RETINAL ARTERY OCCLUSION IN A DIVER

A A L Hsu, T M Wong, J How, J Tan, K T Tan

ABSTRACT

The clinical manifestation of decompression disorders is highly variable, ranging from mild rashes or joint pains to central nervous system symptoms like scotomata, paralysis and death. The diagnosis is easily overlooked, especially if an occupational history is not obtained. Recompression treatment with hyperbaric oxygen is the specific treatment for decompression sickness and air embolism. Prompt recognition and treatment are vital to recovery. However, there is a place for treatment of decompression disorders and embolism even when significant delay of up to 14 days has occurred. This case report discusses decompression disorders in relation to an unskilled fisherman diver who presented with retinal artery occlusion. Decompression disorder leading to retinal artery occlusion is a very rare presentation. The difficulty of diagnosis is discussed as well as the result of delayed hyperbaric treatment.

Keywords: decompression disorder, recompression, hyperbaric oxygen, embolism, fisherman diver.

SINGAPORE MED J 1992; Vol 33: 299-301

INTRODUCTION

Decompression disorders may occur as a result of various mechanisms and they manifest in a large range of signs and symptoms. Retinal Artery Occlusion is but one of the presentations of gas embolism in the circulation as a consequence of decompression disorders. In Rivera's[1] study of 935 cases of decompression sickness(DCS), he found that visual disturbances were seen in 6.8% of all cases of DCS. He also noted that in 1.4% of DCS cases studied, visual disturbances presented as an initial symptom. Delay in receiving treatment resulted in poor recovery from DCS.

Retinal and visual disorders are also mentioned as one of the main symptoms in a paper by Davis on Altitude Decompression Sickness. In their study of 145 cases, 34% developed neurological symptoms as a result of DCS. Symptoms like blurred vision, diplopia, nystagmus, scintillating scotomata, visual field defects or blindness were the common visual presentations seen.They reported that those cases with neurologic manifestations also accounted for all the residual deficits following treatment. However, aviators rarely get hit by spinal cord DCS, unlike divers. Conversely, divers do get neurological symptoms but more commonly suffer from spinal cord-related symptoms like paresis and paralysis, sensory loss, proprioception loss, and loss of bowel and bladder function.

In other cases of diving-related neurological signs and symptoms, air embolism from barotrauma or rapid decompression is the commonest causative agent. In a study by Brooks et al[2] on pulmonary barotrauma in submarine-escape trainees and the treatment of the cerebral arterial air embolism, he reported visual abnormalities for 11 out of 101 cases. In the study of cerebral artery gas embolism, Gorman[3] reported that a similar proportion(11.2%) of cases developed visual symptoms.However, none of the last 2 studies made any mention of the exact visual deficit noted.

Thus this patient of ours did not suffer from spinal cord-related symptoms but just the scotoma. In spite of the substantial delay of 7 days, he was, nonetheless, treated with hyperbaric oxygen and there was a significant improvement of the area of scotoma.

CASE REPORT

A 41-year-old man was admitted to hospital with painless left visual field blurring. Ophthalmic examination revealed normal visual acuity in the right eye but a visual acuity of counting fingers at 2 feet in the left eye. There was left central scotoma with inferonasal extension. The left supero-temporal retina was pale with a few small haemorrhages and the macula was oedamatous. The rest of the physical examination was normal. The diagnosis of occlusion of the left supero-temporal branch of the retina artery was made. He was treated with oculomassage, intravenous acetozolamide (Diamox) and bed rest in the hospital.

On the next day, further information was obtained from the patient. He had been a fisherman/scuba diver for the past 26 years.He had a history of spinal decompression sickness 9 years ago. He was treated with recompression and rehabilitation and recovered completely one year later. The patient made a recent trip to the South China Sea (about 5 days' boat trip from Singapore). He did a total of 6 dives over a period of 3 days. His last dive was 6 days prior to his admission. He made 2 dives,with no decompression stops, on that day. The first lasted 30 minutes at a depth of 20 metres (60 feet) and the second was 4 hours later lasting 120 minutes at the same depth.He developed pain over the left maxillary region during the descent of his second dive. The pain recurred during the ascent and it lasted 10 minutes. This was associated with vague left visual disturbance. As the disturbance appeared to be trivial, he ignored it. He only realized a visual field defect at home 6 days later.He did not volunteer this information till on direct questioning as he was unaware of the possible relationship between diving and his visual problem. He had no past medical illness such as hypertension, diabetes mellitus and

Department of Medicine III
Singapore General Hospital
Outram Road
Singapore 0316

A A L Hsu, M Med(S'pore), MRCP(UK)
Resident

K T Tan, M Med(S'pore), MRCP(UK)
Senior Registrar

Department of Ophthalmology
Singapore General Hospital

J Tan, FRCS
Registrar

Naval Medicine and Research Centre

T M Wong, Dip DHM
Major (Dr)

SMO RSN Office
Republic of Singapore Navy

J How, Dip DHM
Colonel (Dr)

Correspondence to: Dr A A L Hsu

cardiovascular disease. Investigations including blood counts,coagulation and lipid profile, erythrocyte, sedimentation rate, glucose tolerance test, electrocardiography and chest X-rays were normal.

A diagnosis of embolism of a branch of the left retinal artery was suspected and the patient was referred to the Naval Medicine and Research Centre for treatment with hyperbaric oxygen. With recompression therapy, visual acuity improved from counting fingers at 2 feet prior to treatment, to 6 feet following treatment.Unfortunately, when he was reviewed 18 months later there was no further improvement. There was macular scarring and left inferonasal field defect with central scotoma.

DISCUSSION

Central retinal artery occlusion

There are many causes of central retinal artery occlusion. The commonest cause arises from the insufficiency of the circulation to the retina as a result of atherosclerosis,diabetes, and embolic phenomenon due to hypercoagulability of the blood. Rarely, the condition has been related to connective tissue diseases like systemic lupus erythematosus and the various forms of arteritis. However, in some cases, no specific cause is found[4].

Occlusion of the central retinal artery clinically presents as a painless, sudden, often complete loss of vision and typical ophthalmologic changes within a few hours or even minutes of the event[5]. However, in some cases there may have been prodromal symptoms of visual disturbances for a variable period before the actual onset of the occlusion. The presentation of branch retinal artery occlusion may be subtle in an unwary person as the visual loss is sectoral, the supero-temporal branch being most frequently involved (as in this case).The diagnosis of obstruction of a branch artery is sometimes confirmed by the discovery of a pale node which may be an actual embolus or an opaque patch in the wall of the affected vessel in addition to the presence of segmental ischaemic fundal changes.

The most likely causal mechanism in our patient is due to sinus barotrauma or DCS.

Sinus Barotrauma

The main complaint by the diver was that of pain of the sinuses during both the descent and the ascent phase prior to the onset of the visual field defect. This condition is known as sinus barotrauma and it is one of the commonest decompression disorders seen by the Diving Medicine specialist. The other areas that are affected include the middle ear and rarely, the lungs (pulmonary barotrauma). Sinus barotrauma causing arterial embolism of the retinal vessel is extremely uncommon. However, it must be considered upon exclusion of associated medical diseases and in the presence of barotrauma.

Sinus barotrauma can occur during descent: this is then termed as sinus barotrauma of descent. It may also occur during ascent. The injury in sinus barotrauma of descent is due to the inability of the sinuses to equalise pressures with the ambient pressures during the dive. Obstruction of the ostia is the main reason and is due to mucosal odema from rhinitis or URTI. The increased difference of pressures between the sinus and the nasal/oral cavity leads to progressive mucosal congestion and oedema. Rupture of submucosal capillaries may result, leading to bleeding into the sinus cavity. The bleeding into the cavity seen in sinus barotrauma may be seen radiologically as a fluid level in the sinus. Mucosal thickening may also be seen in sinus barotrauma. This is a result of the tearing away of the mucosa from the sinus wall associated with oedema and bleeding. The blood in the sinus also effectively reduces the volume of the sinus and assists in the equalisation of the pressure in the sinus with ambient pressure. During the ascent, the gas in the sinus re-expands to the original volume, and may displace the blood in the cavity, leading to divers complaining of blood in their masks, or blood-stained discharge when coughing or blowing their noses.

Sinus barotrauma of ascent may occur during this time if the ostia remain obstructed during ascent. The expanding gas increases the pressure in the sinus cavities. When the pressure of the gases in the sinuses exceeds the pressure in the blood vessels, embolism may occur. The patient had complaints of pain in the sinuses during descent. The likely problem at this time is sinus barotrauma of descent. As the diver ignored his symptoms and carried on diving, the sinuses could have equalised eventually with the ambient pressure, after a period of trauma. It may have, as described above, bled into the sinus cavity. During the ascent, the sinus may have remained blocked. The re-expansion of gases in the sinus caused increased pressure in the sinus. This resulted in extravasation of the gas in the sinus into the left ophthalmic artery, which lies in close proximity to the maxillary sinus[6]. This is the most likely cause of retinal artery occlusion in our patient.

The visual defect may diminish or even pass off completely (amaurosis fugax) - the shorter the duration of the condition, the more likely is the recovery to be complete.However, in most cases, there is little recovery of visual defect unless the circulation is restored promptly. The degree of reversibility appears to correlate with presenting visual acuity and duration of visual impairment. If the condition lasts some days, recovery is usually incomplete due to irreversible ischaemia, but even then there are cases which do recover after several weeks have passed[7]. In our patient, there was a 6-day delay before medical consultation and another one more day delay before the history of diving was elicited and recompression commenced.

Decompression Sickness

Decompression sickness occurs in divers who ascend to the surface too rapidly after spending time at great depths. Nitrogen is the inert gas responsible for decompression sickness. This gas is absorbed by various organs in the body according to the partition coefficient of nitrogen between the lungs and the blood. It is also affected by the partial pressures of the inspired gas within the blood (Henry's Law). As the uptake of gas during the diving phase is proportional to the duration and depth of the dive, there is usually a large amount of gas in the body at the end of a long and deep dive. In order to ascend safely, one must proceed at a rate where the lungs are able to excrete the excess nitrogen. If the diver ascends too rapidly, then supersaturation of the nitrogen in the body occurs, and bubbles of nitrogen will be formed. Our patient dived for 30 minutes at a depth of 20 metres, followed by a repetitive dive 4 hours later to the same depth for 120 minutes. According to the Royal Navy Diving Tables, this dive is considered a repetitive dive and the times for decompression should be calculated as such. In this case, the diver should have done decompression stops at 9 metres for 5 mins, 6 metres for 30 mins and 3 metres for 45 mins. As the diver was not aware of these requirements, the rapid decompression may result in supersaturation of the blood and tissue fluids with nitrogen. This in turn leads to precipitation of nitrogen bubbles in his blood. These divers do not appreciate the dangers of uncontrolled diving and as such frequently become affected by decompression sickness. Decompression sickness may have manifested as retinal artery occlusion in this patient, although no other neurological signs were detected. Usually, severe decompression sickness manifesting as retinal artery occlusion will also be associated with other severe central nervous system symptoms, reflecting the fairly generalised embolic phenomenon seen in decompression sickness.

Decompression sickness as a cause of blindness and central scotomata has been reported in various diving manuals and books on Diving Medicine. However, less information was available on the effects of central retinal artery occlusion by air embolism. Typical reports based on the hospital setting describe air embolism presenting after gynaecological procedures, open heart surgery, craniotomies and pneumothorax. Most reports present symptoms of unconsciousness, drowsiness and neurological deficits. In some cases apparent recovery occurred without intervention. Fatalities have also been described.

The final common pathway in severe decompression-related air embolism is the resulting end-organ hypoxia and necrosis.The definitive treatment for all gas embolism is recompression therapy with hyperbaric oxygen breathing. This applies for hospital iatrogenic air embolism as well.

This case report illustrates the typical delay in arriving at the diagnosis of decompression-related disorder presenting as retinal artery occlusion.

Treatment

Treatment is directed towards increasing retinal blood flow and to dilating its arterioles thereby increasing the blood supply to the retina, dislodging emboli and overcoming retinal spasm that may be present. The usual treatment-digital massage, paracentesis, recumbency, carbon dioxide and oxygen (carbogen) inhalation, anticoagulants, retrobulbar-injected and systemically-administered drugs - is with a few exceptions, ineffective[8] unless done promptly.

The definitive treatment of central retinal artery occlusion caused by air embolism or decompression sickness is hyperbaric oxygenation. The principles behind hyperbaric oxygenation are:

a. *Compression of the bubble size by increased pressures*
The bubbles obey Boyle's Law which states: When the temperature is kept at a constant,the volume of a gas varies inversely with the applied pressure. Following the production of bubbles during decompression, these bubbles coalesce and grow in the venous circulation. The lung acts as a gas exchanger for these bubbles as they traverse the pulmonary circulation. Spontaneous resolution of venous bubbles can occur if the rate of gas exchange is not exceeded. If there is a large amount of bubbles, then arterial embolism of these bubbles occur and they lodge in arterioles of various organs. Large numbers of bubbles coalesce to form larger bubbles which can even obstruct large blood vessels, with dire consequences.With recompression, when the bubbles are subjected to 2 atmospheres of pressure, the volume of the bubbles will half. This allows dislodgement of the bubbles from the larger blood vessels to smaller blood vessels, reducing the area of hypoxia. Some bubbles may remain in the very small arterioles even following recompression, and this may account for resistance to therapy.

b. *Inspiration of a high partial pressure of oxygen*
An increase in inspired oxygen tension reduces the partial pressure of inspired nitrogen. This increases the partial pressure gradient between the nitrogen in the blood and the nitrogen within the alveolar gas. As a result of the gradient, there is a faster rate of removal of nitrogen from the bubbles in the blood. The large partial pressure of oxygen also increases the dissolved oxygen content in the blood. This increases the dissolved oxygen content of the blood up to 6 mls per 100 ml of blood at 3 atmospheres of pure oxygen breathing. The dissolved oxygen can reverse the hypoxia in tissues obstructed by nitrogen bubbles by diffusion of oxygen from adjacent, well-perfused tissue. This increases the survivability of hypoxic tissues from ischaemic insult, while in the meantime, the obstructing bubbles are being eliminated.

The success rate of such treatment, naturally, is dependent on the speed of diagnosis and duration between moment of incidence and onset of therapy. In Meyer's Study, 5 out of the 6 patients they treated within 8 hours showed improvement of at least 5 Snellen lines. The 6th patient's vision improved from no light perception to counting fingers at 3 metres. Poor results however were seen in those patients treated after 8 hours[9].

Delayed presentation - the role of recompression

In delayed recognition, and in this case, delayed presentation to the admitting unit, a poor prognosis is expected. This is because the obstruction is unrelieved and the hypoxic damage is severe. The fact that there is some reduction of the area of visual loss after the 4 courses of hyperbaric oxygenation demonstrates that there are always areas of marginal perfusion which benefit from hyperbaric oxygenation. This is because angiogenesis and fibroplastic activity are enhanced by hyperbaric oxygenation, especially in hypoxic tissues. Therefore, it is the recommendation of the authors that a trial of hyperbaric oxygenation be carried out for the patient to salvage those areas of surviving tissue with marginal blood supply. There is a significant benefit of recompression therapy with hyperbaric oxygen in reducing the morbidity in cases of decompression sickness[10,11]. In How's study on the rehabilitation of paralysed divers, cases which presented with significant delay are still treated with recompression therapy, followed by aggressive rehabilitation. This resulted in a complete recovery of 43.2% of divers with DCS, and another 37.3% of divers with almost complete recovery[12].

Early diagnosis is critical as the onset of early treatment improves the prognosis tremendously. The morbidity of delayed recognition is significant as the patient's vision cannot be restored once permanent hypoxic damage has occurred. The importance of obtaining an occupational history cannot be overstressed, especially in dealing with relatively healthy young individuals who have no history of hypertension, diabetes and atherosclerosis.

CONCLUSION

An awareness of the varied manifestations of decompression disorder should alert the doctor to the diagnosis. We believe that decompression barotrauma should be added as a differential diagnosis of retinal artery occlusion where the history is suggestive. The occupational history must be taken for all cases of neurologic disturbances, especially when it occurs in a previously healthy individual. Early diagnosis and immediate recompression can lead to better outcome of treatment.

REFERENCES

1. Rivera JC: Decompression Sickness among Diversan analysis of 935 cases. EDU Research Report 1-63. US Navy,Washington, 1963.
2. Brooks GJ, Green RD, Leitch DR: Pulmonary Barotrauma in Submarine Escape Trainees and the Treatment of Cerebral Arterial Air Embolism.Aviation, Space and Environmental Medicine Dec 1986:1201-7.
3. Gorman DF: Arterial gas embolism as a consequence of Pulmonary Barotrauma. In: Desola J.ed. Diving and Hyperbaric Medicine. Proceedings of the IX Congress of the European Undersea Biomedical Society. Barcelona: EUBS, 1978:347-68.
4. Richards EA, Shirley HW, David GC: Central Retinal Artery Occlusion. Am J Ophthalmol 1975;79(3):374-81
5. Isaac CM: Textbook of the Fundus of the Eye. 3rd Ed. Churchill Livingstone 1980.
6. Otolaryngology and Sport Scuba Diving. Ann Otol Rhinol Laryngol 1985 (Suppl) ;115:1-12.
7. James JA, Larry EM: Visual prognosis following treatment of acute central retinal artery occlusion. Br J Ophthalmol 1980,640:913-7.
8. Brian RY, Thomson JR: Treatment of Acute Central Retinal Artery Occlusion. Mayo Clin Proc 1978;53:408-10.
9. Meyers RA, Bray P: Delayed treatment of serious Decompression Sickness. Ann Emerg Med 1985;14(3):254-7.
10. Yap CL: Decompression Sickness among Singapore Fishermen Divers. M Sc thesis. National University of Singapore 1982.
11. Kenneth WK: Delayed treatment of Dysbarism. JAMA 1982;247(18):2555-8.
12. How J: The rehabilitation of the paralysed diver. In: John L ed. Proceedings of the 13th Undersea Medical Society Workshop. Parmentier,Phd. 1984.