

Pure motor hemiparesis in a case of vertebrobasilar arterial ectasia

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Summary. A case of pure motor hemiparesis due to a pontine lacunar infarct is reported. Infarction was related to vertebrobasilar ectasia identified on CT, MRI and MR angiography. MR studies provide accurate information on anatomical location, residual lumen, partial thrombosis, mass effect on brain stem and CSF pathways and vascular complications.

Key words: Intracranial aneurysm – Basilar artery – Ectasia – Magnetic resonance imaging – Magnetic resonance angiography

In 1965, Fisher and Curry [1] defined pure motor hemiplegia as paralysis of the face, arm and leg on one side without any other neurological finding, except for dysarthria. Since the paralysis was usually incomplete, the term pure motor hemiparesis (PMH) was considered more appropriate. PMH is commonly caused by a small, deep cerebral infarct or lacune located somewhere along the corticospinal pathway. However, several other causes of PMH have been described, including haemorrhages, tumours, multiple sclerosis or infections [2]. We report a case of PMH due to vertebrobasilar ectasia (VBE) identified on magnetic resonance imaging (MRI) and MR angiography (MRA).

Case report

A 67-year-old right-handed hypertensive man was admitted because of a right hemiparesis. Over about 6 months, he had developed a progressive gait disorder caused by weakness and hypertonia of the right leg. His wife had also noticed unmotivated crying and laughing. On the day of admission, his clinical state had suddenly worsened with marked weakness of the right limbs without associated signs. Neurological examination showed a right hemiparesis, more marked in the leg, a slight central facial paresis, and moderate dysarthria without dysphagia. Tendon reflexes were very brisk on both sides but plantar responses were flexor. The right arm was flaccid while the leg was markedly spastic. Detailed examination of sensation, visual fields, ocular movements, coordination and cognitive functions was normal.

CT showed several foci of calcification in the left cerebellopontine angle. Contrast-enhancement showed a round, hyperdense mass deforming the left side of the pons. Additional hyperdense areas were seen in the parasellar region. These findings were suggestive of ectasia of the basilar artery (BA) and both carotid siphons.

MRI was performed at 2 T, with a multisection, spin-echo technique. T1-weighted images were obtained in sagittal, coronal, and axial planes with repetition time (TR) 480 ms and echo time (TE) 13 ms. T2-weighted images were obtained in the axial plane with TR 2600 ms and TE 22 and 80 ms. The right vertebral artery (VA) appeared elongated and tortuous. The BA originated in the left cerebellopontine angle cistern and was markedly elongated, its bifurcation indenting the floor of the third ventricle (Fig. 1). Its inferior half was markedly enlarged, with the appearance of a fusiform aneurysm deforming the left side of the pons (Fig. 1). Evidence of a large patent portion within the aneurysmal lumen was given by signal void or isointensity on T1- and T2-weighted sections (Figs. 1, 2). However, the residual lumen appeared of heterogeneous intensity, suggestive of partial thrombosis. A lacunar infarct was identified in the left paramedian part of the pons (Fig. 2).

Three-dimensional time-of-flight MRA was performed on a 1.5 T superconducting magnet, with fast imaging using steady precession (FISP) sequences, TR 36 ms, TE 7 ms. A field of view of 22 cm was used. The MRA showed an elongated right VA, an ectatic BA, and dilated carotid siphons (Fig. 3).

Other laboratory tests, Doppler ultrasonography of cervical arteries and abdominal aorta, chest radiography, ECG and two-dimensional echocardiography were normal. The patient's state progressively improved and he was discharged home receiving acetylsalicylic acid, and having physiotherapy. Six months later, he had a residual spastic weakness of the right leg.

Discussion

Our patient presented with a right PMH caused by a small pontine infarct in the territory of the paramedian perforating branches of an ectatic BA. Prior to his stroke, he had progressive weakness of the right leg which could be attributed to the compression of the left anterior part of the lower pons by the ectatic vessel. PMH is the most common lacunar syndrome, accounting for about half of cases [2]. The frequency of lacunar infarction involving the basis pontis is underestimated by CT. MRI identifies lacunes more accurately [3].

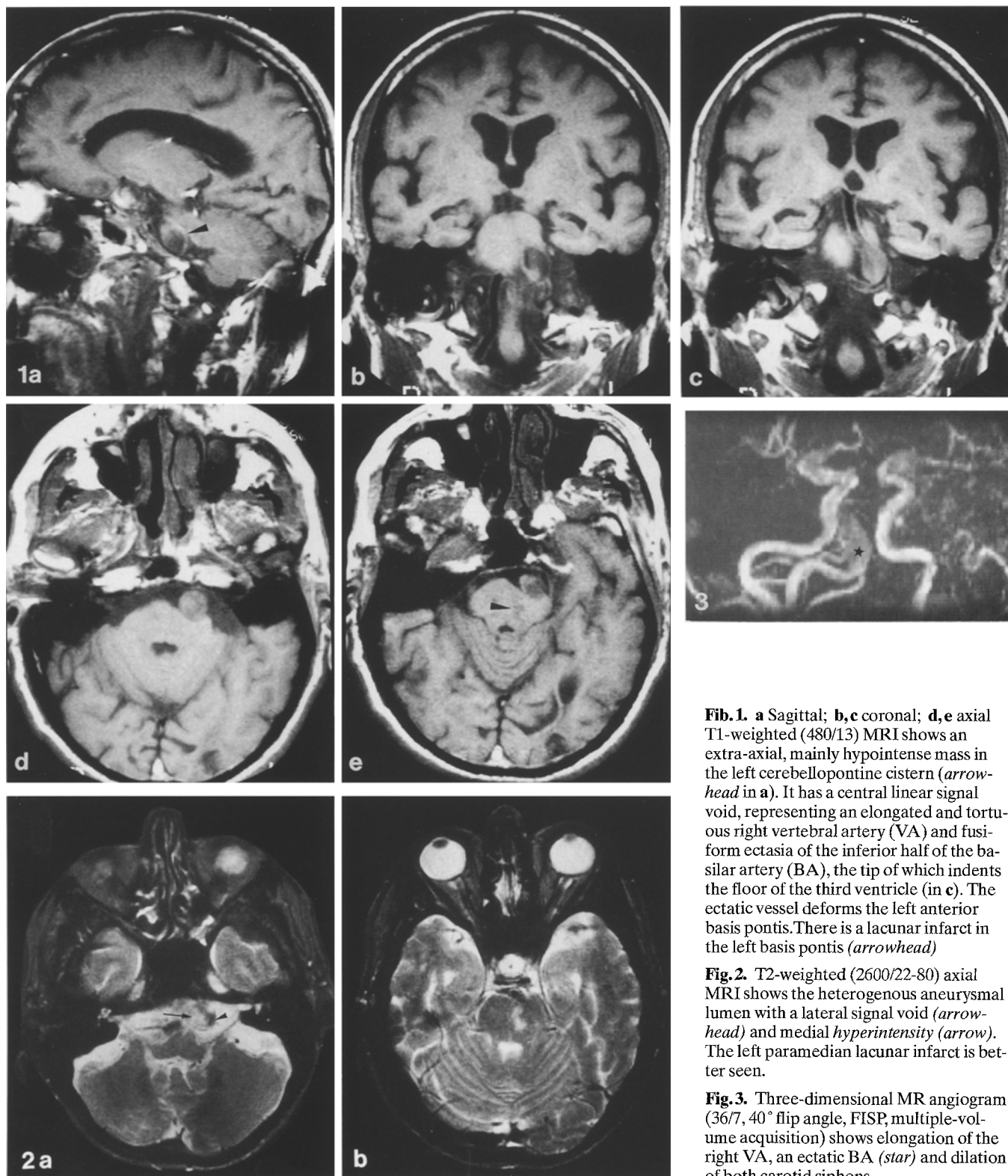


Fig. 1. a Sagittal; b, c coronal; d, e axial T1-weighted (480/13) MRI shows an extra-axial, mainly hypointense mass in the left cerebellopontine cistern (*arrowhead* in a). It has a central linear signal void, representing an elongated and tortuous right vertebral artery (VA) and fusiform ectasia of the inferior half of the basilar artery (BA), the tip of which indents the floor of the third ventricle (in c). The ectatic vessel deforms the left anterior basis pontis. There is a lacunar infarct in the left basis pontis (*arrowhead*)

Fig. 2. T2-weighted (2600/22-80) axial MRI shows the heterogenous aneurysmal lumen with a lateral signal void (*arrowhead*) and medial *hyperintensity* (*arrow*). The left paramedian lacunar infarct is better seen.

Fig. 3. Three-dimensional MR angiogram (36/7, 40° flip angle, FISP, multiple-volume acquisition) shows elongation of the right VA, an ectatic BA (*star*) and dilation of both carotid siphons

VBE is a rare cause of stroke. Yu et al. [4] found a ectasia of the vertebrobasilar system in 17, 11 of which were symptomatic, in 10,000 consecutive vertebral angiograms. Despite the fact that CT has simplified the recognition of VBE [5], the small number of patients in recent series [6–8] confirms the rarity of symptomatic cases. Symptoms

can result from compression (of cranial nerves, brain stem or CSF pathway), from stroke (ischaemia rather than rupture) or both, as in our case. The ectatic arterial wall shows pathological changes consisting of extensive defects in the internal elastic lamina and severe reticular fibre deficiency in the media [9]. Whether these changes result from

atherosclerosis [7] or from an underlying structural arterial defect [9] remains controversial. Whatever the pathogenesis, severe atherosclerosis and intraluminal thrombus usually coexist with these morphological features.

Pessin et al. [10] studied the mechanisms of infarction in patients with VBE. Thrombosis of the ectatic vessel usually causes a massive, fatal brain-stem infarct. Arterial embolism may account for infarction in the territory of the terminal or circumferential branches. A paramedian pontine infarct, as in our case, can be due to obstruction of a perforating arteriole at its origin by atheroma or intraluminal thrombus. Distortion of the perforating vessels, due to an elongated, tortuous BA, could also contribute to ischaemia. Most patients with VBE are hypertensive: arteriolar lipohyalinosis may therefore also play a causative role in lacunar infarcts. Two previous cases of PMH were reported in association with VBE [6, 8] indicating that infarction in the perforating artery territories may co-exist with large vessel obstruction [2].

MRI can be used for showing VBE and its consequences [11–15]. It shows the deformation of the pons, perianeurysmal oedema, and mass effect on the CSF pathways. Elongation and tortuosity of the vertebrobasilar arterial system can be easily demonstrated on T1-weighted coronal sections. The parent vessel, the patent portion of the aneurysmal lumen, and the arteries arising from the ectatic segment usually show signal void on T1- and T2-weighted MRI, indicating rapid or turbulent flow. Signal void is replaced by high signal intensity when flow is slow in the ectatic vessel [12]. MRI often demonstrates the continuity of one or both VA with the dilated BA. However, distinguishing VBE from a giant saccular aneurysm or an arteriovenous malformation can be difficult [13].

The structural characteristics of the dolichoectatic artery can be delineated on CT or MRI. CT is superior to MRI for detecting mural calcification. Peripheral calcification appears as a rim of high attenuation on unenhanced CT, hypointense on MRI, but the latter can be missed or mistaken for haemosiderin [13]. Partial thrombosis appears as an area of non-enhancement on contrast-enhanced CT. MRI characteristics of intraluminal thrombus are complex and variable [12].

MRA provides additional informations on intracranial vascular lesions [16, 17]. However, slow flow lesions such as a partially thrombosed giant aneurysm or vascular ectasia may be poorly visualized due to lack of flow-related enhancement [16]. In our case, a large patent lumen, with rapid or turbulent flow, explained good visualization of the VBE. Since VBE is not surgically treatable, cerebral angiography should be restricted to patients in whom the nature of the vascular lesion remains doubtful after CT and MR studies, especially when it resembles a giant saccular aneurysm or an arteriovenous malformation.

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