Risk of Stroke due to Spontaneous Cervical Artery Dissection

Jianchang Chen¹, Xiang Zhou¹, Chao Li² and Bernard M.Y. Cheung²

Abstract

Although spontaneous cervical artery dissection (SCAD) is generally a rare contributor to a stroke, this condition triggers a considerable percentage of the strokes that are observed in young to middle-aged patients. We herein report the findings of a patient who presented with a stroke and a severe headache. A diagnosis of SCAD was made following a series of examinations. The patient had high-grade stenosis in the cervical artery and received carotid angioplasty along with stenting. A diagnosis of SCAD should be suspected if a patient who is less than 50 years of age presents with a stroke and a severe headache, and CT or an MRI scan rules out hemorrhage.

Key words: stroke, SCAD

(Intern Med 52: 2237-2240, 2013)
(DOI: 10.2169/internalmedicine.52.0109)

Introduction

Spontaneous cervical artery dissection (SCAD), which is also known as spontaneous carotid artery dissection, is a non-traumatic separation that occurs in the wall of the internal carotid arteries or the vertebral arteries. This condition results in either stenosis or an aneurysm of the vessel. SCAD accounts for only about 2% of all ischemic strokes (1, 2). An intense headache may occur in up to 75% of the patients with SCAD, as the cervical arteries are heavily innervated by pain fibers (3). We herein report the case of an SCAD patient who presented with an intense headache.

Case Report

A 48-year-old man presented to the Emergency Department (ED) of The Second Affiliated Hospital of Soochow University with an intense headache that had persisted for 6 hours. The headache was located around the left-side frontotemporal region and was affecting the ipsilateral parieto-occipital region. His medical history revealed that he had been hypertensive for the previous 5 years and had experienced paroxysmal atrial fibrillation (AF) for the previous 3 years. He was not being regularly treated or monitored for either the hypertension or the arterial fibrillation. There was no history that would be suggestive of either head or cervical trauma, and there was no indication of any previous infection.

Upon examination, our patient did not have a fever, but he did appear pale. His blood pressure was 140/80 mmHg and his heart rate was approximately 86 per minute with absolute arrhythmia. The amplitude of his first heart sound was variable. An examination of the nervous system showed that he had nominal aphasia, right homonymous hemianopsia, acalculia and difficulty reading. AF was confirmed on the electrocardiogram (ECG). Magnetic resonance imaging (MRI) demonstrated a left-sided occipitotemporal watershed infarction (Fig. 1). Transesophageal echocardiography (TEE) did not reveal any thrombi in the left atrium or the left auricle. Digital subtraction angiography (DSA) excluded atherosclerosis in our patient and showed evidence of left internal cervical artery dissection (ICAD) that arose approximately 1.5 cm distally to the carotid bulb and extended for 6.2 cm. An intramural aneurysm that was 15.6 mm in length was noted in the origin of the internal carotid artery along with high-grade stenosis (75%) that was also demonstrated by

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Received for publication January 27, 2013; Accepted for publication May 19, 2013
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Figure 1. MRI showing the left-sided occipitotemporal watershed infarction.

Figure 2. DSA showing the cervical artery before and after the stent placement for SCAD.

In the general population, SCAD is an uncommon cause of strokes. However, SCAD accounts for about 25% of strokes in patients less than 45 years of age (4). SCAD leads to an accumulation of blood within the artery layers, and can eventually generate an intramural aneurysm which may spread along the vessel walls (5). The resulting aneurysm may worsen existing stenosis and lead to ischemic stroke. Recent studies have demonstrated through imaging that strokes in patients with cervical artery dissection were most frequently associated with artery-to-artery embolization (6).

One of the most common risk factors for SCAD is hypertension. Hypertension, which is a global health burden, is increasingly prevalent among young people. As hypertension is a potent risk factor for stroke, and the control of hypertension in the Chinese general population is not good, hypertension is therefore the major cause of stroke in China (7). A recent case-control study suggested that hypertension is as much of a risk factor for SCAD-related strokes as it is for other causes of stroke such as cerebral atherosclerosis (8). Our patient had a 5-year history of hyperten-

Aspirin and clopidogrel were immediately administered in the Emergency Department. However, his condition did not improve even with antithrombotic therapy. Therefore, carotid angioplasty and stent placement were undertaken to relieve the high-grade stenosis in the origin of the internal carotid artery. After angioplasty, DSA showed that the aneurysm had completely disappeared and the state of hypoperfusion in the distal brain territory was significantly relieved (Fig. 2, right). The initial neurological symptoms disappeared almost completely within 30 days of the stenting procedure, and the symptoms of stroke did not reoccur.

Discussion

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Our patient presented with a headache and neurological deficits (Table 1). These symptoms indicated a stroke, which could be hemorrhagic, so CT or MRI was indicated. As hemorrhage was immediately ruled out by MRI, we suspected a cerebral infarction that might be related to AF. TEE excluded left-atrial appendage thrombus. DSA showed ICAD. In view of the absence of a history of head trauma, the diagnosis of spontaneous internal artery dissection was made.

Stenting was performed as the specific treatment for our case. Previous studies have indicated that cervical artery dissection is associated with connective tissue abnormalities, suggesting that there may be a structural defect in the extracellular matrix of the arterial wall (9). Although there was a risk in performing carotid angioplasty and stenting in these fragile vessels, antithrombotic therapy alone was not able to reverse the high-grade stenosis that we observed. When there is no recovery following the administration of antithrombotic therapy, carotid angioplasty and stenting should be considered for cases of SCAD in order to avoid prolonged ischemia (10).

The prevalence of SCAD is underestimated (22), and the diagnosis is frequently made at the postmortem examination (1). Although headache, neck pain, a transient loss of vision, Horner’s syndrome, stroke and other ischemic signs are commonly presenting symptoms and signs in SCAD patients, these are too non-specific for a definitive diagnosis (Table 2). Furthermore, patients who are asymptomatic or have only minor transient symptoms could potentially remain undiagnosed. Aside from the aforementioned symptoms and signs, the risk factors for SCAD should also be

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**Table 1. Characteristics of the Patient with Spontaneous Cervical Artery Dissection**

<table>
<thead>
<tr>
<th>Presentation</th>
<th>History</th>
<th>Symptoms and signs</th>
</tr>
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<tbody>
<tr>
<td>6-hour intense headache</td>
<td>HT for 5 years</td>
<td>Persistent left frontotemporal headache</td>
</tr>
<tr>
<td></td>
<td>Paroxysmal AF for 3 years</td>
<td>BP: 140/80 mmHg</td>
</tr>
<tr>
<td></td>
<td>HT and AF not regularly treated or monitored</td>
<td>HR: 86 per minute, irregular</td>
</tr>
<tr>
<td></td>
<td>No head or cervical trauma, or previous infection</td>
<td>Nominal aphasia, right-sided homonymous hemianopsia, agraphia, and dyslexia</td>
</tr>
</tbody>
</table>

**Investigations:**
- ECG: AF
- MRI: Left-sided occipitotemporal watershed infarction
- TEE: No thrombi in the left atrium and auricle
- DSA: Left ICAD and intramural aneurysm in the origin of the internal carotid artery with high-grade stenosis


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**Table 2. A Summary of Other SACD Reports in Asia 2007-2013**

<table>
<thead>
<tr>
<th>Gender</th>
<th>Age</th>
<th>Region</th>
<th>Clinical Presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>74</td>
<td>Japan</td>
<td>Presented with 1h dyspnea and dysphonia. Imaging studies showed a right cerebral hemisphere infarction and dissection of the brachiocephalic artery to the right ICA.</td>
</tr>
<tr>
<td>Male</td>
<td>49</td>
<td>Japan</td>
<td>Presented with disturbance of consciousness and left hemiparesis. MRI revealed cortical infarction. MRI showed right ICAD.</td>
</tr>
<tr>
<td>Male</td>
<td>40</td>
<td>Taiwan</td>
<td>Presented with acute vertigo, vomiting, and right facial numbness after a bout of sneezing. Neurological examination showed right Horner’s syndrome and facial hemi-anesthesia.</td>
</tr>
<tr>
<td>Female</td>
<td>34</td>
<td>Japan</td>
<td>Presented with sudden onset of severe headache. There was a history of high blood pressure and smoking.</td>
</tr>
<tr>
<td>Male</td>
<td>58</td>
<td>Taiwan</td>
<td>Presented with a change in consciousness with left hemiplegia. Angiography showed right mid-CCA extending to ECA and ICA with ICA occlusion.</td>
</tr>
<tr>
<td>Female</td>
<td>64</td>
<td>Korea</td>
<td>Presented with drowsiness. CT confirmed a subarachnoid hemorrhage with hydrocephalus. Angiogram showed a small aneurysm in the right ICA.</td>
</tr>
<tr>
<td>Male</td>
<td>65</td>
<td>Japan</td>
<td>Presented with headache and transient visual disturbance in the right eye. 3D-CT showed an intimal flap in the right petrous internal carotid artery.</td>
</tr>
<tr>
<td>Male</td>
<td>40</td>
<td>Japan</td>
<td>Presented with sudden decrease of vision in the right eye. Cerebral MRI and cerebral angiography showed evidence of ICAD.</td>
</tr>
<tr>
<td>Male</td>
<td>56</td>
<td>Japan</td>
<td>Presented with disturbance in consciousness and left hemiparesis. MRA demonstrated the right ICAD.</td>
</tr>
<tr>
<td>Male</td>
<td>42</td>
<td>Taiwan</td>
<td>Presented with headaches in the left retro-orbital and temporal areas. MRA provided evidence of ICAD.</td>
</tr>
<tr>
<td>Male</td>
<td>38</td>
<td>Japan</td>
<td>Presented with acute-onset left-sided chest pain, dizziness, and vomiting. CT provided evidence of ascending cervical artery aneurysm and dissection of vertebral artery.</td>
</tr>
</tbody>
</table>

ICA: internal carotid artery, CT: computerized tomography, ICAD: internal carotid artery dissection, MRI: magnetic resonance imaging, CCA: common carotid artery, ECA: external carotid artery, MRA: magnetic resonance angiography
taken into consideration when making a diagnosis. In the past, atherosclerosis has not been considered as an important cause of SCAD. In recent years, however, more attention has been paid to the potential influence of the traditional risk factors for atherosclerosis when a diagnosis of SCAD is made. Atherosclerosis has been shown to be associated with SCAD, although the underlying mechanism has not yet been elucidated (23). It is possible that SCAD patients who have cerebral atherosclerosis that may compromise their cerebrovascular circulation may be more likely to present with neurological deficits. There is evidence that the traditional risk factors for stroke, such as hypertension, are more commonly found in SCAD patients (8). Therefore, in patients with traditional risk factors for stroke, the possibility of SCAD should be considered if the patient has one or more symptoms or signs that are suggestive of SCAD.

In conclusion, the clinical signs of SCAD can be nonspecific. Clinicians should therefore keep in mind the possibility of a diagnosis of SCAD if a patient who is less than 50 years of age presents with a severe headache, and either CT or an MRI scan has ruled out hemorrhage.

The authors state that they have no Conflict of Interest (COI).

References


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