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Caisson Disease among Recreational Divers: Review Literature

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ABSTRACT

Introduction: Caisson disease is one of the most common emergencies in divers with very common signs and symptoms that clinicians must be careful to identify based on a complete history and physical examination. The disease also requires immediate and definitive management, usually hyperbaric therapy for a better prognosis.

Content: This article discussed the types, pathophysiology, and management of caisson disease caused by the release of nitrogen gas bubbles into the blood or tissues during or after a pressure drop in the environment. This is a disease that can be prevented by paying attention to existing risk factors.

Summary: Caisson disease or decompression sickness is a collection of symptoms that are often encountered in archipelagic countries and can affect anyone diving for seafood or recreational activities. The symptoms are common, but by knowing the pathophysiology, clinicians can identify the disease and immediately perform therapy, thereby preventing complications such as arterial a gas embolism (AGE) so the mortality and morbidity due to caisson disease can be reduced.

Keywords: Caisson disease; diving; partial pressure; nitrogen

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Introduction

Indonesia is an archipelago composed of approximately 17,508 large and small islands scattered around the equator with a tropical climate. Indonesia's marine area is estimated at 5.8 million km2 with a coastline of 81,000 km and is the country with the longest coastline in the world. Diving has become the activity of choice for local and foreign tourists to enjoy the beauty of the Indonesian sea. [1]

Tourist and those people who live in coastal areas or fishermen looking for marine creatures for their livelihoods by diving are at risk of causing decompression sickness. Decompression sickness is a syndrome associated with the formation and increase in the size of bubbles in tissues and/or blood when the partial pressure in gases in the blood and tissues exceeds the pressure outside the body (ambient).[2]

The incidence of decompression sickness for recreational diving is estimated at three cases per 10,000 dives, while for occupational diving slightly higher with a range from 1.5 to 10 cases per 10.000 dives. The incidence in Europe ranges from 10 to 100 cases per year, but in Indonesia, there was no data available. However, the number of fishermen in Indonesia who show symptoms of decompression sickness is 15.3%. Research in Kepulauan Seribu, DKI Jakarta documented that 6.91% of fishermen experienced decompression sickness, while research on Bungin Island, NTB, showed that 57.5% of divers showed symptoms of decompression sickness in the form of joint pain and mild hearing loss to deafness as much as 11.3%. [3]

Etiology and Risk Factors

A couple of factors are involved within the pathogenesis of the DCS. Divers with bronchial asthma, atrial septal illness, patent foramen ovale, or obesity are greater vulnerable to increase DCS. The intensity of the dive under the sea surface, the temperature of the water, and the rate of ascent are considered the main contributory factors to thedevelopment of DCS. when divers ascend at a velocity of nine–10 meters/min, they have minimum threat of growing DCS. If the ascent is faster (>19 meters/min), the chance of DCS is considerably higher.18 Caisson ailment takes place whilst a speedy discount in stress (e.g., throughout ascent from a dive, go out from a hyperbaric chamber, or ascent to altitude) causes gases previously dissolved in blood or tissue to form bubbles in blood vessels, it occurs in approximately 2 to 4/10,000 dives among leisure divers. The incidence is better amongst business divers exposed to greater depths and longer dive times with the subsequent risk factors: bloodless temperature diving, acting flight after diving, and long and or deep dives.[4,5]

Pathophysiology

Bubble formation is considered the number one mechanism of harm in decompression sickness. Divers take in inert gas (nitrogen whilst respiratory air) into tissues while respiratory compressed gasoline during a

dive, with greater fuel absorbed on deeper or longer dives. all through the ascent to sea degree, the partial pressure of dissolved gases in tissues can exceed ambient stress, leading to the formation of bubbles in the ones tissues or blood passing through them. this can additionally arise at some point of rapid ascent from sea degree to high altitude. The resulting venous gasoline emboli are small (19 to 700 μm) however very commonplace after diving or exposure to fast altitude. these emboli are generally filtered via the pulmonary capillaries and are asymptomatic. however, venous gasoline emboli can attain the arterial stream via overwhelming the filtering ability of the pulmonary capillary network or thru intrapulmonary or intracardiac proper-to-left shunts, which include atrial septal defects and patent foramen ovale (PFO).[6]

The presence of a PFO increases the chance of decompression sickness of the cerebrum, spinal wire, internal ear, and pores and skin, possibly due to small, arterialized venous gas emboli arriving in saturated tissue capillaries after diving springing up thru inward diffusion of a gas. The vulnerability of the brain, that is rather perfused (thus unexpectedly dissipating inert gasoline) and unlikely to saturate after diving, may additionally get up from publicity to high numbers of small arterialized venous fuel emboli, probably coalescing to shape larger bubbles, as has been suggested after a strongly wonderful bubble comparison take a look at for PFO.[6]

Bubble formation in the tissue might also motive mechanical disruption and focal hemorrhage, in particular inside the white remember of the mind. Even small intravascular bubbles may additionally have bodily outcomes, with inflammatory and thrombogenic host responses. Small doses of arterial gas may also provoke a progressive lower in cerebral blood waft, an impact abolished by means of neutrophil depletion. Intravascular bubbles can detach endothelial cells from the underlying basement membrane, ensuing in impaired regulation of vascular tone, plasma leakage, and hypovolemia. thru this mechanism, a excessive venous fuel embolism can injure pulmonary capillaries and induce pulmonary edema. Even without gross mechanical damage, bubble touch with the endothelium can provoke brief receptor capacity vanilloid ion channel commencing, calcium inflow, mitochondrial disorder, and cell death. adjustments inside the coagulation machine after decompression consist of a moderate lower in circulating platelets due to activation and extended consumption, in addition to an increase in circulating fibrin monomers.[6,7]

Manifestations of decompression illness in mice were decreased by way of pre-treatment with glycoprotein IIb/IIIa receptor antagonists. Male mice proof against decompression sickness have improved prothrombin time and reduced circulating issue X degrees. supplement activation has also been said. Circulating microparticle ranges increase after diving. The motive of this increase is uncertain, but micro debris may additionally play a pro-inflammatory function in decompression sickness.[7]

Fisiologis and Clinical Menifestation

Nitrogen is plenty greater soluble in fatty tissue than in different sorts; consequently, tissues with a

excessive-fat content (lipids) usually have a tendency to absorb more nitrogen than do special tissues. The frightened machine consists of about 60 percentage lipids. Bubbles forming within the brain, spinal cord, or peripheral nerves can motive paralysis and convulsions (divers' palsy), problems with muscle coordination and sensory abnormalities (divers' staggers), numbness, nausea, speech defects, and man or woman modifications. whilst bubbles collect within the joints, ache is typically excessive and mobility is limited. The term bends is derived from this discomfort, due to the fact the affected person generally is not capable of straighten joints. Small nitrogen bubbles trapped below neath the pores and skin may additionally reason a purple rash and an itching sensation known as divers' itches. Generally, those signs and symptoms via bypass in 10 to 20 minutes. excessive coughing and trouble respiratry, referred to as the chokes, imply nitrogen bubbles within the respiration gadget. different signs and symptoms embody chest ache, a burning sensation even as respiratory, and severe shock.[19]

Diagnosis

The diagnosis of decompression sickness depends on the patient's history and physical examination, with no reliance on specific supporting examinations. This is because magnetic resonance imaging (MRI) and computed tomography (CT) scans have low sensitivity in assisting the diagnosis of decompression sickness. The exception for thoracic plain photographs is considering would have taken a long time to perform the examination. A plain thoracic photograph is done to see if the patient has a pneumothorax which is a contraindication to pure O2 administration. Suspicion of decompression sickness can be inferred if the patient presents with symptoms of decompression sickness and a history of diving within the previous 24 hours. A thorough neurological examination should be performed in patients with suspicion of decompression sickness to look for other disorders that are not complained of during history taking.[8]

Based on severity, the symptoms of decompression sickness are divided into two groups, namely type 1 and type 2 decompression sickness symptoms. Decompression sickness symptoms consist of joint pain, muscle pain, muscle weakness, fatigue and, skin symptoms. Symptoms of type 2 decompression sickness involve abnormalities of the central nervous system (stroke), respiratory system, and cardiovascular system.[9] The most common symptoms are joint pain in 58% of cases, muscle pain in 35%, and low back pain in 7%. The anatomical locations of joint pain in order from most frequent to least frequent were shoulder, elbow, knee and ankle. The second most common symptom of decompression sickness was paresthesia as in 63.4% of cases. Other accompanying symptoms can include headache, fatigue, malaise, hives, purpura marmorata, chest pain, dyspnea, nausea and vomiting, anorexia, cramps, and spasms. [10] Decompression sickness can also cause thrombocytopenia due to the attachment of platelets to nitrogen gas bubbles. Possible neurological deficits can also occur in decompression sicknesses such as cognitive abnormalities, cranial nerve lesions, and spinal cord dysfunction due to the destruction of white matter due to the formation of micro thrombus in

the spinal circulation. Other manifestations may include hearing loss such as tinnitus and vertigo. The worst possibility of decompression sickness is paralysis or even death. [11]

Management

The selection of suitable remedy is based on by means of the severity of the medical findings and instances. important situations of treatment pathways are shown in the determine beneath. should we use severe manifestation in bracket (type II) and moderate manifestation (type I) characteristics.

Recompression in a hyperbaric chamber is the definitive remedy for decompression sickness. one of the dreams of recompression is to reduce the bubble extent, thereby lowering signs and symptoms as a result of mechanical disruption of tissues and disposing of ischemia. the usage of recompression has been acknowledged to be effective, in comparison to no recompression, since the overdue 1800s, specially whilst applied early after harm. but, if the partial pressure of nitrogen within the frame is better, it calls for an extended and greater gradual decompression.

Evaluation

All casualties requiring recompression therapy in a hyperbaric chamber should be referred to the hospital for evaluation and monitoring, even if the casualty appears fully conscious. Referral to the hospital should be done as soon as possible unless the patient is confirmed dead.

Prognosis and Complications

A commonplace hassle is arterial gas embolism (AGE). signs and symptoms of AGE develop unexpectedly and are regularly complained of heavily. In a examine conducted by using Leitch & inexperienced (1989), AGE signs and symptoms took place all through ascent to the surface, at the surface, or within five minutes of arriving on the surface in 107 of 116 dive instances (92%). Ascent to the floor is regularly speedy, with breath-keeping resulting from panic after an underwater coincidence. despite the fact that, AGE can arise throughout regular ascent. medical manifestations commonly imply brain involvement, with arterial fuel frequently disbursed in a couple of cerebrovascular territories, with multifocal manifestations. Inside the examine mentioned above, the most not unusual preliminary manifestation become a lack of recognition (in 39% of instances), accompanied by way of confusion (37%), dizziness and presyncope (30%), hemiplegia (27%), visible modifications (21%), headache (20%), dysphasia (eleven%), and seizures (eleven%). In about half of the cases, there are obvious signs of underlying pulmonary barotrauma, which include chest ache and hemoptysis. Symptomatic restoration happens in about 1/2 of the cases, however there may be recurrence in some of those cases.

Having a records of decompression sickness will increase the hazard of a comparable occasion inside the destiny. prognosis depends at the severity and additionally depends on factors which includes speed of

first treatment, get entry to a referral clinic and definitive remedy.[11,16]

Conclusion

Decompression sickness is a syndrome caused by the formation of air bubbles in tissues or blood vessels as a result of dissolved noble gases (generally nitrogen) not being completely expelled through the lungs. The main risk factors for decompression sickness in divers are dive depth, duration, frequency and rate of ascent to the surface. Decompression sickness is divided into two based on the symptoms present in divers, mild type or type 1 with symptoms of joint and muscle pain and skin abnormalities such as itching and blueness and severe type that reaches the central nervous system or type 2. The most common signs and symptoms of decompression sickness are joint pain, muscle pain, and tingling/paresthesia in the extremities. However, complications such as paralysis and even death can also occur. Management of decompression sickness begins with the administration of 100% oxygen at atmospheric pressure and if symptoms persist then definitive therapy should be given in the form of recompression with a hyperbaric chamber. That decompression sickness for drivers is preventable by educating them about safety stop and not rising to the surface quickly also avoiding the risk factors.

Conflict of Interest

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