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ТОФУСНАЯ ПОДАГРА КАК ПРИЧИНА СИНДРОМА ЗАПЯСТНОГО КАНАЛА И ДИСФУНКЦИИ СУХОЖИЛИЙ СГИБАТЕЛЕЙ КИСТИ

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Tophaceous Gout Causing the Carpal Tunnel Syndrome and Flexor Digitorum Dysfunction: A Case Report

Резюме

Синдром запястного канала — самая частая периферическая компрессионная мононейропатия, к развитию которой может приводить множество заболеваний и состояний, в том числе, формирование подагрических тофусов в различных структурах канала. В публикации приведен обзор литературы и описание клинического случая синдрома запястного канала у пациента 58 лет с тофусной подагрой и неконтролируемой гиперурикемией. Особенностью случая является сочетание компрессионной нейропатии срединного нерва и дисфункции сухожилий сгибателей пальцев кисти в результате тофусного поражения.

Ключевые слова: *синдром запястного канала, тофусная подагра, тофусная тендопатия, компрессионная нейропатия, хирургическая декомпрессия срединного нерва, внутрисухожильные тофусы*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

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Abstract

Carpal tunnel syndrome is the most common peripheral compression neuropathy and can be caused by many diseases and conditions, including the formation of gouty tophi in various structures of the tunnel. This publication provides a review of literature and a case report on Carpal tunnel syndrome in a 58-year-old male patient with tophaceous gout. The case is characterized by the extremely rare combination of median nerve compression and tendons dysfunction due to the tophi deposits in the flexor tendons of the hand.

Key words: *carpal tunnel syndrome, tophaceous gout, tophaceous tendinopathy, compression neuropathy, surgical decompression of the median nerve, histological presentation of intratendinous tophi*

Conflict of interests

The authors declare that this study, its theme, subject and content do not affect competing interests

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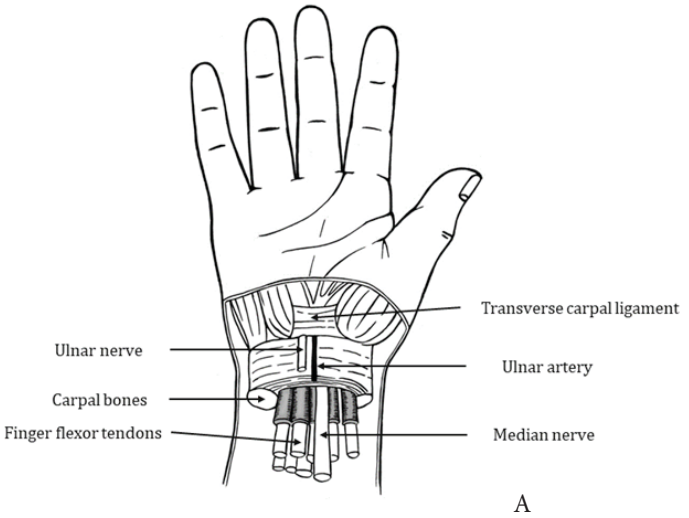
CT — carpal tunnel, CTS — carpal tunnel syndrome, US — ultrasound

Introduction

The carpal tunnel is a bone-fibrous tunnel formed by wrist bones and the transverse carpal ligament, or flexor retinaculum. The carpal tunnel is 2.5 cm long on average. According to ultrasound (US), the anteroposterior diameter of this channel is on average 10.4 mm (6.8–13 mm) [1].

The median nerve and nine flexor tendons with their tenosynovial sheaths pass through this canal (see Figures 1 A, B). The median nerve is located directly under the transverse carpal ligament and between the synovial sheaths of the flexor tendons of fingers.



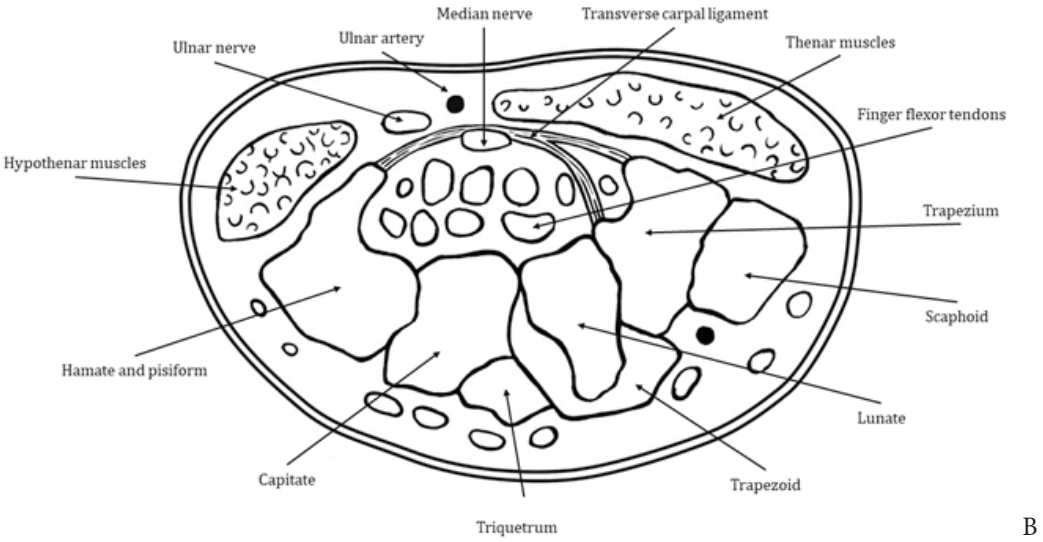


Figure 1. *Anatomy of the carpal tunnel. Longitudinal (A) and transverse (B) sections [2]*

Anteriorly, the carpal tunnel is bounded by the transverse carpal ligament, stretched between the tubercle of the scaphoid and the trapezium from the lateral side, the hook and pisiform bones from the medial side. Posteriorly and from the sides, the tunnel is bounded by the carpal bones and their ligaments. The eight carpal bones are articulated, forming an arc, with the convex facing the back of hand, and the concavity facing the palm.

The median nerve passes in the tunnel, as well as nine flexor digitorum tendons with their tenosynovial sheaths: 4 tendons of the deep flexors, 4 tendons of the superficial flexors of the fingers, and 1 tendon of the long flexor of the thumb.

Clinical Presentation of Carpal Tunnel Syndrome (CTS)

Signs of CTS are neuropathic pain, as well as sensory, vasomotor, trophic and motor deficits. These changes are localized in the innervation area of the median nerve, i.e., in I–III fingers of the hand and the radial half of the fourth finger. However, they can spread to the ventral surface of the forearm. Symptoms are most significant at night and in the first hours after waking up; during daytime they are triggered by any actions that cause nerve ischemia (fixed or habitual repeated flexion/ extension in the wrist joint, vertical static position of the forearm — driving a car, typing, holding a phone during conversation, etc.).

Neuropathic aching or bursting pain is accompanied by a burning sensation and paresthesias. Numbness of fingers and impaired temperature sensitivity are noted. Vasomotor reactions are manifested by pallor and uneven color of the skin, hypothermia of fingers. At later stages, neurotrophic disorders develop in the form of the hypotrophy of hand muscles, primarily in the thenar region. Movement disorders also emerge in the later stages and are manifested by weakness of the abduction of the thumb, impaired fine motor skills of fingers and decreased strength in hand [3].

The estimated prevalence of CTS in the adult population ranges from 1 to 5% [4]; women-to-men ratio is 3-10:1; peak incidence is in 45–60 years [5].

There are several groups of causes of CTS:

- fibrosis of the transverse carpal ligament: idiopathic or associated with overload (regular monotonous flexor/extensor movements in the wrist joint) as well as idiopathic transverse ligament hypertrophy;
- impaired bone anatomy of CT: fractures of the wrist and radius in a typical place and their consequences, destructive changes due to arthritis, Paget's disease, acromegaly, congenital features (square wrist sign);
- mass lesions of CT structures: neoplasms of the median nerve, tendon ganglion, lipoma, gouty tophi;
- tissue edema or infiltration and increased interstitial pressure: obesity, amyloidosis, diabetes mellitus, chronic heart failure, chronic kidney disease, pregnancy, oral contraceptives use, menopause, hypothyroidism, injuries of hand;
- finger flexor tenosynovitis: septic, aseptic (traumatic, overload), rheumatic (rheumatoid arthritis, seronegative spondylitis, gout);
- taking aromatase inhibitors (anastrozole); reactive tenosynovitis as hypothetical mechanism [6].

One of the most rare causes of median nerve compression neuropathy is the deposition of gouty tophi in various structures of CT.

Here is a description of a case report on CTS caused by the accumulation of tophi in the tendons of finger flexors.

Case Report

Patient B., 58, complaints of numbness and weakness in the I, II, and III fingers of the right hand, as well as difficulty with balling up a right hand into a fist over the previous 1–2 years. A history of gout since the age of 20, disease onset with arthritis of I metatarsophalangeal joint. Then, for a long time (more than three decades), the disease proceeded sufficiently benignly. Before the age of 40, gout attacks affecting alternately right and left metatarsophalangeal joints of the I finger occurred every 2–3 years. After 40 years, attacks occurred with the same frequency, but mainly affected ankles and knee joints. Patient's condition during the past three years has worsened: rapid formation of multiple gouty tophi in the joints of hands, feet and Achilles tendons; weekly attacks of arthritis of the joints of fingers and toes.

Hyperuricemia reached 700 μmol/l but the patient received no adequate treatment: for several years he took allopurinol at the dose of 100 mg irregularly, and during the past two years he took 200 mg/day, so, hyperuricemia was at the level of 500–560 μmol/l. To stop gout attacks, he used diclofenac sodium in different doses.

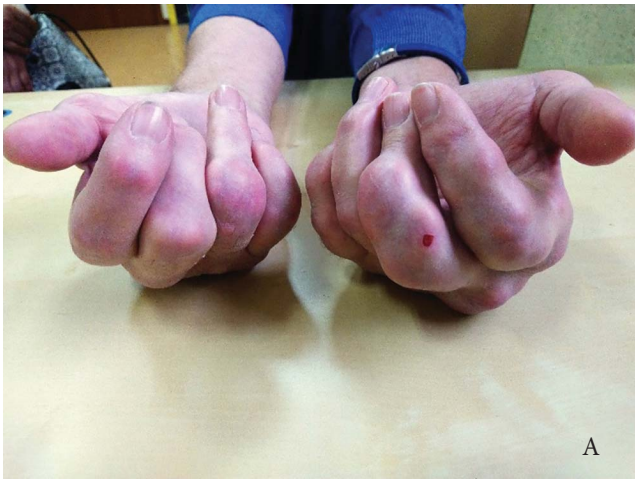
Examination revealed multiple large tophi and accumulation of small tophi on hands, extensor surfaces of elbows, feet, as well as in the area of Achilles tendons; active oligoarthritis of the joints of fingers and toes (Fig. 2 A, B).

Examination of the right hand revealed the bulging of dense tissue on the palmar surface in the area of the wrist joint and 3 cm proximal (Fig. 3). Significant hypotrophy of the thenar, sharp decrease in overall sensitivity and loss of vibration sensitivity in I–IV fingers were also noted. Fist clenching is significantly limited. Phalen's test cannot be performed due to the restriction of movements in wrist joints. Tinel's test is sharply positive.

Ultrasound revealed decreased volume of the carpal tunnel, chondropathy and chronic synovitis of the right wrist joint. Flexor tendons of the II–V fingers were examined at the level of the carpal canal, as well as in the distal region of the forearm and along the palmar surface of the hand. Tendons are thickened, densified, of irregular structure. In their thick part, gouty tophi are visible: conglomerates of mass hyperechoic inclusions without clear contours. There is no differentiation of tendon tissue in these areas; slightly pronounced vascularization is determined along the periphery of the tophus via Doppler US; most pronounced changes are observed in the tissues of the superficial flexor of the III finger. Dynamic test revealed impaired tendon excursion (block at the level of carpal ligament) and compression of the median nerve (Fig. 4 A, B).

Anatomical integrity of the median nerve is preserved throughout its entire length; at the level of the elbow joint, nerve echogenicity is without changes, the differentiation of nerve fibers is preserved. There are no signs of compression at the level of round pronator.

At the level of the middle third of forearm, diameter of the median nerve is 0.27 cm, fibers are clearly differentiated. At the level of carpal tunnel, the nerve is deformed,



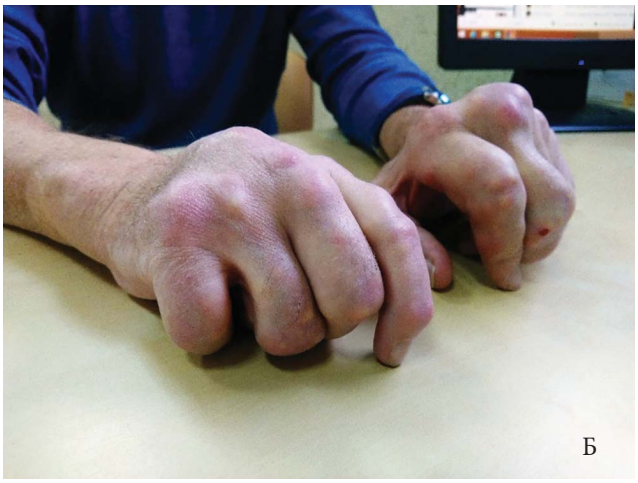


Figure 2 (A, B). *Multiple tophi, accompanied by an inflammatory reaction, active arthritides of numerous joints of the hands, restricted flexion of the fingers*

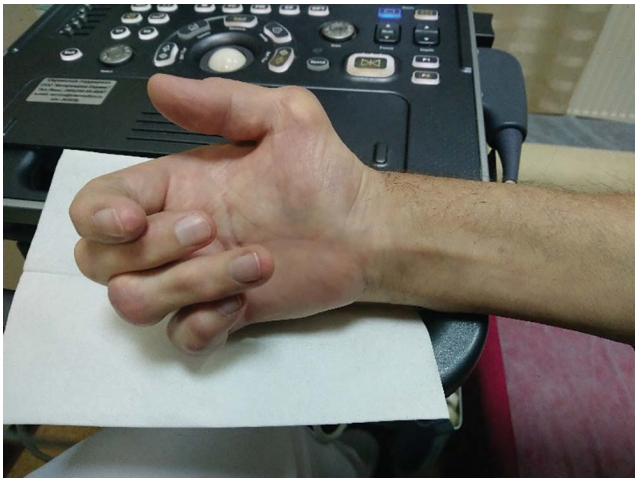


Figure 3. *Bulging in the area of the carpal tunnel of the right hand*

compressed by the thickened tendons of the flexors of fingers (Fig. 4 a, b). Size of the nerve in the tunnel is 0.14 cm (length), 0.91 cm (diameter); length/diameter ratio — 1:6. Carpal ligament thickness is 0.13 cm; ligament tissue is hyperechoic (fibrosed). Circumference of the median nerve at the level of ligament — 1.1 cm, circumference proximal to carpal ligament — 2.1 cm, distal — visualization was impossible (due to the compacted tendon tissue). A thickening of the nerve is determined proximal to the carpal tunnel, over about 3 cm to 0.9 x 0.36 cm in diameter. Echogenicity is reduced, differentiation of fibers is smoothed, nerve membrane is thickened, densified, hyperechoic, in CDI regimen — with signs of minor vascularization.

Therefore, the patient was diagnosed with compression neuropathy of the median nerve due to the compression by intratendinous tophi, as well as tendopathy with impaired tendon excursion, with a primary lesion of the superficial flexor of the third finger.

The patient underwent planned surgical treatment: dissection of carpal ligament, excision of gouty tophi in flexor tendons (Fig. 5).

According to histology results, large gouty tophi were revealed in tendon tissue consisting of the accumulations of uric acid crystals surrounded by a thin connective tissue capsule with numerous macrophages and giant multinucleated cells (Fig. 6).

Subsequently, the patient stayed at the place of residence where he received no adequate rehabilitation treatment due to limitations in local healthcare. Six months after the surgery, sensitivity in the fingers of the right hand did not recover; thenar hypotrophy persisted, there was some improvement in the mobility of fingers (flexion increased by 10–15 degrees). Electroneuromyography (ENMG) was not performed. With 350 mg of allopurinol, blood uric acid level was 435 μmol/l. It was recommended to increase the dose of allopurinol in order to achieve target uric acid level of 300–360 μmol/l.

Thus, despite the potential reversibility of the lesion, the patient failed to achieve any significant restoration of sensitivity and function of the hand. This is due to the extensive tophus lesions, untimely surgical intervention and lack of rehabilitation treatment.

Discussion

Gout is a systemic tophaceous disease characterized by the deposition of monosodium urate crystals in different tissues that results in inflammation in individuals with hyperuricemia due to environmental and/or genetic factors [7]. Gout medications are aimed at maintaining uric acid level at the level of <360 μmol/l (<300 μmol/l in case of severe tophaceous gout) [7]. Uncompensated hyperuricemia leads to the formation of tophi that happens, on average, 10 years after the disease onset [8].

Tophi are the deposits of monosodium urate crystals in different tissues. The most typical of them are subcutaneous tophi on auricles, as well as clusters in the olecranon bursa. Tophi usually form on hands and feet, in the area of joints and tendons, where they can be

located both subcutaneously and intraarticularly, in the thickness of the synovial membrane, intraligamentously and intratendinously, as well as intraosseously. It is also known that monosodium urate can be deposited in any joints, including intervertebral discs, as well as internal organs. Cases of gouty tophi in the organs of respiratory system, eye structures, nails, mammary glands, kidneys, liver, heart valves, pancreas, and intestines were described [9–17].

In rare cases, tophi appear in the carpal tunnel, which leads to CTS. The first report on this subject dates back to 1958 [18], and so far, there are no more than 100 such descriptions in English-language sources [19]. In general, carpal tunnel syndrome due to tophus lesion is rare and amounts to 0.6–2% in the etiology structure of this neuropathy [19–21].

Causes of the neuropathy of the median nerve with gout, as well as other causes of CTS, can be divided into two groups: increased volume of structures contained in the tunnel and decreased internal caliber of the tunnel [22]. The first group of causes includes, first of all, tophus infiltration and inflammatory edema of tenosynoviums and tendons [20, 21, 23, 24]. Thickening of the transverse ligament due to tophus deposits can lead to decreased caliber of the tunnel [25], along with parietal or intraosseous formation of tophi [26, 27]. Direct tophus lesion of the median nerve is known to be possible [28]. Therefore, the specific feature is that CTS secondary to gout may develop via several pathomorphological mechanisms. This should be taken into account when examining patients with CTS that developed with underlying gout.

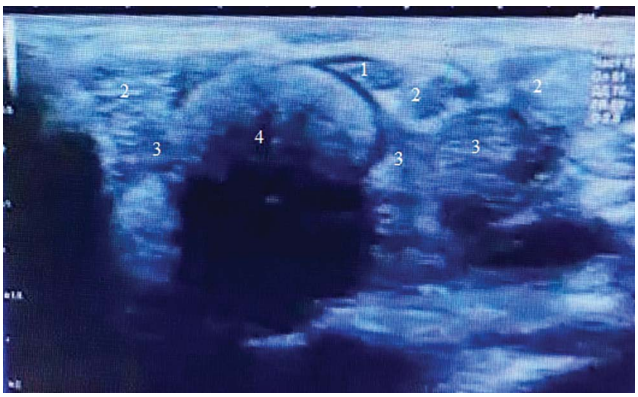


Figure 4 A. *Ultrasound scanning of the wrist joint (palmar surface, cross section through the carpal tunnel)*
1 — the median nerve is compressed by the thickened third flexor digitorum superficialis tendon, containing a large tophus (4); the height of the nerve is reduced, the diameter is widened; the carpal ligament is thick and dense
2 — II-V superficial flexors of the fingers;
3 — II-V profound flexors of the fingers;
4 — Gouty tophi conglomerate in the third superficial flexor tendon

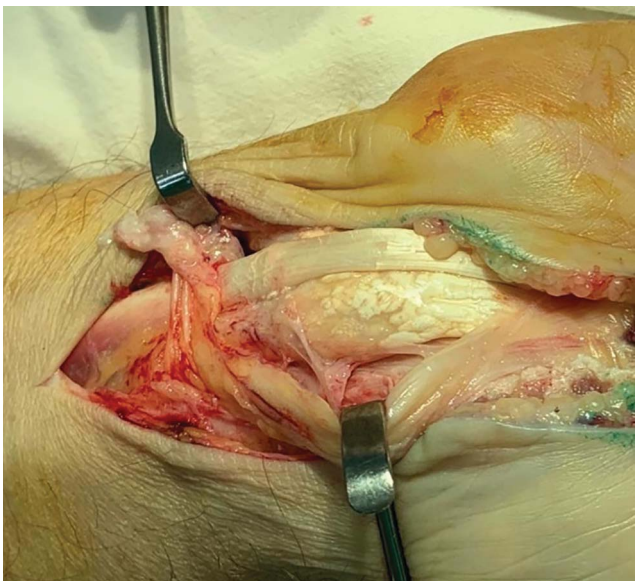


Figure 5. *Intraoperative photograph. Extensive deposits of gouty tophi on the surface and in the thick of the flexor tendons of the right hand*

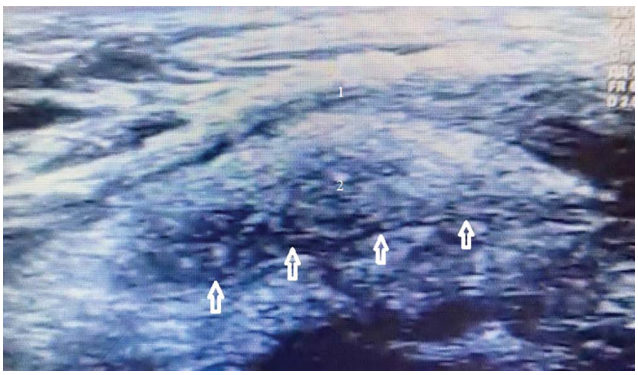


Figure 4 B. *Ultrasound scanning of the wrist joint (palmar surface, longitudinal section through the carpal tunnel)*
1 — the median nerve is compressed by the thickened third flexor digitorum superficialis tendon (2), containing tophi conglomerates (arrows); the thick carpal ligament is above the nerve

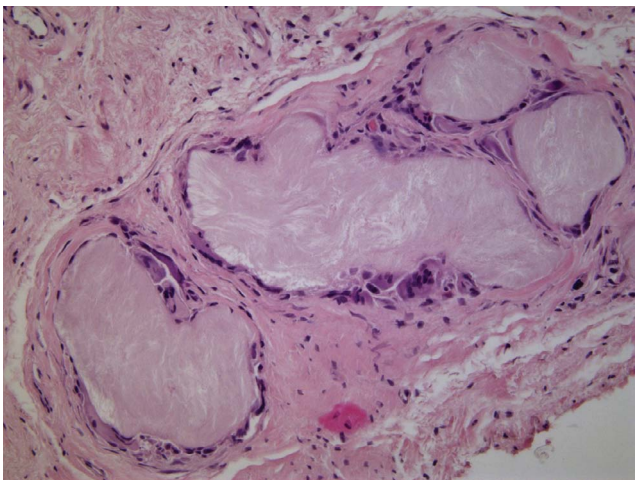


Figure 6. *Intraoperative photograph. Median nerve with signs of compression and venous stasis*

In addition, this clinical case draws attention to tendopathy as one of the variants of tophaceous lesion. Tophi are usually found in the Achilles tendon, the tendon of quadriceps femoris and patellar ligament, as well as in extensors of fingers and toes [8]. This usually does not lead to any significant impairment of biomechanics. However, in the case of tophus deposition in hand flexors, their impaired excursion in the carpal tunnel is possible, up to complete blockade. In turn, this leads to motor dysfunction of the hand in the form of difficulty or inability to bend fingers [29]. Tophaceous lesion of flexor tendons is quite rare; over the past 37 years, only 43 cases were described in English-language literature [30]. However, this possibility should be taken into account as one of the possible causes of deficit in finger movements in patients with gout.

Conclusion

This clinical case is indicative if the variety of causes of CTS, which may include deposition of tophi. On the other hand, it indicates a much wider spectrum of gout complications that, among other things, includes neurological and biomechanical disorders. This case is especially interesting due to a combination of two rare lesions in one patient: deposition of tophi in flexor tendons led to the compression neuropathy of the median nerve, and at the same time, significantly impaired the function of tendons themselves. Such a combination is extremely rare and, to date, is described in no more than 20 observations [30]. However, even a few reports no longer allow us to consider the discussed combination as a unique event but, on the contrary, as one of the variants of gout that requires timely diagnosis and adequate surgical tactics.

Вклад авторов

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Author Contribution

All the authors contributed significantly to the study and the article, read and approved the final version of the article before publication

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