Case Report

Acute renal failure secondary to rosuvastatin-induced rhabdomyolysis

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ABSTRACT

Rhabdomyolysis is a condition characterized by muscle necrosis and can range from asymptomatic elevation of serum muscle enzymes to life-threatening cases associated with extremely elevated enzyme levels, electrolyte imbalances, and acute renal failure (ARF). Here, we present the case of a 76-year-old woman who developed rhabdomyolysis and ARF secondary to an increase in her rosuvastatin from 10 to 40 mg. In this report, we present a case of a 76-year-old woman who presented with decreased appetite and reduced urine output, presumed to be due to a recent increase in her rosuvastatin dosage leading to rhabdomyolysis.

Key words: Acute renal failure, Creatinine kinase, Rhabdomyolysis, Rosuvastatin

The incidence of rhabdomyolysis is 1.6/100,000 person-years. The rate of statin-induced rhabdomyolysis is 0.3–13.5 cases/1,000,000 statin prescriptions as per the US Food and Drug Adverse Reporting System database [1].

As statin-induced rhabdomyolysis is rare, we would like to present this case of acute renal failure (ARF) caused by rosuvastatin-induced rhabdomyolysis.

CASE REPORT

A 76-year-old female presented to the emergency with decreased appetite, lethargy, slight confusion, and reduced urine output.

On initial assessment, her pulse rate was 90 beats/min, Respiratory rate of 18/min, temperature 36.5 C, blood pressure 101/65 mmHg, and a Glasgow Coma Scale of 15. There was no edema or paroxysmal nocturnal dyspnea.

She had a past medical history of hypertension, chronic heart failure, dyslipidemia, diabetes type 2, renal stones treated with lithotripsy, developed ARF secondary to rhabdomyolysis and was admitted to intensive care unit (ICU). Due to a history of heart failure, the patient was seen by a cardiologist 1 month before her admission, and her rosuvastatin was increased from 10 to 40 mg. Ten days prior to her presentation, she had decreased appetite and reduced urine output, which became progressively worse. She did have a fall, which reduced her mobility.

On presentation to the emergency department, she was anuric, confused, and found to be in ARF. Her creatinine was 1,588, urea was 77.8, potassium was 8, pH was 7, and creatine kinase (CK) was 20,894. Her computed tomography kidney, ureter, bladder and head were normal. She was transferred to the ICU and had an echocardiogram, which was unremarkable. Rosuvastatin was stopped immediately, and nephrology was consulted, who started a trial of intravenous (IV) plasmalyte. Her CK came down to 10,000 after a few days, and her ARF resolved well. Subsequently, her CK started rising again and peaked at 93,000, but she remained asymptomatic. Rheumatology reviewed her and started prednisolone with a presumed diagnosis of inflammatory myositis. Her CK came down within a few days, she was mobile and discharged home.

She had electromyography studies and a myositis screen. A rheumatology follow-up was done, and they ruled out inflammatory or necrotizing myositis. As per the rheumatologist, her condition was in keeping with rhabdomyolysis due to the high dose of statins. Her reduced intake, reduced mobility, and dehydration may have contributed to statin-induced rhabdomyolysis, which in turn caused ARF.

As she was treated with steroids, her blood glucose level did go up, so her medication was adjusted. Once her steroids were tapered off and stopped, her diabetic control improved.

She was followed by general internal medicine as an outpatient for a few months. As all her markers were normal, including CK, renal profile, and she was back to her baseline mobility and physical activity, so she was discharged from medicine and rheumatology.

CASE DISCUSSION

Rhabdomyolysis is a syndrome caused by muscle necrosis and leakage of muscle contents in systemic circulation. It has a wide

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range of manifestations ranging from asymptomatic elevation of muscle enzymes to serious consequences associated with high enzyme levels, electrolyte imbalance, and ARF. Clinical presentation depends on the severity of illness, which may include muscle pain and weakness and tea-colored urine [1].

The causes of rhabdomyolysis include infections, metabolic and electrolyte disorders, drugs, toxins, or physical factors such as trauma, ischemia, hypoxia, and immobilization. The risk of statin-induced rhabdomyolysis can be increased by several factors, i.e., hepatic or renal impairment, hypothyroidism, diabetes mellitus, or use of concurrent myotoxic medications [2,3].

Statins are HMG-CoA reductase inhibitors and thereby reduce cholesterol synthesis. They are widely used for primary and secondary prevention of coronary, cerebral, and peripheral ischemic vascular events. Although mostly well tolerated, myotoxicity is a rare but well-documented adverse effect of all statins is dose-dependent. In any clinical case of statin-induced myotoxicity, we can find multifactorial causation and risk factors can be modifiable (statin dose-related, renal/hepatic impairment and tissue hypoxia, use of other drugs which can exacerbate statin toxicity) versus non-modifiable (Asian ethnicity, female sex, low body mass index, elderly and pharmacogenetic risk factors) [4].

Canzonieri *et al*. have mentioned a report of an 82-year-old patient presenting with asthenia, muscle pain and tenderness, and decreased muscle strength. Their patient had been started on a high dose of atorvastatin 3 weeks prior due to acute myocardial infarction. His CK was elevated on admission, and a diagnosis of rhabdomyolysis was made. His CK and liver function tests (LFT) continued to rise despite the suspension of statin. Saline infusion was started, and then immunosuppression with methylprednisolone was initiated, after which the patient's lab markers and symptoms improved [5]. The standard care of rhabdomyolysis includes urine alkalinization, aggressive IV hydration, and short-term renal replacement therapy [1].

The therapeutic management of rhabdomyolysis includes supportive management and prevention of complications at the first level and the second level includes finding out and removal of potential causative agents and precipitating factors [4].

Ezad *et al*. have mentioned a case of statin-induced rhabdomyolysis with a 67-year-old patient presenting with myalgia and weakness in the upper and lower limbs resulting in reduced mobility on a background of chronic obstructive pulmonary disease and ischemic heart disease. His initial labs showed raised CK and LFTs. He was taking simvastatin 80 mg and had been started on clarithromycin prophylaxis a few days before admission. A diagnosis of statin-induced rhabdomyolysis was made and IV fluids were started with suspension of both statin and clarithromycin. He developed acute kidney injury with worsening renal function, LFT, and CK. However, he eventually responded to medical management, and renal replacement therapy was not required [6].

CONCLUSION

As we are prescribing more and more statins every day, we should look at the potential risk factor of statin-induced myopathy in the clinical setting of other risk factors. In our case, the patient had multiple risk factors i.e., female sex, elderly, diabetic, reduced mobility, and dehydration due to reduced oral intake. The precipitating factor in this case was a recent increase in rosuvastatin dose from 10 to 40 mg. Consequently, the patient went into ARF secondary to rhabdomyolysis. She had supportive treatment in intensive therapy unit, i.e., IV hydration, and the offending drug was stopped, which prevented the need for dialysis. Within a few days, she was discharged home with fenofibrate, which she is tolerating well.

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