Massive portal venous gas embolism after scuba diving

Romain Siaffa[1], Marc Luciani[2], Bruno Grandjean[2], Mathieu Coulange[3,4]

[1] *Navy Medical Department, Naval base of Toulon, Toulon, France*
[2] *Department of Hyperbaric Medicine, Notre-Dame de la Miséricorde Hospital, Ajaccio, France*
[3] *Department of Hyperbaric Medicine, Sainte-Marguerite Hospital, APHM, Marseille, France*
[4] *UMR MD2, Aix Marseille University and Institute of Biological Research of the Army, Marseille*

Corresponding author: *Romain Siaffa, Sainte-Anne Military Hospital, 2 Boulevard Sainte Anne, 83000 Toulon, France*
romain.siaffa@intradef.gouv.fr

Key words
Decompression sickness; Liver; Gastrointestinal tract; Hyperbaric oxygen therapy

Abstract
(Siaffa R, Luciani M, Grandjean B, Coulange M. Massive portal venous gas embolism after scuba diving. Diving and Hyperbaric Medicine. 2019 March 31;49(1):61–63. doi: 10.28920/dhm49.1.61-63. PMID: 30856669.)
Introduction: Portal venous gas from a diving injury is an infrequent finding and only a few cases are described. We report a case of severe decompression sickness (DCS) associated with a massive amount of gas in the portal and mesenteric veins.
Case report: A 49-year-old man suffered from DCS after two deep dives on the same day. He presented with cutaneous, neurological and pulmonary symptoms associated with hypoxaemia. He had no abdominal pain. A computed tomography (CT) scan showed large quantities of hepatic and portal venous gas and excluded other explanations for its presence. All symptoms disappeared with hyperbaric oxygen therapy and there were no further complications.
Discussion and conclusion: The role of portal venous gas in DCS is not obvious. Isolated portal venous gas seems to cause no obvious harm. Medical imaging should be considered for differential diagnosis and to prevent some complications, especially in divers presenting with abdominal pain.

Introduction

Gas embolism is one of the main causes of diving injury and can happen after decompression sickness (DCS), in which large quantities of inert gas are released by the tissues. Gas embolism can also occur after lung and possibly hollow organ barotrauma during ascent, especially if the ascent is rapid. The condition may lead to severe neurological disability, and hyperbaric oxygen treatment (HBOT) is needed urgently. In this report, a diver with severe decompression sickness (DCS) exhibited massive and unusual gas formation in the liver and gastrointestinal tract.

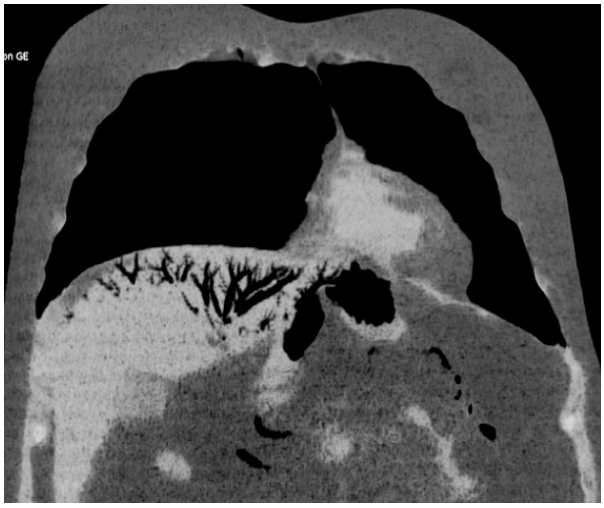
Case report

A 49-year-old, obese (body mass index 34 $kg \cdot m^{-2}$) man who was a moderately experienced diver (three years of diving, 35 dives in the past year) had a first dive to a maximum depth of 60 metres' sea water (msw) (15-minutes (min) bottom time, 52-min total dive time). After three and three-quarter hours resting, he made a second dive. He stayed one min at 40 msw (maximal depth) and 12 min at 25 msw, with a total dive time of 53 min. All decompression procedures were fully respected. He did not ingest a gaseous beverage or heavy meal before diving. Approximately 20 min after surfacing, he experienced asthenia, chest tightness, shortness of breath, vertigo, and mental confusion. He was treated immediately with oxygen (O_2) 15 $L \cdot min^{-1}$ via a mask and transferred to the hyperbaric centre at Ajaccio Hospital, Corsica. On admission, clinical examination revealed vertigo and a pruritic erythematous rash on the abdomen, evoking skin bends. O_2 saturation was 91% on air. Haemodynamic parameters and the rest of the clinical review were normal. Blood tests showed hypoxaemia (P_aO_2 = 55 mmHg), mild haemoconcentration (haematocrit 55%), and C reactive protein (CRP) was 30 $g \cdot L^{-1}$. Because of the initial dyspnoea and hypoxaemia, the patient underwent a computed tomography (CT) scan, which excluded pleural or pulmonary injury (i.e., pneumothorax, pleural effusion, pulmonary oedema, or infection). However, CT showed portal venous gas, looking like an intrahepatic 'dead tree'. There was also gas within mesenteric vessels. No other abnormality was seen on abdominal CT scan. An ENT consultation found that the vertigo was of a central origin.

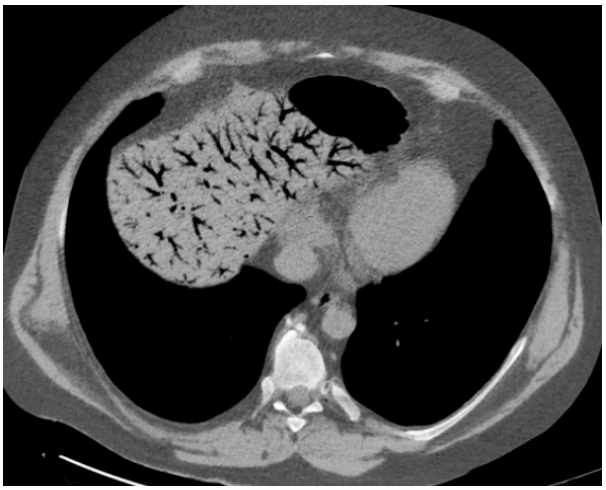
The patient underwent hyperbaric oxygen treatment (HBOT) using a six-hour table (405 kPa maximum pressure, nitrox 80/20, then nitrox 70/30 and finally 100% O_2), intravenous hydration and corticosteroids (40 mg methylprednisone thrice daily, then prednisone 20 mg thrice daily for seven days' total treatment). Four further HBOT (152 kPa, 90 min, 100% O_2) were given, with full recovery of all symptoms and signs of DCS. Transcranial Doppler demonstrated a right-to-left shunt which further investigation confirmed to be a persistent foramen ovale (PFO). He remained asymptomatic and was discharged on the fourth day post incident to return

Figure 1
Abdominal CT; (A) coronal reconstruction and (B,C) axial slides showing portal venous gas with a hepatic 'dead tree' appearance; gas is also present in mesenteric vessels (A, C)

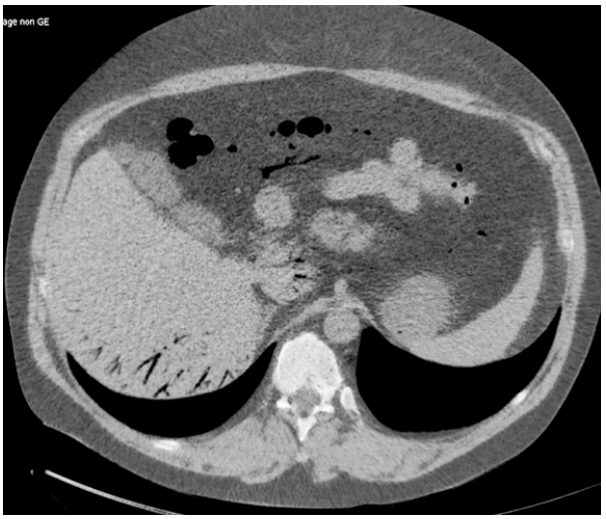
A



B



C



by sea to mainland France. Although now symptom-free, a further four HBOT were administered as an outpatient in Nice. Two months later the PFO was closed and he has returned to diving without any problems.

Discussion

Portal venous gas is a radiological diagnosis. It is defined as a radiolucency of vascular branches within 2 cm beneath the liver capsule. The condition is usually considered a rare and critical disease and may be caused by sepsis (e.g., appendicitis, cholecystitis, diverticulitis), stomach or bowel lining disease (e.g., mesenteric ischaemia, peptic ulcer, inflammatory bowel disease), or an excessive pressure inside hollow organs (e.g., barotrauma, blunt trauma, endoscopy).[1] However, the origin of portal venous gas is not found in 15% of cases. DCS is considered a rare cause of hepatic/portal venous gas, and digestive tract barotrauma (from stomach or bowel) is the primary differential diagnosis.[2] Its pathological significance is not clearly understood and remains controversial. One case has been reported of portal venous gas resulting from barotrauma after a rapid ascent.[3] This occurred after having a heavy meal and gaseous drink just before the dive, but the diver also had symptoms of DCS, and the off-gassing process could also explain the portal venous gas. This case is important because the patient developed a portal thrombosis. Therefore, the authors considered this risk requires further investigation in severe DCS. In another case of DCS exhibiting portal venous gas, abdominal pain was reported in addition to neurological and cutaneous symptoms.[4]

The decision to recompress was driven largely by the presence of neurological symptoms and rash, in addition to the radiological finding of apparently symptomatic portal gas. However, when it is isolated or asymptomatic, the evolution of portal venous gas may be a typically benign process and may be more common than presently appreciated. For example, portal venous gas was found in four of nine divers with DCS investigated with CT for unrelated symptoms such as dyspnoea.[5] Although other case reports have been published, the pathological significance of portal venous gas is not obvious. It seems that a poor prognosis is not related to isolated portal venous gas, but rather to associated severe DCS symptoms. Medical imaging, mainly CT, is essential for differential diagnosis.

We strongly recommend CT scanning when divers present with abdominal symptoms and there is doubt about the cause. CT scans are now easily available in many centres without important loss of time to HBOT. Since a small incremental delay to recompression is unlikely to adversely affect outcome in presentations of typical latency,[6] there seems little potential for harm in pursuing such investigations. A quick, non-invasive device, such as hepatic ultrasound and Doppler, might also be useful, further reducing the delay to recompression.

Conclusion

Portal venous gas may be present in cases of DCS but appears to be a radiological-clinical mismatch; that is, few or no symptoms despite positive CT scan images. Early CT helps in the differential diagnosis and should be performed when divers present with abdominal pain of uncertain cause. A cohort study would indicate the true incidence of portal venous gas after diving.

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Acknowledgement

The diver initially was lost to follow up but subsequently contacted by phone, at which time he gave verbal consent for publication of his case and radiology.

Conflicts of interest and funding: nil

Submitted: 26 April 2018; final revision 08 January 2019
Accepted: 19 January 2019