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Fatal scuba diving incident with massive gas embolism in cerebral and spinal arteries

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Abstract CT and MRI have the potential to become useful adjuncts to forensic autopsy in the near future. The examination of fatal injuries facilitates a profound experience in the clinical–radiological examination of these cases; the more severe findings in corpses with autopsy verification can help one to understand the tiny signs seen in clinical cases of surviving victims. We present the case of a 44-year-old male diver who died from severe decompression sickness after rapid ascent from approximately 120 m. Post-mortem CT and MRI studies of the brain and spinal cord revealed extensive gas inclusions in cerebral arteries, spinal arteries and cerebrospinal fluid (CSF) spaces, while the intracranial venous sinuses remained unaffected. These findings were confirmed at autopsy. Appropriate imaging techniques can help forensic pathologists to aim their autopsies at findings that might otherwise remain undetected.

Keywords Scuba diving · Gas embolism · Post-mortem imaging

Introduction

In the course of an ongoing study [1] on the value of post-mortem digital imaging in forensic pathology that is approved by both the local ethics committee and the juridical authorities, the deceased are examined by standard protocols with both multi-slice CT and MRI. We report the case of a fatal barotrauma where sectional imaging revealed uncommon findings that were later confirmed at autopsy.

Case report

A 44-year-old, experienced, male scuba diver went for a dive to a depth of approximately 120 m. He used a "Trimix" diving gas composition, containing 9.3% oxygen, 53.4% helium and 33.9% nitrogen, and a "Nitrox" diving gas, containing 55.4% oxygen and 46.3% nitrogen. Contrary to common diving principles he was diving alone; eye-witnesses reported that he suddenly surfaced at great speed, flailing his arms and

calling for help. He was pulled into a boat where he died almost instantly. His body was transferred to the Institute of Forensic Medicine; the case was included in the above mentioned study.

Imaging

Imaging studies were performed with the body wrapped in two artefact-free body bags (Rudolf Egli AG, Bern, Switzerland); neuroradiologists and technicians were blind to the subject's identity or appearance.

A CT study of the brain and spine was performed approximately 36 h after death (GE LightSpeed QX/i, General Electric, Milwaukee, Wis., USA). Four hundred and two overlapping, axial, 1.25 mm sections (0.625 mm table feed) were acquired from the head; the craniocervical junction was examined with another block of 76 sections.

MRI was performed immediately after the CT examination (GE Signa Horizon 1.5 T, General Electric). A double-echo (proton density T2-weighted) axial spin echo sequence (TR 4,000 ms, TE 15/90 ms, 5 mm slice thickness, 1 mm gap) and an inversion recovery sequence (TR 11,002 ms, TE 217 ms, TI 2,200 ms) were acquired from the head. Although the sequences were basically the same as used in routine clinical imaging, TR and TE were modified by empirically established correction factors for body temperature (unpublished data).

Image post-processing, including 2D and 3D reformations for both modalities, was performed on an Advantage Windows 4.0 workstation (General Electric).

Autopsy

Autopsy was performed by a board-certified forensic pathologist approximately 60 h after the death. According to standard forensic autopsy techniques, all three body cavities were opened. Special techniques were used for the asservation of gas from the heart ventricles and abdomen to allow gas chromatographic analysis of the gaseous material. Histo-neuropathologial examination of various brain regions was carried out (haematoxylin–eosin (H&E) staining and CD3, CD20, CD45, CD68, myeloperoxidase and glial fibrillary acidic protein, GFAP, immunohistochemistry), as well as toxicological blood alcohol and common drugs analyses.

Results

CT showed air-equivalent attenuation values in numerous intracranial arteries, the orbits, and parts of the CSF system (Fig. 1). MRI revealed massive gas embolism of intracerebral arteries (Fig. 2). Perivascular oedema or haemorrhage was evident on T2-weighted MR images (Fig. 3). The venous sinuses appeared slightly hyperattenuated compared to white matter and did not show

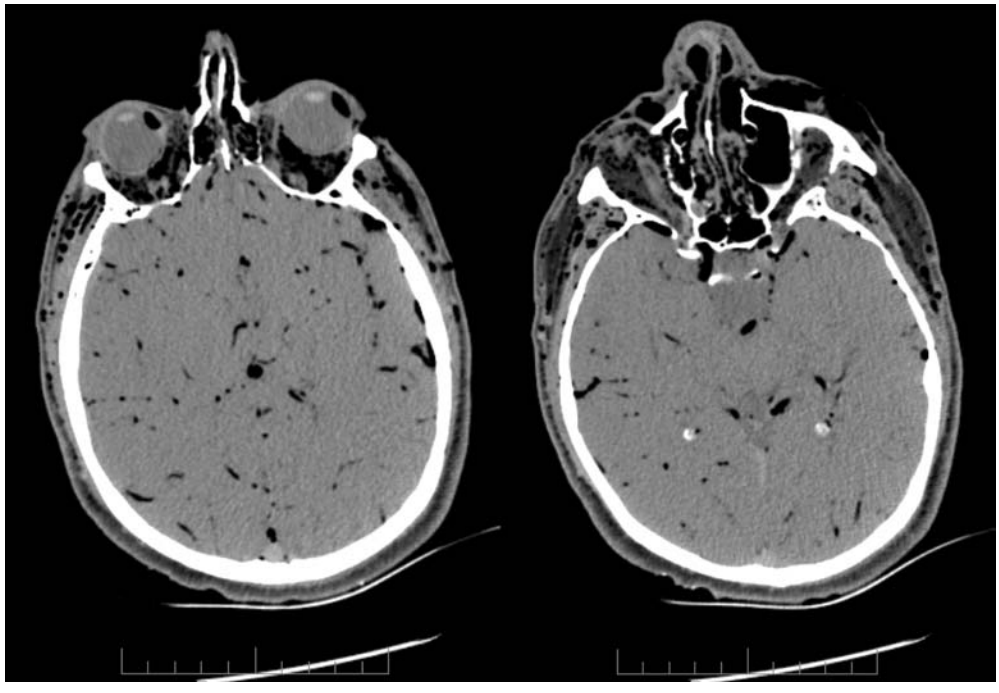


Fig. 1 Thin-section CT shows gas inclusions in the orbits and diffusely distributed in the subarachnoid space. Note hyperattenuation in the superior sagittal sinus, due to post-mortem thrombotic material

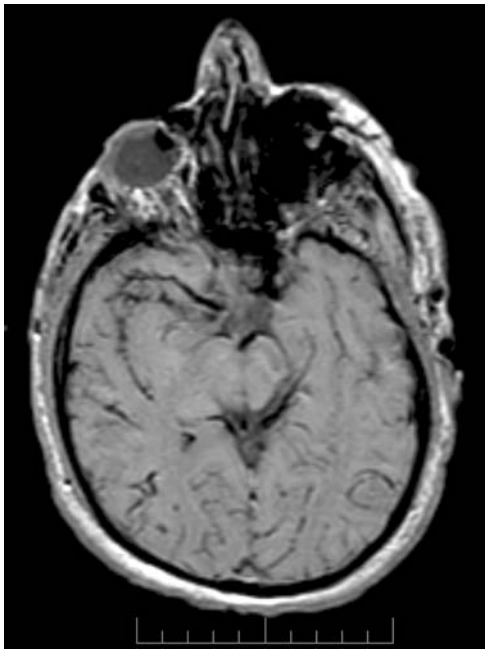


Fig. 2 Massive gas embolism is seen in the right middle cerebral artery, the left internal carotid artery, and the left posterior cerebral artery (T1-weighted 5 mm section, TR 270 ms, TE 4.2 ms)

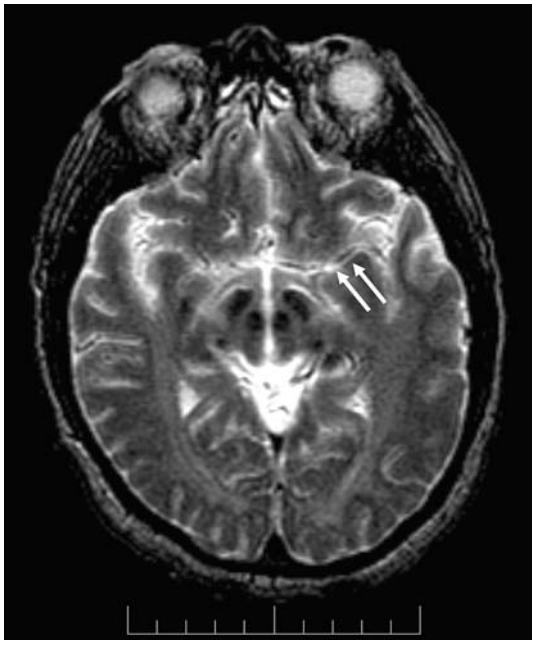


Fig. 3 This axial, fluid-attenuated inversion recovery (FLAIR) image (TR 11,002 ms, TE 217 ms, TI 2,200 ms) shows hyperintense signal around the middle cerebral artery in the left Sylvian fissure (*arrows*)

gas inclusion (Figs. 1 and 4), while some cortical veins draining into the superior sagittal sinus did show air-equivalent attenuation values in the CT examination (Fig. 4). The cranial portion of the anterior spinal artery and its lateral branches were filled with gas to an extent that allowed us to acquire a ''negative contrast angiogram'' of these vessels (Fig. 5).

At autopsy, findings included massive gas embolism in the superficial cerebral arteries and veins (Fig. 6a). Congestion of blood was found in the capillaries. The basilary artery, as well as the anterior spinal artery, showed substantial gas inclusion (Fig. 6b). There were only minor focal arteriosclerotic changes to the cerebral and spinal arteries without any significant stenoses. Brain weight was 1,600 g; there were prominent signs of cerebral swelling. No cerebral haemorrhages could be detected macroscopically. The meninges were slightly turbid. The heart cavities and contiguous blood vessels were ballooned with gas; there was no blood in the heart. The foramen ovale was closed. Massive gas intrusion was also found in soft tissues of the whole body, in the abdominal cavity and the intestinal walls.

Neurohistopathological examination revealed scattered perivascular plasma exudates and acute perivascular haemorrhages (Fig. 7) as well as oedematous changes of cerebral tissue. The blood vessels were partially congested or ''empty'' and dilated due to bubble formation (Fig. 7a). The walls of several meningeal and intraparenchymal blood vessels, as well as the vicinity of these vessels, were infiltrated by lymphocytes, mostly CD3-immunoreactive T lymphocytes. (Fig. 8).

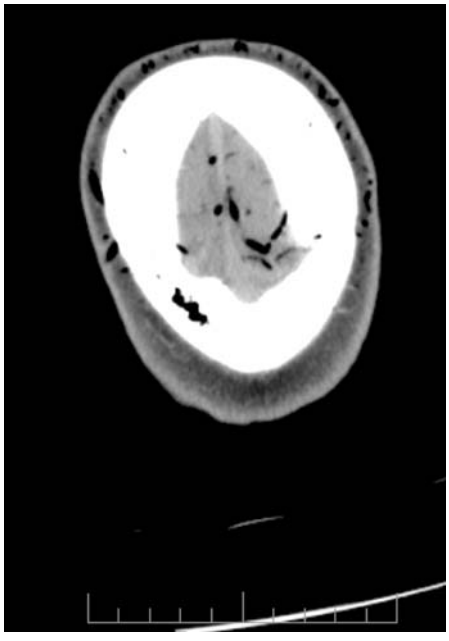


Fig. 4 Gas inclusions in cortical veins draining into the superior sagittal sinus seen in a CT slice. This sinus itself again shows hyperattenuation

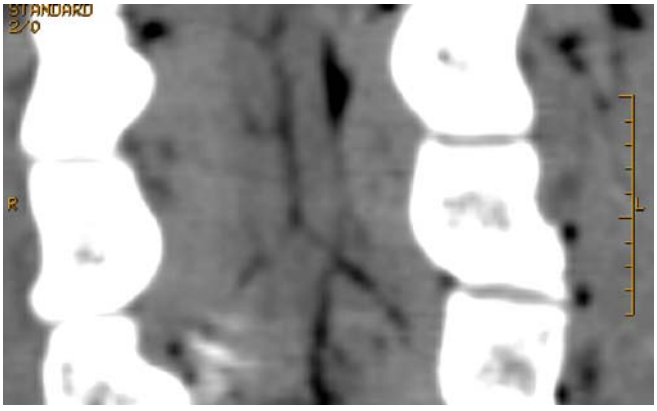


Fig. 5 This CT reformation shows gas in the anterior spinal artery. A "negative contrast angiogram" can be obtained due to the massive gas filling of the vessels

The toxicological examinations revealed no ethanol or drugs in the blood or urine. Chemical analysis of gas collected from different body cavities showed high percentages of oxygen as well as of nitrogen and helium. A post-mortem artefact from the formation of putrefaction gas could therefore be excluded. The oxygen fraction in the body cavities was considerably higher than that in the "Trimix" diving gas used. The diving gear was found to be functional by expert technicians. The cause of death was determined to be massive gas embolism due to decompression sickness.

Discussion

Arterial gas embolism (AGE, pulmonary origin) or decompression sickness (DCI; gas-bubble formation in tissues and in the blood) are caused by rapid decompression, which, in both cases, finally leads to the formation of gas bubbles in the blood [2]. The most common form of barotrauma is middle ear squeeze; more serious complications comprise neurological or pulmonary symptoms. Dysbaric osteonecrosis is a well-known long-term complication, especially for professional deep-sea divers and tunnel workers [3]. Drowning, however, remains the most common cause of fatal diving accidents (60%). A 1997 study reported 1.3 fatal DCI cases per 100,000 dives, and a 1999 study counted 3–9 deaths among 100,000 divers per year in the USA [4, 5]. Recompression (hyperbaric oxygen treatment) remains the therapy of choice in cases of arterial gas embolism and DCI. The importance of a controlled ascent and the observation of no-decompression limits are well known to properly trained divers. With regard to the high percentage of oxygen found in the body cavities, the apparent reason for the subject's reported rapid ascent was oxygen toxicity. According to the diving computer

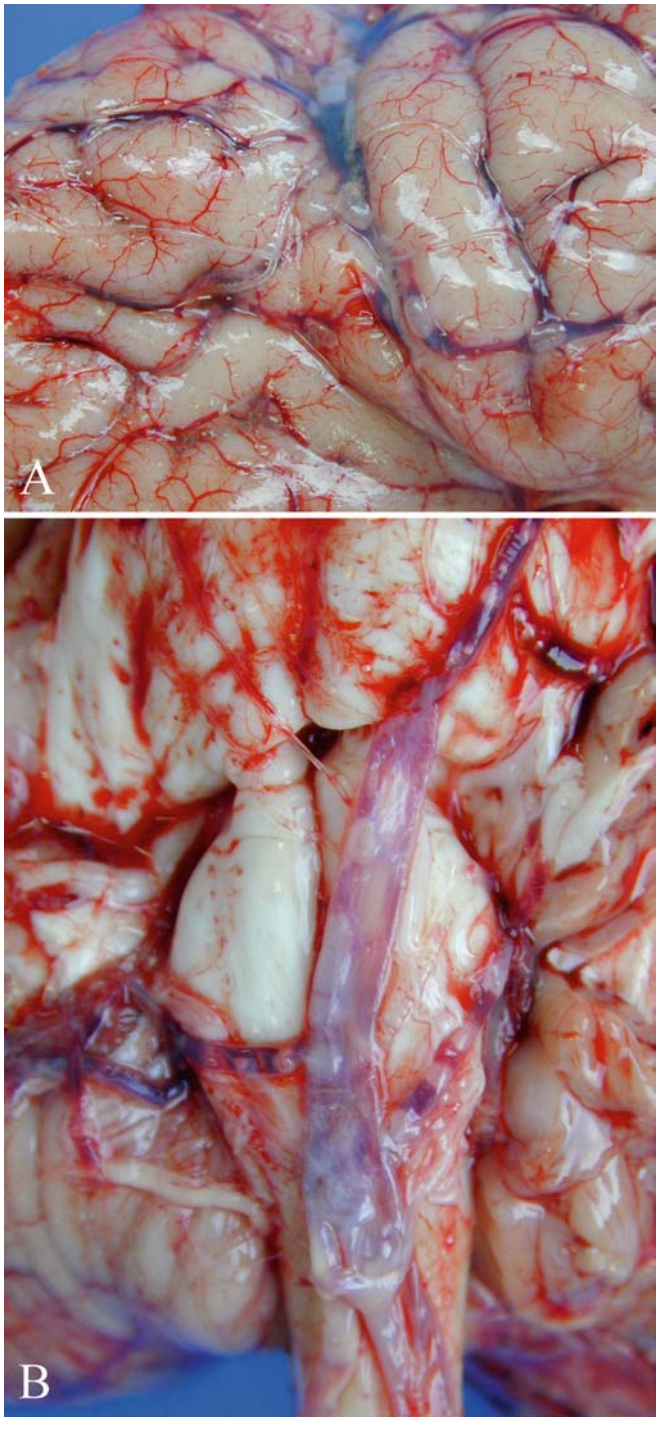


Fig. 6 Autopsy findings. **a** Gas bubbles are present in the superficial cerebral blood vessels, and the small meningeal veins are congested. **b** The basilar artery and its branches are filled with gas bubbles, which show a "pearl necklace"-like configuration

analysis, the "Trimix" diving gas had been used up when the diver had still been at a depth of approximately 120 m. As a consequence, he had had to use the "Nitrox" gas mixture containing a high percentage of oxy-

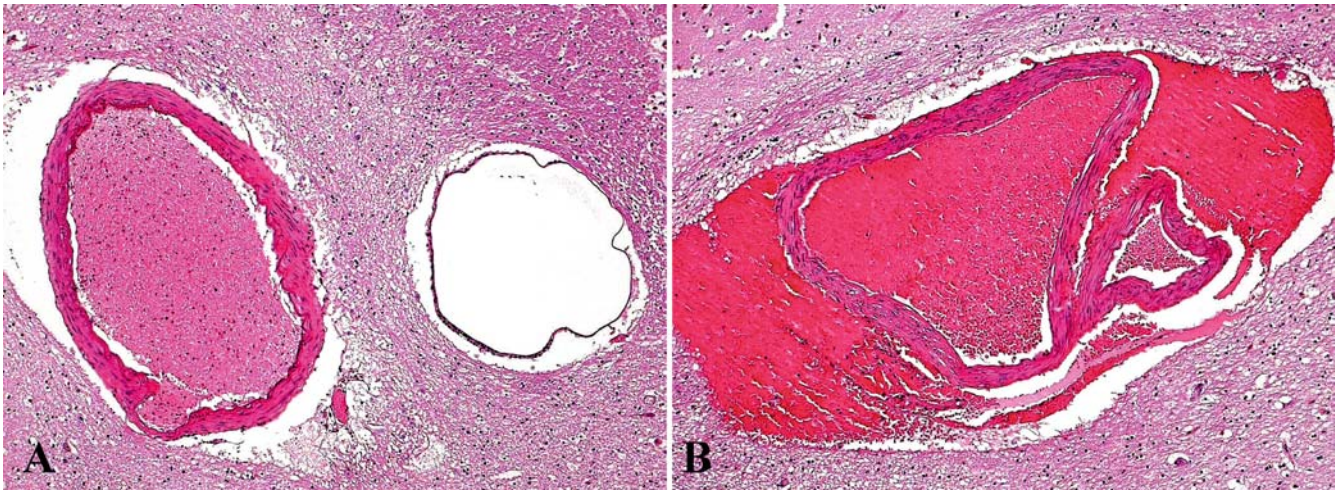


Fig. 7 **a** The histological brain tissue specimen shows an optically empty (*right*) next to a congested (*left*) intracerebral blood vessel (paraffin section, H& E, ×40). **b** Fresh perivascular haemorrhage in the frontal white matter (paraffin section, H&E, ×50)

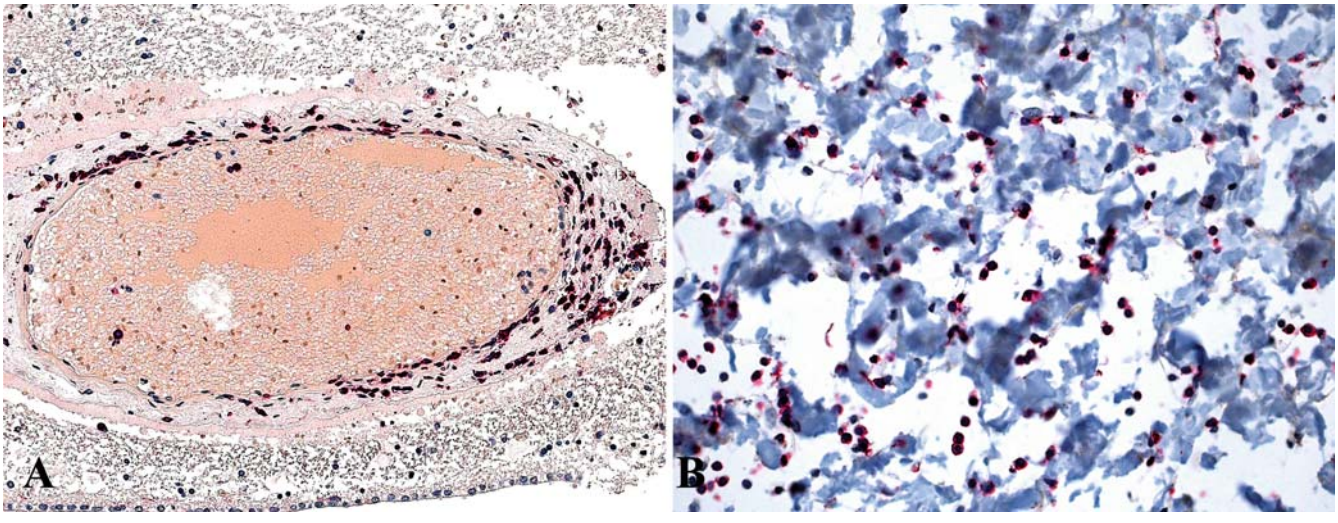
gen, which had led to oxygen accumulation in the tissue, causing the fatal rapid ascent.

The radiological and autopsy findings in the actual case comprised AGE and DCI, both leading to extensive, uncommon, gas-bubble formation in the intracerebral blood vessels. The diver suffered gas embolism while still alive, and it was not a postmortem artefact, as shown by vital signs which were present at histo-neuropathological examination and can only occur when the blood circulation is intact (e.g. perivascular oedema, parenchymal haemorrhage). As the time of survival was short, no marked ischaemic lesions were detectable in the brain and spinal cord at autopsy or on the CT scans and MR images. The minor focal lymphocytic infiltration of vessel walls and perivascular regions in the leptomeninges and the frontalbasal brain tissue without parenchymal necrosis was compatible with a slight inflammation, possibly a viral infection, which might have gone unnoticed by the patient.

The post-mortem application of CT and MRI techniques in forensic cases has been unusual in the past years. However, several forensic institutions are actually evaluating the routine use of CT techniques in fatal trauma cases in view of traumatological diagnosis and the possibilities of forensic reconstruction [6–9], while post-mortem forensic MRI is still rare [1, 10–12].

In diving incidents, CT and MRI can be superior to autopsy with regard to the detection of gas bubbles in the meningeal vessels and the spinal artery and the differentiation from the blood-filled sinuses. At autopsy, a considerable number of air bubbles always disappear

Fig. 8 **a** Infiltration of a blood vessel in the vicinity of the anterior horn of the lateral ventricle by CD3-immunoreactive T cells (paraffin section, ×100). **b** Diffuse infiltration of a slightly fibrotic area of the leptomeninges by CD3-immunoreactive T lymphocytes (paraffin section, ×150)



into the blood vessels when the dura mater is opened. This complicates the diagnosis of the presence and the extent of pre-autopsy, non-artificial air embolism. As in the actual diving accident, appropriate imaging is a useful adjunct to forensic autopsy and can provide excellent information about findings that can be underestimated or even overseen at autopsy. Additionally, the post-mortem radiological examination of forensic cases—allowing a direct comparison between radiological and traumatopathological findings—will offer the possibility of increasing the radiologist's experience in the evaluation of severe and uncommon trauma findings.

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