Open Journal of
Clinical & Medical
Case Reports

Volume 5 (2019)
Issue 10

ISSN: 2379-1039

Blood pressure control in a patient with subclavian steal syndrome and abdominal aneurysm: A clinical dilemma

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Abstract

Subclavian steal syndrome is a condition resulting from subclavian artery stenosis or occlusion, infrequently leading to syncope. We report a 92-year-old man with syncope due to subclavian steal syndrome, for which treatment was difficult because of the coexistence of an abdominal aortic aneurysm. Non-invasive treatment was scheduled for the subclavian artery stenosis and abdominal aortic aneurysm, considering his age and preferences. The clinical course was complicated by recurrent syncope due to unstable blood pressure. Cardiopulmonary arrest occurred 10 days after admission and a ruptured abdominal aortic aneurysm was found during autopsy imaging.

Keywords

aortic aneurysm; blood pressure; subclavian steal; syncope

Introduction

Syncope is defined as a sudden loss of consciousness and postural tone followed by spontaneous recovery due to insufficient blood flow in the brain [1,2]. The causes of syncope vary [1,2], e.g., cardiogenic, orthostatic, and neutrally mediated, and remain undiagnosed in approximately one-third of cases [3,4]. We report a case of syncope due to a rare cause, subclavian steal syndrome, for which treatment was difficult because of the coexistence of an abdominal aortic aneurysm.

Case Report

A 92-year-old man was transferred to the emergency department of our hospital because of transient loss of consciousness. The patient was found unconscious and leaning against the bath tub with fecal incontinence. He had gradually become conscious and returned to normal within a few minutes. The patient denied antecedent trauma and reported that he had had two other incidences of syncope within one

year, both of which resolved spontaneously within seconds. The patient had a previous history of surgery for chronic subdural hematoma, oral cavity cancer, aspiration pneumonia, and cognitive impairment. He had not taken any regular medication. He did not drink, had stopped smoking three years earlier, and had no known allergies.

On arrival, he was drowsy with a Glasgow Coma Scale of 14. His blood pressure was 103/65 mmHg in the left arm, his pulse was 52 beats per minute, his body temperature was 36.1°C, his respiratory rate was 24 breaths per minute, and his oxygen saturation was 98% while breathing ambient air. His pupils were equal (2.5 mm in diameter), round, and reactive to light. Strength on manual muscle testing was 5 out of 5 in the arms and legs, and sensation to light touch, and deep-tendon reflexes of the arms and legs were also normal. There was a painless, pulsatile mass in the abdomen. The remainder of the examination, including cranial nerves, was normal.

A normal sinus rhythm without ST-T changes or QT prolongation was observed on the electrocardiogram. His chest radiograph was normal. His white cell count was 5,800/μl with 75.7% neutrophils, hemoglobin was 10.0 g/dl, and platelet count was 191,000/μl. The C-reactive protein level was 0.78 mg/dl, the urea nitrogen level was 29 mg/dl, and the creatinine level was 1.33 mg/dl. The blood glucose level and ammonia level were normal, as were the lipid profiles, electrolytes and results of liver function tests. The D-dimer level was elevated to 27.34 μl/l (reference value, ≤1.0). Echocardiography demonstrated normal size and function of both ventricles.

Computed tomography (CT) of the brain, obtained without administration of contrast material, revealed no active lesion with a small amount of fluid in the subdural space (Figure 1A), a finding consistent with the previous history of surgery for chronic subdural hematoma. CT images of the chest and abdomen with and without contrast material were initially considered to be unremarkable except for an abdominal aortic aneurysm with a maximum diameter of 48 mm (Figures 1B and 1C). There was no evidence of pulmonary embolism or aortic dissection leading to syncope. The patient was admitted for further examination of transient loss of consciousness.

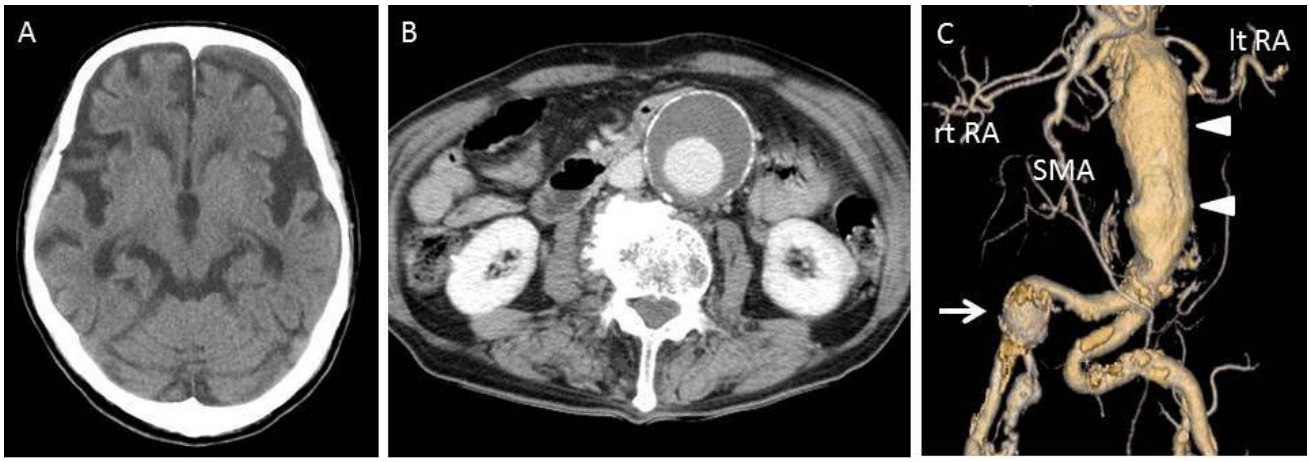

Figure 1: An axial CT image of the brain shows a small amount of subdural effusion in the left frontal region, a finding consistent with a left-sided chronic subdural hematoma (A). An abdominal aortic aneurysm is shown with a clear margin and a maximum diameter of 48 mm (B); the three-dimensional reconstruction revealed a fusiform aneurysm (C, arrowheads) between the renal arteries (RA) and the bifurcation of the iliac arteries, in addition to a localized aneurysm in the right common iliac artery (C, arrow). SMA = superior mesenteric artery.

The patient was later found to have a higher blood pressure in the right arm than in the left arm by more than 40 mmHg in systole. On review of CT scans of the chest, total occlusion at the entry site of the left subclavian artery was suspected (Figure 2A). CT angiography clearly visualized the anatomy around the left subclavian artery (Figure 2B), leading to a diagnosis of syncope due to subclavian steal syndrome. No significant arrhythmia was observed on Holter electrocardiography and monitor recording. Surgical treatment or endovascular intervention for the subclavian artery stenosis and abdominal aortic aneurysm were not planned considering his age and preferences. The patient was advised not to use his left arm extremely.

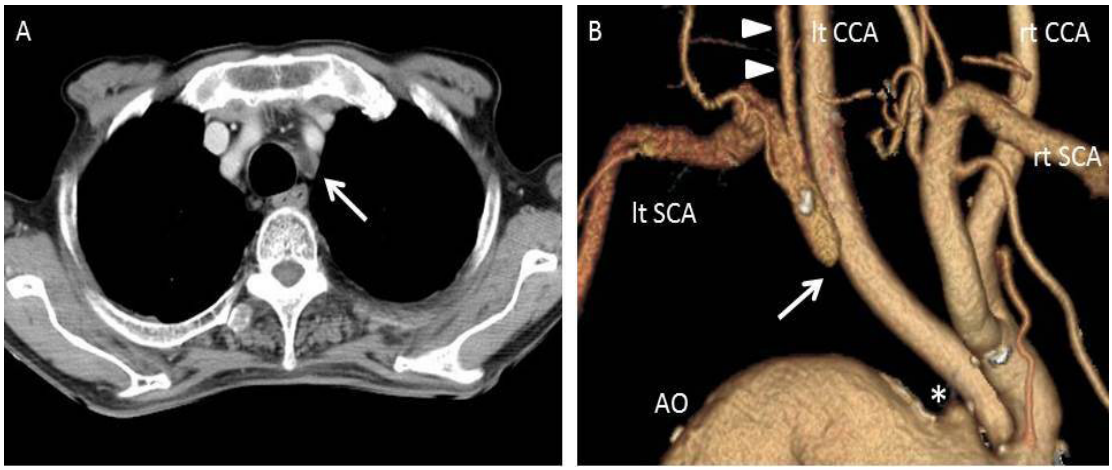

Figure 2: An axial CT image of the chest showing a non-enhanced area of the left subclavian artery (SCA) (A, arrow). The posteroanterior view on CT angiography demonstrates total occlusion of the left SCA from the entry site (B, asterisk) to the white arrow. Note that the vertebral artery (B, arrowheads) is branched from the non-occluded SCA. AO = aorta; CCA = common carotid artery.

The clinical course was complicated by unstable blood pressure. Nifedipineat 40 mg daily was administered and azilsartan at 20 mg daily was later added because of the high systolic blood pressure (e.g., 194 mmHg in the right arm and 145 mmHg in the left arm), but syncope recurred four times during eight days after admission, along with normal vital signs except for decreased systolic blood pressures (e.g., <140 mmHg in the right arm and <90 mmHg in the left arm). Both antihypertensive agents were withdrawn and acute abdominal pain developed the next day. No change in the abdominal aortic aneurysm was noted on follow-up CT. However, cardiopulmonary arrest occurred the following day. Autopsy was declined by his relatives and autopsy imaging revealed a ruptured abdominal aortic aneurysm.

Discussion

The current patient had had three incidences of syncope before admission, and was found to have a different blood pressure in the left and right arms. Three-dimensional reconstruction of CT images demonstrated total occlusion of the left subclavian artery at the entry site. The patient was diagnosed with syncope due to subclavian steal syndrome, although other causes, such as seizure and arrhythmia, cannot be completely ruled out.

Subclavian steal syndrome, which was first reported in 1960 by Contorni [5] and conceptualized in 1961 by Reivich et al. [6], is a condition associated with blood flow reversal in the vertebral artery as a result of significant stenosis or occlusion of the proximal subclavian artery or the innominate artery [7,8]. The exact prevalence of subclavian steal syndrome remains unclear in the general population, but is likely

rare; by cerebrovascular Doppler examination, subclavian steal syndrome was found in 1.2% of more than 25,000 patients [9] or 5.3% of 7,881 scans [10]. Conditions in patients with subclavian steal syndrome vary from being asymptomatic to causing ischemia of the upper extremities or brain [7,8]. It is reported that approximately 5% of patients with subclavian steal syndrome exhibit neurological symptoms [9,10], as in our case.

Acquired subclavian steal syndrome is likely to predominantly develop in the left side (82.3%) [10], probably due to the acute angle of the origin of the left subclavian artery (i.e., at risk for atherosclerosis) [11]. Causes of this condition include atherosclerosis, typically among older male patients, as in our case, whereas Takayasu arteritis, although rare, can lead to this condition, especially in young female patients [7]. It should be noted that a difference in arm blood pressure may indicate subclavian steal syndrome, although imaging modalities, e.g., Doppler sonography, computed tomography, magnetic resonance, and angiography, are required to confirm the diagnosis [7]. In the current case, the blood pressure difference between the right and left arms was a clue to this rare condition as the cause of syncope. The prevalence of symptoms related to subclavian steal syndrome was reported to increase with blood pressure differences in the right and left arms, e.g., 1.38% for 20 to 30 mmHg and 38.5% for ≥50 mmHg [7,10].

Therapeutic intervention is required only by a small percentage of patients with subclavian steal syndrome [7], with an incidence of 1.4% of all and 18.4% of symptomatic patients [10]. Treatment includes subclavian bypass surgery or endovascular intervention; the patency rate was greater than 90% at 10 years [12,13] in the former and 84.5% at 5 years in the latter [14]. Given the lower invasiveness and acceptable long-term outcome of endovascular therapy, it may be considered for patients with subclavian artery stenosis. However, recanalization was not achieved with this technique in up to 30% patients with subclavian artery occlusion [15]. Our patient did not undergo therapeutic intervention even though his blood pressure was difficult to control. It remains unclear whether intervention for the occluded subclavian artery would have stabilized the blood pressure and prevented the abdominal aortic aneurysm from rupturing in the current patient.

Conclusion

In conclusion, we report a case of syncope in which a difference in blood pressure between the arms was useful for the diagnosis of subclavian steal syndrome. The coexistence of an aortic abdominal aneurysm made treatment difficult.

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Manuscript Information: Received: October 21, 2018; Accepted: July 03, 2019; Published: July 15, 2019

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Citation: Saeki M, Kawasaki T, Hori M, Kikkawa Y, Oyamada H. Blood pressure control in a patient with subclavian steal syndrome and abdominal aneurysm: A clinical dilemma. Open J Clin Med Case Rep. 2019; 1559.

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