Osteonecrosis, osteochondrosis, and osteochondriti

Osteonecrosis, osteochondrosis, and osteochondritis dissecans 4703

ESSENTIALS Osteonecrosis Osteonecrosis is ischaemia of bone caused by a range of condi- tions, including trauma, which cause intravascular or extravascular obstruction of blood flow to bone. Many cases are associated with pro-thrombotic conditions (e.g. sickle cell disease, antiphospholipid syndrome); Osteonecrosis can be asymptomatic, but if progressive often leads to secondary (adjacent) joint destruction. Diagnosis is made by magnetic resonance imaging. Aside from treatment of any underlying cause, treatment options include analgesics, vasodila- tors, and surgery. Osteochondritides The osteochondritides are trauma-induced focal disturbances of cartilage, either in joints (articular) or at a periarticular epiphyseal plate or at a tendon or ligament insertion (apophysis/enthesis). Lesions typically occur in active children and adolescents and re- spond to biomechanical modifications and pain relief. Articular osteochondrosis which develops into a ‘dissecans’
lesion often starts as a small area of bone compression, which
can progress to a partially or fully detached osteochondral frag- ment. Such lesions require mechanical protection and careful monitoring, but when severe may be successfully treated with surgery. Osteonecrosis Introduction Osteonecrosis is regional ischaemic skeletal injury, which can be caused by trauma, drugs (e.g. glucocorticoids), or systemic conditions—metabolic, haematological or endocrine (e.g. sickle cell disease, antiphospholipid syndrome). If severe and/or

prolonged, is chaemia can cause skeletal cell death leading to necrotic bone, which can compromise regional skeletal integrity and strength and lead to fracture, damage to adjacent cartilage, and deformity. Conversely, osteonecrosis can be symptomatically silent and persist, with no or minimal symptoms, for years in some cases. Aetiology Both direct mechanical interruption to the blood supply and systemic factors affecting blood delivery to bone (Box 20.5.1) contribute to the development of osteonecrosis. A genetic con tribution to is suggested by occurrence in twins and case clus ters in families (e.g. idiopathic osteonecrosis of the femoral head). Most genetic association studies have pointed to polymorphisms in genes involved in coagulation and fibrinolytic processes. Positive associations with osteonecrosis have been made with: the presence of factor V Leiden mutation (G1691A), homozygosity for the 4G allele of plasminogen activating inhibitor-1 (PAI-1), VEGF-634G/C, and allele 4a of the endothelial nitric oxide syn thase gene (idiopathic hip osteonecrosis). There is debate as to whether there is a genetic influence to protection against osteo necrosis in some populations and with reference to glucocorticoid risk. A mutation in COL2A2, dominantly inherited, has been found in three families with osteonecrosis (type 2 collagen is the major structural protein in cartilage). In a series of patients with osteonecrosis, 83% had one or more positive results for a range of procoagulant disorders. Of these, resistance to activated protein C and anticardiolipin antibodies were the most common, affecting 50% and 26.7% of the study group versus 7.5% and 1% of healthy controls, respectively. Epidemiology Orthopaedic case estimates over 15 years ago suggested there were 10 000–20 000 new cases of adult osteonecrosis annually in the United States of America. Current estimates for osteonecrosis of the femoral head alone are that 20 000 to 30 000 new patients are diagnosed with hip osteonecrosis annually; accounting for ap proximately 10% of the 250 000 total hip arthroplasties done an nually in the United States. The actual incidence will likely now be increased given mild cases may be symptomatically silent. Males are more commonly affected (3–4:1), and most patients are under 50 years of age. Spontaneous osteonecrosis of the knee, which particularly affects women over the age of 50 years, has 20.5 Osteonecrosis, osteochondrosis,
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SECTION 20 Disorders of the skeleton 4704 a prevalence of over 9% in imaging studies in women aged over 65 years. In osteonecrosis of the femoral head there are bilat eral lesions in 75%. Some other diseases-specific frequencies are shown in Box 20.5.1. The incidence of bisphosphonate-related osteonecrosis of the jaw is low. Reports of osteonecrosis of the jaw submitted to manufacturers indicates a reporting rate of less than 1 per 100 000 patient treatment years. Most cases of osteonecrosis of the jaw occur in patients suffering from malignant diseases (n = 117; 92.8% in one recent series). In this series, the commonest ma lignancies were breast cancer (n = 57; 45.2%), multiple myeloma (n = 37; 29.4%), and prostate cancer (n = 13; 10.3%). In risedronate clinical trials in osteoporosis patients, there have been no cases of osteonecrosis of the jaw reported in ap proximately 20 000 patients studied for up to three years. Clinical data from the HORIZON-PFT study, which evaluated once- yearly zoledronic acid 5 mg in 7736 women with postmenopausal osteoporosis over three years showed no difference in the inci dence of osteonecrosis of the jaw between the treatment and placebo group, with one case reported in each group. In a German country-wide study in cancer patients, where IV bisphosphonates are primarily used as adjunct therapy in multiple myeloma and metastatic breast cancer (in high cumulative dose regimes) the reported rate is 95 per 100 000 patient treatment years. In an other retrospective study of 200 oncology patients osteonecrosis of the jaw incidence rate was 1 in 28 patients per year of treat ment. Osteonecrosis of the jaw risk with zoledronic acid was five fold

higher than that with pamidronate or ibandronic acid. The risk of osteonecrosis of the jaw also increased by 40-fold after dental surgery. Pathogenesis Ischaemia occurs after mechanical interruption of blood delivery, nontraumatic intravascular occlusion, thrombosis, cholesterol, ex panding nitrogen bubbles, or other mechanisms that lead to critical ischaemia. With regard to the latter the following may be rele vant: the transition with age from well vascularized red, to poorly vascularized fatty, marrow; an increase in adipocytes in medullary spaces (e.g. secondary to chronic alcohol excess) or adipocyte size (e.g. fat accumulation with glucocorticoids/Cushing's) increasing pressure in sinusoids. Following ischaemia, deposition of vascularized connective tissue can accumulate at the interface between necrotic and normal bone. Calcification can follow and any necrotic tissue remains as an 'is land' of inviable bone. The pathogenesis of osteonecrosis of the jaw associated with high-dose or long-term bisphosphonates use is not fully known but is thought to be a combination of the long-term effects of bisphosphonate-induced low bone turnover, periodontal bacterial colonization (actinomyces is common) with suppurative inflam mation, antiangiogenic effects of the drug and any 'osteonecrosis- associated' comorbidity or factors (see Box 20.5.1). Osteonecrosis of the jaw also appears to be associated with VEGF gene polymorphism in Asian populations. Clinical features Local skeletal features include pain or discomfort, though osteonecrosis can exist without causing symptoms, often for long periods of time. Pain evolution may denote the onset of subchondral bone collapse. Pain quality is often deep-seated, un remitting, and changes little with posture or movement. If sec ondary adjacent joint damage evolves and progresses then features of mechanical joint disease will evolve—causing pain and discom fort on movement, stiffness, swelling, and a functional impact accordingly. Both symptoms arising directly from osteonecrotic bone and secondary joint symptoms however, are not specific. There may be tenderness on palpating over osteonecrotic bone and signs of joint effusion if the lesion has affected the adjacent joint causing cartilage loss or microfracture. Passive joint move ment clarifies the degree of pain from intra-articular pathology associated with the lesion. Differential diagnosis Deep-seated persistent, unmodifyable skeletal pain with movement also raises the possibility of fracture, skeletal tumours, osteomyelitis, and Paget's disease of bone, as well as osteonecrosis. Distinguishing Box 20.5.1 Conditions associated with osteonecrosis Trauma: Fractures and fracture-dislocations, Legg–Calvé–Perthe dis- ease, orthopaedic procedures Glucocorticoids (GCs), Cushing's syndrome and disease: Osteonecrosis is associated with acute repeated high-dose pulse steroid and high- cumulative prednisolone dose/steroid dose equivalent. GCs cause hypertrophy and hyperplasia of marrow fat cells and lipid deposition of osteocytes possibly by diverting marrow mesenchymal stem cells to adi- pocytes vs. osteogenic cell differentiation Alcohol excess: Risk increases greater than threefold in those con- suming 40 units per week or more. Drugs (other): Cocaine; bisphosphonates and denosumab (mono- clonal antibody to RANKL) both causing osteonecrosis of the jaw; oral contraceptives (rare); protease inhibitors; thalidomide Gaucher disease: In the total population of 5894 ICGG Gaucher Disease Registry patients, 544 experienced at least one episode of osteonecrosis; associated with anaemia Systemic lupus erythematosus: Osteonecrosis was reported in up to 27% of patients taking GCs, often multifocal; the risk is higher if there is hypertriglyceridemia Solid organ and haematopoietic cell transplantation: Likely multifactorial risks, but mainly associated with GC use Sickle cell disease (SCD): Prevalence very high most commonly humeral head (28–48% prevalence); osteonecrosis of the femoral head is most prevalent in patients with SCD-SS α-thalassemia; Pro- thrombotic risk: Overall up to 50% of cases of multifocal osteo- necrosis are associated with identifiable pro-thrombotic conditions (e.g. Factor V Leiden; PA1–1; antiphospholipid syndrome); Dysbaric osteonecrosis (caisson disease): Predominately occurs in fem- oral head and proximal

humerus in divers, caisson, and tunnel workers. Avoided in divers by assiduous decompression; incidence lowest in mili- tary divers, highly monitored HIV: The use of protease inhibitors is a significant risk, possibly through causing hyperlipidemia. HIV-associated antiphospholipid syndrome may be a contributory cause Malignancy: Childhood leukemias: poor prognosis associated with lesions occupying more than 30% of the femoral head volume; 80% of hips collapsed in under two years of diagnosis and 50% required arthroplasty Other probable associations: Pancreatitis, hyperlipidaemia, diabetes mellitus, pregnancy, hyperuricaemia/gout

20.5 Osteonecrosis, osteochondrosis, and osteochondritis dissecans 4705 fracture from osteonecrosis where there has been trauma is of ob vious importance, though following trauma, presentation with frac ture is likely to be much earlier than with osteonecrosis. Paget's is extremely rare in young adults and children; osteomyelitis is often associated with features of preceding generalized infection and con current systemic symptoms. Investigations—imaging Generally, the diagnosis of osteonecrosis is made on careful in terpretation of imaging investigations. In most cases, radiographs alone are insufficient to make the diagnosis though they should be routinely requested. Though there are characteristic computed tom ography (CT) and bone scintigraphy appearances, the gold standard for making a diagnosis is magnetic resonance (MR) imaging. It is well-recognized that imaging abnormalities can precede symptoms though this is more likely with MR and bone scintigraphy than with radiographs. Moderately advanced osteonecrosis can be detected with radio graphs with some specific features (e.g. rim of sclerosis may become visible with a radiolucent subchondral crescent—necrotic bone). Progression of the lesion is characterized by collapse of subchondral bone and osteoarthritis of the adjacent joint. On MR, T1 images typically display linear patterns of ab normal low signal and a 'double-line sign' on T2 or fat suppressed sequences, which depicts a high signal intensity reparative inter face of vascular reactive bone adjacent to necrotic subchondral bone. Lines are often serpiginous and generally increase in ap parent volume with progression of the lesion. Such features are highly suggestive of osteonecrosis in the absence of bone expan sion and adjacent soft-tissue lesion extension (e.g. as often occurs with tumour and infection). Bone scintigraphy appearances vary with the age of the lesion. Initially there may be prominent photopenic areas but later there is intense and sometimes patchy radiopharmaceutical accumulation, sometimes surrounding a photopenic area, though if the ischaemic area is small then poor resolution often dictates there is just intense radiopharmaceutical localization seen. It is worth considering in some cases to proactively obtain imaging of other skeletal sites. There is a bilaterality in a sizeable minority of patients with osteonecrosis of the femoral head and in some patients with systemic factors disease may be polyostotic. Radiological staging of osteonecrosis of the femoral head has been established in detail combining clinical and imaging features (e.g. after Ficat see Table 20.5.1) though in practical terms, the Steinberg or Association Research Circulation Osseous (ARCO) systems are simpler and adaptable to use in day to day practice (Table 20.5.2). Investigations—laboratory Laboratory investigations should aim to help discriminate the cause of focal skeletal lesions (osteonecrosis, infection or malignancy) and rule out or establish any metabolic, endocrine, systemic inflamma tory or autoimmune conditions: • Haematological—FBC/CBC, ESR, or PV, pro-thrombotic screen including testing for lupus anticoagulant and anticardiolipin antibodies; • Biochemistry—renal, urate, lipid screen, C-reactive protein, serum and urine protein electrophoresis, liver and bone profile tests including parathyroid hormone and 25-hydroxyvitamin D, fasting glucose; • Immunological—Antinuclear antibody (ANA)/extractable nuclear antigens (ENAs), complement studies, immunoglobulins; • Microbiology—blood cultures if systemically unwell; consider HIV

testing. Treatment—general considerations Initial management steps should include pain control, addressing remediable underlying systemic causes/associated disease, agreeing and planning what amount of weight or load bearing of the affected bone is permissible, patient education about osteonecrosis, and factoring in the patient's view on treatment objectives in full clinical context. In the absence of data from robust controlled studies of treatment the management of osteonecrosis (whether considering osteonecrosis of the femoral head, osteonecrosis of the jaw or any other site/cause for osteonecrosis), is often based on the stage of the lesion and degree of effect of the adjacent joint, which may be: • Early/asymptomatic disease with reversible cause or repair or revascularization possible before the collapse of the subchondral bone. Notably also, small lesions detected with MR typically do not progress and conservative measures are appropriate. • Late disease with subchondral bone collapse where arthroplasty is considered. Table 20.5.1 Practical staging of osteonecrosis of the femoral head (after Ficat and based on consensus of the subcommittee of The Nomenclature of the International Association on Bone Circulation and Bone Necrosis) Stage Findings 0 Patient asymptomatic, radiograph normal, histology shows some osteonecrosis I Patients may have symptoms; CT and radiographs unremarkable; osteonecrosis is considered likely from MR and/or bone scintigraphy; histology
is abnormal II Patient is symptomatic; radiographs abnormal (osteopenia, osteosclerosis, cysts); subchondral radiolucency is absent; MR findings diagnostic III Patient symptomatic; radiographic signs include subchondral lucency (crescent sign) and collapse; shape of femoral head preserved on
CT/radiographs; subclassification on extent of crescent of % of articular surface (IIIa <15%; IIIb 15–30%; IIIc >30%) IV Flattening or collapse of femoral head present; joint space may be irregular; CT more sensitive than radiographs; subclassified on the extent of collapsed surface V Radiograph findings include narrowing of the joint space, osteoarthritis with sclerosis of acetabulum, and marginal osteophytes VI Findings include extensive destruction of the femoral head and joint

 Treatment—osteonecrosis of the femoral head Early medical treatment should include a period of nonweight- bearing (e.g. four to eight weeks). Effectiveness of non-weight-bearing may be blunted by poor adherence in nonsymptomatic patients. Though bisphosphonates have been reported (in anecdotal and small series) to be therapeutic in early osteonecrosis of the femoral head, a recent metanalysis showed study quality is variable and overall that bisphosphonates are not effective. Data were derived from five studies (of 329 patients with 921 patient-years of follow-up). There is no orthopaedic consensus on the optimal surgical treat ment of osteonecrosis of the femoral head. Core decompression involves removing bone from the medullary cavity or drilling mul tiple smaller holes through the bone surface. The cavity may be then filled with a vascularized fibular graft or by nonvascularized cortical bone. Osteotomy attempts to shift skeletal loading from the nec rotic segment but subsequent joint replacement is technically more difficult. Limited joint replacement (hemi-resurfacing) preserves the bone for later arthroplasty and is an option for femoral head collapse in younger patients. Skeletal stem cells combined with impaction bone grafting is a novel treatment translated to the treatment of osteonecrosis of the femoral head Treatment—osteonecrosis of the jaw Review of published cases (almost 5000 cases 2003–14) suggests minimally invasive surgical treatment was the treatment most used. Adjunctive treatments included laser, growth factors, antibiotics, hyperbaric oxygen, and ozone. There are no randomized controlled studies, studies do not use outcomes consistently, and there is a var iety of study designs. Clinical trials with larger samples are required to provide sufficient information for each treatment. Studies have

not re vealed that affected patients have any obvious genetic predisposition. Clearly any antiresorptive osteoporosis treatment needs to be discontinued. Increasing bone turnover using teriparatide or abaloparatide is an untested but reasonable option to consider in early osteonecrosis of the jaw lesions. Treatment—sickle cell disease and pro-thrombotic conditions A 2012 Cochrane Database Review revealed a lack of evidence in therapy for osteonecrosis in sickle cell disease. Vasodilators such as calcium channel blockers and the prostacyclin analogue iloprost (as continuous infusion) have been used to reduce pain in sickle crises and may have a role in preventing osteonecrosis though the issue has not been resolved from studies. There are detailed guidelines for managing antiphospholipid syn drome, though little specifically advised for osteonecrosis. General treatment principles apply and formal anticoagulation necessary given the infarction of bone as ‘end organ infarction’; see guidelines on antiphospholpid syndrome for example at: https://b-s-h.org.uk/ guidelines/. Daily treatment with low-molecular-weight heparin for 12 weeks can resolve early osteonecrosis-associated with pro-thrombotic con ditions. Warfarinization may then be necessary long-term. Areas of uncertainty, controversy, and future developments There is uncertainty as to whether it is possible to ‘profile’ patients for their risk of osteonecrosis. Given the likelihood of multiple risks, it may be able to define a profile of risk factors then design appro priate preventive studies. Uncertainty exists also as whether surgical intervention prior to arthroplasty is effective, compared to conser vative therapy. Indeed there is debate and controversy as to the merit of decompression compared with osteotomy and either procedure compared to conservative therapy alone. Comparative analyses of treatment modalities are lacking. There is likely to be progress from research into osteonecrosis of the jaw, given the concern that large numbers of (bisphosphonate- treated) patients may be at risk. Future developments are likely to focus around establishing osteonecrosis of the jaw risk profile of pa tients prior to bisphosphonate treatment. Osteochondrosis and osteochondritis dissecans Introduction Osteochondrosis is a trauma-induced focal disturbance of cartilage in a joint (articular), at a periarticular epiphyseal plate or tendon or ligament insertion (apophysis/enthesis) (Table 20.5.3). Lesions typically occur in active children and adolescents. Osteochondritis may be associated with a delay in growth-associated endochondral ossification, with a potential consequence of joint or other biomech anical deformity. Where the lesion is associated with cleft formation through articular cartilage, then the lesion is termed ‘dissecans’. Table 20.5.2 Steinberg and Association Research Circulation Osseous (ARCO) classifications of osteonecrosis of the femoral head Steinberg (2001) ARCO (1992) Stage I Normal radiographs Normal radiographs Stage II Femoral head lucency/sclerosis Demarcating sclerosis in femoral head, no collapse Stage III Subchondral collapse without femoral head flattening, ‘crescent sign’ Femoral head collapse, ‘crescent sign’, no joint space narrowing Stage IIIa Collapse <3 mm Stage IIIb Collapse >3 mm Stage IV Subchondral collapse, femoral head flattening, normal joint space Osteoarthritic degenerative changes Stage V Flattening with joint space narrowing, acetabular changes, or both Stage VI Advanced degenerative changes, secondary osteoarthritis For Steinberg: Stages I through IV are classified by per cent of femoral head involvement: A <15%, B 15–30%, C >30%. These size modifiers are considered predictors of femoral head collapse. Small lesion size and more medial location are considered prognostically favourable.

 Aetiology The cause of osteochondrosis may be multifactorial. Acute episodes of trauma and repetitive microtrauma may lead to microfractures in perichondral bone which heal poorly. There are some data to suggest,

that in children and adolescents, osteochondritis may occur through a mismatch of the ability of tissues to withstand re petitive or acute trauma, at a critical stage, or with excessive vel ocity, of growth. Though ischaemia has been postulated as a cause of osteochondritis and osteochondritis dissecans no studies have demonstrated osteo necrosis in specimens of excised osteochondral fragments. Evidence exists for genetic influence in certain lesions (e.g. case reports of various lesions of osteochondritis in monozygotic twins) though comparative prevalence studies in monozygotic vs. dizygotic twins have not been reported. Genome-wide association and proteomic studies of ex-vivo equine joint material in osteochondritis dissecans and controls, are being pursued but are as yet inconclusive Epidemiology The osteochondroses typically affect growing and active children and adolescents. Males are affected more than girls. Osteochondritis dissecans occurs typically in young athletes particularly in the knee, ankle, and elbow. The annual incidence of osteochondritis dissecans of the knee is about 10 per 100 000 with incidence of hospitalized patients between 1 to 2 per 100 000 of the population. However, accurate incidence data for all individual lesions are not available; though notably calcaneal apophysitis (Sever's disease) appears to be quite common (4/1000 children). Pathophysiology Articular osteochondrosis which progress to a 'dissecans' lesion, often start as a small area of bone compression, which progress to a partially (stage 2), detached (stage 3) or detached and displaced (stage 4) osteochondral fragment. In histological studies early fea tures include chondrocyte hypertrophy, fibrous degeneration associated with collagen type I deposition, and chondrocyte dedif ferentiation. Dissected fragments obtained at surgery often contain large numbers of viable cells. Clinical and imaging features Patients present with progressive activity-related focal pain, though some osteochondritis dissecans lesions can cause mild or even no symptoms for long periods of time. Detachment of the osteochondral fragment in osteochondritis dissecans may pre cipitate joint effusion and mechanical symptoms of joint locking, catching, and giving way. Multiple systems for classifying osteochondritis dissecans have been reported, both specific to lesional site and generalized systems. No system has been universally accepted. Radiographs may reveal a well-circumscribed area of sclerotic subchondral bone separated from the remainder of the epiphysis by a radiolucent line. Correlative histopathology and MRI studies in juvenile knee osteochondritis dissecans suggest cyst-like foci in the subchondral bone, bone marrow oedema, and relatively thick unossified epiphyseal car tilage. Breaks in the subchondral bone plate occur with fibrovascular tissue commonly found. Cleft spaces near the cartilage-bone inter face occur in most cases. Focal bone necrosis and inflammation are Table 20.5.3 The osteochondroses Type Site (eponym) Details Articular Metatarsal head (Freiberg's) Typically 2nd metatarsal head; bilateral in 10% Hip (Legg–Calvé–Perthes) Slipped capital femoral epiphysis; 4–10 years of age; more complications if

> 8 years of age; is bilateral in 10% Navicular (Köhler's) 3–7 years of age; male to female ratio 5:1; occasionally bilateral Talus Lateral lesions most likely to be associated with single trauma trigger Lunate (Kienböck's) Rare <15 years of age; commonly males; may be associated with short ulna relative to radius Nonarticular—at enthesis/apophysis Discrimination from enthesitis associated with Juvenile SpA and enthesitis-related arthritis is essential Vertebral end plate (Scheuermann's) 13–17 years of age; male to female ratio equal; usually lower thoracic more than upper lumbar; often several vertebrae affected (3–5); kyphosis can develop subsequently in late adolescence or gradually over

> years—a late diagnosis (age 50–70 yrs) of ‘previous Scheuermann’s’ is not unusual Tibial tubercle (Osgood–Schlatter) Apophysitis of insertion of patellar ligament; typically 10–15 yrs old; more often males than females; bilateral in 25% Inferior patella pole (Sinding–Larsen–Johansson) ‘Jumper’s knee’; typically male adolescents involved in sports and exercise Base of fifth metatarsal (Iselin’s) 9–15 yrs old; typically sports-related trauma Calcaneus (Sever’s) ‘Traction apophysitis’ at Achilles’ tendon insertion; incidence 4/1000 children age 6–17 yrs old Epiphyseal plate Ulna medial epiphyseal at elbow (Panner’s) Avulsion apophysitis from pitching in Little League baseball termed ‘Little League elbow’; typically males <16 yrs old; associated with increased height velocity; extensively reported in sports medicine literature Medial/proximal tibia (Blount’s) ‘Tibia vara’; infantile <3 yrs old or late onset Slipped capital femoral epiphysis Commonest adolescent hip disorder; 20% bilateral; risk factors include obesity, coxa profunda, femoral, or acetabular retroversion, obesity, hypothyroidism, hypopituitarism, renal osteodystrophy; 25% risk of progression to osteonecrosis; almost all cases need surgery

SECTION 20 Disorders of the skeleton 4708 infrequent MR findings. A defect in the hyaline cartilage represents displacement of an unstable lesion. Irregular ossification is a radiological differential diagnosis of osteochondritis dissecans, and is common—typically prevalent (66% of boys and 41% of girls in one series) in younger children (3– 12 years of age). Bone scintigraphy is normal in such cases. Differential diagnosis In adolescents, the main differential diagnosis of osteochondritis to consider is in regard of apophyseal lesions. Another term for an apophysis is an enthesis. Enthesitis is the typical lesion which occurs in enthesitis-related arthritis and the juvenile spondyloarthritis conditions. In adults, articular osteochondritis should be distin guished from osteochondral fracture, osteonecrosis, and degen erative change alone. Osteochondritis at an apophysis/enthesis is a far less frequent diagnosis of insertional ligament or tendon symptoms in adults compared with enthesopathy. The latter invari ably occurs either as a manifestation of spondyloarthritis—thus is an enthesitis (e.g. as in psoriatic arthritis)—or is a mechanical and/or painful lesion associated with diffuse idiopathic skeletal hyperostosis. Treatment The osteochondroses and most juvenile cases of osteochondritis dissecans respond well to nonoperative care. Measures include modi fication of activities, off-loading, analgesia, taping, and a stretching regime to release traction on affected apophyseal sites. Failure to heal within six months requires consideration of surgical treatment. For some osteochondritis lesions there are some known predictors of failure of conservative therapy recorded in the orthopaedic lit erature, thus planning management strategy with an orthopaedic specialist is important at the outset. Adherence with conservative treatment can be poor because symptoms typically recede long be fore bone healing has occurred. There are various surgical procedures reported for both adult and juvenile articular osteochondral cases. There are no random ized controlled trials. For persistent osteochondritis dissecans lesions, despite conservative therapy, the most frequently used technique involves arthroscopic drilling into the affected areas with fixation of the fragment using autologous osteochondral plugs or bio-absorbable polymer screws (which slowly degrade, al lowing healing of the fixed fragment). A viable fragment requires at least 3 mm of subchondral bone. Removing large lesions from weight-bearing areas does not achieve a good outcome in most studies unless accompanied by curettage, drilling, or placement of osteochondral plugs

(mosaicplasty). Newer techniques include autologous osteochondral or chondro cyte implantation or matrix-induced chondrogenesis. These tech niques have generally been employed for osteochondritis dissecans of the knee. Short-term follow-up studies are promising with careful case selection. Areas of uncertainty, controversy,
and future developments There is considerable debate, evident from the orthopaedic literature, on when to intervene surgically for some articular osteochondral lesions. The problem is underscored by the observation that many patients can tolerate and manage their lesions conservatively; indeed lesions can be asymptomatic for long periods. The second main issue is in regard of diagnosis of apophyseal lesions in children and ado lescents, specifically discriminating lesions from spondyloarthritis- related enthesitis lesions. FURTHER READING British Society for Haematology. Guidelines. http://www.bcshguidelines. com/documents/antiphospholipids_2012.pdf Bruns J, Werner M, Habermann C. (2018). Osteochondritis dissecans: etiology, pathology, and imaging with a special focus on the knee. Cartilage, 9, 346–62. Fliefel R, et al. (2015). Treatment strategies and outcomes of bisphosphonate-related osteonecrosis of the jaw (BRONJ) with characterization of patients: a systematic review. Int J Oral Maxillofac Surg, 44, 568–85. Gille J, et al. (2010). Mid-term results of autologous matrix-induced chondrogenesis for treatment of focal cartilage defects in the knee. Knee Surg Sports Traumatol Arthrosc, 18, 1456–64. Gómez-Puerta JA, et al. (2013). High prevalence of prothrombotic abnormalities in multifocal osteonecrosis: description of a series and review of the literature. Medicine Baltimore, 92, 295–304. Lafforgue P (2006). Pathophysiology and natural history of avascular necrosis of bone. Joint Bone Spine, 73, 500–7. Martí-Carvajal AJ, Solà I, Agreda-Pérez LH (2012). Treatment for avas cular necrosis of bone in people with sickle cell disease. Cochrane Database Syst Rev, 16, CD004344. Mont MA, Jones LC, Hungerford DS (2006). Nontraumatic osteo necrosis of the femoral head: ten years later. J Bone Joint Surg Am, 88, 1117–32. Sandro Pereira da Sliva J, et al. (2019). Genetic predisposition for medication-related osteonecrosis of the jaws: a systematic re view. Int J Oral Maxillofac Surg, pii: S0901-5027(19)31111-7. doi: 10.1016/j.ijom.2019.04.014. Sultan AA, et al. (2019). Classification systems of hip osteonecrosis: an updated review. Int Orthop, 43, 1089–95. Wall E, Von Stein D (2003). Juvenile osteochondritis dissecans. Orthop Clin North Am, 34, 341–53. Yuan HF, Guo CA, Yan ZQ (2016). The use of bisphosphonate in the treatment of osteonecrosis of the femoral head: a meta-analysis of randomized control trials. Osteoporosis Int, 27, 295–9.

Revision #1
Created 2026-01-22 16:41:10 UTC by Omar Ayman
Updated 2026-01-22 16:41:10 UTC by Omar Ayman