Lippincott Pharmacology Practice Exam (Sample)

Study Guide

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Questions

1. **What effect does nitroglycerin have when taken with PDE-5 inhibitors?**

   A. It increases heart rate

   B. It can cause life-threatening hypotension

   C. It enhances the effect of PDE-5 inhibitors

   D. It has minimal side effects

2. **A 32-year-old man with opioid addiction presents with cough due to a viral infection. What is the appropriate symptomatic treatment for his cough?**

   A. Guaifenesin/dextromethorphan

   B. Guaifenesin/codeine

   C. Benzonatate

   D. Montelukast

3. **Which pair of antituberculosis drugs has the potential to worsen a patient's gout symptoms?**

   A. Rifampin and isoniazid

   B. Ethambutol and pyrazinamide

   C. Rifampin and ethambutol

   D. Isoniazid and ethambutol

4. **Which is most appropriate for management of a woman diagnosed with postmenopausal osteoporosis with no history of fractures?**

   A. Alendronate

   B. Calcitonin

   C. Denosumab

   D. Raloxifene

5. **For an obese diabetic patient with a history of heart failure, which medication should be added without causing weight gain?**

   A. Linagliptin

   B. Glimepiride

   C. Pioglitazone

   D. Inhaled insulin

6. Which drug is useful in treating sinus bradycardia?

A. Atropine

B. Cisatracurium

C. Neostigmine

D. Succinylcholine

7. For a patient diagnosed with gastroesophageal reflux disease, which medication is most appropriate?

A. An antacid such as aluminum hydroxide

B. Dicyclomine

C. Granisetron

D. Esomeprazole

8. Which of the following best describes the mechanism of action of milrinone in heart failure?

A. Decreases intracellular calcium

B. Increases cardiac contractility

C. Decreases cAMP

D. Activates phosphodiesterase

9. What is the proposed mechanism of cardioprotection from low-dose aspirin?

A. Aspirin preferentially inhibits COX-2 to reduce thromboxane A2 levels.

B. Aspirin preferentially inhibits COX-1 to reduce thromboxane A2 levels.

C. Aspirin preferentially inhibits COX-2 to lower prostacyclin levels.

D. Aspirin preferentially inhibits COX-1 to lower prostacyclin levels.

10. What recommendation should be given to prevent phototoxicity when using fluoroquinolone therapy?

A. Use sunscreen and avoid excessive UV exposure

B. Take the medication at night

C. Take with food

D. Drink with 1 L of water daily

Answers

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

Explanations

1. What effect does nitroglycerin have when taken with PDE-5 inhibitors?

A. It increases heart rate

B. It can cause life-threatening hypotension

C. It enhances the effect of PDE-5 inhibitors

D. It has minimal side effects

Nitroglycerin is a nitrate that is commonly used for the management of angina pectoris, as it induces vasodilation and improves blood flow to the heart. When nitroglycerin is taken in conjunction with phosphodiesterase type 5 (PDE-5) inhibitors, such as sildenafil, tadalafil, or vardenafil, it can lead to a dangerous drop in blood pressure. This is because both medications promote vasodilation through different mechanisms, which can synergistically enhance the effects of each other in terms of lowering vascular resistance and causing hypotension. When the blood vessels dilate too much, the body's ability to maintain blood pressure is compromised, potentially resulting in life-threatening scenarios such as severe hypotension. This risk is heightened in individuals who may already have underlying cardiovascular issues or are on other medications that can lower blood pressure. Thus, the interaction between nitroglycerin and PDE-5 inhibitors is clinically significant, as it underscores the importance of avoiding the combined use of these drugs to prevent serious adverse effects.

2. A 32-year-old man with opioid addiction presents with cough due to a viral infection. What is the appropriate symptomatic treatment for his cough?

A. Guaifenesin/dextromethorphan

B. Guaifenesin/codeine

C. Benzonatate

D. Montelukast

The selection of benzonatate for the symptomatic treatment of cough in this patient is appropriate due to several reasons. Benzonatate is a non-narcotic cough suppressant that works by anesthetizing the stretch receptors in the lungs and pleura, reducing the urge to cough. This is particularly advantageous for a patient with a history of opioid addiction, as avoiding narcotic-based cough treatments helps to minimize the risk of relapse or further substance dependency. In contrast, the other options include medications that pose risks or may not be suitable. Guaifenesin/dextromethorphan contains dextromethorphan, which is an antitussive but can have potential for misuse, especially in individuals with a background of substance abuse. Although guaifenesin is an expectorant and could help with mucus clearance, the inclusion of dextromethorphan would not be optimal in this context. Guaifenesin combined with codeine includes an opioid component, which is contraindicated in a patient with opioid addiction due to the high risk of addiction and adverse effects associated with opioid use. Montelukast is primarily an anti-inflammatory medication used for asthma and allergic rhinitis, and is not indicated for the treatment

3. Which pair of antituberculosis drugs has the potential to worsen a patient's gout symptoms?

A. Rifampin and isoniazid

B. Ethambutol and pyrazinamide

C. Rifampin and ethambutol

D. Isoniazid and ethambutol

The correct answer highlights the combination of ethambutol and pyrazinamide as having the potential to worsen a patient's gout symptoms. Pyrazinamide, in particular, is known to increase uric acid levels in the blood, which can exacerbate gout. When the uric acid levels rise, it can lead to the formation of urate crystals that accumulate in the joints and tissues, resulting in painful gout flares. Ethambutol does not directly affect uric acid levels significantly, but it is often used in combination therapy for tuberculosis alongside pyrazinamide, which can further compound the effects of increased uric acid from pyrazinamide. As a result, while ethambutol alone may not worsen gout, when combined with pyrazinamide, the overall risk of gout symptoms may be heightened due to the latter's impact on uric acid metabolism. This understanding of pyrazinamide's effects relates specifically to its mechanism of action and pharmacodynamic profile, making this pair particularly pertinent for patients with a history of gout or hyperuricemia. Decisions for treatment must carefully consider these side effects, especially in patients vulnerable to gout flares.

4. Which is most appropriate for management of a woman diagnosed with postmenopausal osteoporosis with no history of fractures?

A. Alendronate

B. Calcitonin

C. Denosumab

D. Raloxifene

For a woman diagnosed with postmenopausal osteoporosis and without a history of fractures, the most appropriate management is the use of alendronate. Alendronate is a bisphosphonate that effectively reduces bone resorption and increases bone mineral density, which is crucial for preventing future fractures in individuals at risk for osteoporosis. It is particularly beneficial for postmenopausal women because it has been shown through multiple clinical studies to significantly reduce the incidence of vertebral and hip fractures. Other options, while they may have their indications, are less optimal in this specific context. For instance, calcitonin is generally less effective than bisphosphonates and is typically utilized in those who cannot tolerate other treatments or in acute situations like fracture pain. Denosumab, while effective and used in cases of osteoporosis, is often reserved for patients who have already experienced fractures or for those who have not responded to other therapies. Raloxifene, a selective estrogen receptor modulator, provides benefits in terms of reducing the risk of vertebral fractures but does not have the same strength of evidence for reducing non-vertebral fractures as bisphosphonates like alendronate do. Thus, alendronate stands out as the most appropriate

5. For an obese diabetic patient with a history of heart failure, which medication should be added without causing weight gain?

A. Linagliptin

B. Glimepiride

C. Pioglitazone

D. Inhaled insulin

Linagliptin is an appropriate choice for an obese diabetic patient with a history of heart failure because it is a DPP-4 inhibitor that typically does not cause weight gain. Instead, many patients taking linagliptin may experience weight neutrality or even modest weight loss. This characteristic is particularly important for patients who are overweight or obese and may have comorbid conditions such as heart failure. In contrast, glimepiride and pioglitazone are associated with weight gain. Glimepiride, a sulfonylurea, can stimulate insulin secretion, which may contribute to weight gain as it promotes increased insulin levels that facilitate fat storage. Pioglitazone, a thiazolidinedione, is also linked to weight gain due to fluid retention and adipocyte differentiation. Inhaled insulin, while effective for controlling blood glucose, can also lead to weight gain as it provides exogenous insulin directly. Insulin therapy typically promotes weight gain as it can increase appetite and leads to fat accumulation. Therefore, linagliptin stands out as the preferred option in this clinical scenario not only for its efficacy in managing blood glucose levels but also for its favorable effect on weight, making it suitable for an obese patient with diabetes and heart

6. Which drug is useful in treating sinus bradycardia?

A. Atropine

B. Cisatracurium

C. Neostigmine

D. Succinylcholine

Atropine is effective in treating sinus bradycardia due to its role as an anticholinergic agent that inhibits the action of the vagus nerve on the heart. The vagus nerve releases acetylcholine, which slows down the heart rate. By blocking this action, atropine reduces vagal tone, resulting in an increased heart rate. This mechanism makes atropine particularly valuable in emergency situations where a rapid increase in heart rate is necessary to restore adequate circulation. The other medications listed do not have a direct role in managing bradycardia. Cisatracurium, for instance, is a neuromuscular blocker used mainly during anesthesia to facilitate intubation and muscle relaxation but does not influence heart rate. Neostigmine is a cholinesterase inhibitor that increases acetylcholine levels, which could worsen bradycardia rather than alleviate it. Succinylcholine is also a neuromuscular blocker used primarily for rapid sequence intubation and does not have effects on heart rate. Therefore, atropine is the clear choice for addressing sinus bradycardia effectively.

7. For a patient diagnosed with gastroesophageal reflux disease, which medication is most appropriate?

A. An antacid such as aluminum hydroxide

B. Dicyclomine

C. Granisetron

D. Esomeprazole

In the context of gastroesophageal reflux disease (GERD), the most appropriate medication is a proton pump inhibitor (PPI) like esomeprazole. This class of medications is specifically designed to reduce gastric acid production by inhibiting the proton pump in the stomach lining, thereby providing effective relief from the symptoms of GERD, such as heartburn and acid regurgitation. Esomeprazole not only alleviates symptoms but also promotes healing of the esophagus in cases of erosive esophagitis caused by acid reflux. Other options, while they may have roles in other contexts, do not address the underlying issue of acid overproduction in GERD. Antacids like aluminum hydroxide provide temporary relief by neutralizing existing stomach acid but do not prevent the production of acid over time, which is crucial for long-term management of GERD. Dicyclomine is an anticholinergic used primarily for irritable bowel syndrome and does not target the acid-related issues of GERD. Granisetron, a serotonin receptor antagonist, is used for the prevention of nausea and vomiting and is not relevant to the treatment of GERD. Therefore, esomeprazole stands out as the most effective choice for managing the

8. Which of the following best describes the mechanism of action of milrinone in heart failure?

A. Decreases intracellular calcium

B. Increases cardiac contractility

C. Decreases cAMP

D. Activates phosphodiesterase

Milrinone is a phosphodiesterase inhibitor that primarily works by increasing the levels of cyclic adenosine monophosphate (cAMP) within cardiac cells. The elevation of cAMP leads to enhanced cardiac contractility, which is a crucial aspect of milrinone's therapeutic effects in heart failure. When cAMP levels rise, this prompts a cascade of intracellular signals that culminate in increased calcium availability for myocardial contraction, thereby improving the heart's pumping efficiency. This mechanism of action is particularly beneficial in heart failure, where the heart's ability to contract and pump blood is compromised. By enhancing contractility, milrinone effectively helps to alleviate symptoms of heart failure and improves hemodynamic performance. Other options relate to processes that are not part of milrinone’s mechanism of action. Milrinone does not decrease intracellular calcium; instead, it facilitates a more efficient use of calcium in the contractile process. It also does not decrease cAMP; on the contrary, it increases cAMP levels, leading to improved cardiac function. Lastly, while milrinone does indeed inhibit phosphodiesterase, the more direct and relevant outcome in the context of heart failure treatment is the increase in cardiac contractility that arises from elevated cAMP levels.

9. What is the proposed mechanism of cardioprotection from low-dose aspirin?

A. Aspirin preferentially inhibits COX-2 to reduce thromboxane A2 levels.

B. Aspirin preferentially inhibits COX-1 to reduce thromboxane A2 levels.

C. Aspirin preferentially inhibits COX-2 to lower prostacyclin levels.

D. Aspirin preferentially inhibits COX-1 to lower prostacyclin levels.

Low-dose aspirin is known for its cardioprotective effects primarily due to its mechanism of action on the cyclooxygenase (COX) enzymes, particularly COX-1. At low doses, aspirin irreversibly inhibits COX-1 in platelets, which leads to a significant reduction in the production of thromboxane A2. Thromboxane A2 is a potent vasoconstrictor and promoter of platelet aggregation; thus, lowering its levels helps to prevent the formation of blood clots within the arteries. The cardioprotective mechanism of low-dose aspirin is particularly relevant in the context of preventing cardiovascular events such as heart attacks and strokes. By inhibiting COX-1, aspirin effectively decreases platelet aggregation, which is crucial in maintaining vascular patency and reducing the risk of thrombotic events that can arise from atherosclerosis. This mechanism is why the choice indicating that aspirin preferentially inhibits COX-1 to reduce thromboxane A2 levels is the most accurate in this context. Understanding the role of aspirin in modifying platelet function helps clarify its use in clinical settings for the prevention of cardiovascular complications.

10. What recommendation should be given to prevent phototoxicity when using fluoroquinolone therapy?

A. Use sunscreen and avoid excessive UV exposure

B. Take the medication at night

C. Take with food

D. Drink with 1 L of water daily

The recommendation to use sunscreen and avoid excessive UV exposure is crucial in preventing phototoxicity when using fluoroquinolone therapy. Fluoroquinolones, a class of antibiotics, have been associated with increased sensitivity to sunlight, which can lead to adverse skin reactions when patients are exposed to ultraviolet (UV) light. This heightened sensitivity may manifest as rashes, burns, or other dermatological reactions. By advising patients to apply sunscreen, particularly one with a high SPF, they can create a protective barrier against harmful UV rays, significantly reducing the chances of experiencing these solarium-induced side effects. Additionally, recommending that patients minimize sun exposure, particularly during peak UV radiation hours, contributes to preventing phototoxic reactions. Other options, while important in their respective contexts, do not provide direct benefits for managing or preventing phototoxicity. Taking the medication at night does not address the issue of UV exposure during the day. Taking it with food may enhance absorption or reduce gastrointestinal side effects but has no effect on phototoxicity. Drinking 1 liter of water daily is sensible for hydration but is unrelated to preventing reactions to sunlight. Therefore, the correct recommendation focuses on sun protection strategies to mitigate the risk of phototoxicity with fluoroquinolone therapy.