

CAMLPR HEMATOLOGY PRACTICE TEST (SAMPLE)

STUDY GUIDE



**SAMPLE STUDY GUIDE
WITH LIMITED QUESTIONS**

**VISIT US ONLINE FOR
THE FULL VERSION STUDY GUIDE**

<https://camlprhematology.examzify.com>



Copyright © 2026 by Examzify - A Kaluba Technologies Inc. product.

ALL RIGHTS RESERVED.

No part of this book may be reproduced or transferred in any form or by any means, graphic, electronic, or mechanical, including photocopying, recording, web distribution, taping, or by any information storage retrieval system, without the written permission of the author.

Notice: Examzify makes every reasonable effort to obtain accurate, complete, and timely information about this product from reliable sources.

SAMPLE

Table of Contents

Copyright	1
Table of Contents	2
Introduction	3
How to Use This Guide	4
Questions	5
Answers	8
Explanations	10
Next Steps	15

SAMPLE

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

SAMPLE

- 1. Which malaria parasite is classically associated with Schuffner's stippling in infected RBCs?**
 - A. Plasmodium ovale
 - B. Plasmodium falciparum
 - C. Plasmodium vivax
 - D. Babesia
- 2. Which finding on electrophoresis is most characteristic of the multiple myeloma case?**
 - A. Hypercalcemia
 - B. Anemia
 - C. Abnormal gamma M-spike
 - D. Rouleaux formation
- 3. A CBC is performed with Hgb 60 g/L, Hct 0.290/L, RBC $3.0 \times 10^{12}/L$, WBC $18.0 \times 10^9/L$, PLT $40 \times 10^9/L$, MCV 91 fL, MCH 29 pg, MCHC 329 g/L, RETIC 10%, Peripheral Smear: Schistocytes, helmet cells, burr cells. Coagulation Testing: PT 40 sec, APTT 65 sec, TT 35 sec, D-dimer >500 ng/mL. Additional Testing: uBIL 80 $\mu\text{mol}/L$, LDH 700 U/L. What is the presumptive diagnosis?**
 - A. Disseminated Intravascular Coagulation
 - B. Thrombotic Thrombocytopenic Purpura
 - C. Hemolytic Uremic Syndrome
 - D. Vitamin K deficiency
- 4. How is the Hgb histogram measured?**
 - A. Measured at 540 nm against a standard
 - B. Enzymatic colorimetric method at 600 nm
 - C. Measured directly by spectrophotometry at 450 nm
 - D. Freed hemoglobin converted to cyanide pigment and measured at 525 nm, compared to reagent blank
- 5. A CBC shows HbC predominance on electrophoresis with rectangular crystals on smear. What is the presumptive diagnosis?**
 - A. Sickle cell disease
 - B. Sickle cell trait
 - C. Hemoglobin C
 - D. Iron deficiency anemia

- 6. When must testing occur for coagulation-based specimens?**
- A. PT testing within 24 hours; APTT within 4 hours; if on heparin, spin within 1 hour
 - B. PT within 1 week; APTT within 2 days
 - C. PT within 24 hours; APTT within 24 hours
 - D. PT within 1 hour; APTT within 2 hours
- 7. A case with >20% blasts, Auer rods, and t(8;21) positive. Which AML subtype is this?**
- A. M2
 - B. M3
 - C. M0
 - D. M5
- 8. What is the primary physiological plasmin inhibitor?**
- A. antithrombin
 - B. α 2-antiplasmin
 - C. fibrinogen
 - D. thrombin
- 9. Which diagnosis is suggested by tear-drop (dacrocytes) RBCs and a dry tap with bone marrow fibrosis and a positive JAK2 mutation?**
- A. Idiopathic myelofibrosis
 - B. Hairy cell leukemia
 - C. Acute myeloid leukemia
 - D. Chronic myeloid leukemia
- 10. What is the purpose of the mucin clot test?**
- A. To identify RBCs
 - B. To test for protein content
 - C. Acetic acid is added to detect turbidity and ensure sample is synovial fluid
 - D. To measure viscosity

Answers

SAMPLE

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

SAMPLE

Explanations

SAMPLE

1. Which malaria parasite is classically associated with Schuffner's stippling in infected RBCs?

- A. Plasmodium ovale
- B. Plasmodium falciparum
- C. Plasmodium vivax**
- D. Babesia

Schuffner's stippling are fine, eosinophilic dots in the cytoplasm of red blood cells that are infected by certain malaria parasites. This pattern is most strongly associated with Plasmodium vivax (and can also be seen with Plasmodium ovale), reflecting changes in reticulocytes that the parasite causes as it develops. On a blood smear, P. vivax usually also enlarges the infected red blood cells and presents with trophozoites and multiple abnormalities in the cytoplasm, all of which together point toward this species. In contrast, Plasmodium falciparum tends to show multiple small ring forms and characteristic banana-shaped gametocytes and typically lacks Schuffner stippling; Babesia can mimic some malaria features but shows Maltese cross tetrads in red blood cells rather than Schuffner dots. Therefore, the presence of Schuffner's stippling most strongly indicates Plasmodium vivax.

2. Which finding on electrophoresis is most characteristic of the multiple myeloma case?

- A. Hypercalcemia
- B. Anemia
- C. Abnormal gamma M-spike**
- D. Rouleaux formation

Multiple myeloma causes a clone of plasma cells to produce a single type of immunoglobulin, which appears on serum protein electrophoresis as a monoclonal spike. This sharp, narrow band in the gamma region—the abnormal gamma M-spike—is the hallmark seen on electrophoresis, reflecting the overproduction of one immunoglobulin by malignant plasma cells. While hypercalcemia and anemia are common clinical features and rouleaux can be seen on a blood smear, they are not the distinctive electrophoresis pattern.

3. A CBC is performed with Hgb 60 g/L, Hct 0.290/L, RBC $3.0 \times 10^{12}/L$, WBC $18.0 \times 10^9/L$, PLT $40 \times 10^9/L$, MCV 91 fL, MCH 29 pg, MCHC 329 g/L, RETIC 10%, Peripheral Smear: Schistocytes, helmet cells, burr cells. Coagulation Testing: PT 40 sec, APTT 65 sec, TT 35 sec, D-dimer >500 ng/mL. Additional Testing: uBIL 80 $\mu\text{mol}/L$, LDH 700 U/L. What is the presumptive diagnosis?

A. Disseminated Intravascular Coagulation

B. Thrombotic Thrombocytopenic Purpura

C. Hemolytic Uremic Syndrome

D. Vitamin K deficiency

Disseminated intravascular coagulation is a consumptive coagulopathy where widespread activation of coagulation causes microthrombi formation and simultaneous consumption of platelets and clotting factors, leading to both thrombosis and bleeding. Here, several findings line up with that process. The blood smear shows schistocytes, helmet and burr cells—typical of microangiopathic hemolysis from intravascular fibrin strands. The patient has marked thrombocytopenia (low platelets) and a severe anemia with reticulocytosis, LDH elevation, and unconjugated bilirubin rise, all pointing to ongoing hemolysis from intravascular clots. Coagulation tests are markedly abnormal: prolonged PT and aPTT indicate consumption of clotting factors, and the thrombin time is extended, consistent with a defective coagulation cascade due to factor consumption. An elevated D-dimer reflects fibrin degradation from widespread clot formation, which is a hallmark of DIC. Taken together, these features—MAHA with schistocytes, thrombocytopenia, prolonged coagulation times, and high D-dimer—most strongly support disseminated intravascular coagulation rather than other microangiopathies where coagulation tests are typically normal.

4. How is the Hgb histogram measured?

A. Measured at 540 nm against a standard

B. Enzymatic colorimetric method at 600 nm

C. Measured directly by spectrophotometry at 450 nm

D. Freed hemoglobin converted to cyanide pigment and measured at 525 nm, compared to reagent blank

Hemoglobin is measured colorimetrically by converting all Hb in the sample to a stable cyanmethemoglobin pigment and then measuring its color intensity. This conversion uses Drabkin's reagent, which contains ferricyanide and cyanide; Hb released from red cells is oxidized and forms cyanmethemoglobin. The resulting pigment has a strong, stable color whose absorbance is proportional to the Hb amount in the sample, in line with Beer-Lambert law. The absorbance is read with a spectrophotometer at about 525–540 nm, and a reagent blank is subtracted to account for any color from the reagents themselves. This setup provides a wide linear range and reliable results across common sample conditions, which is why it's the standard method. Other options don't fit because they describe different approaches or wavelengths not normally used for total Hb quantification with this well-established method.

5. A CBC shows HbC predominance on electrophoresis with rectangular crystals on smear. What is the presumptive diagnosis?

- A. Sick cell disease
- B. Sick cell trait
- C. Hemoglobin C**
- D. Iron deficiency anemia

The main idea is that HbC crystals in red cells are highly characteristic of hemoglobin C. When electrophoresis shows HbC as the predominant hemoglobin, it points to homozygous hemoglobin C disease rather than other conditions. In HbC disease, most of the hemoglobin is HbC with little or no HbA present, whereas in HbC trait you'd expect a mix of HbA and HbC rather than HbC dominance. Sick cell disease or trait would show hemoglobin S on electrophoresis, not HbC, and iron deficiency anemia does not produce HbC crystals or HbC dominance. So the pattern described—the HbC predominance on electrophoresis plus rectangular HbC crystals on smear—fits hemoglobin C disease.

6. When must testing occur for coagulation-based specimens?

- A. PT testing within 24 hours; APTT within 4 hours; if on heparin, spin within 1 hour**
- B. PT within 1 week; APTT within 2 days
- C. PT within 24 hours; APTT within 24 hours
- D. PT within 1 hour; APTT within 2 hours

Coagulation tests are very sensitive to preanalytic handling, so timing matters to ensure accurate results. Prothrombin time (PT) is relatively stable and can be reliably tested within 24 hours after collection if the specimen is stored properly. Activated partial thromboplastin time (APTT) is more time sensitive and should be completed within about 4 hours to avoid drift from factor degradation or artefacts. When a patient is on heparin, the sample's accuracy is more easily affected, so processing should be rapid by separating plasma within 1 hour to minimize the anticoagulant's impact and obtain a valid result. This combination—PT within 24 hours, APTT within 4 hours, and rapid spin within 1 hour if on heparin—best reflects standard preanalytical guidelines.

7. A case with >20% blasts, Auer rods, and t(8;21) positive. Which AML subtype is this?

- A. M2**
- B. M3
- C. M0
- D. M5

The key idea is that a specific cytogenetic abnormality defines this AML subtype. The translocation t(8;21)(q22;q22) RUNX1-RUNX1T1 is a classic hallmark of AML with maturation, i.e., FAB classification M2. When blasts exceed 20% and Auer rods are present, the presence of t(8;21) points strongly to M2, because this translocation is strongly associated with AML showing granulocytic maturation. Other subtypes have different defining features: M3 is acute promyelocytic leukemia typically with t(15;17); M0 is minimally differentiated AML with little maturation; M5 is monocytic leukemia with predominant monocyte lineage. So the combination given aligns best with M2.

8. What is the primary physiological plasmin inhibitor?

- A. antithrombin
- B. α 2-antiplasmin**
- C. fibrinogen
- D. thrombin

Plasmin is the enzyme that dissolves clots by breaking down fibrin, so the body needs a strong brake to prevent excessive bleeding. The primary physiological inhibitor of plasmin in circulation is α -2 antiplasmin. It binds free plasmin with very high affinity, forming a stable complex that rapidly inactivates plasmin and stops further fibrinolysis. This keeps the break-down of clot proteins in check while clotting and repair processes continue elsewhere. Other choices—antithrombin mainly inhibits thrombin and other coagulation proteases, fibrinogen is a clotting substrate, and thrombin is a coagulation enzyme—do not serve as the main plasmin inhibitor in normal physiology.

9. Which diagnosis is suggested by tear-drop (dacrocytes) RBCs and a dry tap with bone marrow fibrosis and a positive JAK2 mutation?

- A. Idiopathic myelofibrosis**
- B. Hairy cell leukemia
- C. Acute myeloid leukemia
- D. Chronic myeloid leukemia

Tear-drop red cells, a dry tap on bone marrow aspiration, marrow fibrosis, and a positive JAK2 mutation all point to primary myelofibrosis. Tear-drop cells arise because the fibrotic marrow distorts developing red cells as blood formation shifts to alternate sites (extramedullary hematopoiesis). The fibrotic marrow makes it hard to aspirate, producing a dry tap. The JAK2 V617F mutation is common in myeloproliferative neoplasms, and its presence strongly supports a myeloproliferative process like primary myelofibrosis rather than an acute leukemia or lymphoid leukemia. Hairy cell leukemia can involve the marrow and cause cytopenias and sometimes a dry tap, but it typically shows hairy cells and a different mutation profile (not predominantly JAK2). Acute myeloid leukemia would present with blasts in blood/marrow rather than a fibrotic dry tap pattern. Chronic myeloid leukemia centers on BCR-ABL and lacks the characteristic tear-drop RBCs with marrow fibrosis and JAK2-driven pathology.

10. What is the purpose of the mucin clot test?

- A. To identify RBCs
- B. To test for protein content
- C. Acetic acid is added to detect turbidity and ensure sample is synovial fluid**
- D. To measure viscosity

Mucin clot testing hinges on the idea that synovial fluid contains mucin, which gives it a slimy, viscous character. When acetic acid is added, this mucin tends to form a visible clot or gel, producing turbidity. If a clot forms, it supports that the fluid is synovial in origin and that mucin is present in adequate quantity. This makes the test a quick check to confirm that the sample is indeed synovial fluid and to assess mucin content, rather than measuring how much protein is present, identifying red blood cells, or directly sizing viscosity.

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://camlprhematology.examzify.com>

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

SAMPLE