

# Vascular Disorders of the Posterior Circulation – An Anatomico-Clinical Overview

## INTRODUCTION

The posterior cerebral circulation consists of the vertebrobasilar system (vertebral arteries, basilar artery, posterior inferior cerebellar arteries, anterior inferior cerebellar arteries, superior cerebellar arteries, posterior cerebral arteries) (Fig. 1-3) and supplies the following anatomical structures: upper cervical spinal cord, medulla oblongata, pons, cerebellum, mesencephalon, thalami, occipital lobes, and parts of the temporal and parietal lobes (Fig. 4). Strokes in the posterior circulation comprise approximately 10% to 15% of all strokes<sup>1</sup> and are more common in men than in women<sup>2</sup>. As in supratentorial strokes, the most common etiology is ischemic (approximately 80% of cases), with the proportion of haemorrhagic strokes being similar to that seen in the anterior circulation. Although intraparenchymatous haemorrhage is slightly more frequent, while subarachnoid haemorrhage seems to be less common in the posterior territory<sup>3</sup>.

## AETIOLOGY

The risk factors for posterior circulation infarction do not differ from those for anterior circulation strokes. However, a history of prior stroke, but not transient ischaemic attacks, may be more frequently encountered in patients with posterior circulation strokes<sup>2</sup>. The structures most commonly affected by infarction are the brain stem (60%) and cerebellum (50%) and so are caused by lesions of basilar and/or vertebral arteries (up to 50%)<sup>4</sup>. The most common cause is basilar artery (BA) stenosis or occlusion (approximately 40% of patients) with the proximal and middle segments of BA being the most frequent site of occlusion<sup>5</sup>. Some patients may have stenosis and/or occlusion of the extracranial or intracranial part of the vertebral artery, or posterior cerebral arteries lesions. In rare instances, a dolichoectasy of basilar artery or vertebral artery may be encountered<sup>4</sup>.

Small artery disease is a presumed cause of stroke in 15% of patients, whereas cardiac embolism (e.g. from thrombus associated with an akinetic left ventricle, atrial fibrillation, or paradoxical embolism through a PFO) is a causative factor of stroke in approximately 13% of

patients. In an equal proportion of patients (i.e. 13%) there may be more than one possible cause of stroke including arterial stenosis and/or occlusion, lacunar lesions or a potential cardiogenic source of embolism, whilst in 10% of cases no potential cause of stroke can be identified.

However, there are two types of infarction in the posterior territory which are highly suggestive of a particular etiology. Isolated unilateral or bilateral brainstem infarcts (involving midbrain and/or pons) are associated with basilar stenosis, while isolated cerebellar infarctions are associated with cardioembolism<sup>4</sup>.

Other etiologies of stroke such as for example, dissection of basilar artery or vertebral arteries (Figure 5), are rarely reported in patients with posterior circulation disorders. Cerebral venous and/or sinus thrombosis limited to the posterior circulation territory are casuistic.

## CLINICAL FEATURES

In posterior circulation infarctions severe headache and vomiting are more frequently seen than in anterior circulation strokes<sup>2</sup>. The common signs and symptoms include bulbar or pseudobulbar palsy, vertigo and dizziness, hemiparesis, tetraparesis, cerebellar ataxia, eye movement disorders, changes in levels of consciousness and neuropsychological dysfunction. Indeed the organisation of the brainstem relative to its vascular supply leads to a number of well-recognised syndromes. So ischemic lesions in the territory supplied by posterior inferior cerebellar artery may give the lateral medullary syndrome (Wallenberg's syndrome) [characterised by ipsilateral facial sensory disturbances, nystagmus, dysphagia, dysarthria, Horner's syndrome with contralateral hemibody dissociated sensory disturbances]. The symptoms of anterior inferior cerebellar artery occlusion are similar to those of Wallenberg's syndrome, but can additionally be differentiated by the presence of limb and trunk ataxia, tinnitus, deafness, and facial nerve involvement. Superior cerebellar artery occlusion can be distinguished by the predominance of cerebellar symptoms and a trochlear nerve palsy<sup>6</sup>.

Basilar artery occlusion may give a large spectrum of neurological signs and symptoms of which the most



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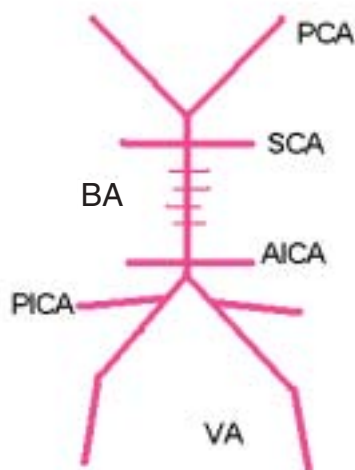


Figure 1. A schematic drawing of the posterior circulation arteries. VA: vertebral artery; PICA: posterior inferior cerebellar artery; AICA: anterior inferior cerebellar artery; BA: basilar artery; SCA: superior cerebellar artery; PCA: posterior cerebral artery.



Figure 2. Magnetic resonance angiography showing normal posterior circulation arteries (abbreviation unfolded in figure 1). SA: subclavian artery; V1-V3: extracranial segments of vertebral artery; V4: intracranial segment of vertebral artery.

frequent are motor deficits ranging from monoparesis to tetraplegia, and also dysarthria, vertigo, nausea and vomiting, headaches, alterations of consciousness, ataxia, and sensory disturbances<sup>7</sup>. Embolic occlusion of the distal part of basilar artery at the point at which it branches into two posterior cerebral arteries, gives a dramatic picture of the so called 'top-of-the-basilar syndrome'. This clinical entity is characterised by severe impairment of consciousness, usually bilateral oculomotor palsies, visual fields defects, cerebellar symptoms, and hemiplegia or tetraplegia.

Multiple infarctions in the posterior circulation are found in approximately 10% of patients with infratentorial and supratentorial infarcts being most commonly found to coexist, whilst concomitant brainstem lesions are less common. In such patients hemianopia, and cerebellar signs predominate and are due typically to vertebral and/or basilar artery steno-occlusive disease rather than cardioembolism. Multiple infratentorial strokes are less frequently seen, and are characterised by a combination of brainstem and cerebellar signs. Lacunar lesions are seen in the majority of these cases and the most uncommon scenario is multiple brainstem and posterior cerebral artery strokes<sup>8</sup>. However there exists a rare condition which consists of infarction in the posterior inferior cerebellar artery and the posterior cerebral artery territories, caused by occlusive disease of the intracranial vertebral artery, with bilateral occlusion of the posterior inferior cerebellar artery together with distal embolism to posterior cerebral artery. A term proximal-distal syndrome of the posterior circulation was coined to describe this clinical entity that is characterised by bilateral axial ataxia with visual field defects<sup>9</sup>.

Overall the most common etiology of posterior cerebral artery territory infarction is cardioembolism, followed by embolism of undetermined origin, and artery-to artery embolism although in approximately 20% of cases the etiology remains unknown. Ischemic strokes in the territory supplied by posterior cerebral arteries are usually manifest as severe headaches, visual field defects (homonymous hemianopia), sensory signs, motor deficits, and cognitive deficits<sup>10</sup>.

The occlusion of small penetrating branches of basilar artery gives rise to the lacunar lesions. There are four classical lacunar syndromes that may be linked to the posterior circulation strokes. Pure sensory stroke may be caused by the lesions confined to the pons, or thalamus.

Pure motor hemiparesis may be caused by the pontine lesion. Thalamic or pontine lacunae may give symptoms of sensorimotor stroke. Ataxic hemiparesis may be caused by a pontine lacunar lesion<sup>4</sup>.

## PROGNOSIS

The outcome in patients with posterior circulation infarction is quite good, with a 30-day case fatality of <4% and less than 20% of patients being left with severe disability after one month, and approximately 30% of patients having no disability<sup>11</sup>. However, half of the patients with BA occlusion have a poor outcome (death or severe disability) with dysarthria, pupillary disorders, decreased levels of consciousness, bulbar symptoms, multiple posterior circulation infarcts, and cardiac embolism all being predictors of poor outcome<sup>5,8</sup>.

Intraparenchymatous bleeding in the posterior fossa is usually a medical emergency, as pontine haemorrhages are lethal in 60% to 90% of cases, depending on the extent of haemorrhage and location (Figure 6)<sup>12,13</sup>. Massive pontine haemorrhages are characterised by sudden onset of coma, respiratory disturbances, tetraplegia with posturing, characteristically with pinpoint pupils, and lower cranial nerves palsies. Large cerebellar haemorrhage may present with severe headache, vomiting, hemi ataxia, nystagmus, and eventually coma. The symptoms of small thalamic hemorrhage may resemble those of lacunar infarction and may include contralateral hemisensory symptoms and usually less pronounced contralateral hemiparesis (Figure 7). In some of these cases upgaze paresis may be encountered. Large thalamic hematomas cause more complex clinical syndromes, as the affected territory is bigger and has a one-month case fatality approaching 25% - this being highest with posterolateral haemorrhages. The predictors of death are initial level of consciousness, meningeal signs, size of haemorrhage with ventricular extension, and the presence of hydrocephalus<sup>14</sup>.

## CONCLUSION

Posterior circulation strokes do not differ much from anterior circulation strokes in risk factors. However, they are distinct in their symptomatology, as different anatomical structures are involved. Different clinical symptomatology may also be explained by a more frequent arterial branch disease in the territory of the basilar artery that gives classical lacunar syndromes. Furthermore the verti-

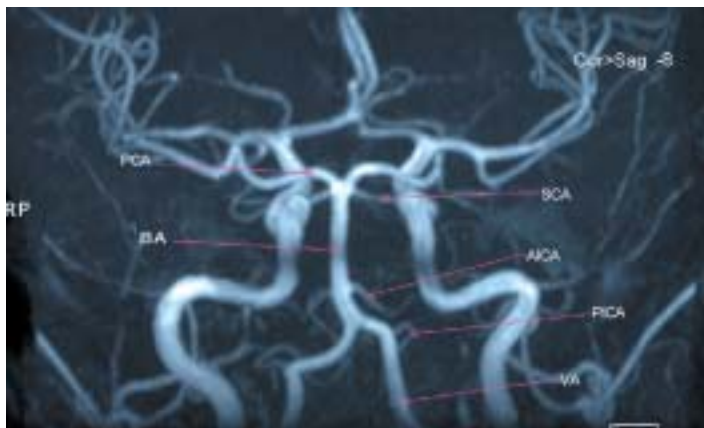


Figure 3. Magnetic resonance angiography with a detailed view of the intracranial part of posterior circulation (abbreviation unfolded in figure 1).

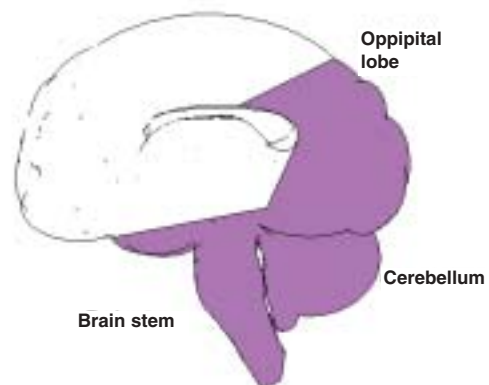


Figure 4. A schematic drawing (medial aspect of a sagittal section through the brain) of the territory supplied by posterior circulation arteries (thalamus not shown).

cal arrangement of the posterior circulation arteries as opposed to the horizontal localisation of the anterior circulation arteries may explain the preponderance for the multiple infarcts at the different levels in the posterior circulation, while multiple infarcts in the anterior circulation are much rarer.

From a clinical point of view it is important to remember that some clinical syndromes such as basilar thrombosis, or posterior fossa haemorrhage are medical emergencies and require prompt diagnosis and treatment.

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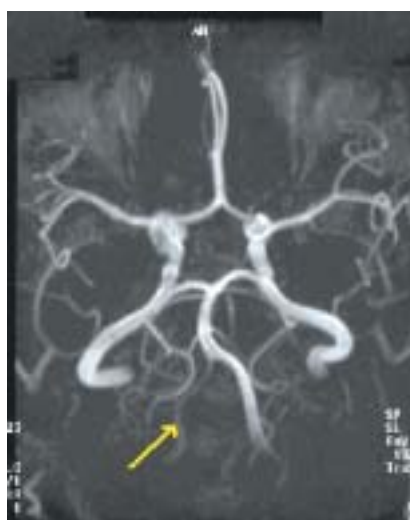


Figure 5 B.

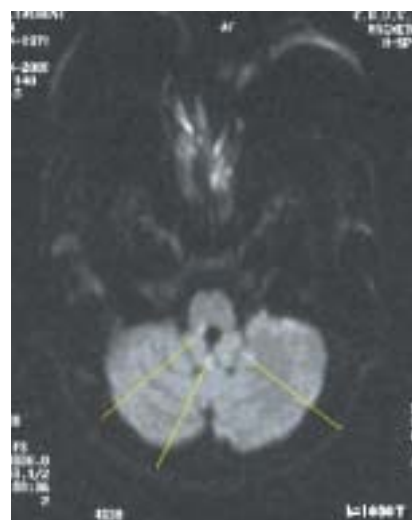
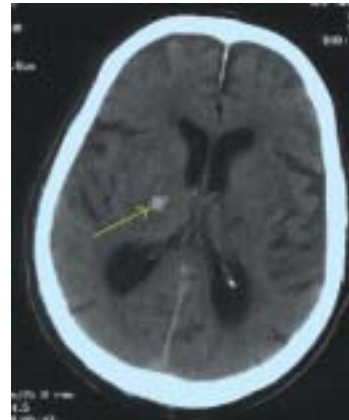


Figure 5 C.

**Figure 5 A.** Magnetic resonance (time-of-flight) angiography of neck and intracerebral vessels showing an occlusive dissection of the right vertebral artery (segment V2-V4) (A: yellow arrow: normal vertebral artery; B: yellow arrow largely diminished flow in intracranial right vertebral artery). Magnetic resonance (Diffusion-weighted imaging) showing a right lateromedullary infarction, and bilateral small ischaemic lesions (yellow lines) in the territory of posterior inferior cerebellar artery (C).



**Figure 6.** Acute phase magnetic resonance imaging T2 showing a secondary pontine haemorrhage (cavernoma).



**Figure 7.** Acute phase cerebral computed tomography showing a hypertensive right thalamo-capsular haemorrhage.