Abstract
Decompression illness (DCI) develops during or after diving. Pulmonary decompression illness (‘Chokes’) is rarely seen because the affected individual usually dies in the water. We encountered a rare and interesting case. A 60-year-old man complained of leg pain after diving. Despite rapid transfer to a nearby hospital, advanced respiratory failure and shock had set in. He was then transferred to our hospital for hyperbaric oxygen therapy (HBOT). On account of his poor general condition, we initially treated him in the intensive care unit without HBOT, where he showed extreme hyperpermeability and a high level of serum procalcitonin (PCT; 20.24 ng/mL). Despite large-volume fluid therapy, severe intravascular dehydration and shock status remained. We assume that the injured endothelial cells induced vascular hyperpermeability and increased levels of inflammatory cytokines leading to the high serum PCT level. PCT might be a useful stress marker of endothelial damage and severity in DCI, including Chokes.

Key words: chokes, decompression illness, hyperbaric oxygen therapy, intensive care unit, procalcitonin, vascular hyperpermeability.

Case
A 60-year-old man was diving for fish using a hookah (air-line diving system). He was nearly 30 m below the surface for about 3 h. He rose to the surface after finishing his job and soon afterward complained of lower leg pain. He was taken to a hospital nearby, where air bubbles were detected in the right ventricle using ultrasound; plain computed tomography showed multiple air bubbles in the subclavian, inferior mesenteric and femoral veins. Immediately, he was transferred to our hospital for hyperbaric oxygen therapy (HBOT). On arrival, he was in severe shock and was intubated. Blood pressure was 85/60 mmHg despite administering maximal doses of dopamine, dobutamine and noradrenaline. Moreover, vasopressin was administered at 6 U/h. Although i.v. crystalloid (12 000 mL) and colloid (1000 mL) were given, a collapsed inferior vena cava and haemoconcentration persisted (haemoglobin, 17.4 g/dL; haematocrit, 51.9%). Serum procalcitonin (PCT) level was 20.24 ng/mL without any evidence for bacterial infection. Pulse oximetry saturation reached
88% and chest radiography revealed bilateral lung effusion (Fig. 1). We diagnosed ‘Chokes’ (pulmonary decompression illness) and continued treatment in the intensive care unit (ICU). Because he showed no neural defects and getting worse about blood pressure, we decided not to perform HBOT. However, he continued i.v. colloid and used airway pressure release ventilation to combat respiratory failure. Ten days later, he was extubated and discharged from the ICU, and 17 days following this he was discharged from our hospital, ambulatory with no subsequent complications.

Discussion

Decompression illness (DCI) shows various clinical manifestations. Pain is the most common symptom, and Chokes is characterised by the triad of substernal pain, cough and dyspnoea. In the clinical setting, however, all three symptoms rarely show simultaneously, and are sometimes misdiagnosed (e.g. immersion pulmonary oedema or water aspiration). Chokes is a rare complaint (only 2% of all DCI cases); almost all patients die in the water before treatment can be given. Therefore, we rarely encounter this condition. In fact, the chief complaint in the present case was lower leg pain instead of typical symptoms. This could easily be misdiagnosed, but respiratory failure and the oxygen saturation level both became progressively worse. A diagnosis of water aspiration was ruled out by the history of his present illness (i.e. no evidence of drowning and symptoms did not appear until he was out of the water). Immersion pulmonary oedema is seen after only a few minutes in the water, and typically, the symptoms start before the ascent. We ruled out these differential diagnoses and correctly diagnosed Chokes, enabling immediate commencement of appropriate treatment. Because he was unstable, HBOT was not administered; otherwise, HBOT using the US Navy treatment table 6 regimen could have been used. However, this therapy is time-consuming (4 h, 53 min) and occasionally puts the patient in danger in case of sudden worsening in the HBOT chamber. Administering HBOT to critically ill patients requires specialised equipment and personnel with ICU skills and knowledge of the physiology and risks unique to HBO exposure. In our hospital, it takes so much time to transfer from ICU to HBOT chamber. Having considered the risks and benefits of HBOT, we opted not to use it on safety grounds. He was haemodynamically unstable and showed no neurological defects.

Severe hyperpermeability was a feature of this case. This was considered to be induced by intravascular air bubbles that damaged the vascular endothelial cells, resulting in severe intravascular dehydration and prolonged shock status, despite the large volumes of i.v. crystalloid administered. Generally, post-DCI shock is caused by loss of i.v. fluid, and DCI accompanied by haemoconcentration usually signifies a poor prognosis. Because most of the crystalloid administered was likely to shift into the pleural and abdominal cavities, that was switched to i.v. colloids administration. After that, the feature of haemoconcentration was gradually improved to the normal condition.

Finally, an interesting aspect was the unusually high level of serum PCT in the absence of bacterial infection (he was diving as usual). We found this incidentally. In general, serum PCT levels increase following bacterial infection. This increase shows the greatest sensitivity and specificity for differentiating patients with septic systemic inflammatory response syndrome. In bacterial infections, inflammatory cytokines (tumor necrosis factor-α, interleukin-1, interleukin-8, etc.) induce the release of PCT, but in viral infections, interferon-γ suppresses PCT production. However, the definite mechanism is unknown; this implies that serum levels of PCT might be high in DCI because of inflammatory cytokines being released following endothelial damage caused by intravascular air bubbles. False positive results for PCT have been reported in newborn babies, burns, heat stroke and other conditions, but none in DCI. Our case therefore suggests that PCT might be a useful marker for diagnosing DCI.

Figure 1. Bilateral lung consolidation and pleural effusion in our patient.
stress marker for endothelial damage and severity of DCI, including Chokes. However, this shows only possibility and we need some more investigation about this.

Competing interests
None declared.

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References