eoss-conf.com

ISSUE
№46

EUROPEAN OPEN
SCIENCE SPACE

COLLECTION OF SCIENTIFIC PAPERS

1ST INTERNATIONAL
SCIENTIFIC
AND PRACTICAL
CONFERENCE

SCIENTIFIC RESEARCH: EMERGING THEORIES AND PRACTICAL BREAKTHROUGHS

JULY 28-30, 2025, EDINBURGH, SCOTLAND

EUROPEAN OPEN SCIENCE SPACE

Proceedings of the 1st International Scientific and Practical Conference

"Scientific Research: Emerging Theories and Practical Breakthroughs"

July 28-30, 2025

Edinburgh, Scotland

Collection of Scientific Papers

Edinburgh, 2025

UDC 01.1

Collection of Scientific Papers with the Proceedings of the 1st International Scientific and Practical Conference «Scientific Research: Emerging Theories and Practical Breakthroughs» (July 28-30, 2025. Edinburgh, Scotland). European Open Science Space, 2025. 148 p.

ISBN 979-8-89704-972-1 (series)
DOI 10.70286/EOSS-28.07.2025

The conference is included in the Academic Research Index ReserchBib International catalog of scientific conferences.

The conference is registered in the database of scientific and technical events of UkrISTEI to be held on the territory of Ukraine (Certificate №534 dated 16.06.2025).

The materials of the collection are presented in the author's edition and printed in the original language. The authors of the published materials bear full responsibility for the authenticity of the given facts, proper names, geographical names, quotations, economic and statistical data, industry terminology, and other information.

ISBN 979-8-89704-972-1 (series)

EUROPEAN OPEN SCIENCE SPACE

Section: Medicine

PATHOLOGICAL AND HISTOLOGICAL EXAMINATION OF SELECTED SKELETAL SITES IN DEFORMING OSTEODYSTROPHY

Sukhonosov Roman
Ph.D., Associate Professor
Department of Human Anatomy, Clinical Anatomy, and Operative Surgery
Nadozirna Sofiia
Student, 2nd Medical Faculty
Ushakova Mariia
Intern Doctor
Halycha Mariia
Student, 2nd Medical Faculty
Kharkiv National Medical University, Kharkiv, Ukraine

Relevance: Deforming osteodystrophy is a rare but clinically significant skeletal disease characterized by chronic disruption of bone remodeling processes. Lesions in certain skeletal regions cause pronounced morphological changes accompanied by deformities and loss of functional stability of the bones. Pathoanatomical and histological studies allow for detailed investigation of these processes, clarification of pathogenetic mechanisms, and provide a foundation for improving diagnostic approaches.

The aim of the study: To characterize morphological changes in specific skeletal regions in deforming osteodystrophy through pathoanatomical and histological analysis, to determine the features of bone remodeling and their significance for diagnosis and understanding of the disease pathogenesis.

Materials and methods: Theoretical: review and analysis of scientific and methodological literature. Practical: original research.

Results and conclusions: Despite the fact that almost any bone may be involved, the axial skeleton is predominantly affected; in numerous cases, lesions of the sacrum, pelvic bones, spine, skull, and femur accounted for approximately 70-80%.

Pathoanatomical features vary from bone to bone and depend on the stage of the disease. In the skull, areas of focal demineralization are clearly identified, where there is an almost complete absence of compact bone in the inner and outer tables of the calvaria, and the diploë comes almost directly to the surface. The osteolytic area has a purple-red color due to prolonged presence of bone marrow and increased vascularization, which is clearly visible through the thinned bone and depends on the disease stage. The frontal and occipital bones are most commonly affected, and the process extends across the cranial sutures. The demineralized area may border more

typical lesions of deforming osteodystrophy, where the skull is thickened and has a grayish-white color. One may observe alternating zones of lytic and osteoblastic phases of the disease. The changes occur mainly in the calvaria and are most pronounced in the occipital region.

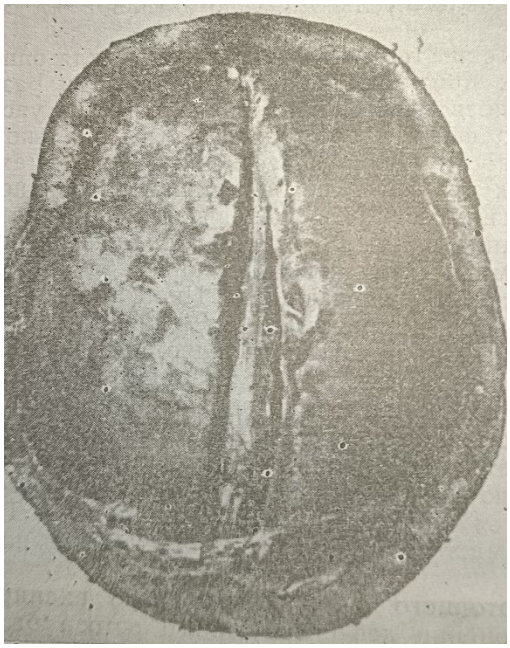

Fig. 1 (Massive skull thickening causing significant reduction of intracranial space)

Diffuse involvement of the pelvic bones is more often seen in the polyostotic form of the disease. The pelvic outlet may be narrowed. The sacrum is affected when the pelvic bones are involved. In cases of involvement of the superior pubic ramus, the process is sharply limited by the symphysis. Within the affected segment(s), the involvement of a vertebra or sacrum is usually total, though changes are more pronounced in the bodies than in the arches and processes.

In long bones, deforming osteodystrophy usually begins at one end and spreads toward the diaphysis. The thickened compact bone has a grayish-red coloration, unlike the normal creamy tone. These two features are relatively easy to distinguish. Sometimes, deforming osteodystrophy of the bone adjacent to a joint leads to osteoarthritis. However, radiological data suggest a low frequency of degenerative joint disease.

The histological picture of bone in deforming osteodystrophy depends on the stage of the disease. The initial stage of osteoclastic resorption is seen at the border between the affected and adjacent normal tissue. The number of resorption lacunae increases in the existing bone trabeculae, which may be accompanied by fibrosis of the intertrabecular bone marrow, though not always. Osteoclasts in deforming osteodystrophy are larger than normal and have a significant number of nuclei (up to 100). In compact bone, the same increased osteoclastic activity is observed as in cancellous bone, and slight fibrosis is noted in the osteons. Vascularization increases

in both types of bone tissue. Osteoblastic activity is always observed following resorption.

Adjacent to the zone of active osteoclasts is the area of osteoblastic activity, where coarse-fibered bone tissue is formed, followed by lamellar bone developing on the newly formed trabecular base.

The intertrabecular space contains vascular fibrous tissue where new bone forms. Small clusters of hematopoietic cells may persist. All this characterizes the active or osteoblastic phase of the disease, during which, after alternating periods of osteoclastic and osteoblastic activity, the classical mosaic structure of bone tissue in deforming osteodystrophy is formed. This pattern, more or less specific to the pathology, arises because bone is resorbed and rebuilt in a more random than physiologically determined manner.

The mosaic pattern is usually observed in sections stained with hematoxylin and eosin, where cement lines are seen inside disorganized bone, and is clearly visible under polarized light microscopy, where lamellae are oriented in different directions in neighboring areas of the same bone. When interpreting this situation, caution is required, as similar changes may occur due to alternating resorption and bone formation in hyperparathyroidism, especially in renal osteodystrophy.

In the inactive phase of the disease, coarse trabecular bone and thickened disorganized compact bone are formed of randomly arranged bone blocks, giving a mosaic appearance. Most of the bone surface lacks osteoclasts and osteoblasts, although mild residual cellular activity may be observed. The fibrous tissue filling the intertrabecular spaces during the active stage is replaced by adipocytes and hematopoietic tissue.

Although the symptoms of deforming osteodystrophy in the final inactive stage are well known, it is always important to consider this condition when examining bone biopsies taken from middle-aged and elderly patients. The resorptive phase is not always easily recognized, especially in the absence of the mosaic pattern. There is a potential confusion between newly formed periosteal bone and osteosarcoma, as well as between hyperparathyroidism and deforming osteodystrophy. Errors are also possible in differential diagnosis with fibrous dysplasia, callus, and osteosclerotic metastatic carcinoma.

Histomorphometric studies of deforming osteodystrophy using double tetracycline labeling showed that increased bone density results from excessive bone matrix production by osteoblasts. The increase in bone cell populations results from an elevated “birth rate” of basic multicellular units. Osteoblast-covered surfaces are significantly enlarged and cause an increase in the volume and surface area of osteoid in deforming osteodystrophy. There is a marked increase in trabecular resorption surfaces and periosteocytic spaces, although this likely reflects differences between woven and lamellar bone rather than periosteocytic osteolysis. The potential risks associated with interpreting a focal disease with inconsistent signs depending on the activity phase, compared to evaluating a generalized bone disease, should be evident.