

1. What is the quickest-responding physiological mechanism to reduce serum K^+ ?

- A. Aldosterone stimulation of Na/K ATPase
- B. β -agonist stimulation of muscle Na/K ATPase
- C. Administration of a loop or thiazide diuretic
- D. Administration of a large water load

2. You are flying over the Andes on a small plane which is about to crash into the mountains. Since you remembered Cuenoud's talk about the ligamentum arteriosum causing aortic rupture, you instruct everyone to turn backwards. Everyone survives the crash, and people celebrate their near death experience by eating lots of candy. One of the passengers is a Type I diabetic, however, and his insulin vial got lost in the crash. He tells you, "I know you're not (yet) a doctor, but please, do something, my vision's all blurry!" What potassium-regulating mechanism is not at work in his body?

- A. Complete lack of insulin allows glucagon to stimulate the Na/K ATPase unopposed, driving down the serum potassium level
- B. ECF hyperosmolality causes flow of potassium rich water into the extracellular space, driving up the serum potassium level
- C. Panic at not finding his insulin bottle causes a release of epinephrine, which stimulates the β -adrenergic Na/K ATPase, driving down the serum potassium level
- D. Ketoacidosis causing an efflux of potassium out of cells in exchange for serum H^+ , raising the potassium level

3. What is *not* a cause of hypokalemia?

- A. Thiazide diuretics
- B. Hyperaldosteronism
- C. Vomiting
- D. Type IV Renal Tubular Acidosis
- E. Type I Renal Tubular Acidosis

4. What theoretical electrolyte manipulation could cause muscle *paralysis*?

- A. Increase serum $[K^+]$ by 2 Meq
- B. Increase intracellular $[K^+]$ by 2 Meq
- C. Decrease serum $[K^+]$ by 2 Meq
- D. Decrease intracellular $[K^+]$ by 2 Meq

5. A 60 yo man on regular checkup patient shows orthostatic hypotension and a U wave on EKG. He says he is on a "water pill" due to having a "bad heart" but doesn't remember exactly what drug he is taking. You suspect he has an electrolyte abnormality, and the lab values confirm your suspicions. What would be a logical next step in his treatment?

- A. Administration of Calcium Gluconate
- B. Albuterol
- C. Adding an aldosterone antagonist to supplement his "water pill"
- D. Treatment with a drug that increases cardiac preload

6. A patient's renal biopsy shows crescents. You order a test to find out what's in his serum. What would you be LEAST likely to find?

- A. anti-step antibodies
- B. anti-NC1 α -3 collagen IV antibodies
- C. P-anca
- D. C-anca
- E. anti-HBV antibodies

7. Where is ammonia synthesized in the kidney?

- A. The same area where 90% of the bicarb is reabsorbed.
- B. The same area where aldosterone has its effect
- C. The same area where ADH acts
- D. The same area affected in Type 1 RTA

Mix and match: RTA

- A. Type 1
- B. Type 2
- C. Type 3
- D. Type 4

8. Impaired distal H^+ secretion

9. Kind of like the 5th aortic arch

10. May show very high levels of 18-hydroxycorticosterone

11. Causes hypokalemia

12. clinically manifested by acidemia and hyperkalemia

13. Management of chronic renal failure: What would you NOT recommend for someone in chronic renal failure with a GFR of 30 ml/min?

- A. A diet low in phosphorous
- B. Supplemental vitamin D
- C. phosphorous-binding medications (i.e. Tums)
- D. recombinant PTH

14. What does the most data say the most common cause of ESRD in the US is?

- A. diabetes
- B. hypertension
- C. glomerulonephritis
- D. cystic kidney

15. What is the most common cause of death in ESRD patients?

- A. cardiac
- B. septicemia
- C. voluntary withdrawal from dialysis
- D. uremia
- E. HUS/TTP

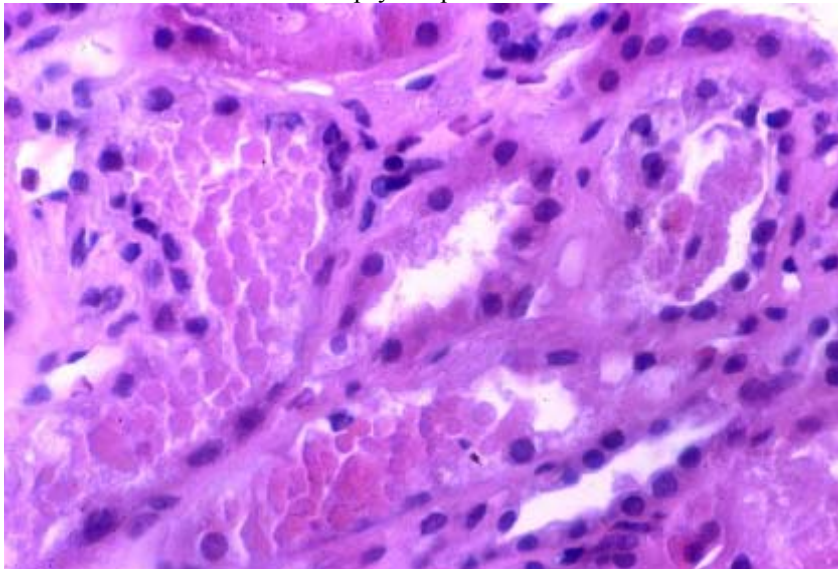
16. What would you expect the approximate FENa to be of an ESRD patient with destruction of 80% of his glomeruli? Assume the patient is eating a normal diet, and is not restricting salt or water.

- A. 0.01%
- B. 2%
- C. 10%
- D. 50%

17. What continues to rise and rise as GFR continues to fall in an ESRD patient?

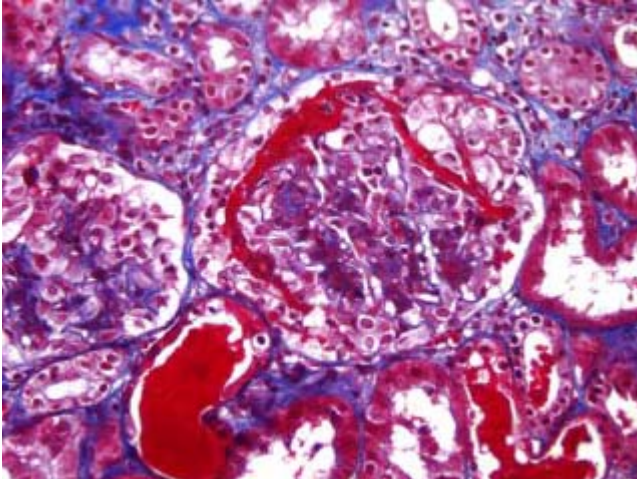
- A. serum phosphage
- B. serum calcium
- C. serum PTH
- D. serum Na

18. What is TRUE about this biopsy sample?



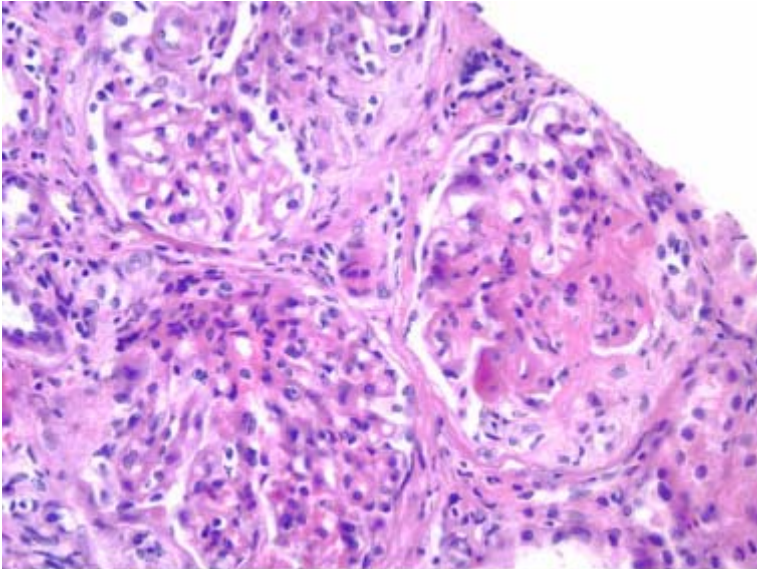
- A. This is an irreversible lesion
- B. The cells sloughing off into the lumen underwent apoptosis as the end result of a molecular chain of events that involved decreased intracellular calcium
- C. This patient could have had a systolic blood pressure of 80
- D. This pathophysiology occurs more frequently in outpatients compared to inpatients
- E. Walker says I have AIDS

19. What is TRUE about this lesion? (it is trichrome stained where fibrin is red)



- A. The fibrin in bowman's space is the result of parietal epithelial cell proliferation
- B. The fibrin in bowman's space is the result of visceral epithelial cell proliferation
- C. The GBM is thickened
- D. The patient would show anasarca
- E. The patient has a poor prognosis

20. What is NOT true about this slide?



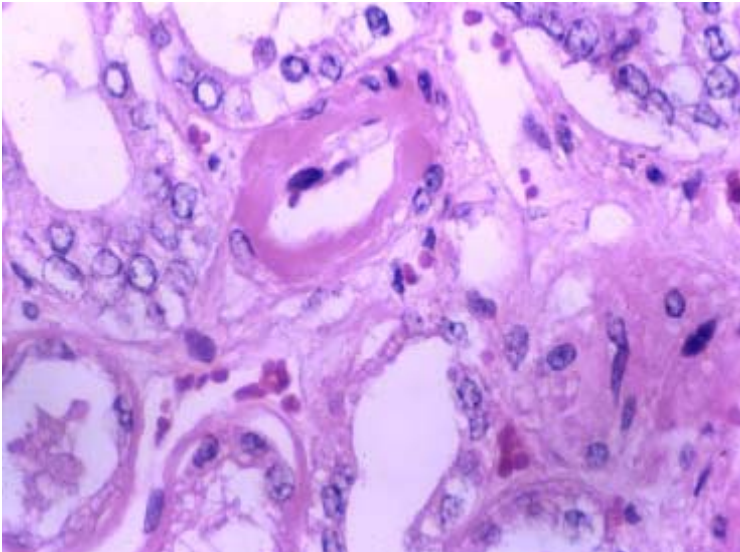
- A. The three glomeruli are in three different stages of disease
- B. This patient may have a blood pressure of 140/90
- C. The three glomeruli probably have very different blood flow
- D. There is evidence of normalcy, hypertrophy, and sclerosis
- E. There may be effacement of parietal epithelial foot processes on EM
- F. This could represent many different diseases or stages of diseases
- G. This represents a diffuse segmental process

21. What is not true about this pathology?



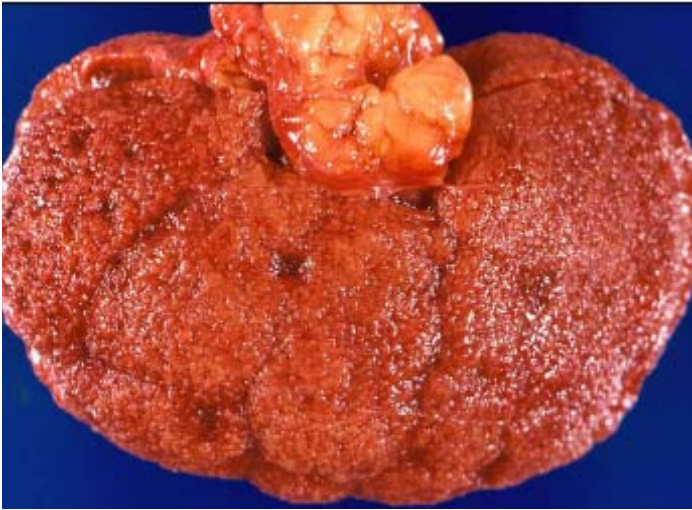
- A. It most likely represents a mutation on chromosome 16
- B. This patient is at an increased risk for stroke
- C. This patient may have abdominal discomfort
- D. It most likely represents a mutation in p53
- E. This is not Von-Hippel-Lindau syndrome

22. What is this a hallmark of?



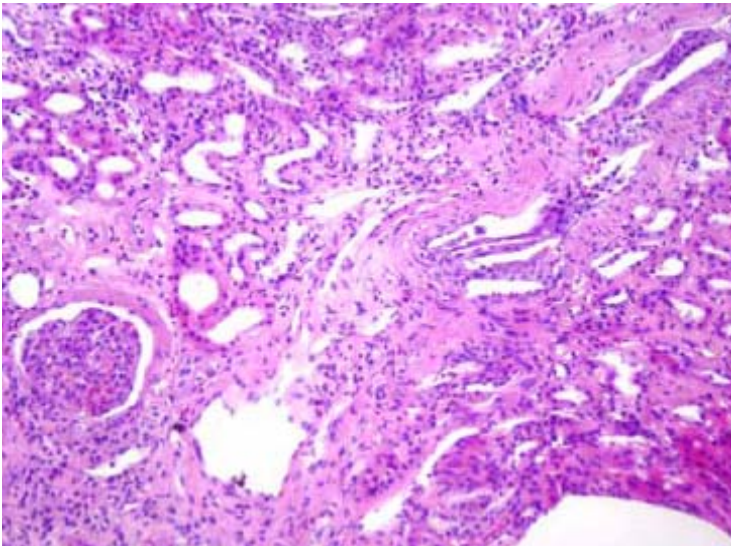
- A. Benign nephrosclerosis
- B. Malignant nephrosclerosis
- C. Minimal change disease
- D. Renal cell carcinoma
- E. Von Hippel-Lindau syndrome

23. What was probably NOT present in this patient?



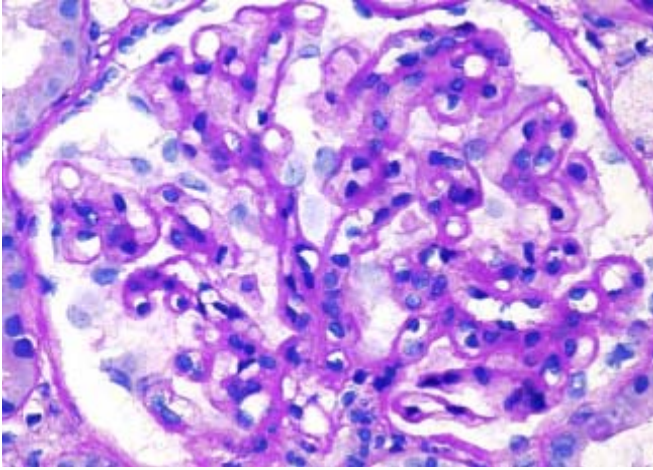
- A. An increased risk of stroke
- B. Blood pressure over 190 systolic
- C. Sclerotic vessels
- D. intimal fibrosis of arteries
- E. focal segmental glomerulosclerosis

24. What is shown in this slide?



- A. Von Hippel-Lindau syndrome
- B. Malignant nephrosclerosis
- C. Cholesterol embolus
- D. Focal Segmental Proliferative glomerulonephritis

25. What is TRUE about this?



- A. You would expect to see subepithelial electron dense humps on EM
- B. You would expect to find IgA in the mesangium on IF
- C. You would expect to find a negative IF and loss of foot processes on EM
- D. You would expect to find spikes and loops with silver stain

Mix and Match: glomerular disease (more than once, you know the drill)

- A. diffuse proliferative glomerulonephritis
 - B. focal segmental glomerular sclerosis
 - C. focal segmental proliferative glomerulonephritis
 - D. membranous nephropathy
 - E. minimal change disease
26. may be caused by henoch schonlein purpura
27. subepithelial “humps” on EM
28. berger’s disease is a familial form
29. subepithelial deposits on EM
30. RBC casts in urine, IgG immunofluorescence
31. can be caused by hypertension

32. What is not a complication of acute renal failure?

- A. pulmonary edema
- B. hyperkalemia
- C. hypernatremia
- D. hypocalcemia
- E. metabolic acidosis

33. What is true about renal clear cell carcinoma?

- A. RCCC is a malignant tumor derived from distal tubules
- B. RCCC has a 5 year mortality rate approaching 90%
- C. RCCC is the most common renal cell malignancy
- D. Foamy macrophages are apparent with LM

34. What the hell is Von Hippel-Lindau syndrome?

- A. An autosomal recessive mutation on chromosome 3
- B. A mutation leading to uninhibited transcriptional elongation steps during RNA synthesis
- C. A familial syndrome with increased risk of berry aneurisms
- D. Kind of a funny name. But not that funny. Still a little funny though. Was it two people? Von Hippel *and* Lindau? Or was it one bald old german guy? Well, I got kind of curious, and found their mascot: I am not making this up.

here she is, the von hippel mascot:



And here's The Man (Landau came later):



Answers

1. B. All of these choices will lower serum K⁺. However, β-adrenergic stimulation (by epinephrine) is one of the main components of the very fast “intrinsic” K⁺ regulatory system that moves K⁺ into/out of cells. All other choices work at the level of the kidney, which runs the slower “extrinsic” system of potassium regulation.
2. A. Insulin stimulates the Na/K ATPase, not glucagon. In the absence of insulin, the plane crash survivor is at risk of *hyperkalemia* due to the just-mentioned unstimulated Na/K pumps, and the reasons described in B and D.
3. D. Type IV Renal Tubular Acidosis causes *hyperkalemia* because of low aldosterone. Aldo stimulates K⁺ efflux into the lumen. Therefore, hyperaldo would cause K⁺ wasting. Type I RTA causes K⁺ wasting because hydrogen ions cannot be excreted by the α-intercalated cell. Thiazide is a K⁺ wasting diuretic because it causes an increase in Na⁺ and reabsorption of sodium causes an excretion of K⁺. Vomiting causes a metabolic alkalosis, which drives K⁺ into cells in exchange for intracellular H⁺.
4. C. Hypokalemia makes it harder to fire action potentials (leading to paralysis), while hyperkalemia makes it easier (leading to inappropriate firing). Since the concentration of K⁺ is much much less in the ECF, then a slight reduction of the ECF [K⁺] would have a profound influence on the cell's resting potential: The cell's resting potential would become much more negative, making it harder to reach threshold and thus harder to fire. 2 Meq manipulations of the intracellular levels of [K⁺] would have negligible effect.
5. C. A U wave is indicative of *hypokalemia*. In this case, it's likely that the patient got hypokalemia from his diuretic. Hypokalemia can cause loss of smooth muscle tone (hence the orthostatic hypotension). An aldosterone antagonist is an example of a potassium-sparing diuretic that can help to alleviate hypokalemia caused by a diuretic. Calcium gluconate is used to stabilize the cell membrane for the treatment of *hyperkalemia*. Albuterol is a β-agonist that would exacerbate hypokalemia by stimulating the Na/K ATPase (it's used to treat hyperkalemia). Also, he has CHF, and in CHF you want to reduce preload, not raise it.
6. E
7. A
8. A
9. C
10. D
11. A
12. D
13. D
14. A
15. A
16. C
17. C
18. C
19. E
20. G
21. D
22. A
23. B
24. C
25. D
26. C
27. A
28. C
29. D
30. A
31. B
32. C
33. C
34. B