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Pseudogout is a Form of Arthritis Caused by Crystal Deposits

Siniša Franjić

Independent Researcher

*Corresponding author: Siniša Franjić, Independent Researcher.

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Abstract

Pseudogout is a condition characterized by joint inflammation caused by the accumulation of crystals in the synovial fluid found within the joints. This leads to swelling and discomfort in the affected areas. Generally, this condition presents in individuals who are 60 years or older. While it primarily impacts the knees, other joints may also be involved. The key difference between pseudogout and standard gout is the presence of calcium pyrophosphate crystals in pseudogout, whereas gout is due to urate crystals. During a pseudogout episode, the joints that are impacted typically swell, feel warm, and are quite painful. Episodes of joint inflammation manifest as acute swelling, warmth, stiffness, and discomfort, lasting from several days to a few weeks, often resolving on their own afterward.

Keywords: Gout, Pseudogout, Arthritis, CPPD, Health.

Introduction

Pseudogout is an acute inflammatory disorder that predominantly impacts the larger joints where calcium pyrophosphate (CPP) dihydrate crystals accumulate in the connective tissues [1]. The frequency of sudden attacks is roughly the same between genders. Women who have been diagnosed with osteoarthritis (OA) and CPP experience a higher occurrence. Around 50% of cases are found in those over the age of 84, while 36% occur in those aged 75 to 84, and 15% in individuals from 65 to 74 years of age. Regarding affected joints, the knee is involved approximately 50% of the time, with other joints like wrists, shoulders, ankles, feet, and elbows also being impacted.

Gout

Gout is traditionally characterized as an acute form of monoarthritis, typically affecting the first metatarsophalangeal (MTP) joint and more broadly, the foot, as well as the knee and sometimes the hands, wrists, and elbows [2]. The acute form of gout is clinically indistinguishable from pseudogout, which involves calcium pyrophosphate crystals instead of uric acid as the crystallized substance responsible. Gout can manifest in an intercritical form wherein uric acid may lead to other systemic complications, such as kidney stones. Chronic gout represents a destructive joint condition that can lead to considerable disability.

The inflammatory response seen in gout arises from the presence of precipitated uric acid crystals within the joint. The reaction involves the influx of neutrophils that release non-specific inflammatory substances, which contribute to the severe inflammation, rather than the “needle-like” nature of the crystals themselves being the sole cause. While elevated uric acid levels increase the chances of a gout attack, these high levels are not mandatory for establishing a gout diagnosis. Men are more frequently affected than women. Factors that can predispose individuals to gout include obesity, kidney insufficiency, use of thiazide diuretics, cyclosporine treatment, high purine diets, excessive alcohol consumption, and family history of the condition.

Gout is categorized into four phases: (1) asymptomatic crystal accumulation in tissues, (2) episodes of acute gout, (3) intercritical phases that occur post-acute attack but prior to the following flare, and (4) chronic gout characterized by chronic arthritis symptoms and/or tophi [4]. The initial episode of gout can frequently be misidentified as cellulitis, as it manifests with swelling and discomfort, typically in a single joint, along with redness and warmth. Traditionally, a gout attack affects the first toe's metatarsophalangeal joint, referred to as podagra, though it can affect any joint in the body. In certain instances, untreated gout can resolve on its own within a timeframe of 3 to 10 days, leaving no lingering signs or symptoms. During an acute

episode, the serum uric acid levels may appear normal or even diminished, likely due to the existing presence of urate crystals. Nevertheless, uric acid readings are beneficial for tracking treatment efficacy during interattack periods. Imaging studies like radiographs may reveal cystic alterations in the joint surface, displaying punched-out lesions and soft tissue calcifications. These imaging results are nonspecific and can also occur in conditions such as osteoarthritis and rheumatoid arthritis. When assessing patients who may have gout, it is crucial to inquire about any recent injuries or trauma. Following an injury, an increase in urate concentration can be detected in the synovial fluid. While imaging techniques are typically unnecessary for gout diagnosis, a history of injury may justify imaging to eliminate the possibility of a fracture.

Infections usually affect only one joint when they are of bacterial origin in more than 90% of cases. Infectious arthritis predominantly occurs in larger joints, such as the knee, hip, and shoulder. Chronic monoarticular arthritis or involvement of a few joints may result from fungi or mycobacterial infections. For acute polyarticular arthritis affecting multiple joints, origins may include endocarditis or a disseminated gonococcal infection. Microorganisms can invade joints in three primary ways: (1) through direct penetration from surgery, bites, or trauma, (2) via hematogenous spread from an unrelated site of infection, or (3) through extension from a nearby infected joint. Alongside an arthrocentesis and analysis of synovial fluid, additional tests such as blood cultures, Gram staining and culture, complete blood count (CBC), and erythrocyte sedimentation rate (ESR) should be conducted.

Individuals with certain risk factors are more susceptible to infectious arthritis, including those with alcohol dependence, cancer, diabetes, undergoing hemodialysis, those with immune deficiencies like HIV, patients on immunosuppressive medications such as corticosteroids, individuals with persistent medical issues like endocrine, pulmonary, or liver diseases, hemophiliacs, and users of intravenous drugs. Bacterial joint infections are most frequently found in individuals with rheumatoid arthritis due to chronic joint inflammation compounded by steroid use, which increases vulnerability to Staphylococcus aureus infections. Patients with HIV might encounter joint infections involving pneumococcus, Salmonella, or even Haemophilus influenzae. Intravenous drug users are particularly at risk for infections from streptococcus, staphylococcus, gram-negative organisms, or Pseudomonas.

Predisposing Factors

A. Older Adults [1]
B. Injury or surgery to joints
C. Hospital stay or illness
D. Genetic chondrocalcinosis
E. Endocrine or metabolic conditions:
1. Gout
2. Hyperparathyroidism
3. Hemochromatosis
4. Hypophosphatasia
5. Hypothyroidism
6. Low magnesium levels
7. Gitelman syndrome
8. Hemosiderosis

Risk Factors

Apart from advancing age, various other elements contribute to the likelihood of developing pseudogout [4].

- Joint damage resulting from an accident or surgical procedure
- **Hereditary factors:** certain individuals may have a natural tendency to experience pseudogout and may begin showing signs at a younger age
- **Elevated iron levels:** individuals with a genetic condition that leads to the accumulation of excess iron in the body (hemochromatosis) are at a higher risk of developing pseudogout
- **Additional conditions:** various conditions impacting metabolism or hormonal glands, like hyperparathyroidism, are linked to the onset of pseudogout.

Causes

The cause behind the formation of the crystals remains unclear [5]. It is possible that there is a hereditary factor involved. This suggests that your body has inherited a particular method for handling calcium crystals. There are specific risk elements associated with pseudogout. For instance, it is more prevalent among men who are over 60 years old. Additional risks encompass thyroid-related issues, renal failure, or metabolic disorders concerning calcium or iron.

Acute pseudogout episodes are connected to the emergence of crystals within the joint fluid. The body's immune system responds to these crystals. A certain category of white blood cells known as polymorphonuclear neutrophils (PMNs) is involved. While white blood cells primarily work to combat infections, they also react to these crystals. Another group of white blood cells, macrophages, consumes both bacteria and crystals. Often, these white blood cells secrete harmful substances aimed at destroying bacteria. However, these substances can inadvertently damage nearby tissues and cartilage, leading to inflammation characterized by intense pain, swelling, and redness.

CPPD

Calcium pyrophosphate crystal deposits (CPPD) in fibrocartilage and hyaline cartilage (chondrocalcinosis) can lead to sudden crystal-induced arthritis known as "pseudogout," chronic degenerative joint disease, and continuous inflammatory polyarthritis termed "pseudorheumatoid arthritis." CPPD may also remain symptomless and be discovered incidentally during radiographic evaluations showing chondrocalcinosis [6]. The occurrence of CPPD rises with increasing age. Conditions such as hyperparathyroidism, familial hypocalciuric hypercalcemia, hemochromatosis, and low magnesium levels increase the likelihood of CPPD, though most instances occur without any related condition.

Pseudogout is typically observed in individuals aged 60 and above. It is marked by sudden, recurring arthritis episodes, which are rarely chronic, primarily affecting major joints like the knees and wrists, and it almost always occurs with radiographic evidence of chondrocalcinosis in the involved joints. The crowned dens syndrome, which arises from pseudogout affecting the atlantoaxial joint along with "crown-like" calcifications surrounding the dens, presents with extreme neck pain, stiffness, and high fever, potentially resembling meningitis or giant cell

arteritis. Similar to gout, pseudogout commonly arises within 24 to 48 hours following significant surgery. The detection of weakly positively birefringent calcium pyrophosphate crystals in joint aspirates confirms the diagnosis. Non-steroidal anti-inflammatory drugs (NSAIDs) can alleviate acute episodes. Colchicine, dosed at up to 1. 8 mg orally during the initial 24 hours, followed by 0. 6 mg once or twice daily until symptoms subside, has shown efficacy. Joint aspiration and an intra-articular injection of triamcinolone, ranging from 10 to 40 mg based on joint size, can also be beneficial, as can initiating oral prednisone at 30 to 50 mg and tapering it over a week to ten days.

For patients who cannot use other therapies, IL-1 inhibitors such as anakinra and canakinumab may be administered. For individuals experiencing three or more attacks annually, preventive treatment with colchicine at a dosage of 0. 6 mg taken orally twice each day can help lessen episodes. The degenerative joint disease related to CPPD can affect areas not typically involved in osteoarthritis, such as the glenohumeral joint, wrist, and the patellofemoral region of the knee. The "pseudorheumatoid arthritis" linked with CPPD impacts the metacarpophalangeal joints and wrists.

The condition known as "pseudogout syndrome" was initially characterized by Kohn in 1962 and pertains to sudden episodes of synovitis triggered by CPPD crystals [7]. Arthritis linked to CPPD is recognized as the third most prevalent form of inflammatory arthritis, and its incidence tends to escalate with advancing age. Arthropathies involving CPPD crystals can exhibit a range of clinical manifestations, which complicates diagnosing the condition, as diverse presentations lead to uncertainty in diagnosis. Numerous terms exist to refer to CPPD and its variants, contributing to the confusion experienced by medical professionals, researchers, and patients alike. As stated by the European Alliance of Associations for Rheumatology (EULAR), there are at least four distinct clinical forms: 1) asymptomatic CPPD; 2) osteoarthritis (OA) accompanied by CPPD; 3) acute arthritis due to CPP crystals; and 4) chronic inflammatory arthritis associated with CPP.

Additional conditions cited in the literature include pseudo-polymyalgia rheumatica (pseudo-PMR), pseudo-neuropathic arthropathy, and tumoral CPPD. Efforts are ongoing to establish new classification criteria through an international process driven by data and expert input to develop CPPD classification standards. Management of acute CPPD focuses on strategies employed in gouty arthropathy, incorporating various medications such as colchicine, non-steroidal anti-inflammatory drugs (NSAIDs), corticosteroids, and interleukin-1 inhibitors. Addressing chronic CPPD proves challenging from an evidence-based perspective due to a lack of randomized controlled trials, though there exists some low-level evidence supporting the use of hydroxychloroquine, methotrexate, colchicine, and steroids.

Acute Pseudogout

Individuals experiencing acute pseudogout typically display acute oligoarthritis accompanied by class II joint fluid and the presence of calcium pyrophosphate crystals within the synovial fluid [8]. Pseudogout resembles gout in patients who are middle-aged or older. Unlike gout, the knee joint is the most frequently affected area in this condition. Usually, serum uric acid levels are within the normal range. Chondrocalcinosis may be observed, though it is not necessarily present in the joint experiencing acute involvement. Regardless of where it is found, the existence of chondrocalcinosis does not serve as a definitive diagnostic criterion. A conclusive diagnosis hinges on detecting calcium dihydrate (pyrophosphate) crystals in the synovial fluid.

Crystal Analysis

The aspiration of synovial fluid followed by crystal analysis under compensated polarized light microscopy remains the established standard for aiding in diagnosis [7]. While bright field microscopy enables the recognition of the distinct morphology of CPP crystals as shapes resembling rhombuses, thin bars, and parallelepipeds, compensated polarized light microscopy further elucidates birefringence. CPP crystals may often need careful examination of the synovial fluid for accurate identification, as they might show a lack of birefringence, can be engulfed by cells, and are often found lodged within vacuoles. Allocating additional time for thorough analysis of the sample can be essential for reaching a diagnosis, as crystal distribution might be sparse. Proper identification of CPP crystals in the synovial fluid of a suspected case is crucial for making an accurate diagnosis. Nevertheless, it is vital that the analysis of CPP is conducted by a sufficiently skilled clinician or laboratory technician to prevent the risk of reporting false-negative findings. Research conducted thus far has revealed inconsistencies in the accurate identification of CPP crystals by clinicians and laboratory personnel. The diligence and training of the observers play a critical role, and insufficient experience could impede proper diagnosis.

CT

A computerized tomography (CT) scan was conducted due to concerns regarding a possible growth in the left temporomandibular joint (TMJ) [9]. The imaging evaluation revealed a large radiopaque mass (with a maximum diameter of 39 mm) that encased the head of the left condyle, which showed signs of degeneration alongside the glenoid fossa, where minor defects were also visible in the fossa's roof. Considering the patient's initial clinical profile and the radiological results, differential diagnoses included tumor or similar diseases, such as pigmented villonodular synovitis (PVNS), synovial chondromatosis, and the risk of malignant chondrosarcoma in the right temporomandibular joint (TMJ). The patient underwent a surgical excision of the tumor under general anesthesia via a preauricular approach, followed by dissection of surrounding tissues. During the operation, it was discovered that the mass was an encapsulated soft tissue growth that originated from and encompassed the periphery of the articular disk. Once the capsule was cut, a nodular, soft, gritty granulomatous tissue was revealed. This tissue was removed in two distinct sections.

Arthroplasty was carried out using a burr to reshape the mandibular condyle appropriately. The incision was then closed with layered sutures. Upon microscopic evaluation, nodules containing birefringent crystalline substances and calcifications were noted, which correlated with reactive chondroblasts and histiocytes. The condition of the crystals during processing confirmed the presence of calcium pyrophosphate crystal deposition, commonly referred to as pseudogout, in the TMJ. The patient experienced an uncomplicated recovery without any facial weakness and reported an improvement in maximum inter-incisal opening

from 19 mm to 36 mm during the annual follow-up appointment after the surgery. A leftward deviation of the mandible was observed only at maximum opening, and no alterations in occlusion were noted. Pain and swelling had entirely subsided.

Diagnosis

Diagnosing pseudogout can prove to be challenging [5]. A physician will assess multiple joints, including those causing pain and those that do not, to identify patterns. The doctor may request x-rays of the affected joint, which might reveal crystal deposits within the soft tissue. The most conclusive method for making the diagnosis involves extracting fluid from a joint and having it analyzed in a laboratory. This step is crucial to differentiate it from an infection. Crystals associated with gout and pseudogout can be identified under a microscope. Each crystal type presents distinct characteristics when examined under polarized light. Each variety of crystal has its own unique configuration and appearance. Gout crystals resemble needles, while pseudogout crystals take on a rhomboid shape. The absence of bacteria under microscopic examination indicates a lesser likelihood of infection. In the presence of no bacteria, identifiable calcium crystals, and PMNs, this combination suggests the occurrence of pseudogout. It is possible for both a crystal-associated issue and an infection to be present simultaneously.

The x-rays can be beneficial as they reveal various alterations. Gout deteriorates the bones and joints in the hand and wrist. This condition is marked by numerous tiny cystic erosions located on the bones at the joint surfaces. An x-ray indicating pseudogout might display calcification in the soft tissues surrounding the joints. Urate levels in the blood are frequently high in cases of gout, whereas uric acid levels remain typical in pseudogout. The count of white blood cells (WBC) might be increased in these scenarios. Lastly, assessing kidney function is crucial. Many medications for pseudogout can be influenced by whether kidney function is adequate or impaired. Thus, understanding kidney health is essential when suggesting treatments.

Management

The approach to treating acute pseudogout involves extracting the crystals via joint aspiration, administering NSAIDs or colchicine during the episode of inflammation, injecting glucocorticoids into the joint when feasible, and restricting joint movement for a brief period [10]. There is no strong evidence to support the extraction of crystals or the prevention of crystal accumulation, which are performed solely for diagnosis and to alleviate pain. When only a joint or two are affected, intraarticular injections can provide the greatest relief; if multiple joints are involved, opting for an NSAID or colchicine is a superior choice. Due to the intense pain experienced during acute flare-ups, limited weight-bearing may be necessary temporarily as symptoms begin to resolve.

For individuals experiencing recurrent pseudogout episodes, considering prophylactic colchicine treatment at a dosage of 0. 6 mg twice daily is advisable. In a group of ten individuals with recurrent flare-ups, colchicine led to a significant reduction in the frequency of episodes within one year, dropping from 32 episodes to 10.

Conclusion

Pseudogout represents a type of arthritis triggered by crystal accumulation within and around the joints, characterized by abrupt and painful swelling in one or more joints. These distressing episodes may persist from days to weeks. The knee joint is the most commonly affected area in this condition. Although labeled as calcium pyrophosphate dihydrate crystal deposition disease, the name "Pseudogout" is widely used owing to the condition's resemblance to gout. Both disorders arise from the buildup of crystals in the joints, although the crystals involved differ between the two. The precise cause of crystal development in the joints leading to pseudogout remains uncertain, but the likelihood of developing this disease increases with age.

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