RESEARCH ARTICLE

Immersion pulmonary edema: an analysis of 31 cases from Oceania

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ABSTRACT

Aim: To review incidents of immersion pulmonary edema (IPE) from Oceania, to determine the demographics, diving parameters, and comorbidities that may be related to this disorder.

Method: Incidents of IPE, most of which were documented by Divers Alert Network Asia-Pacific (DAN AP) or reported in our medical literature, were analyzed. They included interviews with the survivors and a review of available medical records. Only incidents diagnosed as IPE by specialist diving physicians or pathologists with experience in the investigation of diving accidents were included.

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 msw for 25 minutes. Six victims (19%) had previous episodes of IPE. There were nine recorded fatalities in this cohort.

Medical comorbidities were recorded in 68%, with 42% being cardiac. The latter included valvular disease in 29%, transient cardiomyopathies in 26% and dysrhythmias in 16%.

Conclusion: IPE was more likely in middle-aged females, in experienced divers, and during ascent or after surfacing. Commonly reported associations such as exertion, stress, cold exposure, negative inspiratory pressure, hypertension, overhydration, tight wetsuit, aspiration and certain medications were identified.

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

Pathological and historical perspective

Pulmonary edema is a well-described clinical entity with established causes, recognized symptomatology, standardized investigations and accepted treatments [1]. If it occurs during immersion, then the diagnosis is more problematic, and the etiological factors are more numerous and less validated.

Pulmonary edema develops when fluid accumulates in the lung tissue or alveoli [1]. If the pressure gradient between the pulmonary capillary and the alveolar space exceeds the functional integrity of the capillary/alveolar membrane, fluid accumulates in the alveoli. Thus, a rise in pulmonary capillary pressure, or a reduction in alveolar pressure, or damage to the alveolar-capillary membrane, may contribute, alone or in combination, to pulmonary edema. Also, capillary hydrostatic pressure may exceed the plasma oncotic pressure, favoring transudation of fluid into the alveolus and cause pulmonary edema.

Pulmonary edema in scuba divers (SDPE) was established as a bona fide, if rare, condition when reported in 1981 [2]. It presents clinically with dyspnea, fatigue, cough, frothy expectoration which is often blood-stained, hypoxia and auscultatory signs of pulmonary edema. It is confirmed radiologically by chest X-ray and/or pulmonary CT scan. The clinical symptoms, diagnostic investigations and treatments are not contentious, and so are not analyzed further in this report. Other diving disorders that may contribute to pulmonary edema need to be excluded and are described elsewhere [3].

The early SDPE case series comprised only a handful of otherwise healthy subjects, selected to validate the entity of SDPE and exclude other diagnoses [2-5]. Nevertheless,

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the major initiating factor, the physiological effects of immersion per se, was complicated by two other observed stressors: systemic hypertension and excessive physiological responses to cold exposure [2,4].

Pulmonary edema while swimming was observed, occasionally, in victims who also experienced SDPE. Later it was reported in very fit swimmers, after extreme exertion or overhydration. This latter condition was labeled swimming-induced pulmonary edema (SIPE) and was recorded especially in combat/military swimmers undergoing severe endurance training [6,7]. More pulmonary edema cases were described in triathletes (during their aquatic activities) and even in other species (race horses). There was a presumed relationship between SIPE and the well-described exercise-induced pulmonary edema [8,9].

The entities SDPE and SIPE are collectively termed immersion pulmonary edema (IPE). The demographics of these subgroups differ, as do some of the other contributors and comorbidities.

Subsequently, other series from the Northern Hemisphere included cases with cardiac disorders, either permanent or transitory, associated with SDPE [10-12]. Fatalities from SDPE were reported from the Southern Hemisphere [13].

In a seminal study at Duke University, subjects who had reported symptoms consistent with IPE were monitored and compared to controls who had not [14,15]. They were exposed to immersion, hyperbaria and hyperoxia. Higher pulmonary artery pressures and pulmonary capillary wedge pressures were found in those with IPE, indicating that IPE can be a form of hemodynamic pulmonary edema. It was deduced that cardiopulmonary disease may be a common risk factor in the recreational divers/swimmers (with a mean age of 47.8 ± 11.3 years), whereas pulmonary hypertension may be more relevant in military divers/swimmers (mean age 23.3 ± 6.4 years). They concluded that the role of underlying cardiopulmonary dysfunction may be underestimated, especially in older swimmers and divers, and an episode of IPE should prompt the evaluation of cardiac and pulmonary function.

SDPE had evolved from a presumed disorder of healthy divers into a disease of older divers with multiple comorbidities, especially cardiac [16].

An increasing number of divers have presented with IPE over the last decade, possibly because of the increasing age of divers, a greater recognition of this disorder during that period and a greater use of specific cardiac investigations.

Most of the clinical series described to date are from Europe and North America, with the demographics, diving profiles, possible contributors and comorbidities reflecting the diving population of those regions. Our report is based on cases in Oceania (the Indo-Pacific region), and so complements the previous series. More detailed summaries of the individual cases on which this report is based are available [17].

METHODS

For reporting of 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 Western Australia; and the Queensland Office of the State Coroner.

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, and a review of clinical information and investigations. These 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.

A search was made of the Divers Alert Network Asia-Pacific (DAN AP) internal fatality database and associated autopsies for diving-related deaths in Australia during the same period. In addition, a Medline search was conducted using the terms "diving," "immersion" and "pulmonary edema" to identify cases reported within Oceania. Several of these were followed up with the authors in order to elicit 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 autopsy findings. Other diving related causes of pulmonary edema were excluded.

We have not itemized the already well-established features of IPE, such as symptomatology and treatment. We have instead documented the features associated with IPE. These may be causative, provoking or contributing factors.

The demographic records were compiled and included age, gender, maximum depth of immersion, dive duration,

Table 1. Demographics of IPE cohort

age (yr)	mean (SD) 53 (12), range 21-67
gender F/M	18/13 (58% female)
maximum dive depth (msw)	mean (SD) 19 (18), range 0-87
incident depth (msw)	mean 14, range 0-70
dive duration (min)	mean (SD) 25 (14), range 4-56
previous episodes of IPE	6/31 (19%)
dive experience* (n = 26)	novice 6/26, moderate 4/26, very 16/26

*novice , <10 dives, moderate 10-50, very > 50

type of diving (snorkel, scuba air, nitrox, rebreathers), depth and time of incident commencement, dive experience and previous IPE incidents. Possible contributing factors, as described previously by others, were also noted when this information was available. These cases often occurred in remote localities, and medical records were sometimes unreliable or incomplete. As a result, the incidences of these possible contributing factors are likely to be underestimates.

The possible aggravating factors, comorbidities and physiological stressors included:

- negative inspiratory pressure (regulator resistance, reduced gas supply, depth, spatial orientation and counterlung positioning);
- chronic cardiac pathology (mitral or aortic valve disease, ischemic heart disease, myocardial fibrosis, ventricular hypertrophy, etc.);
- transient cardiac disorder, (dysrhythmias, stress cardiomyopathies);
- hypertension;
- cold exposure;
- aspiration;
- overhydration;
- drugs;
- ascent or surfacing ;
- anxiety/stress;
- exertion;
- tight wetsuit;
- others.

Clinical summaries of the incidents put these associations and possible contributing factors in context and are available [17].

The subject's decision to continue with an immersion/diving activity after an IPE event was noted in some cases. However, this information was often not available.

The autopsy findings in our cohort were recorded, and the number of cases ending in death was noted. The survivor cohort was derived from victims who received medical attention for IPE and thus was likely an underestimate of the total incidence of IPE.

Some associations, such as medication usage, were recorded but without clinical knowledge of their relevance. Others, such as negative inspiratory pressure, were deduced from the dive data.

The transient cardiomyopathy (stress cardiomyopathy, takotsubo cardiomyopathy and other reversible myocardial disorders such as reversible myocardial dysfunction – SCM/TC/RMD) was assessed based on the clinical data available. As these transient cardiomyopathies are not well defined, an arbitrary checklist was designated, viz: The diver must have a confirmed diagnosis of IPE and have no evidence of coronary artery disease or myocardial ischemia on conventional cardiac investigation or autopsy. With this prerequisite, three of the following five characteristics were required:

- transient ST-T changes on the ECG, from the time of the incident;
- transient elevation of cardiac enzymes (especially troponins) from the time of the incident;
- ventricular wall motion dysfunction;
- excessive stress events;
- typical myocardial histopathology at autopsy.

RESULTS

Details of 31 cases of IPE from Oceania, from January 2002 to May 2018, inclusive, were documented [17]. There were two surface snorkelers, 22 scuba air divers and seven nitrox divers, which included three closed-circuit rebreathers (CCR). The relevant demographics are shown in Table 1.

Death, with autopsy findings, coroners' inquests and boards of inquiry, occurred in 9/31 cases. Cardiac anomalies were substantial but diverse in 13/31 cases. These included:

- mitral or aortic valve disease of greater than mild severity in 9/31;
- transitory cardiomyopathies (SCM/TC/RMD) in 8/31 cases;
- ventricular wall dyskinesis in 6/31;
- a significant rise in serum troponin level was recorded in 8/10, a slight rise of 4x normal in one and no rise in another. In none of these cases was there other evidence of myocardial ischemia or infarction'
- dysrhythmia clinical history (other than ventricular premature beats) in 5/31;
- autopsy findings included left ventricular hypertrophy (5/9), mitral or aortic valve disease (4/9), myocardial fibrosis (3/9) and contraction bands (3/9).

Left heart impairment, exacerbated by situational factors, was the presumed contributor to IPE in most of these cases. Detailed and more specific cardiac information is available elsewhere [17].

Hypertension was present in 7/31 cases. Excessive exertion was specifically noted in the clinical history in 12/31 cases, and anxiety/stress in 11/31.

It is possible that drugs may have contributed to between 6-10/31 incidents, mainly beta blockers [18], but also sympathomimetics, arrhythmogenics, diclofenac and once with possible edema-inducing pregabalin.

Excess negative inspiratory pressure was likely in 3/31 of cases, based on subjective assessments. This was implied from the divers' impressions, regulator resistance, and a low-on-air situation, postural effects with surfacing and CCR dynamics.

A tight wetsuit or buoyancy compensation device was noted in 6/31 cases.

Possible slight aspiration or swallowing of seawater was elicited on specific interrogation on three occasions. This was an admitted possibility, more than an observation, and was not followed by the typical salt water aspiration syndrome [3]. One case specified swallowing sea water. In the one definite case of aspiration, this occurred on a previous dive a day earlier than the incident and without clinical symptoms supervening

Only 5/31 incidents were in cold water (15°C or lower), and even then, there was appropriate insulation worn and rarely an excessive cold sensation described. At least 10/31 were in waters > 20°C; full temperature range was -1°C to 30°C.

Voluntary over-hydration was possible in 4/31.

The maximum dive depth averaged 19 msw. Symptoms commenced at an average depth of 14 msw after an average of 24 minutes diving, and worsening upon ascent and surfacing.

Some individual cases had very specific possible but contentious contributing factors, such as asthma, diabetes, an adrenal adenoma, a dopamine-secreting non-malignant vagus paraganglioma, and gas toxicity.

Only 7/31 incidents could be classified as idiopathic IPE, and some of these may well have been so designated because of inadequate or delayed interrogation and investigation. Possible contributors to the IPE incidents are shown in Table 2.

DISCUSSION

IPE Demographics

Explanations for most of the possible contributing factors are postulated elsewhere [1-5,8,18,19].

In the Oceania case series, the clinical symptoms of IPE replicated those of other series [2,4,5,10,11,12,19]. In most of the larger Northern Hemisphere IPE case series, females dominated, as did older divers, and they were diving in cold water (< 15°C) to an average depth of 37 msw [5]. The earlier series excluded those with cardiac comorbidities.

The demographic factors in this Oceania IPE series are consistent with the various North Hemisphere series in that they were older than the average scuba diver, with females predominating – reversing the gender ratio of the diving population [20]. Most were experienced divers. There was also a high incidence of recurrences.

The diving circumstances of the Oceania cohort did differ in some respects from the Northern series. Our cases were in warmer waters, with shallower shorter dives and less decompression stress. They had an overwhelming predominance of cardiac co-morbidity and a higher fatality rate.

Immersion

The one common feature in this disorder is the exposure to immersion. This causes a central blood shift, which increases pulmonary capillary pressure and the cardiac preload. Immersion with cold exposure increases vasoconstriction and systemic hypertension, with an increased afterload on the left heart [8,18,19,21]. These two factors together create an increased pulmonary capillary/alveolar gradient favorable to the development of pulmonary edema. Engorgement of the pulmonary vessels may contribute to capillary stress failure [1].

Table 2. Associations and possible contributors to IPE

	frequency	%
ascent or surfacing	21/29	72
medical comorbidity	21/31	68
cardiac comorbidity	13/31	42
mitral/aortic valve dysfunction	9/31	29
transient cardiomyopathy	8/31	26
dysrhythmia	5/31	16
exertion	12/31	39
anxiety/stress	11/31	35
drugs	6-10/31	26
hypertension	7/31	23
tight wetsuit/BCD	6/31	19
cold water exposure	5/31	16
aspiration/swallow water	5/31	16
overhydration	4/31	13
negative inspiratory pressure	3/31	10
nil identified other than immersion	7/31	23

Immersion and the many physiological implications of diving may trigger the development of IPE. This and previous studies attempt to recognize these aggravating factors.

Without any evident contributing factors, other than immersion effects, IPE could be classified as idiopathic. If there are evident provoking or contributing factors, other than immersion, IPE could be described as symptomatic, to be qualified by the contributing factors.

Comorbidity

Previously reported subgroups of Australian divers have had 28-34% medical comorbidities with only 2-3% being cardiac [20]. However, the prevalence of comorbidities in the non-IPE Australian diver groups is likely to be underestimated, as they were not subjected to the same degree of medical examination as this Oceania IPE series.

In the Oceania IPE series, medical comorbidity was present in 68% (mean age 53 ± 12 yr) of the divers, with 62% of these being cardiac. In a Duke University study, 36 IPE subjects were identified (mean age 50.11 ± 10.8 years), of whom 72.2% had one or more significant medical comorbidities [14].

Ascent and surfacing

Ascent to the surface and exiting the water is required once IPE has developed [3]. However, aggravation of IPE on ascent and after surfacing was encountered in both the Oceania and Northern Hemisphere series [5,19,22, 23]. This accentuation of IPE could be explained by:

- spatial positioning effects on negative static lung loads, whenever the diver is ascending vertically or in the head-out position on the surface;
- reduction of respiratory oxygen pressures during ascent;
- redistribution of pulmonary edema fluid from the expansion of intrathoracic gas, due to Boyle's law;
- The natural progression of the disease with time.

Cardiac disorder

Chronic cardiac pathology is recognized as a cause of left ventricular failure and may be a basis on which IPE develops. The pathologies include cardiac ischemia, coronary artery disease and structural disease, especially with mitral and aortic valve dysfunction.

Transient cardiac disorders are also associated with IPE, either as contributors or sequelae. These include dysrhythmias and transient cardiomyopathies.

A recent review highlighted the increasing association of IPE with cardiac dysfunction and deaths [16]. This and multiple case reports also demonstrated an association of IPE and takotsubo cardiomyopathy [22,23,24]. Transient cardiomyopathies encompass such clinical subdivisions as SCM, TC, "atypical TC" and RMD, depending on which characteristic appears more prominant [25]. Coronary artery disease needs to be excluded for these diagnoses. In one series of 54 divers with SDPE, 28% were found to have RMD, including takotsubo. Most had elevated cardiac troponin levels, electrocardiograhic and/or echocardiographic abnormalities [26]. These disorders could be either a consequence of IPE or a contributor to it.

Takotsubo cardiomyopathy was first described in 1990. It is a clinical syndrome that occurs in the absence of significant coronary artery pathology, but presents as an acute, reversible disorder of the heart, with dyspnea or pain. It may be related to a catecholamine release causing a "stunning" of the myocardium. A stressful physical or emotional trigger is often, but not always, present. Women are affected in 90% of incidents, recurrences are common and there is a fatality rate of about 5% for those hospitalized. There is a transient left ventricular wall dyskinesis, often presenting as a ballooning and usually demonstrated by TTE. ECG abnormalities are often suggestive of ischemia. There is a modest elevation of cardiac enzymes and troponins [25]. Because it is transient, delay in investigation can result in a failure to diagnose this disorder.

The incidence of transient cardiac abnormalities is almost certainly an underestimate, as during the chronological first half of this series, cardiac investigations were less frequent and less extensive. During that period the association of IPE and transient cardiac disease was only just being recognized. Thus, for example, cardiac troponin levels were performed in only 3/10 in the earlier half of the series.

Autopsy findings of SCM/TC vary from insignificant cardiac histology to focal necrotic lesions, inflammatory changes, interstitial fibrosis and contraction bands (myocytolysis) – especially in the left ventricle and possibly dependent on the survival time.

Exertion

During strenuous exercise, the lung must accommodate a more than doubling of the pulmonary vascular flow [1]. Elevated pulmonary artery and left atrial pressure, coupled with a decreased intrathoracic pressure during inspiration, results in increased capillary transmural pressures and the exudation of fluid from the capillary to the alveolus.

Anxiety/stress

Anxiety is a contributing factor to many of the IPE scenarios. It is associated with hypertension, increased respiration, cardiac disorders such as ischemia, dysrhythmias and SCM/TMD/TC.

Equipment constriction

This may contribute to increased work of breathing, centralization of blood volume, and anxiety. However, any relationship between IPE and a tight wetsuit or buoyancy compensator remains conjectural.

Drugs

Drugs may influence the likelihood of IPE. Negative inotropes, including beta blockers, weaken muscular contraction and have various cardiorespiratory effects. Sympathomimetics may potentiate hypertension, dysrhythmias or SCM/TC/TMD. The contribution of drugs was conjectural and could not be quantified in this report.

Hypertension

Although systemic hypertension may play a role in increasing the afterload on the left heart, and despite its association with the other cardiac stressors (e.g., cold exposure, anxiety and exertion), this was not as frequent as anticipated. Nevertheless, its incidence was greater than recently reported in some other Australian diver cohorts, including divers of a similar age [20].

Minor aspiration/swallowing of seawater

The occurrence of subclinical minor aspiration or swallowing of seawater in the Oceania series did not replicate the drowning syndromes or the saltwater aspiration syndrome, as described [3]. It may still have contributed to IPE by damaging the integrity of the pulmonary alveolar-capillary barrier, if seawater was inhaled directly, through a snorkel, or sprayed from a leaking regulator.

Negative-pressure inspiration

Negative respiratory pressure may occur under certain circumstances. This reduces intra-alveolar pressures, increasing the capillary-to-alveolar gradient and may enhance pulmonary capillary engorgement. Negative respiratory pressure may occur from a poorly tuned regulator, with increased resistance to breathing, increased gas density with depth, and increased respiratory volumes with exertion or anxiety. With CCRs, the added respiratory resistances of the equipment, the relative position of the counterlung and the spatial orientation of the diver may influence the extent of the negative inspiratory pressure.

Cold

Exposure to cold may intensify systemic hypertension and thus increase afterload on the left heart, but thermal stress was not particularly frequent in this series. Most incidents occurred in temperate or tropical waters and with adequate thermal protection. Thermal protection does not preclude the cooling effect of the gas laws, however. As respiratory gases are decompressed prior to inhalation the temperature of the gas decreases. This is inevitable with use of open-circuit scuba. Cold could potentially increase cardiac preload, enhancing redistribution of blood volume to the central circulation.

Voluntary overhydration

This was not common and not excessive in this series, as it was in others. The rationale for its use was to reduce decompression sickness.

Oxygen

Although there is no disagreement regarding the value of oxygen supplementation in treatment of IPE, there is no such consensus regarding its preventative effect. Seven of our cases were classified as technical divers employing a higher partial pressure of oxygen (PPO_2) near the end of the dive in which they developed IPE.

Recurrences

As IPE is purportedly a rare event, recurrences imply a predisposition. This recurrence may be based on individual susceptibilities, possibly from one of the medical comorbidity disorders, or contributory diving and environmental conditions that are then replicated to produce a recurrence.

The current experience with IPE, especially related to recurrences, cardiac implications and fatalities, has imposed a more conservative approach to resumption of scuba diving or snorkeling. This is discussed elsewhere [3,16].

LIMITATIONS

As with most other clinical series of IPE, it is likely that there is a selection bias by recording the more severe and fatal events, because these are the ones likely to be investigated or be reported.

Many cases of IPE, even some relatively severe cases, rapidly improve after retrieval from the water and thereby avoid medical intervention. Some IPE events are misdiagnosed as an aspiration/drowning incident and other cases are only reported in the internet blogospheres. Thus, it is reasonable to assume that IPE is often undiagnosed, and its frequency underestimated.

There is particular difficulty in differentiating between IPE and drowning at autopsy and cases of IPE may be attributed to drowning [3,27,28].

The associations of some transient factors could in some cases be the result of IPE rather than its causation.

It was not possible to incriminate or quantify certain possible contributors. These include: cold-water exposure (as there were far more cases in temperate and tropical waters), the influence of drugs, and the relative influence of negative inspiratory pressures (as these were unrecorded and thus a matter of clinical conjecture).

CONCLUSION

In this series of 31 IPE incidents from Oceania, cardiac disorders dominated the co-morbidities. These included mitral or aortic valve disease, stress cardiomyopathy, takotsubo or reversible myocardial disorder and dysrhythmias.

Coronary artery disease, dysrhythmias and structural cardiac pathology should be investigated and excluded in possible IPE cases.

Prior to 2000 there were few cases of SCM/TC/RMD or other takotsubo-type cardiomyopathies being reported, and none with IPE. As over a quarter of our cases were identified as having a transient cardiomyopathy, it is likely that this association with IPE is not coincidental. All cases of IPE should include an early chest X-ray or CT scan, ECG, TTE and cardiac biological markers, including cardiac troponins. If abnormal, these tests should be repeated.

Other suggested contributing factors such as exertion, cold exposure, negative inspiratory pressure, hypertension, overhydration, tight wetsuit, aspiration and certain medications were also identified in various cases.

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

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