CASE REPORT

20-year-old injury: an unusual cause of stroke

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SUMMARY

A 62-year-old man presented with a 2-day history of headache, nausea and dizziness on a background of recently diagnosed stage 2c prostate carcinoma and controlled hypertension. Imaging confirmed bilateral cerebellar strokes; on further investigations, he was found to have undiagnosed subclavian steal syndrome following severe injury to his left arm more than 20 years earlier.

BACKGROUND

This case highlights unconventional mechanisms of stroke.

CASE PRESENTATION

A 62-year-old man presented with a new onset headache of 2 days along with nausea and dizziness after going to the toilet. His medical history was significant for Gleason 3+4 locally invasive prostatic adenocarcinoma diagnosed 6 months previously and well-controlled hypertension. He was otherwise healthy with no previous history of cerebrovascular events or other medical problems. He was an exsmoker for 20 years with a 10 pack-year history.

General examination was remarkable for mild ataxia, nystagmus and dysidiadochokineses. His symptoms and headache resolved shortly after admission.

Of note, on closer questioning at a later stage, the patient admitted to mild claudication of the left arm when abducting that arm greater than 90° as well as a history of falling off a ladder and severely injuring his left shoulder 20 years ago.

INVESTIGATIONS

His initial haematological and biochemical investigations confirmed normal cholesterol and blood sugar levels and normal inflammatory markers. A 24-hour Holter and blood pressure monitors revealed maintenance of sinus rhythm and well-controlled blood pressures. Of note, bilateral arm systolic blood pressures only differed by an average of 10–15 mm Hg.

A CT and MRI of the brain revealed bilateral acute cerebellar infarcts, a chronic right cerebellar infarct with multiple petechial haemorrhages. No metastatic disease was visualised (figure 1).

The MR angiography of his vascular supply revealed a proximal occlusion of the left subclavian artery with extensive collaterals from the left vertebral artery to the subclavian artery distal to the stenosis (figure 2) which was suggestive of a subclavian steal phenomenon. It was also noted that this gentleman had a non-dominant right vertebral

Figure 1 MRI of the brain revealing bilateral cerebellar infarcts. The yellow arrow showing right cerebellar infarct and red arrow showing left cerebellar infarct.

artery that was atretic distally with and a mildly irregular mid-basilar artery.

A CT arch angiogram also confirmed a 1.7 cm segmental occlusion of the left subclavian artery as well as demonstrating collaterals from the left vertebral artery to the left subclavian artery in more detail (figure 3).

The development of a collateral circulation suggested that the patient had a longstanding subclavian stenosis with a potential for steal phenomenon to occur.

Other investigations carried out were carotid ultrasound Dopplers which confirmed patent carotids bilaterally and a transthoracic echocardiogram which showed an aneurysmal intra-atrial septum. However,

Figure 2 MR reconstruction of carotid angiogram. The red circle highlights the subclavian artery stenosis and the yellow arrows point to the multiple collaterals.

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Figure 3 Three-dimensional arch aortogram. The red circle and white arrow locates the left subclavian artery stenosis.

a subsequent bubble study with echocardiogram excluded the presence of a patent foramen ovale.

TREATMENT

He was given subcutaneous enoxaparin 40 mg during his admission and started on 300 mg aspirin for 2 weeks followed by 75 mg aspirin lifelong. He was also prescribed atorvastatin 40 mg, perindopril 5 mg and dutasteride/tamsulosin 0.5/0.4 mg for his prostate symptoms.

His case was discussed with the vascular team who felt that he was not for surgical intervention as there was no evidence that hypoperfusion secondary to his subclavian artery occlusion was the cause of his posterior circulation stroke and that intervention would not alter future stroke risk. Further, they felt that his left arm claudication symptoms were too minimal for surgery to be recommended.

At the time of admission, our patient was awaiting an elective radical prostatectomy for prostate cancer. In view of the stroke, this was postponed for 6 months with the start of hormonal treatment and radiotherapy in the interim.

DISCUSSION

The cerebellum receives its blood supply from the cerebellar arteries which originate from the vertebral and basilar arteries. Strokes in this region account for 2–3% of strokes that occur each year.¹

Cerebellar infarction often presents with common and non-specific symptoms such as dizziness, nausea and vomiting, unsteady gait and headache.² Neurological examination may reveal abnormalities in coordination, gait, balance, speech and nystagmus. Subclavian steal stenosis was found to occur 3–4% in the general population,³ and in 17% of 6 534 cases in the joint study of extracranial arterial occlusion,⁴ with atherosclerosis as the main risk factor. The subclavian steal syndrome is characterised by a subclavian artery stenosis located proximal to the origin of the vertebral artery causing retrograde flow of blood from the vertebrobasilar artery circulation to supply the subclavian artery and arm during exertion. This can result in vertebrobasilar insufficiency.⁵

Although subclavian artery stenosis is not rare, flow reversal within the vertebral artery is present in only a minority of these cases (2.5% in 6534 cases). Of those with angiographic steal, only 5.3% (9/168) resulted in neurological symptoms.⁴ This emphasises the point that flow reversal within the vertebral artery does not always result in neurological symptoms. Therefore, for a neurological deficit to occur, other factors must coexist,^{6 7} most commonly concurrent carotid stenosis⁸ or neurovascular instability.

The mainstay of treatment includes angioplasty, endarterectomy and patch aortosubclavian artery bypass or carotid-subclavian artery bypass. Indications for surgery for subclavian artery stenosis include vertebrobasilar insufficiency and upper extremity ischaemia.⁶

There is limited evidence for medical treatment in subclavian steal syndrome; however, it is often treated with anticoagulation or antiplatelet medications,⁹ with the management of risk factors for atherosclerosis.

POSSIBLE MECHANISMS OF CEREBELLAR INFARCTION IN THIS PATIENT

Hypoperfusion due to steal phenomenon

In a patient with subclavian stenosis, the exercise-induced increased upper limb blood flow demand can be met by diverting ('stealing') blood flow from the ipsilateral vertebral artery into the subclavian artery distal to a severe stenosis by retrograde flow. This phenomenon is possible because of the unique anatomic disposition of the vertebral and subclavian arteries, as anastomosis of the vertebral arteries from the circle of Willis provides a potential natural conduit for bypass of an occluded subclavian artery (on the left) or brachiocephalic artery (on the right;¹⁰ figure 4).

Turbulence of alternating anterograde and retrograde blood flow in the vertebral artery to the subclavian artery

While there are limited studies on flow turbulence resulting in thrombus formation, animal studies show that in vivo there was significantly more thrombi by weight occurring in turbulent flow shunts compared to laminar flows (180 ± 30 mg vs 0.9 ± 0.6 mg) and there was a linear relationship between the degree of turbulence to the weight of thrombi formation.¹¹

Prothrombosis with malignancy

There is an associated increased risk of stroke in cancer patients.

When cells of the monocyte or macrophage lineage interact with malignant cells, they release tumour necrosis factor, interleukin 1 (IL-1) and IL-6 causing endothelial damage and thereby converting the vascular lining over which blood flows to

Figure 4 Subclavian steal syndrome (black dotted lines representing obstruction at the proximal left subclavian artery, black arrow indicating normal blood flow and red arrow indicating retrograde flow through the left vertebral artery).

a thrombogenic surface. The interaction between tumour cells and macrophages also activates platelets, factor XII and factor X. This leads to the generation of thrombin and thrombosis. Substances in tumour cells such as cysteine proteases and tissue factor have procoagulant or thromboplastin-like activity.¹²

All the mechanisms above can interact to promote thrombosis and contribute to the occurrence of recurrent cerebellar strokes in this patient in the absence of radiological evidence of atherosclerosis.

Learning points

- ▶ Subclavian artery stenosis occurs in 3–4% of the general population with atherosclerosis as a main risk factor.
- ▶ Among patients with proven angiographic steal phenomenon, only 5.3% (9/168) resulted in neurological symptoms.
- ▶ In a patient with subclavian artery stenosis who has neurological symptoms, other factors must be considered, most commonly concurrent carotid stenosis, neurovascular instability and in our patient's case, concurrent malignancy.

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from previous research and publications were collected by LVWL. This article was fine tuned by LD and OCM prior to submission.

Competing interests None.

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REFERENCES

- 1 Kelly PJ, Stein J, Shafqat S, *et al.* Stroke: functional recovery after rehabilitation of stroke. *J Am Heart Assoc* 2001;32:530–4.
- 2 Edlow JA, Newman-Toker DE, Savitz SI. Diagnosis and initial management of cerebellar infarction. *Lancet Neurol* 2008;7:951–64.
- 3 English JA, Carell ES, Guidera SA, *et al.* Angiographic prevalence and clinical predictors of left subclavian stenosis in patients undergoing diagnostic cardiac catheterization. *Catheter Cardiovasc Interv* 2001;54:8–11.
- 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 Psillas G, Kekes G, Constantinidis J, *et al.* Subclavian steal syndrome: neurotological manifestations. *ACTA Otorhinolaryngol Ital* 2007;27:33–7.
- 6 Sheehy N, MacNally S, Smith CS, *et al.* Contrast-enhanced MR angiography of subclavian steal syndrome: value of the 2D time-of-flight 'localizer' sign. *AJR Am J Roentgenol* 2005;185:1069–73.
- 7 Bornstein NM, Norris JW. Subclavian steal: a harmless haemodynamic phenomenon? *Lancet* 1986;ii:303–5.
- 8 Hennerici M, Klemm C, Rautenberg W. The subclavian steal phenomenon: a common vascular disorder with rare neurologic deficits. *Neurology* 1988;38:669.
- 9 Redmond KC, Barry MC, Kavanagh E, *et al.* Bilateral subclavian steal syndrome. *Ir J Med Sci* 2002;171:44–5.
- 10 Chimowitz MI, Lynn MJ, Howlett-Smith H, *et al.* Comparison of warfarin and aspirin for symptomatic intracranial arterial stenosis. *N Engl J Med* 2005;352:1305–16.
- 11 Stein PD, Sabbah HN. Measured turbulence and its effect on thrombus formation. *J Am Heart Assoc* 1974;35:608–14.
- 12 Bick RL. Cancer-associated thrombosis. *N Engl J Med* 2003;349:109–11.

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