Foundations of Medicine

Case Q&A Study Resource

Table of Contents

Introduction

During my university career, my preferred study method involved active recall and question and answer in a case-based format, which is why this resource is designed this way. By using a "Triple-Step Discovery" method (Scenario, Hint, and Deep-Dive) the material moves away from passive reading and mimics the cognitive load of a real exam, forcing the brain to "reach" for the answer before the logic is revealed. To use this resource effectively, first read the clinical scenario carefully. If stuck, refer to the hint to narrow the focus to the correct physiological system or anatomical landmark before finally reviewing the deep-dive. This deep-dive section is crucial as it highlights the foundational logic and the clinical application, which is exactly how UQ integrates their assessments. Finally, the high-yield "exam tips" at the end of the manual can be used for a rapid-fire review in the 48 hours leading up to exams to solidify must-know facts.

Module 1: Renal & Fluid Balance

Scenario 1: The Metabolic Cost of Reabsorption

The Case: A 74-year-old female with chronic hypertension is admitted with signs of acute-on-chronic renal failure. Laboratory results show a significant drop in GFR. You are asked to consider which part of her nephron is currently under the most metabolic stress as it attempts to maintain homeostatic solute reabsorption despite decreased perfusion.

The Question: Which specific segment of the nephron is characterized by the highest density of mitochondria and the most significant oxygen consumption per gram of tissue?

The HINT: Recall your histology and transport kinetics. Which segment is responsible for the "bulk" reabsorption (approx. 65-70%) of filtered sodium, water, and nearly 100% of glucose? High-volume active transport requires massive amounts of ATP.

The Deep-Dive Overview:

- **The Answer: The Proximal Convoluted Tubule (PCT).**
- **Foundational Logic:** The PCT cells are packed with mitochondria to power the **Na+/K+ ATPase pumps** on the basolateral membrane. This creates the electrochemical gradient necessary for secondary active transport (e.g., SGLT2 for glucose).
- **Clinical Logic:** Because the PCT is so metabolically "expensive," it is the first area to fail during **ischemia**. This leads to **Acute Tubular Necrosis (ATN)**, where epithelial cells slough off and form "muddy brown casts" in the urine—a classic UQ exam "trigger" phrase.

Scenario 2: G-Protein Signaling and Water Retention

The Case: A patient is diagnosed with **Central Diabetes Insipidus** following a head injury. They are producing large volumes of dilute urine because their posterior pituitary is no longer secreting Vasopressin (ADH). You decide to treat them with Desmopressin (a synthetic ADH analogue).

The Question: Upon binding to the V2 receptor in the collecting duct, which secondary messenger pathway is activated to trigger the insertion of Aquaporin-2 channels?

The HINT: ADH V2 receptors are G-protein coupled receptors (GPCRs). Does this specific pathway involve the cleavage of PIP2 (IP3/DAG) or the activation of Adenylyl Cyclase?

The Deep-Dive Overview:

- **The Answer: Gs → Adenylyl Cyclase → cAMP → Protein Kinase A (PKA).**
- **Foundational Logic:** This is a textbook example of GPCR signaling. Binding to the **Gs-coupled** V2 receptor activates Adenylyl Cyclase, which converts ATP to **cAMP**. PKA then phosphorylates vesicles containing **Aquaporin-2**, causing them to fuse with the apical membrane.
- **Clinical Logic:** In **Nephrogenic** Diabetes Insipidus, the hormone is present, but the receptor or signaling pathway is broken (often due to Lithium toxicity). Understanding the signaling pathway helps you differentiate between a "brain" problem (Central) and a "kidney" problem (Nephrogenic).

Scenario 3: The RAAS Cascade and Pressure Regulation

The Case: A 28-year-old female is found to have a significant narrowing of her right renal artery (Renal Artery Stenosis). Her blood pressure is 170/105 mmHg. Her kidneys perceive this low flow as "low blood pressure" and activate a compensatory hormonal cascade.

The Question: Which cell type in the kidney senses this drop in perfusion pressure and releases the initial enzyme required to start the RAAS cascade?

The HINT: These cells are modified smooth muscle cells located in the afferent arteriole. They work in tandem with the *macula densa* of the distal tubule.

The Deep-Dive Overview:

- **The Answer: Juxtaglomerular (JG) Cells.**
- **Foundational Logic:** The **Juxtaglomerular Apparatus (JGA)** is the kidney's "thermostat" for pressure. JG cells release **Renin** in response to: 1. Decreased stretch (baroreception), 2. Sympathetic stimulation (beta1), or 3. Decreased NaCl delivery sensed by the macula densa.
- **Clinical Logic:** Renin converts Angiotensinogen to Angiotensin I. The "logic" trap in exams is the role of **Angiotensin II**, which causes potent vasoconstriction and stimulates **Aldosterone** release from the adrenal cortex to increase sodium/water reabsorption.

Scenario 4: Osmotic Shifts and Tonicity

The Case: An endurance runner collapses after a marathon. In an attempt to rehydrate, they consumed 10 liters of pure water over 4 hours without electrolyte replacement. They are now confused and having seizures. Laboratory tests show a serum sodium of 118 mmol/L (Severe Hyponatremia).

The Question: In this state of "Hypotonic Overhydration," what is the net movement of water relative to the patient's brain cells, and what is the primary risk?

The HINT: Water always follows the higher concentration of solutes (Osmosis). If the blood is "dilute" (hypotonic) compared to the "salty" interior of the cells, where will the water go?

The Deep-Dive Overview:

- **The Answer: Water moves into the cells; Risk of Cerebral Edema.**