Diagnosis

Diagnosis

Clinical - Diagnosis is predominantly clinical with confirmation using other tests, as outlined below . Biomarkers Raised inflammatory markers (erythrocyte sedimentation rate [ESR], C-reactive protein [CRP] and white cell count [WCC]) are characteristic of acute infection, but they are neither su ffi ciently sensitive nor su ffi ciently specific to rule infection in or out. Recently , new synovial fluid markers (α -defensin and calprotectin) have shown high accuracy in diagnosis of PJI. Imaging Plain radiographs can demonstrate dead bone, periosteal reaction, involucrum formation and loosening of implants. However, they are often normal in the first few days of infec - tion. A normal radiograph does not exclude infection. Over time, radiographs will show progressive implant loosening, bone lysis or sequestration in chronic osteomyelitis. Ultrasonography is ideal for identifying soft-tissue collec - tions and joint e ff usions and can be used to guide bone biopsy and aspiration. Computed tomography (CT) scans are helpful in assessing bone union of infected fractures. Small sequestra and cortical erosions are best seen with CT and these scans can be used to plan surgery for excision of dead bone (Figure 43.3a). Isotope bone scans are of very limited value as they are non-specific and give no information that may guide diagno 18 sis or treatment. The combination of F-fluorodeoxyglucose 18 positron emission tomography (FDG-PET) with a CT scan allows localisation of active infection in chronic osteomyelitis 18 and may facilitate planning of surgery . FDG-PET/CT is not specific to infection and so cannot reliably distinguish infective from aseptic loosening around implants. Magnetic resonance imaging (MRI) scanning is the investi gation of choice, in the absence of metal implants. It is highly sensitive and specific, showing all components of the disease (Figure 43.3b). However, it can overestimate the extent of infection when ther e is widespread reactive oedema. α β Microbiological diagnosis Good treatment starts with a reliable microbiological diagno - sis. Superficial swabs from wounds or spontaneously draining - pus are unreliable. Cultures from these do not reflect the pathogens in the bone. Microbiological samples may be falsely negative if antibiotics are given first. Synovial tissue samples are particularly important in producing a higher diagnostic yield for infection with mycobacteria or fungi. In chronic infections, particularly those involving pros - thetic material, multiple biopsy samples are needed. It is rec - - ommended to take at least five tissue samples; each one with a separate, sterile instrument. Samples should be promptly transferred to the laboratory with clinical details of the infec - tion. Cultur e should be maintained for at least 10 days in mus - culoskeletal infections to allow identification of slow-growing

Classi /f_i cation of Group(s) of organisms infection Gram positive Staphylococci Streptococci Cutibacteria Enterococci Gram negative Enterobacteriaceae Pseudomonas spp. Kingella kingae Haemophilus spp. Neisseria spp. Others Fungi, especially Candida spp. Mycobacterium tuberculosis Atypical mycobacteria (Mycobacterium marinum, Mycobacterium ulcerans) Mixed Any combination of the above organisms ‘Culture negative’ No growth from cultures, but diagnosis of infection made on clinical/radiological/ histopathological grounds GI, gastrointestinal; HIV, human immunode /f_i ciency virus. Examples of speci /f_i c organisms and context in which infection

occurs Staphylococcus aureus (commonest across all settings) Coagulase-negative staphylococci (common in implant-associated infection) -Haemolytic streptococci, including Streptococcus pneumoniae, Streptococcus milleri group and Streptococcus viridans (in implant-associated infection) -Haemolytic streptococci (e.g. Streptococcus pyogenes, Streptococcus agalactiae) Increasingly recognised in implant-associated infection and septic arthritis of the shoulder Common in diabetic foot infection and chronic osteomyelitis Escherichia coli (especially at extremes of age) Klebsiella spp. Salmonella spp. (particularly associated with sickle cell disease) Associated with diabetic foot infections, osteomyelitis underlying chronic wounds/ulcers, patients heavily exposed to a hospital environment and/or prior antibiotics Common cause of septic arthritis in children under 4 years Haemophilus in /f_l uenzae (consider in non-immunised children) Neisseria meningitidis Neisseria gonorrhoeae (consider risk factors for sexually transmitted infection) Cause infection in immunocompromised and/or heavily antibiotic-exposed hosts. Common after prolonged use of negative-pressure wound therapy Present without pulmonary disease Geographical distribution Common with HIV May be a component of disseminated infection in HIV-infected patients; also cause post-surgical infection in immunocompetent hosts More common after trauma, recurrent surgery and with poor wound healing and sinuses, or resulting from contiguous spread from an infected source (e.g. skin, GI tract) Most common in patients who have had recent antimicrobial exposure prior to surgical sampling

organisms such as C. acnes . A positive tissue diagnosis is con firmed when phenotypically identical organisms are cultured from at least two of the five tissue samples. A single positive culture may suggest infection. It is also possible to culture organisms from remo implants that have been subjected to ultrasonic vibration to disrupt biofilm (sonication). Summary box 43.3 Principles of diagnosis /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF The histological diagnosis of infection (rather than other sources of inflammation) depends on identifying organisms on a Gram stain or the presence of a neutrophilic infiltrate. Histology can directly diagnose tuberculosis and atypical mycobacterial osteomyelitis (caseating and non-caseating granulomas), actinomycosis and fungal hyphae. The presence of five or more polymorphonuclear neutrophils per high- powered field is diagnostic in fracture-related joint infections and PJIs. Histology is valuable in confirming the presence of infection in culture-negative cases.

(b) Figure 43.3 (a) T ransverse computed tomography scan of the femur with a central sequestrum, sinus and cortical bone erosion. (b) netic resonance imaging scan of the same femur with better resolution of the medullary infection and soft-tissue involvement. ESR and CRP are neither sensitive nor speci /f_i c in making a diagnosis of bone infection Plain radiographs may be normal in the early phase Ultrasonography is valuable for identifying /f_l uid/pus collections MRI is usually the investigation of choice Super /f_i cial swabs are of no value in identifying the organism causing deep infection If the patient is on antibiotics, cultures may be falsely negative Multiple biopsy specimens should be obtained to optimise microbiological diagnosis A neutrophil in /f_i ltrate on histology can con /f_i rm infection

Diagnosis

Aspiration and/or biopsy of intra-articular fluid or tissue will allow a Gram stain to be performed (although this is positive in only about one-third of infected cases). Culture of a causative organism

from synovial fluid is diagnostic (positive in 80–90%) but results are delayed by the time taken to grow and identify the organism in the laboratory . A high WCC in joint fluid 3 (e.g. 50–150 /uni00A0 000 cells/mm), with a neutrophil predominance (>90%), is characteristic of infection. However, other inflam - matory conditions can also cause a raised cell count, and crystals may be seen in infected joints as well as in gout or pseudogout. The limited sensitivity of direct microscopy and Gram stain and the time taken to obtain a positive culture should not delay early treatment for the infection. The decision to perform a sur gical washout and give antibiotics should be based on the clinical picture. Summary box 43.5 Presentation of septic arthritis /uni25CF /uni25CF). /uni25CF - /uni25CF -

Children may be toxic and febrile but adults may have only a low-grade fever Usually symptoms affect only one joint, often with pre-existing arthropathy The joint is swollen and held in a characteristic ‘position of comfort’ Any movement causes extreme pain

Diagnosis

Infection should be suspected in any patient with a leaking wound over an implant, unresolved pain or new pain around a previously pain-free implant. Routine blood tests may be - helpful in acute infection but are often falsely reassuring. The European Bone and Joint Infection Society (EBJIS) has produced diagnostic criteria for PJI. Infection is confirmed if there is a sinus communicating with the joint or there is a high synovial fluid WCC (>3000/µL), a positive microbiologi cal culture or positive histology (more than five polymorphs per high-power field) (Figure 43.5) Plain radiographs may show features of loosening of a chronically infected prosthesis, and ultrasound ma y identify associated collections. Nuclear scans cannot reliably distinguish aseptic loosening from PJI.

Infection unlikely All findings negative A B Clinical A Clear alternative reason for Clinical features implant dysfunction C-reactive protein B Laboratory • Leukocyte count ≤1500 Synovial fluid • PMN ≤65% • All cultures negative Microbiology • No growth on sonication Histology Negative C Radiology Negative 3-phase isotope Nuclear imaging bone scan Figure 43.5 The European Bone and Joint Infection Society de /f_i nition of prosthetic joint infection. CFU, colony-forming unit; CRP , C-reactive protein; HPF , high-power /f_i eld; PMN, polymorphonuclear cells. (Reproduced with permission from McNally MA, Sousa R, Wouthuyzen-Bakker M et al . Infographic: The EBJIS de /f_i nition of prosthetic joint infection: a practical guide for clinicians.

Diagnosis

- In the early phase (2–3 days) radiographs may be normal but MRI will show bone oedema and periosteal elevation. After 5–7 days, plain radiographs may show subtle abnormality with osteopenia and periosteal new bone formation. WCC and CRP are often elevated in the early phase. Treatment should not be delayed pending investigations. Diagnosis

Plain radiographs can delineate soft-tissue swelling, subperi - osteal reaction, bone destruction and sequestra. CT scans are good for cortical bone imaging (Figure 43.8). MRI is the imag - ing test of choice (see General principles of orthopaedic infection). Blood tests are often normal in chronic osteomy - e from deep elitis. Confirmation of the diagnosis is with cultur 18 surgical samples and histology . FDG-PET CT scanning can be helpful for surgical planning.

Figure 43.8 showing diaphyseal chronic osteomyelitis. clearly shows the sequestration of the lateral cortex and overlying new bone formation (involucrum).

Diagnosis

Clinical - Diagnosis is predominantly clinical with confirmation using other tests, as outlined below . Biomarkers Raised inflammatory markers (erythrocyte sedimentation rate [ESR], C-reactive protein [CRP] and white cell count [WCC]) are characteristic of acute infection, but they are neither su ffi ciently sensitive nor su ffi ciently specific to rule infection in or out. Recently , new synovial fluid markers (α -defensin and calprotectin) have shown high accuracy in diagnosis of PJI. Imaging Plain radiographs can demonstrate dead bone, periosteal reaction, involucrum formation and loosening of implants. However, they are often normal in the first few days of infec - tion. A normal radiograph does not exclude infection. Over time, radiographs will show progressive implant loosening, bone lysis or sequestration in chronic osteomyelitis. Ultrasonography is ideal for identifying soft-tissue collec - tions and joint e ff usions and can be used to guide bone biopsy and aspiration. Computed tomography (CT) scans are helpful in assessing bone union of infected fractures. Small sequestra and cortical erosions are best seen with CT and these scans can be used to plan surgery for excision of dead bone (Figure 43.3a). Isotope bone scans are of very limited value as they are non-specific and give no information that may guide diagno 18 sis or treatment. The combination of F-fluorodeoxyglucose 18 positron emission tomography (FDG-PET) with a CT scan allows localisation of active infection in chronic osteomyelitis 18 and may facilitate planning of surgery . FDG-PET/CT is not specific to infection and so cannot reliably distinguish infective from aseptic loosening around implants. Magnetic resonance imaging (MRI) scanning is the investi gation of choice, in the absence of metal implants. It is highly sensitive and specific, showing all components of the disease (Figure 43.3b). However, it can overestimate the extent of infection when ther e is widespread reactive oedema. α β Microbiological diagnosis Good treatment starts with a reliable microbiological diagno - sis. Superficial swabs from wounds or spontaneously draining - pus are unreliable. Cultures from these do not reflect the pathogens in the bone. Microbiological samples may be falsely negative if antibiotics are given first. Synovial tissue samples are particularly important in producing a higher diagnostic yield for infection with mycobacteria or fungi. In chronic infections, particularly those involving pros - thetic material, multiple biopsy samples are needed. It is rec - - ommended to take at least five tissue samples; each one with a separate, sterile instrument. Samples should be promptly transferred to the laboratory with clinical details of the infec - tion. Cultur e should be maintained for at least 10 days in mus - culoskeletal infections to allow identification of slow-growing

Classi /f_i cation of Group(s) of organisms infection Gram positive Staphylococci Streptococci Cutibacteria Enterococci Gram negative Enterobacteriaceae Pseudomonas spp. Kingella kingae Haemophilus spp. Neisseria spp. Others Fungi, especially Candida spp. Mycobacterium tuberculosis Atypical mycobacteria (Mycobacterium marinum, Mycobacterium ulcerans) Mixed Any combination of the above organisms ‘Culture negative’ No growth from cultures, but diagnosis of infection made on clinical/radiological/ histopathological grounds GI, gastrointestinal; HIV, human immunode /f_i ciency virus. Examples of speci /f_i c organisms and context in which infection occurs Staphylococcus aureus (commonest across all settings) Coagulase-negative staphylococci (common in implant-associated infection) -Haemolytic streptococci, including Streptococcus pneumoniae, Streptococcus milleri group and Streptococcus viridans (in implant-associated

infection) -Haemolytic streptococci (e.g. Streptococcus pyogenes, Streptococcus agalactiae) Increasingly recognised in implant-associated infection and septic arthritis of the shoulder Common in diabetic foot infection and chronic osteomyelitis Escherichia coli (especially at extremes of age) Klebsiella spp. Salmonella spp. (particularly associated with sickle cell disease) Associated with diabetic foot infections, osteomyelitis underlying chronic wounds/ulcers, patients heavily exposed to a hospital environment and/or prior antibiotics Common cause of septic arthritis in children under 4 years Haemophilus in /f_l uenzae (consider in non-immunised children) Neisseria meningitidis Neisseria gonorrhoeae (consider risk factors for sexually transmitted infection) Cause infection in immunocompromised and/or heavily antibiotic-exposed hosts. Common after prolonged use of negative-pressure wound therapy Present without pulmonary disease Geographical distribution Common with HIV May be a component of disseminated infection in HIV-infected patients; also cause post-surgical infection in immunocompetent hosts More common after trauma, recurrent surgery and with poor wound healing and sinuses, or resulting from contiguous spread from an infected source (e.g. skin, GI tract) Most common in patients who have had recent antimicrobial exposure prior to surgical sampling

organisms such as C. acnes . A positive tissue diagnosis is con firmed when phenotypically identical organisms are cultured from at least two of the five tissue samples. A single positive culture may suggest infection. It is also possible to culture organisms from remo implants that have been subjected to ultrasonic vibration to disrupt biofilm (sonication). Summary box 43.3 Principles of diagnosis /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF The histological diagnosis of infection (rather than other sources of inflammation) depends on identifying organisms on a Gram stain or the presence of a neutrophilic infiltrate. Histology can directly diagnose tuberculosis and atypical mycobacterial osteomyelitis (caseating and non-caseating granulomas), actinomycosis and fungal hyphae. The presence of five or more polymorphonuclear neutrophils per high- powered field is diagnostic in fracture-related joint infections and PJIs. Histology is valuable in confirming the presence of infection in culture-negative cases.

(b) Figure 43.3 (a) T ransverse computed tomography scan of the femur with a central sequestrum, sinus and cortical bone erosion. (b) netic resonance imaging scan of the same femur with better resolution of the medullary infection and soft-tissue involvement. ESR and CRP are neither sensitive nor speci /f_i c in making a diagnosis of bone infection Plain radiographs may be normal in the early phase Ultrasonography is valuable for identifying /f_l uid/pus collections MRI is usually the investigation of choice Super /f_i cial swabs are of no value in identifying the organism causing deep infection If the patient is on antibiotics, cultures may be falsely negative Multiple biopsy specimens should be obtained to optimise microbiological diagnosis A neutrophil in /f_i ltrate on histology can con /f_i rm infection

Diagnosis

Aspiration and/or biopsy of intra-articular fluid or tissue will allow a Gram stain to be performed (although this is positive in only about one-third of infected cases). Culture of a causative organism from synovial fluid is diagnostic (positive in 80–90%) but results are delayed by the time taken to grow and identify the organism in the laboratory . A high WCC in joint fluid 3 (e.g. 50–150 /uni00A0 000 cells/mm), with a neutrophil predominance (>90%), is characteristic of infection. However,

other inflam - matory conditions can also cause a raised cell count, and crystals may be seen in infected joints as well as in gout or pseudogout. The limited sensitivity of direct microscopy and Gram stain and the time taken to obtain a positive culture should not delay early treatment for the infection. The decision to perform a sur gical washout and give antibiotics should be based on the clinical picture. Summary box 43.5 Presentation of septic arthritis /uni25CF /uni25CF). /uni25CF - /uni25CF -

Children may be toxic and febrile but adults may have only a low-grade fever Usually symptoms affect only one joint, often with pre-existing arthropathy The joint is swollen and held in a characteristic ‘position of comfort’ Any movement causes extreme pain

Diagnosis

Infection should be suspected in any patient with a leaking wound over an implant, unresolved pain or new pain around a previously pain-free implant. Routine blood tests may be - helpful in acute infection but are often falsely reassuring. The European Bone and Joint Infection Society (EBJIS) has produced diagnostic criteria for PJI. Infection is confirmed if there is a sinus communicating with the joint or there is a high synovial fluid WCC (>3000/µL), a positive microbiologi cal culture or positive histology (more than five polymorphs per high-power field) (Figure 43.5) Plain radiographs may show features of loosening of a chronically infected prosthesis, and ultrasound ma y identify associated collections. Nuclear scans cannot reliably distinguish aseptic loosening from PJI.

Infection unlikely All findings negative A B Clinical A Clear alternative reason for Clinical features implant dysfunction C-reactive protein B Laboratory • Leukocyte count ≤1500 Synovial fluid • PMN ≤65% • All cultures negative Microbiology • No growth on sonication Histology Negative C Radiology Negative 3-phase isotope Nuclear imaging bone scan Figure 43.5 The European Bone and Joint Infection Society de /f_i nition of prosthetic joint infection. CFU, colony-forming unit; CRP , C-reactive protein; HPF , high-power /f_i eld; PMN, polymorphonuclear cells. (Reproduced with permission from McNally MA, Sousa R, Wouthuyzen-Bakker M et al . Infographic: The EBJIS de /f_i nition of prosthetic joint infection: a practical guide for clinicians.

Diagnosis

- In the early phase (2–3 days) radiographs may be normal but MRI will show bone oedema and periosteal elevation. After 5–7 days, plain radiographs may show subtle abnormality with osteopenia and periosteal new bone formation. WCC and CRP are often elevated in the early phase. Treatment should not be delayed pending investigations. Diagnosis

Plain radiographs can delineate soft-tissue swelling, subperi - osteal reaction, bone destruction and sequestra. CT scans are good for cortical bone imaging (Figure 43.8). MRI is the imag - ing test of choice (see General principles of orthopaedic infection). Blood tests are often normal in chronic osteomy - e from deep elitis. Confirmation of the diagnosis is with cultur 18 surgical samples and histology . FDG-PET CT scanning can be helpful for surgical planning.

Figure 43.8 showing diaphyseal chronic osteomyelitis. clearly shows the sequestration of the lateral cortex and overlying new bone formation (involucrum).

Diagnosis

Clinical - Diagnosis is predominantly clinical with confirmation using other tests, as outlined below . Biomarkers Raised inflammatory markers (erythrocyte sedimentation rate [ESR], C-reactive protein [CRP] and white cell count [WCC]) are characteristic of acute infection, but they are neither su ffi ciently sensitive nor su ffi ciently specific to rule infection in or out. Recently , new synovial fluid markers (α -defensin and calprotectin) have shown high accuracy in diagnosis of PJI. Imaging Plain radiographs can demonstrate dead bone, periosteal reaction, involucrum formation and loosening of implants. However, they are often normal in the first few days of infec - tion. A normal radiograph does not exclude infection. Over time, radiographs will show progressive implant loosening, bone lysis or sequestration in chronic osteomyelitis. Ultrasonography is ideal for identifying soft-tissue collec - tions and joint e ff usions and can be used to guide bone biopsy and aspiration. Computed tomography (CT) scans are helpful in assessing bone union of infected fractures. Small sequestra and cortical erosions are best seen with CT and these scans can be used to plan surgery for excision of dead bone (Figure 43.3a). Isotope bone scans are of very limited value as they are non-specific and give no information that may guide diagno 18 sis or treatment. The combination of F-fluorodeoxyglucose 18 positron emission tomography (FDG-PET) with a CT scan allows localisation of active infection in chronic osteomyelitis 18 and may facilitate planning of surgery . FDG-PET/CT is not specific to infection and so cannot reliably distinguish infective from aseptic loosening around implants. Magnetic resonance imaging (MRI) scanning is the investi gation of choice, in the absence of metal implants. It is highly sensitive and specific, showing all components of the disease (Figure 43.3b). However, it can overestimate the extent of infection when ther e is widespread reactive oedema. α β Microbiological diagnosis Good treatment starts with a reliable microbiological diagno - sis. Superficial swabs from wounds or spontaneously draining - pus are unreliable. Cultures from these do not reflect the pathogens in the bone. Microbiological samples may be falsely negative if antibiotics are given first. Synovial tissue samples are particularly important in producing a higher diagnostic yield for infection with mycobacteria or fungi. In chronic infections, particularly those involving pros - thetic material, multiple biopsy samples are needed. It is rec - - ommended to take at least five tissue samples; each one with a separate, sterile instrument. Samples should be promptly transferred to the laboratory with clinical details of the infec - tion. Cultur e should be maintained for at least 10 days in mus - culoskeletal infections to allow identification of slow-growing

Classi /f_i cation of Group(s) of organisms infection Gram positive Staphylococci Streptococci Cutibacteria Enterococci Gram negative Enterobacteriaceae Pseudomonas spp. Kingella kingae Haemophilus spp. Neisseria spp. Others Fungi, especially Candida spp. Mycobacterium tuberculosis Atypical mycobacteria (Mycobacterium marinum, Mycobacterium ulcerans) Mixed Any combination of the above organisms ‘Culture negative’ No growth from cultures, but diagnosis of infection made on clinical/radiological/ histopathological grounds GI, gastrointestinal; HIV, human immunode /f_i ciency virus. Examples of speci /f_i c organisms and context in which infection occurs Staphylococcus aureus (commonest across all settings) Coagulase-negative staphylococci (common in implant-associated infection) -Haemolytic streptococci, including Streptococcus pneumoniae, Streptococcus milleri group and Streptococcus viridans (in implant-associated infection) -Haemolytic streptococci (e.g. Streptococcus pyogenes, Streptococcus agalactiae) Increasingly recognised in implant-associated infection and septic arthritis of the shoulder Common in diabetic foot infection and chronic osteomyelitis Escherichia coli (especially at extremes of age)

Klebsiella spp. Salmonella spp. (particularly associated with sickle cell disease) Associated with diabetic foot infections, osteomyelitis underlying chronic wounds/ulcers, patients heavily exposed to a hospital environment and/or prior antibiotics Common cause of septic arthritis in children under 4 years Haemophilus in /f_l uenzae (consider in non-immunised children) Neisseria meningitidis Neisseria gonorrhoeae (consider risk factors for sexually transmitted infection) Cause infection in immunocompromised and/or heavily antibiotic-exposed hosts. Common after prolonged use of negative-pressure wound therapy Present without pulmonary disease Geographical distribution Common with HIV May be a component of disseminated infection in HIV-infected patients; also cause post-surgical infection in immunocompetent hosts More common after trauma, recurrent surgery and with poor wound healing and sinuses, or resulting from contiguous spread from an infected source (e.g. skin, GI tract) Most common in patients who have had recent antimicrobial exposure prior to surgical sampling

organisms such as C. acnes . A positive tissue diagnosis is con firmed when phenotypically identical organisms are cultured from at least two of the five tissue samples. A single positive culture may suggest infection. It is also possible to culture organisms from remo implants that have been subjected to ultrasonic vibration to disrupt biofilm (sonication). Summary box 43.3 Principles of diagnosis /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF The histological diagnosis of infection (rather than other sources of inflammation) depends on identifying organisms on a Gram stain or the presence of a neutrophilic infiltrate. Histology can directly diagnose tuberculosis and atypical mycobacterial osteomyelitis (caseating and non-caseating granulomas), actinomycosis and fungal hyphae. The presence of five or more polymorphonuclear neutrophils per high- powered field is diagnostic in fracture-related joint infections and PJIs. Histology is valuable in confirming the presence of infection in culture-negative cases.

(b) Figure 43.3 (a) T ransverse computed tomography scan of the femur with a central sequestrum, sinus and cortical bone erosion. (b) netic resonance imaging scan of the same femur with better resolution of the medullary infection and soft-tissue involvement. ESR and CRP are neither sensitive nor speci /f_i c in making a diagnosis of bone infection Plain radiographs may be normal in the early phase Ultrasonography is valuable for identifying /f_l uid/pus collections MRI is usually the investigation of choice Super /f_i cial swabs are of no value in identifying the organism causing deep infection If the patient is on antibiotics, cultures may be falsely negative Multiple biopsy specimens should be obtained to optimise microbiological diagnosis A neutrophil in /f_i ltrate on histology can con /f_i rm infection

Diagnosis

Aspiration and/or biopsy of intra-articular fluid or tissue will allow a Gram stain to be performed (although this is positive in only about one-third of infected cases). Culture of a causative organism from synovial fluid is diagnostic (positive in 80–90%) but results are delayed by the time taken to grow and identify the organism in the laboratory . A high WCC in joint fluid 3 (e.g. 50–150 /uni00A0 000 cells/mm), with a neutrophil predominance (>90%), is characteristic of infection. However, other inflam - matory conditions can also cause a raised cell count, and crystals may be seen in infected joints as well as in gout or pseudogout. The limited sensitivity of direct microscopy and Gram stain and the time taken to obtain a positive culture should not delay early treatment for the

infection. The decision to perform a sur gical washout and give antibiotics should be based on the clinical picture. Summary box 43.5 Presentation of septic arthritis /uni25CF /uni25CF). /uni25CF - /uni25CF -

Children may be toxic and febrile but adults may have only a low-grade fever Usually symptoms affect only one joint, often with pre-existing arthropathy The joint is swollen and held in a characteristic ‘position of comfort’ Any movement causes extreme pain

Diagnosis

Infection should be suspected in any patient with a leaking wound over an implant, unresolved pain or new pain around a previously pain-free implant. Routine blood tests may be - helpful in acute infection but are often falsely reassuring. The European Bone and Joint Infection Society (EBJIS) has produced diagnostic criteria for PJI. Infection is confirmed if there is a sinus communicating with the joint or there is a high synovial fluid WCC (>3000/µL), a positive microbiologi cal culture or positive histology (more than five polymorphs per high-power field) (Figure 43.5) Plain radiographs may show features of loosening of a chronically infected prosthesis, and ultrasound ma y identify associated collections. Nuclear scans cannot reliably distinguish aseptic loosening from PJI.

Infection unlikely All findings negative A B Clinical A Clear alternative reason for Clinical features implant dysfunction C-reactive protein B Laboratory • Leukocyte count ≤1500 Synovial fluid • PMN ≤65% • All cultures negative Microbiology • No growth on sonication Histology Negative C Radiology Negative 3-phase isotope Nuclear imaging bone scan Figure 43.5 The European Bone and Joint Infection Society de /f_i nition of prosthetic joint infection. CFU, colony-forming unit; CRP , C-reactive protein; HPF , high-power /f_i eld; PMN, polymorphonuclear cells. (Reproduced with permission from McNally MA, Sousa R, Wouthuyzen-Bakker M et al . Infographic: The EBJIS de /f_i nition of prosthetic joint infection: a practical guide for clinicians.

Diagnosis

- In the early phase (2–3 days) radiographs may be normal but MRI will show bone oedema and periosteal elevation. After 5–7 days, plain radiographs may show subtle abnormality with osteopenia and periosteal new bone formation. WCC and CRP are often elevated in the early phase. Treatment should not be delayed pending investigations. Diagnosis

Plain radiographs can delineate soft-tissue swelling, subperi - osteal reaction, bone destruction and sequestra. CT scans are good for cortical bone imaging (Figure 43.8). MRI is the imag - ing test of choice (see General principles of orthopaedic infection). Blood tests are often normal in chronic osteomy - e from deep elitis. Confirmation of the diagnosis is with cultur 18 surgical samples and histology . FDG-PET CT scanning can be helpful for surgical planning.

Figure 43.8 showing diaphyseal chronic osteomyelitis. clearly shows the sequestration of the lateral cortex and overlying new bone formation (involucrum).

Revision #1
Created 2025-12-31 15:16:28 UTC by Omar Ayman
Updated 2025-12-31 15:16:28 UTC by Omar Ayman