Bones and Joints VIVAs (Pathology)

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2015.1.B.2

Question 4 Fracture Healing **Subject:** Path LOA: 1	Describe the steps in fracture repair process	1 **haematoma** fills fracture gap – provides fibrin mesh framework (hrs) 2 **influx** inflam cells, fibroblasts, new vessels (days) 3 haematoma organising -> **procallus** 4 osteoprogenitors deposit trabeculae of woven bone – **ossification** -> **bony callus** (2-3 weeks) 5 callus matures, **remodelling** (6 weeks)	4 of 5 steps Logical sequence
	How does remodelling of callus occur?	Initial large volume of callus – portions not physically stressed are resorbed, reducing callus size/altering contour	Physical stress, resorption
	What factors can impede the healing of a fracture?	**Inadequate immobilisation**, marked displacement, **infection** (open fractures/FBs), systemic factors (nutrition, smoking…)	2 bold and 1 other
	(Supplementary – if time remaining) How are fractures classified?	Complete/incomplete, open/closed, comminuted, displaced, pathologic, stress	

2014.1.C.2

Question 4 Osteomyelitis **Subject:** Path LOA: 1	1.Describe pathogenesis of osteomyelitis. (Prompt what organisms cause osteomyelitis?)	***Local bone injury** and organism entry, **blood borne** organisms, neighbouring source entry. ***Staph Aureus** > 80% of pyogenic ones Others E coli, Kl Pneum, Ps Aerug from IVDU and GU, haemophilus influenza, Gp B Streptococcus. 50% no orgs found.	1.Bold + 1 to pass other organism
	2.What changes occur to the bone?	***Acute inflammation, necrosis, abscess** Sclerosis, involucrum and sequestrum, lytic focus and surrounding necrosis- periosteal elevation	2.Bold to pass
	3.What are the pathological sequelae of osteomyelitis?	***** **Chronic up to 25%, resolve, deformity and bone destruction**, severe sepsis, pathological fracture, endocarditis, SCC, sarcoma.	3.Bold

2013.2.D.2

PATHOLOGY Question 3 LOA: 1	1. Describe the pathogenesis of osteomyelitis. Prompt: How would this patient have suffered a bony infection of his jaw?	**Local infection** related to extraction of tooth **Blood borne** **Spread from neigbouring** gingival source.	2/3
	2. What organisms cause osteomyelitis?	**Staph Aureus majority** >80% pyogenic E Coli, Kl Pneum, Pseudo A, from GU tract or IVDU H Infl and GBS in neonates Viruses, Fungi, Parasites, TB, syphilis also About 50% no orgs found.	Staph A and 1 other
	3. What changes occur in the bone?	**Acute inflammation and necrosis**, abscess formation Sclerosis and **involucrum** formation Deformity and sequestrum formation, Draining sinus Characteristic lytic focus surrounded by zone of necrosis on X ray, lifting of periosteum 5-25% become chronic inflammation	Bold
	4. What are the clinical consequences of osteomyelitis?	Resolution after Rx with IV antibiotics and drainage Conversion to chronic O myelitis Deformity and bony destruction Severe sepsis syndrome, ARF etc.	2 to pass

2013.1.1

Question 5 Gout LOA: 2	1. What are the causes of gout?	**Hyperuricaemia:** 1. Primary Gout (90%; often idiopathic): Overproduction (diet, unknown enzyme defects); Reduced filtration/excretion with normal production. 2. Secondary Gout (10%; known cause, secondary effect is gout): Leukaemias/tumor lysis/psoriasis, inborn errors of metabolism (overproduction with increased excretion); Chronic renal disease (reduced excretion).	Hyperuricaemia + 1 Primary and 1 Secondary cause Or 1 overproduction and 1 decreased excretion
	2. Describe the pathogenesis of acute gouty arthritis. Prompt- What are the steps involved?	1. Hyperuricaemia 2. **Precipitation of urate crystals into joints** (in synovium / cartilage) 3. **Release of crystals into synovial fluid** (?trauma) 4. **Inflammatory response initiated** – crystals phagocytosed by macrophages and neutrophils; release of inflammatory mediators by macrophages (interleukins, cytokines (IL-1B)); resulting in further neutrophil chemotaxis; neutrophils also release inflammatory mediators (free radicals, leukotrienes (LT B4), lysosomal enzymes) – acute arthritis.	Bold to pass
	3. (only if needed) What factors contribute to the conversion of asymptomatic hyperuricaemia into gout	Age & duration of hyperuricaemia; genetic predisposition/etoh/obesity/drugs e.g. thiazides/lead toxicity	

2012.1.1

Question 2 Fracture healing LOA: 1	**How do fractures heal?** Prompt: What are the timeframes of these stages?	1 Haematoma formation/fibrin mesh - hrs 2 Inflammatory cell influx - days 3 Fibroblast/ Osteoprogenitor cells-procallus 4 Organised haematoma - 1wk, 5 Woven bone , bony callus - 2-3 wks 6 Callus maturation remodelling - 6 wks	Must have reasonable sequence and approximate times, at least 4 components to sequence
	What factors impair fracture healing?	Inadequate immobilisation, severe displacement, vascular compromise, infection /FBs, poor nutrition, systemic illnesses	**At least 3**

2011.2.1

Question 5 Gout LOA: 2	Describe the morphological features of gout (Prompt pathological features)	**Acute arthritis-crystallisation of urates within or around joint** An event possibly trauma initiates the release of crystals into the synovial fluid. **Chronic arthritis** with repeated attacks- formation **tophi** in the inflamed synovial membrane and periarticular tissue **Nephropathy**- deposit urate crystals in kidney and formation **uric acid stones**	3 Bold to pass including arthritis
	What are the causes of gout?	**Hyperuricaemia** **Primary** (90%)-enzyme defects unknown (85-90%), known enzyme defect eg. HGPRT deficiency-rare overproduction of uric acid, under excretion or increased excretion **Secondary**-increased nucleic acid turnover eg. leukaemia(overproduction and excretion), CRF (reduced excretion with normal production), inborn errors of metabolism (over production and excretion)	Need hyperuricaemia plus 1 primary and 1 secondary cause to pass

2011.2.3

Question 5 Rheumatoid Arthritis LOA: 2	1.What is the pathogenesis of Rheumatoid arthritis?	Triggered by exposure of genetically susceptible host to an arthritogenic antigen resulting in chronic inflammatory change. Continuing autoimmune reaction with activation CD4 helper T cells and inflammatory mediators and cytokines that destroy the joint **Genetic susceptibility**- associations with HLA-DRB1 alleles **Environmental arthritogens-** unclear what-various microbial agents implicated- none proven **Autoimmunity**-once inflammatory synovitis initiated-autoimmune reaction T cells result in chronic destruction.	Auto immune plus one other
	2.What are the extra articular manifestations of rheumatoid arthritis	**2 Rheumatoid nodules –**elbows forearms, lumbar **Fibrinoid necrosis** of lymphocytes **Vasculitis –** purpuric, nail bed, neuropathy, ulcers	At least 3
	3 What are the long term complications of RA?	3 Joint destruction, renal failure,	Any details

2011.1.1

Question 5. **Osteomyelitis**	1. **Describe the pathogenesis of osteomyelitis** PROMPT; how do organisms reach the bone?	3 basic methods of infection • **blood born** (haematogenous) • **local infection** (extension contiguous site) • **trauma /surgery** (direct implantation)	**2/3**
	3. What Bacterial **organisms cause osteomyelitis?** (good candidates differentiate by age; Neonatal versus adults)	• **S Aureus** • Gp B strep (neonatal) • S Aureus (> 80%) Surgery/open fractures mixed Patient with UTI or IV drug user • E. Coli, Pseudomonas, Klebsiella	**S Aureus and 1 other**
	2. What are the changes in the bone that occur in **osteomyelitis**	• New bone around area of necrosis • Involucrum • Abscesses • Sclerosis • Deformity • Sequestrum • Draining sinus	3 items

2010.2.3

Question 3.5 Osteoarthritis	1. What factors lead to osteoarthritis 2. Describe the pathological changes that occur in an affected joint 3. Describe the major clinical features of osteoarthritis	1.1. Genetic & environmental (mechanical) 1.2. Age – virtually ubiquitous (80-90%) after 65 1.3. Other exacerbating diseases e.g. Obesity, diabetes, injury, abnormal joints, 2.**Chondrocyte injury** 1.3.1. Early OA: **chondrocytes proliferate (cloning) and secrete inflammatory mediators**, collagens, proteoglycans, and proteases which initiates secondary inflammatory changes. 1.3.2. Later OA: repetitive injury and chronic inflammation lead to chondrocyte drop out, marked loss of cartilage, and extensive **subchondral bone changes** 3.Mostly **asymptomatic** <50y.o. 1.4. Deep, achy **pain** worse with use, **morning stiffness**, **crepitus,** and **limited ROM** 1.5. **Oligoarthritis 95%** (occas generalized/early) 1.6. Impingement on spinal foramina by osteophytes results in cervical and lumbar nerve root compression and **radicular pain**, muscle spasms, **muscle atrophy**, and **neurologic deficits**. 1.7. Common: hips, knees, lower lumbar and cervical vertebrae, PIP, DIP of the fingers, 1st carpoMC joints, and 1st TarsoMT joints. Not wrists, elbows, shoulders	1. 2/4 answers 2. 2/3 bold, 3. 2/4

2008.1

10/4/2008 Q4	Describe the pathological features of gout	**Hyperuricaemia**	
		Acute arthritis Precipitation of urate crystals into the joint/s An event (sometimes minor trauma) releases crystals into synovial fluid Cascade occurs resulting in intense inflammatory reaction (complement activated, chemotaxis of neutrophils and macrophages with phagocytosis and activation of lysosymal enzymes, leukotrienes, prostaglandins and free radicals **Chronic arthritis and formation of tophi** which are urate deposits in synovium and periarticular areas **Nephropathy** – deposition of urate in kidney as well as formation of **uric acid stones**	Pass criteria: 3/4
	What are the causes of gout?	**Primary** – enzyme defect unknown (90%) (overproduction, underexcretion or increased excretion) - rare enzyme defect (HGPRT deficiency) **Secondary** (10%) Increased nucleic acid turnover e.g leukaemias (overproduction and excretion) Chronic renal disease (decreased excretion) Inborn error metabolism (complete HGPRT deficiency – Lesch-Nyhan syndrome) overproduction and excretion	Need primary & 1 secondary to pass.