaction between implants and bone tissue. The most favorable outcome of this interaction is considered to be osseointegration, a concept introduced by P.-I. Brånemark, who described it as a direct structural and functional connection between living bone and the implant surface without the presence of intervening connective tissue [2]. Successful osseointegration is regarded as one of the key conditions for long-term clinical success in implant-supported prosthetic rehabilitation [1,6]. The phenomenon of osseointegration was first identified accidentally during Brånemark's experimental studies of bone microcirculation using an optical chamber implanted into rabbit tibial bone. Since that discovery, extensive scientific research has been devoted to understanding the biological and physiological mechanisms involved in

implant integration, and the amount of available data continues to increase [3–6].

According to A. A. Kulakov, integration of an implant into bone tissue should be considered a dynamic biological process based on the interaction between living tissues and inert biomaterials. This interaction involves the maintenance of compensatory, adaptive, and homeostatic mechanisms that ensure stable coexistence within a unified system. Criteria indicating successful integration include the absence of inflammatory, allergic, or necrotic reactions in peri-implant tissues, the development of morphofunctional bone characteristics at the implant–bone interface, and the long-term preservation of these structural properties [7,10].

According to published studies, several patterns of tissue interaction may develop at the implant–bone interface. In the classical interpretation proposed by P.-I. Brånemark, osseointegration represents direct contact between living bone tissue and the implant surface without the interposition of connective tissue. Fibro-osseointegration, in contrast, is characterized by the formation of a connective tissue layer composed of collagen fibers and coarse fibrous tissue between the implant and the surrounding bone. Another possible outcome is connective tissue integration, in which the implant becomes completely encapsulated by fibrous connective tissue. The literature generally considers the first two variants as acceptable physiological responses, whereas complete fibrous encapsulation is regarded as a sign of implant rejection.

For many years, the theory of blood clot retraction was considered the predominant explanation for the development of osseointegration [10,16]. Based on this concept, implant integration begins with the osteoconduction stage, during which mesenchymal cells and osteoblasts migrate toward and

adhere to the implant surface through the residual fibrin clot. This phase is followed by osteoinduction, characterized by the formation of new bone tissue and mineral deposition within the developing extracellular matrix. The final regenerative stage is bone remodeling, a prolonged process involving alternating cycles of bone resorption and new bone formation around the implant surface.

Implant placement itself is regarded as a form of surgical trauma that initiates inflammation, early bone resorption, and a cascade of vascular and cellular responses aimed at tissue regeneration. Adequate blood circulation and vascular integrity in the surgical area play a crucial role in successful healing, since impaired blood supply or ischemia increases the risk of fibrous or cartilaginous tissue formation instead of normal bone regeneration [1,5,11,8].

Studies have demonstrated that even under conditions of high insertion speed and excellent primary implant stability, a microscopic gap of approximately 60 μm may persist between the implant and adjacent bone tissue. In cases of increased surgical trauma, this space may locally expand to 100–500 μm. The gap becomes filled with blood and tissue fluid containing proteins and biologically active molecules essential for initiating osseointegration. Surface properties of titanium implants influence the adsorption and spatial arrangement of these proteins, while membrane receptors of surrounding cells interact directly with the implant surface, promoting initial cellular attachment [19,20]. At the early stages of osseointegration, extracellular matrix proteins such as fibronectin, together with transmembrane adhesion molecules known as integrins, play a major role in cellular recognition and attachment to the implant surface [21,22]. Blood originating from the bone bed forms a fibrin clot enriched with platelets, growth factors, insulin-like growth factor, transforming

growth factor, and other mediators that stimulate angiogenesis and tissue repair [13,18,23]. The fibrin scaffold facilitates the migration of osteogenic cells toward the implant surface under the influence of platelet-derived growth factors. These mediators also attract fibroblasts and undifferentiated precursor cells to the fibrin matrix, promoting their proliferation and differentiation [15]. The biological events occurring during this phase largely determine the дальнейший успех интеграции имплантата. Stable adhesion of the clot to the implant surface and formation of fibrin connections between viable bone tissue and the implant create favorable conditions for osteogenic cell proliferation and subsequent bone tissue formation along the fibrin network

At the same time, implant integration solely due to contact osteogenesis is rather an idealized model. Most likely, both contact and distant osteogenesis processes occur simultaneously in different areas of the "bone-implant" zone.

The latter is characterized by the formation of bone tissue not on the surface of the implant, but on the surface of the surrounding bone: osteogenic cells of the implant bed synthesize bone matrix towards the surface of the implant.

Already in the first days after implant placement, osteoblasts attach to its surface. These cells synthesize osteogenesis marker proteins — osteopontin, osteocalcin, sialoprotein — which promote the adhesion of osteogenic cells to the implant and the fixation of mineral compounds in the newly formed organic bone matrix. The next stage is the formation of a collagen matrix directly on the implant surface, followed by the deposition of an osteoid substance that transforms into bone tissue [19].

Mineralization of the bone matrix is associated with the accumulation of calcium and phosphorus ions and is ensured not only by calcium-binding

proteins, but also by the participation of phospholipids and chondroitin sulfate of the ground substance [4,17]. Subsequently, young bone tissue undergoes a long-term structural reorganization. This stage—remodeling—combines two opposing processes: bone resorption and new bone formation. Resorption of immature bone tissue is mainly carried out under the action of matrix metalloproteinases secreted by osteoclasts, which is accompanied by an increase in acid phosphatase activity. At the same time, the formation of new bone in the direction of the implant surface is ensured by the high functional activity of osteoblastic cells and is accompanied by the expression of alkaline phosphatase [22]. The remodeling process is closely dependent on the load conditions on the implant and ultimately leads to the replacement of immature bone with a more mature and functionally complete structure.

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