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The acute ischemic stroke following a water jump. Review of the literature

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Abstract

Dissection of the cervical arteries during water-related sports and recreations occurs rarely. The aim of our study was to present a patient with a carotid dissection following a water jump, and to examine the literature in this field. A 23-year-old male patient, 1.71 m tall and 73 kg in weight, experienced a temporarily loss of consciousness, amnesia for the event, neck pain, moderate aphasia, right hemiparesis, and supranuclear facial weakness a few hours after jumping into a swimming pool 2.5 m deep. Since an artery dissection was suspected, a computerized tomographic angiography was performed, which showed the left internal carotid artery dissection. Ultrasonography revealed an intramural mass of 14 mm in length, a larger diameter of this segment (7.8 mm), and a significant lumen narrowing and blood flow reduction. Magnetic resonance imaging confirmed a left internal carotid artery dissection, and showed a large ischemic region in the territory of the left middle cerebral artery. The patient had a relatively good outcome, due to antiplatelet and anticoagulant therapy, and was discharged with a moderate aphasia and a mild weakness of the right arm and leg. The carotid artery dissection following a recreational water jump seems to be very rarely reported.

Keywords: Carotid artery; Dissection; Ischemic stroke; Water sports; Water jump

1. Introduction

Water-related sports and recreations are of a great medical significance, since various bodies of water, including swimming pools, are visited, for example, by over 370 million people every year in the US alone, i.e. by persons who are at risk of certain injuries.[1] Similarly, only in France there are almost 2 million private swimming pools used by at least 6 million people.[2] Water-related accidents can be accompanied by injuries of the cervical spine, spinal cord, neck and head tissues, brain, and some other organs.[3-11] A subsequent dissection of the neck and head arteries in these instances is one of the least frequent injuries.[12-18]

Dissection of the cervical or cranial arteries is a rare event even in the general population, since it occurs in only 2.5-3.0 individuals per 100,000 people annually.[16] Dissection is usually related to an intimal tear and a resultant penetration of blood into the wall of an artery with a consecutive mural hematoma formation leading to lumen narrowing or occlusion, and occasionally a pseudoaneurysm or thrombus formation.[16,19,20] This can compromise the spinal or cerebral blood flow and cause ischemia of the cervical spinal cord, brain, retina or some peripheral nerves.[16,21] or hemorrhage.[4,20,22]

Dissections in general are relatively often caused by direct blunt or mild trauma and rarely by penetrating trauma under various circumstances, including water-related situations.[14-18] Since the latter events are rare, we decided to investigate in detail a possible mechanism of the injury, the computerized tomographic (CT) and magnetic resonance (MR) characteristics, and the clinical course and outcome of a young patient with a water-induced dissection of the internal carotid artery.

Cases report

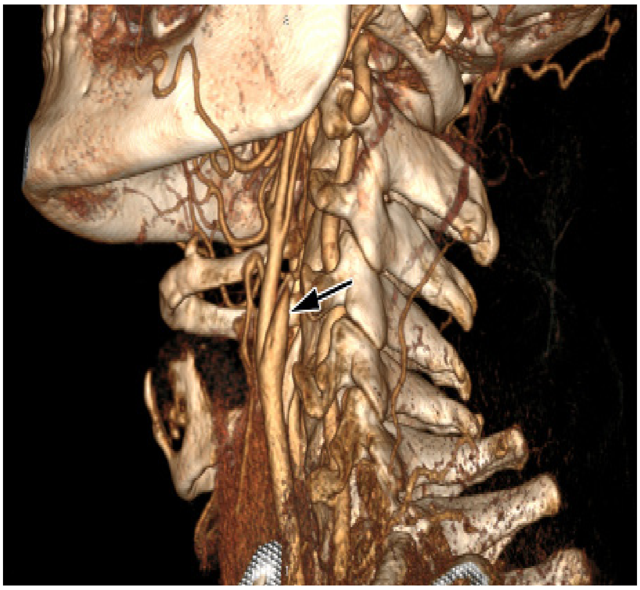
A 23-year-old right-handed male patient, 1.71 m tall and 73 kg in weight, was injured on his summer vacation in a foreign country by jumping into a swimming pool 2.5 m deep. After jumping head-first from a running start, he lost consciousness for a while but fortunately was rescued immediately from the water. Nevertheless, amnesia for the event was noticed thereafter, and a few hours later speech disorder appeared, a right limbs weakness, and neck pain. Examination in the Emergency Unit of the University Hospital confirmed aphasia and right hemiparesis, but a CT examination showed no disorder. However, MR imaging performed the next day revealed a large ischemic area in the left cerebral hemisphere and signs of brain edema. Since arterial dissection was suspected, CT angiography (CTA) was performed, which presented the left internal carotid artery dissection (ICAD). The patient received 50 mL of 20% Manitol, 100 mg of ASA, and 5000 IU of Tinzaparin.

He was transferred eight days later to our hospital, where a neurological examination revealed a right hemiparesis, mild facial weakness, moderate aphasia and dysphasia of both motor and sensory types, some reading and writing difficalties, and right-left orientation disorder, whilst other symptoms and signs were absent. The patient denied alcohol or drug abuse, and there were no signs of any chronic, congenital or genetic disease, although a connective tissue disorder could not be excluded. The laboratory analyses were normal. The cardiologic examination, including blood pressure, electro- and echocardiography, was unremarkable.

Ultrasonography showed normal vertebral arteries and the right internal carotid artery (ICA), whilst the left ICA presented an intramural mass of 14 mm in length, a larger diameter of this segment (7.8 mm as compared to 3.6 mm at other levels), a significant lumen stenosis and minimal blood flow. The CT examination revealed a large ischemic region in the left cerebral hemisphere, whilst the bones, muscles, and internal organs appeared normal.

MR brain and neck imaging was performed thereafter using both T1- and T2-weighted sequences, diffusion-weighted images (DWI), fluid-attenuated inversion recovery (FLAIR), and fat saturation. MR confirmed the left cervical and cavernous ICA dissection (Figs. 1 and 2), and a large infarction in the territory of the left middle cerebral artery (Figs. 3 and 4).

Fig. 1. MR angiography of the neck with contrast-technique TRIX showing a tapering occlusion of left ICA due to its dissection



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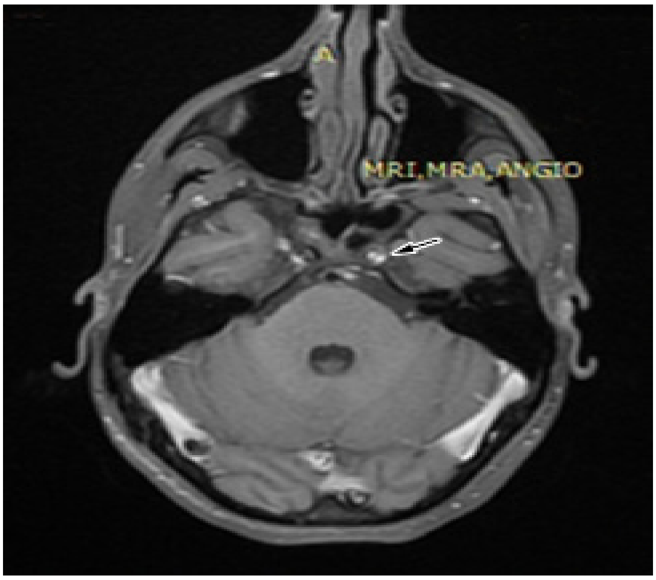
Fig, 2. Postcontrast axial 3D T1 FSPGR sequences presenting a double lumen of the cavernous segment of left ICA



Feg. 3. Axial DWI with restricted diffusion of a subacute infarct of the left MCA

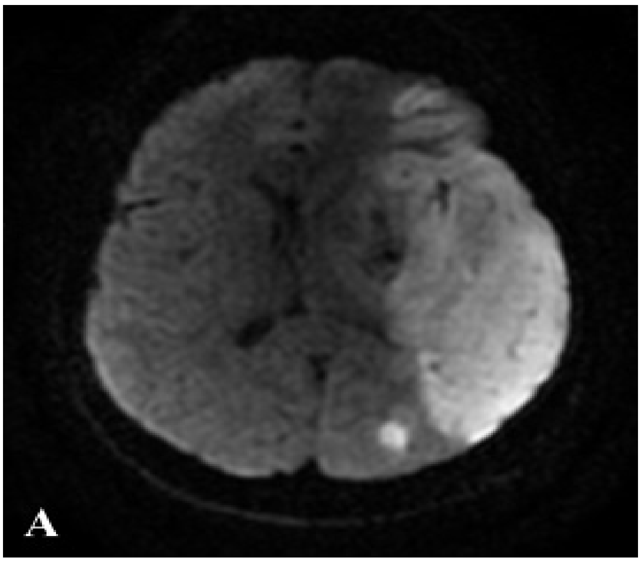
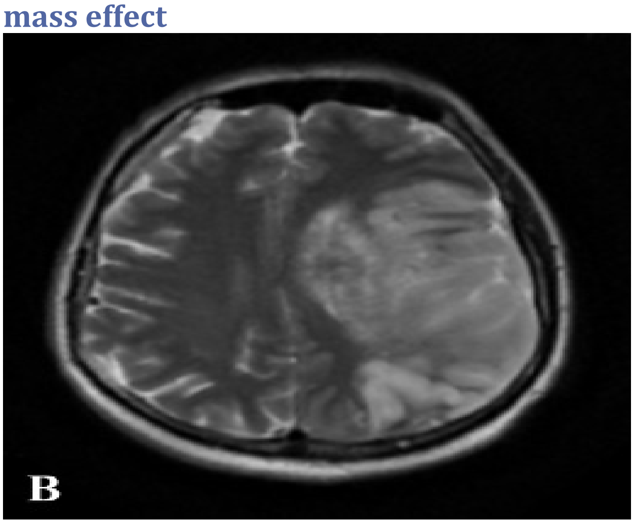


Fig. 4. Axial T2W/SE. A left MCA infarct with mass effect



The ischemic region comprised cortical areas of the precentral gyrus, and posterior portions of the superior, middle and inferior frontal gyri, as well as the postcentral, supramarginal and angular gyri, a part of the superior parietal lobule, the superior and middle temporal gyri, and the insulo-opercular region. The centrum semiovale and corona radiata at these levels were also affected, as well as most of the putamen.

The neuropsychological examination revealed moderate signs of Broca's, Wernicke's, and transcortical aphasia and dysphasia, and certain difficulties in right-left orientation. Some reading, writing, repetition and naming disorders were observed, e.g. phonemic and verbal paraphasia and paragraphia, and moderate comprehension disturbances of the spoken language. The patient continued with antiplatelet and anticoagulant therapy. Surgical or endovascular interventions were not indicated, since he was admitted with a definite lesion of the left hemisphere. He was discharged nine days later with a mild hemiparesis, but with a moderate aphasia. His clinical status was evaluated on the National Institutes of Health Stroke Scale (NIHSS) as 9, and as Rankin 2, respectively. After three months of therapy, the lumen of the left ICA was normal in size.

Discussion

Water-related injuries and epidemiology

Various types of water-related recreations and sports, e.g. swimming, diving, scuba diving, surfing, fishing, boating, sailing, water skiing, water sliding, waterboarding, speedboat or personal watercraft usage, etc., can be occasionally associated with accidental falling or jumping into water and consecutive spine injury, head or brain trauma, cervical arteries dissection, or some other traumatic lesions.[4-14,21,22-40]

Water accidents are relatively rare, so that in North America, for instance, they occur in up to 15,000 cases each year, mostly in 1.9-4.6 young individuals per 1 million population, with a peak at the age of 19.[37,38] Among them, diving injuries are commonly experienced by 6-10% of individuals,[3,11,26,37] whilst swimming injuries comprise 12% of 10,000 participants in this country. Between15% and 87% of water accidents occur in swimming pools, almost equally at public places and at home, and

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others on seaside, rivers, lakes or, rarely, torrents.[1,2,5,9,10,15,28,29,35,37,38,40,41] In spite of being of a relatively rare occurrence, the consequences of water-related injuries can be grave and even fatal occasionally.

Water jumps are mainly associated with injuries caused by striking the head on the bottom of a pool, or a river, lake, sea or ocean bed.[2,5,9,15] Some of them were reported as accidental or suicidal jumps from a bridge,[28,29,42-45] while yet others as recreational jumps into shallow or deep water,[14,26,28-30,34,36,38] including swimming pool jumps,[1,2,10,32] and the remaining ones as other types, e.g. water sliding, bobbling, "balconing," kitesurfing, sea waves and accidental motorcycle water jumps, landing of skydivers, etc.[9,15,17,24,30-34,46-49] As for swimming pool jumps, they are more often performed from the pool edges than from a springboard or platform.[2,7,10,17,32,.41,50]

Mechanism of injury

Axial force is commonly expressed in these cases, with subsequent cervical or other vertebrae fractures or dislocations, which can be accompanied by a spinal cord lesion, or the neck, head and brain injuries.[3,5-9,11] In fact, the neck and head regions are most frequently injured (37%).[1,32] Brain concussion is noticed sporadically. [4,5.10,14] The mentioned injuries have a lethal outcome in up to 8% of patients.[14,37,43]

The physical mechanism of a water jump was experimentally examined in certain animal models, especially regarding diving birds, some of which can enter the seawater surface at a speed of 24 m per second without a head or neck injury.[51] There are several phases while entering the water. First, the impact phase, which lasts from the moment of touching water to head submersion, when the axial drag is strong but the hydrodynamic forces are still weak. This is followed by the air cavity phase, i.e. a formation of an air layer around the head and neck, before the moment when the chest touches the water surface. In this phase, strong hydrostatic pressure force acts on the head and neck causing their deceleration, whilst the axial drag causes the rest of the body to continue descending with the resultant axial compressive load on the neck. Obviously, this phase has the greatest potential for neck injuries, which is avoided in these birds by a specific cervical spine anatomy, including its S-shaped appearance, and very strong neck muscles that are activated immediately before diving in order to stabilize the cervical spine and other neck structures.[51] The last or submerged phase, which closes the air cavity, occurs after the chest impacts the water.

However, the head and neck anatomy of birds and humans is significantly different.[51,52]. In fact, the head in humans is not so aerodynamic as in birds. In addition, it is much larger and with a much greater surface area, meaning that the force of water impact is much stronger than in birds, which can result in head injuries, loss of consciousness or in brain concussion.[4,5,10,14,34] In addition, the human cervical spine, in spite of the cervical lordosis, is not S-shaped, and the relatively weak human neck muscles are arranged in a different manner than in the mentioned birds.[51] Due to that, axial compression force causes a biomechanical instability of the cervical spine and trauma of the neck structures, including the cervical arteries, which is the basis for their injury.[6,12,14,15,17,18,40,41,46]

The axial compression load is transmitted from the head to the cervical spine through occipital condyles.[51] However, the force acts then not only on the spine, but also on other neck structures due to the load transformation into axial, lateral and anteroposterior share, with the addition of translational and rotational deformities.[53,54] It was estimated that the force velocity of 4.2–5.5 m/s produces a mean impact force of 5.3 kN, which is sufficient to cause neck injury in humans.[54] In addition, due to such an impact velocity, there is insufficient time for the activation of the neck muscles, so that their protective role on neck structures in humans is missing.

In the case of head-first water jumping, a great axial load is exerted onto the head and neck, especially if they are not protected by stretching the arms in front of the head. The force intensity primarily depends on the acceleration, especially on the height of a jump, but also of the head surface area, body mass, and angle of entry.[37,51] Our patient performed a jump from a running start, without any indication that he had touched the pool bottom. However, it is not known whether he performed a neck hyperextension or hyperrotation. In any case, he lost consciousness for a while, due to the intense force of the water impact on his head. However, the neck structures are even more susceptible to injury due to a relatively thin cervical spine, and to a weakness of the soft tissues and muscles, the latter being not activated immediately after entering the water.[51]

Arterial dissection

As for cervical arteries dissections, they occur extremely rarely in the mentioned situations (Table 1). **Table 1. Water-related arterial dissections**

Authors	Total No. of patients	Types of contact with the water	Percentages or No. of patients	Affected arteries	Ischemia
Nelson, 1995	1	Scuba diving	1	ICA	Yes
Konno et al, 2001	/	/	/	VA	/
Gibbs et al, 2002	/	/	/	ICA	/
Skurnik & Sthoeger, 2005	/	/	/	ICA	/
Bartsch et al, 2009	/	/	/	ICA	/
Kocyqit et al, 2010	/	/	/	PICA	/
Hafner et al, 2011	/	/	/	ICA	/
Chojdak-Łukasiewicz, 2014	/	/	/	VA	/
Fukuoka et al, 2014	/	/	/	ACA	/
Alonso Formento et al, 2016	/	/	/	ICA	/
Wasik et al, 2017	15	/	15	ICA	/
Furtner et al, 2006	/	Springboard diving	/	Both ICAs	/
Mohaghegh & Hajian, 2015	/	Swimming	/	VA	/
Brajkovic et al, 2013	/	Diving	/	ICA	/
Rosińska et al, 2015	/	Diving	/	ICA	/
Alawadhi, 2019	/	Diving	/	VA	/
Pego-Reigoza et al, 2005	/	Sea wave	/	ICA	/
Akbas et al, 2006	/	Waterslide	/	ICA	/
Alboudi etal,2018	/	Waterslide	/	VA	No
Lumsden et al, 2012	/	Whiplash	/	ICA	/
Fridley et al,2011	/	Wakenboarding	/	ICA	Yes
Schlem et al, 2015	26	Scuba diving	53.7%	ICA, VA	/
		Swimming	26.2%		
		Springboard	3.8%		
		Waterboarding	3.8%		
		Water skiing	3.8%		
		Surfing	3.8%		
		Kite surfing	3.8%		

For example, in a group of 64 patients involved in water accidents, only one had a dissection.[37] In other instances, only case reports were presented.[6,12,14,15,17.18,40,55-65] Schlemm et al.[17] enrolled 115 case reports from literature involving 190 sports-related dissections, and among them only 26 water-related cases. In the latter instances, injuries most often affected scuba divers[12, 17,18, 21,35,55-58,61-65] and swimming individuals,[1,2,4,5,10,24,32] whilst other injuries were extremely rare, i.e. a single individual in each type (Table 1). Our patient experienced a left carotid artery dissection. The left and right ICADs were reported to be almost equal in frequency, as was the involvement of the anterior circulation and the posterior circulation.[6,12,14,17,18, 40,41, 46,56,59,62.66-68] The latter circulation was much rarely involved than the former (Table 1). Dissections most often affect the cervical and rarely the intracranial or cervicocranial arteries. Bilateral carotid or vertebral dissections are very rare, i.e. less than 1%.[17,41,46,63] (Table 1).

As regards the mechanism, the axial (craniocaudal) compressive load acts not only on the head and the cervical spinal cord, but also on the soft tissue of the neck, due to the mentioned load transformation, but also due to a direct transverse neck hydrodynamic impact and deceleration during the air cavity phase with different force absorption by various neck tissues.[4.37,51] Due to that, direct hydrodynamic compression force is possible on the carotid and vertebral arteries.[51]

Symptomatology

As for the consequences of water-related dissections, Horner's syndrome was often manifested in these patients due to compression or ischemia of the sympathetic fibers within the carotid plexus or the cervical sympathetic trunk.[12,18,65] However, a lumen stenosis or occlusion, caused by a developed intramural hematoma, may lead to the spinal cord or brain hypoperfusion or ischemia. Transient ischemic attack (TIA) or ischemic stroke (IS) following a carotid or vertebral artery dissection were noticed in a relatively small number of water-induced injuries. [46,55,57, 63,67-70] In some of these patients the middle cerebral artery region was affected with a consecutive hemiparesis, facial palsy and speech disorders, which are occa sionally associated with headache and facial pain. Only one patient had ischemia in the anterior cerebral artery territory.[72] The remaining patients presented ischemia in the vertebral artery region, [14,17,21,40,56,60-62,73] and one in the PICA territory.[65] (Table 1). The latter patients manifested ataxia, dizziness, vertigo, drowsiness, vomiting, paresthesia, or facial or tongue weakness due to cerebellar or brain stem infarctions. Cortical blindness, as a consequence of ischemia of the posterior cerebral artery region, is a very infrequent event.[39]

The present patient

Our patient showed, firstly, right hemiparesis and supranuclear facial weakness, which improved during hospitalization. They were caused, as it is well known, by ischemia of the primary cortical motor area mainly of the precentral gyrus, and damage of the corresponding pyramidal and corticobulbar fibers within the corona radiata.[74] In addition, he also manifested speech disorders, that is, signs of a moderate Broca's, Wernicke's, and combined transcortical aphasia and dysphasia. He also showed certain difficulties in right-left orientation.

In general, Broca's aphasia occurs predominantly due to a lesion of the posterior part of the inferior frontal gyrus.[74] Wernicke's aphasia is mainly caused by a lesion of the posterior part of the superior temporal gyrus, and motor and sensory transcortical aphasia and dysphasia by damage of the fibers (mainly the peri-insular arcuate fascicle) interconnecting the frontal Broca's area, the temporal Wernicke's area, and the adjacent supramarginal and angular cortices.[74,75] The supramarginal cortex is crucial for reading, whilst the inferior temporo-occipital region is essential for writing.[76] Difficulties in right-left orientation can be explained by ischemia of the posterior parietal cortex.[77] As already mentioned, our patient was discharged with mild hemiparesis, but with a moderate aphasia due to damage of the dominant speech areas.

Conclusion

Sports or recreational water jumping without touching the bottom of a water body are rarely associated with arterial dissection. Our patient recounted with an internal carotid dissection following a recreational water jump due to which he experienced a serious ischemic stroke.

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