Hyperbaric Oxygen Therapy for A Critically Ill Patient Suffering From Severe Decompression Sickness

Colonel (Dr) Andrew Ng Wei Aun* & Colonel (Dr) Muhd Yusof Abu Bakar**

*Department of Anaestheasiology, 94 Armed Forces Hospital, Terendak Camp, Melaka, Malaysia
**Institute of Underwater and Hyperbaric Medicine, 96 Armed Forces Hospital, Royal Malaysian Navy, Lumut, Perak, Malaysia

ABSTRACT

BACKGROUND A 39 years old diver presented to the emergency department of 96 Armed Forces Hospital Lumut with altered conscious level after an air scuba dive to 56 metres depth. He did not receive any first aid or oxygen resuscitation throughout his transport from the dive site to our hospital. He was intubated in the emergency department and stabilized in the intensive care unit before initiation of recompression therapy in the hyperbaric chamber. However, within 24 hours, the diver succumbed due to severe decompression sickness (DCS). Our case demonstrates a classical presentation of severe DCS and discuss some of the issues in providing hyperbaric oxygen therapy for a critically ill patient.

KEYWORDS Air Scuba Dive, Recompression Therapy, Hyperbaric Chamber, Decompression Sickness (DCS)

INTRODUCTION

Decompression sickness (DCS) is caused by bubbles within the body, which formed when there is a reduction in ambient pressure. Examples include a scuba diver ascending from a depth and a fighter pilot ascending to altitude. The physical basis of bubbles formation is based on Henry's Law. A scuba diver is more at risk of developing DCS when he dives deeper, stays underwater longer and ascends to the surface too rapidly. The deeper and longer a scuba diver remains underwater, the greater the amount of gas that will dissolve in the body tissues. When the scuba diver subsequently returns to the surface, the dissolved gas will emerge as bubbles due to a reduction in ambient pressure. If the rate of ascent exceeds a certain limit, the number of bubbles produced will overwhelm the body's ability to filter these bubbles. Bubbles will travel within the vascular system and be distributed throughout the body, causing various pathophysiological effects. Signs and symptoms of DCS range from mild pain to severe paralysis, unconsciousness and death.

We present a case of a diver suffering from severe DCS after a deep dive followed by a rapid ascent. Although intubated given and intensive level of care while undergoing recompression therapy, he sucuumbed to that given diving illness. This case demonstrates some of the issues when treating a critically ill patient in a hyperbaric chamber.

CASE PRESENTATION

A 39 years old ex-military officer who was working as a recreational dive instructor went for a dive with his dive buddy, a 55 years old certified commercial diver, off the coast of Tanjung Piandang, Perak. Both were contracted divers for a local government agency to obtain sand samples. Presumably, both were relatively experienced divers.

They were diving from a boat using standard scuba gears and standard air cylinder. It was unknown if they had prior planning for diving depth, bottom time or decompression stops. They only used a single cylinder but had arranged a standby cylinder at 20 metres depth. There were no first aid equipment or resuscitation oxygen on the boat.

The dive commenced at 12 noon and the dive computers of the divers showed both of them dived to 56 metres depth. Bottom time was 8 minutes. Realizing their cylinders were emptying fast, both divers ascended rapidly to the surface without any decompression stops. The diver's buddy initially stopped at 20 metres, attempting to use the standby cylinder, but subsequently followed him to the surface.

The diver climbed on board the dive boat and fell unconscious immediately after taking off his dive gear. His dive buddy developed nausea, shortness of breath and weakness of both lower limbs 10 mins after surfacing. Both divers were transported by the dive boat directly from the dive site to Royal Malaysian Navy (RMN) Base Lumut without receiving any first aid or oxygen. They arrived at the emergency department of Armed Forces Hospital Lumut at 1600H, about 3.5 hours after surfacing from the dive.

At the emergency department, the diver had Glasgow Coma Scale (GCS) of 8/15 (E4V2M2), blood pressure of 118/73mmHg, pulse rate of 130/min and saturation of 91% on room air. Cardiovascular and lungs examination were unremarkable. Electrocardiogram showed supraventricular tachycardia. Arterial blood gas (ABG) revealed metabolic acidosis with a pH of 7.16, pCO2 36mmHg, pO2 159mmHg, HCO3 12.5 mmol/L, lactate 6.3mmol/L and base excess of 13. Chest X ray did not show any pneumothorax.

He was intubated due to poor GCS and was planned for recompression therapy. However, post-intubation he became hemodynamically unstable and was transferred to the Intensive Care Unit (ICU) for stabilization prior to recompression therapy.

In the ICU, an arterial line and a central venous catheter were inserted. Noradrenaline infusion was commenced to sustain his blood pressure.

At 0030H, he was transferred from ICU to the hyperbaric unit for recompression therapy. Prior to recompression, his blood pressure was 100/60mmHg, pulse rate was 110/min and central venous pressure was 2mmHg. He was on 0.04mcg/kg/min of noradrenaline infusion and was sedated with 1mg/hr of midazolam/morphine combination. Full blood count was unremarkable except for slight leucocytosis (14.2 x 109/L) and mildly increased haematocrit (58%). ABG revealed partially compensated metabolic acidosis with a pH of 7.31, pCO2 30mmHg, pO2 243mmHg, HCO3 20 mmol/L, and lactate 4.8mmol/L.

Prior to recompression, the air in the endotracheal tube cuff was replaced with saline while the intravenous normal saline bottle was vented with a needle. The patient was connected to a Siare hyperbaric certified ventilator (Mode: Synchronized Intermittent Mandatory Ventilation (SIMV) with pressure support. Settings: pressure control 14mmHg, pressure support 8mmHg rate 16/min, positive end-expiratory pressure (PEEP) 8mmHg, I:E ratio 1:2, achieved tidal volume 420-470ml). Noradrenaline and midazolam/morphine infusion were continued using 2 hyperbaric certified syringe pumps (B.Braun Perfusor). He was treated with USN Treatment Table 6.

During the treatment, the diver's blood pressure was labile, requiring constant adjustment of vasopressor infusion rate. There was an initial sharp rise reading in the blood pressure during compression which was sustained for 15-20mins. Subsequently, the blood pressure swung between a mean arterial pressure of 50mmHg to 110mmHg for the first hour. Thereafter the blood pressure slowly dropped as treatment proceeded. Towards the end of the treatment, noradrenaline infusion was 0.1mcg/kg/min. Heart rate similarly increased during the initial part of the treatment from 110/min to 140/min but subsequently stabilized around 70-90/min. A total of 3.5L of normal saline were infused during the treatment.

Ventilatory parameters were relatively stable throughout the treatment with slightly increasing inspiratory pressure and PEEP to maintain adequate tidal volume (Pressure control from 14 mmHg to 18mmHg; PEEP from 8mmHg to 10mmHg). Sedation was maintained with 1mg/hr of midazolam/morphine infusion. No neuromuscular blockage agent was given. There was no noticeable motor movement during the treatment. Urine output was low at a total of 110mls throughout the treatment.

Recompression therapy was completed at 0700H and the diver was transferred back to the ICU with a plan for the second session of recompression therapy 12 hours later. However, in the ICU, he continued to deteriorate and succumbed at 12 noon the same day.

DISCUSSION

This diver dived to 56 metres, well beyond the allowed depth limits for air scuba diving. Furthermore, when he ran out of air, he ascended rapidly without performing decompression stops. Based on safe diving practice, he should have ascended not more than 9 metres/min and perform a decompression stop at 6 metres depth for 4 mins [1]. He became unconscious almost immediately on getting back on the boat. Any signs and symptoms that occur immediately after diving are likely due to DCS. In this case, the boatman recognised this, which explained the decision he took to travel directly from the dive site to RMN Base Lumut as the nearest location with a hyperbaric chamber. However, he failed to administer first aid or oxygen therapy. The mainstay of early resuscitation and treatment for suspected DCS is to provide the diver with as close to 100% oxygen as possible, for as long as possible, while arranging for expedite transfer for recompression therapy. The oxygen provided has a dual function – a. to alleviate hypoxia from bubble effect; b. to hasten bubble resolution by creating a diffusion gradient [2].

The definitive treatment for DCS is recompression therapy. It is generally accepted that the earlier recompression therapy is instituted, the better the outcome. In this case, the diver was planned for early recompression therapy. However, he became unstable post intubation and recompression therapy had to be delayed. The decision to treat an unstable patient can be a difficult one. Risk versus benefit considerations has to be taken into account by the treating hyperbaric physician in consultation with the intensive care team. On one hand, early recompression therapy may result in the patient becoming less unstable. On the other hand, an unstable, critically ill patient may deteriorate inside the chamber where access to optimal intensive care may be limited due to space, equipment, lighting and staff deficiency. For this case, it is unknown if the delay in recompression therapy by 5-6 hours would have had an impact on the outcome, especially since there was already a delay from the time of the incident to arrival in the hospital.

Treating an intubated, critically ill patient in a hyperbaric chamber requires a sound knowledge of physic and physiological changes in the hyperbaric environment. On top of that, equipment, staffing and patient preparation are also important. Boyle's Law state that volume of a gas changes inversely proportional to pressure. When pressure increases, the volume of gas is reduced. Thus, prior to recompression, the air in the endotracheal tube cuff has to be replaced by saline. Otherwise, during compression, the cuff will collapse leading to a large leak. Likewise, venting of fluid bottles is required to eliminate the risk of a sudden increase in fluid delivered during compression (descend) and backflow of fluid during decompression (ascend) [3].

The blood pressure and heart rate of this patient initially increased significantly during the start of compression. There may be a few causes for this.

It was found that hyperoxia causes vasoconstriction and increase in systemic vascular resistance (SVR) leading to elevated systolic blood pressure [4]. However, increased SVR will lead to a drop in cardiac output, and in critically ill patients who may not be able to compensate adequately, this may lead to a drop-in blood pressure. Pain from sinus and middle ear barotrauma (Boyle's Law) during compression may also explain the elevated blood pressure and heart rate. Finally, some authors have found that pressure changes can affect the performance of certain syringe pump causing erratic delivery of vasoactive drugs thus influence changes in blood pressure and heart rate. As a general rule, all critically ill patients should have invasive monitoring in the chamber to allow close monitoring and timely intervention.

In this patient, the respiratory parameters were relatively stable and there were no significant problems with ventilatory support throughout the treatment. However, in some critically ill patients, especially those with pre-existing lung pathology, a hyperbaric condition may have potentially adverse effects on the respiratory system. At depth, gas density increases. This causes increased in airway resistance due to the increased likelihood of turbulent flow (higher Reynolds number). Consequently, the patient's work of breathing is also increased (in non-ventilated patients). In a ventilated patient, the changes in gas density, airway resistance, flow rate and ambient pressure can affect the performance of a mechanical ventilator [5]. It is thus imperative that the treating physician is familiar with the type of ventilator being used (time-cycled, pressure-cycled or volume-cycled ventilators), and how pressure changes affect these ventilators. The use of 100% oxygen during hyperbaric oxygen therapy will lead to washing out of nitrogen from the lungs, causing absorption atelectasis. PEEP is often used in mechanically ventilated patients to recruit and stabilize alveoli and to prevent atelectasis. However, studies have shown that hyperbaric condition can significantly alter the level of PEEP and may lead to cardiovascular compromise (reduction of preload from excessive PEEP) and risk of barotrauma (auto-PEEP and overextension of lung units). Thus, airway pressure must always be monitored closely in conjunction with repeated careful assessment of patient throughout the treatment while checking and readjusting the ventilator settings [6].

CONCLUSION

Our case illustrates a classical presentation of severe DCS in a diver diving beyond depth limitation and ascending too rapidly. Early recognition of DCS, followed by resuscitation and providing 100% oxygen, while arranging for urgent transport to a hyperbaric facility are essential for the best outcome. Furthermore, treating an intubated, critically ill case of DCS in a hyperbaric chamber is complicated because it requires extensive knowledge of the possible effects of the hyperbaric condition on equipment performance and ultimately on patient care. Subsequently, applying this knowledge to provide as close to intensive care unit level of care in the recompression chamber is vital for the best possible clinical outcome.

REFERENCES

1. United States Navy Diving Manual 2016. Air Decompression Table; Table 9-9.
2. Edmonds C, Bennett M, Lippmann J, Mitchell S 2016. First aid and emergency treatment. Diving and Subaquatic Medicine 5th edn, CRC Press, USA, p557-566.
3. Weaver LK. Hyperbaric oxygen in the critically ill. Crit Care Med. 2011;39:1784-91.
4. Mathieu D, Favory R, Collet F et al 2006. Physiologic effects of hyperbaric oxygen on hemodynamics and microcirculation. Handbook on Hyperbaric Medicine, Springer, Netherlands, p75-101.
5. Mathieu D, Ratzenhofer-Komenda B, Kot J. Hyperbaric oxygen therapy for intensive care patients: position statement by the European Committee for Hyperbaric Medicine. Diving Hyperb Med. 2015 Mar;45(1):42-6.
6. Youn BA, Houseknecht R, DeAntonio A. Review of positive end-expiratory pressure and its application in the hyperbaric environment. J Hyperbaric Med 1991; 6(2):87-99.

Correspondence: Colonel (Dr) Andrew Ng Wei Aun. Department of Anaesthesiology of 94 Armed Forces Hospital, Terendak Camp, Melaka, Malaysia. Tel: +60124108808 e-mail: dranwa@gmail.com