ALTITUDE DECOMPRESSION SICKNESS

M K Lim
J How
C M Peng
R Rajan

Aeromedical Centre
Headquarters Republic of Singapore Air Force
Paya Lebar Airport
Singapore 1953

M K Lim, MBBS, D Av Med, MSc (OM)
Senior Medical Officer RSAF
and Head

C M Peng, MBBS, D Av Med
Aviation Medical Officer

Naval Diving Medical Centre
Republic of Singapore Navy
Singapore 2775

J How, MBBS, Dip DHM
Senior Medical Officer RSN

R Rajan, MBBS
Medical Officer

SYNOPSIS

Altitude decompression sickness and diving decompression sickness share a common aetiological basis and have similar clinical manifestations. But there are distinct differences as well. This paper reports two cases of altitude decompression sickness occurring in the Republic of Singapore Air Force and highlights some interesting aspects of the condition.

INTRODUCTION

Aviators and deep sea divers operate in essentially unnatural environments in which alterations in ambient pressures can give rise to decompression sickness. While the effects of diving exposure have been recognised and studied for over 100 years, decompression sickness occurring at altitude did not gain prominence until World War II, when low pressure altitude chambers came into common use, and when aircraft became capable of attaining high altitudes.

Between 1939 and 1943, numerous reports, including those by Matthews in England (1), Hornberger and Benzinger in Germany (2) and Rosenblum in the Soviet Union (3) drew attention to the disorder. Despite the similarities in clinical manifestation between "caisson disease" and "aviators' bends", the relationship between the two conditions was not established. Even as early as 1945, Downey (4) and Behnke (5) had advocated using recompression therapy to treat altitude decompression sickness. But it was not until the mid-1960s that hyperbaric therapy, which has been routinely administered for diving decompression sickness cases since the 1930s, became fully accepted as the treatment for altitude decompression sickness (6).

The following two cases of altitude decompression sickness are the first to be reported in the Republic of Singapore Air Force. One occurred in a fighter pilot during a routine training flight while the other was a pilot trainee undergoing altitude physiology training in a decompression chamber. Both were successfully treated with hyperbaric therapy.

Case No. 1

Lieutenant MSH, a 23-year old Republic of Singapore Air Force pilot, flew in a Hawker Hunter single-seat aircraft at 30,000 feet for about 20 mins. He then descended to 22,000 feet, and was about to commence aerobatic manoeuvres when he suddenly felt as though hit on the head with a hammer. He experienced a severe sharp pain at the back of the head and "saw stars".

He was momentarily disorientated. The aircraft dropped a few hundred feet before he was able to regain control. He radioed to air traffic control, but experienced difficulty in speaking. His speech became slurred. He managed to land safely, and was brought by ambulance to the medical centre.

Clinical examination revealed an anxious looking but otherwise healthy young pilot, covered with perspiration. The pulse was 100 beats per minute, regular, and blood pressure was 100/60 mm Hg (lying) and 95/60 mm Hg (sitting). He was fully conscious and rational, and gave a coherent history. He remembered the warning light for cockpit pressurisation failure had flickered at 30,000 feet, but he had ignored it, thinking the light was malfunctioning. His speech had now returned to normal, and the pain at the back of the head and the scintillating spots in the visual fields had disappeared. There was diminished sensation to pin prick over a 6 cm x 1 cm patch of scalp in the right occipital region.

Decompression sickness with neurological and skin manifestations was considered the most likely diagnosis. The Naval Diving Unit was alerted while the patient was kept under close observation in the sick-bay, and given 100% oxygen via a face mask.

Half an hour later, the patient complained of dull pain in the right knee joint and right hip joint, but there was no more hypoaesthesia over the scalp. In view of the onset of joint symptoms, the patient was transferred by ambulance to the Naval Diving Unit for recompression therapy.

On arrival at the Naval Diving Unit 45 minutes later, he complained of numbness again over the right occipital region and this time, over the tips of his right fingers as well. Pain over the right hip and right knee had worsened. Clinical examination before therapeutic recompression revealed hypoaesthesia over the right occipital region and tips of fingers of the right hand. The affected joints appeared normal but there was pain on extreme flexion and extension.

He was treated according to RN Table 62. One pint of Rheomacrodex was administered intravenously. After 20 minutes of recompression to 2.8 atmospheres (60 feet depth), all symptoms were alleviated.

Clinical examination after recompression therapy was essentially normal. He was further given 6 hours of surface oxygen therapy intermittently (1 hour oxygen: 15 minutes air). Blood investigations including FBC, PCV, and ESR were all normal. CXR and EEG were also normal.

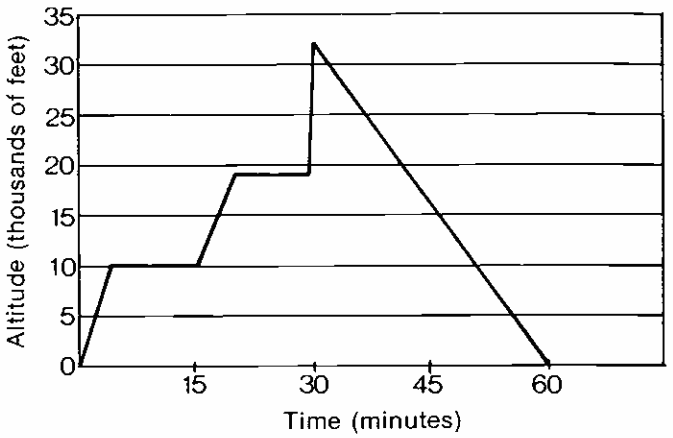
The patient recovered fully and was discharged the next day. Follow-up review indicated total recovery.

Case No. 2

SSW, a 20-year old student pilot, underwent altitude physiology training in the Mark III Altitude Chamber, along with other trainees.

The chamber flight profile consisted of: ascent to 10,000 ft at the rate of 4,000 ft/min; pause at 10,000 ft for mask seal check; resumption of ascent to 18,000 ft and rapid decompression to 33,000 ft in 3 secs. (See Fig. 1) All subjects were breathing a mixture of air and oxygen via pressure-demand oxygen regulators.

FIG 1: ALTITUDE CHAMBER FLIGHT PROFILE



Upon rapid decompression to 33,000 ft SSW complained of numbness and a tingling sensation in his right arm, and a dull ache in his right wrist and elbow. He was switched from air-mix to 100% oxygen, and immediate descent commenced, at a rate of 4,000 feet per minute. He reported slight relief of the symptoms. In retrospect, SSW claimed that the tingling sensation over the right upper limb was first felt at 18,000 feet but he had ignored it then.

On examination after reaching ground level, he was conscious and rational but slightly apprehensive and perspiring. His pulse was 100/min., regular, and the blood pressure was 140/90 mm Hg (sitting). There were no neurological deficits, but there was subjective weakness and residual ache in his right upper limb.

Decompression sickness was considered the most likely diagnosis. The Naval Diving Unit was alerted and the patient transferred over for further management.

He was treated according to RN Table 61. After 20 minutes at a depth of 60 feet all symptoms were alleviated. He was then given 4 hours of oxygen therapy intermittently (1 hour oxygen: 15 minutes air). The patient recovered fully and was discharged the next day.

Review 4 months later indicated complete recovery Long Bone X-rays were normal.

DISCUSSION

Decompression sickness following exposure to high altitude is in most respects, similar to that occurring after decompression from a high-pressure environment.

Limb pain (the "bends"), respiratory disturbances (the "chokes"), skin changes ("the creeps"), and various disturbances of the central nervous system (the "staggers") are common to both conditions. In fact the trend has been to apply the knowledge gained over the years from the study of diving decompression sickness to decompression sickness occurring at altitude.

The exact mechanisms involved in producing the variety of symptoms are still incompletely understood, though there is little doubt that the underlying aetiology is the formation of inert gas bubbles in the body (7). Using Doppler ultrasound techniques, it is now possible to demonstrate the presence of these bubbles in the circulation, even before the onset of symptoms (8).

While the underlying aetiology is similar, the circumstances under which decompression sickness occurs in diving and in flight are not identical. No matter how high the aviator ascends, the difference between his peak altitude and that at sea level cannot exceed 760 mm Hg — whereas in the case of the diver or caisson worker at 4 to 5 atmospheres, the total change in pressure on return to sea level would obviously be greater than 760 mm Hg. Furthermore, for the aviator, immediate descent upon experiencing symptoms is itself a form of recompression therapy. These fundamental differences may account for some distinct dissimilarities in clinical manifestation between altitude decompression sickness and diving decompression sickness.

One striking feature of aviators' bends, for example, is the rapid and complete recovery which occurs in the vast majority of cases, and an absence of permanent sequelae. It has been known since the beginning of this century, that a significant proportion of compressed air workers develop dysbaric osteonecrosis in later life. Typically, these lesions comprise areas of ischaemic necrosis in the head and neck of the femur, the head of the humerus and, less commonly, the lower end of the femur and upper end of the tibia. The overall incidence of radiographically identifiable lesions among some 2,000 caisson workers registered with the British Medical Research Council is about 20% (9). But to-date, inspite of several radiological surveys of groups of individuals exposed repeatedly to altitudes above 25,000 feet, only 3 cases of dysbaric osteonecrosis due to altitude exposure have been reported in the literature (10).

Another notable difference is the rareness of permanent neurological damage and spinal cord involvement with aerial decompression (4). Unlike divers in which there is a predilection for spinal cord involvement, there is a predilection among aviators for brain involvement — giving rise typically to transient blurred vision, scintillating scotomata and hemianopia. These often occur in conjunction with a headache resembling migraine, or more rarely, transitory hemiplegia, monoplegia, aphasia or fits (10).

Skin manifestations common in diving, are less common in altitude decompression sickness. Chokes are also comparatively rarer (4). If these are untreated. however, they may progress to irreversible shock and a fatal outcome.

To-date, 17 fatal cases of altitude decompression sickness have been reported in the open literature (4). Almost all began as simple bends pain, progressing to neurological and respiratory manifestations, and developing irreversible shock after several hours. Various extensive fluid-replacement schemes and drug regimens failed to alter the course in those aviators. All these cases occurred prior to 1959, before recompression therapy was used in the treatment of altitude decompression sickness.

In both our cases, the patients were recompressed to 60 feet (2.8 atm abs) while breathing 100% oxygen. The entire procedure took 135 minutes to complete. After therapeutic recompression, patients were given 100% oxygen for 4-6 sessions of 1 hour oxygen and 15 minutes air at the surface. Breathing 100% oxygen is aimed at removal of nitrogen from the body while the use of intervening air reduces the hazard of hyperbaric oxygen toxicity.

The value of adjuvant drugs in the management of decompression sickness has been reviewed (11). These include plasma expanders, heparin, corticosteroids, antiplatelet agents, vasodilators, vasopressors, bronchodilators and diazepam. In one of our cases, Rheomacrodex, a low molecular weight dextran was used to combat microcirculatory stasis and correct for loss of fluid to the extravascular compartment.

Protection against altitude decompression sickness in modern day aircraft is mainly through cabin pressurization. An aircraft with a 5 psi cabin pressure differential will maintain a cabin pressure altitude of approximately 17,000 feet while operating at an actual altitude of 40,000 feet. Above 50,000 feet, the protection provided by cabin pressurization must be supplemented through the use of pressurized suits (12). No pressurization system is failure-proof, however, as one of the present two cases illustrates. In fact, the greater the ceiling altitude attainable through cabin pressurization, the greater is the potential for sudden decompression. Lately, the development of the Anglo-French supersonic passenger plane "Concorde" has necessitated studies into the consequences of window "blow-out" at 50,000 — 70,000 feet altitude at which it cruises (13). Other problems encountered with military flying are decompression occurring as a result of enemy action and ejection at high altitude.

Gradual decompression by stage ascent, as practised by deep sea divers has, for practical reasons, no place in aviation. This is because in order to avoid nitrogen bubble formation, it would require a uniform ascent of only 78 feet per minute or a stage ascent of 156 feet per minute (14), both of which are clearly unacceptable in aviation.

On the other hand, denitrogenation by breathing 100% oxygen, which has been found to be not practical in diving, offers great possibilities in aviation. Indeed, the only effective prophylactic measure against altitude decompression sickness is pre-breathing 100% oxygen before flight. Approximately 40% of the nitrogen stores of the body are eliminated during 30 minutes of pre-oxygenation. It has been shown that breathing pure oxygen at ground level for 15 minutes will reduce the incidence of bends at 38,000 feet by approximately 50% while 1 hour of pre-oxygenation will reduce the incidence at the same altitude by 95% (15).

The question as to why only a small proportion of persons exposed to low pressures succumb to decompression sickness and to post-descent shock remains unanswered. A number of factors have been shown to increase susceptibility to altitude decompression sickness, including the rate of ascent, final altitude reached, age, previous exposure, physical activity, and obesity

(12, 16, 17, 18). After a single episode, susceptibility to altitude decompression sickness is increased. Moreover, there is a tendency for the bends pain to recur in the same location (16). As for the effects of exercise, the incidence has been reported to be more than 3 times as great in persons exercising than in those at rest, with the region most affected being the part which is exercised (16).

Finally, bends rarely occur below 18,000 feet unless the subject has been exposed to a hyperbaric environment such as scuba diving prior to ascent. For this reason, personnel should avoid flying or performing low pressure chamber runs for 24 hours following scuba diving, compressed air dives, or high pressure chamber runs (19).

CONCLUSION

Exposure to reduced atmospheric pressure at high altitude or in low pressure chambers exposes man to the risk of developing decompression sickness. Doctors responsible for the health care of flying personnel must be alert for such incidents. Early recognition of the different manifestations of this condition is essential, if delays in starting proper therapy are to be avoided. The importance of close coordination between aeromedical and naval medical diving facilities cannot be overemphasised.

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