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Severe Rhabdomyolysis Complicated by Dialysis-Requiring Acute Kidney Injury and Acute Liver Injury: A Case Report

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Abstract:
Severe rhabdomyolysis may cause life-threatening acute kidney injury and multiorgan dysfunction. A 63-year-old Malaysian man with coronary artery disease, recent percutaneous coronary intervention, hypertension and dyslipidaemia was admitted in September 2026 with rhabdomyolysis, acute kidney injury and acute liver injury with coagulopathy. Creatine kinase reached 59,267 U/L, and serum creatinine was 228 µmol/L on 1 September. Rosuvastatin-associated muscle toxicity was considered alongside occult infection and inflammatory causes, although drug causality was not established. The patient developed anuria and required eight renal replacement therapy sessions comprising conventional haemodialysis and sustained low-efficiency dialysis. His course was complicated by anaemia requiring transfusion and acute liver injury considered clinically consistent with ischaemic hepatitis. Urine output subsequently improved, with a polyuric phase documented from 28 September 26. He was discharged on 1 October 26 with nephrology follow-up; however, maintenance haemodialysis was required at follow-up on 21 October 26. Liver biochemistry had normalised by that visit. This case highlights the importance of investigating rhabdomyolysis systematically, managing severe kidney injury promptly and documenting longer-term renal outcomes.
Keywords—rhabdomyolysis; acute kidney injury; haemodialysis; rosuvastatin; statin-associated myopathy; acute liver injury

I. INTRODUCTION

Rhabdomyolysis results from skeletal muscle injury with release of creatine kinase, myoglobin and electrolytes into the circulation. Acute kidney injury (AKI) can follow through pigment-associated tubular injury, vasoconstriction and intratubular obstruction. Causes include trauma, exertion, seizures, infection, metabolic abnormalities, inflammatory myopathy and drugs. Statins are a recognised but uncommon cause of severe rhabdomyolysis. We report severe rhabdomyolysis with dialysis-requiring AKI and concurrent acute liver injury in a patient with recent coronary intervention and rosuvastatin exposure. [1, 2]

II. CASE PRESENTATION

A 63-year-old Malaysian man, a former smoker, had coronary artery disease treated with two coronary stents in July 2026, hypertension, dyslipidaemia and eczema. He had been prescribed rosuvastatin 20 mg; the initiation date was not documented. An echocardiogram performed in August 2025 showed preserved left ventricular systolic function (ejection fraction 56%) and moderate aortic regurgitation. He was admitted on 8 September 2026.

Creatine kinase was reported at 59,267 U/L, with serum creatinine 228 µmol/L on 1 September 2026. Renal function subsequently deteriorated with anuria. The clinical team considered rosuvastatin adverse drug reaction, severe occult or atypical infection and an inflammatory condition as possible explanations. Abdominal and pelvic ultrasonography on 9 September showed increased bilateral renal echogenicity without hydronephrosis; kidney lengths were 12.5 cm and 11.9 cm. Minimal ascites was noted. These findings did not establish pre-existing chronic kidney disease.

The patient also developed acute liver injury (AST 2,594 U/L; ALT 1,352 U/L) with coagulopathy (PT 16.10, INR 1.2 and APTT 46.2), considered likely to represent ischaemic hepatitis. Hepatitis B and C screening results were non-reactive.

III. MANAGEMENT AND HOSPITAL COURSE

A temporary right femoral venous catheter was inserted and renal replacement therapy commenced. Eight sessions were documented: haemodialysis on 9 September (2 hours), 10 September (4 hours), 22 September (4 hours), 26 September (4 hours) and 29 September (4 hours); and sustained low-efficiency dialysis on 12, 15 and 19 September (6 hours each). Exact dialysis indications and haemodynamic data were unavailable.

Possible infection was treated with intravenous piperacillin-tazobactam 2.25 g four times daily and oral doxycycline 100 mg twice daily. No confirmed infective source was documented in the provided summary. Intravenous N-acetylcysteine was administered from 9 to 13 September for acute liver injury; the treating team documented subsequent resolution of the liver injury and coagulopathy.

Anaemia was attributed to chronic disease, possible haemolysis and blood loss from the femoral vascular access site or gastrointestinal tract. The patient received fresh frozen plasma on 11 September and packed red blood cells on 12 and 15 September. Faecal occult blood testing was positive on 15 September, but endoscopy was not performed because the patient declined the procedure. The

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latest documented haemoglobin concentration was 8.4 g/dL, and serum creatinine at discharge was 520 µmol/L. At follow-up on 21 October 2026, the patient required maintenance haemodialysis. Liver biochemistry had normalised by that date (AST 29 U/L; ALT 3 U/L).

IV. OUTCOME

Urine output improved, with a polyuric phase documented from 28 September 2026. The final recorded haemodialysis session during the admission was completed on 29 September. He was discharged on 1 October 2026 with predialysis review and nephrology follow-up to reassess the need for further dialysis. At follow-up on 21 October 2026, he required maintenance haemodialysis, despite the earlier improvement in urine output. Liver biochemistry had normalised.

V. DISCUSSION

A creatine kinase of 59,267 U/L is consistent with severe skeletal muscle injury. Rhabdomyolysis-associated AKI is biologically plausible in a patient progressing to anuria, but neither kidney biopsy nor urinary sediment findings were available to establish the precise pathology. Ultrasonography excluded obstruction but increased echogenicity alone does not prove chronic kidney disease. [1, 2]

Rosuvastatin exposure following recent coronary intervention was a clinically important possible precipitant. If weakness or creatine kinase elevation persisted after statin cessation, immune-mediated necrotising myopathy would warrant consideration [3, 4].

Renal replacement therapy treats complications of severe AKI, such as refractory electrolyte imbalance, acidosis, volume overload or uraemia, rather than serving as routine treatment solely for elevated creatine kinase. The documented use of conventional and sustained low-efficiency dialysis reflects substantial renal dysfunction, but specific indications cannot be inferred. Improvement in urine output is compatible with a recovery phase; complete renal recovery requires biochemical follow-up. [1, 2]

Acute liver injury and coagulopathy complicated the clinical course. Rhabdomyolysis can elevate aminotransferase concentrations through muscle release, whereas a diagnosis of ischaemic hepatitis generally requires compatible liver biochemistry and haemodynamic evidence. Concurrent anaemia and possible gastrointestinal bleeding further complicated management in the setting of recent coronary stenting. Nevertheless, the patient's liver biochemistry had normalised by follow-up on 21 October 2026.

VI. CONCLUSION

Severe rhabdomyolysis can lead to dialysis-requiring acute kidney injury and multiorgan complications. In this patient, rosuvastatin exposure raised concern for a drug-associated cause, although alternative explanations could not be excluded. Eight renal replacement therapy sessions were followed by improved urine output and a documented polyuric phase during admission. Despite this initial improvement, the patient required maintenance haemodialysis at follow-up on 21 October 2026. Liver biochemistry subsequently normalised. This case highlights the importance of multidisciplinary management and continued renal follow-up after severe rhabdomyolysis.

TABLE I. DOCUMENTED RENAL REPLACEMENT THERAPY

Date (2026)	Modality	Duration
9 September	Haemodialysis	2 hours
10 September	Haemodialysis	4 hours
12 September	Sustained low-efficiency dialysis	6 hours
15 September	Sustained low-efficiency dialysis	6 hours
19 September	Sustained low-efficiency dialysis	6 hours
22 September	Haemodialysis	4 hours
26 September	Haemodialysis	4 hours
29 September	Haemodialysis	4 hours

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DECLARATIONS

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DISCLOSURE

Harafinova Harman Shah had full access to all of the data in this case report and takes complete responsibility for the integrity of the data and the accuracy of the data presented. The author approved the final version for submission.

ETHICS STATEMENT

This is a single-patient case report; institutional review board approval was not required at Hospital Pengajar Sultan Zainal Abidin. The report was conducted in accordance with the Declaration of Helsinki.

CONFLICTS OF INTEREST

The author declares no conflicts of interest.

DATA AVAILABILITY STATEMENT

The data supporting the findings of this case report are available from the corresponding author upon reasonable request. The data are not publicly available because of patient privacy considerations.

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