F2011MS48 – PKD

In a patient with ADKPD, which is the primary mechanism for the development of kidney failure?

A. Increase in the number of cysts
B. Hypertension
C. Increase in size of existing cysts
D. Peri-cyst fibrosis
E. Recurrent haemorrhage of cysts

I think a.

From Uptodate-

Early cysts begin as dilatations of intact tubules that are in contact with the nephron and fill by glomerular filtration [27,75]. In contrast, enlarging cysts lose their connection to functioning nephrons as they reach a size of more than 2 to 3 mm. Cyst growth in this setting results from secretion of fluid into the cysts (not glomerular filtration) and is associated with hyperplasia of the cyst epithelium that may reflect underlying maturational arrest.

F2015CA64 - Sodium bicarbonate in CKD

What is the main benefit of sodium bicarbonate in the treatment of Chronic Metabolic Acidosis in Chronic Renal Failure?

A. Decrease Potassium excretion
B. Decrease Phosphate excretion
C. Increase responsiveness to erythropoietin
D. Increase glucose uptake in skeletal muscle
E. Increase protein production

I think e.

Effects of sod bicarb in CKD are seen in:

- Progression of CKD: Good evidence to that.
- Bone health- Bones help with buffering of some of the metabolic acidosis with the release of calcium and phosphate from bone. Preventing this change may minimize the degree of negative calcium balance and prevent or delay the progression both of osteopenia and of hyperparathyroid bone disease.
- Nutritional status: Uremic acidosis can increase skeletal muscle breakdown and diminish albumin synthesis, leading to muscle wasting and muscle weakness.

F2015CA47 - Young adult CKD aetiology

A 27 year old man presented with hypertension and proteinuria with eGFR of 54. Renal ultrasound shows a shrunken left kidney and normal right kidney. What is the likely diagnosis?

A. Renal cortical dysplasia
B. Renal agenesis
C. Renal artery stenosis
D. Reflux nephropathy
E. Renal medullary cystic disease

I wonder if the answer is B. RAS is unlikely in a 27-year-old, if the choice was FMD or renovascular HTN (RVH-which includes both FMD and RAS), would have been the correct answer.10-15% of all RVH is FMD which is usually seen below 40 age.

From Uptodate:

Children with a solitary kidney are at risk for long-term CKD, which is thought to be due to glomerular hyperfiltration.

In a population-based study, 353 of 979,630 screened 17-year old conscripts were identified with a solitary kidney. Kidney injury was more common in the group with a solitary kidney compared with those with two kidneys (42 versus 24 percent) and all three components of kidney injury were more prevalent in the group with a solitary kidney; high BP (32 versus 23 percent), proteinuria (18 versus 0.4 percent), and eGFR <90 mL/min/1.73 m^2 (12 versus 0.1 percent) [42]. In this cohort, multivariate analysis showed higher body mass index, male sex, and smaller kidney length were associated with kidney injury

F2015MS37 - Sodium bicarbonate in CKD
In patients with metabolic acidosis due to chronic kidney disease, sodium bicarbonate has been shown to improve outcomes. Sodium bicarbonate achieves this effect by which primary mechanism?

A. Increased proximal tubule potassium excretion
B. Decreased H+ absorption
C. Increased proximal tubule phosphate excretion
D. Increased efferent arteriolar dilatation
E. Increased afferent arteriole dilatation

I am not sure, none of the choices make sense to me. We know that metabolic acidosis leads to the following (from Uptodate):

Bone resorption and osteopenia [11,29-34]
●Increased muscle protein catabolism [7,35-42]
●Aggravation of secondary hyperparathyroidism [43,44]
●Reduced respiratory reserve and exhaustion of body buffer systems, resulting in increased severity of acute intercurrent illnesses [45]
●Reduced Na^+-K^+-ATPase activity in red blood cells [46] and myocardial cells [47], which could impair myocardial contractility and produce heart failure [48]
●Endocrine disorders such as resistance to growth hormone and insulin, and hypertriglyceridemia [7,35,49]
●Systemic inflammation [50,51]
●Hypotension and malaise [6,7]
●Cognitive dysfunction

Mechanism of action of sod bicarb- Dissociates to provide bicarbonate ion which neutralizes hydrogen ion concentration and raises blood and urinary pH.

2008B3 - renal allograft dysfunction

A 45-year-old male receives a kidney transplant from his brother. Ten days after the operation, the transplant function appears to worsen, with serum creatinine rising from 140 µmol/L to 190 µmol/L [60 - 120 µmol/L].
What is the most likely cause of the rise in creatinine?
A. Ureteric obstruction.
B. Calcineurin inhibitor toxicity.
C. Polyoma (BK) virus nephropathy.
D. Acute rejection.
E. Acute tubular necrosis (ATN).

2009B15

A 55 year old gentleman receives a cadaveric renal transplant. Over the following 3 days there is no measurable fall in serum creatinine. A radionucleotide MAG-3 scan (image shown) shows good perfusion and concentration of the radiotracer with no excretion.
What is the likely diagnosis?
A. Hyperacute rejection
B. Acute Tubular Necrosis
C. Cysclosporin Nephrotoxicity
D. Ureteric Obstruction
E. Venous Thrombosis

Answer to 2008B3

The question is a bit vague. We do not have the Calcineurin drug levels with reference ranges (possibility of CNI toxicity vs. rejection). Not clear whether any predisposing event for Acute Tubular Necrosis. Definitely can rule out BK as it is too early in the piece. Mostly guided by the typical timing of Acute Rejection post-transplant, I will choose D-acute rejection.

Answer to 2009B15

Agree with B-ATN. Delayed Graft Function expected with deceased donor transplant, with nuclear scan suggestive of ATN.

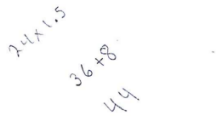
13. A 40 year-old man with renal failure of unknown origin receives a deceased donor kidney transplant. His past history includes renal anaemia, hypertension and Crohn's disease for which he required extensive intestinal surgery including resection of bowel. Post-transplant, he passed 1-2 litres of urine per day for the first week however the creatinine was slow to fall and dialysis was restarted. Three weeks later, he remains anuric and dialysis dependent. Kidney biopsies performed at weeks 1, 2 and 3 after transplant were all reported as normal. Regular ultrasound studies have shown good perfusion of the kidney with no obstruction.

The most likely diagnosis is:
A. Antibody mediated rejection
B. Prolonged delayed graft function
C. Recurrent Oxalosis (secondary)
D. Transplant renal artery stenosis
E. BK nephropathy

It is most likely C-Secondary Oxalosis. With extensive intestinal resection, oxalate doesn't bind with the intestinal luminal Ca and thus gets absorbed. The history of initial urine production and then becoming anuric also is suggestive. Only confusing issue is biopsies being reported as normal- we should be able to see the oxalate deposits on biopsy. I will go with C.

A 72-year-old woman with a history of type 2 diabetes mellitus, congestive heart failure (CHF), and renal failure is admitted for nausea, vomiting, and shortness of breath. Her medications include insulin and frusemide. Her weight is 60 kg. Admission laboratory values are shown below:

Na^+ = 140 mmol/L	[135 – 145]	pH = 7.40	[7.36 – 7.44]
K^+ = 4.1 mmol/L	[3.5 – 5.2]	pCO2 = 40 mmHg	[35 – 45]
Cl^- =95 mmol/L	[95 – 110]	pO2 = 90 mmHg	[80 – 100]
HCO_3^- = 24 mmol/L	[22 – 32]		
Creatinine = 360 umol/L	[70 – 110]		
Urea = 19 mmol/L	[3 – 8]		
Glucose = 9 mmol/L	[3.0 – 7.7]		
Albumin = 41 g/L	[40 – 50]		



She has which of the following?

A. Metabolic acidosis and respiratory alkalosis

B. Metabolic acidosis and metabolic alkalosis

C. Respiratory acidosis and metabolic acidosis

D. Respiratory alkalosis and metabolic alkalosis

E. Metabolic acidosis, metabolic alkalosis, and respiratory alkalosis

Confusing but I would vote for a; logic being with the bicarb going down here with someone in renal failure, I would expect the CO2 to go up with the formula-

Arterial PCO2 = Serum HCO3 + 15 (discussed below)

In this case Pco2 (40) is almost = Serum HCO3 (24) + 15

From Uptodate:

-When a metabolic acid-base disorder reduces the serum HCO3 (metabolic acidosis) or increases the HCO3 (metabolic alkalosis), there should be an appropriate degree of respiratory compensation moving the PCO2 in the **same direction** as the serum HCO3 (falling in metabolic acidosis and rising in metabolic alkalosis)

-When a respiratory acid-base disorder causes the PCO2 to increase (respiratory acidosis) or decrease (respiratory alkalosis), compensation occurs in two phases. There is an immediate, small change in serum HCO3 (in the same direction as the PCO2 change), which is due to whole body buffering mechanisms. If the respiratory disorder persists for more than minutes to hours, the kidneys respond by producing larger changes in serum HCO3 (again, in the **same direction** as the PCO2). These HCO3 changes mitigate the change in pH. Renal compensations are mediated by increased hydrogen ion secretion (which raises the serum HCO3 concentration) in respiratory acidosis and decreased hydrogen ion secretion and urinary HCO3 loss (which reduces the serum HCO3 concentration) in respiratory alkalosis. The renal compensation takes three to five days for completion.

Response to metabolic acidosis — Respiratory compensation for metabolic acidosis causes the arterial PCO2 to fall approximately 1.2 mmHg (0.16 kPa) for every 1 mEq/L reduction in the serum HCO3 concentration. The respiratory response to metabolic acidosis begins within 30 minutes [11] and is complete within 12 to 24 hours.

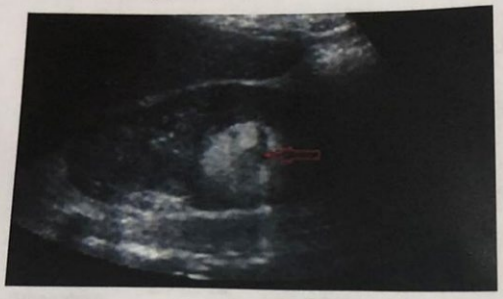
- Arterial PCO2 = Serum HCO3 + 15

Response to metabolic alkalosis — The respiratory compensation to metabolic alkalosis should raise the PCO2 by approximately 0.7 mmHg (0.09 kPa) for every 1 mEq/L elevation in the serum HCO3 concentration [10,18,19]. A very-easy-to-use relationship to determine if the PCO2 is appropriately increased in response to metabolic alkalosis is [20]:

- PCO2 = HCO3 + 10

Question 22

Ms Granger is a 25 year old female who presents to Emergency with left flank pain of sudden onset. She tells you she has a history to tuberous sclerosis and is 30 weeks pregnant. Her blood pressure is 90/60 mmHg. The full blood count showed: Haemoglobin 9.1 g/L, White cell count 12.2 x 10^9/L, Platelet 350 x 10^9/L. An urgent ultrasound was performed, which showed a round 5 cm highly echogenic lesion in the left kidney (red arrow).



Which is the most relevant intervention for this clinical problem?

A. Intravenous desmopressin.
B. Emergency Caesarean and delivery.
C. Renal artery embolization.
D. Urgent nephrectomy.

C. Renal artery embolissation

Yes I think C. Tuberous sclerosis comes with renal angiomyolipomas (AML).

Bleeding from renal AMLs can be mild or catastrophic resulting in hemorrhagic shock, loss of function of the affected kidney, and death [6,7]. Patients with AML who develop active bleeding should receive resuscitative measures (if hemodynamically unstable) and, if feasible, undergo prompt angiography and selective artery embolization (SAE) to stop the bleeding.

A 75 year old man has known myeloma with a lambda light chain detected in his serum (3 gm/l) and urine. He has been referred because of the development of proteinuria over the last 4 months (recently quantitated at 3.5 gm/day, normal < 0.12 gm/day). There is no significant haematuria but his serum creatinine is mildly elevated at 106 umol/l (normal < 90 umol/l). On ultrasound the kidneys are 11.2 cm each in length. The most likely histologic finding on renal biopsy would be:

A. type 1 mesangiocapillary glomerulonephritis

B. light chain deposition disease

C. membranous glomerulonephritis

D. primary amyloidosis

E. myeloma cast nephropathy

D-primary amyloidosis

Yes agree, is AL amyloidosis as pt has 3.5 G proteinuria. If it was a rise in creatinine with non or sub nephrotic proteinuria then the answer would be E.

Causes of AKI in myeloma:

- **Tubular**

Light chain cast nephropathy (myeloma kidney) : 30-50%

Interstitial nephritis/fibrosis : 20-30%

Acute Tubular Necrosis : 10%

- **Glomerular**

Amyloidosis : 10%

Monoclonal immunoglobulin deposition disease (light chain deposition disease) : 5%

Rare: Cryoglobulinemia/MCGN, C3 GN, DDD, Fibrillary GN and immunotactoid glomerulopathy

- Others: Hypercalcemia, use of NSAIDs for bone pain and IV contrast

Haemodialysis patients are prone to significant weight gains between dialysis episodes, due to the accumulation of fluid. This can result in significant BP drops during dialysis. In a patient who has been on regular dialysis for 2 years, which of the following may help stabilise intradialytic BP:

A. Low dialysate sodium
B. Haemodiafiltration
C. High dialysate sodium
D. Pre-dialysis Midodrine
E. Beta-blockers

My vote here would go to b- HDF. High dialysate sodium is realistically not used, at least I have never seen it being used.

- Hemofiltration (HF) uses only convection and no diffusion
    - large volume of replacement fluid (25 to 50 L per day) infused into either the inflow or outflow blood line and both this replacement fluid and excess fluid in the patient are removed by UF
    - Larger sized molecules cleared better by HF in comparison to HD
    - Used in the slow, continuous renal replacement therapies (CRRT) for the sick and hypotensive patient in ICU
    - No dialysis fluid (dialysate) needed
- Hemodiafiltration (HDF) utilizes convective in combination with diffusive clearance i.e., combination of HD and HF (More expensive than HD)

- HDF allows increased clearance of larger-molecular-weight molecules compared to HD
- HDF requires the infusion of significant amounts (usually 15 to 30L per session) of infusate to replace the ultrafiltrate
- **<u>Better tolerated in those with lowish BP</u>**

QUESTION 93

A 60-year-old female admitted under the surgical team following abdominal surgery is commenced on total parenteral nutrition (TPN). She develops thirst and constipation with the following biochemical profile:

Corrected calcium	2.85 mmol/L	(2.1 – 2.6)
Creatinine	130 mmol/L	
Urea	10 mmol/L	
eGFR	44 ml/min	
Vitamin D level	15 nmol/L	
PTH	9.5 pmol/L	(0.5 – 5)

You initiate IV fluids to correct the hypercalcemia. What is the next best test to determine the cause?

A. Neck ultrasound
B. Parathyroid uptake scan
C. 25(OH) vitamin D level
D. 24 hour urine calcium collection

C

Yes agree with C.Although it is a strange Q. Hypophosphatemia is more common along with hypokalemia. Technically I suppose you could argue that excessive Vit D in TPN can cause a high Ca.