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Research Progress on the Correlation between Pseudogout and Periprosthetic Infection after Knee Joint Replacement Surgery

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Abstract: Knee osteoarthritis (KOA) is one of the most common degenerative joint diseases in the elderly, and knee arthroplasty is an important treatment for end-stage KOA, which can effectively improve knee pain and function. However, postoperative joint swelling and pain are common clinical complications. Pseudogout caused by calcium pyrophosphate deposition and periprosthetic infection are the most confusing causes. The core strategies of the two treatments are completely different, and differential diagnosis is the core difficulty in clinical practice. The purpose of this paper is to systematically review the metabolic background and triggering factors, clinical characteristics, and key points of identification of periprosthetic infection in pseudogout after knee arthroplasty, so as to provide a reference for clinical optimization of the differential diagnosis process and reduction of misdiagnosis.
Keywords: Pseudogout; Knee osteoarthritis; Periprosthetic infection; Knee replacement

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1. Introduction

Knee osteoarthritis (KOA) is a common orthopedic disease in older adults, characterized pathologically by degeneration of articular cartilage and clinically by chronic pain, joint stiffness, and limited mobility, which seriously affects patients' quality of life [1]. With population aging and rising obesity rates [2], the prevalence of KOA has increased year by year, making it one of the leading causes of disability in older adults and imposing a heavy burden on society and families. When KOA progresses to the end stage, conventional conservative treatment is often ineffective, and surgical intervention is usually required [1].

Knee arthroplasty is an effective means of treating end-stage KOA, relieving knee pain, correcting deformity, and restoring joint function [3]. However, postoperative knee swelling and pain are the most common clinical manifestations and are often closely associated with periprosthetic joint infection (PJI). Although the incidence of PJI is relatively low, for affected individuals it can cause the suffering associated with multiple surgeries and long-term antibiotic treatment, seriously affecting functional recovery and long-term implant survival [4]. Therefore, rapid and accurate identification of the nature of postoperative joint swelling and pain is a critical clinical task.

Pseudogout is the acute form of calcium pyrophosphate deposition disease (CPPD); it is caused by the deposition of calcium pyrophosphate (CPP) crystals in the joint and the triggering of an acute inflammatory response, and it occurs predominantly in older adults [5]. The knee is the most commonly involved site in pseudogout, followed by the wrist; the shoulder, hip, and ankle may also be affected [6]. Epidemiological surveys show that prevalence increases with age [7]. The clinical manifestations of pseudogout mainly include sudden severe joint pain, redness, swelling, elevated skin temperature, joint effusion, and systemic inflammatory responses such as fever, which overlap considerably with the manifestations of PJI, making differentiation extremely difficult and posing a major clinical challenge.

Therefore, accurate and timely differentiation between these two conditions is of great clinical significance. However, existing research is relatively limited, and clinicians lack sufficient awareness of this issue. Against this background, from the practical perspective of orthopedic clinical practice, this article systematically reviews the metabolic background and clinical features of pseudogout after joint arthroplasty and the key points for differentiating it from PJI, aiming to provide a reference for clinicians to optimize the differential diagnosis process and reduce misdiagnosis and unnecessary surgical intervention.

2. Current epidemiological status

2.1. Current incidence of postoperative CPPD

The clinical features of CPPD in native joints are relatively well defined. However, when it occurs after joint arthroplasty, its clinical features show certain particularities. Accurate recognition of these features is a prerequisite for timely postoperative diagnosis and avoidance of misdiagnosis. A large number of clinical studies have reported the occurrence of pseudogout after knee arthroplasty, with a very wide range of onset times. Yahia *et al.* [8] reported 7 cases of acute crystal arthritis after joint arthroplasty, with onset from 7 days to 9 years after surgery; 5 cases were confirmed by synovial fluid examination to be caused by calcium pyrophosphate deposition. Matsubara *et al.* [9] reported an individual case with the first episode occurring 14 years after surgery. These cases indicate that the risk window for postoperative pseudogout extends throughout the entire postoperative period rather than being limited to the early postoperative stage.

2.2. Difficulty in differentiating postoperative pseudogout from PJI

The acute attack of CPPD closely resembles PJI, and the two overlap extensively in clinical symptoms, inflammatory markers, and synovial fluid appearance [10]. Clinicians' insufficient understanding of postoperative pseudogout, together with the lack of reliable differential markers, leads to a high misdiagnosis rate. A systematic review by Mashayekhi *et al.* [11] showed that a considerable number of patients received unnecessary surgery and antibiotic treatment because of misdiagnosis. This situation highlights the complexity and urgency of differentiating CPPD from PJI.

3. Risk factors and metabolic background of postoperative pseudogout

In some patients, the acute attack of CPPD is an acute inflammatory response triggered by the formation of calcium pyrophosphate crystals in joint tissues and their release into the joint cavity under external stimulation [12]. In knee arthroplasty, local surgical trauma and systemic metabolic factors superimpose on each other, jointly constituting the pathological basis for crystal deposition and acute attacks.

3.1. Hypercalcemia and calcium pyrophosphate crystal deposition

Hypercalcemia is one of the recognized metabolic risk factors for CPPD [13]. From a microscopic mechanistic perspective, the promotion of crystal deposition by hypercalcemia is not a passive precipitation of calcium salts but an active process involving cell signal transduction. Xie *et al.* [14] suggested that elevated extracellular Ca^{2+} concentration or an imbalance in the Ca^{2+}/Mg^{2+} ratio is a key initiating factor promoting the formation of CPP crystals in cartilage. Specifically, a high-calcium environment can stimulate chondrocytes to release adenosine triphosphate, which activates P2 purinergic receptor signaling pathways, leading to intracellular calcium overload and mitochondrial dysfunction, ultimately promoting the binding of pyrophosphate to calcium ions to form CPP and its deposition in the cartilage matrix. This mechanism provides a key microscopic bridge between hypercalcemia and CPPD, indicating that hypercalcemia can cause CPP deposition through supersaturation of free calcium in the cartilage matrix.

3.2. Multiple upstream pathways leading to postoperative hypercalcemia

Multiple independent upstream factors can indirectly increase the risk of CPPD by elevating serum calcium. In the specific context of joint arthroplasty, these factors may act alone or synergistically.

3.2.1. Hyperparathyroidism

Primary hyperparathyroidism is one of the most common causes of hypercalcemia and is characterized by hypercalcemia and markedly elevated parathyroid hormone (PTH) levels, or inappropriately normal PTH levels [15]. Calcium homeostasis is regulated by calcitonin, vitamin D, and parathyroid hormone [16]; excessive PTH production causes hyperparathyroidism, which in turn leads to hypercalcemia.

3.2.2. Vitamin D excess and *CYP24A1* gene variants

Abnormal vitamin metabolism is another important pathway leading to hypercalcemia and can be divided into endogenous and exogenous situations. Exogenous vitamin D excess is usually caused by long-term inappropriate intake of extremely high doses of vitamin D preparations. Excessive vitamin D supplementation can lead to excessively high serum levels of 25-hydroxyvitamin D, increasing intestinal and renal absorption of calcium and phosphorus and increasing osteoclast activity, thereby causing hypercalcemia [17].

Endogenous vitamin D accumulation mainly arises from loss-of-function mutations in the *CYP24A1* gene. CYP24A1 encodes 24-hydroxylase, which is the key rate-limiting enzyme for the degradation of 1,25-dihydroxyvitamin D [18]. When CYP24A1 has an inactivating mutation, active vitamin D cannot be properly degraded and continuously accumulates; even low-dose or conventional-dose vitamin D supplementation can then cause hypercalcemia. This genetically determined high sensitivity to vitamin D has also been reported in infantile acute hypercalcemia [19]. However, substantial evidence indicates that adults carrying the same mutation can also develop symptoms. Baudart *et al.* [18] reported a 52-year-old patient who presented with CPPD; etiological screening revealed a *CYP24A1* gene mutation, and laboratory tests showed

serum calcium at the upper limit of normal and elevated 1,25-dihydroxyvitamin D. This case report provides a new explanatory dimension for susceptibility to CPPD after knee arthroplasty: patients carrying this gene variant may develop pseudogout after surgery even if preoperative vitamin D supplementation is within the normal range, because of the combined effects of immobilization, stress, and other factors.

3.2.3. Postoperative immobilization and increased bone resorption

In addition to the above endocrine and genetic factors, immobilization after joint arthroplasty itself can be an independent promoting factor for hypercalcemia. Reduced limb activity can enhance osteoclast activity and bone resorption, releasing Ca^{2+} from bone into the blood and consequently elevating serum calcium [20]. For patients with a pre-existing tendency toward hypercalcemia, the superimposed effect of postoperative immobilization may push Ca^{2+} above the threshold for triggering CPPD.

3.3. Comprehensive model of pseudogout after joint arthroplasty

Overall, the occurrence of CPPD after joint arthroplasty is not determined by a single factor but is the result of the combined action of the systemic metabolic background and local surgical trauma. This understanding provides a theoretical framework for comprehending the pathogenesis of postoperative CPPD and also provides a basis for identifying high-risk patients during the perioperative period and formulating preventive strategies.

To facilitate systematic understanding of the core features of CPPD after knee arthroplasty, the key points are summarized in **Table 1**.

Table 1. Core features of pseudogout after knee arthroplasty

Item	Key points	References
Definition	Acute form of CPPD, caused by intra-articular deposition of calcium pyrophosphate crystals that induces acute inflammation.	[5]
Epidemiology	More common in older adults; the knee is frequently involved.	[6,7]
Systemic metabolic factors	Hypercalcemia, hyperparathyroidism, vitamin D excess, *CYP24A1* gene variants.	[13,15–22]
Local triggers	Surgical trauma, postoperative immobilization.	[12,20]
Clinical manifestations	Sudden joint pain, swelling, elevated skin temperature, limited mobility; may be accompanied by fever and systemic inflammatory response.	[8,10,21]
Treatment principles	Acute attack: nonsteroidal anti-inflammatory drugs, colchicine, glucocorticoids.	[31]

Note: The above clinical features are not specific in the postoperative setting and require comprehensive judgment to improve diagnostic accuracy.

4. Differential diagnosis between pseudogout and PJI

Differentiating postoperative CPPD from PJI is the core clinical difficulty. The following discussion addresses three aspects: clinical manifestations, auxiliary examinations, and comprehensive differential diagnosis.

4.1. Common features of clinical manifestations and routine tests

During an acute attack of CPPD, CPP crystals detach from the cartilage surface into the joint cavity and

cause an acute inflammatory response, manifesting as sudden joint redness, swelling, pain, elevated skin temperature, joint effusion, and markedly limited range of motion [12]. Some patients also have fever, but body temperature generally does not exceed 38.5°C; fatigue and other systemic symptoms may also occur [21].

The clinical manifestations of PJI are more varied. Acute PJI also presents mainly with joint redness, swelling, heat, pain, and wound drainage; severe cases may have high fever and chills. Chronic PJI is more insidious, often presenting with persistent joint pain, mild swelling, and progressive decline in joint function; some patients may also develop late manifestations such as implant loosening and sinus tract formation [22,23]. However, it is difficult to make an accurate differentiation between the two based solely on clinical manifestations, and auxiliary examinations are also required.

Routine blood tests and synovial fluid tests also have obvious limitations. A systematic review by Bradley *et al.* [21] found that postoperative inflammatory marker values closely resemble the thresholds for PJI. Al-Jabri *et al.* [4] summarized that C-reactive protein can be significantly elevated both after surgery and in infection; erythrocyte sedimentation rate can continue to rise for up to one year after joint arthroplasty. Although novel synovial fluid markers (such as alpha-defensin and leukocyte esterase) show high sensitivity and specificity in the diagnosis of PJI [24], they cannot diagnose CPPD, and the gross appearance of synovial fluid in both conditions is turbid and inflammatory [25]. For these reasons, relying on a single indicator makes accurate differentiation between the two diseases difficult.

4.2. Differential value of synovial fluid crystal analysis and imaging

Synovial fluid crystal analysis is currently the most direct means of differentiation. The ACR/EULAR classification criteria include the detection of CPP deposition in synovial fluid analysis as one of the core diagnostic bases for CPPD [13]. However, the criteria also note that this method has a certain false-negative rate and inter-observer variability in interpretation. It should be noted that synovial fluid testing should be performed within 12–24 hours after aspiration, and infection and CPP deposition can coexist [26]. Therefore, after joint aspiration, crystal analysis and bacterial culture should be performed simultaneously rather than used as substitutes for each other.

Imaging has auxiliary value in differential diagnosis. Radiography can show chondrocalcinosis [27]. PJI on radiographs mostly shows nonspecific changes such as periprosthetic osteolysis and osteopenia; in late stages, periosteal reaction or implant loosening may be seen, which can serve as an important means of differential diagnosis [28]. Conventional CT can more clearly display fine calcifications not obvious on radiographs, and spectral CT reconstruction can better reflect the contrast between the affected joint and the normal joint [29]. Musculoskeletal ultrasound has high sensitivity for cartilage calcification and meniscal calcification [30]. MRI is mainly used to evaluate the extent of soft tissue involvement in PJI and can clearly show inflammation, abscess, and sinus tracts in the soft tissues around the joint [28]. Therefore, when suspicious signals appear, radiography, CT, musculoskeletal ultrasound, and other auxiliary examinations can be combined for differentiation.

4.3. Optimization strategy for the differential diagnosis process

Given that the above examination methods each have blind spots and that a single indicator cannot reliably distinguish the two diseases, clinical differentiation should follow a hierarchical and stepwise strategy. After joint aspiration, crystal analysis and bacterial culture should be performed simultaneously, and different

pathways should be assigned according to the results, as summarized in **Figure 1**.

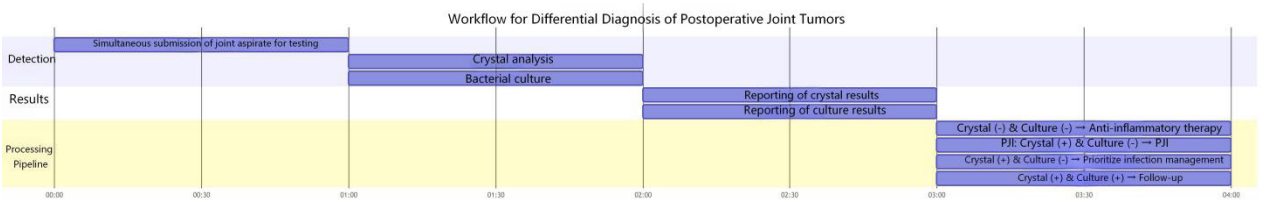


Figure 1. Flowchart for the differential diagnosis of postoperative joint swelling and pain.

To facilitate understanding of the differentiation between postoperative pseudogout and PJI, the key points are summarized in **Table 2**.

Table 2. Differentiation between pseudogout and periprosthetic joint infection

Differential dimension	Pseudogout	Periprosthetic joint infection	References
Systemic symptoms	May have fever, often low-grade	May have high fever and chills	[21]/[22,23]
Local manifestations	Joint redness, swelling, heat, pain, limited mobility	Joint redness, swelling, heat, pain, limited mobility, or accompanied by sinus tract formation and implant loosening	[10,21]/ [22,23,33]
Inflammatory markers	May be markedly elevated in the acute phase, but no characteristic threshold	May be markedly elevated; dynamic monitoring is more valuable	[4,21]
Synovial fluid examination	CPP crystals visible	No crystals	[13]
Bacterial culture	Negative	Positive	[26]
Imaging	Chondrocalcinosis visible on radiography, CT, musculoskeletal ultrasound, etc.	MRI can show the extent of soft tissue involvement	[27–30]/[28]
Treatment principles	Conservative treatment	Surgical intervention	[31]/[32,33]

5. Management strategies and significance

5.1. Fundamental differences in treatment pathways

The management strategies for postoperative CPPD and PJI are completely different, which is precisely why correct differential diagnosis is important.

Acute attacks of CPPD can be controlled with medications, mainly nonsteroidal anti-inflammatory drugs, colchicine, and glucocorticoids [31]. Most patients can achieve relatively rapid symptom relief with oral medications. In contrast, the treatment principle for PJI is primarily surgical. The 2024 guidelines of the British Orthopaedic Association propose a tiered management strategy for PJI [32]: milder cases may undergo debridement combined with antibiotic treatment and implant retention; for cases in which infection is difficult to control, revision surgery is required. The two diseases are diametrically opposed in treatment direction, drug selection, and surgical decision-making. Misdiagnosis not only leads to unnecessary treatment but may also delay the optimal timing of intervention.

5.2. Clinical significance of accurate differential diagnosis

Accurately distinguishing postoperative CPPD from PJI is important in multiple respects, mainly reflected in

the following three points.

The impact on patient prognosis is the most direct. CPPD is an inflammatory disease that can be controlled with medication; timely diagnosis and short-term drug treatment can control symptoms, and joint function is generally not affected [31]. Once misdiagnosed, however, it means long-term antibiotic treatment, unnecessary surgical trauma, and the suffering associated with postoperative rehabilitation. If PJI is not correctly diagnosed early, bacterial biofilms may firmly adhere to the implant surface within weeks, forming a protective barrier against antibiotics and ultimately causing implant–bone interface destruction, bone defects, and implant loosening [22].

Accurate differentiation also has an important impact on the rational allocation of healthcare resources. The cost of revision surgery is usually 2–3 times that of primary arthroplasty. In addition, prolonged hospitalization, repeated imaging examinations, multidisciplinary consultations, and long-term antibiotic treatment bring direct and indirect medical expenditures, and the economic burden caused by misdiagnosis is substantial [33].

In addition, accurate differentiation can reduce patients' psychological burden. As mentioned above, once misdiagnosed as PJI, the uncertainty during long-term treatment and rehabilitation can cause obvious anxiety, which in turn affects rehabilitation training and quality of life [34]. After a clear diagnosis, patients can engage more actively in rehabilitation training. This point is often overlooked in clinical practice, but its impact on final prognosis should not be underestimated.

6. Conclusion and prospects

Postoperative CPPD and PJI show highly similar manifestations after knee arthroplasty, but their treatments are diametrically opposite; accurate differentiation is key to affecting patient prognosis. The two conditions converge through the common endpoint of crystal inflammation and bacterial infection—acute arthritis—but there are fundamental differences in the source of onset, disease course, and management strategies. In brief, CPPD is a crystal-induced aseptic inflammation managed mainly with medication, whereas PJI is an infectious disease caused by bacterial invasion and managed primarily with surgical intervention. Current understanding of this intersecting field is still in its infancy; existing evidence is mostly based on case reports, and systematic differential diagnosis workflows and targeted perioperative management plans are lacking. Future research should focus on the clinical features of the two conditions, explore rapid tools for differentiating infection from crystal detection, and conduct prospective studies in high-risk populations, so as to provide a basis for establishing diagnostic and treatment standards suited to domestic practice.

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