# ITE Nephrology Practice Test (Sample)

## Study Guide


**Everything you need from our exam experts!**

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# Introduction

**Preparing for a certification exam can feel overwhelming, but with the right tools, it becomes an opportunity to build confidence, sharpen your skills, and move one step closer to your goals. At Examzify, we believe that effective exam preparation isn't just about memorization, it's about understanding the material, identifying knowledge gaps, and building the test-taking strategies that lead to success.**

**This guide was designed to help you do exactly that.**

**Whether you're preparing for a licensing exam, professional certification, or entry-level qualification, this book offers structured practice to reinforce key concepts. You'll find a wide range of multiple-choice questions, each followed by clear explanations to help you understand not just the right answer, but why it's correct.**

**The content in this guide is based on real-world exam objectives and aligned with the types of questions and topics commonly found on official tests. It's ideal for learners who want to:**

- **Practice answering questions under realistic conditions,**
- **Improve accuracy and speed,**
- **Review explanations to strengthen weak areas, and**
- **Approach the exam with greater confidence.**

**We recommend using this book not as a stand-alone study tool, but alongside other resources like flashcards, textbooks, or hands-on training. For best results, we recommend working through each question, reflecting on the explanation provided, and revisiting the topics that challenge you most.**

**Remember: successful test preparation isn't about getting every question right the first time, it's about learning from your mistakes and improving over time. Stay focused, trust the process, and know that every page you turn brings you closer to success.**

**Let's begin.**

# How to Use This Guide

**This guide is designed to help you study more effectively and approach your exam with confidence. Whether you're reviewing for the first time or doing a final refresh, here’s how to get the most out of your Examzify study guide:**

### 1. Start with a Diagnostic Review

**Skim through the questions to get a sense of what you know and what you need to focus on. Your goal is to identify knowledge gaps early.**

### 2. Study in Short, Focused Sessions

**Break your study time into manageable blocks (e.g. 30 – 45 minutes). Review a handful of questions, reflect on the explanations.**

### 3. Learn from the Explanations

**After answering a question, always read the explanation, even if you got it right. It reinforces key points, corrects misunderstandings, and teaches subtle distinctions between similar answers.**

### 4. Track Your Progress

**Use bookmarks or notes (if reading digitally) to mark difficult questions. Revisit these regularly and track improvements over time.**

### 5. Simulate the Real Exam

**Once you're comfortable, try taking a full set of questions without pausing. Set a timer and simulate test-day conditions to build confidence and time management skills.**

### 6. Repeat and Review

**Don’t just study once, repetition builds retention. Re-attempt questions after a few days and revisit explanations to reinforce learning. Pair this guide with other Examzify tools like flashcards, and digital practice tests to strengthen your preparation across formats.**

**There’s no single right way to study, but consistent, thoughtful effort always wins. Use this guide flexibly, adapt the tips above to fit your pace and learning style. You've got this!**

# Questions

1. **A patient with metabolic acidosis has a positive urine anion gap and hyperkalemia. This pattern most strongly suggests which RTA?**
   - **A. Proximal RTA**
   - **B. Type 4 RTA**
   - **C. Distal/type 1 RTA**
   - **D. Diuretic-induced acidosis**

2. **What is the recommended rate and timeline for correcting hypernatremia?**
   - **A. 5 mEq/L per hour**
   - **B. No more than 1 mEq/L per hour with 50% correction in 24-35 hr and complete correction in 3-7d**
   - **C. 0.1 mEq/L per hour**
   - **D. 2 mEq/L per hour**

3. **What is the classic immunofluorescence pattern seen in anti-GBM disease (Goodpasture syndrome) involving the glomerular basement membrane?**
   - **A. Granular IgG and C3 deposition along the GBM.**
   - **B. Linear staining of the glomerular basement membrane.**
   - **C. Mesangial IgA deposition.**
   - **D. Subepithelial immune complex humps on EM.**

4. **ADPKD has an increased risk of which renal cancer type?**
   - **A. Oncocytoma**
   - **B. Papillary RCC**
   - **C. Chromophobe RCC**
   - **D. Clear cell RCC**

5. **Which antacid components pose toxicity risk in CKD patients?**
   - **A. Calcium-containing**
   - **B. Aluminum-containing**
   - **C. Magnesium-containing**
   - **D. Both aluminum- and magnesium-containing**

**6. Idiopathic membranoproliferative GN biopsy features include which of the following?**

- **A. Hypercomplementemia with normal light microscopy**
- **B. Hypocomplementemia with thickened capillary loops on light microscopy; immune deposits on electron microscopy**
- **C. Crescent formation with pauci-immune deposits**
- **D. Subepithelial humps on EM**

**7. In acute hyperkalemia, which measure is immediately used to stabilize cardiac membranes?**

- **A. IV Calcium Gluconate**
- **B. Kayexalate**
- **C. Insulin plus Dextrose**
- **D. Inhaled Beta-Agonist**

**8. Gitelman syndrome is best described by which profile?**

- **A. High potassium and magnesium with high renin and aldosterone**
- **B. Low potassium and magnesium with low renin and aldosterone**
- **C. Normal potassium and magnesium with normal renin**
- **D. Low potassium and magnesium with low renin and aldosterone**

**9. What is the typical normal range for the urinary anion gap in healthy individuals?**

- **A. 0-10**
- **B. 60-80**
- **C. -10 to 0**
- **D. 30-50**

**10. Timing of hematuria in IgA nephropathy after a mucosal infection is best described as which of the following?**

- **A. Immediately**
- **B. 1-2 weeks after**
- **C. 1-2 days after**
- **D. 1-2 months after**

# Answers

1. B
2. B
3. B
4. B
5. D
6. B
7. A
8. D
9. D
10. C

# Explanations

**1. A patient with metabolic acidosis has a positive urine anion gap and hyperkalemia. This pattern most strongly suggests which RTA?**

**A. Proximal RTA**

**B. Type 4 RTA**

**C. Distal/type 1 RTA**

**D. Diuretic-induced acidosis**

**Urine anion gap reflects how much ammonium (NH4+) the kidneys are excreting to counteract metabolic acidosis. In metabolic acidosis, the kidneys should boost NH4+ excretion, and this NH4+ is balanced in urine by accompanying chloride. When NH4+ excretion is high, chloride in urine rises and the urine anion gap tends to be negative. If NH4+ excretion is impaired, the chloride balance is reduced and the urine anion gap becomes positive. Hyperkalemia points to reduced aldosterone action or resistance, which characterizes Type 4 RTA. In this form, diminished aldosterone effect lowers NH4+ production and excretion, giving a positive urine anion gap, while potassium tends to be elevated because of reduced distal potassium secretion. Distal RTA can also show a positive urine anion gap, but it typically presents with hypokalemia rather than hyperkalemia. Proximal RTA usually preserves ammonium excretion and tends to have a negative urine anion gap with hypokalemia. Diuretic-induced acidosis can cause a positive gap, but the accompanying hyperkalemia specifically points toward aldosterone-related (Type 4) RTA. So the combination of metabolic acidosis, a positive urine anion gap, and hyperkalemia is most consistent with Type 4 RTA.**

**2. What is the recommended rate and timeline for correcting hypernatremia?**

**A. 5 mEq/L per hour**

**B. No more than 1 mEq/L per hour with 50% correction in 24-35 hr and complete correction in 3-7d**

**C. 0.1 mEq/L per hour**

**D. 2 mEq/L per hour**

**Correcting hypernatremia must be gradual because brain cells adapt to high osmolality and rapid shifts can cause dangerous cerebral edema. The safest plan sets a ceiling on how fast the sodium can fall and uses a staged correction over days rather than hours. No more than about 1 mEq/L per hour is allowed, and a practical target is to reduce roughly half of the total sodium deficit within the first 24 to 35 hours, then finish correction over about 3 to 7 days. This approach balances the need to reverse the hypernatremia with the risk of osmotic shifts affecting the brain. For example, if the serum Na is 160 mEq/L and your target is 140 mEq/L, the deficit is 20 mEq/L. Aiming for about 10 mEq/L drop in the first day or so would meet the guideline, with the remainder achieved gradually over the following days, provided the patient's volume status and underlying cause are corrected appropriately.**

## 3. What is the classic immunofluorescence pattern seen in anti-GBM disease (Goodpasture syndrome) involving the glomerular basement membrane?

**A. Granular IgG and C3 deposition along the GBM.**

**B. Linear staining of the glomerular basement membrane.**

**C. Mesangial IgA deposition.**

**D. Subepithelial immune complex humps on EM.**

**The key feature is a linear pattern of IgG (often with C3) along the glomerular basement membrane on immunofluorescence. This occurs because the autoantibodies in anti-GBM disease bind uniformly to antigens present across the entire basement membrane, producing a smooth, continuous line rather than discrete deposits. This linear staining is distinctive and helps separate anti-GBM disease from immune complex–mediated conditions, which show granular deposits. The other patterns align with different diseases: granular IgG/C3 along the GBM corresponds to immune complex GN, mesangial IgA deposition points to IgA nephropathy, and subepithelial humps on EM are typical of post-streptococcal (post-infectious) GN.**

## 4. ADPKD has an increased risk of which renal cancer type?

**A. Oncocytoma**

**B. Papillary RCC**

**C. Chromophobe RCC**

**D. Clear cell RCC**

**ADPKD increases the risk of renal cell carcinoma, and the subtype most consistently linked to this condition is papillary RCC. In patients with ADPKD, papillary RCC is more commonly observed than the clear cell, chromophobe, or oncocytic subtypes, which explains why this option is the best choice. Clinically, if a patient with ADPKD develops a new renal mass or a change in existing cysts on imaging, RCC—often of papillary histology—should be considered.**

## 5. Which antacid components pose toxicity risk in CKD patients?

**A. Calcium-containing**

**B. Aluminum-containing**

**C. Magnesium-containing**

**D. Both aluminum- and magnesium-containing**

**In CKD, reduced kidney function means the body can't clear certain minerals from the bloodstream efficiently, so antacids containing those minerals can accumulate and cause toxicity. Aluminum-containing antacids tend to deposit in bone and brain when they build up, leading to bone disease and neurotoxicity, including encephalopathy in advanced cases. Magnesium-containing antacids can cause hypermagnesemia; as magnesium levels rise, patients may develop weakness, hypotension, slowed reflexes, and in severe cases, cardiac or respiratory depression. Calcium-containing antacids can raise calcium levels and contribute to vascular calcification, but their toxicity risk is not as high or acutely toxic as the aluminum and magnesium forms in CKD. Therefore, the components that pose the clearest toxicity risk in CKD are aluminum- and magnesium-containing antacids.**

## 6. Idiopathic membranoproliferative GN biopsy features include which of the following?

**A. Hypercomplementemia with normal light microscopy**

**B. Hypocomplementemia with thickened capillary loops on light microscopy; immune deposits on electron microscopy**

**C. Crescent formation with pauci-immune deposits**

**D. Subepithelial humps on EM**

**Idiopathic membranoproliferative GN is classically immune complex–mediated with consumption of complement. The biopsy shows thickened capillary loops with a double-contour (tram-track) pattern on light microscopy from mesangial proliferation and GBM splitting, and electron microscopy reveals immune deposits in subendothelial or intramembranous locations. Serum complement levels are typically low due to consumption by the classical pathway activated by these immune complexes. This combination makes the described pattern—hypocomplementemia with thickened capillary loops on light microscopy and immune deposits on electron microscopy—the best fit. The other options point to different processes: hypercomplementemia doesn't fit MPGN; crescents with pauci-immune deposits suggest RPGN from vasculitis; subepithelial humps are more characteristic of postinfectious GN.**

## 7. In acute hyperkalemia, which measure is immediately used to stabilize cardiac membranes?

**A. IV Calcium Gluconate**

**B. Kayexalate**

**C. Insulin plus Dextrose**

**D. Inhaled Beta-Agonist**

**The key idea is that in acute hyperkalemia the first priority is to protect the heart from the electric instability caused by high potassium. Calcium given intravenously stabilizes the myocardial membrane quickly by raising the threshold for depolarization, which reduces excitability and prevents life-threatening arrhythmias. This protection occurs within minutes, giving you time to employ treatments that lower the potassium level itself. Importantly, calcium does not lower potassium—it simply buys time for other measures to work. Options that shift potassium into cells or remove it from the body (like insulin with dextrose or beta-agonists, or GI binders) act on potassium levels, not membrane stability, and Kayexalate has a slower onset. So, intravenous calcium gluconate provides the immediate cardiac membrane stabilization needed in acute hyperkalemia.**

## 8. Gitelman syndrome is best described by which profile?

**A. High potassium and magnesium with high renin and aldosterone**

**B. Low potassium and magnesium with low renin and aldosterone**

**C. Normal potassium and magnesium with normal renin**

**D. Low potassium and magnesium with low renin and aldosterone**

**Gitelman syndrome is a distal convoluted tubule defect in the Na-Cl cotransporter, producing a thiazide-like tubulopathy. This causes salt wasting with mild volume depletion, leading to low potassium and low magnesium in the blood. It also usually causes metabolic alkalosis and hypocalciuria. A distinguishing feature is that renin and aldosterone levels are not elevated; they are typically low to normal, unlike Bartter syndrome where renin and aldosterone are high due to more pronounced volume depletion. Therefore the profile of low potassium and magnesium with low renin and aldosterone best fits Gitelman syndrome.**

## 9. What is the typical normal range for the urinary anion gap in healthy individuals?

**A. 0-10**

**B. 60-80**

**C. -10 to 0**

**D. 30-50**

**The urinary anion gap is a practical way to estimate ammonium excretion by the kidneys and is calculated from the urine concentrations of sodium, potassium, and chloride: (Na+ + K+) − Cl−. In healthy individuals, the kidneys excrete acid mainly as ammonium (NH4+), which is accompanied by chloride to maintain electroneutrality. This higher chloride content in urine makes the gap positive, and the typical magnitude clinicians observe in a normal person falls in the range of about 30–50 mEq/L. So, a positive urinary anion gap around 30–50 mEq/L reflects normal ammonium handling by the kidneys. This value also helps interpret acid-base disorders: a negative or near-zero gap suggests robust ammonium excretion (as seen with diarrhea-driven metabolic acidosis), whereas a persistently positive gap can point toward impaired ammonium excretion as in certain renal tubular acidoses. Keep in mind that diet, urine flow, and timing of collection can slightly influence the exact number, but 30–50 mEq/L is a commonly cited normal range.**

## 10. Timing of hematuria in IgA nephropathy after a mucosal infection is best described as which of the following?

**A. Immediately**

**B. 1-2 weeks after**

**C. 1-2 days after**

**D. 1-2 months after**

**IgA nephropathy typically presents with hematuria that occurs soon after a mucosal infection because mucosal IgA–containing immune complexes form in response to the infection and deposit in the glomerular mesangium. This leads to hematuria within a short window, usually within 1–2 days after the illness starts. The timing helps distinguish it from other post-infectious glomerulonephritides, where the lag is longer (like 1–3 weeks). So the best description is that hematuria appears about 1–2 days after the mucosal infection.**

# Next Steps

**Congratulations on reaching the final section of this guide. You've taken a meaningful step toward passing your certification exam and advancing your career.**

**As you continue preparing, remember that consistent practice, review, and self-reflection are key to success. Make time to revisit difficult topics, simulate exam conditions, and track your progress along the way.**

**If you need help, have suggestions, or want to share feedback, we'd love to hear from you. Reach out to our team at hello@examzify.com.**

**Or visit your dedicated course page for more study tools and resources:**

**https://itenephrology.examzify.com**

**We wish you the very best on your exam journey. You've got this!**