Case Report

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A patient with severe rhabdomyolysis and high levels of creatinine kinase had renal functions fully recovered after haemodialysis: a case report

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Abstract

Rhabdomyolysis is diagnosed based on the levels of blood biomarkers such as creatine kinase (CK), but the use of CK levels to predict long-term renal function remains controversial. This current report presents a case with a very high CK level with the presentation of acute kidney injury (AKI) who regained full renal function. A 29-year-old man, in a manic mood and presenting with dyspnoea, was admitted to hospital following an episode of ketamine use along with a history of drug abuse. The laboratory analyses identified rhabdomyolysis (CK, 35 266 U/l) and AKI (serum creatinine, 3.96 mg/dl). Despite treatment with intravenous normal saline (4000 ml/day), his CK level reached at least 300 000 U/l. He underwent 13 sessions of haemodialysis and his renal function fully recovered. The final measurements were serum creatinine 1.0 mg/dl and CK 212 U/l. These findings support the view that the predictive power of CK level on AKI is limited, especially regarding long-term renal function. Close follow-up examinations of renal function after haemodialysis are mandatory for patients with rhabdomyolysis.

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Introduction

Rhabdomyolysis is diagnosed based on the levels of blood biomarkers like creatine kinase (CK) and myoglobin. CK levels that exceed five-times the upper limit of the normal range suggest a diagnosis of rhabdomyolysis.[1] However, the use of CK levels to predict long-term renal function in patients with rhabdomyolysis remains controversial. For example, a recent case study described a 32-year-old man, diagnosed with rhabdomyolysis and acute kidney injury (AKI), whose CK level was normal (156 U/l).[2] Renal biopsy of this patient also confirmed severe acute tubular injuries with positive myoglobin casts.[2] The present case report describes a similar patient that experienced initial severe rhabdomyolysis (CK $\geq$300 000 U/l), who subsequently regained full renal function following haemodialysis.

Case report

A 29-year-old man, in a manic mood and experiencing dyspnoea, was admitted to Taichung Veterans General Hospital, Taiwan in December 2018 following an episode of ketamine use along with a history of drug abuse during the previous 6 months. He was handcuffed and brought in by police officers due to illicit behaviour. He also displayed persistent uncontrolled limb movements, with or without physical restraints. He had no history of hypotension nor any previous exposure to renal toxins, but he appeared hypertensive and tachypnoeic. His vital signs were as follows: blood pressure, 193/116 mmHg; heart rate, 86 beats/min; respiratory rate, 30 breaths/min; and body (blood) temperature, 36.4°C. He was moody, excited and complained of myalgia and dark urine. Laboratory analyses identified rhabdomyolysis as follows: CK, 35 266 U/l; aspartate aminotransferase (AST), 466 U/l; alanine transaminase (ALT), 143 U/l; lactate dehydrogenase, 2285 U/l; and AKI (serum creatinine, 3.96 mg/dl; blood urea nitrogen, 37 mg/dl). Findings were all negative on brain computed tomography scans and lumbar puncture. He was subsequently treated with intravenous normal saline (4000 ml/day) together with alkalization procedures. The urine output was up to 1500 ml on the first day but it decreased to 500 ml on the second day. Anuria was noticed on the third day. His CK level nevertheless reached 300 000 U/l, a condition that had persisted over 3 days after admission. In fact, his CK levels were so high that they had exceeded the maximum detectable level of the measuring device. On the third day, his serum creatinine level increased to 15.23 mg/dl, the AST level to 4395 U/l and the ALT level to 2663 U/l. He then received a series of 13 haemodialysis sessions. The results of renal function tests showed the following: urinalysis, 3+ of occult blood and 2+ of proteinuria without haematuria (0–2/high power fields of red blood cells). Rhabdomyolysis-related AKI was clinically diagnosed, but without a renal biopsy. After 11 days of hospitalization, the patient was discharged with a cuffed vascular catheter for further haemodialysis if necessary. Finally, his renal functions recovered fully and his haemodialysis treatment was stopped. The final measurements were as

follows: serum creatinine, 1.0 mg/dl; CK, 212 U/l. His entire treatment course is summarized in Figure 1. Written informed consent for the publication of patient information was provided by this patient. No picture of this patient was presented in this study and all data were anonymized.

Discussion

For this current patient, the causes of his rhabdomyolysis were traumatic (multiple trauma and crush injuries) as well as non-traumatic (extreme exertion and ketamine abuse) in origin. Several studies have reported on ketamine-related rhabdomyolysis.[3,4] Ketamine can induce agitation and extended muscular activity, which ultimately may lead to rhabdomyolysis.[3,4] Metabolic myopathy being a possible cause was not considered likely in this case because there were no recurrent episodes, exercise intolerance or a family history. Even without the renal biopsy and measured myoglobulin levels (the culprit of AKI in rhabdomyolysis), multifactorial rhabdomyolysis-related AKI can readily be diagnosed clinically. The myoglobulin cannot be removed by haemodialysis.

The extremely high level of CK that was observed in this current case simply reflected the large muscle mass of this young man (171 cm in height, 70 kg in weight) in addition to other unknown factors. The exact CK level of this current patient could not be determined due to device limitations, but it was at least 300 000 U/l. This current patient might

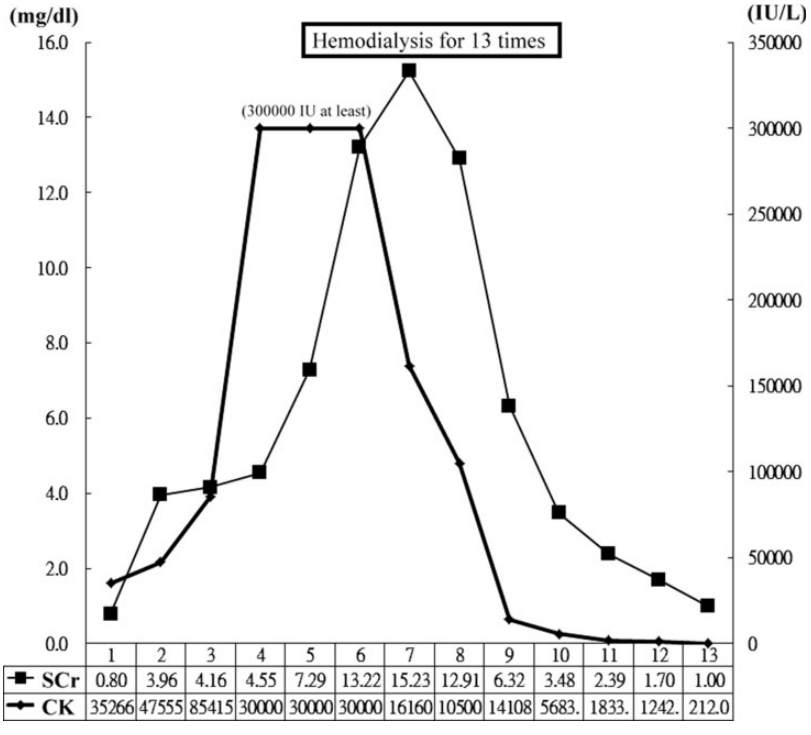

Figure 1. Changes in serum creatinine (SCr) and creatine kinase (CK) levels during the treatment of rhabdomyolysis-related acute kidney injury in a 29-year-old man who was admitted to hospital in a manic mood and experiencing dyspnoea following an episode of ketamine use along with a history of drug abuse.

well have the second highest, if not the highest CK level reported in the literature to date. For example, the case of a 19-year-old man with a head injury and severe rhabdomyolysis (650 000 U/l) was previously reported and after receiving haemodialysis for 24 days to treat the AKI, his renal functions also completely recovered.[5] However, the haemodialysis only provided supportive care and was not able to remove the myoglobulin.[5]

The predictive power of CK for AKI was comprehensively reviewed in a previous meta-analysis, which concluded that the aetiology of rhabdomyolysis (traumatic/nontraumatic) strongly affects the predictive role of CK on renal functions.[6] Rhabdomyolysis shows a better correlation with renal function in crush-induced AKI.[6] A recently reported case of a patient that had used recreational drugs demonstrated little correlation between CK level and AKI.[2] CK levels do not appear to predict the long-term outcome of renal dysfunction, as demonstrated in the present case and in a previously reported case.[5] Both patients regained full renal function after haemodialysis even when they initially presented with extremely high levels of CK ($\geq$300 000 or 650 000 U/l).[5] Although this current case does not present any innovative treatment, it does serve to highlight the fact that the predictive value of CK in rhabdomyolysis is very limited and this should be borne in mind when clinicians are treating patients with rhabdomyolysis in clinical practice.

In conclusion, the predictive power of CK on AKI in patients with rhabdomyolysis may not be uniformly simple, especially regarding long-term renal function. Extremely high presenting levels of CK do not necessarily indicate that patients will be unable to completely regain their renal function. Close follow-up examinations of renal function after haemodialysis are mandatory for patients with rhabdomyolysis.

Declaration of conflicting interest

The authors declare that there are no conflicts of interest.

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