CASE REPORT

Acute kidney injury caused by decompression illness successfully treated with hyperbaric oxygen therapy and temporary dialysis

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Abstract A 52-year-old Japanese male professional diver was referred to our hospital for decompression illness (DCI). After 1 h of diving operation at 20 m below sea level, he complained of dyspnea, chest pain, and abdominal pain. He dove again, intending to ease the symptoms, but the symptoms were never relieved. He dove for a total of 4 h. No neurological abnormalities were observed. Computed tomography images revealed portal venous gas and mesenteric venous gas, in addition to bubbles in the femoral veins, pelvis, lumbar canal, intracranial sinuses, and joints. Hyperbaric oxygen therapy (HBOT) was immediately administered. His symptoms improved after the first course of HBOT, however, the patient had anuria for almost 36 h after admission and exhibited acute kidney injury (AKI). Serum creatinine and creatine kinase (CK) levels were increased to maximal values of 6.16 mg/dL and 18,963 U/L, respectively. Blood flow signals were not detected on kidney Doppler ultrasound. We considered that AKI was caused by blood flow impairment and capillary leak syndrome due to DCI in addition to rhabdomyolysis secondary to arterial gas embolism in the skeletal muscles. Temporary dialysis was required to correct the acidemia and electrolyte disturbance. Diuretic phase was initiated, and the patient was put off dialysis on day 3. Serum creatinine and CK levels returned to normal on day 11. He

was successfully treated without any complications. Although AKI is a rare manifestation, we should consider AKI risk in patients with severe DCI.

Keywords Acute kidney injury · Arterial gas embolism · Decompression illness · Decompression sickness

Introduction

Decompression illness (DCI) describes various symptoms resulting from intravascular and extravascular bubbles formed by decompression [1]. It is classified into two categories: decompression sickness (DCS) and arterial gas embolism (AGE). DCS results from tissue damage and circulatory disturbance caused by bubble formation by the dissolved inert gas. It is further divided into type I (mild form; involving only the skin, musculoskeletal system, and lymphatic system) and type II (serious form; involving other organs such as the inner ears, central nervous system, and lungs: the chokes and circulatory system). AGE is caused by arterial gas from the alveolar gas and venous gas [2]. Each type occurs because of sudden decompression. DCI could be a life-threatening disease even with appropriate treatment. Hyperbaric oxygen therapy (HBOT) is a standard treatment for DCI [3]. Although the US Navy Recompression Therapy Table (USNTT) 6 is commonly used for the treatment of type II DCS [4], there are a limited number of hospitals in Japan that can provide USNTT 6, because DCI is not a common disease in our country and the maintenance cost of the equipment is high.

A retrospective study of scuba diving-associated DCI in the US military committee in Okinawa, Japan, showed that the annual incidence of DCI and fatalities were 13.4 and 1.3 cases per 100,000 dives, respectively. The overall

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scuba diving-associated DCI was estimated to be 1 case per 7400 dives [5]. The occurrence of acute kidney injury (AKI) in association with DCI is rare. We encountered a case of severe DCI with AKI, which was successfully treated with HBOT and temporary dialysis. Our case report proposes the risk consideration of AKI, especially in severe DCI patients.

Case report

Herein, we report the case of a 52-year-old Japanese male professional diver with more than 30 years of experience in diving operations conducted at 20 m below sea level. He was working alone to detach the lodged anchor of a ship from another's anchor. It required 1 h to finish. When he came up to the surface, he soon experienced dyspnea, abdominal pain, and chest pain. He also felt lightheaded and fatigued. However, he did not go to the hospital directly. Instead, he dove again, intending to ease the symptoms. He initially dove deeper than 20 m below sea level and then repeatedly ascended and dove in accordance with his symptoms. After almost 3 h of diving, he finally reached the surface. His symptoms were relieved immediately after the diving, but they subsequently recurred. He was immediately taken to a local hospital, and DCI was suspected. Because there was no equipment for administering HBOT at the hospital, the patient was referred to our hospital. However, there was a considerable distance between hospitals, and the travel time was 3 h. Thus, the patient arrived at our hospital >6 h after the onset of his initial symptoms. He had a history of one episode of DCS, which occurred at the age of 32 years. Hospitalization was not needed at that time. Other history was unremarkable, except for well-controlled hypertension. His maintenance medication was irbesartan (100 mg). He smoked two packs of cigarettes per day.

At initial presentation, his height was 175 cm, body weight was 97.4 kg, and body mass index was 31.8 kg/m². He was afebrile (37.1 °C) and normotensive (104/65 mmHg), but had sinus tachycardia (129 beats per min), tachypnea (respiratory rate, 24 breaths per min), and oxygen desaturation (SpO₂, 95% with the administration of 8 L oxygen via a mask). The patient had generalized mild edema, and the inferior vena cava (IVC) was found to be collapsed (≤5 mm) on ultrasound evaluation. Cutis marmorata [6] was observed on the lower extremities. No neurological abnormality was observed. Computed tomography (CT) images revealed portal venous gas and mesenteric venous gas, in addition to bubbles in the femoral veins, pelvis, lumbar canal, intracranial sinuses, and joints (Fig. 1). Gas bubbles in kidneys were not found on CT images. Electrocardiography showed no ischemic

changes. Echocardiography showed no wall motion abnormalities and right-to-left shunt.

Arterial blood gas examination showed non-compensated metabolic acidosis (pH, 7.358; pCO₂, 24.8 mmHg; pO₂, 79.5 mmHg; HCO₃⁻, 13.6 mmol/L) with lactate accumulation (6.36 mmol/L). His blood count was as follows: white blood cells (WBC), 40,300/μL; red blood cells, 603 × 10⁴/μL; hemoglobin (Hb), 19.3 g/dL; hematocrit (Hct), 56.7%; and platelets, 28.2 × 10⁴/μL. He had no electrolyte disturbance, but had renal impairment (blood urea nitrogen, 18.8 mg/dL; creatinine, 2.35 mg/dL) and hypoalbuminemia (serum albumin, 3.0 g/dL). Slight elevation of creatine kinase (CK, 444 U/L) was observed. Coagulation test was almost normal (prothrombin time-international normalized ratio, 1.08; activated partial thromboplastin time, 35.6 s; and fibrinogen, 414 mg/dL).

Based on his clinical manifestations, laboratory tests, and CT images, he was diagnosed with severe DCI. We immediately performed HBOT. USNTT 5 was used (2.8 atm for 2.5 h) due to the limitation of the faculty. After the first course of HBOT, an arterial line was placed to monitor the patient's blood pressure. Bubbles entered the line, and the risk of AGE was considered. His symptoms improved after the first session of HBOT, and we planned two sessions of HBOT per day for immediate improvement.

However, CK was remarkably elevated (18,963 U/L), and renal function was worsened on day 2. Creatinine was increased by 4.23 mg/dL. Arterial blood gas evaluation indicated that acidemia was also worsened (pH, 7.285; pCO₂, 22.8 mmol/L; pO₂, 99.3 mmol/L; HCO₃⁻, 10.6 mmol/L with 2 L of O₂ via a nasal cannula), and hyperkalemia (6.1 mEq/L) was observed. Blood flow was not detected on kidney Doppler ultrasound (Fig. 2). Although sufficient amount of fluid infusion was continued to correct intravascular volume depletion by monitoring IVC diameter, the 24-h urine output after admission was almost zero. The patient had stage 3 AKI according to clinical practice guideline on AKI 2012 of the Kidney Disease: Improving Global Outcome [7]. We considered emergent dialysis to correct acidemia and electrolyte disturbance of the patient. Because the patient was hemodynamically unstable, we started continuous renal replacement therapy (CRRT). The conditions of CRRT were as follows: mode, continuous hemodiafiltration; dialysis membrane, SHG-1.0 (Toray Medical, Tokyo, Japan); and anticoagulant, nafamostat mesilate. The blood, dialysate, substitute, and filtration flow rates were 100 mL/min, 400 mL/h, 400 mL/h, and 800 mL/h, respectively. After 12-h CRRT, the diuretic phase was initiated, and the patient was off dialysis on day 3. The results of urinalysis on day 3 were as follows: urine specific gravity, 1.016; urine myoglobin, 3800 ng/mL; urine β₂-microglobulin,

Fig. 1 Computed tomography images on the day of admission. **a** Massive portal venous gas. **b** Symmetrical femoral vein bubbles. **c** Mesenteric venous gas. **d** A bubble within the lumbar canal.

e Bubbles in the shoulder joint and humerus. **f** Bubbles in the hip joint and femur. **g** A bubble in the superior sagittal sinus and **h** intracranial bubbles

1281 $\mu\text{g/L}$; urine *N*-acetyl- β -D-glucosaminidase, 31.8 U/L; urine sodium, 99 mEq/L; and FE_{Na} , 1.99%. These test results supported the presence of rhabdomyolysis, and AKI in this case was not mainly caused by prerenal failure. On the same day, follow-up CT was performed, and we

confirmed that the venous gas and bubbles had disappeared. Creatinine was increased by maximal value of 6.16 mg/dL on day 3 but returned to normal on day 11 (0.86 mg/dL). CK level, WBC count, and Hb level also decreased to normal ranges. HBOT was reduced to one

Fig. 2 Kidney ultrasound images on day 2. We used Aplio™ 300 (Toshiba Medical Systems, Tochigi, Japan) for the evaluation. (The upper figures) In the pulse wave Doppler mode, the structure of the renal artery was visualized; however, blood flow signals were weak

session per day from day 7 and completed on day 11. The patient was discharged on day 12 without any complications.

Discussion

In DCI, bubbles may cause both ischemic and reperfusion injuries by impairing the vascular endothelium [8]. Increasing sheared stress may lead to the release of endothelial membrane fragments, also known as microparticles (MPs), which appear to activate neutrophils and interact with the platelet membrane [9]. Increased MP levels have been observed in patients with renal diseases and have been associated with arterial and endothelial dysfunction [10]. Impaired endothelial function may cause hypovolemic shock owing to extravasation of plasma. Gempp et al. reported a case of capillary leak syndrome (CLS) in a recreational diver [11]. CLS could be caused by increased vascular permeability resulting from endothelial injuries caused by gas bubbles and causes hemoconcentration [11]. AKI is commonly observed in patients with CLS, and appropriate fluid management is an essential part

and undetectable. (The lower figures, on the right) Even in the Superb Micro-Vascular Imaging mode, which can detect slow blood flow, there was no detectable blood flow in the renal parenchyma

of the treatment [12]. The Hct increases significantly after diving, and this increase is weakly correlated to the maximum depth of the dive [13]. In the current case, we considered that remarkable leukocytosis and hemoconcentration were caused by increased neutrophil activation due to MPs and intravascular volume depletion due to CLS.

Rhabdomyolysis is caused by injury to the skeletal muscle fibers and AKI is a serious complication. Myoglobin is a causative agent of renal impairment [14]. Rhabdomyolysis associated with diving could be caused by AGE in skeletal muscles. Smith et al. reported a correlation between serum CK elevation and clinical outcomes, and they suggested CK as a biological marker for evaluating the size and severity of the gas embolism [15]. In the present case, we considered bubbles in the arterial line as the evidence of AGE.

We could find only three case reports of AKI caused by diving-associated DCI in the English literature [16–18] (Table 1). We considered that rhabdomyolysis, secondary to AGE, was not the main cause of AKI in this case, because the patient had renal impairment and anuria, even with slightly elevated CK levels initially. Viecelli et al.

Table 1 Comparison of previously reported three cases of acute kidney injury caused by decompression illness and the present case

	Case 1	Case 2	Case 3	Case 4
Reference	[16]	[17]	[18]	The present case
Reported year	2014	2015	2017	2017
Age	27	29	61	52
Sex	M	M	M	M
Diving depth	15 m	30 m at maximum 20 m on average	54 m at maximum on the first dive 48 m at maximum on the second dive	20 m
Diving time	15 min (three times of ascent)	30 min	N/A	4 h in total
Initial CK level (U/L)	N/A	909	N/A	444
Peak CK level (U/L)	893	1289	N/A	18,963
Initial creatinine level (mg/dL)	1.36	1.92	1.87	2.35
Peak creatinine level (mg/dL)	13.7	2.62	1.87	6.16
Clinical symptoms	Blurred vision and paralysis followed by abdominal pain	Tachypnea and tachycardia	Fatigue, vertigo, nystagmus, weakness of the lower extremities, hypotension, and tachycardia	Dyspnea, chest pain, abdominal pain, lightheadedness, fatigue, and myalgia
Other findings	Tc-99 m-MAG3 scan result was consistent with ATN	Increased bilateral parenchymal echogenicity of kidney on ultrasound	Hemoconcentration (Hct, 65% and Hb was 22.1 g/dL), leukocytosis (WBC, 40,450/ μ L), PTE on CT scan without signs of DVT, multiple cerebral thromboembolism on MRI, and PFO on TEE	Portal venous gas, mesenteric venous gas, bubbles in veins, cerebral venous sinuses, and lumbar canal and joints No signal of blood flow on kidney ultrasound
Treatments	Fluid infusion with HBOT	Normal saline infusion without HBOT	Fluid infusion with catecholamine use with HBOT	Fluid infusion with HBOT and temporary dialysis
Clinical outcome	Complete recovery	Complete recovery	Slight unsteadiness remained	Complete recovery

CK creatine kinase, M male, N/A not available, Tc-99 m-MAG3 technetium-99 m-mercaptoacetyltriglycine, ATN acute tubular necrosis, HBOT hyperbaric oxygen therapy, Hct hematocrit, Hb hemoglobin, WBC white blood cell, PTE pulmonary thromboembolism, CT computed tomography, DVT deep venous thrombosis, MRI magnetic resonance imaging, PFO patent foramen ovale, TEE transesophageal echocardiography

performed a renogram on a DCI patient with AKI; the result was compatible with that of acute tubular necrosis (ATN) [16]. In that case, the authors concluded that ATN was caused by DCI, not rhabdomyolysis, because CK was not remarkably high (maximum of 893 U/L) and myoglobinuria was negative [16]. In a case similar to ours, Kalpa et al. reported a severe case of DCI with AKI that showed remarkable hemoconcentration (Hct 65%), indicating the existence of CLS [18]. We could not find any published case reports in which kidney biopsy was performed. Pathological evaluation of the kidneys in patients

with DCI has been rarely reported, even including autopsy case reports. Ninomiya et al. reported an autopsy case of a patient with DCI in his 50s who showed a small number of fat emboli in the glomeruli, arterioles, and tubules [19]. The authors concluded that the fat emboli were an evidence of DCI [19]. In an old animal experimental study, gas bubble emboli were observed in the renal artery and vein [20]. In another study, air emboli in the kidney veins were observed microscopically in a specimen taken from a stranded beaked whale that showed DCI-like lesions, similar to those in humans and laboratory animals [21]. In

the current case, the exact cause of AKI could not be ascertained histologically because kidney biopsy was not performed due to the improvement of renal function soon after the diuretic phase. CT with contrast was not performed to evaluate renal blood flow in the present case because of the risk of contrast-induced nephropathy. Although the kidney ultrasound findings were insufficient to determine the cause of renal blood flow impairment, we considered that it was caused by air emboli in the arterioles and venules, in addition to possible fat emboli that were undetectable by CT scan and could be among the causes of AKI. Intravascular volume depletion due to CLS with a long time course until the initiation of fluid treatment and rhabdomyolysis due to AGE were also causes of AKI in the present case. Xu et al. reported associations between short latency time from surfacing to the onset of symptoms and severity of DCI in addition to delay of HBOT and poor clinical outcome in a large single center study [22]. Other predisposing factors in this case were the use of angiotensin II receptor blocker and smoking history. Buch et al. reported that cigarette smokers tended to experience more severe DCI symptoms than non-smokers [23].

We reported an interesting case of AKI caused by DCI that was successfully treated with HBOT and temporary dialysis. Despite the presence of severe manifestations with short latency time and delayed initiation of HBOT, our patient had a good clinical course. Although AKI is not well recognized and commonly reported as a complication of DCI, we should consider the risks of AKI, especially in patients with severe DCI. To ascertain the exact etiology of AKI in DCI patients, further clinical studies are needed, including kidney biopsy.

Compliance with ethical standards

Conflict of interest The authors declare no conflict of interest associated with this manuscript.

Human and animal rights The article does not contain studies with human or animal participants.

Informed consent Informed consent was obtained from the patient.

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