CASE REPORTS

Magnetic resonance angiography in subclavian steal syndrome

Paul D Flynn, David J Delany, Huon H Gray

Abstract

A case is reported of a patient with the subclavian steal syndrome in whom the reversed blood flow of the vertebral artery was shown by phase encoded magnetic resonance angiography.

(Br Heart J 1993;70:193-194)

Reversed blood flow in the vertebral artery associated with neurological dysfunction was described first in 1961 by Reivich *et al*,¹ although the concept of retrograde flow in the cerebral circulation had been presaged by Willis in his "cerebri anatome" of 1664.² Reivich *et al* described two patients with stenosis of the left subclavian artery proximal to the origin of the left vertebral artery, and

showed angiographically that blood in the vertebral artery was flowing from the verte-brobasilar junction to the subclavian artery. Furthermore in experiments on anaesthetised dogs it was shown that occlusion of the sub-clavian artery resulted in a reversal of flow in the ipsilateral vertebral artery, and increased flow in the contralateral vertebral and both carotid arteries. Antegrade basilar and total cerebral blood flow were, however, reduced. It was postulated that, with subclavian stenosis, activity that increased the reversed verte-bral flow, such as exercise of the ipsilateral limb, could result in transient vertebrobasilar or cerebral hypoperfusion. This phenomenon was labelled "the subclavian steal syndrome" by Fisher.³

Subsequently, subclavian steal was reported in many other patients and detected either by arteriography⁴ or more recently by Doppler ultrasound of the extracranial arteries.^{5,6} We report here a case of subclavian steal syndrome in which the steal was shown non-invasively by phase contrast magnetic resonance angiography (MRA).

Case report

A 72 year old woman was admitted a few hours after a collapse at home. She had been well, and had been standing at her kitchen sink washing elderberries for 20 minutes when she gradually developed some mild backache. A few minutes later she slumped backwards and was caught and lowered to the floor by her husband. She did not lose consciousness but seemed disoriented. There had been no chest pain, no palpitation, no premonitory aura, or any other epileptiform features. Her general practitioner was called, and on arrival he noted weak pulses in her left arm and arranged her admission to hospital with a diagnosis of a possible thoracic aortic dissection. She had fainted 11 years previously, but otherwise she had been fit and well. She had smoked cigarettes for many years.

Examination showed a regular pulse at 80 beats per minute, and confirmed the relative weakness of the left arm pulses with a differential blood pressure of 148/80 mm Hg in the right arm and 112/80 mm Hg in the left. A left-sided carotid bruit was noted. It was at this point that the patient remarked that, as a former nurse, she had on occasion felt her own pulses and had noted their weakness in her left arm for some years. There were no other cardiovascular signs and neurological examination was normal.

Department of
Cardiology, Wessex
Cardiothoracic
Centre, Southampton
General Hospital,
Southampton
P D Flynn
D J Delany
H H Gray

Correspondence to:
Dr D J Delany, Department
of Cardiology, Wessex
Cardiothoracic Centre,
Southampton General
Hospital Southampton
SO9 4XY.

Figure 1 Spin echo coronal MRI in the plane of the aorta and subclavian, and vertebral artery junctions. The absence of flow in the proximal subclavian artery (arrow) was confirmed on transverse images.

Figure 2 Phase contrast MRA in the same plane as fig 1. Flow encoding is black for cephalic and white for caudal. The vertebral artery positions are shown by line markers.

Normal investigations included a full blood count, biochemical screen (including glucose), measurement of a series of cardiac enzymes, and electrocardiograms. Her chest x ray film was normal and showed no evidence of mediastinal widening. On computed tomograph scanning of the thoracic aorta there was atheromatous disease of the descending thoracic and abdominal aorta but no evidence of dissection. It was noted that the left subclavian artery did not opacify with contrast. Spin echo MRI images confirmed occlusion of the left subclavian (fig 1), and MRA with flow encoding gradients was performed showing

antegrade flow in the right vertebral artery, but retrograde flow in the left vertebral artery, constituting a classical subclavian steal (fig 2). Thus a combination of MRI and MRA was able both to exclude other diagnostic considerations and accurately and definitively make the true diagnosis without the need for any invasive procedure.

Discussion

The combination of history, physical findings, and retrograde flow in the left vertebral artery on MRI was considered diagnostic of the subclavian steal syndrome. In the 1960s treatment of this condition was usually surgical but Fields and Lemak in 1972 showed no significant difference in mortality between those patients who had undergone surgery and those who had been managed conservatively.⁴ The proportion of patients achieving a good outcome was also similar, and the rate of strokes was marginally higher in the surgical group. In 1988 Hennerici *et al* showed that most patients with isolated unilateral subclavian steal were symptom free, and suggested that in most of those who were not, the symptoms were most likely to result from coexistent cerebrovascular disease. In support of this they followed up 54 patients with unilateral subclavian steal alone with Doppler examinations over seven years. Eleven patients died, only one of them from a stroke. Eight further patients had transient ischaemic attacks, all of whom were shown on repeat Doppler examination to have developed carotid artery stenosis.

The consensus now seems to be that isolated subclavian steal is a relatively benign phenomenon, but that it may be associated with other cerebrovascular disease for which it could be considered a marker. Our patient was advised to stop smoking if possible, and to avoid any prolonged exertion of her left arm, especially while standing. Other intervention will only be considered if she develops further evidence of disabling vertebrobasilar insufficiency.

1 Reivich M, Holling HE, Roberts B, Toole JF. Reversal of blood flow through the vertebral artery and its effect on cerebral circulation. *N Engl J Med* 1961;265:878-85.
2 Willis T. Practice of physick. London: S Pordage, 1684; Part 6: 59.
3 Fisher CM. A new vascular syndrome—"the subclavian steal". *N Engl J Med* 1961;265:912-3.
4 Fields WS, Lemak NA. Joint study of extracranial arterial occlusion. VII. Subclavian steal—a review of 168 cases. *JAMA* 1972;222:1139-43.
5 Bornstein NM, Norris JW. Subclavian steal: a harmless haemodynamic phenomenon? *Lancet* 1986;ii:303-5.
6 Hennerici M, Klemm C, Rautenberg W. The subclavian steal phenomenon: a common vascular disorder with rare neurologic deficits. *Neurology* 1988;38:669-73.