clinical cases DDIs

Date of report 07 Sep 2022

Reported case interaction between **Cobicistat** and **Atorvastatin**

Drugs suspected to be involved in the DDI

Perpetrator
Cobicistat

Daily Dose
150 (mg)

Dose adjustment performed
No

Administration Route
Oral

Start date
Unknown

End date
Jan. 1, 2022

Victim
Atorvastatin

Daily Dose
40 (mg)

Dose adjustment performed
No

Administration Route
Oral

Start date
Unknown

End date
Ongoing

Complete list of drugs taken by the patient

Antiretroviral treatment

Darunavir/Cobicistat/Emtricitabine/Tenofovir-AF

Complete list of all comedications taken by the patient, included that involved in the DDI

Atorvastatin 40 mg qd

Clinical case description

Gender

Male

Age

59

eGFR (mL/min)

>60

Liver function impairment

No

Description

59-year-old male with long-term HIV infection. Always on cART containing boosted protease inhibitors. No virologic failures. Currently on Darunavir/cobicistat/FTC/TAF. Last viral load determination <20 copies/mL; CD4+ T cell count 53 cells/mm3. Heavy smoker, hypercholesterolemia. 10-year risk of MI or death 15% (Framingham Risk Score). On treatment with atorvastatin 40 mg qd. NO evidence of statin-associated toxicity (liver enzyme levels and CK within normal range). Lipid levels above the target (LDL 115 mg/dL; target <70 mg/dL). Darunavir/cobicistat/FTC/TAF was changed to Bictegravir/FTC/TAF. Three months after the change viral load continued <20 copies/mL, LDL-c levels decreased to 55 mg/dL. This is an example on how ritonavir and cobicistat may interfere with statins mechanism of action, limiting their efficacy.

Clinical Outcome

Loss of efficacy

Drug Interaction Probability Scale (DIPS)

Score

7 - Probable

Editorial Comment

Atorvastatin is a substrate of the hepatic transporter OATP1B1 which factiliates its entry in the liver, the site of action and elimination. Darunavir/cobicistat inhibits OATP1B1 and therefore can limit the entry of atorvastatin in the liver which may impait its lipid lowering effect. An analysis of the Swiss HIV Cohort Study (Courlet P et al. J Antimicrob Chemother 2020) reported indeed that insufficient lipid control was observed with boosted protease inhibitors despite high atorvastatin concentrations likely explained by the inhibition of OATP1B1 but also their less favorable effect on lipid. Target lipid values were more often achieved with unboosted integrase inhibitors due to both their favourable DDI profiles and neutral effect on lipids. The authors conclude that integrase inhibitor-based regimens should be favoured in patients with refractory dyslipidaemia.

University of Liverpool Recommendation

■ Potential interaction - may require close monitoring, alteration of drug dosage or timing of administration

For more information click here

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