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Crystal-induced arthritis in prosthetic joints: a systematic review of clinical features, diagnosis, management, and outcomes

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Abstract

Background To summarize clinical presentations, baseline characteristics, diagnosis, treatment, and treatment outcomes through a systematic review of cases of crystal-induced arthritis in prosthetic joints in the literature.

Methods A systematic review of case reports and case series was performed according to Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. A literature search was performed through PubMed/MEDLINE, Google Scholar, Embase, Cumulative Index to Nursing & Allied Health, and Web of Science. We identified case reports/case series in English of adult patients presenting with crystal-induced arthritis (gout, calcium pyrophosphate deposition disease) in prosthetic joints. Articles that met the inclusion criteria were utilized for qualitative data synthesis.

Results We found 44 cases of crystal-induced arthritis in prosthetic joints from 1984 to 2021. Crystal-induced arthritis in periprosthetic joints most frequently affects patients who had knee arthroplasty and most often presents as monoarticular arthritis that is usually acute in onset. However, several cases in the literature involved patients who had bilateral knee replacements and presented with a concurrent flare of gout or calcium pyrophosphate deposition disease in bilateral knees. Patients with crystal-induced arthritis in prosthetic joints show elevated white blood cell counts with neutrophil predominance and respond favorably to anti-inflammatory treatments, usually within one week. In many cases, crystal-induced arthritis was challenging to differentiate from prosthetic joint infection, with approximately one-third of patients undergoing surgical intervention and 35% receiving antibiotic treatment.

Conclusion Crystal-induced arthritis in prosthetic joints can mimic prosthetic joint infections and should always be considered in the differential diagnoses of joint pain in prosthetic joints. We present the first systematic review of crystal-induced arthritis in prosthetic joints to increase awareness of the diagnosis and proper management.

Keywords Gout, Calcium pyrophosphate deposition disease, Crystal-induced arthritis, Prosthetic joints

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Introduction

Crystal-induced arthritis is characterized by joint inflammation due to crystal deposition. The primary etiologies of crystal-induced arthritis include gout and calcium pyrophosphate deposition disease, which involve monosodium urate (MSU) and calcium pyrophosphate dihydrate (CPPD) crystal deposition, respectively. Crystal-induced arthritis is common, with gout affecting approximately 1–4% of adults worldwide and calcium pyrophosphate deposition disease having an estimated prevalence of 4–7% among adults in the United States and Europe [1]. In contrast, crystal-induced arthritis in prosthetic joints is poorly characterized, with only a few case reports in the orthopedic literature [2].

Total arthroplasty, especially total hip and knee arthroplasty, is increasingly used in treating arthritis. The number of total hip and knee arthroplasties is anticipated to increase significantly over the next few decades as populations in advanced countries age. More patients are undergoing joint arthroplasty at a younger age. With the number of arthroplasties increasing and more adults undergoing arthroplasty, research into one of the common causes of arthroplasty failure-periprosthetic joint infection (PJI) [2] and conditions that may mimic PJI is critical to improving the quality of care [3–5].

Infection control is crucial in managing PJI and therefore involves surgical intervention and antibiotic therapy in most cases. In contrast, most reported cases of crystal-induced arthritis in prosthetic joints have been managed medically with agents like colchicine and nonsteroidal anti-inflammatory drugs [6]. Consequently, unnecessary surgery could result from crystal-induced arthritis presumed to be PJI. Therefore, this study aimed to systematically review the literature to characterize cases of crystal-induced arthritis in prosthetic joints.

Methods

This systematic review was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [7, 8]. Before initiating the literature search, the study protocol was registered with The International Prospective Register of Systematic Reviews (PROSPERO), PROSPERO ID: CRD42022362899. Specifically, MKH, an information services and instruction librarian, was enlisted to conduct an extensive systematic search through PubMed/MEDLINE, Google Scholar, Embase, Cumulative Index to Nursing & Allied Health (CINAHL), and Web of Science. The language was limited to English. The search strategy (Appendix A) involved relevant keywords, including gout, calcium pyrophosphate deposition disease, crystal-induced arthritis, prosthetic joint, and patient population (adult patient). De-duplication and screening of articles were undertaken using Covidence, a web-based collaboration software platform that streamlines the production of systematic and other literature reviews [9]. Two authors (HS and JD) independently screened all titles and abstracts obtained from the literature search from 1984 to 2021. The remaining articles underwent a full-text assessment to determine eligibility based on the inclusion criteria, including the article must be written in English, it must be a case report or case series, but not a review, the patient must be 18 years old or more, and present with proven crystal-induced arthritis of a prosthetic joint without another diagnosis (infection ruled out). Any disagreements between the two reviewers were resolved with discussion or the involvement of a third reviewer (SYL). A standardized data collection form that followed the PRISMA and Cochrane Collaboration guidelines for systematic reviews was used to obtain information regarding the name of authors, year of publication, country of origin, study characteristics (symptoms. age, gender, comorbidities, the reason for prosthetic joints, locations of affected joints, number of affected joints, laboratory findings: serum WBC (white blood cell) count, CRP (c-reactive protein), ESR (erythrocyte sedimentation rate), serum urate, means of diagnosis, synovial fluid analysis (synovial fluid WBC, synovial fluid polymorphonuclear leukocytes (PMNs), type of crystals), treatments, time course (time from prosthetic surgery to onset of symptoms, time from onset to diagnosis, time from therapeutic initiation to symptomatic resolution), and limitations. We calculated descriptive statistics to summarize the clinical characteristics of the included cases. We conducted analyses using JMP statistical software, version 15.1 (SAS Institute Inc., Cary, NC).

Results

Figure 1 shows a PRISMA flow diagram summarizing the identification, screening, eligibility, and inclusion and exclusion processes of the studies involved. The initial MEDLINE, Embase, Web of Science, CINAHL, and Google Scholar databases review yielded 405, 879, 381, 64, and three articles, respectively. We removed three hundred thirty-nine duplicate studies. A total of 1393 articles were screened based on their relevance and type, whereas 1341 were either review articles, editorials, or focused on matters irrelevant to the research question and were excluded from the study. We evaluated 55 articles for full-text review. Review articles or articles that did not meet our inclusion criteria were excluded. As a result, 36 articles, including 44 cases from case reports and series, were included in the review) (Appendix B) [2, 10–43].

Table 1 presents the baseline demographics, diagnostic findings, chief clinical symptoms, indications for prosthetic joints, and affected joints from the individual cases ($n=44$). The median age of the included cases was

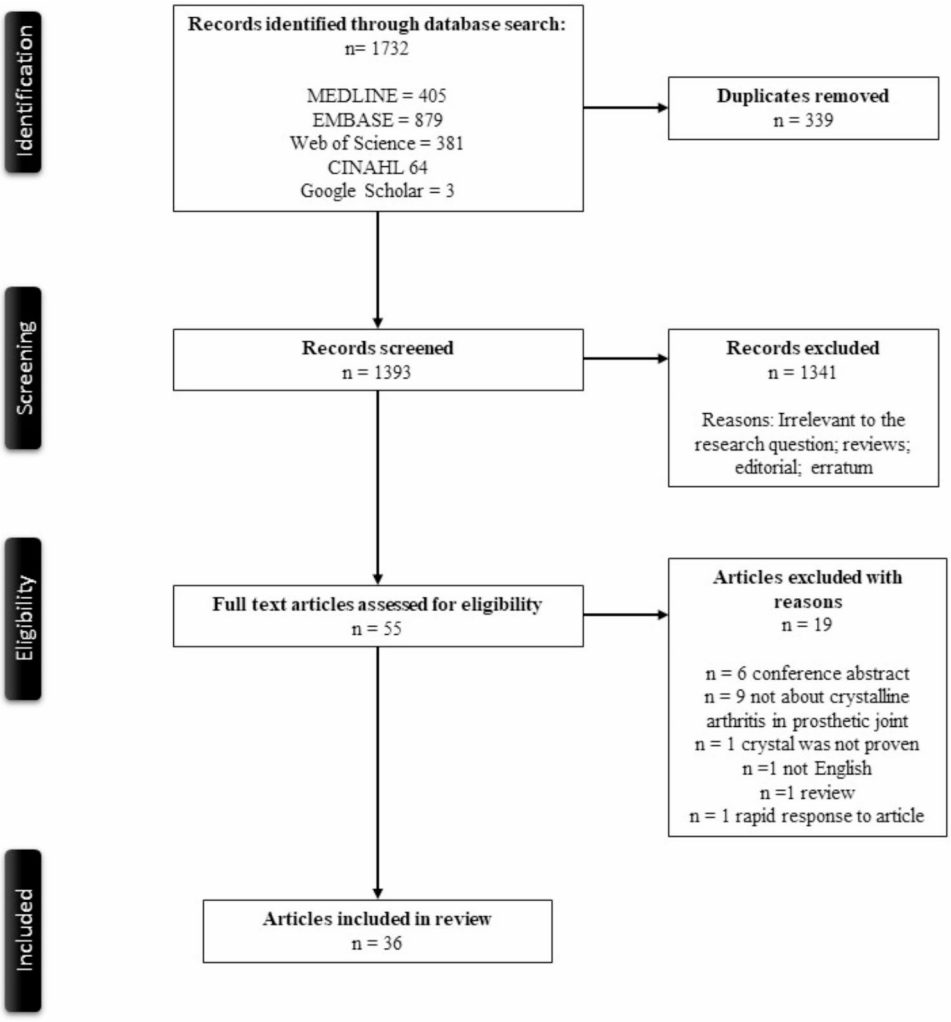

Fig. 1 PRISMA flow diagram

71.0 years (interquartile range [IQR] 61.8–77.0) without skewed deviation in terms of sex. History of gout and CPPD was found in 44.8% (13/29) and 5.0% (1/20), respectively. The median time from onset to diagnosis was three days (range 1–28 days) The most common symptoms included joint pain (97.6%), joint swelling (100%), and warmth (100%) of the affected joint. 47.5% of patients had a fever. The most common indication for prosthetic joints was osteoarthritis (59.1%, 26/44). Approximately 90% of cases involved prosthetic knees, followed by the hips and metatarsophalangeal joints. In terms of initial presentation, 80% of patients were monoarticular. Approximately 15.9% of cases involved two prosthetic joints, most commonly the contralateral joint. Of the cases involving two joints where the contralateral joints were affected, 6 cases occurred in prosthetic knees (the patient had bilateral knee replacement), and 1 case occurred in contralateral prosthetic metatarsophalangeal joints (the patient had bilateral silicone interposition arthroplasty). In contrast, two cases occurred in the contralateral native joint.

Table 2 presents laboratory data and diagnostic findings. According to the available data, the median serum urate level was 8.9 mg/dl (IQR 8.1–10.4). Of 16 cases that reported serum urate level only 2 cases had a uric acid measurement of less than 6.0 mg/dl. Synovial fluid analysis was remarkable for a median WBC count of 22.0×10^3/μL (IQR 9.5–40.1). The median synovial fluid polymorphonuclear neutrophil percentage was 90% (IQR 81.5–95.0). There was no skewed deviation regarding monosodium urate and CPPD crystals, 56.8% and 45.5% respectively. 68% of cases were diagnosed with arthrocentesis. 7.3% were diagnosed directly with synovial biopsy/synovectomy. 19.5% of patients were diagnosed based on arthrocentesis, followed by synovial biopsy or synovectomy. The median time from prosthetic surgery to the onset of symptoms was 7.0 years (IQR 0.19-10.0).

Table 1 Baseline characteristics of patients with gout or calcium pyrophosphate deposition disease in prosthetic joints

Characteristic	*N* (%) or Median (IQR)
Age (years)	71.0 (61.8–77.0)
Sex	
Male	21/44 (47.7)
Female	23/44 (52.3)
Pertinent Medical History	
Gout	13/29 (44.8)
CPPD	1/20 (5.0)
Rheumatological disease	1/10 (10.0)
Diabetes mellitus	11/18 (61.1)
Chronic kidney disease	10/18 (55.6)
Hypertension	14/19 (73.7)
Medication use	
Allopurinol	8/14 (18.2)
Chief Symptoms	
Fever	19/40 (47.5)
Joint pain	41/42 (97.6)
Joint swelling	37/37 (100)
Erythema	17/22 (77.3)
Warmth of affected joints	23/23 (100)
Decreased range of motion	30/41 (73.2)
Reason for Prosthetic Joints	
Osteoarthritis	26/44 (59.1)
Unspecified	15/44 (34.1)
Others	3/44 (6.8)
Affected Prosthetic Joints	
Knee (includes 6 cases of bilateral knee arthroplasty involvement)	41/44 (93.2)
Hip	2/44 (4.5)
1st MTP	1/44 (2.3)
Number of Affected Joints on Presentation	
Monoarticular Prosthetic Joint	35/44 (79.5)
Bilateral-Contralateral Prosthetic Joints	7/44 (15.9)
Polyarticular Including Native Joints	2/44 (4.5)

Abbreviations CPPD, calcium pyrophosphate dihydrate crystal deposition disease; MTP: metatarsophalangeal

* Prevalence here is defined as the number of cases reported the variable divided by the number of the total cases

Table 2 Laboratory findings and diagnostic patterns of patients with gout or calcium pyrophosphate deposition disease in prosthetic joints

	Prevalence (%) *	Median (IQR)
Laboratory Findings		
Serum WBC (10^3/μL)	27/44 (61.4)	12.4 (8.2–14.4)
ESR (mm/h)	24/44 (54.5)	67.0 (33.5–97.5)
CRP (mg/L)	29/44 (65.9)	59.0 (14.2–215.8)
Serum urate (mg/dL)	16/44 (36.4)	8.9 (8.1–10.4)
Synovial fluid WBC (10^3/μL)	30/44 (68.2)	22.0 (9.5–40.1)
Synovial fluid PMNs (%)	25/44 (56.8)	90.0 (81.5–95.0)
Type of Crystals		
Monosodium urate	25/44 (56.8)	
CPPD	20/44 (45.5)	
Hydroxyapatite	1/44 (2.3)	
Time from Prosthetic Surgery to Onset of Symptoms (years)	44/44 (100)	7.0 (0.19–10.0)
Time from Therapeutic Initiation to Symptomatic Resolution (days)	31/44 (70.5)	3.0 (1.0–6.0)
Means of Diagnosis		
Arthrocentesis	30/41 (68.2)	
Synovial biopsy or synovectomy	3/41 (7.3)	
Arthrocentesis, followed by synovial biopsy or synovectomy	8/41 (19.5)	

Abbreviations CPPD, calcium pyrophosphate dihydrate crystal deposition disease; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; IQR, interquartile range; PMN, polymorphonuclear; WBC, white blood cell

* Prevalence here is defined as the number of cases reported the variable divided by the number of the total cases

Table 3 Treatment and outcomes of patients with gout or calcium pyrophosphate deposition disease in prosthetic joints

	N (%) or Median (IQR)
Treatment	
Surgery	13/44 (29.5)
Systemic glucocorticoid	11/44 (25.0)
Intraarticular glucocorticoid	2/44 (4.5)
Colchicine	18/44 (40.9)
Antibiotics	15/43 (34.9)
NSAIDs	21/44 (47.7)
Allopurinol	8/44 (18.2)
Time from Therapeutic Initiation to Symptomatic Resolution (days)	4.0 (2.3–7.0)
Death	0/44 (0)

Abbreviations NSAID; nonsteroidal anti-inflammatory drug

* Prevalence here is defined as the number of cases that reported the variable divided by the number of the total cases

Table 3 presents treatments and outcomes. Colchicine, Non-steroidal anti-inflammatory drugs (NSAIDs), and oral/intraarticular steroids were most frequently used. Approximately 35% of patients received antibiotics, and 29.5% underwent surgical intervention. It took a median of 4.0 days (IQR 2.3-7.0) until the resolution of symptoms from the initiation of treatment.

Discussion

In the present study, we thoroughly reviewed case reports of crystal-induced arthritis in prosthetic joints. This is the first systematic review of crystal-induced arthritis in prosthetic joints to increase awareness of the diagnosis and proper management and to clarify detailed clinical presentations, treatments, and time course of symptoms. Crystal-induced arthritis in periprosthetic joints is acute in onset, most frequently affects the knee, and usually presents as monoarticular arthritis. However, there were

several cases in the literature where the patient had bilateral knee replacements and presented with a flare of gout or calcium pyrophosphate deposition disease in bilateral knees. Patients with crystal-induced arthritis in prosthetic joints show elevated WBC count with neutrophil predominance in synovial fluid and respond favorably to anti-inflammatory treatments, including systemic glucocorticoids, colchicine, and NSAIDs, usually within one week. It appears that crystal-induced arthritis in prosthetic joints shows almost similar synovial fluid findings and treatment responses as native joint crystal-induced arthritis. These findings make it challenging to differentiate periprosthetic joint infection from crystal-induced arthritis in prosthetic joints, especially on initial presentation. Our study summarizes the clinical characteristics of crystal-induced arthritis in periprosthetic joints, providing insight for the multidisciplinary team of internists, rheumatologists, orthopedists, and infectious disease physicians involved in patient care.

We found a total of 44 cases of crystal-induced arthritis in prosthetic joints since 1984, when the first case of gout following joint arthroplasty was reported [26]. In contrast, CPPD following a major joint arthroplasty was not reported until 2007 [44]. Crystal-induced arthritis in prosthetic joints is an uncommon diagnosis, but it may be under-reported [35]. Routine testing for crystals may not be routinely performed at many centers when synovial fluid is aspirated from prosthetic joints, and there is the possibility that an inflammatory response from gout or CPPD crystals may cause cases reported as culture-negative prosthetic joint infection. Because of this, there is a paucity of studies and research on crystal-induced arthritis in prosthetic joints. Almost all patients presented with joint pain, swelling, and warmth in the affected joints, while most also had erythema and decreased range of motion. Only about half of patients had a fever. The onset of symptoms to diagnosis was variable (1–28 days) with a median of 3 days, compared to gout and acute CPP crystal arthritis flares, which are typically acute in onset (maximum pain noted within 24 h) [45, 46]. Over half of the patients had elevated serum WBC count, ESR, and CRP. Synovial WBC count was elevated with median polymorphonuclear neutrophils of 90%. About one-third of the patients underwent treatment for presumed PJI with surgery and antibiotics. This result is consistent with literature describing the difficulty distinguishing between crystal-induced arthropathy and PJI presentations in prosthetic joints [2, 12].

Prosthetic joint infection is a common and severe postoperative complication that may be challenging to differentiate from aseptic causes of inflammation, like crystal-induced arthritis, as both may present similarly with symptoms like acute joint pain, swelling, and erythema [2, 45, 46].

The challenge in clinical practice is that no single test provides a definitive diagnosis of prosthetic joint infection. There is a significant overlap between findings found in prosthetic joint infection and crystal-induced arthritis in prosthetic joints [5]. In 2018, the Musculoskeletal Infection Society and the Infectious Diseases Society published criteria to standardize the diagnosis of prosthetic joint infection [47]. While the 2018 criteria have a 97.7% sensitivity and 99.5% specificity, many preoperative minor criteria overlap with crystal-induced arthritis, including elevated inflammatory markers, synovial PMNs, and synovial white blood cell count. Besides specific signs such as sinus tract evidence of joint communication, many criteria were based on an intraoperative diagnosis. Alpha-defensin testing was a minor criterion in the 2018 International Consensus Meeting criteria for PJI. Alpha-defensin is an antimicrobial peptide produced by the innate immune system, and a positive alpha-defensin test has been shown to have a sensitivity of 69–100% and specificity of 94–98% for PJI [35, 48]. While promising, further research is necessary to assess the validity of alpha-defensin testing in periprosthetic crystal-induced arthropathy.

Prompt arthrocentesis and identification and verification of synovial fluid are critical for diagnosis. Ideally, for suspected PJI, arthrocentesis is performed in a sterile environment in an operating theatre (by orthopedics) or interventional radiology suite to prevent sample contamination [49]. However, clinical decision-making is even more complicated, considering that both conditions may present concurrently in the same joint [3, 4], and a missed PJI can have devastating consequences. Further efforts are needed to help develop methods or systems to reliably differentiate PJI and crystal-induced arthritis, especially preoperatively, because treatment, prognosis, and healthcare utilization differ significantly for both conditions. Increased awareness is essential, with consideration given to testing for crystals in synovial fluid samples obtained from prosthetic joints as recommended per guidelines where septic prosthetic arthritis is suspected. Only monosodium urate crystals and calcium pyrophosphate crystals can be identified on light microscopy, while wet preparation with alizarin red stain is needed to identify the presence of calcium hydroxyapatite crystals [50].

A high clinical suspicion is needed to make an accurate diagnosis. Crystalline arthritis flares can occur during the treatment of trauma, as well as before and after surgery [51]. In certain cases, crystalline arthritis flares can be triggered by surgical procedures [52]. We found that 44.8% of patients had a history of gout, while just 5% reported a history of CPPD. As half of the patients had no history of gout or CPPD, crystal-induced arthritis in prosthetic joints should be suspected, even in patients

not reporting a prior history of gout or calcium pyrophosphate deposition disease. Viriyavejkul et al. reported that CPPD crystals were present in 52.9% of patients who underwent knee arthroplasty in a case series of 102 patients [53]. However, almost all patients were unaware of the presence of calcium crystals. Identifying chondrocalcinosis on prior radiographs, while not diagnostic, may be helpful in the diagnosis. Further, we found a wide range of time to presentation, from a few days after surgery to decades after surgery, suggesting crystal-induced arthritis should always be suspected in cases with prosthetic joint pain regardless of the surgery date. Close coordination of care with a multidisciplinary team, including rheumatologists, infectious disease experts, and orthopedic surgeons closely coordinating care, may be optimal in management. Critical aspects of the appropriate management include promptly verifying synovial fluid for the presence of crystals and assessing the probability of infection. Confirmation of a favorable response to anti-inflammatory treatments (NSAIDs, colchicine, and prednisone) is essential. In our review, most cases were resolved within seven days of treatment. Ultimately, the patient may undergo surgical intervention due to difficulty distinguishing PJI and crystal-induced arthritis. In general, intraarticular corticosteroid injections into prosthetic joints are not recommended due to the increased risk of prosthetic joint infections [54].

While the mechanism of crystal-induced arthritis in prosthetic joints is not wholly understood, several authors have provided several suggestions [6, 55]. Calcium pyrophosphate deposition disease crystals are manifestations of metabolic derangement that originate from the cartilage. CPPD crystals deposit in the cartilage leading to joint damage [6, 56, 57]. Implantation of prosthetic joints does not remove all of the cartilage in the joint; therefore, CPPD crystals can still be formed from the persistence of native cartilage (i.e., in the patella with certain types of knee replacement procedures). Furthermore, cartilage can also be formed after joint replacement surgery by cartilaginous metaplasia around the prosthetic joint [6, 55]. Similarly, the pathophysiology of a gout flare requires the presence of synovial tissue. Synovial remnants may persist in the prosthetic joint. Neosynovial tissue also may develop around the prosthesis post-surgery [55]. When monosodium urate crystals are deposited within the synovial tissue, this leads to a gout flare.

Although both time from symptom onset to diagnosis and symptom resolution following appropriate therapy was relatively quick, this delay in diagnosis and treatment adds unnecessary days of hospitalization and, thus, unnecessary costs to both the patient and the hospital. While research into effectively identifying crystal-induced arthritis is needed, consideration should be given to optimizing the situation for patients who undergo arthroplasty. Data from the United States National Inpatient Sample 1998–2014 found that gout was independently associated with an 18% increased risk of discharge to a non-home setting. Gout was also found to increase the length of stay by 8% [58]. Further research is needed to determine if better gout management can reduce the increased healthcare utilization of gout in patients undergoing arthroplasty. Notably, in our study, only very few patients with gout had urate lowering therapy. Optimizing urate control may be beneficial in reducing the risk of gout after arthroplasty; however future research is needed. Of interest, Harato and Yoshida reported the use of prophylactic NSAIDs in a patient with a high risk of flare [25]. Further studies are needed to clarify the utility of prophylactic treatment and determine patients at increased risk of developing crystal-induced arthropathy in prosthetic joints. Some authors have noted the possibility of a more aggressive synovectomy during joint replacement for high-risk patients [59]. However, this remains to be investigated further because synovectomy does not produce improved pain or range of motion outcomes and is associated with increased blood loss and operative time [59].

Strengths of this study include a comprehensive systematic review of all cases reported in the literature of crystal-induced cases in prosthetic joints, involving an experienced librarian, and a multidisplinary team of board certified rheumatologists and orthopedics surgeons following PRISMA guidelines. It provides insight into an understudied area where further research is needed because of the increasing number of joint arthroplasties used to treat arthritis as the population ages from a rheumatology perspective, where the literature has been mainly reported in the orthopedic literature. Several limitations of this study should also be discussed. There were no laboratory results or joint x-rays before prosthetic joint replacement in certain articles, and we could not contact authors to obtain data not mentioned in the literature. Secondly, we did not include review articles, conference abstracts, or preprints, leading to uncertainty in the evidence level discussed.

In conclusion, crystal-induced arthritis in prosthetic joints is a rare condition that presents similarly to PJI. This uncertain clinical picture often leads to unnecessary treatment such as antibiotics and surgery, exposing the patient to the risks involved with those treatments without any benefits. Thus, we present the first systematic review of crystal-induced arthritis in prosthetic joints to increase awareness of the diagnosis and proper management. Crystal-induced arthritis should always be considered in the differential diagnosis of joint pain in prosthetic joints. Prompt diagnosis and treatment with typical crystal-induced arthritis medications should

result in rapid resolution of symptoms and, thus, prevent unnecessary treatment and increased length of hospital stay.

Abbreviations

MSU	Monosodium urate
CPP	Calcium pyrophosphate
CPPD	Calcium pyrophosphate dihydrate
PJI	Periprosthetic joint infection
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses
CINAHL	Cumulative Index to Nursing & Allied Health
WBC	White Blood Cell
CRP	C-reactive protein
ESR	Erythrocyte sedimentation rate
PMNs	Polymorphonuclear leukocytes
IQR	Interquartile range
NSAIDs	Non-steroidal anti-inflammatory drugs

Supplementary Information

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Supplementary Material 1

Supplementary Material 2

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Author contributions
HS- study design, data collection, interpretation of data, drafting, and revising the manuscript. JD - data collection, interpretation of data, drafting, and revising the manuscript. BS - study design, data collection, interpretation of data, drafting, and revising the manuscript. LT - data collection, interpretation of data, drafting, and revising the manuscript. YN - interpretation of data, drafting, and revising the manuscript. MK - study design, data collection. CN - study design, data collection, interpretation of data, drafting, and revising the manuscript. SYL - study design, data collection, interpretation of data, drafting, and revising the manuscript. All authors read and approved the final manuscript.

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Data availability
The data presented in this study are available on request from the corresponding author.

Declarations

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Competing interests
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