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Arthritis in the intensive care unit

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Septic arthritis

Nongonococcal bacterial arthritis

Overview

Bacterial arthritis is a rapidly progressive, destructive joint disease. The incidence of septic arthritis is approximately 2 to 10 per 100,000 in the general population but is significantly higher in patients with rheumatoid arthritis and other joint diseases [1]. Despite advances in surgical and medical treatment, septic arthritis carries a mortality rate of up to 10% in the general population, with permanent damage to the affected joint occurring in 25% to 50% of cases [1–3]. Morbidity and mortality rates reach as high as 65% and 56%, respectively, in patients with rheumatoid arthritis, polyarticular arthritis, or other significant underlying disease [4]. In the intensive care unit (ICU), septic arthritis is more often associated with severe sepsis and with chronic, debilitating disease, such as renal failure, diabetes mellitus, or malignancy. Accordingly, the mortality rate of septic arthritis in the ICU ranges from 20% to 75%, depending on the severity of the acute and underlying illnesses [4]. Frequently encountered reasons for ICU care of patients with septic arthritis include respiratory failure, decreased level of consciousness, acute renal failure, hypotension, and other multiorgan manifestations of septic shock.

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Pathophysiology

Bacterial arthritis results most commonly from hematogenous spread of infection from distant sites during transient or persistent bacteremia [5]. Bacterial arthritis can also arise from contiguous bone or soft tissue infection or from direct inoculation of organisms into the joint, usually from traumatic injury or during surgery. In ICU patients, septic arthritis may result from seeding of the joints during bacteremia associated with pneumonia, bacterial endocarditis, or other distant infections. Bacterial growth occurs rapidly in an enclosed joint space, stimulating synovial cells to release cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin (IL)-1β, which induce migration of inflammatory cells into the synovial fluid and tissues. Phagocytosis of the bacteria by synovial inflammatory cells and polymorphonuclear (PMN) cells leads to formation of microabcesses within the synovium as well as release of collagenases and other proteolytic enzymes. Synovial cell necrosis can be demonstrated within 48 to 72 hours, as enzymes released by the phagocytic cells destroy the articular cartilage. Because these proteases can remain in the joint even after treatment of the infection, ongoing inflammation and damage to cartilage and bone may persist [5,6].

Staphylococcus aureus is the causative organism in many cases (37–56% in certain series) of bacterial arthritis in adults and generally causes the most severe illness [4,5]. Streptococcal species are the second most common cause of nongonococcal septic arthritis, particularly group A streptococcus, which usually infects the joint through skin or soft tissue infection. Group B, C, and G streptococcal species may also cause bacterial arthritis through the gastrointestinal or genitourinary tracts. Gram-negative bacilli, such as *Escherichia coli, Klebsiella pnemoniae, Pseudomonas aeruginosa,* and *Salmonella enteriditis*, are common causes of bacterial arthritis in susceptible individuals, including chronically ill children and adults with diabetes mellitus, cancer, sickle cell disease, or human immunodeficiency virus (HIV) infection. Anaerobic and polymicrobial joint infections are also more likely to occur in these patients, usually as a result of intra-abdominal or pelvic infections or after trauma or surgery [3,5].

Clinical features

Nongonococcal arthritis presents as an acute monoarthritis in 80% of cases [7]. Symptoms include the acute onset of joint pain and swelling in approximately 85% of patients, with decreased active and passive range of motion. Fever is present in less than half of the cases [2,3]. The most commonly affected joints are the knee (30% of cases), hip (16%), ankle (6%), shoulder (8%), wrist (4%), and elbow (9%) [3].

Polyarticular involvement occurs in approximately 20% of cases of septic arthritis [7,8]. As with monoarthritis, *S. aureus* is the most frequent causative organism, and the knee is the most commonly affected joint. Polyarticular arthritis is more likely to occur in patients with rheumatoid arthritis or other comorbid conditions and is associated with a much worse prognosis than septic monoarthritis. In one retrospective study, 40% of patients admitted to the hospital with

polyarticular septic arthritis required ICU transfer, and 40% of those died of sepsis (compared with 18% and 4%, respectively, of patients with monoarthritis) [4].

Risk factors

Several conditions predispose to septic arthritis. In one large-scale, prospective study, significant risk factors for developing septic arthritis were age of 80 years or above, diabetes mellitus, rheumatoid arthritis, recent joint surgery, and presence of a hip or knee prosthesis with or without skin infection [1].

Rheumatoid arthritis, particularly severe, long-standing, seropositive rheumatoid arthritis, is an important risk factor for septic arthritis. The associated use of oral or intra-articular steroids further increases the risk [9,10]. Crystal-induced arthritis, such as gout and calcium pyrophosphate dihydrate (CPPD) crystal deposition disease, and severe osteoarthritis may also coexist with bacterial arthritis [11]. Intensivists should maintain a high level of suspicion for bacterial arthritis in these patients, because the presentation often mimics an exacerbation of their underlying joint disease. Delay in diagnosis increases the risk of permanent joint damage. [12].

Patients with HIV infection, with or without associated intravenous drug use, are also at higher risk for infectious arthritis. As is the case for immunocompetent patients, *S. aureus* and streptococcal species are the most common organisms [13,14].

Intravenous drug use is a major risk factor for bacterial arthritis, presumably because of transient bacteremia caused by frequent injections. *S. aureus* is the usual pathogen, and these patients are more susceptible than others to methicillin-resistant *S. aureus*. In contrast to septic arthritis in non–drug users, the joints commonly affected in intravenous drug users are the hip, sternoclavicular, sacroiliac, and other joints in the axial skeleton [15,16].

Musculoskeletal symptoms are present in more than 40% of patients with infective endocarditis [17,18]. Rheumatologic manifestations of infective endocarditis include arthralgias, myalgias, peripheral arthritis, septic bursitis, septic discitis, and sacroiliitis [19]. Peripheral arthritis associated with bacterial endocarditis may be infectious or immune-mediated, and aspiration of fluid from the affected joint may reveal a septic or aseptic inflammatory arthritis [20]. Septic arthritis in infective endocarditis is strongly associated with intravenous drug use and with *S. aureus* infection [18,19]. Common sites are the sternoclavicular, sacroiliac, wrist, knee, and ankle joints.

Diagnosis

Synovial fluid analysis. The diagnosis of bacterial arthritis is made by examination of synovial fluid obtained by arthrocentesis or by surgical drainage of the affected joint. Bacteria and PMN cells can be directly observed on Gram's stain. Cultures of the synovial fluid are positive in approximately 90% of cases of nongonococcal arthritis, provided that the patient has not received antibiotics and that the synovial fluid is immediately inoculated onto the culture medium or into

aerobic and anaerobic blood culture bottles [21]. The diagnosis of septic arthritis is suggested by a synovial cell leukocyte count over 50,000 cells/mm^3 with more than 80% PMNs; however, other inflammatory arthritides, such as rheumatoid arthritis and gout, may also present with high leukocyte counts. Measurement of glucose and lactate dehydrogenase (LDH) levels in the fluid is generally not helpful in differentiating between bacterial arthritis and other causes of inflammatory arthritis.

Other diagnostic tests. In contrast to synovial fluid cultures, blood cultures are positive in only 50% of cases of nongonococcal arthritis [21]. Culture of wounds, urine, and other possible sources of infection may aid in identifying the causative organism. Peripheral blood leukocytosis and an elevated erythrocyte sedimentation rate (ESR) or C-reactive protein are commonly found but are nonspecific.

Radiologic evaluation. Evidence of joint destruction is usually not visible on plain-film radiographs for 2 weeks or more. Early changes that may be seen on radiograph include joint effusion and arthritis. Joint space narrowing and joint erosions become evident later in the disease. The presence of gas within the joint of a nonsurgical patient suggests infection with *E. coli* or anaerobic organisms. Computed tomography (CT), MR imaging, and radionuclide imaging may be useful, especially in evaluation of the axial joints, and are more sensitive than plain-film radiographs early in the disease [22].

Differential diagnosis

Other causes of acute arthritis that can mimic bacterial arthritis include gout, calcium pyrophosphate dihydrate deposition disease (CPDD), viral, mycobacterial, fungal, or gonococcal arthritis, traumatic arthritis, or hemarthrosis. Systemic illnesses may also present with arthritis that mimics bacterial arthritis; these conditions include Lyme disease, rheumatoid arthritis, Reiter's syndrome, and bacterial endocarditis.

Treatment

Antibiotics. Treatment with antibiotics should be started in all cases of proven or suspected bacterial arthritis once the appropriate specimens are obtained for culture. Initially the antibiotic regimen is based on the Gram's stain of synovial fluid. A penicillinase-resistant penicillin is the usual choice for gram-positive cocci; in penicillin-allergic individuals, clindamycin or vancomycin can be used. Gram-negative bacilli should be treated initially with an antipseudomonal penicillin or cephalosporin, plus an aminoglycoside. In patients with hospital-acquired arthritis or in other patients in whom methicillin-resistant *S. aureus* is the suspected pathogen, vancomycin is the drug of choice. If the Gram's stain is negative but synovial fluid is purulent, empiric therapy can include a penicillin or cephalosporin for healthy individuals and broad-spectrum antibiotics, such as

those mentioned previously, for immunocompromised or elderly patients. Most antibiotics exceed the minimum inhibitory concentrations in the joint [5,23].

Once the organism is identified and the sensitivity profile is determined, the antibiotic regimen may be altered based on sensitivity, toxicity, cost, and avoidance of promoting selection of resistant organisms (particularly with vancomycin). Antibiotic therapy should be given for 4 weeks. In healthy patients who respond well to initial parenteral therapy, the last 2 weeks of therapy may be given orally. Shorter courses of treatment may be effective in children [5].

Surgical treatment. Drainage of infected synovial fluid is the other essential component of therapy for septic arthritis and may be accomplished by several methods. Closed-needle aspiration is usually effective in draining small joints, such as the wrist or ankle; it may also be the initial mode of drainage for the knee. Arthroscopic drainage may be more effective than needle aspiration for drainage of the knee; it may also be used to drain the shoulder and other joints which are less accessible to needle aspiration. Open surgical drainage is generally required for infections of the hip or sacroiliac joints or of any joint that cannot be adequately drained by needle or arthroscopic techniques [24,25]. Repeated drainage of the infected joint is often necessary. Serial analysis and culture of the synovial fluid should be performed to assess response to therapy.

Prosthetic joint infection

Infection of prosthetic joints is a serious complication that occurs as a result of direct inoculation of bacteria into the joint during surgery or as a result of perioperative bacteremia. The rate of early prosthetic joint infection–that is, infections within 12 months of the surgery–is approximately 2%, whereas the rate of late infection (12 months or more after surgery) is approximately 0.60% per year [26]. The most common causative organisms are *S. aureus* (50%), polymicrobial (33%), gram-negative bacteria (10%), and anaerobes (5%) [27]. The frequency of early infections has decreased significantly because of improved surgical techniques and perioperative antibiotic therapy. Infection of a prosthetic joint causes sepsis and loosening of the prosthesis, and removal of the prosthesis along with a prolonged course of antibiotics is usually necessary.

Gonococcal arthritis

Overview

Disseminated gonococcal infection (DGI) is the most common cause of septic arthritis in young adults. Gonococcal arthritis is preceded by mucosal infection with *Neisseria gonorrhoeae* through the endocervix, urethra, rectum, or oropharynx. The primary infection is often asymptomatic and may precede the development of DGI by days to months. Bacteremia and septic arthritis occurs in approximately 1% of patients with local infection. All sexually active individuals are susceptible to DGI, but those at higher risk include menstruating or pregnant

women and patients with inherited late complement deficiencies or with other immunodeficient conditions [28].

Clinical features

DGI typically presents with the acute onset of fever, skin lesions, tenosynovitis, and arthritis. Tenosynovitis in DGI commonly involves the small joints and tendons of the hands, feet, wrists, and ankles. In less than half of patients, oligoarthritis or polyarthritis may develop in a migratory, additive, or diffuse pattern. Skin lesions are also diffuse and varied, including erythematous macules, papules, pustules, and necrotic lesions, which may be present at different stages or simultaneously. In a given individual DGI may be manifested predominantly by a syndrome of fever, tenosynovitis, and dermatitis or predominantly by arthritis with large, purulent joint effusions. Rare complications of DGI include endocarditis and pericarditis [28–30].

Diagnosis

The diagnosis of DGI is made by identifying *N .gonorrhoeae* in the blood, synovial fluid, or skin lesions. Blood cultures are positive in less than 50% of patients and are more likely to be positive in those with predominant fever, rash, and tenosynovitis. Culture of the synovial fluid is positive in less than 50% of patients with significant arthritis. Leukocytosis in the synovial fluid of 30,000 to 100,000 cells/μL with predominance of PMNs supports the diagnosis but does not help differentiate DGI from nongonococcal arthritis. Culture of the genitourinary tract gives the highest yield; culture of other possible sites of infection, such as the pharynx and the rectum, should also be taken. The differential diagnosis of DGI includes other infectious diseases, such as viral infections, mycobacterial infections, Lyme disease, hepatitis B, and *Neisseria meningitidis*, as well as systemic conditions such as rheumatoid arthritis and systemic lupus erythematosus.

Treatment

Treatment of DGI should begin with a third-generation cephalosporin such as ceftriaxone, or, if the culture shows a sensitive strain, with penicillin, and is continued for 7 days. Initially, antibiotics are given parenterally, followed by oral administration if the symptoms are resolving. Oral antibiotics that are effective in treating clinically improving DGI include penicillinase-resistant penicillins, second-generation cephalosporins, and ciprofloxacin. Concurrent or empiric treatment for *Chlamydia trachomatis* infection with doxycycline or azithromycin is also advised [28,31].

Other causes of infectious arthritis

Polyarthritis may occur as a result of infection with alphaviruses, adenovirus, coxackieviruses, rubella virus, human parvovirus, and other viruses [32]. Hepatitis B infection can cause an immune-mediated polyarthritis which may precede

jaundice by days to weeks. Mycobacteria species are infrequent causes of infectious arthritis and typically follow an indolent course. Fungal species are another uncommon cause of arthritis. For example, *Cocidioides immitis*, a soil fungus indigenous to the Southwestern United Sates and parts of Mexico and Central and South America, may cause arthritis in patients with primary or disseminated disease. *Histoplasma capsulatum,* which is found in the Mississippi and Ohio River valleys, may cause an acute polyarthritis, often in conjunction with the appearance of erythema nodosum or erythema multiformae. *Blastomyces dermatitides*, also endemic to the Mississippi and Ohio River Valleys, may cause acute monoarthritis along with pulmonary infection. Lyme disease, caused by infection with *Borrelia burgdorferi*, is often found in the Northeastern, mid–Atlantic, and Midwestern United States where its vector, the *Ixodes* tick, is indigenous; arthritis is a feature of late disease and is usually chronic.

Crystal-associated arthritis

Gout

Overview

The annual incidence of gout is approximately 1.6 per 1000 in men and approximately 0.5 per 1000 in women [33]. The typical age of onset is 45 to 64 years, although onset after age 65 often occurs. The incidence and prevalence of gout are both increased with serum uric acid levels greater than 10 mg/dL [34,35]. Acute gout usually occurs in hyperuricemic individuals, but in intensive care patients it can occur after a sudden drop in uric acid level caused by trauma, surgery, or serious infection [36]. Other risk factors for the development of gout are increased body mass index, hypertension, alcohol consumption, and renal disease [37,38]. Patients who are taking medications that inhibit uric acid secretion are also at risk for developing gout; these medications include diuretics, cyclosporine, azathioprine, ethambutol, pyrazinamide, and niacin [35]. Use of iodinated contrast media, for example in angiography or CT studies, may precipitate attacks in hospitalized patients, as can the use of parenteral nutrition [39].

Pathophysiology

Acute gouty attacks are caused by precipitation of monosodium urate crystals in the synovial space and consequent stimulation of an inflammatory response by PMN cells and phagocytic synovial cells. The subsequent release of a number of inflammatory mediators, including IL-6, IL-8, IL-1, TNF-α, and C5a sustain the inflammatory process [40]. The presence of monosodium crystals in the joint is necessary but not sufficient to trigger an attack of gout, as evidenced by the presence of these crystals in asymptomatic joints. Factors that affect crystal nucleation and growth and the balance between pro- and anti-inflammatory cytokines are also important in causing a gouty attack [41].

Clinical features

Gout presents as an acute monoarthritis in 85% to 90% of patients [42]. Sudden onset of severe pain and tenderness of the joint, accompanied by swelling, warmth, and erythema, are typical features. The most commonly affected joint is the first metatarsal; other joints of the lower extremities, such as the ankle and knee, are also commonly involved, whereas involvement of the fingers, wrists, and elbows occurs less frequently. Fever may be present. An attack of acute gout can last days to weeks, after which there may be spontaneous resolution of the symptoms. The usual course of the disease is one of intermittent attacks and remissions, followed by a chronic phase marked by polyarticular arthritis and the formation of tophi.

Gout in elderly persons may present with several atypical features: there is a higher proportion of female patients and of polyarticular involvement, a predisposition for involvement of the small joints of the fingers, a higher frequency of fever or even delirium accompanying acute attacks, and development of tophi earlier in the disease [39]. Because of these atypical features, a high index of suspicion is required to make the diagnosis in elderly persons and to distinguish gout from other causes of polyarthritis such as osteoarthritis. Tophi in patients with osteoartritis are often mistaken for or may be superimposed on Heberden's nodes [43].

Diagnosis

The diagnosis of gout is made by identification of monosodium urate crystals in synovial fluid or tissue. These crystals are needle-shaped and negatively birefringent when examined under polarized-light microscopy. The leukocyte count in the synovial fluid during an acute attack usually ranges from 5000 to 75,000 cells/mL and is composed predominantly of PMN cells. [44]. The serum uric acid level is usually elevated but may be normal, particularly after trauma, surgery, or other major stressors [36]. The differential diagnosis of acute gout includes pseudogout and other crystal-associated arthropathies, as well as infectious arthritis.

Treatment of acute gout

The aim of treatment of acute attacks of gout is to relieve symptoms and to terminate the attack by suppressing inflammation. A number of agents may be used for this purpose, including nonsteroidal anti-inflammatory drugs (NSAIDs), colchicine, corticosteroids, and corticotropin.

Colchicine is the oldest drug used to treat acute gout. It is effective in relieving symptoms in 75% to 80% of patients, especially when used early in the attack [45]. The use pf colchicine is often limited by gastrointestinal (GI) side effects such as abdominal cramps, diarrhea, and nausea and vomiting, and by its potential for serious renal, hepatic, and bone marrow toxicity. Colchicine is given orally, 0.5 to 0.6 mg every hour, until symptoms resolve, GI toxicity develops, or the cumulative dose reaches 6.0 mg (less if renal or liver disease is present). Intravenous colchicine has also been used with success but is associated with a greater risk of serious systemic toxicity, as well as with local thrombophlebitis or extravasation injury [36,46]. For these reasons, the drug is usually avoided in the

elderly and in patients with renal or hepatic disease. In patients with normal liver and kidney function, the risk of toxicity may be reduced by limiting each intravenous dose to 2 mg or less, with the total cumulative dose not exceeding 4 mg in 24 hours and the total course of therapy not exceeding 7 days [47].

Nonsteroidal anti-inflammatory drugs are generally the first-line agents for treatment of acute gout and usually relieve symptoms within 1 to 3 days of initiating therapy [45,48]. The NSAID that has been used classically for acute gout is indomethacin, although other NSAIDs are equally effective. Nonsteroidal anti-inflammatory drugs should be avoided in patients with peptic ulcer disease or renal insufficiency. Extreme caution should accompany their use in elderly persons because of their higher susceptibility to renal toxicity.

Corticosteroids are increasingly used to treat acute gouty attacks. Septic arthritis should be ruled out by analysis and culture of the joint fluid before initiating steroid therapy. Intra-articular injection of methylprednisolone or corticosteroid esters after joint aspiration can provide rapid relief of pain and stiffness [39]. Systemic corticosteroids may also be used and are particularly helpful in polyarticular attacks of gout; they may be given in doses of prednisone starting at 40 to 60 mg per day and tapered over 7 to 10 days [49]. Alternatively, injections of corticotropin (40–80 units) given intravenously, intramuscularly, or subcutaneously, or a single intramuscular injection of triamcinalone (40–60 mg) are equally efficacious [34,35,50–52]. These regimens are particularly useful in patients who cannot tolerate therapy with NSAIDs or colchicine and in those who cannot take drugs orally.

Calcium pyrophosphate dihydrate crystal deposition disease

Overview

Calcium pyrophosphate dihydrate crystal deposition disease is commonly referred to as ''pseudogout'' because of its similarities to gout. The disease is characterized by precipitation of CPPD crystals into the articular cartilage (causing chondrocalcinosis) and subsequent inflammation. The mechanisms by which these crystals are formed and by which their deposition triggers the inflammatory reaction are not well understood. Most cases are idiopathic, although familial forms do exist [53,54]. The prevalence of the disease, manifested as chondrocalcinosis with or without osteoarthritis, is 10% to 15% in the population aged 65 to 75 years and more than 40% in those over age 80 years [35]. A number of endocrine and metabolic disorders are associated with CPPD deposition; these disorders include hyperparathyroidism, hemochromatosis, hypomagnesemia, hypophosphatasia, Wilson's disease, and possibly hypothyroidism [55–57].

Clinical features

Much like gout, CPPD deposition disease is characterized by acute episodes of mono- or oligoarthritis lasting days to weeks, with asymptomatic periods between attacks. The knee is the most commonly affected joint, although any joint may be involved. Like gout, acute attacks of pseudogout may be spontan-

eous or triggered by trauma, surgery, or severe medical illness, such as myocardial infarction [35,39,58].

Diagnosis

Definitive diagnosis of pseudogout is made by demonstration of typical radiographic calcifications and the presence of crystals in the synovial fluid that show no or weakly positive birefringence under polarized-light microscopy. Probable disease is diagnosed when only one of these criteria is present. Alternatively, CPPD crystals in synovial fluid may also be identified by chemical analysis or X-ray diffraction. The differential diagnosis includes infectious arthritis, gout, and other crystal-induced arthropathies.

Treatment

Acute attacks of pseudogout are usually treated with NSAIDs. Aspiration of joint fluid with concurrent injection of steroids (in the absence of septic arthritis) is also effective in relieving acute inflammation and may shorten the symptoms' duration [48]. Intravenous colchicine has also been used with some success, but the associated toxicity greatly limits its use [59].

Other crystal-induced arthropathies

Arthritis can also occur as a result of deposition of other calcium-containing crystals, such as calcium oxalate, hydroxyapatite, and octacalcium phosphate. Clinical manifestations include arthritis, bursitis, tenosynovitis, and the "Milwaukee shoulder syndrome" [60,61]. Calcium oxalate crystals are an uncommon cause of arthritis in patients receiving hemodialysis [62].

Hemarthrosis

Overview

Hemarthrosis, or bleeding into the joint space, is an uncommon problem in the ICU. The causes of hemarthrosis can be categorized as traumatic and nontraumatic. Nontraumatic causes include hemophilia A and B (the incidence is almost 100% in severe hemophiliacs), warfarin therapy (particularly when the international ratio is greater than twice normal), pigmented villonodular synovitis, hemangioma, sickle cell anemia, Charcot's joint, myeloproliferative disease, gout, pseudogout, thrombocytopenia, synovioma, scurvy, arteriovenous fistula, osteonecrosis, and synovial metastases [3,63].

Clinical manifestations

The most common site for hemarthrosis is the knee joint. Acute hemarthroses in hemophilia originate from the subsynovial venous plexus underlying the joint capsule and produce a tingling or burning sensation, followed by intense pain.

Typically, the joint is swollen, hot, and tender to palpation, with erythema of the overlying skin. Joint mobility is greatly compromised by pain and stiffness. In hemophiliacs, recurrent hemarthrosis may occur with minimal or no trauma, usually affects the same joints, and can lead to chronic arthritis.

Patients with hemarthrosis caused by anticoagulation therapy with warfarin have a similar clinical presentation. They often remain symptomatic until anticoagulation is reduced or discontinued [64].

Treatment

Replacement of deficient clotting factors results in symptomatic relief of acute hemarthrosis in hemophiliacs. Other therapeutic interventions include arthrocentesis, analgesics, and joint immobilization for a period of approximately 2 days, with subsequent passive range-of-motion exercises [63]. Surgical synovectomy has been shown to reduce the severity of synovitis and the incidence of recurrent hemarthrosis in patients with frequent or persistent hemarthroses despite adequate conservative therapy. Radioisotopic synovectomy using yttrium-90 and dysprosium-165 has also been successful in these cases [63].

Management of hemarthrosis caused by anticoagulation therapy includes careful joint aspiration and prompt discontinuation of warfarin. Supplementation with vitamin K or fresh frozen plasma is usually not warranted, given the minor and transient morbidity of hemarthrosis in this setting [65].

Summary

Acute arthritis in critically ill patients may be caused by local or systemic infection, by a flare of chronic joint disease such as rheumatoid or crystal-associated arthritis, or by less common entities such as hemarthrosis. Diagnosis requires analysis of synovial fluid, and appropriate treatment is based on its findings. Prompt diagnosis and treatment are usually necessary to prevent the significant morbidity associated with these conditions.

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