BACKGROUND

Decompression sickness (DCS) is caused by the rapid change of the surrounding pressure while ascending.\(^1\) As during descent, increased environmental pressure leads nitrogen to reach the bloodstream through the alveoli, which usually present in a dissolved form, while rapid ascend causes nitrogen to form bubbles,\(^2\) leaving a short-time window for these bubbles to be resolved and re-absorbed.\(^3\) Entrapment of these bubbles in specific tissues could lead to blood flow obstruction and vessel spasm, as well as activation of the platelets and the clotting cascade,\(^4\) causing tissue hypoperfusion. DCS is usually classified into two types, DCS I is characterized by mild cutaneous manifestations such as mottled or marbled rashes together with musculoskeletal pain; and DCS II is considered a
more severe form with neurological symptoms of poor coordination and confusion, together with cardiopulmonary manifestations that could present as dyspnea and retrosternal pain.5

2 | CASE PRESENTATION

A fit and well 54-year-old Fisherman, who is a moderate smoker, an occasional alcoholic with no notable medical history or chronic medications, was referred to the emergency department with dyspnea, lethargy, severe generalized arthralgia, and mild confusion after rapidly ascending of a saturation diving of 50 m sea depth. On initial assessment, the patient was dyspneic, alert, and responsive with a Glasgow coma scale of 14. On examination, there was bilateral joints pain, which was aggravated by movement without signs of inflammation. The neurological, chest, cardiac, and abdominal examinations were unremarkable, with a blood pressure of 90/60 mm Hg, a pulse of 95 Bpm, saturation of 90%, respiratory rate of 24/minute, and a temperature of 36.6°C; the calculated well’s pulmonary embolism score was 3.

3 | INVESTIGATIONS

At the time of presentation, the patient was evaluated by the emergency core medical trainee, where high-flow oxygen was initiated and a multi-disciplinary team of cardiology, pulmonology, and neurology was gathered to assess the patient’s symptoms. The initial investigation was electrocardiography (ECG), and it showed a normal ECG (Figure 1). The patient was then escorted to the radiology division, and a posterior–anterior chest X-ray (PA-CXR) was obtained and it showed a normal CXR (Figure 2). Meanwhile, the ordered blood tests revealed the following: normal D-dimer, normal hemoglobin, platelets, glucose, ESR, creatinine, urea, potassium, and sodium; however, it revealed elevated white blood cells of 13 (normal value 4500_11000) and CRP of 73 mg/L (normal value 0_7 mg/L). Arterial blood gas (ABG) showed metabolic acidosis, with a PH of 7.32, PCO2 of 28, Hco3 of 15, and Po2 of 63 mm Hg. After ensuring the patient was stable, he was escorted by the cardiology intern for emergency transthoracic echocardiography (TTE) to exclude the presence of structural heart diseases; where it showed normal left ventricular, normal right ventricle, and no valvular disease; yet, spontaneous echo contrast inside the right cardiac chambers was witnessed, similar to agitated saline echo testing (Figure 3).

During the examination, the patient was asked to do the Valsalva maneuver by taking a deep breath and holding it, and the aim was to evaluate the presence of patent foramen ovale (PFO) using color-flow Doppler, which could have an important role in DCS, and no flow was appreciated.

4 | DIFFERENTIAL DIAGNOSIS

After evaluating the clinical picture, together with the patient’s history, physical examination, and relevant investigations, a differential diagnosis of pneumothorax, pulmonary embolism, aspiration pneumonia, and DCS was established. Pneumothorax was excluded due to the normal chest auscultation and normal PA-CXR. Aspiration pneumonia was also excluded due to the normal examination and normal PA-CXR; while pulmonary embolism (PE) was excluded due to the well’s PE score, normal D-dimer, and the ABG findings.

5 | TREATMENT

Following admission, the patient was initially treated with 100% oxygen. Intravenous saline solution was also used for rehydration with a dose of 100 ml/h; and finally, due to the joint pain and the inability to walk, prophylactic subcutaneous (S.C) Enoxaparin was initiated with a dose of 40 mg/day. Meanwhile, the HBOT center was immediately contacted, and the first session was arranged.

FIGURE 1 Patient’s electrocardiogram upon admission
6 | OUTCOME AND FOLLOW-UP

A significant clinical and hemodynamic improvement was observed within 1 h of the first HBOT session, with a blood pressure of 135/75, a respiratory rate of 17/minute, a heart rate of 75 Bpm, and an oxygen saturation of 98%. Joint pain was mildly reduced, and the patient was able to stand by himself, though support was needed to walk around the hospital room. The patient was reevaluated using TTE within 1 h after the HBOT session, and surprisingly, the right cardiac chambers bubbles had fully vanished with no additional pathological findings (Figure 4), and he was discharged within 48 h of admission. Upon discharge, the patient’s chief complaint was mild resistant arthralgia that was alleviated using paracetamol, and he was able to walk and support himself.

7 | DISCUSSION

The diagnosis of DCS relies mostly on clinical suspicion; however, the patient’s presentation intersects with several differential diagnoses that should be excluded before establishing the final diagnosis. The symptoms of dyspnea and chest pain after a rapid ascent suggest the diagnoses of pneumothorax and post-diving aspiration pneumonia, and chest X-ray is a useful method to exclude these conditions. While pulmonary embolism should also be considered, this patient’s ABG was not compatible with the diagnosis, and the pretest probability with normal D-dimer could help to exclude the condition. The initial management for suspected DCS is the application of oxygen, as experimental studies showed a quicker decrease in the bubble load with the use of oxygen compared with air-breathing. Simultaneously, low molecular weight heparin could also be advised in DCS patients with reduced movement abilities to reduce the risk of DVT. Finally, HBOT is also present as one of the most important treatment lines. The main principle of this treatment is to deliver 100% inhaled oxygen inside a pressurized chamber equal or above 1.4 atmospheres absolute (ATA), which has a significant role in the pathology of DCS. The application of ultrasound for detecting microbubbles in DCS patients has been more common. In this case, TTE was used for three purposes, to exclude any structural heart disease, detect microbubbles, and search for the presence of left–right shunts (PFO). However, it has low specificity as a bubble screening tool; another drawback is obtaining a clear ultrasound image, as suitable echocardiographic windows might not be accessible in the emergency settings. Yet, this modality could be used as part of point-of-care ultrasound.
This report suggests that echocardiography is a useful screening tool to detect microbubbles in patients with suspected DCS. To support our hypothesis, a literature search was performed using PubMed and the Cochrane Library to evaluate similar case report findings. Four published cases were identified (Presented in Table 1), and the reference list of these studies was manually screened for similar case reports. Although patients’ reported symptoms varied, all of these studies used ultrasound as a screening tool, and the most common locations for detected microbubbles were the right ventricle and inferior vena cava. Three studies detected bubbles in several other locations using CT scan; three studies described the use of HBOT, and improvement was reported in two studies. Three studies concluded that the use of CT following ultrasound could be a useful method to diagnose DCS.

In conclusion, echocardiography in the current case helped in the diagnosis of suspected DCS, and the purpose of the patient admission to the cardiology department was to monitor these microbubbles’ behavior using TTE after the first HBOT session and to see if the disappearance of these microbubbles could reflect clinical improvement or could be related to the overall prognosis. We found that these bubbles disappeared within 1 h of HBOT, and significant improvement was noticed during the same period, which could suggest possible applicability of TTE in diagnosing and monitoring DCS patients.

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**TABLE 1** Identified published cases of bubbles detection using ultrasound in DCS patients

| Case          | Yanagawa et al., 2021<sup>13</sup> | Jitsuiki et al., 2020<sup>14</sup> | Kondo et al., 2018<sup>15</sup> | Boussuges et al., 2008<sup>16</sup> |
|---------------|----------------------------------|----------------------------------|-------------------------------|----------------------------------|
| Age           | 53 years                         | 26 years                         | 65 years                      | N/A                              |
| Diving depth  | 21 meters                        | 26 meters                        | 24 meters                     | N/A                              |
| Chief complaint| Abdominal pain and dyspnea       | Scotoma, headache and fatigue    | Epigastric pain               | N/A                              |
| Used modalities| US and CT scan                   | US and CT scan                   | US and CT scan                | Echocardiography                 |
| US bubbles’ location | Portal vein and RV             | IVC                             | IVC                           | LV/RV                           |
| HBOT          | Yes                              | Yes                             | Yes                           | N/A                              |
| Bubbles vanishing time | Within 2 days                   | N/A                             | N/A                           | N/A                              |
| Discharge     | Within 6 days                    | Within 2 days                   | N/A                           | N/A                              |
| Reported complications | Memory disturbance               | N/A                             | N/A                           | N/A                              |

Abbreviations: CT, computed tomography; HBOT, hyperbaric oxygen therapy; IVC, Inferior vena cava; LV, left ventricle; N/A, not applicable; RV, right ventricle; US, ultrasound.
CONFLICT OF INTEREST
None.

AUTHOR CONTRIBUTIONS
Mohammad Ramadan was the first to review the patient and performed the initial TTE with a discussion with Allam Harfoush. The second TTE was done by Allam Harfoush with a discussion with Mohammad Ramadan. Follow-up was done by both. The case was written by Allam Harfoush as the corresponding author and Mohammad Ramadan and Hanady Hamdallah as the co-authors.

ETHICAL APPROVAL
No ethics committee approval is warranted as it was a clinical case.

CONSENT
Written informed consent was obtained from the patient’s relative to publish this report in accordance with the journal’s patient consent policy.

DATA AVAILABILITY STATEMENT
Data are available upon request to the corresponding author (Full blood tests and TTE clips before and after HBOT), please email the corresponding author, it was also uploaded to be reviewed by the CCR editorial board during submission.

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