Improvement in renal prognosis with prompt hemodialysis in hyperosmolar hyperglycemic state-related rhabdomyolysis
A case report
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Abstract
Rationale: Acute kidney injury is common and correctable in patients with a hyperosmolar hyperglycemic state (HHS). Nevertheless, hyperglycemic crisis may also contribute to the development of rhabdomyolysis, which can worsen renal function and lead to high mortality in such patients.

Patient concerns: Herein, we report a case of hyperosmolar hyperglycemic state-related rhabdomyolysis and acute renal failure with an excellent outcome.

Diagnosis: A 26-year-old Asian female with underlying paranoid schizophrenia presented with newly diagnosed type 2 diabetes mellitus complicated with HHS. Her renal function deteriorated rapidly in spite of standard management for hyperglycemic crisis. Rhabdomyolysis was subsequently diagnosed according to the high levels of serum creatine kinase (CK) (37,710 U/L, normal range: 20–180 U/L) and myoglobin (5167.7 ng/mL, normal range: 14.3–65.8 ng/mL).

Interventions: After treatment failure of intravenous hydration plus loop diuretic agent for rhabdomyolysis related acute renal failure, temporary hemodialysis was performed 3 times to relieve oliguria and pulmonary edema.

Outcomes: Her renal function recovered well after temporary renal replacement therapy.

Lessons: Rhabdomyolysis is a complication of HHS. Delayed detection can be fatal, and timely renal replacement therapy can result in an excellent prognosis. Therefore, it is crucial for clinicians to detect and treat such patients as early as possible to avoid impairing their renal function.

Abbreviations: CK = creatine kinase, Cr = creatinine, DKA = diabetic ketoacidosis, HHS = hyperosmolar hyperglycemic state.

Keywords: hyperosmolar hyperglycemic state, rhabdomyolysis

1. Introduction
A hyperosmolar hyperglycemic state (HHS) is one of the most serious acute complications of diabetes. The rate of hospital admissions for HHS accounts for less than 1% of all primary diabetic admissions.[1] The mortality rate for patients with HHS is between 10% and 20%, which is approximately 10 times higher than that for diabetic ketoacidosis (DKA).[2]

Rhabdomyolysis, a term used to describe the rapid breakdown of striated muscle, is characterized by rupture and necrosis of muscle fibers. The classic triad of symptoms includes muscle pain, weakness, and dark urine, although more than 50% of patients do not complain of muscle pain or weakness.[3] Rhabdomyolysis is of clinical concern because it can cause acute renal failure, disseminated intravascular coagulation, cardiac arrest and arrhythmias, and significant electrolyte abnormalities, all of which can result in significant morbidity or mortality.[4]

About 20% of patients admitted for HHS have rhabdomyolysis, with an estimated mortality rate of about 35.5%.[5] The clinical features of rhabdomyolysis vary in severity from asymptomatic to acute renal failure requiring hemodialysis, and can be lethal if physicians ignore the possibility.

The clinical course between the 2 conditions is poorly understood. Herein, we report a case of acute renal failure following hyperosmolar hyperglycemic state-related rhabdomyolysis with an excellent outcome after a timely diagnosis and prompt treatment. The experience from this case may help physicians improve treatment and outcomes for such patients.

2. Case report
In December 2015, a patient with newly diagnosed diabetes mellitus and the acute complication of hyperglycemic crisis presented at Chang Gung Memorial Hospital. This patient was a 26-year-old Asian female with underlying paranoid schizophrenia for which she was under routine medical treatment (risperidone, trihexyphenidyl, and clonazepam). She was also obese, with a body mass index of about 31.99 (body weight: 85
kg, body height: 163 cm). A few days before admission, she became irritable, experienced polydipsia, consumed many beverages and subsequently suffered from nausea, vomiting, and loss of appetite. Her status of auditory hallucinations, slurred speech, and bilateral hand tremors had not changed. Finally, she developed profound general weakness and was transferred to our emergency department for treatment. A physical examination revealed no specific abnormalities, except for dry oral mucosa, obese appearance, and mild fever. Hyperglycemia (1408 mg/dL glucose), hyperosmolarity (osmolality = 395 mOsm/kgH2O), positive serum ketone body without metabolic acidosis (Table 1) were noted, and HHS with ketosis was diagnosed. Prompt, aggressive fluid replacement, continuous insulin infusion (0.1 IU/kg/h) and potassium correction were started. Because she had a mild fever, empiric ceftriaxone was prescribed to cover bacterial infection and a subsequent switch to levofl oxacin was made. As dehydration is more severe in patients with HHS than in DKA, the incidence of acute renal failure in patients with diabetic crisis (DKA and HHS) was 25%. As dehydration is more severe in patients with HHS than in DKA, the incidence of acute renal failure in patients with diabetic crisis (DKA and HHS) was 25%.

Acute kidney injury is common and correctable in patients with a hyperglycemic crisis. In a study by Singhal et al, the incidence of acute renal failure in patients with diabetic crisis (DKA and HHS) was 25%. As dehydration is more severe in patients with HHS than in DKA, the incidence of acute renal failure in HHS may be higher. The cause of kidney injury is most often volume depletion, which can be reversed by adequate volume expansion, and renal replacement therapy is seldom necessary.

In our case, the patient’s urine output declined in spite of fluid resuscitation, and she subsequently suffered from pulmonary congestion and dependent edema. Careful re-evaluation of the etiology of acute renal failure is critical to allow for proper therapy of HHS with an unstable clinical course following standard treatment. In our case, continuing intravenous hydration alone would have resulted in inevitable respiratory failure because of pulmonary edema. Fortunately, rhabdomyolysis was diagnosed immediately, and her renal function had an excellent prognosis after temporary hemodialysis therapy. The common causes of rhabdomyolysis include trauma, extreme exertion, epilepsy, and toxin-related myopathy (such as that caused by alcohol and statins). Occasionally, hyperglycemic crisis can contribute to the development of rhabdomyolysis. Inadequate muscle energy storage, hyperosmolarity, or severe hypophosphatemia during hyperglycemic crisis are thought to be associated with rhabdomyolysis, however, the mechanism is not fully understood.
not well understood.\textsuperscript{[9–12]} If physicians ignore this rare complication of HHS, then the risk of mortality for patients with HHS is increased.\textsuperscript{[5]}

In our case, her critical condition, including pulmonary edema and electrolyte imbalance, was reversed after prompt hemodialysis following HHS and rhabdomyolysis-related oligouria and acute renal failure. Her renal function improved after heme pigment casts were removed by dialysis and tubular obstruction was relieved. Urine volume, as well as serum Cr and CK levels, were restored to normal range, and temporary hemodialysis was successfully stopped. In such cases, another etiology of rhabdomyolysis that should be considered is neuroleptic malignant syndrome caused by antipsychotic medications.\textsuperscript{[13]} However, the dosages of antipsychotic medications in our case had not changed before the onset of HHS and rhabdomyolysis, and no limb rigidity or seizures were observed. Therefore, neuroleptic malignant syndrome was not suspected, and her antipsychotic medications were continued throughout the clinical course after consultation with a psychiatrist.

4. Conclusion

In conclusion, rhabdomyolysis is not an uncommon complication of HHS, but delayed detection can be fatal. The early recognition of rhabdomyolysis, followed by prompt fluid resuscitation and timely renal replacement therapy can result in an excellent prognosis of survival and renal recovery.

Author contributions

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