ABSTRACT: We present a case of partial Wallenberg syndrome also called partial lateral medullary syndrome, a hemorrhagic or ischemic stroke of the area fed by the posterior inferior cerebellar artery and the clinical manifestation depends on the extension of the lesion: dorsal-ventral, medial-lateral and rostro-caudal. Five types have been described. Our patient had headache, hoarseness, right upper extremity, right hemithorax and right upper gluteal hypothermalgesia implicating the involvement of the cervical, the thoracic and part of lumbar fibers of the left lateral spinothalamic tract and the ambiguous nucleus; an entity not described before. The imaging done to our patient disclosed the dissection of the left vertebral artery. He was treated with anticoagulation with gradual improvement in his symptoms.

CASE PRESENTATION

A 55-year-old male patient with hypertension and dyslipidemia was admitted to the medical ward because of right upper extremity hypothermalgesia noted five days prior to presentation. The patient discovered this deficiency when he was trying to get a bottle of cold water and noticed it was not cold in his right hand but in his left hand. Back in the history he notes hoarseness plus a severe continuous aching occipital headache since five days that disturbed his sleeping. He denies any head trauma, visual disturbances or diplopia, dysphasia, dysphagia, facial or body dysesthesia or weakness, nausea, vomiting, dizziness, vertigo, imbalance, or alteration of level of consciousness. He is on indapamide 1.5 mg once daily, simvastatin 20 mg once daily and aspirin 100 mg daily. He had bilateral inguinal herniorrhaphy five years ago; his family history is irrelevant; he does not smoke neither drink alcohol. His vital signs disclosed an elevated systolic blood pressure of 155 mmHg, regular heart beats of 86 beats per minute and a normal temperature and respiratory rate. The physical exam showed a symmetric face, no carotid bruits; normal heart beats, good bilateral breath sound, normal exam of the abdomen. His neurological exam was as follows: normal cranial nerves, pupils were reactive equal to light and accommodation, the corneal reflex was present bilaterally, no
nystagmus, normal oral cavity with preserved gag reflex, uvular and palatal symmetry. The tongue moved well and there was no facial weakness. The motor power was 5/5 in all muscle compartments. Gait and cerebellar signs (rapid alternating movement, finger-to-nose, Romberg test, and tandem gait) were normal. The reflexes were preserved 2/4. However, the abnormal was the sensory exam: sensation to pain and heat was absent in the right upper extremity, right hemithorax and extending to the upper outer part of the right gluteal area. Blood metabolic profile was normal and his glucose level was normal.

Brain MRI was done and showed hypersignal band in the lateral medulla consistent with ischemia with abolition of the flow void signal in the left vertebral artery as seen in figures 1, 2, 3. Subsequent cerebral angiography showed dissection of the left vertebral artery (Fig. 4).

DISCUSSION

We presented a case of partial Wallenberg syndrome. Wallenberg syndrome is the infarction (ischemic or hemorrhagic) of the lateral medulla and the clinical manifestation depends on the three-dimension extension of the lesion: dorsal-ventral, medial-lateral and rostrocaudal extension (Fig. 5). The syndrome includes vertigo, hoarseness and dysphagia, Horner’s syndrome and cerebellar ataxia on the same side of the lesion, and hypothermalgesia involves the ipsilateral face and the opposite trunk and limbs (crossed sensory symptoms).

The contra lateral deficits in pain and temperature sensation from the body are due to involvement of the lateral spinothalamic tract; the ipsilateral loss of pain and temperature sensation from face are due to involvement of spinal

![Figure 1](image1.png)

**Figure 1.** Abolition of the flow void in the left vertebral artery (white circle).

![Figure 2](image2.png)

**Figure 2.** Abolition of the flow void in the left vertebral artery (white circle).

![Figure 3](image3.png)

**Figure 3.** Hypersignal in left lateral medulla (arrow).

![Figure 4](image4.png)

**Figure 4.** Angiography showing dissection of the left vertebral artery (large white arrow) showing filling defect.

![Figure 5](image5.png)

**Figure 5.** The somatotopic distribution of the spinothalamic and trigeminothalamic tracts in the medulla (with the permission of the authors [1]).
trigeminal nucleus; dysphagia, hoarseness, diminished gag reflex are caused by involvement of the ambiguous nucleus; the involvement of the vestibular system causes vertigo, diplopia, nystagmus, vomiting; the involvement of the central tegmental tract causes palatal myoclonus and the involvement of the inferior cerebellar peduncles causes ipsilateral cerebellar signs including ataxia. Five different subtypes of Wallenberg syndrome were described in the literature by Zhang et al. [1] (Fig. 6, 7).

- **TYPE I**: Far dorsal lateral medullary infarction ( ), causing ipsilateral face hypothermalgesia and contralateral body hypothermalgesia.
- **TYPE II**: Enlarged dorsal lateral infarction ( ), including the ventral trigeminothalamic tract causing ipsilateral and contralateral face hypothermalgesia and contralateral hypothermalgesia.
- **TYPE III**: Midlateral medullary infarction ( ) causing contralateral face and body hypothermalgesia.
- **TYPE IV**: Far lateral infarction ( ) causing hypothermalgesia in the ipsilateral face and the contralateral lower trunk and leg.
- **TYPE V**: Restricted mediolateral infarction ( ) causing hypoalgesia only in the contralateral face, arm and upper trunk, without involvement of the ipsilateral face.

**Figure 6.** Subtypes I & II of Wallenberg Syndrome (reproduced with authorization from the authors [1]).
Figure 7. Subtypes III, IV & V of Wallenberg Syndrome (reproduced with authorization from the authors [1]).
Our patient had only hoarseness and sensory symptoms in the right upper extremity, right hemithorax extending to the right upper thigh area with the corresponding anatomical deficit in the left lateral medulla that should correspond to the left ambiguous nucleus and the fibers of the cervical, thoracic and lumbar area of the left lateral spinothalamic as shown by the MRI.

Of the cross syndromes described in the literature [2], none of the cross syndromes would fit to this patient and the anatomical defect and the clinical manifestation corresponds to a partial Wallenberg syndrome. The vessels to the brainstem come from the vertebrobasilar system: the posterior inferior cerebellar artery, the anterior inferior cerebellar artery, the superior cerebellar artery, the posterior cerebral artery, and the pontine artery. Each of these vessels sends small branches (a few or many) into the underlying brain stem structures along its course. Other vessels penetrate the brain stem from the basilar artery. Small medullary and spinal branches of the vertebral artery make up a third group of vessels. The vessels that are responsible for causing the Wallenberg syndrome are the branches of the vertebral artery or, most commonly, the posterior inferior cerebellar artery [3].

The correlation between radiologic, vascular and clinical findings has been discussed in various articles. Ross et al. [4] described four patients in 1985 with Wallenberg syndrome with a correlation to MRI. Early MRI would show the lesions and allow for intervention if needed. In our patient immediate MRI was done and showed the abolition of the flow void and the angiography showed dissection of the left vertebral artery and treatment was initiated to save the brainstem from further damage.

Kim et al. [5] wrote about the correlation between the vascular lesions and the MRI of 34 patients with different patterns of Wallenberg syndrome. All their patients underwent angiography and their findings suggest that the larger infarct are associated with multiple vessel involvement, dissection, poor collaterals and the smaller infarcts are associated with single vessel, good collaterals and long standing atherothrombotic or embolic conditions. Also the speed of development of the vascular lesion determines the eventual size of the infarct and consequent clinical syndromes.

Kim [6] described in his article the symptoms-radiological correlation in 130 patients with acute lateral medullary syndrome where he noted that the most sensory symptoms-signs were the most frequent manifestation (96%) and that the limb/body involvement without trigeminal involvement (isolated limb/body pattern) in 21%. Headache occurred in 52%, dull, aching, throbbing and paroxysmal, started with or before the onset of other symptoms-signs and subsided in several days or weeks. They occurred most often in the ipsilateral occipital area, followed by the frontal region. According to the author, in patients with caudal infarcts, there are less frequent presence of dysphasia and dysarthria; less bilateral trigeminal sensory pattern, and more frequent isolated limb/body sensory pattern and sensory gradient worse in the leg than those with rostral lesions attributed to the lateral-superficial configuration of the infarct. The most lateral infarct produced lesion-isolated limb/body and sensory gradient worse in the leg due to anatomical characteristics of the medulla: the spinothalamic fibers from the upper extremities and torso run medially deeper whereas those from the lower extremities run laterally more superficially. Angiography showed that infarction and dissection are the most common causes; dissection was seen more often in patients with caudal than rostral lesions.

Our patient had minimal symptoms related to the involvement of the left lateral cervical and thoracic spinothalamic tract conducting pain and temperature sensation from the right upper extremity, right hemithorax and the upper outer part of the right gluteal area with a coherent MRI of the brain stem. The sparing of the other component of the medulla excluded the occurrence of the other items of the Wallenberg syndrome. These may be explained by the dissecting left vertebral artery, a rapid occurrence, with subsequent thrombus that showered small emboli to the brain stem causing these symptoms.

Our patient was started on oral anticoagulation and his condition remained stable and he was discharged home. On follow-up at seven months, he remained anticoagulated with hypothermalgesia at the upper-external gluteal area only.

REFERENCES