Vertebral artery dissection: not a rare cause of stroke in the young

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Abstract

We hereby describe a 42-year-old lady who developed vertebral artery dissection following a head injury. The clinical features and management of the condition are discussed.

Keywords: vertebral artery dissection, vertebral artery injury, young stroke

Case report

A 42-year-old woman presented with history of left frontal headache, vomiting, dizziness, hyper-somnolence and right-sided visual disturbance. Two days earlier, she had sustained a head injury in the occipital region. She had history of hypertension. On admission, she was alert and conscious (GC Scale 15/15), apyrexial, haemodynamically stable (blood pressure 170/90, pulse rate 80/min), and she had a right homonymous hemianopia but no other neurological deficits. Funduscopic examination was unremarkable. CT of the brain revealed left occipital and posterior parietal infarcts. Subsequently, she became drowsy and developed mild weakness and poor coordination in the right arm, which spontaneously improved within a few hours. Five days later, an magnetic resonance angiogram (MRA) of the head and neck revealed absent flow in the left vertebral artery with a dissection flap (Figure 1), appearances consistent with left vertebral artery dissection. She was prescribed aspirin for 2 weeks and was anti-coagulated with warfarin 2 weeks after the event. She was commenced on amlodipine for hypertension. A year later, her hemianopia is still persistent.

Discussion

Cervical arterial dissections account for about 20% of young strokes, though vertebral dissections are up to five times less common than carotids. Vertebral artery dissection (VAD) has an estimated incidence of around 1.0 per 100,000 [1] and is slightly more common in females with average age
affected in the fifth decade [2, 3]. VAD can be spontaneous or can follow blunt cervical trauma. Sub-intimal dissections swell and occlude the artery. The reduced blood flow rate and endothelial injury encourage thrombus formation, with the inevitable emboli causing brain stem and cerebellar infarctions. The sub-adventitial dissections form a pseudo aneurysm, which can rupture again causing a sub-archanoid haemorrhage. Bilateral dissections are not uncommon and were seen in two-third of patients in a series by Mokri et al. [4] and in 28% of patients studied by Schievink et al. [5].

Headache and/or neck pain are the prominent symptoms and tend to be ipsilateral to the dissection and commonly posteriorly distributed [2], though our patient had a frontal headache albeit on the same side as the dissection. In a retrospective analysis of 26 patients by Saeed et al. [3], headache was present in 88% of patients along with vertigo in 57%, unilateral facial paraesthesia in 46% and visual field defects in 15%.

The incidence of posterior circulation stroke syndrome following vertebral artery injury (VAI) was 24% in the series by Biffl et al. [6], whereas other studies had quoted an incidence of around 5%. The common types of stroke are lateral medullary and cerebellar strokes, a consequence of vascular occlusion. Visual symptoms are varied but common. Saeed [3] reported visual field defects in 15% of his patient series. In 52 patients with VAD, Hicks et al. [7] found that 86% had ophthalmologic findings such as diplopia (45%), nystagmus (37%), ocular misalignment (3%), Horner's syndrome (27%), decreased corneal sensation (22%), ptosis (16%), blurred vision (14%) and visual field defect (10%). Hence, presence of visual symptoms in the setting of head injury should alert one to the possibility of VAI. The majority of dissected lesions seem likely to stabilise within a few months. Prognosis appears promising with most of the patients making a complete or very good recovery [4, 8]. After the first month of dissection, the risk of recurrent dissection is about 1% per year [5].

Management

The primary aim in VAI management is to prevent stroke and to improve neurological outcome. There are no randomised trials, though the general consensus is to treat such patients with anti-coagulant therapy provided no contra-indication exists, on the basis that the presumed mechanism of stroke in arterial dissections is embolic rather than haemodynamic in origin [1]. The rationale for anti-coagulation is to minimise clot formation, propagation and embolisation [9]. Studies [6, 9, 10] indicate that in blunt cervical artery injury, anti-coagulation is beneficial in terms of improved neurological outcome and improved survival. A recent population-based study of patients with spontaneous cervical artery dissections has also shown anti-coagulation to be beneficial [1]. For individuals who would not tolerate anti-coagulation, anti-platelet therapy should be considered. The optimal duration of the treatment remains unclear, though a 3–6 months course is advocated [9, 10].

Key points

- In the context of neurological features and head injury, consider cervical arterial dissection as a possibility.
- Visual symptoms in the setting of head injury should alert one to the possibility of VAI.
- Sub-archanoid bleeds can follow dissections from pseudo aneurysm formation.

Conflicts of interest

None

References