



# Drugs for hyperlipidemia

- SUMMARY. (Slide 2)
- MCQs. (Slide 3 and 4)
- SAQ. (Slide 5)

أبدا لم يفت الاوان لكي تبدأ

| Target                    | Class  | Drug   | Action  | ADRs  | Effect (%change)  | Notes   |
|---------------------------|--|--|---|---|---|---|
| Exogenous<br>Cholesterol  | Bile Acid<br>sequestrants                        | Cholestyramine<br>Colestipol<br>Colesevelam                  | Form an insoluble complex with the bile acids and salts, preventing their reabsorption from the intestine and disrupting the enterohepatic circulation of bile acids. | GI distress, constipation, decreased absorption of other drugs (Statins, Ezetimibe Chlothiazides, Digoxin, Warfarin) Colesevelam does not interfere with absorption of other drugs  | ↓ LDL ↑ HDL No change in triglycerides                    | Contraindications:  1- Complete biliary obstruction  2- Chronic constipation  3-Severe hypertriglyceridemia (TG >400 mg/dL) |
|                           | Cholesterol absorption inhibitor                 | Ezetimibe  | Ezetimibe reduces C absorption. Therefore, ezetimibe reduces the flux of C from the intestine to the liver.   | Not common  GIT disturbance, headache, fatigue, artheralgia & myalgia   | ↓ LDL( 14-18),<br>↑ HDL (1-3)<br>↓Triglyceride (2)        | Monotherapy: primary prevention of low risk CHD.  Combination therapy: safe: with statins or fibrates                       |
| endogenous<br>cholesterol | Statins<br>(HMG-Co A<br>Reductase<br>Inhibitors) | Simvastatin Lovastatin Atorvastatin Pravastatin Rosuvastatin | potent competitive inhibitors of HMG-CoA reductase  | Common: Headache, myalgia, fatigue, GI intolerance, and flu-like symptoms Hepatotoxicity, Teratogenicity. Muscle aches, or weakness associated with an elevation of creatine kinase (CK) released from muscles, are the best indicator of statin-induced myopathy | ↓LDL 18-55%  ↑ HDL 5-10%  ↓TG & VLDL 10-30%               | It is important to check CK & liver enzymes regularly upon administration of statins.                                       |
|                           | Niacin<br>(Nicotinic<br>Acid)                    |  | In adipose tissue: it binds to adipose nicotinic acid receptors In liver:inhibits hepatocyte diacylglycerol acyltransferase-2 In plasma: it increase LPL activity     | Flushing (+aspirin) Hyperglycemia, Hyperuricemia, GI distress, hepatotoxicity   | ↓LDL (15-30),<br>↑HDL (15-35)↓<br>Triglyceride (20-50)    | Contra-indications Gout – Peptic ulcer – Hepatotoxicity – Diabetes mellitus   |
|                           | Fibrates   | Clofibrate<br>Fenofibrate<br>Gemfibrozil                     | They increase gene transcription for lipoprotein lipase (LPL) leading to increased catabolism of TG in VLDL and chylomicrons  | Dyspepsia, gallstones, myopathy   | ↓LDL (5-20), ↑HDL<br>(10-20)<br>↓Triglyceride (20-<br>50) | Pregnant or nursing women ,Renal impairment,Gall-bladder disease.In alcoholics  |

#### Online Quiz ..



✓ <a href="https://www.onlineexambuilder.com/pharmacology-anti-hyperlipidemia/exam-144715">https://www.onlineexambuilder.com/pharmacology-anti-hyperlipidemia/exam-144715</a>

**Q**:**b** 

Answers

|   |  |  |   | liver via the enterohepatic circulation?   |                |
|---|--|--|---|--|----------------|
| A) Niacin.  | B) Fenofibrate.  | C) Cholestyramine.   | D) Fluvastatin.   |  |                |
| would you give to her to A) Stop taking the levothyro B) Take levothyroxine 1 hou C) Switch cholestyramine to | avoid a drug interaction keepsine as it can interact with cour before cholestyramine on o colestipol as this will elimin | between her cholestyramine a<br>cholestyramine.<br>an empty stomach. |   | styramine and levothyroxine (thyroid hormone   | ). What advice |
| Q3: Which of the following A) Patients with diabetes  |  | more likely to experience myants with renal insufficiency.           | algia (muscle pain) or myor<br>C) Patients with gout.         | pathy with use of HMG CoA reductase inhibitors  D) Patients with hypertriglyceridemia.   | s?             |
| Q4: Which one of the fol<br>A) Fenofibrate.   | lowing drugs decreases cl<br>B) Niacin.  | nolesterol synthesis by inhibit<br>C) Cholestyramine.                | ing the enzyme 3-hydroxy-<br>D) Lovastatin.                   | -3- methylglutaryl coenzyme A reductase?   |                |
| lushing and itchy feeling   | that he thinks is related to minutes prior to taking niacir  | the niacin. Which of the follown.  B) Ad                             | ving options can help him m<br>minister aspirin 30 minutes af | ides and low HDL levels. He is complaining of an unanage this adverse effect of niacin therapy? fter taking niacin. iacin to immediate-release niacin. | uncomfortable  |
| •   | •  | lipidemia with Pravastatin for choices is the best option?           | the past 6 months Her pl                                      | hysician wishes to add an additional agent to bl   | ock absorption |
| A) Niacin.  | B) Colesevelam.  | C) Gemfibrozil.  | D) Ezetimibe.   |  | <b>Q:9</b>     |

### Cont.... MCQs

| Q7: Patient comes into the ER wi have caused his gall stones?     | th gallstones, after furthe                            | er investigations You find ou | it that he is on drugs to treat his hype  | erlipidemia. Which of the following | drugs could           |
|---|--|-------------------------------|---|-------------------------------------|-----------------------|
| A) Niacin.  | B) Fenofibrate   | C) Ezetimibe                  | D) Colesevelam  |                                     |                       |
| •   | olesterol about 28 mg/dl                               | His physician wishes to b     | mia. His most recent lipid panel reveal egin treatment to increase his HDL clas?  |                                     |                       |
| A) Colesevelam.   | B) Niacin.   | C) Simvastatin.               | D) Ezetimibe.   |                                     |                       |
| Q9: Patient with diabetes has hypothese A) Niacin.  B) Statins.   | perlipidemia, Which drug<br>C) Colestipol. D) Ezeti    |                               | e used in his case due the risk of deve   | elopment of Hyperglycemia?          |                       |
| Q10: Patient with hypercholester creatine kinase. Which drugs did |  | tion of two anti hyperlipide  | mic drugs, After 4 days the patient co  | omplaining of sever myalagia and i  | ncrease in            |
| A) Cholestyramine & Lovastatin                                    | . B) Fenofibr  | rate & Ezetimibe.             | C) Lovastatin & Fenofibrate.  | D) niacin & Ezetimibe.              |                       |
| vitamins D and E supplement, W                                    | hat is the most likely drug                            |                               | ncrease in LDL, The doctor prescribes   | s one of the an4 hyperlipidemia dru | ugs with              |
| A) Statins.   | B) Colestipol.   | C) Ezetimibe.                 | D) Nicotinic acid   |                                     |                       |
| •   | cholesterol level of 131 m                             | •••                           | agent for the past 6 months. He also has a gent for the past 6 months. He also has a general section of 510 mg/dL, and there is respectively. | •                                   | 13:B<br>12:C          |
| A) Ezetimibe.   | B) Niacin.   | C) Colestipol.                | D) Gemfibrozil.   |                                     | 11:B                  |
| Q13: Which one of the following A) Elevated blood pressure.       | is the most common side  B. Gastrointestinal disturbar | **                            |   |                                     | Answers<br>8:8<br>A:9 |
| 4   |  |                               |   |                                     |                       |



A 72-year-old male who is treated for hyperlipidemia with high-dose atorvastatin for the past 6 months. He also has a history of renal insufficiency. His most recent lipid panel shows an LDL cholesterol level of 131 mg/dL, triglycerides of 510 mg/dL, and HDL cholesterol of 30 mg/dL.

#### Q1: What is the mechanism of action of Atorvastatin?

t is an example of Statins which are potent competitive inhibitors of (HMG-CoA) reductase which is an important enzyme in cholesterol synthesis in the liver

Q2:Is there anything special about Atorvastatin?
Yes, it has long half-life (14 hours) so we can take it at any time of the night.

Q3: What are the most serious side effect of statin group, that we have to be aware of them by frequent lab investigation?

Q4: If his physician wishes to add an additional agent for his hyperlipidemia. Which of choices is the best option in his case? why?

#### Q5: list three of contraindication for this drug ^^?

1-Gout. 2-Peptic ulcer..

3-Diabetes mellitus.

Q6: why we can not use any drug from Resins group in this case?

Because he has high level of TG and The bile acid binding resins can raise triglycerides more and more which will worse his situation.

#### Q7: why we can not use any drug from Fibrate group in this case?

He has a history of renal insufficiency so we can not combine the fibrate with statin because the incidence of myopathy may increase, he also may develop Rhabdomyolysis.









## Editing file

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