Internal carotid artery dissection in stroke from scuba diving: A case report

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Gibbs III JW, Piantadosi CA, Massey EW. Internal carotid artery dissection in stroke from scuba diving. Undersea Hyperb Med 2002, 29(3):167-171-Although diving with compressed air is generally safe, neurological problems resulting from infarction in SCUBA diving are well known, including arterial gas embolism and decompression sickness (caisson’s disease, bends) involving the brain and spinal cord. While air gas embolism forms the overwhelming majority of causes for stroke in divers, internal carotid artery (ICA) dissection is another potential mechanism for central nervous system infarction in the setting of SCUBA diving. A 38 year-old female, who presented with complaints of headache, nausea, vomiting, and left sided hemiparesis after rapid ascent to the surface from a depth of 120 feet of seawater was initially treated for decompression illness in a hyperbaric chamber. Further neurological workup revealed a right ICA dissection. This case demonstrates the dangers of ICA dissection following rapid ascent to the surface from underwater and emphasizes an interesting presentation of stroke associated with SCUBA diving.

internal carotid artery dissection, SCUBA diving, stroke, occupational hazards

Carotid artery dissection is an uncommon cause for stroke and is predominantly observed in young to middle aged persons. Dissection of the ICA frequently occurs in association with abnormal neck movements or direct trauma to the neck (1). This case illustrates carotid dissection occurring in the setting of deep diving with rapid ascent to the surface.

CASE REPORT

A 38 year old, right-handed, white female had a rapid ascent to the surface after several hours of diving. She failed to pause with ascension from 120 feet of seawater (fsw) due to a presumed episode of confusion and disorientation. She was an experienced diver of over 7 yrs,
with many dives of greater than 100 fsw. She lost consciousness at 5-10 fsw, and then she slowly floated to the surface. She was immediately retrieved and received 100% supplemental oxygen by facemask. She revived within 1 min on the deck and experienced extreme nausea, vomiting, and headache without focal neurological features. Her diving partners contacted emergency helicopter services, and she presented to the hospital 4 hr later.

In the emergency department, she was vomiting and complained of a dull bilateral headache, but she was alert and produced a history. She complained of mild left sided upper and lower extremity weakness but no alteration in sensation. There were no visual symptoms or vertigo, but she complained of upper anterolateral right cervical neck discomfort. She denied past medical problems and did not take any medications. She denied alcohol, tobacco, or other drug use. Her family history included dementia.

She was pale, tired, and anxious. Vital signs were temperature 37.5°C, blood pressure 112/97 mm Hg, heart rate 84, and respiratory rate 20. The arterial oxygen saturation was 98% on 2 liters of supplemental oxygen. On neurological exam, her Folstein Mini-Mental State Exam score was scored 26/30; she made 1 error on delayed recall, was unable to copy a design, and was only able to accomplish 3 successive serial 7's. She demonstrated a mild non-fluent expressive aphasia with occasional word-finding difficulties and had intermittent difficulty repeating phrases. Cranial nerve examination exhibited a right gaze preference but full extraocular movements. Motor examination displayed a left pronator drift with strength 4/5 in the left biceps and hamstrings. Sensation was normal symmetrically, and reflexes were 2+ in all extremities, with no pathological reflexes present. Gait was normal based, and Romberg testing was negative.

Axial CT imaging of the brain revealed an acute right cortico-parietal infarct (Fig. 1A). She was immediately placed in recompression on a US Navy Table 6 with gradual decompression for presumed air gas embolism (AGE) or serious decompression sickness (DCS), her symptoms began to slowly improve over the subsequent days with complete resolution of her left sided hemiparesis, but her non-fluent expressive aphasia was intermittently detected. Axial magnetic resonance imaging (MRI) of the head with diffusion-weighting confirmed the presence of an acute infarct (Fig. 1B).

**Figure 1.** A. Axial CT of the brain without contrast. The axial CT image reveals a wedge shaped, focal area of low attenuation in the right parietal cortex consistent with a cerebral infarct. There is no evidence of an extra-axial
fluid collection, hemorrhage, or hydrocephalus. B. Axial diffusion and flair MRI imaging. Diffusion-weighted (i) and flair (ii) imaging exhibits an increased signal in the right parietal region consistent with recent, acute infarction that is correspondingly observed on CT imaging.

Magnetic resonance arteriography (MRA) (Fig. 2B) was performed to further investigate possible etiologies for her stroke as she was having continued right anterior cervical neck discomfort. MRA of the intra- and extracranial ICA vasculature revealed a decrease in the caliber of the right ICA at the base of the skull consistent with probable dissection with luminal hematoma (Fig. 2B). Therefore, a confirmatory computerized tomographic angiogram (CTA) was performed to more completely investigate the presence for a right ICA dissection (Fig. 2A). The CTA revealed a high cervical right ICA dissection with the greatest narrowing centered at the skull base with approximately 75% luminal reduction (Fig. 2A)

Figure 2. A. Axial CTA source image at the level of the skull base. Notice the approximate 75% narrowing at the level of the skull base of the right ICA (black arrow) in comparison to the left ICA (white arrow). B. 3-dimensional time-of-flight MRA of the intra- and extracranial carotid circulation. The focal signal abnormality at the level of the distal cervical ICA represents a mural hematoma as a result of a focal area of dissection (white arrows). The narrowing of the carotid artery at this level resulted in an acute infarct of the right parietal cortex.

The patient was started on intravenous heparin therapy for management of an ICA dissection and was converted to coumadin. She remained anticoagulated for 6 months, and then therapy was discontinued. Follow-up examination and CTA were normal (not shown).

DISCUSSION

Cervical ICA dissection is common etiology for cerebral infarction among young adults, accounting for 10-20% of ischemic stroke cases (1). Predisposing factors for spontaneous
dissection include fibromuscular dysplasia (2), cystic medial necrosis (3), Marfan’s syndrome (4), accumulation of mucopolysaccharides (5), and minor abnormalities of the internal elastic lamina (6). Mechanical forces involving penetrating injuries, chiropractic manipulation, hyperextension of the neck during athletic events, inadvertent intraoperative laceration, and percutaneous carotid angiography have also been implicated (7). However, the etiology of the majority of cases of ICA dissection remains unknown.

Serious brain pathology from SCUBA diving is generally attributable to the effects of air emboli to the brain, not complications such as ICA dissection and is treated by recompression (8). ICA dissection with SCUBA diving has been reported previously in only one instance (9). Most injuries reported in the setting of diving involve physiological problems from arterial gas embolism and decompression sickness (reviewed in 8-11). Although our patient began to improve during recompression therapy in the hyperbaric chamber, its therapeutic benefit in the setting of a carotid dissection is currently unknown. Recently, it was demonstrated that there is a high incidence of microemboli distal to the ICA dissection that may cause cerebral infarction (12). Of additional interest in our patient was the presence of a probable “crossed aphasia” which refers to a combination of aphasia and left hemiparesis. Right hemispheric language dominance ranges in incidence from 0.38-1.8% in the clinical population (13). Also, while her cerebral infarction was parietal in location, her neurological exam infarction had frontal aspects as she demonstrated a gaze deviation, cognitive changes, and aphasia that suggested associated edema into the frontal lobe. While local trauma to the carotid vasculature from the buoyant tank or from strap pressure was another possible cause of the ICA dissection, it is also possible that the lesion was spontaneous and linked to SCUBA diving only coincidently.

The typical patient with a carotid artery dissection presents with complaints of ipsilateral pain of the head, face, or neck, Horner’s syndrome (14), and cerebral or retinal ischemia several hours to days later (15). The presence of any 2 of these elements of this classical triad should initiate an investigation for ICA dissection (reviewed in 1). As both neck pain and cerebral ischemia were observed in our case, a search for an ICA dissection was necessitated. Ultrasound is useful in the initial assessment of patients thought to have an ICA dissection, but confirmatory tests are almost always currently indicated. Conventional angiography has been considered to be the gold standard in the diagnosis of ICA dissections; however, magnetic resonance techniques also provide high resolution of the ICA and can exhibit the intramural hematoma itself (16) (cf Fig. 2B). MRA is rapidly replacing arteriography as the gold standard in the detection of ICA dissection (16). A CTA was utilized in our patient to non-invasively confirm the presence of the ICA dissection but is normally not needed for the diagnosis of an ICA dissection (Fig. 2B).

Current therapy for carotid artery dissection to prevent future thromboembolic complications recommends heparin intravenously followed by coumadin for 3 to 6 months, unless there is an intracranial extension of the dissection (1). No randomized studies exist for the utilization of coumadin in ICA dissection but indirect evidence suggests the appropriateness of coumadin. Transcranial Doppler studies exhibit a high frequency of distal intracranial microemboli from the dissection (12). As many as 85% of patients may have excellent recovery, and reoccurrence of carotid arterial dissection is rare (5). Similarly, our patient’s follow-up CTA after 6 months of anticoagulation to assess the continued presence of the right ICA dissection was normal. Therefore, carotid dissection represents another cause for stroke and neurological complaints in divers that could potentially be underdiagnosed or overlooked by medical personnel.
REFERENCES