Case reports

Persistent extravascular bubbles on radiologic imaging after recompression treatment for decompression sickness: A case report

Juan C Dapena[1], Corine A Lansdorp[2], Simon J Mitchell[3,4]

[1] *Navy Medicine Operational Training Center, Hyperbaric Medicine Department, Pensacola (FL), USA*
[2] *Amsterdam University Medical Centre, location AMC, Department of Anaesthesiology/Hyperbaric Medicine, Amsterdam, The Netherlands*
[3] *Department of Anaesthesiology, School of Medicine, University of Auckland, New Zealand*
[4] *Department of Anaesthesia, Auckland City Hospital, Auckland, New Zealand*

Corresponding author: *Dr Juan C Dapena, Navy Medicine Operational Training Center, Hyperbaric Medicine Department, 220 Hovey Rd, Pensacola, Florida, 32508, USA*
juan.c.dapena.mil@mail.mil

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Abstract

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Decompression sickness (DCS) is a condition arising when dissolved inert gas in tissue forms extravascular and/or intravascular bubbles during or after depressurisation. Patients are primarily treated with 100% oxygen and recompression, which is often assumed to lead to resolution of bubbles. After this, repeated hyperbaric exposures can be provided in case of persistent symptoms, with oxygen delivery to ischaemic tissues, anti-inflammatory properties and reduction of oedema considered the main mechanisms of action. In this case report we present the history and imaging of a diver diagnosed with DCS that was treated with two US Navy Treatment Table 6 recompressions, but who still had multiple extravascular bubbles apparent on CT-imaging after these hyperbaric treatments. Based on these findings we hypothesise that, contrary to general belief, it is possible that large extravascular bubbles can persist after definitive treatment for DCS.

Introduction

Decompression sickness (DCS) is a condition arising when dissolved inert gas in tissue forms extravascular and/or intravascular bubbles during depressurisation.[1] It is well known for its appearance in divers, although it can appear in any person breathing an inert gas (most commonly nitrogen), who is subject to an ambient pressure reduction at a rate that exceeds the rate of washout of the gas. It can also be seen, for example, in tunnel and caisson workers and aviators flying an unpressurised aircraft at high altitude.

The diagnosis of DCS is based on clinical manifestations, with a wide range of possible signs and symptoms, depending on the location of the bubbles.[1] Symptoms frequently reported are pain, numbness/paraesthesia and constitutional symptoms such as headache, light-headedness and fatigue. More severe symptoms such as alteration of mental status, loss of consciousness, spinal cord syndromes and cardiovascular complications are also seen.

The primary treatment for DCS is breathing 100% oxygen during recompression (hyperbaric oxygen treatment [HBOT]). The resulting increase in the gradient between the pressure of nitrogen in bubbles and alveoli washes out the inert gas, leading to resolution of bubbles.[1] In case of persistent symptoms after the initial treatment, repeated HBOT can be provided. Anecdotally, it is often assumed that bubbles are unlikely to persist after an initial definitive HBOT session and consequently that the benefit of additional treatments comes from oxygenation of ischaemic tissues, anti-inflammatory effects and reduction of oedema, rather than actual bubble resolution. There is certainly a marked lack of evidence to the contrary. One report describes the recurrence of low grade venous gas emboli in three subjects following a US Navy Treatment Table 5 administered for mild DCS symptoms arising after a 48 hour shallow saturation dive.[2,3] The implications of this for bubble resolution by longer recompression protocols (such as the US Navy Treatment Table 6, USN TT6) administered after more typical non-saturation bounce dives are unknown.[3] Moreover, to our knowledge, there is no direct evidence at all for persistence of extravascular bubbles after a definitive HBOT recompression.

Here we present the case of DCS treated with two USN TT6 recompressions,[3] but who still had multiple large extravascular bubbles on CT-imaging after these treatments.

Case report

The patient gave permission for his case and radiology to be reported. A 37 year-old male presented after diving off coastal USA. He had performed six repetitive and strenuous dives to a maximum depth of 44 metres' sea water (msw) while spear fishing the day before, breathing nitrox (29% oxygen, 71% nitrogen). The patient reported to have been out diving for about 3.5 hours with an average bottom time of 11 minutes per dive. He reported performing safety stops during ascent at 5 msw for his first three dives, but did not recall safety stops for his last three dives or the rate of ascent of his dives. Unfortunately, the actual data of the diving computer including information on the surface intervals was not available during the consultation.

At the end of the third dive the patient started experiencing pain in his right shoulder, which improved during his fourth and fifth dive but persisted after the sixth. He described it as a burning pain extending up to the base of the neck and as far distally as both elbows, with the pain being extreme during the ride back to shore. The patient also reported chest pain while surfacing from the fifth dive, resolving upon completion of the sixth dive. The next morning the pain in the shoulders and neck was still present and there was soft tissue swelling over both shoulders and upper arms. His girlfriend also noticed swelling of the left hand side of the face. The patient then self-referred to a local hospital for evaluation.

There were no previous diving injuries within his two years of diving experience. There was no other relevant medical history and no allergies or intoxications, other than tobacco use equivalent to five pack years. A neurological examination was normal.

The patient was diagnosed with musculoskeletal and lymphatic DCS, and was treated with a USN TT6 beginning 25 hours after completion of diving.[3] Upon completion of his first treatment his pain had reduced from 10/10 to 6/10 and there was incomplete resolution of his upper limb and facial swelling. Due to minimal improvement of his symptoms a computed tomography (CT) scan was performed, showing a small amount of gas in the manubriosternal joint (Figure 1A) and along the posterior to superior-medial aspect of the right glenohumeral joint (Figure 2A), with no signs of pulmonary barotrauma.

Because of the ongoing complaints, the attending physician contacted the staff of the Undersea and Hyperbaric Medicine fellowship programme in New Orleans. The recommendation was made to give the patient a 2 h break and then provide another USN TT6. After completion he reported persistent pains in the shoulders at a lower intensity, and persistent proximal swelling of both arms. The patient signed himself out of the hospital and drove over nine hours to New Orleans to be evaluated by the fellowship programme staff.

After arrival in New Orleans the patient still had residual pain. A repeated physical examination, as well as a detailed neurological evaluation, was normal except for obvious swelling of both upper arms. Another CT scan of the chest was performed and compared to the previous scan by a radiologist. A small amount of gas in the manubriosternal joint similar to the earlier CT was found (Figure 1B), as well as a small focus of gas anterior to the left sternoclavicular joint (Figure 3B) that was not demonstrated earlier (Figure 3A). The focus of gas that was previously seen along the posterior to superior-medial aspect of the right glenohumeral joint (Figure 2B) was no longer present.

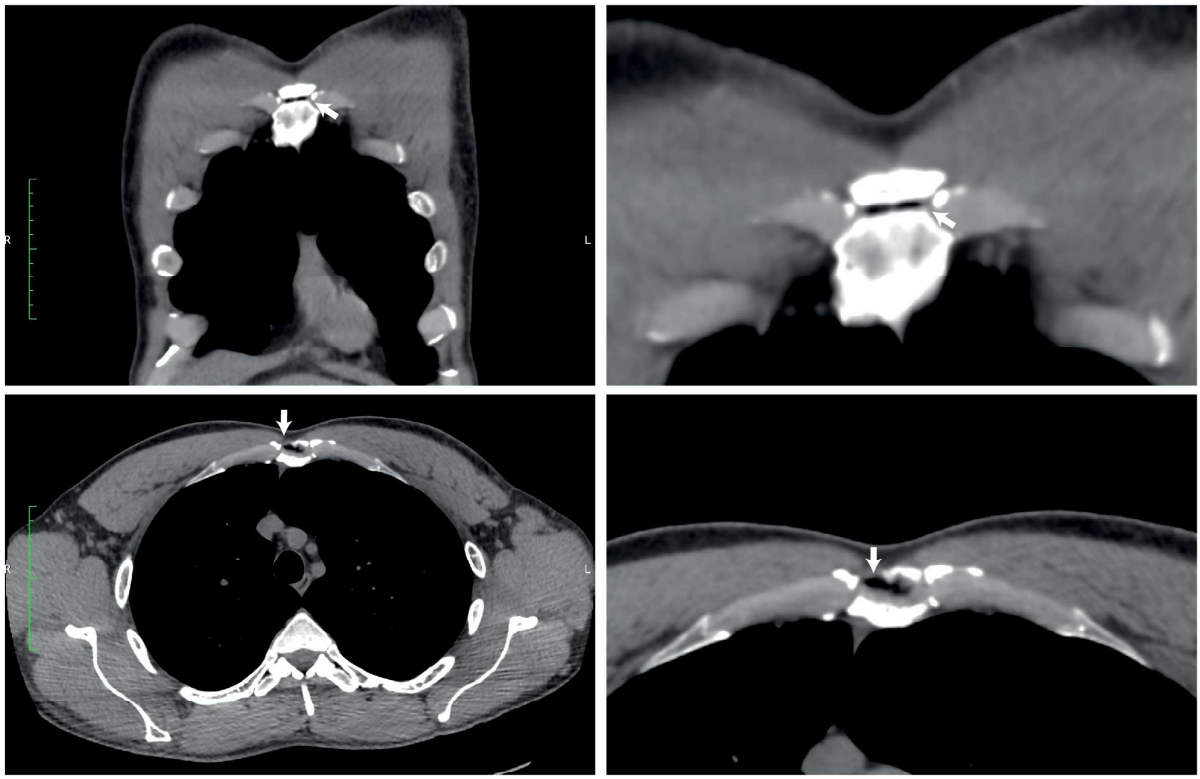
Because two USN TT6 were provided within a short interval and symptoms were relatively mild at this point, the patient was discharged and re-evaluated the next day. He then still had residual aches of the neck and persistent swelling and discomfort of both upper extremities. A single US Navy Treatment Table 9 was provided with partial resolution of his aches.[3] Due to non-medical and unrelated issues, the patient departed the institution after this treatment. A prescription order for a transthoracic echo (to rule out a patent foramen ovale) and magnetic resonance imaging of the neck, thorax and the spinal cord was provided but never carried out by the patient. He returned to spear fishing seven weeks after the incident.

Discussion

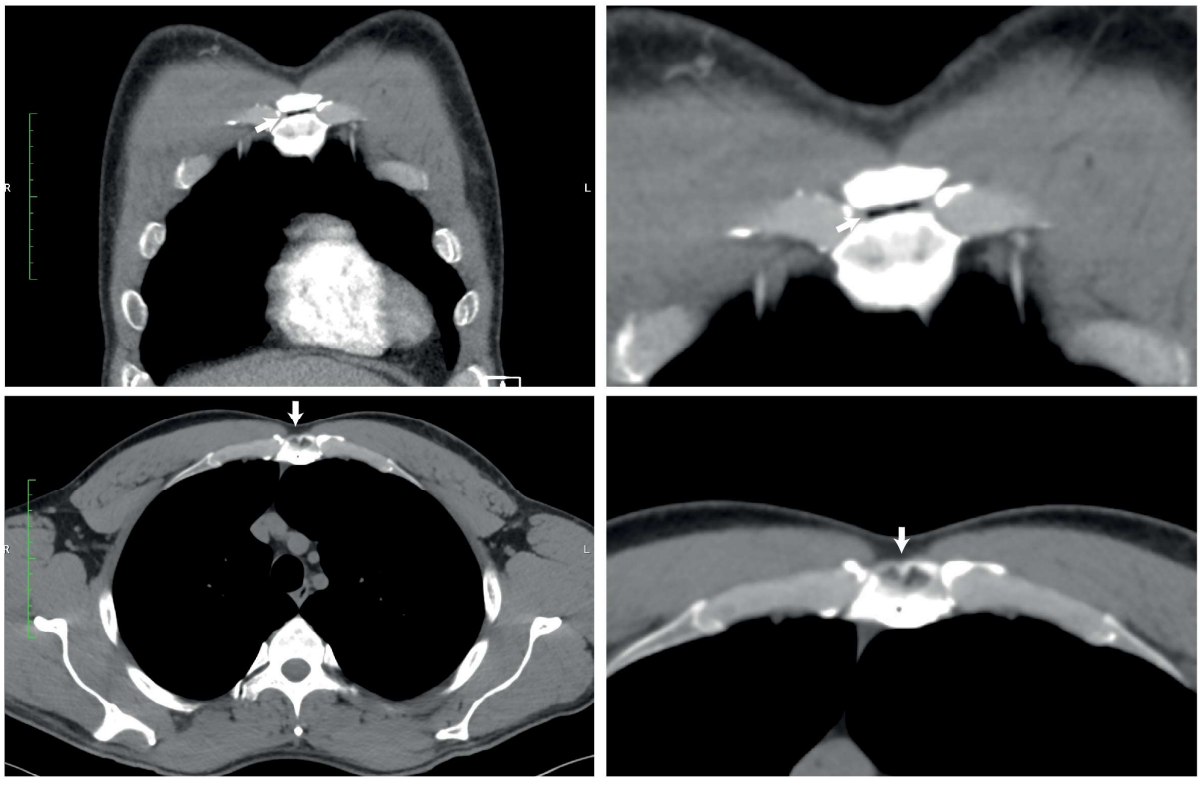
To our knowledge this is the first documented case of persistent extravascular bubbles identified by radiologic imaging after initial and subsequent recompression for DCS that would arguably be perceived as 'adequate'. Indeed, gas persisted in a common location after two USN TT6 recompressions. This is interesting because it challenges a common assumption that bubbles formed during decompression are very unlikely to persist after treatment with definitive HBOT protocols.

This assumption is largely based on the physical mechanisms of bubble resolution in response to HBOT. Apart from the direct effect of recompression on bubble size due to Boyle's law, by increasing the nitrogen pressure in compressed bubbles while at the same time reducing the nitrogen pressure in the alveoli toward zero, HBOT establishes a substantial gradient for diffusion of nitrogen out of bubbles into the blood, and thence to alveoli for elimination. This should be a potent driver for bubble elimination.[1] The likely efficacy of HBOT in this regard is supported by animal experiments, in which air bubbles injected into the spinal cord of decompressed rats all disappeared after breathing 100% oxygen at ambient pressure, even without recompression.[4]

Figure 1
Coronal and axial CT-images of the chest showing a gas focus (arrow) in the sternomanubrial joint, after recompression one (A) and recompression two (B)



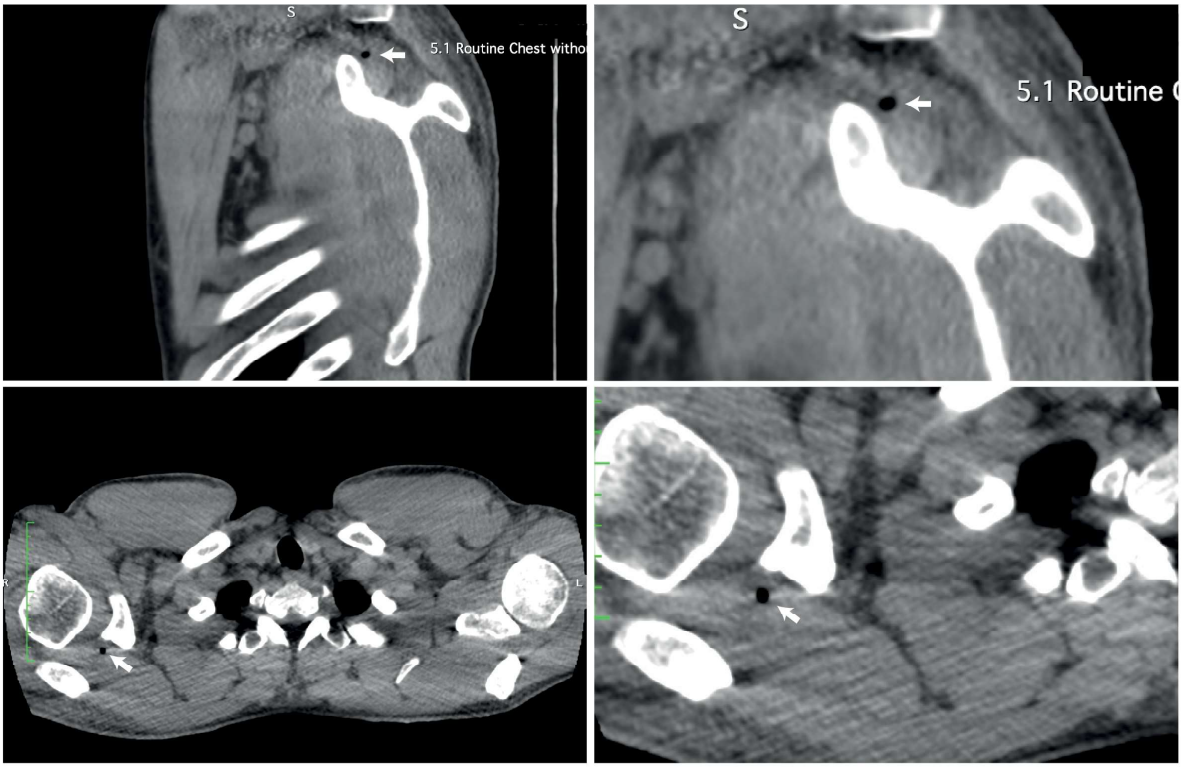
A



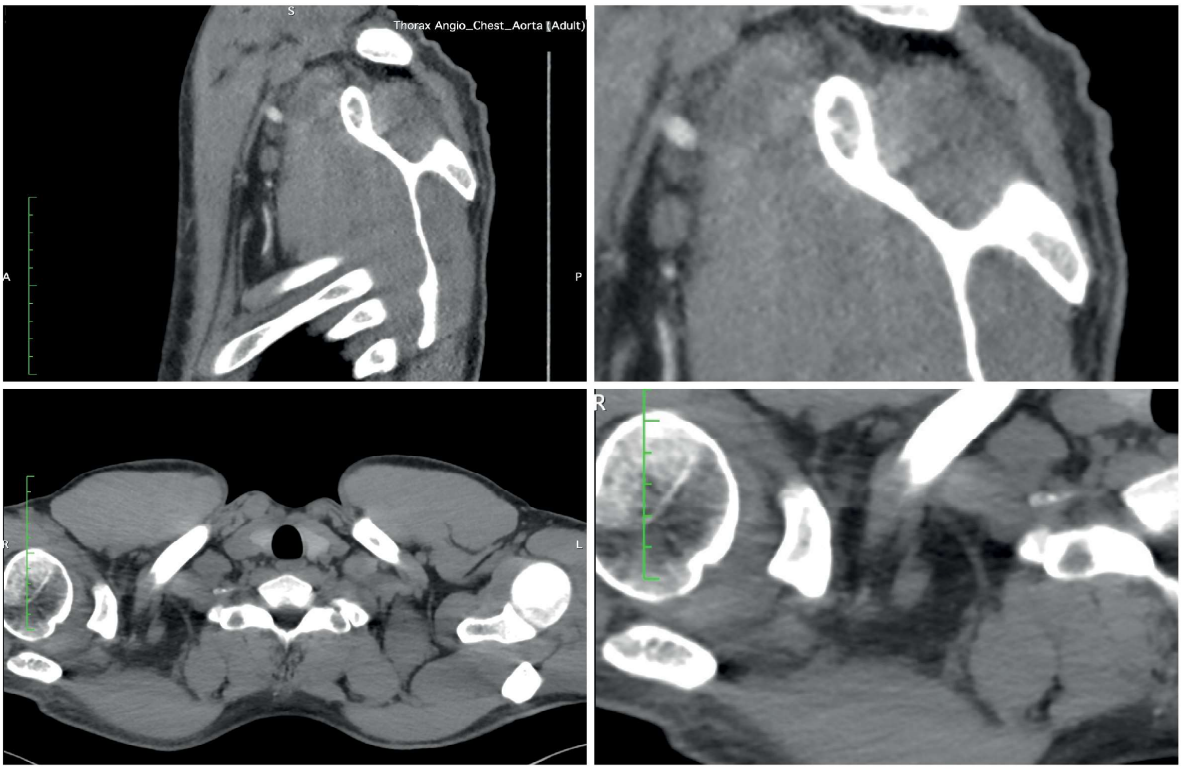
B

Figure 2

Sagittal and axial CT-images of the chest showing a gas focus (arrow) near the right glenohumeral joint and tip of the supraspinatus muscle after recompression one (A), with resolution after recompression two (B).



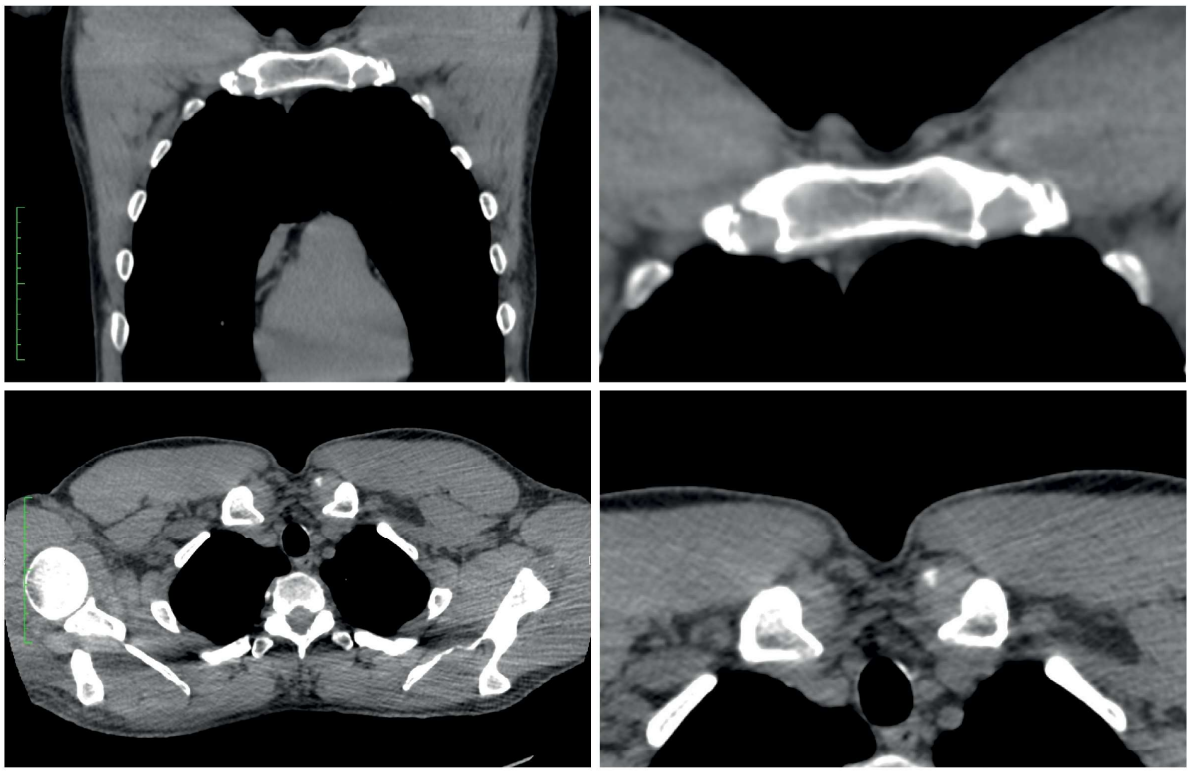
A



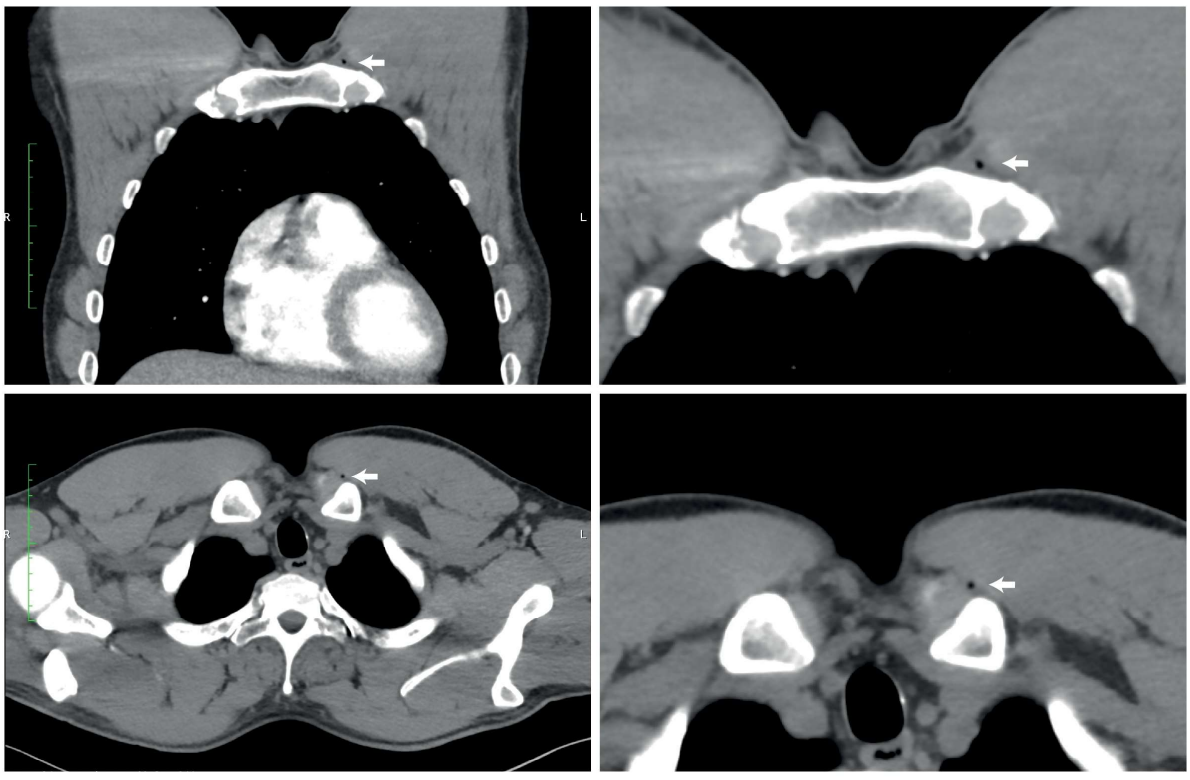
B

Figure 3

Coronal and axial CT-images of the chest showing gas foci (arrows) located anterior to the left sternoclavicular joint and posteromedial to the left pectoralis major muscle after recompression treatment two (B), that were not seen after recompression one (A)



A



B

The potential for venous bubbles (venous gas emboli – VGE) to recur after recompression for DCS has previously been reported.[2] Three of four subjects who completed a US Navy Table 5 treatment for mild DCS arising after a shallow saturation dive exhibited recurrent VGE formation after VGE initially disappeared during the recompression. The authors hypothesised that hyperoxic vasoconstriction during HBOT resulted in decreased tissue perfusion and nitrogen washout, and thus subsequently allowed further VGE formation after reperfusion. In the present case it seems possible that hyperoxic vasoconstriction in 'slow' musculoskeletal tissue (typically exhibiting low perfusion) may have contributed to the lack of efficacy of HBOT in clearing some bubbles from that tissue. Whatever the mechanism responsible, the present case appears to demonstrate persistence of extravascular bubbles (as opposed to VGE) following two USN TT6 recompressions with considerable latency before and between treatments; this represents a previously unrecorded degree of resistance to resolution of diving-related tissue bubbles despite multiple definitive HBOT recompressions.

The finding of a new bubble anterior to the left sternoclavicular joint (that was not present on the first CT scan) was unexpected, although this might be explained based on the technical limitations of CT imaging. It is possible that the gas focus was already present at the time of the first CT, but it was missed because the thickness of the CT slices exceeded the size of the bubble itself.

We acknowledge the possibility of an artefactual source of the gas seen in these CT scans. For example, another possible explanation for persistent bubbles is the so-called 'vacuum phenomenon', which refers to the radiological finding of gas (suspected to be nitrogen) in a joint.[5] It can be a consequence of trauma, inflammation, or cavitation. Radiologically it can be confused for DCS, being differentiated only through clinical correlation. In this case, a diver exhibited signs and symptoms of DCS in the broad anatomic locations in which the bubbles were seen, and did not report previous trauma or rheumatological conditions, decreasing the likelihood of vacuum phenomenon as an explanation for the radiologic findings. Furthermore, two out of three bubbles were not located within a joint.

We also acknowledge that pulmonary barotrauma (PBT) with mediastinal emphysema and extension of gas into the neck cannot be absolutely ruled out as a contributor to, or even the cause of, this diver's symptoms. However, we consider this a less likely explanation than DCS based on the dive and clinical history, symptom pattern, and the distribution of residual gas in sites other than those most affected in a typical case of PBT with mediastinal emphysema.[6] In any event, any debate about bubble origin is largely irrelevant to the key point of this report: that definitive HBOT did not completely resolve the resulting tissue bubbles that seem highly likely to be diving-related.

It is acknowledged that the performance of multiple radiologic investigations in this case was unusual. DCS is a clinical diagnosis and it is not common practice to perform advanced radiologic studies before or between treatments, although CT scans have been contributory to diagnosis of post-dive abdominal pain in several instances by demonstrating intravascular gas in the splanchnic circulation,[7,8] and to informing the differential diagnosis in diver presenting with cerebral and pulmonary symptoms.[9] In contemplating such investigations the balance between clinical value versus radiation exposure and cost must be carefully considered. Typically, however, patients with residual symptoms receive tailing treatments until complete resolution or lack of further improvement occurs, without radiological guidance.[10]

Conclusions

Persistent bubbles most likely arising from DCS were found on CT imaging after repeated definitive hyperbaric treatments. Based on these findings and the lack of an adequate alternative explanation, it is plausible that large extravascular bubbles can persist after definitive HBOT treatment for diving-related illness. If confirmed, this finding has important implications for topics of debate that recur in diving medicine, such as the reasons why flying after definitive recompression for DCS could be associated with recurrence or worsening of symptoms. Although routine decisions about initial and repeat treatment for DCS should remain clinically guided, it would be interesting if selected cases with residual symptoms are similarly investigated in the future, allowing for further insight into this phenomenon.

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Conflicts of interest and funding

Professor Mitchell is the Editor of *Diving and Hyperbaric Medicine* Journal and so had no role in managing the review process or the decision to accept this manuscript. These matters were managed by the European (deputy) editor Dr Lesley Blogg. There were no other conflicts of interest.

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