Electroencephalography and magnetic resonance imaging in neurological decompression sickness.

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[1] Department of neurology, Haukeland University Hospital, [2] Section of hyperbaric medicine, Department of occupational medicine, Haukeland University Hospital, [3] Neuroradiological Division, Department of radiology, Haukeland University Hospital, [4] Department of Occupational medicine, Haukeland University Hospital, [5] Department of Hyperbaric medicine, University of Bergen, Norway.

Grønning M, Risberg J, Skeidsvoll H, Moen G, Aanderud L, Troland K, Sundal E, Thorsen E. Electroencephalography and magnetic resonance imaging in neurological decompression sickness. Undersea Hyperb Med 2005; 32(6):397-402. The purpose of this study was to evaluate the use of electroencephalography (EEG) and magnetic resonance imaging (MRI) in the clinical evaluation of acute decompression sickness (DCS) in the central nervous system (CNS). Twenty-one patients treated because of acute DCS in the CNS during 1999-2001 were included, 15 patients with clinical cerebral DCS and five with clinical spinal cord DCS. Seven patients had abnormalities in their EEG, five with a cerebral DCS and two with spinal cord DCS. MRI showed high intensity lesions in the spinal cord in four patients with clinical spinal cord DCS and in one with clinical cerebral DCS. Cerebral lesions were not identified by MRI in any patient. In conclusion, EEG showed unspecific abnormalities in only one third of the cases. Conventional MRI with a 1.5 T scanner may be of help in the diagnosis of DCS in the spinal cord, but not in the brain. EEG and MRI have low sensitivity in the diagnosis of acute DCS in the CNS. Recompression treatment of DCS should still be guided by clinical neurological examination and assessment of symptoms.

INTRODUCTION

Neurological decompression sickness (DCS) is a result of free gas bubbles located in the central nervous system (CNS). During decompression inert gas supersaturation is present in the tissues and in the blood stream. The inherent instability of supersaturation may result in free gas being evolved in stationary bubbles locally in tissues or in bubbles transported as gas microemboli in the blood stream. DCS may involve any part of the central nervous system. The symptoms may vary from cognitive symptoms or slight sensory disturbances to more severe paralysis and loss of function. In the most severe cases the condition may be life threatening. The clinical symptoms usually occur within the first hour after the dive, in most cases within three and rarely as long as 24 hours after the dive (1). Neurological decompression sickness is associated with electroencephalographic (EEG) abnormalities (2-4), but the findings are not specific for this condition. Although there are several MRI studies on divers(4-6), few emphasize the diagnostic value of MRI in acute DCS (7-9). In this study we report the findings from EEG and MRI in 20 patients with neurological decompression sickness examined and treated in our hospital during the period 1999-2001.

METHODS

Facilities, Subjects, and Protocols

All patients with neurological decompression sickness were included who received recompression therapy in 1999-2001 at the Haukeland University Hospital, were inpatients at the Department of Neurology and were followed up by a neurologist. They were referred to the Department of Neurology after their first recompression treatment, which was initiated by the diving contingency team at the Department of Hyperbaric Medicine. The first treatment was recompression to 2.8 atmospheres absolute (ATA) according to the US Navy Table 6, supplemented by daily hyperbaric oxygen treatments at 2.4 ATA for 90 minutes breathing 100 % oxygen. The number of supplemental hyperbaric treatments depended on the symptoms and the clinical findings of the individual patient. Included in this study are 20 patients, examined with EEG and MRI during the first 2-7 days after the accident when they still had symptoms and/or clinical neurological findings of the disease. One patient had MRI 10 days after the incident. Eighteen were sports divers and two were professional divers. Fifteen patients, four women and 11 men with a mean age of 28 years (range 17-38 years) had definite cerebral symptoms (see Table 1). Fourteen of these had positive clinical neurological signs when examined after the first treatment. Five patients, all men with a mean age of 38 years (range 18-57 years) had symptoms and signs from the spinal cord only.

EEG and MRI recordings

The EEG was a 21-channel recording with electrode placement according to the 10-20 system. All EEGs were recorded at the same neurophysiological laboratory and read independently by two neurophysiologists. The MRI studies were done with a 1.5 Tesla (T) scanner. The examination included T1-weighted proton density (PD) and T2- weighted images. In addition axial diffusion weighted (DW) images of the cerebrum were done in all patients. T1 and T2 images of the spinal cord were done in patients with possible spinal cord involvement. The MRI's were evaluated independently by two neuroradiologists.

RESULTS

Cerebral decompression sickness

In the fifteen patients who reported cerebral symptoms, 14 had positive clinical neurological findings and five had

Table 1. The average age of the patients, the proportion with clinical neurological findings after the first and last recompression treatment (1 and 2), and the proportion with abnormal results on EEG and MRI scan.

	N	Average age (range)	Positive neurol. 1	EEG	MRI	Positive neurol. 2
Cerebral DCS	15	28 (17-38)	14/15	5/15	1/15	3/15
Spinal DCS	5	38 (18-57)	5/5	2/5	4/5	5/5

abnormalities in the EEG, 4-6 Hz theta activity in the temporal or frontal regions on the day after the first recompression treatment. At discharge from hospital only one of these five had EEG abnormality. All had normal findings on MRI of the brain (Case 1). One of these patients, however, had a lesion in the thoracic part of the spinal cord on T2 weighted MRI, compatible with an ischemic lesion. After the last recompression treatment only three of these 15 patients had minor positive clinical neurological findings.

Spinal cord decompression sickness

Five patients had symptoms and clinical signs from the spinal cord only. These had clinically myelopathy after both the first and the last recompression treatment although the neurological findings had significantly regressed in four patients. Two had abnormal EEG's, one having 4-6 Hz theta activity in the frontal region and one generalized slow wave activity. They did not however have a second EEG at discharge from hospital. Four had high intensity lesions in T2-weighted MRI indicating ischemic lesions. One of these had normal findings on the MRI of the cerebrum and the spinal cord on examination the first day of the incident but high signal lesions on T2 weighted images in the cervical and thoracic parts of the spinal cord when examined one week later. In one patient the lesion was located in the cervical area and in two patients in the thoracic area of the spinal cord, consistent with the clinical neurological findings. One patient with a severe spastic paraparesis had normal findings on MRI, Case 2.

Case 1

A 22-year-old woman had two SCUBA dives to 13 meters. Immediately after the last dive she complained of vertigo and nausea. One hour after the dive she received recompression treatment on US Navy Table 6. When examined after the first treatment she was unsteady with positive Romberg test, hyperreflexia in her left arm and leg, asymmetric abdominal reflex and slightly asymmetric plantar reflexes. The EEG showed focal abnormality with 4-6 Hz theta waves in her right temporal region. MRI of the brain was normal.

Case 2

A 53 year-old man had two dives to maximum 26 meters lasting for 30-40 minutes with a three-hour surface interval. Immediately after the second dive his lower limbs became weak and numb. During the transport to the hospital he experienced urinary incontinence. At the beginning of hyperbaric oxygen treatment he had severe paresis in both legs. After the first treatment he had a severe spastic paraparesis with hyperreflexia and Babinski's sign and touch hyposensitivity below the level of T6. Sitting in bed he had postural instability. MRI of the cerebrum and the spinal cord did not show any abnormalities. EEG showed abnormal 4-6 Hz activity in both temporal regions. Somatosensory evoked response (SER) from the tibial nerve showed significantly increased latency bilaterally indicating a pathologic process in the spinal cord. During repeated hyperbaric oxygen treatments he regained some function. However, one week after the accident he was still not able to walk. He was referred to his local hospital for rehabilitation. One month later he had a spastic paraparesis but was able to walk a short distance with crutches and he had regained almost normal urinary control. One week and one month after the DCS MRI did still not show any abnormality.

DISCUSSION

Fourteen of the 15 patients with cerebral decompression sickness and all five patients with spinal decompression sickness had positive findings on the clinical neurological

examination even after the first recompression treatment. Five of the patients with cerebral disease and two of the patients with spinal cord DCS had abnormal EEG. MRI showed abnormalities in the spinal cord in five patients, in one with cerebral DCS and in four out of the five with spinal cord DCS.

The finding of a low fraction of the patients having abnormal EEG is consistent with the finding of Todnem et al (2). In their study EEG was recorded 1-3 days after completed hyperbaric oxygen treatment. They found abnormality in EEG in four out of 21 divers with neurological decompression sickness, one having slow waves and sharp potentials in the right temporal and occipital regions; the others increased slow waves in both hemispheres. Increased slow wave activity in EEG was described in 7 out of 20 sports divers with neurological decompression sickness after the first recompression treatment in the study of Bjørnstad et al. (3). Other authors have reported EEG findings in divers with histories of neurological decompression sickness with divergent results (6,10). Sipinen found that the proportion of abnormal EEG's was significantly higher in a group of divers with history of DCS compared with a control group (6). Murrison compared the EEGs of 68 divers with histories of neurological decompression sickness with 45 non-diver controls, and found that they were electroencephalographically indistinguishable (10). The EEG abnormalities are not specific but may indicate multifocal central nervous system lesions.

In all of our patients the MRI of the brain was normal. Reuter et al examined 16 divers with dysbaric injuries after the initiation of therapeutic recompression and found ischemic cerebrovascular lesions in 6/8 patients with arterial gas embolism and in 2/8 divers with DCS (7). In the study of Sipinen et al. (6) four of 25 divers who had been treated in a pressure chamber due to decompression sickness during the previous 6 years, had hyperintense lesions in the white matter of the brain. The lesions were thought to be of vascular type. However, no information of the age of these divers or the possible occurrence of hypertension or other risk factors for cerebrovascular disease was given. All 24 healthy controls and 29 healthy naval divers who had not suffered DCS had normal brain MRI. In the study of Yanaganawa et al. MRI showed lesions in the brain in 9 out of 25 divers (5). Age, diving history, smoking and alcohol consumption were highest for those with CNS lesions. Our MRI results in patients with spinal cord decompression sickness are in accordance with the findings of Sparacia et al. (8). In two divers with spinal cord decompression sickness they found on T2-weighted images increased signal intensity in the dorsal columns of the spinal cord in one patient at levels C3-Th1 and a focal area at Th9 level, and in the other patient at level C2-C4. In the study of Levin et al however, MRI within two weeks after the decompression sickness in two divers revealed subcortical white matter lesions whereas no lesion of the spinal cord was demonstrated despite the fact that one diver clinically had a spinal cord decompression sickness (9). In the study Warren et al. cranial MRI was abnormal in three out of four patients with cerebral gas embolism and MRI documented spinal cord abnormalities in three out of 12 patients with decompression sickness and spinal cord symptoms (11). Thus, a normal MRI of the brain or the spinal cord does not disqualify the diagnosis neurological decompression sickness.

There are still controversies regarding the pathophysiology of decompression sickness. Arterial bubbles may be trapped in the cerebral circulation if they form columns that are long enough to occupy several generations of branching arterioles such that the net surface tension pressure acting on the column exceeds cerebral perfusion pressure (12). This

is most likely to occur in small arterioles, and the boundary between the grey and the white matter, which is rich in such vessels (13). It is in this region in particular that ischemia has been demonstrated following experimental gas embolism of the brain (14) and in which acute tissue infarction has been demonstrated in an animal model of DCS (15). We could, however, not demonstrate any lesions in this area by MRI.

Other mechanisms may be of importance in DCS of the spinal cord. Spinal cord infarction in DCS may be caused by obstruction of cord venous drainage at the level of the epidural vertebral venous system as shown in a study of anesthetized dogs (16). In this study there were widespread and severe hemorrhages in the spinal cord white matter, with remarkable sparing of the grey matter. A later study of acute neurological decompression illness in pigs showed however, a somewhat different histopathologic picture (17). Sixteen of 80 pigs that suffered functionally from decompression sickness died. Petecchial hemorrhages were grossly visible in the spinal cord of 73% of the survivors and in 63% of the fatalities. White matter hemorrhages in the spinal cord were generally more numerous and extensive than those affecting the grey matter; however, grey matter hemorrhage was associated with increasing disease severity. In our study MRI showed spinal cord lesions in four of five cases with spinal cord DCS.

In our patients the conventional MRI techniques using a 1.5 T scanner did not show pathological changes in the brain. This may be explained by low sensitivity of this technique, but may also indicate that the pathophysiology of cerebral DCS in particular, is more complicated and not necessarily of structural origin.

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