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A Case of Severe Acute Kidney Injury Induced by Near-Drowning

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Abstract: A 23-year-old man began drowning when he suffered a leg spasm while snorkeling in the ocean. After being rescued, he showed consciousness disturbance and a pale face. On arrival, chest roentgen showed decreased radiolucency in the bilateral lung fields. The level of uric acid was 18.4 mg/dl. He received tracheal intubation for mechanical ventilation with positive end-expiratory pressure. After admission to the intensive-care unit, his respiratory function improved, and he was extubated on the second hospital day. However, he became complicated with oligouria and showed a daily increase in his creatinine level. He also became complicated with lung edema due to acute renal failure, so intermittent hemodialysis therapy was started on the fourth hospital day. After six hemodialysis sessions, he spontaneously obtained sufficient urine flow, and the levels of both creatinine and blood urea nitrogen decreased without hemodialysis. He was ultimately discharged on foot. Acute renal failure after nearly drowning to death is not very rare, so physicians should pay close attention to this complication in near-drowning patients. Hyperuricemia may contribute to the occurrence of acute renal failure after nearly drowning.
Keywords: drowning; acute renal failure; uric acid.

INTRODUCTION

Near-drowning represents an insult that can affect all organ systems. A common pathway for injury is hypoxemia, acidosis, and hypoperfusion[1]. However, most literature on drowning and near-drowning focus on pulmonary insults, such as pulmonary edema and aspiration pneumonia [2]. Recently, reports of immersion- or near-drowning-induced acute kidney injury have been increasing [2-6]. We herein report a case of severe acute kidney injury induced by nearly drowning.

CASE PRESENTATION

A 23-year-old man began drowning when he suffered a leg spasm while snorkeling in the ocean. He had no significant medical or family history. After being rescued, he showed consciousness disturbance and a pale face. He was transported to our hospital by a physician-staffed helicopter on arrival; his vital signs were as follows: Glasgow Coma Scale, E3V5M6; blood pressure, 76/46 mmHg; heart rate, 141 beats per minute with atrial fibrillation; and percutaneous saturation 92% under 10 L/minute of oxygen. He had moist rales on auscultation. Electrocardiogram and cardiac sonography findings were negative. Chest roentgen showed decreased radiolucency in the bilateral lung fields. He received tracheal intubation for mechanical ventilation with positive end-expiratory pressure. Chest computed tomography (CT) depicted consolidations in both dorsal lung fields, suggesting aspiration (Figure 1), so he was administered tazobactam/piperacillin.

The main results of a blood analysis are shown in Table 1. After admission to the intensive-care unit, his respiratory function improved, and he was extubated on the second hospital day. However, he became complicated with oligouria and showed a daily increase in his creatinine level (Figure 2), so all drugs were stopped. He also became complicated with lung edema due to acute renal failure, so intermittent hemodialysis therapy was started on the fourth hospital day. After six hemodialysis sessions, he spontaneously obtained sufficient urine flow, and the levels of both creatinine and blood urea nitrogen decreased without hemodialysis. He was ultimately discharged on foot and introduced to a hospital far from our institution but near his home.

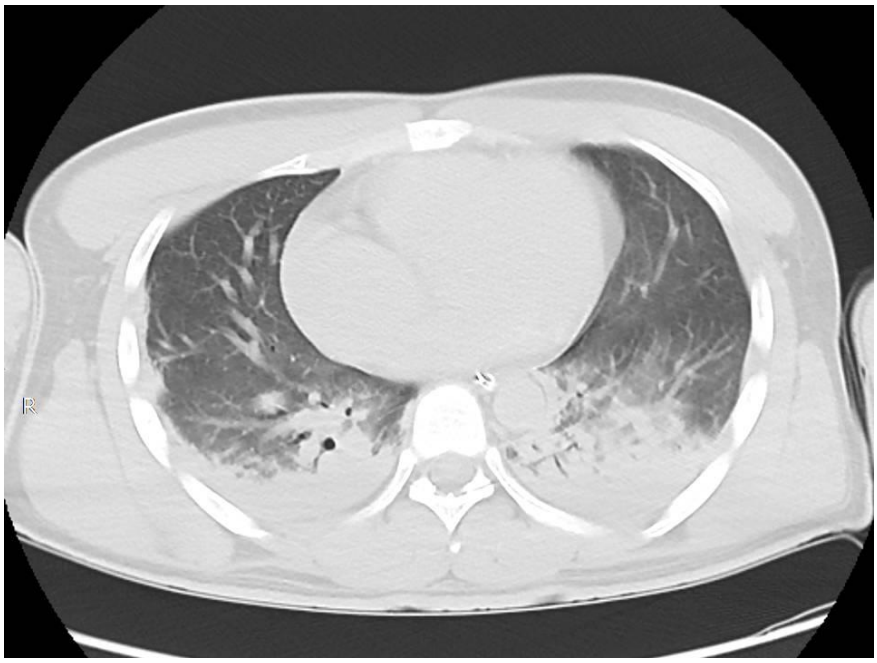

Fig-1: Chest computed tomography (CT) on arrival. CT depicted consolidations in both dorsal lung fields, suggesting aspiration.

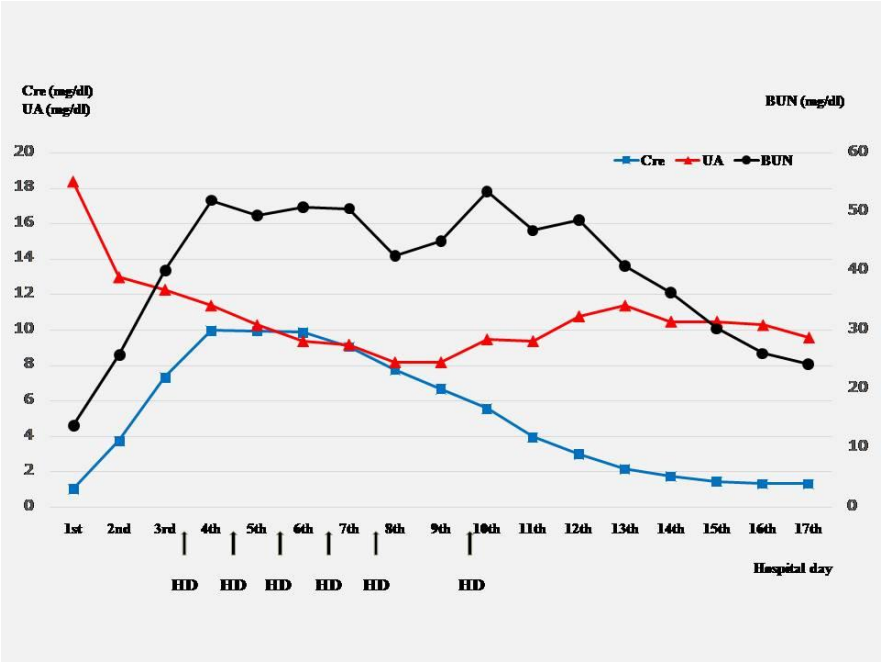

Fig-2: Time course of the creatinine, uric acid and blood urea nitrogen levels. The levels of creatinine and blood urea nitrogen initially increased and then decreased without hemodialysis after the 12th hospital day. The initial level of uremic acid was 18.4 mg/dl.

Cre, creatinine; UA, uric acid; BUN, blood urea nitrogen; HD, hemodialysis

DISCUSSION

Gorelik *et al.* reported the results of an evaluation of the medical records of 95 patients rescued from nearly drowning in the Mediterranean Sea [3]. Among them, 42 (43%) developed acute kidney injury after nearly drowning. Acute kidney injury after nearly drowning was associated with the need for resuscitation and mechanical ventilation as well as with the calculated volume of ingested seawater (extrapolated from the increase in the plasma sodium level) and the degree of acidemia, lactemia, and ventilatory failure, similar to the present patient. They summarized the pathogenesis of acute kidney injury after near-drowning as conceivably multifactorial and primarily including rhabdomyolysis, a systemic inflammatory response associated with multiorgan failure, and hypoxic renal injury related to anoxic insult due to a reduced renal blood flow [3].

Table-1: Blood laboratory findings on arrival

Arterial blood gas analysis under 10 L/minute oxygen			
pH	7.251	PO_2	132 mmHg
PCO_2	33.8 mmHg	HCO_3^-	14.4 mmol/l
Base excess	-11.7 mmol/L	Lactate	11.2 mmol/L
Cell blood count			
White blood cells	6.9 g/dl	Albumin	4.4 g/dl
Glucose	97 mg/dl	HbA1C	5.4 %
Aspartate aminotransferase	26 IU/l	Alanine aminotransferase	22 U/l
Serum biochemical data			
Total protein	6.9 g/dl	Albumin	4.4 g/dl
Glucose	97 mg/dl	HbA1C	5.4 %
Aspartate aminotransferase	26 IU/l	Alanine aminotransferase	22 U/l
Creatine phosphokinase	224 IU/l	Total bilirubin	0.5 mg/dl
Blood urea nitrogen	13.9 mg/dl	Creatinine	1.02 mg/dl
Sodium	141 mEq/l Potassium		
Coagulation			
Activated partial thromboplastin time	24.8 (27.4) sec		
Prothrombin time	13.3 (12.1) sec		
Fibrinogen	257 mg/dl		

The present patient showed extremely high levels of uric acid in a blood examination on arrival. Pimentel *et al*. reported on the level of serum uric acid in neonatal rats subjected to hypoxia-induced ischemia. The uric acid levels were significantly higher after eight days in the hypoxia-ischemia insult group than in the control group, returning to baseline levels by 60 days after insult [7]. One of the mechanisms suggested to underlie this result was the effect of xanthine oxidase, an enzyme that converts hypoxanthine to xanthine and xanthine to uric acid in response to hypoxic-ischemic insult [8]. In addition, renal failure itself increases the level of uric acid due to decreased uric acid clearance. Furthermore, uric acid can increase after tissue damage due to hypoxia induced by drowning [9]. Hyperuricemia can deteriorate the renal function [10]. Accordingly, the marked hyperuricemia in the present case may have contributed to the complication of acute renal failure.

CONCLUSION

Acute renal failure after nearly drowning to death is not very rare, so physicians should pay close attention to this complication in near-drowning patients. Hyperuricemia may contribute to the occurrence of acute renal failure after nearly drowning.

Conflict of interest statement

The authors declare no conflicts of interest in association with this study.

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