Volume 3, Issue 5, May 2025 ISSN (E): 2938-3765

CLINICAL FEATURES OF CALCIUM PYROPHOSPHATE ARTHROPATHY CHONDROCALCINOSIS

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Abstract
One of the current problems in rheumatology is microcrystalline arthritis.This article describes calcium pyrophosphate arthropathies, their specific etiology, classification, clinical course, and diagnostic methods.

Keywords: Microcrystalline arthritis, calcium pyrophosphate arthropathy, clinic, diagnostics.

Introduction

One of the urgent problems of modern medicine is microcrystalline arthritis, which is a type of joint disease. Over the past 10 years, the incidence of these diseases has increased. Also, their high prevalence among the working-age population is also contributing to the increase in disability. In particular, the latent form of gout is increasing due to microcrystalline arthritis, which is associated with the peculiarities of lifestyle and nutrition, as well as the improvement of diagnostics. Therefore, early detection and proper treatment of these diseases, as well as dispensary observation, are of great importance in preparing a practicing physician for the profession and in his future practical activities.

Regardless of the field of specialization, every doctor should know the symptoms characteristic of microcrystalline arthritis and be able to use the right tactics in managing these patients. In this regard, it is of great importance for the therapist to be able to identify rheumatological diseases, diagnose and choose the right treatment in a timely manner.

DEFINITION

Microcrystalline arthritis is a disease characterized by the deposition of crystals in the synovial fluid, the development of synovitis due to their impregnation of the joint and periarticular tissues. Depending on the type of crystals, the following forms of microcrystalline arthritis are distinguished: [1,2]:

- Gout - deposition of uric acid salt crystals (sodium monourate);
- Calcium-pyrophosphate arthropathy (deposition of pyrophosphate crystals, calcium-pyrophosphate arthritis, chondrocalcinosis, pseudogout)
- Hydroxyapatite arthropathy (deposition of calcium phosphate crystals, calcium-fixing periarthritis)
- Calcium oxalate crystal deposition

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Pathogenic factors:

A. Primary-pathogenic crystals:

Urates - in gout.

Biological crystals - in injuries of joints and periarticular tissues.

Synthetic crystals - when traminolone is injected into the joint.

B. Secondary-pathogenic crystals:

In osteoarthritis, the accumulation of Ca pyrophosphate crystals is assumed.

In chronic inflammatory processes, calcium phosphate accumulates. In hemodialysis arthritis, oxalates accumulate due to hypocalcemia, which develops as a result of impaired vitamin D metabolism.

Arthritis - ascorbic acid is converted into oxalates.

Causes of acute microcrystalline inflammation:

1. Separation of crystals from local areas due to mechanical tissue injury.
2. Reduced dissolution of crystals and reduction in size (for example, at the beginning of hypouricemic therapy)
3. Increased protein levels in the acute phase due to competitive inflammation.

• Calcium-pyrophosphate arthritis (chondrocalcinosis, pseudogout) is one of the diseases that belongs to the group of microcrystalline arthritis and has been increasing in recent years.

Calcium-pyrophosphate arthropathy (pyrophosphate crystal deposition disease, calcium-pyrophosphate arthritis, chondrocalcinosis, pseudogout).

Chondrocalcinosis is a rare inflammatory disease of the joints, in which crystals of pyrophosphate and other calcium salts accumulate and precipitate in the joint. Chondrocalcinosis belongs to the group of crystalline arthropathy of the knee, hip and other joints. This disease is considered the 3rd most common disease after rheumatoid arthritis, gout. The causes of chondrocalcinosis of the meniscus and various joints can be:

• primary (cause - heredity),
• secondary (cause - another disease that disrupts metabolism),
• idiopathic (unknown cause).

The etiology is unclear. Often this disease occurs in conjunction with injuries, hypomagnesemia, hyperparathyroidism, gout, and hemochromatosis. Calcium pyrophosphate deposition in the joints is characteristic due to secondary degenerative and metabolic changes in damaged tissues and in the elderly. In some cases, the term "pseudo" is used in relation to gout due to the similarity of symptoms with other joint diseases [3]:

• "pseudogout" (Atype)
• "pseudorheumatoid arthritis" (Vtype)
• "pseudo-osteoarthritis" (with acute attacksStype, without attacksDtype),
• "asymptomatic" (Ytype)
• "pseudoneuropathic" (Ftype)

Recognized risk factors include: advanced age, osteoarthritis (OA), joint injuries, metabolic diseases, genetic predisposition and family history.

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In developed countries, increased life expectancy and iatrogenic factors leading to hypomagnesemia: abuse of loop diuretics, proton pump inhibitors, calcineurin inhibitors (cyclosporine, tacrolimus) (which disrupt calcium metabolism) are considered as factors leading to comorbidity. The incidence of acute episodic pyrophosphate arthropathy is increasing in hyperparathyroidism and end-stage renal failure (due to metabolic disorders).

Epidemiology: The presence of a hereditary predisposition, autosomal dominant inheritance, and onset by the age of 40 have been reported in the literature. Idiopathic (sporadic) calcium pyrophosphate arthropathy is rare in patients under 55 years of age, especially in cases where joint injury or knee meniscectomy have not been performed. In a large study conducted in the UK, the prevalence of calcium pyrophosphate arthropathy of the knee, taking into account age, gender, and symptoms of knee pain, was 4.5% in patients over 40 years of age. Chondrocalcinosis occurs in 1 in 10 people aged 60 to 75 years, in 1 case over 80 years, and only a small percentage develop clinical manifestations. The main age is over 60 years. Men and women can suffer equally.

Pathogenesis: In the early stages of the disease, calcium pyrophosphate accumulates in the cartilage of the joint. An acute attack of arthritis develops when it moves from the cartilage to the joint cavity, and this condition can be caused by hypocalcemia, enzyme changes due to infection, and biomechanical loads on the joint. Over time, the joint structure changes, the articular surface becomes smooth, and the bone deforms.Idiopathic/sporadic, but early-onset familial forms of the disease are also common. The autosomal dominant form of the disease is associated with mutations in the inorganic pyrophosphate transporter ANKH [5]. Defects in inorganic pyrophosphate (PPi) metabolism, impaired chondrocyte differentiation, and alterations in the extracellular matrix are closely associated with calcium pyrophosphate dihydrate crystal deposition, and play a central role in the pathogenesis of calcium pyrophosphate dihydrate crystal deposition disease. Activation of the NLRP3 inflammasome with subsequent activation of caspase-1, as well as processing and secretion of interleukin (IL)-1β, induce an inflammatory response to calcium pyrophosphate dihydrate crystals.

Clinic: The disease can develop for a long time without any symptoms. With the exacerbation of chondrocalcinosis of the knee or other joints, the following symptoms appear:

• sharp, sharp pain that increases during movements and touch;
• swelling of the diseased joint;
• limitation of mobility;
• redness of the skin in the area ofthe affected joint and an increase in local temperature;
• absence of fever and subcutaneous nodules characteristic of gout.

The disease manifests itself in several forms:

1. Latent chondrocalcinosis is more common in the hip, knee, interphalangeal joints, intervertebral discs, it occurs in the pubic symphysis and is diagnosed only by radiological examination .

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2. Acute arthritis or "pseudogout atypical" (25%): pain, hyperemia, swelling in the joint, reaches a maximum within 12-36 hours. In 50% of cases, the knee joint is affected. Arthritis attacks last for several weeks. In the inter-attack period, the shape and function of the joints do not change.

3. Chronic ("pseudo-rheumatoid arthritis-like arthritis type V" occurs in 5% of cases: morning stiffness, deformity and hereditary predisposition are characteristic. Synovitis of the joints of the hands, chronic symmetrical inflammatory polyarthritis. In about 5% of cases, 2-3 metacarpophalangeal joints of the hands are affected, there is a tendency to symmetry. Elbow, wrist and knee joints are also involved. There is short-term pain, swelling and stiffness of a paroxysmal nature, prone to spontaneous relief. Radiological examination does not show bone erosion, but reveals signs of chondrocalcinosis. There are similarities in symptoms with rheumatoid arthritis, but there are no laboratory signs of RA.

4. "Stip" pseudo-osteoarthritis with acute attacks is a common clinical form (observed in about 50% of patients). It is most often detected in elderly women. The knee joint is mainly affected, sometimes the hip joint. In addition, joints that are not typical for primary arthrosis are involved - the wrist, forearm, metacarpophalangeal, elbow and shoulder. Possible swelling of the joints, morning stiffness, pain when moving, limitation of movement. Radiological examination reveals signs of chondrocalcinosis in the affected joints.

5. Pseudoankylosing spondylitis (type D). This is a variant with predominant damage to the spine with persistent pain and stiffness. Calcium pyrophosphate crystals are deposited in the intervertebral discs, articular cartilage, synovium, ligamentum flavum, intervertebral bursae, and sacroiliac joints. Radiographs reveal nonspecific changes in the form of a decrease in the height of the intervertebral discs, sclerosis of the subchondral parts of the vertebrae, and osteophytes. Pyrophosphate crystals are deposited around the odontoid process (in the transverse atlas ligament and pterygoid ligaments) and in the anterior atlanto-occipital ligament, which is called the "crown" or "horseshoe". With a backward shift, myelopathy may also develop due to the destruction of the odontoid process and the deposition of calcium pyrophosphate in the atlantoaxial joint. "Type D without attacks" -

6. Asymptomatic variant (type E). Asymptomatic chondrocalcinosis is most commonly found in the knee, metacarpophalangeal joints, hip, wrist, annulus fibrosus of intervertebral discs, pubic symphysis, and spine. Incidental finding of chondrocalcinosis on plain radiographs or other imaging does not necessarily indicate that the patient should be treated for arthritis caused by PFC crystal deposits.

7. Pseudo-neurotrophic variant (type F). Severe joint destruction and neuropathy. Sometimes the deposition of pyrophosphates leads to rapid destruction of the joints, which is accompanied by pain, deformation and dysfunction. Unlike arthropathy, there is no sensory impairment in neurological diseases (syringomyelia, etc.).

8. The pseudoosteoarthrosis form is similar to the clinical and radiological signs of osteoarthrosis, however, signs of chondrocalcinosis are found on the radiological . Also, unlike osteoarthritis, patients have flexion contractures and varus deformities.

9. Destructive arthritis of the Charcot type of arthropathy: pronounced destructive changes in the shoulder, hip, knee joints are characteristic.

10. Lumbosciatica is similar to the clinic of intervertebral disc prolapse.

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11. Rheumatic polymyalgia: morning stiffness, pain in the shoulder, neck, spine, buttocks and thigh muscles, mainly women.

Diagnosis: Based on anamnesis, objective examination and general blood, urine and biochemical blood tests, as well as determination of the level of trace elements in the blood, examination of synovial fluid and radiological results.

In the synovial fluid, calcium pyrophosphate crystals are detected in the form of rhomboids or rods.

Radiological examination: mainly in the tendons of the shoulder, hip, knee joints, in the form of focal or linear shadows of calcium salts are detected.

Also, ultrasound of the joints helps to assess the volume of synovial fluid and study changes in the structure of the cartilage;

• MRI - this method best detects tissue damage (joint capsule, cartilage, muscle, tendons and other structures), and also detects inflammation. Bones and tendons can also be calcified

Diagnostic criteria for calcium pyrophosphate arthropathy [5]:

1) Detection of calcium pyrophosphate crystals in the tissues or synovial fluid of the affected joint using "detection methods" (e.g., characteristic "powder diffraction" radiographs)

2A) Detection of mono- or triclinic crystals in the synovial fluid that show weak birefringence (or no birefringence) under a polarizing microscope.

2B) Presence of typical chondrocalcinosis on radiographs (punctate and linear calcifications in fibrous and hyaline cartilage and articular capsules, especially if these changes are symmetrical).

2C) Presence of typical signs of PFC crystal deposition in articular hyaline cartilage or fibrocartilage by ultrasound.

3A) Acute arthritis of the knee, wrist, or other large joints.

3B) Chronic arthritis affecting the knee, hip, wrist, forearm, elbow, shoulder, or metacarpophalangeal joints with acute attacks.

Definite diagnosis: Criteria 1 or 2A must be present

Probable diagnosis: 2A or 2B or 2C

Presumptive diagnosis: 3A or 3B

Comparative diagnosis:

The disease should be excluded first: In patients with seronegative RA-like symptoms that appear after 55 years, in patients with clinical signs in atypical joints (elbow, shoulder, wrist, metacarpophalangeal), in patients with polymyalgia rheumatica

Symptoms	**Gout**	**Calcium pyrophosphate arthritis**
Gender and age	Males, over 40 years old	Males and females over 60 years old
Location	1st plus phalanx joint	Knee joint
Morphology	Urate crystals	Calcium pyrophosphate crystals
Radiological examination	“Sealed foci”	Chondrocalcinosis
Ultrasound examination	Tophus in tissues	Chondrocalcinosis

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Complications: if treatment is not carried out or the diagnosis of the disease is not made in a timely manner, the following complications develop over time: severe pain syndrome, arthritis, joint deformation, decreased mobility of the diseased joint, if the legs are affected - O-shaped deformation.

Treatment: Temporary immobilization of the affected areas with a splint is recommended. Medications include indomethacin or diclofenac 150-200 mg/day for 12-14 days, colchicine 0.6–1.2 mg/day to prevent attacks.

Surgical treatment: Aspiration of synovial fluid. Large crystals are removed arthroscopically.
Prevention of the disease: there are no specific preventive measures. There are only general recommendations that are best followed:
• proper, nutritious and varied nutrition;
• sufficient physical activity;
• maintaining a balance of work and rest;
• timely prevention and treatment of acute and chronic diseases;
• regular monitoring by doctors.

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