Cerebral Magnetic Resonance Imaging of compressed air divers in diving accidents.

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Gao GK, Wu D, Yang Y, Yu T, Xue J, Wang X, Jiang YP. Cerebral Magnetic Resonance Imaging of compressed air divers in diving accidents. Undersea Hyperb Med 2009; 36(1):33-41. To investigate the characteristics of the cerebral magnetic resonance imaging (MRI) of compressed air divers in diving accidents, we conducted an observational case series study. MRI of brain were examined and analysed on seven cases compressed air divers complicated with cerebral arterial gas embolism CAGE. There were some characteristics of cerebral injury: (1) Multiple lesions; (2) larger size; (3) Susceptible to parietal and frontal lobe; (4) Both cortical grey matter and subcortical white matter can be affected; (5) Cerebellum is also the target of air embolism. The MRI of brain is a sensitive method for detecting cerebral lesions in compressed air divers in diving accidents. The MRI should be finished on divers in diving accidents within 5 days.

INTRODUCTION

Recreational Scuba (self-contained underwater breathing apparatus) diving has attracted a lot of people in western countries. However, most of the professional divers used the surface-supplied light diving equipment in northern China. Although the overall incidence of diving-related injury is small, the increasing number of dives performed each year will result in an increasing number of previously healthy individuals who will be affected by diving accidents. To many people, the terms ‘decompression illness’ (DCI) ‘decompression sickness’ (DCS) and ‘the bends’ are synonymous. However, although there should be no difference in the immediate treatment of anyone suffering from these conditions, the terms do not necessarily mean the same. Decompression Illness (DCI) is an umbrella term for both decompression sickness (DCS) and Arterial Gas Embolism (AGE) (1). Brain imaging of DCI has been reported by several authors (2-5); however, to our knowledge, few previous reports have described cerebral MRI characteristics associated with AGE.

MATERIALS AND METHODS

From September 2002 to April 2005, seven patients, male, 23-35 years old (30.7±4.9 years), were referred to the Department of Hyperbaric Medicine, No. 401 hospital of PLA in Qingdao of China for diving related injuries. AGE was diagnosed by the following criteria: Symptom onset time of less than 15 minutes post-dive; Presence of cerebral neurological signs, symptoms, or findings; and Symptom duration greater than 15 minutes. Histories including rapid ascent, out-of-gas, cardiopulmonary symptoms, pneumothorax, or mediastinal or subcutaneous emphysema
increased the confidence of AGE diagnosis (1). All of the divers included in the study were examined by the same Neurologist (the director of the department), who is experienced in the field of Diving Medicine, and diagnosed with AGE. Information relevant to each dive and the subsequent clinical condition are shown in Table 1, see opposite page.

MRI Scans were performed on a Novus prototype operating system at 1.5 T. Axial head images were obtained with T1-weighted (TR/TE, 500ms/20ms), T2-weighted (TR/TE, 4500ms/120ms) and FLAIR (TR/TE, 10000ms/128ms). Ti-weighted images with contrast enhancement were not performed.

The interval between the diving accident and the initial neuroimaging study ranged from 1 day to 5 days. Neurological examinations were performed by the same neurologist to characterize the extent of clinical compromise. Patients were treated with therapeutic hyperbaric recompressions and auxiliary therapy at the Department of Hyperbaric Medicine in No. 401 hospital of PLA Qingdao.

RESULTS

We analyzed MRI images from each of the seven cases and characterized the lesions that were larger than 1× 1cm² by location and size. These results are summarized by Table 2. And we also calculated the number of diver CNS lesions location in brain (Table 3).

On the MRI examinations, abnormal areas were of increased signal intensity on T2-weighted images and FLAIR images. All seven cases involved predominantly cortical and the subcortical white matter.

CASE REPORTS

Case I: Cerebral arterial gas embolism (CAGE)

A 35-year-old male, healthy professional diver at work, digging for abalone supplied by compressed air. The water temperature was 25°C. After a first dive to 26m for 40 mins, he resurfaced for a 1 hour break on the ship. 20 minutes into his second dive, he began to experience difficulty breathing so he blew up from the bottom. A few minutes later he lost his consciousness. At 6 h after the accident he was admitted to hyperbaric oxygen department of No.401 hospital. He was diagnosed as CAGE, and treated immediately with 100% oxygen, intravenous fluids, a bolus of 10 mg of intravenous dexamethasone put in the decompression chamber (United States Navy treatment table II – using oxygen, total time was four and a half hours).

An MRI scan of the brain was undertaken within 24 h of the injury. Axial T2WI and FLAIR images reveal multiple lesions of high signal within left occipital lobe, both basal ganglia, both centrum semiovale and both the frontal lobes (Figure 1,2). After 10 sessions of hyperbaric oxygen and other medical treatment, the patient was partially recovered and discharged from our department.

Case II: Cerebral arterial gas embolism and spinal cord DCS

A 35-year-old male professional diver, previously healthy, diving for abalone using surface supplied light diving equipment. His diving depth was 20 msw, total bottom time was 30 mins, and he was supplied by compressed air from the ship. A mechanical failure in the ship’s pump caused a disruption in the supply of compressed air to the diver. He experienced difficulty breathing and immediately blew up from bottom. He had symptoms of chest pain and then lost consciousness. The signs of pneumoderma in the neck was found. Six hours later, the patient was admitted to hyperbaric medicine department of No.401 hospital for recompression therapy (US Naval treatment table II).

He was diagnosed as having pulmonary barotraumas and spinal cord DCS. After the recompression therapy and auxiliary treatment, an MRI scan of the brain was undertaken within
msw: meter sea water

**Table 1.** Diving stats and related clinical condition

<table>
<thead>
<tr>
<th>No.</th>
<th>Age (msw)</th>
<th>Gender</th>
<th>Total bottom time (mins)</th>
<th>Neurological symptoms and signs of diving accidents</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>35,M</td>
<td>26</td>
<td>70</td>
<td>dizziness, unconsciousness</td>
</tr>
<tr>
<td>2</td>
<td>37,M</td>
<td>20</td>
<td>30</td>
<td>paraplegia, unconsciousness; pneumoderma</td>
</tr>
<tr>
<td>3</td>
<td>25,M</td>
<td>25</td>
<td>80</td>
<td>emphysema, unconsciousness; vomiting; pneumoderma</td>
</tr>
<tr>
<td>4</td>
<td>33,M</td>
<td>18</td>
<td>120</td>
<td>paraplegia, unconsciousness; mediastinal emphysema</td>
</tr>
<tr>
<td>5</td>
<td>34,M</td>
<td>17</td>
<td>90</td>
<td>unconsciousness</td>
</tr>
<tr>
<td>6</td>
<td>23,M</td>
<td>28</td>
<td>20</td>
<td>left hemiparesis, unconsciousness</td>
</tr>
<tr>
<td>7</td>
<td>30,M</td>
<td>14</td>
<td>90</td>
<td>dizziness, unconsciousness</td>
</tr>
</tbody>
</table>

**Table 2.** Cerebral MRI manifestations and characteristics

<table>
<thead>
<tr>
<th>No.</th>
<th>Lesions location</th>
<th>Manifestation</th>
<th>Size (cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Occipital + Frontal lobe</td>
<td>Patchy</td>
<td>2×4; 2×3</td>
</tr>
<tr>
<td>2</td>
<td>Left centrum semiovale</td>
<td>Patchy</td>
<td>2×2</td>
</tr>
<tr>
<td>3</td>
<td>Left frontal lobe</td>
<td>Patchy</td>
<td>5×3</td>
</tr>
<tr>
<td>4</td>
<td>Right cerebellum</td>
<td>Patchy</td>
<td>2×2</td>
</tr>
<tr>
<td>5</td>
<td>Central white matter + Left cerebellum</td>
<td>Patchy</td>
<td>2×4; 2×4; 1×2</td>
</tr>
<tr>
<td>6</td>
<td>Both parietal region and frontal region</td>
<td>Patchy</td>
<td>5×4; 6×4</td>
</tr>
<tr>
<td>7</td>
<td>Both frontal lobes + Central white matter</td>
<td>Patchy</td>
<td>1×2; 2×2; 8×3</td>
</tr>
</tbody>
</table>

**Table 3.** Location of the CNS lesions

<table>
<thead>
<tr>
<th>Location</th>
<th>No of divers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cortical</td>
<td>6</td>
</tr>
<tr>
<td>Subcortical</td>
<td></td>
</tr>
<tr>
<td>Central white matter</td>
<td>6</td>
</tr>
<tr>
<td>Basal ganglia</td>
<td>1</td>
</tr>
<tr>
<td>Cerebellum</td>
<td>2</td>
</tr>
</tbody>
</table>

**Figs. 1, 2.** Axial T2WI and FLAIR images reveal multiple lesions of high signal within left occipital lobe, both basal ganglia, both centrum semiovale and both the frontal lobes.
48 h of the injury. Axial T2 weighted image reveals two areas of high signal within left centrum semiovale of subcortical (Figure 3). Electromyography shows C6-7 minor injuries. His strength had grade 1/6 in right lower limbs. The patient received 2.5 ATA HBO treatment daily for 1.5 h for 30 days. His neurological status improved gradually. At 30 days after the accident, his right lower limbs function had improved to grade 5/6. He was discharged from the hospital.

Case III: Pulmonary barotraumas complicated with cerebral arterial gas embolism

A 25-year-old male, healthy professional diver, digging for abalone. He made 2 dives to a depth of 25 msw for a total bottom time of 80 minutes. The water temperature was around 10°C and the breathing gas was compressed air supplied by the ship. A mechanical failure in the ship’s pump caused a disruption in the supply of compressed air to the diver. He experienced difficulty breathing and immediately blew up from bottom. One minute after resurfacing, he lost consciousness. Two hours later, he awoke and appeared hematemesis and paraplegia. At 6 h after the accident, he was admitted to department of hyperbaric medicine and was treated in the decompression chamber (United States Navy treatment table II).

An MRI scan of the brain was undertaken within 24 h of the injury. Axial FLAIR image reveals patchy shadows of high signal within both cerebella and right centrum semiovale. (Figure 6,7. See page 39 for Fig.7)

Case IV: Cerebral arterial gas embolism and spinal cord DCS

A 33-year-old male, healthy professional diver, digging for abalone. His diving depth was 18 msw, which he repeated 4 times for a total bottom time of 80 mins. The water temperature was around 5°C and the breathing gas was compressed air supplied by the ship. A mechanical failure in the ship’s pump caused a disruption in the supply of compressed air to the diver. He experienced difficulty breathing and immediately blew up from bottom. About two minutes later, he lost consciousness. Two hours later, he was recovery from his coma and appear hematemesis and paraplegia. At 6 h after the accident, he was admitted to department of hyperbaric medicine and was treated in the decompression chamber (United States Navy treatment table II).

An MRI scan of the brain was undertaken within 24 h of the injury. Axial FLAIR image reveals patchy shadows of high signal within both cerebella and right centrum semiovale. (Figure 6,7. See page 39 for Fig.7)

Case V: Cerebral arterial gas embolism

A 34-year-old male, digging for abalone. He wore surface supplied light diving equipment, and breathed compressed air supplied by the ship. He made two dives to 17 msw for a total bottom time of 90 minutes. A mechanical failure in the ship’s pump caused a disruption in the supply of compressed air to the diver. He experienced difficulty breathing and immediately blew up from bottom. He lost consciousness upon resurfacing. He awoke one hour later with paralysis of the left leg. He was admitted at 3 h after the accident to the hyperbaric medicine department of No.401 hospital to receive treatment in the hyperbaric chamber (United States Navy treatment table II). An MRI scan of the brain was performed within 1 days of the injury. Axial FLAIR
Fig. 3. Axial T2 weighted image reveals two areas of high signal within left centrum semiovale of subcortical.

Fig. 4. Axial FLAIR image reveals patchy shadow of high signal within left frontal lobe.

Fig. 5. Axial FLAIR image reveals patchy shadows of high signal within both frontal lobes.

Fig. 6. Axial FLAIR image reveals patchy shadows of high signal within both cerebella.
image reveals patchy shadows of high signal within subcortical central white matter of both frontal lobes (Figure 8). After 15 sessions HBO treatment and other medical treatment, the patient was partially recovered and was discharged from our department.

**Case VI: Cerebral arterial gas embolism**

A 23-year-old male with 3 years diving experience, digging for abalone. He wore surface supplied light diving equipment, and breathed compressed air supplied by the ship. He dived to a depth of 28msw for a total bottom time of 20 mins. A mechanical failure in the ship’s pump caused a disruption in the supply of compressed air to the diver. He experienced difficulty breathing and immediately blew up from bottom. About ten minutes later, he lost his consciousness and appeared hematemesis. At 3 h after the accident, he was admitted to hyperbaric medicine department of No.401 hospital and to be put in the decompression chamber (United States Navy treatment table II). Four hours later, he regained consciousness.

An MRI scan of the brain was undertaken within 1 day of the injury. Axial FLAIR image reveals multiple patchy shadows of high signal within subcortical central white matter of left parietal lobe and both frontal lobes (Fig 11, see page 40). Axial FLAIR image reveals multiple patchy shadows of high signal within subcortical central white matter of both parietal lobes and both frontal lobes (Fig 12, see page 40). After 15 sessions hyperbaric oxygen treatment and other medical treatment, the patient was fully recovered and discharged from our department.

**DISCUSSION**

During ascent to the surface, a diver is constantly exposed to alterations of ambient pressure. Barotrauma refers to tissue damage that occurs when a gas-filled body space (e.g., lungs, middle ear) fails to equalize its internal pressure to accommodate changes in ambient pressure. The behavior of gasses at depth is governed by Boyle’s law: the volume of a gas varies inversely with pressure. Arterial gas embolism develops when free air enters the pulmonary vasculature and is carried to the heart and arterial circulation. A large proportion of air bubbles can reach the brain, occlude blood vessels and cause stroke-like events. The most common signs and symptoms of arterial gas embolism are neurological, although pulmonary symptoms may also be present. In more than 80 percent of patients, symptoms develop within five minutes of reaching the surface, but they also can occur during ascent or after a longer surface interval.
Fig. 7. Axial FLAIR image reveals patchy shadows of high signal within right centrum semiovale.

Fig. 8. Axial FLAIR image reveals patchy shadows of high signal within subcortical central white matter of both frontal lobes.

Fig. 9. Axial FLAIR image reveals multiple patchy shadows of high signal within centrum semiovale and subcortical central white matter of both parietal lobes and both frontal lobes.

Fig. 10. Axial T2 weighted image reveals patchy shadows of high signal within subcortical central white matter of frontal lobes.
The pathophysiology and mechanism of injury in scuba diving accidents is still somewhat controversial and has been reviewed in detail (2-5). In Chinese northern part, most of the professional divers use surface-supplied light diving equipment. The diver inhales the compressed air through a long surface supplied gas pipe link up to the ship. The longer surface-supplied gas pipe, the higher the risk of diving accidents. All the cases in this group were caused by the pump failures that disrupted air supply. Cerebral injuries likely are due to arterial gas embolization, either from pulmonary barotraumas with direct rupture into the arterial system or from paradoxic embolization through previously unrecognized right-to-left shunts (that may only be open during abnormal pressure conditions found during diving). Cerebral air embolism generally occurs within minutes of surfacing, with cognitive dysfunction the predominant feature (1). Up to 41 % of patients may be unconscious when first seen. In this group of cases, most of the divers appear cognitive dysfunction or coma at the surface.

Although Gronning(6) described the use of brain MRI in the diagnosis of diving-induced neurologic dysbarism syndromes, these illnesses have not been investigated extensively with neuroimaging methods. MRI has not played a major role in diving accident management, primarily because of the poor sensitivity of lesion detection with conventional cerebral CT scanning as documented. In addition, imaging of spinal cord ischemic lesions, which constitute the majority of diving-related injuries, has not been feasible, although a recent paper suggests that spinal cord damage may not be as prevalent in DCS/DCI as once thought (7). Thus, until recently physical examination has remained the only objective method of patient evaluation. Our cases study supports the use of MRI for diving induced cerebral injuries.
In these cases of suspected CAGE, MRI detected more than fifteen larger lesions in seven patients. All of the cases, the divers’ accidents were due to failure of the gas supply and rapid resurfacing. They suffered from CAGE certified by MRI. We analyzed the characteristics of cerebral MRI, as following:

- Multiple lesions: all the seven cases were injured by CAGE, and manifestation shows multiple lesions. We suspect that gas embolism can go to anywhere through the arterial vessel easily. So the cerebral MRI showed multiple lesions or loci. 
- Larger size: In these cases, cerebral injuries areas larger more than 1×1cm² were calculated. Major locus area were larger more than 6×4cm² or 8×3cm² (Fig10 and Fig 12). We believe that bubbles can go to the larger arterial vessels, such as the middle cerebral artery, and block the blood stream. Susceptible to parietal lobe and frontal lobe: Six cases affected the parietal lobe and frontal lobe (Table 2). It is possible that when the diver stands up or laying down, the bubbles embolism can go to the parietal lobes and frontal lobes easily because of specific gravity. Injury affected not only cortical grey matter but also subcortical white matter: Cortical infarcts have been reported with experimental air embolisms as well as with clinical air embolisms. However, some white-matter involvement was present in the cases reported by Jensen and Reuter (8, 9). In this group, six cases were injured in cortical grey matter except case 2 (Table 3) and MRI showed injuries in subcortical white matter in all seven cases. In all the cases, the injury affecting the subcortical white matter was detected by MRI. CAGE can also affect cerebellum (e.g. case 4 and 5). Of interest is the predominant involvement of subcortical white matter in our series (Fig. 1-12). In addition, the size and location of lesions may be influenced further by vasoconstriction and vasodilatation responses of arterial vessels to gas emboli.

In general, our cases showed that severe AGE more than DCS can injure mainly the brain. The dive profile suggested suspected form of nervous system injury. MRI is a valuable method in the diagnosis of AGE and it should be finished in 1-5 days in diving accidents. The dive profile and suspected form of nervous system injury is pretty clear and consistent with CAGE.

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REFERENCES