

COMPREHENSIVE OSTEOPATHIC MEDICAL LICENSING EXAMINATION (COMLEX) LEVEL 2 PRACTICE EXAM (SAMPLE)

STUDY GUIDE



SAMPLE STUDY GUIDE WITH LIMITED QUESTIONS

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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	16

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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. What is the next step in the management of severe coarctation of the aorta in a newborn?**
 - A. IV antibiotics
 - B. IV prostaglandin E1
 - C. Immediate surgery
 - D. Cardiac catheterization

- 2. What finding in direct immunofluorescence can show inflammatory activity in the skin?**
 - A. Granular IgA deposits
 - B. Linear IgG deposits
 - C. Fibrinogen at the dermal-epidermal junction
 - D. IgE in the papillary dermis

- 3. Which organism is commonly responsible for acute infectious lymphangitis?**
 - A. Escherichia coli
 - B. Streptococcus pyogenes or MSSA
 - C. Staphylococcus epidermidis
 - D. Clostridium perfringens

- 4. What pattern of lymphadenopathy is associated with follicular lymphoma?**
 - A. Progressive lymphadenopathy
 - B. Waxing and waning or slowly progressing LAD
 - C. Rapidly enlarging LAD
 - D. Localized lymphadenopathy

- 5. Which x-ray finding is characteristic of osteosarcoma?**
 - A. Fractured bone
 - B. Destruction of medullary and cortical bone
 - C. Calcification in soft tissue
 - D. Joint effusion

- 6. Which antibody is associated with drug-induced lupus?**
- A. Anti-Ro (SSA)
 - B. Scl-70 antibody
 - C. Anti-histone
 - D. Anti-Jo-1
- 7. In a Grade III ankle sprain, which ligaments are compromised?**
- A. Ankle joint capsule only
 - B. Anterior talofibular and calcaneofibular ligaments
 - C. All ligaments including anterior, calcaneofibular, and posterior talofibular
 - D. Only the posterior talofibular ligament
- 8. What causes the eosinophilic infiltrate seen in Loeffler endocarditis?**
- A. Parasitic infection
 - B. Allergic reaction
 - C. Malignancy
 - D. Bacterial infection
- 9. What is the greatest risk factor for meconium aspiration syndrome?**
- A. Multiple gestation
 - B. Advanced maternal age
 - C. Post mature gestational age
 - D. Maternal smoking
- 10. What is a common symptom of Dandy Walker malformation?**
- A. Hydrocephalus
 - B. Seizures
 - C. Spasticity
 - D. Hypotonia

Answers

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

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Explanations

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1. What is the next step in the management of severe coarctation of the aorta in a newborn?

- A. IV antibiotics
- B. IV prostaglandin E1**
- C. Immediate surgery
- D. Cardiac catheterization

In the management of severe coarctation of the aorta in a newborn, initiating IV prostaglandin E1 is a critical next step. This medication serves to maintain ductal patency, which is essential in cases of coarctation where systemic and pulmonary blood flow may be compromised. In the context of aortic coarctation, especially in neonates, the condition can lead to severe complications if the blood supply to the lower body is restricted. Prostaglandin E1 works by keeping the ductus arteriosus open, thus allowing for collateral circulation to develop and ensuring that blood flow can reach the lower extremities and vital organs. This is particularly crucial in cases of critical coarctation, where the newborn may be at risk for significant end-organ dysfunction. Once stabilized with prostaglandin E1, further definitive treatment, such as surgical intervention or other approaches, can be planned based on the clinical scenario and the infant's overall condition. The use of IV antibiotics, while important in other contexts such as sepsis, does not address the urgent hemodynamic needs associated with coarctation. Immediate surgery may be necessary in some cases, but it would typically follow stabilization with prostaglandin E

2. What finding in direct immunofluorescence can show inflammatory activity in the skin?

- A. Granular IgA deposits
- B. Linear IgG deposits**
- C. Fibrinogen at the dermal-epidermal junction
- D. IgE in the papillary dermis

The presence of linear IgG deposits seen in direct immunofluorescence is indicative of ongoing inflammatory activity in the skin, particularly in the context of autoimmune blistering diseases, such as pemphigus vulgaris or bullous pemphigoid. In these diseases, IgG antibodies target specific components of the skin, leading to a characteristic linear pattern of deposition along the basement membrane zone. This pattern is associated with significant inflammatory response and blister formation. In the context of pemphigoid, for example, the linear IgG deposits point to the binding of antibodies to hemidesmosome proteins, leading to dermal-epidermal separation, which manifests as blisters. The detection of these antibodies highlights the underlying pathophysiological processes that contribute to the inflammation and clinical symptoms observed in these conditions. Other potential findings, such as granular IgA deposits, fibrinogen at the dermal-epidermal junction, and the presence of IgE in the papillary dermis, can reflect different types of dermatologic conditions or processes, but they do not directly signify acute inflammatory activity in the same manner as linear IgG deposits do in the context of autoimmune dermatoses.

3. Which organism is commonly responsible for acute infectious lymphangitis?

- A. Escherichia coli
- B. Streptococcus pyogenes or MSSA**
- C. Staphylococcus epidermidis
- D. Clostridium perfringens

Acute infectious lymphangitis is commonly caused by *Streptococcus pyogenes*, a β -hemolytic streptococcus known for its role in skin and soft tissue infections, particularly following skin breaks. This organism is capable of rapidly spreading through the lymphatic system, leading to the characteristic symptoms of lymphangitis such as red streaking along the lymphatic vessels, fever, and local pain or tenderness. In some cases, Methicillin-sensitive *Staphylococcus aureus* (MSSA) can also be involved in the condition, particularly in patients with skin infections or other predisposing factors. The pathogenesis involves bacterial entry through a cut or abrasion, triggering an inflammatory response in the lymphatic channels. *Streptococcus pyogenes* is particularly efficient at this, as it can adhere to and invade host cells, causing necrosis and suppuration, ultimately leading to lymphatic inflammation. While other organisms such as *Escherichia coli*, *Staphylococcus epidermidis*, and *Clostridium perfringens* may be involved in different types of infections or conditions, they are not typical pathogens associated with acute infectious lymphangitis. *E. coli* is more often implicated in urinary tract infections or intestinal diseases, *Staphylococcus epidermidis* is typically a skin

4. What pattern of lymphadenopathy is associated with follicular lymphoma?

- A. Progressive lymphadenopathy
- B. Waxing and waning or slowly progressing LAD**
- C. Rapidly enlarging LAD
- D. Localized lymphadenopathy

Follicular lymphoma is characterized by a specific pattern of lymphadenopathy that generally presents as waxing and waning or slowly progressing lymphadenopathy. This indolent cancer originates from B lymphocytes and typically exhibits a more gradual increase in lymph node size rather than an acute or rapid enlargement. Patients may experience periods where their lymphadenopathy appears to improve or stabilize, followed by phases of progression. This pattern reflects the nature of follicular lymphoma as a low-grade non-Hodgkin lymphoma, which can often escape immediate detection and may not present symptoms until it has reached a more advanced stage. In contrast, progressive lymphadenopathy suggests a more aggressive disease process and may align more with other lymphomas, while rapidly enlarging lymphadenopathy would be indicative of a more aggressive malignancy such as diffuse large B-cell lymphoma. Localized lymphadenopathy does not fit the typical pattern seen in follicular lymphoma, as this condition often involves multiple lymph node regions rather than being confined to a single area. This highlights the subtle but important clinical features of follicular lymphoma in distinguishing it from other forms of lymphatic malignancies.

5. Which x-ray finding is characteristic of osteosarcoma?

- A. Fractured bone
- B. Destruction of medullary and cortical bone**
- C. Calcification in soft tissue
- D. Joint effusion

Osteosarcoma is a type of malignant bone tumor that typically affects the long bones, particularly around the knee (distal femur and proximal tibia). A hallmark of osteosarcoma on an X-ray is the destruction of both the medullary (inner) and cortical (outer) bone. This radiographic finding reflects the aggressive nature of the tumor and its ability to invade the surrounding bone structure. In osteosarcoma, one can also see a characteristic "sunburst" pattern of bone formation or periosteal reaction due to the rapid bone growth associated with the tumor. However, the destruction of normal bone architecture—both of the medullary cavity and cortex—is essential for the diagnosis. This destructive process often leads to a mixed lytic and blastic appearance on imaging, which is very distinct compared to other conditions. While the other answer choices may relate to various bone or soft tissue conditions, they do not specifically represent the typical imaging findings associated with osteosarcoma. For instance, fractured bones can appear in several different scenarios, calcification in soft tissue can suggest other pathologies such as dystrophic calcification or calcifying tumors, and joint effusion is often seen with injury or inflammatory conditions but is not indicative

6. Which antibody is associated with drug-induced lupus?

- A. Anti-Ro (SSA)
- B. Scl-70 antibody
- C. Anti-histone**
- D. Anti-Jo-1

Drug-induced lupus erythematosus is characterized by the presence of specific antibodies, with anti-histone antibodies being the most commonly associated with this condition. These antibodies are directed against histones, which are proteins found in cell nuclei. Their presence is indicative of drug-induced lupus, typically appearing in patients who have been exposed to certain medications such as procainamide, hydralazine, or isoniazid. In drug-induced lupus, the clinical presentation resembles systemic lupus erythematosus (SLE), but it usually resolves after discontinuation of the offending drug. The anti-histone antibody serves as a diagnostic marker for this condition, differentiating it from other connective tissue diseases. This is why recognizing anti-histone antibodies is crucial in identifying and managing drug-induced lupus appropriately.

7. In a Grade III ankle sprain, which ligaments are compromised?

- A. Ankle joint capsule only
- B. Anterior talofibular and calcaneofibular ligaments
- C. All ligaments including anterior, calcaneofibular, and posterior talofibular**
- D. Only the posterior talofibular ligament

In a Grade III ankle sprain, complete rupture of the affected ligaments occurs, leading to significant instability of the ankle joint. Specifically, this type of sprain typically involves all key ligaments that provide stability to the lateral aspect of the ankle. The anterior talofibular ligament, calcaneofibular ligament, and posterior talofibular ligament are all compromised. The anterior talofibular ligament connects the talus and fibula and is often injured in inversion injuries. The calcaneofibular ligament, which extends from the fibula to the calcaneus, is also commonly involved in this type of sprain. The posterior talofibular ligament, while less commonly injured, can be compromised in more severe sprains. As a result, a Grade III ankle sprain is characterized by damage to all three of these ligaments, leading to the instability in the ankle that is consistent with this classification. This full rupture necessitates extensive rehabilitation and may require surgical intervention due to the loss of ligamentous support in the ankle, making option C the correct choice. The other options reflect either partial involvement of the structures or focus on a single ligament, which does not align with the complete disruption seen in a Grade III

8. What causes the eosinophilic infiltrate seen in Loeffler endocarditis?

- A. Parasitic infection**
- B. Allergic reaction
- C. Malignancy
- D. Bacterial infection

*The eosinophilic infiltrate observed in Loeffler endocarditis is primarily caused by a parasitic infection, specifically related to the presence of certain parasites like *Strongyloides stercoralis* or *Toxocara canis*. This condition is characterized by an infiltration of eosinophils, which are a type of white blood cell that typically responds to parasitic infections and allergic reactions. In Loeffler endocarditis, the underlying parasitic infection leads to an immune response that results in significant eosinophilia, contributing to the inflammation and damage in the heart tissue and valves. The eosinophils in this condition are believed to play a role in both the pathogenesis of the disease and the inflammatory response against the parasites. The distinction between various causes like allergic reactions, malignancy, or bacterial infections lies in the fact that they do not primarily trigger the same type of eosinophilic infiltration as seen in Loeffler endocarditis. While allergic reactions can cause eosinophilia, in the context of Loeffler endocarditis, the initiating factor is specifically the parasitic infections and their subsequent immune-mediated effects on cardiac tissues.*

9. What is the greatest risk factor for meconium aspiration syndrome?

- A. Multiple gestation
- B. Advanced maternal age
- C. Post mature gestational age**
- D. Maternal smoking

Meconium aspiration syndrome (MAS) occurs when a newborn inhales a mixture of meconium and amniotic fluid into the lungs around the time of delivery. The greatest risk factor for MAS is post-mature gestational age, which is defined as being beyond 42 weeks of pregnancy. As the gestation period extends, the likelihood of the fetus passing meconium increases. This can happen because the fetal stress related to post-maturity often leads to meconium-stained amniotic fluid. When the fetus inhales this stained fluid, the meconium can obstruct their airways and lead to respiratory distress, resulting in MAS. In this context, although factors like multiple gestation, advanced maternal age, and maternal smoking can contribute to complications during pregnancy and delivery, they are not as directly linked to the incidence of meconium aspiration syndrome as post-maturity is. Hence, the association with post-mature gestational age is crucial in understanding the greatest risk for this condition.

10. What is a common symptom of Dandy Walker malformation?

- A. Hydrocephalus**
- B. Seizures
- C. Spasticity
- D. Hypotonia

Dandy Walker malformation is a congenital brain malformation characterized by the enlargement of the fourth ventricle, cystic dilation of the cerebellar vermis, and the possible involvement of the posterior fossa. One of the hallmark symptoms associated with this condition is hydrocephalus, which can occur due to the obstruction of cerebrospinal fluid (CSF) pathways. The malformation can lead to an accumulation of CSF, increasing intracranial pressure and causing symptoms such as headaches, vomiting, and developmental delays. While seizures, spasticity, and hypotonia can be associated with various neurological conditions, they are not as directly connected to Dandy Walker malformation as hydrocephalus. Hydrocephalus is particularly significant because it results from the structural abnormalities caused by the malformation, making it a defining and common symptom of this condition. Recognizing hydrocephalus as a primary symptom is crucial for proper diagnosis and management of patients with Dandy Walker malformation.

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:

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

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