Vertebral Artery Injury and Cerebellar Stroke While Swimming: Case Report

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SUMMARY A twenty-five year old woman suffered the acute onset of dysequilibrium followed by headache, nausea, vomiting, vertigo, and slurred speech while swimming. Brain imaging revealed a right cerebellar infarct. Intravenous digital subtraction angiography showed a hypoplastic right vertebral artery and focal narrowing of the dominant left vertebral at the level of the C1-C2 junction. The patient was treated with aspirin and dipyridamole and immobilized for two weeks. She achieved almost complete recovery. Repeat angiography showed resolution of the left vertebral artery defect. Other cases of posterior circulation infarction associated with head turning during sports and ordinary activities are reviewed.

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OBSTRUCTION TO VERTEBOBASILAR PERFUSION resulting from cervical rotation was first documented during the early part of this century in the German literature.1 There have since been numerous reports of cerebral, cerebellar, and spinal cord infarction associated with head movement. The most common setting is chiropractic manipulation of the neck; three hundred sixty such cases have been registered with the Stroke Council of the American Heart Association2 and thirty-eight documented in the literature.3-4

Sports and ordinary activities involving head turning have also been associated with vertebrobasilar insufficiency (table 1). We report the first case of posterior circulation stroke during swimming.

Case Report

A twenty-five year old lefthanded woman was transferred from another hospital with headache, vomiting, slurred speech, and loss of balance.

The previous day she had been swimming freestyle in a pool with her face kept continuously rotated approximately ninety degrees to the left for about an hour. She was forced to make an abrupt change in direction to avoid a child and suddenly felt as if an undercurrent were pulling her down and to the right. Nauseated, she struggled to poolside, suffered a brief episode of vertigo, and vomited. She had a full cervico-occipital ache, slurred speech, and right-sided clumsiness with loss of equilibrium. Over the subsequent twenty-four hours her speech and balance improved, but her headache worsened and she vomited repeatedly.

The general medical examination was normal. The patient’s speech was dysrhythmic and side-to-side alternating movements of the tongue were slow and uneven. There was no bulbar weakness. She held her head tilted approximately thirty degrees to the right and had a broad-based, unsteady gait with a tendency to fall to the right. There was right arm and leg dysmetria and dysdiadochokinesia.

Computerized transaxial tomography (CT) one day after onset revealed a low-density area in the right cerebellum extending anteriorly and to the midline which did not enhance following intravenous contrast (fig. 1). Nuclear magnetic resonance imaging two days later showed involvement of the right cerebellar hemisphere without primary injury to or impingement upon the brainstem (fig. 2). Plain films and CT of the cervical spine were unremarkable. Intravenous digital subtraction angiography (DSA) demonstrated focal narrowing of the left vertebral artery at the C1-C2 level; the right vertebral was hypoplastic (fig. 3).

The patient was kept on strict bedrest in a hard cervical collar for two weeks and maintained on aspirin 80 mg per day and dipyridamole 25 mg three times a day. Over this time her neurologic signs resolved almost completely, the residua being a head tilt and gait unsteadiness when fatigued. Repeat DSA four weeks after the onset of symptoms revealed resolution of the defect in the left vertebral artery (fig. 4).

Discussion

Swimming involves a variety of head movements which could lead to compromise of vertebral artery flow. Our patient was swimming a modified crawl in which she kept her head in continuous rotation to the left at angles greater than those necessary to produce vertebral artery obstruction in the cadaver,1,11 during arteriography,12 and intraoperatively as measured by an electromagnetic flowmeter.13 Dissection of the extracranial internal carotid artery during swimming has recently been encountered by the present authors and reported by Luken, et al.14

That clinical sequelae of head rotation are rare suggests that predisposing factors are necessary for ischemia to occur. Various authors have stressed the association of vertebral artery anomalies, tortuosity, and atherosclerosis as well as the duration and force of head rotation,12,15,16 but these have not been consistent features.3 In the present case the vertebral artery ipsilateral to the side of cerebellar infarction was hypoplastic while the contralateral, dominant vertebral was focally narrowed at the C1-C2 junction. The patient made a sudden forceful movement in an unspecified

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direction that was superimposed on a prolonged rotation to the left.

This is the first case in which DSA has been used to document vertebral artery injury associated with head rotation. We chose DSA because vertebral arteriography in patients with recent infarction carries a greater risk of central nervous system complications relative to non-vertebral arteriography and because DSA is associated with a low risk of procedure-related CNS complications. DSA images the posterior circulation well and should prove useful in demonstrating lesions at all levels of vertebral artery injury associated with head rotation — C1-C2, C6, and the occipitoatlantal joint.

This is also the first case in which resolution of such a lesion following treatment with anti-platelet agents and immobilization has been angiographically documented. We administered aspirin and dipyridamole rather than heparin because focal neurologic symptoms and signs had not progressed over the twenty-four hours prior to admission. While clinical data are limited and therapeutic approaches must be individualized, in selected cases anti-platelet therapy may be beneficial.

FIGURE 3. DSA showing focal narrowing of the left vertebral artery at the level of the C1-C2 junction (arrow). The right vertebral artery is hypoplastic.
### Table 1

<table>
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<th>Vascular Pathology</th>
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<td>Gymnastics (5) (parallel bars)</td>
<td>18/M</td>
<td>spinal cord infarction, lower cervical</td>
<td>anterior spinal artery occlusion</td>
<td>neurological examination and arteriography</td>
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<td>Calisthenics (5)</td>
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<tr>
<td>Calisthenics (6) (stretching exercises)</td>
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<td>spinal cord infarction, lower cervical</td>
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<td>Yoga (5)</td>
<td>28/F</td>
<td>left cerebellar infarct</td>
<td>left vertebral artery narrowed at C1-C2</td>
<td>neurological examination and arteriography</td>
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<tr>
<td>Yoga (7)</td>
<td>25/M</td>
<td>left dorsolateral medullary syndrome, onset 2 hrs after at exercise:</td>
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<tr>
<td></td>
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<td>right dorsolateral medullary syndrome, evolving over minutes:</td>
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<td>Archery (8)</td>
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<td>right vertebral artery spasm aneurysm distal to spasm</td>
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<td>Driving (9)</td>
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<td>Driving (9) (in reverse)</td>
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<td>right vertebral artery occlusion at C1-C2 and left vertebral artery stenosis at foramen magnum</td>
<td>neurological examination and arteriography</td>
</tr>
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### References


**Figure 4.** DSA showing resolution of the left vertebral artery defect (arrow) illustrated in Figure 3.


17. Wishart D: Complications in vertebral angiography as compared to non-vertebral cerebral angiography in 447 studies. AJR 113: 527-537, 1971


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