



Letter to the Editor

Vertebral artery loop in a case of recurrent transient global amnesia[☆]

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1. Introduction

Transient global amnesia (TGA) denotes a temporary anterograde amnesia and a mild reduction of retrograde episodic memory, while immediate recall ability is preserved. TGA is of sudden onset, lasts about 4–6 h, and typically no focal neurological findings are appreciated on the clinical exam. The medial temporal lobe supplied by the posterior cerebral circulation has been identified as the affected anatomical correlate, however, pathophysiology remains elusive [1]. A broad spectrum of conditions may trigger TGA, including emotional stress, migraine, or physical effort. On the other side, the annual risk for recurrence of TGA is exceedingly low even after repetitive exposure to triggers [2].

We report a woman who presented with recurrent episodes of TGA, each occurring exclusively after swimming. Imaging revealed an unusual anatomy of her right vertebral artery (VA). We speculate that repetitive head rotation while swimming may have resulted in transient posterior cerebral hypoperfusion due to abnormal VA anatomy.

2. Case presentation

A 74-year old woman went swimming freestyle vigorously for 20 min at the north-eastern coast of the United States. After she got out of the water, disorientation and anterograde as well as retrograde memory loss were noticed by her partner. Although her symptoms resolved within one hour, she was brought to the emergency room. Her history revealed a similar episode with transient memory loss following freestyle swimming two years prior to her current admission. The past episode had also resolved spontaneously within one hour. She states that when she crawls with maximum force, she rotates her head only to her left side for breathing.

On admission, physical and neurological examination was unremarkable. No focal motor or sensory findings were appreciated. Range of motion of her head was normal around all axes. An electrocardiogram was normal. An electroencephalogram (EEG) showed left

and right temporal polymorphic slowing suggestive of mild independent bitemporal cerebral dysfunction. Magnetic resonance imaging (MRI) revealed no abnormal diffusion restriction or susceptibility signal loss. CTA of the intra- and extracranial vessels demonstrated patent arteries and no aneurysms, dissections, or atherosclerotic changes were found. Of interest, all extracranial vessels were extraordinarily tortuous with the right vertebral artery (VA) forming a loop ranging from the foramen transversarium at C3/4 through the neural foramen into the spinal canal and back (Fig. 1). The right VA loop was trapped between an osteophyte and uncovertebral joint formation, narrowing the vessel's caliber. We suspected position-dependent compromise of blood flow in the VA as a contributing factor for recurrent TGA. Oral low-dose aspirin was administered, and the patient was advised to avoid excessive head rotation in particular while crawling freestyle. One year later, the patient reported no new episodes of TGA.

3. Discussion

TGA represents a common entity affecting 5 per 100,000 persons each year in the United States [1]. Differential diagnoses include transient ischemic attacks, arterial dissections, and temporal seizures which are all often accompanied by focal neurologic deficits. It is suspected that in TGA local hypoperfusion or electrophysiological abnormalities affecting memory relevant structures of Papez's circuit including the hippocampus, the parahippocampal gyrus, and the medial temporal lobe, result in memory impairment. Above structures are supplied by the vertebro-basilar system. Individuals with VA pathologies may therefore present with TGA [3].

The VA is subject to considerable mechanical forces when rotating the head. During head rotation, the ipsilateral atlantoaxial joint is fixed, whereas the opposite joint moves forward. Therefore, the VA contralateral to the direction of head rotation is exposed to the largest shear forces. Symptoms of vertebrobasilar insufficiency in that setting typically include vertigo, tinnitus, or drop attacks [4]. Five cases of vertebrobasilar insufficiency due to swimming documented with

[☆] Statistical analysis was not necessary for this case study and review of images or literature.

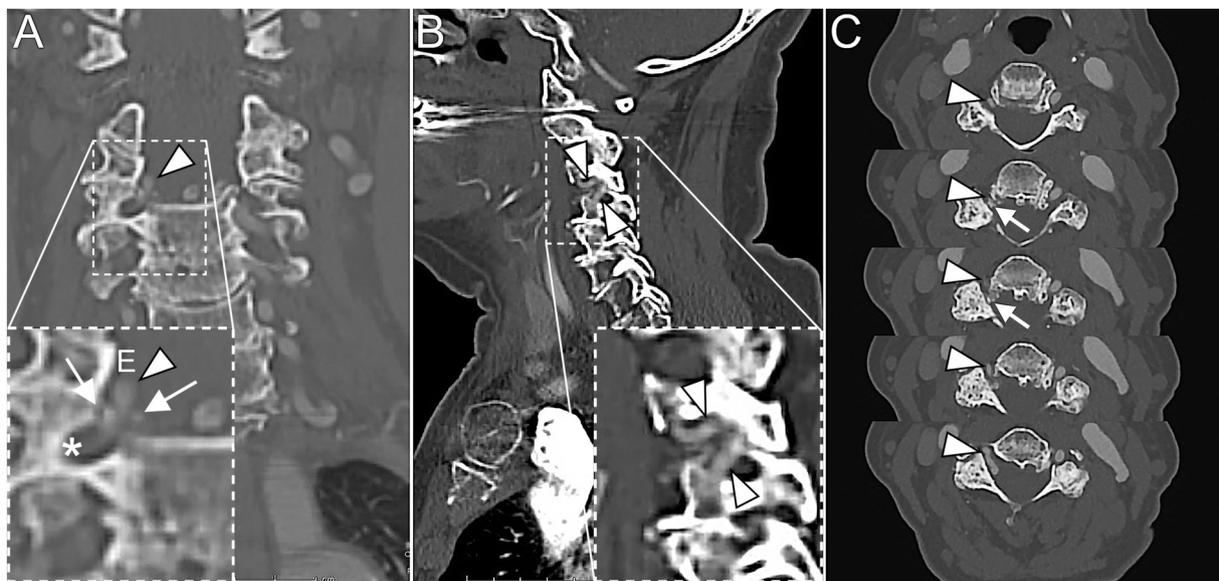


Fig. 1. Computed tomography angiography of extra- and intracranial vessels. Coronal (A), oblique (B), and axial (C; caudal to cranial from top to bottom) sections demonstrate a loop of the right vertebral artery extending from the vertebral canal through the neural foramen into the epidural space of the cervical spinal canal (arrow heads). On the coronal image (A) the vertebral artery (arrow heads) is seen entering the epidural space (E) through the neural foramen (asterisk) while its lumen is narrowed by an osteophyte and uncovertebral joint formation (arrows in insert). On the oblique view (B) the loop is best seen as it is captured in a single longitudinal section. On the axial sections the compression of the vertebral artery (arrow heads) by osteophytes (arrows) is noted as it enters through the neural foramen (first three cross sections from top) while the caliber appears normal as the vessel forms the loop and exits the neural foramen (bottom two cross sections).

imaging studies have been reported in the medical literature, all in the setting of vertebral dissection [5]. An unusually tortuous right VA compromised by degenerative cervical spine changes was found in our patient presumably giving rise to position-dependent posterior cerebral hypoperfusion. Osteophytes and degenerative changes were previously described to compress the VA during head rotation giving rise to clinical symptoms of vertebrobasilar insufficiency [6]. Reproducibility of the syndrome on two occasions lends further support to our hypothesis but in the absence of direct demonstration of the underlying pathophysiology some diagnostic uncertainty remains. We cannot provide imaging evidence of reduced cerebral perfusion during head turn, and we cannot exclude that head rotation may also have compromised blood flow in the tortuous left VA aggravating the cerebral hypoperfusion. EEG findings were consistent with bitemporal pathology favouring a vascular event but we were not able to rule out a seizure as a contributing factor.

Our case provides a possible structural basis for recurrent TGA. Even in the presence of a structural abnormality prognosis may remain favourable [7].

Author contributions

Philipp Karschnia, study concept and design, analysis and interpretation of data, drafting of manuscript.

Robert K. Fulbright, analysis and interpretation of data, revising of manuscript.

Joachim M. Baehring, study concept and design, analysis and interpretation of data, revision of manuscript.

Co-investigators

None.

Authors disclosure

Philipp Karschnia Mr. Karschnia reports no disclosures.
Robert K. Fulbright Dr. Fulbright reports no disclosures.
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Declarations of interest

None.

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