**Question 1**

How long does it usually take for a post streptococcal of the pharynx to develop glomerulonephritis?

A 6 months

B 3 months

C 5 days

D 2 weeks

Explanation D

Post streptococcal glomerulonephritis usually appears 1-4 weeks after a pharyngeal or skin infection (impetigo). PSG occurs most frequently in children aged 6-10yrs, but adults of any age can be affected.

**Question 2**

What is the most common cause of chronic pyelonephritis?

A Pregnancy

B Chronic catheterisation

C Chronic obstructive pyelonephritis

D Chronic vesicoureteral reflux

Explanation D

Chronic pyelonephritis is a disorder in which chronic tubulointerstitial inflammation and scarring are associated with pathological involvement of the calyces and pelvis. Chronic pyelonephritis is an important cause of end stage chronic kidney disease. This condition remains an important cause of kidney destruction in children with severe lower urinary tract abnormalities

Chronic pyelonephritis can be divided into two forms

Chronic reflux nephropathy-more common

Chronic obstructive pyelonephritis

Note: both conditions can result in bilateral pyelonephritis. Vesicoureteral reflux occurs in 1-2% of otherwise normal children

**Question 3**

In the diagnosis of malignant renal hypertension, which of the following statements is correct?

A Occurs in 5-10% of individuals with an elevated blood pressure

B Malignant hypertension only occurs in patients with previous hypertension

C The morphology picture of onion skinning is proportional to the degree of renal failure

D Due to kidney dysfunction, there is decreased levels of renin

Explanation C

Malignant hypertension is the form of renal disease associated with malignant or accelerated phase of hypertension. This pattern of hypertension may occasionally develop in normotensive individuals but is more often super imposed on pre-existing benign hypertension, secondary forms of hypertension or underlying chronic renal disease (glomerulonephritis or reflux nephropathy). It is an uncommon condition occurring in1 to 5% of patients with elevated blood pressure. Occurs more often in younger people and the black population.

The initial insult is some sort of vascular damage to the kidneys. This results in increased vessel permeability, focal death of cell walls and platelet deposition. This leads to fibrinoid necrosis of the arterioles and small arteries, swelling of the intima layer and vascular thrombosis. The kidneys become markedly ischaemic. With severe involvement of the afferent arteriole, the renin angiotensin system is activated. (Increasing the levels of plasmin renin dramatically)

The microscopic changes reflect the pathogenic events of malignant hypertension. There is intimal thickening caused by concentric proliferation of smooth muscle cells and collagen which accumulates in a layered configuration (together with accumulation of proteoglycans and plasma proteins). This gives an onion skinning appearance. This lesion is also called hyperplastic arteriolitis and correlates with renal failure in malignant hypertension

**Question 4**

Regarding post infectious glomerulonephritis which of the following statements is correct?

A Occurs 1- 4 weeks post impetigo

B Is due to the toxic effect of streptolysin on the basement membrane

C Is due to Group B alpha-haemolytic streptococcus

D Most patients will develop renal failure

Explanation A

Acute glomerulonephritis occurs most frequently in children aged 6-10yrs, 1-4 weeks after a streptococcal infection of the pharynx or skin (impetigo). It is due to a group A beta haemolytic streptococcus. The streptococcal antigenic component responsible for the immune reaction has eluded identification for years but most of the evidence suggests streptococcal pyogenic endotoxin B. More than 95% of affected children will recover.

Note: impetigo is a skin infection caused by both Staphylococcus aureus (most commonly), and Streptococcus pyogenes

**Question 5**

Which of the following is correct in relation to nephrotic syndrome?

A Albumin is lost, other globulins are unaffected

B Hypertension Your Answer

C There is alteration to serum lipid levels

D Decreased interstitial fluid volume

Explanation C

The manifestations of nephrotic syndrome include: massive proteinuria, hypoalbuminaemia, generalised oedema (increased interstitial fluid), hyperlipidaemia and lipiduria

**Question 6**

Which of the following is not a nephrotoxic cause of acute tubular necrosis (ATN)?

A Erythromycin

B Radiographic contrast material

C Aminoglycosides

D Lead

Explanation A

Causes of nephrotoxic ATN include:

- Gentamicin

- Radiographic contrast agents

- Heavy metal poisoning

- Organic solvents

**Question 7**

Concerning acute tubular necrosis (ATN), which of the following statements is correct?

A Cephalosporins are not a causative agent

B Nephrotoxic causes are associated with a poor prognosis

C Rhabdomyolysis is not a cause

D ATN is the commonest cause of acute renal failiure

Explanation D

Acute tubular necrosis/injury

Renal failure can be caused by

ATN

Organic vascular obstruction

Sever glomerular disease

Acute tubulointerstitial nephritis, most commonly due to hypersensitivity to drugs

Massive infection, pyelonephritis

DIC

Urinary obstruction

ATN

Ischaemic type-due to decreased or interrupted blood flow

Systemic thrombosis/embolism

DIC

Decreased effective circulating volume-hypovolaemic shock

Microangiopathies of the renal blood vessels

Direct toxic injury to the tubules by

Endogenous agents (myoglobin, Hb, monoclonal light chains)

Exogenous agents (drugs, radiocontrast, heavy metals, solvents, antibiotics-gentamicin)

Other antibiotics are implicated. No examples are given in the textbook.

Web search-there are cases of cephalosporins causing nephrotoxic ATN (cephalexin, cephaloridine and cephalothin)

Nephrotoxin ATN has a better prognosis than ischaemic ATN.

Rhabdomyolysis releases nephrotoxin substances from damaged myocytes (myoglobin) and damaged myocytes sequester extracellular fluid provoking hypovolaemic decrease in renal perfusion.

ATN accounts for about 50% of cases of acute renal failiure (the most commonest cause)

Acute tubulointerstitial nephritis

Drugs — Virtually any drug can cause AIN, although only a few have been reported with any frequency

The most common drug causes of AIN now include

Nonsteroidal anti-inflammatory agents (NSAIDs), including selective cyclooxygenase (COX)-2 inhibitors

Penicillins and cephalosporins

Rifampin

Antimicrobial sulfonamides, including trimethoprim-sulfamethoxazole

Diuretics, including loop diuretics such as furosemide and thiazide-type diuretics

Ciprofloxacin and to a lesser degree, other quinolones

Cimetidine only rare cases have been described with other H-2 blockers such as ranitidine

Allopurinol

Proton pump inhibitors such as omeprazole

Indinavir

5-aminosalicylates eg, mesalamine

**Question 8**

Regarding acute tubular necrosis (ATN), which of the following statements is correct?

A It is associated with hyperkalemia in recovery

B The non-oliguric form has a better recovery

C It is associated with ischaemic cortical cells

D 80% are associated with anuria

Explanation B

ATN is associated with hypokalaemia in the recovery phase. The tubules are still damaged so large amounts of water, sodium and potassium are lost in the flood of urine. Up to 50% of patients may have non-oliguric ATN which is associated with high urine volumes. This type of ATN generally follows a more benign clinical course. Ischaemic ATN is associated with focal tubular epithelial necrosis. The straight portion of the proximal tubule and the ascending thick limb in the renal medulla are especially vulnerable. There is rupture of the basement membrane and occlusion of tubular lumen by casts. The lesion occurs in a skip like pattern along the nephron with large gaps in between. The 3 stages of ATN include: initiating stage, maintenance stage (decreased urine output) and a recovery stage.

**Question 9**

Ischaemic tubular necrosis (ITN) is associated with which of the following?

A Predominantly PST and ascending loop of Henle necrosis

B A maintenance stage with polyuria

C Intact basement membranes

D Cast obstruction of the proximal tubule

Explanation A

Ischaemic ATN is associated with focal tubular epithelial necrosis. The straight portion of the proximal straight tubule (PST) and the ascending thick limb in the renal medulla are especially vulnerable, but focal lesions may occur in the distal tubule, often in conjunction with casts. In the toxic type extensive necrosis is present along the PST (proximal straight tubules) segments with many toxins. However the ascending loop of Henle and the DCT are also involved. In both types the lumens of the DCT and collecting ducts contain casts. There is rupture of the basement membrane and occlusion of tubular lumen by casts. The lesion occurs in a skip like pattern along the nephron with large gaps in between. The 3 stages of ATN include: initiating stage, maintenance stage (decreased urine output) and a recovery stage.

Initiating phase: slight decrease in urine volume with a slight rise in BUN

Maintenance phase: sustained decrese in urine output, salt and water overload, rising BUN, hyperK, metabolic acidosis and manifistations of uraemia

Recovery phase: steady increase in urne volume, loss of water, sodium and potassium.

ATN is associated with hyperkalaemia in the maintenance phase and hypokalaemia becomes a problem in the recovery phase.

Up to 50% of patients with ATN do not have oliguria and instead have increased urine volumes. This so called non- oliguric ATN which is associated with high urine volumes. This type of ATN generally follows a more benign clinical course and is associated particularly with nephrotoxins

**Question 10**

Regarding the hepatorenal syndrome, which of the following statements is correct?

A It only occurs in kidneys with existing disease

B Urine has a high sodium concentration

C The kidney maintian the ability to concentrate urine

D It is irreversible

Explanation C

In hepatorenal syndrome- there is an appearance of renal failure in patients with severe liver disease, in whom there are no intrinsic renal disturbances (no intrinsic morphological or functional cause). Kidney function promptly improves if hepatic failure is reversed. Pathophysiology appears to consist of decreased renal perfusion pressure, followed by renal vasoconstriction. Because the renal ability to concentrate urine is retained, oliguria with hyperosmolar urine devoid of proteins and low in sodium occurs.

**Question 11**

Which of the folowing statments is correct regarding urolithiasis?

A Presence of hypercalcaemia implies renal insufficiency

B A patient with leukemia is likely to make cystine calculi

C Struvite stones are made up of magnesium-ammonium-phosphate

D Calcium is the major component in 35% of calculi Your Answer

Explanation C

Leukaemia causes uric acid stones due to the high cell turnover and resulting hyperuricaemia.

Hypercalcuria can be due to absorptive (gastrointestinal hyper absorption) or renal (intrinsic impairment of renal tubule reabsorption) impairments.

Calcium oxalate stones 70% Struvite stones 15% Uric acid stones 5-10% Cysteine stones 1%

**Question 12**

Regarding pyelonephritis, which of the following statements is correct?

A 85% of infections are caused by Gram negative bacteria

B Ureteral obstruction makes haematogenous infection less likely

C Infection is less likely during pregnancy

D Papillary necrosis and perinephric abscess are common seqelae

Explanation A

Infection is more likely in pregnancy. 4-6% of pregnant women have bacteruria and 20-40% will develop a symptomatic infection if not treated. Haematogenous infection is more likely to occur in the presence of ureteral obstruction, in debilitated patients and in patients receiving immunosuppressive therapy. It is associated with non-enteric organisms such as staphalococcus or fungi. Patchy interstitial suppurative inflammation and tubular necrosis are the hallmarks of acute pyelonephritis

**Question 13**

Alkalinisation of urine may precipitate which of the following renal calculi?

A Calcium oxalate stones

B Uric acid stones

C Struvite stones

D Cystine stones

Explanation C

Uric acid stones form in acid urine- pH<5.5. If the urine is alkaline, uric acid remians soluble and does not precipitate out. This forms the basis of treatment of uric acid calculi. Struvite stones develop when the pH is higher than 7.2 and ammonia is present in the urine. Calcium stones-calcium oxalate develops in acid urine and calcium phosphate develops in alkaline urine (pH >7.2). Cystine stones are caused by genetic defects in the renal reabsorption of aminoacids, including cystine, leading to cytinuria. Stones form at low urinary pH.

**Question 14**

Which of the following statements is true about adult polycystic kidney disease (PKD)?

A Mitral valvular disease is present in 20 - 25% of patients Correct Answer

B Most die from a ruptured berry aneurysm

C It can cause chronic renal failure in 2% of affected patients

D It is an autosomal recessive disease

Explanation A

PKD is an autosomal dominant (adult) kidney disease. It is relatively common affecting 1 of every 400-1000 live births. It accounts for 5-10% of cases of Chronic Renal Failure (CRF) requiring transplantation or dialysis. Extra-renal congenital abnormalities are common, 40% have polycystic liver disease. Intracranial berry aneurysms arise in the circle of Willis and Sub-arachnoid Haemorrhage (SAH) account for 4-10% of individuals. Mitral valve prolapse and other cardiac valvular anomalies occur in 20-25% of patients, but most are asymptomatic. Ultimately about 40% of adult patients die of coronary or hypertensive heart disease, 25% of infection, 15% of ruptured berry aneurysms or hypertensive intracerebral bleed.

Note: there is a form of autosomal recessive (childhood) polycystic kidney disease which is genetically distinct from adult PKD

PKD1 mutation causes CRF in 95% of patients by age 70 PKD2 mutation causes CRF in 45% of patients by age 70 Either way.

Therefore, adult PKD is autosomal dominant and not recessive, making the statement: It is an autosomal recessive disease-wrong

**Question 15**

A 5yr old girl presents to the emergency department with malaise, fever, nausea, oliguria and haematuria. You identify red cell casts in her urine. What is the most likely diagnosis?

A Membranous glomerulopathy

B Post streptococcal glomerulonephritis

C Focal, segmental glomerulonephritis

D Minimal change disease

Explanation B

Poststerptococcal glomerulonephritis usually appears 1-4 weeks after a streptococcal throat or skin infection. It occurs most frequently in ages 6-10yrs, but adults of any age can be affected. The A beta haemolytic streptococci are the causative agents, but only certain strains are nephrogenic. More than 90% of cases being traced to types 12, 4 and 1.

It is an immune mediated disease-type III hypersensitivity reaction.

Clinical picture: abrupt malaise, fever, nausea, oliguria and haematuria 1-2 weeks after recovery form a sore throat. The patients have red cell casts in the urine, mild proteinuria (<1gm/day), periorbital oedema and mild to moderate hypertension. More than 95% of cases eventually recover with only conservative therapy of maintaining a salt and water balance. Few progress to a chronic glomerulonephritis with or without recurrence of an active nephritic picture. Adults present more atypically with the sudden appearance of hypertension., oedema with the elevation of the renal urea and creatinine.

Minimal change disease is associated with children, but it typically does NOT present with haematuria.

Note: 5,7,10,11,13