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## **Case Report**

# Position Dependent Carotid Impingement Causing Recurrent Strokes

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### Abstract

We report the case of a young man with recurrent strokes over a four year period, all occurring after leaning forward. He had suffered damage to the right subclavian and right carotid arteries in a car accident 20 years prior. Review of history and imaging concluded that all of his infarcts had been in the distribution of the right carotid artery. CT angiogram revealed that a segment at the origin of the right common carotid artery was adjacent to the sternum and kinked at the point of contact. Proposed mechanism of infarcts is position dependent intermittent vessel damage causing thrombosis and distal embolization. The patient underwent surgical repair, with no further events. This case highlights the importance of evaluating structures adjacent to vessels in patients with cryptogenic strokes.

## **ABBREVIATIONS**

**MRI:** Magnetic Resonance Imaging; **MRA:** Magnetic Resonance Angiogram; **CT:** Computed Tomography; **MCA:** Middle Cerebral Artery; **PCA:** Posterior Cerebral Artery; **TIAs:** Transient Ischemic Attacks.

## **CASE PRESENTATION**

A 39 year old man came to Neurology clinic for a second opinion. His general health status was good, with none of the common stroke risk factors. At age 19, he had been in a car crash with trauma to the chest requiring surgery to repair damage to the aorta, right subclavian, and right common carotid arteries. He recovered well, with no symptoms until age 36 when he had his first stroke. Upon straightening up after leaning over during yard work, he developed a "head rush" feeling followed by visual problems, dysarthria, left facial droop and left hand numbness. Most of the symptoms resolved in a couple days, with residual left upper quadrantanopsia. An angiogram showed thrombus in the right carotid bulb, for which he underwent carotid endarterectomy. He was discharged on warfarin, aspirin, and atorvastatin.

Six months later, he had another episode of left hand numbness after bending forward. His anticoagulation was subtherapeutic, and MRI was positive for right cortical stroke. Symptoms resolved after about a week. Four months later he was admitted for a transient episode of "head rush" after bending over, was again found to be subtherapeutic on warfarin, and placed on heparin IV. The following day, he complained of increased vision

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problems, and was found to have a right occipital intracerebral hemorrhage. Anticoagulation was stopped and he was discharged on clopidogrel.

After that admission, he developed migrainous headaches consisting of pain on the right side of his face, head, and eye in association with flashing lights in the right eye. He also noticed recurrent episodes of the "head rush" feeling after leaning forward. These were sometimes followed by transient feelings of warmth and paresthesias on his right face.

Eight months later, he developed frequent episodes of lightheadedness and tingling in his right arm after actively using the arm. He was found to have right subclavian stenosis and subsequently underwent axillo-axillary bypass. The right arm symptoms and lightheadedness resolved, but the migraines and "head rush" episodes persisted.

Six months later, he had another episode of dizziness and left hand numbness after bending over. MRI revealed a right cortical stroke, and evaluation with MRA head and neck, EEG, echocardiogram, and hypercoagulable labs was unremarkable. Four months later, he had an episode while leaning over to shovel snow of head rush followed by flushing of his right face, left sided weakness, dysarthria, and confusion. MRI showed multiple small infarcts in the right MCA and PCA territories. His symptoms improved to residual left hand weakness only.

At this point, he presented to our clinic for further evaluation. Based on his history and prior imaging, all of his strokes and TIAs appeared to be in the distribution of the right internal carotid

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artery (including the occipital infarcts, secondary to a large right posterior communicating artery and diminutive P1 segment of the right posterior cerebral artery). To better evaluate his vasculature, a CT angiogram was performed. Initial report was of patent vessels throughout. Careful attention to the origin of the right common carotid artery showed that the vessel was in contact with the sternum, with a kink in the vessel at the area of contact (Figure 1). Bending forward could put additional pressure on the vessel in this location, causing vessel injury, with subsequent thrombus formation and artery to artery embolization. It could also cause transient hypoperfusion leading to the head rush sensations, with warmth and paresthesias on the right side of the face likely a result of changes in blood flow in the external carotid artery.

The imaging findings and proposed mechanism of strokes were discussed with the patient, and the decision made to proceed with surgical reconstruction.

## SURGICAL DESCRIPTION

The axillo-axillary bypass graft previously placed across the midline was dissected out, and extensive adhesiolysis was performed to free the heart from the anterior chest wall, and to dissect out the ascending aorta. The previous aorto-to-carotid and aorto-to-subclavian bypass grafts were avoided to prevent embolization of debris within them. The ascending aorta was then clamped with a partial occluding clamp and an opening made about 3 cm above the aortic valve. The proximal end of a customdesigned bifurcated Dacron graft (Terumo, Vascutek, Scotland,



Figure 1 Imaging of the right common carotid artery origin.

A. Pre-operative CT angiogram, Arrow shows likely location of positiondependent carotid origin impingement

B. Post-operative CT angiogram

\*Connection to right common carotid artery. ^Connection to right axillary artery (pre-op this is an axillo-axillary bypass, post-op this is an aorto-axillary graft).

UK) was anastomosed to the ascending aorta. The right common carotid artery was then transected and anastomosed to the graft. The previous aorto-carotid graft was freed from the manubrium posteriorly, transected as proximally as possible to its origin from the aorta, and removed. The previous axillo-axillary bypass graft was then transected and anastomosed to the free end of the new bifurcated graft, and the remainder of the previous axillo-axillary graft was resected and removed. A Doppler device was used to confirm good flow in both the right common carotid artery and the right axillary artery, and the chest was closed.

The patient was kept on dual antiplatelet agents (Plavix 75mg daily and aspirin 81mg daily) for 3 months and then switched to 81 mg aspirin alone. He recovered well from the surgery and reported no further episodes of focal symptoms or head rush. His migraine headaches also resolved post-operatively.

## DISCUSSION

Boney abnormalities causing vessel damage are a rare but important cause of stroke, as the underlying lesion may be amenable to surgical correction. One of the authors has previously reported a case in which a congenital boney abnormality of the occiput was found to be causing recurrent vertebral artery damage and strokes [1]. Cerebral embolism can also be seen with damage to the subclavian artery from a cervical rib in the thoracic outlet syndrome [2]. Typically, the carotid artery is not in a position where it can be damaged by surrounding structures, although an intriguing association has been found between styloid process length and carotid dissection [3]. In this case, the prior injury and reconstruction brought the carotid into an unusual position relative to the sternum.

This case demonstrates that careful review of not just the vessel lumen, but also the surrounding structures can sometimes reveal a cause of stroke. CT angiogram is an imaging modality that allows for excellent visualization of both vessels and surrounding boney structures. 3-dimensional reconstruction programs that allow for rotation of the image and viewing from alternative angles can sometimes reveal lesions that are not immediately apparent in standard imaging protocols.

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