A REVIEW OF CASES OF PULMONARY BAROTRAUMA FROM DIVING

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ABSTRACT

Pulmonary barotrauma is a condition where lung injury arises from excessive pressure changes. Five cases of pulmonary barotrauma from diving have been seen at the Naval Medicine and Research Centre from 1970 to 1991. Four suffered surgical emphysema while one developed cerebral arterial gas embolism (CAGE). These cases are presented and discussed, including the predisposing factors, common signs and symptoms and essential treatment of this condition.

Keywords: *Pulmonary barotrauma, cerebral arterial gas embolism.*

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INTRODUCTION

Pulmonary barotrauma is a condition where lung injury occurs as a result of excessive pressure changes. It arises because of the inverse relationship between the volume of a given mass of gas and its absolute pressure, representing a clinical sequelae of Boyle's Law.

The excessive change in pressure has always been emphasized but it is the change in volume of the enclosed gas that causes the tissue damage; it is also an individual's susceptibility to volume change that determines, whether one will suffer from barotrauma for a given pressure change. Thus, since volume changes are proportionally greater around 1 atm pressure, it is around this pressure zone that barotrauma often manifests[1].

Pulmonary barotrauma used to be peculiar to divers, aviators and chamber attendants as they experienced in their work great changes in breathing gas pressures. More recently, with the advent of mechanical ventilation, anaesthetists are also very concerned about this condition[2]. It occurs commonly enough with the various jet ventilators[3] but has also been reported during cardiopulmonary resuscitation with manual ventilators[4]. More rarely, pulmonary barotrauma can result from exposure to a rapidly changing pressure wave either underwater or in the air, for instance, from a bomb blast. Inhalational drug abuse (cocaine)[5] and toxic gas inhalation[6] can also cause pulmonary barotrauma.

The clinical manifestations of pulmonary barotrauma include signs of pulmonary tissue damage, surgical emphysema, pneumothorax and arterial gas embolism. Five cases of pulmonary barotrauma arising from diving are presented and discussed. These were obtained from the case record files of the Naval Medicine and Research Centre (NMRC), Republic of Singapore Navy, from 1970 to 1991.

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CASE REPORTS

Case 1

HKK, aged 26, a naval seaman made a SCUBA dive on the morning of 20 May 1976. His dive depth varied from 3 to 10 metres. After an hour, he went to his reserve air and started to surface.

Immediately on surfacing, he felt a pain in the whole chest that was aggravated by deep breathing. On reaching shore, he noticed a change in his voice and that his neck was swollen with a crackly feel to the skin. Earlier that morning, he had snorkled to 5 metres but could not equalise his ears.

When seen, his vital parameters were normal except for slight tachypnoea. He spoke with a soft, low husky voice. There was very slight central chest pain on deep inspiration and mild dysphagia. Subcutaneous emphysema was evident from the neck down to the upper chest. The lungs were clear and the heart sounds were normal but faint. There were no neurological signs. Both tympanic membranes had Grade I aural barotrauma. X-rays showed pneumomediastinum with prominent subcutaneous emphysema in the neck region.

HKK was put on a regime of 100% free flow oxygen with air breaks for 48 hours.

His recovery was uneventful with no signs nor symptoms of air embolism although the recompression chamber was initially prepared and kept on standby. His voice returned to normal within 12 hours. The dysphagia lasted 2 days, while the subcutaneous emphysema resolved by day 17. The follow-up X-rays confirmed resolving surgical emphysema. Pulmonary function test with the vitalograph done 3 weeks later did not reveal any changes from the one done 2 months earlier. There were no permanent sequelae.

Case 2

HC, aged 42, a sports diver with one and a half years' experience but without any formal professional training in diving.

On 2 September 1984 at 1300 hours, half an hour into a dive to 30 metres off the coast of Mersing, he was seen to suddenly surface. When the safety boat reached him, he was found to be unconscious with no pulse or respiration. There was blood-stained froth coming from his mouth. His life jacket was inflated and his SCUBA tank was empty.

Cardiopulmonary resuscitation was started and the pulse and respiration returned after 12 minutes although he remained unconscious. He was placed on his side and evacuated to a hospital in Mersing (6 hours away) where he was noted to be in coma. After stabilisation he was flown to Singapore by helicopter. During the flight he threw two fits. He arrived at the Department of Neurosurgery, Tan Tock Seng Hospital, on

3 September at 1200 hours. He was still in coma with a decerebrate response to pain.

He was referred to the NMRC on 4 September at 2300 hours - about 58 hours after the diving accident. A presumptive diagnosis of pulmonary barotrauma with cerebral arterial gas embolism was made based on the history.

Hyperbaric treatment was instituted with oxygen (US Navy extended table 6A) for a duration of 7 hours. No definite changes were noted in his coma state immediately after treatment but he was noted to be in a lighter state on the third day following treatment. A day later he was able to withdraw from pain. Seven days post-treatment he was able to open his eyes. In the following weeks, his condition gradually improved, aided by rehabilitative physiotherapy.

Today, though confined to a wheelchair, he is able to carry out most of his daily activities such as toilet, dressing, eating and movement.

Case 3

CPC, aged 35, a naval mechanic, made a series of training dives on 25 November 1986, each lasting 1 to 2 hours. The deepest depth was 10 metres. The first three dives were made without encountering any problem. His fourth dive was at night and he recalled that he was holding his breath on and off as he was trying to conserve his air. After an hour and a half, he felt sudden pain in his throat and started to ascend.

On reaching the surface, he noticed that his voice was soft and hoarse while his throat felt sore. He also experienced retrosternal chest tightness that was associated with slight difficulty in breathing. There was a crackly sensation around the neck. He rested awhile and felt much better.

At the medical centre the next morning, he appeared well and no abnormal signs except for subcutaneous emphysema over his neck. There were no auscultatory findings. His X-rays revealed surgical emphysema in both the mediastinum and neck region.

He was started on 100% free flow oxygen and put on an oxygen/air regime for 24 hours while in hospital.

There was a prompt, uncomplicated recovery.

Case 4

Immediately after his first compass dive of 60 minutes at 5 metres on 18 July 1989, RR, aged 20, a naval diver, experienced central chest tightness. He also said there was slight breath-holding during the dive as he was attempting to prolong it as long as possible.

After resting for about 4 to 5 hours, he felt much better and went for a night dive of similar depth and duration. After this dive, the central chest tightness worsened and this time it was associated with tightness of the neck muscles, throat pain, dysphagia and left otalgia. He decided to rest for a day to see if the symptoms would resolve spontaneously and only sought medical attention two days after the incident.

When seen, his otalgia had resolved, the chest discomfort had improved though he still had throat pain and mild dysphagia. There was no change in voice quality. Examination revealed normal tympanic membranes, some subcutaneous crepitus in the neck and supraclavicular areas and slight mediastinal hyperresonance. There were no neurological signs or symptoms.

He was given free flow oxygen and sent for X-rays. The X-rays showed some gas in the superior mediastinum and neck region.

His symptoms resolved spontaneously with only very slight chest pain on deep inspiration four days post-incident. His lung function parameters were normal.

Case 5

On 18 February 1991, 20 to 30 minutes after ascent from his second SCUBA Jackstay dive to 9 metres, LKT, aged 20, naval diver, experienced mild tightness and heaviness of the chest which radiated up to the neck. This discomfort resolved somewhat with rest and he proceeded on with the night dive. During this dive, he did not feel any chest pain but had to ascend after about half an hour as he got entangled with his lifeline. He claimed there was no panic ascent.

On reaching surface, he again developed chest tightness which gradually became worse. There was no throat pain nor voice change.

He sought medical attention immediately and was given free flow oxygen. Clinically, he was well. There was no skin crepitus. A mediastinal crunch, however, could be heard. X-rays confirmed pneumomediastinum without subcutaneous emphysema.

He was not given intermittent oxygen therapy as his condition was mild. There was rapid recovery as confirmed by serial X-rays.

DISCUSSION

Decompression pulmonary barotrauma is the result of overdistension and rupture of lung tissue by expanding gas when subjected to a reduction of ambient pressure. Compression pulmonary barotrauma is rare and occurs when the limit of lung compressibility is exceeded. In pulmonary barotrauma of ascent, rupture of alveolar septa allow escape of gas into interstitial spaces which may track along perivascular sheaths and cause mediastinal emphysema and pneumothorax. It may further extend into the pericardium, the retroperitoneum and subcutaneous tissues of the neck. Systemic arterial gas embolism can also occur. However, the precise pathological events in the lung parenchyma which precede and accompany arterial gas embolism is still unknown. Alveolar-vascular membrane disruption is the favoured theory as the base of marginal alveoli which abut on pulmonary vascular sheaths appear to be one area of weakness in the face of alveolar overdistension[9]. Recently, diffuse alveolar haemorrhage[10] and pneumomyocardium[11] have also been reported.

Generally, critical alveolar wall tension can be reached after just one breath of a breathing mixture at raised environmental pressure before returning to the surface. Experiments in animals have demonstrated that intratrachael pressures of about 80 mmHg[12] or transpulmonic pressures of 60 - 70 mmHg[13, 14] are sufficient to cause rupture of alveolar septa. Such critical pressure changes could occur by ascent from a depth of just over one metre, and gas embolism leading to death has followed ascent from a depth of 2 metres.

Failure to exhale adequately during ascent is one causal factor in the production of pulmonary barotrauma and is prone to occur when a diver makes an emergency ascent in a state of panic. However, overzealous attempts to exhale during rapid ascent through water has also been postulated to predispose divers to air-trapping as the airways tend to narrow and act as check valves during forced expiration at low lung volumes[15]. Voluntary breath-holding in an attempt to prolong a dive may lead to barotrauma if sufficient pressure change occurs during the breath-holding episode, as when following a length of rope anchored as varying depths (Jackstay). Cases 3 and 4 had this element of breath-holding. An emphysematous bulla is also a well known predisposition[16]. Retention of air in a segment supplied by a diseased bronchus is another causal factor[17]. Shearing forces set up by tethering of alveoli or pleural adhesions from scarring and fibrosis can also predispose to

barotrauma. Even mild conditions like coryza and influenza can make a diver susceptible to this condition[18], as was probably so in Case 1.

Be that as it may, a few predisposing factors are usually evident, especially in uncomplicated pulmonary barotrauma. In 140 patients, Leitch and Green[19] found only six cases where medical conditions predisposed the pulmonary barotrauma although the authors did not state what investigations were performed to exclude pre-existing pulmonary pathology. At NMRC, all new diver trainees are required to undergo a thorough medical examination including spirometric assessment and a large chest X-ray before commencement of training. Divers suspected of having asthma are given the Histamine Challenge Test. These medicals are subsequently conducted annually.

Arterial gas embolism is the most dangerous complication of pulmonary barotrauma. Only a small volume in the systemic arterial circulation is necessary to produce severe disturbances. In the head-up erect position, the brain receives the bulk of embolic air, while in the feet-up inverted position, the coronary vessels are preferentially embolized[20]. Other tissues affected may include the spleen, liver, kidney or limbs. Serious effects may result from blockage of cerebral or coronary vessels by bubbles in the order of 30 microns to 2 mm in diameter. Elliot et al reported arterial gas embolism with central nervous system involvement in 79 (90%) out of 88 cases of pulmonary barotrauma[21]. Leitch and Green found arterial gas emboli in 117 (84%) out of 140 cases of pulmonary barotrauma[19]. Electroencephalographic studies can further disclose evidence of cerebral gas embolism in the absence of any other suggestive manifestation[22]. Hence, embolisation to the cerebral circulation is common. Case 2 was the only sports diver among the 5 and he did not have any formal training in diving. The other 4 cases of pulmonary barotrauma showed no clinical evidence of neurological involvement. All had only surgical emphysema affecting the mediastinum and subcutaneous tissues. It is interesting to note, however, that air embolism is more commonly encountered in cases of mediastinal emphysema than in cases of pneumothorax[14].

Unconsciousness or collapse is the most common presentation of arterial gas embolism (30% - Elliot et al[21], 38% - Leitch & Green[19], 45% - Brooks et al[23]). Other manifestations include confusion, aphasia, visual disturbances, vertigo, convulsion, paresthesia, varying degrees of paresis, cardiac-type chest pain and skin marbling. Symptoms occur acutely, most often immediately or within 1 to 2 minutes of ascent.

However, it must be noted that many patients with cerebral arterial gas embolism experience some degree of spontaneous recovery, albeit usually transient. This is probably due to the redistribution of emboli from the cerebral arterioles distally into the venous circulation. Many will subsequently relapse, even in those with apparently complete recovery, with either the same symptoms or more frequently, a different set of symptoms[24]. This may be because progressive vascular occlusion from downstream vessel coagulopathy causes failure of collateral reperfusion or because re-embolisation occurs[25].

In uncomplicated pulmonary barotrauma, dyspnoea, cough and haemoptysis are common symptoms of lung tissue damage. As can be seen from our case reports, voice change into a hoarseness or brassy monotone (from left recurrent laryngeal nerve paresis), a feeling of fullness in the throat, dyspnoea, dysphagia and retrosternal discomfort are the usual complaints if surgical emphysema has occurred. Symptoms may appear immediately in severe cases, which may include syncope or shock from tamponade, or may be delayed several hours in lesser cases. The clinical features of pneumothorax resulting from diving are similar to those from other causes. Tension pneumothorax may develop from either coughing or subsequent exposure to subatmospheric pressure eg in an aircraft.

Treatment of air embolism is urgent, must be instituted immediately and must take precedence over treatment for all other manifestations of pulmonary barotrauma. Serious symptoms which develop immediately after ascent may be regarded as air embolism and treated accordingly until a definite diagnosis has been made. Immediate recompression to 50 metres (6 atm) with air decreases the gas bubbles to one-sixth their volumes and allow them to pass through most vessels. Once moving, bubble dissipation and absorption are expedited. Any delay in therapy allows progress of the blood-bubble interaction which leads to intravascular coagulation, increased capillary permeability, oedema, haemoconcentration and infarction. Initial compression to 18 metres with oxygen is also practised in some centres[19]. Ascent on an oxygen therapeutic table is begun when possible. The use of hyperbaric oxygen allows more oxygen to dissolve in the plasma, alleviating the ischemia while the high PO_2 retards progression of cerebral oedema by inducing cerebral vasoconstriction.

In the absence of a recompression chamber, the patient should be placed in the 30 degree head down left lateral position to reduce risk of further systemic embolisation. This posture, however, should not be maintained for more than 20 to 30 minutes because of the likelihood of aggravating cerebral oedema. Oxygen should be administered by mask, both to reduce the existing emboli and to ensure that subsequent ones, if any, are composed of oxygen instead of air.

A major factor determining the success of treatment is the time from onset of symptoms until the patient is given adequate recompression therapy. In general, the shorter this interval, the better the result. In a review of 117 cases of cerebral arterial gas embolism by Leitch, it was found that the cut-off time for favourable outcome was about 4 hours. The likelihood of achieving a cure was reduced to less than 50% when the delay exceeded 4 hours. However, this should not deter a trial of treatment even in the late case. Human cases have been successfully treated up to 24 hours after onset. In our case, HC was started on treatment 58 hours after the diving accident. Although there was no complete cure, he recovered sufficiently to lead an acceptable quality of life.

For cases of surgical emphysema, air embolism or pneumothorax must first be excluded, and if in doubt, treatment for these should take priority. When symptoms are mild, free flow 100% oxygen will increase the gradient for removal of nitrogen from the emphysematous areas. Air breaks are required to prevent collapse of alveoli. If symptoms are severe, therapeutic recompression using oxygen is necessary. Surgical decompression may have to be resorted to as well.

In the four-year survey of the mortality of British Divers, Crockford and Dyer[26] in 1975 traced a fatal accident rate of 11.7 to 15.6 per 1,000 men years of exposure, placing diving as the occupation with the highest risk.

A proper medical screening before pursuing diving as a sport or a profession is therefore absolutely necessary. Instruction in proper diving techniques and avoidance of diving when down with upper respiratory tract infection is also vital. As a continuous safeguard, all divers should be medically examined yearly. A recurrence of pulmonary barotrauma tends to be worse than the first incident and is more likely to include arterial gas embolism[27]. Thus all divers who suffer pulmonary barotrauma should be prevented from further diving.

Military diving institutions have little problem enforcing such regulations with safe, reliable guidelines provided in

exhaustive manuals. However, it may not be possible to do the same thing for commercial diving establishments and sports diving schools. Diving is not for the unwary or untrained. A local authoritative regulating body should be set up to oversee all non-military diving set-ups to ensure that the intrinsically high risk of diving is always minimised.

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