Subclavian fusiform aneurysm causing partial subclavian steal syndrome. Case report.

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Abstract

We present a 55-year-old man with repeated transient symptoms of vertebrobasilar arterial pathology. Physical examination detected no signs that would indicate the stenosis of the proximal subclavian artery segment. Ultrasound detected hemodynamic disorder of right vertebral artery, which correspond to a partial subclavian steal syndrome. Angiography shows fusiform aneurysm of ipsilateral subclavian artery in which central part is the base of hypoplastic vertebral artery. In the literature are rare reports of cases of partial subclavian steal syndrome caused by aneurysmal expansion of subclavian artery.

Keywords: subclavian steal syndrome, aneurysm, subclavian artery, ultrasonography

Introduction

Subclavian steal syndrome (SSS) is the phenomenon of reverse blood flow in the vertebral artery (VA), which is usually caused by stenosis or occlusion of the subclavian artery (SA) proximal to the vertebral artery origin [1,2]. Generally this pathology is diagnosed by ultrasound (US). Partial subclavian steal syndrome (PSSS) is characterized by alternating flow direction, typical appearance of hemodynamic spectrum and is also caused by stenosis of SA [3]. Subclavian artery aneurysms (ASA) are a rare phenomenon according to the number of reports in the medical literature, and can present clinically diverse symptoms, including local events (compression syndromes, thrombosis, or rupture), but also distal embolization and stroke [4].

Case report

The patient at the age of 55 years was examined at the Department of Neurology. For 15 years he has been treated with medication for type 2 diabetes mellitus. He was nonsmoker in the last 15 years. He had no history of hypertension, no previous injury to the head, neck, or shoulders. Within the last month before the first neurological examination he described two episodes of dizzy while getting up out of bed, with impossibility of standing and tendency for falling down. Disturbances lasted about one minute. Neurological examination register only impaired vibration sensation in the feet and extinguished reflexes of triceps surae, possibly as part of the initial chronic diabetic distal polyneuropathy. Also, not audible vascular murmur over carotid arteries (CA) and SA was found. Repeated measurements of blood pressure in upper arms were normal (120/70 mmHg), with no difference between the two arms. Peripheral arterial pulsations in the hands were normal.

Transcranial Doppler failed to detect the right VA and the left VA was registered with normal flow. Extracranial color Doppler (7.5MHz linear transducer,
Krunoslav Buljan et al

Aloka 5000, Tokyo, Japan) found marginal calcified plaques in the proximal segments of both internal CA without hemodynamic disturbances and a narrow right VA (2 mm in diameter), with low flow velocities (peak systolic velocity 0.21 m/s), and hemodynamic spectrum corresponding to PSSS (altering direction of circulation of the dominant antegrade flow and retrograde flow in late systole) (fig 1). The left VA had normal hemodynamic spectrum. Computed tomography angiography (CTA) confirmed the fusiform aneurysm of the right SA (fig 2). The largest diameter of the aneurysm was 15.6 mm, with proximal diameter of the aneurysm of 9 mm and distal of 8.7 mm. From the center of the aneurysm the starting point of the VA was identified together with the V1 segment of hypoplastic VA.

Over the next 6 months the patient had occasionally minimal symptomatology, which may correspond to transient disturbances of vertebrobasilar arterial circulation. He explained also of moderate short-term pain in the projection of the right clavicular region. Repeated ultrasound examination and CTA (5 months after the first angiography) showed continued hemodynamic disturbance in right VA and no changes in the size or shape of the right SA aneurysm. Surgical or endovascular treatment of the ASA was postponed.

Discussion

According to Meila et al in 99.8% of cases, the right vertebral artery originates from the right subclavian artery [5]. US of the vertebral arteries is a reliable method of examination of the vertebral arteries [6,7]. Usually it is the only diagnostic tool sensitive enough to allow the detection of the early stages SSS [3,8]. PSSS is regularly associated with the stenotic process of SA proximally from the VA departure [3] or rarely, VA origin [9], but not with ASA. Even though it is unusual, the manifestation of ASA in our case was the simultaneous occurrence of the vertebral arterial disorders. VA waveform changes in our case match early stage of SSS, where retrograde flow is lower than antegrade. VA waveform changes in our case are very similar to PSSS phenomenon, which is caused by stenosis SA. With an increasing degree of stenosis SA, the VA systolic flow becomes bidirectional with a reversal from mid to late systole [10], as in our case. According to a number of authors such VA waveform match stenosis SA between 55-80% [11-13]. Roh et al described surgical and endovascular procedure of aneurysm of the right subclavian artery which included the origin of the right vertebral artery [14]. However, they did not describe possible hemodynamic changes of the vertebral artery. Both in ASA and stenosis SA, pathophysiological mechanism of hemodynamic changes in SA is similar. The intraluminal pressure drop happens in the departure of VA. In the literature, we found only one case described where the ASA caused SSS [15]. Unlike our case, it was an aneurysm of the proximal segment of SA, with positive signs at physical examination - difference in the blood pressure - between the upper arms and impaired peripheral arterial pulsations on the side of aneurysm.

Conclusion

Ultrasound detection of PSSS phenomenon can sometimes indicate an aneurysmal expansion of the subclavian artery in the VA origin area.

References