Clinical problem solving: Mental confusion and hypoxaemia after scuba diving

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Bubbles; Decompression sickness; Differential diagnosis; Hyperbaric oxygen therapy; Respiratory symptoms; Stroke

Abstract
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Introduction: We report a case of a diving accident associating both cerebral symptoms and signs of respiratory impairment after two dives. The objective is to describe the process for obtaining the diagnosis.
Case report: A 52-year-old man experienced mental confusion associated with hypoxaemia after surfacing. All decompression procedures were fully respected. The diver had a spatio-temporal disorientation accompanied by a marked tendency to fall asleep spontaneously. He had no dyspnoea and no cough, but crepitations at both lung bases were found with oxygen saturation at 80%.
Conclusions: In this clinical case, cerebral magnetic resonance imaging and chest computed tomography scan helped to exclude other pathology that would have necessitated urgent transfer rather than urgent hyperbaric treatment. The imaging is particularly useful in case of cerebral and respiratory symptoms following scuba diving.

Introduction

When clinical symptoms are observed after scuba diving, it is important to identify the type of diving accident but also to look for a differential diagnosis. The diagnosis is primarily based on the patient history and diving circumstances. But in certain situations, it may be necessary to carry out first-line complementary tests. Herein, we report a case of a diving accident associating both cerebral symptoms and signs of respiratory impairment after scuba diving. The objective is to describe the process for obtaining the diagnosis.

Case report

A 52 year-old man who experienced mental confusion and hypoxaemia following a dive, was hospitalised at the hyperbaric centre of the Sainte-Anne military hospital in Toulon (France) for seven days in September 2018.

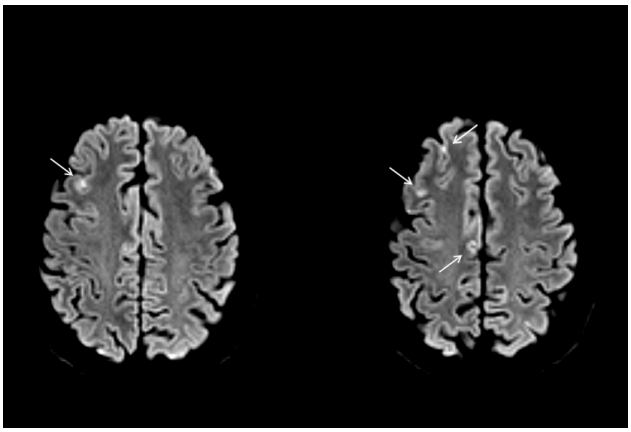
The patient was an experienced scuba diver of German nationality with several hundred dives without incident. He was in the south of France on a diving holiday. He had no significant medical past history, other than past surgical operations for inguinal hernia and nasal septoplasty. There were no known allergies and no medical treatments.

He began his vacation with a first scuba air dive at a maximum depth of 31 metres' sea water (msw) for 35 min total duration with a 3 min safety stop at 5 msw, surfacing at 10:35 am. In the afternoon, a second dive was carried out (surface interval = 4 h) using a nitrox mixture (28% oxygen, 72% nitrogen) to 26 msw for 42 min with a 3 min safety stop at 5 msw. He came out of the water at 3:30 pm without any physical problems reported. All decompression procedures were fully respected. No environmental risk factors were found.

At 4:00 pm he collapsed without loss of consciousness but exhibiting severe fatigue with nausea and pallor. He was treated immediately with oxygen (O_2) 15 L·min^{-1} and transferred by helicopter to the hyperbaric center of Toulon, France. At this point, the diver was confused with a Glasgow coma scale score of 13 but did not have any sensory-motor deficits. His temperature was 37.4°C, pulse 100 beats·min^{-1}, blood pressure 118/81 mmHg, respiratory rate 18 breaths·min^{-1} and O_2 saturation 80%. Blood glucose and the electrocardiogram were normal.

On admission to the hyperbaric centre at 5.30 pm, clinical examination revealed a spatio-temporal disorientation accompanied by a marked tendency to fall asleep

Figure 1
Cerebral MRI scan showing hyperintensities in the right frontal lobe (diffusion sequences)



spontaneously. The patient was stimulated constantly to keep him awake. The neurological examination did not show a sensory-motor deficiency, with no tendon reflex anomalies and a negative Babinski test; cerebellar and vestibular syndrome symptoms were also absent.

The diver remained haemodynamically stable but the peripheral O_2 saturation on air was 80%. He had no dyspnoea and no cough, but the pulmonary examination revealed crepitations at both lung bases. Arterial blood gases taken during air breathing showed hypoxaemia, with the $PaO_2 = 55$ mmHg (7.3 kPa). Chest and cardiac ultrasonography was immediately carried out and did not find any ultrasound lung comets or pneumothorax; however, circulating venous bubbles were detected in the right heart. The blood samples showed a leukocytosis with polynuclear neutrophils at 21,400 per µL (normal < 7700), natriuretic peptides (NT-ProBNP) at 233 ng·L^{-1} (normal < 84), positive D dimers at 1.55 mg·L^{-1} (normal < 0.5), lactate dehydrogenase (LDH) at 413 IU·L^{-1} (normal < 225), while haematocrit and albumin were normal. He had no cholestasis, no hepatic cytolysis and no inflammatory syndrome.

In view of the presenting neurological signs, possible diagnoses were stroke or cerebral decompression sickness (DCS). It was therefore decided that a cerebral MRI should be done urgently, before any recompression, to eliminate the diagnosis of ischaemic or haemorrhagic stroke that would require specific care. In addition, a computed tomography (CT) scan of the chest was performed to eliminate the diagnosis of pulmonary embolism but also to look for signs of immersion pulmonary oedema (IPO) or pulmonary barotrauma.

The cerebral MRI showed the presence of hyperintensities in the right frontal lobe, with the diffusion sequence indicating several cortical foci of ischaemia suggestive of an embolic cause; probably bubbles given the context of diving (Figure 1). No other abnormalities were observed at the cerebral level. A sphenoid-ethmoid sinusopathy with bilateral mastoiditis was also observed.

The CT chest scan did not show any changes to suggest IPO. There was no evidence of a pulmonary embolism. In addition, no pneumomediastinum and pneumothorax were found and so the diagnosis of pulmonary barotrauma was discounted. The presence of bilateral small areas of atelectasis at the pulmonary bases was noted. On the upper abdominal sections, the presence of hepatic portal venous gas was observed (Figure 2), which prompted extension of the investigation to the abdominal-pelvic region. This examination also showed the presence of venous gas in the right femoral vein (Figure 3).

Following the imaging investigations, the patient was recompressed at 7.10 pm for hyperbaric oxygen treatment (HBOT) using the treatment tables and adjunctive medical treatments (including intravenous rehydration with 2 L of normal saline over 5 h, methylprednisolone, acetylsalicylic acid, and lignocaine) currently applicable in our centre. He received an initial 5 h oxygen table (equivalent to a US Navy Treatment Table Six), two further short heliox treatments at 2.8 atmospheres absolute (atm abs) over the next 24 hours, followed by daily HBOT sessions at 2.5 atm abs for five days. Improvement across the treatment period was observed, with the neurological signs of confusion disappearing at the end of the first treatment, while the hypoxaemia gradually regressed over 24 hours.

A secondary work-up was then carried out to investigate the presence of a persistent (patent) foramen ovale (PFO), using transcranial Doppler and trans-oesophageal echocardiography. These tests revealed the presence of a massive spontaneously shunting PFO with an aneurysmal atrial septum. In the following days, a cardiac ultrasound and pulmonary functional testing were normal. A cranio-facial CT scan showed the presence of a left maxillary polyp, with a left ethmoid sinus opacity.

Discussion

Diving accidents bring together a set of varying clinical entities and separate pathophysiological mechanisms.[1,2] The most frequent diving accident see at our centre is DCS, which is thought to be caused by formation of bubbles in the body during decompression.[9]

The central nervous system and especially the spinal cord is frequently affected in DCS.[3] The brain can also be affected as a result of emboli passing from the right to left side of the heart via a PFO, which is found 80% of cerebral DCS cases.[4] The differential diagnosis of brain damage is primarily represented by ischaemic or haemorrhagic stroke that may occur in a diving context.[5]

Pulmonary lesions caused by SCUBA diving are IPO, pulmonary barotrauma, drowning or pulmonary DCS, of which the last is less common. With regard to the current

Figure 2
Abdominal CT scan (coronal reconstruction) showing portal venous gas

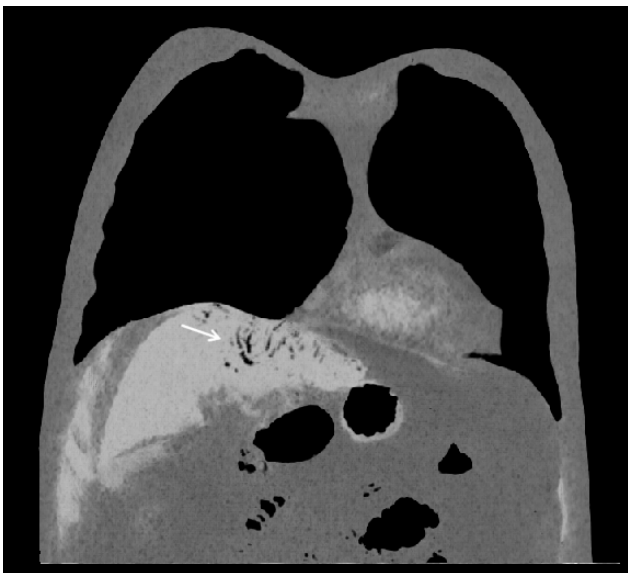


Figure 3
Pelvic CT scan (coronal reconstruction) showing gas in the right femoral vein

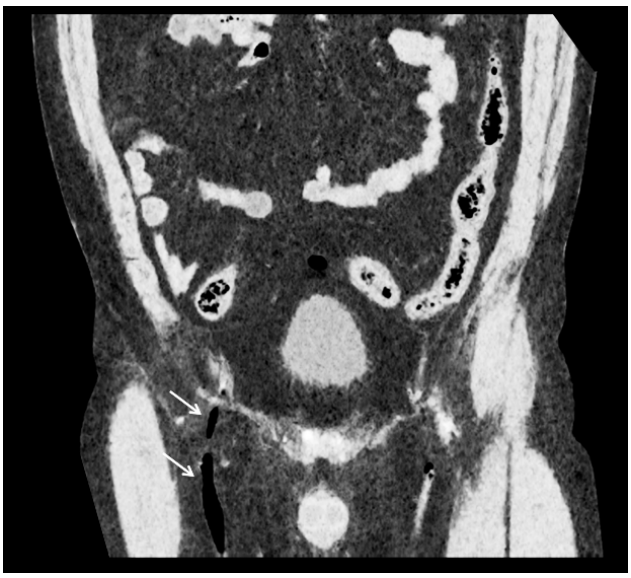
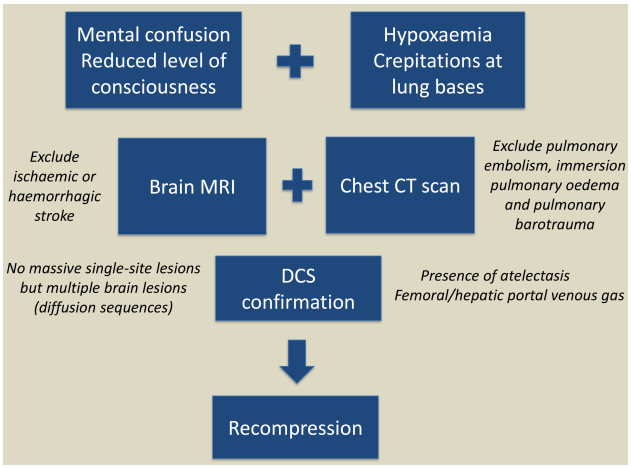


Figure 4
Decision-making process employed in managing this case



epidemiology of diving accidents, the primary aim of a clinical examination would be to investigate if IPO is present, whose origin is multifactorial.[6–8]

A pulmonary barotrauma, which is related to variations in pressure and alveolar volume, must be investigated in the case of rapid ascent and incomplete exhalation, while in the underwater environment inhalation of sea water is always possible. More rarely, a cardio-respiratory decompression sickness can also occur, when the pulmonary circulation is congested by the venous return of high numbers of circulating bubbles.[1,2]

In diving accidents with clinical signs of pulmonary damage or a high suspicion of pulmonary pathology (based on the diving history), early chest CT scanning may help clinicians exclude IPO and also to ensure the absence of barotrauma lesions such as a pneumothorax or a pneumomediastinum which could have contraindicated hyperbaric treatment.

In case of isolated respiratory symptoms, the treatment is based on normobaric oxygenation, except for the case of a cardio-respiratory decompression sickness, which requires hyperbaric recompression.[2,9]

In the presence of cerebral neurological symptoms after a dive, the problem is to determine whether the patient should be sent to a hyperbaric unit or to a stroke centre, as they are not necessarily available in the same place. Unpublished epidemiological data from our hyperbaric centre show that the differential diagnosis of stroke is more common than often thought.[5] In view of the specific care required for stroke patients, doctors practicing in hyperbaric centres should eliminate the diagnosis of stroke and perform a cerebral MRI before recompression if there is doubt.[9]

Finally, this diving accident illustrates the remarkable number of bubbles that may evolve during decompression; in this case responsible for a DCS with both cerebral and pulmonary impairment. The formation of decompression bubbles was authenticated by echocardiography and the thoraco-abdominal CT scan which showed femoral and hepatic portal venous gas. The cerebral involvement was revealed by the presence of several cortical hyperintensities observed in the MRI diffusion sequence. These diffusion sequences suggested recent ischaemic injury. Given this clinical picture, the history of diving, and considering the absence of unifocal haemorrhage or ischaemia on the MRI scan, the diagnosis of stroke was discounted.

The mechanism of cerebral embolic injury was suggested by the presence of a large spontaneously shunting PFO. The mechanism of pulmonary involvement was suggested by the elimination of other diagnoses such as IPO, pulmonary barotrauma and pulmonary embolism. The presence of atelectasis in the pulmonary bases was probably related to bubble-induced lung damage.[10] The various organ injuries found were probably related to the physical and inflammatory effects of this significant formation of bubbles.

In this clinical case the use of radiological examinations was indispensable to confirm whether hyperbaric treatment or another care plan was necessary. We were able to confirm the diagnosis of DCS with cerebral and respiratory impairment (Figure 4). The radiological examinations were carried out urgently, which did not delay recompression.

References

1 Vann RD, Butler FK, Mitchell SJ, Moon RE. Decompression illness. Lancet 2010;377:153–64. doi: 10.1016/S0140-6736(10)61085-9. PMID: 21215883.

2 Francis TJR, Mitchell SJ. Manifestations of decompression disorders. In Brubbak AO, Neuman TS, editors. Bennett and Elliot's physiology and medicine of diving, 5th ed. London: WB Saunders; 2003. p. 578–99.

3 Blatteau JE, Gempp E, Simon O, Coulange M, Delafosse B, Souday V, et al. Prognostic factors of spinal cord decompression sickness in recreational diving: Retrospective and multicentric analysis of 279 cases. Neurocrit Care. 2011;15:120–7. doi: 10.1007/s12028-010-9370-1. PMID: 20734244.

4 Gempp E, Lyard M, Louge P. Reliability of right to left shunt screening in the prevention of scuba diving related-decompression sickness. Int J Cardiol. 2017;248:155–8. doi: 10.1016/j.ijcard.2017.08.059. PMID: 28864135.

5 Bourron Alexandra. Signes neurologiques en sortie de plongée: accident de décompression à expression cérébrale ou accident vasculaire cérébral. Thèse, soutenue le 18 janvier 2019. Faculté de médecine de Marseille. Sciences du Vivant(q-bio). 2019. French. Available from: https://dumas.ccsd.cnrs.fr/dumas-02117296. [cited 2019 June 19].

6 Gempp E, Louge P, Henckes A, Demaistre S, Heno P, Blatteau J-E. Reversible myocardial dysfunction and clinical outcome in scuba divers with immersion pulmonary edema. Am J Cardiol. 2013;111:1655–9. doi: 10.1016/j.amjcard.2013.01.339. PMID: 23497776.

7 Peacher DF, Martina SD, Otteni CE, Wester TE, Potter JF, Moon RE. Immersion pulmonary edema and comorbidities: case series and updated review. Med Sci Sports Exerc. 2015;47:1128–34. doi: 10.1249/MSS.0000000000000524. PMID: 25222821.

8 Castagna O, Regnard J, Gempp E, Louge P, Brocq FX, Schmid B, et al. The key roles of negative pressure breathing and exercise in the development of interstitial pulmonary edema in professional male SCUBA divers. Sports Med Open. 2018;4(1):1. doi: 10.1186/s40798-017-0116-x. PMID: 29299780. PMCID: PMC5752643.

9 Blatteau JE, de Maistre S, Louge P, Gempp E. Diagnostic approach for the management of diving injuries – experience of the hyperbaric center of Sainte-Anne hospital, Toulon France. In: Denoble P, Marroni A, editors. Differential diagnosis of decompression illness workshop proceedings. Durham (NC): Divers Alert Network; 2019. p. 85.

10 Geng M, Zhou L, Liu X, Li P. Hyperbaric oxygen treatment reduced the lung injury of type II decompression sickness. Int J Clin Exp Pathol. 2015;8:1797–803. PMID: 25973070. PMCID: PMC4396314.

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