

A Case of Pancreatic Enzyme Elevation Due to Use of Statins

Statin Kullanımına Bağlı Pankreatik Enzim Yükselmesi Olgusu

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ABSTRACT

It is well known that statin usage is linked with side effects such as myalgias, hepatic enzyme elevation and rhabdomyolysis. Pancreatic injury and lipase elevation may be a rare side effect of statins. In this case report, we present a 61-year-old male with the history of cerebrovascular disease (CVD) and prostate cancer which experienced elevation of lipase levels after use of statins. We tried different types of statins for the patients condition of pancreatic injury and in this case report, the possible reasons of statin induced pancreatic injury are underlined.

Keywords: Pancreas, Statin, Lipase

ÖZET

Statin kullanımıyla birlikte kas ağrıları, karaciğer enzim yüksekliği ve rhabdomyoliz görülebilmektedir. Pankreas hasarı ve buna bağlı lipaz yüksekliği statinin nadir görülen ciddi istenmeyen etkilerindedir. Bu vaka sunumunda, statin kullanımından sonra lipaz seviyelerinde yükselme görülen, serebrovasküler hastalık ve prostat kanseri öyküsü bulunan 61 yaşında bir erkek hastayı değerlendirdik. Bu hastada olası pankreatik hasar görülen hastada farklı statin gruplarını denedik ve statin kullanımına bağlı olası pankreas hasar mekanizmalarını ele aldık.

Anahtar Kelimeler: Pankreas, Lipaz, Statin

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INTRODUCTION

Statins are the most commonly prescribed cholesterol-lowering agents on the market; they are inhibitors of the HMGCoA reductase enzyme and that gives them direct and efficient pharmaceutical features on the management of dyslipidemia. Statin-induced serum amylase and lipase increase is an unusual condition. We report a 61-year-old male whose laboratory results indicating serum amylase and lipase increase with statin use; that was resolved upon rearranging the prescription with different types of statins and it recurred after re-administration of the same prescription.

CASE REPORT

We present a case of a 61-year-old male with a history of stroke and prostate cancer. He came to our clinic with a complaint of high blood pressure and dyslipidemia. He denied alcohol consumption and smoking. The blood pressure value was 155/94 mmHg and pulse rate was 72 bpm and rhythmic. Fasting blood glucose level was 99mg/dl, HbA1c ratio was %6.1 and LDL was 102mg/dl. Initial prescription included: atorvastatin 40mg 1x1, valsartan/ hydrochlorothiazide 80 /12.5 mg 1x1, clopidogrel 100 mg 1x1 and tamsulosin 0,4mg 1x1.

After three months of drug usage, a blood test was carried out. The serum lipase level was found 109 mg/dl. (normal range 0-60 mg/dl.) The other biochemical blood levels were found in normal ranges. The drug was changed from atorvastatin 40mg 1x1 to rosuvastatin 10 mg 1x1 and after a month the laboratory tests showed a significant increase in lipase levels. Physical examination findings of the patient were normal. Signs such as nausea, vomiting and abdominal pain associated with acute pancreatitis were not detected. According to laboratory data, serum lipase was 476,1 mg/dl that lead to a shift to atorvastatin 10 mg 1x1 for approximately one month. The results were indicating increased LDL levels (140mg/dl) because the patient had a history of stroke, the recommended target of LDL levels in guidelines are below 70 mg/dl and for this reason, the prescription changed to pravastatin 40 mg 1x1. The increase in the lipase levels showed recurrence, and it turns back to normal levels within a month but the LDL levels were detected 151 mg/dl. For this reason, pitavastatin 2 mg 1x1 started and after a month with pitavastatin 2mg usage, the level of LDL was 119 mg/dl and the lipase level was 76 mg/dl.

DISCUSSION

Drug-induced acute pancreatitis(AP) is seen as a rare condition among the other causes of AP. Determination of drug-induced pancreatic enzyme increase based on clinical suspicion and detailed investigation on drug history. An increase in serum amylase and lipase with drug induction is unusual but the incidence may be increasing. Chintanabonia et. al.reported a case report on drug-induced AP and according to the study serum lipase levels show more specificity compared to serum amylase levels and have higher diagnostic value (1). There is no significant drug-drug interaction between the agents listed on the initial prescription besides they were usual combinations used in the conditions include cerebrovascular diseases and dyslipidemia. Nevertheless, two case reports described the occurrence of AP in patients treated with combined salicylates and statins, which are commonly prescribed drugs for coronary artery disease (2,3).

The exact mechanism of the increase in serum amylase and lipase levels with the statin induction is still unknown, but there are a few case reports that attribute it to CYP3A4 metabolism (4). This was considered because results showed no relapse with pravastatin in which metabolism occurs by glucuronidation reactions with very minimal intervention of CYP3A4 enzyme. Also, Deshpande PR et al. reported a case report on atorvastatin-induced AP which resolved after drug withdrawal and it was stated even with atorvastatin monotherapy an increase in pancreatic enzymes may occur (5). It has been mentioned that statin-induced AP may occur with the use of different types of statins as seen in our case (6). This could prove that pancreatic injury caused by statins may be a class effect of statins which means it could be seen with the use of a different type of statins.

The possible mechanisms for statin-induced AP may include direct toxic effects to the pancreas and accumulation of a toxic metabolite. The preclinical studies demonstrated statins may exhibit antitumor effects in pancreatic cancer cell lines in vitro and animal models in vivo through cell cycle arrest. Statins are HMG-CoA analogs and they interfere with protein synthesis by inhibiting mevalonate and pathways that are involved in cell growth, proliferation, survival of the pancreatic cells and this mechanism may lead to the injury of pancreatic cells (7).

The precise prediction could not be made for the statin use and occurrence time of the AP symptoms which is also mentioned in the case study of Chintanabonia et al. on statin-induced AP, there was no consistent latency period for statin-associated AP as noted in previous case reports (1).

CONCLUSION

Drug-induced pancreatic enzyme elevation is a condition that should be strongly considered regardless of the duration of the statin therapy when other likely causes had been ruled out. In our case different with a different type of statin, the possible pancreatic injury continued so it is thought that statin-induced pancreatic injury may be a class effect. Rearrangement of the prescription is an effective procedure for preventing further relapses.

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