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ESSENTIALS Bacterial causes of arthritis—Septic arthritis is the most serious cause of one or more hot swollen joints. A causative organism can be iden- tified in about 80% of cases, with Staphylococcus aureus the most common, followed by Streptococcus and gram-negative organisms. The key diagnostic investigation is microscopy and culture of aspir- ated joint fluid. Management is with drainage of bacteria, pus, and debris from the joint, along with antibiotics. Consensus is that these should be given intravenously for up to two weeks, or until clinical signs improve, followed by oral antibiotics for four weeks. Prosthetic joint infection is a particular challenge requiring specialist care. Viral causes of arthritis—Arthralgia and/or arthritis are common oc- currences with many viral infections, particularly parvovirus, hepa- titis B and C, rubella, human immunodeficiency virus, α-(including chikungunya) and dengue viruses. Joint manifestations are usually sudden in onset, correlate with the onset of clinical illness, and gen- erally self-limiting, but can persist following infection with chikun- gunya and dengue. Bacterial causes of arthritis Introduction There is a broad differential diagnosis for the patient presenting with one or more hot swollen joints (Fig. 19.7.1). Septic arthritis is the most serious of these; it is a medical emergency with a case fatality rate of 11% for monoarticular disease and 30% for polyarticular dis- ease. Considerable morbidity is also associated with the condition; a poor functional outcome is seen in 24%, while osteomyelitis com- plicates 8% of cases. Epidemiology The incidence of septic arthritis is 2–10 per 100 000 and is thought to be rising due to the combination of an ageing population, greater use of immunosuppressive therapies and increased orthopaedic proced- ures such as arthroscopies. Septic arthritis typically occurs in the very old and very young. Risk factors for septic arthritis are presented in Table 19.7.1. The presence of pre-existing joint pathology is an important risk factor, with the risk greater in rheumatoid arthritis compared to osteoarthritis. Rheumatoid arthritis is present in 15% of cases of monoarticular and 50% of cases of polyarticular septic arthritis. The increased risk with rheumatoid arthritis is likely to be multifactorial; rheumatoid

arthritis itself is a risk factor for infection, while therapy for this condition also confers additional risk. UK national registry data shows an increased risk of septic arthritis in rheumatoid arth- ritis patients treated with anti-TNF therapy when compared to those treated with conventional disease-modifying antirheumatic 19.7 Infection and arthritis Graham Raftery and Muddassir Shaikh Acute monoarthritis Gout Calcium pyrophosphate Bacterial Fungal Mycobacterial Osteoarthritis Avascular necrosis Spontaneous haemarthrosis Reactive arthritis Rheumatoid arthritis Trauma Infection Lyme disease Inflammatory arthritis Psoriatic arthritis Other causes Crystals Calcium oxalate Hydroxyapatite Pigmented villonodular synovitis Fig. 19.7.1 Causes of acute monoarthritis. Table 19.7.1 Risk factors for septic arthritis Rheumatoid arthritis Joint prosthesis/recent joint surgery Diabetes Alcoholism Intravenous drug abuse Leg ulceration Low socioeconomic status Pharmacological immunosuppression

section 19 Rheumatological disorders 4458 drug therapies; this increased risk is greatest in the early months following initiation of this treatment. Local and distant risk fac- tors for infection may be present. Local factors include hip or knee prosthesis and recent arthroscopy. Septic arthritis is recognized following intra-articular injection, but the absolute risk associated with this procedure is low (three infections per 7900 procedures). Of the distant risk factors, skin infection is of particular importance. Diagnostic criteria Criteria for the diagnosis of septic arthritis proposed by Newman have been widely accepted. The diagnosis of septic arthritis is made when one of the following four points is met:

1. Organism is isolated from affected joint
2. Organism is isolated from elsewhere (in the context of clinical suspicion of septic arthritis of one or more joints)
3. There is histological or radiological evidence of joint infection
4. There are clinical features of septic arthritis with turbid fluid as- pirated from joint (usually in context of prior antibiotic therapy) Pathogenesis Study of human septic arthritis pathogenesis is challenging. A murine model of septic arthritis developed by Tarkowski has been used to study bacterial virulence and host response with a focus on Staphylococcus aureus-induced disease. This mouse model of septic arthritis has similarities to human arthritis; the arthritis is induced by haematogenous spread while the rate of progression and severity of joint damage also mirror human disease. Bacterial virulence The virulence of Staphylococcus aureus comes from several bacterial factors. A capsule is expressed by many strains and acts as a phys- ical barrier inhibiting phagocytosis. Adhesins are cell surface com- ponents which facilitate bacterial colonization of host tissue; each strain of Staph Aureus can express different adhesins. Inactivation of adhesins by vaccination has a protective effect for mice exposed to Staph Aureus. Staphylococcal protein A (a cell wall anchored protein) is another virulence factor. Strains expressing this pro- tein are associated with more severe arthritis and increased mor- tality. Staphylococcus aureus produces many toxins and enzymes. A subset of ‘superantigen’ toxins stimulate T lymphocytes to release pro-inflammatory cytokines with systemic effects such as fever and hypotension. Superantigen producing strains of Staphylococcus aureus are more likely to induce septic arthritis of an increased severity and associated with greater mortality when compared to strains not producing superantigen toxins. The host response Neutrophils have an important role in the early response to Staphylococcus aureus infection. Peptides produced by Staphylo coccus

aureus act as a chemoattractant for neutrophils. A deficiency in neutrophils increases the severity of disease, as does comple- ment deficiency. Macrophage derived cytokines facilitate joint damage but these cytokines also have a protective role. TNFα has a role in bacterial clearance and is associated with reduced mor- tality, while IL1 receptor signalling is involved in host protection during Staphylococcus aureus infection. B lymphocytes are not sig- nificantly involved in septic arthritis whereas T lymphocytes are actively involved in the disease process. Cytokines expressed by T lymphocytes have a mixed role: interferon-γ is implicated in joint destruction but is protective in early septic arthritis; IL-10 acts as an anti-inflammatory molecule and a lack of IL-10 causes enhanced joint destruction; IL-4 acts as an inhibitor to phagocytosis resulting in decreased clearance of bacteria. Causative organisms The distribution and sensitivities of pathogens causing septic arth- ritis has changed little over the last 20 years. A causative organism is identified in 82% of cases. Staphylococcus aureus is the most com- monly identified organism, followed by Streptococcus. Methicillin- resistant Staphylococcus aureus (MRSA) is responsible for around 10% of Staphylococcus aureus infections. While Staphylococci and Streptococci are also the most prevalent organisms in all at risk sub- groups, gram-negative organisms are often seen in the very young and very old, patients with diabetes, and intravenous drug users. Intravenous drug users are prone to infections with an array of or- ganisms, including pseudomonas, anaerobes, and fungi. The role of immunosuppression is important. In patients treated with anti-TNF therapies developing septic arthritis, Staphylococcus aureus is the most commonly reported organism, with MRSA re- corded in 11% of cases. Organisms that rarely cause septic arthritis in the immunocompetent can be seen in patients treated with anti- TNF therapies; cases of septic arthritis with intracellular organisms such as salmonella and listeria have been documented. Prosthetic joint infection is typically caused by Staphylococcus, with coagulase negative Staphylococci and MRSA more prevalent than in native joint infection. Clinical features The typical presenting symptoms are joint pain, redness, and swelling, with restriction in range of movement of the affected joint. Symptoms usually evolve over a few days up to three weeks, with a median time to hospital admission of seven days. The knee is the most frequently affected site. Polyarticular disease is present in around 15% of patients with a mean of three joints involved. Pain in a joint with known arthropathy is likely to be disproportionate to the typical pain experienced in that joint. Symptoms suggestive of infection (fever, sweats, and rigors) may not be present and raised temperature on admission to hospital is only seen in around 60% of patients. Investigations An algorithm for management of the hot swollen joint in adults is shown in Fig. 19.7.2. Tests of synovial fluid and blood Synovial fluid should be aspirated from the affected joint and sent for gram stain and culture prior to antibiotic therapy. Gram stain is positive in around 50% of patients and culture is positive in 67%. In addition to gram stain and culture, synovial fluid should also be analysed for crystals by polarizing light microscopy. Suspected pros- thetic joint infection should be referred to an orthopaedic surgeon and aspirated in theatre under full aseptic technique. Blood cultures should always be taken, and as with joint aspir- ation this should be done prior to antibiotic therapy to increase the likelihood of a positive microbiological diagnosis. Blood cultures

 are positive in 33% to 46% of cases and can be so when the joint aspirate is negative. White cell count (WCC), C-reactive protein (CRP), and erythrocyte

sedimentation rate (ESR) should all be checked and are usually raised. However, each of these can be normal in patients with septic arthritis and this finding does not refute the diagnosis. Particular caution should be exercised in patients taking immunosuppressive therapies; for example, Tocilizumab profoundly suppresses C-reactive protein production by the liver via its inhibition of IL-6. Patient presents with acute increase in pain ± swelling in one or more joints GP History examination Clinical impression septic arthritis No definite alternative diagnosis Definite alternative diagnosis Inflammatory arthritis Crystal arthritis Haemarthrosis Trauma Bursitis/cellulitis Treat as appropriate Refer for urgent A&E or specialist assessment Self referral to A&E History examination MUST ASPIRATE and other investigations NOT SEPTIC Diagnosis SEPTIC ARTHRITIS Empirical antibiotic treatment (as per local protocol) Alter if necessary once results available Management of septic arthritis in secondary care Admit patient to hospital (rheumatology or orthopaedics according to local custom) Ensure synovial fluid sample is taken, with blood and any other relevant culture samples prior to starting antibiotics Commence antibiotics as per protocol Joint should be aspirated to dryness as often as required (either by needle aspiration or arthroscopically) Seek rheumatology or orthopaedic advice if in doubt Further imaging e.g. MRI–osteomyelitis may require surgical intervention If there is lack of resolution despite treatment consider the following: Incorrect causative organism Modification of antibiotic therapy Seek specialist advice Alternative foci of infection or systemic sepsis Fig. 19.7.2 Algorithm for the management of hot swollen joint in adults. Reproduced from Coakley G et al. (2006) BSR & BHPR, BOA, RCGP and BSAC guidelines for management of the hot swollen joint in adults. Rheumatology (Oxford), 45(8), 1039–1041, by permission of Oxford University Press.

section 19 Rheumatological disorders 4460 Renal and liver function should also be checked to assess end organ damage, which may inform choice of antibiotic. Investigations to identify a distant source for infection should be tailored to the clinical presentation and may include chest X-ray, urine culture, wound and ulcer swab culture. Similarly, investiga- tions to look for a haematogenous source of infection should be considered (e.g. echocardiography). Endocarditis and discitis can precede, coexist with, or complicate septic arthritis (especially due to Staphylococcus aureus) in up to 5% of patients. If gonococcal septic arthritis is suspected the microbiology labora-tory should be informed before joint aspiration as delay in culturing the joint aspirate can lower the yield for gonococcus growth. Gonorrhoea nucleic acid amplification testing (NAAT) should be requested from the following sites in those with possible gonococcal septic arthritis, ensuring the correct swabs are used: heterosexual men—first pass urine sample and throat swab; men who have sex with men—first pass urine sample, throat swab and self taken rectal swab; women-self-taken vaginal swab, self-taken rectal swab and throat swab. Molecular diagnostic techniques such as broad range polymerase chain reaction targeting 16S ribosomal RNA will increasingly have a role to play in culture negative septic arthritis. These are particularly useful where patients have received antibiotic therapy prior to cul- ture, and also in identifying anaerobes and other organisms such as Kingella Kingae, where conventional culture yield is low. White cell count, C-reactive protein, and erythrocyte sedi- mentation rate cannot reliably differentiate septic arthritis from noninfective causes. Serum procalcitonin is an emerging biomarker which may be useful in this regard; a small number of studies have shown procalcitonin to be sensitive and specific in early septic arth- ritis, and serum procalcitonin does not rise in acute crystal arthritis. The main limitation of procalcitonin measurement is that it is likely to be raised in any bacterial infection, and many patients with a hot joint will have nonarticular infection at presentation. In such pa- tients procalcitonin has no discriminant function. Furthermore, the limited evidence in suspected

prosthetic joint infection has found procalcitonin to be less sensitive than C-reactive protein. Imaging Plain radiographs do not have a role in the diagnosis of septic arth- ritis but may offer clues to an alternative diagnosis, such as the presence of chondrocalcinosis caused by calcium pyrophosphate deposition. Current imaging modalities cannot reliably differentiate septic arthritis from other acute inflammatory joint pathology, but MRI is useful in the assessment of osteomyelitis. Ultrasound can be useful to guide joint aspiration, particularly from deep joints such as the hip. Management Antibiotics There is a paucity of high-quality evidence on antibiotics for native joint septic arthritis and no clear preferred antimicrobial strategy. Consensus is that intravenous antibiotics should be given for up to two weeks, or until clinical signs improve, followed by oral anti- biotics for four weeks. An exception is gonococcal arthritis, which is treated with a one-week course of a third generation cephalosporin. UK guidance on antimicrobial therapy for septic arthritis is shown in Table 19.7.2. The choice of empiric treatment should adequately treat Staphylococcus aureus and Streptococcus, as these are the most likely pathogens in all patient groups; antibiotic choice should also be informed by patient risk factors and local patterns of organism prevalence and drug resistance. Drainage Bacteria, pus, and debris should be removed from the joint. The op- tions for this include closed needle aspiration, or surgical drainage by arthroscopy or open arthrotomy. There is a lack of evidence to favour one approach over another, with little difference in mortality or length of hospital stay. Whichever technique is chosen, it should be repeated as required. In patients who have a suboptimal response to needle aspiration, arthroscopic aspiration or open arthrotomy should be used. Historically, open arthrotomy has been the preferred technique for septic arthritis of the hip although there is evidence that arthroscopy may be a safe and effective technique in this setting. Prosthetic joint infection Prosthetic joint infection is a particular challenge. The presence of foreign material within the joint provides a platform for biofilm for- mation, an organized bacterial colony that grows on the implant surface. Bacteria within the biofilm may transiently have reduced metabolic activity and become less susceptible to antibiotics reliant on cellular replication for their mechanism of action. The biofilm is difficult to eradicate. Effective management of prosthetic joint infection requires a sur- gical strategy combined with appropriate antibiotic therapy. Multiple factors influence the choice of surgical strategy. In patients with a well-fixed prosthesis within 30 days of prosthesis implantation or with less than three weeks of infectious symptoms, debridement, antibiotics, and implant retention can be considered. For infections that occur more than 30 days following arthroplasty, joint revision is almost always required. Joint revision can be performed as a one- stage or two-stage procedure, with the latter being more commonly employed. A two-stage procedure separates joint removal, debride- ment and closure (the first-stage) from subsequent re-insertion by weeks or months. A one-stage exchange may be chosen if the patient is not able to undergo multiple operations and providing there is good soft tissue and good bone stock. Novel therapeutics Corticosteroids Joint destruction in septic arthritis occurs as a consequence of the in- flammatory response to infection. The suppression of the inflamma- tory response with corticosteroids has been proposed as a potential adjuvant treatment. Two randomized, placebo controlled studies have looked at the administration of intravenous dexamethasone in add- ition to antibiotics for management of septic arthritis in children. This was found to be safe and led to a more rapid clinical improvement than standard treatment. There was also significantly less residual joint dysfunction at 6 and 12 months in the dexamethasone group. However, at present there is insufficient evidence to advocate the use of corticosteroids in the management of adult septic arthritis. Bisphosphonates Bisphosphonate therapy has been investigated in animal models of Staphylococcus aureus septic arthritis. The addition of zoledronic

19.7 Infection and arthritis 4461 acid to antibiotics and corticosteroids led to less osteoclast activity and less severe arthritis. While the authors propose this triple treat- ment be considered in human septic arthritis, there is insufficient evidence to support this approach at the present time. Viral causes of arthritis Introduction Arthralgia and/or arthritis are common occurrences with many viral infections. Joints complaints are a common manifestation of infections with parvovirus, hepatitis B and C, rubella, α- and dengue virus, and joint symptoms are observed less commonly in associ- ation with a wide variety of other viral infections (e.g. herpes viruses including Epstein–Barr virus, mumps, human immunodeficiency virus, and enteroviruses). Aetiology and pathogenesis A variety of different mechanisms have been described which enable viruses to initiate rheumatic symptoms. Direct invasion of the joint has been proposed as a mechanism utilized by ru- bella and rubella vaccine virus, parvovirus, alphaviruses, and en- teroviruses. Both rubella and rubella vaccine virus are thought to grow preferentially in synovial tissues and have been cultured from joint tissues. Formation and deposition of immune com- plexes (containing viral particles as the antigen component) as a result of humoral response to viral infection is thought to be the mechanism behind arthritis seen with hepatitis B, hepatitis C, and parvovirus. Molecular mimicry may also occur, whereby antibodies directed against viral antigens cross react with tissue antigens. The modes of transmission of viruses causing arthritis are shown in Table 19.7.3. Clinical features Viral arthritides typically present with symmetric polyarticular dis- ease which can be in the form of arthralgia or arthritis. These mani- festations are usually sudden in onset, correlate with the onset of clinical illness, and generally self-limiting, although infections due to rubella virus, parvovirus, and alphavirus can cause persistent joint symptoms. However, even with persistent symptoms, viral arthritis very rarely leads to deforming and destructive joint dis- ease. Musculoskeletal manifestations of some of the common viral arthritides are discussed next. Hepatitis B virus Up to 25% of patients with acute hepatitis B virus infection develop joint symptoms in the prodromal phase. The arthritis most commonly affects hands and knees, although other large joints can be affected as well. The joint symptoms are usually self-limiting and resolve with the onset of jaundice, and there are no reports of resulting joint damage. Polyarthritis in the setting of chronic hepatitis B virus infection is less common, but can occur as part of clinical syndromes due to im- mune complex mediated diseases such as hepatitis B virus-associated polyarteritis nodosa and essential mixed cryoglobulinemia. Hepatitis C virus About one-fifth of patients with chronic hepatitis C virus infection complain of arthralgia. Two-thirds of these patients have symmet- rical inflammatory arthritis involving small joints of the hands, re- sembling rheumatoid arthritis but without erosions; the remaining one-third have oligoarthritis. The arthritis generally improves with antiviral treatment. Symptomatic treatment is usually with nonsteroidal anti-inflammtory drugs. Hydroxychloroquine has been used, but other conventional disease-modifying agents employed in the treatment of rheumatoid arthritis (such as methotrexate) are of limited value because of problems with hepatotoxicity. Biological agents including etanercept have been used for the treatment of in- flammatory arthritis in patients with chronic hepatitis C virus in- fection. Arthritis can also be one of the manifestations of mixed cryoglobulinemia seen with hepatitis C virus infection. Parvovirus Parvovirus B19 is the cause of fifth disease or erythema infectiosum. Less than 10% of children with this syndrome have joints symptoms, Table 19.7.2 Empirical antibiotic choice in suspected septic arthritis Patient group Antibiotic choice No risk factors for atypical organisms Flucloxacillin 2 g qds iv. Local policy may be to add gentamicin i.v. If penicillin allergic, Clindamycin 450–600 mg qds i.v. or 2nd or 3rd generation cephalosporin i.v. High risk of Gram-negative sepsis (elderly, frail, recurrent UTI, recent abdominal surgery) 2nd or 3rd generation cephalosporin, e.g. cefuroxime 1.5 g tds i.v. Local policy may be to add flucloxacillin i.v. to 3rd

generation cephalosporin. Discuss allergic patients with Microbiology—Gram stain may influence antibiotic choice MRSA risk (known MRSA, recent inpatient, nursing home resident, leg ulcers or catheters, or other risk factors determined locally) Vancomycin i.v. plus 2nd or 3rd generation cephalosporin i.v. Suspected gonococcus or meningococcus Ceftriaxone i.v. or similar dependent on local policy or resistance i.v. drug users Discuss with microbiologist ITU patients, known colonization of other organs (e.g. cystic fibrosis) Discuss with microbiologist Reproduced from Coakley G et al. (2006) BSR & BHPR, BOA, RCGP and BSAC guidelines for management of the hot swollen joint in adults. Rheumatology (Oxford), 45(8), 1039–1041, by permission of Oxford University Press. Table 19.7.3 Viral causes of arthritis according to mode of transmission Blood borne Hepatitis B Hepatitis C HIV Arthropod borne α-viruses including (chikungunya virus) Dengue Respiratory acquired Parvovirus Rubella

section 19 Rheumatological disorders 4462 while more than half of adults complain of joint involvement, where parvovirus is responsible for up to 15% of cases of acute arthritis. In children joint involvement can be oligoarticular and asym- metric, often involving the knees. The arthritis in adults is typically of acute onset, symmetrical and polyarticular, commonly affecting metacarpophalangeal and proximal interphalangeal joints, and it may be mistaken for rheumatoid arthritis. Joint symptoms typically resolve within a few weeks but can persist for several months in 20% of patients. Rubella Up to one-third of patients with rubella or given rubella virus vac- cine develop arthritis. Following administration of rubella vaccine joints symptoms usually develop after two weeks. The onset of arth- ritis due to rubella infection is seen within a few days before and after the appearance of rash. Usually small joints of hands and wrists are affected; aside from the knees, involvement of other joints is less common. Tenosynovitis has also been described. Symptoms usually resolve within a couple of weeks. Alpha viruses Alphaviruses (including chikungunya virus) are transmitted to verte- brate hosts by arthropod vectors (usually mosquitoes). Joints involve- ment is observed in almost all patients with symptomatic infections with alphaviruses and is usually symmetrical and polyarticular, often involving small joints of hand and feet. The onset of joint symptoms in chikungunya is abrupt and severe. Symptoms usually resolve in six months, although some patients with chikungunya can have per- sistent symptoms lasting for several years. Chronic erosive inflamma- tory arthritis has also been reported in patients with persistent joint symptoms following chikungunya infection. Dengue virus Rheumatic manifestation are a major clinical feature of dengue virus infection and up to three-quarters of patients develop musculoskel- etal manifestations, which can include involvement of muscles, tendons, joints, and bone. Typically the patient complains of marked muscle and joint pain (also described as bone-break fever). Symptoms usually resolve within a few weeks, but chronic arthritis has been reported. HIV Several disorders with musculoskeletal manifestations have been described in HIV-infected patients. HIV-associated arthritis usually involves lower limbs in an oligoarticular pattern, but involvement of other joints can also occur in a mono or polyarticular distribution. The illness is typically self-limiting and resolves within six weeks. Often no treatment other than nonsteroidal anti-inflammtory drugs is required. However, a few patients can have a prolonged course of arthritis, and disease-modifying antirheumatic drugs used in the treatment of rheumatoid arthritis such as sulfasalazine, hydroxychloroquine, methotrexate and anti-TNFα agents, have been used in those not responding to symptomatic treatment and to nonsteroidal anti-inflammtory drugs. Other chronic arthritides such as spondyloarthropathies (reactive arthritis, psoriatic arthritis and undifferentiated spondyloarthro pathy) and rheumatoid arthritis have also been described in patients with HIV. Whether HIV infection plays a pathogenic role in causing these disorders or is a

coincidental finding remains unclear. They are usually treated in the same way as in patients without HIV infection. However, risk of infections with immunosuppression in HIV-infected patients has to be weighed up against the potential benefits. Agents such as sulphasalazine and hydroxychloroquine, which are not con- sidered to be particularly immunosuppressive, can be used. There is a consensus that treatment with more potent immunosuppressive agents such as methotrexate and anti-TNFα therapies can also be con- sidered in patients with well controlled HIV infection, such as those with undetectable viral load and CD4 count of more than 200. Diffuse infiltrative lymphocytosis syndrome (DILS) can mimic Sjögren's syndrome and presents with massive parotid gland swelling, sicca symptoms, lymphadenopathy, and arthralgias. Lymphocytic infiltration of other organs can lead to polymyositis, renal tubular acidosis, hepatitis, interstitial pneumonitis, and peripheral neur- opathy. DILS has been reported in up to 4% of HIV-infected patients, although the introduction of highly active antiretroviral therapy (HAART) has resulted in significant reduction in the in- cidence. Autoantibodies (Ro, La, and Rheumatoid factor) typically seen in patients with Sjögren's syndrome are absent in patients with DILS. Treatment is usually symptomatic and aimed at improving sicca symptoms. Immunosuppressive treatment may be required for those with pulmonary or renal involvement. Following the initiation of highly active antiretroviral therapy in HIV-infected individuals about 10–30% of patients develop para- doxical deterioration of clinical status due to worsening of pre- existing infectious processes (the pre-existing infections may have been previously diagnosed and treated, or they may be subclinical). This phenomenon is called immune reconstitution inflammatory syndrome (IRIS), which is attributable to the rapid recovery of the immune system following introduction of highly active antiretro- viral therapy, resulting in the host regaining capacity to mount an inflammatory response to a pre-existing infection. In addition to the inflammatory response to opportunistic infections affecting the musculoskeletal system, IRIS can lead to the development of new rheumatic autoimmune disorders and reactivation of pre-existing autoimmune disorders. The reported disorders include rheumatoid arthritis, systemic lupus erythematosus, and sarcoidosis. Depending on the severity of symptoms these autoimmune conditions, treat- ment with corticosteroids may be warranted, especially in those with severe and persistent disease. Investigations In a patient presenting with arthralgia/arthritis there is no consensus on routine testing for viral infections as the possible aetiology when a specific viral infection is not suspected. Clinical clues, patient risk factors and local epidemiology may suggest a possible viral aetiology behind arthralgia/arthritis and prompt further investigation. Serologic testing using immunoassay for both IgM and IgG antibodies to viral components is the commonly used method. The testing must be directed against the specific viruses suspected to be involved, based upon both epidemiologic data and clinical features. Direct testing of serum for viral specific nucleic acid by polymerase chain reaction (PCR) can also be performed. Several viruses have been isolated from joint fluid or synovium in individuals with clinical signs of arthritis, but viral isolation from joint tissue has very little role in routine clinical practice due to cost, difficulty in isolating the virus, and because only a small number of viruses directly infect the joint.

19.7 Infection and arthritis 4463 Management Since most viral arthritides are self-limiting and of short duration, treatment is primarily aimed at relieving symptoms with analgesic agents (e.g. acetaminophen) and nonsteroidal anti-inflammtory drugs. In general there is no need for specific antiviral therapy, al- though the underlying condition may require specific treatment (e.g. hepatitis B, hepatitis C, HIV). Immunosuppressants and gluco- corticoids are of limited value and generally not required, other than in those very few who develop chronic arthritis. FURTHER READING Coakley G, et al. (2006). BSR & BHPR, BOA, RCGP and BSAC guidelines for management of the hot

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