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Original article

Crystal-induced arthritis after arthroplasty: 7 cases

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ABSTRACT

Objectives: To describe the occurrence in prosthetic joints of crystal-induced arthritis (CIA) defined as the deposition within the synovial membrane and/or joint cavity of calcium pyrophosphate dehydrate (CPPD) (chondrocalcinosis), sodium urate (gout), or hydroxyapatite.
Methods: We retrospectively reviewed the 7 cases of prosthetic-joint CIA seen between 1993 and 2013 at a medical-surgical center specialized in the management of osteoarticular infections.
Results: The 4 females and 3 males ranged in age from 67 to 79 years. Acute CIA occurred at the knee in 6 patients (5 with total knee arthroplasty and 1 with unicompartmental knee arthroplasty) and at the hip in 1 patient (with total hip arthroplasty). Time from arthroplasty to CIA varied from 7 days to 9 years. An abrupt onset was a consistent feature, with pain, complete loss of function, and local evidence of inflammation. A single patient had a fever and 6 patients had laboratory evidence of systemic inflammation. Joint aspiration showed hemarthrosis in 3 patients and inflammatory joint fluid with 20,000 to 79,000 neutrophils/mm^3 in 6 patients. Joint fluid cultures were negative in 6 patients. CPPD crystals were evidenced in 5 patients, including 1 who also had hydroxyapatite crystals detected by electron microscopy after alizarin red staining. Monosodium urate crystals were found in 1 patient. The remaining patient had both CPPD crystals and positive cultures for *Campylobacter fetus*. In 5 patients, treatment with colchicine or a nonsteroidal antiinflammatory drug ensured prompt control of the symptoms and systemic inflammation. The patient with total hip arthroplasty underwent joint aspiration for hemarthrosis. In 1 patient, an intraarticular injection of triamcinolone hexacetonide improved the symptoms and systemic inflammation. The patient with *Campylobacter fetus* infection was treated with antibiotics, excision of the abscess, and synovectomy.
Conclusion: CIA may occur after arthroplasty, within synovial membrane remains or neosynovium developed around the prosthetic joint. CIA is a manifestation of a metabolic disease that persists and can reactivate after surgery. Routine testing for crystals is rarely performed in patients with sterile arthritis of a prosthetic joint, and crystals are difficult to detect in joints with hemarthrosis; consequently, the frequency of prosthetic-joint CIA may be underestimated. Although rare, CIA should be considered routinely when symptoms suggesting septic arthritis develop in a prosthetic joint, in order to avoid unnecessary prolonged antibiotic therapy and, in some cases, surgery. The treatment is usually simple.

1. Introduction

Crystal-induced arthritis (CIA) is due to the deposition of microcrystals within the synovial membrane or joint cavity. The most common form is gout, in which the crystals are composed of monosodium urate (MSU). The deposition of calcium pyrophosphate dehydrate (CPPD) crystals is known as chondrocalcinosis. Hydroxyapatite crystals may also cause CIA. The usual presentation is acute arthritis with severe joint pain, local evidence of inflammation and, in some cases, a fever. These symptoms may suggest septic arthritis, which can occur concomitantly. Infection is a severe complication of joint replacement surgery that is difficult to diagnose and to treat. CIA of prosthetic joints, in contrast, is a rare occurrence of which few cases have been described [1–15], resulting in low awareness among clinicians.

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Table 1
Clinical presentation in 7 patients with prosthetic-joint crystal-induced arthritis.

Patients	Sex	Age at diagnosis Year of diagnosis	Type of prosthesis	Indication	Time to symptom onset	Fever	Initial CRP, mg/L	Preop. X-ray	Joint fluid	Treatment	Follow-up CRP, mg/L	Antibiotic therapy
1	F	83 years 2013	THA right	Hip OA	4 years	No	Negative	No linear calcifications	Hemarthrosis Negative cultures	Colchicine	Negative	No
2	M	79 years 2010	TKA right	Knee OA	7 days	39°	250	Linear calcification	Hemarthrosis Cells 22,000/mm^3 Neu. 97% Cultures-CPPD	Colchicine	Negative	Yes, for 48 h
3	M	75 years 2012	UKA left	Medial tibiofemoral OA	28 days	No	255	Linear calcification	Hemarthrosis Cells 21,000/mm^3 Cultures-CPPD	Colchicine	Negative	No
4	F	73 years 1993	TKA right	Knee OA	3 years	No	76	Linear calcification	Cells 24 600/mm^3 CPPD Hxdroxy-apatite Cultures-CPPD	Naproxen	Negative	No
5	M	67 years 2013	TKA right	Rheumatoid arthritis	9 years	No	59	No linear calcifications	Cells 26,000/mm^3 Cultures-CPPD	Local glucocorticoid and Colchicine	17	No
6	F	72 years 2012	TKA left	Knee OA	31 days	No	182	No linear calcifications	Cells 21,000/mm^3 MSU	Colchicine	Negative	Yes
7	F	77 years 2011	TKA right	Knee OA	15 days	No	244	Linear calcification	Cells 79,000/mm^3 CPPD *Campylobacter fetus*	Antibiotics	40	Yes

The objective of this study was to describe cases of prosthetic-joint CIA seen at a medical-surgical center specializing in the management of osteoarticular infections.

2. Methods

We conducted a retrospective study at the rheumatology and orthopedic surgery departments of a single center, the Diaconesses Croix Saint-Simon Hospital, Paris, France, which houses a referral center for complex osteoarticular infections (CRIOA). We searched the CRIOA database for cases of prosthetic-joint arthritis seen between January 1993 and December 2013. We identified 1510 cases of prosthetic-joint inflammatory arthritis including 941 at the hip, 517 at the knee, 34 at the shoulder, and 5 at other joints. We selected patients with total hip arthroplasty (THA), total knee arthroplasty (TKA), or unicompartmental knee arthroplasty (UKA). Among them, we identified patients who underwent joint aspiration to investigate acute symptoms affecting the prosthetic joint and who met the following criteria for CIA: microcrystals visible in the joint aspirate; or radiological evidence of articular CPPD deposition disease of another joint or of the operated joint before the arthroplasty; and absence of another diagnosis; and favorable outcome during systemic or local antiinflammatory therapy. One of the joint aspirate samples was stained with alizarin red then examined under the electron microscope, which showed crystals of hydroxyapatite.

3. Results

The study included 7 patients, 4 females and 3 males ranging in age from 67 to 79 years (median, 73 years). Table 1 reports their main characteristics. TKA was by far the most common site, with 5 cases; the other sites were UKA in 1 patient and THA in 1 patient. Time from arthroplasty to symptom onset varied from 7 days to 9 years; it was less than 1 month in 4 cases and ranged from 4 to 9 years in 3 cases. The abrupt onset of pain with complete loss of function was a consistent feature. Local evidence of inflammation was apparent at the knee ($n = 6$) and a large swelling at the hip ($n = 1$). A fever was noted in a single patient (39 °C). None of the patients had chills. Laboratory evidence of systemic inflammation was found in 6 of the 7 patients (median C-reactive protein level, 182 mg/L).

Joint aspiration showed hemarthrosis in 3 patients and inflammation with 20,000 to 79,000 neutrophils/mm^3 in 6 patients. Of the 6 patients with sterile joint fluid cultures, 5 had CPPD crystals, including 1 who also had hydroxyapatite crystals; the remaining patient had MSU crystals. Finally, 1 patient had CPPD crystals and acute *Campylobacter fetus* infection.

Six patients had a documented history of CPPD deposition disease or gout. The patient with THA and hemarthrosis (patient #1) had previously experienced similar episodes, which had remained unexplained, despite a 20-year history of severe polyarticular CPPD deposition disease affecting the spine, knees, wrists, and shoulders. One patient had rheumatoid arthritis with erosions but met clinical and ultrasound criteria for disease remission, with no signs of structural progression.

In 6 patients, the joint radiographs taken before the arthroplasty procedure showed cartilage calcifications typical for articular CPPD deposition disease; these calcifications were located within the replaced joints in 4 patients and at other joints in 2 patients. None of the patients had radiological evidence of prosthetic loosening.

Treatment was with colchicine in 5 patients and a nonsteroidal antiinflammatory drug in 1 patient. The symptoms and laboratory evidence of inflammation responded promptly in 5 of these 6 patients. In a patient who responded only partially to colchicine, an intraarticular injection of the glucocorticoid triamcinolone hexacetonide was effective in improving the symptoms and systemic inflammation. Another patient required aspiration to treat hemarthrosis of a prosthetic hip and was then given colchicine to prevent a recurrence. The remaining patient had *C. fetus* infection in addition to CIA and was treated with excision, joint lavage, synovectomy, and antibiotic therapy for 3 months.