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NEPHROLOGY & UROLOGY | CASE REPORT

Severe acute kidney injury associated with immersion into seawater

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Abstract: Near drowning is an important, but rarely reported precipitant of acute kidney injury (AKI). The underlying pathophysiology is thought to be ischaemic kidney injury, but diagnostic uncertainty has arisen due to reports of isolated AKI in the absence of ischaemic damage to other organs. Here we describe a case of immersion into seawater resulting in near drowning and severe AKI. Our case highlights the need for assessment of kidney function in this situation. Based on the clinical presentation, we propose that ischaemic injury does indeed occur, and we make suggestions on the management of such patients in the absence of histopathological information obtained from renal biopsy.

Subjects: Nephrology; Physiology; Emergency Medicine

Keywords: acute kidney injury; acute kidney tubular necrosis; near drowning; renal circulation

1. Introduction

Near drowning, widely accepted as survival beyond 24 h after being unable to breath due to immersion in water, presents to acute care services and is commonly associated with mortality or long-term neurological sequelae (Quan, Mack, & Schiff, 2014). It can cause cardiac dysrhythmias, respiratory compromise and may be associated with concomitant trauma (Salomez & Vincent, 2004).

Among the numerous causes of acute kidney injury (AKI), near drowning is an infrequently reported association, with a poorly understood pathophysiology. Although cardiopulmonary arrest or overt circulatory shock in the context of near drowning can lead to multiorgan failure encompassing AKI, only 20% of near drowning AKI patients suffer circulatory compromise (Spicer et al., 1999).

ABOUT THE AUTHORS

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PUBLIC INTEREST STATEMENT

People that survive immersion into sea water can suffer life threatening complications, sometimes days after the initial event. There is currently no published guidance on the management of kidney failure in this setting. Here we describe a case in which a man survived a fall into sea water, resulting in complete immersion and near drowning. He then presented to hospital some 24 h later feeling unwell at which time the kidneys were found to be non-functioning. We offer advice on the management of such patients, which in this case resulted in apparent complete recovery of kidney function. Dissemination of reports such as ours should hopefully bring about consensus on how best to manage such patients, including agreement on the issue of renal biopsy.

Additionally, patients can present with isolated AKI sometimes days after the incident (Miki, Takeda, Yamamoto, & Kusano, 2013). It is unknown whether immersion into fresh water or seawater influences AKI severity or outcomes in near drowning.

Ischaemia-reperfusion injury (IRI) resulting from either reflex aspiration of water into the lungs, or laryngospasm, has been proposed as an explanatory mechanism for AKI in near drowning (Layon & Modell, 2009), but this does not satisfactorily explain isolated AKI in the absence of other end-organ damage such as central neurological or myocardial dysfunction. A surge in renin-angiotensin levels after whole-body immersion has also been postulated as a cause for such AKI but the levels or activities of these factors have not been systematically evaluated in near drowning cases, so this is unproven (Gooden, 1992). When organ dysfunction does occur in other systems it often lacks full explanation; for example, electrocardiograms of near drowning victims frequently demonstrate features of Takotsubo cardiomyopathy, not accounted for by conventional myocardial ischaemia (Omar, Sprenker, Bosco, Mangar, & Camporesi, 2015) whilst non-cardiogenic adult respiratory distress syndrome (ARDS) can paradoxically present in a delayed fashion, sometimes hours after near drowning (Buggia, 2014).

Despite the infrequency of near drowning AKI reports in the literature, one case series suggests a 50% incidence of AKI within a cohort of 30 near drowning casualties (Spicer et al., 1999). The majority (73%) of these patients had mild, self-limiting AKI. However, 27% had severe AKI with a creatinine greater than 300 µmol/l. Severe cases are very seldom reported and may require acute haemodialysis until recovery of renal function but there is currently no accepted system to predict which casualties may develop AKI, the severity of their AKI or the need for haemodialysis. Suggested prognostic indicators for AKI include low arterial pH, low serum bicarbonate, low base excess, and dipstick haematoproteinuria, although these have not been prospectively validated (Spicer et al., 1999). Interestingly, the requirement for cardiopulmonary resuscitation was not predictive of AKI (Spicer et al., 1999). Lymphocytosis has been suggested to predict renal impairment in the context of near drowning, and although cellular components of the immune system including lymphocytes may participate in renal IRI (Jang, Ko, Wasowska, & Rabb, 2009), there could be a more specific immunological mechanism relating to near drowning that is yet to be defined.

Whilst two case reports of isolated AKI after near drowning describe histopathological findings in keeping with acute tubular necrosis (ATN) (Alp et al., 2016; Seong et al., 2012), the remainder of cases have no associated histopathology, generating uncertainty as to the underlying pathophysiology of this rarely reported condition and the broad spectrum of presentation ranging from mild to severe AKI. Additionally, the inconsistent presence or absence of supporting histopathological information in the published cases indicates a lack of consensus as to whether a kidney biopsy is beneficial to the management of AKI associated with near drowning.

Here we describe the case of a 42 year old male warehouse worker who suffered severe oliguric AKI associated with near drowning, secondary to total body immersion in sea water. We suggest that his AKI can be explained by ischaemic ATN and make suggestions on the management of such individuals.

2. Case summary

Our patient, who had no significant past medical or family history, suffered an accidental fall whilst on a fishing trip, resulting in total immersion into cold sea water for approximately 5 min. He swam to safety, did not require cardiopulmonary resuscitation and was conscious throughout the incident, with no apparent trauma. He then presented to hospital 24 h afterwards, complaining of new onset nausea and fatigue.

Physical examination detected only mild epigastric tenderness while blood pressure at presentation was 122/63 mm Hg, pulse rate was 104 bpm, oxygen saturations were 100% and temperature

was 36.8°C. Blood glucose measurement was 7.0 mmol/l and urine dipstick demonstrated low-level haematoproteinuria (1 + blood, trace protein).

Serum biochemistry confirmed AKI (Table 1); creatinine had been normal at 78 µmol/l only 4 months prior but was 187 µmol/l at presentation, rising to 451 µmol/l, 48 h later. Liver function tests (LFTs) demonstrated hepatic injury with an alanine transaminase level of 20,348 units/l, but a normal alkaline phosphatase measurement of 120 units/l. Prothrombin time was raised at 35 s in keeping with hepatocellular injury, whilst C-reactive protein was only mildly raised at 16 mg/l. Lactate dehydrogenase (LDH) levels were markedly raised at 9,924 IU/L and venous lactate was also raised at 2.9 mmol/l, suggestive of tissue injury. Creatinine kinase measurements were only mildly raised at 351 unit/l and returned to normal within 24 h of presentation.

Microscopic examination of the urine failed to demonstrate red blood cell casts or other features of glomerulonephritis. A 12 lead electrocardiogram and posterior-anterior chest X-ray were also normal. Doppler-assisted abdominal ultrasound performed the day following admission demonstrated normal appearances of both kidneys and bladder with only mild diffuse fatty infiltration of the liver.

Other tests including viral serology (hepatitis A, B, C, D, Epstein Barr Virus, Cytomegalovirus), HIV tests, leptospirosis IgM and cultures, and anti-streptolysin O titre were all normal (Table 1). Blood and urine cultures failed to support growth of any organisms. Autoantibody measurements including liver-kidney microsomal, mitochondrial M2, scleroderma and lupus antibodies were all normal (Table 1).

Table 1. Laboratory parameters

Urea	12.8 mmol/l	Hemoglobin	14.7 g/dl
Creatinine	187 µmol/l (peak 451 µmol/l)	Platelets	176
Sodium	137 mmol/l	White count	7.3
Potassium	3.8 mmol/l	Lymphocytes	0.82
Bicarbonate	27 mmol/l	Neutrophils	6.29
Glucose	7.4 mmol/l	Eosinophils	0.02
Ionised calcium	1.0 mmol/l	Haptoglobin	0.67 g/l
Phosphate	0.88 mmol/l	Anti streptolysin O	182 IU/ml
CRP	16 mg/l		
Lactate	2.8 mmol/l	Blood culture	Negative
ALT	20,348 units/l	Urine culture	Negative
Bilirubin	82 µmol/l		
Albumin	43 g/l	Legionella culture	Negative
Alkaline phosphatase	120 units/l	Leptospira IgM	Negative
LDH	9,979 units/l	CMV IgG	Negative
PT	31 s	EBV IgM	Negative
APTT	31 s	ANA antibody	Negative
Fibrinogen	2.9 g/l	Mitochondrial antibody	Negative
Hepatitis A IgM/IgG	Negative	Smooth muscle antibody	Negative
Hepatitis B antibody	Negative	Liver-kidney antibody	Negative
Hepatitis C antibody	Negative	Ds-DNA antibody	Negative
Hepatitis D/E IgM	Negative	Sm antibody	Negative
Magnesium	1.3 mmol/l	Ro/La antibodies	Negative
Paracetamol	Undetectable	Scl-70 antibodies	Negative
Creatine kinase	351 unit/l	Centromere antibodies	Negative
		EBNA antibody	Negative

Note: Values shown are those at the point of presentation unless otherwise stated.

Seventy-two hours after admission, serum creatinine dropped to 427 µmol/l and the patient became polyuric. Creatinine continued to resolve to 201 µmol/l at the point of discharge 6 days later. Within a further 2 weeks creatinine normalised to 89 µmol/l. At no point did the patient become sufficiently acidotic, hyperkalaemic, uraemic or fluid overloaded to require acute haemodialysis. Whilst the high PT was successfully treated with vitamin K, other liver function tests also normalised during the follow-up period.

3. Discussion

Most studies on near drowning report complications such as cardiopulmonary arrest, multiorgan failure, pulmonary oedema, pneumonia and hypoxic brain injury. Other reports highlight rarer haematological complications including haemolysis and coagulopathy (Layon & Modell, 2009). Only very rarely is near drowning reported to cause AKI. Our case of near drowning, together with other published reports, demonstrates the importance of assessing renal function in such casualties by measurement of urine output and serum biochemistry, even days after the event.

With regard to the previously described prognostic indicators for AKI associated with near drowning, our patient did not have a low bicarbonate level or a raised lymphocyte count. He did have dipstick haematoproteinuria at presentation, a non-specific indicator of renal injury, which completely resolved by the time of discharge. Whilst the previous case series of 15 patients reported a correlation between severe AKI and higher levels of dipstick proteinuria, our case exhibited only trace levels of dipstick proteinuria but severe AKI. The normal bicarbonate level, trace proteinuria and normal lymphocyte count, makes our case unique from the published series (Spicer et al., 1999) and we therefore suggest that the use of these prognostic factors should undergo prospective evaluation in near drowning to determine their predictive value for AKI.

The nature of this patient's presentation with contemporaneous hepatocellular injury and oliguric AKI, together with the time to renal recovery, heralded by polyuria, are highly suggestive of ATN. In the absence of drugs, toxins or sepsis, renal ischaemia is the remaining cause of this ATN which corresponds with the raised LDH and lactate levels arising from ischaemia. The precise mechanism(s) underlying this ischaemia remains undefined.

We suggest that where no other precipitant can be identified, cases of even severe AKI associated with near drowning can be managed with appropriate supportive care, including haemodialysis where indicated. We also propose that such patients could be spared a renal biopsy procedure, with the attendant haemorrhagic risks from deranged coagulation, which is unlikely to alter the management beyond supportive care in the context of ischaemic ATN. As shown here, in the absence of secondary complications of AKI or other adverse events, normalisation of estimated GFR should be an anticipated long-term outcome for these patients.

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