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Histological manifestations of wrist osteoarthritis and their dependence on the duration and severity of the disease

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Abstract

Introduction The possibilities of medical visualization of changes in the articular cartilage and subchondral bone in wrist osteoarthritis are limited. There are few studies devoted to its histological manifestations.

The **purpose** f the work was to determine the relationships between histological features of wrist osteoarthritis and the duration and stage of SLAC/SNAC syndrome.

Material and methods The surgical material of 12 patients who underwent resection of the proximal row of wrist bones or removal of the scaphoid bone and intercarpal arthrodesis was studied. In nine patients, the duration of the disease was shorter than four years and in three it was from 10 to 22 years. Stage I SLAC / SNAC syndrome was detected in two patients, stage II — in six, stage III — in four. Cartilage changes were assessed using the international OARSI scale, the prevalence of subchondral bone necrosis was determined semi-quantitatively (from 0 to 3 points) in 3–10 fields of microscopic views of the from each patient.

Results The OARSI score varied from 1–2 to 5 points if the duration of the disease was shorter than four years and from 3–4 to 4–5 points if it continued from 10 to 22 years. The osteonecrosis score in the compared subgroups was 3 (2÷3)(0–3) and 3 (2÷3)(2–3), $p = 0.11$. In SLAC/SNAC stage I, the OARSI score variability ranges from 1–2 to 4, in stage II — from 2 to 4–5, in stage III — from 3–4 to 5. The osteonecrosis score in the compared subgroups was 2 (1÷2)(1–3), 3 (2÷3)(1–3), and 3 (2÷3)(0–3) ($P^{1-2} = 0.03$; $P^{2-3} = 0.62$; $P^{1-3} = 0.02$).

Discussion The SLAC/SNAC syndrome can be of two types, progressive and stagnant. In the second type, the disease is asymptomatic for a long time. Regardless of the cause of SLAC / SNAC syndrome, all patients with wrist osteoarthritis experience irreversible osteonecrosis of the subchondral bone and bone marrow, which probably reflects the degree of acute or chronic damage to the vessels that feed the bone.

Conclusion With a general tendency for greater degenerative changes in the articular cartilage at a higher stage of SLAC/SNAC syndrome, their histological manifestations vary between individuals at each stage. Osteonecrosis of the subchondral bone is more common in SLAC / SNAC stages II–III than in stage I.

Ключевые слова: SLAC / SNAC syndrome, wrist osteoarthritis, articular cartilage, subchondral bone, histology

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INTRODUCTION

The wrist consists of eight small bones and many articulations between them, which are easily injured, and is a component of the wrist joint, one of the most complex joints in the human body that provides multidirectional movements of the hand relative to the forearm [1].

The most common injuries that lead to post-traumatic osteoarthritis of the wrist are ruptures of the scapholunate ligament or non-union of scaphoid fractures that may cause subsequent development of progressive collapse of the wrist, respectively SLAC and SNAC syndromes (scapholunate advanced collapse and scaphoid nonunion advanced collapse) [2]. As literature reviews show, injuries to the scapholunate ligament are often combined with fractures of the radius or scaphoid bones or other wrist injuries [3]. Injuries to the scapholunate ligament can occur secondarily in crystal arthropathies [4]. Moreover, untreated idiopathic avascular osteonecrosis of the lunate and scaphoid bones (Kienböck's disease and Preiser's disease) can also lead to progressive collapse and osteoarthritis of the wrist [5].

The common factor in the development of SLAC and SNAC syndromes is instability of the scaphoid bone. Therefore, they manifest themselves in a similar and predictable sequence of degenerative changes in the wrist joints [6–10], which are clinically significant: pain during exercise and at rest, restricted range of motion and reduced strength of the wrist grip. The diagnosis and progression of the disease are clarified by radiography, computed tomography and magnetic resonance imaging [6, 11], as well as arthroscopy [12].

Histological characteristics of wrist osteoarthritis are the subject of several studies that established significant correlations between destructive changes in the pseudoarthrotic surface of the scaphoid bone with duration of the injury, hypertrophy of the villi of the synovial layer of the wrist joint capsule with the severity of contracture, osteonecrosis in the scaphoid bone with pain severity [13]. In recent years, pain in osteoarthritis has been linked also with damage to the bone marrow of the subchondral bone [14]. These are grounds for continuing studies of the histological manifestations of wrist osteoarthritis, which is necessary to understand the pathogenesis of the disease and develop treatment tactics.

The **purpose** f the work was to determine the relationships between histological features of wrist osteoarthritis and the duration and stage of SLAC/SNAC syndrome.

MATERIALS AND METHODS

The surgical material from 12 patients (10 men and two women) operated on in 2023–2024 for wrist osteoarthritis was analyzed. The patients underwent resection of the proximal row of wrist bones or quadrilateral arthrodesis.

The age of the patients at the time of surgery ranged from 20 to 65 years (41.50 ± 15.88), the age at the time of injury or the onset of the first symptoms of the disease ranged from 17 to 64 years (35.86 ± 17.04).

Ten patients had a history of trauma (83 %): low-energy in eight (fall from standing height on the hand), high-energy in two (road accident in one and a ball hit in sports in the other), two patients denied trauma. The time since the injury or the onset of pain syndrome in the case of injury denial varied from eight months to 22 years (5.64 ± 7.6).

Four patients (33 %) were diagnosed with SLAC syndrome, and the remaining eight patients were diagnosed with SNAC syndrome. According to the radiological finding, stage I osteoarthrosis (classifications of Vender et al., Watson & Ballet [15]) was detected in two patients, stage II in six patients, and stage III in four patients. Based on radiometric data, DISI (instability of the intercalary segment) deformity was detected in 10 patients (83 %).

The study was conducted in accordance with the ethical standards of the 2013 Helsinki Declaration and approved by the Ethics Committee of the institution (protocol dated 07.10.2022 No. 2 (72)).

The surgical material (removed lunate and/or scaphoid bone or fragments of the latter) was fixed in a 10 % neutral formalin solution. After decalcification in a mixture of formic and hydrochloric acid solutions, the material was subjected to histological processing in a HISTOSAFETM INFILTRATM apparatus for vacuum processing of tissues (ErgoProduction LLC, Russia).

Histological micropreparations (paraffin sections produced with HM 450 Thermo Scientific microtome (USA) were stained with hematoxylin and eosin or the Masson three-color method. An AxioScope. A1 microscope with an AxioCam ICc 5 camera and Zenblue software (CarlZeissMicroImagingGmbH, Germany) was used to obtain digital images of the micropreparations. Changes in the articular cartilage were assessed according to the international OARSI scale [16] in a blinded manner.

The extension of subchondral bone necrosis was determined semi-quantitatively (0 to 3 points) in 3–10 fields of view from each patient (78 fields in total) at an instrumental magnification of 400×: 0 points — no signs of necrosis; 1 — signs of osteocyte death involve up to a third of the visual field area; 2 — up to two-thirds of the visual field area; 3 — up to three-thirds (100 %) of the visual field (manifestation of necrotic changes).

For statistical processing of quantitative data, the Excel Attestat application, version 9.3.1 (developer I.P. Gaidyshev, certificate of registration in Rospatent No. 2002611109) was used. Given the small number of compared patient subgroups, hypotheses about differences were tested using the nonparametric Mann – Whitney criterion.

RESULTS

In most patients (n = 9), the disease duration did not exceed four years, and in three patients it ranged from 10 to 22 years (Table 1). In the cases of disease duration of less than 4 years (n = 9), the articular cartilage assessment according to the OARSI scale varied from 1–2 to 5 points, with a disease duration of 10 to 22 years — from 3–4 to 4–5 points. In SLAC/SNAC stage I, the variability of OARSI assessments was from 1–2 to 4, with SLAC/SNAC stage II — from 2 to 4–5, with SLAC/SNAC stage III — from 3–4 to 5. All patients were found to have extended subchondral bone necrosis, which occupied from 56.67 % to 100 % of the tested area.

The lowest articular cartilage damage (grade 1–2) was observed in one patient (Fig. 1). The articular surface was not disfibered; destructively altered chondrocytes and empty cellular lacunae were detected in the superficial zone. The basophilic line was not detected, the subchondral bone plate was absent in some places, and areas of bone marrow pannus penetration were noted (Fig. 1a). In the subchondral zone, the integrity of the trabecular network was disrupted, osteolysis was pronounced, bone trabeculae with small interstitial osteonecrosis were encountered (Fig. 1b), signs of reparative osteogenesis were noted – osteoblasts on the surface of bone trabeculae (Fig. 1c). Fatty bone marrow, large accumulations of sludged erythrocytes, and erythrocytes outside the vessels predominated in the intertrabecular spaces (Fig. 1c).

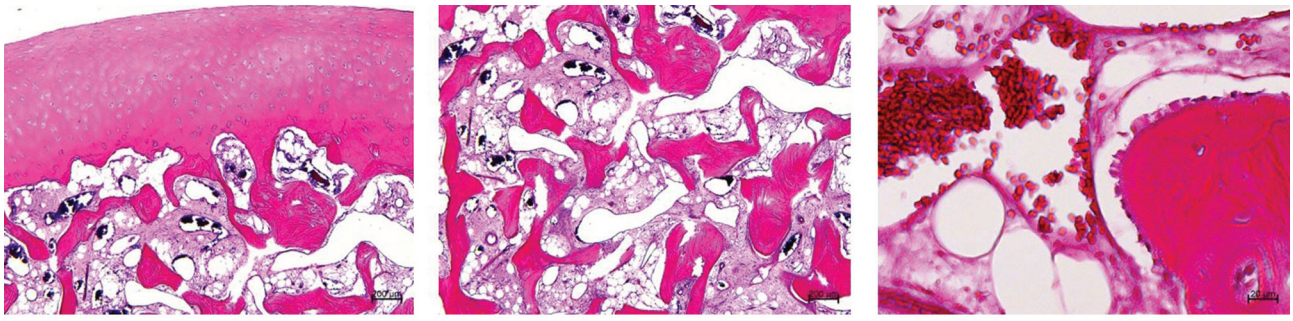
OARSI grade 3 cartilage damage was also detected in one patient (Fig. 2). The articular cartilage was present throughout, the superficial zone was defibered, detachment of small cartilage fragments was pronounced, and areas with synovial pannus were encountered (Fig. 2a). The thickness of the calcified cartilage was increased and amounted to ½–2/3 of the non-calcified cartilage.

In the subchondral zone, the integrity of the trabecular network was disrupted, the trabeculae had small interstitial osteonecroses (Fig. 2 b), and signs of reparative osteogenesis were noted (Fig. 2c). Bone marrow fibrosis was present and there were sludged erythrocytes in the vessels.

Table 1

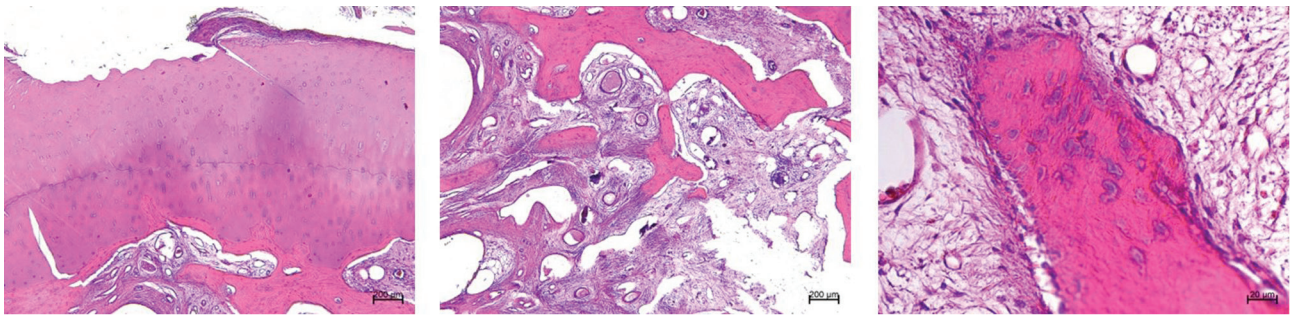
Main characteristics of patients and of surgical material

No	Age, years	Time since injury, years	Radiographic stage SLAC / SNAC	Cartilage evaluation (OARSI)	Osteonecrosis of subchondral bone (% of tested area)
1.	48	2,33	SNAC II	4	100.00
2.	23	2	SLAC II Lunate bone necrosis	4–5	75.00
3.	41	20	SNAC II	4	93.33
4.	61	1,5	SLAC III	4–5	93.33
5.	41	10	SNAC III	3–4	86.67
6.	51	22	SNAC III	4–5	100.00
7.	20	2,25	SNAC I	1–2	56.67
8.	31	0,75	SLAC II	2–3	76.67
9.	36	1,17	SNAC II	2	89.00
10.	21	3,6	SNAC I Aseptic necrosis of the distal pole of the scaphoid bone	4	77.67
11.	60	1,42	SLAC II	2	100.00
12.	65	0,67	SLAC III	5	70.83



a b c

Fig. 1 Photos of micropreparation of patient L, 20 years old (SNAC I, disease duration 2.3 years, OARSI 1–2: (a) general view of the articular cartilage; (b) subchondral zone; (c) sludged erythrocytes, osteoblasts on the surface of bone trabecula. Hematotoxylin and eosin staining. Magnification ×40 (a, b), ×400 (c)



a b c

Fig. 2 Microphotos of preparations of patient P, 31 years old (SLAC II, 0.75 years old, OARSI 3): (a) focus of defiberingin the superficial zone, synovial pannus; (b) trabeculae with interstitial osteonecrosis, bone marrow fibrosis; (c) newly formed trabeculae surface lined with active osteoblasts. Hematoxylin and eosin staining. Magnification ×40 (a, b), ×400 (c)

OARSI grade 3–4 cartilage lesions were detected in four patients (two with a disease history of less than four years and two from the group with a history of 10 to 22 years). Throughout the entire length, detachment of the superficial and intermediate zones of cartilage is pronounced, so that the articular surface is represented by a deep zone over a large area (Fig. 3a). Areas of replacement of the deep cartilage zone with bone tissue and a new formation of the basophilic section were detected (Fig. 3b). Pronounced necrosis of the subchondral bone was accompanied by smooth resorption, delamination of the ground substance of trabeculae along the adhesion lines. Signs of reparative osteogenesis were not pronounced. Unilocular and bilocular cysts were encountered (Fig. 3c).

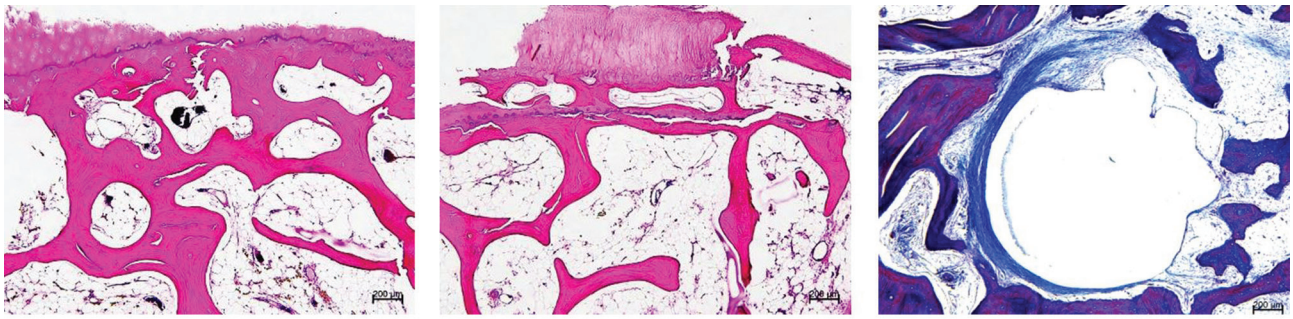


Fig. 3 Microphotos of preparations of patient I, 48 years old (SNAC II, disease duration 2.33 years old, OARSI 3–4): (a) the articular surface is represented by a defibered deep zone of cartilage; (b) progression of the bone formation process towards the articular surface, neoplasm of the basophilic section; (c) subchondral zone, cyst with a thick fibrous capsule. Staining with hematoxylin and eosin (a, b) and the Masson trichrome method (c). Magnification ×40

Cartilage damage grade 4–5 was also detected in four patients (three with a disease history of up to four years, one with a history of 10 to 22 years). The articular cartilage was defibered in some places, covered in other places with synovial pannus; there were giant isogenic groups of chondrocytes in the defibering foci (Fig. 4a). Areas of replacement of the deep and intermediate zones from the side of subchondral zone with bone marrow pannus; there was new formation of the subchondral bone plate covered with connective tissue on top (Fig. 4b). Over a large area there was a thin layer of fibrous tissue. Pronounced osteonecrosis of the subchondral bone, rarefaction of bone trabeculae and signs of osteoclastic resorption, cysts were seen. Signs of reparative osteogenesis are weakly expressed.

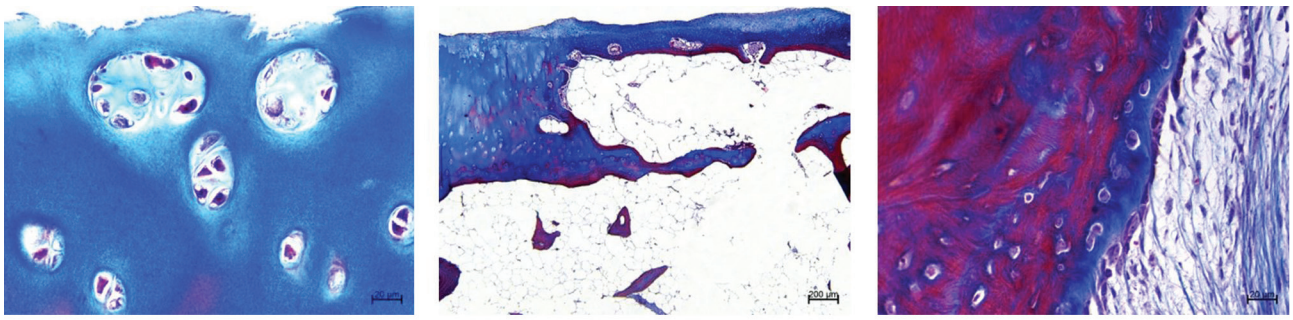


Fig. 4 Microphotos of preparations of patient D, 61 years old, (SLAC III, duration of disease 1.5 years, OARSI 4–5: (a) multi-membered isogenic groups of cells, (b) replacement of articular cartilage with bone marrow pannus, neoplasm of the basophilic section, (c) active osteoblasts on the surface of the subchondral bone plate, bone marrow fibrosis. Staining with the Masson trichrome method. Magnification ×400 (a, c), ×40 (c)

Articular cartilage damage grade 5 was detected in one patient with a disease history of less than a year (Fig. 5), who denied injury. The articular surface was represented by exposed bone tissue since the articular cartilage was absent (Fig. 5a). Severe osteonecrosis, osteolysis, stratification

of the ground substance of bone trabeculae was present (Fig. 5b). There was necrotic detritus in the intertrabecular spaces. Foci of reparative osteogenesis, newly formed trabeculae and extended areas of fibrous replacement of bone marrow are noted (Fig. 5c).

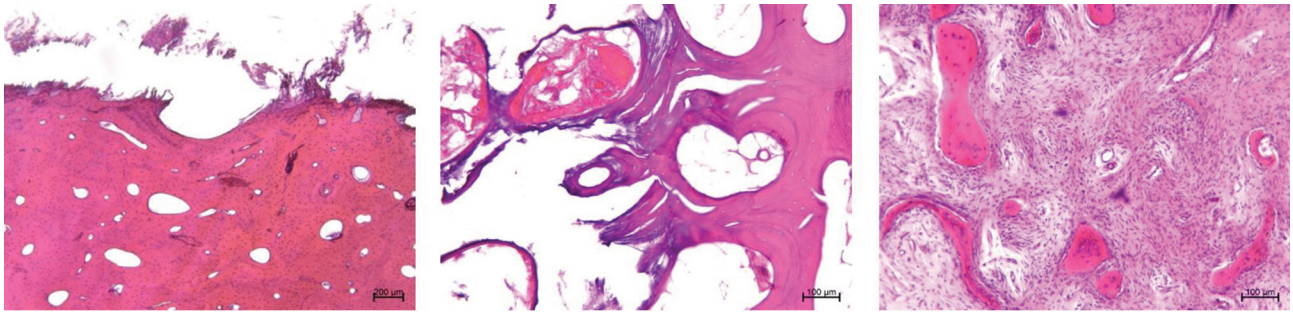


Fig. 5 Microphotographs of patient K., 65 years old (SLAC III, 0.67 years of disease duration, OARSI 5): (a) articular surface is represented by subchondral bone; (b) osteolysis, stratification of the main substance of bone trabeculae; (c) foci of reparative osteogenesis. Hematoxylin and eosin staining. Magnification ×40 (a), ×100 (b, c)

The osteonecrosis score varied from 0 to 3 points in patients with disease history of up to four years and from 2 to 3 points in patients with disease history of 10 to 22 years, the differences being statistically insignificant (Table 2). The median osteonecrosis score was lower in SLAC/SNAC stage I compared to stages II and III at a significance level of 0.05 (Table 3).

Table 2

Osteonecrosis evaluation (points) in regard to disease duration

Duration of SLAC / SNAC syndrome	Evalustion in points Me (Q1÷Q3) (min–max)	*p*
Less than 4 years (*n* = 9)	3 (2÷3) (0–3)	0.11
From 10 to 22 years (*n* = 3)	3 (2÷3) (2–3)	

Table 3

Osteonecrosis evaluation (points) in regard to osteoarthritis stage

Osteoarthritis stage	Evalustion in points Me (Q1÷Q3) (min–max)	*p*
SLAC / SNAC I (*n* = 2)	2 (1÷2) (1–3)	P^{1-2} = 0.03*
SLAC / SNAC II (*n* = 6)	3 (2÷3) (1–3)	P^{2-3} = 0.62
SLAC / SNAC III (*n* = 4)	3 (2÷3) (0–3)	P^{1-3} = 0.02*

Note: * — $p < 0.05$ Mann – Whitney test

DISCUSSION

This is the first study that assessed the articular cartilage damage according to the international OARSI scale and used semi-quantative evaluation of subchondral bone necrosis in wrist osteoarthritis caused by the developed SLAC / SNAC syndromes.

The hyaline cartilage of the wrist bones is several times thinner than the cartilage of large joints, but it is not weight-bearing [17]. Therefore, the development of wrist osteoarthritis is preceded in most cases by acute trauma, including in the group of patients studied by us. Chronic trauma to the wrist during manual labor is not considered a proven factor in wrist osteoarthritis [18], but repetitive microtraumas of the wrist in elite athletes are considered a significant etiological factor [19]. Other authors described widening of the scapholunate space of nontraumatic etiology, which with age causes progressive instability and osteoarthritis of the wrist [20]. However, some patients may not remember the episode of the injury.

As the study showed, regardless of the anamnesis, degeneration of the cartilage of the articular surfaces of the wrist bones developed in most cases within a period of less than four years, and in some cases, less than a year. However, subchondral bone necrosis was detected in all patients and was irreversible, since in no case was it classified as class 1, in which ischemic events do not disrupt the continuity of bone trabeculae, despite the death of osteocytes [21]. Disruption of continuity, lysis and rarefaction of bone trabeculae were expressed in all the cases studied, which reflects the nature and consequences of wrist collapse. The intertrabecular spaces contained predominantly fatty bone marrow; in most patients, areas of its fibrous replacement and cystic degeneration were detected.

The obtained findings suggest the limited possibilities of organ-preserving and chondroplastic surgeries in osteoarthrosis of the wrist. According to the international literature reports, radiofrequency chondroplasty performed during wrist arthroscopy creates a high risk for the survival of chondrocytes [22]. A method of bone marrow stimulation thorough arthroscopic drilling has long been recommended for the relief of chronic post-traumatic pain in the wrist, but the technique has not been widely used in clinical practice [23]. One patient after conservative treatment failure for osteoarthritis of the wrist underwent juvenile cartilage allograft plasty [24]; osteochondral auto- and allografts were used to treat cartilaginous defects of the wrist in small groups of two and three patients [25, 26], and costal cartilage grafts as a spacer were used in the treatment of patients with Kienböck's disease [27, 28]. Information on the effectiveness of these methods is currently insufficient.

The gold standard of surgical treatment of SLAC/SNAC syndrome today remains “salvage” operations (resection of the proximal row of wrist bones and quadrilateral arthrodesis), which allow to relieve pain and maintain acceptable functions of the hand [29, 30]. However, the problem of optimal treatment in young patients with initial manifestations of wrist osteoarthritis remains unsolved.

For the development of treatment technologies, further study of clinical and pathological variants of the course of wrist osteoarthritis depending on the mechanism of injury, types of activity, gender and age of patients seems promising. The data obtained indicate that SLAC / SNAC syndrome can have two types of its course, progressive and stagnant. The second type of the disease remains asymptomatic for a long time. Regardless of the cause of SLAC / SNAC syndrome (rupture of the scapholunate ligament, fracture of the scaphoid bone or idiopathic necrosis of the scaphoid or lunate bones), wrist osteoarthritis is accompanied by irreversible osteonecrosis of the subchondral bone and bone marrow in all cases. This fact probably reflects the severity of acute or chronic damage to the vessels feeding the bone.

CONCLUSION

With a general tendency for greater degenerative changes in the articular cartilage at a higher stage of SLAC/SNAC syndrome, their histological manifestations vary between individuals at each stage. Osteonecrosis of the subchondral bone is more common in SLAC / SNAC stages II-III than in stage I.

Conflict of interests *None.*

Ethical approval *The study was approved by the institutional ethics board, protocol (72) dated 07.10.2022).*

Informed consent *All patients gave informed consent on participation in the study.*

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