COMAT Foundational Biomedical Science (FBS) Practicce Exam (Sample)

Study Guide

Everything you need from our exam experts!

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Questions

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1. What is a key characteristic of hypochromic microcytic anemia?

A. Cigar-shaped red blood cells

B. High reticulocyte count

C. Normal-sized red blood cells

D. Increased white blood cell count

2. What treatment protocol is commonly referred to by the acronym "MONA" in the management of myocardial infarction?

A. Magnesium, Oxygen, Nitroglycerin, Aspirin

B. Morphine, Oxygen, Nitroglycerin, Aspirin

C. Metoprolol, Oxygen, Nitroglycerin, Antiplatelet

D. Metoprolol, Oxygen, Nitroglycerin, Aspirin

3. Where are leptin receptors primarily located in the body?

A. Hypothalamus

B. Liver

C. Pituitary gland

D. Pancreas

4. What is the preferred treatment for acid reflux?

A. Antacids

B. Proton pump inhibitors (e.g., omeprazole)

C. Histamine-2 blockers

D. Prokinetics

5. Which metabolic condition is associated with ethylene glycol poisoning?

A. Respiratory acidosis

B. Metabolic acidosis with an anion gap

C. Metabolic alkalosis

D. Hypercapnia

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6. What type of hypersensitivity is characterized by Hashimoto's thyroiditis?

A. Type I

B. Type II

C. Type III

D. Type IV

7. What is a primary effect of testosterone toxicity in females?

A. Masculinization

B. Increased bone density

C. Menstrual irregularities

D. Infertility

8. Alpha1-selective adrenergic blockers are used to manage which condition?

A. Bradycardia

B. Hypertension

C. Hypotension

D. Heart failure

9. Which type of hypersensitivity reaction is involved in the early phase of allergic rhinitis?

A. Type II hypersensitivity

B. Type I hypersensitivity

C. Type III hypersensitivity

D. Delayed-type hypersensitivity

10. What genetic mutation is characteristic of Myotonic Dystrophy Type 2?

A. CTG repeat on DMPK

B. CCTG repeat on ZNF9

C. GAA repeat on FXN

D. ATG repeat on MYH7

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Answers

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

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Explanations

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1. What is a key characteristic of hypochromic microcytic anemia?

A. Cigar-shaped red blood cells

B. High reticulocyte count

C. Normal-sized red blood cells

D. Increased white blood cell count

The key characteristic of hypochromic microcytic anemia is the presence of smaller than normal (microcytic) red blood cells that also have a reduced hemoglobin content, leading to their hypochromic appearance, which signifies a lighter color when viewed under a microscope. This condition is commonly associated with iron deficiency anemia, where there is insufficient hemoglobin production due to a lack of iron. As a result, the red blood cells produced are smaller and paler than typical healthy red blood cells. The changes in cell morphology and the characteristics of hypochromia are fundamental in diagnosing microcytic anemia. Other options do not align with the defining features of this type of anemia. High reticulocyte counts would indicate an active production of red blood cells, which is less common in hypochromic microcytic anemia unless there is a compensatory response to a recent loss of red blood cells. The presence of normal-sized red blood cells contradicts the definition of microcytic anemia, and increased white blood cell counts are indicative of other conditions, not typically associated with hypochromic microcytic anemia.

2. What treatment protocol is commonly referred to by the acronym "MONA" in the management of myocardial infarction?

A. Magnesium, Oxygen, Nitroglycerin, Aspirin

B. Morphine, Oxygen, Nitroglycerin, Aspirin

C. Metoprolol, Oxygen, Nitroglycerin, Antiplatelet

D. Metoprolol, Oxygen, Nitroglycerin, Aspirin

The acronym "MONA" refers to a specific treatment protocol that is widely used in the management of myocardial infarction (MI). The components of MONA include Morphine, Oxygen, Nitroglycerin, and Aspirin. Morphine is utilized for its analgesic properties to relieve pain and anxiety, which can help decrease heart workload and improve comfort in patients experiencing chest pain during an MI. Oxygen is administered to improve tissue oxygenation, particularly in cases where the patient shows signs of hypoxia or respiratory distress. Nitroglycerin acts as a vasodilator, helping to relieve angina and improve coronary blood flow by relaxing the smooth muscle in blood vessels, thus reducing myocardial oxygen demand. Lastly, Aspirin is given for its antiplatelet effect, which is crucial in preventing further clot formation in the coronary arteries. This combination addresses both the immediate symptoms associated with a myocardial infarction and aims to improve outcomes by preventing further ischemic damage. The protocol is essential in the acute phase of MI, making it a cornerstone in emergency cardiac care. While other combinations of medications and treatments may be beneficial in myocardial infarction management, MONA specifically stands out for its established importance in initial treatment.

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3. Where are leptin receptors primarily located in the body?

A. Hypothalamus

B. Liver

C. Pituitary gland

D. Pancreas

Leptin receptors are primarily located in the hypothalamus, which is a crucial region of the brain that plays a significant role in regulating energy balance, hunger, and body weight. When leptin, a hormone produced by adipose (fat) tissue, is released into the bloodstream, it binds to these receptors in the hypothalamus. This binding initiates a signaling cascade that helps to suppress appetite and increase energy expenditure, thereby contributing to the maintenance of body weight. The hypothalamic pathways influenced by leptin receptors also interact with various neural circuits that control food intake and energy expenditure. Understanding the predominant role of the hypothalamus in leptin action is essential for comprehending conditions such as obesity, where leptin signaling may become disrupted. Other locations considered for leptin receptors, such as the liver, pituitary gland, and pancreas, do have some receptors present, but their primary action related to appetite regulation occurs in the hypothalamus. This distinct localization underlines why the hypothalamus is the correct answer regarding leptin receptor distribution.

4. What is the preferred treatment for acid reflux?

A. Antacids

B. Proton pump inhibitors (e.g., omeprazole)

C. Histamine-2 blockers

D. Prokinetics

Proton pump inhibitors (PPIs), such as omeprazole, are considered the preferred treatment for acid reflux, also known as gastroesophageal reflux disease (GERD). These medications work by blocking the proton pumps in the stomach lining, which are responsible for producing stomach acid. By significantly reducing the production of this acid, PPIs help alleviate the symptoms of acid reflux and promote healing of any esophageal irritation or damage that may have occurred due to excessive acid exposure. In many cases, PPIs have been shown to be more effective than other options because they provide a longer duration of acid suppression, which allows for better symptom control and healing of the esophagus. Additionally, PPIs can be used for both short-term relief and long-term management of GERD, making them a versatile choice in treating this condition. Other treatments, while useful in certain contexts, do not provide the same level of acid suppression or long-term symptom relief. Antacids neutralize existing stomach acid for quick relief but do not prevent future acid production; thus, they are typically used for mild symptoms. Histamine-2 blockers can reduce acid production but are generally less potent than PPIs. Prokinetics may help enhance gastrointestinal motility and prevent reflux but do not

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5. Which metabolic condition is associated with ethylene glycol poisoning?

A. Respiratory acidosis

B. Metabolic acidosis with an anion gap

C. Metabolic alkalosis

D. Hypercapnia

Ethylene glycol poisoning is associated with metabolic acidosis with an anion gap due to the production of toxic metabolites such as glycolic acid and oxalic acid after ingestion. When ethylene glycol is metabolized in the liver, it is converted into these acids, which lead to an accumulation of hydrogen ions in the blood, resulting in a decrease in blood pH (acidosis). This increase in hydrogen ions also causes a widening of the anion gap, which is calculated by the difference between measured cations and measured anions in the serum. The presence of an increased anion gap metabolic acidosis is a hallmark of ethylene glycol toxicity, which can manifest with symptoms such as nausea, vomiting, and central nervous system effects. Prompt recognition and treatment are essential to prevent severe complications and potential mortality. In contrast, respiratory acidosis, metabolic alkalosis, and hypercapnia are not directly related to ethylene glycol poisoning and involve different pathophysiological mechanisms.

6. What type of hypersensitivity is characterized by Hashimoto's thyroiditis?

A. Type I

B. Type II

C. Type III

D. Type IV

Hashimoto's thyroiditis is classified as a Type IV hypersensitivity reaction, which is characterized by cell-mediated immune responses rather than antibody-mediated ones. In this autoimmune condition, the immune system attacks the thyroid gland, leading to inflammation and ultimately hypothyroidism. In Type IV hypersensitivity, T lymphocytes (particularly CD4+ T-helper cells and CD8+ cytotoxic T cells) play a central role. Their activation leads to the recruitment of other immune cells, causing tissue damage. In the case of Hashimoto's thyroiditis, the body produces autoantibodies against thyroid peroxidase and thyroglobulin, but the primary damage is driven by the cellular immune response rather than antibodies. This understanding distinguishes Hashimoto's from other types of hypersensitivity. For instance, Type I hypersensitivity involves IgE-mediated allergic responses, Type II hypersensitivity is characterized by antibody-mediated attacks on specific cell types or tissues, and Type III involves immune complexes that can lead to inflammation and tissue damage. Therefore, the involvement of T cells and cellular mechanisms in Hashimoto's thyroiditis aligns it with Type IV hypersensitivity.

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7. What is a primary effect of testosterone toxicity in females?

A. Masculinization

B. Increased bone density

C. Menstrual irregularities

D. Infertility

The primary effect of testosterone toxicity in females is masculinization. This phenomenon occurs because elevated levels of testosterone can lead to the development of male secondary sexual characteristics. Common manifestations of masculinization include hirsutism (excessive hair growth in male-pattern distribution), deepening of the voice, and changes in body composition, such as increased muscle mass and decreased body fat. The underlying mechanism involves the androgenic effects of testosterone, which can result in alterations to hormone levels and a shift in the balance of estrogen and androgen levels. Such changes can profoundly impact the physical characteristics and biological functions typically associated with female physiology. In contrast, while increased bone density, menstrual irregularities, and infertility can occur due to hormonal imbalances, they are not the primary effects of testosterone toxicity in the same way that masculinization is. Increased bone density might be seen with certain hormonal changes, but it is generally not a direct or common effect of testosterone toxicity itself in females. Menstrual irregularities and infertility may arise from hormonal disruptions caused by testosterone, but again, these are not the most defining or primary consequences of such toxicity.

8. Alpha1-selective adrenergic blockers are used to manage which condition?

A. Bradycardia

B. Hypertension

C. Hypotension

D. Heart failure

Alpha1-selective adrenergic blockers are primarily used to manage hypertension. These medications work by blocking alpha1-adrenergic receptors on smooth muscle in blood vessels, leading to vasodilation and a subsequent decrease in blood pressure. This mechanism is beneficial in treating high blood pressure, helping to reduce the workload on the heart and lower the risk of cardiovascular complications. While alpha1 blockers may also have some applications in other conditions, such as benign prostatic hyperplasia, their key role in managing hypertension distinguishes them in this context. They are not typically used to treat bradycardia, hypotension, or heart failure directly, as those conditions require different therapeutic approaches or agents to address the specific physiological needs of patients.

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9. Which type of hypersensitivity reaction is involved in the early phase of allergic rhinitis?

A. Type II hypersensitivity

B. Type I hypersensitivity

C. Type III hypersensitivity

D. Delayed-type hypersensitivity

The early phase of allergic rhinitis is characterized by a Type I hypersensitivity reaction. This type of hypersensitivity, also known as an immediate hypersensitivity reaction, involves the activation of IgE antibodies that are produced in response to an allergen. When an individual with a sensitized immune system encounters the same allergen again, the allergen cross-links the IgE antibodies bound to mast cells and basophils, leading to their degranulation. This process releases various mediators such as histamine, leukotrienes, and prostaglandins, which cause the characteristic symptoms of allergic rhinitis, including sneezing, nasal congestion, and itching. Understanding the mechanism of Type I hypersensitivity is crucial, as it underscores how allergens trigger an exaggerated immune response in susceptible individuals. This reaction can occur within minutes of exposure to the allergen, marking the early phase of its onset. In contrast, other types of hypersensitivity, such as Type II, Type III, and delayed-type hypersensitivity, involve different immune mechanisms and timelines, making them less relevant to the immediate symptoms experienced in allergic rhinitis.

10. What genetic mutation is characteristic of Myotonic Dystrophy Type 2?

A. CTG repeat on DMPK

B. CCTG repeat on ZNF9

C. GAA repeat on FXN

D. ATG repeat on MYH7

Myotonic Dystrophy Type 2 is primarily associated with a specific genetic mutation characterized by the expansion of a CCTG repeat in the ZNF9 gene. This mutation leads to the symptoms associated with this form of muscular dystrophy, which include muscle stiffness (myotonia), weakness, and other systemic manifestations. The CCTG repeat expansion causes abnormal RNA processing and contributes to the pathophysiology of the disease through a gain-of-function mechanism, where the mutant RNA interferes with normal cellular processes. This understanding of the genetic basis of Myotonic Dystrophy Type 2 is crucial, as it aids in diagnosis and potential therapeutic approaches. Other choices include mutations linked to different conditions: the CTG repeat is associated with Myotonic Dystrophy Type 1, the GAA repeat pertains to Friedreich's ataxia, and the ATG repeat is not commonly associated with a known muscular dystrophy condition. Therefore, the distinct identification of the CCTG repeat on ZNF9 is what firmly aligns with Myotonic Dystrophy Type 2.

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