DNB
Master Solutions in Orthopedics
Volume - 3



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Paper 1 | DNB Othopedics (Dec 2020)

Question 1.

Describe Shoulder Stabilizers. Describe Clinical Tests for Shoulder Instability

Answer 1.

Anatomic control of glenohumeral joint stability can be divided into static (e.g., ligaments and tendons) and dynamic (e.g., muscular contraction) factors (Table 1). The cooperative role that these factors play is complex, and no single factor is responsible for glenohumeral joint stability. Similarly, no single lesion is responsible for clinical instability (Table 2). Contemporary approaches to the treatment of glenohumeral joint instability are directed at restoring normal anatomy and biomechanics, as opposed to simply constraining motion, as has been historically described.

Dynamic factors (i.e., rotator cuff and biceps) are affected by their level of conditioning and strength. Scapulothoracic motion is a more subtle, but equally important, dynamic factor helping to maintain shoulder stability. To add to this complex equation, an interaction between static capsuloligamentous factors and dynamic muscular factors, mediated through proprioception, has been postulated.

Factors maintaining joint STABILITY

Static Factors

1. Articular components
2. Articular version
3. Glenoid labrum
4. Negative intra- articular pressure
5. Adhesion cohesion
6. Capsule and ligament
7. Rotator cuff

Dynamic Factors

1. Rotator cuff
2. Biceps brachii
3. Scapular rotators
4. Proprioception

Normal And Abnormal Anatomy And Biomechanics

Stability Factor	Pathoanatomy
Glenoid version	Congenital: abnormal version; dysplasia Fracture causing abnormal version
Humeral version	Congenital: abnormal version; dysplasia Fracture/surgery causing abnormal version
Articular congruity	Congenital: dysplasia Acquired: fracture, Bankart lesion, osteoarthritis
Labrum	Bankart lesion Fraying secondary to laxity

Capsuloligamentous	Traumatic tear, cumulative microtrauma with plastic deformation Congenital laxity Loss of proprioceptive feedback
Negative intraarticular pressure	Capsular tear Rotator interval defect Lax capsule
Rotator cuff deficiency	Traumatic tear, cumulative microtrauma
Biceps	SLAP lesion Tendon rupture
Scapulothoracic motion	Dyskinesis: fatigue and weakness of serratus Long thoracic nerve palsy
SLAP, superior labrum from anterior to posterior.	

Anatomic and biomechanic dysfunction leading to glenohumeral instability results from varying levels of applied stress (i.e., a single traumatic event vs. cumulative microtrauma), the relative risk of injury associated with an activity, the quality and integrity of the static stabilizers, and the strength and conditioning of the dynamic stabilizers. An individual's susceptibility for glenohumeral instability is dependent on these factors.

Tests for Instability

Anterior	1. Apprehension test 2. Jobe Relocation test 3. Bony apprehension test
Posterior	1. Posterior apprehension test 2. Jerk test 3. Porcellini test
Multi-direction	1. Sulcus sign

Anterior instability test:

- **Apprehension test**
- **Bony apprehension test**
- **Jobe relocation test remove**

1. Apprehension test

Provocative test for anterior instability can be done in sitting / standing / supine position. The arm is abducted to 90° with elbow flexed 90°. Now the arm is externally rotated to 90°. Further external rotation is done slowly and if the patient has recurrent dislocation the patient exhibits apprehension rather than pain and the test is positive. If patient has pain then it can be other causes like posterior impingement

Bony apprehension test: In bony apprehension test,the apprehension is present at lower ranger of abduction / external rotation. There could be mid range instability due to the glenoid bone loss.

2. Jobe's Relocation test:

In supine position, place the arm in 90° abduction and full external rotation. At this point the patient feels apprehension. This is the first part of the test. Now with other hand examiners pushes the humeral head posteriorly (relocation). This is the next part. As the head relocates the apprehension decreases and this test is very sensitive test for anterior instability. Now the third part of the test is done. Remove this posterior force now without indicating it to the patient. The apprehension re-appears. This is called release and here the apprehension increases.

Posterior instability: less common- 2 percent of instability.

- Posterior apprehension
- Porcellini test

1. Posterior apprehension:

Patient in sitting position. Now ask the patient to relax and then take the arm into adduction and internal rotation with elbow flexed to 90°. In this position patient will have apprehension if there is a posterior instability.

2. Porcellini test:

With the examiner standing behind the patient, the patient's arm is forward flexed to 90, adducted 10–15, and maximally internally rotated. After stabilizing the scapula with one hand, the patient is asked to elevate the arm against the resistance and the pain and strength were noted.

The second test is done with the thumb of the examiner's hand was placed just lateral to the posterior joint line maintaining a moderate anterior force in order to prevent the humeral head from subluxating posteriorly, and then the maneuver is repeated. The test is considered positive if the pain with or without weakness is noticed during the first maneuver was reduced or eliminated during the second maneuver.

Inferior instability:

Sulcus test:

The examiner pulls the elbow down and observes the acromion. If there is a gap on lateral aspect or a sulcus visible, it denotes inferior laxity. The distance between inferior margin of acromion to the humeral head is measured.

Distance-

1 cm- 1+ sulcus

1-2 cms- 2+ sulcus

>2 cms- 3+ sulcus

Other instability tests:

Laxity – Anterior / Posterior laxity:

1. The anterior drawer test:

The patient is made supine. Considering that the left shoulder has to be examined, the examiner holds the affected limb in his right axilla. The shoulder should be in around 80- 100 degree abduction and 20 degree forward flexion and in external rotation. The examiner then holds the proximal humerus with his right hand and gives a counter pressure over the coracoid process with the left thumb and gives an anterior translation with the right hand. Always compare with other side. Normally more than 50% of head should not come out of glenoid cavity. This test is usually negative in all other positions of abduction or any other position

2. Rockwood test for anterior instability

In sitting position with the arm tucked by the side of body perform external rotation the arm maximally. Then the same is performed at 45, 90, 120°. If apprehension felt at 45, 90, 120° then test is positive. Usually at 0 degree there will be no apprehension.

Question 2.

Classify Surgical Site Infections, Management of 5-day Surgical Site Infection in Plate Osteosynthesis.

Answer 2.

According to Center for Disease Control and Prevention (CDC, United States), Surgical Site Infection (SSI) is an infection that occurs after surgery in that part of the body where surgery took place within 30 days of a procedure or within 90 days (up to 1 year if the implant is left in place) if an open reduction, spinal fusion or implantation of hip/knee prosthesis is done and the infection appears to be related to the procedure. Diagnostic criteria for SSI include:

- Fever > 380c (deep surgical wound infection)
- Painful spreading erythema indicating cellulitis
- Purulent discharge observed on direct examination (incisional surgical wound infection)
- Tenderness (deep surgical wound infection)
- Localized swelling (edema)
- Redness or erythema

Classification depending on plane of infection

CDC classified SSI into superficial (subcutaneous), deep (deep soft tissue, fascia and muscle) or organ/space (bone and joints considered as organ/ space). Organ / space or implant infections are mostly based on biofilm that requires removal of all surfaces and involved implant for eradication. Several categories of wound have been defined for guiding the extent of preventive measures (Eg. Debridement) while addressing the issue of primary injury / procedure. Closed fractures are considered clean wounds, while open fracture as contaminated, established infections (infected non-union/ infected hardware, osteomyelitis) are considered dirty wounds and minor break in the sterile surgical field is considered

as clean contaminated wound. Infected stab wound is not an SSI, and is rather classified as superficial or soft tissue infection depending upon the extent.

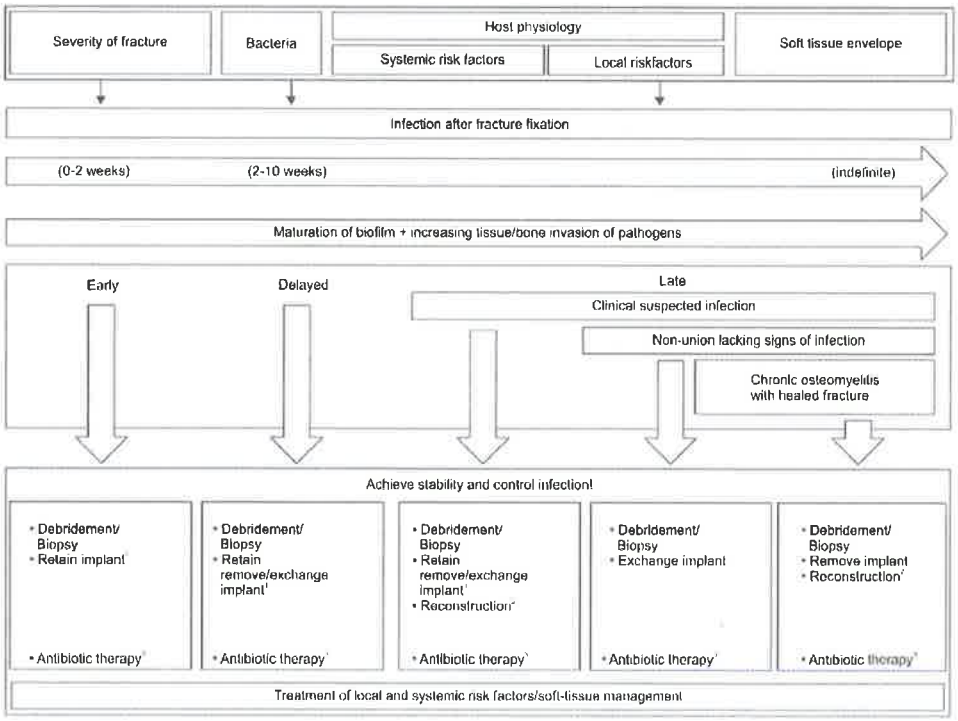
Classification depending on time post surgery

0-2 weeks- early

2-10 weeks- delayed

More than 10 weeks- late infection

Treatment



Treatment of early infection after plate osteosynthesis

Problems

- Colonization of hardware can occur intraoperatively,
- Biofilm formation may proceed within days,
- Implant serves as the nidus for infection and complicating healing/ treatment

What to do to the Plate?

- Biofilm formation is in an immature stage
- Fulminant osteomyelitis is often not yet present
- In early infections, consolidation can be achieved despite the presence of an infection, as long as the osteosynthesis construct remains stable
- Retention of the plate is hence advocated

Advantages of plate osteosynthesis

- The implant can be debrided without disturbing the stability
- This is not the case of an intramedullary device (might require an exchange as well) Treatment involves antibiotic therapy and tissue debridement.

Debridement

- Soft tissue debridement
- Careful revision of hematomas since they are a suitable growth medium for bacteria
- Plate wash and debridement

The purpose of surgery is to augment the host's immune response. Debridement results in removal of dead and avascular tissues, reduces the bacterial and bacterial products. Surgery is not required for all cases but in

conditions when pus is aspirated, or in radiographic changes of osteomyelitis suggestive of necrotic material, pus and chronic inflammation. If the above-mentioned conditions are not present, antibiotic trial can be given for 36 to 48 hours after culture sensitivity has arrived. If patient still does not respond, either wrong antibiotic is been given or abscess has formed

Antibiotic

- Post debridement, a 12-week course of antibiotic therapy with retained implants
- If implant is removed then 6 weeks of antibiotic to be given
- Debridement reduces the bacterial load and may clear an immature biofilm
- However additive systemic antibiotics will treat the remainder of the infection.
- Once the fracture has healed it is strongly recommended to remove the implant to reduce the risk of a recurrent infection.

For effective treatment

- Identification of organism
- Check for its antibiotic susceptibility.
- Appropriate antibiotic should be chosen based on minimal inhibitory concentration and serum bactericidal concentration.
- Antibiotic should be bactericidal.
- Sufficient concentration of antibiotics should be delivered over the affected area in order to destroy the organism.

Antibiotic delivery

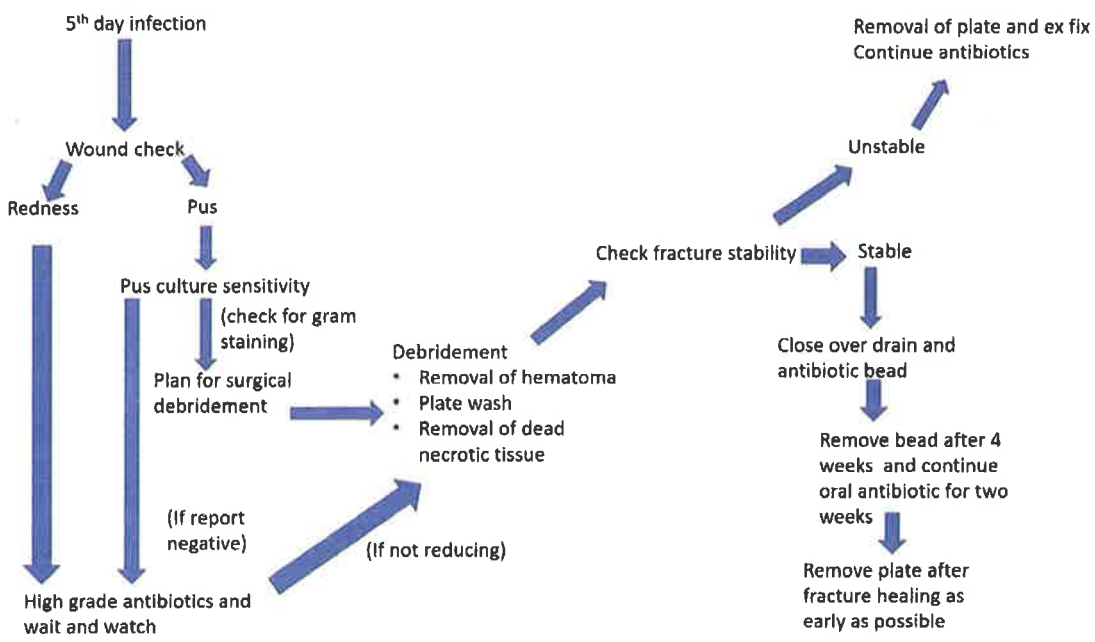
- Oral antibiotics is the most common mode to be used
- Intravenous application may be required for infections that do not respond to oral antibiotics.
- Local delivery of antibiotics is also beneficial. Polymethyl methacrylate (PMMA) beads impregnated with heat-stable antibiotics (vancomycin, tobramycin, and gentamicin)

Advantages of local delivery systems

- High antibiotic concentration is present in 2 to 3 cm area around each bead. With vancomycin and tobramycin, the peak concentration of antibiotic delivered to local tissue occurs on the first day and lasts for approximately 1 week.
- Delivering antibiotics locally in high concentration avoids systemic toxicity

Disadvantages of local delivery systems

It has to be removed surgically within 4 weeks. (another alternative is the biodegradable collagen – gentamicin sponge and also has the advantage of providing higher antibiotics concentration than PMMA bead).



Question 3.

Describe Anatomy of Articular Cartilage. OA changes in Cartilage

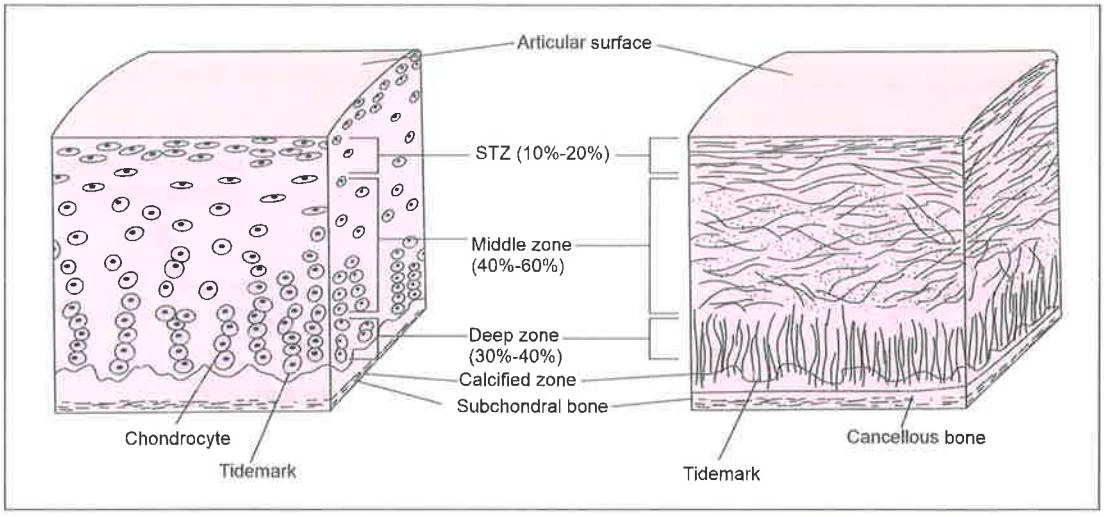
Answer 3.

Normal structure

- Articular cartilage consists primarily of extracellular matrix with a sparse population of cells
- Its made of hyaline cartilage of 2-4mm thick
- It does not have its own blood vessels, lymphatic vessels and nerves
- It has a lowlevel of metabolic activity,
- There is little or no cell division or cell death in normal adult articular cartilage expect it has chondrocytes, making up only about 1% of volume of adult human articular cartilage
- The articular cartilage needs to be elastic and have high tensile strength to functional normally.
- The unique mechanical properties of articular cartilage depend on the extracellular matrix
- The extracellular matrix consists of two components (help to retain water within the matrix)
  - Tissuefluid
  - The framework of structural macromolecules consisting of type II collagen fibers, proteoglycans andnon-collagenousproteins and glycoproteins,
- Tissue fluid and structural macromolecules are produced in the appropriate amounts and assembled and organized into a highly ordered molecular framework by the chondrocytes
- The collagen matrix gives cartilage its form and tensile strength.
- Proteoglycans and noncollagenous proteins bind to the collagenous network, help stabilize the matrix macromolecular framework and help chondrocytes bind to the macromolecules of the network

Histology of the cartilage

- Various zones of articular cartilage
  - The superficial zone, the middle zone, the deep zone, and the calcified zone
- Within each zone, 3 regions can be identified
  - The pericellular region, the territorial region, and the interterritorial region.
- **Zones of cartilage**



- **Superficial zone**
  - The thin superficial (tangential) zone protects deeper layers from shear stresses
  - 10% to 20% of articular cartilage thickness.
  - The collagen fibers of this zone (primarily, type II and IX collagen) are packed tightly and aligned parallel to the articular surface

- It contains a relatively high number of flattened chondrocytes, and the integrity of this layer is imperative in the protection and maintenance of deeper layers.
- This zone is in contact with synovial fluid and is responsible for most of the tensile properties of cartilage, which enable it to resist the sheer, tensile, and compressive forces imposed by articulation.

- **Middle (transitional) zone**
  - Immediately deep to the superficial zone
  - It provides an anatomic and functional bridge between the superficial and deep zones.
  - It represents 40% to 60% of the total cartilage volume,
  - It contains proteoglycans and thicker collagen fibrils.
  - In this layer, the collagen is organized obliquely, and the chondrocytes are spherical and at low density.
  - Functionally, the middle zone is the first line of resistance to compressive forces
- **Deep zone**
  - Is responsible for providing the greatest resistance to compressive forces, given that collagen fibrils are arranged perpendicular to the articular surface.
  - The deep zone represents approximately 30% of articular cartilage volume.
  - It contains the largest diameter collagen fibrils in a radial disposition, the highest proteoglycan content, and the lowest water concentration.
  - The chondrocytes are typically arranged in columnar orientation, parallel to the collagen fibers and perpendicular to the joint line.
- **The tide mark**
  - It distinguishes the deep zone from the calcified cartilage.
  - The collagen fibrils are arranged perpendicular to the articular cartilage.
  - The calcified layer plays an integral role in securing the cartilage to bone, by anchoring the collagen fibrils of the deep zone to subchondral bone.
  - In this zone, the cell population is scarce and chondrocytes are hypertrophic.

Regions

- **Pericellular matrix**
  - This is a thin layer adjacent to the cell membrane, and it completely surrounds the chondrocyte.
  - It contains mainly proteoglycans, as well as glycoproteins and other noncollagenous proteins.
  - This matrix region may play a functional role to initiate signal transduction within cartilage with load bearing.
- **Territorial matrix**
  - Surrounds the pericellular matrix;
  - It is composed mostly of fine collagen fibrils, forming a basketlike network around the cells.
  - This region is thicker than the pericellular matrix, and it has been proposed that the territorial matrix may protect the cartilage cells against mechanical stresses and may contribute to the resiliency of the articular cartilage structure and its ability to withstand substantial loads
- **Interterritorial region**
  - It is the largest of the 3 matrix regions;
  - It contributes most to the biomechanical properties of articular cartilage.
  - This region is characterized by the randomly oriented bundles of large collagen fibrils, arranged parallel to the surface of the superficial zone, obliquely in the middle zone, and perpendicular to the joint surface in the deep zone.
  - Proteoglycans are abundant in the interterritorial zone

Pathogenies of Primary osteoarthritis

- The only cells in the articular cartilage is the chondrocytes ,these are thought to be long lived cells
- Therefore aging profoundly alters chondrocyte function and matrix structure and functioning
- There is an age-related reduction in the number of chondrocytes in articular cartilage
- Chondrocytes cell senescence can result in phenotypical alteration of cells, called the

senescent secretory phenotype.

- This phenotype is characterized by increased production of cytokines and growth factors.
- Which in turn lead to matrix degradation and reducing matrix synthesis and repair, and ultimately lead to joint degeneration
- Chondrocytes become less responsive to growth factors (IGF-1)with aging
- Age-related changes in size, structure and sulfation of aggrecan, the molecules in cartilage matrix that hold water is seen which leads to change in the biophysical properties of cartilage matrix and therefore reducing its resiliency and tensile strength
- Since articular cartilage has a relatively lowturn-over rate and there is accumulation of advanced glycation end-products, resulting from spontaneous non-enzymatic glycation of proteins.
- Increased collagen cross-linking due to modification of collagen by AGE formation renders the cartilage more brittle with increased fatigue failure.
- There is also increasing oxidative stress due tochronic production of **endogenous reactive oxygen species(ROS or "free radicals")**which plays an important role in aging and in the link between aging and osteoarthritis.
- Increased levels of ROS can damage DNA, including mitochondrial DNA, thereby affecting cell viability, and contributing to the disruption of extracellular matrix homeostasis.
- Increased levels of ROS may also contribute to the senescent secretory phenotype and the reduced sensitivity of chondrocytes to IGF-1

Aging change	Contribution to OA
Accumulation of cells exhibiting the senescent secretory phenotype	Increased cytokine and MMP production stimulates matrix degradation
Oxidative stress/ damage, decreased water concentration, increased collagen cross linking, decrease tensile strength and stiffness in the superficial layers	Increased susceptibility to cell death and reduced matrix synthesis, increased water concentration, loss of tensile strength and stiffness throughout
Decreased levels of growth factors and decreased growth factorresponsiveness	Reduced matrix synthesis and repair
Increased AGE formation	Brittle tissue with increased fatigue failure

Question 4.

Define Bone Remodeling, What is the Difference between Osteoporosis and Osteopenia. Explain the Management of Osteoporotic Vertebral Fractures with a Flowchart.

Answer 4.

The skeleton is a metabolically active organ that undergoes continuous remodeling throughout life. Bone remodeling involves the removal of mineralized bone by osteoclasts followed by the formation of bone matrix through the osteoblasts that subsequently become mineralized. The remodeling cycle consists of three consecutive phases: resorption, during which osteoclasts digest old bone; reversal, when mononuclear cells appear on the bone surface; and formation, when osteoblasts lay down new bone until the resorbed bone is completely replaced. Bone remodeling serves to adjust bone architecture to meet changing mechanical needs and it helps to repair micro-damages in bone matrix preventing the accumulation of old bone. It also plays an important role in maintaining plasma calcium homeostasis. The regulation of bone remodeling is both systemic and local.

- Osteoporosis is a systemic skeletal disease characterized by low bone mass and micro-architectural deterioration of bone tissue, leading to enhanced bone fragility and a consequent increase in fracture risk. According to BMD measurements a T score less than – 2.5 is considered to be osteoporotic and is classified as severe if there is an associated fracture history due to osteoporosis.

Osteopenia is when there is lower bone density but the reduction is not sufficient to cause a fragility fracture and is defined as a T score between -1 to -2.5.

Management of vertebral osteoporotic fracture

Diagnosis of "fragility" fracture

↓

Identify underlying disease*

↓

History and examination*

↓

Physical examination*

↓

Laboratory tests*

↓

Imaging*

↓

General fracture management*

↓

Fixation principles in osteoporotic bone

- Relative stability instead of absolute stability
- Indirect, functional reduction (not anatomical reduction)
- Locked splinting with long plates or nails
- Load distribution (no peak stresses)
- No interfragmentary compression
- Secondary bone healing with callus formation
- No mixture of principles and methods
- Consider vertebroplasty/kyphoplasty if clinically stable

↓

Rehabilitation

  - Goal is to improve strength, balance, position sense, reactions to
- Improve level of function / independence
- Maximize mobility
- Decrease risk of falls
- Decrease risk of fractures

↓

Secondary prevention

  - Treat and monitor underlying disease
- Regular physical activity
  - Maintaining safe ambulatory status, independent ADLs
  - Daily limb and core home exercise routine
- Sufficient intake of calcium and vitamin D
  - daily 1000-1500 mg calcium, 400-800 IU vitamin D
  - by foods or foods and supplements combined
- Adequate nutrition
- Avoid cigarettes, excess alcohol

↓

Prevent future fractures

Basics

- Nutrition, exercise, fall prevention strategies
- Modify risk factors (smoking, excess alcohol)
- Treat co-morbidities (i.e., endocrine disorder?)
- Pharmacological agents
  - Daily 1000-1500 mg calcium, 400-800 IU vitamin D
    - Through diet and supplements
  - Osteoporosis medications (Table)
- FRAX - fracture Risk Assessment Tool

Underlying diseases*

- Severe chronic liver or kidney diseases
- Steroid medication (>7.5mg for more than 6 months)
- Malabsorption (eg. Crohn's disease)
- Rheumatoid arthritis
- Systemic inflammatory disorders
- Hyperthyroidism
- Primary hyperparathyroidism
- Antiepileptic medication

History*

- Family history of OP
- Menarche / Menopause
- Nutrition
- Medications → (past and present)
- Level of activity
- Fracture history
- Fall history & risk factors for falls
- Smoking, alcohol intake
- Risk factors for secondary OP
- Prior level of function

Physical examination*

- Height
- Weight
- Limb exam
  - ROM, strength, deformity, pain, neurovascular status
- Spine exam
  - pain, deformity, mobility
- Functional status

Laboratory tests*

- ESR / CRP
- Blood count
- Calcium
- Phosphate
- Liver function tests
  - Alkaline Phosphatase (ALP)
  - GGT
- Renal function studies
- Basal TSH
- Intact PTH
- Protein-immunoelectrophoresis
- Vit D (25 and 1.25)

Imaging*

- Bone mineral density
  - Establish severity of osteoporosis
  - Baseline for monitoring treatment efficacy
- Spine radiograph
  - vertebral fracture assessment
  - Screening for patients with
    - Back pain
    - Loss of height > 4 cm
    - Progressive kyphosis General fracture management*
- Stabilize patient, pain relief
- Fracture care
- Changes in cortical bone
  - Decreased thickness
    - Less working length of implants
      - ☐ Less purchase in cortex
  - Increased Haversian canal areas (lacunae formation)
    - Increased weakness
    - Predisposition to low-energy fractures
- Changes in cancellous bone
  - Less and thinner trabeculae
    - With fewer, often broken interconnections Reduced cut-out resistance and bone voids

Question 5.

What is the Pathogenesis of Gout. How do you Manage a Case of Gout.

Answer 5.

- Disorder of purine metabolism characterized by hyperuricaemia, deposition of monosodium urate monohydrate crystals in joints and peri-articular tissues and recurrent attacks of acute synovitis.
- Late changes include cartilage degeneration, renal dysfunction and uric acid urolithiasis.

Pathology

- Nucleic acid and purine metabolism normally proceeds, through complex pathways, to the production of hypoxanthine and xanthine; the final breakdown to uric acid is catalysed by the enzyme xanthine oxidase.
- Monosodium urate appears in ionic form in all the body fluids; about 70 per cent is derived from endogenous purine metabolism and 30 per cent from purine-rich foods in the diet. It is excreted (as uric acid) mainly by the kidneys and partly in the gut.
- Urate is poorly soluble, with a plasma saturation value of only 7 mg/dL (0.42 mmol/L). This concentration is commonly exceeded in normal individuals and epidemiological studies have identified entire populations (for example the Maoris of New Zealand) who have unusually high levels of serum uric acid.
- The term 'hyperuricaemia' is therefore generally reserved for individuals with a serum urate

concentration which is significantly higher than that of the population to which they belong (more than two standard deviations above the mean); this is about 0.42 mmol/L for men and 0.35 mmol/L for women in western Caucasian peoples.

- By this definition, about 5 per cent of men and less than 1 per cent of women have hyperuricaemia; the majority suffer no pathological consequences and they remain asymptomatic throughout life.
- Uric acid is the normal breakdown product of purine metabolism. An abrupt increase in production or ingestion of purine-containing compounds can overwhelm the renal excretory mechanism, resulting in increased uric acid levels in serum and related body fluid spaces.
- Predisposing factors include an inherited deficiency aggravated by male gender and dietary indulgences in meat and red wine .
- Typically, monosodium urate crystals precipitate in supersaturated solutions.
- Lower temperatures decrease the solubility of uric acid, explaining the propensity of toe and finger joint involvement.
- Repetitive microtrauma, such as occurs in the first metatarsophalangeal joint of the great toe and the distal interphalangeal joints of the hand, explains greater involvement of these joints.
- Urate crystals are deposited in minute clumps in connective tissue, including articular cartilage; the commonest sites are the small joints of the hands and feet. For months, perhaps years, they remain inert.
- Then, possibly as a result of local trauma, the needlelike crystals are dispersed into the joint and the surrounding tissues where they excite an acute inflammatory reaction.
- Individual crystals may be phagocytosed by synovial cells and polymorphs or may float free in the synovial fluid.
- With the passage of time, urate deposits may build up in joints, peri-articular tissues, tendons and bursae; common sites are around the metatarsophalangeal joints of the big toes, the Achilles tendons, the olecranon bursae and the pinnae of the ears.
- These clumps of chalky material, or tophi (L. *tophus* = porous stone), vary in size from less than 1 mm to several centimetres in diameter. They may ulcerate through the skin or destroy cartilage and peri-articular bone.

Factors predisposing to hyperuricaemia

1. Older age, male gender
2. Genetic enzyme defects, hyperparathyroidism
3. Haemolytic disorders, myeloproliferative disorders
4. Obesity, diabetes, hypertension
5. High consumption of red meat, hyperlipidaemia
6. Chronic inflammatory diseases
7. Long-term use of aspirin or diuretics
8. Alcohol abuse

The acute attack

- The typical onset - an acute and dramatically painful arthritis that begins at night in a single joint. The commonest sites are the metatarsophalangeal joint of the big toe, the ankle and finger joints, and the olecranon bursa. Occasionally, more than one site is involved. The attack usually comes out of the blue but may be precipitated by minor local trauma, operation, intercurrent illness, unaccustomed exercise or alcohol consumption. In a small segment of patients, joint symptoms are preceded by nephrolithiasis. Associated fever, chills, swelling, and periarticular erythema may be confused with cellulitis or septic arthritis. As the inflammation subsides, desquamation of the skin overlying the affected joint may be noted.
- The area can be incised and drained by an unsuspecting surgeon.
- Sometimes the only feature is acute pain and tenderness in the heel or the sole. Hyperuricaemia is present at some stage, though not necessarily during an acute attack. However, while a low serum uric acid makes gout unlikely, hyperuricaemia is not 'diagnostic' and is often seen in normal middle-aged men.
- Initial attacks subside spontaneously over 3 to 10 days, even without specific therapy. Patients are asymptomatic until the next episode, even though monosodium urate crystals may be aspirated from the involved joints or even from joints that have not been affected. Over time, attacks occur more frequently, involve more joints, persist for longer periods, and may result in end-stage arthritis.

- The true diagnosis can be established beyond doubt by finding the characteristic negatively birefringent urate crystals in the synovial fluid. A drop of fluid on a glass slide is examined by polarizing microscopy. Crystals may be sparse but if the fluid specimen is centrifuged a concentrated pellet may be obtained for examination.

CHRONIC GOUT

- Recurrent acute attacks may eventually merge into polyarticular gout. Joint erosion causes chronic pain, stiffness and deformity; if the finger joints are affected, this may be mistaken for rheumatoid arthritis. Chronic recurrent attacks of gouty arthritis and persistent hyperuricemia may result in the deposition of uric acid crystals in the synovium, subcutaneous tissue, bursae, subchondral bone, and helix of the ear. Resultant *tophi* (uric acid deposits with surrounding histiocytic reactions) typically appear an average of 10 years after the initial gouty attack. Tophaceous material is characteristically chalky white and exhibits uric acid crystal morphology on examination under polarized light microscopy. A large tophus can ulcerate through the skin and discharge its chalky material.
- Renal lesions include calculi, due to uric acid precipitation in the urine, and parenchymal disease due to deposition of monosodium urate from the blood. Kidney stones of uric acid are 10 times more common among patients with symptomatic gouty arthritis than among normouricemic individuals. Related renal dysfunction, proteinuria, and hypertension may be seen in patients with gout, but these are usually mild.

X-rays

- During the acute attack x-rays show only soft-tissue swelling.
- Chronic gout may result in joint space narrowing and secondary osteoarthritis.
- Tophi appear as characteristic punched-out 'cysts' or deep erosions in the para-articular bone ends; these excavations are larger and slightly further from the joint margin than the typical rheumatoid erosions. Advanced destruction in joints affected by gout is characterized by subchondral erosions with a sclerotic margin; these are round or oval, suggesting pressure-related erosion of adjacent bone. The joint space is relatively well preserved until late in the disease.
- Occasionally, bone destruction is more marked and may resemble neoplastic disease
- In chronic gout, massive deposits of monosodium urate crystals can be found around the joints and tendon sheaths, causing nerve compression, such as carpal tunnel syndrome.
- Intratendinous monosodium urate crystal deposition can lead to tendon rupture.
- Amputation may be necessary because of the extreme bony disruption resulting from gout.



Fig:Largeperiarticular excavations – tophi consisting of uric acciddeposits

Diagnosis

- The presence of hyperuricemia alone does not establish the diagnosis of gout; the uric acid level may be elevated, and an acute attack of gout may never occur. Conversely, during an acute attack of gout, the uric acid level may be normal. Aspiration of a joint provides the only definitive diagnosis of gout
- confirmed by demonstration of *intracellular, negatively birefringent monosodium urate crystals.*
- These needle-shaped crystals appear yellow under polarized light microscopy.
- Synovial fluid white counts may be normal or in the range of 100,000/mm^3.
- If high white cell counts are present, the gouty effusion may be mistaken for septic arthritis until crystals are identified and joint cultures are found to be negative.

- Serum uric acid levels are important in the long-term management of gout but are of limited use in the diagnosis of acute gout because these levels may be normal at the time of an acute gouty attack.

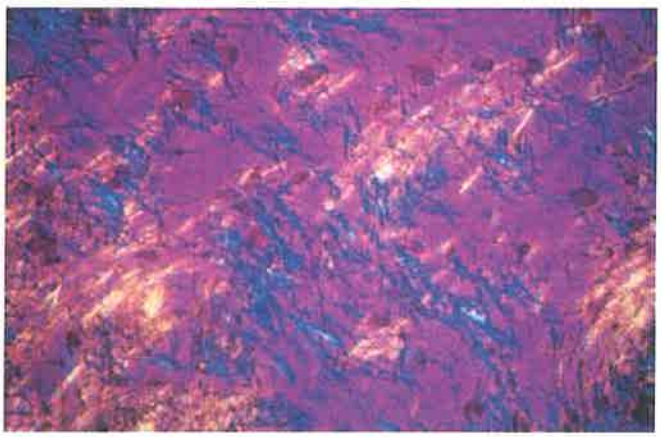


Fig:Uratecrystals are needle-like, 5–20 μm long and exhibit strong negative birefringence

- *Rheumatoid arthritis (RA)* Polyarticular gout affecting the fingers may be mistaken for rheumatoid arthritis, and elbow tophi for rheumatoid nodules. In difficult cases biopsy will establish the diagnosis. RA and gout seldom occur together.

Treatment

- Treatment approaches include medications to diminish the inflammatory response of an acute attack and reduction of serum uric acid to minimize the risk of recurrent attacks and progressive joint destruction.

The acute attack

- The acute attack should be treated by resting the joint, applying ice packs if pain is severe, and giving full doses of a non-steroidal anti-inflammatory drug (NSAID).
- Colchicine, one of the oldest of medications, is less effective and may cause diarrhoea, nausea and vomiting. *Colchicine* interferes with leukocyte migration and function, and 90% of patients with acute gout have decreased pain and swelling when colchicine is initiated within a few hours of disease onset. Although quite effective, oral colchicine is associated with gastrointestinal adverse effects, and approximately 80% of patients treated with colchicine experience cramping diarrhea and/or nausea and vomiting. Therefore, in patients who do not have severe pain, NSAIDs are the most common medications used in treatment of an acute gout attack.
- A tense joint effusion may require aspiration and intra-articular injection of corticosteroids.
- Red meat and alcohol comsumption should be avoided in an acute episode. Oral corticosteroids are sometimes used for patients who cannot tolerate NSAIDs or in whom NSAIDs are contraindicated.
- *The sooner treatment is started the sooner is the attack likely to end.*

Interval therapy

- Chronic gout and hyperuricemia are managed by first determining whether the patient is a perpetual overproducer or underexcreter of uric acid.
- Overproducers are treated with *xanthine oxidase inhibitors*, such as allopurinol, and underexcreters are managed with a *uricosuric agent*, most commonly probenecid.
- Between attacks, attention should be given to simple measures such as losing weight, cutting out alcohol and eliminating diuretics.
- Dieatary restricrions include restriction of red meat and alcohol comsumption, decreasing soda intake. No restriction on vegetarian food is advocated.
- Urate-lowering drug therapy is indicated if acute attacks recur at frequent intervals, if there are tophi or if renal function is impaired. It should also be considered for asymptomatic hyperuricaemia if the plasma urate concentration is persistently above 6 mg/dL (0.36 mmol/L). However, one must remember that this starts a life-long commitment and many clinicians feel that people who have never had an attack of gout and are free of tophi or urinary calculi do not need treatment.
- *Uricosuric drugs* (probenecid or sulfinpyrazone) can be used if renal function is normal.
- However, allopurinol, *a xanthine oxidase inhibitor*, is usually preferred, and for patients with renal complications or chronic tophaceous gout allopurinol is definitely the drug of choice.
- Urate-lowering drugs should never be started before the acute attack has completely subsided, and they should always be covered by an anti-

inflammatory preparation or colchicine, otherwise they may actually prolong or precipitate an acute attack.

- Patients who suffer an acute attack of gout while already on a constant dose of urate-lowering treatment should be advised to continue taking the drug at the usual dosage while the acute episode is being treated.

Surgery

- With prolonged urate-lowering therapy, adjusted to maintain a normal serum uric acid level (less than 0.36 mmol/L), tophi may gradually dissolve.
- However, ulcerating tophi that fail to heal with conservative treatment can be evacuated by curettage; the wound is left open and dressings are applied until it heals.
- Surgery for tophaceous deposits rarely is indicated, unless an important structure is compressed or if the patient cannot tolerate uric acid–lowering measures.
- In addition to tophus excision and débridement, other procedures that may benefit a patient with gout include tenosynovectomy, tendon repair or transfer for tendon ruptures, carpal tunnel release, and appropriate management of joints that have been destroyed by gouty arthritis.

Febuxostat

Indications

Febuxostat is an inhibitor of the enzyme xanthine oxidase and is FDA-approved for the chronic management of hyperuricemia in patients diagnosed with gout. Febuxostat is prescribed to patients for which allopurinol is not efficacious or patients who experience severe side effects with allopurinol.

Mechanism of Action

Febuxostat is a non-purine selective inhibitor of the enzyme xanthine oxidase, which is involved in purine catabolism. The xanthine oxidase enzyme catalyzes two reactions that ultimately generate uric acid from hypoxanthine. Febuxostat is a potent, selective inhibitor of xanthine oxidase, forming a stable complex with both the reduced and oxidized form of the enzyme, thereby inhibiting its function.

Administration

Febuxostat is administered orally with an initial dose of 40 mg per day. The dose can be increased to 80 mg per day in patients that do not achieve a serum uric acid level of less than 6 mg/dL after two weeks of treatment with 40 mg so long as the patient does not have severe renal impairment (defined as a clearance of 15 to 29 mL/min). Upon initiating treatment with febuxostat, gout flares may occur. Therefore, flare prophylaxis with either a non-steroidal anti-inflammatory drug (NSAID) or colchicine is recommended concurrently with febuxostat treatment and may continue for up to six months.

Adverse Effects

Common adverse drug effects (greater than 1% of the febuxostat-treated group based on multiple randomized, controlled clinical studies ranging in length from 6 to 12 months):

- Liver function abnormalities, dizziness, arthralgia, nausea, and rash.

Question 6.

Define Neurogenic Shock. Describe in Detail Clinical Diagnosis of Level of Spinal Cord Injury.

Answer 6.

This is a condition of autonomic dysregulation seen in the patients with spinal cord injuries. It is most commonly seen in patients with traumatic injuries to the spinal cord most commonly cervical and thoracic spine injuries. The trauma leads to a state of shock caused by unopposed parasympathetic action with a complete loss of sympathetic response. It is of vital importance to diagnose and manage aggressively to prevent any secondary injuries. However it is a diagnosis of exclusion as the most common cause of shock after trauma is hemorrhagic shock.

It is important to distinguish the two as the management differs drastically. The autonomic instability is manifested by triad of hypotension, bradycardia, and temperature dysregulation.

As per the most widely accepted consensus the accepted criteria is systolic blood pressure of <100 mm Hg and heart rate of less than 80 per minute. The other causes of neurogenic or spinal shock apart from trauma are spinal

anesthesia (most common), Guillain-Barre syndrome, autonomic nervous system toxins, transverse myelitis.

Spinal shock is defined as acute loss of motor, sensory and reflex functions below the level of injury. This is generally associated with neurogenic shock. It is manifested by absence of anal wink and bulbocavernosus reflexes and by flaccid paralysis. It is a temporary phenomenon and recovers usually in 24 to 48 hours even in severe injuries but can persist for weeks or rarely months. There is no specific treatment for spinal shock.

Determining neural levels:

Anatomy

- Vertebral column has a total of 33 Vertebrae.
- 7 Cervical, 12 Thoracic, 5 Lumbar, 5Sacral - 5 segments fused in one block, 4 Coccyx - 4 segments fused in one block.
- 31 spinal segments and Nerves
- 8 cervical, 12 Thoracic, 5 Lumbar, 5 sacral and one Coccyx
- Total no of vertebral discs: 23
- Spinal cord starts from C1 and ends at the level of L1 or L2. Here the cord ends in the form of conus medullaris. From here there is a ligament that connects the spinal cord to the coccyx and gives stability to the cord.
- L1 to L5 has the cauda equine, which is a filamentous fibrous thread like Structure.

Cord Segment Level	
Vertebral level Neural level	Vertebral level Neural level
Cervical Add 1	Cervical Add 1
Thoracic (1-6) Add 2	Thoracic (1-6) Add 2
Thoracic (7-9) Add 3	Thoracic (7-9) Add 3
T10 L1 and L2	T10 L1 and L2
T11 L3 and L4	T11 L3 and L4
T12 L5	T12 L5
Lumbar I Sacral elements	Lumbar I Sacral elements

E.g., C7 vertebral body is opposite to cord segment 8, cord finishes at T12 level on conus medullaris at L1.

The level of injury for a person with SCI is the lowest point on the spinal cord below which sensory feeling and motor movement diminish or disappear. The level is denoted by the letter-and- number name of the vertebra at the injury site.

Sensory level: The sensory level is determined by performing an examination of the key sensory points within each of the 28 dermatomes on each side of the body (right and left) and is the most caudal, normally innervated dermatome for both pin prick (sharp/dull discrimination) and light touch sensation. This may be different for the right and left side of the body.

Motor level: The motor level is determined by examining a key muscle function within each of 10 myotomes on each side of the body and is defined by the lowest key muscle function that has a grade of at least 3 [on manual muscle testing (MMT) in the supine position], providing the key muscle functions represented by segments above that level are judged to be intact (graded as a 5 on MMT). This may be different for the right and left side of the body.

Neurological level of injury (NLI): The NLI refers to the most caudal segment of the spinal cord with normal sensory and antigravity motor function on both sides of the body, provided that there is normal (intact) sensory and motor function rostrally. The segments at which normal function is found often differ by side of the body and in terms of sensory and motor testing. Thus, up to four different segments may be identified in determining the neurological level, i.e., R(ight)- sensory, L(eft)-sensory, R-motor, L-motor. The single NLI is the most rostral of these levels.

Sensory scores: This term refers to a numerical summary score of sensory function. There is a maximum total of 56 points each for light touch and pin prick (sharp/dull discrimination) modalities, for a total of 112 points per side of the body. This can reflect the degree of neurological impairment associated with the SCI.

Motor scores: This term refers to a numerical summary score of motor function. There is a maximum score of 25 for each extremity, totaling 50 for the upper limbs and 50 for the lower limbs. This score can reflect the degree of neurological impairment associated with the SCI.

Incomplete injury: This term is used when there is preservation of any sensory and/or motor function below the neurological level that includes the lowest sacral segments S4-S5 (i.e. presence of "sacral sparing"). Sensory

sacral sparing includes sensation preservation (intact or impaired) at the anal mucocutaneous junction (S4-5 dermatome) on one or both sides for light touch or pin prick, or deep anal pressure (DAP). Motor sacral sparing includes the presence of voluntary contraction of the external anal sphincter upon digital rectal examination.

Complete injury: This term is used when there is an absence of sensory and motor function in the lowest sacral segments (S4-S5) (i.e. no sacral sparing).

Zone of partial preservation (ZPP): This term, used only with complete injuries, refers to those dermatomes and myotomes caudal to the sensory and motor levels that remain partially innervated. The most caudal segment with some sensory and/or motor function defines the extent of the sensory and motor ZPP respectively and are documented as four distinct levels (R- sensory, L-sensory, R-motor, and L-motor).

Clinical Pearls:

Appreciation of light touch and pin prick sensation at each of the key points is separately scored on a three-point scale, with comparison to the sensation on the patients' cheek as a normal frame of reference:

0 = absent

1= altered (impaired or partial appreciation, including hyperesthesia) 2 = normal or intact (similar as on the cheek)

NT = not testable

Light touch sensation is tested with a tapered wisp of cotton stroked once across an area not to exceed 1cm of skin with the eyes closed or vision blocked.

Pin prick sensation (sharp/dull discrimination) is performed with a disposable safety pin that is stretched apart to allow testing on both ends; using the pointed end to test for sharp and the rounded end of the pin for dull.

The strength of each muscle function is graded on a six-point scale:

0 = total paralysis.

1 = palpable or visible contraction.

2 = active movement, full range of motion (ROM) with gravity eliminated.

3 = active movement, full ROM against gravity.

4 = active movement, full ROM against gravity and moderate resistance in a muscle specific position.

5 = (normal) active movement, full ROM against gravity and full resistance in a muscle specific position expected from an otherwise unimpaired person.

NT= not testable (i.e. due to immobilization, severe pain such that the patient cannot be graded, amputation of limb, or contracture of >50% of the range of motion).

Question 7.

What is Fat Embolism? Clinical Features, Investigations, Diagnosis and Treatment.

Answer 7.

Fat Embolism Syndrome

Fat embolism refers to the presence of fat globules in vital organs and peripheral circulation after fracture of a long bone or other major trauma. Fat embolism syndrome reflects a serious systemic manifestation as a consequence to these emboli.

- Fat embolism is a common phenomenon. It is more commonly seen in patients with multiple fractures and in fractures involving lower limbs especially femur.
- Fat originates from the site of trauma, particularly from the injured marrow. Fat globules > 10 mm are considered significant.

Clinical Presentation

- Early warning signs are a slight rise in temperature and pulse rate (tachycardia)

The classical triad of fat embolism syndrome is:

1. Respiratory symptoms: Dyspnea or tachypnea.
2. Neurological symptoms: Confusion or disorientation.
3. Petechial rash: In axilla, neck, periumbilical area, conjunctiva of lower lid, front and back of chest, shoulder.
   - Fat 1 is rare in children.