



Decompression Illness Symptoms in a 29-Year-Old Female after a Treatment

Table 9: A Case Report



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Introduction/Background: Arterial gas embolism (AGE) is a rare but serious complication of hyperbaric oxygen therapy (HBOT). The etiology of an AGE following HBOT in a previously healthy patient can be elusive and often subclinical.

Case presentation: A 29 year old female with no previous pulmonary history completed her twelfth routine multiplace HBOT to 45 fsw for complications of surgical wounds. She emerged from the chamber at her normal baseline. During the next 40 minutes, while she received wound care, her mental status began to decompensate. She was placed on high flow oxygen. On exam she was unable to recognize her spouse and she demonstrated a mild, but noticeable, motor imbalance. Immediate chest x-ray revealed no pneumothorax. With the suspected diagnosis of DCI, the decision was made to treat her with a USN Treatment Table 6.

Results: During recompression, her symptoms began to improve as she descended through 45 fsw. When she reached 60 fsw, she had complete resolution of symptoms and her neurological examination was normal. Subsequent workup included a transesophageal echocardiogram with “bubble interrogation” and high resolution CT scan of the chest. No right-to-left shunt could be induced on bubble interrogation. The CT scan however, did show mild regions of air trapping in the bilateral lower lobes not noted on previous chest x-ray report (see Figure 1). This finding was felt to provide an explanation for the patient’s clinical course. She was disqualified from any future diving and instructed not to participate in scuba and snorkeling.

Discussion: Pulmonary barotrauma is a well known complication of compressed air diving.¹ Trapped gas expanding in the alveolar space can damage the surrounding vessels. The resulting intravascular bubbles in the left-sided circulation can become lodged in the end arteries of the systemic circulation (see Figure 2).² The emboli cause pathological changes downstream by two mechanisms: a reduction in perfusion and an inflammatory response to the bubble.³ Emboli that become lodged in the cerebral vasculature may manifest symptoms from mild confusion, headache and minor motor deficit to complete hemiparesis, loss of consciousness, convulsions, coma or even cardiopulmonary arrest. The most important diagnostic criterion is the patient’s history. The clinical suspicion of AGE is based on the initial neurological symptoms and the direct temporal relation between the symptoms and the causative event (in our case a decompression exposure).² Symptoms of AGE due to pulmonary barotrauma typically occur within minutes of the initial insult. In a 2005 Divers Alert Network report, the vast majority of divers with cerebral symptoms usually caused by AGE became symptomatic within one hour (see Figure 3).⁴ There is a reported case of cerebral AGE with symptom latency of 48 hours.⁵ This is believed to be a very rare exception. Initial treatment of gas embolism consists of supportive management, administration of the highest concentration of oxygen available and keeping the patient in the supine position. For AGE, HBOT is the treatment of choice, as soon as cardiopulmonary stabilization has been achieved.²

Conclusions: Mental status change in a patient after a routine hyperbaric treatment is anything but routine. The expression of Boyle’s law in pulmonary barotrauma leading to AGE is well documented in the diving community; however, there is a paucity of reported cases involving HBOT.⁶ This report is offered to remind us that AGE due to pulmonary barotrauma can occur after routine treatments involving the use of hyperbaric oxygen.

References:

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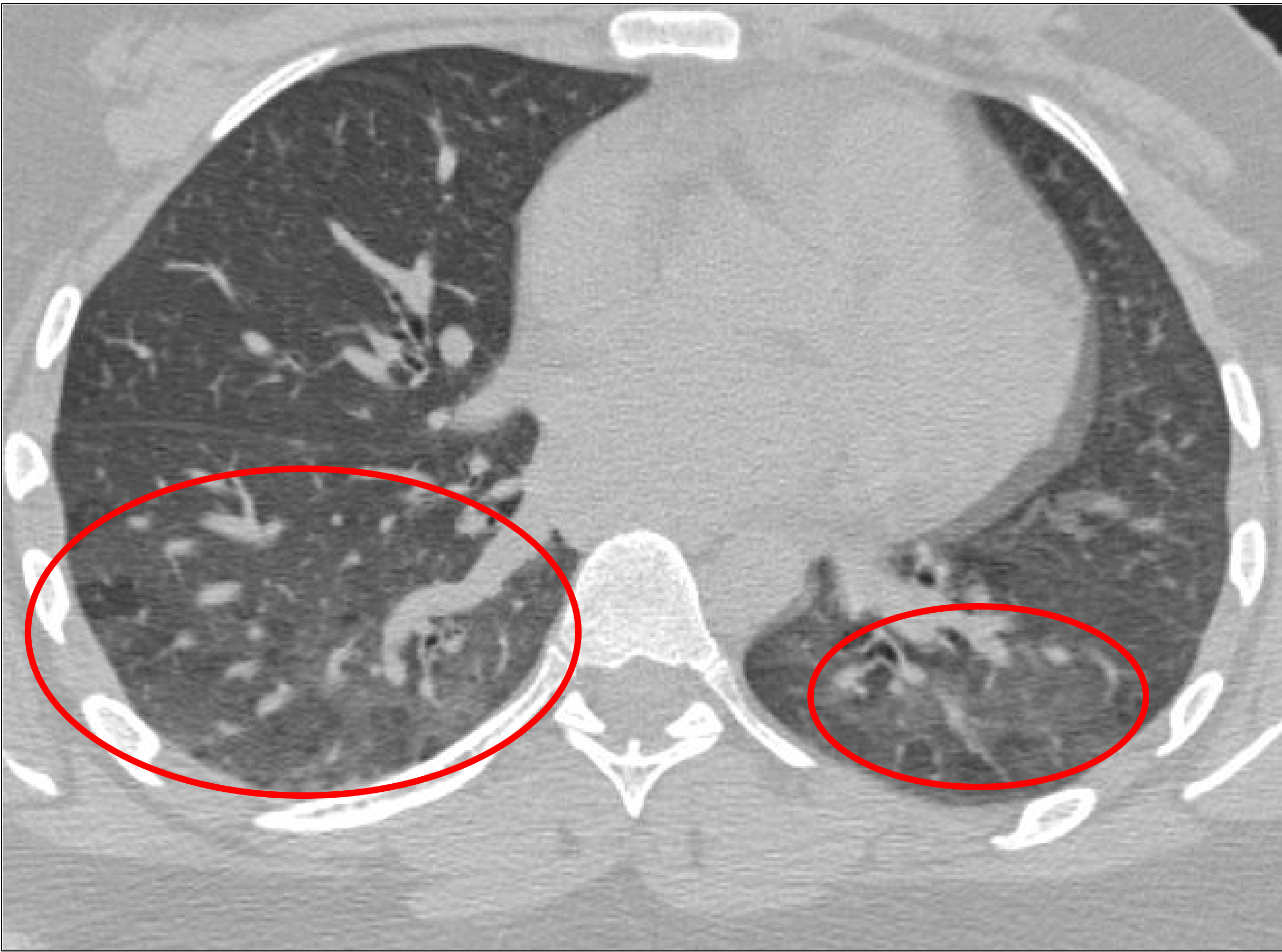


Figure 1. CT scan showing mild regions of air trapping in the bilateral lower lobes.

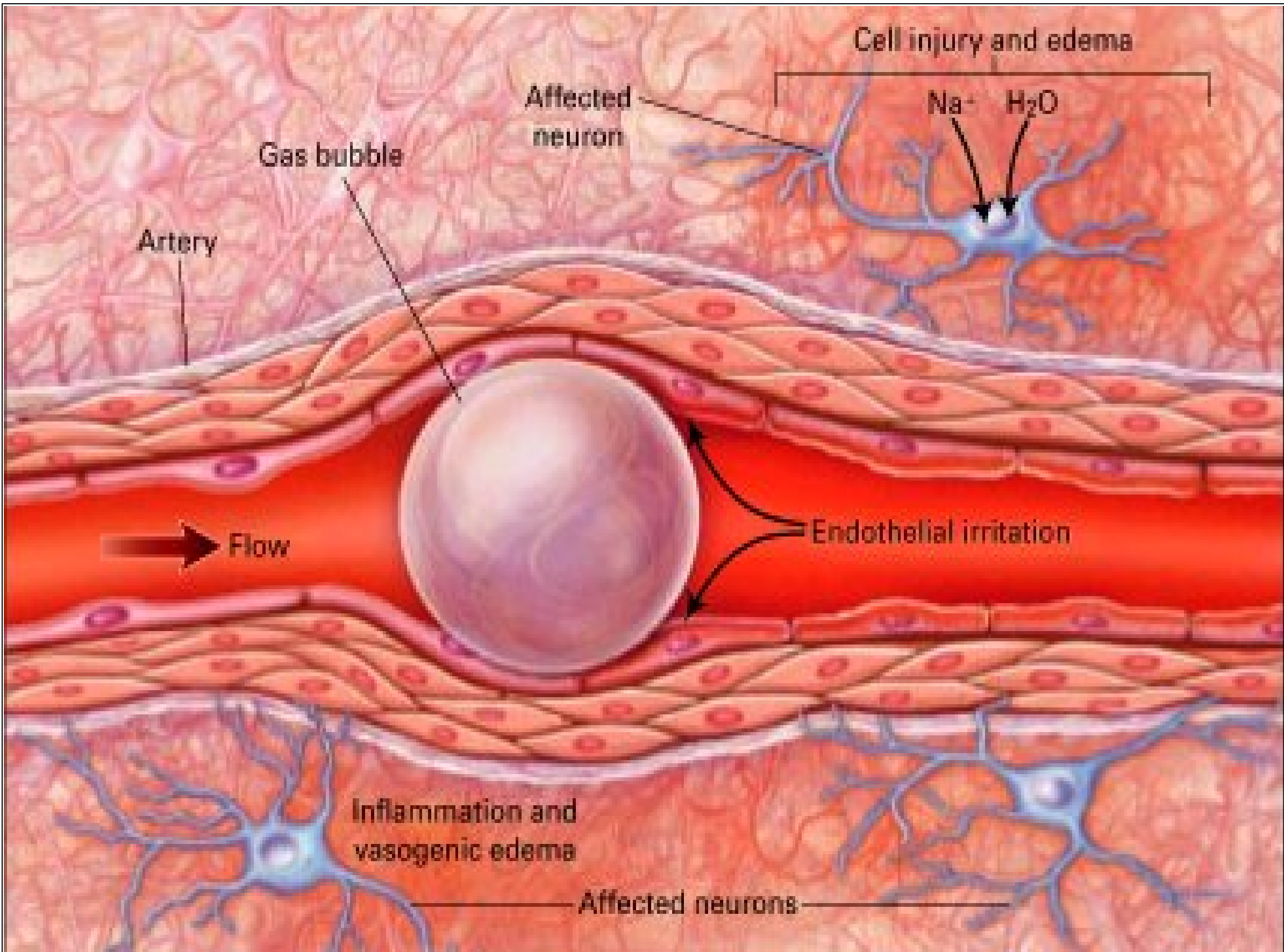


Figure 2. Schematic of a gas embolus lodged in a systemic end vessel.

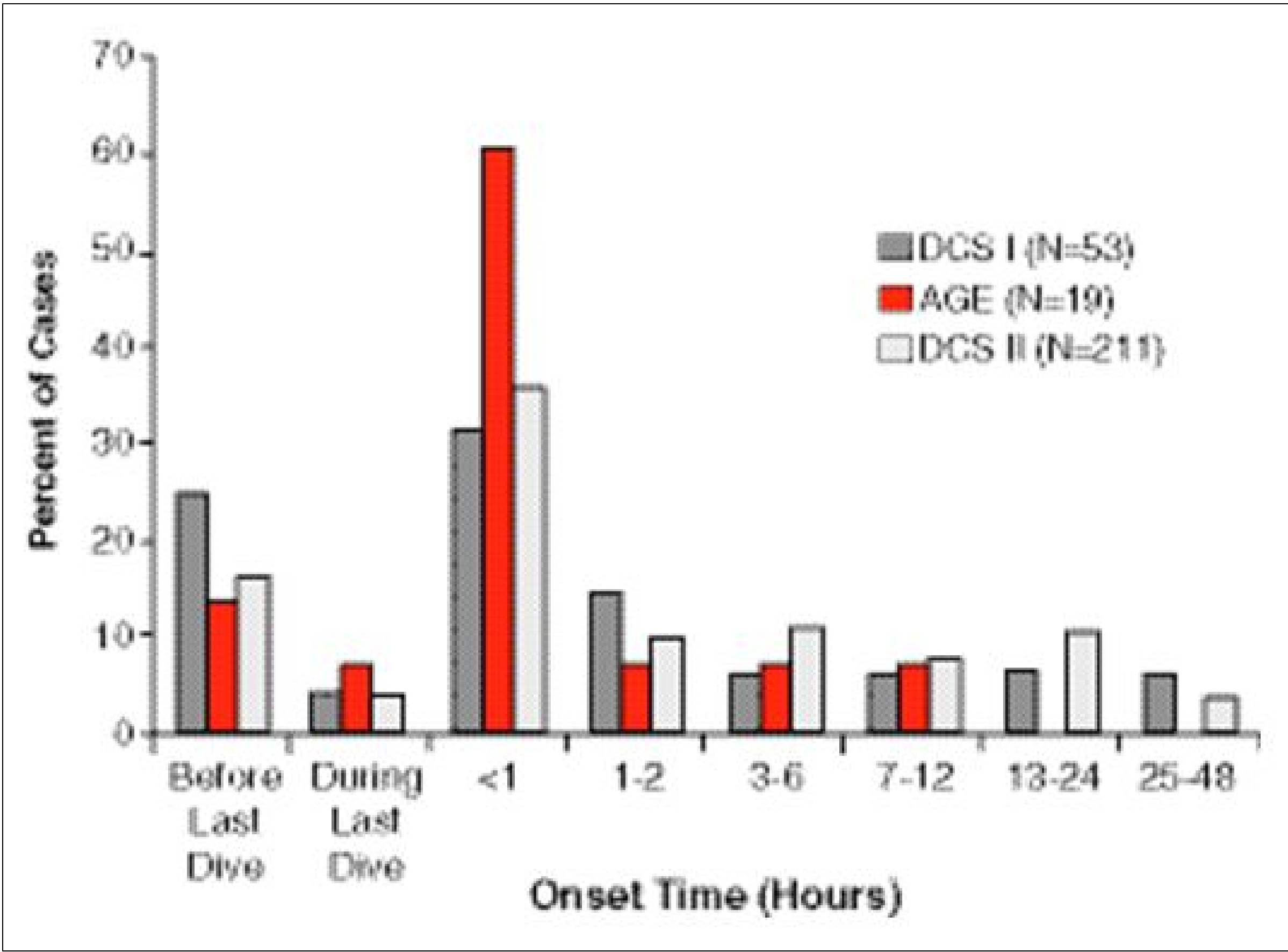


Figure 3. Latency of onset of DCI symptoms.