Lateral medullary infarct with alternating and dissociated sensorimotor deficits: Opalski's syndrome revisited

Hermann, D M; Jung, H H; Bassetti, C L
Lateral medullary infarct with alternating and dissociated sensorimotor deficits: Opalski’s syndrome revisited

Dirk M. Hermann, MD;¹,² Hans H. Jung, MD;² Claudio L. Bassetti, MD²
Department of Neurology, University Hospitals of ¹Essen, Germany, and ²Zurich, Switzerland

Number of figures: 1
Number of tables: 1
Number of references: 11
Number of words: 749

Correspondence:
Prof. Dr. Dirk M. Hermann
Department of Neurology
University Hospital Essen
Hufelandstr. 55
D-45122 Essen, Germany
Phone: +49 201 723 2814
Fax: +49 201 723 5534
e-mail: dirk.hermann@uk-essen.de

Keywords: stroke, lateral medulla, Wallenberg’s syndrome.
In 1946, Opalski described two patients with lateral medullary stroke presenting with alternating hypaesthesia (trigeminal hypaesthesia with contralateral limb/trunk hypalgesia), ipsilateral hemiparesis and hemiataxia [1]. Eight years later, two similar patients were reported by Cywinski et al. [2]. It was controversially discussed whether hemiparesis in atypical Wallenberg’s syndrome results from extension of a lateral medullary infarct into the pyramidal tract [3-7] or from hypotonic dysregulation of motor loops [8,9]. MRI inconsistently showed pyramidal tract involvement [3-7]. We present a patient with a hitherto undescribed Opalski variant with alternating and dissociated sensory symptoms in addition to motor hemiparesis, in whom neuroradiological and electrophysiological studies were performed.

This 73-year-old man presented with right lateropulsion, mild headache, dizziness, swallowing and speech problems starting two days before admission. As risk factors, arterial hypertension, hypercholesterolemia and nicotine abuse were noted. Neurological deficits consisted of right-sided Horner’s syndrome, facial hypaesthesia, glossopharyngeal palsy, facio-brachio-crural hemiparesis (M4) (Fig.1A) with extensor plantar responses and hyperreflexia, mild ataxia and brachio-cranial hemihypaesthesia. On the left side, hypothermalgesia was found.

MRI showed a lateral medullary infarct that extended ventrally to the most lateral pyramidal tract and caudally to the upper spinal cord (Fig.1B). Motor-evoked potentials revealed prolonged central conduction times to the right, but not left abductor digiti minimi (10.2ms; <8.7ms) and anterior tibial (17.5ms; <17.1ms) muscles (Fig.1C). Tibial nerve sensory-evoked potentials exhibited delayed upper cervical but not lumbar responses on the right but not left side (35.5ms; <34.3ms). Hence, all deficits could be attributed to a single medullary lesion.

The cause of hemiparesis in lateral medullary stroke is controversially discussed. In 130 patients with MRI-documented lateral medullary infarcts [10], Kim identified twelve patients with ipsilesional tactile hypaesthesia, of which seven had mild ipsilesional hemiparesis [8]. In view of mild motor deficits and absent reflex abnormalities, Kim raised doubts whether hemiparesis was attributed to a pyramidal lesion, arguing in favour that the hemiparesis represented a spino-cerebellar hypotonic hemisindrome [9].

Opalski’s [1] and Cywinski et al.’s [2] patients differ from Kim’s cases as there were clinical signs of corticospinal tract injury (accentuated reflexes, Babinski’s sign) (Table). Reflex abnormalities and/or Babinski’s sign were rarely described ever since:
Of six patients only one had typical reflex abnormalities [7], while the others did not [3-6]. The question remains open as whether motor paresis in these patients reflected true corticospinal lesions. Possibly, injuries to aberrant fibres crossing through the brainstem [11] might contribute to motor deficits.

While contralesional hypothermalgesia is typical in Opalski’s syndrome (Table), dissociated sensory symptoms have never been reported. In all earlier patients, tactile deficits were absent on the paretic side, indicating an alternating, but not truly dissociated syndrome. Our patient exemplifies that both alternating and dissociated sensory deficits may be induced by a single lower brainstem lesion. We suggest that the eponym Opalski’s syndrome should be reserved for patients presenting with ipsilesional hemiparesis, hyperreflexia and Babinski’s sign, and contralesional hypothermalgesia.

References


**Fig.1** Photograph (A), MRI (B) and motor evoked potentials (C) in Opalski variant. Note the right-sided arm paresis (A) following lateral medullary stroke that extends to the pyramidal tract and cervical spinal cord (B). Motor evoked potentials in abductor digiti minimi muscles (C) exhibit delayed responses after cortical, but not cervical or peripheral stimulation on the right (R), but not left (L) side. White arrows in (B) indicate the infarct, black arrows in (C) the minimal corticomuscular latency.
**Table** Neurological observations in the historical patients by Opalski [1] and Cywinski et al. [2], as well as in more recent cases of autopsy [3] or MRI [4-7]-confirmed lateral medullary infarcts considered as Opalski’s syndrome

<table>
<thead>
<tr>
<th>Age</th>
<th>Stroke etiology</th>
<th>Ipsilesional deficits</th>
<th>Contralesional deficits</th>
<th>Associated findings</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>57 yrs.</td>
<td>Unknown</td>
<td>Mild hemiparesis (FBC) with hyperreflexia/ Babinski’s sign, hypaesthesia te/pa (F), hemiataxia</td>
<td>Hypaesthesia te/pa (BC)</td>
<td>Gait ataxia</td>
<td>Opalski [1]</td>
</tr>
<tr>
<td>30 yrs.</td>
<td>Unknown</td>
<td>Severe hemiparesis (BC) with hyperreflexia, hypaesthesia te/pa (F), Horner syndrome</td>
<td>Hypaesthesia te/pa (T)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>63 yrs.</td>
<td>Athero-thrombotic</td>
<td>Mild hemiparesis (BC) with hyperreflexia/ Babinski’s sign, hypaesthesia te/pa (F: V₁,2), hemiataxia (BC), position sense disturbance, Horner syndrome</td>
<td>Hypaesthesia te/pa (BC)</td>
<td>-</td>
<td>Cywinski et al. [2]</td>
</tr>
<tr>
<td>60 yrs.</td>
<td>Athero-thrombotic</td>
<td>Mild hemiparesis (BC) with hyperreflexia, hypaesthesia te/pa (F: V₂,3), mild hemiataxia (B), Horner syndrome</td>
<td>Hypaesthesia te/pa (BTC)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>78 yrs.</td>
<td>VA occlusion</td>
<td>Severe hemiparesis (FBC)</td>
<td>Hypaesthesia te/pa (BC)</td>
<td>Dysarthria</td>
<td>Dhamoon et al. [3]</td>
</tr>
<tr>
<td>51 yrs.</td>
<td>VA occlusion</td>
<td>Mild hemiparesis (FBC), hypaesthesia te/pa (F), hemiataxia</td>
<td>Hypaesthesia te/pa (BC)</td>
<td>Dysarthria, dysphagia</td>
<td>Hommel et al. [4]</td>
</tr>
<tr>
<td>51 yrs.</td>
<td>Unknown</td>
<td>Mild hemiparesis (FBC), hypaesthesia ta (F), hemiataxia, Horner syndrome</td>
<td>Hypaesthesia te/pa (BC)</td>
<td>Gait ataxia, nystagm</td>
<td></td>
</tr>
<tr>
<td>69 yrs.</td>
<td>VA occlusion</td>
<td>Mild hemiparesis (FBC), hypaesthesia ta (F), Horner syndrome</td>
<td>Hypaesthesia ta (BC)</td>
<td>Gait ataxia, dysarthria, dysphagia</td>
<td>Montaner et al. [5]</td>
</tr>
<tr>
<td>42 yrs.</td>
<td>Unknown</td>
<td>Hemiparesis, (BC), hypaesthesia te/pa (F)</td>
<td>Hypaesthesia te/pa (BC)</td>
<td>Gait ataxia</td>
<td>Kimura et al. [6]</td>
</tr>
<tr>
<td>57 yrs.</td>
<td>Unknown</td>
<td>Hemiparesis (BC) with hyperreflexia, hypaesthesia te/pa (F), Horner syndrome</td>
<td>Hypaesthesia te/pa (BC)</td>
<td>Gait ataxia, dysarthria, dysphagia</td>
<td>Sanahuja et al. [7]</td>
</tr>
<tr>
<td>73 yrs.</td>
<td>VA occlusion</td>
<td>Mild hemiparesis (FBC) with hyperreflexia/ Babinski’s sign, hypaesthesia ta (FBC)/ te/pa (F), position sense disturbance, hemiataxia, Horner syndrome</td>
<td>Hypaesthesia te/pa (BTC)</td>
<td>Dysarthria, dysphagia</td>
<td>Hermann et al.</td>
</tr>
</tbody>
</table>

F, facial; B, brachial; T, truncal; C, crural; ta, tactile, te, temperature; pa, pain; VA, vertebral artery