Pneumomediastinum and the use of hyperbaric oxygen treatment

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Abstract

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Pulmonary barotrauma may occur in diving and can result in a spectrum of injuries referred to as pulmonary over-inflation syndrome (POIS). Pneumomediastinum is a part of the POIS spectrum and only rarely results in respiratory symptoms. We present a case of a civilian diver who developed pneumomediastinum with respiratory symptoms which did not respond to normobaric 100% oxygen. After investigation for pneumothorax, he underwent hyperbaric oxygen treatment which resulted in significant alleviation of his symptoms. This is a novel case example of this treatment algorithm.

Introduction

Pulmonary over-inflation syndrome (POIS) is the spectrum of injuries that can result when pulmonary barotrauma (PBT) occurs. This barotrauma is the result of expanding gas within the lungs which occurs typically during ascent in a water column. The mechanism is explained by Boyle's law which states that pressure and volume are inversely related.[1] Thus, as the pressure decreases up a water a column, the volume of gas in a diver's lungs increases. This gas should be released through normal exhalation during ascent. Pathology arises when anything inhibiting or affecting the rate of gas release from the lungs occurs during the dive.[1] This report presents an example of PBT in a diver that resulted in symptomatic pneumomediastinum which responded to hyperbaric oxygen treatment (HBOT).

Case presentation

The patient gave permission for publication of this case report.

An otherwise healthy 32-year-old male made a planned recreational dive to 24 metres' seawater (msw) breathing air on open circuit scuba with a bottom time of 12 minutes and total dive time 50 minutes. At the end of the dive, he began to experience chest tightness within 3 msw of reaching the surface. On the surface he had obvious facial swelling, voice changes, chest pain and throat tightness with difficulty breathing. He was wearing a dive watch that he reported sounded no safety alarms throughout his dive. Attempts to adjust his buoyancy compensating device (BCD) within the first two minutes of his dive resulted in an undesired ascent in the water column from 10 to 4 msw. His dive partner was with him during the entire dive but did not make this ascent in the water column. The dive partner remained asymptomatic.

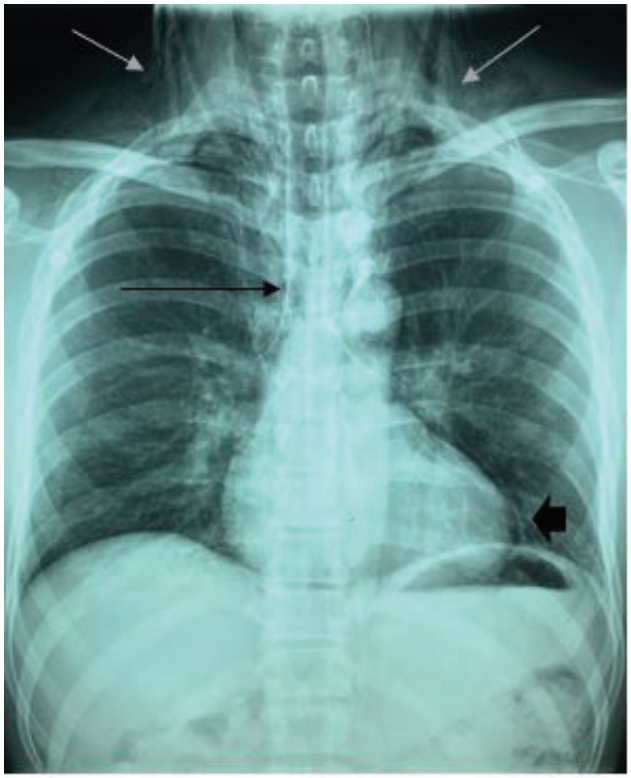
He was taken to the local emergency room where he was in obvious respiratory discomfort, leaning forward with hands on knees, with a respiratory rate of 26–30. His oxygen saturation (96–98%), heart rate (70–80·min^{-1}) and blood pressure (120–130/70-80) were all within normal limits. A 12-lead electrocardiogram showed normal sinus rhythm. He was started on normobaric 100% oxygen. On examination, there were notable voice changes and palpable crepitus along the cheeks and anterior neck and upper chest bilaterally. He had significant central chest discomfort throughout his respiratory cycle which was more notable during inspiration. Breath sounds were clear and were present bilaterally. A full neurological exam including mental status, motor, sensation, deep tendon reflexes and cerebellar signs demonstrated no deficits. His oxygen saturation was 100% during oxygen breathing. After 60 minutes of oxygen treatment, his respiratory rate had not decreased, he continued to demonstrate respiratory distress and there was only minimal improvement of his chest pain and dyspnoea. Chest radiographs showed pneumomediastinum, subcutaneous emphysema tracking up the neck and into the face and possible pneumopericardium. There was no pneumothorax

(Figure 1). Given the normal electrocardiogram, no cardiac blood tests were drawn.

Given the minimal alleviation of symptoms with supplemental oxygen and no contraindication to hyperbaric oxygen, the decision was made to treat him in the hyperbaric chamber located nearby. The chamber utilised was a standard Navy double-lock recompression chamber. Treatment was instigated within four hours of the initial injury. The US Navy Diving Manual recommends shallow recompression in the setting of severe pneumomediastinum in the absence of a pneumothorax. The table consists of breathing 100% oxygen at the shallowest depth of relief (usually 5–10 feet of seawater [fsw]) for one hour or longer if needed.[8] The chamber was initially pressurised to 131.7 kPa (1.3 atmospheres absolute, 3 msw, 10 fsw equivalent), and the patient was placed on 100% O_2 via a built in breathing system (BIBS) mask. Symptoms of chest and throat tightness, as well as voice changes, resolved 10 minutes into HBOT, and his respiratory rate normalised to 14–16·min^{-1}. There were no issues on compression or decompression in the chamber. He remained in the chamber at a pressure of 131.7 kPa for 60 total minutes. He had no recurrence of symptoms after surfacing. The subcutaneous emphysema resolved clinically approximately 72 h after the initial injury. Four weeks later he underwent thoracic computed tomography (CT) scanning without contrast, which was negative for any pulmonary abnormalities or residual injury.

Figure 1

Chest radiograph demonstrating pneumomediastinum with distinct outlining of the trachea (thin black arrow). Partial 'halo sign' (thick black arrow) along the left border of the cardiac silhouette. Extensive subcutaneous emphysema tracking bilaterally up the neck (thin gray arrows)



Discussion

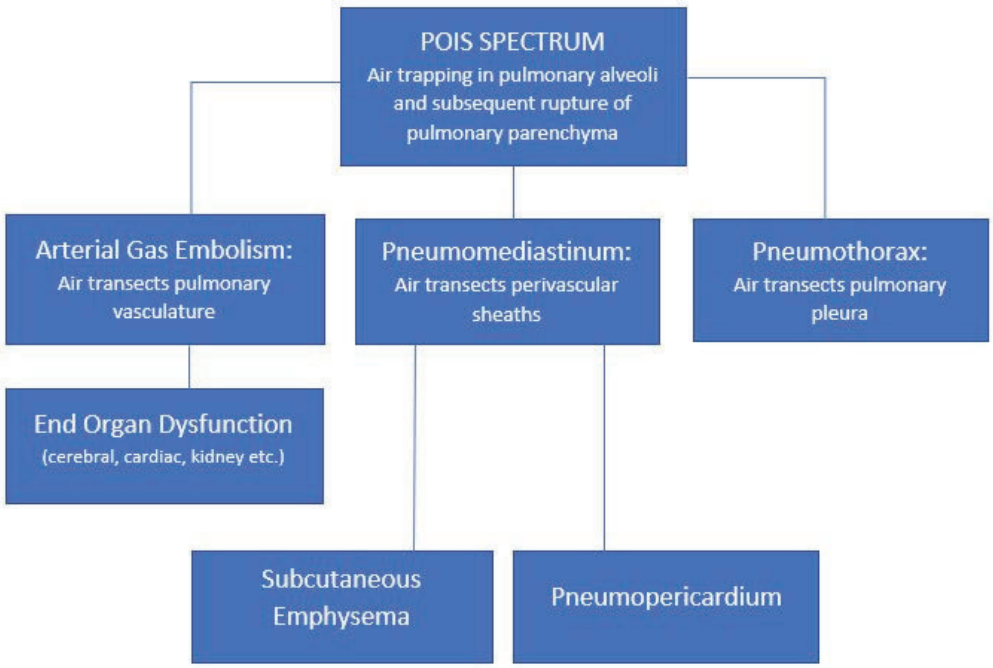
PBT is the second most common type of barotrauma after ear/sinus barotrauma.[2] POIS refers to the spectrum of injuries that result specifically from PBT which range from life threatening arterial gas embolism (AGE) to subcutaneous emphysema. As air expands within the lung parenchyma and reaches the necessary pressure difference that air can transect into the pulmonary vasculature, the perivascular sheaths and/or the pleura, resulting in one or a combination of POIS injuries (Figure 2).[1] Isolated pneumomediastinum results from air migration along the perivascular sheaths and pulmonary interstitium to the hilum to enter the mediastinal space.[1]

Risk factors for developing POIS are related directly to a diver's ability to appropriately exhale the expanding gas in the lungs. Rapid ascent, decreased forced expiratory volume (FEV), anatomic weakness in the pulmonary parenchyma and pulmonary obstruction are several risk factors commonly discussed in the literature.[1,3,4] This patient had no history of pulmonary disease such as asthma or recurrent bronchitis, or indications that he would have limitations to his respiratory flow-volume curve. However, no pulmonary function tests were conducted. The most severe consequence of POIS would be AGE, which he did not have. There is no clear explanation why some people develop AGE during a POIS injury and others do not.[1,5]

Most pneumomediastinum events are asymptomatic or mildly symptomatic.[6] Standard treatment for symptomatic pneumomediastinum is normobaric 100% oxygen.[6] Treatment with oxygen should alleviate most symptoms. Currently no definitive guidelines exist for what constitutes 'severe' pneumomediastinum. This patient was demonstrating signs of respiratory distress and elevated RR. Treatment with supplemental oxygen provided minimal relief of these symptoms which was the main factor in determining to undertake HBOT.

The chest X-ray was suspect for a potential pneumopericardium. This is generally rare and often benign however it can become life-threatening causing tamponade and cardiopulmonary failure.[3] Radiographic signs may include a decrease in the cardiac silhouette, or 'small heart sign', along with the heart being partially or completely surrounded by air, the 'halo sign'.[3] More commonly, the 'halo sign' finding is not attributable to pneumopericardium but instead further evidence of pneumomediastinum, with the anterior portion of the pleural reflection off the left border of the heart.[1] Although our patient had central chest discomfort, given his normal blood pressure, heart rate and 12-lead electrocardiogram we did not think there was clinical evidence of cardiac compromise. Had there been any

Figure 2
POIS spectrum diagram. These injuries can occur in conjunction or isolation of one another.



evidence to the contrary, cardiac blood investigations would have been a reasonable first step in work up.

The patient did have clinically obvious subcutaneous emphysema, which caused obvious symptoms with his facial swelling and voice changes. Subcutaneous emphysema is a part of the POIS spectrum and most typically ranges from asymptomatic to mildly symptomatic, presenting most commonly with soft tissue swelling/crepitus and voice changes.[7] However, symptoms can be severe and result in airway and/or vascular compromise.[7,8] The challenge then becomes how to successfully manage this type of airway, potentially under positive pressure ventilation, without further exacerbating the instigating pulmonary injury. For this specific patient, had his respiratory status deteriorated and there was concern for worsening tracheal obstruction, intubation may have become necessary. Carefully managing tidal volumes and positive end-expiratory pressure is critical in a patient like this.[8] Also considering other invasive means of trapped gas release, such as subcutaneous drain placement on low suction in areas of gas accumulation, may become necessary in order to avoid worsening the obstruction.[7] In this case, his respiratory discomfort could have been secondary to the subcutaneous emphysema he experienced or, more likely, a combination of the pneumomediastinum and the subcutaneous emphysema. This is further supported by the fact that after treatment in the chamber he clinically improved while there was still obvious clinical evidence of subcutaneous emphysema in the soft tissues of his face and neck.

Only one other reported case of an isolated POIS injury without AGE was found in which symptoms were manifested and treated with HBO_2. That case is from the 1950s; a male performing submarine escape training from 30 msw depth who developed symptomatic POIS injury without AGE or radiographic evidence of pneumothorax on a plain chest film. He was compressed to the depth of near complete symptomatic relief at approximately 608 kPa (50 msw equivalent) and subsequently underwent a US Navy Treatment Table 3.[9] As described above, the present case did not require such extensive recompression in order to achieve relief of symptoms. The patient was taken directly to 131.4 kPa (3 msw equivalent) on 100% oxygen with near complete resolution of symptoms within 10 minutes and was kept at depth for one hour. The US Navy Diving Manual recommends 'shallow' recompression in the setting of severe pneumomediastinum in the absence of a pneumothorax. The table consists of breathing 100% oxygen at 116.5–131.4 kPa (1.5–3 msw equivalent) for one hour or longer if needed.[10] Had this patient not experienced relief of symptoms, treatment could have progressed to deeper depth, while weighing the risk-to-benefit ratio of prolonged recompression to symptom relief.

Pneumothorax is the least common manifestation of PBT, however, the clinical consequences of compressing a patient with a pneumothorax could be severe given the risk of developing a tension pneumothorax during ascent.[1,3,11] Pretreatment evaluation of a PBT patient for pneumothorax is recommended when time and patient stability permit.

There was no evidence of pneumothorax on posterior-anterior and lateral chest radiographs. However, as a recent case report demonstrates, a negative CXR does not mean there is no pneumothorax.[12] There is always the potential a subclinical pneumothorax could be present during treatment and treating providers should be prepared to perform chest tube thoracostomy if this complication were to develop.[3,13–15] Clinical ultrasound and CT scans are more sensitive when compared to radiographs; however, they are not perfect modalities. In addition to being more sensitive for detecting a pneumothorax, a CT can evaluate for the presence of pulmonary bullae or bleb disease with more accuracy compared to plain radiograph.[13–17] These structural abnormalities are known risk factors for POIS injuries.[18] In retrospect, despite the negative CXR and reassuring exam, given the expedience and increased sensitivity, a point of care ultrasound at bedside would have been the ideal imaging to obtain for this patient to ensure he did not have a small pneumothorax that was missed on the radiograph prior to compressing in the chamber.

While the aetiology of this diver's injury is ultimately unknown, it is likely that during his initial uncontrolled ascent from 10 to 4 msw he may have unintentionally and unknowingly held his breath while trying to adjust his BCD. Prior studies which examined breath holding during ascent, found that out-of-air situations or panic due to unfamiliarity with equipment are the most common causes of PBT.[1,2] Given the type of injury and ultimate uncertainty regarding the aetiology, a CT was obtained post-injury to assess for the presence of any pulmonary abnormalities. Despite the negative imaging, and that this patient is an experienced diver with no previous episodes of injury, it was explained to him that given this POIS event his risk of recurrent injury may be higher if he chose to dive again, including the potential for more severe consequences including AGE.[19] For this specific patient the increased risks should be carefully considered before engaging in diving in the future.

Conclusions

This case provides a clinical example of HBOT in the setting of symptomatic pneumomediastinum with clear benefit to the patient. There is a paucity of documented cases of treating symptomatic POIS in the absence of AGE with HBOT. A suitable treatment table is offered as an option in the US Navy Dive Manual for more severe cases of pneumomediastinum in the absence of a pneumothorax. Questions of resource cost, availability and symptom severity should be considered when determining whether HBOT is a good option for a patient with symptomatic pneumomediastinum.

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