Renal VIVAs (Pathology)

shakEM

Aug 2015

2015.1.B.3

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	

2014.1.B.1

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.**

2013.1.2

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

2012.2.3

Q5 Acute Kidney Injury LOA: 2	1. What causes 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.	One example for each bolded and then at least one other cause.
	2. How does urine output often change with time following acute kidney injury?	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.	Know initial decrease followed by diuresis

2012.1.3

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

2011.2.1

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.

2010.2.1

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

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