RESEARCH ARTICLE

Immersion pulmonary edema: case reports from Oceania

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ABSTRACT

Introduction: We aimed to document identified cases of immersion pulmonary edema (IPE) in divers from Oceania (the Indo-Pacific region) from January 2002 to May 2018, inclusive.

Method: Cases were identified using various sources, including searches of the Divers Alert Network Asia-Pacific (DAN AP) Fatality Database, published case reports, and interviews with survivors who had reported their incident to DAN AP.

Where available, investigations, pathology and autopsy results were obtained. Only incidents diagnosed as IPE by diving physicians or pathologists with experience in the investigation of diving accidents were included. Individual case histories and outcomes, together with brief individual summaries of the associations and possible contributing factors were recorded.

Results: Thirty-one IPE incidents in divers from Oceania were documented. There were two surface snorkelers, 22 scuba air divers and seven nitrox divers which included three closed-circuit rebreathers (CCR). The mean (SD) age was 53 (12) years, 58% of victims were females, and the average dive profile was to a maximum depth of 19 meters of seawater for 25 minutes. Six victims (19%) had previous episodes of IPE. There were nine recorded fatalities. Cardiac anomalies dominated the associated or possible contributing factors. These included valvular disease in 29%, transient cardiomyopathies in 26% and dysrhythmias in 16%.

Conclusions: Previously reported associations of IPE such as exertion, stress, cold exposure, negative inspiratory pressure, hypertension, overhydration, ascent or surfacing, tight wetsuit, aspiration and certain medications were identified. Cardiac conditions were frequent and included chronic disorders (valvular pathology, coronary artery disease) and transient disorders (dysrhythmias, transient myocardial dysfunction, takotsubo or stress cardiomyopathy). It is likely that the chronic cardiac disorders may have contributed to the IPE, whereas the transient cases could be either sequelae, contributors or coincidental to the IPE.

INTRODUCTION

Reviews have documented the history, clinical features, diagnosis, investigations, pathology, treatments, comorbidities and possible contributing factors for immersion pulmonary edema (IPE) [1].

We investigated 31 incidents of IPE in divers from Oceania (the Indo-Pacific region). Most occurred within Oceania and include all the regional cases of which we are aware from January 2002 to May 2018. The following is a summary of the individual cases, with references, indicating clinical and pathological aspects.

We have documented the individual IPE cases, their demographics, dive parameters, clinical history and investigations, while noting the associations or possible contributors in each case. The purpose of the paper is to provide a more detailed picture of the variety of scenarios associated with suspected IPE, and record how these have been managed

METHODS

For fatal cases, ethics approval was received from the Victorian Department of Justice Human Research Ethics Committee (to access data from the Australian National Coronial Information System); the Royal Prince Alfred Hospital Human Research Ethics Committee; the Coronial Ethics Committee of the Coroner's Court of

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Western Australia; and the Queensland Office of the State Coroner. In non-fatal cases victims provided informed, written consent to review and publish their medical and diving data in a non-identifiable form and provided these data for this purpose.

Cases included those reported to DAN AP by survivors, instructors and diving physicians from January 2002 to May 2018, inclusive. These were followed up with interviews where possible, as well as a review of clinical information and investigations.

In some cases, such as those involving fatalities, clinical information was not available. In others, only the formal conclusions were obtained regarding pathology tests and investigations. This is evident in the case reports. In most of these, medical histories, reports and discharge summaries were accessed, all with patients' approval. Any positive symptomatology, such as hypertension, was recorded. However, in the absence of specific documentation, negative clinical features were usually not recorded. Investigation results, both positive and negative, were recorded in the case histories.

A search was made of the DAN AP internal fatality database and associated autopsies for diving-related deaths in Australia during the same period. At autopsy, IPE needs to be differentiated from other disorders, especially drowning [1-5]. Thus, such cases associated with drowning and aspiration syndromes, pulmonary decompression sickness, pulmonary barotrauma, oxygen toxicity, other gas contaminations, and some marine animal toxicities, are excluded. In addition, a Medline search was conducted using the terms "diving," "immersion" and "pulmonary edema" to identify published cases within Oceania. Several of these cases were followed up with the authors in order to obtain further details.

Only cases validated by a diving physician or pathologist with experience in diving accident investigation were included. The main criteria were: symptomatology being related to immersion, clinical and radiological evidence of pulmonary edema, and/or consistent autopsy findings.

Demographic records were compiled and included: Age, gender, maximum depth of immersion, dive duration, type of diving (snorkel, scuba air, nitrox, rebreather), depth and time of incident commencement, diving experience and previous IPE incidents. The possible contributing factors, as suggested previously by others, were also noted when this information was available.

These cases often occurred in remote localities, and medical records were often incomplete. As a result, the prevalence of these possible contributing factors may be underestimated.

In most, the authors have commented on specific factors that may have been associated with the individual case but which have previously been observed in other series [1]. Some of these associations may have contributed to the IPE, but others may be coincidental or even consequences of the IPE.

RESULTS

CASE 1 (2002) [6,7]

The victim was a 51-year-old, healthy and physically fit female (body mass index/BMI of 24 kg/m^2) with no known cardiac history. During her 20 scuba dives over two years she experienced two episodes where she became exhausted, dyspneic, confused, cyanotic, panicky and required assistance. On another occasion she aborted the dive before submerging, as she had become dyspneic after a surface swim.

On her fatal dive, to a maximum of 12 meters of seawater (msw) for a total of 12 minutes, she performed three well-controlled ascents due to unspecified reasons. On the final surfacing, she was breathing heavily and panicking, although she remained conscious and coherent and swam to a mooring. Once there, her breathing deteriorated, she expectorated pink, frothy sputum and lost consciousness before being towed to shore. Cardiopulmonary resuscitation (CPR) was unsuccessful. Witnesses believed that she had not aspirated any water. Air consumption calculations indicated a respiratory minute volume (RMV) of 37 L.min^{-1}.

Autopsy

The autopsy showed pulmonary edema, resuscitation artefacts and cerebral edema. There were no supportive signs of drowning. Hilar node sarcoidosis was identified, but there was no evidence of pulmonary barotrauma (PBT) or coronary artery disease (CAD). Structural cardiac pathology was detected at autopsy. This included mitral and tricuspid valve degeneration with ballooning of the tricuspid valve and microcalcification of the bundle of His. The myocardium exhibited contraction band necrosis.

Comments

Although unclear of its import in this case, mitral valve degeneration may be associated with mitral incompetence and propensity to increased pulmonary vascular pressures and pulmonary edema. It may be associated with dysrhythmias as well.

Contraction bands are often associated with takotsubo cardiomyopathy (TC) and other stress cardiomyopathies

(SCM) [8-10] but are a non-specific post mortem indicator of stressed myocardium from various causes. The sympathomimetic influences which could contribute to TC/SCM/transient myocardial dysfunction (TMD) include the observed anxiety/panic episodes – and exertion in this case. There is no supportive evidence that these disorders were contributory. Without appropriate premorbid investigations, these differential diagnoses would be conjectural only.

Three previous transitory incidents were consistent with IPE.

CASE 2 (2002) [11]

A 31-year-old female with no relevant medical history and using no medication was undertaking her second pool dive at a depth less than 4 meters for 90 minutes in a water temperature of 20°C. She developed dyspnea, a wheezy cough, watery, slightly pink, sputum and became cyanotic. There was no aspiration of water, overhydration, vigorous exercise, inadequate thermal protection, or other obvious triggers to IPE.

Medical examination

There were widespread crepitations on auscultation, and her oxygen saturation on air was 88%. While breathing 50% oxygen, her partial pressure of oxygen in arterial blood (PaO_2) was 63 mmHg, with an oxygen saturation of 92%. A chest X-ray (CXR) showed pulmonary edema. Extensive cardiopulmonary investigations were negative, including electrocardiogram (ECG), transthoracic echocardiogram (TTE), stress test, histamine and hypertonic saline challenges. Other diving and non-diving explanations were excluded, and it was deduced that she suffered IPE. She returned to diver training and completed 50 dives without incident.

Comments

This case exemplified the concept of IPE occurring in a healthy diver.

CASE 3 (2004) [12]

A 52-year-old mildly obese female, an inexperienced snorkeler, was snorkeling off a tropical island. She was taking a beta-blocker and a calcium channel blocker for hypertension.

After 45 minutes, she tried to return 200 meters to shore, but was very fatigued and noted a bubbling in her chest. When she reached the shore she was dyspneic, with copious pink, frothy sputum; and cyanotic. On shore, she noted possible angina-type symptoms.

Medical examination

On hospitalization, the next day, she was much improved, but still had pulmonary edema and an oxygen saturation of 93%. Echocardiogram (ECG) revealed peaked T-waves in the lateral chest leads. Transthoracic echocardiogram (TTE) showed an area of inferolateral left ventricular wall hypokinesis and mild mitral regurgitation.

TTE two weeks later indicated that the left ventricular ejection fraction was reported as normal, with no evidence of regional dysfunction or mitral regurgitation. Cardiac isoenzymes and coronary angiography were normal.

Nine months later her hypertension was labile but persistent, and a right submandibular lump was noted. Dopamine levels were nine times normal. She underwent resection of a dopamine-secreting non-malignant right vagus paraganglioma. She was well on follow-up 14 years later.

Comments

Hypertension per se has been incriminated in IPE causation, as has the use of beta-blocker drugs [1,8,11]. The temporary TTE anomalies were consistent with SCM/TC; this is supported by excess dopamine production. This sympathomimetic action contributes to stress effects by significantly increasing heart muscle contraction force. Mitral regurgitation was evident soon after the incident, but not later.

Of note, the combination of a beta blocker and a calcium channel blocker is interesting, as this combination is usually contraindicated [13].

CASE 4 (2007) [14]

A 72-year-old male with a BMI of 25 kg/m^2 was a very experienced diver. He had a history of coronary artery disease (CAD), and a thallium stress test at age 63 showed exercise-induced myocardial ischemia of the lateral wall of the left ventricle. CAD was treated successfully by coronary artery bypass graft (CABG) at age 65, with no subsequent symptomatology and no significant abnormalities prior to the diving incident. The last stress ECG was normal, one month previously.

On the second day of a remote diving holiday, he performed a 50-minute multilevel dive to a maximum depth 15 msw, with a five-minute safety stop at 3-5 msw. The water temperature was 27°C, and there was a slight current. The dive was innocuous, without any excessive exertion. There were no rapid ascents, saltwater aspiration or other incidents.

At the safety stop he noticed increasing dyspnea, but, attributed this to a low-on-air situation or regulator resistance. He ascended and remained on the surface for about 10 minutes, during which time he had inflated his buoyancy compensation device (BCD) and adopted a head-out vertical position. He noted increasing difficulty in breathing from both his demand valve and his snorkel. He also observed the sensation of fluid crackling (rales, crepitations) in his lungs. He attempted to relieve the dyspnea by pulling on the neck of his tight wetsuit, without effect. Dyspnea, fatigue and the sounds of pulmonary fluid, cough and expectoration of frothy sputum were all aggravated by exertion, continued after he boarded the dive vessel, and then diminished over the next three hours.

After a 4.5-hour surface interval he felt normal and so dived again, on an almost identical profile and environment, but without any incident or difficulty. For this second dive he had dispensed with the tight wetsuit and original demand valve. The new regulator produced no excessive resistance to breathing. On surfacing, he assumed a horizontal position and breathed through his snorkel.

Subsequent clinical developments

After a five-year period of uneventful frequent snorkeling and scuba diving, he developed swimming-induced IPE (SIPE) on three separate occasions, all during moderate exertion snorkel swims. He subsequently developed angina of effort and effort-induced dyspnea, despite sestamibi CT scans and angiographic verification of well-functioning coronary grafts.

Resting TTE showed mild left ventricular hypertrophy (LVH) with normal cavity and function. A stress TTE showed hypokinesis of the inferior and septal walls, resulting in moderate systolic impairment. This segmental dysfunction improved during recovery. The TTE also demonstrated moderate aortic stenosis and mild mitral valve incompetence. The aortic stenosis subsequently became severe and ultimately required an aortic valve replacement.

Comments

This case allowed a comparison of different potential IPE contributors associated with almost identical dives, with both IPE and non-IPE consequences.

Scuba IPE was associated with previous cardiac pathology and coronary artery stenosis (corrected by CABG). Years later it developed again while snorkeling, with mild mitral incompetence and moderate aortic stenosis, but with no evidence of CAD. Moderate aortic stenosis and mild mitral incompetence may increase pulmonary vascular pressures, and this may be aggravated by exertion. Furthermore, challenges associated with immersion may cause mild mitral incompetence to worsen.

In the snorkeling IPE incidents, there were possible environmental contributors, such as exertion and head-out immersion.

CASE 5 (2007) [15]

A 45-year-old female was undertaking her fourth dive on a course. She did not disclose a history of migraine and adult-onset attention deficit disorder, for which she was taking 25-30 mg of dexamphetamine daily.

She was diving at a depth of 26 msw as part of Advanced Open Water training when she signaled that she was low on air, despite her gauge indicating a pressure of 120 bar. The instructor handed her his alternate regulator while he breathed from her regulator, finding no abnormality. She requested to surface; this was controlled by the instructor. She became progressively more dyspneic on ascent. Frothy sputum and vomit was evident when she reached the surface. Shortly afterward, she became unconscious and was towed to shore, where CPR was provided for an extended period but was unsuccessful. There was still 90 bar remaining in her tank.

Autopsy

The autopsy revealed pulmonary edema and post-mortem decompression artefact. The heart showed no structural pathology, but there was minor coronary artery disease, and the atria were dilated. On histology, the only significant finding was a fine patchy replacement fibrosis in the myocardium. There were equivocal signs of cerebral arterial gas embolism (CAGE) which may have been related to CPR.

Comments

The description of this event is highly suggestive of IPE at depth. The cause for this cannot be ascertained from the available evidence but the pre-existing presence of a left bundle-branch block (LBBB), atrial dilatation and sympathomimetic medication may have increased the risks of a dysrhythmia.

CASE 6 (2009)

This 63-year-old female had made more than 500 dives. She was taking proton pump inhibitors for reflux esophagitis, escitalopram for anxiety/depression, and hormone replacement therapy (HRT).

She suffered two separate IPE-related incidents while diving. On the first occasion, she made a dive in calm waters with a temperature of 19°C, completing an uneventful dive to 10.9 msw for 49 minutes. After a surface interval of one hour 47 minutes, she re-entered the water. By this time there was a slight swell and surface current. She was swamped by a wave and may have aspirated "a small amount of seawater" and coughed. During descent she felt short of breath and, on reaching 15.3 msw she remained breathless, despite stopping to slow and control her breathing. After swimming, she became more breathless and decided to abort the dive. She commenced a controlled ascent but shortened her 5-msw safety stop, as she was becoming increasingly dyspneic and incapacitated. The total dive time was 25 minutes.

When she surfaced, her buddy towed her to the dive boat. She was dragged onboard, as she was too weak to assist herself. She lay down and was provided with oxygen (O_2). She was gasping for breath, cyanotic and coughing up pink, frothy sputum.

Medical examination

She was evacuated to hospital by an air ambulance. On arrival, she was dyspneic, cyanotic and with reduced O_2 saturation (86%). CXR and computer tomography (CT) scan revealed evidence of pulmonary edema. Troponin level was elevated significantly. ECG showed abnormalities, including T-wave inversion. TTE showed an overall reduction in left ventricular ejection fraction to 53%, with global hypokinesis.

On the following morning she developed retrosternal chest pain extending to the left clavicle. Coronary artery angiogram was normal, with no evidence of ischemia. The TTE and ECG reverted to normal in a few days, on discharge, consistent with TC/SCM/TMD. Respiratory function tests (RFT) and provocative tests were normal. The final diagnosis was acute pulmonary edema, possibly aggravated by minimal saltwater aspiration and followed by myocardial damage. ECG, TTE and troponins were abnormal, but transitory. After much discussion she decided to continue with selective diving, against advice.

CASE 7 (2010) [Incident 2 of Case 6]

This now 64-year-old female had a previous incident of IPE, described above. She returned to diving. Five months later, after a nitrox course, a repeat diving medical examination, more dives and a tropical diving holiday, she experienced another, albeit milder, episode of IPE.

The conditions were less favorable – surface chop on a large swell, with a strong underwater current and a water temperature of 25°C. She descended to a depth of 30 msw, fighting the current and running short of air (down to 10 bar). On surfacing, she was exhausted and breathless. She was evacuated to hospital by air ambulance.

Medical examination

Oxygen was administered. CXR confirmed pulmonary edema. Blood O_2 saturation was reported to be reduced significantly. Echocardiography was undertaken the next day and was normal. A month later a cardiac MRI and lung function tests, including hypertonic saline provocation were conducted, again with normal results.

The patient returned to diving with restrictions that included appropriate thermal insulation, avoidance of exertion and a low tolerance to abort the dive if she became dyspneic. At the time of follow-up, she had completed approximately 100 further dives (in tropical waters) without incident.

Comments

This was a comparatively minor incident. It may have been aggravated by exertion, stress and increased inspiratory regulator resistance due to a low-on-air situation and resultant increased negative inspiratory pressure.

CASE 8 (2010) [16]

This 49-year-old woman was severely obese (BMI of 41 kg/m^2), with mild hypertension, hypercholesterolemia, anxiety and depression. She was taking paroxetine, alprazolam and levonorgestrel. She had been treated with diuretics for ankle edema and glyceryl trinitrate for possible angina.

Cardiac investigations showed no evidence of infarction. A thallium exercise ECG showed changes during maximal exercise and scan abnormalities were suggestive of reduced blood flow to the anterior wall of the left ventricle. The report stated that this could have been an artefact due to the overlying breast tissue. These changes were asymptomatic and normalized post exercise

She also suffered episodes of dyspnea on land, requiring hospitalization. Chest X-ray showed non-specific changes, and a CT pulmonary angiogram showed no evidence of pulmonary embolism or focal lung or pleural abnormality. She was subsequently prescribed salbutamol, although there was no definitive indication of asthma.

To improve her fitness, she enrolled in a diving course, but did not disclose the above information. During her

first open water dive, she waded for 70 meters, then surface-swam for a few minutes to reach a buoy 170 meters from shore. Wearing 17 kg of weights, she descended very briefly to about 0.5-1 msw before ascending and complaining of dyspnea and "feeling sick". She was observed to be breathing excessively and with a slight wheeze. She was towed to shallower water but was panicking. She coughed when a wave splashed over her face. She then requested salbutamol, which she self-administered four times. She soon deteriorated, became unresponsive and cyanotic with yellow, frothy sputum coming from her mouth. A defibrillator indicated pulseless electrical activity, rapidly decreasing to asystole. Resuscitation was continued for 30 minutes but the victim failed to respond.

Autopsy

Apart from pulmonary edema, there was no evidence of drowning or PBT. The main findings were of an enlarged heart and mild atherosclerosis of the coronary arteries. There were no features suggestive of arrhythmogenic cardiomyopathy. There was some mitral valve thickening of the anterior leaflet, with shortening and thickening of the papillary muscle (possibly mild mitral valve prolapse). Histology confirmed the fatty infiltration of the heart. The atrioventricular (AV) node showed mild muscular hypertrophy and myxoid change in some vessels, as well as in the mitral valve. There was no evidence of asthma.

Comments

Some chronic cardiac pathology was present, but not to a degree to cause death per se. A history of angina could have been a result of microvascular cardiac ischemia, as there was only minimal coronary artery atherosclerosis.

Dysrhythmias may have been a consequence of the undoubted psychological stress, extreme exertion by her standards, mitral valve disease and sympathomimetic drugs (especially salbutamol). Speculatively, these factors could also be contributory to SCM/TC, which is also consistent with the clinical history and the reduced blood flow to the anterior wall of the left ventricle.

CASE 9 (2010) [17,18]

This 51-year-old female was medically and physically fit, apart from childhood eczema and being overweight (BMI of 38 kg/m^2). She was a very experienced and well-qualified diver.

During a 30-meter surface swim against a moderate current she may have aspirated a small amount of seawater when she was swamped by a wave. She developed dyspnea as she rested at a marker buoy prior to descent.

She experienced progressive dyspnea and fatigue a few minutes after reaching 12 msw and aborted the dive. Cough supervened, together with copious pink sputum, wheezing and rattling sounds in the chest during respiration. Paramedics attended, and she was taken to hospital by ambulance.

Medical examination

On hospitalization, pulmonary edema and a transitory obstructive airways disorder were demonstrated. She responded quickly with O_2 and bronchodilators.

A month later the respiratory function tests, asthma provocation and cardiac assessments (including TTE and stress ECG) revealed no abnormality.

She was diagnosed with IPE. Although diving medical experts disagreed regarding her fitness to continue to dive, she did so and completed over 50 more dives uneventfully until her fatal dive, described below.

Comments

Exertion, and possibly aspiration, may have contributed to this IPE. Cardiopulmonary investigations revealed no evidence of chronic disease, although these follow up investigations were delayed and thus transient cardiac pathology such as SCM/TC would not have been detected. She conformed to the diagnosis of idiopathic IPE.

Case 10 (2011) [Incident 2 of Case 9] [17,18]

One year after suffering the IPE incident described above, and without any abnormality on extensive cardiorespiratory investigations, this now 52-year-old very experienced female diver undertook a night dive. After a 30-meter surface swim, she descended to a maximum depth of 18 msw. At 14 msw, and after 25 minutes she signaled her buddy to abort the dive. They re-traced their route and ascended slowly up the reef, the victim repeatedly indicating that she was "not OK." After 37 minutes when they reached the surface, she was dyspneic with cough, expectoration, an audible wheeze, and was incapacitated. She was towed 100 meters to shore, where CPR was performed, unsuccessfully.

Autopsy

The principal anatomical finding was pulmonary edema, with no additional evidence of drowning or PBT. Histological examination of the heart confirmed minor bridging of the left anterior descending (LAD)

coronary artery and showed interstitial hemorrhage and some associated contraction bands within the posterolateral left ventricle wall.

Comments

Moderate exertion may have contributed to the IPE. Resumption of diving after a previous IPE was controversial. Prompt investigations after the earlier incident may have allowed evaluation of a SCM/TC. Contraction band necrosis of the left ventricle myocardium at autopsy can have several causes, including SCM/TC [9].

CASE 11 (2011)

This 27-year-old female tourist was visiting a tropical island and undergoing her second day of an Open Water Diver course.

She had no relevant cardiorespiratory history. She was learning tasks at a maximum depth of 3 msw when she developed a tightness in her chest, difficulty in breathing, and a cough. She was adamant that she had not aspirated seawater (although she may have on the previous day). On surfacing, she developed more coughing and copious pink, frothy sputum.

Medical examination

A local physician recorded the presence of bilateral crepitations on auscultation.

Medical evacuation took several hours, during which she was given O_2 and improved so much that, apart from the CXR which supported the diagnosis of IPE, she was not subjected to cardiac investigations. She was discharged a day later.

Comments

Possibly due to the inadequate interrogation and investigation, no possible contributing factors were recorded. The water temperature was 28-29°C, and there was no excessive exertion required. Without further details she would be assessed as idiopathic IPE.

CASE 12 (2011)

This 59-year-old male had a history of childhood wheezing. He had a BMI of 30 kg/m^2 and hypertension (160/100 mmHg) for which he was taking an angiotensin II receptor antagonist and a beta blocker.

He was an experienced diver, using air and nitrox to a maximum depth of 35 msw. On three previous occasions he had experienced "shortness of breath" related to anxiety during a dive or on surfacing. He attributed this once to a "leaking regulator," but this was repaired and not evident in the other incidents.

He descended to a depth of 16-18 msw in unpleasant conditions, including cold weather, a water temperature of 11°C, surge and choppy surface conditions.

After about 20 minutes he noticed a sensation he described as "breathing wet" from his regulator, although he was certain that the regulator was not malfunctioning. He normally noticed a dry mouth while diving but not on this occasion (or on his previous three episodes of dyspnea). The visibility was poor, and he was feeling cold and anxious, so he decided to abort the dive and ascended with his buddy to the safety stop at 3-5 msw. While there, he noticed difficulty breathing so he decided to ascend after a dive time of 30 minutes. On surfacing, he experienced increased breathing difficulty and began to cough vigorously, expectorating white, frothy sputum with a pink tinge. He described a "rattling" in his chest when he coughed but did not notice a wheeze.

On the surface for about 10 minutes, he assumed a head-out vertical orientation, with BCD inflated. He remained very short of breath, with tightness in the chest and continuous coughing. Upon boarding the boat, he was observed to be cyanotic and was given O_2 via a demand valve. His difficulty in breathing eased after about 10 minutes.

Specifically, he did not notice any restriction to breathing from his regulator during the dive, no aspiration, and there was still approximately 100 bar cylinder pressure on completion of the dive.

Medical examination

In hospital, during the next few hours, he was given 100% O_2, and when this was occasionally withdrawn it was resumed due to a reduction in blood oxygen saturation (SaO_2).

Chest X-ray showed interstitial lung markings suggestive of pulmonary edema. Raised troponin levels (2.05 ug/L [normal < 0.05]) were consistent with recent myocardial damage. His ECG was initially normal, but later showed some ischemic changes, with widespread anterior T-wave changes. A coronary angiogram performed the next day was normal.

Cardiac catheterization revealed no critical coronary stenosis. The left ventricular end diastolic pressure was markedly elevated at 36 mmHg. There were segmental wall motion abnormalities consistent with stress-induced cardiomyopathy (takotsubo), with apical and distal inferior wall hypokinesis.

A TTE performed some days later, revealed normal left ventricle (LV) cavity size and systolic function, mild left ventricular hypertrophy (LVH) and inferoapical hypokinesis. There was mild thickening of the mitral and aortic valves, with mild aortic regurgitation and mildly elevated pulmonary artery pressure.

He performed 12 conservative dives in benign conditions over the following five years but has since given up diving.

Comments

IPE predisposing factors included treated hypertension, previous episodes of anxiety with diving and episodes of dyspnea with diving, possibly due to IPE. On this dive he developed IPE after deciding to abort due to cold and anxiety and while ascending.

Cardiac investigation revealed mild aortic and mitral valvular pathology and no significant coronary artery disease. There was elevation of troponin levels and evidence of left ventricular wall motion abnormalities which may have been pre-existing but which could also be consistent with a resolving acute TC/SCM/TMD. There was also post-event evidence of pulmonary hypertension and diastolic dysfunction which, if present pre-event, could have been predisposing factors as well.

CASE 13 (2011) [17]

This 55-year-old male had a past history of tightness in the chest, hypertension, dysrhythmias and tachycardias induced by exercise, and post-exercise syncope. Cardiological consultation and TTE 15 years earlier revealed a slightly thickened mitral valve leaflet. Cardiac consultations and TTE three years prior to the diving incident revealed mild aortic and mitral regurgitation with mild left atrial dilatation. Repeated cardiac assessments indicated no other significant structural abnormalities or evidence of coronary artery ischemia.

Despite his cardiac issues, he was physically very fit (BMI of 26 kg/m^2) cycling three times per week. He was undertaking physiotherapy for back pain and took diclofenac. He was a recently qualified, inexperienced diver with newly purchased gear.

He carried his own scuba gear 150 meters to the site without obvious cardiorespiratory problems or dyspnea, although his back was uncomfortable. The water temperature was 18°C, there was a mild surface current/chop, and visibility was less than 3 meters. With two companions, he descended to 4-5 msw. He repeated OK signals until, after four minutes, he signaled a need to surface. He did so with a companion. The ascent rate was described as "normal." Bloody fluid was observed coming from his nose. He indicated that he would return to shore by swimming under the pier to a ladder. A divemaster watched him from the landing. He was seen to float into view on the other side of the pier. His BCD was inflated, and he was floating on his back, unconscious. His head was above water, and his lips appeared cyanotic. Blood-stained frothy sputum was oozing from his mouth.

Rescue and retrieval were complicated, as they were unable to get him onto the landing. He was eventually towed to shore, where resuscitation was performed. The time from when he was first noticed to be unconscious and until CPR was commenced was eight to 10 minutes. He was cyanotic with fixed, dilated pupils. His airway was soiled with froth, vomit and water. Intubation and resuscitation were difficult with sputum and vomit present. The defibrillator indicated that the victim was in asystole and Advanced Life Support was implemented. After about 20 minutes there was return of spontaneous circulation (atrial fibrillation/AF).

Medical examination

He was transported to hospital. A CT brain scan was performed and reportedly demonstrated global hypoxic ischemia. Other relevant investigations included CXR. Pulmonary edema was evident as well as bilateral pleural effusions. A brain CT showed no gas emboli. He died several hours later.

Autopsy

The heart weight was normal but, the atria appeared dilated, and both ventricles were hypertrophic. The mitral valve showed thickening and myxoid degeneration of the anterior valve leaflet consistent with mitral valve prolapse. There was less than 10% narrowing of the LAD coronary artery. Histology of the heart showed mild to moderate perivascular and pericellular fibrosis and scattered microscopic subendocardial scars in the left ventricle. No contraction band necrosis was described.

The upper airways contained thin blood-stained fluid mixed with gastric contents. The lungs were heavy, congested and edematous. There was 150 mL of straw-colored fluid in the right pleural cavity and 100 mL in the left. There was an early aspiration pneumonia. No additional evidence of drowning or PBT was present.

Comments

IPE was a likely diagnosis. There was an absence of convincing evidence of cardiac ischemia. Pulmonary edema was severe, and the clinical deterioration with

ascent was consistent with this diagnosis. Mitral valve pathology with atrial dilation may also have contributed to IPE or to a fatal dysrhythmia.

The dive profile and autopsy essentially excludes PBT and decompression sickness (DCS) as initiating causes. The inflated BCD could have aggravated the dyspnea in the same manner as a tight wetsuit.

CASE 14 (2012)*

This 41-year-old female had no significant medical history and was on no medications. She engaged in moderate exercise and had a BMI of 25.2 kg/m^2. She was a moderately experienced air and nitrox diver, with a history of 47 dives. Her first incident was relatively innocuous, the second almost fatal.

Incident 1: During a dive to 28.4 msw for 21 minutes, she was relaxed and noticed no breathing difficulty or resistance to breathing during the dive. She felt fatigue on surfacing and complained of dyspnea and difficulty boarding the boat. She felt dizzy, with shortness of breath, a non-productive cough, subsequent weakness and difficulty with walking.

After a surface interval of approximately 2.5 hours she felt OK and made a dive to 9 msw for 27 minutes. The returning surface swim was in calm water for about 30 meters. She initially used a snorkel, but she developed shortness of breath, and she was much slower than the other swimmers. All symptoms resolved that day.

Incident 2: Three months later. She descended to 36 msw breathing Nitrox 30 for 17 minutes, in good conditions and a water temperature of 17°C. This was her deepest dive, and she was with her instructor. She possibly swallowed some water while on the surface prior to diving, as she was negatively buoyant due to a BCD problem that had caused some distress. She was breathing excessively and overexerting herself until at 15 msw her BCD problem was rectified, and she achieved neutral buoyancy. The next portion of the dive was uneventful: She was swimming well and without any distress. However, at around 12 minutes into the dive and after two to three minutes at the maximum depth, she noted a "fuzzy light-headedness" and wanted to ascend. At 15 msw she had to stop to "catch her breath." She again indicated that she wished to surface, which she did in a diagonal slow ascent. She recalls a "choking sensation" on inhalation. Her companion held her and controlled the ascent, bypassing the safety stop. He noticed a dark fluid from her mouthpiece at about the 7-msw depth. On surfacing she coughed and expectorated pink, frothy sputum. She was unable to inform her buddy that she could not breathe.

She has no memory of the subsequent rescue, but the instructor stated that there was no aspiration during this time. She was hauled onto the boat, unconscious, although breathing weakly, placed into the recovery position and received O_2 via a non-rebreather mask. After a few minutes of O_2 breathing, she began to breathe more easily and became responsive, despite producing large quantities of pink, frothy sputum.

Subsequent testing of the regulator revealed no source of leakage or water entry from the second stage, but there was a possible first stage problem, with the high-pressure seat housing cap which may have reduced the flow volume at some pressures. At no stage was this evident during testing, but could theoretically have resulted in negative-pressure inhalation.

Medical examination

She was evacuated to the nearest hyperbaric unit for assessment. The doctors concluded that recompression was not required, and she was admitted for management of suspected IPE. After about two hours on O_2 and CPAP, her breathing became much easier.

A CXR demonstrated pulmonary edema, which was partly resolved on discharge and normal on the third day. Substantially elevated troponins were noted on admission (182 ng/L) (decreasing to 126 ng/L on day of admission), with leukocytosis of 18,220, 92% neutrophils. Respiratory function testing revealed a forced expiratory volume (FEV) 1.0 / forced vital capacity (FVC) 65%/63% of predicted levels during hospitalization. A month later, the results were 80%/90% predicted.

Specialist cardiac assessment a month later, which included ECG, stress test and TTE, revealed a first-degree and right bundle-branch block (BBB), and a workload of 13.4 METS without any abnormality of valvular action, ventricular function, wall thickness or ischemia.

She subsequently performed two problem-free dives to 10 msw but has since decided to stop diving for fear of a recurrence.

Comments

Higher-than-normal O_2 partial pressures (e.g., as seen with nitrox) have been implicated in IPE [19], although some physiological evidence suggests that this is unlikely [20,21].

*Although this diver had two likely incidents of IPE, it is only recorded as a single case as there was no medical assessment and diagnosis following the first incident.

Excessive negative buoyancy, anxiety and exertion may have contributed to the second incident. High troponin levels are often associated with transient myocardial damage. As formal cardiological assessment was delayed a month, the normal findings are not incompatible with a TC/SCM/TMD diagnosis. Both IPE instances were aggravated by ascent to the surface. Regulator problems may have increased negative inspiratory pressures.

CASE 15 (2012)

A 50-year-old diver with a history of 90 dives was fit and involved in a variety of physical sports. He had no significant medical history.

He performed a boat dive, with a water temperature of 13°C, wearing a drysuit and reported feeling warm and comfortable. His regulator had been serviced recently.

On the first dive of the day he went directly to 37 msw before working up to 20 msw. There was a very strong current; he was working hard, using a lot of air. He was low on air at the safety stop, so he changed to his pony bottle and surfaced. He had not noticed any significant breathing resistance from either regulator used.

After a three-hour surface interval, during which he felt fine, he made another dive to 30 msw, working up the reef to 25 msw. At this point, he noticed that he "did not feel right." He decided to ascend; at about 10 msw his breathing became labored. Thinking this was likely due to his regulator, he changed to his pony bottle, but this made no difference. He ascended to the safety stop at 5 msw. After a few minutes there, he decided to terminate the dive, as breathing had become too strenuous. On surfacing, he found it extremely difficult to breathe. He needed assistance to board the boat and was weak, breathless, wheezing and coughing up pink, frothy sputum. Once on the boat, the crew removed his drysuit and he was given O_2. He was evacuated to hospital by air ambulance.

Medical examination

In hospital he received O_2 and CPAP and improved substantially over one hour. His SaO_2 was 88% on air.

The CXR showed pulmonary edema. Initial cardiac examinations were normal. ECG showed sinus tachycardia with no evidence of ischemia. Troponins levels were not measured.

Cardiac assessment six weeks later revealed repeated ventricular ectopic rhythms on ECG, mild concentric left ventricular hypertrophy on TTE, together with borderline biatrial dilatation.

The victim ceased diving and swimming but engages in regular, energetic cycling and has had no subsequent medical issues over the six years since the IPE incident.

Comments

Negative-pressure inhalation was possible in the first dive, due to a low-on-air situation. Both dives were physically demanding, against currents.

Apart from these possible provoking factors, this case is consistent with idiopathic IPE.

CASE 16 (2012) [22]

A 58-year-old male nurse had a past history of anxiety and depression, reflux esophagitis, paroxysmal atrial fibrillation (AF) and shoulder pain. He was obese (BMI of 38.1 kg/m^2). He was reported to have been non-compliant with his AF medications and had four emergency department admissions over the previous seven years, on one occasion requiring cardioversion. His AF appeared to be occurring more frequently.

He suffered multiple episodes of palpitations with durations from one to six hours. His cardiologist had reportedly recommended a pacemaker, but no device had been fitted. His medications were esomeprazole, ibuprofen, metoprolol and tadalafil, with amiodarone being suspended. He was also taking analgesics for shoulder pain.

He was previously an experienced diver but had not been diving for 20 years until recently participating in some shallow river and shore dives. On the diving medical questionnaire, he failed to declare his cardiac conditions. He participated in three uneventful shallow dives.

On the first "deep dive" of the course, he breathed nitrox with 32% O_2. Conditions were fine, and the water temperature was 16°C.

After about 10 minutes at 30 msw, during which diving skills were assessed, the group ascended to 20 msw for additional tasks. On further ascent to 14 msw the victim lagged and signaled that he was out of air and wished to ascend, despite his contents gauge reading 130 bar. He then ascended quickly in a prone position. On the surface, he was unconscious, floating facedown with his regulator out. He was "bubbling and foaming a brownish liquid from his mouth." CPR was attempted but was unsuccessful. Gurgling sounds were heard with each rescue breath. His dive equipment was found to be in good condition and fully serviceable.

Autopsy

Pulmonary edema was present. There was no additional evidence of drowning, PBT or DCS. The heart weighed 506 g (n = 331–469 g) with a globose shape and dilation of the right atrium and both ventricles. There was a 30% stenosis of the left anterior descending coronary artery. Histology of the heart showed mild patchy subendocardial and perivascular fibrosis and myocyte hypertrophy but no acute ischemic changes.

Comments

The IPE was likely associated with a cardiac etiology. A history of clinically uncontrolled paroxysmal atrial fibrillation was probably significant as the immersion-related challenge could have triggered further AF. The resultant hemodynamic effects, possibly influenced by the multiple cardiac drugs, including beta blockers, may have aggravated pulmonary edema.

CASE 17 (2013)

A 61-year-old female had no previous medical history but was found to have a left BBB detected on an ECG performed during workup for cosmetic surgery several years earlier.

After 12 previous uneventful dives, she completed one dive to 22 msw for 35 minutes, took a surface interval of 90 minutes, then made a 19-msw dive for 24 minutes in warm tropical waters off a remote island. The current was strong, and after 10 minutes she began to have trouble breathing. She had 120 bar of air remaining. She started to panic at depth, realizing that she could not continue the dive, and began to ascend, controlled by her companion.

Upon surfacing, she was totally incapacitated and needed to be rescued onto the boat. There she was semiconscious "unable to get enough air." She then lapsed into unconsciousness for about 45 minutes, during which time she was gasping and coughing up fluid and variously "white, yellow and pinkish foam." She became apneic for about a minute until she was administered rescue breathing and given supplemental O_2. She then became tachypneic and tachycardic.

She was evacuated by boat and vehicle to a local clinic. At that stage she remained on O_2 and her breathing settled. Subsequently, well after the incident, she thought that she might have breathed in some water around the mouthpiece when she started to panic.

Medical examination

This was cursory, and no more information was available. She has not pursued further diving but has snorkeled in tropical waters without incident. She has reported no subsequent medical events.

Comments

Overexertion was noted, as were anxiety/panic, all risk factors for TC/SCM/TMD syndromes. However, without echocardiogram or troponin evidence, such diagnoses are speculative. There was no evidence of previous cardiac illness, except for left BBB. A history of some aspiration of seawater was proposed, but this was in retrospect and not reported at the time.

Without definite evidence, this case may be included as an idiopathic IPE, although other possible causes could be considered.

CASE 18 (2013)

This 52-year-old man with a BMI of 29 kg/m^2 was physically very fit, but with factor V Leiden deficiency and asymptomatic non-Hodgkins lymphoma. A recently qualified diver, he routinely coughed on surfacing after dives. He performed two dives to a maximum depth of 24 msw, each for 22 minutes. He coughed a little on exiting the first dive. Neither dive involved exertion, and he did not aspirate water.

He was about to leave the bottom during the second dive when he started to cough. His tank pressure was 60 bar, but there was no noticeable resistance to breathing. He ascended to a 4-msw safety stop when coughing became continuous. He surfaced and managed to board the boat with assistance, and while coughing up pink frothy sputum. He then improved rapidly.

Medical examination

On examination in hospital his SaO_2 was 83%; he was given supplemental O_2. Apart from a slight cough, he was asymptomatic the next day. CXR verified pulmonary edema. TTE and ECG were normal. There was a slight elevation in troponin levels.

A month later his RFT and expiratory spirometry were normal or above predicted levels. Five months after the incident a cardiac assessment showed normal ECG, exercise stress ECG, and exercise TTE.

He has subsequently made several shallow, exertion-free dives without incident.

Comments

Apart from immersion, no likely contributing factors were elicited. The small troponin increase was not explained. It could imply a possible cardiac involvement, although it does occur in settings other than cardiac ischemia secondary to coronary artery disease, and has been reported frequently in cases of IPE [23]. Otherwise, this case conforms to idiopathic IPE and was so designated.

CASE 19 (2013)

This experienced diver was 57 years old, with atrial fibrillation and a history of transient ischemic attacks for which he was anticoagulated on rivaroxaban. On the fourth day of a diving holiday, he was using a Pelagian rebreather (Rebreather Lab, Thailand, 2010) with back-mounted counterlungs. Because of leg cramps, he took extra electrolyte and fluid supplements pre-dive.

The plan was for a 180-minute cave dive with a maximum depth of 13 msw and an average depth of 4 msw. He felt he was struggling with his breathing at times and at about 56 minutes he realized he would not complete the dive plan. After a dive time of 75 minutes he was exhausted and faint and was sucking the mask onto his face uncontrollably during inspiration. He felt his breathing was difficult and that he was not getting enough air. He converted to open-circuit and felt better, breathing well. He then reverted to the closed-circuit rebreather (CCR) without further difficulty.

On surfacing, he noted a small blob of a light pink substance from his mouth. With every breath, he noticed a "gurgling sound and feeling in his chest." On the boat he had difficulty climbing the steps and had problems breathing. He improved after 15-20 minutes.

Medical examination

A diving doctor suggested a carbon dioxide toxicity with possible IPE. The next day he had a CXR, which showed a small amount of congestion indicative of pulmonary edema, and a normal ECG. Pulmonary crepitations were heard.

He discontinued diving for several years but remained enthusiastic. He returned to some relatively basic diving and snorkeling, completing around 30 uneventful dives in good conditions. Four years after his incident, a TTE showed mitral sclerosis and mild mitral incompetence, mild pulmonic regurgitation with normal pulmonary artery pressures. A stress ECG showed atrial fibrillation with a controlled ventricular response. There was no evidence of cardiac ischemia.

Comments

It is possible that the initial symptoms were due to hypercapnia, as diagnosed. Negative inhalation pressures may have been caused or exacerbated by the back-mounted counterlung and the minimal increased density by breathing gases at depth. Negative static lung load itself could predispose to IPE by augmenting intrapulmonary blood volume.

TTE demonstrated mitral valve sclerosis and regurgitation, which would be expected to have increased pulmonary venous pressure. Closed-circuit rebreather (CCR) units commonly run with a partial pressure of oxygen (PPO_2) of 1.3 ATA, which may be expected to result in a reduction in pulmonary arterial resistance. This, associated with increased venous pressure, overhydration and negative inhalation pressure as described above as well as the presence of atrial fibrillation may have tipped this otherwise reasonably fit (11-MET fitness test) man into pulmonary edema. It is of interest that the individual received some symptomatic relief when he switched to open-circuit, which would have both reduced the negative inspiratory pressure and the PPO_2.

The relatively unimpressive CXR may be explained by the investigation being done the day after the subject was asymptomatic.

CASE 20 (2014) [6]

This 56-year-old female was fit and healthy, except for an incident of chest tightness and dyspnea for a few days 10 months previously. This was investigated, but no cardiac anomaly was found. She also had a couple of episodes of shortness of breath on strenuous exertion, which were not investigated. She took no medications, and her BMI was 25 kg/m^2.

She had minimal snorkeling experience. The water temperature was 15-16°C, but she was wearing a wetsuit as she swam among dolphins. During the third swim she complained that she could not breathe, and began to cough, expectorating copious frothy sputum. She continued to deteriorate after being rescued into the safety boat and was very weak, cyanotic and vomiting before becoming unconscious. She died soon after hospitalization.

Autopsy

This verified the pulmonary edema and noted cardiomegaly, but no evidence of cardiac ischemia. There was no additional evidence of drowning.

Comments

Cold-water exposure may have contributed, but this is countered by the use of a wetsuit and the absence of depth exposure. Stress is likely, because of inexperience in snorkeling and the exertion required.

The patient had previously experienced repeated incidents of exertional dyspnea, which could have indicated a temporary cardiac anomaly such as dysrhythmia or TC/SCM/TMD, or more simply a lack of adequate physical fitness for the exertion undertaken. Cardiomegaly implies some cardiovascular pathology.

CASE 21 (2015) [24]

This 67-year-old female was a very experienced air/nitrox diver (>1,800 dives over 12 years), usually diving in warm tropical waters and sometimes against strong currents.

She had no cardiac or respiratory problems. Her BMI was 21 kg/m^2 and she took pregabalin for orthopedic problems, rosuvastatin for hypercholesterolemia and esomeprazole for possible gastric reflux. Six months previously, she had a cardiac CT calcium score of 100 (normal). Hypercholesterolemia was well controlled. Transthoracic stress echocardiography was normal.

She went diving in gentle conditions; mild current, good visibility, and a temp of 19°C. She wore an additional wetsuit vest, as she had been cold while diving the previous day. The additional buoyancy necessitated a head-down descent.

The dive was uneventful, until at 15 minutes and at the maximum depth of 20 msw, when she became aware of "not feeling right" and a slight difficulty in breathing. She indicated that she wanted to ascend, which they did slowly over eight minutes, to the 5-6 msw mark for a five-minute safety stop. There, the dyspnea increased, and they ascended to the surface. She became increasingly short of breath, started coughing and expectorating pink, frothy sputum. She felt a rattling sensation in the chest. She was assisted onto the boat, laid supine and administered high-concentration O_2. She was evacuated to hospital by helicopter.

Medical examination

A diagnosis of acute pulmonary edema was made, and chest crepitations were noted.

A CXR verified the diagnosis of pulmonary edema, with reduced SaO_2. Troponin T levels were markedly elevated to 4,052 ng/L. The ECG showed premature ventricular complexes, left axis deviation and non-specific T-wave abnormalities on lateral leads. A TTE (one hour after admission) showed moderate segmental impairment of systolic function, a left ventricular ejection fraction (LVEF) of 38% and extensive anterolateral, lateral and posterior hypokinesis with mild mitral incompetence. Coronary angiogram (within hours of admission) revealed moderate segmental LV dysfunction (mid-anterior-lateral and inferoposterior hypokinesis with sparing of the apical and basal walls) with only mild diffuse coronary artery disease, not requiring any intervention.

Treatment consisted of 100% O_2, CPAP, diuretics, aspirin and clopidogrel. She was well, without the need for further treatment, after six hours.

A repeat TTE (six days later) showed normal LV and RV size and function; LVEF 62%; Grade 1 (abnormal relaxation) diastolic dysfunction with normal estimated filling pressures; normal estimated R heart pressures (RVSP = 34%); mild (grade ¼) mitral and tricuspid regurgitation. LFT was normal and there were no subsequent abnormal cardiological investigations, or symptomatology.

With the above findings a diagnosis of IPE with takotsubo cardiomyopathy was made.

Comments

She did not consider this to be a stressful dive. There was no excessive resistance to breathing through the regulator. There was no aspiration of seawater and no bubbling sensation in the second-stage regulator. The wetsuit was not overtight. She had no previous or subsequent cardiorespiratory problems. The dive history all but excluded dysbaric illnesses and aspiration, and air consumption (12.4 L.min^{-1}), validated her denial of "stress".

Clinical symptoms and radiology were consistent with IPE. Apparent aggravation during ascent may have simply reflected the natural progression of the problem or worsening of hypoxia as the inspired PO_2 fell. The transiently abnormal cardiac investigations indicated a TC/SCM/TMD, which may have been the cause of the event, although it is possible that TC was precipitated by IPE developing from other causes. There were no predisposing factors other than immersion.

CASE 22 (2015)

This 36-year-old male was a very experienced technical diver, self-described as "somewhat obese" and with hypertension. He was diving overseas, in water temperature of around 4°C, using a CCR. He had overhydrated in an attempt to prevent decompression sickness.

He descended to a depth of 70 meters of fresh water (mfw) before a short 'bounce' to 87 mfw for a few minutes. On returning to 70 mfw, he was aware of difficulty in

breathing and decided to terminate the dive. During ascent he developed coughing into his equipment. He was very dyspneic, exhausted and distressed on surfacing, with a wheeze and a crackling sound in his lungs. His sputum was copious and frothy. He had carried out appropriate decompression, including breathing 100% O_2 from 6 mfw to the surface.

He breathed surface O_2 for four hours, during which time his symptoms improved, becoming asymptomatic after six hours. Decompression sickness was dismissed by the diving medical expert consulted, as symptoms developed at almost maximum depth.

Medical examination

A delayed cardiological assessment was unremarkable apart from hypertension, which was treated.

During a six month layoff, he attended to his obesity and undertook training to improve his physical fitness. He then resumed his diving activities, against medical advice, but not in such cold water and without overhydration. He has performed more than 100 dives without recurrence of problems.

Comments

This case illustrates the possible contributions of immersion, hypertension, cold exposure and overhydration.

CASE 23 (2015)

This 58-year-old female diver (BMI of 27 kg/m^2) had a double mastectomy, hysterectomy, retinal detachments, a right knee replacement and two, small below-knee DVTs, all some years previously. For a month prior to the current diving incidents, she experienced considerable stress.

She had made 3,500 dives over 35 years, including deep and some technical diving, and had experienced two possible episodes of DCS (one aviation-induced). On this occasion, she was on an overseas trip and had arranged some diving. She wore a hired drysuit and 11 kg of weights. On the first day, she had a problem-free dive in a freshwater stream (-1°C).

The next day, she made a dive to 20 meters for 30 minutes on a hydrothermal vent. The sea was very calm with no current, and the water temperature was -1°C. The gradual descent took about 10 minutes, as did the ascent to the safety stop. However, halfway through the dive, the drysuit leaked. She was getting wet and feeling colder but persevered. No other issues were evident. She stated that she did not feel nervous or worried. Toward the end of the safety stop, she developed difficulty breathing, taking rapid, shallow breaths. The symptoms appeared when she was in a head-up, vertical position during the ascent.

Upon surfacing, she was unable to remove her gear to board the boat and required assistance. She developed some coughing but did not observe the nature of the sputum. She then vomited. She felt cold and needed to be warmed. She felt weak and noticed crackling sounds in her chest – more on the left side – and a sensation of wheezing for about 10 minutes. All symptoms resolved after about an hour.

The second incident occurred three days later at the same site. The water temperature was again -1°C; conditions were calm with no current. The dive was to 14 msw for 30 minutes in duration, with slow descent and ascent. The suit remained dry, and the dive appeared to be problem-free until, at about 3 msw near the end of the dive, she suffered the same sensation of breathlessness and associated symptoms as previously described. The symptoms developed when she was in a head-up vertical position, during the ascent.

This was more incapacitating than the first incident, with the diver being rescued and towed to the boat. The chest symptoms were bilateral; she felt weaker and required assistance. She was offered O_2, which she rejected, and she spat out some fluid during the walk to the car. She did not feel well the following day but did not seek medical attention, although some chest tightness persisted.

Medical examination

Medical assessment and cardiological consultations and investigations were performed the following week when she had returned home.

She had mild hypertension and hypercholesterolemia. The initial ECG revealed atrial extrasystoles and ischemic ST-T changes in lateral leads. Repeat ECG showed sinus rhythm with inverted T waves in V3-6. ECG a week later, when the patient was asymptomatic, revealed sinus rhythm with a first degree AV block and ischemic ST-T changes in the anterolateral leads.

A TTE indicated that the LV was of normal size, although there was mild segmental impairment of the left ventricular systemic function, with hypokinesis of the distal anterolateral wall and, more prominently, of the distal anterior wall. There was further hypokinesis of the mid- to distal inferolateral wall and inferolateral aspect of the apex. There was impaired diastolic function, an ejection fraction of 50%, and the left atrium (LA) was mildly

dilated. Troponin was 276, 219 and 144 ng/L on consecutive days, and 17 three weeks later (n < 16). Coronary angiogram, chest CT and cardiac MRI were then normal.

She was treated with statins over the following few weeks, but side effects required these to be suspended. TTE a month later showed sinus rhythm and normal left biventricular size and function. Left ejection fraction was 73%, valvular function was normal, and there were no segmental wall motion abnormalities.

Further follow-up three months later confirmed a normal ECG. The TTE indicated that LV systolic function had completely normalized, and there was no significant valvular abnormality. There was mild left atrial dilatation and impaired diastolic function.

The cardiologist diagnosed takotsubo cardiomyopathy.

Comments

Takotsubo cardiomyopathy was diagnosed, based on the atypical echocardiographic findings (which reversed over the following weeks), abnormal ECG findings (that also reversed over this time), troponin levels that were still high, but reducing, when tested nine to 11 days post incident, and the absence of coronary artery ischemia on investigation. Although TC may have been the cause of IPE, it is also possible that it was precipitated by IPE developing from other causes.

CASE 24 (circa 2015)[25]

A 21-year-old medically and physically fit trainee military diver was exposed to maximal exertion, including an extremely strenuous 2-km ocean swim in tropical water of 30°C. To avoid dehydration, he had consumed 500-1,000 mL of water prior to the swim. In the 30 minutes post-swim, he complained of dyspnea and chest tightness, cough and blood-stained sputum. There was no history of aspiration. He was promptly examined by the attending medical officer.

Medical examination

Over the next two hours his O_2 saturation was 90% and he was given supplementary O_2. A CXR showed bilateral perihilar congestion with air space opacities in the right lower lobe and retrocardiac region, indicating pulmonary edema. His ECGs were normal, as was the heart, radiologically. Cardiac enzymes were normal, but creatine kinase was elevated, possibly related to some mild rhabdomyolysis. Renal and cardiac investigations were negative, including hematological and biochemical screenings.

Over the next two days, he remained afebrile and became clinically well, with CXR and muscle enzymes reverting to normal. He resumed military diver training without incident.

Comments

This case was typical of the swimming-induced pulmonary edema (SIPE) described in healthy military/combat swimmers and triathletes [26,27]. The diagnosis was SIPE aggravated by extreme aquatic exertion (exercise-induced IPE), and possibly overhydration.

CASE 25 (2016)

This female diver was aged 51 years and had a BMI of 35.5 kg/m^2. She gave a history of no significant illnesses, apart from currently experiencing menopause. Her comprehensive diving medical revealed moderate hypertension, possibly related to recent and significant social stress. She had a normal ECG, a normal stress ECG with a Bruce protocol to 9.5 minutes, without issue. A stress TTE showed normal wall motion at rest with all segments hyperdynamic immediately post exercise. There was no evidence of cardiac ischemia.

She engaged in snorkeling on a regular basis, without difficulty and a day earlier had commenced her Open Water Scuba course, with pool and initial open water dive to a maximum of 10 meters, without incident. On the second day, she attempted her second open water dive. Prior to the dive, she noticed palpitations and a "racing" heart rate, which occurred when walking 100 meters to the dive site. She felt very agitated in the water before descent, while in the head-out position. Following a 10-meter swim, she descended to 1 msw depth, when she was distressed by tachycardia, wheezing and rattling in her chest, aggravated by respiration.

She had no exposure to cold, and she was wearing both a wetsuit and a thermal vest. The wetsuit was not excessively tight. Initially no resistance to breathing was observed, no removal of the regulator took place, and there was no aspiration of seawater.

She decided to ascend and after discussion with her diving instructor she rested on the surface for around six minute, during which time her instructor also noted she had a rattling sound with respiration. They decided to descend to a depth of 6 msw to conduct dive exercises. Before she reached this depth, probably around 5 msw, her heart was racing, and the rattling sensation was more evident. She requested ascent and they did so. She then required help in returning to the beach and in removing some of her diving equipment.

During the return 100-meter walk up the beach, she had respiratory distress, including the rattling inspirations and about 10 episodes of coughing and expectoration of blood-tinged (bright red and then becoming pink) sputum. She commenced O_2 breathing at 10-15 minutes after the dive. Dyspnea and inspiratory rattling was noted by all observers. An ambulance was requested, and she was transported to hospital. A subsequent check on the performance of the regulator revealed no obvious abnormalities.

Medical examination

In the ambulance, her SaO_2 was 88% on air; she appeared pale and lethargic, with ongoing blood-stained, frothy sputum. She was admitted to hospital where she remained on O_2 and was largely asymptomatic in less than an hour.

A CXR demonstrated pulmonary edema. Initial troponin T was elevated at 127 ng/L, reducing the next day to 81 ng/L. TTE revealed a LV of normal size and systolic function, borderline concentric LVH, borderline LA dilatation, raised PA systolic pressure of 45 mmHg and mild tricuspid regurgitation.

A follow-up CT coronary angiogram showed normal coronary arteries and a zero-calcium score. A Holter monitor revealed no abnormalities apart from infrequent atrial and ventricular ectopic beats. On subsequent examinations the only anomaly observed was a BP of 148/100. An ECG was normal with sinus rhythm and a TTE showed normal systolic function and no valvular abnormality. Her pulmonary artery pressure had returned to normal.

She was advised not to dive and to avoid snorkeling as well. The provisional diagnosis was IPE and reversible myocardial dysfunction (RMD).

Comments

This was a middle-aged, healthy female who experienced IPE with evidence of a mild reversible myocardial injury. Other than the association of IPE with hypertension there were no obvious causes.

CASE 26 (2016)

A 63-year-old very experienced male diver was diving alone in a protected, shallow bay. After an hour he surfaced and was seen waving an emergency glow stick and heard screaming for help. Lifesavers from an adjoining beach reached him with a rescue board. They conversed with him as they removed some of his equipment. He became cyanotic and unconscious and died during the 10-minute ride to the life savers' clubhouse.

Autopsy

This revealed pulmonary edema in an obese (BMI of 34 kg/m^2) male. There was no additional evidence of drowning or PBT.

Other relevant findings included asymmetrical cardiac hypertrophy affecting the left ventricle and interventricular septum, with non-specific features affecting the heart muscle. The coronary arteries were patent, and there was no evidence of ischemia. Histological examination of the heart muscle showed non-specific features which may be associated with systemic hypertension.

Histologically there was marked myocyte hypertrophy of the left ventricle, with focal areas of subendocardial replacement fibrosis. There was also increased perivascular fibrosis. An adrenal adenoma was found.

Comments

While the changes in the lungs could have arisen from several causes, the pathologist suggested that IPE must be considered as a likely cause.

CASE 27 (circa 2016) [28]

This 58-year-old male was a very experienced scuba diving instructor. He had been diagnosed with moderate mitral valve incompetence, but his diving was not restricted. There was a possible history of an asthmatic reaction with a respiratory infection.

Six months after the mitral valve diagnosis, he noticed dyspnea and cough after swimming exertion. This cleared and two days later he made a dive to 18.7 msw for 56 minutes in water of over 20°C. The dive included a 5-msw safety stop, where he breathed 100% O_2 as a training exercise. After he resumed his ascent, he developed severe dyspnea that worsened when he surfaced. He removed his wetsuit, which felt tight, and resumed breathing 100% O_2. Despite this, he developed a cough and then hemoptysis.

Medical examination

He was hospitalized; his SaO_2 read 96%, on O_2. The CXR and CT scan demonstrated pulmonary edema.

The pulmonary manifestations continued despite the administration of O_2 and antibiotic treatment and remained present for five days. He received three hyperbaric treatments to exclude decompression illness. This complicated the clinical presentation, as did the development of a pyrexia of 39°C.

Apart from raised C-reactive protein and B-type natriuretic peptide (BNP) results, there were no other manifestations suggestive of a community-based pneumonia, DCS or PBT. All other investigations were normal. The mitral valve incompetence was verified.

Echocardiograms performed after recovery demonstrated severe mitral valve regurgitation, which was treated surgically with a mitral valve replacement.

Comments

This case report's uniqueness lies in the protracted nature of pulmonary symptoms beyond the anticipated duration of IPE once the victim was rescued.

Mitral valve incompetence can result in both increased pulmonary vascular pressures and pulmonary edema [29]. Therefore, it may also be a contributor to the development of IPE. However, the duration of pulmonary edema in this case seems exceptional and may be influenced by the severe mitral regurgitation. Where this is due to annular dilation, LA loading such as occurs with immersion may result in acute aggravation of the regurgitation.

The pulmonary symptoms experienced two days before the major IPE incident could well be either swimming-induced IPE, or the development of pulmonary infection, subsequently treated by antibiotics. Pulmonary infection supervening on IPE could also be an explanation for the prolongation of symptoms.

The tight sensation from the wetsuit could be either a contributing factor to IPE or merely an aggravator to the IPE symptoms.

CASE 28 (2017)

This 61-year-old female was overweight with non-insulin dependent diabetes, but no longer taking medication and with no known complications. Her diving medical examiner advised against diving.

On her first open water scuba dive, she donned her equipment, including 8.1 kg of weights, and walked slowly but with breathlessness 50 meters to the beach. She complained that her wetsuit felt too tight. Conditions were "ideal," and the water temperature was 20°C.

The maximum depth at the site was 8 msw; average depth during the dive 4.2 msw. After 30 minutes, and at a depth of 4 msw, she grabbed her instructor and spat out her regulator. The instructor immediately purged her regulator and replaced it. She was wide-eyed, breathing rapidly and shallowly. She clutched her throat before becoming unconscious.

The instructor controlled their ascent and towed her 30-50 meters to shore, with her head being supported out of the water by another diver. The instructor was confident that she had little opportunity to aspirate water. She had white, frothy sputum coming from her mouth. She became unconscious and apneic.

Trained lifesavers were available, and an AED was attached within 10-15 minutes of the incident, but no shock was advised. Continued resuscitation and transfer to a nearby hospital resulted in return of spontaneous circulation after prolonged asystole.

Medical examination

A coronary angiogram was unremarkable. A ventriculogram, showed moderate segmental systolic dysfunction with good basal and apical function, but hypokinesia of mid-anterior and inferior walls. Troponin rose from 25 ng/L on arrival at hospital to 540 ng/L ($n < 16$).

She failed to respond to treatment and died six days later. The autopsy revealed focal contraction bands in the LV, mild thickening of the LV (14 mm) and focal moderate CAD up to 50-70%. There was evident organizing bronchopneumonia and cerebral edema with ischemic changes.

Comments

Ventricular dysfunction was demonstrated; however, its significance after prolonged asystolic arrest and CPR is unclear. The history of excessive exertion for her limited physical capability, and probable anxiety/panic some 30 minutes into the dive is suggestive of an acute underwater event. A TC/SMC/TMD explanation for her IPE is possible, with the pathology in the mid-ventricular region [30], but it is not possible to determine whether these cardiac changes contributed to the IPE event, or were a consequence of myocardial injury during her cardiac arrest. A tight wetsuit could have aggravated the situation.

CASE 29 (2017)

This female diver aged 55 years (BMI of 25.8 kg/m^2) was in good health and physically fit, doing competitive long-distance swimming and regular fitness training. Two years earlier, she was investigated by a cardiologist for weakness and dizziness; it was observed that she had a low pulse rate. However, ECG, stress TTE and Holter monitor readings were normal. At age 38 her brother had suffered from dysrhythmias, paroxysmal atrial fibrillation (PAF). He experienced five such episodes that required electrical cardiac reversion to restore sinus rhythm.

She had made 55 previous dives, all incident-free and mainly in tropical waters. On this occasion, the water temperature was 6–10°C. She was not cold, due to the effective wetsuit she wore, although she had to carry 17 kg of weights to compensate for this. She was anxious for her less experienced buddy diver, her daughter.

They descended to 10-15 msw. She was slightly negatively buoyant. After 10 minutes her daughter indicated that she wanted to ascend. The victim found it harder than expected and, at about 8 msw, began to breathe hard, causing her to feel more anxious. On surfacing she was cyanotic, and her breathing was "wet, rattly and wheezy." On reaching shore, she was very weak, wheezing and coughing frothy, pink sputum. She felt some relief by kneeling on all fours, but not by lying supine. She was evacuated to hospital.

Later review of the equipment revealed an air consumption of 84 bar in the 14-minute dive, greater than was customary for her.

Medical examination

She received relief of her symptoms within one to two hours by breathing O_2, but signs of pulmonary edema persisted for several more hours. These resolved with O_2 and bilevel positive airway pressure (BiPAP). She was given low-dose aspirin and was hospitalized and investigated for six days.

On admission, a CT revealed evidence of pulmonary edema. There was no evidence of pulmonary embolus. TTE revealed sinus rhythm and showed a mild degree of global systolic dysfunction (EF 51-53%). There was a possible mild hypokinesis of the anterior interventricular septum. Initial troponin levels were 156, peaked at 195, then fell to 20 two days later.

A persantine myocardial perfusion imaging (MIBI) test showed no evidence of ischemia. CT angiography revealed no coronary artery disease. Myocardial perfusion revealed no scintigraphic evidence for ischemia, but with equivocal left ventricular systolic function. Paroxysmal atrial fibrillation/ flutter was demonstrated, up to 145 bpm with associated palpitations. A 24-hour Holter monitor showed sinus rhythm with multiple episodes of paroxysmal atrial fibrillation (47% of total).

A month later the ECG and TTE were normal. Over the next two months she had episodes of palpitations and atrial fibrillation. This was treated by her cardiologist with sotalol and flecainide, but these were not well tolerated. Three months after the incident she resumed swim training in a pool, but this, like all other exercise, triggered episodes of PAF and was terminated when she felt unwell. Subsequent treatment focused on reducing exercise-induced dysrhythmias.

Comments

Atrial fibrillation is a recognized risk factor for pulmonary edema and may have been the precipitating factor in this patient. However, whether this was PAF-induced IPE, or IPE-induced PAF in a predisposed subject is not known.

CASE 30 (2017)

This 55-year-old man, a very experienced technical diver with more than 8,000 dives, had no cardiorespiratory problems and was physically fit, with a BMI of 28.3 kg/m^2. He conducted a feet-first descent to 48 msw using a CCR with over-the-shoulder counterlungs. He then swam horizontally until some 10-12 minutes into the dive. His old wetsuit was a little tight and needed to be stretched prior to the dive. The water was colder than he was used to, and he had also consumed 500 mL or more of water just prior to diving. There was no aspiration or any exceptional resistance from the CCR. Visibility was good and there was no current. Exertion and stress were minimal.

He developed some difficulty in breathing, with cough and a gurgling sound in his chest. He decided to abort the dive and made an ascent in a vertical head-up position. His symptoms became worse during ascent and he aborted his decompression staging.

On the surface his symptoms worsened, and copious sputum was blood-stained, with a greenish tinge. On board the boat he felt exhausted, but improved rapidly over 20 minutes, although some symptoms persisted, and his chest felt "tight and raw." He was exhausted for several hours. That night he slept in a sitting position. He was back to normal approximately 12-18 hours later.

Medical examination

No investigations were performed at the time, but his general practitioner sent him for cardiac assessment several weeks later. At that time there were no abnormalities detected on ECG or TTE, but his blood pressure was 140/100 mmHg.

He has since conducted another 26 dives in tropical waters, thermally comfortable while wearing a lycra suit, and using his CCR. On one dive, he became slightly short of breath and felt a gurgling in his chest and aborted without further issues. He is now commencing treatment for hypertension and is being investigated for sleep apnea.

Comments

The initial IPE may have been contributed to by cold-water exposure, overhydration and, possibly, a tight wetsuit although none of these triggers were present in the subsequent episode.

CASE 31 (2018)

This 71-year-old male (BMI of 27.1 kg/m^2) had a history of amaurosis fugax and a knee replacement. Two years prior to the incident, a TTE revealed a subaortic valve membrane and minor valvular aortic stenosis. The remainder of the TTE was unremarkable, with normal left ventricular size and function, normal right ventricular size and function and normal pulmonary pressures. He was advised that he could undertake strenuous exercise and was assessed by that doctor as fit to dive. He was taking clopidogrel, rosuvastatin and aspirin. Overall, he had performed more than 1,000 dives over 36 years, without major incident.

On this occasion he made a dive in tropical waters (28°C) to a maximum depth of 20 msw for 42 minutes using nitrox (31%). On descending to about 4 msw, he had trouble with the buoyancy of his camera equipment and surfaced to remedy the problem. This procedure required some exertion, after which he redescended. At 16 msw and after 20 minutes underwater, during which he encountered no current and was not anxious, he developed coughing, which he did into his regulator while he continued the dive. The coughing increased as he ascended from 18 msw and was greatest after surfacing. The cough produced blood-tinged sputum and he was dyspneic. He became more concerned when he became very tired, requiring many rest stops when walking up the steps back to his accommodation. The symptoms began improving soon after he rested but lasted for some hours.

Medical examination

A CXR on admission showed pulmonary edema. The ECG showed sinus bradycardia. Troponin was 120 ng/L around 12 hours post-incident and dropped to 20 ng/L the next day (n = 0-20). D-dimer reduced from 1,090 to 201 ng/mL over the same period (n = 0-600). The TTE indicated mild aortic stenosis, thickened aortic valve and mild aortic regurgitation as well as a reverse EA ratio indicative of impaired diastolic relaxation. He was discharged and advised that he could perform mild diving the next day.

Follow-up testing on return to Australia confirmed exercise-induced LV dysfunction with exercise-induced left BBB. There was no obstructive CAD.

Comments

Several factors in this case may have contributed to the observed pulmonary edema. His past history of subaortic stenosis and valvular changes as well as his diastolic dysfunction and the vasodilator effects of a higher O_2 partial pressure may all have contributed to elevated pulmonary capillary pressures in the setting of increased preload from immersion. While the troponin rise might well have been initially thought to be indicative of an acute myocardial infaction with associated acute pulmonary edema, this was subsequently discounted with a coronary scan.

CONCLUSION

A total of 31 diagnosed IPE incidents in divers were documented. There were two surface snorkelers, 22 scuba air divers and seven nitrox divers that included three closed-circuit rebreathers (CCR). The mean (SD) age was 53 (12) years, 58% of victims were females, and the average dive profile was to a maximum depth of 19 msw for 25 minutes. Six victims (19%) had previous episodes of IPE. There were nine recorded fatalities.

Factors previously identified by others and which may have been contributors were noted. These included: exertion, stress, cold exposure, negative inspiratory pressure, hypertension, overhydration, ascent or surfacing, tight wetsuit, aspiration and certain medications.

Cardiac conditions were frequent and included chronic disorders (valvular pathology, coronary artery disease) and transient disorders (dysrhythmias, transient myocardial dysfunction, takotsubo or stress cardiomyopathy). It is likely that the chronic cardiac disorders may have contributed to the IPE, whereas the transient episodes could be either the sequelae, contributors or coincidental to the IPE.

This series supports the hypothesis that the more elderly IPE subjects are likely to have comorbidities and be susceptible to IPE recurrences and fatalities unless the contributing factors are able to be identified and addressed.

Conflict of interest and funding statements

The authors have declared that no conflict of interest exists with this submission.

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