دعمارنجم

Osteoarthritis

Osteoarthritis (OA) is a chronic disorder of synovial joints in which there is progressive softening and disintegration of articular cartilage accompanied by new growth of cartilage and bone at the joint margins (osteophytes), cyst formation and sclerosis in the subchondral bone, mild synovitis and capsular fibrosis.

Risk factors

Joint dysplasia Disorders such as congenital acetabular dysplasia and Perthes' disease presage a greater than normal risk of OA in later life.
Trauma Fractures involving the articular surface are obvious precursors of secondary
Occupation There is good evidence of an association between OA and certain occupations
Bone density It has long been known that women with femoral neck fractures seldom have OA of the hip. This negative association between OA and osteoporosis
Obesity The simple idea that obesity causes increased joint loading and therefore predisposes to OA may be correct – at least in part.
Family history Women whose mothers had generalized OA are more likely to develop the same condition.

Signs and Symptoms

Pain It is often quite widespread, or it may be referred to a distant site
Stiffness is common; characteristically it occurs after periods of inactivity, but with time it becomes constant and progressive.
Swelling may be intermittent (suggesting an effusion) or continuous (with capsular thickening or large osteophytes).
Deformity may result from capsular contracture or joint instability
Loss of function

Imaging
(narrowing of the 'joint space') sclerosis of the subchondral bone under the area of cartilage loss, cysts close to the articular surface, osteophytes at the margins of the joint and remodelling of the bone ends on either side of the joint. Late features may include joint displacement and bone destruction.

Complications

Capsular herniation Osteoarthritis of the knee is sometimes associated with a marked effusion and herniation of the posterior capsule (Baker's cyst).

Loose bodies Cartilage and bone fragments may give rise to loose bodies, resulting in episodes of locking.

Rotator cuff dysfunction Osteoarthritis of the acromioclavicular joint may cause rotator cuff impingement, tendinitis or cuff tears.

Spinal stenosis Longstanding hypertrophic OA of the lumbar apophyseal joints may give rise to acquired spinal stenosis. The abnormality is best demonstrated by CT and MRI.

Spondylolisthesis In patients over 60 years of age, destructive OA of the apophyseal joints may result in severe segmental instability and spondylolisthesis (socalled 'degenerative' spondylolisthesis, which almost always occurs at L4/5).

Differential diagnosis of osteoarthritis
Avascular necrosis
Inflammatory arthropathies Rheumatoid arthritis, ankylosing spondylitis and Reiter's disease
Polyarthritis of the fingers
Diffuse idiopathic skeletal hyperostosis (DISH)

EARLY TREATMENT

(1) maintain movement and muscle strength; (2) protect the joint from 'overload';

(3) relieve pain; and (4) modify daily activities.

INTERMEDIATE TREATMENT

Joint debridement (removal of loose bodies, cartilage tags, interfering osteophytes or a torn or impinging acetabular or glenoid labrum) may give some improvement. This may be done either by arthroscopy or by open operation

LATE TREATMENT

Realignment osteotomy
Joint replacement
Arthrodesis

OSTEONECROSIS

MAIN CONDITIONS ASSOCIATED WITH NON-TRAUMATIC OSTEONECROSIS

Infections
- Osteomyelitis
- Septic arthritis

Haemoglobinopathy
- Sickle cell disease

Storage disorders
- Gaucher's disease

Caisson disease
- Dysbaric osteonecrosis

Coagulation disorders
- Familial thrombophilia
- Hypofibrinolysis
- Hypolipoproteinaemia
- Thrombocytopenic purpura

Other
- Perthes' disease
- Cortisone administration
- Alcohol abuse
- SLE (? increase in antiphospholipid antibodies)
- Pregnancy (? decreased fibrinolysis; ? fatty liver)
- Anaphylactic shock
- Ionizing radiation

SLE, systemic lupus erythematosus

Clinical features
The earliest stage of bone death is asymptomatic; by the time the patient presents, the lesion is usually well advanced. Pain is a common complaint. ‘click’ in the joint, stiff and deformed. in advanced cases there may be fixed deformities.

Imaging

X-ray changes appear3 months after the onset of ischaemia , subchondral bone sclerosis, thin fracture line just below the articular surface –the *‘crescent sign’ , In the late stages, collapsed*
segment. necrotic portion separates from the parent bone as a discrete fragment‘joint space’ retains its normal width because the articular cartilage is not destroyed until very late.

MRI is the most reliable way of diagnosing marrow changes and bone ischaemia at a comparatively early stage.

TREATMENT
EARLY OSTEONECROSIS
oral alendronate. reduce loading of weight-bearing joints may help osteotomy will help to preserve the anatomy while remodelling proceeds. This approach is applicable especially to the hip and knee.

INTERMEDIATE STAGE OSTEONECROSIS
there is structural damage and distortion of the articular surface realignment osteotomy – either alone or combined with curettage and bone grafting of the necrotic segment – has a useful role. arthrodesis will relieve pain and restore stability.

LATE STAGE OSTEONECROSIS
Destruction of the articular surface may be give rise to pain and severe loss of function

Three options are available:
(1) non-operative management, concentrating on pain control, modification of daily activities and, where appropriate, splintage of the joint;
(2) arthrodesis of the joint, e.g. the ankle or wrist; or
(3) partial or total joint replacement, the preferred option for the shoulder, hip and knee.