Case Report

The Mystery of the Swollen Knee

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Abstract

We described a case of a 59-year-old male with fevers, right knee swelling, non-inflammatory synovial fluid with negative gram stain and without crystals. Ultrasound examination of the periarthicular structures of the knee demonstrated intra-tendinous patellar tendon tophaceous deposits and associated deep-infrapatellar gout bursitis. This case highlights the use of bedside ultrasound in the real-time diagnosis of patients with monoarticular inflammatory arthritis, direct quantification of the extent of crystal deposition disease, accurate assessment of inflammatory involvement of joints, bursae, tendons and soft tissues becoming a distinctive imaging modality for evaluation of crystal arthropathies.

INTRODUCTION

Gout is an inflammatory disease induced by the precipitation of monosodium urate (MSU) - crystals in a variety of tissues, joints, periarthicular, and soft-tissues. Imaging modalities such as musculoskeletal ultrasound play an important role in the diagnosis, evaluation and treatments of patients with crystal-induced arthritis. Furthermore, this case illustrates the importance of ultrasound in the detection, localization and aspiration of joint fluid for the presence of MSU- crystals under polarized microscopy.

CASE PRESENTATION

54-year-old man was admitted to the hospital after four days of severe pain and swelling of the right knee, associated with subjective fevers. The patient was discharged from the hospital one-week prior, after a right big toe distal phalanx amputation for osteomyelitis. He was at home for three days after the surgery when he developed swelling and pain of his right knee. The symptoms worsened over the next three days and he was unable to ambulate, at which point he returned to the hospital. He reported mild discomfort in the right knee for a few months prior to admission, especially with kneeling. He denied any locking or giving out of the right knee and had no prior history of trauma. No other joints were painful, swollen or tender. On occasions, he felt hot but did not check his temperature. In the emergency room he was a febrile and received intravenous (IV) vancomycin and Unasyn.

MEDICAL HISTORY

He suffered from insulin dependent diabetes mellitus, diabetic neuropathy, chronic kidney disease stage III, coronary artery disease, stroke, seizure disorder, obstructive sleep apnea, chronic obstructive pulmonary disease (COPD), hypertension, and osteomyelitis with 2 prior amputations of his 1st and 2nd distal phalanxes of the right foot. He had a history of Osgood-Schlatter disease in childhood.

Medications and allergies

He was taking Augmentin 875/125mg BID, Aspirin 81mg, Dilantin 200mg TID, Humalog Insulin, Metoprolol 25mg BID, Nexium 40mg, Norvasc 7.5mg, Pravachol 40mg and Advair Diskus 250/50mcg/BID. He was allergic to Codeine.

Family and social history

The patient denied any family history of rheumatologic disorders, including psoriasis. He lived independently by himself and denied alcohol abuse, smoking and illegal drug use prior to admission.

Review of systems

He complained of mild low back pain and shortness of breath, subjective fevers and inability to ambulate. He denied morning stiffness, history of tick bite, eyes pain or erythema, no skin rashes suggestive of psoriasis and no bowel symptoms.

Physical examination

The vital signs were within normal range with a temperature of 98.7F. The right knee had a large effusion and extremely limited range of motion (Figure 1A). It was tender to palpation and warm to touch without erythema. The rest of the joints were without effusions or synovitis and had normal range of motion. No tophaceous deposits were identified over the ear, elbow or other joints. The remainder of the physical exam was unremarkable.

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Laboratory evaluation

WBC 13 cells/mm³, Hemoglobin 11 g/dl, Platelets 264,666/mm³, Creatinine 1.4mg/dl, ESR 103 mm/h, CRP 43 mg/l, Uric acid 5.9 mg/dl. Urine analysis had 100mg/dl proteinuria, +1 blood, without bacteria. Two blood cultures were drawn and had negative Gram stains.

The chest radiography was normal and Doppler ultrasonography of the calves did not demonstrate the presence of thrombi. The radiography of the right knee showed a moderate effusion and a small ossicle in the patellar tendon consistent with prior history of Osgood-Schlatter disease in childhood (Figure 2).

The right knee was aspirated and yielded clear, non-inflammatory fluid (Figure 3A) with a nucleated cell count of 110 cells/mm³. Gram stain, culture and crystal analysis was negative.

CASE SUMMARY

54 year-old man with recent history of osteomyelitis of his right toe, status post amputation, returns to the hospital after four days of right knee pain and severe swelling. The synovial fluid aspirated from the right knee was found to be non-inflammatory with negative gram stain and without crystals.

Hospital course

Musculoskeletal ultrasound was used to further evaluate the right knee effusion and adjacent structures as well as the contralateral knee for comparison (Figure 1B). Standard scans of the knees were obtained based on the European Society of Musculoskeletal Radiology (ESSR) standardized scans [1]. These include both longitudinal and transversal planes: quadriceps tendon, suprapatellar and parapatellar joint recesses, patellar tendon, medial, lateral and posterior knee. The femoral trochlea could not be visualized by ultrasound due to the patient’s inability to flex the knee. Evaluation of the suprapatellar, medial and lateral recesses of the knee showed moderate anechoic signal on gray scale that was consistent with the moderate effusion (Figure 1B) as seen on X-ray. There was no appreciable synovitis and negative for power-Doppler signal. On MSUS, the presence of crystalline deposits seen in crystal-induced arthritis have a characteristic sonographic pattern with MSU crystals deposition appearing as hyperechoic deposits on the surface of the synovial-hyaline or articular cartilage interphase known sonographically as the “double contour sign” while CPP-crystals appear as intra-cartilaginous specks within the hyaline cartilage [2]. Both of which were not appreciated under ultrasound. Microtophi can sometimes be seen in soft tissues or synovium as floating hyperechoic foci resulting in the classic sonographic “snow storm appearance” [3].

Evaluation of the extensor mechanism of the knee showed the normal fibrillar pattern of the patellar tendon with hyperechoic, linear density identified at the insertion of the patellar tendon at the tibial tuberosity (Figure 4). These hyperechoic linear densities exhibited post-acoustic shadowing consistent with a bony fragment. The location correspond to the ossicle seen on the knee X-ray (Figure 2B). The MSUS findings were characteristic of an Osgood-Schlatteras previously described in the literature [4]. In additions, there was moderate effusion in the deep infrapatellar bursae containing some hyperechoic signals. An intense power-Doppler signal was identified within the inferior margin of the patellar tendon (Figure 5 (B and D)). Longitudinal and transverse views of the patellar tendon of affected and non affected knee are shown in Figure 5 (A and C). On further
examination of the patellar tendon proximally by ultrasound, we were able to confirm an heterogeneous hyperechoic formation on gray-scale (Figure 5 (B and D)). The round formation extended through the entire tendon thickness, disrupted the normal fibrillar pattern of the tendon and contained multiple hyperechoic specs w/o post-acoustic shadowing. A large deep infrapatellar bursitis was also identified under the intratendinous lesion. The differential diagnosis of this lesion included an infectious abscess, gouty tophus, heterotropic calcification, chronic partial tear of the tendon with calcifications, benign or malignant tumors.

Using ultrasound guidance, the deep infrapatellar bursa was aspirated. The aspirated fluid was pink and cloudy (Figure 4A (B)) and had 43,000 nucleated cells/mm³. Under polarized light microscopy, the synovial fluid revealed many intra and extra cellular monosodium urate (MSU) crystals (Figure 4B). Both the knee and the deep infrapatellar bursa were injected with triamcinolone acetonide and he was able to bear weight and ambulate 3 days later. The final diagnosis in our case was an acute gout flare with patellar tendon tophus, infrapatellar bursitis and sympathetic effusion of the knee joint.

**DISCUSSION**

The evaluation of a hot knee with a large effusion is usually straightforward and does not require additional imaging. The history, physical examination, routine labs and a complete synovial fluid analysis will indicate the diagnosis in the majority of cases. In our patient however, the non-inflammatoty effusion and the absence of crystals in the synovial fluid made the usual etiologies unlikely and failed to explain the subjective fevers, elevated WBC and ESR.

US allowed us to identify the intra-tendinous tophus and infrapatellar bursitis which revealed the presence of MSU-crystals under polarized light microscopy and settled the diagnosis. Tophi have a fairly characteristic appearance under ultrasound evaluation [5]. They are usually heterogeneous and hyperechoic, with poorly defined contours and hypoechogenic halos. Using the power Doppler signal, active inflammation can be detected to confirm active inflammatory disease. MSUS appears as sensitive if not better than the MRI for the evaluation of tendons [6]. A study evaluating asymptomatic hyperuricemic patients found tophi by MSUS inside the tendons in one third of patients, which suggests that tendons are a preferred location for early MSU crystal deposition [7].

There are only a few case reports in which the initial gout manifestation is a tophus causing patellar tendinitis [8,9]. The reported cases occurred in athletes and a man with history of Osgood-Schlatter disease [8]. The latter case shared many of the uncommon findings of our case: the intra-patellar tendon tophus as the initial gout manifestation, normal uric acid value and a history of Osgood-Schlatter disease with a residual ossicle in the patellar tendon. Tophi tend to form at friction points such as the extensor surface of the elbows. It is possible that chronic recurrent micro-traumas to the patellar tendon favored MSU-crystal depositions and tophi formation.

The treatment of intra-tendinous tophaceous gout follows the general treatment guidelines with a few caveats. Acute attacks are best treated with intra-articular or peri-tendinous glucocorticoid injections. They take longer to resolve and occasionally the symptoms do not respond to injections. Surgical excision (tophectomy) might be useful in the latter situation. Serum uric acid lowering therapy is employed after the attack subsides, with concomitant flare prophylaxis for 6 months. The target for the serum uric acid level should lower, at <5 mg/dl in an attempt to dissolve the tophi in a timely fashion. The time to tophi resolution is directly dependent upon the median serum uric acid [10]. The lower the uric acid concentration, the quicker the tophus dissolves. In a case series, pegloticase was used in two patients with tophaceous gout of the hands [11]. The uric acid concentration dropped close to zero and it took 12 weeks for the tophi to dissolve. The choice of the uric acid lowering agent...
will vary depending on the response to injections, recurrences and desired level of activity. Oral agents such as allopurinol and febuxostat, will likely take longer to decrease the uric acid and dissolve the tophus, but they should be considered first line agents if the clinical situation permits.

This brief case highlights three important things to consider in our daily rheumatology practice when dealing with a monoarthritis. In the presence of a hot, swollen knee with normal synovial fluid analysis that fails to explain the overall clinical and laboratory scenario, attention should be given to the adjacent structures such as the patellar tendon and infra-patellar bursa that may provide diagnostic clues. This is the first reported case in which a tophus in the patellar tendon causes a non-inflammatory knee effusion as initial manifestation of gout. If this possibility is not considered, unnecessary workup and treatment could ensue. The high likelihood of infection in this case would have required surgical washout and long-term antibiotics, which would not have improved the symptoms, leading to expensive readmissions. Furthermore, it highlights the utility of ultrasound with power-Doppler signal in the diagnosis, detection, localization and aspiration of joint fluid for evaluation under polarized light microscopy [12]. US proves to be an extremely useful, inexpensive and accurate tool to aid the clinical examination of challenging cases involving the knee or any other affected joint and facilitates corticosteroid injections under ultrasound guidance [13], when nonsteroidal anti-inflammatory drugs and oral therapy are not tolerated or contraindicated. Lastly, our case raises the question whether patients with a history of Osgood-Schlatter disease have an increased risk of developing tophi in the patellar tendon. This hypothesis will certainly need further evaluation with a formal study.

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Author contributions

All authors were involved in the patient’s care, in obtaining the MSUS images and in the drafting of the article. All authors approved the final version of the manuscript to be published.

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