Case Report

A 60-year-old male was sent to the emergency room for acute-onset right ankle pain with mild fever in the early morning.

The doctor, on duty at that time, took the patient’s history. The patient, a business manager in a trading company, had many social engagements. He was a social drinker, with occasional excessive drinking. He had been taking diuretics for hypertension for 2 years. He had drunk a large amount of alcohol at a party on the previous night. He was awoken by right ankle pain at 02:00. Initially, he thought it was an ankle sprain from playing golf. However, the pain became increasingly severe, and was associated with redness, swelling, tenderness and local heat. He wanted painkillers and struggled to get out of bed. He fell to the ground and woke his wife. Subsequently, he was sent to our emergency room.

The physical examination noted an obese gentleman with an acute ill-looking appearance, body temperature of 38°C, pulse rate of 110/min, respiratory rate of 25/min, blood pressure of 160/86 mmHg, normal chest and abdomen, with no open wound over the integument. Acute synovitis was noted over the right ankle joint, with severe pain at mild motion. To clarify the etiology of the arthritis, the doctor aspirated 5 mL of synovial fluid from the right ankle joint. The fluid was yellowish and mildly turbid with some whitish crystals. Under the impression of acute crystal-induced arthritis, colchicine and diclofenac were prescribed. In addition, local ice packing was suggested.

The blood test results were as follows: white blood cell count (WBC), 12,500/mm³; hemoglobin, 13.5 g/dL; platelets, 310,000/mm³; erythrocyte sedimentation rate (ESR), 15 mm/hr; creatinine, 1.2 mg/dL; uric acid, 9.6 mg/dL; C-reactive protein (CRP), 2.1 mg/dL; fasting blood sugar, 125 mg/dL. The synovial fluid analysis revealed: WBC, 15,000/mm³; polymorphonuclear neutrophils (PMN), 95%; glucose, 90 mg/dL; and many intracellular and extracellular needle-shaped crystals by light microscopy with strong negative birefringence under
polarizing microscopy. Monosodium urate (MSU) crystals were considered. A Gram stain was performed at the same time, but no suspicious pathogens were noted.

A definite diagnosis of acute gouty arthritis (GA) was made according to the positive MSU crystals in the synovial fluid. Three-day treatments of colchicine and diclofenac were prescribed to the patient. He was followed up in a rheumatologic clinic after this 3-day treatment.

Introduction

Acute arthritis is a common condition encountered in daily clinical practice, in both emergency rooms and rheumatologic clinics. Crystal-induced arthritis is the most common cause of acute arthritis in the elderly. However, other arthritis types or other conditions mimicking arthritis should be ruled out before making a definitive diagnosis. According to the affected joint count, acute arthritis can be classified into acute monoarthritis (such as crystal-induced arthritis, septic arthritis, and hemarthrosis) and acute polyarthritis (such as acute exacerbation of rheumatoid arthritis, atypical gout, and nodal osteoarthritis) (Table 1). Because the severity of each disease is variable, we should pay greater attention to how we handle elderly patients. If a patient presents with extreme pain, fever, suspicious septic arthritis, severe tophaceous gout, gastrointestinal tract bleeding, and unstable systemic condition (renal dysfunction, heart disease, liver disease, and poor blood sugar control), admission for further evaluation and management is mandatory. If a patient presents without these conditions, they can leave the emergency room after initial management and receive further treatment in an outpatient clinic.

Table 1. **Differential diagnosis of acute arthritis**

<table>
<thead>
<tr>
<th>Acute monoarthritis</th>
<th>Acute polyarthritis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crystal-induced arthritis</td>
<td>Atypical gout</td>
</tr>
<tr>
<td>Gout</td>
<td>Nonsuppurative septic arthritis</td>
</tr>
<tr>
<td>Pseudogout</td>
<td>Rheumatoid arthritis</td>
</tr>
<tr>
<td>Septic arthritis</td>
<td>Psoriatic arthritis</td>
</tr>
<tr>
<td>Hemarthrosis</td>
<td>Nodal osteoarthritis</td>
</tr>
<tr>
<td>Osteoarthritis</td>
<td>Palindromic rheumatism</td>
</tr>
<tr>
<td>Hallux valgus with bunion</td>
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</tbody>
</table>

The steps for evaluating a patient with acute arthritis are as follows:

1. **History taking.** This is the first step of the diagnosis. The history taking should include the onset and duration of arthritis, characteristics of the pain, aggravating or relieving factors, previous episodes, diet, alcohol consumption, and exercise. In addition, it should include any history of trauma, traveling, organism or sexual exposure, past disease and drug histories, and family histories of arthritis, immunologic diseases or urolithiasis.

2. **Physical examination.** Alongside a general physical examination, local findings, such as acute inflammatory reactions, joint deformity, tophi or nodules, and open wounds, should be examined.

3. **Laboratory tests.** Blood tests, including a complete blood count, biochemical analyses (uric acid, renal and liver function, cholesterol and triglyceride, and blood sugar levels), inflammatory markers (CRP and ESR) and rheumatoid factor, are helpful. Urinary tests can detect hematuria, proteinuria, 24-hour uric acid excretion, and 24-hour creatinine clearance. To rule out cellulitis and septic arthritis from direct inoculation or hematogenous dissemination, a blood culture should be performed in febrile patients, and a pus culture for a suppurative wound.

4. **Synovial fluid analysis.** A definite diagnosis of acute arthritis cannot be made by history taking and physical examination alone. An arthrocentesis can be performed on a swollen joint, as long as there are no contraindications, such as overlying infection or severe dermatitis, to relieve the swelling, avoid joint damage, and diagnose the etiology of the arthritis. If septic synovial fluid is not likely, a local steroid injection can be given to rapidly relieve the inflammation. Synovial fluid can be divided into four groups: non-inflammatory, inflammatory, septic, and hemorrhagic (Table 2). The appearance, viscosity, protein, glucose, white and red cell counts, and PMN percentages under light microscopy differ among these four groups. Crystal-induced arthritis is highly suspected if intracellular needle- or rhomboid-shaped crystals are noted. A definite diagnosis can be made according to the refringence of the crystals under polarizing microscopy. Gram staining and bacterial cultures with a sensitivity test should be carried out if septic arthritis is suspected.
5. Imaging studies.

- Plain film. The presentation of acute arthritis on plain film is usually soft tissue swelling (Figure 1). Other findings are not necessarily associated with the pathology of this episode. For example, most elderly people show osteoarthritic changes on knee X-rays. However, these are not usually the cause of acute arthritis. Acute arthritis usually takes longer or needs several episodes to develop characteristic joint damage, such as bone erosion and tophi.

- Soft tissue sonography. Acute arthritis usually presents as a hypoechoic lesion, with synovial hyperplasia and hypervascularity. Aspiration of synovial fluid under echo guidance will facilitate a correct diagnosis.

### Gout

GA is the most common crystal-induced arthritis. The pathogenesis is abnormal purine metabolism and/or decreased urate excretion, such that MSU crystals are deposited in joints or other tissues to induce an inflammatory reaction. GA can be divided into primary and secondary cases. Ninety percent of primary GA is due to decreased urate excretion. Secondary GA is mostly from renal diseases, diseases causing high cell turnover such as myeloproliferative or lymphoproliferative disorders, psoriasis, chemotherapy for malignancy, and the side effects of drugs.

### Risk factors

Hyperuricemia is the most important biochemical basis of gout. Higher levels of serum uric acid lead to higher incidences of GA\(^1\). Generally speaking, the serum uric acid level increases with age. The level rises by 1–2 mg/dL after adolescence in males. In females, however, it stays at a low level before menopause and a very low incidence of gout in premenopausal females is noted. In Taiwan, the prevalences of hyperuricemia are around 25.8% and 15.0% in males and females, respectively\(^2\). Depressed renal excretion of uric acid owing to some renal diseases, loop or thiazide diuretics, and low-dose aspirin use

### Table 2. Synovial fluid analysis

<table>
<thead>
<tr>
<th>Disease category</th>
<th>Appearance</th>
<th>Viscosity</th>
<th>WBC ($\times 10^3$/mm$^3$)</th>
<th>PMN (%)</th>
<th>Protein (g/dL)</th>
<th>Glucose (% plasma)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-inflammatory</td>
<td>&gt; 4 mL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Osteoarthritis</td>
<td>Straw, clear</td>
<td>High</td>
<td>1–3</td>
<td>&lt;30</td>
<td>2–4</td>
<td>80–100</td>
</tr>
<tr>
<td>Traumatic arthritis</td>
<td>Straw, clear</td>
<td>High</td>
<td>1–3</td>
<td>&lt;30</td>
<td>2–4</td>
<td>80–100</td>
</tr>
<tr>
<td>Inflammatory</td>
<td>&gt; 4 mL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rheumatoid arthritis</td>
<td>Straw, cloudy</td>
<td>Low</td>
<td>10–30</td>
<td>&gt;90</td>
<td>5–8</td>
<td>80</td>
</tr>
<tr>
<td>Reiter’s syndrome</td>
<td>Straw, cloudy</td>
<td>Low</td>
<td>10–30</td>
<td>&gt;90</td>
<td>5–8</td>
<td>80</td>
</tr>
<tr>
<td>Gout</td>
<td>Straw, clear</td>
<td>Low</td>
<td>10–30</td>
<td>&gt;90</td>
<td>5–8</td>
<td>80</td>
</tr>
<tr>
<td>Septic</td>
<td>&gt; 4 mL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gonococcal arthritis</td>
<td>Straw, slightly cloudy</td>
<td>Very low</td>
<td>5–25</td>
<td>&gt;90</td>
<td>5–8</td>
<td>10–50</td>
</tr>
<tr>
<td>Other bacterial arthritis</td>
<td>Creamy, thick</td>
<td>Very low</td>
<td>&gt;50</td>
<td>&gt;90</td>
<td>7–8</td>
<td>10–50</td>
</tr>
<tr>
<td>Mycobacterium</td>
<td>Creamy, thick</td>
<td>Very low</td>
<td>10–20</td>
<td>&gt;60</td>
<td>7–8</td>
<td>10–50</td>
</tr>
<tr>
<td>Hemorrhagic</td>
<td>&gt; 4 mL, red</td>
<td>High</td>
<td>As blood</td>
<td>As blood</td>
<td>As blood</td>
<td>100</td>
</tr>
</tbody>
</table>

WBC = white blood cell count; PMN = polymorphonuclear neutrophils.
are common in patients with hypertension and cardiac diseases, especially in the elderly. Other risk factors for hyperuricemia include race, genes, obesity, fast body weight change, drugs (antituberculosis agents such as ethambutol and pyrazinamide, niacin, cyclosporine, and didanosine), drinking alcohol, high purine intake, hypercatabolic state, extreme exercise, trauma, seasonal or climate change, and chemical exposure.

Clinical manifestations

The clinical manifestations of GA can be divided into four stages: asymptomatic hyperuricemia; acute GA; intercritical gout; and chronic tophaceous gout. Initially, it presents as recurrent acute monoarthritis, mostly in the lower extremity joints. Podagra (Figure 2), comprising GA at the first metatarsophalangeal (MTP) joint, is the first manifestation in 60% of gout patients. Furthermore, 90% of GA patients have podagra during the disease course. Other joints frequently involved are the dorsal foot, ankle, knee, finger, wrist, and elbow joints in that order. Acute GA usually awakens the patient in the middle of the night after overeating and drinking or after extreme exercise. It presents as marked local redness, swelling, heat and tenderness, and even a bed sheet covering or a wind cannot be tolerated. Low-grade fever and weakness are usually associated. The symptoms peak at around 24–48 hours without suitable treatment. However, atypical gout usually presents as chronic polyarthritis at the upper or lower extremity joints in the elderly, often associated with tophi. In this subtype, acute attacks with severe inflammatory signs are very uncommon.

While taking the patient’s history, metabolic syndrome or syndrome X, including hyperuricemia, hypertension, type 2 diabetes mellitus, hypertriglycerideremia, atherosclerosis and obesity, warn of the existence of gout. In addition, a family history of gout, urolithiasis and metabolic syndrome imply the suspicion of gout. By physical examination, we can note local inflammatory reactions and severe pain even with mild contact or motion. Tophi are often located around inflamed joints, earlobes and olecranon bursa in patients with a long history of GA.

Laboratory tests

Blood tests reveal leukocytosis, increased serum uric acid and inflammatory markers (CRP and ESR). Biochemical tests often reveal elevated triglyceride, creatinine and fasting blood sugar. Proteinuria, hematuria and even casts in urinalysis stress a work-up of renal dysfunction or urolithiasis.

Synovial fluid analysis

Arthrocentesis can be done directly or under sono-graphic guidance. The characteristics of synovial fluid in acute GA are as follows: low viscosity; semi-transparency to straw-colored; glucose in synovial fluid at about 80% of blood glucose; white cell count elevation to 10,000–50,000/mm³ with PMN >90%. It should be differentiated from septic arthritis. MSU crystals in GA are needle-shaped, free (extracellular) or engulfed (intracellular) by PMN under light microscopy, and exhibit strong negative birefringence under polarizing microscopy (Figure 3). If septic arthritis is suspected, Gram staining and bacterial cultures should be carried out before antibiotic treatment.

Imaging studies

1. Plain film. Soft tissue swelling is usually the single finding in acute GA. However, bone erosion, joint

![Figure 2](https://example.com/fig2.png)

Figure 2. Podagra: acute gouty arthritis over the first metatarsophalangeal joint.

![Figure 3](https://example.com/fig3.png)

Figure 3. Monosodium urate crystals: needle-shaped, strongly negatively birefringent under polarized microscopy.
space narrowing, tophi in the subchondral space and bone marrow, osteoporosis, the characteristic “punch-out lesion” and “overhanging sign” (Figure 4), and even fractures are evident after episodes of recurrence.

2. Soft tissue sonography. In acute stages, a hypoechoic lesion can be seen. The sensitivity for bone erosion is higher with sonography than with plain film. In addition, aspiration of an area that is difficult to approach can be carried out under the guidance of sonography.

3. Renal sonography. Long-term history of GA, chronic tophaceous gout and symptoms of urolithiasis suggest the necessity of renal echography to search for a probable renal stone or other renal diseases.

Classification criteria

Clinically, the definite diagnosis of GA is based on the characteristic MSU crystals in aspirated synovial fluid from inflamed joints or in tophi. Other features, such as acute monoarthritis, hyperuricemia and response to colchicine treatment, can provide clues toward diagnosis. The classification criteria of acute GA suggested by the American Rheumatism Association are (adapted from Wallace et al.6): (1) presence of characteristic urate crystals in the joint fluid; (2) a tophus proven to contain MSU crystals; or (3) presence of six or more of the following 12 clinical/laboratory/radiologic phenomena:

1. More than one attack
2. Maximal inflammation developed within 1 day
3. Attack of monoarticular arthritis
4. Joint redness
5. First MTP joint painful or swollen
6. Unilateral attack involving the first MTP joint
7. Unilateral attack involving tarsal joint
8. Suspected tophus
9. Hyperuricemia
10. Asymmetric swelling within a joint (X-ray)
11. Subcortical cysts without erosion (X-ray)
12. Negative culture from a joint fluid during attack

Treatment

Acute GA

Colchicine, an inhibitor of WBC chemotaxis, should be given as soon as possible. The effectiveness rate is up to 90%. However, it drops to 75% if prescribed after 12–24 hours. The acceptable dose is 1–2 mg/day7. In addition, a full dose of a nonsteroidal anti-inflammatory drug (NSAID) can be used if the patient can tolerate it. Local injection of a corticosteroid into severely inflamed joints is very effective. Systemic corticosteroid or adrenocorticotropic hormone injection can be carried out for severe oligoarticular or polyarticular attacks, or attack at sites not amenable to aspiration8. It should be stressed that this is not the time to commence urate-lowering therapy. If a patient has taken a hypouricemic agent, they should maintain the treatment. We have learned that a fluctuation in the uric acid levels will induce further attacks of GA.

Intercritical gout and chronic gout

Urate-lowering therapy: After elimination of risk factors and diet control for gout, patients should initiate hypouricemic agent treatment if they have serum uric acid levels above 9 mg/dL, three or more acute attacks per year, tophaceous gout or renal dysfunction. The target serum uric acid level is less than 6 mg/dL. The hypouricemic agents can be divided into three categories:

1. Uricosuric agents. Benzbrromarone increases renal urate excretion by inhibiting proximal renal tubular reabsorption of urate. This category of drug is indicated for a renal urate excretion rate of <600 mg/day, provided that the patient has neither urolithiasis nor renal dysfunction (creatinine clearance <35 mL/min).

2. Xanthine oxidase inhibitors. Allopurinol can facilitate the dissolution of tophi and inhibit the formation of tophi and nephrolithiasis. It is indicated for a daily urate excretion rate >1,100 mg, 24-hour creatinine clearance <35 mL/min, tumor lysis syndrome,
urolithiasis or tophaceous gout. Its severe side effect, toxic epidermal necrolysis, should be monitored. Furthermore, interactions with other drugs should be kept in mind for elevated or depressed drug concentrations when other drugs are taken concurrently. Recently, febuxostat, a non-purine xanthine oxidase inhibitor, was developed. This drug seems more potent and safer in renal-impaired patients than allopurinol. However, it has not yet been approved for clinical use.

3. Recombinant urate oxidases. This is a new category of drug. Intravenous rasburicase transforms uric acid to the more water-soluble allantoin, which is readily excreted via the kidney. The drug has been used successfully to prevent dialysis in tumor lysis syndrome. However, the antigenicity of rasburicase may limit its application to long-term therapy, and the optimized regimen for refractory tophaceous gout has not been established. A subcutaneous pegylated uricase with a longer half-life and lower antigenicity was developed. However, these drugs are not available on the Taiwan market.

For prevention of an acute attack, oral colchicine can be given at 0.5–1.0 mg/day for patients with frequent GA attacks and in the first months of urate-lowering therapy, or at 0.5–1.0 mg statim for patients with gouty prodromes.

*Asymptomatic hyperuricemia*

It is not necessary to treat asymptomatic hyperuricemia. However, it is necessary to monitor the serum uric acid and creatinine levels to identify the underlying causes of hyperuricemia and gout, and to treat the underlying metabolic syndrome (syndrome X, such as hypertension, type 2 diabetes mellitus, hyperlipidemia, atherosclerosis and obesity). Urate-lowering therapy is considered for more than three episodes of acute GA per year, positive family history of gout or urolithiasis, 24-hour renal excretion of urate > 1,100 mg, or serum uric acid > 9 mg/dL for 6 months even under diet control.

*Diet and lifestyle remodeling*

All GA and hyperuricemic patients should follow the rules of avoiding high-purine foods (animal visceral organs, sardines, oysters, clams, crabs, etc.) and large amounts of alcohol. Drinking more water to maintain at least 2,000 mL of urine output, keeping an ideal body weight, suitable exercise, and avoidance of extreme exercise and injury, stress and cold environments are all important.

**Pseudogout**

Pseudogout, another common crystal-induced arthritis, is an inflammatory disease caused by accumulation of calcium pyrophosphate dihydrate. Like GA, the clinical manifestation is mainly acute monoarthritis, with less polyarthritis, usually associated with systemic symptoms such as fever and malaise. Differential diagnosis for the two diseases is necessary. The most commonly affected joints are the knee, wrist and shoulder in pseudogout, whereas the first MTP joint, dorsal foot and ankle are frequently involved in GA. In addition, the inflammation in pseudogout is usually milder than that in GA. The risk factors for pseudogout include elderly age, trauma, genes, and metabolic abnormalities (hyperparathyroidism, hemochromatosis, Wilson’s disease, etc.).

Leukocytosis and elevated CRP and ESR are common in pseudogout. Synovial fluid analysis reveals similar findings to gout, except for differently shaped crystals. The calcium pyrophosphate dihydrate crystals in pseudogout are short and rhomboid, with weak positive birefringence under polarizing microscopy, and are, therefore, different from the needle-shaped crystals with strong negative birefringence in GA. A plain film usually shows osteoarthritis, with chondrocalcinosis (Figure 5), bone cysts and bone spurs. Avascular necrosis or fractures may develop after prolonged inflammation.

The treatment for pseudogout relies on symptom control. Colchicine, NSAIDs or local steroid injection is effective in acute episodes. The response is usually slower than that in gout.

*Figure 5. Chondrocalcinosis, a frequent finding in X-ray, is most commonly noted at the knee (arrows) and wrist.*
Septic Arthritis

Septic arthritis can be divided into gonococcal and non-gonococcal infections. The latter is far more common in the elderly, and manifests as acute suppurative monoarthritis. *Staphylococcus aureus* is the major pathogen, and streptococcal species, Gram-negative aerobes and anaerobes are unusual. On the other hand, gonococci, viruses, *Borrelia burgdorferi* (Lyme disease), mycobacteria and fungi induce nonsuppurative polyarthritis with a slower disease course. Septic arthritis usually attacks diseased joints. For example, rheumatoid arthritis joints are prone to septic arthritis from hematogenous spreading of bacteria as a result of synovial hyperplasia. Other risk factors for septic arthritis are gout, pseudogout, trauma, immunocompromised individuals, intravenous drug addiction, and joint replacement.

Acute suppurative arthritis has a rapid disease course. Patients usually manifest acute arthralgia and limited range of motion, or acute-on-chronic arthralgia. Massive joint effusion is usually present. The most commonly involved joint is the knee, but the hip, shoulder, ankle and wrist can also be involved. It is difficult to differentiate septic arthritis from GA or pseudogout by clinical symptoms/signs and laboratory tests. The diagnosis of septic arthritis relies on the identification of a pathogen in the synovial fluid. A cloudy-creamy effusion, with a white cell count >50,000/mm³, PMN >90% and elevated protein and depressed sugar contents, suggests the probability of septic arthritis. A positive Gram stain can confirm septic arthritis, but a negative result cannot rule it out. On the other hand, a positive crystal examination cannot exclude septic arthritis owing to the possible coexistence of gout and septic arthritis. A bacterial culture of the synovial fluid should be carried out to identify the definitive pathogen and facilitate treatment. For some patients, tuberculous or fungal cultures are necessary.

Antibiotic treatment should be initiated as soon as possible if septic arthritis is suspected. Parenteral antibiotics are prescribed according to the results of Gram staining or the empirical antibiotics for the local regions. After an antibiotic sensitivity test becomes available, the antibiotic can be changed to a more suitable one. Antibiotic treatment should continue for 3–4 weeks, except in cases of gonococcal septic arthritis for which 2 weeks is adequate. Repeated joint tapping is necessary for a large amount of joint effusion to attenuate joint damage. Surgical drainage is reserved for joints in patients who respond poorly to antibiotics, undergo tapping too frequently, have deeply located areas, or exhibit periarticular soft tissue infection. The prognosis of septic arthritis is poor, and permanent joint damage is frequent with the exception of gonococcal infection.

Hemarthrosis

The most common etiology of hemarthrosis in the elderly is joint contusion or sprain. Other etiologies include bleeding tendencies from other diseases and tumors. The knee, elbow and ankle are frequently involved. Patients usually present with acute onset of joint swelling and pain. Severe muscle spasm is noted at physical examination. Joint tapping is the most valuable method to relieve the pain and inflammatory reactions, and to help the diagnosis, provided no bleeding tendency is present. Hematomatous joint fluid indicates a recent hemorrhage. However, the joint fluid turns yellowish after 3–5 days. Synovial fluid analysis shows a non-inflammatory synovial fluid with a white cell count <2,000/mm³ and negative Gram staining.

Ice packs should be prescribed for the injured joint. After the acute stage, a contrast bath can relieve joint swelling. NSAIDs improve the pain and inflammation. A protective orthosis is then used to prevent further injury. After the pain subsides, a repeat physical examination is necessary to check for meniscus tears and cruciate ligament rupture. Soft tissue sonography or magnetic resonance imaging should be arranged for a suspicious lesion. Orthopedist consultation is necessary for arthroscopy.

Osteoarthritis

Osteoarthritis usually presents as chronic arthritis. However, acute exacerbation is possible especially after overusing a joint. Tapping of the joint effusion can help the diagnosis and relieve the symptoms. A local steroid injection can be performed to depress the inflammatory reactions if infection is unlikely.

Other Conditions Mimicking Acute Arthritis

Cellulitis and hallux valgus with bunion formation are easily mistaken for acute monoarthritis. Comprehensive
history taking and a physical examination help with the diagnosis. It should be kept in mind that joint aspiration and injection should not be performed if cellulitis is suspected.

**Acute Polyarthritis**

Atypical gout and nonsuppurative septic arthritis have slower disease courses, but may also present as acute arthritis. Other chronic polyarthritis types, such as rheumatoid arthritis and psoriatic arthritis, flare up acutely from time to time. Other arthritis types, such as palindromic rheumatism and nodal osteoarthritis, rarely cause permanent joint damage. However, Heberden's nodes and Bouchard's nodes may be mistaken for tophi and these should be carefully differentiated.

**Conclusion**

Great care should be taken when dealing with acute arthritis in the elderly. Differential diagnosis and correct treatment to prevent permanent joint damage are mandatory.

**References**