

# Disorders of Calcium and Phosphate Metabolism 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

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- 1. In the management of hypomagnesemia-related hypoparathyroidism, which intervention is essential?**
  - A. Calcimimetics**
  - B. Phosphate restriction**
  - C. Calcium supplementation**
  - D. Magnesium repletion**
  
- 2. Which is a major cardiovascular finding in hypercalcemia?**
  - A. Hypotension**
  - B. Hypertension**
  - C. Syncope**
  - D. Edema**
  
- 3. What is the significance of alkaline phosphatase in bone metabolism disorders?**
  - A. Marker of osteoblastic activity and bone turnover; elevated in rickets, osteomalacia, and high-turnover bone disease.**
  - B. Marker of osteoclast activity.**
  - C. Marker of renal function clearance.**
  - D. Marker of calcium absorption in gut.**
  
- 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 statement about primary hypoparathyroidism is true?**
  - A. PTH is low**
  - B. PTH is high**
  - C. PTH is normal**
  - D. PTH varies**

- 6. Which of the following is a major renal manifestation of hypercalcemia?**
- A. Hematuria**
  - B. Proteinuria**
  - C. Kidney stones**
  - D. Flank pain**
- 7. What is the pathophysiology of XLH?**
- A. PHEX mutation leads to increased FGF23 activity causing renal phosphate wasting and decreased 1,25(OH)2D.**
  - B. PHEX mutation leads to decreased FGF23 activity causing renal phosphate retention and increased 1,25(OH)2D.**
  - C. FGF23 mutation causes increased phosphate reabsorption and increased 1,25(OH)2D.**
  - D. Vitamin D receptor mutation leading to phosphate wasting.**
- 8. In pseudohypoparathyroidism, which laboratory pattern is observed?**
- A. Hypocalcemia, hyperphosphatemia, with elevated PTH**
  - B. Hypercalcemia, hypophosphatemia, with elevated PTH**
  - C. Hypocalcemia, hypophosphatemia, with low PTH**
  - D. Hypercalcemia, hyperphosphatemia, with low PTH**
- 9. What is the main difference in PTH levels between primary hyperparathyroidism and familial hypocalciuric hypercalcemia?**
- A. Primary hyperparathyroidism presents with low PTH and high calcium**
  - B. Primary hyperparathyroidism presents with elevated PTH and hypercalcemia**
  - C. Familial hypocalciuric hypercalcemia presents with normal or mildly elevated PTH and hypocalciuria**
  - D. Familial hypocalciuric hypercalcemia presents with low PTH and hypercalciuria**

- 10. Which conditions are associated with increased risk of vascular calcifications due to mineral bone disorder?**
- A. Osteoporosis with vertebral compression**
  - B. CKD-MBD; secondary or tertiary hyperparathyroidism; use of calcium-based phosphate binders**
  - C. Hypervitaminosis D with hypercalcemia**
  - D. Paget disease of bone**

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## Answers

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

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## **Explanations**

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**1. In the management of hypomagnesemia-related hypoparathyroidism, which intervention is essential?**

- A. Calcimimetics**
- B. Phosphate restriction**
- C. Calcium supplementation**
- D. Magnesium repletion**

Magnesium is a crucial cofactor for both PTH secretion and PTH signaling at target tissues. When magnesium is low, the parathyroid gland struggles to release PTH and the kidneys and bones don't respond properly to PTH. This creates hypocalcemia that won't correct just by giving calcium or restricting phosphate. Replenishing magnesium restores normal PTH release and action, allowing calcium to rise toward normal and phosphate handling to improve. Clinically, this means treating with magnesium repletion (often IV magnesium sulfate for acute/severe cases) is the essential step. Once magnesium is corrected, calcium supplementation can be adjusted as needed, but the foundational intervention is repleting magnesium. Other options, like using calcimimetics, would further suppress PTH and worsen calcium levels, and phosphate restriction alone doesn't address the underlying Mg deficiency or PTH axis.

**2. Which is a major cardiovascular finding in hypercalcemia?**

- A. Hypotension**
- B. Hypertension**
- C. Syncope**
- D. Edema**

When calcium levels are high, calcium ions enter vascular smooth muscle cells more readily, causing increased contraction of the vessel walls. This raises systemic vascular resistance and elevates arterial pressure, so hypertension emerges as a prominent cardiovascular effect of hypercalcemia. The heart can also become more contractile and its conduction can be affected, which can accompany the high blood pressure, but the standout finding is the rise in blood pressure. Other options—like hypotension, edema, or syncope—are less typical as direct, primary cardiovascular manifestations of hypercalcemia (they may occur in certain contexts, such as severe dehydration or complex clinical pictures), but hypertension best captures the usual cardiovascular impact of elevated calcium.

**3. What is the significance of alkaline phosphatase in bone metabolism disorders?**

- A. Marker of osteoblastic activity and bone turnover; elevated in rickets, osteomalacia, and high-turnover bone disease.**
- B. Marker of osteoclast activity.**
- C. Marker of renal function clearance.**
- D. Marker of calcium absorption in gut.**

Alkaline phosphatase reflects osteoblastic activity and bone formation, i.e., bone turnover. Osteoblasts ramp up ALP when they're forming bone, and levels rise when there's active matrix mineralization or increased bone formation. In conditions like rickets and osteomalacia, mineralization is defective, so osteoblasts work harder to lay down new bone, driving up ALP levels. This makes ALP a useful marker of bone formation and turnover, not of bone resorption. It's not a marker of osteoclast activity (that role is served by other markers), nor does it measure renal clearance or gut calcium absorption. Clinically, bone-specific ALP helps track bone formation and response to therapy, since total ALP can be elevated for non-bone reasons (liver or intestinal sources).

**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 statement about primary hypoparathyroidism is true?

- A. PTH is low**
- B. PTH is high**
- C. PTH is normal**
- D. PTH varies**

In primary hypoparathyroidism, the parathyroid glands fail to produce sufficient parathyroid hormone. Since PTH is the hormone that raises blood calcium by acting on bone, kidneys, and the gut (via active vitamin D), its deficiency leads to low calcium levels. At the same time, phosphate is not adequately excreted by the kidneys, so phosphate tends to be elevated. This pattern—hypocalcemia with a low, inappropriately low or absent PTH level after a calcium check—defines primary hypoparathyroidism. The other scenarios, where PTH is high or normal, point to secondary causes or resistance rather than a primary gland failure, making the statement that PTH is low the correct one.

6. Which of the following is a major renal manifestation of hypercalcemia?

- A. Hematuria**
- B. Proteinuria**
- C. Kidney stones**
- D. Flank pain**

When calcium is high, the kidneys handle more calcium by excreting it in the urine, making the urine supersaturated with calcium salts. This favors precipitation and stone formation, especially calcium oxalate stones, so calcium-based kidney stones are the major renal manifestation of hypercalcemia. Hematuria or proteinuria can occur but are not the defining renal feature, and flank pain is typically a symptom that arises with passing a stone rather than a direct result of hypercalcemia itself. Chronic hypercalcemia can also impair the kidney's concentrating ability and lead to nephrocalcinosis or diabetes insipidus, but the classic renal issue doctors look for with elevated calcium is calcium stone formation.

## 7. What is the pathophysiology of XLH?

- A. PHEX mutation leads to increased FGF23 activity causing renal phosphate wasting and decreased 1,25(OH)2D.
- B. PHEX mutation leads to decreased FGF23 activity causing renal phosphate retention and increased 1,25(OH)2D.**
- C. FGF23 mutation causes increased phosphate reabsorption and increased 1,25(OH)2D.
- D. Vitamin D receptor mutation leading to phosphate wasting.

XLH is driven by loss-of-function mutations in PHEX that lead to increased FGF23 activity. FGF23 acts on the kidney to promote phosphate wasting by downregulating the sodium-phosphate cotransporters in the proximal tubule, which lowers phosphate reabsorption and causes hypophosphatemia. It also suppresses 1 $\alpha$ -hydroxylase, reducing production of 1,25-dihydroxyvitamin D (calcitriol), further decreasing intestinal phosphate (and calcium) absorption. The combination of low phosphate and low calcitriol impairs bone mineralization, producing rickets in children and osteomalacia in adults. The description that matches XLH is a PHEX mutation leading to increased FGF23 activity with renal phosphate wasting and decreased calcitriol. A statement claiming decreased FGF23 activity would predict phosphate retention and increased calcitriol, which does not fit XLH.

## 8. In pseudohypoparathyroidism, which laboratory pattern is observed?

- A. Hypocalcemia, hyperphosphatemia, with elevated PTH**
- B. Hypercalcemia, hypophosphatemia, with elevated PTH
- C. Hypocalcemia, hypophosphatemia, with low PTH
- D. Hypercalcemia, hyperphosphatemia, with low PTH

Pseudohypoparathyroidism shows end-organ resistance to parathyroid hormone, so the body can't respond to PTH even though it may be produced in high amounts. PTH's normal actions raise serum calcium and lower serum phosphate by increasing calcium reabsorption and decreasing phosphate reabsorption in the kidney, stimulating bone resorption, and promoting activation of vitamin D. When target tissues resist PTH, calcium remains low and phosphate stays high, and the parathyroid glands respond by secreting more PTH. The result is a pattern of low calcium, high phosphate, with elevated PTH levels, which is why this option best fits pseudohypoparathyroidism. In contrast, commonly seen patterns in other conditions help distinguish them: primary hyperparathyroidism typically has high calcium with low phosphate and high PTH; hypoparathyroidism has low calcium with high phosphate and low PTH; PTH-independent hypercalcemia shows high calcium with low PTH.

**9. What is the main difference in PTH levels between primary hyperparathyroidism and familial hypocalciuric hypercalcemia?**

- A. Primary hyperparathyroidism presents with low PTH and high calcium**
- B. Primary hyperparathyroidism presents with elevated PTH and hypercalcemia**
- C. Familial hypocalciuric hypercalcemia presents with normal or mildly elevated PTH and hypocalciuria**
- D. Familial hypocalciuric hypercalcemia presents with low PTH and hypercalciuria**

In hypercalcemia, how PTH behaves helps distinguish these two scenarios. Primary hyperparathyroidism involves autonomous overproduction of PTH by the parathyroid glands, so PTH remains high even with high calcium. That elevated PTH drives bone resorption, kidney reabsorption of calcium, and active vitamin D, leading to hypercalcemia. In familial hypocalciuric hypercalcemia, a calcium-sensing receptor defect shifts the calcium set-point, so PTH is not appropriately suppressed and stays normal or only mildly elevated despite high calcium. The kidneys then reabsorb calcium more avidly, causing hypocalciuria. So the key difference is that primary hyperparathyroidism presents with elevated PTH, whereas familial hypocalciuric hypercalcemia shows normal to mildly elevated PTH with hypercalcemia.

**10. Which conditions are associated with increased risk of vascular calcifications due to mineral bone disorder?**

- A. Osteoporosis with vertebral compression**
- B. CKD-MBD; secondary or tertiary hyperparathyroidism; use of calcium-based phosphate binders**
- C. Hypervitaminosis D with hypercalcemia**
- D. Paget disease of bone**

Vascular calcifications in mineral bone disorder are driven by disruptions in calcium and phosphate handling that occur in chronic kidney disease. When kidneys fail to excrete phosphate effectively, serum phosphate rises and, together with reduced active vitamin D, triggers secondary hyperparathyroidism. The resulting rise in parathyroid hormone drives bone turnover and mobilizes calcium, and the combination can push the calcium-phosphate product toward precipitation in vascular walls. Adding to this, calcium-based phosphate binders used to treat hyperphosphatemia increase the calcium load, further elevating the calcium-phosphate product and promoting vascular calcification. This combination—CKD-MBD with secondary or tertiary hyperparathyroidism and use of calcium-containing phosphate binders—explains the increased risk. Other listed conditions don't fit this specific mechanism. Osteoporosis with vertebral compression centers on bone fragility, not on systemic mineral disturbances driving vascular calcification. Paget disease involves abnormal bone remodeling but not the mineral disorder that promotes vessel calcification. Hypervitaminosis D can cause hypercalcemia, but it's not the same CKD-specific pathway linking phosphate retention, secondary/tertiary hyperparathyroidism, and calcium-phosphate-mediated vascular calcification.

## 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](mailto: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!**

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