

## PULMONARY CT SCANNING SHOULD BE CONSIDERED IN DOUBTFUL DCS TO ELIMINATE PULMONARY GAS EMBOLISM

Ş. Aktaş<sup>1</sup>, S. Aydın<sup>1</sup>, H. A. Nogay<sup>1</sup>, M. Çimşit<sup>1</sup>

Deniz ve Sualtı Hekimliği Anabilim Dalı, İstanbul Tıp Fakültesi, Çapa, 34390,  
İstanbul/Türkiye

### SUMMARY

In decompression sickness when the definite diagnose is not possible, CT scanning of the pulmonary system is essential. Many clinicians may be faced problems to differentiate DCS from pulmonary gas embolism, especially when the ascent rate is less than 10 m/min, bottom time is within the "0" deco limits according the tables.

Four patients who developed neurologic symptoms after SCUBA diving were suspected and CT scanning were performed, which were showing air trapping lesions in the lungs. One patient developed symptoms two times, one developed four times and the rest only once.

Key Words: Pulmonary CT, Air embolism, decompression sickness

### INTRODUCTION

Although the sign and symptoms may be similar, decompression sickness and air embolism have different origins and mechanisms. Treatment of these diseases have same principles because the formed pathologies are mostly alike (Elliott, 1982; Davis, 1990). In decompression sickness main reason is to stay at depths more than certain time limits and not having sufficient decompression stops while surfacing. But in embolism the air is generated from the lungs because of insufficient expiration (Edmonds, 1980).

Decompression sickness mostly involves spinal cord while air embolism settles in brain and the symptoms develop relatively much earlier than DCS (Pearson, 1984). Decompression sickness may occur even within no decompression limits. Individual properties of divers are also important. Air embolism might also be seen in slow ascent rates depending to air trapping lesions in the lungs individually. Differential diagnosis gains importance during treatments but mostly in giving diving permission after the treatment.

### MATERIALS AND METHODS

CASE 1: 16 year old male SCUBA diver. He was permitted to dive after a medical examination including his chest X-Ray. He performed a dive to 32 meters 18 minutes bottom time and his ascent rate was 10 mts/min. During surfacing he experienced vertigo, nausea and disorientation. On the boat he had difficulty in walking due to his balance disorder. His gait was slightly atactic and he had mild dysmetria. Otoscopic examinations revealed normal findings in both ears. Audiograms and tympanograms were normal but ENG showed areflexia in the left side which didn't respond to 20, 30 and 44 °C caloric stimulations. Hypoc excitability was detected in the right side. ENG result was complete left vestibular loss and incomplete right vestibular dysfunction. This patient was diagnosed as air embolism and CT examination of the lungs showed bullous formation.

CASE 2: A 44 year old, male, Cypriot SCUBA diver. In 1986 after surfacing a 30 minutes dive from 18 meters he passed out for a few minutes. In London his cardiac examinations, EEG, brain CT were found normal. This event was attribu-

ted to insecticide use prior to dive. In 1988, after surfacing a 45 minutes dive from 15 meters he experienced unconsciousness, strength loss and tingling sensation in extremities. His complaints disappeared after ten minutes (He had used insecticide prior to dive.) In 1990, he had lost consciousness during surfacing after a vigorous dive to 18 meters for 30 minutes. His attender pulled him out and he stayed semiconscious for 30 minutes on the boat. In 1991, he experienced blurring in the vision, numbness in the face and in the both hands after surfacing from 21 meters dive of 35 minutes duration. He swam to the shore and symptoms disappeared after an hour. One year later he applied to our department for medical examination. His pathologic findings were decreased vibration sensation in the lower extremities (right affected more than the left) and hypoesthesia in the right cruris. Patient diagnosed as an air embolism and CT examinations of the lungs showed bullous emphysematous lesions in the both sides.

CASE 3: 36 year old, male SCUBA diver. After having a normal medical examination result, he had dive for two years usually with a deco brain. He made a dive to 20 meters for ten minutes and he spent another 50 minutes at 10 meters. After 3.5 hour surface interval he made his second dive to 27 meters for 10 minutes. During his ascent he spent 20 minutes between 15-9 meters. When he ascent to 6 meters he experienced tingling in his face, a sudden sharp pain in his back and five minutes after surfacing numbness and strength loss developed in his left leg. He made water recompression attempts two times to 9 meters. He felt normal at nine meters but in ten minutes after surfacing he felt worse than before. Being given 8 mg. dexamethasone IM, 1.5 liters fluid orally, Aspirin 500 mg and 100% O<sub>2</sub> inhalation during transportation, he was transferred in a pressurised aircraft. After four hours he was recompressed. After ten HBO sessions he was completely normal. MRI investigations of the brain and spine was normal. Since decompression sickness diagnose was not certain air embolism was suspected and CT examination of the lungs showed bullous formations.

CASE 4: 38 year old, male, SCUBA diver. He made a dive to 70-75 feet depth, duration 25 minutes. According to his history ascent rate was normal; approximately 10 mts/min. His left side paralysed soon after surfacing and he had difficulty in speaking for two hours, then he returned to completely normal. Six months later he went to Cleveland Clinics Foundation in US for medical examinations. Detailed examinations including MRI of head and spine were performed. A noise induced hearing loss and high cholesterol levels were detected. He was given a diet program and an advise to wear ear noise protectors during gun-shooting. After his detailed medical examinations he was told that he could dive again. On his first dive, he dived to 142 feet for 13 minutes and ascent to 60 feet for 10 minutes and surfaced. Ten minutes after surfacing he felt a sharp pain in his abdomen and in his back. Strength loss and numbness developed in both legs. Symptoms faded so he didn't ask for medical assistance. Four hours after the dive he became worse. He couldn't urinate and defecate. He came to our department after 17 hours of the accident. He was recompressed to 60 feet and after the first treatment he became much better. Further daily HBO sessions accompanied with rehabilitation gave good result. After 12 sessions HBO treatment was discontinued and only rehabilitation programme was applied. He respond well to therapy and dehospitalized only having some degree of difficulty in urinating. Because of the uncertainty in differential diagnoses between DCS and air embolism CT scanning of the lungs was performed. Small nodular densities in upper lobes of both lungs and posterior subpleural bullae were detected.

## DISCUSSION

Isolated vestibular decompression sickness can be seen rarely in air diving (Edmonds,1980;Farmer,1984) and it is reported in grossly omitted decompression cases in air diving (Aktaş,1990).Case 1 had made a mild dive which was within 0 deco limits.Bilateral isolated vestibular involvement accompanied other cerebellar signs (ataxia and dysmetria) lead us to a central pathology because there were no pathology in hearing tests.Also inner ear barotrauma can be eliminated because there was no ear clearing difficulty in his diving history.Bilateral involvement without any other DCS sign made us to think an emboli of the Scarpa ganglion of the cerebellum could be the cause of these findings and this might happen even in the absence of high ascent rates.CT scanning of the lungs showed the lesions which were not detected with the ordinary chest X-Rays.

In case 2 who had several attacks following short dives without having cardiac abnormality predisposing to DCS,We thought insecticide usage prior dive or an underlying pulmonary pathology as the cause. Since there were no contact with insecticide before the last two dives, pulmonary pathology gained more probability.However his ascent rates were slow,an air trapping lesion might be the cause and CT scanning of the lungs confirmed that.

The symptoms of case 3 were showing similarities to DCS.Though it was a multilevel dive,the use of decobrain and the analyses of diving profile made it unlikely that it was a decompression sickness.Although the ascent rate was slow,the pulmonary air embolism was possible if air trapping lesions were available CT examination of the lungs after the treatment showed the bullae which were not detected prior chest x-Rays.

In the first attack of case 4 ; his diving profile,onset and the character of the symptoms lead us to a pulmonary embolism.Diving profile in his second attack was very mild for causing a DCS but location and the development of the symptoms made the differential diagnosis very difficult.Although he passed detailed medical examination even including MRI of the brain and the spine,nodular findings on the chest x-Ray and his tuberculosis history combined with his first attack would have led us to order CT scanning of the lungs.In this case after the treatment the diver underwent a CT examination of the lungs before the last decision about his diving life.After CT scanning he was not allowed to dive because of the air trapping lesions.

## CONCLUSION

Air trapping lesions are eliminating factors from diving but with ordinary chest x-Ray examination,sometimes it is difficult or not possible to detect these pathologies.Although CT scanning is giving better results, it is non practical and expensive even in countries where tuberculosis incidence is high.In doubtful DCS cases CT scanning of the lungs must be performed not only selection of the treatment but also for the decision about further diving life.

## ACKNOWLEDGEMENT

We here wish to thank to Dr.Turgay Osman for the audiologic assessment of the cases.

#### REFERENCES

- Aktaş, Ş., Aydın, S., Osman, T., Çimsit, M. (1990) Severe omitted decompression resulted inner ear decompression sickness in air diving. Undersea Biomedical Research Supplement to vol 17. Abs. no. 170.
- Davis, J.C. (1990) Treatment of decompression sickness and arterial gas embolism. In: Diving Medicine, Bove, A.A. and Davis, J.C. (eds.) WB Saunders co. second ed. New York, pp. 249-261.
- Edmonds, C. (1980) Decompression sickness. In: Diving and Subaquatic Medicine. Edmonds, C., Lowry, C., Pennefather, J. (eds.) DMC. 2<sup>nd</sup> ed. Sydney, pp. 129-185.
- Edmonds, C. (1980) Hearing loss and disorientation. In: Diving and Subaquatic Medicine. Edmonds, C., Lowry, C., Pennefather, J. (eds.) DMC. Second Ed. Sydney, pp. 383-407.
- Elliott, D.H., Kindwall, E.P. (1982) Manifestation of the decompression sickness. In: The Physiology and Medicine of Diving. Bennett, P.B., Elliott, D.H. (eds.) Bailliere Tindall, third ed. London, pp. 461-473.
- Farmer, J.C. (1984) Inner ear decompression sickness. In: The Physician's Guide to Diving Medicine. Shilling, C.W., Carston, C.B., Mathias, R.A. (eds.) Plenum press, London and New York, pp. 312-316.
- Pearson, R.R. (1984) Diagnoses and treatment of gas embolism. In: The Physician's Guide to Diving Medicine. Shilling, C.W., Carston, C.B., Mathias, R.A. (eds.) Plenum press, London and New York, pp. 333-361.