



# Decompression Illness in a Scuba Diver With Significant Esophageal Injury

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## ABSTRACT

Scuba divers are at risk of decompression illness, of which arterial gas embolism is the most feared consequence. Severe complications involving the gastrointestinal tract are rare. In this report, we describe a case of an experienced scuba diver who was forced to rapidly ascend because of a mechanical failure. His course was complicated by severe esophageal mucosal injury including multiple ulcerations. Although he improved clinically, an esophageal stricture subsequently formed, which required dilation. This seems to be the first case report of significant esophageal injury resulting from decompression illness in a scuba diver.

## INTRODUCTION

Decompression illness (DCI) can occur after scuba diving. The term covers both arterial gas embolism and decompression sickness (DCS).<sup>1</sup> In arterial gas embolism, alveolar gas or venous gas emboli are introduced into the arterial circulation, whereas DCS is caused by in situ bubble formation from dissolved inert gas. Both of these are rare occurrences after diving for which the incidence is decreasing.<sup>1</sup>

In this report, we will describe an experienced scuba diver who, because of a mechanical failure, was forced to rapidly ascend. His course was complicated by significant esophageal injury, something which has not previously been reported.

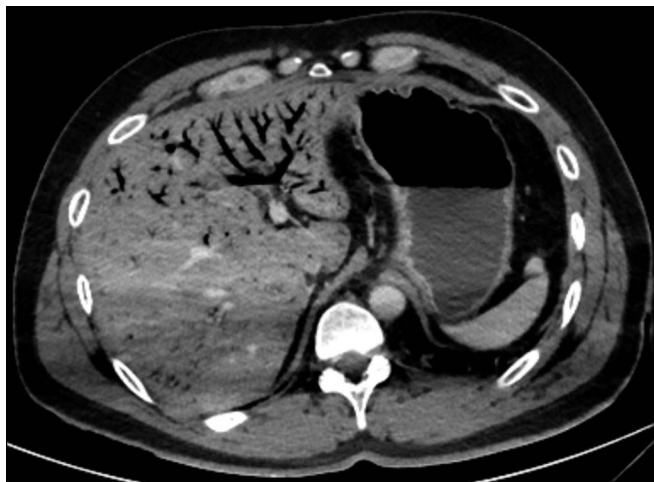
## CASE REPORT

A healthy 37-year-old man, without any significant medical history, was working as a divemaster and scuba instructor. On the day of his presentation, he dove to a depth of 64 m. At a depth of 44 m, mechanical problems occurred, and he was forced to rapidly ascend the rest of the way without stopping. Immediately afterward, he felt acute abdominal pain and numbness of his left leg and tongue. He was immediately transferred to a local hospital.

On arrival, his vital signs revealed a blood pressure of 110/70 mm Hg, pulse rate of 116 bpm, respiratory rate of 20 breaths per minute, and a saturation of only 80% on room air (90% with 2-L oxygen through nasal cannula). He was intubated. Physical examination revealed moderate abdominal tenderness and abdominal distention, as well as cutis marmorata. An emergent computed tomography (CT) scan was performed. The abdominal CT demonstrated branching gaseous foci in the liver, findings consistent with gas filling the entire portal venous system (Figure 1). Gas was also noted in the mesenteric veins, and there was pancreatic edema.

He was transferred to our tertiary medical center for hyperbaric oxygen therapy and intensive care treatment. On admission, laboratory studies showed evidence of rhabdomyolysis (creatinine kinase 6,500  $\mu$ /L [normal 10–170]), acute hepatitis (aspartate aminotransferase 340 [normal 5–38], alanine aminotransferase 400 U/L [normal 4–41]), acute pancreatitis (lipase 345 U/L [normal 13–60] and amylase 179 U/L [normal 28–100]), acute renal failure (creatinine 2.69 mg/dL [normal 0.7–1.2]), elevated lactate dehydrogenase (1,975 U/L [normal 240–480]), and elevated C-reactive protein of 237 mg/L (normal 0.3–5.0). Chest CT was normal with a normal esophageal appearance.

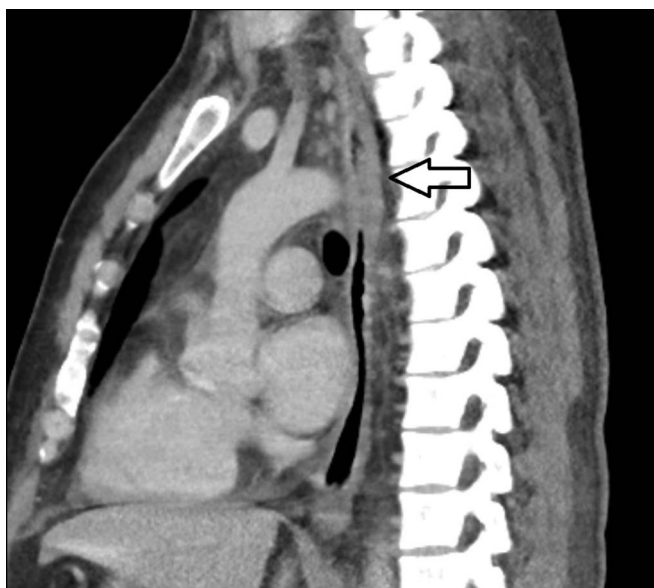
He was treated in the hyperbaric oxygen chamber with significant clinical and laboratory improvement. However, on the fourth day of hyperbaric therapy, he complained of severe chest pain with odynophagia and dysphagia. An emergent CT was performed to



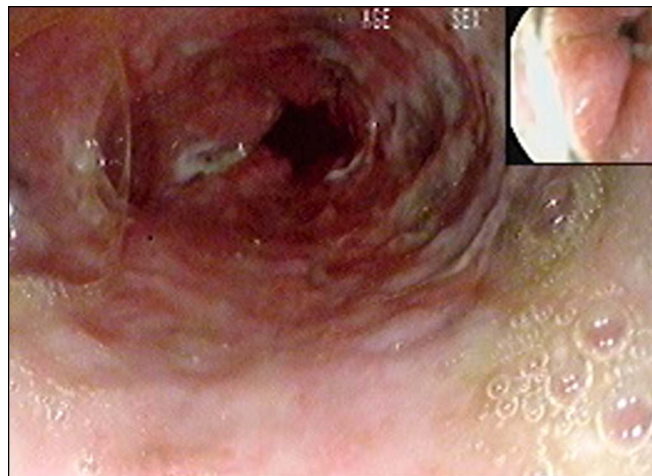
**Figure 1.** Computed tomography scan showed gas filling the portal system.

rule-out a perforation. It showed a thickened esophageal wall without signs of perforation (Figure 2, arrow). A gastroscopy was performed, which demonstrated numerous ulcers with erythematous and friable mucosa in the lower third of esophagus (Figure 3). In addition, a 1 cm clean-based ulcer was noted in the antrum, although the remainder of the gastric mucosa was normal. Esophageal biopsies revealed heavy acute inflammatory infiltrate, hemosiderin and fibrinopurulent exudate, with negative stains for viral and fungal infection (Figure 4). He was treated with high-dose proton pump inhibitors and intravenous fluids, after which clinical improvement was noted. He was discharged after 9 days of hospitalization on esomeprazole 40 mg daily.

Given the severity of his esophagitis, a repeat gastroscopy was planned to ensure adequate mucosal healing. At the time that



**Figure 2.** Computed tomography scan revealed thickened esophageal walls (arrow).

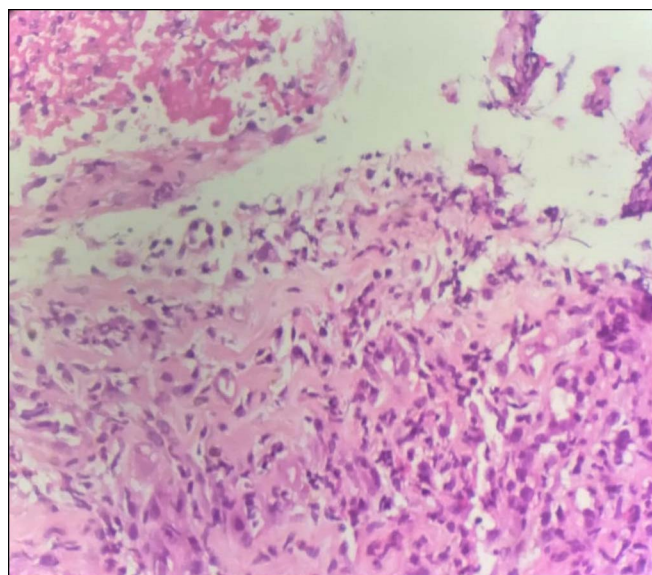


**Figure 3.** Endoscopy revealed multiple ulcers in the distal esophagus with erythematous and friable mucosa.

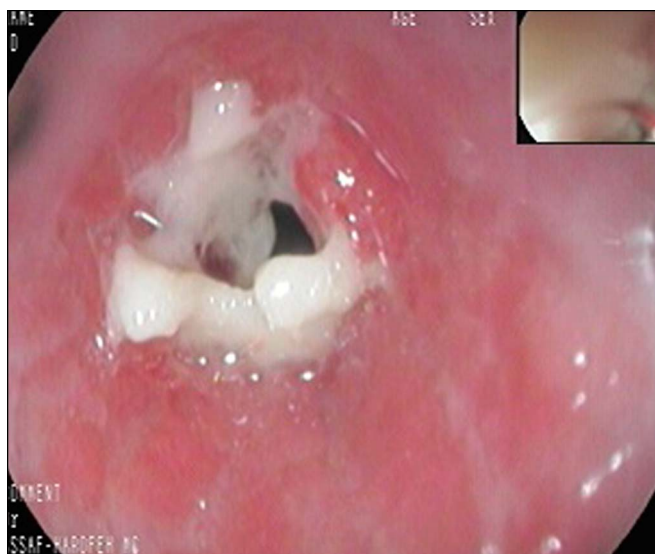
this was performed, 8 weeks after his previous endoscopy, he still reported dysphagia to solid foods. The endoscopy revealed a stricture in the midesophagus with a luminal diameter of approximately 8 mm and length of 3 cm requiring the use of a pediatric gastroscope (Figure 5). Otherwise, the esophageal mucosa seemed normal. The stricture was dilated with a through-the-scope balloon to a maximum diameter of 12 mm. During a telephone conversation a month later when he missed his follow-up appointment, he reported feeling well with no further symptoms. He has not returned to our clinic since.

## DISCUSSION

In this report, we described a case of esophageal injury in a scuba diver. Scuba divers are at risk of several types of injuries.



**Figure 4.** Biopsies from the distal esophagus revealed acute inflammatory infiltrates with hemosiderin and fibrinopurulent exudate (40× magnification).



**Figure 5.** Repeat endoscopy 8 weeks after presentation revealed an esophageal stricture.

Barotrauma is the most common form of diving-related injury and develops when an air-filled body cavity is unable to balance its pressure with the environment because of changes in ambient pressure.<sup>2</sup> Barotrauma usually occurs to a scuba diver during descent, and the lungs and inner ear are the most common sites to be involved.<sup>2,3</sup>

DCI, however, tends to occur to a diver because of the ascent from deep waters.<sup>1</sup> Arterial gas embolism is the most serious potential sequela of this. Arterial gas emboli can result from the passage of gas bubbles into the pulmonary veins and from there into the systemic circulation. Air emboli can damage the pulmonary, cutaneous, musculoskeletal, and lymphatic systems and can be deadly when they enter the cerebral or coronary arteries. The mainstay of treatment for DCI is hyperbaric oxygen therapy.<sup>1,4</sup>

DCS occurs from in situ bubble formation from dissolved inert gas.<sup>1</sup> With DCS, many people suffer from abdominal discomfort, what is often referred to as “the bends.”<sup>1</sup> Additional gastrointestinal symptoms may include nausea, vomiting, abdominal distention, or fecal incontinence.<sup>5</sup> However, only a handful of cases have described severe gastrointestinal manifestations. These have included 3 cases reports of ischemic colitis because of air embolism, 2 cases of gastric perforation, and single reports of mesenteric venous thrombosis, diaphragmatic rupture, and esophageal variceal bleeding.<sup>5–12</sup> The diagnosis of DCI is clinical, although laboratory abnormalities such as elevated creatinine kinase, transaminases, and lactate dehydrogenase are often noted.<sup>13</sup>

To the best of our knowledge, this is the first reported case of esophageal injury because of diving-related complications. The exact etiology of his esophageal injury is not clear. Whether because of an air embolism or DCS, or just given his overall state of inflammation and sickness, he may have been predisposed to

ischemia in the distal esophagus, which has relatively less vascularization compared with the rest of the esophagus.<sup>14</sup> It is possible given his overall condition that he also experienced reflux of gastric content, which contributed to the injury in the distal esophagus. He was treated supportively with fluids and aggressive acid-blocking medication, and thankfully, he had a good outcome.

In conclusion, we present here the first case of significant esophageal injury in a patient with DCI after scuba diving. Physicians treating patients with DCI should be aware of this if their patient develops symptoms of chest pain, dysphagia, or odynophagia.

## DISCLOSURES

Author contributions: E. Avivi and DZ Yovel reviewed the literature and drafted the manuscript. H. Shirin critically reviewed the manuscript. DL Cohen reviewed the literature, critically reviewed the manuscript, and is the article guarantor.

Financial disclosure: None to report.

Informed consent was obtained for this case report.

Received January 4, 2022; Accepted June 6, 2022

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