

Case Report

A Case of Anterior Cerebral Artery Dissection Caused by Scuba Diving

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A 51-year-old man was admitted with right hemiparesis during scuba diving, without headache. Brain magnetic resonance (MR) imaging depicted high-intensity areas in the left superior frontal and cingulate gyri on diffusion-weighted imaging. Dissection of the anterior cerebral artery (ACA) was detected using axial MR angiography and 3-dimensional MR cisternography. Dissection of the ACA during and after scuba diving has not been reported before. Dissection of the arteries should be included in the differential diagnosis when neurologic symptoms occur both during and after scuba diving, even if the patient does not experience headache. Furthermore, the combination of MR cisternography and MR angiography is useful to detect ACA dissection. **Key Words:** Anterior cerebral artery dissection—ischemic stroke—scuba diving—3-dimensional MR cisternography.

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Introduction

Ischemic stroke in scuba diving is unusually attributable to decompression illness (decompression sickness and arterial gas embolism).¹ Dissection of intra- or extracranial arteries in scuba divers is only rare²⁻⁹ and that of the anterior cerebral artery (ACA) is not reported yet. We describe a patient who experienced ACA dissection during scuba diving and presented at the emergency department with acute ischemic stroke.

Case Report

A 51-year-old man who had no history of hypertension, diabetes mellitus, dyslipidemia, and smoking

was complained of sudden right hemiparesis that developed during scuba diving, without headache. An instructor was aware of his anything odd, and he was raised up to the surface and taken to the nearest general hospital in the ambulance. He had more than 20 years of experience with scuba diving. The whole dive, including pre- and postdiving preparation, took 1.5 hours. The actual dive reached a depth of only 20 m and lasted 1 hour. General physical findings on admission showed blood pressure of 126/60 mm Hg and heart rate of 65 beats/min with sinus rhythm. Neurologic findings showed only right hemiparesis. The National Institutes of Health Stroke Scale score was 2. Electrocardiography showed sinus rhythm. Brain computed tomography did not show cerebral hemorrhage or acute infarct findings. Brain magnetic resonance (MR) imaging at 1 hour after onset depicted hyperintense areas in the left superior frontal and cingulate gyri on diffusion-weighted imaging, and MR angiography showed stenosis of the left ACA (Fig 1). Tissue plasminogen activator (.6 mg/kg) was administered intravenously at 80 minutes after onset. Hemiparesis subsequently improved slightly. Administration of aspirin (100 mg/d) was started 24 hours later.

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Received January 23, 2014; revision received February 18, 2014; accepted February 21, 2014.

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1052-3057/\$ - see front matter

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<http://dx.doi.org/10.1016/j.jstrokecerebrovasdis.2014.02.016>

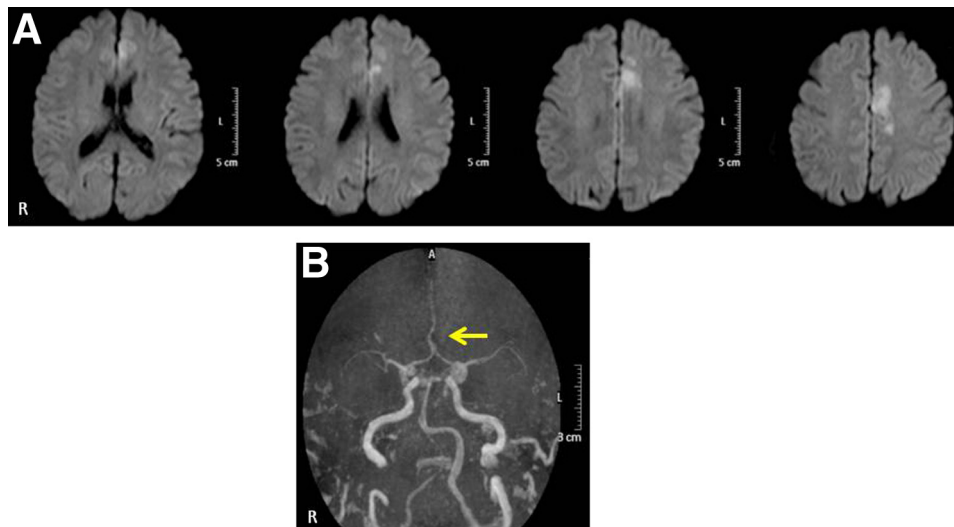


Figure 1. (A) Diffusion-weighted magnetic resonance (MR) image at onset. Hyperintense areas in the left superior frontal and cingulate gyri were seen. (B) MR angiography at onset, showing stenosis of the left A2 segment (arrow).

Three days after the onset, the patient was transferred to our hospital. Neurologic findings on admission to our hospital showed only slight right hemiparesis. The Barre' sign was positive in the right side. The grip power was 30 kg bilaterally. On day 4, he could walk by himself without any assistance. Echocardiography did not show any thrombus, and wall motion was normal. Transesophageal echocardiography did not show patent foramen ovale or atrial septal aneurysm. Using contrast injected under valsalva strain, presence of a right-left shunt was not confirmed. Three-dimensional computed tomography angiography showed stenosis of the A2 segment of the ACA. At 9 days after onset, we performed MR imaging for the second time and thin-section 3-dimensional MR cis-

ternography¹⁰: this technique can easily demonstrate the outer contours of the ACA without the influence of slow flow, turbulence, or thrombus in the inner lumen. MR angiography revealed double lumen finding. The dissection of the ACA was confirmed. MR cisternography also showed that enlarged outer contours with luminal stenosis of ACA led us to diagnose a dissection of this artery (Fig 2). Neurologic symptoms further improved in the following days. The patient was discharged after 20 days.

Discussion

The cause of ischemic stroke in this patient was the arterial dissection of left ACA but not decompression

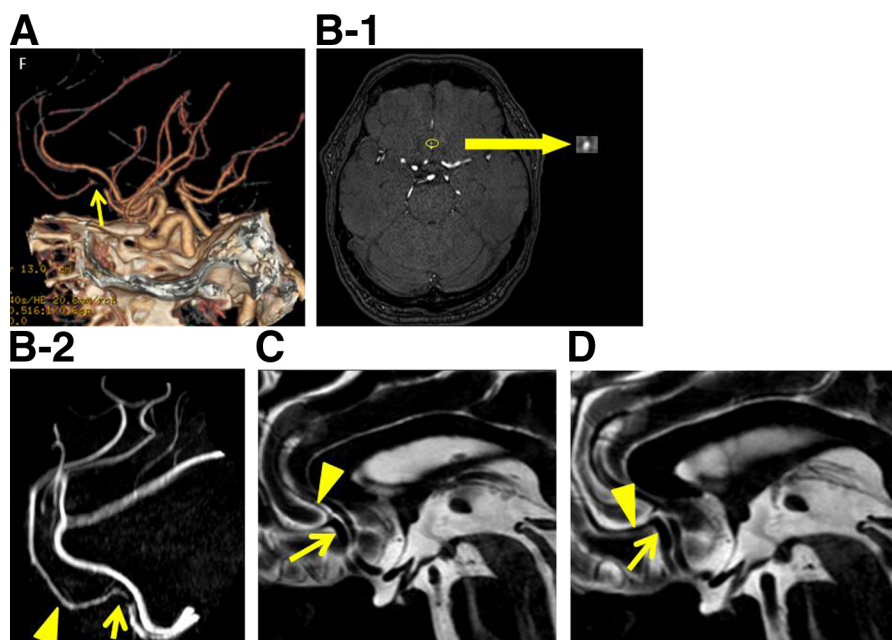


Figure 2. (A) Brain 3-dimensional computed tomographic angiography on day 20 after onset, showing stenosis of the left A2 segment (arrow). (B-1) Axial magnetic resonance (MR) angiography showed double lumen finding (circle), (B-2) MR angiography shows stenosis of the left A2 segment (arrow) and callosomarginal artery (arrowhead), but the pericallosal artery is not depicted because of occlusion. (C) MR cisternography shows fusiform dilatation of the outer contours of the left A2 (arrow) and collapse of the pericallosal artery (arrowhead). (D) MR cisternography shows fusiform dilatation of the outer contour at the narrowing segment of the left A2 (arrow) and callosomarginal artery (arrowhead).

illness. Decompression illness is caused by bubbles in blood or tissue during or after a reduction in environmental pressure.¹

Several reports have described arterial dissection,²⁻⁹ including dissection of the internal carotid artery in 5 cases, vertebral artery in 3 cases, and posterior inferior cerebellar artery in 1 case, during or after scuba diving. However, to the best of our knowledge, dissection of the ACA during scuba diving has not been reported. Dissections of cervical arteries are a common cause of stroke in young patients, most frequently because of abnormal neck movements, local trauma, or abnormalities of the vascular connective tissue or fibromuscular dysplasia.¹¹⁻¹³ Stroke-like symptoms during diving are usually a sign of dysbaric air embolism. If symptoms start immediately or shortly after surfacing, decompression sickness or pulmonary barotrauma is most often the cause. Some patients with dissection initially receive treatment for suspected decompression sickness but show further progression in the following days or do not respond to hyperbaric oxygenation therapy.⁴⁻⁶ A history of not responding to hyperbaric oxygenation or further evolution of neurologic symptoms may be indicative of arterial dissection.

The occurrence of arterial dissection does not depend on diving parameters such as duration, maximum depth, or speed of surfacing.⁴⁻⁸ Causes proposed to trigger vessel dissection in scuba divers are exaggerated head movements, also complicated by the necessity to bear the weight of diving gear, and the demanding physical exercise required during immersion/emersion. The other is related to the intracranial location of ACA. The cerebral falx impairs the ACA directory by the minor trauma, and ACA is stretched or sheared because of abrupt deviation of callosum.¹⁴ In Japan, the dissection accounts for 43% of ACA territory infarction.¹⁵ If neurologic symptoms occur during or after scuba diving, dissection of the arteries should be suspected, even if the patient does not have headache. Furthermore, MR cisternography can easily demonstrate the outer contours of ACA without the influence of slow flow, turbulence, or thrombus in the inner lumen. The combination of MR cisternography and MR angiography is useful for detecting ACA dissection.

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