

Asian Journal of Pharmaceutical and Health Sciences

www.ajphs.com



Statin induced temporary accommodation inertia - a report

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ARTICLE HISTORY

Received: 03.11.2012

Accepted: 03.12.2012

Available online: 10.02.2013

Keywords:

Atorvastatin, Accommodation inertia, Dyslipidemia.

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ABSTRACT

Statin medications are mainstay of treatment for patients with dyslipidemia and coronary artery diseases. Statin has rarely some adverse effects like rhabdomyolysis and myositis. There are several reported cases of eye muscle weakness or paralysis leading to diplopia, ophthalmoplegia and blepheroptosis. But no literature reported any cases of transient accommodation inertia. This study reported one case of atorvastatin induced transient accommodation inertia leading to temporary blurring of vision. It was found from this study that Not only atorvastatin has rare side effects like myositis, rhabdomyolysis, external ophthalmoplegia, but it can cause transient ciliary muscle paralysis.

INTRODUCTION

tatin medications are mainstay of treatment for patient with dyslipidemia and coronary artery diseases. Statins or HMG-CoA reductase inhibitors (competitive) are effective in blocking hepatic synthesis of cholesterol. Rarely some severe adverse effects occur like rhabdomyolysis, and milder one related to myopathy. Exercise exacerbates these side effects, probably due to inflammation [1]. In the Journal Ophthalmology 115 (12) April 2000, Fraunfalder and Richards reported 256 eye conditions related to muscle balance that have been associated with the use of statin drugs. The conditions reported were diplopia (double vision), blepheroptosis (ptosis of drooping eyelid) or ophthalmoplegia (weakness or paralysis of the muscles controlling one or both eyes) [2]. According to WHO criteria also, the relationship between statin therapy and diplopia, ptosis, or ophthalmoplegia is possible and the Mechanism by which they may occur is either myositis of the extraocular muscles, the levator palpebrae superioris muscles, or both. Minor increases in creatine kinase (CK) activity in plasma are observed in some patients receiving reductase inhibitors, frequently associated with heavy physical activity. Rarely, patients may have marked elevations in CK activity, often accompanied by generalized discomfort or weakness in skeletal muscles. If the drug is not discontinued, myoglobinuria can occur, leading to renal injury. Myopathy may occur with monotherapy, but there is an increased incidence in patients also receiving certain other

drugs. Genetic variation in an anion transporter (OATP1B1) is associated with severe myopathy and rhabdomyolysis induced by statins. [3] But we have not found any report of temporary accommodation inertia leading to transient reversible blurring of vision after extensive literature review. In this respect this article is unique.

CASE REPORT

A 38 year male, Clarke by occupation, presented with chronic headache and vertigo. His blood pressure and pulse were 168/90 mmHg and 88/minute respectively and other systemic examinations were within normal limit. His blood biochemistry reports showed total cholesterol, triglyceride, LDL and HDL levels were 286 mg/dl, 296 mg/dl, 150 mg/dl and 45 mg/dl respectively. He had no significant past history. He was started on Tabs Amlodipine 2.5 mg and Tab Atorvastatin 10 mg once daily. After about 7 10 days of taking the treatment the patient noticed transient blurring of vision in the initial phase of reading or looking at near objects. He consulted an Ophthalmologist for the problem. His ophthalmological examination was within normal limit except temporary accommodation inertia persistent for 2 seconds only and then there was clearing of vision [4]. On examination the patient had vision 6/6 (N6) in both eyes. NPA (near Point Accomodation) 14 centimeter, AA (Accomodation Amplitude) 7D, no ptosis, no opacity found in lens. Intra-ocular pressure was within normal limit. Patient had no history of Diabetes, Anemia, ocular trauma and intraocular surgery. Patient

does not take any antidepressant, antipsychotic, antihistaminic, antianxiety, antispasmodic and diuretic medicines. He is nonalcoholic and had no history of attack of CVA and any other debilitating illness. After about one week of treatment patient suddenly developed diarrhea and stopped taking both the drugs just to notice with surprise that he could see near objects clearly. He was then advised to restart the drugs. He had recurrence of similar symptoms. As per medical advice, then he first stopped tab Amlodipine just to see that temporary blurring continues. Then he stopped taking tab Atorvastatin and continued tab Amlodipine with disappearance of such symptom after about 5 days of stoppage. He was then advised to consult a neurologist. Neurological examination was within normal limit. Anti Acetylcholine Receptor Antibody was absent in his blood, Hb 125 gm/L, Fasting Blood glucose level 4.3 mmol/L, Post Prandial Blood glucose 5.4 mmol/L, TSH level 2.2 IU/L, free T4 1.1 ng/ml, and serum CK 170 IU/L (high normal) were all within normal range. He was then advised to start Rosuvastatin 10 mg at night time and had recurrence of same symptoms. He then stopped taking that and was advised to start with Fenofibrate 200 mg and gradually had disappearance of that symptom.

RESULT AND DISCUSSION

Clinically the patient was absolutely normal except high blood pressure and transient accommodation inertia. We have excluded all the factors which can affect eye muscles. We have found all those parameters are within normal limit. Only transient ciliary muscle palsy has lead to temporary accommodation inertia. It was additionally found that with the stoppage of Atorvasatin, patient was relieved of the symptom within 5 days time.

CONCLUSION

There are reported cases of atorvastatin induced diplopia, ophthalmoplegia and ptosis. But atorvastatin induced temporary ciliary muscle palsy is yet to be reported. If atorvastatin causes such problem, it should be stopped immediately. This is also true for other drugs in the same group for the particular patient. So such patient should be advised with hypolipidemic drug from a different group.

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