Brain Stem: Medial and Lateral Medullary Syndrome

Brain stem syndromes are characterized by affliction of precise structural entities with specific consequent clinical manifestations. Relevant appraisal of the medulla's anatomy and blood supply is essential to understanding the peculiar features of medullary syndromes. In this article, we emphasize clinical characteristics of medial medullary syndrome and lateral medullary syndrome considering the medulla's topographical organization. We conclude with therapy and prognoses.

Organization and Blood Supply of Medulla

The medulla oblongata is the terminal segment of the brain stem, which continues, at the foramen magnum, as the spinal cord. The cross-section shows the structures at the level of the pyramids and the inferior olivary nucleus.

Constituting the medial medulla
Pyramids: decussating corticospinal tracts, located between the median fissure and ventrolateral sulcus, constitute the pyramids.

Hypoglossal nuclei and nerve fibers: arise from the meeting point of the pons and medulla.

Medial longitudinal fasciculus: connects various cranial nerve nuclei, such as III, VI, and VIII, with the spinal cord, bringing about coordination in eye, body, and neck movements.

Medial lemniscus: mediating position, vibration, and fine touch sensations. The posterior column fibers from the gracile and cuneatus nuclei cross, as internal arcuate fibers, and continue further in the contralateral medial lemniscus to the thalamus.

Collectively constituting the lateral medullary segment

Inferior cerebellar peduncle: mainly concerned with proprioception, integration, and coordination of motor tasks. The following tracts traverse the inferior cerebellar peduncle:

<table>
<thead>
<tr>
<th>Tract</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dorsal spinocerebellar tract</td>
<td>Conveys unconscious proprioception from the spinal cord</td>
</tr>
<tr>
<td>Climbing fibers from the inferior olivary nucleus to the cerebellum</td>
<td>Provides input to the cerebellum for motor coordination</td>
</tr>
<tr>
<td>Vestibulo-cerebellar tract</td>
<td>Relays vestibular information to the flocculonodular lobe (vestibulocerebellum)</td>
</tr>
<tr>
<td>Cuneo-cerebellar tract (originates in the ipsilateral accessory cuneate nucleus)</td>
<td>Mediates unconscious proprioceptive information from the neck and upper limb</td>
</tr>
</tbody>
</table>

Vestibular nuclei: located on the dorsolateral regions of the medulla and pons, it is comprised of four sub-nuclei. It gives rise to the vestibular component of
the vestibule-cochlear nerve (eighth cranial nerve), which is important for maintaining balance, orientation, and proprioception of the head and body in the space. The subnuclei are:

- Inferior vestibular nucleus (Spinal or descending nucleus)
- Medial vestibular nucleus (Schwalbe nucleus)
- Lateral vestibular nucleus (Deiter nucleus)
- Superior vestibular nucleus (Bechterew nucleus)

**Spinal nucleus of the trigeminal nerve:** gives rise to the trigeminothalamic tract, which mediates pain and temperature sensations from the face.

**Nucleus ambiguus:** gives rise to the branchiomotor supply of IX, X, and XI cranial nerves.

**Solitary nucleus:** the rostral part mediates taste sensation, while the dorsal segment is responsible for visceral sensations and the carotid reflex.

**Descending sympathetic fibers:** proceed towards the ciliospinal center of Budge in the cervical spinal cord.

**Spinothalamic tract:** mediates pain and temperature sensations from the body to the thalamus.

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**Blood supply**

The medulla’s blood supply comes from the vertebrobasilar trunk and its branches. The two vertebral arteries join to form the basilar artery. Three types of end arteries supply the medulla:

- Long circumferential vessels
- Short circumferential vessels
- Paramedian perforators.

The major long circumferential vessels and their vascular territories are:
<table>
<thead>
<tr>
<th>Vessel</th>
<th>Vascular territory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior spinal artery (ASA)-branch of the vertebral artery (VA)</td>
<td>Medial medulla</td>
</tr>
<tr>
<td>Posterior inferior cerebellar artery (PICA)-branch of the VA</td>
<td>Posterolateral medulla and part of cerebellum</td>
</tr>
</tbody>
</table>

Depending on the vascular territory involved, occlusion of specific branches may lead to specific symptoms and manifestations.

**Medial Medullary Syndrome**

First described in 1908 by Spiller, medial medullary syndrome is characterized by damage to and dysfunction of the medial medullary structures.

Dejerine introduced the triad of contralateral hemiplegia, sparing the face, contralateral loss of deep sensation, and ipsilateral hypoglossal paralysis to this disease, now also known as Dejerine Syndrome. It is also variably known as hypoglossal alternating hemiplegia, inferior alternating syndrome, and lower alternating hemiplegia.

**Lateral Medullary Syndrome**

Damage to the lateral medulla leads to lateral medullary syndrome. Gaspard Vieszseux first described it in 1808. It is also known as ‘Wallenberg syndrome.’ The following variants are of interest:

**Opalski syndrome**: lateral medullary syndrome with ipsilateral hemiplegia

**Babinski-Nageotte syndrome**: lateral medullary syndrome with contralateral hemiplegia.

The lateral medullary syndrome may be complete or partial, depending on the vessel and
the subsequent vascular territory involved.

All brain stem syndromes are ‘crossed syndromes’ involving the ipsilateral cranial nerve and contralateral motor weakness.

Epidemiology

There are very few clinically and pathologically proven cases of medial medullary syndrome. Lateral medullary syndrome is more common. According to a study, the highest incidence is seen in middle-aged males at 50-60 years. Hypertension, diabetes, smoking, and atherosclerosis are the major risk factors.

Pathogenesis of Medullary Syndrome

Medial medullary syndrome is usually caused by infarction following vascular insult to the medial medulla. Occlusion of the ASA or damage to the paramedian perforators culminates in medial medullary syndrome.

Infarction of the lateral medulla, following insult to the PICA, the vertebra-basilar trunk, or brain stem perforators, gives rise to lateral medullary syndrome.

The ectogenesis is similar in both syndromes.

Infarction and vascular insult:

- **Thrombosis**: secondary to atherosclerosis and hypertension (most common); less likely due to hypercoagulable states
- **Embolic occlusion**: seen secondary in cocaine abuse, medullary neoplasms, radionecrosis, hematoma, neck manipulation, trauma, and bullet injury to the vertebral artery
- Spontaneous, post-traumatic, or iatrogenic vertebral artery dissection
- Post-traumatic or iatrogenic vertebral-basilar trunk occlusion/injury
- **Vasculitis**
- Post-infective vasculitis or occlusion
- Post-endovascular intervention
- Post-surgical intervention for vascular anomalies like AVM
- Post-surgical intervention of the spine
- Other rare causes are:
  - Structural lesions such as brain tumors
  - Hemorrhage
  - Vascular anomalies: leading to “steal phenomenon,” causing secondary hypoperfusion of the medulla
  - Idiopathic.

Classification of Medullary Syndrome

Based on the location of the infarct on imaging, there are three types of medial medullary syndrome:

- **Ventral**: contains the pyramid (most common)
- **Middle**: includes the medial lemniscus
- **Dorsal**: includes the medial longitudinal fasciculus.
Symptoms and Diagnosis of Medullary Syndrome

**Brain stem strokes** are acute ischemic events, characterized by vomiting, vertigo, diplopia, headache, ataxia, hiccups, and dysarthria. Facial paresis, though rare, can be seen due to the involvement of few aberrant descending fibers. It can cause many system ailments, such as vestibule-cerebellar, bulbar, sensory, respiratory, or autonomic systems.

More often due to a systemic cause, such as **hypertension** and **atherosclerosis**, medial medullary syndrome is often **bilateral** with a poor prognosis.

The tracts involved, with resultant specific symptoms in medial medullary syndrome, can be summarized in the following manner:

<table>
<thead>
<tr>
<th>Tract involved in medial medullary syndrome</th>
<th>Manifestation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoglossal nerve fibers</td>
<td>Ipsilateral tongue weakness, deviation of the tongue to the ipsilateral side</td>
</tr>
<tr>
<td>Corticospinal nerve fibers in the &quot;pyramid&quot;</td>
<td>Contralateral motor weakness</td>
</tr>
<tr>
<td>Medial lemniscus</td>
<td>Contralateral posterior column sensations affection-propiroception, vibration, and fine touch with relative sparing of pain and temperature</td>
</tr>
<tr>
<td>Medial longitudinal fasciculus (MLF)</td>
<td>MLF disruption leads to “internuclear ophthalmoplegia,” characterized by failure to adduct the contralateral eye</td>
</tr>
</tbody>
</table>

Clinical recognition of lateral medullary syndrome depends on identifying the **triad of ipsilateral hyperalgesia of the face, Horner’s syndrome, and ipsilateral ataxia**.

<table>
<thead>
<tr>
<th>Tract involved in lateral medullary syndrome</th>
<th>Manifestation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferior cerebellar peduncle</td>
<td>Ipsilateral ataxia, ipsilateral appendiceal cerebellar signs as dysmetria, intentional tremors</td>
</tr>
<tr>
<td>Vestibular nuclei</td>
<td>Vomiting, vertigo, nystagmus, diplopia.</td>
</tr>
<tr>
<td>Trigeminothalamic tract from the spinal nucleus of the trigeminal nerve</td>
<td>Loss of pain and temperature sensation from ipsilateral half of the face</td>
</tr>
<tr>
<td>Nucleus ambiguus and the solitary nucleus</td>
<td>Lower cranial nerve dysfunction-hoarse voice, nasal twang, dysphonia, dysphagia, decreased gustation, weak gag, regurgitation, aspiration, and increased risk of aspiration pneumonia</td>
</tr>
<tr>
<td>Lateral spinothalamic tract</td>
<td>Loss of pain and temperature sensation from the contralateral half of the body</td>
</tr>
<tr>
<td>Descending sympathetic fibers</td>
<td>Ipsilateral Horner’s syndrome (miosis, ptosis, anhydrosis, and loss of ciliospinal reflex)</td>
</tr>
<tr>
<td>Central tegmental tract</td>
<td>Palatal myoclonus</td>
</tr>
</tbody>
</table>

**Symptoms of lateral medullary syndrome**

- Vertigo
- Nausea and vomiting
- Blurred vision
- Imbalanced sitting posture
- Horizontal or rotational nystagmus
- Loss of sensation of pain and temperature on half of the face
- Loss of sensation of pain and temperature on the contralateral half of the trunk
- Hoarseness
- Diplopia
- Dysphagia
- Hiccups
- Dysphonia
- Horner’s syndrome
- Poor gag reflex
- Ataxia of both limb and gait
- Bradycardia

**Diagnosis**

After assessing the present symptoms, a neurological examination should be performed to differentiate between true stroke and mimicking stroke by this syndrome. **HINTS (head- impulsive-nystagmus- test of skew)** is a three-step oculomotor examination for determining the presence of infarction in lateral medulla.

**Imaging studies** are the most common preliminary investigations. **CT brain (plain)** is the initial investigation, mainly to rule out hemorrhage. **MRI diffusion** detects infarcts within a few minutes. CT and MRI scans determine the infarction’s exact location. The etiology can be detected by performing studies like **CT angiography, MRI angiography, and arterial neck Doppler**. For large vessel disease, **digital subtraction angiography** is the gold standard.

**Therapy and Prognosis of Medullary Syndrome**

Management is usually conservative. **Blood pressure regulation** with cerebral perfusion maintenance must be established immediately. In the case of ischemic stroke, blood thinners, like heparin or warfarin, reduce the blockage in the arteries supplying the lateral side of the medulla. The long-term outlook depends on active rehabilitation and symptomatic treatment.

Repetitive transcranial magnetic stimulation will rehabilitate patients with dysphagia due to Wallenberg syndrome.

Patients with lower cranial nerve weakness due to lateral medullary syndrome are prone to **aspiration pneumonia**. They might benefit from **Ryle’s tube feeds** or diverted feeding techniques such as **gastrostomy**. Patients who require **tracheostomy** usually have guarded prognoses.

The role of surgery, though minimal, can be summarized as:

- For structural lesions
- For vascular lesions like AVM
- For vertebrobasilar stenosis, an endovascular intervention can also be performed.

Medullary syndromes are **lethal in many patients**. Those who survive are usually afflicted by **central post-stroke pain**.

**Summary**

**Brain stem** syndromes are ‘**crossed syndromes,**’ usually secondary to **ischemic strokes** and infarcts.

**Medial medullary syndrome** is frequently caused by **occlusion of the ASA or**
Vertebral artery. It is identified by ipsilateral tongue weakness, ipsilateral tongue deviation, and contralateral motor weakness.

**Lateral medullary syndrome** is commonly due to PICA or vertebral artery occlusion. It manifests as a triad of ipsilateral ataxia, ipsilateral Horner’s syndrome, and ipsilateral hyperalgesia of the face. Loss of pain and temperature sensation in the contralateral half of the body is seen.

These medullary syndromes are diagnosed based on the **clinico-radiological correlation**. Management is usually conservative, with **high mortality and morbidity**.

**References**


DeJong’s Textbook of Neurology.

Youman’s Textbook of Neurological Surgery.


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