Disorders of Calcium and Phosphate Metabolism Practice Test (Sample)

Study Guide

BY EXAMZIFY

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. Which statement best describes CKD-related secondary hyperparathyroidism?

- **A. PTH is suppressed due to phosphate retention**
- **B. PTH remains within normal range**
- **C. PTH is elevated due to parathyroid hyperplasia and hypocalcemia**
- **D. PTH becomes undetectable**

2. What is the PTH challenge response in PPHP?

- **A. Urinary cAMP does not change**
- **B. Urinary cAMP decreases**
- **C. Urinary cAMP increases > 300 mmol/L, as in normal patients**
- **D. Urinary cAMP increases but only modestly**

3. Albright's hereditary osteodystrophy phenotype is associated with which form?

- **A. PHP Type 1A**
- **B. PHP Type 1B**
- **C. PPHP**
- **D. All forms equally**

4. Which statement is true regarding PPHP?

- **A. Represents the double- pseudo form with resistance to PTH but no ectopic phenotype**
- **B. Represents the double- pseudo form with AHO phenotype**
- **C. Is the same as PHP Type 1A**
- **D. Is characterized by primary hyperparathyroidism**

5. Which scenario is associated with hyperphosphatemia through cellular lysis releasing intracellular phosphate?

- **A. Rhabdomyolysis**
- **B. Renal failure**
- **C. Increased calcium citrate complexation**
- **D. Massive transfusion**

6. In primary hyperparathyroidism, the majority of cases are due to which finding?

A. Parathyroid hyperplasia

B. Carcinoma

C. Single adenoma

D. Adenoma in all four glands

7. Which statement best describes osteitis fibrosa cystica?

A. It is a bone disease with high turnover and elevated alkaline phosphatase

B. It is a bone disease with low turnover and low alkaline phosphatase

C. It is a disease with increased bone density

D. It is not associated with hyperparathyroidism

8. Hyperphosphatemia in renal failure is explained by which mechanism?

A. Inability to excrete phosphate

B. Crush injury

C. Massive cellular lysis releasing phosphate

D. Increased calcium citrate complexation

9. In familial hypocalciuric hypercalcemia, PTH level is best described as?

A. Normal to slightly high

B. Elevated markedly

C. Suppressed

D. Undetectable

10. Inheritance of PHP Type 1B is:

A. Autosomal dominant

B. Autosomal recessive

C. UNCLEAR

D. X-linked

Answers

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

Explanations

1. Which statement best describes CKD-related secondary hyperparathyroidism?

A. PTH is suppressed due to phosphate retention

B. PTH remains within normal range

C. PTH is elevated due to parathyroid hyperplasia and hypocalcemia

D. PTH becomes undetectable

In CKD, the balance of calcium and phosphate is disrupted, and that disruption drives secondary hyperparathyroidism. The failing kidneys can't excrete phosphate effectively, so phosphate builds up in the blood. High phosphate promotes hypocalcemia (and, in CKD, there's also reduced production of calcitriol, which lowers intestinal calcium absorption). Low calcium stimulates the parathyroid glands to release more PTH, and with ongoing stimulus they undergo hyperplasia. The result is a chronically elevated PTH level driven by hypocalcemia and parathyroid gland growth, which is exactly what secondary hyperparathyroidism describes. This differs from primary hyperparathyroidism, where PTH is high despite normal or high calcium due to a glandular problem, not kidney-related phosphate and vitamin D disturbances.

2. What is the PTH challenge response in PPHP?

A. Urinary cAMP does not change

B. Urinary cAMP decreases

C. Urinary cAMP increases > 300 mmol/L, as in normal patients

D. Urinary cAMP increases but only modestly

PTH challenge testing looks at how the kidney's tubules respond to parathyroid hormone by measuring urinary cAMP production. In PPHP, the renal tissues are not resistant to PTH, so the PTH signal activates Gs and adenylate cyclase, driving a robust rise in urinary cAMP—similar to normal individuals. That means after the PTH challenge you see a marked increase in urinary cAMP, reflecting intact PTH receptor signaling in the kidney. If there were resistance to PTH, you'd expect little to no rise (or only a very small one), which is what happens in classic pseudohypoparathyroidism. So a strong, normal-like urinary cAMP increase after PTH confirms PPHP rather than a PTH-resistant form.

3. Albright's hereditary osteodystrophy phenotype is associated with which form?

A. PHP Type 1A

B. PHP Type 1B

C. PPHP

D. All forms equally

Albright's hereditary osteodystrophy describes the skeletal and subcutaneous changes seen with GNAS-related hormone resistance. This AHO phenotype is most characteristically linked to pseudohypoparathyroidism type Ia, where a mutation on the maternal GNAS allele causes resistance to PTH in multiple tissues along with the distinctive AHO features. Those features include short metacarpals with brachydactyly, a round face, short stature, and sometimes subcutaneous ossifications. If the GNAS mutation is on the paternal allele, you can see the AHO phenotype without hormone resistance (pseudopseudohypoparathyroidism). Conversely, the other PHP type with primarily hormone resistance but minimal AHO features is less classically tied to the full AHO picture. So, the classic association of Albright's phenotype is with pseudohypoparathyroidism type Ia.

4. Which statement is true regarding PPHP?

A. Represents the double- pseudo form with resistance to PTH but no ectopic phenotype

B. Represents the double- pseudo form with AHO phenotype

C. Is the same as PHP Type 1A

D. Is characterized by primary hyperparathyroidism

PPHP (pseudopseudohypoparathyroidism) sits in the pseudohypoparathyroidism spectrum and is defined by the Albright hereditary osteodystrophy (AHO) phenotype without biochemical resistance to parathyroid hormone. In PPHP, you typically see the skeletal/soft-tissue features of AHO—short stature, brachydactyly, rounded face, subcutaneous calcifications—but calcium, phosphate, and PTH levels respond normally to PTH; there is no renal resistance to PTH. Thus the important point about PPHP is the presence of AHO features without PTH resistance. The other statements are not correct for PPHP: PHP Type IA involves PTH resistance and shares AHO features, primary hyperparathyroidism is a different disorder driven by excess PTH production, and PPHP is not simply the same as PHP Type 1A. The take-home is that PPHP presents with AHO phenotype but normal PTH signaling.

5. Which scenario is associated with hyperphosphatemia through cellular lysis releasing intracellular phosphate?

A. Rhabdomyolysis

B. Renal failure

C. Increased calcium citrate complexation

D. Massive transfusion

Phosphate is predominantly intracellular, so when cells break apart, their stored phosphate floods into the bloodstream. In rhabdomyolysis, there is rapid breakdown of skeletal muscle cells, releasing large amounts of intracellular phosphate (along with potassium and myoglobin) into the circulation. This direct release drives hyperphosphatemia. Renal failure raises phosphate mainly by reduced excretion, not by spilling intracellular phosphate from lysed cells. Increased calcium citrate complexation changes calcium handling rather than releasing phosphate from cells. Massive transfusion can alter electrolytes through donor cell contents and citrate effects, but the classic mechanism of hyperphosphatemia from intracellular phosphate release is most characteristic of rhabdomyolysis.

6. In primary hyperparathyroidism, the majority of cases are due to which finding?

A. Parathyroid hyperplasia

B. Carcinoma

C. Single adenoma

D. Adenoma in all four glands

The main idea here is that primary hyperparathyroidism is most often caused by a single parathyroid adenoma. A solitary adenoma in one gland produces excess PTH autonomously, driving elevated calcium levels, while the other glands are typically normal or suppressed. This pattern accounts for the largest share of cases—about eighty-something percent. The remaining cases usually involve multigland disease, such as hyperplasia affecting multiple glands, or rarely parathyroid carcinoma. The notion of an adenoma in all four glands is not the typical presentation and doesn't fit the common pattern, which is why a single adenoma best explains the majority of primary hyperparathyroidism.

7. Which statement best describes osteitis fibrosa cystica?

A. It is a bone disease with high turnover and elevated alkaline phosphatase

B. It is a bone disease with low turnover and low alkaline phosphatase

C. It is a disease with increased bone density

D. It is not associated with hyperparathyroidism

Osteitis fibrosa cystica is the skeletal manifestation of excessive parathyroid hormone activity, so it represents a high-turnover bone disease. PTH ramps up bone resorption by osteoclasts and, in response, osteoblasts try to keep up, leading to rapid bone remodeling with fibrous tissue replacement and cystic, brown-tumor–like changes. Because this process involves active osteoblastic activity to form new bone, the marker of bone formation—alkaline phosphatase—is elevated. This combination—high bone turnover with an elevated alkaline phosphatase—best fits the condition. A low-turnover scenario wouldn't reflect the PTH-driven remodeling, increased bone density isn't characteristic, and the disease is classically linked to hyperparathyroidism, not independent of it.

8. Hyperphosphatemia in renal failure is explained by which mechanism?

A. Inability to excrete phosphate

B. Crush injury

C. Massive cellular lysis releasing phosphate

D. Increased calcium citrate complexation

In renal failure, hyperphosphatemia mainly happens because the kidneys lose the ability to excrete phosphate as the glomerular filtration rate falls. Phosphate is normally filtered by the glomeruli and largely eliminated via the urine; with fewer functioning nephrons, phosphate clearance drops and serum phosphate builds up. While massive cellular breakdown (crush injuries or widespread lysis) can raise phosphate by releasing it from cells, that mechanism is not what explains the chronic rise in phosphate seen with reduced kidney function. Increased calcium citrate complexation isn't the driver of elevated phosphate in this context, as it alters calcium handling rather than causing renal phosphate retention.

9. In familial hypocalciuric hypercalcemia, PTH level is best described as?

A. Normal to slightly high

B. Elevated markedly

C. Suppressed

D. Undetectable

In familial hypocalciuric hypercalcemia, a loss-of-function mutation in the calcium-sensing receptor makes the parathyroid glands and kidney "think" calcium is lower than it really is. Because the calcium-sensing system is reset, the normal feedback that should suppress PTH when calcium rises is blunted. As a result, PTH secretion stays in the normal range or only mildly elevated despite hypercalcemia. This contrasts with conditions like primary hyperparathyroidism, where PTH is typically clearly elevated, and with situations where PTH would be suppressed. The key takeaway is that PTH is not appropriately suppressed in FHH; it remains normal to slightly high. The accompanying hypocalciuria (low urinary calcium) is another characteristic feature that reflects the same CaSR defect in the kidney.

10. Inheritance of PHP Type 1B is:

A. Autosomal dominant

B. Autosomal recessive

C. UNCLEAR

D. X-linked

The main idea is that inheritance patterns can be unclear when the disorder is driven by epigenetic changes rather than a single gene mutation. Pseudohypoparathyroidism type 1B is caused by methylation defects at the GNAS locus, which disrupt PTH signaling. Because these epigenetic changes can arise de novo or be transmitted in a nonclassic Mendelian way (and imprinting means the effect depends on which parent the abnormal allele comes from), there isn't a consistent, simple autosomal dominant or recessive pattern across all families. This combination of sporadic cases and parent-of-origin effects makes the inheritance pattern itself unpredictable, so it's labeled unclear.

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://disordersofcaandpometabolism.examzify.com

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