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REVIEW ARTICLE

Posterior circulation ischaemic stroke—a review part I: anatomy, aetiology and clinical presentations

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Abstract
Posterior circulation ischaemia is a clinicopathological condition with complex symptomatology associated with an infarction within the vertebrobasilar arterial system. Posterior circulation strokes account for about 20–25% of all ischemic strokes and remain a significant cause of patient disability and mortality. Diagnosis can be challenging because presenting symptoms are often non-focal and because there is a substantial overlap in symptoms and signs of ischaemia in the anterior circulation. Despite better imaging techniques, diagnosis and treatment of life-threatening conditions, such as basilar artery occlusions, are often delayed. Therefore, early detection of symptoms and causes of posterior circulation ischaemia is essential for choosing the most appropriate therapy. In this review, we summarise the anatomy, aetiology, typical presentations and characteristic findings of common strokes resulting from disease in the vertebrobasilar arterial system.

Keywords Vertebrobasilar arterial system · Posterior cerebral ischaemia · Basilar artery occlusion

Introduction

Posterior circulation ischaemia (PCI) is a clinicopathological condition associated to an infarction within the vertebrobasilar arterial system, mainly in the brainstem (48% of the cases) and in the posterior inferior cerebellar artery (PICA) territory (36% of the cases) [1]. PCI accounts for about 20–25% of all ischemic strokes and has an annual adjusted incidence of 18 per 100,000 person-years (95% confidence interval, 10/100,000 to 26/100,000) [1–3]. Even if the overall mortality at 1 month has been estimated at 3.6–11%, this condition remains a significant cause of patient disability and is associated with a high risk of recurrent stroke [4–8].

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Early recognition of PCI symptoms is essential to ensure a correct clinical diagnosis and a proper therapy can only be offered through an accurate detection of the underlying cause. This review aims to provide a smooth and reliable tool for promptly recognising PCI. For this purpose, we summarise the anatomical classification, aetiology and clinical presentations of common strokes resulting from disease in the vertebrobasilar arterial system and focus on the key differences between posterior and anterior circulation stroke.

Posterior circulation anatomy

The vertebrobasilar arterial system receives only 20% of cerebral blood flow and supplies the posterior portion of the brain including the brainstem, the thalami, the cerebellum and parts of the occipital and temporal lobes [6].

Vertebral arteries

Vertebral arteries (VAs) usually arise from the subclavian arteries (occasionally directly from the aortic arch) and travel cranially through the transverse foramina of the cervical vertebrae [9]. Both VAs join at the pontomedullary junction to form the basilar artery. VAs usually have a luminal diameter of 3–5 mm [5] and are conventionally divided into four segments (Fig. 1):

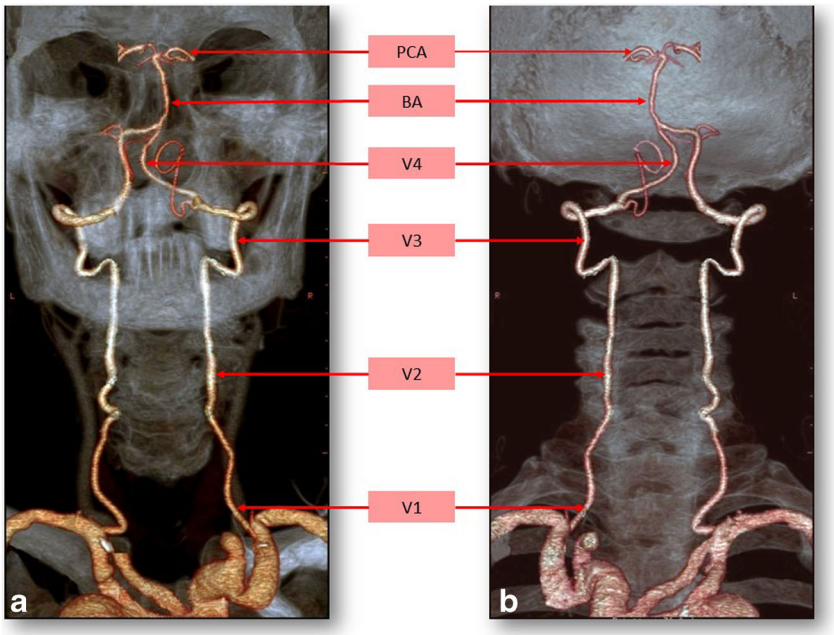

Fig. 1 Postprocessed frontal (**a**) and dorsal (**b**) CTA images of the normal extracranial and intracranial posterior circulation. The images show the four segments of the vertebral arteries (V1, V2, V3 and V4), the basilar artery (BA) and the posterior cerebral arteries (PCA)

- V1 (the pretransverse segment): from the origin to the transverse foramen of C6.
- V2 (the transverse segment): from the transverse foramen of C6 to the transverse foramen of C2.
- V3 (the suboccipital segment): from the foramen transversarium of C2 to the atlanto-occipital membrane. This segment forms a loop that allows free movements of the head and the neck [5].
- V4 (the intracranial segment): from the point where the VAs pierce the atlanto-occipital membrane to their confluence to form the basilar artery at the medullopontine junction.
- The intracranial part of each VA gives rise to the following branches:

1. The *posterior spinal artery* supplies the gracile and cuneate fasciculi and the inferior cerebellar peduncle [9].
2. The *anterior spinal artery* which unites to its fellow of the opposite side and supplies a paramedian region of the lower medulla and spinal cord [9]. Occlusion of one anterior spinal artery produces medial medullary syndrome (see later).
3. The *posterior inferior cerebellar artery* winds around the dorsolateral surface of the medulla to supply the lateral bulbar region, the posteroinferior cerebellar hemisphere and the inferior portion of the vermis [9]. Occlusion of the *posterior inferior cerebellar artery* (PICA) produces the lateral medullary syndrome (see later).

Basilar artery

Basilar artery (BA) originates at the pontomedullary junction by the union of the two VAs (Figs. 1 and 2). It travels rostrally along the anterior surface of the medulla and the pons until it bifurcates into the two posterior cerebral arteries. Branches of the BA include:

1. The *anterior inferior cerebellar artery* (AICA) arises from the proximal BA and wraps around the anterolateral aspect of the lower pons to supply the inferolateral portion of the pons and the anteroinferior surface of the cerebellum (Fig. 2) [9]. The *labyrinthine artery*, usually originating from the AICA (in ~ 15% of cases, it can also branch directly from the BA), is the main arterial supply to the vestibular apparatus and cochlea [9].
2. The *pontine arteries* are numerous small vessels, which penetrate the pons to supply the medial (paramedian branches), anterolateral (short circumferential arteries) and posterolateral (long circumferential arteries) aspects of the pons [9].
3. The *superior cerebellar artery* (SCA) arises from the distal BA, passes laterally just inferior to the oculomotor nerve and winds around the pons-midbrain junction. SCA typically supplies the lateral and posterior aspects of the upper pons and midbrain and the superior surface of the cerebellum (Fig. 2) [9].
4. The *posterior cerebral artery* (PCA) arises by the terminal bifurcation of the BA, passes laterally, above the SCA, and winds around the cerebral peduncles to supply the

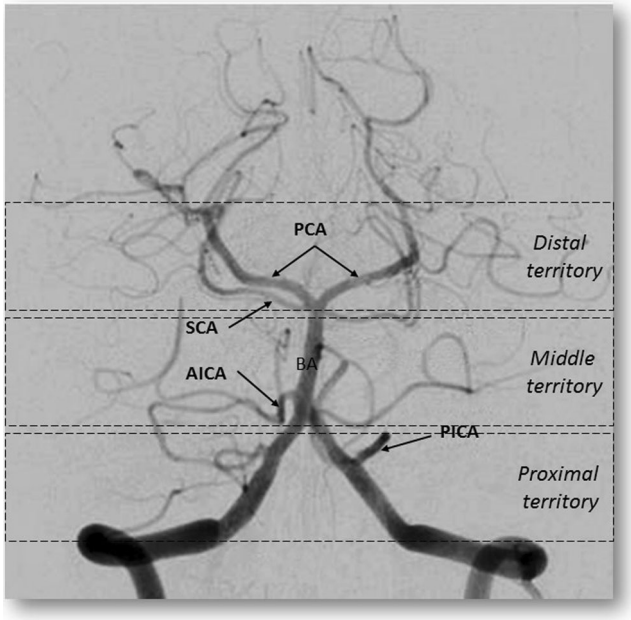

Fig. 2 Cerebral angiography showing the normal vertebrobasilar arterial system. Posterior circulation is subdivided in proximal, middle and distal intracranial territory, according to Caplan et al. [6]. AICA = anterior inferior cerebellar artery, BA = basilar artery, PCA = posterior cerebral artery, PICA = posterior inferior cerebellar artery, SCA = superior cerebellar artery

midbrain, the hypothalamus and the thalamus via *penetrating branches* (Figs. 1 and 2) [9]. Cortical branches of this artery supply the inferior and medial aspects of the temporal and occipital lobes (the posterior temporal artery, the internal occipital artery), the primary visual cortex (the calcarine artery) as well as the lateral surface of the hemisphere (the parieto-occipital artery) [9]. PCA is schematically divided into four segments: P1 (within the interpeduncular cistern) from the origin to the posterior communicating artery, P2 (within the crural and ambient cistern) from the posterior communicating artery to the posterior aspect of the midbrain, P3 (quadrigeminal segment) from the posterior aspect of the midbrain to the calcarine fissure and P4 (calcarine segment) distally to the anterior limit of the calcarine fissure [9, 10].

Common anatomical variants of posterior circulation

VAs Asymmetry in luminal diameter of VAs is common, and approximately 70% of people have a left dominant VA [5, 11].

VA hypoplasia (VAH) is a congenital variation in the size of the VAs defined by a V4 diameter of ≤ 2 mm and an asymmetry ratio of ≤ 1:1.7 of both VAs [11]. This condition has a prevalence ranging between 1.9 and 26.5% and has been shown to confer an increased probability of ischaemic stroke in the vertebrobasilar territory [11–14]. Using whole-brain CT perfusion, 42.4% of patients with VAH showed hypoperfusion in the territory of the ipsilateral PICA even in the absence of manifest PCI [11]. In another study, patients with VA asymmetry had twice as many pontine infarctions (ipsilateral to the smaller vessel) as those with symmetric VAs [15].

PICA PICA is bilaterally or unilaterally absent respectively in 2% or 10% of cases [16]. In 10% of cases, PICA arises from the BA [16]. Frequently, there is a reciprocal relationship in PICA/AICA anatomy, with an inverse relationship between the sizes of the two. A common finding is a large PICA with a small or absent ipsilateral AICA and contralateral PICA (the "PICA-AICA" variant). Alternatively, patients may have a dominant AICA and a small or absent ipsilateral PICA ("AICA-PICA" variant) [17].

BA BA fenestration refers to a duplication of a portion of the artery and usually occurs at the lower end of the vessel. The prevalence of this anatomic variant ranges between 0.3% and 0.6%, depending on the technique used, and may predispose to posterior circulation (PC) aneurysm formation [18].

Congenital asymmetric blood flow to the vertebrobasilar junction may cause BA curvature and elongation [19]. The resulting inner wall shear stress and torsion of the pontine perforating arteries may cause endothelial injury that predisposes to local thrombosis [19]. In one study by Hong et al. [19], it has been demonstrated that in 72% of patients with BA curvature, pontine infarction occurred contralateral to the side of the BA displacement.

Hypoplasia of the BA is rare and often associated with a bilateral fetal origin of PCA (see below) [20] or, less commonly, with persistent trigeminal artery. Such vessel, reported in 0.1–0.6% of angiographic or MR-angiographic series [21–23], forms an anastomosis between the carotid artery and the BA or the cerebellar arteries. Another even rarer anastomosis between anterior and PC is represented by the persistence of the hypoglossal artery [24, 25]. Finally, persistence of proatlantal intersegmental artery may form a connection between carotid artery and extracranial VAs [24].

PCA In about 10% of people, the P1 segment is absent, and the ipsilateral PCA arises directly from the intracranial internal carotid artery. This variant is referred to as a fetal PCA [26]. In 15.1% of individuals, a partial fetal PCA, an anatomic variant with a hypoplastic P1 segment, is present [26]. In such cases, especially if the anterior communicating artery is nonfunctioning, an infarct in the PCA territory may indicate that ipsilateral carotid stenosis is symptomatic [27].

The artery of Percheron The thalamus on either side is supplied by the thalamoperforating arteries, which are multiple small branches arising from ipsilateral P1 segment of PCA [28]. Variations in the thalamoperforating arteries have been

described in autoptic studies and are not uncommon [28]. Artery of Percheron is an anatomic variant in which a single unpaired thalamoperforating artery arises from the P1 segment of PCA to supply the bilateral medial thalami with a variable contribution to the rostral midbrain [29, 30]. Occlusion of this artery will lead to bilateral infarction of paramedian thalami, with or without midbrain involvement [30]. Clinical features include hypersomnolence, amnesia, coma, aphasia/dysarthria, ocular movement disorders (including vertical gaze palsy), and pupillary abnormalities in different combinations [31–33]. Of note, bilateral thalamic infarction was most commonly associated with unilateral or bilateral P1 segment hypoplasia [34].

Aetiology

Common causes

Stroke mechanisms responsible for PCI and anterior circulation ischaemia (ACI) are quite similar. Most common causes of vertebrobasilar ischaemia are, in fact, atherosclerosis, embolism, penetrating small-artery disease and arterial dissection [6, 35–37].

A. Embolism

In a large hospital registry study (the NEMC-PCR) of 407 patients with PC stroke, embolism was the most common cause (40%), and a cardiac source of embolism was reported in 24% [6]. In the Hallym stroke registry, only 11% of 591 Korean patients with PC strokes had cardiac sources of embolism [36]. In another study of 2545 Chinese patients, cardioembolism caused infarction in a significantly smaller proportion of patients with PCI (5.4%) than in patients with ACI (13.3%) [37]. The low rate in the cardiogenic embolism in PCI has been explained by the fact that the PC receives approximately 20% of cerebral blood flow so that only a fifth of cardiac emboli may end up within the PC [6].

B. Atherosclerosis

Large-vessel atherosclerotic disease is responsible for 32–35% of PC strokes [6, 38]. The most common extracranial location of atherosclerotic occlusive disease within the PC is the V1 segment of the VA [6, 39]. Atherosclerotic plaques may begin in the subclavian artery and extend into the ostia of the extracranial VAs or begin within the most proximal portion of the extracranial VAs [6, 40, 41]. The severity of PC strokes due to extracranial VA stenosis correlates with plaque characteristics, such as surface irregularity and morphology of the lesion [40, 41].

Intracranial atherosclerotic disease (IAD) is more prevalent among Asian (54%), Afro-American (11%) and Hispanic (6%) stroke patients than in Caucasians (with rates reportedly as low as 1% in the USA) [42–47]. However, IAD in Caucasians might be underestimated, as shown by the very different prevalence rates found in several reports, ranging between 1% and 43% [43, 48]. In a post-mortem study by Mazighi et al. on 339 patients with stroke (259 patients with brain infarction and 80 patients with brain haemorrhage), the overall prevalence of intracranial plaques (i.e. non-stenotic plaques and plaques inducing a stenosis of the arterial lumen > 30%) was 59.0% (95% CI, 53.8 to 64.2) and the prevalence of intracranial plaques inducing a stenosis of the arterial lumen > 30% was 37.2% (95% CI, 32.0 to 42.3) [48]. In the same study, BA appeared to be the predominant location for stenosis > 30% in 15.9% of patients [48]. In a recent prospective, multicenter, hospital-based, transcranial ultrasound study on 1134 Italian patients with acute ischemic stroke, IAD has been identified as a cause of first-ever acute ischemic stroke in the 8.7% of the overall study population and in the 3.2% of patients with PCI [49].

Intracranial PC occlusive lesions may occur anywhere along the BA and in the V4 segment of the VAs at or near the vertebral-basilar artery junction, whereas atherosclerosis of the PCAs is less common [6, 41, 50].

Ischaemia due to large-vessel atherosclerotic disease may result from tissue hypoperfusion, in situ thrombosis, or artery-to-artery thromboembolism. The intra-arterial embolism is the most likely cause of PC ischaemia in patients with proximal extracranial VA stenosis, as suggested by results from the NEMC-PCR registry as well as by the detection of circulating emboli on transcranial Doppler ultrasound distal to the vertebral stenosis [6, 51]. Vertebrobasilar insufficiency due to haemodynamic changes is a rare cause of PC stroke, demonstrated only in 13 of 407 patients of the NEMC-PCR registry and usually caused by bilateral intracranial VA disease or tandem extracranial and intracranial lesions [6, 35].

C. Penetrating small-artery disease

Occlusion of single deep penetrating arteries arising from the intracranial VA, BA and PCA causes lacunar infarcts in the PC. Topographically, in a study on the correlations between lesion patterns on neuroimaging and the causes of stroke (TOAST etiological groups), small-artery disease has been found to be the main cause of PCA territory infarcts (52.1%) [52].

Most of PC lacunar infarcts are caused by lipohyalinotic thickening of the wall of the small arteries [53]. Alternatively, an embolic cause is assumed, either artery-to-artery embolism or cardioembolism [53]. Moreover, Caplan first demonstrated in 1989 that a single subcortical infarction (SSI), traditionally called "lacunar infarction," can also be produced by atherosclerosis occurring in the parental artery by blocking the orifice of branching arteries [53, 54]. SSI associated with parental artery disease (SSIPAD) is relatively common in Asian

populations where intracranial atherosclerosis is prevalent [54]. In a recent study of Taiwan patients, BA atheromatous branch occlusive disease was responsible for 28.0% of PC strokes [55]. SSIPAD caused by PCA atherosclerosis has been found in 7–22% patients with lateral thalamic infarction [54, 56, 57]. In an angiogram study by Kim, 67 out of 123 patients with lateral medullary infarction were considered to have SSIPAD [58]. In another MRA-based study of 86 patients with medial medullary infarction, VA atherosclerotic disease was identified in 53 (62%) patients, with stroke resulting primarily via SSIPAD [59].

D. Arterial dissection

Arterial dissection is more frequently a cause of PCI than of ACI [6, 60]. It has been recently reported that up to one-quarter of PCI is related to an arterial dissection [55, 60]. VA dissection has an estimated incidence of 1–1.5 per 100,000 per year and predominantly affects patients in their fourth decade of life [60, 61]. VA dissections are usually found in either the V2 or V3 segment (where the artery winds around the lateral masses of the upper cervical vertebrae) [62]. In the Asian population, cervicocranial arterial dissection involves the intracranial more frequently than extracranial arteries [60, 63–66]. In a recent series of 74 Asian patients with PCI associated with arterial dissection, 87.5% of them had an extracranial VA dissection extending to intracranial VA and/or BA [60]. However, intracranial extension has been demonstrated to carry a higher risk of haemorrhagic events (i.e. subarachnoid haemorrhage) and mortality [67].

Cervical artery dissection may occur spontaneously or result from a cervical trauma, including neck manipulation [68]. In VA dissection as well as in carotid dissection, stroke seems to be related to an embolism from a thrombus at the site of dissection, rather than a reduction in haemodynamic flow secondary to luminal compression [68].

Uncommon causes of PCI

Intracranial artery dilatative arteriopathy

Intracranial artery dilatative arteriopathy (IADE) refers to the dilatation, elongation, widening and tortuosity of the intracranial VAs and BA [69]. Dolichoectasic arteries are characterised by an abnormally large external diameter and a thin arterial wall [69]. Histologically, the affected arteries show deficiencies in the muscularis and internal elastic lamina with irregular thickness and fibrosis of the media [69]. Degeneration of the internal elastic lamina (tunica media) has well documented in pathological specimens of BA and MCA of patients with IADE and lysosomal storage disorders, such as Pompe disease and Fabry disease [70–72]. Moreover, vertebrobasilar IADE has been associated with aortic dilatation, ectasic coronary arteries, Marfan syndrome and autosomal dominant polycystic kidney disease [73].

The following mechanisms may explain the pathophysiology of clinical manifestations of the IADE:

- Acute brain ischaemia. The estimated 5-year complication risk for ischemic stroke is 17.6% (95% CI, 12.4–22.8) [73]. A reduction in flow velocity in the dilated arteries predisposes to a local thrombus formation that may occlude the affected vessel or cause an artery-to-artery embolism. Alternatively, elongation and angulation of arteries can stretch and distort the orifices of arterial branches leading to the occlusion of penetrating branches [35, 73].
- Compression of cranial nerves, brainstem or third ventricle [35].
- Vascular rupture with haemorrhagic stroke or subarachnoid haemorrhage (SAH) [73].
- Long-term prognosis of IADE has been associated with vertical elongation, lateral displacement and diametric changes of dilated arteries over time [74].

1. Subclavian steal syndrome
2. Large-vessel vasculitides
3. Fabry disease
4. MELAS
5. Posterior reversible encephalopathy syndrome (PRES)
6. Bow hunter syndrome [75]

The global prevalence rate of rare causes of PCI, as assessed in several large stroke registries, is variable, ranging between 3% and 26% [55].

Clinical features

The following factors contribute to making the diagnosis of PCI challenging:

- Due to the broad area of brain tissue supplied from the vertebrobasilar arterial system, PCI usually gives rise to a broad range of signs and symptoms and rarely causes only one symptom.
- Ischemic events in the anterior circulation (AC) and PC can share some clinical features (i.e. hemiparesis, hemianopsia and dysarthria) so that it can be challenging to identify the vascular territory by clinical symptoms and signs alone [6]. Argentino et al. found that approximately 10% to 20% of patients with a very early clinical diagnosis (within 5 h of the onset) of presumed AC stroke eventually had a PCI [76]. In a study that compared the frequency of symptoms and signs in the AC and PC in a series of 1174 consecutive acute stroke patients, the authors observed that several neurological deficits generally associated with

ACI, such as homolateral paralysis, central facial/lingual palsy and hemisensory deficits, were the most frequent clinical features also in patients with PCI [77]. In another recent study, aiming to identify specific features of PC and AC strokes, patients with PCI more often showed decreased consciousness, visual field defects and vestibulocerebellar signs but less hemisyndromes, dysarthria and cognitive symptoms compared to patients with ACI [78].

Furthermore, it has been demonstrated that the prehospital triage face-arm-speech-test (F.A.S.T.) score, developed to identify patients with acute stroke, is less useful in the diagnosis of PC ischemic events than carotid artery events [79]. Likewise, the National Institutes of Health Stroke Scale (NIHSS), commonly used to evaluate the signs and symptoms of stroke, tends to underestimate the severity of PCI [80]. Recently, the expanded-NIHSS (eNIHSS) has been proposed [81]. This scale adds signs more specific for PC events such as vertical gaze palsy, nystagmus, Horner syndrome, IX and XII nerve palsy and truncal ataxia [81].

- Non-focal symptoms not fully meeting the National Institute of Neurological Disorders and Stroke (NINDS) criteria for TIA, such as non-rotatory dizziness, vertigo, dysarthria, wooziness, confusion, headache and amnesia, are frequent in patients with PCI [82]. In a prospective, population-based incidence study in Oxfordshire, UK (Oxford vascular study), only 8% of transient brainstem symptoms preceding a vertebrobasilar stroke fulfilled the NINDS criteria for TIA [83]. In another observational study in the Netherlands, focal symptoms were associated to non-focal symptoms (i.e. unsteadiness, non-rotatory dizziness or a general feeling of being unwell) in 70% of patients with symptomatic VA stenosis [84]. Transient non-focal brainstem symptoms have been associated with the risk of subsequent stroke in the PC and seem to occur more frequently in patients with asymptomatic VA stenosis than CA stenosis [84].
- Symptoms and signs pointing to brainstem involvement and having a high diagnostic specificity for PCI, such as crossed motor deficits, crossed sensory deficits, oculomotor nerve palsy and Horner's syndrome, have a low prevalence [77].

Common symptoms of PCI

The frequency of the most common posterior circulation signs and symptoms has been evaluated in three single large centre observational studies: the NEMC-PCR, the ischemic posterior circulation stroke in the state of Qatar registry (IPCS-SQR) and the Chengdu Stroke Registry (CSR) [6, 7, 82, 85] (Table 1).

The most common signs are unilateral limb weakness, facial palsy, gait ataxia, dysarthria and nystagmus. Symptoms usually reported by patients with PCI are vertigo/dizziness, nausea and vomiting, headache and alteration of consciousness [6, 7, 82, 85].

Syndromes specific of PC vascular territories

To describe the location of infarcts in the vertebrobasilar arterial system, PC has been schematically subdivided by Caplan into proximal, middle and distal intracranial territories (Fig. 2) [6].

The proximal territory includes regions supplied by the intracranial VA: the medulla oblongata and the PICA-supplied cerebellum (Table 2).

The middle territory includes brain regions supplied by the BA up to its SCA branches: the pons and the AICA-supplied cerebellum (Table 3).

The distal territory includes regions supplied by the rostral BA, SCAs, PCAs and their penetrating branches: midbrain,

Table 1 Common signs and symptoms of PCI

		NEMC-PCR %	IPCS-SQR %	CSR %
Signs	Focal weakness	38	61	63.2
	Ataxia	31	65	31.5
	Facial/lingual palsy	31	65	40.7
	Nystagmus	24	48	11.9
	Dysarthria	28	64	25.5
Symptoms	Vertigo/dizziness	47	75	18.9
	Headache	28	–	17.5
	Nausea or vomiting	27	60	33.8
	Disturbed consciousness	5	18	10.3

NEMC-PCR the New England Medical Center Posterior Circulation Registry, *IPCS-SQR* the ischaemic posterior circulation stroke in the state of Qatar registry, *CSR* the Chengdu Stroke Registry

Table 2 Proximal intracranial posterior circulation syndrome

Vascular territory	Side	Clinical findings	Anatomy
		Medial medullary syndrome (Dejerine syndrome)	
ICVA	Contralateral	Brachio-crural hemiparesis	Pyramidal tract
		Hemibody loss of tactile, vibration and position sense	Medial lemniscus
	Ipsilateral	Tongue weakness +/− atrophy	Hypoglossal nucleus
		Lateral medullary syndrome (Wallenberg syndrome)	
ICVA	Contralateral	Hemisensory loss of pain and temperature below the face	Spinothalamic tract
PICA	Ipsilateral	Facial sensory loss of pain and temperature	Spinal trigeminal nucleus
		Dysmetria arm and leg, gait ataxia	Restiform body, cerebellum
		Nystagmus, nausea/vomiting, vertigo	Vestibular nucleus
		Dysphagia, dysphonia	Nucleus ambiguus
		Horner's syndrome	Descending sympathetics
		Hemimedullary infarction	
ICVA	Contralateral	Symptoms of lateral medullary infarct	See above
PICA		Brachio-crural hemiparesis	Pyramidal tract
		Cerebellar infarction	
PICA	Ipsilateral	Labyrinthine vertigo syndrome	Medial vermis
		Vertigo + nystagmus, gait ataxia, truncal lateropulsion, limb incoordination	Lateral cerebellar hemisphere

ICVA intracranial vertebral artery

thalamus, SCA-supplied cerebellum and PCA territories [6] (Table 4).

Non-specific symptoms of PCI

1. *Dizziness and vertigo* are two terms used interchangeably by patients to indicate common non-specific symptoms. Dizziness is responsible for nearly 7.5 million ambulatory visits per year and is usually described as a feeling of light-headedness or lack of mental clarity [86]. Specifically, vertigo more describes a sensation of spinning and usually indicates dysfunction of the peripheral vestibular or central vestibulo-cerebellar system [86].

Vertigo and/or dizziness caused by posterior circulation disease is usually associated with other brainstem or cerebellar

Table 3 Middle intracranial posterior circulation syndrome

Vascular territory	Side	Clinical findings	Anatomy
		Locked-in syndrome	
Proximal BA	Bilateral	Quadriplegia	Bilateral cortical spinal tracts
		Bifacial paralysis	Bilateral corticobulbar tracts
		Horizontal gaze paralysis	Bilateral fasciculus of CN VI
		Dysarthria, tongue and mandibular weakness	Bilateral corticobulbar tracts
		Inferior ventral pontine syndrome	
Proximal BA	Contralateral	Arm and leg weakness	Pyramidal tracts
	Ipsilateral	Horizontal gaze paralysis	CN VI
		Nuclear facial palsy	CN VII
		Inferior medial pontine syndrome	
Proximal BA	Contralateral	Arm and leg weakness	Pyramidal tracts
		Hemisensory loss	Medial lemniscus
		Internuclear ophthalmoplegia	Medial longitudinal fasciculus
	Ipsilateral	Horizontal gaze palsy	PPRF or CN VI
		Facial nerve palsy	VII nucleus/fascicle
		Lateral pontine syndrome (Marie-Foix syndrome)	
Proximal BA	Contralateral	Arm and leg weakness	Corticospinal tracts
		Hemisensory loss of pain and temperature	Spinothalamic tracts
	Ipsilateral	Dysmetria arm and leg	Cerebellar tracts
		Ventral mid-pontine syndrome	
Middle BA	Contralateral	Arm and leg weakness	Corticospinal tracts
		Tegmental mid-pontine syndrome (Di Grenet syndrome)	
Middle BA	Contralateral	Hemisensory loss of pain and temperature	Spinothalamic tracts
		Hemibody loss of tactile, vibration and position sense	Posterior columns
	Ipsilateral	Global anaesthesia or thermal analgesia of the face. Sometimes paralysis of the masticatory muscles	V nucleus

CN cranial nerve, *PPRF* paramedian pontine reticular formation

Table 4 Distal intracranial posterior circulation syndrome

Vascular territory	Side	Clinical findings	Anatomy
Top of the basilar syndrome			
Distal BA	Bilateral	Somnolence, vivid hallucinations and dreamlike behavior	Ascending reticular activating system
		Vertical gaze paralysis	Superior colliculi
		Cortical blindness, Balint's syndrome (optic ataxia, loss of voluntary but not reflex eye movements, simultanagnosia[a]), amnestic dysfunction, and agitated behavior	Temporal and occipital lobes of both sides
Mesencephalic dorsal tegmental syndrome (Mills' syndrome)			
SCA	Contralateral	Thermal analgesia of the face, arm, trunk and leg	Spinothalamic tracts
	Ipsilateral	Limb ataxia	Superior and middle cerebellar peduncle; superior cerebellar hemisphere
		Horner's syndrome	Descending sympathetics
Ventral mesencephalic syndrome (Weber's syndrome)			
Distal BA	Contralateral	Hemiparesis	Corticospinal tracts
	Ipsilateral	Oculomotor nerve palsy	Oculomotor nerve fibres
Thalamic pain syndrome (Dejerine-Roussy syndrome)			
PCA	Contralateral	Hemisensory loss of superficial and deep sensation	Posteroinferior thalamus
		Hemibody pain	Posteroinferior thalamus
Unilateral PCA syndrome			
PCA	Contralateral	Homonymus hemianopsia (with macular sparing)	Occipital lobe
		Achromatopsia	Ventral occipital cortex
		Alexia without agraphia	Dominant occipital lobe plus splenium of corpus callosum
Bilateral PCA syndrome (Anton's syndrome)			
PCA	Bilateral	Cortical blindness	Both occipital lobes
		Unawareness, denial of blindness, confabulations, visual hallucinations	Both occipital lobes
PCA-MCA border zone regions syndromes			
PCA	Bilateral	Prosopagnosia	Bilateral ventral-mesial-occipital-temporal border zones
		Balint's syndrome	Bilateral occipital-parietal border zones
		Transcortical sensory aphasia	Left temporal-parietal border zone

[a] Simultanagnosia: inability to perceive the visual field as a whole

symptoms. When occurring isolated, these symptoms rarely have a vascular cause. In a study of 1666 patients presenting to the emergency department with dizziness or vertigo, stroke or TIA was diagnosed only in the 3.2% of cases [87]. However, in some cases, isolated vertigo may be due to infarcts in the cerebellar flow territory of the medial branch of the PICA, as well as in the cerebellar nodulus, vestibular nucleus and in the entry zone of the vestibular nerve [88]. Furthermore, rarely, and almost exclusively in patients with diabetes, the occlusion of the labyrinthine artery that usually branches from the AICA may damage the inner ear leading to prolonged vertigo, unilateral hearing loss or both [35, 86, 88].

When vertigo occurs isolated and the neurologic examination is normal, it is not easy to establish the central or peripheral origin of the symptomatology. Usually, central vertigo has an acute onset, lasts days to weeks, is often associated with neurological findings and, if exacerbated by head positioning, appears without latency [86, 88].

Head-Impulse-Nystagmus-Test of Skew (HINTS) is a useful bedside test to distinguish between vertigo caused by peripheral or by central lesions [89]. HINTS has three parts, as synthetically explained below.

- Head impulse test: Subject in the supine position fixes eyes on a central distant target. Rotate head quickly and unpredictably from side to side by about 15°. Normal response is the eyes remain fixed on the target. The abnormal response is that the eyes are dragged off of the target followed by a saccade back to the target when rotating the head toward the side of peripheral vestibular damage.
- Nystagmus: Peripheral vestibular lesions are associated with nystagmus that is always in the same direction, while brainstem and cerebellar lesions cause nystagmus that changes direction with different position of gaze.
- Skew deviation: The test involves covering one eye and seeing if there is a vertical shift in the eye when uncovering. Brainstem and cerebellar lesions usually cause a slight skew deviation.

It has been reported that a normal head impulse, alternating nystagmus and presence of skew deviation have 100% sensitivity and 96% specificity to identify a central cause of vertigo [89] (Table 5).

2. *Headache* in PCI is present in the 46–70% of cases and seems to be caused by irritation of trigeminovascular afferents located in the brainstem [90]. Patients with cerebellar infarcts usually have unilateral headaches ipsilateral to the cerebellar lesions [91].

Table 5 HINTS test

	Brainstem or cerebellar lesions	Peripheral vestibular lesions
Head impulse test	Normal on both sides	Abnormal on one side
Nystagmus	Alternating (fast phase alternating directions)	Unidirectional (horizontal nystagmus increasing in intensity with gaze toward the fast phase)
Skew deviation		Absent

3. *Drop attack* is a sudden loss of postural tone and falling without warning [35, 82]. There is usually no recognised loss of consciousness, and the patient can report on the event. PCI rarely causes isolated drop attacks. Brainstem ischaemia affecting corticospinal tracts subserving motor control of the limbs, in fact, usually causes persistent weakness and is almost always associated with symptoms or findings related to brainstem or cerebellar dysfunction [6, 82].

Differential diagnosis

Several disorders presenting with symptoms and signs of brainstem involvement may mimic PCI. In this regard, it is not useless to emphasize that, in addition to a rational use of instrumental investigations, a scrupulous anamnesis and an accurate neurological examination are indispensable for correctly guiding the diagnosis. Below are some diseases that, if not properly recognized, could incorrectly be diagnosed as PC strokes.

Any rapidly progressive clinical condition with multiple cranial nerve dysfunctions (i.e. cranial polyradiculitis, Miller-Fisher syndrome, botulism or myasthenic crisis) can potentially be mistaken for a brainstem lesion. Clinical findings (reflexes), CSF, nerve conduction studies and serum antibodies ensure a correct diagnosis [92].

Likewise, a careful analysis of clinical findings, as well as of the history, blood chemistry and EEG, allows to quickly recognize *toxic or metabolic disturbances* (i.e. drug abuse or hypoglycaemia) that may present with features resembling cerebrovascular disease.

Central pontine myelinolysis and Wernicke's encephalopathy usually present with brainstem deficits. A history of rapid correction of hyponatraemia or of poor nutritional intake will clarify the diagnosis.

Neuroinflammatory disorders, such as sarcoidosis or Behçet's disease, may acutely affect the brainstem. However, these diseases often have systemic clinical features which are useful for correctly guiding the diagnosis [92].

CNS infection by viruses (i.e. Epstein-Barr virus or West Nile virus), bacteria (i.e. *Listeria monocytogenes*) or fungi may mimic stroke [93]. Clinical findings, cerebrospinal fluid examination and MRI features usually help to get a correct diagnosis.

Acute intracranial haemorrhage affecting brainstem, subarachnoid haemorrhage and tumour mimicking ischaemic stroke can be differentiated from PCI only by imaging [93].

In PRES, due to the involvement of the occipital lobes, visual defects are common and may mimic a PCA stroke. A history of severe hypertension, immunosuppression, renal failure or eclampsia, as well as the finding of bilateral posterior subcortical vasogenic oedema in neuroradiological examinations, will help to clarify the diagnosis [94].

Extensor jerks and spasms and decerebrate posturing arising with BAO are sometimes mistaken for *grand mal seizures* [95]. Likewise, convulsive movements similar to seizures can be seen in brainstem and thalamic strokes. The presence of pupillary and eye movement abnormalities should alert the clinician to the correct diagnosis [96].

Finally, in the presence of a headache associated with PC symptoms, a clinician should also consider the diagnosis of a *migraine with brainstem aura*. This rare type of primary headache disorder, previously referred to as basilar migraine, is characterized by attacks preceded and/or accompanied by transitory focal neurologic symptoms pointing to dysfunction in the region supplied by the BA and its branches [97]. The diagnosis is based on the finding of at least two migraine attacks accompanied by at least two of the following fully reversible symptoms: dysarthria, vertigo, tinnitus, impaired hearing, double vision, ataxia and decreased level of consciousness [97, 98].

Conclusion

PCI is a potentially life-threatening clinicopathological condition associated with a complex and fluctuating symptomatology. Diagnosis can be challenging because anatomical variants of PC are frequent and presenting symptoms are often non-focal. Moreover, there is a substantial overlap of symptoms and signs in the PC and AC strokes, even if patients with PCI more often show at onset decreased consciousness and vestibulo-cerebellar signs. Most common causes of vertebrobasilar ischaemia are atherosclerosis and embolism.

SSIPAD is relatively common in Asian populations where intracranial atherosclerosis is prevalent.

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Compliance with ethical standards

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We performed a review of the literature about the state of art of vertebrobasilar stroke; therefore, our work did not involve neither human participants nor experiments on animals.

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