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QUESTION 1

Your patient is a 58-year-old male who presents with onset of severe substernal chest pain and shortness of breath. An ECG reveals an acute STEMI, and he is on his way to the cardiac catheterization suite for percutaneous coronary intervention. Which of the following drugs used in acute coronary syndromes treated with PCI must undergo oxidation by hepatic P450 enzymes to an active form?

- A. Clopidogrel
- B. Ticlopidine
- C. Eptifibatide
- D. Aspirin
- E. Warfarin

Correct Answer: A

Clopidogrel and ticlopidine are ADP receptor pathway inhibitors. The irreversible inhibition of the ADP-dependent pathway of platelet activation is thought to be the result of covalent modification and inactivation of the platelet P2Y ADP receptor. This receptor is coupled to the inhibition of adenylyl cyclase. Both drugs are prodrugs and undergo conversion to active metabolites in the liver. However, clopidogrel must undergo oxidation by hepatic P450 enzymes to its active form. This is significant because many drugs are metabolized the hepatic P450 enzymes, including statins, and clopidogrel may interact with these medications. Clopidogrel is a second-generation thienopyridine and ticlopidine is a first-generation thienopyridine. Both drugs are indicated in combination with aspirin to prevent stent thrombosis. Eptifibatide is a GPIIb-IIIa receptor antagonist that is used to treat unstable angina and non-ST segment elevation myocardial infarction. Eptifibatide is also used to reduce ischemic events in patients who are undergoing percutaneous coronary intervention. The drug is a synthetic peptide that directly antagonizes the GPIIb-IIIa receptor on the platelet. Aspirin is an antiplatelet drug that works by inhibition of synthesis of prostaglandins. Prostaglandin G2 is the result of a synthesis pathway that is activated by platelets and endothelial cells, and results in localized vasoconstriction and induction of platelet aggregation, as well as causing release of platelet granules. Warfarin is an anticoagulant that acts on vitamin K-dependent reactions in the coagulation pathway. Vitamin K is necessary for hepatic synthesis of coagulation factors II, VII, IX and X, protein C and protein S. Vitamin K-dependent carboxylation is necessary for induction of enzymatic activity of these coagulation factors. Take-home message: Clopidogrel, a second-generation thienopyridine ADP receptor pathway inhibitor, is indicated in combination with aspirin to prevent stent thrombosis in patients who undergo percutaneous coronary intervention after myocardial infarction. Clopidogrel is a prodrug that must undergo oxidation by hepatic P450 enzymes, and therefore may affect the activity of statins and other drugs dependent on the hepatic P450 enzymes.

QUESTION 2

Select the class of Anti-diabetic medication that works in the specified organ to prevent hyperglycemia. Select all that applies. Kidney (G)

- A. Sulfonylureas
- B. Alpha- Glucosidase Inhibitors
- C. DPP4 Inhibitors
- D. Glucagon-like peptide-1 receptor agonists



E. Thiazolidinediones

F. Biguanide

G. SGLT2 inhibitors

Correct Answer: G

SGLT2 inhibitors Sulfonyleureas work in beta cells in the pancreas that are still functioning to enhance insulin secretion. Alpha-Glucosidase Inhibitors stop -glucosidase enzymes in the small intestine and delay digestion and absorption of starch and disaccharides which lowers the levels of glucose after meals. DPP4 blocks the degradation of GLP-1, GIP, and a variety of other peptides, including brain natriuretic peptide. Glucagon-like peptide-1 receptor agonists work in various organs of the body. Glucagon-like peptide-1 receptor agonists enhance glucose homeostasis through: (i) stimulation of insulin secretion; (ii) inhibition of glucagon secretion; (iii) direct and indirect suppression of endogenous glucose production; (iv) suppression of appetite; (v) enhanced insulin sensitivity secondary to weight loss; (vi) delayed gastric emptying, resulting in decreased postprandial hyperglycaemia. Thiazolidinediones are the only true insulinsensitising agents, exerting their effects in skeletal and cardiac muscle, liver, and adipose tissue. It ameliorates insulin resistance, decreases visceral fat. Biguanides work in liver, muscle, adipose tissue via activation of AMP-activated protein kinase (AMPK) reduce hepatic glucose production. SGLT2 inhibitors work in the kidneys to inhibit sodium-glucose transport proteins to reabsorb glucose into the blood from muscle cells; overall this helps to improve insulin release from the beta cells of the pancreas.

Reference: <https://doi.org/10.1093/eurheartj/ehv239>

QUESTION 3

TM is a 78 YOW with a history of hypertension, hypercholesterolemia and arthritis was admitted for proximal arterial fibrillation.

While in the hospital she was placed on diltiazem drip and eventually, converted to oral diltiazem 240mg. Pt's home medication includes Simvastatin 40mg po daily , hydrochlorothiazide 25mg po daily , Lisinopril 20mg daily and Acetaminophen. Her LDL-C is 100mg /dL.

What would be the most appropriate change to make on her therapy?

- A. Increase Simvastatin to 80mg po daily
- B. Keep Simvastatin at 40mg po daily
- C. Change Simvastatin 40mg to Atorvastatin 40mg po daily
- D. Change Simvastatin to Lovastatin 20mg po daily
- E. Discontinue Statins.

Correct Answer: C

Diltiazem has a major drug interaction with Simvastatin. Diltiazem is a CYP3A4 inhibitor, and since Simvastatin is metabolized by CYP3A4, its level can build up and the risk of myopathy increases. It is recommended to switch to a non- CYP3A inhibitor such as Pitavastatin, Pravastatin, or Rosuvastatin, and if Simvastatin is to be kept on it should not exceed 10 mg/day. The same interaction also exists with lovastatin, and the recommendation is to not exceed a total dose of 20 mg/day po of Lovastatin. Given the current options, the best choice is to change to Atorvastatin 40 mg po daily.

Reference: <http://www.fda.gov/Drugs/DrugSafety/ucm256581.htm>



http://circ.ahajournals.org/content/129/25_suppl_2/S1

QUESTION 4

A 22-year-old woman adopted a cat. Shortly thereafter, she developed itchy eyes and persistent rhinorrhea. She was clearly allergic to the pet, but desperately wanted to keep it. She tried taking diphenhydramine, but it had intolerable side effects.

Which of the following is a common effect of this type of medication?

- A. Decreased intraocular pressure
- B. Bradycardia
- C. Xerostomia
- D. Diarrhea
- E. Excessive sweating

Correct Answer: C

Diphenhydramine possesses anticholinergic properties. Xerostomia, or dry mouth, is a common side effect of anti-cholinergic medications, due to anti-muscarinic, parasympatholytic effects. Other adverse reactions may include: ?Mydriasis with blurred vision, photophobia ?Urinary retention ?Constipation ?Anhidrosis ? Hyperthermia ?Tachycardia ?Altered mental status A commonly referenced mnemonic for anti-cholinergic toxicity is "mad as a hatter, red as a beet, dry as a bone, hot as a hare, blind as a bat" to reflect confusion, flushing, dry mouth, hyperthermia and mydriasis, respectively.

QUESTION 5

Which of the following beta-blocker is NOT proven to reduce mortality in patients with Systolic CHF?

- A. Bisoprolol
- B. Nadolol
- C. Carvedilol
- D. Metoprolol succinate
- E. Metoprolol Tartrate

Correct Answer: E

Nadolol is not proven to reduce mortality in patients with systolic CHF. The efficacy of nadolol in HF has not been determined. For patients taking nadolol, it should be used with caution in those with compensated heart failure and patients should be monitored for a worsening of the condition. Bisoprolol, carvedilol, and sustained-release metoprolol succinate are the beta-blockers that have been proven to reduce mortality in patients with systolic CHF. These 3 beta-blockers have been effective in reducing the risk of death in patients with chronic HFrEF. Other beta-blockers were found to be less effective. Bucindolol did not exhibit uniform effectiveness across different populations. Metoprolol tartrate was found to be less effective in HF clinical trials.



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