Genij Ortopedii. 2022. Vol. 28, no. 2. P. 204-210.

Original article

https://doi.org/10.18019/1028-4427-2022-28-2-204-210

Trabecularization of the cortical plate in chronic osteomyelitis

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Abstract

Introduction Literature data and analysis of our own material indicate a change in the density of the long bone cortical plate in patients with chronic osteomyelitis along its extension; however, the nature and degree of structural disorders of the cortical layer beyond the inflammation zone is an unexplored area of radiomorphology. **Objective** To study the structural features of the cortical plate of the femur and tibia outside the inflammation nidus and the change in its structure in the form of trabecularization. **Material and methods** The study is retrospective single-center work of level of evidence IV. In 86 patients with chronic osteomyelitis of the long bones of the lower extremities, using polypositional radiography and multislice computed tomography (MSCT), the features of radiological morphology of the femur and tibia were studied in order to identify structural features and quantify the density of the cortical plate and its trabecularization. **Results** The cause of the inflammatory process in the bone in 83 cases was trauma or surgery, and consequences of hematogenous osteomyelitis in three cases. The most common location of chronic osteomyelitis was the distal femur (24) and tibia (25) bones. Due to long-term disease, nonunion or a bone defect developed in 11 patients. Analysis of MSCT data in 86 patients with chronic osteomyelitis showed that anatomical changes in the femur and tibia and the density characteristics of the cortical plate near the focus of destruction were manifested in 79.5 % of patients with a decrease in the thickness of the cortical plate (up to 2 mm) with complete trabecularization in separate areas with density from 290 to 360HU in 47.7 % of the examined. **Conclusion** Radiological morphological changes in the cortical plate of the femur and tibia in patients with chronic osteomyelitis near the focus of destruction were characterized by a decrease in the thickness and density of the cortical plate, accompanied by its complete or partial trabecularization with a density not exceeding 315.5 ± 38.6 Hounsfield units (HU) in 41 (47.7 %) of 86 patients.

Keywords: chronic osteomyelitis, long bone, MSCT, cortical plate, trabecularization

For citation: Diachkova G.V., Kliushin N.M., Diachkov K.A., Shastov A.L., Larionova T.A. Trabecularization of the cortical plate in chronic osteomyelitis. *Genij Ortopedii*, 2022, vol. 28, no 2, pp. 204-210. https://doi.org/10.18019/1028-4427-2022-28-2-204-210

INTRODUCTION

The study with microCT revealed significant changes in the structure of the cortical bone that are usually observed in old age and in osteoporosis (cortical porosity (cp), cortical thinning (ct) and trabecularization (tr)) [1, 2]. The authors defined "cortical porosity" as a significant increase in the number and merging of pores, "cortical thinning" as a decrease in the thickness of the cortical plate, and the term "trabecularization" as the appearance of trabecular structures within the cortical compartment, proposed by R. Zebaze et al. (2010). It has been suggested that "intracortical remodeling induces porosity and cortical fragments that resemble trabeculae". The opinion expressed enables to assess the risk of fractures and the morphological effects of growth, aging, diseases, and possible treatment [3]. The change in the bone quality by trabecularization is manifested by the deterioration of the microstructure seen as perforation of trabeculae, thinning and loss of the connection between them, as well as cortical layer thinning and increased porosity along with other changes in the bone, such as the composition and collagen cross-linking [4]. Kameo Y. et al. (2020) proposed a new theoretical concept of a mathematical model of cortical and cancellous bone remodeling which should help identify the main features of cortical bone loss due to various causes, in particular by aging, when the load on the bone decreases and resorption processes begin to dominate under the influence of biochemical factors secreted by osteocytes. Computer modeling demonstrates how the bone is modeled as it grows into a mature 3D trabecular structure. A decrease in load leads to bone loss due to thinning of the trabeculae and loss of trabecular connections, while an increase in load leads to thickening of the trabeculae [5]. Analyzing the literature, Y. Bala et al. (2015) suggested that the loss of strength of the cortical layer of the bone is the result of an imbalance of intracortical and endocortical remodeling as a result of a decrease in the mechanical factor (load) and activation of biochemical processes as a result of the activity of osteocytes, which leads to porosity and thinning of the cortex, explaining the brittle fractures associated mainly with age [6]. Huiskestn R. et al. (2000) suggested that biochemical signaling cascades are initiated by mechanical signals. Growth factors such as TGF-b have been shown to be released during the resorption phase. These factors are powerful stimulators of osteoblast formation. Another form of communication that has recently been shown to exist is that cells of the osteoblast lineage that express RANKL are involved in the maturation and activation of osteoclasts that secrete RANK, which is the receptor for RANKL, recognize RANKL through direct intercellular interaction, and then differentiate into osteoclasts [7, 8]. The phenomenon of trabecularization has been studied with microCT and electron microscopy [9, 10, 11]. Chen C. et al. (2018) showed that modern MSCT scanners are suitable for effective quantitative visualization of peripheral bone microarchitectonics, if we focus on certain quantitative indicators [12]. The possibility of the quantitative study of some bone characteristics by MSCT was stated in the works of Holcombe SA et al., (2018) and Zachary S. et al. (2019) [13, 14]. Iori G et al. (2019) showed that the microstructural characteristics of the tibia (cortical bone thickness and the prevalence of large pores) are taken into account as biomarkers of mechanical damage to the femur as an alternative to or in addition to standard DXA [9].

Our previous studies showed that MSCT revealed a decrease in the thickness of the cortical plate beyond the zone of destruction or inflammation, a change in bone density, which differs significantly for cortical and trabecular bones [15].

As far as the studies by Zebaze et al. (2010) were performed using high-resolution peripheral computed tomography and scanning electron microscopy to quantify and compare cortical and trabecular bone, the proposed indicators could not be studied using MSCT, but it is possible to determine the decrease in the thickness of the cortical plate and its layers. MSCT also enabled to qualitatively assess the change in the structure of the cortical plate, which is characteristic of bone sections with a trabecular structure, and to measure its density [3].

Purpose To study the structural features of the cortical plate of the femur and tibia outside the inflammation nidus and the change in its structure such as trabecularization.

MATERIAL AND METHODS

It is a retrospective and a single-centre study of level of evidence IV. The inclusion criteria were chronic osteomyelitis of the femur or tibia without total extension and complete MSCT set of images. Total involvement cases were excluded.

The study was performed in accordance with the ethical standards of the Declaration of Helsinki of the World Medical Association as amended by the Ministry of Health of the Russian Federation, approved by the ethics committee of the Ilizarov NMRC for TO of the Ministry of Health of Russia. All patients signed an informed consent for the publication of the data without their identification.

Statistical processing of density parameters was performed using the Attestat software of Microsoft Excel. To confirm the conclusions about the differences between the results obtained the two groups due to their small samples, the Mann-Whitney U-test was used. The sample parameters given in the tables below had the following designations: M – mean, σ – standard deviation, n – number of patients, p – level of significance. The critical level of significance in testing statistical hypotheses in this study was taken equal to 0.05.

Polypositional radiography and MSCT were performed before treatment in 86 patients with chronic osteomyelitis of the femur and tibia, aged 18 to 65 years, who were treated at the Clinic for Purulent Osteology of the Ilizarov NMRC for TO of the Ministry of Health of Russia. Among them, there were 72 men and 14 women. The cause of the inflammatory process in the bone in 75 cases was trauma or surgery, and the consequences of hematogenous osteomyelitis in eleven cases. In 96.5 % of cases, there was osteomyelitis stage 3 according to Cierny-Mader classification, in 3.5 % of cases it were stages 2 (2) and 4 (1). The most common location of chronic osteomyelitis was the distal femur (24) and tibia (25); in 13 patients, infection was localized in the proximal femur, in 15 cases in the middle third of the tibia, and in 9 cases on the border of the middle and lower third of the tibia. Eleven patients had nonunion or bone defects. The studies were performed on CT systems "Toshiba Aquilion-64", "GE Lihgt Speed VCT". MSCT was performed with a special reconstruction algorithm "BONE". Axial sections were processed in the multiplanar reconstruction (MPR) mode in the coronal and sagittal planes. The structure of the cortical plate was studied at a distance of 3 to 5 cm from the destruction zone in its various sections: in areas adjacent to the periosteum, at a distance of 2 to 7 mm, and in the zone adjacent to the endosteum. Total thickness (histogram plotting) and local density (Hounsfield units – HU) were measured (Fig. 1).

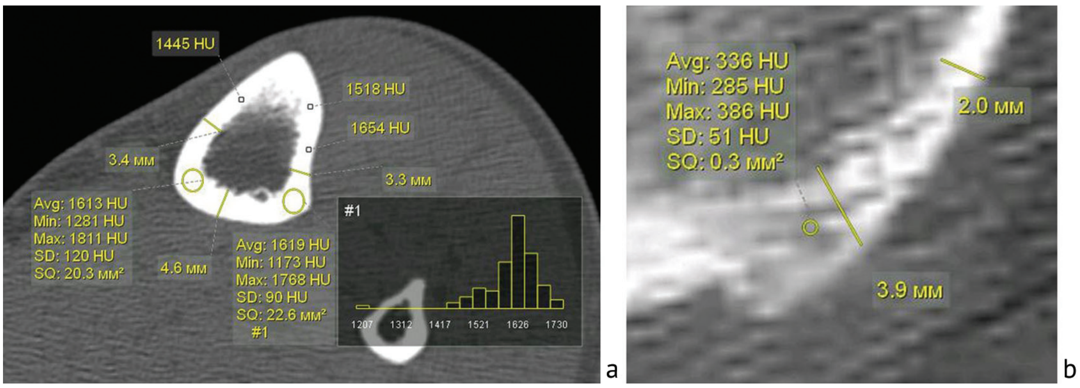

Fig. 1 MSCT of the lower leg of a patient with chronic osteomyelitis. Axial section outside the destruction zone, at a distance of 5 cm from the focus of inflammation; density and thickness of the cortical plate (a); axial section near the destruction zone (b). The thickness of the compact layer is reduced to 2-4 mm, and the density is decreased to 336 HU

RESULTS

The MSCT data in 86 patients showed that anatomical changes in the femur and tibia in chronic osteomyelitis were individual in all patients. Structural disorders were characterized by some common manifestations, but their severity had significant differences. The most pronounced changes were observed in all patients with infection process in the metaepiphyseal region (62 patients). In 24 patients with location of inflammation in the diaphysis, the extent of bone destruction was less.

The densest part of the cortical plate (compact layer) of the femur of the affected limb was 1.5 to 2.4 mm thick, whereas the thickness of the cortical plate was normally 7.8 ± 1.1 mm in the upper third of the femur. In the middle third, the thickness of the cortical plate in the axial section was somewhat greater: 8.4 ± 0.7 mm.

The study of the thickness of the cortical plate in patients with chronic osteomyelitis of the femur and tibia in the proximity to the zone of destruction or surgical intervention (the thickness of the cortical plate was studied outside the zone of surgical intervention) showed that it was significantly smaller than in a healthy limb (Table 1).

Measurement of the density of the cortical plate outside the focus of inflammation and of the healthy limb showed that outside the zone of destruction or surgical intervention the density of the cortical plate was less throughout the entire length of the bone, than of a healthy limb and depended on the duration of the disease (Table 2).

In 92.6 % of patients, the structure and density of the cortical plate at a distance of 3 to 5 cm from the area of inflammation was changed. Trabecularization, its extent, and reduction in density depended on many factors: duration of the disease, number of relapses, and limb functioning. Due to a large range in the duration of the disease (from 1 to 65 years), it was incorrect to calculate the average duration of the disease. In 15.1 % of cases, the disease continued more than 20 years, in 5.8 % from 10 to 15 years, in 8.1 % from 5 to 10 years, in the rest of the cases from 1 to 5 years. Additional means of support were used by 26.7 % of patients.

In 13 patients, the thickness of the compact layer of the cortical plate was reduced by 2-4 mm. At a distance of 4 to 6 mm from the periosteal edge of the cortical plate, areas with a density of 189.20 to 313.45 HU were detected what corresponded to the density of the bone of a trabecular structure. The total thickness of the compact part and the trabecularization zone was not much greater than the thickness of the cortical plate of a healthy limb, since even at a distance from the focus of inflammation, the cortical plate had a structure different from that of a healthy limb at this level. The density of the compact layer of the cortical plate in patients whose disease duration did not exceed 1–2 years was practically normal (Fig. 2a), while in patients with repeated relapses and the duration of the disease for more than five years, the density of the cortical plate decreased much (40–44 %) (Fig. 2b).

In 37 patients, the trabecularization was more pronounced, and the thickness of the compact layer did not exceed 2–3 mm; the rest of the cortical plate had a structure that differed from a typical cortical plate structure with a density no greater than that of the trabecular bone (Fig. 3).

Table 1

Cortical plate thickness in the femur and tibia outside the destruction zone or zone of surgical intervention and unaffected limb

Parameter		Femur (lateral side)1	Tibia (lateral side)
Uninvolved limb			
Cortical plate thickness (axial section)	Upper third	7.1 ± 1.1мм	8.01 ± 0.3
	Middle third	8.4 ± 0.7мм	6.12 ± 0.2
	Lower third	2.5 ± 0.7мм	3.51 ± 0.3
Femur (involved side)			
Cortical plate thickness outside the destruction zone	Upper third	2.1 ± 0.5	–2
	Lower third	1.7 ± 0.1	–

Notes: 1 – on the anterior surface, the thickness of the cortical plate of the tibia in the upper third, given the anatomical features, is much thicker. The thickness of the cortical plate of the femur also slightly differs in circumference, and therefore the measurement was performed in all patients along the lateral surface; 2 – radiological morphological changes in the tibia, with varying degrees of severity, in 86 % of patients spread along the entire length of the bone, and therefore it was impossible to measure the true thickness of the cortical plate with statistical analysis

Table 2

Cortical plate density of the femur and tibia outside the destruction zone or zone of surgical intervention associated with disease duration and of an unaffected limb

Parameter	Femur		Tibia	
	Up to 5 years	More than 5 years	Up to 5 years	More than 5 years
Cortical plate density outside the infection nidus at a distance from 3 to 5 cm	1504.84 ± 148.72	1210.34 ± 245.8	1317.21 ± 103.34	1117.45 ± 276.3
Cortical plate density of an unaffected limb (in the middle third)	1621.17 ± 96.15	–	1458.46 ± 101.15	–

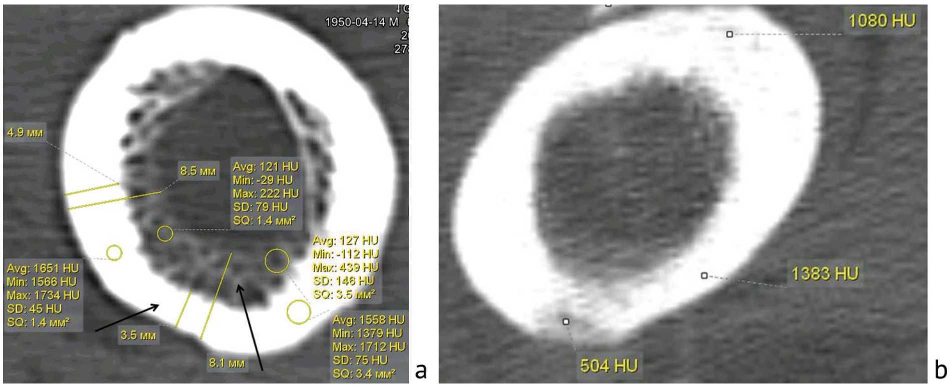

Fig. 2 MSCT of the femur of patient Sh. with chronic osteomyelitis. Axial section outside the destruction zone; density and thickness of the cortical plate in the area of the compact layer of the cortical plate and in the area of trabecularization (arrows) (a); density of the cortical plate in patient M. with a disease duration of 6 years (b)

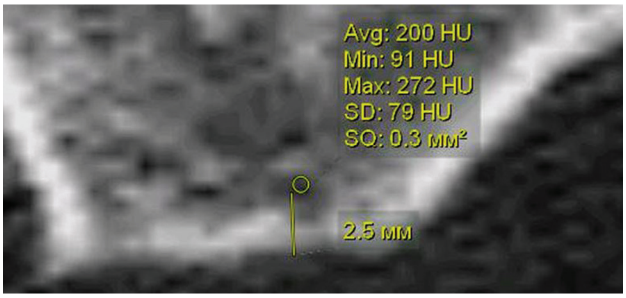

Fig. 3 MSCT of the femur of a 37-year old patient P. Axial projection; density of the bone area from the endosteal side is 200 HU

У 20 больных трабекуляризация распространялась не на всю корковую пластинку, занимала 10–15 % ее окружности, у 16 больных – до 20 %.

In 20 patients, trabecularization did not extend throughout the entire cortical plate, it occupied 10-15% of its circumference, in 16 patients up to 20 %.

As our previous studies have shown, the density of a bone with a trabecular structure varies significantly, especially in various pathologies (deforming arthrosis, congenital shortening of the limb, chronic osteomyelitis), but does not exceed 280–300 HU, with the exception of the talus, which has a fine cancellous structure and density in the range of 380–460 HU. The density in the metaphyseal part of the femur in patients with inflammation located on the border of the middle and lower thirdss reaches 210-230 HU in some areas, but in the resorption zone it may not exceed 70-110 HU. The density in the metaphysis of the contralateral limb in patients with chronic osteomyelitis is also less than normal (Fig. 4).

Bone density at a distance of 2.9 mm from the periosteal edge in patient P., 37 years old, was 302 HU, which corresponds to the density of the trabecular bone. The cortical plate at the maximum distance from the inflammation site retained its normal thickness; there was a moderate change in the structure in a limited area and a slight decrease in density (Fig. 5).

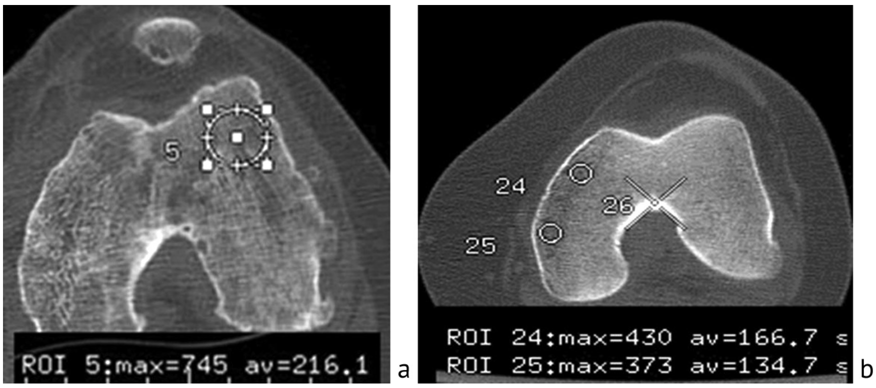

Fig. 4 MSCT of the knee joint in patients with chronic osteomyelitis. Axial section: a femoral condyles in an osteomyelitic nidus located on the border of the middle and lower thirds; b – condyles of the contralateral limb of a patient with chronic osteomyelitis of the femur located in the lower third

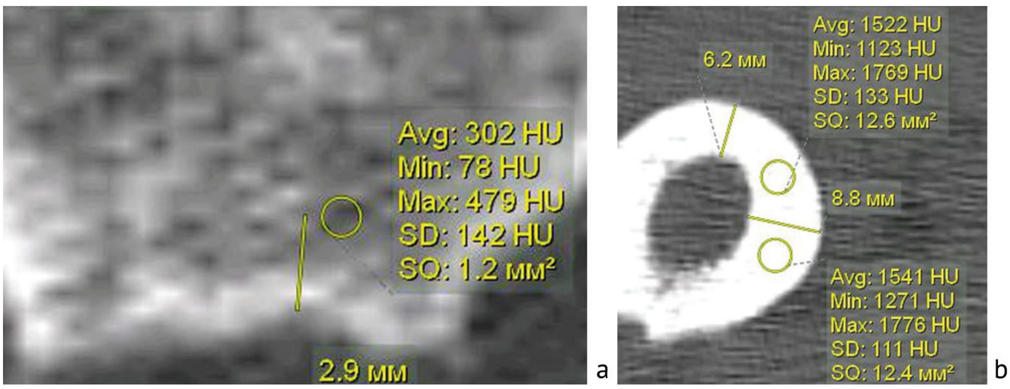

Fig. 5 MSCT of the femur of a 37-year old patient P. with chronic osteomyelitis. Axial section outside the destruction zone (a), axial section at a farthest possible distance from the inflammation nidus (b)

The study of the cortical plate on axial sections also showed that intracortical porosity was extensive, accompanied by thinning of the cortical layer due to the transformation of the part of the cortical plate adjacent to the endosteum. In 12 patients, there was an area with a pronounced heterogeneous structure, the density of which was 215 HU (Fig. 6a, arrow). At a distance of 7.4 mm from the periosteal edge of the cortical plate, but still in the area of the former cortical layer, since its thickness in a healthy limb was 7.6–9.2 mm, the bone density did not exceed 261 HU, which corresponded to the density of the trabecular bone in this zone (Fig. 6b).

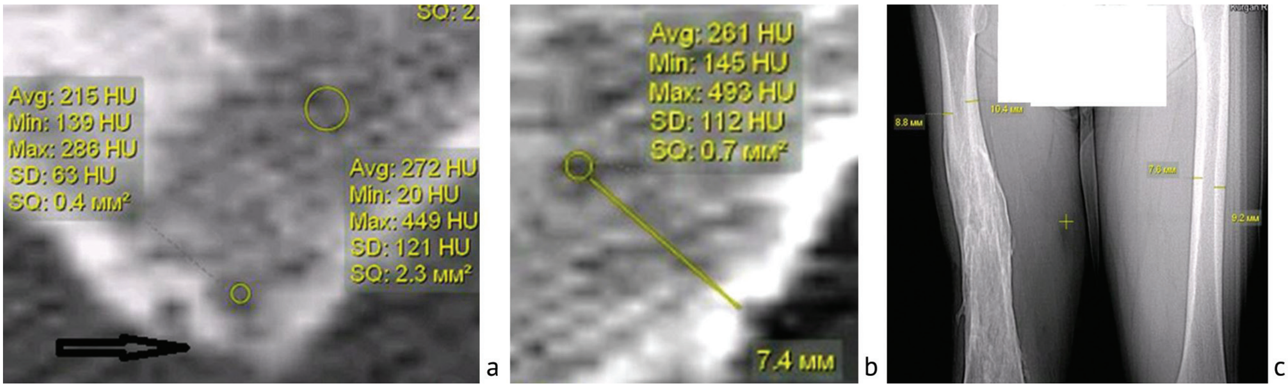

Fig. 6 MSCT of the femurs of patient P., 37 years old, with chronic osteomyelitis: a, b axial section; c topogram. Measurement of cortical plate thickness on a healthy limb

DISCUSSION

The analysis of the structure and density of the cortical plate in patients with chronic osteomyelitis showed that the features of the architectonics and density values in the areas on the endosteal side correspond to the density of the trabecular bone. Although, the MSCT data alone cannot be used to judge on trabecularization of the cortical plate with a quantitative assessment of all indicators (trabecular thickness, pore size) that is revealed with the peripheral computed tomography, it can be assumed that almost a third of the cortical plate acquires a trabecular structure, given its density and architectonics, and that intracortical porosity is extensive and is associated with thinning of the cortical layer due to the transformation of the inner part of the medullary canal into a trabecularized structure of cortical remnants resembling trabeculae [11]. The data obtained can be explained by the study of R.M. Zebaze et al. (2010) that stated that bone loss is cortical rather than trabecular; in osteomyelitis its resembles extensive intracortical porosity and is associated with thinning of the cortical layer due to the transformation of the inner part of the medullary canal into a trabecular structure of the remnants of the cortical plate, as in older people with osteoporosis [3]. In our previous studies, we revealed that in patients with chronic osteomyelitis, a decrease in the thickness of the cortical plate (not in the area of the osteomyelitic nidus where it can be thickened) and its density in many areas where there is no sclerosis and eburnation [15].

There are works that show that changes in bone structure such as trabecularization are associated not only with age-related osteoporosis, but also with some diseases [16, 17, 18]. The data obtained by us in the study of the femur and tibia in patients with chronic osteomyelitis showed that endocortical remodeling with subsequent trabecularization of the cortical plate occurs in most patients with a long course of the disease. The trabecularization is of varying grades, accompanied by a decrease in the thickness of the compact layer of the cortical plate, trabecular bone formation in the area adjacent to the medullary canal, with a density corresponding to the trabecular part in long bones. Radiological morphological changes in the cortical plate are manifested by a decrease in the thickness outside the destruction zone in the adjacent sections in 79.5 % of patients, and in 47.2 % of cases by trabecularization. There is evidence that the change in the structure of the femur in the neck area (decrease in mineral density) leads to a change in the microstructure of the tibia, and the thickness of the compact bone and the dominance of large pores in the cortical plate of the tibia should be taken as a biomarker of the mechanical damage to the femur, as an alternative or in addition to the standard DXA [9]. This coincides with our data, as changes in the tibia were also detected in chronic osteomyelitis of the femur [15, 19]. Undoubtedly, the cause of trabecularization in long bones is not only the inflammatory process, as the main disease, but also dysfunction of the limb, limitation of load or innervation, which additionally causes intracortical remodeling [16, 20–22]. Thus, to various pathomorphological changes in the bone tissue in chronic osteomyelitis that were previously described [23], one more manifestation can be added, that is the phenomenon of trabecularization. The data obtained by us identified this phenomenon of trabecularization in patients with chronic osteomyelitis, accompanied by a significant decrease in the thickness of the cortical plate, a change in the structure of the bone, indicating a reaction to the disease (chronic osteomyelitis), significantly changing the strength properties of the bone and contributing to the spread of the inflammatory process [6].

CONCLUSION

Radiological morphological changes in the cortical plate of the femur and tibia in patients with chronic osteomyelitis in the proximity to the destruction nidus were characterized by a decrease in the thickness and density of the cortical plate, accompanied by its complete or partial trabecularization and its density not exceeding 315.5 ± 38.6 HU in 41 (47.7 %) out of 86 patients.

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The article was submitted 01.02.2021; approved after reviewing 25.03.2021; accepted for publication 26.01.2022.

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