Exam Corner

The Bone & Joint Journal

June 2014 • Answers

Authors: Mr. Emmet Griffiths and Mr. Vikas Khanduja
SpR, East of England

MCQs and EMQs

1. Which of the following accounts for 65% to 80% of the dry mass of flexor tendons?
   Answer: a. Collagen type I

2. With regards to clinical tests for diagnosing pathology in the hand, please select the correct response for each of the following from the list given below
   Answer:
   a. Rigid extension of the distal interphalangeal joint when the patient is asked to extend the middle phalanx against resistance **Boutonniere deformity**
   b. Less interphalangeal flexion when the metacarpo-phalangeal joints are hyperextended as compared with when the metacarpo-phalangeal joints are flexed **Intrinsic plus hand**
   c. Metacarpal grind test **Carpometacarpal arthritis**

   Elson's test as described above demonstrates that with a central slip rupture in Boutonniere deformity the only extension to occur is via the lateral bands, hence the DIPJ becomes rigidly extended. Intrinsic plus hand occurs when spastic intrinsics overcome weak extrinisics in the hand resulting in less flexion at the PIPJ with the MCPJ in extension compared with MCPJ flexion.
   Carpometacarpal Grind test involves griping the metacarpal and using axial compression to compress the CMCJ. If pain is reproduced this is a positive test.

3. Sepsis is a serious medical condition caused by an overwhelming immune response to infection. Which one of the following is NOT true about severe sepsis?
   Answer: c. Initial serum lactate of < 2 mmol/L

   Sepsis is defined as Systemic inflammatory response (SIRS) with infection (SIRS criteria is met if two or more are present: Temperature > 38°C or < 36°C, Pulse > 90 beats/min, Respiratory Rate (RR) > 20 or PaCO2 < 4.3 kPA, WBC > 12000 or < 3000/mm3, Acutely Altered Mentation, Blood glucose > 6.6). Lactate is prognosic for sepsis. Severe sepsis is defined as sepsis with a lactate > 2 mmol/L

4. You have seen a patient with a wound infection to his knuckle after a human bite. Which organism is most commonly associated with this type of an injury?
   Answer: a. Eikenella corrodens

   Gram-negative anaerobic bacillus which is commensal in the human mouth. Pasturella mulocida is commonly seen in cat bites. Acromonas hydrophillia is seen with exposure to fish or amphibians. Pseudomonas aeruginosa is seen in immunocomprimised individuals.

5. A 25-year-old patient involved in a polytrauma is in the intensive care unit following intramedullary nailing for bilateral femoral fractures and an emergency laparotomy after being involved in a road traffic accident. He has significant bleeding from his wound sites, and develops widespread bruising, gangrene of his digits and multi-organ failure. Which one of the following is the most likely pathogenesis?
   Answer: a. Widespread release of tissue factor with activation of intrinsic and extrinsic coagulation pathways

   This patient has developed disseminated intravascular coagulation. The result of activating intrinisic and extrinsic pathways is two -fold. Firstly, thrombi form throughout the microvascaular system, secondly, this results in depletion of coagulation factors and platelets causing bleeding.

Vivas

Adult Pathology

A 45-year-old male presents with left shoulder pain following a fall while intoxicated. These are his radiographs and CT obtained in A & E (Figs. 1a-c)

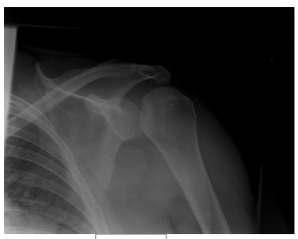


Fig. 1a

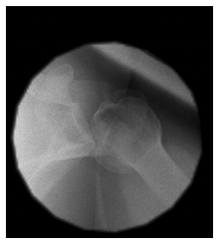


Fig. 1b

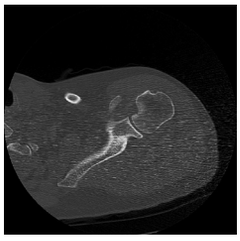


Fig. 1c

1. What is the diagnosis and what is the mechanism of injury?
   Answer: Acute traumatic left posterior shoulder dislocation on a background of chronic recurrent dislocations. Traumatic posterior dislocations occur during a high-energy injury if an axial force is applied with the shoulder in a position of internal rotation, forward elevation, and adduction. Traumatic dislocation may also be sustained as a result of sustained contraction of the internal rotators during a seizure that occurs with the arm at the side.

2. What does the CT scan show and how is it caused?
   Answer: Well-reduced left glenohumeral joint with an osteochondral impression fracture (also termed an encoche fracture or a reverse Hill-Sachs lesion). It is produced as the anterior aspect of the humeral head impacts on the posterior aspect of the glenoid. If the shoulder is not relocated at an early stage, the head defect enlarges and becomes corticated as a result of the grinding effect of rotational movements. With prolonged dislocation, secondary deformity of the articular cartilage of the humeral head may develop. This "ping-pong-ball" effect is due to resorption of the subchondral bone of the humeral head when it is not subjected to physiologic loading.

3. List the potential complications?
   Answer: Irreducible (locked) dislocation; acute re-dislocation; recurrent posterior instability; fractures of the glenoid rim or proximal humerus; nerve palsy (axillary nerve most common); chronic pain; joint stiffness; shoulder weakness or deformity; osteonecrosis; post-traumatic osteoarthritis; rotator cuff tear; functional incapacity; a complication of an ancillary stabilization procedure or treatment with an arthroplasty.

4. List the operative options for the defect demonstrated in the CT.
   1. McLaughlin procedure: transfer of the tendon of subscapularis into the Hill-Sachs defect
   2. Modified McLaughlin procedure: transfer of the osteotomised lesser tuberosity with the attached subscapularis into the Hill-Sachs defect
   3. Rotational osteotomy of the humerus.
   4. Disimpaction, elevation and bone grafting (autologous or allograft) of the depressed osteochondral humeral head fracture
   5. Osteochondral humeral or femoral head allograft replacement of the defect
   6. Posterior Bankart repair (opemn or arthroscopic)
   7. Hemicap hemiarthoplasty
   8. Hemiarthoplasty (or total shoulder replacement if there is degenerate change)

Trauma

A 75-year-old lady presents with an injury to her right hip following a heavy fall after tripping over a kerb. These are her radiographs obtained following the fall (Fig. 2).

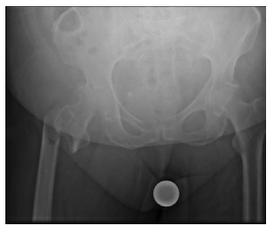


Fig. 2a

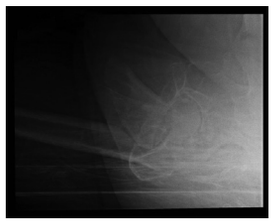


Fig. 2b

1. Describe the radiographs.
   Answer: Plain AP radiograph centred on both hips and lateral radiograph of the right hip demonstrating an oblique subtronchanteric fracture of the right proximal femur. Notably there is an area of sclerosis around the medial cortex. The fracture site itself has a rounded appearance. The fracture is displaced laterally with rotation. On the lateral there is flexion of the proximal fragment. There is also osteoarthritic change to both hips, most prominent in the right hip with loss of joint space, osteophytes, sclerosis and subchrondral cyst formation.

2. What is the association between arthritis of the hip and the type of hip fractures?
   Answer: Due to the reduced range of movement associated with arthritic change creating a relatively rigid lever arm through the femoral head and neck, intertrochanteric and subtronchanteric fractures are much more common than subcapital femoral fractures.

3. What is the rationale behind this pattern of injury?
   Answer: The sclerotic margin and rounded edges of this fracture point towards a bisphosphonate fracture. These patients have generally been taking bisphosphonates greater than one year. The action of bisphosphonates blocks osteoclastic bone breakdown resulting in an increase in microtrabecular defects creating areas of weakness or stress lines. They are most commonly seen in the subtrochanteric region of the proximal femora but can occur in other areas.

4. How would you manage this patient?
   Answer: Full history and examination including history of hip pain prior to injury, weight loss, systemic symptoms, current medications.
   Fixation of fracture with a long intramedullary nail and fixation into the femoral neck or a toal hip replacement to manage osteoarthritis at the same time.

5. What are the potential difficulties that you would envisage during surgery in this case?
   Answer:
   Positioning: The patient has arthritic hips and therefore positioning of the contralateral leg may pose problems in gaining adequate imaging.
   Intra-operative: Displaced short oblique fractures can be difficult to reduce intra-operatively for closed intramedulllary fixation due to the pull of muscular attachments. The iliopsoas flexes the hip with its attachment to the lesser trochanter. Distally the shaft is externally rotated by the gluteus maximus. It is not uncommon for a varus deformity to remain following reduction of hip flexion. Maintaining reduction will also be difficult as the fracture may be too transverse for a reduction forceps. If a hip replacement is planned then potential difficulty can be encountered during the neck osteotomy, hip dislocation, and reduction and also stabilisation of the fracture.
   Post-operative: Mobilisation of the patient will be challenging due to the arthritic hip above the fracture Bisphosphonates have been linked with reduced callus formation on bone healing.

Hands

A seven-year-old girl fell off her horse and sustained a closed postero-lateral dislocation of her left dominant elbow. She under went a closed reduction under general anaesthesia within six hours of injury. In the review clinic one week later she was noted to have difficulty in bending the tip of her left thumb and index finger. There was no associated numbness in her hand (Figs. 3a-c).

Fig. 3a

Fig. 3b

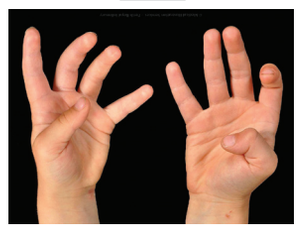


Fig. 3c

1. Describe the clinical photographs?
   Answer: The photographs show the inability to flex the thumb interphalangeal joint and index finger distal interphalangeal joint of the left hand. This indicates loss of function of the flexor pollicis longus of the left thumb and flexor digitorum profundus of the index finger (Figs 1 and 2).
   In addition there is inability to approximate the tip of thumb and index finger to make an 'O' on the left hand (instead a triangle is formed) as compared with the other side (Fig. 3). This is sometimes referred to as 'Kiloh-Nevin sign' where the patients with anterior introsseous nerve (AIN) palsy due to

inability to flex the interphalangeal joint of thumb and distal interphalangeal joint of the index finger cannot not make a complete 'O' and instead compose a triangle.

2. What do you think is the probable diagnosis?
   Answer: The loss of function of the flexor pollicis longus of the left thumb and flexor digitorum profundus of the index finger without associated numbness in the distribution of median nerve in this case indicates that this is a case of AIN palsy. Alternatively, loss of sensation in the median nerve distribution along with these motor deficits would have pointed towards a median nerve lesion which is more commonly seen with elbow dislocation.

3. What is the aetiology of this condition?
   Answer: Most commonly an associated lesion of AIN palsy with elbow dislocation is a neurapraxia following a traction injury. However, it is important to rule out entrapment of the median nerve in the elbow joint.
   Bilateral non traumatic AIN palsy would indicate 'Parsonage-Turner syndrome' or brachial plexus neuritis.

4. How would you further manage this problem?
   Answer: In the present case there are no 'worrying symptoms and signs' of entrapped nerve hence the management would be close observation of the neurological status of the patient with mobilisation of her elbow to avoid stiffness.
   Suspicion of nerve entrapment should be raised in cases with difficult reductions, intense pain following successful reduction, the deterioration of neurological deficit or an increase in joint space on post-reduction radiographs. Cases where nerve is suspected an urgent MRI should be ordered to define the location of the nerve prior to exploration.
   Signs of recovery would be indicated by some evidence of movement in the flexor pollicis longus of the left thumb and flexor digitorum profundus of the index finger.
   No signs of improvement by four to six weeks should prompt further investigation in form of nerve conduction tests to define the lesion. An MRI or CT scan may be requested in cases where entrapment of the nerve by soft-tissue or fracture callus is suspected. Depending on the results of these investigations a referral should be made to the local peripheral nerve injury unit for advice on further management.

5. What do you know about the prognosis of this injury?
   Answer: Neurapraxia of AIN following elbow dislocation recovers satisfactorily. Cases of delayed onset palsy due to entrapment in the fracture callus need exploration with neurolysis of the nerve with good functional results unless due prolonged delays there are intrinsic changes in the nerve itself and the muscle units it supplies.

Children's Orthopaedics

Have a look at this radiograph (Fig. 4).

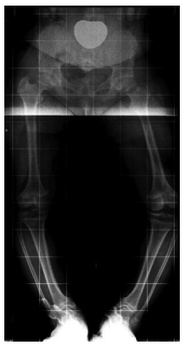


Fig. 4

1. What condition does this 11-year-old have?
   Answer: Achondroplasia

2. What are the typical musculoskeletal abnormalities evident on this radiograph?
   Answer: Relative fibular overgrowth causes varus deformity of the proximal and distal tibial joint lines. Lateral collateral ligament laxity at the knee causes a dynamic varus with lateral thrust at the joint (seen here as opening of the lateral compartment). Relative trochanteric overgrowth, with coxa vara is seen in the proximal femora. Tombstone iliac wings and a Champagne glass pelvic inlet are also characteristic of Achondroplasia.

3. What are the other orthopaedic manifestations of this condition?
   Answer: In the upper limb, there may be flexion deformities of the elbows, with posterior bowing of the ulna and also dislocation of the radial heads. The trident hand (2nd, 3rd and 4th digits separated and equal in length) is characteristic. The vertebrae are also affected and can cause spinal pathology such as spinal stenosis and thoracolumbar kyphosis.

4. What are the non-orthopaedic problems associated with this condition?
   Answer: Foramen Magnum stenosis can cause cervical cord compression. Communicating hydrocephalus; typical achondroplastic facies include frontal bossing and midface hypoplasia. These children may also have developmental delay and respiratory problems such as sleep apnoea.

5. What is the genetic mutation responsible for this condition, and what effect does this have?
   Answer: Achondroplasia is caused by a single point mutation is in the gene encoding the Fibroblast Growth Factor Receptor 3 (FGFR3). The FGFR3 is believed to regulate bone growth by limiting enchondral ossification. Two mutations in the FGFR3 gene are responsible for 99% of cases of achondroplasia. These mutations lead to prolonged receptor activation after ligand binding, and resulting in excessive growth limitation. Soluble FGFR3 has successfully been used in mice to act as a decoy for FGF in order to restore normal growth in achondroplasia.

6. What is the mode of inheritance of this condition?
   Answer: It is Autosomal Dominant

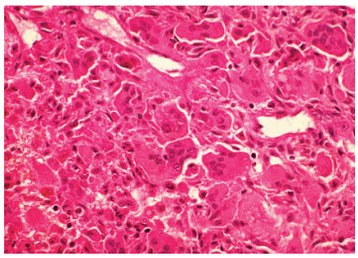
7. This child's parents do not have this condition. How is this possible?
   Answer: A large proportion (80%) of cases occur due to a spontaneous mutation.

8. What are the general principles of limb reconstruction in these patients?
   Answer: It is widely accepted that deformity should be corrected in order to restore the mechanical axis of the lower limbs, either by guided growth or osteotomy. Stature lengthening for achondroplasia is a controversial issue and should be performed only at the patient's wishes. There is evidence that lower limb lengthening improves the patient's quality of life, but as the goal of treatment should be a final height over 5 feet, multiple repeated lengthening procedures are required. Lengthening of the humerii should also be considered.
   Pre-operatively, one should clinically and radiologically screen for spinal stenosis, as this may be exacerbated by surgical lower limb lengthening.
   In general, bone and soft-tissue lengthens more easily in achondroplasia than in congenital longitudinal deficiencies,

allowing greater lengthenings per segment per procedure (up to 10 cm or 40%). The regenerate bone forms and consolidates faster than normal in achondroplasia, and so premature consolidation is a possible complication. A recent study has suggested that lengthening procedures result in reduced growth at the distal femoral and proximal tibial physes.

Basic Science

1. What stain has been used (Fig. 5) and how is it performed?



Answer: H&E Stain (Haematoxylin and eosin). This is one of the most common histology preparations. Haematoxylin (Haematin and Alunminium) is applied to the slide, this dyes nuclei blue (and also stains chrondrocytes a similar colour). A solution of Eosin Y is then added, this turns eosinophillic structures (intra and extracellular proteins) a spectrum of colours between red and orange. Structures that remain white in this stain include adipocytes, myelin and neuroal axons.

2. This is a bone biopsy from a lytic epiphyseal lesion. What is the diagnosis?

Answer: Giant cell Tumour (GCT) – Neoplastic mononuclear nuclei are present in this slid. Multiple giant cells can be seen which have similar nuclei to the mononuclear stromal cells in large numbers (10 to 50).

3. What are the treatments for this type of tumour?
Answer:
Non-operative: Radiotherapy (higher risk of malignant transformation, generally used for nonoperable tumours)
Bisphosphates (reduce local recurrence 30% to 4%)
Monoclonal antibody (Denosumab) – relatively new treatment. Blocks Rank Ligand to prevent osteoclastic action of mononuclear stromal cells.
Operative: Curettage and reconstruction – allows joint to be preserved, leading to better long term outcomes. Local recurrence 10% to 25% (with adjuvant – phenol/hydrogen peroxide this reduces to 3%). Bone graft or cement are commonly used to fill the defect. Resection/Amputation – Most commonly used in hand GCT

4. What is the nature of the pulmonary lesions that may be associated with this tumour?
Answer: Giant cell tumours metastasis to the lungs in a benign form in 2.6% of cases. These present as discrete nodules which are groups of giant cell tumours. Resection, radiotherapy and chemotherapy have been utilised to treat. A long term study demonstrated a mortality rate of 17% at a mean of 11.9 yrs. Risk factors for pulmonary involvement include recurrent lesions and primary GCT in the distal radius. GCT can also undergo secondary malignant change, specifically linked to previous radiotherapy or multiple

References

1. **Kokkalis ZT, Mavrogenis AF, Ballas EG, Papanastasiou J, Papagelopoulos PJ.** Modified McLaughlin technique for neglected locked posterior dislocation of the shoulder. *Orthopedics* 2013;36:e912-6.
2. **Gerber C, Lambert SM.** Allograft reconstruction of segmental defects of the humeral head for the treatment of chronic locked posterior dislocation of the shoulder. *J Bone Joint Surg [Am]* 1996;78-A:376-382.
3. **Wooten C, Klika B, Schleck CD, et al.** Anatomic shoulder arthroplasty as treatment for locked posterior dislocation of the shoulder. *J Bone Joint Surg [Am]* 2014;96-A:e19.
4. **Prasarn ML, Ahn J, Helfet DL, Lane JM, Lorich DG.** Bisphosphonate-associated femur fractures have high complication rates with operative fixation. *Clin Orthop Relat Res* 2012;470:2295-2301.
5. **Isaacs JD, Shidiak L, Harris IA, Szomor ZL.** Femoral insufficiency fractures associated with prolonged bisphosphonate therapy. *Clin Orthop Relat Res* 2010;468:3384-3392.
6. **Beverly MC, Fearn CB.** Anterior interosseous nerve palsy and dislocation of the elbow. *Injury* 1984;16:126-128.
7. **Babal JC, Mehlman CT, Klein G.** Nerve injuries associated with pediatric supracondylar humeral fractures: a meta-analysis. *J Pediatr Orthop* 2010;30:253-263.
8. **Ramachandran M, Birch R, Eastwood DM.** Clinical outcome of nerve injuries associated with supracondylar fractures of the humerus in children: the experience of a specialist referral centre. *J Bone Joint Surg [Br]* 2006;88-B:90-94.
9. **Kim DH, Murovic JA, Kim YY, Kline DG.** Surgical treatment and outcomes in 15 patients with anterior interosseous nerve entrapments and injuries. *J Neurosurg* 2006;104:757-765.
10. **Fernandes JA, Devalia KL, Moras P, et al.** Screening for spinal stenosis in achondroplastic patients undergoing limb lengthening. *J Pediatr Orthop B* 2014;23:181-186.
11. **Kim SJ, Balce GC, Agashe MV, Song SH, Song HR.** Is bilateral lower limb lengthening appropriate for achondroplasia?: midterm analysis of the complications and quality of life. *Clin Orthop Relat Res* 2012;470:616-621.
12. **Song SH, Kim SE, Agashe MV, et al.** Growth disturbance after lengthening of the lower limb and quantitative assessment of physeal closure in skeletally immature patients with achondroplasia. *J Bone Joint Surg [Br]* 2012;94-B:556-563.
13. **Saleh M, Burton M.** Leg lengthening: patient selection and management in achondroplasia. *Orthop Clin North Am* 1991;22:589-599.
14. **Blackley HR, Wunder JS, Davis AM, et al.** Treatment of giant-cell tumors of long bones with curettage and bone-grafting. *J Bone Joint Surg [Am]* 1999;81-A:811-820.
15. **Xu SF, Adams B, Yu XC, Xu M.** Denosumab and giant cell tumour of bone-a review and future management considerations. *Curr Oncol* 2013;20:e442-e447.
16. **Balke M, Hardes J.** Denosumab: a breakthrough in treatment of giant-cell tumour of bone? *Lancet Oncol* 2010;11:218-219.
17. **Klenke FM, Wenger DE, Inwards CY, Rose PS, Sim FH.** Giant cell tumor of bone: risk factors for recurrence. *Clin Orthop Relat Res* 2011;469:591-599.
18. **Tse LF, Wong KC, Kumta SM, et al.** Bisphosphonates reduce local recurrence in extremity giant cell tumor of bone: a case–control study. *Bone* 2008;42:68-73.
19. **Thomas D, Henshaw R, Skubitz K, et al.** Denosumab in patients with giant-cell tumour of bone: an open-label, phase 2 study. *Lancet Oncol* 2010;11:275-280.
20. **Rock M.** Curettage of giant cell tumour of bone: factors influencing local recurrences and metastasis. *Chir Organi Mov* 1990;75(1 Suppl):204-205.
21. **Siebenrock KA, Unni KK, Rock MG.** Giant-cell tumour of bone metastasising to the lungs. A long-term follow-up. *J Bone Joint Surg [Br]* 1998;80-B:43-47.