ACEM Primary Examination Vivas > Pathology > Renal
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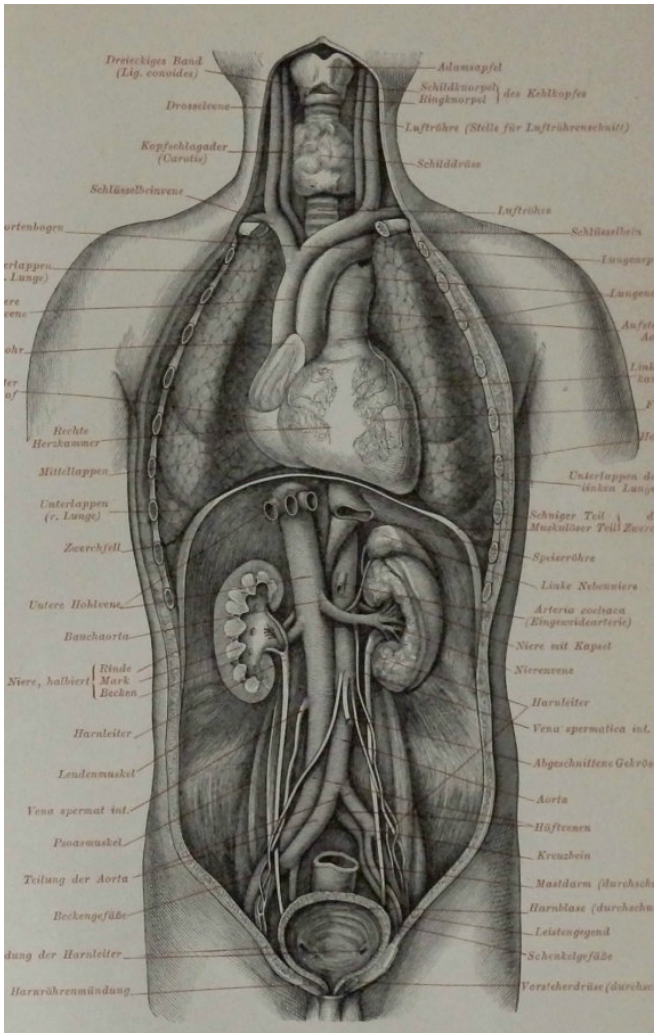

Acute Kidney Injury 2015-2-C

Stem: Moving onto Pathology			
Question 4 Acute Kidney injury / rhabdomyolsysis **Subject:** Path LOA: 2	1. Define Acute Kidney Injury 2. What are the causes of AKI (please give examples)?	Clinico-path entity, **acute reduction of renal function** with morphologic **tubular injury (usually).** Reversible. 1. **Ischaemia**/abnormal blood flow. Systemic – assoc with thrombosis (HUS, TTP, DIC) or hypovolaemia. Intra-renal – angiopathies, malignant HT 2. **Toxic injury to glomeruli/tubules** – myoglobin, drugs, contrast 3. Acute tub.int nephritis – hypersensitivity reaction to drugs, IgA nephropathy 4. Obstruction ("post-renal") – tumour, clot, stones	Bold Bold and 1 other category 1 example for each

Acute Kidney Injury 2012-2

Q5 Acute Kidney Injury LOA: 2	1. What causes acute kidney injury? 2. How does urine output often change with time following acute kidney injury?	1. Commonest cause of acute renal failure. **Ischaemia**: hypotension, vasoconstriction, capsular tamponade. **Direct toxic injury**: (aspirin), aminoglycosides, contrast, myoglobin, crystals, protein. Acute tubulointerstitial nephritis (infections, heavy metals, hypersensitivity reaction to drugs). Post renal urinary **obstruction**. DIC, sepsis. 2.Highly variable. a. Initiation phase: decreased urine output with elevation of urea (< 36 hours) b. Maintenance phase: sustained decreased output (40 – 400 ml/day), salt and water overload, uraemia, hyperkalaemia, metabolic acidosis. c. Recovery phase: increased output and hypokalaemia. Increased vulnerability to infection. May last for months.	One example for each bolded and then at least one other cause. Know initial decrease followed by diuresis

Acute Tubular Necrosis 2015-1-B

Stem: Moving onto Pathology. Her biochemistry results show an acute kidney injury			
Question 4 Acute tubular necrosis **Subject:** Path LOA: 2	Define Acute Kidney Injury	Clinico-path entity, **acute reduction of renal function** with morphologic **tubular injury (usually)**	Bold
	What are the causes of AKI (please give examples)?	1 **Ischaemia**/abnormal blood flow. Systemic – thrombosis (HUS, TTP, DIC) or hypovolaemia. Intra-renal – angiopathies, malignant HT 2 **Toxic injury to tubules**– drugs, radio-dye, myoglobin 3 Acute tub.int nephritis – reaction to drugs 4 Obstruction ("post-renal") –tumour, clot	Bold and 1 other category 1 example for each
	Describe the typical clinical course of AKI	Variable 1 Initiation 36 hours – decr UO, incr urea 2 Maintenance – **oliguria**, salt/H2O overload, incr urea/K/H 3 Recovery - **incr urine vol** (up to 3L/d), H2O/Na/K loss. Ur/Cr r/t normal	Oliguric phase, polyuric recovery
	(Supplementary – if time remaining) What are the most likely causes in this 70 year old lady?	Ischaemic injury from hypovol/hypotension from femur # +/- inability to get to water Myoglobin deposition from rhabdo	

Acute Tubular Necrosis 2006-1

TOPIC: Thursday PM – Q4 – Acute Tubular Necrosis ____ **NUMBER:** _____

OPENING QUESTION	What are the causes of acute tubular necrosis?	**COMMENTS**
POINTS REQUIRED	1 Ischaemic: hypotension, vasoconstriction, intracapsular pressure	3 to pass
	2 Direct toxic injury: aminoglycosides, amphotericin, sulfas Other substances: contrast, myoglobin, crystals, proteins	
	3. Acute tubulo-interstitial nephritis	
	4. Urinary obstruction (post-renal)	
	5. DIC, sepsis	
PROMPTS	What substances can damage the renal tubules?	
SECOND QUESTION (if needed)	Describe the phases of acute tubular necrosis?	Need at least 1 and 3
POINTS REQUIRED	1 Initiation phase: decreased urine output, elevated urea, 36 hrs	
	2 Maintenance phase: sustained decreased output, salt and water overload, uraemia, hyperkalaemia	
	3 Recovery phase: increased output and possibly polyuria, hypokalaemia, vulnerable to infection	
PROMPTS	What is the first thing that happens in ATN?	
THIRD QUESTION (if needed)		
POINTS REQUIRED	1	
	2	
	3	
	4	
	5	
	6	
PROMPTS		

Nephrotic Syndrome 2008-1

Q 4. Nephrotic syndrome	What are the manifestations of the nephrotic Syndrome?	1. Massive proteinuria, with the daily loss of 3.5 gm or more of protein (less in children) 2. Hypoalbuminemia, with plasma albumin levels less than 30 gm/L 3. Generalized oedema 4. Hyperlipidemia and lipiduria	Pass criteria:3 out of 4
	What are the mechanisms of the oedema?	1. Loss of colloid osmotic pressure 2. Loss of serum albumin 3. Accumulation of water and sodium in tissues 4. Due to compensatory secretion of aldosterone Mediated by - Hypovolaemia - ↑ ADH - ↑ Sympathetic system	Pass criteria: 3/4

TOPIC: NEPHROTIC SYNDROME_____ **NUMBER: 3** _____

OPENING QUESTION	What are the manifestations of nephrotic syndrome	**COMMENTS**
POINTS REQUIRED	1 Massive proteinuria	First 3 to pass
	2 Hypoalbuminaemia	
	3 Oedema (generalised)	
	4 Hyperlipidaemia	
	5 Lipiduria	
PROMPTS	Anything else?	
SECOND QUESTION (if needed)	What are the underlying processes responsible for these features	
POINTS REQUIRED	1 Derangement of glomerular capillary walls resulting in increased permeability to plasma proteins. Either structural damage or physicochemical alterations > massive proteinuria	Required to pass
	2 Hypoalbuminaemia 1) secondary to above and 2) inability of liver to synthesise enough replacement albumin. 3) Additionally increased renal catabolism of filtered albumin.	Need more than 1 to pass Prompt for 2) and 3)
	3 Generalised oedema secondary to 1) loss of colloid osmotic pressure of blood. Compounded by 2) sodium and water retention due to activation of renin-angiotensin system, enhanced ADH secretion, stimulation of sympathetic system and reduction in natriuretic factors.	
	4 Hyperlipidaemia (complex causation). Causes include increased liver synthesis, abnormal lipid transport and decreased catabolism.	
	5 Lipiduria a combination of increased production (see 4) and increased glomerular permeability (see 1)	
PROMPTS		
THIRD QUESTION (if needed)	What are the causes of the nephrotic syndrome	
POINTS REQUIRED	1 Primary glomerular disease (95% kids and 60% adults) kids: 60% minimal change, 10% each focal segmental, membranoproliferative and other, 5% membranous Adults: 35% focal segmental. 30% membranous. 10% minimal change 10% membranoproliferative, 15% other	PGN required to pass. Should know difference in causation between kids and adults.
	2 Systemic disease (mostly adults) diabetes, amyloidosis, SLE, drugs (NSAID's, penicillamine), infections (malaria, hepatitis B&C, HIV), malignancies, other (bee-sting allergy, hereditary nephritis)	
PROMPTS	Q 3 IS OPTIONAL ADDITIONAL OPTIONAL QUESTION	

Post Strep Glomeurolonephritis 2010-2

Question 1.5 Post Streptococcal GN	1. Describe the aetiology and pathogenesis of post streptococcal glomerulonephritis.	1.1 **Group A β-hemolytic streptococci** (eg: 90% types 12, 4, and 1) 1.2 Typically post pharyngeal/skin infections (impetigo) - sometimes epidemic, partic in overcrowded insanitary conditions 1.3 An **immunologically mediated disease** ? Type 2/ or 3 type e.g. ? Circulating or antigen deposit disease. 1.4 Granular **immune deposits in the glomeruli** (IgG & C3) - partic GBM- leading to leaking glomeruli. 1.5 Streptococcal antigen found in the glomeruli. 1.6 Complement activation – low serum complement 1.7 Elevated titres of anti streptococcal Ab 1.8 Nephritis associated streptococcal plasmin receptor NAPlr, Strep pyogenic exotoxin B (SpeB), zSPeb	1. 2 x Bold + 1 others
	2. Describe the clinical features of post Streptococcal GN.	1. 1 to 4 weeks after a streptococcal infection of the pharynx or skin (impetigo). 1.1. Malaise, fever, nausea, oliguria, and **haematuria** 1.2. Red cell casts, mild **proteinuria** (usually < 1 gm/day), periorbital and other **oedema**, mild to moderate **hypertension** 1.3. 95% will recover quickly in 1-3 weeks, 4 % chronic, 1% severe acute renal failure. Adult onset has worst prognosis 1.4. Depleted C3 and almost always Strep Ags.	2. 2 x Bold + 2 others

TOPIC: POST STREPTOCOCCAL GLOMERULONEPHRITIS **NUMBER:** _____ 4

OPENING QUESTION	What is the pathogenesis of post streptococcal glomerulonephritis?	**COMMENTS**
POINTS REQUIRED	1 Initiated by Group A Beta-haemolytic streptococcal infection (commonly pharyngitis or impetigo), 90% due to types 1, 4 or 12 (cell wall M protein)	Required
	2. Antibody and immune complex production over 1-4 weeks	Required
	3. Immune complex deposition in glomeruli. (IGG and C_3)	Required
	4. Acute proliferative glomerulonephritis	Required
PROMPTS	Prompt for any ommissions	
SECOND QUESTION (if needed)	What is the clinical course of the disease in children?	
POINTS REQUIRED	1 Recent streptococcal infection as above.	Required if not mentioned previously
	2 Abrupt onset of fever, malaise, nausea, oliguria and haematuria.	Required
	3 Oedema	Required
	4 Mild to moderate hypertension	Required
	5 Red cell casts in urine and mild proteinuria	
	6 95% fully recover (with complete resolution of glomerular lesions), 4% chronic GN, 1% rapidly progressive GN. Prolonged and persistent proteiniuria or abnormal GFR = poorer prognosis	Required
PROMPTS		
THIRD QUESTION (if needed)	How does the clinical course differ in adults?	
POINTS REQUIRED	1 Less benign	Required
	2 Sporadic cases @ 60% fully recover. (Better recovery rates in epidemic outbreaks)	
	3 In the remainder glomerular lesions resolve less quickly or not at all. Prolonged proteinuria, haematuria and hypertension. Outcomes slow resolution, chronic GN or rapidly progressive GN	
PROMPTS		

TOPIC: Thursday AM – Q4 – Post-streptococcal GN _____ **NUMBER:** _____

OPENING QUESTION	Infections by what types of streptococci can lead to glomerulonephritis?	**COMMENTS**
POINTS REQUIRED	1 Strains of Group A beta haemolytic strep with M protein in wall	
	3 Rarely others	
PROMPTS	What groups of streptococci are there?	
SECOND QUESTION (if needed)	What is the cause of the glomerular damage?	Pass: immune complex plus 2 others
POINTS REQUIRED	1 Latent period of 1+ weeks	
	2 Immune complex disease	
	3 Several possible antigens involved	
	4 Antigens have affinity for glomeruli	
	5 Also activates alternative complement cascade	
PROMPTS	Do streptococci produce any harmful antigens?	
THIRD QUESTION (if needed)	What abnormalities are seen in the urine in a patient with glomerulonephritis?	
POINTS REQUIRED	1 Haematuria	
	2 Proteinuria	
	3 Cellular casts	
	4 Other debris	
	5	
PROMPTS	What happens when glomerular membranes are damaged?	

Pre Eclampsia 2008-1

Q5. Pre-eclampsia	What are the proposed pathogenesis and consequences of pre-eclampia?	Placental ischaemia is the key feature leading to 1.Reduction in PGI2, PGE2, 2.Inc renin/angiotensin II, 3.Inc thromboxane and endothelial dysfunction, 4.Resulting in systemic hypertension & DIC.	Pass criteria: placental ischaemia & 1 other point.
	Describe the clinical course of pre-eclampsia.	Usually starts after 32 weeks gestation, charcaterised by 1.Hypertension, oedema and proteinuria. 2.Headache and visual disturbances are common. 3.Ecclampsia is characterised by convulsion and coma.	Need 2/3 to pass.
	Describe the morphological changes in the placenta.	1. Placental infarcts, 2. retroplacental haematoms, 3. villous ischaemia, 4. prominent syncytial knots, 5. thickened trophoblastic basement membrane, 6. villous hypovascularity, 7. fibrinoid necrosis , 8. intramural lipid deposition.	Needs to give 3 to pass.

Pyelonephritis 2015-2-A

Stem: A 40 year old woman presents with left loin pain and fevers. Urine microscopy is performed			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 **Clinical Building Block:** Urine Microscopy	Please describe the abnormalities. What is the most likely diagnosis?	**High poly and RBC counts** with +ve protein and blood (in the absence of epi-clean catch) indicates **infection** In the clinical context c/w **pyelonephritis** +/- stone	Bold to pass
Question 2 Pyelonephritis **Subject:** Path LOA: 2	What organisms cause acute pyelonephritis? Prompt: what are the most common? What steps are involved in ascending infection of the urinary tract? What conditions predispose to acute pyelonephritis?	**G-ve bacilli** (>85%), endogenous organisms E Coli, proteus, klebsiella, enterobacter, strep faecalis(enterococcus) Other: staph, fungi, (viruses in immunocompromised and renal transplant patients) **5 steps:** 1. colonisation distal urethra 2. entry into bladder 3 . urinary tract obstruction / stasis of urine 4. vesicoureteric reflux 5. intrarenal reflux Urinary tract obstruction Instrumentation Vesico-ureteric reflux Pregnancy Female upto 50yrs Males >50 yrs Abnormalities- congenital/acquired DM, Immunosuppression	G-ve & 3 organisms pass Need to explain the concept clearly 4/9 to pass

Pyelonephritis 2012-1

Question 4 UTI	**What organisms cause acute pyelonephritis?** Prompt: what are the most common?	**G-ve bacilli** (>85%), endogenous organisms E Coli, proteus, klebsiella, enterobacter, strep faecalis Other: staph, fungi, (viruses in immunocompromised and renal transplant patients)	**G-ve & 3 organisms pass**
	What steps are involved in ascending infection?	**5 steps:** 1. colonisation distal urethra 2. entry into bladder 3 . urinary tract obstruction / stasis of urine 4. vesicoureteric reflux 5. intrarenal reflux	Need to explain the steps clearly
	What are the features of chronic pyelonephritis?	**Chronic** = chronic reflux or obstruction causes pelvocalyceal damage. Recurrent infections lead to recurrent bouts of renal inflammation and **scarring**	**Bold** & concept

Urinary Tract Obstruction 2013-1

Question 5 Obstructive uropathy LOA: 2	1. What are the causes of urinary tract obstruction?	1. Congenital- urethral valves & strictures; bladder neck obstruction; ureteropelvic narrowing; reflux **Calculi; Prostatic hypertrophy** **Tumors**- prostate; bladder; cervix/uterus; other Inflammation- prostatitis; urethritis; ureteritis; retroperitoneal fibrosis Sloughed papillae, clots; Pregnancy; Uterine prolapse; cystocele Functional- neurogenic (spinal cord/diabetic); dysfunctional; ureter or bladder	Bold plus one other.
	2. What are the clinical features of acute obstruction?	**2. Pain** due to distension or Sx of underlying process e.g. renal colic, LUTS in prostatic disease asymptomatic (in Unilateral complete or partial) Polyuria and nocturia. Calculi, HT, distal tubular acidosis- (In Bilateral partial) oligo/anuria, hyperkalaemia, incr urea & creat- (in Complete bilateral)	Bold
	3. What are the possible clinical sequelae of urinary tract obstruction?	3. Infection Stone formation Atrophy/hydronephrosis/obstructive uropathy (if chronic)- => renal failure Complications of renal failure.	3/5

Urinary Tract Obstruction 2011-2

Question 4 LOA: 2	1 What conditions cause urinary tract obstruction?	Extrinsic and intrinsic causes **Intrinsic** – Congenital abnormalities: posterior uretheral valves, urethral strictures, etc, **Calculi** Tumours Inflammation: prostatitis, ureteritis, urethritis Blood clots Sloughed papillae **Extrinsic** Tumours BPH Retroperitoneal fibrosis Pregnancy Uterine prolapse and cystocoele Functional disorders: neurogenic bladder	6 causes including calculi to pass (must demonstrate knowledge of intrinsic and extrinsic causes but needn't use words)
	2. Describe the progression of effects of unrelieved obstruction of a ureter.	Reduced GFR Progressive **dilation** of the proximal ureter, renal pelvis and calyces (hydronephrosis) Renal **parenchymal atrophy** Blunting apices of the pyramids Interstitial inflammation leading to interstitial fibrosis Enlargement of kidney Eventual result is a large (15-20cm) thin walled **non-functional** cystic structure.	Dilation, parenchymal atrophy and loss of function to pass.

Urolithiasis 2014-1-C

Stem: We are moving onto pathology.			
Question 4 Pre-eclampsia **Subject:** Path LOA: 2	4.1 Describe the pathogenesis of pre - eclampsia.	**4.1 Endothelial dysfunction,** vasoconstriction leads to hypertension, increase vascular permeability causing proteinuria & oedema.	Bold + 1 to pass
	4.2 What is the clinical course of pre eclampsia?	4.2 > 34 weeks typically has **HT, oedema, proteinuria** Headache and visual disturbance **Eclampsia** is progression to seizures and coma	2/3 bold to pass (prompt: what happens in untreated pre-eclampsia?)
	4.3 What morphological changes occur in the placenta?	4.3 Infarcts, haematomas, villous ischaemia, syncytial knots, fibrinoid necrosis	1 to pass

Urolithiasis 2014-1-B

Stem: A 40 yo man presents to ED with renal colic			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
The first questions is in regard to pathology			
Question 1 Urolithiasis (Robbins pp 962-963) **Subject:** Path LOA: 1	1.What are the main types of renal calculi? *Prompt: What are the common constituents of renal calculi?* 2.What conditions in urine favour stone formation? 3. What are the complications of ureteric calculi?	**1.Calcium** oxalate and phosphate (70%); 2. Struvite or triple (magnesium ammonium phosphate) (15-20%); 3. Uric acid (5-10%); 4. Cystine (1-2%) **2.** Increased concentration of stone constituents; changes in urinary pH; decreased urine volume; bacteria 3. pain, haematuria, **infection, obstructive renal impairment**	**1.Calcium + 1 other to pass** **2. 2 to pass** **3. 1 bold and 1 other.**

Urolithiasis 2007-2

TOPIC: Urolithiasis **NUMBER: Q3**

OPENING QUESTION	**What are the types of urinary calculus?**	**COMMENTS**
POINTS REQUIRED	1. **Ca oxalate** 70%	mandatory
	Idiopathic hypercalcuria 50%	3/6 with examples
	Hypercalciuria & hypercalcaemia 5% - (hyperparathyroid, bone diseases, Sarcoid)	
	Hypercalcaemia only 5% - (increased absorption, renal, idiopathic, fasting)	
	Hyperoxaluria 5% - 4.5% enteric - 0.5% Primary.	
	Hypocitraturia 1%	
	Unknown 15-20%	
	2 **Magnesium ammonium phosphate (Struvite) (triple phosphate)** 15% - (staghorn calc)	mandatory
	. Urea splitting organisms.... - (proteus, some staph),	1/2
	3 **Uric acid**	mandatory
	Raised uric acid Over 50% normal uric acid and excretion (pH <5.5)	½?
	4. **cysteine 1-2%**	bonus
SECOND QUESTION	**What is the pathogenesis of renal stones?**	
	Increased concentration of stone constituents Change in urinary pH Decreased urine volume Infection – bacteria Deficiency in inhibitors-pyrophosphates, Diphosphonates, Citrate, Glycosaminoglycans, Osteopontin, nephrocalcin-	3 out of 6

Urolithiasis 2006-1

TOPIC: Friday AM – Q 4 – Urolithiasis _____ **NUMBER:** _____

OPENING QUESTION	What different substances can renal calculi be composed of?	**COMMENTS**
POINTS REQUIRED	1 calcium (oxalate and/or phosphate) 80%	Calcium and at least 1 other.
	2 struvate (magnesium/ammonium/phosphate) 15%	
	3 uric acid 5%	
	4 cystine 1%	
	5 Others (metabolic byproducts, drugs)	
PROMPTS	What can become concentrated in the urine and crystallize?	
SECOND QUESTION (if needed)	What factors may lead to formation of struvate calculi?	2 to pass
POINTS REQUIRED	1 Anatomical: polycystic, horseshoe, sponge, diverticulae, reflux	
	2 Concentration of urine	
	3 Substrate formed by urea-splitting bacteria (proteus, klebsiella, pseudomonas).	
	4 Metabolic causes	
PROMPTS	Are there any general factors that could lead to formation of crystals?	
THIRD QUESTION (if needed)	What are the potential complications of renal calculi?	
POINTS REQUIRED	1 Infection	
	2 Renal parenchymal damage	
	3 Obstruction	
	4 Bleeding	
	5 Pain	
	6 Renal failure	
PROMPTS	What can happen if urine flow is obstructed?	