Pseudorheumatoid disability man with chronic tophaceous gout: a case report

RODICA TRĂIȘTARU1, VIORELA ENĂȘCĂUȘ2, DIANA KAMAL3, CAMELIA FOARFĂ4, OTILIA ROGOVEANU1

1) Department of Physical Medicine and Rehabilitation
2) Department of Family Medicine
3) Emergency County Hospital, Craiova
4) Department of Pathology
University of Medicine and Pharmacy of Craiova

Abstract

Gout is a type of inflammatory arthropathy that affects the peripheral joints and results from the accumulation of monosodium urate (MSU) crystals in the synovial fluid and other tissues. This disease is the most common form of inflammatory arthritis in men over 40 years of age. The fundamental biochemical abnormality in gout is an increase in serum urate (SU) concentration. These needle-like crystals induce not only acute episodes of inflammatory process into the surrounding area, but also, in the long-term history of the disease, chronic inflammation that is associated with changes in articular and periarticular structures. The next step caused by deposited MSU crystals is represented by the tophus formation and chronic gouty synovitis. The presence of tophi has been associated with greater physical functional disability in gout patients. We presented a case of severe chronic tophaceous gout in a 48-year-old man with chronic hand arthritis and urolithiasis, to point the significance of complex assessment (clinical, functional, imagistic and histological exams) in the diagnosis of a soft tissue lesion, especially in hands.

Keywords: gout, monosodium urate crystals, tophi, disability.

Introduction

Chronic gout is a type of inflammatory arthropathy that affects the peripheral joints and results in the accumulation of monosodium urate (MSU) crystals in the synovial fluid and other tissues, typically follows a clinical course, first with years of asymptomatic hyperuricemia (the basic underlying metabolic abnormality) followed by acute intermittent attacks, and eventually with chronic arthritis with the formation of tophi [1–4].

Presently, gout is one of the most common rheumatic diseases of adulthood and is a debilitating disease whose prevalence appears to be increasing worldwide due to many factors (e.g., aging populations, changes in diet – obesity and lifestyle – ethanol use and medications) despite a better understanding of its risk factors and pathophysiology, and the availability of reasonably effective treatment [5–7].

The increased prevalence of gout has been linked to the increased prevalence of comorbidities associated with hyperuricemia, such as high blood pressure, obesity, metabolic syndrome, type 2 diabetes, and chronic kidney disease [5].

Gout is the most common form of inflammatory arthritis in men over 40 years of age. The fundamental biochemical abnormality in gout is an increase in serum urate (SU) concentration. The monosodium urate (MSU) crystals may form and can deposit in joints and periarticular tissues when high SU concentrations are reached (6.8 mg/dL or 0.41 mmol/L at 37°C) [8]. These needle-like crystals induce not only acute episodes of inflammatory process into the surrounding area, but also, in the long-term history of the disease, chronic inflammation that is associated with changes in articular and periarticular structures [9]. The next step caused by deposited MSU crystals is represented by the tophus formation and chronic gouty synovitis [8].

Arthritis caused by gout (gouty arthritis is confirmed if monosodium urate crystals are present in the synovial fluid) accounts for millions of patients visits annually, and the prevalence is increasing [10]. In the past 50–60 years, the prevalence of gout has increased in accordance with the high prevalence of metabolic syndrome and the increase in the average weight of people. The Western World appears to be in the midst of the third great gout epidemic of all time [7].

The accumulation of MSU crystal in tissues also leads to nephrolithiasis, and urate nephropathy [10]. Gout can coexist with many diseases including rheumatoid arthritis, ankylosing spondylitis, and even systemic lupus erythematosus but these associations are rare and even controversial. Furthermore, subcutaneous tophaceous deposits can look like those from other rheumatic disorders, such as nodules in rheumatoid arthritis and lesions in multicentric reticulohistiocytosis, which may lead to further diagnostic confusion [11, 12]. The presence of tophi has been associated with greater functional physical disability in gout patients [13, 14].

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urolithiasis, to point the significance of complex assessment (clinical, functional, imagistic and histological exams) in the diagnosis of a soft tissue lesion, especially in the joints of the hands.

Patient, Methods and Results

Our report describes a significantly disabled man with previously diagnosed and inadequately treated chronic tophaceous gouty arthropathy (CTGA).

History

A 48-years-old man was admitted in the hospital complaining of an intense pain in both hands, especially the 1st, 2nd and 3rd fingers, significantly disability of prehension and upper limb mobility. No other significant joint complains were mentioned.

He described the slowly building of tophaceous deposits in and around the small joints of the hands and first metatarsophalangeal joints (MTPJ) as having started five years ago. Urate-lowering therapy for gout (Allopurinol, 300 mg daily to maintain the serum uric acid level less than 6 mg/dL) is initiated after the development of tophi, but there was no compliance to the treatment.

In the last year, the tophi around second and third bilateral hand fingers had presented a rapid growth with inflammatory signs. His therapy for acute gout was non-steroidal anti-inflammatory drugs and colchicines.

The first attack (a polyarticular clinical attack) was before the age of 35 years. This aspect could raise the suspicion of an inborn enzymatic defect. After the first gout attack, our patient was informed about the modifiable risk factors (high-purine diet, alcohol use, diuretic therapy) but he did not respect these recommendations.

Previous medical history showed primary arterial hypertension, hypertensive cardiomyopathy and nephrolithiasis. He followed a long-term diuretic therapy.

His mother was diagnosed with type 2 diabetes mellitus.

Clinical exam

Our non-smoker and normal weight patient was in the fourth development stage of clinical gout – severe chronic gouty arthritis (due to the numerous, complicated tophi affected more than four joints) (Figure 1).

Physical examination revealed swollen painful fingers, with limited mobility, joint deformity, numbness in right hand and significant dysfunction in prehension and daily activities. The patient developed periarticular masses (tophi), 4–8 mm in diameter, on both lateral sites of the 1st, 2nd and 3rd fingers phalanges, and around the both first metatarsophalangeal joints. The overlying skin was erythematous, with yellow points in one site, through drained clear fluid. All masses did not appear attached to the underlying bony structures.

He had not presented any other masses surrounding the lower or upper limb tissue joints, but on the helix of the both ears we found 2 mm diameter yellow mass.

The second and the third stages of chronic gout were historically described by the patient. The clinical exam of all organs and systems (including cardiovascular system) was in normal limits.

Disability, impairment, and hand function were assessed for the studied patient using a gout-specific HRQoL instrument, the Gout Impact Scale (GIS), to measure the association of gout characteristics with HRQoL (high-related quality of life). GISs were scored from 0 to 100, with higher scores on each scale indicating “worse condition”. In our patient, the score of GIS was 81, indicating a greater disease impact in quality of life.

Laboratory tests

The basic laboratory tests (peripheral blood with erythrocyte sedimentation rate, blood chemistry findings – blood sugar level, immunological tests – absence of rheumatoid factor and anti-CCP antibodies, urinary analysis) were within normal limits, with only the three pathologic values: serum urate concentration was 6.8 mg/dL, hypercholesterolemia (254 mg/dL) and 10 330/mm³ leukocytes in peripheral blood test.

The EKG sowed normal sinus rhythm, with a QRS axis at 0º, and diffuse flattened T-waves indicating myocardial ischemia.

We performed X-rays of the patient’s hands (Figure 2). The decreased bone density and mild erosive changes at the phalanges, both in the right and left hands were observed. Calcified erosions with sharp margins and overhanging edges are characteristic of gout. The biomechanical axes of the fingers in the right hand were not preserved.

The ultrasonography of the hands (longitudinal ultrasound images of the proximal interphalangeal joint of the third and second right fingers (Figure 3A) and first right finger (Figure 3B), performed with the HD 11 XE Ultrasound System Philips, 12.5 MHz linear sound) was helpful in visualizing the soft tissues in the surrounding joint tissues. The ultrasound observed lesions that were characterized by a heterogeneous amorphous soft tissue swelling with double contour sign and hyperechoic soft-tissue areas, representing tophus (solid arrows) and juxta-articular bone erosions (dashed arrows).
We performed an excisional biopsy with complex histological and immunohistochemical analysis. The biopitic fragment was processed by standard techniques of inclusion in paraffin following the next steps: fixation in 10% buffered formalin, washing with water or 80% alcohol, dehydration – in a graded alcohol clarify – baths of benzene, toluene, xylene and waxing. Standard staining with Hematoxylin–Eosin was used.

For immunohistochemical examination (IHC), the LSAB (HRP) (LSAB – Labeled Streptavidin Biotin, HRP – Horseradish peroxidase) method was used and antibodies such as CD10, CD20, CD45RO, vimentin (Table 1).

Table 1 – Antibodies used in this study

<table>
<thead>
<tr>
<th>Antibody</th>
<th>Source</th>
<th>Clone</th>
<th>Dilution</th>
<th>Antigen retrieval</th>
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<tr>
<td>CD10</td>
<td>LEICA</td>
<td>56C6</td>
<td>1:100</td>
<td>Five cycles citrate buffer</td>
</tr>
<tr>
<td>CD20</td>
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<td>L26</td>
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</tr>
<tr>
<td>Vimentin</td>
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<tr>
<td>CD68</td>
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<td>PG-M1</td>
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<tr>
<td>CD45RO</td>
<td>DAKO</td>
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The macroscopic semblance of the gouty tophi was that of a nodule with a whitey color on section that grew towards the skin where it evacuated its viscous content.

The histological examination of the biotic fragment (Hematoxylin–Eosin stained section) showed the following aspects:

- The histiocytary gouty tophi is a granuloma (foreign body granuloma), which appears as a nodular formation, imprecisely defined, consisting predominantly of phago- cytes histiocytes, leukocytes plus a conjunctive-vascular component (Figure 4, A and B);
- the gouty tophi shows amorphous urate deposits defined as monosodium urate (MSU) crystals, which appear as an acidophilic precipitate, needle-shaped, birefringent and representing a foreign body (Figure 5, A and B);
- MSU crystals are irritants with minimal toxicity, surrounded by inflammatory cells. These are represented by the foreign body giant cells, lymphocytes and plasma cells. Foreign body giant cells are large macrophages, multinucleated, placed in contact with urate deposits (Figure 6, A and B).

Immunohistochemical analysis (IHC):

- CD68 (macrophage marker) was intense and diffuse positive in the foreign body’s giant macrophages (Figure 7, A and B);
- CD10 (common leukocyte antigen) was intense and diffuse positive in the inflammatory cells that form the granulomas (including macrophages) (Figure 8, A and B);
- CD45RO (marker for T-lymphocytes) was intense and focal positive in the remaining T-cells that make up granulomas (Figure 9, A and B);
- CD20 (marker for B-lymphocytes) was intense and focal positive, in very few cells (B-lymphocytes outstanding) around the granulomas (Figure 10, A and B);
- vimentin (mesenchymal marker) was intense and diffuse positive for the fibroblasts and endothelial cells of the conjunctive-vascular component, and in some macrophages (Figure 11, A and B).

After complex assessment of our patient, we applied a complete treatment in accordance with the first ACR evidence and consensus-based pharmacologic and non-pharmacologic management recommendations for gout. We explained to our patient the role of lifestyle interventions in his disease (optimal weight, dietary changes such as reducing intake of animal purines, high-fructose sweeteners, and alcohol, and increasing intake of vitamin C).

Treatment recommendations were individualized based on the patient’s overall health, comorbidities, and willingness to adhere to chronic treatment. The urate-lowering therapy with Allopurinol (300 mg daily), was continued due to reduce serum urate levels to less than 6 mg/dL in order to mobilize and deplete crystals with minimal toxicity. Our patient should have benefited from Febuxostat (other xanthine oxidase inhibitor like Allopurinol) and Pegloticase (a PEGylated uric acid specific enzyme) treatment (a treatment approved and indicated only for the refractory gout patients), but this drug is not available in our country.
Figure 4 – (A and B): The histiocytic gouty tophi appear as a nodular formation, imprecisely defined, consisting predominantly of phagocytes histiocytes, leukocytes plus a conjunctive-vascular component. HE staining, ×40.

Figure 5 – (A and B): The gouty tophi shows amorphous urate deposits defined as monosodium urate (MSU) crystals, which appear as an acidophilic precipitate, needle-shaped, birefringent and representing a foreign body. HE staining, ×100.

Figure 6 – (A and B): Foreign body giant cells are large macrophages, multinucleated, placed in contact with urate deposits. HE staining: (A) ×100; (B) ×200.
Figure 7 – (A and B): CD68 (macrophage marker) was intense and diffuse positive in the foreign body’s giant macrophages. CD68 immunostaining: (A) ×100; (B) ×200.

Figure 8 – (A and B): CD10 (common leukocyte antigen) was intense and diffuse positive in the inflammatory cells that form the granulomas (including macrophages. CD10 immunostaining: (A) ×40; (B) ×200.

Figure 9 – (A and B): CD45RO (marker for T-lymphocytes) was intense and focal positive in the remaining T-cells that make up granulomas. CD45RO immunostaining: (A) ×100; (B) ×200.
Figure 10 – (A and B): CD20 (marker for B-lymphocytes) was intense and focal positive, in very few cells (B-lymphocytes outstanding) around the granulomas. CD20 immunostaining: (A) ×40; (B) ×100.

Figure 11 – (A and B): Vimentin (mesenchymal marker) was intense and diffuse positive for the fibroblasts and endothelial cells of the conjunctive-vascular component, and in some macrophages. Vimentin immunostaining: (A) ×40; (B) ×200.

Non-steroidal anti-inflammatory drugs (NSAIDs), corticosteroids or oral Colchicine are optimal treatments for any acute gout attack. We recommended pharmacologic anti-inflammatory prophylaxis (low-dose NSAID therapy and oral Colchicine) because our patient had clinical evidence of continuing activity of the disease and the serum urate target had not yet been achieved.

The cardiovascular diseases were monitored and the diuretic therapy was interrupted. The Losartan and calcium-blocker drugs were prescribed.

The differential diagnosis was not necessary in the present complete evaluation; the clinical and imagistic aspects were enough to put the positive diagnosis. We took into consideration only two differential diagnoses; gout has the ability to mimic infectious entities as well as metastatic disease.

The prognosis of the disease is variable, in accordance with the patient compliance to the treatment recommendations, especially educational and non-pharmacological aspects.

The possible complications of tophi include soft tissue damage and deformity, joint destruction and nerve compression syndrome, with important physical disability.

Discussion

In the medical literature, most articles described the clinical, functional, imagistic and histopathological features of tophaceous gout, in patients with chronic arthritis, to emphasize the importance of considering this disease entity in the differential diagnosis of other joint diseases or soft tissue disorders [15, 16].

Clinical and functional aspects

In our report, we present the particular onset and evolution in an adult man diagnosed with a significantly debilitating chronic gout. Most common clinical manifestation of gout is represented by a painful arthritis in the lower extremities [15]; our patient is an exception (the painful arthritis is in both hands). In most people, the first clinical attack is usually monoarticular (in MTP joint in roughly 50% of persons), but it could be polyarticular sometimes, like in our patient [7]. All common sites of the urate accumulations (the synovium, articular cartilage of joints, periarticular ligaments, tendons and soft tissue like prepatellar bursa, olecranon bursa, the base of the great toe, the ear helix) and the less frequently (skin of
the fingers, palms, soles, nasal cartilages) have to assess for the complete clinical and functional patient evaluation. In our patient, the gout tophi were present at almost all the small joints of both hands. His affected digits could define as dactylitis; the overlying skin was icteric, erythematous, with lesions that drained clear fluid, a chalky material.

The studied patient is particularly susceptible to developing other metabolic diseases (type 2 diabetes mellitus, hyperlipidemia or dyslipidemia, metabolic syndrome), when confronted with his medical history.

We did not investigate a possible inborn enzyme defect in our patient, but we considered this aspect; the gouty attacks before the age of 30 years in men should raise the suspicion of an inborn enzyme defect or one of several hereditary kidney diseases [7].

Recent researches mentioned that uric acid level in itself is associated with diastolic dysfunction and left ventricular hypertrophy, and is an independent risk factor for congestive heart failure [7]. The uric acid level of our patient was controlled in the past years, so the cardiovascular comorbidities had no disability impact on his quality of life.

Our patient had a lower level of quality of life – QOL (the Gout Impact Scale score was 81, also the serum urate concentration was 6.8 mg/dL); this high disability occur due to structural changes that manifest in the soft tissues and due to the presence of multiple tophi [13, 17]. From the available literature (only few studies have addressed the relationship between serum urate and QOL and/or function – may be in part due to a lack of appreciation until recently of the effects of gout on QOL and in part due to lack of validated specific gout assessment QOL measures), there is no association between the level of uric acid and QOL for patients with chronic gout [8]. The impairment, pain and disability were assessed using various scales, especially for foot structure and foot function. Pain and disability can limit functional capabilities in the individual, compromising quality of life. QOL and function have been shown to be impaired in patients with gout [18]. All patients diagnosed with chronic gout, especially refractory chronic gout, as is our patient, have various physical dysfunctions and decline in quality of life QOL compared to patients without chronic gout [19]. Compared to the general population and to non-gout patients who had one or more self-reported musculoskeletal conditions, patients with severe gout and other co-morbidities, as was our patient, have been shown to have lower SF-36 scale scores, particularly in physical function [20].

**Imagistic aspects**

The musculoskeletal involvement in patients with gout and the subclinical structural damage in asymptomatic individuals with hyperuricemia can been demonstrated and monitored by ultrasound exam [21]. These specific ultrasound findings provide an early non-invasive tool for diagnosis of gout – one of the commonest forms of inflammatory arthritis, mediated by the accumulation of monosodium urate crystals in the superficial portions of the articular cartilage, resulting in an inflammatory response [22].

In our patient, the imagistic approach can be helpful not with the diagnosis of gouty tophi, that were clinically obvious, but with monitoring the response to treatment (medical therapy can be effective for the great majority of unusual tophaceous deposits) [1].

We performed an ultrasound exam in our patient in accordance to international conclusions that ultrasound fulfills the Outcome Measures in Rheumatoid Arthritis Clinical Trials (OMERACT) filter, as a feasible, valid and discriminative measure for evaluating changes in tophus size in gout patients in urate-lowering therapy [23]. Also, the ultrasound imaging may be useful to evaluate the severity of the disease (measurement of structural joint changes), the extent of MSU accumulation (location and magnitude of urate accumulation), and the presence of chronic inflammation in all soft tissue structures around the affected joints [24]. We consider that other imagistic means, such as CT and magnetic resonance imaging (MRI) are not necessary.

We highlighted the hypo echoic and mixed echogenicity of the nodules, with two elements:

- the most useful elementary lesion in gout: the double-contour sign, explained as a hyper echoic, irregular band over the superficial margin of the joint cartilage, produced by accumulation of MSU crystals on the surface of the hyaline cartilage, which increases the interface of the cartilage surface, reaching a thickness similar to the subchondral bone [9, 25];
- the commonly seen surrounding hypoechoic halo probably corresponds to the outer, loose fibrovascular area seen on histology [26].

Our ultrasonography images have a limit – we did not apply the Doppler signal to evaluate the hyper-vascularization.

However, the most reliable method of diagnosing gouty arthritis continues to be needle aspiration of joint fluid and identification of the crystals on polarizing microscopy.

**Histological aspects**

We performed the biopsy in our patient because we are in accordance with the other studies that sustained the definitive diagnosis of gout certainly made by demonstration of MSU crystals in the synovial fluid or biopsy [16].

The means to prepare for histological analysis is particularly important, standardized steps being necessary to be completed to preserve MSU crystals.

In contrast to acute gout, the characteristic and mechanisms of chronic gout have been less intensively examined [27].

Microscopically, the tophus – a pathognomonic lesion of chronic gout – appears as a chronic granulomatous mass of urates, crystalline or amorphous, surrounded by an intense inflammatory reaction, composed of mononucleated and multinucleated macrophages and encased by fibroblasts and dense connective tissue [28, 29]. Schematically (Figure 12), there can be identified three areas of the tophus, inside to outside: area 1 – the central crystalline core; area 2 – the cellular corona area; area 3 – the outer fibrovascular area [30].
The presence of tophi in our patient has only been identified into the elastic and dense fibrous periarticular tissues – two of the three typical sites [31]. The histological exam did not reveal the crystals accumulation in the Haversian system and the bone structure of the fingers was not replaced by tophaceous material.

We consider being important two-histological features in gout tophi: first, the presence of MSU crystals and second, the immune-inflammatory cell complex that surround the deposits of MSU crystals.

The inflammatory nature of MSU crystals was discovered 50 years ago [7]. The MSU crystals have the capacity of initiating an inflammatory response from leukocytes and synovial cells to trigger the release of cytokines that amplify the local inflammatory process [32].

Into the acute gout attack, these crystals are powerful triggers for other inflammatory mononuclear cells (recruited neutrophils, resident macrophages, monocytes, endothelial cells) with phagocytosis properties and significant resolution of MSU crystal-induced inflammation [7]. All studies have revealed that cells around and within the tophus produce cytokines, chemokines, enzymes and other mediators that could generate the bone and joint complex damage. Innate and adaptive immune cells (macrophages, mast cells, plasma cells and B- and T-cells) have been identified within the tophus [27]. The resident and induced macrophages play the fundamental role in the inflammatory cascade response; these cells, like other mentioned mononuclear cells, synthesize cytokines (pro-inflammatory cytokines – IL-1β, IL-6, IL-8, tumor necrosis factor-α and anti-inflammatory cytokines – IL-10, TGF-β1) and produce lytic enzymes (e.g., matrix metalloproteinases) [33]. Local production of matrix metalloproteinases and tumor necrosis factor-α within the tophus may contribute to the development of the joint damage [34].

IL-1β production is an important process that can orient the therapeutic possibilities in the future [7]. Monocytes are stimulated by MSU crystals to express large quantities of IL-1β, due to the inflammasome complexes – an important intracellular mediator in the production of two proinflammatory cytokines IL-1β and IL-8. The term “inflammasome”, defined in 2002, represents a large cytoplasmic complex of several types of proteins that trigger or potentate the inflammatory process (more exactly, because of its ability to initiate IL-1β processing and secretion), and induce cellular pyroptosis, a type of programmed cell death distinct from apoptosis [7, 35].

In our patient, the histological aspects were in accordance with literature data. A variety of cells is presented within the studied tophus. Macrophages were the most numerous cells in the granulomatous mass, where we observed a variety of cells.

We did not study the cell expressing IL-1β that is particularly observed among mononucleated and in multinucleated cells; in the fibrovascular zone, the perivascular expression of cytokine was mentioned [36]. This particular cytokine IL-1β and the cells dualism monocytes–macrophages are considered the most important factors in the formation of the gouty tophus, like in the initiation and resolution phases of acute gout [36].

The immunohistochemical analysis showed increased activity of the cells in the granuloma structure examined. The existence of the markers mentioned in the table, justify the intensity of the inflammatory reaction, with a polyarticular and dysfunctional marked clinical expression.

The studied lymphocyte markers orient us on the intensity of the immune and inflammatory process and arguments the architectural complexity of the tophi. The overall number of CD20+ B-cells was much lower, but CD10+ was significantly present in all inflammatory cells, so the adaptive immune system in gouty tophi in our patient was modified.

Corroborating our patient’s data, we can take into account an innate change in his immune response, because the analysis for macrophages revealed the presence of numerous CD68+ cells, a cell of the innate immune system in gouty tophi [36]. The innate immune system, as distinct from the adaptive immune system of T- and B-cells, plays a pivotal role in the mechanism by which MSU crystals generate and promote an inflammatory response in joints and other soft tissues [37].

In our patient, the MSU accumulation is clinically expressed as gouty hand arthritis, tophi formation with dactylitis aspects and history of nephrolithiasis. The clinical and histological assessments are two arguments that in our patient could not follow the normal pattern for the resolution of gout; this normal pattern includes three phases: removal and neutralization of MSU crystals, clearance of apoptotic cells and a switch of cytokine patterns from the pro-inflammatory to anti-inflammatory [7].

The histological studied aspects were clear for gout tophi. There is no the suspicious for chondrocalcinosis (calcium pyrophosphate dehydrate crystals have small size, rhomboid shape and no positive birefringence) or confusion with rheumatoid nodule (a palisading granuloma) – two of the most common differencing aspects.

**Treatment aspects**

In our patient’s treatment plan, we respected the baseline recommendations for patients diagnosed with gout. We considered that the optimal management of gout include lifestyle changes and three different classes of medication (those for treatment of the acute attack, those for prophylaxis of attacks and those that reduce body burden of uric acid) [38].
The mechanisms through which MSU crystals cause inflammation have also been intensively studied and these insights are likely to affect the therapy of hyperuricemia and gout in the future.

New treatments are available today and on the horizon for tomorrow, which offer a better quality of life for gout sufferers. These include Febuxostat (a non-purine inhibitor of xanthine oxidase with a potentially better combination of efficacy and safety than Allopurinol) and investigational inhibitors of URAT-1 (an anion exchanger in the proximal tubule that is critical for uric acid homeostasis). New abortive treatments include IL-1 antagonists that can cut short the acute attack in one to two days in patients who cannot take non-steroidal anti-inflammatory drugs, Colchicine or corticosteroids. Lastly, antagonists that can cut short the acute attack in one to three days in patients with hyperuricemia that are likely to affect the therapy of hyperuricemia and gout in the future.

Despite the fact that the gouty attacks were not frequent in our patient, these became more severe with a tendency to involve almost all joint fingers of the hand. The MU crystals accumulated around soft tissues generated the tophi especially in hand digits, with an onset mimicking a rheumatoid arthritis.

Our complex data assessment represents an argument for the particular host reaction to MSU crystals, with formation of the tophi – complex and organized chronic inflammatory tissues.

The complete histological and imagistic studies (the longitudinal setting in chronic tophus gout can be available by ultrasound exam) can complement one another and help improve the clinical management of patients with this painful and potentially debilitating disease.

Conclusion
Despite the fact that the gouty attacks were not frequent in our patient, these became more severe with a tendency to involve almost all joint fingers of the hand. The MU crystals accumulated around soft tissues generated the tophi especially in hand digits, with an onset mimicking a rheumatoid arthritis.

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The complete histological and imagistic studies (the longitudinal setting in chronic tophus gout can be available by ultrasound exam) can complement one another and help improve the clinical management of patients with this painful and potentially debilitating disease.

References
Corresponding author
Rodica Trăistaru, Lecturer, MD, PhD, Department of Physical Medicine and Rehabilitation, University of Medicine and Pharmacy of Craiova, 2 Petru Rareș Street, 200349 Craiova, Romania; e-mail: rodicatraistru@hotmail.com

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