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A Rare Cause of Acute Kidney Injury in a Patient with Acute Coronary Syndrome

ABSTRACT

Acute kidney injury (AKI) is a common complication after any percutaneous coronary intervention (PCI), especially in high-risk groups. We describe a rare cause of AKI due to rhabdomyolysis in a patient who underwent a primary PCI for acute myocardial infarction and started on high dose of statins. Patient developed gradual onset weakness and pain of all four limbs and decreased urine output after 4 weeks of starting rosuvastatin requiring dialysis. He had a significant elevation of CPK and positive urinary myoglobin confirming the diagnosis of pigment nephropathy. Rosuvastatin was discontinued and with cautious hydration AKI gradually recovered. A thorough history and review of medications can help in identifying the etiology of AKI. Early diagnosis and intervention in AKI can help improve outcomes.

Key words: Rhabdomyolysis, Pigment nephropathy, Acute kidney injury

INTRODUCTION

AKI is frequently encountered in high risk patients undergoing percutaneous coronary intervention. It is very important to identify the cause of AKI as it has a bearing on treatment options and prognostic outcomes. We describe a situation of statin induced rhabdomyolysis and pigment nephropathy causing AKI after PCI. It is a rare but life-threatening complication of using statins which requires a high index of suspicion. Early diagnosis and prompt treatment can have a favourable outcome in such patients.

CASE DESCRIPTION

A 72-year-old male with diabetes and hypothyroidism was admitted to our hospital with complaints of gradual onset severe weakness of all four limbs, reduced appetite, myalgia and reduced urine output for 1 week. He had undergone a primary angioplasty for myocardial infarction 1 month ago. His prescription included pantoprazole 40 mg, aspirin 75 mg, rosuvastatin 40 mg, thyroxine sodium 50 mcg, metformin 1 g, teneligliptin 20 mg, and Insulin. His creatinine was documented 1.3 mg/dl prior to percutaneous coronary intervention (PCI). Examination revealed dehydration, acidotic breathing, and altered sensorium. Motor examination revealed proximal myopathy and grade 1 power in all four limbs. His vitals were stable. Fundus examination did not reveal changes of diabetic retinopathy or cholesterol emboli (to rule out athero-embolic renal disease). Detailed investigations are listed in Table 1. Serum complements were normal. Creatine phosphokinase (CPK) was markedly elevated and urinary myoglobin levels were high, confirming diagnosis of pigment nephropathy.

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Patient required hemodialysis on admission. Rosuvastatin was stopped and with cautious hydration with isotonic saline and bicarbonate supplementation gradually acute kidney injury (AKI) improved. Patient became dialysis-free after 5 sessions. His serum levels of CPK showed gradual decline after discontinuation of rosuvastatin thereby confirming drug-induced rhabdomyolysis.

DISCUSSION

AKI is a common complication after any PCI, especially in high risk groups. Etiology of AKI can however differ in various patient groups. Common causes include contrast mediated nephrotoxicity, athero-embolic renal disease. Rosuvastatin-induced rhabdomyolysis is not common.[1] The incidence of rosuvastatin-induced rhabdomyolysis is 3/10,000.[2] So this condition could be a red herring in any patient who undergoes PCI. Rhabdomyolysis is a life-threatening condition that can be caused by any statin (muscle symptoms with elevated creatine kinase substantially >10 times the upper limit of normal, with renal impairment and myoglobinuria consistent with pigment

Table 1: Investigations

	On admission	Day 3	Day 6	Day 10	Day 13	1 Year
Hb/PCV/ESR	11/36/108					
BUN	172					
Creatinine	11.5	7.5	5.9	4.5	3.7	1.9
Na/K/Cl/HCO_3	121/7/85/12	132/5.5/110/22				
SGOT/SGPT	435/157		230/102		73/48	35/23
CPK total	5610	4690	1436	1090	670	250
Protein/Albumin	5.7/2.7					
Bilirubin	0.8					
Calcium	6.1					
Phosphorous	11.8					
Uric acid	10.8					
C3/C4 compliment	81/21					
SPEP	Negative					
T3/T4/TSH	1.9/0.6/17.9					
Urine Routine microscopy	Protein 2+ 4–5 PC/hpf 2–3 RBC/hpf Granular cast++					Protein 1+ Granular casts+
Urine Protein/creatinine ratio	3.2					
Urine myoglobin	18714					
USG KUB	Normal size and echo both kidneys					

SPEP: Serum protein electrophoresis

nephropathy).[3,4] Age, renal dysfunction, hypothyroidism, family history of hereditary muscular disorders, previous history of muscular toxicity with another statin or fibrate, grape-fruit juice consumption (more than 1 L/day), alcohol abuse, people of Chinese or Japanese descent, and combination therapy with fibrates are all factors that increase the risk of rosuvastatin-induced myopathy or rhabdomyolysis.[5] Our patient was elderly male with hypothyroidism and eGFR 54/mL/min/1.73 m^2 putting him in high-risk group for rhabdomyolysis. Hypothyroidism has been independently associated with rhabdomyolysis, hence statins should be used judiciously in them.[6] The specific mechanism of statin-induced rhabdomyolysis is uncertain, although the following explanations have been suggested: The first is a roadblock in cholesterol synthesis, which makes the membranes of skeletal muscle cells unstable due to low cholesterol levels.[4] Abnormal intracellular protein signaling due to prenylated protein abnormalities.[7] Finally, a lack of coenzyme Q10 results in aberrant mitochondrial respiratory performance.[8,9] Individuals with a known hypersensitivity to rosuvastatin, as well as those with active liver disease or unexplained chronic elevations in liver transaminases levels (>3 times the upper normal limit [ULN] on two consecutive visits), should avoid taking it. Liver transaminase levels should be tested at baseline, 12 weeks after commencing medication or increasing the dose, and then every 6 months after that. If liver transaminase levels surpass three times the ULN, the dosage should be lowered or the medication stopped. In patients with a history of liver illness or alcohol misuse, rosuvastatin should be administered with caution because it has the potential to raise liver transaminase levels.[1,10] Our patient also had elevated transaminases which normalized on discontinuation of rosuvastatin. Statin-induced myalgia is frequently encountered in practice but quadriparesis as a presenting feature of statin-induced rhabdomyolysis is a rare finding which was seen in our case.[11]

CONCLUSION

In conclusion, clinicians should be aware of the potential for muscle toxicity and rhabdomyolysis, which is associated with statins, especially in high-risk groups. Accordingly, myalgias in patients under rosuvastatin treatment should raise a high index of suspicion and necessitate immediate testing of creatine kinase and myoglobin to exclude life-threatening rhabdomyolysis and prevent subsequent renal complications.

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