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Q:469	<p>469. A 10yo girl presents with pallor and features of renal failure. She has hematuria as well as proteinuria. The serum urea and creat are elevated. These symptoms started after an episode of bloody diarrhea 4days ago. What is the most probable dx?</p> <p>a. TTP b. HUS c. ITP d. HSP e. ARF</p>
Clincher(s)	
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KEY	B
Additional Information	
Reference	
Dr Khalid/Rabia	<p>KEY- B</p> <p>Haemolytic Uraemic Syndrome (HUS) is a triad of Haemolytic anaemia, thrombocytopenia and Renal failure. It is said to be caused most commonly by E.coli O:157H7 which binds to endothelial receptors in the GIT, Renal and central nervous system.</p> <p>Symptoms [abdominal pain, pallor due to anaemia, hematuria and proteinuria, features of renal failure like- nausea/vomiting, swelling of face, hand, feet or entire body etc. and elevated urea and creatinine etc.] start around two weeks after an episode of bloody diarrhea. The diarrhoea is characterised to get bloody after 1-3 days. This scenario is typical for HUS.</p> <p>It is also known to be precipitated by strept pneumonia and some drugs like cyclosporin and tacrolimus.</p>

Q:506	<p>506. A 10yo boy is clinically obese and the shortest in his class. He had a renal transplant last year and his mother is worried that he is being bullied. What is the most probable dx?</p> <p>a. Cushing's syndrome b. Congenital hypothyroidism c. Pseudocushing's syndrome d. Lawrence moon bieder syndrome e. Down Syndrome</p>
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Clincher(s)	
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E	A
KEY	
Additional Information	
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Dr Khalid/Rabia	<p>a. Cushing's syndrome he's on steroids post-renal transplant, Oral steroids is the chief cause of Cushing's syndrome (OHCM, 8th, page 124).</p> <p>Laurance-moon synd. Night blindness due to retinitis pigmentosa, polydactyly are important features (OHCS/8th/648). With no emphasis on more common features, Oral-steroid induced (post renal transplant) Cushing makes more sense.</p> <p>Congenital hypothyroidism Feeding difficulties, Somnolence, Lethargy, Low frequency of crying, Constipation</p> <p>Down's syndrome he is clinically obese not conganital case,down syndrome has cardaic problem and characteristic facial feature and mentalyy retarded so it cant be option,these features are same for lawrence moon but ptnt are mentally retarded whereas kid is studying in normal school rather than special one</p> <p>Pseudocushing's syndromeit is mainly an <u>idiopathic</u> condition.Some frequently occurring illnesses can induce a phenotype that largely overlaps with <u>Cushing syndrome</u> and is accompanied by hypercortisolism</p>

Q:1161	<p>1161. A 14yo boy has been dx with nephrotic syndrome. 5d later he presents with flank pain, hematuria and fluctuating urea levels. A dx of renal vein thrombosis is made. What is the most likely cause for renal vein thrombosis?</p> <p>a. Protein C deficiency</p> <p>b. Vasculitis</p> <p>c. Loss of antithrombin III</p> <p>d. High estrogen levels</p>
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	e. Stasis
Clincher(s)	
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KEY	C
Additional Information	
Reference	
Dr Khalid/Rabia	<p>key : c</p> <p>Complications of nephrotic syndrome include:</p> <ul style="list-style-type: none"> • Decreased resistance to infections, due to urinary immunoglobulin loss. • Increased risk of arterial and venous thrombosis, due to loss of antithrombin III and plasminogen in the urine, combined with an increase in hepatic synthesis of clotting factors. Adults with membranous nephropathy are at particular risk • Acute kidney injury • Chronic kidney disease may occur as a result of an underlying cause - eg, amyloidosis or diabetes. <p>Increased risk of osteitis fibrosa cystica and osteomalacia due to loss of vitamin D-binding protein</p>

Q:1535	<p>1535. A 75yo man has urinary symptoms of hesitancy, frequency and nocturia. Rectal exam: large hard prostate. What is the most appropriate inv?</p> <p>a. CA 125 b. CA 153 c. CA 199 d. CEA e. PSA</p>
Clincher(s)	
A	
B	
C	

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D	
E	
KEY	E
Additional Information	
Reference	
Dr Khalid/Rabia	<p>Answer: E Prostate-specific antigen (PSA)</p> <p>Cancer type: Prostate cancer Tissue analyzed: Blood PSA is produced exclusively by epithelial prostatic cells, both benign and malignant. It is also found in the serum. Serum PSA is currently the best method of detecting localised prostatic cancer and monitoring response to treatment but it lacks specificity, as it is also increased in most patients with benign prostatic hyperplasia.</p>

Q:1554	<p>1554. An elderly lady presents with confusion. She is afebrile but complains of dysuria for 2 days duration. What is the definitive dx investigation?</p> <p>a. Blood culture b. Urine nitrites c. CT head d. ECG e. IVU</p>
Clincher(s)	
A	
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E	
KEY	B
Additional Information	
Reference	
Dr Khalid/Rabia	<p>Key: Urine Nitrites (B) Reason: According to the OHCM pg. 288, if symptoms are present, dipstick the urine and treat empirically if nitrites or leucocytes are positive while awaiting culture of an MSU. ECG and CT Head are useless tests and IVU doesn't make sense because we aren't looking for an obstructive cause. You only go for imaging studies if it is recurrent and there is failure with standard treatment. Most accurate test –</p>

	<p>URINE CULTURE.</p> <p>Discussion:</p> <p>Urinary tract infection (UTI) - this implies the presence of characteristic symptoms and significant bacteriuria from kidneys to bladder. Many laboratories regard 10⁵ colony-forming units per millilitre (cfu/ml) as the threshold for diagnosing significant bacteriuria.</p> <p>Organisms:</p> <p>Several micro-organisms are known to cause UTI, but the majority of infections will be produced by three organisms:</p> <ul style="list-style-type: none"> * Escherichia coli * Staphylococcus saprophyticus * Proteus mirabilis <p>Infection with less common organisms is more likely to occur in patients who have underlying pathology and/or frequent infections, are immunosuppressed, or who are catheterised. Organisms which may produce infection under these circumstances include:</p> <ul style="list-style-type: none"> * Klebsiella spp. * Proteus vulgaris * Candida albicans * Pseudomonas spp. <p>Signs: Fever, abdominal/ loin tenderness, foul smelling urine.</p> <p>Symptoms: Frequency, dysuria, urgency, hematuria, suprapubic pain.</p> <p>Tests:</p> <ul style="list-style-type: none"> * Dipstick urine – nitrites or leukocytes positive mean UTI. Treat empirically. * MSU sample for culture and sensitivity. * CBC, Urea & Electrolytes, Blood culture if systemically unwell. <p>Imaging: usually done in males, children or persistent cases.</p> <ul style="list-style-type: none"> * USG and refer to urology for assessment. * CT-KUB. * Urodynamics. <p>Management:</p> <ul style="list-style-type: none"> * Drink plenty of fluids. * Trimethoprim 200mg x 12 hourly PO OR Nitrofurantoin 50mg x 6 hourly PO for 3-6 days OR Amoxicillin 500mg x 8 hourly PO. * Second-line: Co-Amoxiclav 7 day course PO.
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Q:1565	<p>A 75yo man presents with ARF. He has been troubled by recurrent epistaxis but over the last 3wks he reports to have coughed up blood too. What is the single most likely positive antibody?</p> <ol style="list-style-type: none"> a. P ANCA b. C ANCA c. Anti Ro d. Anti DS DNA e. Anti centromere
	<i>Renal failure and lung- Wegener's Granulomatosis or Good pastures syndrome</i>

	(type 4 collage found in lungs – alveola- so he is alveolar and basement membrane)
Clincher(s)	
A	
B	
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KEY	B
Additional Information	
Reference	
Dr Khalid/Rabia	<p>Key: C ANCA (B)</p> <p>Reason: This patient has Wegener's Granulomatosis which affects the upper and lower respiratory tract and presents with renal insufficiency. It is C ANCA positive and is treated by steroids or cyclophosphamide.</p> <p>Discussion: Also called granulomatosis with polyangiitis (so vessels affected). Affects the upper respiratory tract, lungs and kidneys.</p> <ul style="list-style-type: none"> * Nasal obstruction, ulcers, epistaxis. Destruction of nasal septum causing a characteristic saddle nose deformity. * Rapidly progressive GN with crescent formation, hematuria and proteinuria can occur. * Cough, hemoptysis, pleuritis. <p>Investigations:</p> <ul style="list-style-type: none"> * C-ANCA, Raised ESR and CRP. * Urine R/E to check for proteinuria or hematuria. If present, renal biopsy to confirm. * Chest X-ray – Nodules and fluffy infiltrates of pulmonary haemorrhage. * CT Scan – Diffuse alveolar haemorrhage. <p>Treatment:</p> <ul style="list-style-type: none"> * Severe disease should be treated with steroids and cyclophosphamide or rituximab to induce remission. * Methotrexate or azathioprine for maintenance. * Patients with severe renal disease may benefit for plasma exchange. * Co-Trimoxazole should be given as prophylaxis against P. jiroveci infection.

Q:1703	<p>A 34yo man had a 4mm ureteric stone which he passed in urine. This time he presents with 3cm stone in the right kidney. Single most appropriate treatment?</p> <ol style="list-style-type: none"> No treatment ESWL (extra corp shock wave) Laparotomy Observe
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	e. Operative stone removal
	<i>Less than 4-5mm: increase fluid intake; Less than 1-2cm ESWL; if more than operative – Percutaneous nephrolithotomy</i>
Clincher(s)	
A	
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D	
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KEY	?
Additional Information	<p>OHCM</p> <p><i>Initially:</i> Analgesia, eg diclofenac 75mg IV/IM, or 100mg PR. 225 (If CI: opioids) + IV fluids if unable to tolerate PO; antibiotics (eg cefuroxime 1.5g/8h IV, or gentamicin) if infection. <i>Stones <5mm in lower ureter:</i> ~90–95% pass spontaneously. □fluid intake.</p> <p><i>Stones >5mm/pain not resolving:</i> Medical expulsive therapy: nifedipine 10mg/8h PO 226 or □-blockers (tamulosin 0.4mg/d 227) promote expulsion and reduce analgesia requirements:228 · start at presentation. 229 Most pass within 48h (>80% after ~30d).</p> <p>If not, try extracorporeal shockwave lithotripsy (ESWL) (if <1cm), or ureteroscopy using a basket. 230 ESWL: US waves shatter stone. SE: renal injury, may also cause □BP and DM. 231 Percutaneous nephrolithotomy (PCNL): keyhole surgery to remove stones,when large, multiple, or complex. 232 Open surgery is rare.</p> <p>· <i>Indications for urgent intervention (delay kills glomeruli):</i> Presence of infection and obstruction—a percutaneous nephrostomy or ureteric stent may be needed to relieve obstruction (p642); urosepsis; intractable pain or vomiting; impending ARF;obstruction in a solitary kidney; bilateral obstructing stones. 232</p> <p><i>Prevention General:</i> Drink plenty. Normal dietary Ca²⁺ intake (low Ca²⁺ diets increase oxalate excretion). <i>Specifically:</i> •<i>Calcium stones:</i> in hypercalciuria, a thiazide diuretic is used to · Ca²⁺ excretion •<i>Oxalate:</i> · oxalate intake; pyridoxine may be used p312) •<i>Struvite:</i> treat infection promptly •<i>Urate:</i> allopurinol (100–300mg/24h PO).</p> <p>Urine alkalinization may also help, as urate is more soluble at pH>6 (eg with potassium citrate or sodium bicarbonate) •<i>Cystine:</i> vigorous hydration to keep urine output >3L/d and urinary alkalinization (as above). D-penicillamine is used to chelate cystine, given with pyridoxine to prevent vitamin B6 deficiency</p> <p>PCNL > 2 CM STONE</p> <p>Renal stones: management</p>

Acute management of renal colic

Medication

- the British Association of Urological Surgeons (BAUS) recommend diclofenac (intramuscular/oral) as the analgesia of choice for renal colic*
- BAUS also endorse the widespread use of alpha-adrenergic blockers to aid ureteric stone passage

Imaging

- patients presenting to the Emergency Department usually have a KUB x-ray (shows 60% of stones)
- the imaging of choice is a non-contrast CT (NCCT). 99% of stones are identifiable on NCCT. Many GPs now have direct access to NCCT

Stones < 5 mm will usually pass spontaneously. Lithotripsy and nephrolithotomy may be for severe cases.

Prevention of renal stones

Calcium stones may be due to hypercalciuria, which is found in up to 5-10% of the general population.

- high fluid intake
- low animal protein, low salt diet (a low calcium diet has not been shown to be superior to a normocalcaemic diet)
- thiazides diuretics (increase distal tubular calcium resorption)

Oxalate stones

- cholestyramine reduces urinary oxalate secretion
- pyridoxine reduces urinary oxalate secretion

Uric acid stones

- allopurinol
- urinary alkalinization e.g. oral bicarbonate

*Diclofenac use is now less common following the MHRA warnings about cardiovascular risk. It is therefore likely the guidelines will change soon to an alternative NSAID such as naproxen

Reference	
Dr Khalid/Rabia	

Q:1072	A 61yo man, known smoker, comes to the hospital with complaints of painless hematuria, urgency and dysuria. He has been worried about his loss of weight and reduced general activity. Which inv would be diagnostic of his condition? a. Urine microscopy b. IVU c. CT d. Cystoscopy e. US abdomen f. KUB g. Cystoscopy with biopsy h. Mid stream urine for culture i. Transrectal U																												
Clincher(s)	61yr,smoker,Painless hematuria,loss of weight																												
A	No signs of uti so microscopy is not required here.																												
B	It is an Xray of urinary tract following injection of contrast.not helpful in this case																												
C	CT urogram can be diagnostic but its not the first line																												
D	Cystoscopy alone is not beneficial here																												
E	not diagnostic here																												
KEY	G.cystoscopy with biopsy can diagnose tumour and its type <table><tr><th colspan="3">TNM staging of bladder cancer</th><th>(See also p527)</th></tr><tr><td>Tis</td><td>Carcinoma <i>in situ</i></td><td>Not felt at EUA</td><td></td></tr><tr><td>Ta</td><td>Tumour confined to epithelium</td><td>Not felt at EUA</td><td></td></tr><tr><td>T1</td><td>Tumour in lamina propria</td><td>Not felt at EUA</td><td></td></tr><tr><td>T2</td><td>Superficial muscle involved</td><td>Rubbery thickening at EUA</td><td></td></tr><tr><td>T3</td><td>Deep muscle involved</td><td>EUA: mobile mass</td><td></td></tr><tr><td>T4</td><td>Invasion beyond bladder</td><td>EUA: fixed mass</td><td></td></tr></table> <p>EUA = examination under anaesthetic</p>	TNM staging of bladder cancer			(See also p527)	Tis	Carcinoma <i>in situ</i>	Not felt at EUA		Ta	Tumour confined to epithelium	Not felt at EUA		T1	Tumour in lamina propria	Not felt at EUA		T2	Superficial muscle involved	Rubbery thickening at EUA		T3	Deep muscle involved	EUA: mobile mass		T4	Invasion beyond bladder	EUA: fixed mass	
TNM staging of bladder cancer			(See also p527)																										
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T4	Invasion beyond bladder	EUA: fixed mass																											

Additional Information	<div data-bbox="403 275 804 318">648 Bladder tumours</div> <p>>90% are transitional cell carcinomas (TCCs) in the UK. What appear as benign papillomata rarely behave in a purely benign way. They are almost certainly indolent TCCs. Adenocarcinomas and squamous cell carcinomas are rare in the West (the latter may follow schistosomiasis). UK incidence $\approx 16000/\text{yr}$. $\sigma:\phi \approx 5:2$. Histology is important for prognosis: Grade 1—differentiated; Grade 2—intermediate; Grade 3—poorly differentiated. 80% are confined to bladder mucosa, and only ~20% penetrate muscle (increasing mortality to 50% at 5yrs).</p> <p>Presentation Painless haematuria; recurrent UTIs; voiding irritability.</p> <p>Associations Smoking; aromatic amines (rubber industry); chronic cystitis; schistosomiasis (risk of squamous cell carcinoma); pelvic irradiation.</p> <p>Tests</p> <ul style="list-style-type: none"> • Cystoscopy with biopsy is diagnostic. • Urine: microscopy/cytology (cancers may cause sterile pyuria). • CT urogram is both diagnostic and provides staging. • Bimanual EUA helps assess spread. • MRI or lymphangiography may show involved pelvic nodes. <p>Staging See TABLE.</p> <p>Treating TCC of the bladder</p> <ul style="list-style-type: none"> • Tis/Ta/T1: (80% of all patients) Diathermy via transurethral cystoscopy/transurethral resection of bladder tumour (TURBT). Consider intravesical chemotherapeutic agents for multiple small tumours or high-grade tumours. A regimen of mitomycin c, doxorubicin and cisplatin as maintenance to prevent recurrence is as effective as intravesical BCG (which stimulates a non-specific immune response) and has less SEs. ²⁰ 5yr survival $\approx 95\%$. • T2-3: Radical cystectomy is the 'gold standard'. Radiotherapy gives worse 5yr survival rates than surgery, but preserves the bladder. 'Salvage' cystectomy can be performed if radiotherapy fails, but yields worse results than primary surgery. Post-op chemotherapy (eg M-VAC: methotrexate, vinblastine, adriamycin, and cisplatin) is toxic but effective. Neoadjuvant chemotherapy with CMV (cisplatin, methotrexate and vinblastine) has improved survival compared to cystectomy or radiotherapy alone. ²⁰ Methods to preserve the bladder with transurethral resection or partial cystectomy + systemic chemotherapy have been tried, but long-term results are disappointing. If the bladder neck is not involved, orthotopic reconstruction rather than forming a urostoma is an option (both using ~40cm of the patient's ileum), but adequate tumour clearance must not be compromised. ► The patient should have all these options explained by a urologist and an oncologist. • T4: Usually palliative chemo/radiotherapy. Chronic catheterization and urinary diversions may help to relieve pain.
Reference	Ohcm page 648
Dr Khalid/Rabia	

Q:1186	<p>A homeless lady presents with cough and fever. She complains of night sweats and weight loss. CXR has been done and shows opacity. What is the next appropriate management?</p> <ol style="list-style-type: none"> AFB Mantoux test IFN gamma testing
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	d. Bronchoscopy e. CT
Clincher(s)	Cough with fever,night sweats,wight loss,CXR shows opacity
A	AFB to detect Mycobacterium TB.diagnostic.
B	Can show exposure only
C	nterferon-release assays (IGRAs) represent advances in tuberculosis immunology and evolutionary biology. IGRAs were designed to replace tuberculin skin test (TST) for the diagnosis of latent tuberculosis infection because of their logistical advantages and enhanced specificity over TST. Although IGRAs and TST have been useful in epidemiologic studies, they lack the sensitivity and reproducibility normally expected from diagnostic tests in clinical practice
D	Used to visualize and detect airway pathology and to look for foreign body in the airway
E	Not required
KEY	A
Additional Information	
Reference	
Dr Khalid/Rabia	a. AFB Mantoux test can only tell you "the exposure which may be the past", can't confirm active disease INVESTIGATIONS: 1- CXR 2- Sputum for AFB/culture 3- BAL

Q:1213	What are the side effects of thiazide diuretics? a. Hypocalcemia b. Hyponatremia c. Hypernatremia d. Hyperkalemia
Clincher(s)	Straight foraward
A	
B	
C	
D	
E	
KEY	B

Additional Information	<p>Side-effects Side-effects of thiazides and related diuretics include mild gastro-intestinal disturbances, postural hypotension, altered plasma-lipid concentrations, metabolic and electrolyte disturbances including hypokalaemia (see also notes above), hyponatraemia, hypomagnesaemia, hypercalcaemia, hyperglycaemia, hypochloraemic alkalosis, hyperuricaemia, and gout. Less common side-effects include blood disorders such as agranulocytosis, leucopenia, and thrombocytopenia, and impotence. Pancreatitis, intrahepatic cholestasis, cardiac arrhythmias, headache, dizziness, paraesthesia, visual disturbances, and hypersensitivity reactions (including pneumonitis, pulmonary oedema, photosensitivity, and severe skin reactions) have also been reported.</p> <p>Thiazide diuretics work by inhibiting sodium absorption at the beginning of the distal convoluted tubule (DCT). Potassium is lost as a result of more sodium reaching the collecting ducts. Thiazide diuretics have a role in the treatment of mild heart failure although loop diuretics are better for reducing overload. The main use of bendroflumethiazide was in the management of hypertension but recent NICE guidelines now recommend other thiazide-like diuretics such as indapamide and chlortalidone.</p>
Reference	BNF page 87
Dr Khalid/Rabia	<p>Thiazide diuretics - Bendroflumethiazide, hydrochlorothiazide</p> <p>Low dose to treat hypertension</p> <p>Also used in combination with loop diuretics to treat heart failure (Metolazone)</p> <p>In case of hypertension, if patient is intolerant to calcium channel blockers, thiazide diuretics are prescribed.</p> <p>Side effects include hyponatremia, hypokalemia, hypomagnesemia, hyperuricaemia, metabolic alkalosis, hypotension, hypovolaemia</p>

Q:1238	<p>A 36yo man has been dx with DI. What electrolyte picture is expected to be seen?</p> <ol style="list-style-type: none"> High serum Na, low serum osmolarity, high urine osmolarity Low serum Na, low serum osmolarity, high urine osmolarity Low serum Na, high serum osmolarity, high urine osmolarity High serum Na, high serum osmolarity, low urine osmolarity Normal Na, normal serum osmolarity, normal urine osmolarity
	ADH works on water loss (wont take na with it) and there is more water loss and so more concentration in serum (more Na in serum)
Clincher(s)	
A	

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B	
C	
D	
E	
KEY	

Additional Information	<p>Diabetes insipidus (DI)</p> <p>This is the passage of large volumes (>3L/day) of dilute urine due to impaired water resorption by the kidney, because of reduced ADH secretion from the posterior pituitary (cranial DI) or impaired response of the kidney to ADH (nephrogenic DI). See fig 1.</p> <p>Symptoms Polyuria; polydipsia; dehydration; symptoms of hypernatraemia (p686). <i>Polydipsia can be uncontrollable and all-consuming, with patients drinking anything and everything to hand: in such cases, if beer is on tap, disaster will ensue!</i>¹⁰</p> <p>Causes of cranial DI • Idiopathic (≈50%) • Congenital: defects in ADH gene, DIDMOAD¹ • Tumour (may present with DI + hypopituitarism): craniopharyngioma, metastases, pituitary tumour • Trauma: temporary if distal to pituitary stalk as proximal nerve endings grow out to find capillaries in scar tissue and begin direct secretion again • Hypophysectomy • Autoimmune hypophysitis (p224) • Infiltration: histiocytosis, sarcoidosis² • Vascular: haemorrhage² • Infection: meningoencephalitis.</p> <p>Causes of nephrogenic DI • Inherited • Metabolic: low potassium, high calcium • Drugs: lithium, demeclocycline • Chronic renal disease • Post-obstructive uropathy.</p> <p>Tests U&E, Ca²⁺, glucose (exclude DM), serum and urine osmolalities. Serum osmolality estimate ≈ 2 × (Na⁺ + K⁺) + urea + glucose (all in mmol/L). Normal plasma osmolality is 285–295mOsmol/kg, and urine can be concentrated to more than twice this concentration. Significant DI is excluded if urine to plasma (U:P) osmolality ratio is more than 2:1, provided plasma osmolality is no greater than 295mOsmol/kg. In DI, despite raised plasma osmolality, urine is dilute with a U:P ratio <2. In primary polydipsia there may be dilutional hyponatraemia—and as hyponatraemia may itself cause mania, be cautious of saying "It's water intoxication from psychogenic polydipsia".</p> <p>Diagnosis The water deprivation test aims to test the ability of kidneys to concentrate urine for diagnosis of DI, and then to localize the cause. See box.</p> <p>NB: it is often difficult to differentiate primary polydipsia from partial DI.</p> <p>ΔΔ: DM; diuretics or lithium use; primary polydipsia—this causes symptoms of polydipsia and polyuria with dilute urine. Its cause is poorly understood;⁴ it may be associated with schizophrenia or mania (±Li⁺ therapy), or, rarely, hypothalamic disease (neurosarcoid; tumour; encephalitis; brain injury; HIV encephalopathy). As part of this syndrome, the kidneys may lose their ability to fully concentrate urine, due to a wash-out of the normal concentrating gradient in the renal medulla.</p> <p>Treatment Cranial DI: Find the cause—MRI (head); test anterior pituitary function (p224). Give desmopressin, a synthetic analogue of ADH (eg Desmomet[®] tablets).</p> <p>Nephrogenic: Treat the cause. If it persists, try bendroflumethiazide 5mg po/24h. NSAIDs lower urine volume and plasma Na⁺ by inhibiting prostaglandin synthase: prostaglandins locally inhibit the action of ADH.</p> <p>▶▶ Emergency management • Do urgent plasma U&E, and serum and urine osmolalities. Monitor urine output carefully and check U&E twice a day initially.</p> <ul style="list-style-type: none"> • IVI to keep up with urine output. If severe hypernatraemia, do not lower Na⁺ rapidly as this may cause cerebral oedema and brain injury. If Na⁺ is ≥170, use 0.9% saline initially—this contains 150mmol/L of sodium. Aim to reduce Na⁺ at a rate of less than 12mmol/L per day. Use of 0.45% saline can be dangerous. • Desmopressin 2μg IM (lasts 12–24h) may be used as a therapeutic trial.
Reference	Ohcm page 232,233
Dr Khalid/Rabia	<p>Diabetes Insipidus (D)</p> <p>Diabetes insipidus is a condition in which your ability to control the balance of water within your body does not work properly. The kidneys are not able to</p>

	<p>regulate the amount of water that passes out in the urine. This means that the patient passes large amounts of dilute urine – polyuria.</p> <p>Because of passing more urine, and therefore losing more fluid from the body, to try to compensate for this, the patient becomes thirstier and wants to drink more – polydipsia.</p> <p>Patients with this condition become dehydrated easily. The levels of sodium and potassium salts in the blood can also become unbalanced and too high.</p> <ul style="list-style-type: none"> - Points to look for: - Dilute Urine -> Low urine osmolality - Dehydration -> High serum sodium and hence high serum osmolality
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Q:1256	<p>26yo man presents with painless hematuria. He has no other complaints and on examination no other abnormality is found. What is the most appropriate initial inv to get to a dx?</p> <ul style="list-style-type: none"> a. Cystoscopy b. Midstream urine for culture c. Abdominal US d. MRI spine e. Coag screening
Clincher(s)	Young patient,painless hematuria
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KEY	
Additional Information	
Reference	
Dr Khalid/Rabia	<p>C.Abdominal US. As the patient is young so we can't go for cystoscopy. The initial investigation in this patient should be abdominal US to exclude any pathology in the genitourinary tract. PKD can also be suspected here so US should be done to exclude that aswell first.</p>

Q: 173	<p>A 15yo boy presents with generalized edema. His urinalysis reveals protein +++, eGFR =110. What is the most likely dx?</p> <ul style="list-style-type: none"> a. IgA nephropathy b. Membranous nephropathy c. Minimal change disease d. PSGN e. Lupus nephritis

Clincher(s)	
A	Hematuria, hypertension, oliguria. Post infectious- in nephritic syndrome
B	Nephritic syndrome – more in diabetic
C	Nephrotic- oedema
D	7-21 days after a streptococcal infection
E	More after After drugs (SLE kind)
KEY	The key is C. Minimal change disease. [Points in favour: i) Age 15 yrs ii) Generalized oedema iii) Protein in urine +++ vi) Normal eGFR of 110 (Normal range- 90 to 120 mL/min)].
Additional Information	<p>Minimal Change Disease (MCD, also known as Nil Lesions, Nil Disease, or lipoid nephrosis) is a disease of the kidney that causes nephrotic syndrome and usually affects children (peak incidence at 2–3 years of age).^[1]</p> <div data-bbox="429 701 1133 1574" data-label="Image"> <p>The diagram illustrates the histological changes in Minimal Change Disease. It shows a cross-section of a glomerular capillary wall with podocytes. Labels indicate 'Diffuse loss of foot processes' (the interdigitating structures between podocytes), 'Microvilli' (small projections on the podocyte surface), and 'Vacuolation' (swelling of the podocyte cytoplasm). Below the diagram, text states: 'The three hallmarks of Minimal Change Disease: diffuse loss of podocyte foot processes, vacuolation, and the appearance of microvilli.'</p> </div> <p>Minimal Change Disease is most common in very young children but can occur in older children and adults. It is by far the most common cause of nephrotic syndrome (NS) in children between the ages of 1 and 7, accounting for the majority (about 90%) of these diagnoses.^[2] Among teenagers who develop NS, it is caused by minimal change disease about half the time. It can also occur in adults but accounts for less than 20% of adults diagnosed with NS. Among children less than 10 years of age, boys seem to be more likely to develop minimal change disease than girls. Minimal change disease is being seen with increasing frequency in adults over the age of 80.</p>

People with one or more autoimmune disorders are at increased risk of developing minimal change disease. Having minimal change disease also increases the chances of developing other autoimmune disorders.

Most cases of MCD are idiopathic, but there have been causes of secondary MCD identified, including medications, immunizations, neoplasm, and infection. Case reports and literature reviews have shown an association between MCD and malignancies, particularly hematologic malignancies, such as Hodgkin's disease, non-Hodgkin lymphomas, or leukemias. Colorectal cancer-associated MCD is uncommon and has been reported in only a few cases to date.^[3]

Symptoms^[edit]

The symptoms are [proteinuria](#) (leakage of protein into the urine) and [edema](#) ([water retention](#)). [Nephrotic syndrome](#) (NS) is a general term that refers to the loss of protein in the urine, [hypoalbuminemia](#), and [edema](#). Many conditions are categorized as nephrotic syndromes—minimal change disease is unique, because it is the only one lacking any evidence of pathology on light microscopy (hence the name).

When protein is lost in the urine, the concentration of protein in the blood decreases, thereby reducing the intravascular [Oncotic pressure](#) relative to the interstitial tissue. The subsequent movement of fluid from the vascular compartment to the interstitial compartment manifests as the swelling known as [edema](#). Edema is commonly observed in the feet and legs, particularly in individuals with poorly functioning venous valves, and in the belly or abdomen (ascites), and around the eyes, but can occur anywhere, especially in response to gravity. Additionally, because of this extra fluid that stays in the body, individuals often gain weight and experience fatigue—in many patients, for example, clothes and shoes no longer fit. Some people notice that their urine becomes more frothy or foamy from the excess protein in the urine, and may find that they urinate less often.

Pathology^[edit]

For years pathologists found no changes when viewing specimens under light microscopy; hence the name minimal change disease. With the advent of [electron microscopy](#), the changes now known as the hallmarks for the disease were discovered. These are diffuse loss of [visceral epithelial cells](#) foot processes (podocyte effacement),^[4] vacuolation, and growth of microvilli on the visceral epithelial cells.

The pathology of minimal change disease is unclear and is currently

	considered idiopathic . The pathology does not appear to involve complement , immunoglobulins , or immune complex deposition. Rather, an altered cell-mediated immunologic response with abnormal secretion of lymphokines by T cells is thought to reduce the production of anions in the glomerular basement membrane , thereby increasing the glomerular permeability to serum albumin ^[5] through a reduction of electrostatic repulsion. ^[6] The loss of anionic charges is also thought to favor foot process fusion. ^[1] The etiological agent is somewhat of a mystery but viruses such as EBV , and food allergies have been implicated. Also, the exact cytokine responsible has yet to be elucidated, with IL-12 , IL-18 and IL-13 having been most studied in this regard, yet never conclusively implicated.
Reference	https://en.wikipedia.org/wiki/Minimal_change_disease
Dr Khalid/Rabia	Most common cause of nephrotic syndrome in children is minimal change disease. There will be hypoalbuminemia and peripheral edema too. Electron microscopy shows effacement of podocyte foot processes.. MCD has albumin selective proteinuria. Treatment is with steroids.

Q: 205	46yo man, known case of chronic GN presents to OPD. He feels well. BP = 140/90mmHg. Urine dipstick: protein ++, blood ++ and serum creatinine=106mmol/L. Which medication can prevent the progression of this dx? a. ACEi b. Diuretics c. Cytotoxic meds d. Longterm antibiotics e. Steroids
Clincher(s)	
A	Usually in diabetics : ACE in
B	
C	
D	
E	
KEY	The key is A. ACEI. [renal impairment is delayed by ACEI].
Additional Information	ACEI prevents progreesion to renal failure in the presence of proteinuria. ohcm pg 294 Except when protein too high Aci most importantly prevents proteinuria

Reference	
Dr Khalid/Rabia	

Q: 269	<p>A 66yo man, an hour after hemicolectomy has an urine output of 40ml. However, an hour after that, no urine seemed to be draining from the catheter. What is the most appropriate next step?</p> <p>a. IV fluids b. Blood transfusion c. Dialysis d. IV furosemide e. Check catheter</p>
Clincher(s)	
A	
B	
C	
D	
E	
KEY	<p>Key = E Points in favour = Always check catheter for any obstruction or other abnormality before iv fluids.</p>
Additional Information	<p>As the patient is post surgery, he would have already got enough IV fluids. blood tranfusion also not indicated you will check catheter as it might not be inserted properly or it came out which is not collecting the urine and makes us think there is no urine and the patient might have become anuric.</p>
Reference	
Dr Khalid/Rabia	

Q: 303	<p>A 34yo African-caribbean man with a hx of sarcoidosis has presented with bilateral kidney stones. What is the most likely cause for this pt's stones?</p> <p>a. Hypercalcemia b. Hyperuricemia c. Diet d. Recurrent UTIs e. Hyperparathyroidism</p>

Clincher(s)	
A	
B	
C	
D	
E	
KEY	The key is A. Hypercalcemia.
Additional Information	<p>Dysregulated calcium metabolism is a well-recognized complication of sarcoidosis, resulting in hypercalcaemia (prevalence 5–10%), hypercalcuria (40–62%) and reduced bone density (40–55%).</p> <p>Hypercalcuria is the most common defect of calcium metabolism in sarcoidosis, with a prevalence of 40–62% in published series [3, 4]. Clinically significant hypercalcaemia is less frequent and is generally asymptomatic, occurring in approximately 5% of patients [5]. Long-standing hypercalcaemia and hypercalcuria can cause nephrocalcinosis, which accounts for over half the patients with sarcoidosis who have renal impairment [6] and is the major cause of chronic renal failure. Other renal complications of sarcoidosis due to abnormal calcium metabolism include nephrolithiasis in approximately 10% of patients [7], and more rarely tubular disorders such as nephrogenic diabetes insipidus [8]. Symptomatic hypercalcaemia presenting with dehydration, polyuria and an altered conscious state is a rare but recognized complication of sarcoidosis.</p>
Reference	http://rheumatology.oxfordjournals.org/content/39/7/707.full
Dr Khalid/Rabia	Hypercalcemia in sarcoidosis is due to the uncontrolled synthesis of 1,25-dihydroxyvitamin D3 by macrophages. 1,25-dihydroxyvitamin D3 leads to an increased absorption of calcium in the intestine and to an increased resorption of calcium in the bone.

Q: 369	<p>A pt presents with complete anuria following prolonged hypotension and shock in a pt who bled profusely from a placental abruption. What is the most probable dx?</p> <ol style="list-style-type: none"> Post viral infection Acute papillary necrosis Acute cortical necrosis HUS Renal vein thrombosis
Clincher(s)	
A	
B	

C	
D	
E	
KEY	The key is C. Acute cortical necrosis.
Additional Information	<p>acute cortical necrosis) is a rare cause of acute kidney failure. The condition is "usually caused by significantly diminished arterial perfusion of the kidneys due to spasms of the feeding arteries, microvascular injury, or disseminated intravascular coagulation" and is the pathological progression of acute tubular necrosis.^[1] It is frequently associated with obstetric catastrophes such as abruptio placentae and septic shock, and is three times more common in developing nations versus industrialized nations (2% versus 6% in causes of acute kidney failure).^[citation needed]</p> <p>Causes^[edit]</p> <p>In adults^[edit]</p> <ul style="list-style-type: none"> • Pregnancy related (>50% of cases) <ul style="list-style-type: none"> • Placental abruption • Infected abortion • Prolonged intrauterine fetal death • Severe eclampsia • HIV^[2] • Snake bites^[3] • Binge drinking^[4] • Shock • Trauma • Sickle cell disease^[5] • Systemic lupus erythematosus (SLE)^[6] • Sepsis^[7] • SLE-associated antiphospholipid syndrome^[8] • Vitamin deficiency^[9] • Pancreatitis^[10] • Malaria^[11] • Meningococemia^[12] • Drug-induced toxicity (e.g. NSAIDs, Contrast Media, Quinine,^[13] or ATRA^[14]) <p>Neonatal^[edit]</p> <ul style="list-style-type: none"> • Congenital heart disease • Fetal-maternal transfusion • Dehydration • Perinatal asphyxia

	<ul style="list-style-type: none"> • Anemia • Placental hemorrhage • Severe hemolytic disease • Sepsis^[7] <p>Pathophysiology[edit]</p> <p>The exact pathologic mechanism for RCN is unclear, however the onset of small vessel pathology is likely an important aspect in the etiology of this condition. In general the renal cortex is under greater oxygen tension and more prone to ischemic injury, especially at the level of the proximal collecting tubule, leading to its preferential damage in a sudden drop in perfusion. Rapidly corrected acute renal ischemia leads to acute tubular necrosis, from which complete recovery is possible, while more prolonged ischemia may lead to RCN. Pathologically, the cortex of the kidney is grossly atrophied with relative preservation of the gross structure of the medulla. The damage is usually bilateral owing to its underlying systemic causes, and is most frequently associated with pregnancy (>50% of cases).^[1] It accounts for 2% of all cases of acute kidney failure in adults and more than 20% of cases of acute kidney failure during late pregnancy.^{[15][16]}</p> <p>Diagnosis[edit]</p> <p>While the only diagnostic "gold standard" mechanism of diagnosis en vivo is via kidney biopsy, the clinical conditions and blood clotting disorder often associated with this disease may make it impractical in a clinical setting. Alternatively, it is diagnosed clinically, or at autopsy, with some authors suggesting diagnosis by contrast enhanced CT.^[17]</p> <p>Treatment[edit]</p> <p>Patients will require dialysis to compensate for the function of their kidneys.</p> <p>Prognosis:</p> <p>Cortical necrosis is a severe and life-threatening condition, with mortality rates over 50%.^[citation needed] Those mortality rates are even higher in neonates with the condition due to the overall difficult nature of neonatal care and an increased frequency of comorbid conditions. The extent of the necrosis is a major determinant of the prognosis, which in turn is dependent on the duration of ischemia, duration of oliguria, and the severity of the precipitating conditions. Of those that survive the initial event, there are varying degrees of recovery possible, depending on the extent of the damage.</p>
Reference	https://en.wikipedia.org/wiki/Renal_cortical_necrosis
Dr Khalid/Rabia	There are 2 reasons for this acute cortical necrosis. i) significant diminished

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	arterial perfusion of the kidneys due to spasm of the feeding artery secondary to profuse bleeding from placental abruption ii) DIC secondary to placental abruption.
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Q:386	A 39yo man with acute renal failure presents with palpitations. His ECG shows tall tented T waves and wide QRS complex. What is the next best step? a. Dialysis b. IV calcium chloride c. IV insulin w/ dextrose d. Calcium resonium e. Nebulized salbutamo
Clincher(s)	Tall tented t waves and wide qrs and acute renal failure
A	Persistent hyper kalemia >7mmol/l needs dialysis
B	
C	Once cardio protective agent is given then we give this.
D	Calcium resonium is a polystyrene cation exchange resin which acts to remove excess potassium from the body by exchanging it for the cation ion (Ca++) in the resin. It is a relatively slow method of reducing serum potassium.
E	Can be given if insulin and dextrose can't be given as it helps moves the potassium inside the cell but can increase the pulse.
KEY	B
Additional Information	
Reference	
Dr Khalid/Rabia	IV calcium chloride (both IV calcium gluconate or IV calcium chloride can be used) when there is ECG changes. DX The ECG changes are suggestive of Hyperkalemia. At potassium level of >5.5mEq/L occurs tall tented T waves and at potassium level >7mEq/L occurs wide QRS complex with bizarre QRS morphology.

Q:556	. A pt after transurethral prostatic biopsy. What electrolyte imbalance can he develop? a. Hyperkalemia b. Hyponatremia c. Hypocalcemia d. Hypernatremia e. Hypercalcemia
Clincher(s)	Transurethral prostatic biopsy done

A	
B	
C	
D	
E	
KEY	B
Additional Information	
Reference	
Dr Khalid/Rabia	<p>Transurethral Resection of the Prostate (TURP) Syndrome is a rare but potentially life-threatening complication of a transurethral resection of the prostate TURP procedure.</p> <p>It occurs as a consequence of the absorption into the prostatic venous sinuses of the fluids used to irrigate the bladder during the operation. Symptoms and signs are varied and unpredictable, and result from fluid overload and disturbed electrolyte balance and hyponatraemia. Treatment is largely supportive and relies on removal of the underlying cause, and organ and physiological support. Preoperative prevention strategies are extremely important.</p>

Q:598	<p>A 72yo woman who is taking loop diuretics is suffering from palpitations and muscle weakness.</p> <p>What is the electrolyte imbalance found?</p> <p>a. Na+ 130mmol/l, K+ 2.5mmol/l b. Na+ 130mmol/l, K+ 5.5mmol/l c. Na+ 140mmol/l, K+ 4.5mmol/l d. Na+ 150mmol/l, K+ 3.5mmol/l e. None</p>
Clincher(s)	Loop diuretic electrolyte imbalance
A	
B	
C	
D	
E	
KEY	A
Additional Information	<p><i>Normal values</i></p> <p><i>Na: 135-145mmol/l</i></p> <p><i>K: 3.5-5mmol/l</i></p>
Reference	
Dr Khalid/Rabia	Hypokalemia can occur with loop or thiazide diuretics.

Q:632	<p>A 34yo man complains of arthralgia, abdominal pain and vomiting, a facial rash that is worse in the summer and hematuria. Urea and creatinine are slightly elevated with urinalysis demonstrating red cell casts. PMH is remarkable for childhood eczema. Which inv is most likely to lead to a dx?</p> <p>a. US KUB b. Joint aspiration c. Auto antibodies d. IVU e. Renal biopsy</p>
Clincher(s)	Arthralgia, abd pain, vomiting, facial rash worse in summer, haematuria , urge and creatinine slightly elevated, hvx of childhood eczema
A	
B	
C	
D	
E	
KEY	C
Additional Information	
Reference	
Dr Khalid/Rabia	<p>Dr Rabia HSP mostly in children after a viral infection and with a palpable purpura on buttocks and extensor surfaces. JAKS</p> <p>Si can't be HSP. SLE yes I agree a lot of presentations going in favour of SLE. arthralgias rash photosensitivity renal involvement. I don't recall abdominal pain and vomiting in SLE. SLE - Facial Rash, worsen by sunlight, arthralgia, Nephritis, Other immune problems (eczema)</p> <p>Dr Khalid The key is C. Auto antibodies. [Likely diagnosis is SLE for which auto antibody (anti ds DNA antibody) should be done].</p>

Q:689	<p>A 10yo boy presents with generalized swelling. This has been present for 4days and included swollen ankles and puffiness of the face. It started a few days after he had a mild cold with runny nose. His only PMH was eczema. Urine analysis: hematuria, proteinuria 10g/24h, creat 60umol/l and albumin=15g/l. What is the single most likely dx?</p>
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	a. IgA nephropathy b. HSP c. Minimal change nephropathy d. Wilson's disease e. Cardiac failure
Clincher(s)	Swollen ankles, puffiness of face, proteinuria, hx of eczema, low albumin, mild cold and runny nose.
A	Typical pt is young man with episodic hematuria
B	Systemic variant of IgA nephropathy with flitting arthritis, extensor purpurin rash, abd pain and nephritis (JAKS)
C	No hx of hematuria
D	Not relevant in this scenario
E	Not relevant in this scenario
KEY	A
Additional Information	<p>Illnesses that can precipitate haematuria include urinary tract infection, pneumonia, staphylococcal infection, acute gastroenteritis, influenza and glandular fever.</p> <p>The disease can be highly variable, ranging from microscopic haematuria to rapidly progressive glomerulonephritis</p> <p>Tx: BP control with Acei and steroids for immunosuppression.</p>
Reference	
Dr Khalid/Rabia	<p>the typical presentation of nephritic syndrome as well as we know the patient upper respiratory tract infection then it lead to immune response</p> <p>no hematuria in minimal change</p> <p>HSP presents typically with the following JAKS joints abdomen kidney and skin arthritis, abdominal pain, haematuria and petechiae In hsp there is purpura on extensor surfaces, abdominal pain and nephritis (systemic)</p> <p>iga nephropathy after 2-3 days of cold infection, post strep glomerulonephritis after 1-12wks of URTI</p>

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Q: 992	<p>A 27yo man presents with abdominal pain. He says his urine is dark. Exam: BP=160/105mmHg. What is the most appropriate inv?</p> <p>a. US</p> <p>b. Renal biopsy</p> <p>c. CT</p> <p>d. Urine protein</p> <p>e. Urine microscopy</p>
Clincher(s)	
A	U/s usually confirms
B	Unexplained acute kidney injury or chronic kidney disease, acute nephritic syndromes, unexplained proteinuria and haematuria, systemic diseases associated with kidney dysfunction, suspected transplant rejection
C	
D	
E	
KEY	<p>Ans:A</p> <p>USG will confirm</p> <p>Abdominal pain, hematuria and hypertension are classic feature of autosomal dominant polycystic kidney disease. The disease process usually begins before the age of 30 yrs and renal failure are evident at about 60 yrs of age</p>
Additional Information	<p>Ohcm (p299) or to guide treatment. 29</p> <p>Renal biopsy :</p> <p>Pre-procedure: Check FBC, clotting, group and save. The physician performing the procedure should obtain written informed consent. Ultrasound to delineate anatomy. Stop anticoagulants (aspirin 1 week, warfarin 2–3 days, low molecular weight heparin 24 hours).</p> <p>Contraindications: • Abnormal clotting • Hypertension >160/>90mmHg • Single kidney (except for renal transplants) • Chronic kidney disease with small kidneys (<9cm) • Uncooperative patient • Horseshoe kidney • Renal neoplasms.</p>
Reference	
Dr Khalid/Rabia	

Q:999	<p>999. A 65yo man complains of hematuria, frequency, hesitancy and nocturia. He reports that on certain occasions he finds it difficult to control the urge to pass urine. Urine microscopy confirms the presence of blood but no</p>
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	<p>other features. What is the most probable dx?</p> <ol style="list-style-type: none"> BPH Bladder ca Prostatic ca Pyelonephritis Prostatitis
Clincher(s)	65 yr, hematuria , frequency , hesitancy , nocturia, blood in urinalysis
A	
B	<p>Painless haematuria must be treated as malignancy of the urinary tract until proved otherwise. Assoc with smoking / occup h/o dyes ,paint (aromatic amine),schistosomiasis(usually in developing countries ..Squamous cell tumours usually follow chronic inflammation from stones or indwelling catheters.7th leading cancer in uk .ranking 4th among males</p>
C	Most common cancer in men in uk .over age 65
D	Fever , burning micturation , abd pain
E	
KEY	<p>ANS: C</p> <p>Reason: Prostatic CA, presents with symptoms of LUTS(lower urinary tract symp) initially and in locally invasive disease there can be hematuria,dysuria,incontinence.</p>
Additional Information	<p>Referral guidelines for ca bladder</p> <p>Patientinfo.co</p> <p>Current recommendations are for the following patients to be referred urgently for a urological assessment:</p> <ul style="list-style-type: none"> Adults over 45 years and have unexplained visible haematuria without urinary tract infection. Adults over 45 years with visible haematuria that persists or recurs after successful treatment of urinary tract infection. Adults aged 60 and over and have unexplained non visible haematuria and either dysuria or a raised white cell count on a blood test. <p>A non-urgent referral for bladder cancer is recommended for those people aged 60 and over with recurrent or persistent unexplained urinary tract</p>

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	infection.
Reference	
Dr Khalid/Rabia	

Q:1041	<p>A 79yo man who is being treated with GnRH antagonist for proven adenocarcinoma of the prostate attends a follow up session. What is the most appropriate inv?</p> <p>a. Serum AFP b. Serum PSA c. Serum acid phosphates conc d. Serum ALP isoenzyme conc e. Trans rectal US</p>
Clincher(s)	
A	Liver carcinoma – following up patients
B	urologists rely on rising PSA results to signal that a radical intervention (usually either chemotherapy or radiotherapy) is necessary. This is particularly appropriate for older patients with comorbidities
C	
D	
E	
KEY	B
Additional Information	<p>urologists rely on rising PSA results to signal that a radical intervention (usually either chemotherapy or radiotherapy) is necessary. This is particularly appropriate for older patients with comorbidities, on the basis that they are likely to die of some other cause before a slow-growing prostate tumour has an effect on their lifespan. Such 'active monitoring' is also appropriate for any patient who wishes to avoid the side-effects of interventional management. Most prostate cancers are adenocarcinomas arising in the peripheral zone of the prostate gland</p> <p>Risk factors: Age Black-african Family hx · · Factors such as food consumption, pattern of sexual behaviour, alcohol consumption, exposure to ultraviolet radiation, chronic inflammation and occupational exposure have all been considered as possible risk factors</p> <p>* Local disease: o Raised PSA on screening. o Weak stream, hesitancy, sensation of incomplete emptying, urinary</p>

frequency, urgency, urge incontinence.

- o Urinary tract infection.
- * Locally invasive disease:
 - o Haematuria, dysuria, incontinence.
 - o Haematospermia.
 - o Perineal and suprapubic pain.
 - o Obstruction of ureters, causing loin pain, anuria, symptoms of acute kidney injury or chronic kidney disease.
 - o Impotence.
 - o Rectal symptoms - eg, tenesmus.
- * **Metastatic disease:**
 - o Bone pain or sciatica.
 - o Paraplegia secondary to spinal cord compression.
 - o Lymph node enlargement.
 - o Loin pain or anuria due to ureteric obstruction by lymph nodes.
 - o Lethargy (anaemia, uraemia).
 - o Weight loss, cachexia
- * **Abdominal palpation may demonstrate a palpable bladder due to outflow obstruction.**
- * **DRE may reveal a hard, irregular prostate gland. Indications of possible prostate cancer are:**
 - o Asymmetry of the gland.
 - o A nodule within one lobe.
 - o Induration of part or all of the prostate.
 - o Lack of mobility - adhesion to surrounding tissue.
 - o Palpable seminal vesicles.

Differential diagnosis * All other causes of haematuria (eg, urinary tract infection) and urinary tract obstruction. * Benign prostatic hyperplasia. * Prostatitis. * Bladder tumours.

INV:

PSA

Transrectal needle biopsy

- **Urinalysis** to exclude renal and bladder pathology. Urine sent for microscopy, culture and sensitivities.
- **Renal function tests** to help exclude renal disease.

MRI should be considered for men with a negative TRUS core biopsy to determine whether another biopsy is needed

MRI for staging

Bone scan for mets

The National Institute for Health and Care Excellence (NICE) referral guidelines for suspected cancer state:[11]

- * Men presenting with symptoms suggesting prostate cancer should have a DRE and PSA test after counselling. Symptoms will be related to the lower urinary tract and may be inflammatory or obstructive. Prostate cancer is also a possibility in male patients with any of the following unexplained symptoms: erectile dysfunction, haematuria, lower back pain, bone pain or weight loss, especially in the elderly.

	<p>* Urinary infection should be excluded before PSA testing, especially in men presenting with lower tract symptoms. The PSA test should be postponed for at least one month after treatment of a proven urinary infection.</p> <p>* If a hard, irregular prostate typical of a prostate carcinoma is felt on DRE, then the patient should be referred urgently. The PSA should be measured and the result should accompany the referral.</p> <p>* Patients do not need urgent referral if the prostate is simply enlarged and the PSA is in the age-specific reference range.</p> <p>* In a man with or without LUTS and in whom the prostate is normal on DRE but the age-specific PSA is raised or rising, an urgent referral should be made. Symptomatic patients with high PSA levels should be referred urgently.</p> <p>* If there is doubt about whether to refer an asymptomatic man with a borderline level of PSA, the PSA test should be repeated after an interval of one to three months. If the second test indicates that the PSA level is rising, the patient should be referred urgently.</p> <p>Rx: low risk localized tumor: active surveillance/surgery (personal preference) Intermediate to high risk:</p> <ul style="list-style-type: none"> • Men with intermediate and high-risk localised prostate cancer should be offered a combination of radical radiotherapy and androgen deprivation therapy, rather than radical radiotherapy or androgen deprivation therapy alone. Brachytherapy (a form of radiotherapy in which radiation is targeted directly at the prostate gland) in combination with external-beam radiotherapy is a treatment option for localised prostate cancer. • Men with intermediate and high-risk localised prostate cancer should be offered 6 months of androgen deprivation therapy before, during or after radical external beam radiotherapy. • Continuing androgen deprivation therapy for up to 3 years should be considered for men with high-risk localised prostate cancer. • High-dose rate brachytherapy in combination with external beam radiotherapy should be considered for men with intermediate and high-risk localised prostate cancer. Brachytherapy alone should not be offered to men with high-risk localised prostate cancer. <p>Locally advanced: pelvic radiotherapy Metastasis: bilateral orchidectomy</p> <ul style="list-style-type: none"> • Urinary tract obstruction, acute kidney injury, chronic kidney disease. • Sexual dysfunction: erectile dysfunction, loss of libido. • Metastatic spread: bone pain, pathological fractures, spinal cord compression.
Reference	
Dr Khalid/Rabia	

Q:1057	<p>A 30yo man presents to hosp complaining that his urine has been very dark recently, resembling coffee at worst. He has been under the weather 2wks back and had taken a few days off work with a sore throat and coryzal</p>
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	<p>symptoms. Urine dipstick in hosp returns highly positive for blood and protein. He is admitted for supportive management and is scheduled for a renal biopsy, which shows mesangial proliferation with a positive immune-fluorescence pattern. What is the most probable dx?</p> <p>a. Membranous glomerulonephropathy b. SLE c. Wegener's granulomatosis d. Post – strep GN e. IgA nephropathy</p>
Clincher(s)	
A	
B	
C	
D	
E	
KEY	<p>Dx is Post strep GN History of sore throat, mesangial proliferation and immune fluorescence pattern point to post strep GN. Presentation is usually nephritic syndrome. Renal biopsy isn't performed unless atypical presentation. IF shows IgG and C3 deposits Serology shows inc ASOT and inc C3 Supportive treatment with more than 95% function recovered</p>
Additional Information	
Reference	
Dr Khalid/Rabia	

Q:1059	<p>1059. A 10yo boy is brought to the hosp with a rash over his buttocks a/w abdominal pain and vomiting. In the ED, he is accompanied by his mother and stepfather. His mother had left him for the weekend with the stepfather and was called to come back from holiday as he started to have some hematuria with the rash. Social services had been notified on arrive to hospital. What is the most probably dx?</p> <p>a. NAI b. ITP c. HSP d. ALL e. HUS</p>

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Clincher(s)	Rash on buttocks (extensor), abd pain , vomiting
A	NAI-
B	Idiopathic thrombocytopenic purpura –present in women of child bearing age most common .also occurs in 2-6yrs of children signs of thrombocytopenia ,minor bleeding , easy bruising , petechia ,heamatemesis , melena.no splenomegaly IgG antibodies formed against patients platelets
C	Henoch schonlein purpura HSP) is a systemic variant of IgA nephropathy, causing a small vessel vasculitis. <i>Features:</i> Purpuric rash on extensor surfaces (typically on the legs), fl itting polyarthritis, abdominal pain (GI bleeding) and nephritis. <i>Diagnosis:</i> Usually clinical. Confi rmed with positive IF for IgA and C3 in skin or renal biopsy(identical to IgA nephropathy). <i>Treatment:</i> Same as IgA nephropathy. <i>Prognosis:</i> 15% nephritic patients □ ESRF; if both nephritic and nephrotic syndrome, 50% □ ESRF.
D	Acute lymphocytic leukemia
E	Hemolytic uremic syndrome- follows after diarrhhea
KEY	C) HSP HSP is a Small vessel vasculitis with purpura (non blanching purple papules)- buttocks and extensor surfaces. Young. Glomerulonephritis, arthritis, abd pain (+_ intussusception) may mimic an acute abdomen. Rx is supportive.
Additional Information	
Reference	
Dr Khalid/Rabia	

Q: 94	<p>A 64yo man has been waking up in the middle of the night to go to the bathroom. He also had difficulty in initiating micturition and complains of dribbling. A dx of BPH was made after a transrectal US guided biopsy and the pt was prepared for a TURP. What electrolyte abnormality is highly likely due to this surgery?</p> <p>a. Hypokalemia</p> <p>b. Hypocalcemia</p> <p>c. Hyperkalemia</p> <p>d. Hyponatremia</p>
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	e. Hypernatremia
Clincher(s)	
A	
B	
C	
D	<p>Absorption of fluid used for bladder irrigation (via 3 way catheter) to flush out blood clots and IV fluids all may lead to hypervolaemia and dilutional hyponatremia (Turp Syndrome)</p> <p>(The liquid (glycine) used for irrigation of the bladder and prostate gets absorbed by the venous sinuses leading to hyponatremia, fluid overload and in the some cases glycine toxicity.)</p>
E	
KEY	The key is D. Hyponatremia.
Additional Information	<p>Post splenectomy they always ask about two complications.</p> <p>1. Acute gastric dilatation((they will say patient with signs of shock n some coffee ground color blood was collected on insertion of Ryles tube (NG tube) after splenectomy)). Here answer will be acute gastric dilatation.</p> <p>2. Thrombocytosis is second major post splenectomy complication. They will give u a patient with splenectomy done despite being mobile he developed DVT what might be the cause. U shud mark splenectomy</p>
Reference	
Dr Khalid/Rabia	

Q: 96	<p>A 35yo man presented with hematuria, abdominal swelling and has a BP of 190/140. What is the most diagnostic inv?</p> <p>a. Cystoscopy</p> <p>b. USG</p> <p>c. CT</p> <p>d. Renal biopsy</p>
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	e. Urine analysis								
Clincher(s)	Age and bp is the clincher.								
A									
B	The diagnosis is ADPKD.								
C									
D									
E									
KEY	The key is B. USG.								
Additional Information	The sensitivity of USG for PKD is nearly 100% in those above 20 years of age								
Reference									
Dr Khalid/Rabia	<p>In given case patients age is 35. So the USG diagnostic criteria is: Age 18 – 39 yrs>3 unilateral or, bilateral cysts (here bilateral means if 1 + 1 it is enough).</p> <p>Autosomal dominant polycystic kidney disease (ADPKD) is the most common inherited cause of kidney disease, affecting 1 in 1,000 Caucasians. Two disease loci have been identified, PKD1 and PKD2, which code for polycystin-1 and polycystin-2 respectively</p> <table border="1"> <thead> <tr> <th>ADPKD type 1</th><th>ADPKD type 2</th></tr> </thead> <tbody> <tr> <td>85% of cases</td><td>15% of cases</td></tr> <tr> <td>Chromosome 16</td><td>Chromosome 4</td></tr> <tr> <td>Presents with renal failure earlier</td><td></td></tr> </tbody> </table> <p>The screening investigation for relatives is abdominal ultrasound:</p>	ADPKD type 1	ADPKD type 2	85% of cases	15% of cases	Chromosome 16	Chromosome 4	Presents with renal failure earlier	
ADPKD type 1	ADPKD type 2								
85% of cases	15% of cases								
Chromosome 16	Chromosome 4								
Presents with renal failure earlier									

	<p>Ultrasound diagnostic criteria (in patients with positive family history)</p> <ul style="list-style-type: none"> • two cysts, unilateral or bilateral, if aged < 30 years • two cysts in both kidneys if aged 30-59 years • four cysts in both kidneys if aged > 60 years
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Q: 98	<p>A 65yo man presented with frank hematuria. He has no other urinary symptoms. What is the most appropriate next step that will lead to the dx?</p> <p>a. IVU</p> <p>b. US Abdomen</p> <p>c. Cystoscopy</p> <p>d. Mid-stream urine for culture</p> <p>e. Transrectal US</p>
Clincher(s)	
A	
B	
C	Bladder cancer. Age 65, asymptomatic haematuria. ADPKD [at the beginning there is very few or no symptoms]
D	
E	
KEY	Key is C. Cystoscopy.
Additional Information	<p>Controversy: opinion divided with BPH : Bph with obstructive uropathy causing glomerulonephropathy and accounting for hematuria plus nocturia also suggesting he must be a diabetic so it's BPH here</p> <p>Others say: In exams hematuria suggests a more alarming underlying cause like ca prostate despite da fact that it does occur in BPH as well.</p>
Reference	

Dr Khalid/Rabia	
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Q: 111	A 15yo male has bilateral ankle edema. His BP=110/70mmHg and urinalysis shows protein++++. What is the most likely dx? a. HUS b. IgA nephropathy c. Membranous GN d. Minimal change GN e. Nephrotic syndrome
Clincher(s)	15 yr
A	
B	
C	
D	Points in favour: i) Age 15 ii) Ankle oedema iii) Normotension iv) Heavy proteinuria. Ans. 3. Treatment of choice is steroid (prednisolone). Failure of steroid or frequent relapse (>3) cyclophosphamide.
E	
KEY	. The key is D. Minimal change disease.
Additional Information	
Reference	
Dr Khalid/Rabia	<p>Most common cause of nephrotic in children is minimal change disease. There will be hypoalbuminemia and peripheral edema too. Electron microscopy shows effacement of podocyte foot processes.. MCD has albumin selective proteinuria. Treatment is with steroids.</p> <p>IgA nephropathy is nephritic and will also show HTN and microscopic hematuria and follows upper resp tract infection.</p> <p>Membranous GN also presents as nephrotic but age and since MCD is most common we choose MCD.</p> <p>Nephrotic syndrome itself is not a diagnosis.</p> <p>Minimal change disease(commonest csuse of nephrotic syndrome.in young agegroup) is the key. Loss of foot processes of podocytes leads to proteinuria and hence edema.</p> <p>It's definitely nephrotic synd... While minimal change disease is common in children.. It peaks at 2-3 years old.. This guy being 15 is well above the age range and we have no other clue pointing to a more specific diagnosis</p>

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	Nephrotic syndrome. points of diagnosing is proteinuria, bilateral ankle edema and no hypertension
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Q: 150	<p>A 5yo boy is referred to the hospital and seen with his father who is worried that he has been listless. He is not sure why his GP suggested he should come to the ED and is keen to get some tablets and go home. Exam: tired and irritable, swelling around eyes. Renal biopsy: remarkable for podocyte fusion on EMicros. What is the most probable dx?</p> <p>a. NAI b. Myelodysplastic disease c. HSP d. Membranous GN e. Minimal change GN</p>
Clincher(s)	
A	
B	
C	
D	
E	most common form of nephrotic syndrome in children. facial edema specially around the eyes malaise & easy fatigue ability occurs in same is happening with the pt in given scenario
KEY	The key is E. Minimal change glomerulonephritis. [Podocyte fusion on electron microscopy]
Additional Information	
Reference	
Dr Khalid/Rabia	Most common cause of nephrotic syn in children is minimal change disease. There will be hypoalbuminemia and peripheral edema too. Electron microscopy shows effacement of podocyte foot processes.. MCD has albumin selective proteinuria. Treatment is with steroids.
Q: 1	<p>A 65yo man presents with painless hematuria, IVU is normal, prostate is mildly enlarged with mild frequency. What is the most appropriate next step?</p> <p>a. US Abdomen b. Flexible cystoscopy c. MRI d. Nuclear imaging e. PSA</p>

Clincher(s)	Painless Haematuria, mildly enlarged, mild frequency
A	
B	Painless haematuria → exclude Bladder Cancer
C	
D	
E	
KEY B	Definitive inv to exclude Bladder Cancer
Additional Information	<div> <div>648</div> <div>Bladder tumours</div> <p>>90% are transitional cell carcinomas (TCCs) in the UK. What appear as benign papillomata rarely behave in a purely benign way. They are almost certainly indolent TCCs. Adenocarcinomas and squamous cell carcinomas are rare in the West (the latter may follow schistosomiasis). UK incidence ≈ 1:6000/yr. ♂:♀ ≈ 5:2. Histology is important for prognosis: Grade 1—differentiated; Grade 2—intermediate; Grade 3—poorly differentiated. 80% are confined to bladder mucosa, and only ~20% penetrate muscle (increasing mortality to 50% at 5yrs).</p> <p>Presentation Painless haematuria; recurrent UTIs; voiding irritability.</p> <p>Associations Smoking; aromatic amines (rubber industry); chronic cystitis; schistosomiasis (risk of squamous cell carcinoma); pelvic irradiation.</p> <p>Tests</p> <ul style="list-style-type: none"> • Cystoscopy with biopsy is diagnostic. • Urine: microscopy/cytology (cancers may cause sterile pyuria). • CT urogram is both diagnostic and provides staging. • Bimanual EUA helps assess spread. • MRI or lymphangiography may show involved pelvic nodes. </div>

Surgery

	<p>Haematuria Blood in the urine may arise from anywhere in the renal tract. It is classified as visible (VH—previously known as macroscopic, frank) or non-visible (NVH—found on dipstick or microscopy). Non-visible is subdivided into symptomatic (sNVH—with LUTS—dysuria, hesitancy, urgency, p644) and asymptomatic (aNVH). Dipstick of fresh urine is more sensitive than MSU, which has a high false -ve rate, therefore no need to confirm every positive dip with microscopy.³ However, MC&S of urine is useful for further analysis of cause. Patients with one episode of VH, one of sNVH or persistent aNVH require further assessment.</p> <ul style="list-style-type: none"> • Exclude transient causes: (UTI, vigorous exercise, menstruation). • Check creatinine/eGFR, proteinuria (spot ACR or PCR) and BP. • Painless VH usually means bladder cancer (p648); refer urgently. • In aNVH or if systemic symptoms, consider FBC, ESR, CRP, blood film, clotting (NB do not simply attribute haematuria to anticoagulants/antiplatelet agents).³ • Urine MC&S to look for infection, malignant/inflammatory cells, casts, crystals. • USS and rapid referral to nephrology if rapid decline in GFR or haematuria with proteinuria, casts or dysmorphic cells. Red cell casts ≈ glomerular bleeding.
Reference	OHCM 286 , 648
Dr Khalid/Rabia	<p>Ans. 1. The key is B. Flexible cystoscopy.</p> <p>Ans. 2. Painless hematuria in an elderly (here 65 years old man) indicates carcinoma bladder for which flexible cystoscopy is done.</p> <p>An elderly gentleman complaining of painless hematuria : always exclude bladder cancer</p> <p>The most important and definite Investigation for bladder cancer is a cystoscopy+ Biopsy.</p> <p>Initially : Urine microscopy but it does not rule out CA.</p> <p>Other causes of painless hematuria are rhabdomyolysis , coagulation disorder , prostate cancer , hemolytic anemia , renal tumor , and polycystic kidney disease</p> <p>you can exclude those by absence of :</p> <ol style="list-style-type: none"> 1- History of crush injury for rhabdomyolysis 2- No bleeding from other orifices for coagulation disorder 3- No symptoms of prostatism for Prostate Cancer 4- No signs of anemia 5- No tenderness in loin or masses (renal tumor) 6- No hypertension (in polycystic kidney) <p>although other investigations like Mid urine sample , IVU , may show UTI , other findings like filling defects , etc.. they dont help with diagnosis and prognosis</p> <p>Diagnosis : Bladder CA. (1 in 10,000)</p> <p>Most common : Transitional cell CA. 3x in MEN of 50+ age.</p> <p>Inc factors :</p> <p>Smoking, schistosomiasis, rubber dye industries, White ppl, recurrent infections.</p>

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	Symptoms : Painless hematuria (on and off) Pain in lower abdomen Treatment : TUR with 1 chemotherapy within 24 hours. If needed, BCG is used for next chemo cycles.
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Q: 4	<p>79yo anorexic male complains of thirst and fatigue. He has symptoms of frequency, urgency and terminal dribbling. His urea and creatinine levels are high. His serum calcium is 1.9 and he is anemic. His BP is 165/95 mmHg. What is the most probable dx?</p> <p>a. BPH b. Prostate carcinoma c. Chronic pyelonephritis d. Benign nephrosclerosis</p>				
Clincher(s)					
A					
B					
C					
D					
E					
KEY					
Additional Information					
Reference					
Dr Khalid/Rabia	<p>First to say in this case (almost all features goes in favour of prostatic carcinoma like- frequency, urgency and terminal dribbling are features of prostatism; Age, anorexia and anaemia favours carcinoma prostate diagnosis and it would be accurate presentation if it was hypercalcaemia. But given calcium level is of hypocalcaemic level and it is the main cause of discrepancy of this question). Renal failure can be an association of malignant disease and can cause high BP. Thirst is a feature of hypercalcaemia (here may be erroneously calcium level is given in hypocalcaemic level ; probably a bad recall). Prostate biopsy is the confirmatory diagnosis and others like PSA is suggestive. This is what I could pointed out. If there is any better explanation please place it to correct the answer- any one please.</p> <table border="1"> <tr> <td>Calcium</td><td>2.1-2.6</td></tr> <tr> <td>m</td><td>mmol/l</td></tr> </table>	Calcium	2.1-2.6	m	mmol/l
Calcium	2.1-2.6				
m	mmol/l				

	<p>I think this patient has CKD secondary to prostate CA which leads to hypocalcemia due to vit D def.</p> <p>Osteoblastic metastases — Occasional patients with widespread osteoblastic metastases, particularly those with breast or prostate cancer, have hypocalcemia.</p> <p>Diagnosis : Prostate Cancer Most common CA in men of uk. 1 in 8 men. After 65. Risk factors: Fatty diet, exposure to cadmium, ageing n family history.</p> <p>Symptoms : Poor stream, hesitancy, dribbling, frequency, urgency, poor emptying.</p> <p>Invs : Examine. PSA levels. Confirmatory test : Biopsy.</p> <p>Grading : Gleason Score. 4 or less - well differentiated. 10 yr risk of local progression 25% 5 - 7 - moderately differentiated. 50% risk Over 7 - poorly differentiated. 75% risk</p> <p>Risk assessment PSA levels. Low - <10 and gleason score 6 or below Intermediate - psa 10 to 20 or gleason score 7 High - psa >20 or gleason 8 to 10.</p> <p>Staging : MRI preferred over CT. Treatment : Surgery. Radical prostatectomy. S/E impotence, incontinence of urine. Radiotherapy. External and internal (brachytherapy) HRT to stop TESTOSTERONE. Medicines - LHRH. Goserelin, leuprorelin, triptorelin (act on pitutary) and Flutamide, cyproterone (anti androgenic) Prognosis : variable. Depends on the stage. Complications : UTi, AKI, CKD, sexual dysfunction, metastasis.</p> <p>Note : Prostate CA has increased risk with HYPERCALCEMIA. Not hypo. So the statement seems to be wrong. Even BPH has nothing to do with Ca levels. Benign nephrosclerosis is due to long standing HTN. No link to prostate found.</p>
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Q: 11	An 80yo man presented with pain in his lower back and hip. He also complains of waking up in the night to go to the washroom and has urgency as well as dribbling. What is the most likely dx? a. BPH b. Prostatitis c. UTI d. Prostate carcinoma e. Bladder carcinoma
Clincher(s)	
A	
B	
C	
D	Age, nocturia, urgency and dribbling points towards prostate pathology. Pain of lower back and hip points towards bony metastases from prostate cancer.
E	
KEY D	Symptoms match with PC
Additional Information	
Reference	
Dr Khalid/Rabia	DISCUSSED IN MCQ 4. Ans. 1. D. Prostate carcinoma. Ans. 2. Age, nocturia, urgency and dribbling points towards prostate pathology. Pain of lower back and hip points towards bony metastases from prostate cancer. Ans. 3. Blood test for PSA; Prostate biopsy; MRI [if initial biopsy is negative, to decide repeat biopsy]. Source NICE. Ans. 4. Treatment options: 1. Active treatment [i) radical prostatectomy ii) radical radiotherapy iii) hormone therapy iv) brachytherapy v) pelvic radiotherapy vi) orchidectomy] 2. Active surveillance 3. Watchful waiting 4. Palliative care [Source: NICE].

Q: 34	A 32yo man presented with painless hematuria. He is hypertensive but the rest of the exam is unremarkable. What is the most likely dx? a. Polycystic kidneys b. Ca bladder c. Ca prostate d. TTP
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	e. HUS
Clincher(s)	Painless Haematuria , hypertension
A	Correct answer
B	
C	
D	
E	
KEY A	
Additional Information	
Reference	
Dr Khalid/Rabia	<p>Ans. 1. A. Ans. 2. Painless haematuria at an younger age with hypertension. Ans. 3. Renal ultrasound.</p> <ul style="list-style-type: none"> • autosomal dominant • Gross haematuria following trauma is a classic presenting feature of ADPKD, Advise against participating in contact sports which risk abdominal trauma • polycystic kidneys can produce excess erythropoietin and hence raise Hb • Angiotensin-converting enzyme (ACE) inhibitors or angiotensin-II receptor antagonists are the preferred choice

Q: 71	<p>A 75yo alcoholic presents with a mass up to umbilicus, urinary dribbling, incontinence, and clothes smelling of ammonia. What is the next step in management?</p> <p>a. Urethral catheter b. Suprapubic catheter c. Antibiotics d. Condom catheter e. Nephrostomy</p>
Clincher(s)	
A	Correct
B	
C	
D	
E	

KEY A	Alcohol consumption → retention
Additional Information	
Reference	
Dr Khalid/Rabia	<p>Q. 1. What is the key?</p> <p>Q. 2. What is the cause of this retention?</p> <p>Ans. 1. The key is A. Urethral catheter.</p> <p>Ans. 2. Alcohol consumption (it is rather a less common cause of urinary retention).</p> <p>ACUTE URINARY RETENTION</p> <p>Causes of urinary retention</p> <ul style="list-style-type: none"> In men - BPH, meatal stenosis, <u>paraphimosis</u>, penile constricting bands, <u>phimosis</u>, <u>prostate cancer</u>. In women - prolapse (<u>cystocele</u>, <u>rectocele</u>, uterine), <u>pelvic mass</u> (gynaecological malignancy, uterine <u>fibroid</u>, ovarian cyst), retroverted gravid uterus. In both - <u>bladder calculi</u>, <u>bladder cancer</u>, <u>faecal impaction</u>, gastrointestinal or retroperitoneal malignancy, urethral strictures, foreign bodies, stones. <p>Infectious and inflammatory:</p> <ul style="list-style-type: none"> In men - <u>balanitis</u>, prostatitis and prostatic abscess. In women - acute <u>vulvovaginitis</u>, vaginal <u>lichen planus</u> and <u>lichen sclerosis</u>, vaginal <u>pemphigus</u>. In both - <u>bilharzia</u>, <u>cystitis</u>, <u>herpes simplex virus</u> (particularly primary infection), peri-urethral abscess, <u>varicella-zoster virus</u>. <p>Drug-related:</p> <p>Up to 10% AUR episodes are thought to be attributable to drugs. Those known to increase risk include:</p> <ul style="list-style-type: none"> Anticholinergics (eg, antipsychotic drugs, antidepressant agents, anticholinergic respiratory agents). Opioids and anaesthetics. Alpha-adrenoceptor agonists. Benzodiazepines. NSAIDs Detrusor relaxants. Calcium-channel blockers Antihistamines. Alcohol. <p>Neurological:</p> <p>More often causing chronic retention but may cause AUR:</p> <ul style="list-style-type: none"> Autonomic or peripheral nerve (eg, <u>autonomic neuropathy</u>, <u>diabetes mellitus</u>, Guillain-Barré syndrome, <u>pernicious anaemia</u>, <u>poliomyelitis</u>, radical pelvic surgery, spinal cord trauma, <u>tabes dorsalis</u>). Brain (eg, cardiovascular disease (CVD), MS, neoplasm, <u>normal pressure hydrocephalus</u>, <u>Parkinson's disease</u>). Spinal cord (eg, intervertebral disc disease, <u>meningomyelocele</u>, MS, <u>spina bifida</u> occulta, spinal cord haematoma or abscess, spinal cord trauma, <u>spinal stenosis</u>, spinovascular disease, <u>transverse myelitis</u>, tumours, cauda equina). <p>Other:</p>

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	<ul style="list-style-type: none"> • In men - penile trauma, fracture, or laceration. • In women - postpartum complications (increased risk with instrumental delivery, prolonged labour and <u>Caesarean section</u>),^[2] urethral sphincter dysfunction (Fowler's syndrome). • In both - <u>pelvic trauma</u>, iatrogenic, psychogenic. • <p>MANAGEMENT: Foleys catheter first line. If contraindicated or failed refer to urology or try suprapubic catheterization</p> <p>COMPLICATIONS: UTIs, Renal failure, Post retention diureses and hematuria</p>
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Q:	
Clincher(s)	
A	
B	
C	
D	
E	
KEY	
Additional Information	
Reference	
Dr Khalid/Rabia	

Q:	
Clincher(s)	
A	
B	
C	
D	
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KEY	
Additional Information	
Reference	
Dr Khalid/Rabia	

Q:	

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Clincher(s)	
A	
B	
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KEY	
Additional Information	
Reference	
Dr Khalid/Rabia	

Q:	
Clincher(s)	
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Additional Information	
Reference	
Dr Khalid/Rabia	

Q:	
Clincher(s)	
A	
B	
C	
D	
E	
KEY	
Additional Information	
Reference	
Dr Khalid/Rabia	

Q:1315	A 32yo man presents with 3d of scrotal pain. Exam: thickening of the left
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	testis and it is hot to touch. What is the most appropriate management? a. Analgesia b. Reassurance c. Antibiotics d. Referral to surgeon
Clincher(s)	Scrotal pain and hot to touch
A	
B	
C	
D	
E	
KEY	C
Additional Information	
Reference	
Dr Khalid/Rabia	<p>Epididymo-orchitis</p> <p>Causes :</p> <p>☐ In men under 35 years old, infection is most often due to a sexually transmitted pathogen - eg, Chlamydia trachomatis and Neisseria gonorrhoeae.</p> <p>☐ In men over 35 years old, infection is most often due to a non-sexually transmitted Gram-negative enteric organism causing urinary tract infections - eg, Escherichia coli, Pseudomonas spp. Specific risk factors include recent instrumentation or catheterisation.</p> <p>☐ Signs n symptoms :</p> <p>☐ It usually presents with unilateral scrotal pain and swelling of relatively acute onset.</p> <p>☐ Acute epididymitis is usually unilateral but is bilateral in 5-10% of the patients.</p> <p>☐ Tenderness to palpation on the affected side.</p> <p>☐ Palpable swelling of the epididymis, starting with the tail at the lower pole of the testis and spreading towards the head at the upper pole of the testis with or without involvement of the testicle.</p> <p>☐ There may also be urethral discharge, secondary hydrocele, erythema and/or oedema of the scrotum on the affected side and pyrexia.</p> <p>Clincher here for making the diagnosis is the unilateral tenderness and the hot temperature.</p> <p>Differential would be testicular torsion , but that normally occurs in men who are younger than 20 years though it can occur at any age.</p> <p>A painful swollen testicle in an adolescent boy or a young man should be managed as torsion until proven otherwise</p>

Q:1317	A 32yo man develops hematuria 2wks after a sore throat. What is the dx?
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	a. Post infection nephritis b. IgA nephropathy c. Membranous nephritis d. Glomerulonephritis
Clincher(s)	Hematuria 2 wks after sore throat
A	Present with nephritic picture after strepto infection.
B	IgA nephropathy is common in young kids and in 20% of adults. In Adults it's usually assoc with hx of NSAIDS or Hodgkin lymphoma.
C	Present with picture of nephrotic syndrome
D	Broader term for all glomerular disease.
E	XX
KEY	A
Additional Information	
Reference	
Dr Khalid/Rabia	In, igA nephropathy, symptoms appear 1-3 days after the infection, whereas in Post infection, symptoms appear 1-12 weeks after the sore throat. So, our answer here should be A.

Q:1318	. An elderly man who has anorexia, prostate symptoms and HTN. There are small kidneys on US. What is the dx? a. Hypertensive renal disease b. Prostate ca c. BPH
Clincher(s)	
A	
B	
C	Causes hydronephrosis
D	
E	
KEY	
Additional Information	<u>In Chronic kidney disease the kidneys are usually small, but can be enlarged in infiltrators disorders(amyloid, myeloma),APKD, DM.</u>
Reference	
Dr Khalid/Rabia	Only Hypertensive nephropathy leads to shrunken kidneys. Prostate symptoms are not explained by Hypertension nephropathy unless there is an outflow obstruction and the other two options can be the cause. Should go with answer A. Needs to be discussed though. NOTE : Kidney size is increased in the case of diabetic nephropathy.

Q:1345	<p>A 2yo girl has frequency, urgency and burning micturition. She has some supra pubic tenderness. Which one of the following is the most appropriate initial inv?</p> <p>a. Supra pubic aspiration of urine for C&S b. Clean catch of urine for C&S c. USG d. IVU e. MCUG</p>
Clincher(s)	
A	
B	
C	
D	
E	A micturating/voiding cystourethrogram (MCUG/VCUG) produces X-ray images of the bladder and urinary tract.
KEY	B
Additional Information	
Reference	
Dr Khalid/Rabia	<p>Urinary tract infections (UTI) are more common in boys until 3 months of age (due to more congenital abnormalities) after which the incidence is substantially higher in girls. At least 8% of girls and 2% of boys will have a UTI in childhood</p> <p>Presentation in childhood depends on age:</p> <ul style="list-style-type: none"> ☐ infants: poor feeding, vomiting, irritability ☐ younger children: abdominal pain, fever, dysuria ☐ older children: dysuria, frequency, haematuria ☐ features which may suggest an upper UTI include: temperature > 38°C, loin pain/tenderness <p>NICE guidelines for checking urine sample in a child</p> <ul style="list-style-type: none"> ☐ if there are any symptoms or signs suggestive of a UTI ☐ with unexplained fever of 38°C or higher (test urine after 24 hours at the latest) ☐ with an alternative site of infection but who remain unwell (consider urine test after 24 hours at the latest) <p>Urine collection method</p>

	<p>☐ clean catch is preferable</p> <p>☐ if not possible then urine collection pads should be used</p> <p>☐ cotton wool balls, gauze and sanitary towels are not suitable</p> <p>☐ invasive methods such as suprapubic aspiration should only be used if non-invasive methods are not possible</p> <p>Management:</p> <p>☐ infants less than 3 months old should be referred immediately to a paediatrician</p> <p>☐ children aged more than 3 months old with an upper UTI should be considered for admission to hospital. If not admitted oral antibiotics such as cephalosporin or co-amoxiclav should be given for 7-10 days</p> <p>☐ children aged more than 3 months old with a lower UTI should be treated with oral antibiotics for 3 days according to local guidelines, usually trimethoprim, nitrofurantoin, cephalosporin or amoxicillin. Parents should be asked to bring the children back if they remain unwell after 24-48 hours</p> <p>☐ antibiotic prophylaxis is not given after the first UTI but should be considered with recurrent UTIs</p>
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Q:1347	<p>A young boy presented to the OPD 12wks after renal transplantation with fever and pain in lower abdomen. Renal functions were deranged. Renal biopsy showed immune cell infiltrate and tubular damage. What is the most probable dx?</p> <p>a. Pyelonephritis</p> <p>b. Chronic graft rejection</p> <p>c. Acute rejection</p> <p>d. Drug toxicity</p> <p>e. Graft vs host disease</p>
Clincher(s)	
A	
B	
C	
D	
E	
KEY	C
Additional Information	
Reference	
Dr Khalid/Rabia	<p>Hyperacute rejection : Within minutes of transplant <24 hrs</p> <p>Acute : After one week upto 6 months</p> <p>Chronic : After years due to fibrosis</p>

NEPHROLOGY MERGED UNITY

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Q:	
Clincher(s)	
A	
B	
C	
D	
E	
KEY	
Additional Information	
Reference	
Dr Khalid/Rabia	

Q:	
Clincher(s)	
A	
B	
C	
D	
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KEY	
Additional Information	
Reference	
Dr Khalid/Rabia	

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Q:	
Clincher(s)	
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KEY	
Additional Information	
Reference	
Dr Khalid/Rabia	

Q:	
Clincher(s)	
A	
B	
C	
D	
E	
KEY	
Additional Information	
Reference	
Dr Khalid/Rabia	

Q: 312	<p>A 58yo lady presented with urinary incontinence. She looks anxious for her condition. Urine culture is sterile. Her urodynamic study is normal. What is the next step?</p> <ul style="list-style-type: none"> a. Antibiotics b. Topical estrogen c. Systemic estrogen d. Duloxetine e. Pelvic floor exercise
Clincher(s)	
A	No sign of infection

B	No indication
C	No indication
D	Not first indication
E	Conservative management is preferred before medication
KEY	E
Additional Information	
Reference	
Dr Khalid/Rabia	

Q: 317	A 44yo man presents with periorbital and pedal edema. 24h urine shows 8g of protein/d and serum cholesterol=7mmol/L. Renal biopsy results are awaited. What would be the most likely dx? <ul style="list-style-type: none"> a. Minimal change disease b. Glomerulonephropathy c. Membranous glomerulonephropathy d. FSGS e. IgA nephropathy f. Mesangiocapillary
	Hypertension in FSGN
Clincher(s)	
A	
B	
C	Some people may present as nephrotic syndrome with proteinuria , edema with or without renal failure . Others may be asymptomatic and may be picked up on screening or urinalysis as having proteinuria . A definitive diagnosis of membranous nephropathy requires a kidney biopsy .
D	In children and some adults, FSGS presents as a nephrotic syndrome , which is characterized by edema (associated with weight gain), hypoalbuminemia (low serum albumin , a protein in the blood), hyperlipidemia and hypertension (high blood pressure). In adults it may also present as kidney failure and proteinuria , without a full-blown nephrotic syndrome .
E	
KEY	
Additional Information	
Reference	wiki
Dr Khalid/Rabia	

Q: 329	A man has a BP of 160/90mmHg, proteinuria++. KUB US are equally reduced in size with smooth borders and normal pelvic calyceal system. What is the cause of HTN in the pt?
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	<ul style="list-style-type: none"> a. Chronic glomerulonephritis b. Chronic pyelonephritis c. Bilateral renal artery stenosis d. Essential HTN e. Polycystic kidney
Clincher(s)	
A	
B	
C	
D	
E	
KEY	C
Additional Information	<p>Renovascular disease (Fig 1) Defined as stenosis of the renal artery or one of its branches. Causes: Atherosclerosis (in 80%: >50yrs, arteriopathy: often co-existent IHD, stroke or peripheral vascular disease), fibromuscular dysplasia (10%, younger). Rarer: Takayasu's arteritis, antiphospholipid syndrome, post-renal transplant, thromboembolism, external compression. Signs: □ BP resistant to treatment; worsening renal function after ACE-i/ARB in bilateral renal artery stenosis; 'flash' pulmonary oedema (sudden onset, without LV impairment on cardiac echo). Abdominal carotid or femoral bruits, and weak leg pulses may be found. Tests: USS: renal size asymmetry (affected side is smaller), disturbance in renal blood flow on Doppler US. CT/MR angiography are more sensitive. Renal angiography is 'gold standard', but do after CT/MR as it is invasive (p759). · : Comprehensive antihypertensive regimens (p134), transluminal angioplasty □} stent placement or revascularization surgery. Best medical management is probably superior to revascularization in atherosclerotic disease, see the ASTRAL trial.</p>
Reference	ohcm
Dr Khalid/Rabia	

Q:338	<p>A 40yo male with pre-existing glomerulonephritis having proteinuria and hematuria suddenly deteriorates and presents with oliguria and serum K+=7.8mmol/L, urea=13mmol/L, creat=342mmol/L, GFR=19mL/h. The best management would be?</p> <ul style="list-style-type: none"> a. Calcium supplement b. Calcium resonate enema 30g c. 10units insulin with 50% dextrose d. Nebulized salbutamol e. 10ml of 10% calcium gluconate f. Hemodialysis urgent
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Clincher(s)															
A															
B															
C															
D															
E															
KEY	E														
Additional Information	<table border="1"> <thead> <tr> <th>CKD stage</th><th>GFR range</th></tr> </thead> <tbody> <tr> <td>1</td><td>Greater than 90 ml/min, with some sign of kidney damage on other tests (if all the kidney tests* are normal, there is no CKD)</td></tr> <tr> <td>2</td><td>60-90 ml/min with some sign of kidney damage (if kidney tests* are normal, there is no CKD)</td></tr> <tr> <td>3a</td><td>45-59 ml/min, a moderate reduction in kidney function</td></tr> <tr> <td>3b</td><td>30-44 ml/min, a moderate reduction in kidney function</td></tr> <tr> <td>4</td><td>15-29 ml/min, a severe reduction in kidney function</td></tr> <tr> <td>5</td><td>Less than 15 ml/min, established kidney failure - dialysis or a kidney transplant may be needed</td></tr> </tbody> </table>	CKD stage	GFR range	1	Greater than 90 ml/min, with some sign of kidney damage on other tests (if all the kidney tests* are normal, there is no CKD)	2	60-90 ml/min with some sign of kidney damage (if kidney tests* are normal, there is no CKD)	3a	45-59 ml/min, a moderate reduction in kidney function	3b	30-44 ml/min, a moderate reduction in kidney function	4	15-29 ml/min, a severe reduction in kidney function	5	Less than 15 ml/min, established kidney failure - dialysis or a kidney transplant may be needed
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Reference															
Dr Khalid/Rabia															

Q: 887	<p>A 60yo man presents with severe colicky pain from his right flank radiating to his groin. His urinalysis reveals trace blood cells. What is the single most discriminatory inv?</p> <ol style="list-style-type: none"> US abdomen XR KUB Colonoscopy Upper GI endoscopy Laproscopy
Clincher(s)	Clincher : severe colicky pain, flank pain, traces blood cells in urinalysis
A	
B	

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C	
D	
E	
KEY	a ... corrected : b (if CT KUB is given then choose that for choice investigation for stones)
Additional Information	
Reference	
Dr Khalid/Rabia	Although xray KUB does not show uric acid stones. Around 80% of renal stones are calcium and are seen on xrays. Plus, in this patient, pain is radiating from loin to groin as in uretric stones which are not visualised by US. Colicky pain, flank pain and blood cells in urine point towards renal pathology. When a renal pathology is suspected, US KUB is done, not US abdomen. Colonoscopy, upper GI endoscopy and laproscopy have different indications.

Q: 931	931. A pt had passed a 4mm stone in his urine. He has a 3mm stone in the renal pelvis found on US. What is the management? a. ESWL b. None c. Dormier basket d. Surgery e. PCNL
Clincher(s)	
A	
B	
C	
D	
E	
KEY	B. none
Additional Information	
Reference	
Dr Khalid/Rabia	If the stone is <5mm in lower ureter ,it will pass spontaneously.. if it is >5mm ---> medical therapy (nifedipine ,alpha blocker) if not passed , go for ESWL (If < 1cm) or dormia basket.. PCNL ---> when stone is large ,multiple or complex.. Percutaneous nephrostomy ---> presence or infection or obstruction , to safe the kidney from reflux damage and save the person from Sepsis (if pus collected)

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Q: 941	<p>941. A 75yo man comes in complaining of difficulty in passing urine, poor stream and dribbling at the end of voiding and anorexia. US shows bilateral hydronephrosis. What is the cause of these findings?</p> <p>a. BPH b. Renal stones c. Bladder stones d. Prostatic ca e. UTI</p>
Clincher(s)	
A	
B	
C	
D	
E	
KEY	d. Prostatic ca
Additional Information	
Reference	
Dr Khalid/Rabia	<p>bph is common and BOO signs are present with it earlier but here the anorexia is given which points to carcinoma</p> <ul style="list-style-type: none"> • bladder outlet obstruction: hesitancy, urinary retention • haematuria, haemospermia • pain: back, perineal or testicular • digital rectal examination: asymmetrical, hard, nodular enlargement with loss of median sulcus

Q: 943	<p>A 50yo man presents with the complaints of recurrent UTI and occasional blood in the urine. Some unusual cells have been seen in urine on routine exam. Which of the following inv would you like to carry out now?</p> <p>a. Cystoscopy b. Urine C&S c. XR KUB d. US e. CBC</p>
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Clincher(s)	
A	
B	
C	
D	
E	
KEY	D. US
Additional Information	<u>If frank blood- do cystoscopy</u>
Reference	
Dr Khalid/Rabia	the unusual cells may indicate malignancy , which in turn can be the cause of recurrent UTI .US followed by cystoscopy seems to be the appropriate approach. unusual cell points towards possible malignancy! may be bladder cancer or renal cell carcinoma. So we have two options, for bladder cystoscopy and for renal cell ca US. Before going to more invasive procedure we can think first noninvasive procedure. So US is more logical i think. for uti we can do c/s but that is not the major issue

Q: 989	989. A 45yo known hypertensive man presents with hematuria, proteinuria and edema. What is the definitive dx test for him? a. Urine protein b. Renal biopsy c. Renal function test d. Urine microscopy e. Serum protein
Clincher(s)	
A	
B	
C	
D	
E	
KEY	B
Additional Information	
Reference	
Dr Khalid/Rabia	Pt seems to be suffering from nephritic syndrome , the definitive diagnosis can be made on renal biopsy.

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Q: 990	<p>A 47yo man presents with proteinuria, BP=160/95 mmHg, small kidneys that have smooth renal pelvis. What is the most probable dx?</p> <p>a. GN</p> <p>b. Chronic pyelonephritis</p> <p>c. Unilateral renal artery stenosis</p> <p>d. Multiple myeloma</p> <p>e. ARF</p>
	<p>Nephrotic>CRF>shrunken kidney</p> <p>DM>leading cause of CRF> not small Q</p>
Clincher(s)	
A	
B	
C	
D	
E	
KEY	A
Additional Information	
Reference	
Dr Khalid/Rabia	<p>GN is a common cause of CKD, presenting with proteinuria, hypertension and <u>small kidneys</u>. (nephritic syndrome)</p>

Q:	
Clincher(s)	
A	
B	
C	
D	
E	
KEY	
Additional Information	
Reference	
Dr Khalid/Rabia	

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Q:	
Clincher(s)	
A	
B	
C	
D	
E	
KEY	
Additional Information	
Reference	
Dr Khalid/Rabia	

Q:	
Clincher(s)	
A	
B	
C	
D	
E	
KEY	
Additional Information	
Reference	
Dr Khalid/Rabia	

Q:	
Clincher(s)	
A	
B	
C	
D	
E	
KEY	
Additional Information	
Reference	
Dr Khalid/Rabia	

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Q: 706	<p>A 75yo man with adenocarcinoma of the prostate which has spread outside the capsule of the gland has ARF. What is the most appropriate next inv?</p> <p>a. MRI spine</p> <p>b. Radionuclide bone scan</p> <p>c. Trans rectal US</p> <p>d. US pelvis</p> <p>e. US KUB</p>
Clincher(s)	daignosed case of adenocarcinoma prostate and acute renal failure.
A	MRI done to rule out bone metastasis, which is not the case here.
B	Also done in bone metastasis(no history of bone pain).
C	to rule out local invasion of prostate.
D	confined to pelvis.
E	Most appropriate for evaluation of degree of injury to kidney and ureter due to reflex uropathy. Prostate carcinoma cause obstructive nephropathy(hydronephrosis and dilated ureter).
KEY	E- USG KUB

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Additional Information	<p>Prostate cancer The commonest male malignancy. Incidence: ↑with age: 80% in men >80yrs (autopsy studies). Associations: +ve family history (x2-3 ↑risk, p524), ↑testosterone. Most are adenocarcinomas arising in peripheral prostate. Spread may be local (seminal vesicles, bladder, rectum) via lymph, or haematogenously (sclerotic bony lesions). Symptoms: Asymptomatic or nocturia, hesitancy, poor stream, terminal dribbling, or obstruction. Weight↓ ± bone pain suggests mets. DRE exam of prostate: May show hard, irregular prostate. Diagnosis: ↑PSA (normal in 30% of small cancers); transrectal USS & biopsy; X-rays; bone scan; CT/MRI. Staging: MRI. If contrast-enhancing magnetic nanoparticles used, sensitivity for detecting affected nodes rises from 35% to 90%.²⁵⁰ Treatment: Disease confined to prostate:²⁵¹ Options depend on prognosis (BOX), patient preference and comorbidities. •Radical prostatectomy if <70yrs gives excellent disease-free survival (laparoscopic surgery is as good). The role of adjuvant hormonal therapy is being explored. •Radical radiotherapy (± neoadjuvant & adjuvant hormonal therapy) is an alternative curative option that compares favourably with surgery (no RCTs). It may be delivered as external beam or brachytherapy. •Hormone therapy alone temporarily delays tumour progression but refractory disease eventually develops. Consider in elderly unfit patients with high-risk disease. •Active surveillance—particularly if >70yrs and low-risk. Metastatic disease: •Hormonal drugs may give benefit for 1-2yrs. LHRH agonists, eg 12-weekly goserelin (10.8mg SC as Zoladex LA®) first stimulate, then inhibit pituitary gonadotrophin. NB: risks tumour 'flare' when first used—start anti-androgen, eg cyproterone acetate, in susceptible patients. The LHRH antagonist degarelix is also used in advanced disease. Symptomatic Rx: Analgesia; treat hypercalcaemia; radiotherapy for bone mets/spinal cord compression. Prognosis: 10% die in 6 months, 10% live >10yrs. Screening: DRE of prostate; transrectal USS; PSA (see BOX).</p>
Reference	OHCM
Dr Khalid/Rabia	

Q:711	<p>A 32yo miner is rescued after being trapped under a fallen rock for 4h. After applying a bladder catheter, 15-20ml of reddish brown urine was obtained. HR=120bpm, SBP=100mmHg. What would be the next appropriate step?</p> <ol style="list-style-type: none"> Dopamine IV Fluid challenge Furosemide IV 20% Mannitol IV Antibiotics
Clincher(s)	H/o trauma and red urine.
A	dopamine is given in falling blood pressure due to active haemorrhage

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B	Fluid challenge is given in acute renal injury due to penal causes like trauma(rhabdomolysis)
C	fruosemide is advised for pulmonary oedema due to renal failure.
D	manitol for reducing cerebral oedema.
E	not relevant here.
KEY	B- fluid challenge.

Additional Information	<div data-bbox="523 232 1513 286" data-label="Section-Header"> <h2>292 Acute kidney injury (AKI): management</h2> </div> <div data-bbox="611 297 823 327" data-label="Section-Header"> <h3>General measures</h3> </div> <div data-bbox="611 327 1513 969" data-label="List-Group"> <ul style="list-style-type: none"> • Assess volume status: look for ↓urine volume, non-visible JVP, poor tissue turgor, ↓BP, ↑pulse. Signs of fluid overload: ↑BP, ↑JVP, lung crepitations, peripheral oedema, gallop rhythm on cardiac auscultation. • Aim for euvolaemia:⁴⁷ if difficult balance with risk of fluid overload, consider titrating input hourly, by matching previous hours output + 25mL/h for insensible losses. This is not usually practical outside of an HDU setting as it requires intensive nursing input. If euvolaemic review balance daily, over 24-hour period aim to match input to loss (urine, vomit, diarrhoea, drains) + 500mL for insensible loss (more if T⁺↑). <ul style="list-style-type: none"> ▶ Avoid K⁺-containing fluids unless hypokalaemic. • Stop nephrotoxic drugs: (See 'sick day rules' BOX) eg NSAIDs, ACE inhibitor, gentamicin, amphotericin. Stop metformin if creatinine is >150mmol/L, see p200. Check and adjust doses of renally excreted drugs (ask your pharmacist for the <i>Renal Drug Handbook</i>, a weighty but valuable tome!). • Monitoring: Consider transfer to intensive care or high-dependency unit (ICU/HDU). <ul style="list-style-type: none"> • Check pulse, BP, JVP, and urine output hourly (insert a urinary catheter). Consider inserting a CVP line if on HDU/ICU. • Daily U&Es. • Daily fluid balance chart and daily weight. • Nutrition is vital in the critically unwell patient: aim for normal calorie intake (more if catabolism ↑↑, eg burns, sepsis) and protein ~0.5g/kg/d. If oral intake is poor, consider nasogastric nutrition early (parenteral if NGT impossible, p588). </div> <div data-bbox="611 976 882 1005" data-label="Section-Header"> <h3>Treat underlying cause</h3> </div> <div data-bbox="611 1005 1513 1314" data-label="List-Group"> <ul style="list-style-type: none"> • Pre-renal: Correct volume depletion with appropriate fluids, treat sepsis with antibiotics, consider referral to ICU for inotropic support if signs of shock (see p804). • Post-renal: Catheterize and consider CT of renal tract (CTKUB) and urology referral if obstruction likely cause. If signs of obstruction and hydronephrosis on CT/uss then discuss with urology regarding cystoscopy and retrograde stents or nephrostomy insertion; this buys time to allow treatment of cause of obstruction, eg stone, mass. • Intrinsic renal: ▶ Refer early to nephrology if concern over tubulointerstitial or glomerular pathology, any signs of systemic disease, multi-organ involvement (eg pulmonary-renal, hepatorenal syndromes) or indications for dialysis (see p291). </div> <div data-bbox="611 1321 879 1352" data-label="Section-Header"> <h3>Manage complications</h3> </div> <div data-bbox="611 1350 1513 1536" data-label="List-Group"> <ul style="list-style-type: none"> • Hyperkalaemia (see BOX p293). • Pulmonary oedema (see BOX p293). • Uraemia—may require dialysis if severe or complications, eg encephalopathy, pericarditis, contact renal team. Otherwise symptomatic management. • Acidaemia—may require dialysis, consider sodium bicarbonate orally or IV if in HDU/ICU setting, discuss with nephrology before initiating. </div> <div data-bbox="611 1543 935 1576" data-label="Section-Header"> <h3>Renal replacement therapy</h3> </div> <div data-bbox="611 1574 1513 1729" data-label="Text"> <p>Options in AKI include haemodialysis and haemofiltration, both require large-bore venous access (eg internal jugular line). Haemodialysis is usually done intermittently and allows good clearance of solutes in short periods but requires the patient to be haemodynamically stable. If patients are stable enough for ward-based care this is usually the modality of choice. Patients on the ICU are often</p> </div>
Reference	OHCM pg 292.
Dr Khalid/Rabia	

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Q: 716	<p>A 42yo woman with a PMH (past medical hx) of severe headache treated in the ED presents with signs and symptoms of renal failure. She has been seen by her GP for HTN and abdominal pain with OP inv pending. Which inv is most likely to lead to a dx?</p> <p>a. US KUB b. CT brain c. IVU d. Renal artery Doppler e. Renal biopsy</p>
Clincher(s)	H/o severe headache, HTN and abdominal pain- all suggestive of Polycystic kidney disease.
A	Best investigation is Ultrasound KUB.
B	
C	
D	
E	
KEY	
Additional Information	<p>Autosomal dominant polycystic kidney disease (APKD)</p> <p>Prevalence: 1:1000. 85% of patients have mutations in PKD1 (chromosome 16) and reach ESRF by 50s. Remainder have mutation in PKD2 (chromosome 4) and have a slower course, reaching ESRF by 70s. Can be clinically silent for many years so family screening is important. Signs: Renal enlargement with cysts, abdominal pain ± haematuria (haemorrhage into a cyst), cyst infection, renal calculi, BPT, progressive renal failure. Extra-renal: liver cysts, intra-cranial aneurysm→SAH (subarachnoid haemorrhage, see p482), mitral valve prolapse, ovarian cysts and diverticular disease. Rx: Monitor U&E. ↑BP should be treated aggressively, with target levels of <130/80mmHg⁵⁰ (ACE-i are best choices). Treat infections, dialysis or transplantation for ESRF, genetic counselling. Pain may be helped by laparoscopic cyst removal or nephrectomy. ↑Water intake, ↓Na⁺ intake, and avoiding caffeine may also help.⁵¹ Screening for PKD: Genetic testing for PKD1 is difficult as the gene is large and there are hundreds of described mutations. USS screening offers good sensitivity and specificity depending on age. Age 18-39yrs >3 unilateral or bilateral cysts, 40-59yrs >2 cysts in each kidney, >60yrs >4 cysts in each kidney have good sensitivity and specificity and a positive predictive value close to 100%.⁵² Genetic screening for some PKD2 mutations is available in specialist centres. Screening for SAH with magnetic resonance angiography may be done in 1st-degree relatives of those with SAH + ADPKD. Some screen with no family history, especially for certain occupations (eg pilots).</p>
Reference	OHCM pg 312
Dr Khalid/Rabia	

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Q: 781	A 50yo newly dx with HTN complains of urinary freq and dysuria. The urinalysis reveals presence of white cells and protein. Choose the single most appropriate tx? a. Imipramine b. Adjust diuretics c. Vaginal estrogen d. Trimethoprim
Clincher(s)	UTI
A	
B	
C	
D	First line antibiotic- Trimethoprim.
E	
KEY	D- Trimethoprim.

Additional Information	<div data-bbox="555 271 1554 1317" style="background-color: #e6f2ff; padding: 10px; border: 1px solid #003366;"> <p>Managing UTI¹¹</p> <p>▶ Drink plenty of fluids; urinate often (don't 'hold on').</p> <p>Management of bacterial UTI in adult non-pregnant women</p> <ul style="list-style-type: none"> • Consider empirical treatment for presumed <i>E. coli</i> in otherwise healthy women who present with lower UTI: local sensitivities vary but consider trimethoprim 200mg/12h PO or nitrofurantoin, eg 50mg/6h PO for 3-6d (if normal renal function), amoxicillin 500mg/8h PO. Alternative: cefalexin 1g/12h (if eGFR >40). 2nd line: co-amoxiclav PO (7d course). The latter 2 may cause problems with <i>C. difficile</i>. If there is no response, do a urine culture. • In case of vaginal itch or discharge consider vaginal examination and swabs for other diagnoses, eg thrush, chlamydia, other STIs. • In non-pregnant women with upper UTI, take a urine culture and treat with, eg co-amoxiclav 1.2g/8h IV, then oral when afebrile, complete 7d course. Avoid nitrofurantoin as it does not achieve effective concentrations in urine. Resistance to trimethoprim is common. Non-pregnant women with asymptomatic bacteriuria do not need antibiotics so screening is not recommended. <p>Managing bacterial UTI in pregnant women Get expert help. Any bacteriuria (symptomatic or asymptomatic) should be treated with an antibiotic. Dipstick and urine culture should be repeated at each antenatal visit.</p> <p>Managing bacterial UTI in adult men</p> <ul style="list-style-type: none"> • Take UTI in men seriously as it often results from an anatomical or functional anomaly. Therefore, all men with symptoms of upper UTI, recurrent UTI or who fail to respond to antibiotic therapy should be referred to a urologist. Also consider prostatitis, epididymitis and chlamydial infection. • Bacterial UTI in men may need a 2wk course of a quinolone, such as levofloxacin (if no response, think of prostatitis and treat for 4wks). • Antibiotic therapy of asymptomatic bacteriuria in elderly men (>65yrs old) is not recommended, as it does not reduce morbidity or mortality while it increases the risk of adverse events such as rashes or GI problems. </div>
Reference	OHCM pg 289
Dr Khalid/Rabia	

Q:794	<p>A young boy presented with bilateral periorbital edema, ankle swelling and increase in body weight. What is the most likely dx?</p> <ol style="list-style-type: none"> Chronic heart failure Nephrotic syndrome Renal failure Acute heart failure Glomerulonephritis

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Clincher(s)	Young age, signs of renal failure.
A	
B	Most common cause is nephrotic syndrome due to minimal change disease.
C	
D	
E	
KEY	
Additional Information	B
Reference	
Dr Khalid/Rabia	

Q:865	An 80yo man has a permanent catheter. Catheter specimen urine found lots of e-coli. What is the single most appropriate management as he wants to attend his daughter's wedding next week? a. Change the catheter b. Prolonged antibiotics c. Bladder wash d. Repeat MSU after wedding e. Reassure
	E coli=uTI
Clincher(s)	permenant catheter and Ecoli on finding.
A	change catheter for prevention of sepsis due to UTI.
B	
C	
D	
E	
KEY	A- catheter replacement.

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Additional Information	
Reference	
Dr Khalid/Rabia	

Q:1360	<p>A 40yo man complains of severe colicky loin pain that radiates to his scrotum. He is noted to have microscopic hematuria. No masses are palpated. What is the single most likely cause?</p> <p>a. Acute cystitis b. Bladder ca c. Renal vein thrombosis d. Acute pyelonephritis e. Ureteric calculus</p>
	Most common stone= oxalate
Clincher(s)	severe colicky loin pain and no masses on palpation.
A	
B	
C	
D	
E	Ureteric calculus.
KEY	E- ureteric calculus.

Additional
Information**640 Urinary tract calculi (nephrolithiasis)**

Surgery

Renal stones (calculi) consist of crystal aggregates. Stones form in collecting ducts and may be deposited anywhere from the renal pelvis to the urethra, though classically at **1** Pelviureteric junction **2** Pelvic brim **3** Vesicoureteric junction.

Prevalence Common: lifetime incidence up to 15%. **Peak age:** 20–40yr. ♂:♀≈3:1.

Types •Calcium oxalate (75%) •Magnesium ammonium phosphate (struvite/triple phosphate; 15%) •Also: urate (5%), hydroxyapatite (5%), brushite, cystine (1%), mixed.

Presentation Asymptomatic or: **1 Renal colic:** excruciating ureteric spasms 'loin to groin' (or genitals/inner thigh), with nausea/vomiting. Often cannot lie still (differentiates from peritonitis). **Renal obstruction** felt in the loin, between rib 12 and lateral edge of lumbar muscles (like intercostal nerve irritation pain; the latter is not colicky, and is worsened by specific movements/pressure on a trigger spot). **Obstruction of mid-ureter** may mimic appendicitis/diverticulitis. **Obstruction of lower ureter** may lead to symptoms of bladder irritability and pain in scrotum, penile tip, or labia majora.²²³ **Obstruction in bladder or urethra** causes pelvic pain, dysuria, strangury (desire but inability to void²²⁴) ± interrupted flow. **2 UTI** can co-exist (trisk if voiding impaired); **pyelonephritis** (fever, rigors, loin pain, nausea, vomiting), **pyonephrosis** (infected hydronephrosis) **3** Haematuria **4** Proteinuria **5** Sterile pyuria **6** Anuria.

Examination Usually no tenderness on palpation. May be renal angle tenderness especially to percussion if there is retroperitoneal inflammation.

Tests FBC, U&E, Ca^{2+} , PO_4^{3-} , glucose, bicarbonate, urate. **Urine dipstick:** Usually +ve for blood (90%). **MSU:** MC&S. **Further tests for cause:** Urine pH; 24h urine for: calcium, oxalate, urate, citrate, sodium, creatinine; stone biochemistry (sieve urine & send stone).

Imaging: Spiral non-contrast CT is superior to and has largely replaced IIVU for imaging stones (99% visible) + helps exclude differential causes of an acute abdomen. ▶A ruptured abdominal aortic aneurysm may present similarly. 80% of stones are visible on KUB XR (kidneys+ureters+bladder). Look along ureters for calcification over the transverse processes of the vertebral bodies. IIVU: radio-opaque contrast injected and serial films taken until contrast seen down to level of obstruction. Cannot be interpreted without a plain control. Abnormal findings: failure of contrast to flow to bladder, dense nephrogram (contrast unable to flow from kidney), clubbed/blunted renal calyces (back pressure), filling defects in the bladder. (CI: contrast allergy, severe asthma, pregnancy, metformin.) USS to look for hydronephrosis or hydroureter.

R: Initially: Analgesia, eg **diclofenac** 75mg IV/IM, or 100mg PR.²²⁵ (If CI: opioids) + IV fluids if unable to tolerate PO; antibiotics (eg **cefuroxime** 1.5g/8h IV, or **gentamicin**) if infection. **Stones <5mm in lower ureter:** ~90–95% pass spontaneously. ↑fluid intake. **Stones >5mm/pain not resolving:** Medical expulsive therapy: **nifedipine** 10mg/8h PO²²⁶ or α-blockers (**tamsulosin** 0.4mg/d²²⁷) promote expulsion and reduce analgesia requirements:²²⁸ ▶start at presentation.²²⁹ Most pass within 48h (>80% after ~30d). If not, try extracorporeal shockwave lithotripsy (ESWL) (if <1cm), or ureteroscopy using a basket.²³⁰ ESWL: US waves shatter stone. SE: renal injury, may also cause ↑BP and DM.²³¹ Percutaneous nephrolithotomy (PCNL): keyhole surgery to remove stones, when large, multiple, or complex.²³² Open surgery is rare.

▶**Indications for urgent intervention** (delay kills glomeruli): Presence of infection **and** obstruction—a percutaneous nephrostomy or ureteric stent may be needed to relieve obstruction (p642); urosepsis; intractable pain or vomiting; impending ARF; obstruction in a solitary kidney; bilateral obstructing stones.²³²



Reference

OHCM pg 640

Dr Khalid/Rabia

February 17, 2016

Q:1415	<p>A pt is on loop diuretics. What effect do loop diuretics produce?</p> <p>a. Low Na⁺, low K⁺</p> <p>b. Low Na⁺, normal K⁺</p> <p>c. Normal Na⁺, normal K⁺</p> <p>d. High Na⁺, low K⁺</p> <p>e. High Na⁺, high K⁺</p>
Clincher(s)	mechanism of loop diuretics.
A	
B	
C	
D	
E	
KEY	A- low sodium and low potassium. (Good description below)

Additional
Information**304 Diuretics and their mechanism of action¹**

Renal medicine

Loop diuretics, eg furosemide, bumetanide

Mechanism: Block the $\text{Na}^+/\text{K}^+/\text{2Cl}^-$ co-transporter in the thick ascending limb of the Loop of Henle, hence increase the solute load of the filtrate and reduce water resorption. **Effects:** Significant loss of water and Na^+Cl^- . Ca^{2+} , K^+ and H^+ excretion is also increased. **Clinical use:** They are readily absorbed from the GI tract, and given orally they act within 1 hour. IV the peak effect is seen within 30 minutes so they are useful in treating acute pulmonary oedema. Widely used in peripheral oedema (heart failure, ascites). They can also be used to treat severe hypercalcaemia. **Side effects:** Hypokalaemic metabolic alkalosis (because of K^+ and H^+ excretion), hypovolaemia, ototoxicity, allergic reactions.

Thiazide diuretics, eg bendroflumethiazide, indapamide, metolazone

Mechanism: They inhibit the Na^+Cl^- transporter in the distal convoluted tubule. They also decrease Na^+Cl^- resorption and hence increase water loss. **Effects:** Moderate Na^+ and Cl^- excretion. They result in low urine Ca^{2+} by increasing urinary Ca^{2+} resorption (the opposite effect of loop diuretics). They can cause profound hypokalaemia due to excessive K^+ loss. Excretion of uric acid is reduced, and Mg^{2+} is increased. **Use:** Their action begins within 1-2 hours after ingestion, and lasts for 12-24 hours. Useful in TBP, long-term treatment of oedema (eg heart failure). They can reduce renal stone formation in patients with idiopathic hypercalciuria by decreasing urine calcium concentrations. **Side effects:** Hypokalaemia, hyponatraemia, hypomagnesaemia, metabolic alkalosis, hyperglycaemia, increased serum lipid and uric acid levels (thiazide diuretics are contraindicated in gout).

Potassium-sparing diuretics

Mechanism: Spironolactone and eplerenone are aldosterone antagonists and hence inhibit the sodium-retaining action of aldosterone. Amiloride and triamterene block sodium channels in collecting tubules. Both types decrease K^+ excretion. **Effects:** They increase Na^+ excretion and decrease K^+ and H^+ excretion, there is a mild associated diuresis. The onset of action of spironolactone is very slow, and takes several days to have its full effect. Amiloride has a much faster onset and a duration of action of only 12-16 hours. **Use:** Usually used to control the K^+ wasting caused by loop diuretics or thiazides and are given as combination therapy. Spironolactone and eplerenone have significant benefits in hyperaldosteronism (↑serum aldosterone. Primary = Conn's, rare. Secondary occurs in cirrhosis and heart failure). **Side effects:** Hyperkalaemia is common, metabolic acidosis can occur. Anti-androgenic effects (eg gynaecomastia) are seen with spironolactone, as is GI upset.

Osmotic diuretics Mannitol is a solute that is freely filtered at the glomerulus but poorly reabsorbed from the tubule, so it remains in the lumen and holds water by osmotic effect. The reduction in water reabsorption occurs in the proximal tubule and loop of Henle and also slightly increases Na^+ loss. **Effects:** Mannitol can reduce brain volume and intracranial pressure by osmotically extracting water from the tissue into the blood. **Use:** Haemolysis, rhabdomyolysis, reducing intra-ocular and intra-cranial pressures. **Side-effects:** ↑ Na^+ , headache, nausea, vomiting. In patients who are unable to form urine the transient expansion of the extracellular fluid when water is extracted from intra-cellular fluid can cause pulmonary oedema.

Carbonic anhydrase inhibitors such as acetazolamide act on the proximal tubule to increase excretion of bicarbonate and consequently sodium, potassium and water. This causes alkalinization of the urine and hence a mild metabolic acidosis. They are not used as diuretics but are still available for use in **glaucoma** to reduce the forma-

Reference

OHCM pg 304

Dr Khalid/Rabia

February 17, 2016

Q: 1416	<p>A 6yo girl is being investigated for renal failure. She is found to have a congenital abnormality of the insertion of the ureters into the urinary bladder. What is the single most likely cause for renal failure in this pt?</p> <p>a. SLE b. PKD c. Wilms tumor. d. Acute tubular necrosis e. Reflux nephropathy</p>
Clincher(s)	congenital malformation of urogenital tract,
A	
B	
C	
D	
E	
KEY	E- reflux nephropathy.
Additional Information	
Reference	
Dr Khalid/Rabia	

February 17, 2016

Q:1444	<p>A 60yo man complains of tiredness, lethargy and itching that is severe after a hot bath. He also has nocturia, polyuria and nausea and vomiting. Exam: pallor, pigmentation and generalized edema. What is the single most likely dx?</p> <p>a. Hyperthyroidism</p> <p>b. Lichen planus</p> <p>c. Lymphoma</p> <p>d. Eczema</p> <p>e. Liver failure</p> <p>f. chronic renal failure.</p>
	Polycythemia- in CRF- so itching after hot bath
Clincher(s)	
A	
B	
C	
D	
E	
KEY	F- chronic renal failure.
Additional Information	<div> <div>294</div> <div>Chronic kidney disease (CKD)</div> </div> <p>Definition Impaired renal function for >3 months based on abnormal structure or function, or GFR <60mL/min/1.73m² for >3 months with or without evidence of kidney damage. Classification: 5 stages (Box). Symptoms usually only occur once stage 4 is reached (GFR <30). End-stage renal failure (ESRF) is defined as GFR <15 mL/min/1.73m² or need for renal replacement therapy (RRT—dialysis or transplant).¹⁹</p> <p>Causes</p> <p>1 Diabetes: 20% UK, 43% USA. Type II >> type I</p> <p>2 Glomerulonephritis: commonly IgA nephropathy, also rarer disorders, eg mesangiocapillary GN, systemic disorders, eg SLE, vasculitis</p> <p>3 Unknown: up to 20% in the UK have no obvious cause of CKD, many of these present late with small, shrunken kidneys where a biopsy would be uninformative</p> <p>4 Hypertension or renovascular disease</p> <p>5 Pyelonephritis and reflux nephropathy</p> <p>Rarer causes include obstructive uropathy, which commonly causes AKI but is often reversible, chronic interstitial nephritis (eg myeloma, amyloid), and following previous AKI. Adult polycystic kidney disease (APKD) is the commonest inherited cause of CKD, rare inherited disorders include Alport's syndrome and Fabry's disease (p712).</p>

February 17, 2016

Reference	OHCM pg 294
Dr Khalid/Rabia	
Q: 1445	<p>A 30yo man complains of vague pain in the loin with BP=140/90mmHg. He is found to have proteinuria and hematuria. What is the inv to confirm the dx?</p> <p>a. Abdominal US b. ANCA c. ANA d. Urine microscopy and culture e. Stool culture</p>
Clincher(s)	young patient with vague abdominal pain, proteinuria and hematuria with HTN. always suspect polycystic kidney disease.
A	
B	
C	
D	
E	
KEY	A usg abdomen
Additional Information	
Reference	
Dr Khalid/Rabia	

February 17, 2016