Case Scenario 1: Renal Tubular Dysfunction

Patient Presentation:

A 45-year-old male presents with fatigue, muscle weakness, and polyuria. Blood tests reveal hypokalemia (low potassium), metabolic alkalosis, and normal blood glucose. Urinalysis shows no glucose or protein but high potassium excretion. A renal biopsy is performed.

Histology Findings:

Light microscopy of the kidney shows normal glomeruli. The proximal convoluted tubules (PCT) appear normal with visible brush borders. However, the distal convoluted tubules (DCT) show hypertrophy and increased cellularity.

MCQ Question 1:

Which histological feature is most characteristic of the PCT in this biopsy?

a) Tall columnar cells with no brush border

b) Simple squamous epithelium

c) Simple cuboidal epithelium with a brush border

d) Stratified epithelium with basal striations

Correct Answer: c) Simple cuboidal epithelium with a brush border

MCQ Question 2:

The patient's hypokalemia and metabolic alkalosis are likely due to dysfunction in which tubule segment?

a) Proximal convoluted tubule

b) Loop of Henle

c) Distal convoluted tubule

d) Collecting duct

Correct Answer: c) Distal convoluted tubule

Written Question:

Explain why DCT dysfunction might lead to hypokalemia and metabolic alkalosis in this patient.

Answer:

The DCT is responsible for aldosterone-dependent reabsorption of sodium and secretion of potassium and hydrogen ions. Dysfunction here can lead to excessive potassium secretion (causing hypokalemia) and impaired hydrogen ion secretion (leading to metabolic alkalosis). Hypertrophy of DCT cells may indicate compensatory mechanisms due to chronic electrolyte imbalances.

Case Scenario 2: Glomerular and Tubular Damage

Patient Presentation:

A 60-year-old female with a long history of poorly controlled hypertension presents with worsening edema, proteinuria, and elevated serum creatinine. A kidney biopsy is done to assess damage.

Histology Findings:

The biopsy shows glomerulosclerosis and thickened basement membranes. Additionally, many proximal convoluted tubules show epithelial flattening, loss of brush borders, and protein reabsorption droplets in the cytoplasm.

MCQ Question 1:

What is the likely cause of the protein droplets in PCT cells?

a) Glycogen storage

b) Lipid accumulation

c) Reabsorbed proteins from filtrate

d) Viral inclusions

Correct Answer: c) Reabsorbed proteins from filtrate

MCQ Question 2:

Which part of the nephron is primarily damaged in hypertensive nephropathy?

a) Loop of Henle

b) Collecting duct

c) Glomerulus and PCT

d) Juxtaglomerular apparatus

Correct Answer: c) Glomerulus and PCT

Written Question:

Describe the connection between glomerular damage and the observed changes in the PCT.

Answer:

Glomerular damage increases permeability of the filtration barrier, leading to proteinuria. The PCT, which normally reabsorbs small proteins via pinocytosis, becomes overloaded with protein. This results in visible protein reabsorption droplets in the cytoplasm and eventual tubular epithelial damage, contributing to progressive renal dysfunction.

Case Scenario 3: Urinary Concentration Defect

Patient Presentation:

A 28-year-old female complains of excessive thirst, polyuria, and nocturia. Water deprivation test shows inability to concentrate urine. MRI of the brain is normal. A renal biopsy is considered.

Hypothetical Histology:

If performed, the biopsy might show normal glomeruli and PCT. However, the medullary collecting ducts may show a decrease in principal cells or abnormal expression of aquaporin-2 channels.

MCQ Question 1:

Which cells in the collecting duct are rich in aquaporins and crucial for water reabsorption?

a) Intercalated cells

b) Principal cells

c) Macula densa cells

d) Juxtaglomerular cells

Correct Answer: b) Principal cells

MCQ Question 2:

Which structure is responsible for creating the medullary concentration gradient essential for urine concentration?

a) Proximal convoluted tubule

b) Distal convoluted tubule

c) Loop of Henle

d) Juxtaglomerular apparatus

Correct Answer: c) Loop of Henle

Written Question:

Explain the role of principal cells and aquaporins in urine concentration. How might their dysfunction lead to the patient's symptoms?

Answer:

Principal cells in the collecting ducts express aquaporin-2 channels on their apical membranes, which are inserted in response to ADH. This allows water reabsorption from the filtrate back into the hypertonic medulla, concentrating urine. Dysfunction or deficiency of these channels impairs water reabsorption, leading to dilute urine, polyuria, and compensatory polydipsia.

Case Scenario 4: Juxtaglomerular Apparatus Disorder

Patient Presentation:

A 35-year-old male presents with severe hypertension and hypokalemia. Plasma renin activity is low, and aldosterone levels are high. Ultrasound shows a small adrenal adenoma. Renal biopsy is performed to rule out secondary causes.

Histology Findings:

The glomeruli appear normal. The juxtaglomerular apparatus shows atrophy of juxtaglomerular cells in the afferent arterioles. The macula densa appears normal.

MCQ Question 1:

Which cells secrete renin?

a) Macula densa cells

b) Principal cells

c) Juxtaglomerular cells

d) Extraglomerular mesangial cells

Correct Answer: c) Juxtaglomerular cells

MCQ Question 2:

Atrophy of juxtaglomerular cells would lead to:

a) Increased renin secretion

b) Decreased renin secretion

c) Increased erythropoietin production

d) Hyperplasia of macula densa

Correct Answer: b) Decreased renin secretion

Written Question:

How does the histology correlate with the patient’s low plasma renin and high aldosterone? Discuss the physiological feedback loop involved.

Answer:

Juxtaglomerular cell atrophy leads to decreased renin production, explaining low plasma renin. Normally, renin activates angiotensin II, which stimulates aldosterone release. However, in this case, an adrenal adenoma autonomously secretes aldosterone, causing hypertension and hypokalemia while suppressing renin via negative feedback, leading to JG cell atrophy.

Let me know if you'd like more scenarios or specific adaptations for exam preparation.