Respiratory Variation of Vertebral Arterial Flow in a Patient Scheduled for Coronary Artery Bypass Graft Surgery
– Lesson From Atypical Subclavian Steal Phenomenon –

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Figure 1. (a) Color Doppler and (b) pulsed-wave Doppler ultrasonography indicating unusual subclavian steal phenomenon of the left vertebral artery. (A) Spontaneous breathing. Retrograde blood flow (blue) down the left vertebral artery was observed throughout a respiratory cycle. (B) Deep breathing. Blood flow direction was retrograde (blue) during the expiratory phase (Ex), and antegrade (red) during the inspiratory phase (In). (C) Selective subclavian angiography. (a) Under breath hold at shallow inspiration, a slit-like lesion (arrowhead) was seen at the kinking segment of the left subclavian artery. The left vertebral artery (LVA) and left internal thoracic artery (LITA) were not visualized. (b) Under breath hold at deep inspiration, the kink of the artery was attenuated according to the thoracic cage elevation (white arrow, clavicle; gray arrow, second rib). The slit lesion then disappeared (arrowhead) and the LVA and LITA were visualized on antegrade flow. (D) Volume-rendering multi-detector computed tomography under breath hold at deep inspiration showing only a mild atherosclerotic lesion (arrowhead) at the kinking site of the proximal left subclavian artery.
Atypical Subclavian Steal Phenomenon

Subclavian steal phenomenon” is defined as retrograde blood flow in the vertebral artery associated with a hemodynamically significant stenosis or occlusion of the proximal ipsilateral subclavian artery.1 Most cases of subclavian steal phenomenon, however, are asymptomatic.2 Recently, “coronary-subclavian syndrome” has been highlighted as the pathogenesis of ischemic events in patients undergoing coronary artery bypass graft (CABG) surgery with the left internal thoracic artery (LITA) coronary conduit: flow-limiting stenotic lesion of the left subclavian artery proximal to the LITA can produce myocardial ischemia during the left arm exercise.3 There is increasing evidence that this syndrome is not rare, and may have profound consequences for cardiac ischemic events and long-term survival.3

A 71-year-old female dialysis patient with a history of hypertension and dyslipidemia presented with dyspnea on exertion. She had left forearm internal arteriovenous shunt for dialysis. On admission, vascular bruit was audible in the left supraclavicular fossa. She did not complain of focal neurological symptom or left arm ischemia during arm exercise. On coronary angiography the patient was found to have triple-vessel disease associated with the left main disease.

Doppler ultrasonography indicated unusual respiratory variation of the direction of blood flow in the left vertebral artery. Under spontaneous breathing, retrograde blood flow was observed throughout a respiratory cycle (Figure 1A) suggesting subclavian steal phenomenon. Under deep breathing, however, blood flow direction was retrograde during the expiratory phase, whereas it was antegrade during the inspiratory phase (Figure 1B). On selective subclavian angiography a slit-like shadow defect was seen at the kinking segment of the left subclavian artery proximal to the origin of the left vertebral artery under breath hold at shallow inspiration (Figure 1C-a; Movie S1). The left vertebral artery and the LITA were not visualized. These findings suggested that the slit-like lesion was a hemodynamically significant stenosis. In contrast, under breath hold at deep inspiration, the kink was attenuated according to the thoracic cage elevation, the slit-like lesion disappeared, and the left vertebral artery and the LITA were visualized on antegrade flow (Figure 1C-b; Movie S2). Volume-rendering multi-detector computed tomography (MDCT) under breath hold at deep inspiration indicated only a mild atherosclerotic lesion at the kinking segment of the left subclavian artery (Figure 1D). Accordingly, it was suggested that the change in the luminal narrowing in proportion to the kink at the mild atherosclerotic lesion of the subclavian artery produced the unusual flow pattern of subclavian steal phenomenon, and caused transient normalization of the blood flow in the left vertebral artery during deep inspiration.

It is noteworthy that the vertebral flow pattern was unusual as subclavian steal phenomenon of known etiology. The to-and-fro pattern with retrograde flow in systole can be found in patients with subclavian steal phenomenon before the atherosclerotic lesion develops to severe stenosis or occlusion that results in the complete flow reversal throughout a cardiac cycle.4 To the best of our knowledge, however, variation during the respiratory cycle has not been documented in the vertebral artery flow pattern in patients with subclavian steal phenomenon. In the present case the kink at the mild atherosclerotic lesion of the subclavian artery was responsible for a
rare cause of subclavian steal phenomenon, which had an unusual flow pattern with respiratory variation. The kink of the subclavian artery is not uncommon in patients with arteriosclerosis, particularly elderly patients. Thus, the incidence of this type of subclavian steal phenomenon may be underreported.

The ipsilateral internal arteriovenous shunt might have a significant hemodynamic effect on subclavian steal phenomenon, because the internal shunt would augment the steal flow by decreasing the arterial resistance of the forearm. If the internal shunt had been temporarily occluded (eg, pressed with fingers), the normalization of the vertebral flow during deep inspiration might be attenuated or disappear according to the reduction in the forearm flow. We did not perform the occlusion test, however, because the patient refused it. If the subclavian artery had a fixed stenosis, it is possible that deep inspiration would augment venous return and the flow volume through the internal shunt, which would aggravate subclavian steal phenomenon and increase the reversal flow during deep inspiration. This was not the case, however, for the present patient. The vertebral flow direction was normalized during deep inspiration, suggesting that in this case, the increase of the antegrade subclavian arterial flow by the stretch of the kinking portion overwhelmed the possible augmentation of the shunt flow volume during deep inspiration.

In the present case, we had to consider the feasibility of the LITA as an artery conduit for CABG surgery. To avoid coronary-subclavian steal syndrome, the patient underwent CABG surgery involving grafting of the right internal thoracic artery, but not the LITA, to the left anterior descending artery and saphenous vein grafts to the right coronary artery and the left circumflex artery. Also, aorta-to-the left subclavian artery bypass surgery was performed using a vascular prosthesis to ensure blood flow through the internal shunt in the ipsilateral arm (Figure 2A). After the operation, on MDCT under breath hold at shallow inspiration the left vertebral artery was visualized even though the slit-like lesion was still present at the kinking site of the left subclavian artery. Consistent antegrade flow was also observed in the left vertebral artery during a respiratory cycle (Figure 2B).

In conclusion, we report a case of subclavian steal phenomenon with an unusual flow pattern with respiratory variation. In this case, it was considered that respiratory change of the degree of luminal stenosis at the kinking segment of the proximal left subclavian artery was the cause of subclavian steal phenomenon with transient normalization of blood flow during deep inspiration. MDCT has been established as the non-invasive imaging modality to evaluate not only the presence and severity but also the vulnerability of the coronary and peripheral arterial lesions. Given that the absence, however, of MDCT-proven significant organic stenosis does not preclude the occurrence of subclavian steal phenomenon, the use of multiple imaging modalities (eg, a combination of MDCT and duplex ultrasonography) would be required for preoperative assessment before CABG to avoid coronary-subclavian syndrome.

Disclosures
None.

References

Supplementary Files
Supplementary File 1
Movie S1. Selective subclavian angiography under breath hold at shallow inspiration. A slit-like lesion was observed at the kinking segment of the left subclavian artery proximal to the origin of the left vertebral artery. The left vertebral artery and left internal thoracic artery were not visualized.

Supplementary File 2
Movie S2. Selective subclavian angiography under breath hold at deep inspiration. The kink of the left subclavian artery was attenuated according to the thoracic cage elevation. Subsequently, the slit lesion disappeared and antegrade flow visualized the left vertebral artery and left internal thoracic artery.

Please find supplementary file(s) at http://dx.doi.org/10.1253/circj.CJ-13-0071