

Neuroanatomical basis of Wallenberg syndrome

Bases neuroanatómicas del síndrome de Wallenberg

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Abstract

Wallenberg syndrome, or lateral medullar syndrome, is the clinical presentation of the infarct in the territory of posterior inferior cerebellar artery. Its signs and symptoms include vertigo, nystagmus, diplopia, ipsilateral Horner syndrome, facial ruddiness and dry skin, dysphonia, dysphagia, dysarthria, ipsilateral loss of gag reflex, ipsilateral ataxia, ipsilateral impaired taste, ipsilateral facial pain and paresthesia, decreased ipsilateral blink reflex, contralateral hypoalgesia and thermoanesthesia in the trunk and limbs; and ipsilateral facial hypoalgesia and thermoanesthesia. Neuroanatomical knowledge is essential to its comprehension, study, and diagnosis, because the classic neurological manifestations are easy to explain and understand if function and localization of affected anatomical structures are known as if the posterior cerebral circulation is.

Keywords: Wallenberg. Syndrome. Ischemia. Medulla oblongata.

Resumen

El síndrome de Wallenberg, o síndrome bulbar lateral, es la manifestación clínica del infarto en el territorio de irrigación de la arteria cerebelosa posteroinferior. Su presentación incluye vértigo, nistagmo, diplopía, síndrome de Horner, rubicundez y anhidrosis facial homolateral, disfonía, disfagia, disartria, pérdida homolateral del reflejo nauseoso, ataxia homolateral, disgeusia homolateral, dolor y parestesia faciales homolaterales, pérdida o disminución homolateral del reflejo corneal, hipoalgesia y termoanestesia de tronco y extremidades contralaterales, hipoalgesia y termoanestesia facial homolateral. El conocimiento neuroanatómico es imprescindible para su comprensión, estudio y diagnóstico, ya que sus manifestaciones neurológicas clásicas son fácilmente explicables y entendibles si se conocen la función y la localización de las estructuras anatómicas afectadas, así como la irrigación cerebral posterior.

Palabras clave: Síndrome de Wallenberg. Isquemia. Médula oblongada. Bulbo raquídeo.

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Introduction

First described in 1895 by Polish neurologist Adolf Wallenberg^{1,2}, Wallenberg syndrome, also known as lateral bulbar syndrome, posterior inferior cerebellar artery (PICA) syndrome, lateral medullary infarction, or dorsolateral medullary infarction²⁻⁴, is caused by infarction of the lateral and posterior portion of the lower olivary nucleus of the brain stem (medulla oblongata) just in the territory irrigated by PICA².

Approximately 83% of strokes in the United States of America are from ischemic etiology; out of these, only 20% occur in the vertebrobasilar territory².

The most common and most described causes of Wallenberg syndrome are, of frequency, atherothrombotic occlusion of the vertebral artery, PICA or medullary arteries, cardioembolism, and vertebral dissection^{2,5}. However, there are other less common but well-described causes of Wallenberg syndrome, such as the use of emerging drugs known as “legal highs,”⁶ cocaine use⁷, sarcoidosis⁸, granulomatosis with polyangiitis (Wegener’s granulomatosis)³, giant cell arteritis⁹, scorpion sting¹⁰, and even PICA aneurysm without rupture¹¹.

Medulla oblongata anatomy

The medulla oblongata is usually studied in two portions, one open and one closed, both separated by the obex, with the open portion being cephalic to it (Fig. 1). The medulla oblongata contains various structures, composed of both white and gray matter (Fig. 2).

Nuclei

The nuclei contained by the medulla oblongata at its open portion include the following:

- *Nucleus ambiguus*: it contains the cells responsible for innervating the muscles of the soft palate, pharynx, larynx, and upper esophagus through the glossopharyngeal, vagus, and accessory cranial nerves. It also contains parasympathetic neurons that control heart rate through the vagus nerve^{12,13}.
- *Hypoglossal nucleus*: formed by motor neurons that will constitute the hypoglossal nerve; it gives movement to the tongue. The neuronal fibers that originate in this nucleus exit through of the pre-olivary sulcus, between the pyramid and the olive, to form cranial nerve XII.

- *Dorsal nucleus of vagus nerve*: it is the largest parasympathetic nucleus of the medulla and is composed of motor neurons, whose preganglionic fibers will form part of the vagus nerve when exiting through the retro-olivary sulcus just behind the olive. Its function includes glandular and smooth muscle motor regulation in the viscera of the chest and abdomen.
- *Spinal trigeminal nucleus*: neurons located in this nucleus receive ipsilateral afferents from the trigeminal, facial, glossopharyngeal, and vagus nerves, through the spinal trigeminal tract. Its fibers ascend through the ventral trigeminothalamic tract, which decussates on its trajectory to the contralateral thalamus. Its function is the transmission of general sensations (touch, pain, and temperature) of the head^{12,13}.
- *Olivary nuclei*: include the lower, medial accessory, and dorsal accessory olivary nuclei, and are part of the so-called pre-cerebellar nuclei. These nuclei receive information from the spino-olivary tract originating in the contralateral posterior horn of all spinal segments; it also receives information from the red nucleus (through the dorsal tegmental tract) and ipsilateral cerebral cortex. Its fibers continue toward the cerebellum through the lower cerebellar peduncle. This nuclear complex helps in the coordination of learned movement patterns^{12,13}.
- *Solitary nucleus*: it receives information through the solitary tract, coming from the lower nodes of the hypoglossal, vagus, and geniculate ganglion of the facial nerve. The caudal portion of this nucleus participates in visceral reflexes related to the glossopharyngeal and vagus nerves (nauseous and vagal reflexes), while the cephalic portion receives information on taste coming from the facial and glossopharyngeal nerves.
- *Lower and medial vestibular nuclei*: these nuclei contain neurons that receive afferent stimuli through the vestibulocochlear nerve. Some of its fibers enter the cerebellum through the inferior cerebellar peduncle, others descend through the vestibulospinal tract, and others (mostly coming from the medial vestibular nucleus) are part of the medial longitudinal fasciculus.

White matter

The following axons cross the open portion of the medulla oblongata:

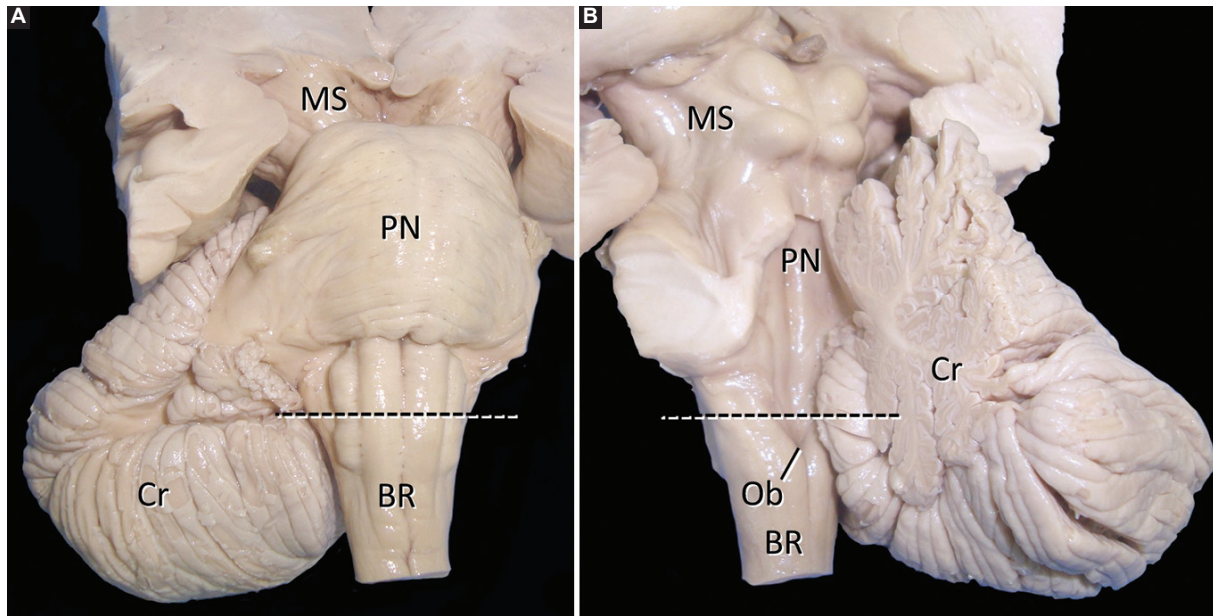


Figure 1. Medulla oblongata external anatomy. **A:** medulla oblongata anterior view. **B:** medulla oblongata left posterolateral view. The dashed line shows the section level of figure 2. The left cerebellar hemisphere was cut. BR: medulla oblongata; Cr: cerebellum; MS: mesencephalic; Ob: obex; PN: bridge.

- *Medial lemniscus*: formed by ascending fibers of the gracile and cuneiform nuclei after decussation. These fibers terminate in the lateral portion of the ventral-posterior nucleus of the thalamus. It transmits conscious proprioceptive information^{12,13}.
- *Ventral spinocerebellar tract*: ascending fibers that, after decussating at its origin, pass through the anterior portion of the lateral cord in the spinal cord, cross the medulla oblongata, and enter the cerebellum at the mesencephalic level through the superior cerebellar peduncle. It transmits unconscious proprioceptive information, mainly from lower limbs^{12,13}.
- *Spinal lemniscus*: it is the combination of spinothalamic and spinotectal tracts. It contains contralateral thermoalgesic information from the trunk and extremities coming from the spinal cord and concludes in the ventral-posterior nucleus of thalamus^{12,13}.
- *Pyramidal tract*: it is the corticospinal pathway when it descends through the medulla oblongata pyramid. It contains motor information and is formed by descending fibers of the upper motor neuron before its decussation^{12,13}.
- *Medial longitudinal fasciculus*: it contains ipsilateral and contralateral fibers originating in vestibular nuclei terminating in the abducens, trochlear, and oculomotor nuclei. The information it transmits allows the coordination of eye movements^{12,13}.
- *Inferior cerebellar peduncle*: it contains cerebellar afferents coming from the spinal cord (through the dorsal spinocerebellar tract), from olivary nuclei, and from vestibular nuclei^{12,13}.
- *Spinal trigeminal tract*: it is formed by ipsilateral fibers of the trigeminal, facial, glossopharyngeal, and vagus nerves terminating in the spinal trigeminal nucleus, transmitting general sensations of touch, pain, and temperature of the head^{12,13}.
- *Solitary tract*: it is formed by fibers from the inferior ganglions of hypoglossal and vagus nerves and the geniculate ganglion of facial nerve and terminates in the solitary nucleus. It transmits information on taste and reflex afferents related to the glossopharyngeal and vagus nerves^{12,13}.
- *Ventral trigeminothalamic tract*: it contains ascending fibers from the spinal trigeminal nucleus that decussate to terminate in the contralateral thalamus. It transmits general sensations of touch, pain, and temperature of the head^{12,13}.
- *Hypoglossal nerve fibers*: axons from neurons located in the hypoglossal nucleus; as they exit through the pre-olivary sulcus, they form the

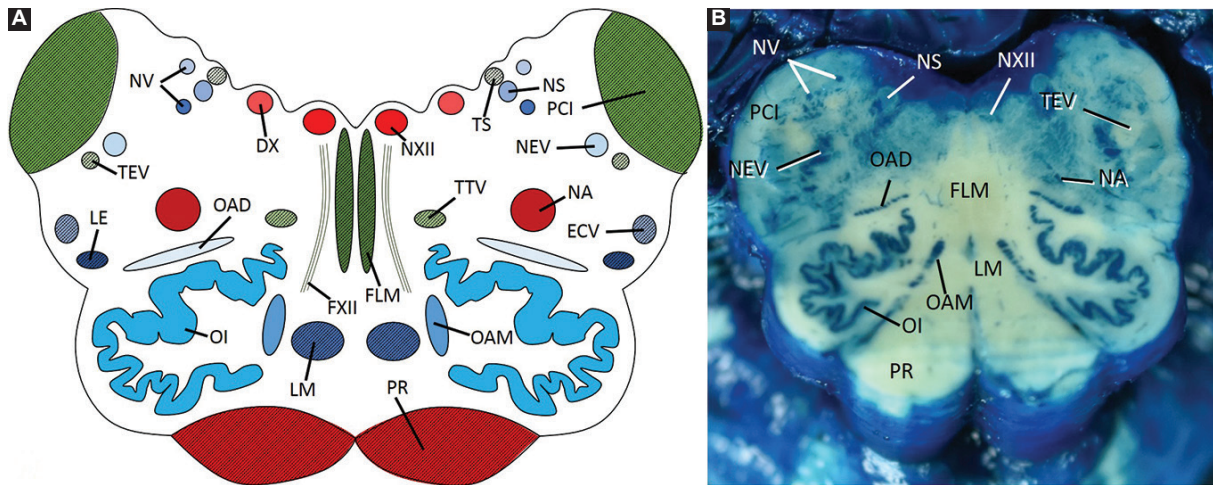


Figure 2. Medulla oblongata internal anatomy (cross-section, Fig. 1). **A:** anatomical diagram. **B:** human medulla oblongata with Mulligan staining. Motor nuclei (red tones): dorsal nucleus of vagus nerve (DX), nucleus ambiguus (NA), and hypoglossal nucleus (NXII). Sensory nuclei (blue tones): spinal trigeminal nerve nucleus (NEV), solitary nucleus (NS), inferior and medial vestibular nuclei (NV); dorsal accessory olivary nucleus (OAD), medial accessory olivary nucleus (OAM), and inferior olivary nucleus (OI). Ascending tracts (blue tones with stripes): ventral spinocerebellar tract (ECV), spinal lemniscus (LE), and medial lemniscus (LM). Descending tracts (red tones with stripes): pyramidal tract (PR). Other tracts and fibers (green shades with stripes): medial longitudinal fasciculus (FLM), inferior cerebellar peduncle (PCI), spinal trigeminal tract (VTE), solitary tract (TS), ventral trigeminothalamic tract (TTV), and hypoglossal nerve fibers (FXII).

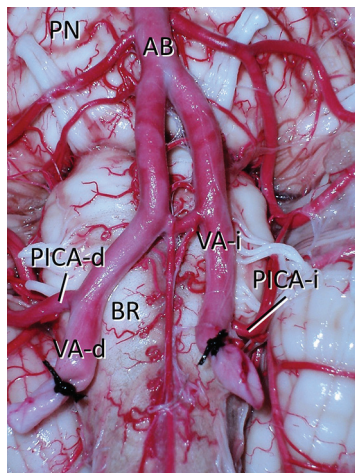


Figure 3. Posterior inferior cerebellar artery (PICA) origin. AB: basilar artery; BR: medulla oblongata; PICA-d: right PICA; PICA-i: left PICA; VA-d: right vertebral artery; VA-i: left vertebral artery; PN: bridge.

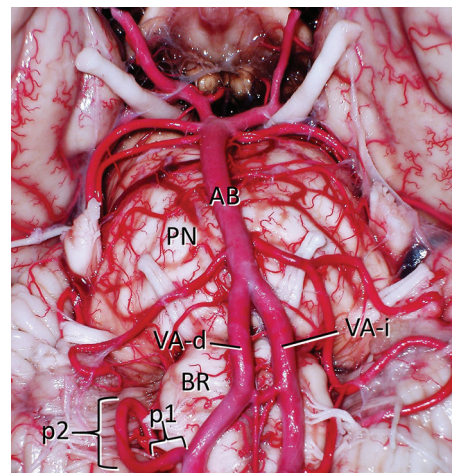


Figure 4. Posterior inferior cerebellar artery (PICA) initial segments. Brainstem posterior view. AB: basilar artery; BR: brainstem; p1: PICA anterior medullary segment; p2: PICA lateral medullary segment; PN: bridge; VA-d: right vertebral artery; VA-i: left vertebral artery.

hypoglossal nerve, responsible for the movement of the tongue^{12,13}.

PICA anatomy

PICA is the most distal and prominent branch of the vertebral artery, and it originates in the intracranial segment of the vertebral artery, in more than 40% of cases in the lateral spinal segment and almost 33%

in the vertebral artery pre-medullary segment (Fig. 3)^{12,14-16}. In some cases, PICA occurs as the terminal branch of the vertebral artery¹⁷. In 5-20% of cases, PICA has an extradural origin¹⁸.

It is present in 94-96% of cases; it arises as a single artery in 90-97% and duplicated in 3-6%¹⁵⁻¹⁹.

PICA is divided, as proposed by Lister, Rodríguez-Hernández, and Rhoton, into five segments designated with the lowercase letter "p" and numbers 1-5.

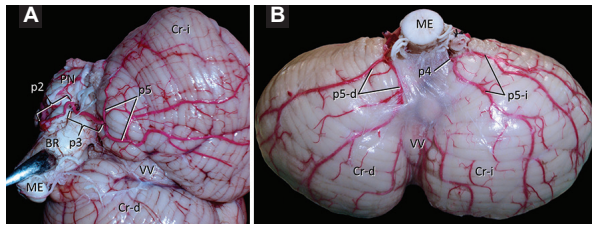


Figure 5. Posterior inferior cerebellar artery (PICA) course. **A:** cerebellar left inferolateral view. **B:** cerebellar inferior view. BR: medulla oblongata; Cr-d: cerebellar right hemisphere; Cr-i: cerebellar left hemisphere; ME: spinal cord; p2: PICA lateral medullary segment; p3: PICA tonsillo-medullary segment; p4: PICA telovelotonsillar segment; p5: PICA cortical segment; p5-d: right p5; p5-i: left p5; PN: bridge; VV: ventral vermis.

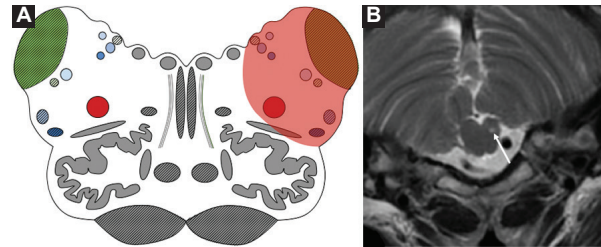


Figure 6. Ischemia territory in Wallenberg syndrome. **A:** anatomical depiction. The red shadow shows left posterior inferior cerebellar artery irrigation territory. The nuclei and affected white matter are shown in color. **B:** T2-weighted MRI image showing ischemia (white arrow).

Segment p1, called anterior medullary segment, is located on the anterior portion of the medulla; segment p2, or lateral medullary segment, courses along the lateral part of the medulla up to the retro-olivary sulcus, just at the origin of the glossopharyngeal, vagus, and accessory nerves (Fig. 4); segment p3, called the tonsillomedullary segment, is related to cerebellar tonsil caudal half; segment p4, or telovelotonsillar segment, is located in the cleft between the tela choroidea and inferior medullary velum rostrally, and superior pole of the cerebellar tonsil caudally; finally, segment p5, called the cortical segment, is located on cerebellar hemispheres cortical surface (Fig. 5)^{19,20}.

Its irrigation territories include the posterior portion of cerebellar hemispheres, inferior portion of the vermis, central cerebellar nuclei, and the choroid plexuses of the fourth ventricle. It gives rise to spinal branches that irrigate the dorsolateral portion of the brainstem^{12,19,21}.

Clinical manifestations

Wallenberg syndrome classical clinical manifestations are due to PICA-irrigated structures ischemia (Fig. 6) and involve the vestibular nuclei, inferior cerebellar peduncle, spinal trigeminal nucleus, spinal trigeminal tract, spinothalamic tract, nucleus ambiguus, and descending preganglionic sympathetic fibers^{12,13}.

Frequently (more than 60% of cases), in Wallenberg syndrome, in addition to lateral medullary area direct injury, there is an infarction of additional areas on the medulla oblongata²².

These manifestations include vertigo, nystagmus, diplopia, Horner syndrome, ipsilateral facial flushing and anhidrosis, dysphonia, dysphagia, dysarthria, ipsilateral nauseous reflex loss, ipsilateral ataxia, ipsilateral dysgeusia, ipsilateral facial pain and

paresthesia, ipsilateral corneal reflex loss or reduction, hypoalgesia and thermoanesthesia of trunk and contralateral limbs, hypoalgesia, and ipsilateral facial thermoanesthesia^{2,12,13,23-25}.

In addition to classic findings, which may or may not occur in their entirety, Wallenberg syndrome has been described as a cause of epicrania fugax²⁶, cervicobrachial dystonia²⁷, central hypoventilation²⁸, neurotrophic and punctate keratopathy^{4,23}, mania²⁹, and cardiovascular autonomic dysfunction (with orthostatic hypotension)³⁰.

Neurological manifestations anatomoclinical correlation

- *Vertigo and nystagmus:* they occur both due to vestibular nuclei direct lesion (mainly inferior) found in PICA irrigation territory and vestibular pathways lesion, particularly in those that communicate with the parieto-insular vestibular cortex^{2,12,13,23,31}. Vertigo is usually of central characteristics in direction of the gaze. Both clinical manifestations may be accompanied by secondary singultus, nausea, and vomiting².
- *Diplopia:* due to injury to collateral pontine and medial longitudinal fasciculus, structures that are involved in conjugate eye movement²³.
- *Horner syndrome, ipsilateral flushing, and facial anhidrosis:* enophthalmos, conjunctival injection, and miosis that are characteristic of the so-called Claude Bernard-Horner syndrome, as well as flushing and facial anhidrosis, are caused by injury of the descending preganglionic sympathetic fibers that cross the medulla oblongata^{2,12,13,21,23}.
- *Dysphonia, dysphagia, dysarthria, and ipsilateral nauseous reflex loss:* these manifestations result from injury to the nucleus ambiguus and some

fibers that give rise to glossopharyngeal and vagus nerves. Dysphagia can be severe in 40% of cases, and up to 100% of patients have some degree of dysphagia; it is usually short-lived and disappears almost entirely in 4-10 weeks. Specifically, dysphagia occurs due to injury to a central pattern generator that centrally controls the swallowing process^{2,12,13,24,25}.

- *Ipsilateral ataxia*: it occurs with a tendency toward ipsilateral lateralization and is the consequence of direct cerebellar lesion by PICA occlusion, due to spinocerebellar fibers injury and by inferior cerebellar peduncle fibers injury^{2,12,13}.
- *Ipsilateral dysgeusia*: occurs due to lesion of the solitary tract and the solitary nucleus².
- *Ipsilateral facial pain and paresthesia and ipsilateral corneal reflex loss or decrease*: spinal trigeminal tract and spinal trigeminal nucleus are affected in Wallenberg syndrome, which explains ipsilateral pain and paresthesia. In addition, these structures participate as afferent pathways of the corneal reflex, necessary for blinking during corneal sensory stimulation; this involvement explains the onset of related keratitis in some cases^{2,4,12,13,23}.
- *Hypoalgesia and thermoanesthesia of trunk and contralateral limbs*: it occurs as a result of a lesion of the spinothalamic tract that ascends through the spinal lemniscus^{2,12,13,23}.
- *Ipsilateral facial hypoalgesia and thermoanesthesia*: caused by spinal trigeminal tract and spinal trigeminal nucleus injury^{2,12,13,23}.

Conclusion

Wallenberg syndrome is the most common clinical presentation of a stroke in the posterior circulation. Its clinical manifestations include a diverse range of signs and symptoms, most of which are easily explained by an anatomical correlation with affected structures. Its study is a clear reflection of the importance of clinically applied neuroanatomy.

Conflicts of interest

The authors declare that they have no conflict of interest.

Ethical disclosures

Protection of human and animal subjects. The authors declare that the procedures that were

followed adhered to the ethical standards of the responsible committee for experimentation on human beings and were in agreement with the World Medical Association and the Declaration of Helsinki.

Confidentiality of data. The authors declare that no patient data appear in this article.

Right to privacy and informed consent. The authors declare that no patient data appear in this article.

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