A Case of Severe Acute Kidney Injury by Near-Drowning

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INTRODUCTION

Acute kidney injury (AKI) secondary to near-drowning is rarely described and poorly understood. Much of the literature of near-drowning has concentrated on the respiratory effects of aspiration of salt and freshwater, and on the management of both early and late respiratory complications such as aspiration pneumonia and adult respiratory distress syndrome (1). However, near-drowning and immersion can have profound effects on other end organs such as cerebral (hypoxic brain injury, cerebral edema) (2), cardiac (atrial fibrillation) (3) and hematologic complications (coagulopathy and hemolysis) (4). Moreover, multisystem failure resulting from near-drowning is also well described (5). Near-drowning induced acute kidney injury (AKI) is not uncommon and is heterogenous clinical entity (6). Although the resultant AKI is usually mild and self-limited, severe cases such as AKI associated with shock, multisystem failure, rhabdomyolysis (7, 8) and isolated AKI can occur (6). Only few cases of severe isolated AKI due to acute tubular necrosis (ATN) resulting from near-drowning exist in the literature. We report a case of near-drowning who developed severe isolated AKI requiring dialysis due to biopsy-confirmed ATN.

CASE DESCRIPTION

A 21-yr-old man was admitted to this hospital because of anuria and nausea on June 11, 2011. He had been well until 3 days before admission, when he went to swim in the lake with his friends. He was exhausted before got back to the shore, and was suffocating. He was unconscious briefly (about 2-3 min) until rescued by his friend. He was transported to the emergency room of another hospital. On physical examination, he was conscious but sleepy; blood pressure was 132/68 mmHg, body temperature was 36.9°C, and pulse was 114 beats per minute; respiration was 20 per minute, and the oxygen saturation was 96% while the patient was breathing ambient air. Laboratory tests revealed serum creatinine level of 1.4 (0.4-1.3) mg/dL, total carbon dioxide (TCO₂) of 9.8 (24-30) mM/L, anion gap of 27.2 mM/L, hemoglobin concentration of 16.7 g/dL, and leukocyte count of 12,300/μL (lymphocyte 41.1%). Electrocardiogram and chest radiography were normal. The patient was discharged after 12 hr observation period. He was admitted to our hospital 3 days later, complaining of being tired, anorexic and anuric. The vital signs were as follows: blood pressure, 140/90 mmHg; pulse, 75 beats per minute; respiration, 20 per minute; and body temperature, 37.6°C. Laboratory findings showed blood urea nitrogen (BUN) of 42.7 (6-26) mg/dL, serum creatinine of 11.5 mg/dL, seum cystatin C level of 3.39 (0.5-1.10) mg/L, aspartate aminotransferase (AST) of 6 (10-40) IU/L, alanine aminotransferase (ALT) of 9 (6-40) IU/L, lactate dehydrogenase (LDH) of 435 (218-472) IU/L, creatine kinase (CK) of 225 (5-217) U/L, myoglobin of 87.9 (15.2-91.2) ng/mL, TCO₂ of 15.4 (20-28) mM/L, anion gap of 19.3 mM/L, hemoglobin concentration of 11.80/μL, leukocyte count of 11,180/μL (segmented neutrophil 86.5%, lymphocyte 8.1%). There was
AKI secondary to near-drowning is not uncommon, but the re-
discussed by Spicer et al. in 1999 (6); usually the term means that
resultant severe AKI requiring dialysis is exceedingly rare: we found
only 7 reported cases, including the present report (6-10). The two other
cases were related with rhabdomyolysis (7, 8), 1 case resulted
from hypothermia (9), 1 case were related with both (10), and 2 cases had isolated AKI (6). Isolated AKI was first
described by Spicer et al. in 1999 (6); usually the term means that
disproportionately severe AKI even several days after the immers-
sion episode who did not require cardiopulmonary resuscitation (CPR) or did not experience shock, hypothermia or myoglobinuria after their near-drowning. This case is the third report
of severe isolated AKI in the literature and first report in Korea. Immersion and drowning injury has been widely reviewed (1-
3), and widespread tissue hypoxia and subsequent reperfusion
injury are thought to be the predominant underlying pathophys-
ology, although hypovolemia and hypothermia may also con-
tribute to tissue damage. The initial event leading to tissue inju-
ry in kidney ischemia-reperfusion injury (IRI) is the acute reduc-
tion of blood flow that produces hypoxia-induced vascular and
tubular dysfunction. Following reperfusion, various intracellu-
lar events occur that lead to cellular dysfunction, apoptosis and
cell death (11). The morphological changes occur mainly in prox-
imal tubules, susceptible to ischemic injury, including loss of
polarity, loss of the brush border (12), and redistribution of inte-
grins and Na+/K+-ATPase to the apical surface (13). Calcium (14)
and reactive oxygen species (15) may also have a role in these
morphological changes, in addition to subsequent cell death
resulting from necrosis and apoptosis. Both viable and nonvia-
cible cells are shed into the tubular lumen, resulting in the forma-
tion of casts and luminal obstruction and contributing to the re-
duction in the GFR (16). Inflammation is also an important early
event leading to intracellular responses that ultimately result in
apoptosis and necrosis. The involvement of the immune system
in kidney IRI is very complex; dendritic cells and macrophages
may contribute early in the innate immune response to kidney
IRI and promote neutrophil infiltration (17), while T regulatory
cells have been shown to suppress the extent of kidney IRI through
an IL-10 dependent mechanism (18). Dendritic cells, neutro-
phils, phagocytic macrophages and lymphocytes participate in
the early phase of kidney IRI as well as the late reparative phase
(19). A retrospective study to assess clinical predictors of near-
drowning associated AKI concluded that lymphocytosis may also predict renal impairment (6). In our case, the patient had
not lymphocytosis but mild lymphocyte infiltration in intersti-
tium was seen.

Metabolic acidosis is common after near-drowning, and is due
to lactic acidosis induced by tissue hypoxia (5, 20). That lactate
was the probable cause of acidosis supported by the increased
anion gap and reversible nature of the acidosis. In this case, un-
fortunately, serum level of lactic acid and arterial blood gas anal-
ysis just after immersion episode were not checked, but severe
high anion gap metabolic acidosis was seen on presentation. The
acidosis was quickly corrected, but the initial mild renal dys-
function progressed to establish severe AKI and required sup-
portive dialysis. It is very similar to previous case reports of iso-
lated AKI (6). Why renal injury predominates in the absence of
other post-immersion injury sequelae is not known. The possi-
ble explanation is renin-angiotensin surge when returning to dry

DISCUSSION

AKI secondary to near-drowning is not uncommon, but the re-

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from hypothermia (9), 1 case were related with both (10), and 2 cases had isolated AKI (6). Isolated AKI was first
described by Spicer et al. in 1999 (6); usually the term means that

no elevation of infectious or immunological marker. Urinalysis showed a trace of blood and 1+ proteinuria. A spot urine protein creatinine ratio was 634.37 mg/g. The hourly urine output was less than 10 mL despite of bolus infusion of normal saline and continuous infusion of furosemide. A chest roentgenogram initially showed mild pulmonary congestion with bilateral pleural effusion. Non-contrast enhanced abdominal computed tomography showed normal sized kidneys without urinary tract obstruction. A bone imaging study using Tc-99m-methylene diphosphonate showed no soft tissue uptake. The patient needed five sessions of dialysis over the succeeding 5 days and serum creatinine level was 5.12 mg/dL on the next day after the last hemodialysis session, then we stopped hemodialysis treatment. On the 9th hospital day, serum creatinine level was 3.05 mg/dL, we planned renal biopsy that had been postponed because of bleeding risk. The next day, serum creatinine level was 1.98 mg/dL and renal biopsy was performed. The renal tubular epithelial cells were denuded and had exfoliating brush borders and intermittent mitosis due to regeneration. The interstitium was edematous and had mild infiltration of lymphocytes, but the glomeruli showed unremarkable finding (Fig. 1). The diagnosis of acute tubular necrosis was retained. The patient’s renal function recovered spontaneously, 1.24 mg/dL 3 weeks later and 1.07 mg/dL 5 weeks later.

Fig. 1. Renal biopsy specimen from the patient with serum creatinine of 1.98 mg/dL. A proximal tubule with denuded epithelium, exfoliating brush borders (black arrows) and mitotic figure (white arrow) is shown (× 400).
land after whole-body immersion may play a role (3). Although the pathology of isolated AKI is controversial, renal hypoxia and subsequent reperfusion injury play major role in developing severe AKI due to ATN in our case. It is plausible that the patient had not hemodynamic instability, hypothermia or rhabdomyolysis that related with AKI after near-drowning.

In summary, we describe a case of severe isolated AKI caused by near-drowning. To the best of our knowledge, this is the first such case report in Korea. Our case differs from the 2 other cases we reviewed in the literature in that our patient’s AKI was confirmed by kidney biopsy as ATN. Although near-drowning associated AKI is usually mild and self-limited, severe AKI required dialysis can occur even several days after immersion event, it is recommended that any patient who presents after near-drowning or immersion should be assessed for potential AKI.

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