Clinical presentation of vertebrobasilar stroke

Vanessa Carvalho, MD[a,*], Vitor Tedim Cruz, MD, PhD[a,b]

Abstract

Vertebrobasilar (VB) stroke is responsible for 20% of all strokes and transient ischemic attacks. Due to the vast cerebral territory it supplies, VB ischemia can present with a wide range of symptoms and signs, sometimes even overlapping with carotid circulation stroke. Furthermore, brain computed tomography, usually performed as initial imaging modality, has a suboptimal visualization of the posterior fossa, making VB stroke an even more challenging diagnosis to any physician. Hence, awareness of the vertebrobasilar anatomy and the clinical presentation of VB ischemia is crucial to promote early recognition of this disorder.

Keywords: cerebrovascular disorders, stroke, vertebrobasilar stroke

Introduction

It is estimated that nearly a fourth of all transient ischemic attacks (TIA) and strokes occur in the vertebrobasilar (VB) vascular territory.[1] Although traditionally VB stroke is regarded as having a more benign outcome when compared to anterior circulation stroke, data is still conflicting, with some studies showing a higher impairment in VB stroke patients,[2] with 21% of death or major disability at 3 months.[3]

Furthermore, screening tools commonly used to assess patients likely to have an acute stroke, such as the Face Arm Speech Test, or to determine patients with TIA or minor stroke with high risk of recurrence (ABCD2 score) have been primarily evaluated in groups of unselected patients with ischemic events, most of them with anterior circulation strokes. Thus, both scales are less effective in the diagnosis and identification of high risk cases of posterior circulation ischemic events,[4] highlighting the importance of the recognition of the VB stroke presentation.

Our purpose is to review the anatomy and the clinical presentation of VB stroke.

Anatomy

The VB vascular system comprises the vertebral, basilar and posterior cerebral arteries and their branches. It feeds the posterior region of the brain, including the brainstem, the thalamus, the cerebellum and areas of the occipital and temporal lobes.[5]

Starting with the vertebral artery, which arises from the subclavian artery on each side, it divides into 4 segments, 3 extracranial and 1 intracranial. The first segment (V1) follows the origin of artery until the entrance in the transverse process of the fifth or sixth cervical vertebrae. The second part (V2) courses within the intervertebral foramina, exiting behind the atlas, giving rise to the third portion (V3) that runs in the direction of the foramen magnum. After piercing the dura and arachnoid at the base of the skull, the fourth segment runs intracranially, and meets the contralateral artery at the midline, in the medullo-pontine junction, to form the basilar artery. This fourth segment gives rise to the posterior inferior cerebellar artery and to the anterior spinal artery (ASA). Of note, asymmetry in diameter is common in the vertebral arteries, and atresia can occur in 15% of the normal individuals.[6]

Cranially, the basilar artery runs in the ventral surface of the pons and gives rise to several important branches, namely the pontine branches, both paramedian, short circumferential and long circumferential, the anterior inferior cerebellar artery and the superior cerebellar artery (SCA). At the interpeduncular fossa, after giving rise to the SCA, the basilar artery divides into the 2 posterior cerebral arteries (PCA). In most patients, the latter often receive contribution from the anterior circulation through the posterior communicating artery. Moreover, in 10% of individuals, PCA arises solely from the carotid artery, the "fetal variant" of the PCA.[7] The PCA is responsible for the vascular supply of the posterior temporal and occipital cortex, and also gives small branches to the midbrain, thalamus, hypothalamus and corpus callosum.[5,8]

Caplan et al, further divided the vertebrobasilar system into proximal intracranial, middle intracranial and distal intracranial territories. Each one implies a different probability of stroke.[3,9]

Clinical presentation

Regarding the clinical presentation of VB stroke, in a clinical series of 407 patients, the most common symptoms experienced by patients were dizziness (47%), unilateral limb weakness (41%), dysarthria (31%), headache (28%), vomiting and nausea (27%); as for clinical signs, the most frequent were unilateral limb weakness (38%), gait ataxia (31%), unilateral limb ataxia

Carvalho and Cruz Porto Biomed. J. (2020) 5:6

Porto Biomedical Journal

(30%), dysarthria (28%) and nystagmus (24%).[10] Hence patients with VB ischemia can present not only with symptoms and signs suggesting posterior circulation but also with symptoms that overlap with carotid system ischemia or with unspecific symptoms such as nausea, vomiting and headache. Signs and symptoms that should prompt us to suspect of VB ischemia are vertigo, ataxia or gait unsteadiness, bilateral sensorimotor deficit, respiratory disfunction, consciousness impairment, cranial nerve (CN) impairment and crossed deficits.[8] However, most patients do not present with a single symptom or sign but with a cluster of manifestations, mirroring the ischemic area. This syndrome can help us to localize the stroke and also to deduce the underlying mechanism: for instance, medial strokes in the brainstem are usually due to lesions of the paramedian branches of the basilar or vertebral artery, and hence due to small vessel disease, while lateral strokes are more likely indicative of disease of the larger circumferential vessel that supplies the region.

Before we discuss the posterior circulation syndromes (see Table 1), we must understand the organization of the brainstem. The brain derives from the anterior end of the neural tube. The neural tube has a ventral portion (basal plate) and a dorsal portion (alar plate). The basal plate will contain the motor neurons and the alar plate will contain the sensory neurons. The development of the fourth ventricle will displace the alar plates laterally, hence the CN nuclei that are ventral and medial are motor and those that are dorsal and lateral are sensory.[11]

Furthermore, we can localize the cranio-caudal level of the lesion using the impaired CN: if there is sign of dysfunction of the glossopharyngeal, vagus, accessory, or hypoglossal (IX–XII) nerves/fascicles, the lesion must lie in the medulla; if the abducens, the facial or the vestibulocochlear (VI–VIII) nerves are impaired, it localizes to the pons and, if there are signs of dysfunction to the oculomotor or the trochlear nerve (III and IV), the lesion is likely in the midbrain. Of note, the trigeminal complex has 3 nuclei that extend from the midbrain until the upper cervical cord, so isolated facial sensory impairment itself does not localize the lesion. Each section of the brainstem can be further divided into 3 longitudinally organized regions: the basis, more anteriorly, where the corticospinal and corticobulbar tracts lie, the tegmentum, in the middle, where we can find most structures, including the CN nuclei and the sensory pathways, and, lying more dorsally, the roof or tectum.[8,12]

Medullary infarctions can be grossly divided into medial and lateral, the latter being far more frequent.[13] Medial medullary stroke (or Dejerine syndrome) is usually due to occlusion of the ASA or of median branches of the vertebral artery. The clinical presentation comprises contralateral hemiparesis (pyramidal tract), contralateral loss of vibration and postural sensation (medial lemniscus) and ipsilateral paresis of the tongue (hypoglossal nucleus/fascicle).[14] Lateral medullary infarct (or Wallenberg syndrome), is usually incomplete, and the classical triad includes ipsilateral Horner syndrome (descending sympathetic pathway), ipsilateral ataxia (inferior cerebellar peduncle) and contralateral hemisensory loss (ascending spinothalamic tract).[15] Patients can also present ipsilateral intention tremor (inferior cerebellar peduncle), ipsilateral facial sensory impairment (spinal tract and nucleus of the trigeminal nerve), saccadic lateropulsion (characterized by an undershoot of contralaterally directed saccades, overshoot of ipsilaterally directed saccades and ipsilateral deviation of vertical saccades),[16] vertigo, nausea and contralateral nystagmus (vestibular nuclei), dysgeusia (solitary nuclei) and paralysis of the ipsilateral palate,

dysarthria and dysphonia (ambiguus nuclei). Other possible clinical symptoms from lateral medullary dysfunction are palatal myoclonus (inferior olive) and skew deviation and diplopia due to lesion to the vestibular nuclei.[17] When both medial and lateral medulla are afflicted, usually due to a VA occlusion, a hemimedullary syndrome (or Babinski syndrome) occurs, with simultaneous contralateral hemiparesis and hemisensory loss, ipsilateral ataxia, Horner syndrome, facial sensory loss, tongue weakness and dysarthria.[8,18]

The pons can be divided into 4 groups, according to its vascular supply: the anterior (further divided into anteromedial and anterolateral), lateral and tegmental regions. Infarction in the anteromedial and anterolateral region are usually due to occlusion of paramedian and circumferential pontine branches of the basilar artery. Lesions in this region can damage the corticobulbar, corticopontocerebellar and corticospinal tracts, leading to contralateral hemiplegia or hemiparesis, facial palsy, ataxia, dysphagia, dysarthria and, less frequently loss of proprioception (medial lemniscus), ipsilateral peripheral facial palsy (facial nucleus/fascicle), ipsilateral lateral rectus palsy (abducens fascicle) and paresis of ipsilateral horizontal gaze (abducens nucleus). If the Medial longitunal fasciculus (MLF) is damaged, the patient can present with internuclear ophthalmoplegia (INO): the ipsilateral eye is unable to adduct, and the contralateral eye can abduct but has horizontal nystagmus when gaze is directed to the contralateral side. Convergence is usually preserved. The classical brainstem syndromes of Raymond and Millard-Gubler can occur due to ventromedial pontine infarctions and are further described in Table 1. Ventrolateral infarctions are rarer and contain the same structures as the ventromedial region so their presentation is usually similar. However, when a stroke is purely ventrolateral, due to the somatotopic organization of the pyramidal tract (face and arms more medial and the legs represented more laterally), there is a disproportionate impairment of the lower limbs. Dorsolateral pons stroke is characterized by ipsilateral ataxia (middle cerebellar peduncle, pontocerebellar fibbers), contralateral loss of pain and temperature of the limbs and trunk (lateral spinothalamic tract), ipsilateral tinnitus and reduced auditory acuity, ipsilateral peripheral facial palsy (facial nucleus/fascicle), loss of facial sensation (trigeminal principal nucleus), paresis of ipsilateral masticatory muscles (trigeminal motor nucleus), vertigo, nausea, vomiting (vestibular nuclei) and ipsilateral lateral rectus palsy. The syndromes of Marie-Foix and Foville and Raymon-Cestan can result of lateral pontine strokes and are described in Table 1. Tegmental lesions are very unusual and usually consist of consciousness impairment, ataxia, skew deviation, vertigo, Abducens nerve palsy and one and a half syndrome. Bilateral medial lesions of the pons cause "locked-in" syndrome: due to lesion of the cortico-bulbar and corticospinal fibers and of the abducens nucleus in a quadriplegic patient, unable to perform horizontal gaze movement, but conscious and able to communicate only through vertical eye movements and blinking.[8,10,18]

Lesions in the basis of the mesencephalon, supplied by proximal branches of the PCA, cause the classical Weber syndrome, with palsy of the ipsilateral oculomotor nucleus (oculomotor fascicle) and contralateral hemiparesis (pyramidal tract). Lesions in the oculomotor nerve can be further divided into nuclear or infranuclear/fascicular.[19] Fascicular oculomotor lesions usually are characterized by ipsilateral involvement of all the oculomotor innervated muscles, sparing the contralateral eye but the degree of impairment of each subtype of ocular muscle palsy might vary, and hence produce a partial oculomotor palsy.[19] Conversely, in nuclear lesions there is unilateral palsy of

| Table 1 | | | | |

Vertebrobasilar ischemic syndromes

Territory	Vascular territory	Eponym	Affected structures	Clinical presentation
Lateral medulla	VA or PICA	Wallenberg syndrome	Descending sympathetic fibers; ICP; Spinotalamic tract; Vestibular Nuclei; Ambiguus Nucleus; Solitary Nucleus; Spinal tract and nucleus of the trigeminal nerve	Ipsilateral: Horner syndrome; ataxia; intention tremor; facial sensory loss; lateropulsion; dysgeusia; palate palsy; Contralateral: hemisensory loss; nystagmus; Other: vertigo; nausea; dysarthria; dysphonia; skew deviation
Medial medulla	Medullar branches of the VA or ASA	Dejerine syndrome	Corticospinal fibers; Medial lemniscus; Hypoglossal nucleus/fascicle	Ipsilateral: Tongue weakness; Contralateral: Hemiparesis; Vibratory and position sense loss
Hemi-medullar	VA (proximal to the PICA); ASA branches	Babinski syndrome	Corticospinal fibers; Medial lemniscus Hypoglossal nucleus/fascicle; Descending sympathetic fibers; ICP; Spinotalamic tract; Vestibular Nuclei; Ambiguus nucleus; Spinal tract and nucleus of the trigeminal nerve; Medial lemniscus	Ipsilateral: Horner Syndrome; Ataxia; Tongue weakness; facial sensory loss; Contralateral: Hemiparesis; sensory loss; Other: dysarthria; nausea; vertigo
Ventral pons	Paramedian branches of basilar artery		Corticobulbar tract; Corticopontocerebellar fibers; Corticospinal tract; Medial lemniscus; Facial nucleus/fascicle; Abducens nucleus/fascicle	Ipsilateral: peripheral facial palsy; lateral rectus palsy; paresis of the ipsilateral horizontal gaze; ataxia; Contralateral: hemiparesis; facial palsy; proprioceptive and vibratory sensory loss; Other: dysphagia; dysarthria
		One-and-a-half syndrome	MLF; Abducens nucleus	Ipsilateral: INO; impaired conjugated gaze
		Raymond syndrome	Abducens fascicle; Corticospinal tract	Ipsilateral: Lateral rectus muscle palsy; Contralateral: Hemiparesis
		Millard-Gubler syndrome	Abducens fascicle; Corticospinal tract; Facial nucleus/fascicle	Ipsilateral: Lateral rectus muscle palsy; facial palsy; Contralateral: Hemiparesis
Lateral Pons	Short or long circumflex arteries of AICA		MCP; Pontocerebellar fibbers; Lateral spinothalamic tract; Facial nucleus/fascicle; Principal trigeminal nucleus; Trigeminal motor nucleus; Vestibular nuclei; Facial nerve fascicle	Ipsilateral: Ataxia; tinnitus; reduced auditory acuity; peripheral facial palsy; loss of facial sensation; paresis of masticatory muscles; lateral rectus palsy; Contralateral: loss of pain and temperature of the limbs and trunk Other: vertigo; nausea; vomiting
	Short or long circumflex arteries of AICA	Marie-Foix syndrome (more caudal lesion)	MCP; Corticospinal tract; Lateral spinothalamic tract	Ipsilateral: ataxia Contralateral: hemiparesis; sensory loss to pinprick sensation and temperature
	Short or long circumflex arteries of AICA	Foville syndrome	PPRF; Facial nucleus/fascicle; Corticospinal tract	Ipsilateral: horizontal gaze paresis; peripheral facial palsy; Contralateral: hemiparesis
	SCA or long circumflex arteries	Raymond-Cestan syndrome (more rostral lesion)	Cerebellum; Medial lemniscus; Spinothalamic tract; Corticospinal tract	Ipsilateral: ataxia; Contralateral: facial and body sensation; hemiparesis
Bilateral Pons	BA	–	Corticospinal tract; Corticobulbar tract: Facial nucleus/fascicle; Reticular formation	Quadriplegia; aphonia/dysphagia; bilateral horizontal gaze paresis; bilateral facial weakness; transient lethargy
Tegmetum Mesencephalon	PCA	Benedikt syndrome	Red nucleus; Oculomotor fascicle	Ipsilateral: Oculomotor palsy; Contralateral: involuntary movements (chorea, tremor, athetosis)
		Claude syndrome	SCP; Red nucleus; Oculomotor fascicle	Ipsilateral: Oculomotor palsy; ataxia. Contralateral: involuntary movements (chorea, tremor, athetosis)
		Nothangel	SCP; Oculomotor fascicle	Ipsilateral: Oculomotor palsy; ataxia
Tectum mesencephalon	PCA or SCA	Parinaud syndrome	Oculomotor nucleus; Superior colliculi; Inferior colliculi; Posterior commissure.	Loss of vertical gaze (++ the upgaze); Convergence-retraction nystagmus; Convergence impairment; Pupillary light-near dissociation; Eyelid retraction (Collier's sign)

(continued)

Table 1

(continued).

Territory	Vascular territory	Eponym	Affected structures	Clinical presentation
Basal Mesencephalon	BA	Weber syndrome	Oculomotor fascicle; Corticobulbar and corticospinal tract	Ipsilateral: III nerve palsy, with mydriasis Contralateral: hemiparesis
Bilateral lesions of the midbrain and cranial structures	BA	–	Midbrain; Thalamus; Medial temporal lobes; Occipital lobes	Impaired vertical gaze; oculomotor cranial nerve palsy; consciousness impairment; delirium; peduncular hallucinosis; quadriparesis; visual field defect; Anton syndrome; Bálint syndrome
Occipital Lobe	PCA—unilateral lesions	–	Occipital cortex	Contralateral: Homonymous Hemianopia
		–	Infracalcarine gyrus	Contralateral: Superior Homonymous Quadrantanopia
		–	Supracalcarine gyrus	Contralateral: Inferior Homonymous Quadrantanopia
	PCA—bilateral lesions	Anton syndrome	Bilateral occipital lesions	Cortical blindness; Denial of the deficits; Confabulation
Thalamic stroke	Tuberothalamic artery (from the PCoA)	–	Reticular nuclei; intralaminar nuclei; ventral anterior and ventrolateral nuclei; anterior nuclei; ventral internal medullary lamina; ventral amygdalofugal pathway; mamillothalamic tract	Fluctuating consciousness; impaired memory and learning; personality changes; apathy; perseveration; language impairment (left) or hemispatial neglect (right); acalculia; apraxia
	Paramedian artery (from P1)	–	Medial dorsal nucleus; intralaminar nuclei; pulvinar, paraventricular; lateral dorsal nucleus internal medullary lamina	Decreased arousal; impaired memory and learning; aphasia (left); hemispatial neglect (right); apathy; agitation; aggression
	Inferolateral artery (from P2)	–	Ventroposterior nuclei; Ventral lateral nucleus; MGN; pulvinar; lateral dorsal nucleus	Sensory loss; hemiataxia; hemiparesis; auditory impairment; behavioural changes; thalamic pain (Dejerine-Roussy syndrome)
	Posterior choroidal artery (from P2)	–	LGN; lateral dorsal nucleus; lateral posterior nucleus; MGN; pulvinar	Visual field defect; sensory loss; weakness; aphasia; memory loss; dystonia; tremor

AICA = anterior-inferior cerebellar artery, ASA = anterior spinal artery, ICP = inferior cerebellar peduncle, LGN = lateral geniculate nucleus, MCP = middle cerebellar peduncle, MGN = medial geniculate nucleus, MLF = Medial longitunal fasciculus, PCoA = posterior communicating artery, PICA = posterior-inferior cerebellar artery, PPRF = Paramedian pontine reticular formation, SCA = superior cerebellar artery, SCP = superior cerebellar peduncle, VA = vertebral artery.[15,19]

the third CN associated with weakness of the ipsilateral and contralateral superior rectus muscle (due to crossed innervation of the medial subnucleus that innervate this muscle) and bilateral incomplete ptosis (since a single caudal subnucleus innervates both elevator palpebrae superioris). However, fascicular lesions are often accompanied by nuclear lesions because the paramedian branches of the top of the basilar artery often supply both structures.[20]

A lesion in the tegmentum will additionally cause contralateral involuntary movements, such as chorea, tremor or athetosis due to lesion to red nucleus (Benedikt's syndrome) or ipsilateral ataxia when involves the superior cerebellar peduncle (Claude's syndrome).[18] Lesions to the tectum of the midbrain can cause Parinaud's syndrome, which is characterized by loss of vertical gaze (with upgaze more severely impaired), convergence-retraction nystagmus, convergence impairment, pupillary light-near dissociation and eyelid retraction (Collier's sign).[18] Similar to "locked-in-syndrome", emboli can lodge in the distal BA and cause bilateral ischemia in the midbrain, thalamus, and temporal and occipital lobes. Patients present with combined symptoms of the ischemic regions, with vertical gaze palsy, pupillary abnormalities, consciousness fluctuation and delirium, vivid visual hallucinations ("peduncular hallucinosis"), visual field defects, and motor and sensory deficits.[21,22]

Occipital infarction, usually due to ACP embolism, usually presents with visual field defects, either contralateral homonymous hemianopia or contralateral inferior or superior quadrantanopia, if the lesion is in the supra or infracalcarine sulcus, respectively. Bilateral lesions can cause cortical blindness or even Anton syndrome, in which the patient, although blind, denies the deficit and has visual hallucinations or confabulates. Bilateral watershed lesions in the parietooccipital junction can cause Balint syndrome, in which patients experience optic ataxia (inability to reach targets using visual guidance), oculomotor apraxia (inability to voluntarily direct gaze), and simultagnosia (described as the inability to synthesize objects within a visual field).[22]

Finally, the VB circulation also supplies the thalamus trough branches from the first (P1) and second (P2) segments of the ACP. Thalamic strokes can have a wide range of clinical presentations, according to the affected nuclei groups. The most common presentation is hemisensory deficit in all the sensory modalities, frequently accompanied by motor deficit due to the proximity to the internal capsule, but thalamic aphasia and behavior abnormalities can occur if the anterior nuclei are involved,[23] as well as visual field defect, tremor or acute impaired consciousness, particularly in bilateral thalamic lesions.[24] Thalamic vascular syndromes are further explained in Schmahmann's

Table 2

Rare causes of vertebrobasilar stroke

Rare causes of VB stroke	Clinical context
Subclavian steal syndrome	Subclavian stenosis prior to the origin of the VA. The ultrasound reveals reverse flow in the VA; different blood pressure values between the 2 arms
Giant cell arteritis	Older patients (>50 Yo), headache, temporal tenderness, increased erythrocyte sedimentation rate and/or C-reactive protein
Fabry's disease	More frequently male patients; cardiac and renal impairment, acroparesthesias, angiokeratoma
MELAS	Young adults, short stature, myopathic features (with myopathic muscle biopsy), lesions in the parieto-occipital/parietotemporal region, often with bilateral involvement, MRI without reduction of apparent diffusion coefficient
PRES	Hypertension, pregnancy, immunosuppressive treatment; patients with seizures, visual disturbances, headache. Vasogenic oedema lesions usually in the parieto-occipital region, often with bilateral involvement

MELAS = mitochondrial encephalopathy, lactic acidosis and stroke-like episodes, PRES = posterior reversible encephalopathy syndrome, VA = vertebral artery, Yo = years-old.[5]

"Vascular syndromes of the thalamus"[24] or in Powel and Hughes' "A chamber of secrets—The neurology of the thalamus: lessons from acute stroke".[25]

The different posterior circulation syndromes are also described in Table 1.

Diagnostic workup and ancillary tests

Brain computed tomography (CT), the most frequently available imaging technique in the emergency department, has a poor sensitivity for the posterior fossae.[26] Hence an MRI, in particular diffusion-weighted imaging (DWI), is a much-preferred imaging scan when a VB stroke is suspected. However, in a recent metanalysis, 6.8% of all acute ischemic events were negative, and posterior circulation stroke was 5-times more likely to be negative on DWI.[27] In fact, in another recent study, 13.7% of the clinically suspected strokes were DWI negative, and 30% of these were located in the brainstem,[28] highlighting the importance of the clinical diagnosis.

As for stroke etiology and subsequent etiological work-up, most frequent mechanisms are embolism, large artery thrombosis and lipohyalinosis.[3,29] If we divide the most frequent etiology by the different vascular territories (proximal, middle and distal), half of the proximal territory infarctions are caused by cardiac and artery-to-artery embolism (from the extracranial vertebral arteries), while the other half is explained by hypoperfusion due to intracranial vertebral occlusive disease. Middle territory stroke is usually due to occlusive lesions of the basilar artery or its branches and more distal territory lesions are attributable to embolism (both cardiogenic or artery-to-artery), the remainder being explained by small vessels disease.[9] Involvement of both distal and middle regions (either alone or in combination) are associated with a greater risk of death or major disability.[3]

Therefore, a comprehensive etiology workup must be performed in all patients, including endovascular imaging, echocardiogram and cardiac rhythm monitorization. Also, one should never forget that some rarer causes of stroke or stroke mimics have some preference for the posterior circulation and should be considered in the proper context[5]—this include vertebral dissection, subclavian steal syndrome, giant cell arteritis, mitochondrial encephalopathy, lactic acidosis and stroke-like episodes (MELAS) and posterior reversible encephalopathy syndrome (PRES) (see Table 2).

Conclusion

Vertebrobasilar stroke often presents as a clinical challenge to the emergency department physician. It is a non-neglectable cause of morbidity and mortality in stroke patients, which can be avoided or minimized with correct recognition and treatment.

The clinical presentation is wide, from mild unspecific symptoms to catastrophic presentations such as locked-in-syndrome or top of the basilar syndrome. Furthermore, the lower sensitivity of brain CT for the posterior fossae and the higher probability of a DWI-negative MRI in these patients, make a high level of suspicion crucial in these patients, in order to recognize and treat VB stroke.

Conflicts of interest

The authors declare no conflicts of interest.

References

[1] Bogousslavsky J, Van Melle G, Regli F. The lausanne stroke registry: analysis of 1,000 consecutive patients with first stroke. Stroke. 1988; 19:1083–1092.

[2] Kim J-T, Park M-S, Choi K-H, et al. Clinical outcomes of posterior versus anterior circulation infarction with low national institutes of health stroke scale scores. Stroke. 2016;48:55–62.

[3] Glass TA, Hennessey PM, Pazdera L, et al. Outcome at 30 days in the New England Medical Center Posterior Circulation Registry. Arch Neurol. 2002;59:369–376.

[4] Gulli G, Markus HS. The use of FAST and ABCD2 scores in posterior circulation, compared with anterior circulation, stroke and transient ischemic attack. J Neurol Neurosurg Psychiatry. 2012;83: 228–229.

[5] Markus HS, van der Worp HB, Rothwell PM. Posterior circulation ischaemic stroke and transient ischaemic attack: diagnosis, investigation, and secondary prevention. Lancet Neurol. 2013;12:989–998.

[6] Cloud GC, Markus HS. Diagnosis and management of vertebral artery stenosis. QJMed. 2003;96:27–54.

[7] Shaban A, Albright KC, Boehme AK, Martin-Schild S. Circle of Willis variants: fetal PCA. Stroke Res Treat. 2013;2013:1059371-6. doi: 10.1155/2013/105937. Epub 2013 Mar 21.

[8] Blumenfeld H. Neuroanatomy Through Clinical Cases. 2nd ed2010; Sinauer Associates,

[9] Caplan LR, Wityk RJ, Glass TA, et al. New England Medical Center Posterior Circulation registry. Ann Neurol. 2004;56:389–398.

[10] Searls DE, Pazdera L, Korbel E, et al. Symptoms and signs of posterior circulation ischemia in the New England Medical Center Posterior Circulation registry. Arch Neurol. 2012;69:346–351.

[11] Swenson RS. Chapter 4—Development [Internet]. Review of Clinical and Functional Neuroscience. Available from: https://www.dartmouth.edu/~rswenson/NeuroSci/index.html. Accessed: 13th August 2020.

[12] Querol-Pascual MR. Clinical approach to brainstem lesions. Semin Ultrasound CT MRI. 2010;31:220–229.

[13] Kameda W, Kawanami T, Kurita K, et al. Lateral and medial medullary infarction: a comparative analysis of 214 patients. Stroke. 2004;35: 694–699.

[14] Akimoto T, Ogawa K, Morita A, Suzuki Y, Kamei S. Clinical study of 27 patients with medial medullary infarction. J Stroke Cerebrovasc Dis. 2017;26:2223–2231.

Carvalho and Cruz Porto Biomed. J. (2020) 5:6

Porto Biomedical Journal

[15] Sacco RL, Freddo L, Bello JA, Odel JG, Onesti ST, Mohr JP. Wallenberg's lateral medullary syndrome: clinical-magnetic resonance imaging correlations. Arch Neurol. 1993;50:609–614.

[16] Tilikete C, Koene A, Nighoghossian N, Vighetto A, Pélisson D. Saccadic lateropulsion in Wallenberg syndrome: a window to access cerebellar control of saccades? Exp Brain Res. 2006;174:555–565.

[17] Brazis PW. Ocular motor abnormalities in Wallenberg's lateral medullary syndrome. Mayo Clinic Proc. 1992;67:365–368.

[18] Burger KM, Tuhrim S, Naidich TP. Brainstem vascular stroke anatomy. Neuroimaging Clin N Am. 2005;15:297–324.

[19] Brazis PW. Localization of lesions of the oculomotor nerve: recent concepts. Mayo Clinic Proc. 1991;66:1029–1035.

[20] De Mendivil AO, Alcalá-Galiano A, Ochoa M, Salvador E, Millán JM. Brainstem stroke: anatomy, clinical and radiological findings. Semin Ultrasound CT MRI. 2013;34:131–141.

[21] Nouh A, Remke J, Ruland S. Ischemic posterior circulation stroke: a review of anatomy, clinical presentations, diagnosis, and current management. Front Neurol. 2014;5:30. 1–16. Published 2014 Apr 7. doi:10.3389/fneur.2014.00030.

[22] Carrera E, Bogousslavsky J. The thalamus and behavior: effects of anatomically distinct strokes. Neurology. 2006;66:1817–1823.

[23] Schmahmann JD. Vascular syndromes of the thalamus. Stroke. 2003;34:2264–2278.

[24] Powell R, Hughes T. A chamber of secrets the neurology of the thalamus: lessons from acute stroke. Pract Neurol. 2014;14:440–445.

[25] Hwang DY, Silva GS, Furie KL, Greer DM. Comparative sensitivity of computed tomography vs. magnetic resonance imaging for detecting acute posterior fossa infarct. J Emerg Med. 2012;42:559–565.

[26] Edlow BL, Hurwitz S, Edlow JA. Diagnosis of DWI-negative acute ischemic stroke. Neurology. 2017;89:256–262.

[27] Brunser AM, Cavada G, Venturelli PM, et al. Diffusion-weighted imaging determinants for acute ischemic stroke diagnosis in the emergency room. Neuroradiology. 2018;60:687–692.

[28] Bogousslavsky J, Regli F, Maeder P, Meuli R, Nader J. The etiology of posterior circulation infarcts. Neurology. 1993;43:1528–1533.