Abstract

Background—Syncope is commonly worked up for carotid stenosis, but only rarely attributed to it. Considering paucity of such cases in literature, we report a case and discuss the pathophysiology.

Design/methods—We report a patient with high-grade bilateral severe internal carotid artery (ICA) stenosis who presented with syncopal episodes in the absence of stroke, orthostatic hypotension, significant cardiovascular disease, or vasovagal etiology. We reviewed all literature pertaining to syncope secondary to carotid stenosis and other cerebrovascular disease.

Results—A 67-year-old man presented with two brief syncopal episodes. History and physical examination was not suggestive of seizure or vasovagal syncope. Other workup was negative for any stroke or syncope secondary to cardiac or vasovagal etiology. Magnetic resonance angiography (MRA) revealed bilateral ICA severe stenosis. This was confirmed by transfemoral carotid vessels angiography. Internal carotid angioplasty and stenting was performed on one side. After this, the patient remained asymptomatic. After one month, carotid endarterectomy (CEA) of contralateral side was performed. Patient remained symptom free after that. On review of literature, we identified only 12 cases of syncope attributable to carotid stenosis and reviewed 24 cases attributable to other cerebrovascular disease.

Conclusion—Syncope secondary to carotid stenosis, especially in the absence of any focal ischemic events is rare. It can only be expected in those patients who have bilateral hemodynamically significant carotid disease, which is unlikely in the absence of any focal ischemic events.

Keywords

Carotid artery stenosis; carotid stenting; carotid endarterectomy; magnetic resonance angiogram; syncope

BACKGROUND

Syncope is common medical problem with reported incidence of 6.2 per 1000 person-years of first time events in the United States [1]. Although common etiology for syncope is vasovagal (21.2%) or cardiac (9.5%) or orthostatic (9.4%) and for 36.6% the cause may be unknown [1]. In patients with negative workup for these etiologies, cerebrovascular disease workup is considered, which may be limited to carotid ultrasound [2].

High-grade stenosis or near occlusion of bilateral ICA is associated with stroke and transient ischemic attack (TIA) due to distal embolism or cerebral hypoperfusion. Bilateral severe stenosis of ICA can potentially cause syncope due to wide spread bilateral cerebral hypoperfusion. This phenomenon is very rare and minimal literature is reported on such patients.

CASE DESCRIPTION

A 67-year-old man with history of hypertension presented with sudden onset of syncope, while he was standing when he suddenly collapsed and hit his head. He woke up a few seconds later on the floor with bleeding from the back of head. After he walked a few steps, he collapsed again. He denied any symptoms of lightheadedness, dizziness, chest pain, or shortness of breath preceding these events. Both times he regained consciousness spontaneously after a few seconds without any weakness. On arrival to the emergency room, his heart rate was 78 beats per minute and systolic blood pressure was 136/65 mmHg. Some laceration and minimal bleeding was noticed on right occipital scalp that was repaired at ER. Electrocardiogram and telemetry monitoring was negative for any arrhythmias. Computerized tomography (CT) scan of the head was normal for stroke or any other
significant pathology. Patient also sustained right shoulder abrasion. Serum troponins were not elevated. Postural blood pressure measurements were negative for any suggestion of orthostatic hypotension. He had active glaucoma and past history of paroxysmal atrial fibrillation but was symptom free for the last three years and is not taking any anticoagulation medication. Patient past history is also positive for coronary artery spasm myocardial infarction four years with normal coronary arteries on coronary angiography and catheterization.

Transthoracic echocardiogram demonstrated normal left ventricular systolic function with slight mid diastolic abnormality. Bilateral carotid ultrasound revealed right internal carotid artery (ICA) peak velocity of 4.24 m/s and left ICA peak velocity of 7.02 m/s and was interpreted as bilateral ICA stenosis of ≥ 70%. The plaque was noted to be predominantly smooth soft with a thin calcific component. Magnetic resonance angiogram (MRA) also demonstrated a short segment of 80% stenosis of proximal right ICA and two short segments of about 60% stenosis involving the left ICA. Left vertebral artery was dominant and right vertebral artery was hypoplastic. Transfemoral carotid angiography confirmed near occlusion of right ICA and > 90% stenosis of left ICA.

In the absence of any other significant cause, the bilateral cerebral ischemia was considered to be the possible cause of syncopal episodes. Left ICA angioplasty and stenting with distal protection was performed to establish revascularization. The procedure was uneventful and blood pressure remained stable both intra- and postoperatively. There was no residual stenosis. Patient was free of syncopal symptoms after revascularization of left ICA. Postprocedure examination revealed no focal neurological deficits and patient was continued on aspirin and clopidogrel postprocedure. It was followed by elective carotid endarterectomy of right ICA one month later. It was also uneventful and patient remains stable. Patient was free of any syncopal symptoms on clinical follow-up at one month, two months, four months and over the phone follow-up after two years.

DISCUSSION

Cerebral autoregulation maintains blood flow to the brain in case of drop in arterial blood pressure. Richard P. White [7], in his study demonstrated that in patients with carotid artery stenosis, this dynamic cerebral auto regulation is impaired [7]. In this study, the cerebral auto regulatory index (ARI) was found to be significantly reduced in middle cerebral artery ipsilateral to a stenosed/occluded carotid artery; mean±SD 3.3±2.2, compared with 5.9±2.1 ipsilateral to nonstenosed artery in patients (p<0.002) and 6.3±1.1 for the normal controls (p<0.0001). The study demonstrated that the ARI was significant progressively reduced with increasing degree of carotid stenosis (ANOVA p=0.0016, Scheffé’s test p<0.05 for 80% to 95% compared with <60%). However, even with severe carotid stenosis (>80%) in some cases, ARI was normal (5/23), and in only a minority of cases, it was severely impaired (7/23). Interestingly, after CEA or angioplasty, there was a significant improvement in ARI in eight subjects, which increased from 4.0±2.3 to 5.9±2.1 (p<0.02) [7]. In all subjects in whom ARI was
outside the normal range preoperatively it returned to normal postoperatively [7].

Parts of the brain that controls consciousness are reticular formation and its ascending pathway [3]. Syncope or temporary loss of consciousness resulting from isolated bilateral severe carotid artery disease is not very well studied or recognized. There have been multiple reports associating posterior circulation insufficiency with syncope. Kubick and Adam in their study were able to find that among 18 patients whose autopsy revealed basilar artery occlusion, seven had history of syncope during hospitalization [4]. In another study, Patrick et al correlated six patients with syncope out of 39 patients with infarction in the vertebrobasilar territory [5]. Also, Kashiwazaki et al [6] reported nine male patients aged 59–83 who presented with syncope with concurrent occlusive carotid artery disease. Unlike our patient, five patients in their study were associated with dehydration or hypotension. Additionally, MRI demonstrated fresh cerebral infarction in the watershed zone that was absent in our patient. In this study, four patients who had developed in appropriate collateral circulation were treated surgically (CEA or superficial temporal to MCA), while other five were treated medically. All nine patients did not report any further episode of syncope after treatment, thus making it debatable to compare and contrast the medical versus surgical treatment options [6]. Subclavian steal phenomenon or vertebral basilar insufficiency caused by the reverse flow into the right vertebral artery are also two possible explanation in our patient. However, there is a need to study the incidence of syncope as presenting symptoms in isolated bilateral severe stenosis or occlusion of internal carotid artery diseases on a larger sample of patients.

Although association of posterior circulation insufficiency with syncope is well established, syncope resulting from isolated bilateral severe carotid artery disease is not very well studied or recognized. On literature search, we found only few previous reports of bilateral ICA stenosis as cause of syncope. Yanagihara et al [3] reported three patients with brief loss of consciousness with coexisting angiographic evidence of bilateral severe stenosis or occlusion of ICA. [3] All three patients were negative for any clinical or EEG seizure activities, significant cardiovascular disease, vasovagal syncope or rhythm disorder except orthostatic hypotension and premature complexes in one patient. All three patient underwent bilateral or unilateral CEA and were free of symptoms after the surgery, strongly suggestive of a correlation of bilateral carotid stenosis with the syncope as in our patient [3].

Figure 2. Digital subtraction angiography illustrating high grade stenosis of right ICA (2a) and left ICA (2b).
Anecdotally, bilateral carotid artery stenosis workup is not uncommon in patients with syncope. Identification of some degree of stenosis during this workup raises the question of its association with the clinical presentation. Our literature review suggests that this is a very rare phenomenon. Physiologically, unilateral reduced blood flow should not affect bilateral reticular formation and its pathways to bilateral hemisphere in a person with intact contralateral cerebral circulation and brain anatomy [8]. For anterior circulation stenosis to be a cause of syncope, bilateral centers for consciousness need to be affected simultaneously, which in subjects without pathological drop in blood pressure, is only expected if there is bilateral carotid stenosis. Based on flow dynamics, a severity of stenosis need to be at least 63% to cause a significant drop in blood flow [9]. With ICA stenosis of 80%, pressure drop of 56 mmHg has been reported [9]. So bilateral severe stenosis of >63% in subjects with negative workup for other syncopal events is probably the only situation when carotid revascularization should be considered.

CONCLUSIONS

Recognition of pathophysiology of reduced blood supply to brain in patients with bilateral severe ICA stenosis as a possible mechanism for syncope will help in further understanding of ICA disease, its progression and the effects of carotid revascularization on quality of life, and may be critical in the early identification of significant ICA stenosis and prevention of syncope or stroke.

REFERENCES