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Quiz – 2014

Question I

The thread diameter of lag screw of dynamic hip screw (DHS) is:

- A) 1 cm.
- B) 2.2 cm.
- C) 1.65 cm.
- D) 1.25 cm.

Question II

Coxa Profunda would classically lead to:

- A) Pincer FAI.
- B) Cam FAI.
- C) Mixed FAI.
- D) Protrusio acetabuli.

Question III

What is the pathology seen in the following image of ankle arthroscopy.

Question IV

316L stainless steel used in orthopaedic implants contains:

- A) Iron, carbon, chromium, nickel.
- B) Iron, carbon, chromium, nickel, aluminium.
- C) Iron, carbon, lead, nickel.
- D) Iron, carbon, chromium, nickel, molybdenum.

Question V

Which of the following is not-true for SCIWORA?

- A) This is defined as spinal cord injury in patient with no visible fracture or dislocation on plain radiograph and CT scan both.
- B) More common in children under 8 years of age.
- C) Usually result from severe flexion or distraction injury
- D) Believed to occur because spinal cord is more elastic than spinal column in children.
- E) Complete spinal injuries are more common in younger children.

Answers

I: (D)

A few points on DHS Implant and technique:

Lag Screw: It has a Thread diameter – 12.5 mm, Pitch – 3 mm, Lead that is the distance travelled by screw with one turn – 18 mm. It has a Tapered thread at tip with Reverse cutting flutes for easy extraction.

Side Plates (Barrel plate): Barrel angle can vary from 150° to 130° in 5 degree increments.

Barrel length: Standard (38 mm) and short (25 mm).

Compression Screw: Used by some surgeons to produce additional compression of fragments 29 & 36 mm length.

DHS Angle Guide: a variable angle guide is available which matches the preoperative Caput-collum-diaphyseal angle (CCD), the angle between the neck and the shaft of the femur in the hip.

DHS Triple Reamer: reams for the lag screw, the barrel and the barrel plate junction.

Tips for successful DHS in intertrochanteric fracture: first and foremost you need to ask:

- 1) Is DHS a viable option for a particular intertrochanteric fracture?

This is because a DHS is based on tension band principle; it fixes the eccentrically loaded hip and needs an intact medial wall or reduced postero-medial cortical contact to have a long in vivo life. The lag screw of DHS fixing the Femoral head slides in the barrel of side plate along neck axis under functional load allowing gradual controlled compression of fracture site, which is so essential for fracture union in an acceptable position.

The lesser trochanter in intertrochanteric fracture is the key and fractures with WIDELY DISPLACED AND BIG lesser trochanter fragment are unstable and not good candidates for DHS fixation. Secondly the lateral wall should be more than 25 mm for DHS viability. Thirdly reverse oblique intertrochanteric fracture and fractures with subtrochanteric extension are not suitable fractures for DHS.

- 2) Next question you should ask yourself can you reduce this fracture maintaining a stable posteromedial contact on both Anteroposterior and Lateral view?

Reduction manoeuvre in most of the fracture cases require abduction and internal rotation but especially unstable/comminuted intertrochanteric fractures require external rotation and lifting of distal femoral shaft for reducing posterior sag.

- 3) You want to ask is can you insert the guide pin in the right place and to the right length and what is that right place and right length in the femoral head?

Insertion of Guide Pin is the MOST IMPORTANT surgical step and Ideally it should be in the patients anatomic CCD angle but 135° barrel plate angle is the best as it is convenient and it is at this angle that the guide wire of DHS can be inserted in the centre of femoral head (in both AP and lateral C-arm images) where the bone is strongest and to the greatest depth. Further though 150° is better as it is in the weight bearing axis which allows for good lag screw barrel slide without jamming. But it is not convenient to insert, increases chance of subtrochanteric fracture and avascular necrosis. Entry point of guide pin for 135° DHS on lateral cortex is at level of lesser trochanter/2.5–3 cm below vastus lateralis ridge. It is 5 mm distal for every 5 degree increase in sideplate angle.

- 4) So what is the correct length of the DHS lag screw?

Ideally it should be as deep in femoral head as possible. But 10 mm short of pilot length (from lateral cortex to subchondral bone) suffices. It should 5 mm short in case of osteoporotic bones. Tip-Apex distance ideally should be less than 24 mm and should be measured after correcting for magnification of the image.

- 5) How to do good reaming and tapping?

Ream under C-arm preferably, look for Guide wire progression and Continue until reamer bottoms out. If guide pin inadvertently gets withdrawn while reaming insert the centring sleeve in to the reamed hole and insert the lag screw in reverse fashion into it; then hammer the guide pin into it. Do not tap osteopenia bone and when zero mark of lag screw inserter aligns with lateral cortex Screw tip will be 10 mm from joint surface, but in osteopenic bone advance additional 5 mm for increased hold. Left side inter trochanteric fracture have been shown to have worse reduction than right as clockwise motion is used by right handed surgeons leading to anterior push on proximal fragment on left side and apposition on right side.

- 6) What is the optimum compression of fracture you should achieve peroperatively and how to do it?

It is better to ACHIEVE 70–80% compression peroperatively. Ways to achieve it are:

- 1) Normally 5 mm of fracture compression is achieved with insertion of compression if the lag screw of aforesaid standard length is used. If you want more use a lag screw 5 mm less but insert it to the desired depth in femoral head and achieve compression by using a compression screw which come in two sizes.
- 2) Loosen traction and impact the fracture with plastic impactor before inserting the shaft screws.
- 3) Axial compression is achieved by inserting screws in eccentric position in dynamic holes of side plate; first hole gives 1 mm and second hole 2 mm compression. 5 mm compression can be achieved with 5 hole plate.

- 7) Do we need to insert Compression screw?

This is controversial; it is not routinely used and surgeons remove it after achieving compression as elucidated above. One good indication for its insertion is if you cannot visualise the lag screw preoperatively in the barrel, insert compression screw. Secondly you must use it when a short barrel plate is used. Be careful NOT to strip the lag screw in osteopenic bone and do not use power! while inserting compression screw.

- 8) How many holes side plate to use?

Generally, for an A1.1 and an A1.2 fracture (AO classification), a two-hole DHS plate is enough.

A four-hole DHS plate for type A 2.

- 9) When to use Short or long barrel side plate?

Standard or long barrel is always better biomechanically. But following are some interesting figures for optimum functioning DHS which need to be considered to know when to use short barrel.

- There should be minimum of 20 mm slide possible (the distance between thread and barrels proximal portion).
- 32 mm is the lag screw thread length.

- Minimum of 22 mm barrel screw engagement is required for stable lag screw barrel mating without jamming.

So all this amounts to $20 + 32 + 22 \text{ mm} = 74 \text{ mm}$; so if you use lag screw of less than or equal to 75 mm use short barrel side plate (though literature varies from 75–80 mm cutoff).

- 10) Before closing an intertrochanteric fracture patient fixed with DHS what 2 thing to do?
 - Fix posteromedial lesser trochanter fragment.
 - C-arm for reduction and compression.
- 11) What is lateral wall in a case of intertrochanteric fracture why is it important and what are the options to fix unstable intertrochanteric fractures?

Can be measured preoperatively on x-rays or preoperatively from the insertion point of guide pin to the distal most fracture line. Should be more than 25 mm for DHS to be successful. If lateral wall is comminuted or less than 25 mm use trochanteric stabilizing plate, intramedullary devices. Use cement or locking plate DHS for pathological/osteoporotic intertrochanteric fractures.

II: (A)

Femoroacetabular impingement is a leading cause of osteoarthritis of the hip in young and active patients. The basic pathology in FAI is an early contact during hip joint motion

between skeletal prominences of the acetabulum and the femur, especially during flexion and internal rotation. There are three types of impingement syndromes: Pincer impingement is the acetabular cause of femoroacetabular impingement and is characterized by focal or general overcoverage of the femoral head. Cam impingement is the femoral cause of femoroacetabular impingement and is due to an aspherical portion of the femoral head–neck junction. Majority of the patients have a combination of both forms of impingement, which is called “mixed type”.

In a normal hip the acetabular fossa lies lateral to the ilioischial line on an AP view. Coxa profunda is said to occur when the floor of the acetabuli touches or crosses over the ilioischial line medially. Protrusio acetabuli occurs when part of the femoral head is crosses over the ilioischial line medially.

The coverage of acetabulum can be quantified by the centre edge angle; an angle less than 25 degrees would indicate an acetabular dysplasia whereas one above 40 degrees would point towards acetabular overcoverage.

However, recently the concept of Coxa Profunda leading to pincer-type of impingement has been questioned. It has been found that Coxa profunda is seen in a variety of hip disorders and asymptomatic hips and therefore should be considered a normal radiographic finding, at least in females.

Nepple JJ, Lehmann CL, Ross JR, Schoenecker PL, Clohisy JC. Coxa profunda is not a useful radiographic parameter for diagnosing pincer-type femoroacetabular impingement. *J Bone Joint Surg Am.* 2013 Mar 6;95(5):417–423. <http://dx.doi.org/10.2106/JBJS.K.01664>.

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III

Negatively brief ringent crystal of mono sodium urate in a patient with gout.

Gout and pseudogout are the most common crystal-induced arthropathies. Gout is caused by monosodium urate monohydrate crystals; pseudogout is caused by calcium pyrophosphate crystals.

- 1) How to diagnose gout?

For typical presentations of gout (such as recurrent podagra with hyperuricaemia) a clinical diagnosis alone is reasonably accurate but not definitive without crystal confirmation. The gold standard is Joint aspiration and synovial fluid analysis for typical crystals. The important thing is Crystalline arthritis and infectious arthritis can coexist or be the antecedent cause. Consequently, in patients with acute monoarticular arthritis/suspected gout, send synovial fluid for Gram stain and culture and sensitivity. During acute attacks, the synovial fluid is inflammatory, with a WBC count higher than $2000/\mu\text{L}$ & possibly higher than $50,000/\mu\text{L}$, with a predominance of polymorphonuclear neutrophils, though low WBC counts are occasionally found.

2) How do the crystal of gout appear?

In gout, crystals of monosodium urate (MSU) appear as needle-shaped/tooth pick intracellular and extracellular crystals. When examined with a polarizing filter and red compensator filter, they are yellow as in the photograph above when aligned parallel to the slow axis of the red compensator but turn blue when aligned across the direction of polarization (i.e., they exhibit negative birefringence). Negatively birefringent urate crystals are seen on polarizing examination in 85% of specimens.

3) What are differential diagnosis of these crystals?

Microscopic analysis in pseudogout shows calcium pyrophosphate (CPP) crystals, which appear shorter than MSU crystals and are often rod shaped with blunt ends or rhomboidal. Under a polarizing filter, CPP crystals change colour depending upon their alignment relative to the direction of the red compensator. They are positively birefringent, appearing blue when aligned parallel with the slow axis of the compensator and yellow when perpendicular. Crystals must be distinguished from birefringent cartilaginous or other debris. Debris may have fuzzy borders and may be curved, whereas crystals have sharp borders and are straight. Corticosteroids injected into joints have a crystalline structure that can mimic either MSU or CPP crystals. They can be either positively or negatively birefringent. The sensitivity of a synovial fluid analysis for crystals is 84%, with a specificity of 100%. As alkalization reduces uric acid crystal solubility and the enzyme uricase can “dissolve” these crystals, reduction by addition of sodium hydroxide or uricase to suspected gout crystal can be helpful.

4) What if fluid is negative for crystals?

Crystals may be absent very early in a flare. Although the sensitivity of this test is inferior, aspiration of synovial fluid from previously inflamed joints that are not currently inflamed or in subsequent flare may reveal urate crystals. Such crystals are generally extracellular.

5) Do we need to do repeat aspiration of joints?

Once a diagnosis of gout is established by confirmation of crystals, repeat aspiration of joints with subsequent flares is not necessary unless infection is suggested or the flare does not respond appropriately to therapy for acute gout.

6) Is measurement of serum uric acid important?

Measurement of serum uric acid is the most misused test as presence of hyperuricemia in the absence of symptoms is not diagnostic of gout. In addition, as many as 15% of patients with symptoms from gout may have normal serum uric acid levels at the time of their attack. Situations that decrease uric acid levels can trigger attacks of gout. In such cases, the patient's medical records may reveal prior elevations of uric acid. The level of serum uric acid does correlate with the risk for developing gout and repeat flares. The 5-year risk for developing gout is approximately 0.6% if the level is below 7.9 mg/dL, 1% if it is 8–8.9 mg/dL, and 22% if it is higher than 9 mg/dL.

7) Is measurement of urine uric acid important?

Renal uric acid excretion should be determined in selected gout patients, especially those with a family history of young onset gout, onset of gout under age 25, or with renal calculi. A 24-hour urinary uric acid evaluation is generally performed if uricosuric therapy is being considered. If patients excrete more than 800 mg of uric acid in 24 h while eating a regular diet, they are overexcretors and thus overproducers of uric acid. These patients (approximately 10% of patients with gout) require allopurinol instead of probenecid to reduce uric acid levels. Furthermore, patients who excrete more than 1100 mg in 24 h should undergo close renal function monitoring because of the risk of stones and urate nephropathy. Kidney and liver function test, blood sugar levels and lipid profile also need to be done.

8) What are the classical features of bone erosion on X-ray?

The radiologic features in soft tissue, bone and joints of patients with chronic tophaceous gout are not usually seen until 6–12 years after the initial attack. The soft tissue findings consist of calcific deposits and eccentric juxta-articular lobulated masses (hand, elbow, knee, ankle and foot). Bone findings include “mouse bites” from erosion of long-standing soft-tissue tophi, bone infarction, “punch-out” lytic bone lesions and sclerosis of margins. Juxta-articular Erosions with overhanging edges are considered pathognomonic for gout, Maintenance of the joint space, Absence of periarticular osteopenia, Location outside the joint capsule, Sclerotic (cookie-cutter, punched-out) borders. Asymmetric distribution among the joints, with a strong predilection for distal joints, especially in the lower extremities.

9) Is there any role of MRI?

MRI with gadolinium is recommended when tendon sheath involvement must be evaluated and when osteomyelitis is in the differential diagnosis.

10) What are the typical clinical features and course of gout?

Gout usually presents as acute episodic inflammatory monoarticular arthritis, but may also manifest as chronic arthritis of 1 or more. In acute attacks the rapid development of severe pain, swelling, and tenderness that reaches its maximum within just 6–12 h, especially with overlying erythema, is highly suggestive of crystal inflammation though not specific for gout. Symptoms in gout are Podagra (1st MTP joint – initial joint manifestation in 50% of gout cases and eventually involved in 90%), Arthritis in other sites the instep, ankle, wrist, finger joints, and knee; in pseudogout, large joints (e.g., the knee, wrist, elbow, or ankle). Monoarticular involvement is more common, though polyarticular acute flares (10%) are not rare, and many different joints may be involved simultaneously or in succession. In acute gout, attacks have maximum intensity within 8–12 h; in pseudogout, attacks resembling those of acute gout or a more insidious onset that occurs over several days. Without treatment, symptom patterns that change over time; attacks can become

more polyarticular, involve more proximal and upper-extremity joints, occur more often, and last longer mimicking rheumatoid arthritis.

Tophi are a pathognomonic feature of gout and are mainly found in articular, periarticular, bursal, bone, auricular, and cutaneous tissues. Renal manifestations of gout include urolithiasis, typically occurring with an acidic urine pH and rarely Chronic interstitial nephropathy.

11) How does it present in elderly people?

Elderly women, particularly women with renal insufficiency who are taking a thiazide diuretic, can develop polyarticular arthritis as the first manifestation of gout. These attacks may occur in coexisting Heberden and Bouchard nodes. Such patients may also develop tophi more quickly, occasionally without prior episodes of acute gouty arthritis.

12) What are the 3 principles of managing gout?

- Treating the acute attack
- Providing prophylaxis to prevent acute flares
- Lowering excess stores of urate to prevent flares of gouty arthritis and to prevent tissue deposition of urate crystals.

13) Do we need to treat asymptomatic hyperuricemia?

As a general rule, asymptomatic hyperuricemia should not be treated. Patients with levels higher than 11 mg/dL who overexcrete uric acid are at risk for renal stones and renal impairment; may be candidates for therapy.

14) Is dietary or activity modification important?

Overall, purine restriction generally reduces serum uric acid levels by no more than 1 mg/mL, with modest impact, and diets with very low purine content are not palatable. Diet modifications alone are rarely able to lower uric acid levels sufficiently to prevent accumulation of urate, but they may help lessen the triggers of acute gout attacks. Purines are found in all protein foods. All sources of purines cannot and should not be eliminated. High-purine foods should be either avoided or consumed only in moderation. Foods very high in purines include organ meats such as sweetbreads, sardines, and mussels. Foods moderately high in purines include mutton, liver, bacon, salmon, kidneys, and turkey.

Patients should avoid excess ingestion of alcoholic drinks (>2/day in male and >1/day servings in female), particularly beer. Moderate wine intake is not associated with increased development of incident gout, but excesses of any form of alcohol in gout patients are associated with acute gout flares. Patients should avoid sodas and other beverages or foods sweetened with high-fructose corn syrup. They should also limit their use of naturally sweet fruit juices, table sugar, and sweetened beverages and desserts, as well as table salt. Patients taking colchicine should avoid grapefruit and grapefruit juice.

Maintaining a high level of hydration with water (at least 8 glasses of liquids per day) may be helpful in avoiding attacks of gout. In view of the association of gout with atherosclerosis,

advise a low-cholesterol, low-fat diet if such a diet is otherwise appropriate for the patient. Weight reduction in patients who are obese can improve hyperuricemia. Ketosis-inducing diets (e.g., fasting) should be avoided. The patient should avoid trauma to the affected joint; otherwise, they should be active.

15) How to medically manage gout as an orthopaedician?

The temptation to treat patients without a proven diagnosis must be resisted. Septic arthritis may clinically resemble gout or pseudogout, and unrecognized septic arthritis can lead to loss of life or limb. Acute treatment of proven crystal-induced arthritis is directed at relief of the pain and inflammation. Nonsteroidal anti-inflammatory drugs (NSAIDs) are the first line drugs though corticosteroids, colchicine, and adrenocorticotrophic hormone (ACTH) are other treatment options. The choice is based primarily on whether the patient has any concomitant health problems (eg, renal insufficiency or peptic ulcer disease). Supplement with topical ice if required.

16) Which NSAID to use?

Although indomethacin is the NSAID traditionally chosen for acute gout, most of the other NSAIDs can be used as well. Select an agent with a quick onset of action. Do not use aspirin, because it can alter uric acid levels and potentially prolong and intensify an acute attack. Cyclooxygenase-2 (COX-2) inhibitors may require higher dosages than are typically used. To control the acute attack, NSAIDs are prescribed at full dosage for 2–5 days. Once the acute attack is controlled, the dosage is reduced to approximately one half to one fourth of that amount. Taper the dosage over approximately 2 weeks. Gout symptoms should be absent for at least 2 days before the NSAID is discontinued.

17) Is colchicine test pathognomonic and what is its role today?

Clinical response to colchicine is not pathognomonic for gout. Colchicine, a classic treatment, is now rarely indicated because of its narrow therapeutic window and risk of toxicity. To be effective, colchicine therapy is ideally initiated within 36 h of onset of the acute attack. Newer recommendations trend toward lowered daily and cumulative doses rather than classic hourly dosing regimens which are no longer recommended. The regimen currently favoured consists of 1.2 mg of colchicine, followed by 0.6 mg 1 hour later to initiate treatment of the early gout flare. Colchicine should also be avoided in patients with compromised renal function, hepatic dysfunction, biliary obstruction, or an inability to tolerate diarrhoea.

18) What is the role of intraarticular steroid?

When comorbid conditions limit the use of NSAIDs or colchicine, and in severe monoarticular attack; a preferred option may be an intraarticular steroid injection, particularly when a large, easily accessible joint is involved. Septic arthritis must be reasonably excluded.

19) Should allopurinol be started during acute attack?

Therapy to control the underlying hyperuricemia generally is contraindicated until the acute attack is controlled (unless kidneys are at risk because of an unusually heavy uric acid load). Starting therapy to control hyperuricemia during an acute attack may intensify and prolong the attack. If the patient has been on a consistent dosage of probenecid or allopurinol at the time of the acute attack, however, the drug should be continued at that dosage during the attack.

20) Is there any role of combination therapy in acute attack?

If the patient does not have an adequate response to initial therapy with a single drug, ACR guidelines advises that adding a second appropriate agent is acceptable. Using combination therapy from the start is appropriate for an acute, severe gout attack, particularly if the attack involves multiple large joints or is polyarticular.

21) What is hyperuricemia and Does every person need uric acid lowering drug after acute attack?

This is controversial but there are some rules. Hyperuricemia, which is variably defined as a serum urate level greater than either 6.8 or 7.0 mg/dL. Control of hyperuricemia generally is not pursued for a single attack but do advise him on lifestyle/dietary modifications and treatment of comorbidities as hyperlipidaemia, hypertension, hyperglycaemia. If the first attack is not severe, however, some rheumatologists advocate waiting for a second attack before initiating such therapy; not all patients experience a second attack, and some patients may require convincing that they need life-long therapy.

The risk of a second attack of gout after the first attack is 62% after 1 year, 78% after 2 years, and 93% after 10 years. The decision to begin therapy depends:

- a) On the baseline serum uric acid levels (>9 mg/dL denotes a higher risk for recurrent gouty arthritis and tophi) on follow-up of an acute attack,
- b) Patients with 1 or more tophi on clinical examination or imaging study
- c) Frequent attacks of acute gouty arthritis (≥ 2 attacks per year).
- d) X-ray changes of gout.
- e) Renal disease is present.

22) What is the therapeutic goal of uric acid lowering drugs?

The therapeutic goal of urate lowering therapy is to promote crystal dissolution and prevent crystal formation. This is achieved by maintaining the serum uric acid below the saturation point for monosodium urate ($\leq 360 \mu\text{mol/l}$ or $\leq 6 \text{ mg/dl}$).

23) In what dose to give uric acid lowering drugs (ULT)?

Allopurinol is an appropriate first line long-term urate lowering therapy. It should be started at a low-dose (100 mg daily and 50 mg/d in kidney disease) and increased by 100 mg every two to four weeks if required till therapeutic goal is

achieved which can exceed 300 mg/day (maximum 800 mg/d) even in kidney dysfunction – “go low, go slow” strategy. If allopurinol toxicity occurs, options include other xanthine oxidase inhibitors (febuxostat in renal impaired patient), a uricosuric agent, or allopurinol desensitisation (the latter only in cases of mild rash). Uricosuric agents such as probenecid and sulphinpyrazone are both effective but probably inferior to allopurinol in lowering SUA. They should not be used in patients with renal impairment/ urolithiasis.

24) How frequently to measure SUA level on ULT?

Initially every 2–3 weeks when ULT dose is titrated. Later on 6 monthly is required.

25) Can use of uric acid lowering drugs precipitate acute attack of gout?

Because allopurinol, febuxostat, and probenecid change serum and tissue uric acid levels, they may precipitate acute attacks of gout. To reduce this undesired effect, colchicine or low-dose NSAID treatment is provided for at least 6 months. In patients who cannot take colchicine or NSAIDs, low doses of prednisone can be considered. When used prophylactically, colchicine can reduce such flares by 85%. Patients with gout may be able to abort an attack by taking a single colchicine tablet at the first twinge of an attack

26) Side effects of allopurinol?

Allopurinol hypersensitivity reactions may develop, which can be severe or fatal. Because a skin rash may progress to a severe hypersensitivity reaction, patients who develop a skin rash should discontinue allopurinol. Hepatotoxicity, bone marrow depression, and interstitial nephritis are rare but serious.

27) How long to give these drugs?

After diagnosis and treatment of an acute gouty arthritis episode, the patient should return for a follow-up visit in approximately 1 month to be evaluated for therapy to lower serum uric acid levels. If patient has been started on ULT and target level of 5–6 mg/dL is achieved and maintained, patients can be seen every 6–12 months and their serum uric acid monitored. ULT Drugs or lifestyle modifications are continued indefinitely after gout resolves to maintain target SUA. Concomitant Attack prophylaxis with ULT is given for 3 month if there was no tophi initially and 6month in patient with tophi after target level SUA is achieved.

28) Are there any new drugs?

Pegloticase is a recombinant porcine-like uricase & is not recommended as first-line ULT agent for any case scenarios. Pegloticase is appropriate for patients with severe gout disease burden and refractoriness to, or intolerance of, conventional and appropriately dosed ULT. IL-1 inhibitors such as canakinumab and rilonacept are useful alternatives for preventing flares.

29) Is Surgical Treatment of Gout indicated?

Surgical interventions become inevitable when the overlying soft tissue of tophus ulcerates and (or) becomes infected. Surgery may also be indicated for tophaceous complications, including joint deformity, compression (e.g., cauda equina or spinal cord impingement), and intractable pain. Tophi involving metaphyses can be curetted. The risk of overlying skin necrosis and delayed wound healing are important issues in (50%) conventional surgical debridement of tophi. A minimally invasive approach to managing tophi reportedly minimizes wound complications. Arthroscopic shaver technique to avoid poor wound healing has also been advocated.

H. Ralph Schumacher, Mark Robbins, Neil Wenger, And Robert Terkeltaub. 2012 American College of Rheumatology guidelines for management of gout. Part 2: systematic non-pharmacologic and pharmacologic therapeutic approaches to hyperuricemia. *Arthritis Care Res (Hoboken)*. 2012 Oct;64(10):1447–1461.

SUMMARY: ACUTE ATTACK (DIAGNOSE PROPERLY, RULE OUT SEPTIC, GET SUA, KFT, LFT LIPID PROFILE; YOUNG PATIENT-MYELO/LYMPHOPROLIFERATIVE DISORDER; DRUG USE) -----

↓
NSAIDS IN FULL DOSE +ICE; THAN LOWER IT----THINK OF INTRAARTICULAR STEROID, NO ALCOHOL

↓
STOP NSAID WHEN ACUTE ATTACK SUBSIDES AND SUA NORMAL FOR 2DAYS----

↓
FOLLOW UP AT 1MONTH

← NEED FOR ULT IF SUA>9, >1 TOPHI,

→ add dietary and lifestyle modification
in all to prevent flares.

Recurrent attack>2/year, renal impairment.

But cover with attack prophylaxis- NSAIDS/COLCHICINE0.6mg

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tents higher than those of steel make an alloy commonly called pig iron that is brittle and not malleable. Unprotected carbon steel rusts readily when exposed to air and moisture. So to decrease corrosion stainless steel was designed; Stainless steel, is a steel alloy with a minimum of 10.5% chromium content by mass. Stainless steels contain sufficient chromium to form a passive film of chromium oxide (passivation), which prevents further surface corrosion by blocking oxygen diffusion to the steel surface and blocks corrosion from spreading into the metal's internal structure, and due to the similar size of the steel and oxide ions they bond very strongly and remain attached to the surface. Passivation only occurs if the proportion of chromium is high enough and oxygen is present. Stainless steel does not readily corrode, rust or stain with water as ordinary steel does, but despite the name it is not fully stain-proof, most notably under low oxygen, high salinity (human body), or poor circulation environments. So for such hostile environments Nickel was added. The first stainless steel used for implants contained ~18wt% Cr and ~8wt% Ni makes it stronger than the steel and more resistant to corrosion. Nickel stabilizes steel and

makes steels virtually non-magnetic and less brittle at low temperatures (better resistance to stress-corrosion cracking). Further addition of molybdenum (Mo) has improved its corrosion resistance (chloride pitting and crevice corrosion) to chlorine environment in body, known as type 316 stainless steel. Afterwards, the carbon (C) content has been reduced from 0.08 to 0.03 wt% which further improved its corrosion resistance to chloride solution, and named as 316L. The “L” means that the carbon content of the alloy is below 0.03%, which reduces the sensitization effect (precipitation of chromium carbides at grain boundaries) caused by the high temperatures involved in welding. Grade 316LVM is preferred where biocompatibility is required (such as body implants, VM-vacuum melting).

V: (D)

Spinal cord injury in children often occurs without evidence of fracture or dislocation. The mechanisms of neural damage in this syndrome of spinal cord injury without radiographic abnormality (SCIWORA) include flexion, hyperextension, longitudinal distraction, and ischaemia. Inherent elasticity of the vertebral column in infants and young children, among other age-related anatomical peculiarities, render the paediatric spine exceedingly vulnerable to deforming forces. The neurological lesions encountered in this syndrome include a high incidence of complete and severe partial cord lesions. Children younger than 8 years old sustain more serious neurological damage and suffer a larger number of upper

cervical cord lesions than children aged over 8 years. Of the children with SCIWORA, 52% have delayed onset of paralysis up to 4 days after injury, and most of these children recall transient paresthesia, numbness, or subjective paralysis. Management includes tomography and flexion-extension films to rule out incipient instability, and immobilization with a cervical collar. Delayed dynamic films are essential to exclude late instability, which, if present, should be managed with Halo fixation or surgical fusion. The long-term prognosis in cases of SCIWORA is grim. Most children with complete and severe lesions do not recover; only those with initially mild neural injuries make satisfactory neurological recovery.

Pang D, Wilberger JE Jr. Spinal cord injury without radiographic abnormalities in children. *J Neurosurg* 1982; 57:114.

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