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CASE REPORT **OPEN ACCESS**

ALTERNATIVE MANAGEMENT OF DECOMPRESSION ILLNESS IN BREATH-HOLDING DIVERS USING NPPV

Priyanka Ganesa Utami[1*], Eka Musridharta[2], Mursyid Bustami[2], Ita Muharram Sari[2], Sardiana Salam[2]

***Correspondence:** priyanka.utami@gmail.com*
[1]National Brain Center Hospital Prof. Dr. Mahar Mardjono, Jakarta, Indonesia
[2]Division of Neuroemergency and Neurocritical Care, National Brain Center Hospital Prof. Dr. Mahar Mardjono, Jakarta, Indonesia

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ABSTRACT

Background: Decompression illness (DI) is a syndrome caused by the formation of gas bubbles during rapid decompression. DI can manifest with neurological manifestations. Divers are at risk of developing DI even in shallow water.
Case: A 25-year-old woman lost consciousness after a training dive. She lost consciousness shortly after surfacing, having previously dived to a depth of four meters. She vomited once and denied seizures. This was her third training dive; the second occurred two days prior, and the first ten days prior. The patient was in good health and had no history of heart or lung problems. A non-contrast head CT scan and MRI revealed ischemic lesions in the bilateral thalamus and cerebellum. Chest X-ray (CXR), transthoracic echocardiography (TTE), echocardiography, and bubble contrast scans showed no abnormalities. The patient was treated with 100% oxygen via non-invasive positive pressure ventilation (NPPV – CPAP mode) and showed improvement in her neurological deficits after 10 days.
Discussion: DI is associated with vascular or extravascular bubbles that form as a result of rapid decompression. Pure oxygen inhalation using HBOT is the main therapy for DI. But, in the absence of HBOT, NPPV can be considered for its effectiveness to achieve a mean arterial pO2 concentration above 500 mm Hg.
Conclusion: Recognizing the signs and symptoms of DI is important because they are nonspecific. Oxygen: 100% oxygen should be administered to those suspected of having DI.

Keywords: breath-holding diving, decompression illness, NPPV

Introduction

Over the past few decades, diving has become a popular recreational activity. Indonesia, an archipelagic nation renowned for its stunning waters, has become a popular destination for recreational diving among tourists. Although diving is relatively safe, serious injury or death can occur, with a mortality rate of 3–6 deaths per 100,000 divers. Decompression Illness (DI) is an injury affecting divers, with consequences ranging from mild to serious.[1] Therefore, the possibility of DI occurring in Indonesia is unavoidable. Unfortunately, hospitals with hyperbaric chambers are very few and unevenly distributed, even in areas renowned for their marine tourism.

Decompression Illness is a clinical syndrome caused by the formation of undissolved gas in the blood and tissues due to rapid decompression.[2] The diagnosis of DI relies almost entirely on the diving history and the presence of a wide variety of clinical signs following decompression. To date, no specific biochemical markers confirm post-decompression bubble-related injuries. Furthermore, the sensitivity of radiological and nuclear examinations is not superior to clinical observation. Therefore, the best way to diagnose DI is to exclude other possible aetiologies that contribute to post-dive decompression symptoms: unexplained pain, skin, cardiovascular, or neurological abnormalities, chronologically related to compressed gas diving, and to rule out other etiologies.[3]

Because DI can result in severe neurological consequences, neurologists must be aware of the signs and symptoms of this disease and how to manage these patients. Our case report demonstrates the benefits of non-invasive positive pressure ventilation (NPPV) as an alternative therapy for managing Decompression Illness in areas without hyperbaric chambers facilities or during transport to hospital with hyperbaric chambers facilities. Furthermore, NPPV is widely available in hospitals throughout Indonesia, making it beneficial in the management of Decompression Illness in areas where hyperbaric chambers are not access able or during transport to hospital with hyperbaric chambers facilities.

Case Report

A 25-year-old woman presented to the emergency room complaining of altered consciousness after a dive training session at a diving facility. The altered consciousness occurred shortly after surfacing and was preceded by non-projectile vomiting. She denied seizures, and her left limb appeared more active. The patient experienced symptoms after diving to a depth of 4 meters and then surfacing. This was her third dive; the second dive was two days prior, and the first dive was ten days prior. The patient experienced no symptoms after the first and second dives. She was in good health and had no history of heart or lung problems.

The patient was taken to a facility with a hyperbaric chamber for recompression treatment, but because the patient was unconscious, the patient was advised to undergo tympanoplasty to maintain pressure on the tympanic membrane and keep the eustachian tube open[4]. Furthermore, the patient must be monitored by qualified medical personnel during the recompression treatment in the hyperbaric chamber. Nevertheless, the family decided to move the patient to our hospital.

The examination in the emergency room revealed altered mental status with a GCS of E2M5V2, BP 115/70, HR 53 bpm, RR 22 breaths per minute, temperature of 36.7 °C, and SpO2 of 97%. We found that the patient had right hemiparesis. Blood gas analysis and chest X-ray (Figure 1) showed no abnormalities. Non-contrast head CT scan (Figure 2) showed bilateral infarction within the thalamus and cerebellum. Non-contrast head MRI (Figure 3) showed similar results: bilateral infarction within the thalamus and cerebellum, accompanied by minimal haemorrhagic transformation in the form of scattered small petechiae. We evaluated for heart valve disorders, a risk factor for DI, but echocardiography, transoesophageal echocardiography, and bubble contrast echocardiography all showed normal results, without any heart valve disorders.

PH	N	7.38
PCO2	N	39
PO2	H	197
HCO3	N	23
CO2	N	24
SpO2	H	98.7
BE	N	-1.3
FiO2	N	36%
PF Ratio	N	547.2

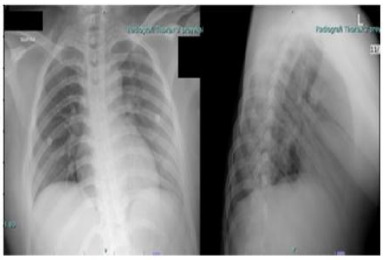

Figure 1. Blood Gas Analysis and Chest X-Ray showed no abnormality

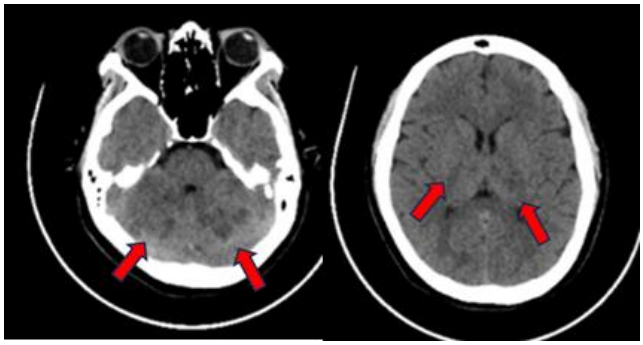

Figure 2. Non-contrast head CT scan showed relatively well-defined hypodense lesions in the bilateral thalamus and cerebellum (red arrow)

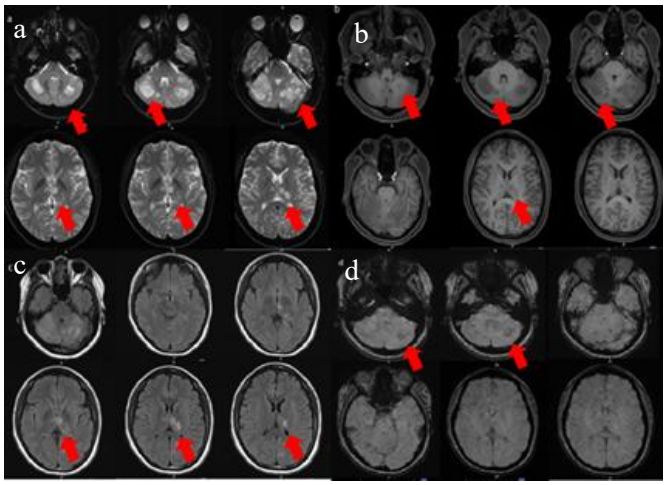

Figure 3. Non-contrast head MRI (a) DWI sequence, (b) T1 sequence, (c) FLAIR sequence showed infarction in the bilateral thalamus and cerebellum, and (d) SWI sequence shows micro bleeding in the form of scattered small petechiae in the bilateral thalamus and cerebellum region (red arrow)

We administered high-flow, high-pressure oxygen therapy using NPPV in continuous positive airway pressure (CPAP) mode, with an oxygen fraction of 50% and positive end-expiratory pressure (PEEP) of 10, with daily blood gas analysis for monitoring. We administered mannitol and dexamethasone to manage cerebral oedema; dopamine and norepinephrine with a target mean arterial pressure (MAP) of 90 mmHg to maintain cerebral blood flow; and maintained the patient's hydration status.

We observed significant clinical improvement in the patient's level of consciousness and motor function after

the second day of NPPV. We continued NPPV for seven days until the patient's level of consciousness and motor strength improved, then gradually tapered it over three days. The patient was then discharged from the neurocritical care unit (NCCU) to the neurologic ward. However, we still observed a sequela of motor aphasia in the patient.

Discussion

When a diver descends below the surface, the body experiences a tremendous increase in pressure. To keep the thorax from collapsing, air must be supplied under high pressure, exposing the blood in the lungs to extremely high alveolar gas pressures. This condition is known as hyperbarism.[5] DI occurs due to undissolved nitrogen gas forming inert gas bubbles (non-reactive gas bubbles)[6,7] in the circulation or tissues after diving.[3,8,9] DI also known as caisson disease (CD), DI, or decompression sickness (DCS).[3] Haldane (1907) classified DI into three categories: type I, joint pain; type II, systemic symptoms or signs, caused by involvement of the central nervous system (CNS) or cardiopulmonary system; and type III, characterized by seizures and death. However, only the first two categories that are accepted internationally.[8]

Nitrogen (N2), which accounts for 78% of the total gas in normal air, is the primary cause of DI. According to Henry's law, the solubility coefficient of each gas determines how much of that gas can dissolve. Furthermore, during diving, this inert gas also experiences an increase in partial pressure. The solubility coefficient of N2 in blood and fat is lower than that of oxygen and carbon dioxide (Table 1). Therefore, nitrogen is less soluble in blood, and even a small partial pressure can cause nitrogen to escape into the free phase.[3,5]

Table 1. Henry's constant for gas solubility in water shows that the nitrogen (N2) solubility coefficient is lower than those of oxygen and carbon dioxide[10]

Substance	Chemical formula	Henry's constant (k) in water at 25°C (mol/L atm)
Oxygen	O_2	1.3×10^{-3}
Carbon dioxide	CO_2	3.3×10^{-2}
Nitrogen	N_2	6.1×10^{-4}
Hydrogen	H_2	7.6×10^{-4}
Ammonia	NH_3	5.6×10^{-2}
Methane	CH_4	1.2×10^{-3}
Sulfur dioxide	SO_2	5.6×10^{-2}

During diving, the body is exposed to higher ambient pressure, increasing the partial pressure of N2. Different compartments absorb nitrogen during this high-pressure exposure.[3] This process is called in-gassing. During decompression, ambient pressure drops, causing nitrogen supersaturation in the body's tissues and blood because the respiratory system cannot eliminate nitrogen. This elimination process is called outgassing. N2 gas usually does not cause symptoms because it is filtered by the pulmonary capillaries, where it diffuses along the concentration gradient into the alveoli. If gas bubbles exceed the filtering capacity of the pulmonary capillaries, they circulate in the systemic circulation. Some dissolve in the blood, and some remain insoluble, forming inert gas bubbles.[3,11]

During decompression, microbubbles enlarge and coalesce into larger bubbles. Gas bubbles can form in both veins and arteries, but they are more common in veins. Arterial gas embolism (AGE) can form in the arteries themselves, originate from surrounding tissue, or enter the arterial system from the venous system through a shunt. Venous gas embolism (VGE) can originate from surrounding tissue via capillaries or form within the veins themselves. Veins have thinner, more porous walls, making them more permeable to gas than arteries. Furthermore, the venous system has lower resistance and a larger diameter than the arterial system, resulting in slower blood flow in the veins than in the arteries. This process further increases the likelihood of gas bubbles forming in the veins.[3,5,12,13] Therefore, gas bubbles are more common in veins than in arteries. Venous bubbles are commonly found after repeated dives, even at relatively shallow depths, as was the case in our patient.[3,5,12] Apart from causing AGE and VGE, these gas bubbles can also enter the tissue and cause tissue damage (Figure 4).[14]

Larger gas emboli can affect larger vessels. The larger the affected vessel, the greater the tissue damage from AGE. Embolic occlusion impairs tissue perfusion. In the central nervous system, gas emboli may produce effects similar to stroke, as seen in our patient. Failure of the electrolyte pumps results in cytotoxic edema. The influx of sodium, chloride, and calcium ions causes fluid to shift into the cells. Excess calcium in the cells also consumes adenosine triphosphate (ATP), which is already essentially depleted.[3,12] This process is characterized by plasma shifting into the intracellular space, edema around the blood vessels (perivascular edema), and paralysis of blood vessels due to vascular-activated substances. This process further reduces oxygen supply to already hypoxic tissues. Furthermore, N2 elimination becomes increasingly difficult due to obstructed blood flow, leading to greater accumulation of undissolved gas and progression of the embolic process.[3,13]

In the blood, gas bubbles can attract a layer of plasma proteins, forming a bubble “skin” composed of plasma glycoproteins, fibrinogen, and gamma globulin. The active surface of this bubble allows platelets and white blood cells to adhere. Over time, platelet activation leads to aggregation and adhesion around the

bubble, entrapping other blood constituents. Cellular blood elements, such as red blood cells, can become entangled in the growing fibrin network. This thickening of the bubble "skin" can reduce diffusion, providing a mechanism for bubble stabilization and survival. Platelet adhesiveness also generally increases in response to bubbles.[12]

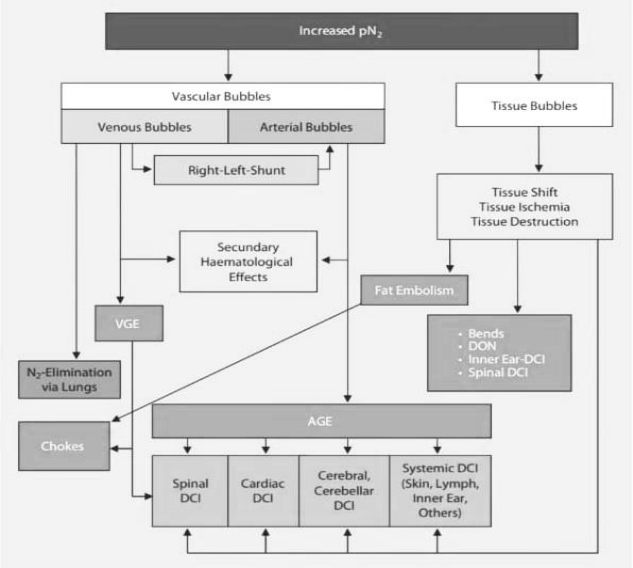

Figure 4. The effect of increasing the partial pressure of nitrogen and the formation of inert gas bubbles in the body[3]

Many factors increase the risk of DI in divers, including obesity, patent foramen ovale (PFO), rapid ascent to the surface, Valsalva maneuver while submerged, breath-hold diving, and the frequency and interval of breath-hold diving.[8,9,15–17] Risk factors for DI with neurological symptoms in breath-hold diving include diving to 20 meters, repeated dives every three hours or more, rapid ascent to the surface, and shorter surface time relative to depth.[15] Our patient was a breath-hold diver with shorter intervals between the second and third dives than between the first and second. Tests measuring N2 saturation and desaturation in a compressed-gas diving model indicate that DI can occur during repeated breath-hold diving with short surface intervals.[16,18] After repeated breath-hold diving, N2 bubbles may form in the small blood vessels of the tissues and flow into the right atrium, but they do not have time to be eliminated by the respiratory system until the next dive.[17]

The air pressure at sea level is 1 atmosphere absolute (ATA). For every 10 M below the water's surface, the pressure increases by 1 ATA. Therefore, if a diver dives to a depth of 10 M, the pressure will increase to 2 ATA.[5,9] At a depth of 10 M, the diver's total gas volume is assumed to be approximately 3.5 L (residual volume + expiratory reserve volume + tidal volume) with normal breathing. At the surface, the total lung capacity is estimated to be 6 L. If the diver holds his breath (breath-holding), the lung volume will increase by 7 L at the surface, 1 L more than the total lung capacity due to the excess air stored during breath-holding.[3,13] However, our patient only dived to a depth of 4 meters; it is likely that the surface pressure experienced by her during the dive increased by less than 0.5 ATA.

Although no one knows what happens to patients during diving, we cannot rule out the possibility that they perform a Valsalva maneuver at depth, either reflexively, during a cough, or by breath-holding. The mechanical effect of the Valsalva maneuver is an increase in intrathoracic and intra-abdominal pressure. Forceful breathing and exhalation against a closed airway increase intrathoracic and intra-abdominal pressure, thereby raising pressure in all blood vessels in the chest and abdomen, including the superior and inferior vena cavae, and in the cardiac chambers.[19] As little as 10% overexpansion of the lungs is sufficient to cause gas embolism. This occurs when the intratracheal pressure reaches 76-80 mmHg, or when the diver swims to the surface while holding his breath after inhaling compressed gas, even from a depth of only 1 meter.[3]

Echocardiography, transesophageal echocardiography, and bubble contrast echocardiography in our patient did not show a PFO or other valvular disorders. In addition, our patient had a normal body mass index (BMI), so the risk of DI due to PFO or obesity can be ruled out. We also obtained normal results on chest X-ray and blood gas analysis, and there were no decompression manifestations in other organs, indicating that the brain was the only organ affected by decompression in this patient. According to the DI classification by Haldane (1907), our patient is classified as DI type II, which is characterized by systemic symptoms or signs caused by involvement of the CNS or cardiopulmonary system.[8]

The initial resuscitation step in patient with decompression illness should be oxygen administration, ideally with an FiO2 of 100%. Pure oxygen increases tissue oxygenation and the partial pressure gradient, thereby reducing the size and number of bubbles by increasing inert gas elimination, preventing additional venous gas emboli, and improving tissue oxygenation.[3,12,20] Therefore, administering pure oxygen with non-invasive measures, such as CPAP, in patients who do not require intubation is reasonable to achieve the highest possible FiO2. [20,22] We administer high-flow, high-pressure oxygen therapy using NPPV in CPAP mode with an oxygen fraction of 50% and a PEEP of 10, accompanied by blood gas analysis monitoring. A study by Kohler et al. [22] compared eight devices for increasing arterial pO2 and found four devices to be most effective in achieving a mean arterial pO2 of approximately 540 mm Hg, including SuperNO2VA, NPPV, high-flow nasal cannula, and a diving regulator.

If non-invasive ventilatory support is used, providers should consider using minimal pressure settings (i.e., just enough to overcome device resistance) to reduce intrathoracic pressure, which can increase left-to-right shunts and further increase the risk of paradoxical embolism in patients with a patent foramen ovale. Furthermore, whether ventilation is performed through invasive or noninvasive means, reexpansion barotrauma can be a contributing factor to decompression illness, and therefore, practitioners should minimise peak pressures whenever possible. Additionally, clinicians should ensure adequate fluid resuscitation to prevent dehydration and achieve adequate circulation.[14,20]

Recompression in a hyperbaric chamber is still remains the definitive treatment for patients with Decompression Illness, including cerebral gas embolism, regardless of its severity according guidelines.[3,20,23] One goal of recompression is to reduce bubble volume, thereby alleviating symptoms caused by mechanical tissue disruption and reducing ischemia by redistributing intravascular bubbles. Oxygen recompression significantly increases the inert gas diffusion gradient from the bubbles to the blood. Hyperbaric oxygen therapy can also reduce leukocyte adhesion and microparticles. Furthermore, the increased dissolved oxygen in the blood improves tissue oxygenation.[21] US Navy guidelines recommend managing DI with initial oxygen recompression in a hyperbaric chamber at 2,8-ATA, and this has been the most commonly used initial recompression protocol since the 1970s. This therapy can be extended or repeated if the patient's response is not optimal due to severe decompression conditions.[14] Therefore, recompression therapy in a hyperbaric chamber should be performed immediately in patients with suspected DI.[22]

However, not all hospitals have hyperbaric chamber facilities. Although NPPV is not a primary treatment modality in DI management and is more often used as first aid during patient transfer, we observed significant improvement in our patient after the second day of NPPV initiation and continued to see improvement for up to seven days of oxygen administration using NPPV, although the patient still had sequelae such as motor aphasia. Given the benefits of NPPV in DI management and its availability in almost all hospitals in Indonesia compared to hyperbaric chambers, oxygen therapy using NPPV can be considered in patients with suspected DI if hyperbaric chamber facilities are not access able or during transport to hospital with hyperbaric chamber facilities.

Conclusion

Decompression illness is a clinical syndrome caused by the formation of undissolved gas in the blood and tissues due to a rapid reduction in environmental pressure. DI often manifests as neurological symptoms; therefore, a neurologist must be able to identify this disease. Given the limited number of hyperbaric chambers in Indonesia, despite its vast waters, and the widespread distribution of NPPV in almost all hospitals in Indonesia, along with the benefits it can provide to patients with DI, oxygen therapy using NPPV can be considered a treatment for DI during patient transfer to a hospital with a hyperbaric chamber, or when a hyperbaric chamber is not available or cannot be reached.

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Declaration of Interests

The author states that there is no conflict of interest to disclose regarding the preparation of this case report manuscript.

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