CASE REPORT

A crossed brain stem syndrome without crossed sensory symptomatology

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SUMMARY

Lateral medullary infarction (LMI) or Wallenberg syndrome is a type of brain stem stroke, more specifically, a type of crossed brain stem syndrome. LMI is a well-described entity with several documented typical characteristics including pain and temperature impairment in the ipsilateral to the lesion side of the face and the contralateral side of the trunk and limbs. We present a case of LMI which describes a patient who presented with atypical features of analgesia and thermoaesthesia on the contralateral side of the face and absence of sensory deficit on the ipsilateral side. We attributed this pattern of involvement to a lesion that affects the ventral trigeminothalamic tract and spares the dorsolateral part of the medulla where the spinal trigeminal tract and its nucleus lie. This case report highlights the presence of atypical presentations of LMI that may initially challenge the physician's diagnostic reasoning.

BACKGROUND

Lateral medullary infarction (LMI) or Wallenberg syndrome is one of the most common crossed brain stem syndromes. In this case, we encountered a patient with an atypical presentation of LMI. His MRI confirmed that the lesion was located in the medulla. The exact location of the lesion was responsible for the atypical presentation of this patient.

CASE PRESENTATION

A 72-year-old man presented with hoarseness, postural instability, vertigo and headaches for 2 days prior to admission. He also complained of difficulty in swallowing both liquids and solids on those 2 days. He denied having any recent head trauma and taking any medications. Ten years earlier, he was involved in a motor vehicle accident where he sustained a head injury but made a complete recovery without any neurological consequences. His family history is not significant; he does not smoke or consume alcohol. Upon examination, the patient's blood pressure was 167/104 mm Hg, heart rate was 55 beats/min and he had a normal temperature and respiratory rate. Upon neurological examination, the patient was oriented in time and space and had horizontal nystagmus with a slow phase to the left. The left pupil appeared slightly constricted, and both pupils were reactive to light. His gag reflex was diminished. The motor power and reflexes were normal and equal in all four extremities. When standing upright, he felt lateropulsion to the left. The

finger-to-nose test revealed dysmetria on the left. Sensation to pain and heat was absent in the right trunk, right limbs and right half of the face. Ear, nose and throat examination revealed paralysis of the left vocal cord. During the course of his illness, he developed refractory hiccups. The rest of the exam was unremarkable.

INVESTIGATIONS

Upon admission, the patient underwent an emergency brain CT. The results were normal.

An emergency MRI was not performed because of the relatively benign clinical course, and the symptomatology highly suggested that this was a case of LMI. After 6 months, an MRI brain scan and a magnetic resonance angiography (MRA) of the cerebral circulation were performed for follow-up.

TREATMENT

- ▶ Acetylsalicylic acid 100 mg once daily orally
- ▶ Nadroparin 2850 anti-Xa IU once daily subcutaneously
- ▶ Atorvastatin 10 mg once daily orally
- ▶ He temporarily received tube feeding due to impaired swallowing ability
- ▶ He was prescribed a diuretic for the uncontrolled hypertension.

OUTCOME AND FOLLOW-UP

The patient was discharged after his neurological symptoms improved, and he was able to swallow liquids and food 3 weeks after his admission. After 6 months, he remains in good general condition,

Figure 1 The magnetic resonance angiography shows occlusion of the left vertebral artery.

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with some residual neurological symptomatology, which includes a mild deficit in pain and heat sensation on the right side of the body and face.

DISCUSSION

The diagnosis of LMI is based on the classical clinical features first described by Wallenberg in 1895.¹ However, the variability of the clinical presentation²⁻⁴ has proven to be much greater than what was initially described. This results from the variable arterial supply of the affected region^{5,6} and seems to correlate with the exact location of the infarct in the medulla.⁷⁻¹⁰ It has been reported that the most common cause of LMI is vertebral artery disease.^{7,8} In this patient, the MRA demonstrated occlusion of the left vertebral artery (figure 1). The patient presented with paralysis of the left vocal cord, nystagmus with a slow phase to the left, ataxia and dysmetria on the left, constricted left pupil, analgesia and thermanaesthesia in the right trunk and limbs. These exemplify the typical features of an LMI and are consistent

with a left-sided lesion in the medulla. The classical description of LMI includes analgesia and thermanaesthesia which affect the ipsilateral side of the face and the contralateral side of the body (figure 2). This is attributed to the involvement of the ipsilateral descending spinal trigeminal tract and its nucleus (which carries pain and heat sensation from the ipsilateral side of the face) and the lateral spinothalamic tract (which carries pain and heat sensation from the contralateral side of the body), respectively. The puzzling feature in this case was the presence of analgesia and thermanaesthesia on the same side of the face and body (figure 3; absence of crossed sensory symptomatology), with the rest of the symptoms suggesting a left-sided lesion in the medulla. After a brief review of the literature, we concluded that, in the present case, the dorsolaterally located spinal trigeminal tract and its nucleus had probably been spared and that the analgesia and thermanaesthesia in the contralateral face were due to the involvement of the ventral trigeminothalamic tract (figure 4). This tract is located more ventrally and medially in the medulla¹¹

Figure 2 The typical presentation of LMI involves sensory deficit in the ipsilateral side of the face and the contralateral side of the trunk and limbs (crossed sensory symptomatology).

Figure 3 In this case, the patient had analgesia and thermanaesthesia on the contralateral side of his face, trunk and limbs. The ipsilateral side of the face remained unaffected (absence of crossed sensory symptomatology).

Figure 4 Image adapted from Duvernoy's *Atlas of the human brain stem and cerebellum*.¹¹ Schematic demonstration of the lesion in the medulla oblongata. The exact location of this lesion (marked with solid parallel lines) affects the lateral spinothalamic tract (which is responsible for pain and heat sensation from the contralateral side of the trunk and limbs) and the more ventrally and medially located ventral trigeminothalamic tract (which is responsible for pain and heat sensation from the contralateral side of the face). The dorsolateral part of the medulla, where the spinal trigeminal tract and its nucleus lie (which are responsible for pain and heat sensation from the ipsilateral side of the face), has probably been spared (area within the broken line).

and consists of crossed second-order neurons that carry pain, temperature and light touch sensation from the contralateral side of the face. The MRI scan (figure 5) showed a ventrally and medially located lesion in the left medulla, sparing the dorsolateral medulla, where the spinal trigeminal tract and its nucleus lie. Therefore, the MRI scan was consistent with our neuroanatomical hypothesis. The results of other relevant studies confirm the mentioned clinical–neuroanatomical correlation in cases of LMI with this pattern of involvement.^{8–10} Zhang *et al*⁹ described five different patterns of LMI: type 1 involves sensory deficit in the ipsilateral face and the contralateral trunk and limbs; type 2, sensory deficit in the contralateral trunk, limbs and face in addition to the ipsilateral face; type 3, sensory deficit in the

contralateral face and body; type 4, sensory deficit in the ipsilateral face and the contralateral lower trunk and leg and type 5, sensory deficit only in the contralateral face, arm and upper trunk, without involvement of the ipsilateral face. Obviously, our patient belongs to the third category. Zhang *et al* attribute this pattern to a lesion that spares the spinal trigeminal tract along with its nucleus but disturbs the ventral trigeminothalamic tract. The hypothesis regarding a more ventral location of the lesion is consistent with the results of Kim,⁸ who described the symptoms of radiological correlation in 130 consecutive patients with LMI. According to this study, the contralateral trigeminal–contralateral limb/body pattern was observed in 25% of the subjects. Kim showed that patients with contralateral trigeminal involvement were more likely to have a ventrally located lesion in the brain stem. Conforto *et al*¹⁰ investigated the correlation between facial sensory abnormalities and lesion topography in eight patients with LMI, and their results showed that contralateral facial sensory abnormalities are related to medial extension of the infarct. In conclusion, sparing the spinal trigeminal tract and its nucleus while disrupting the

Figure 5 This T2 sequence MRI image of our patient shows a high signal (hyperintensity) in the left medulla, which is suggestive of an infarct. The lesion appears to be ventrally and medially extended (probably involving the ventral trigeminothalamic tract). The dorsolateral part of the left medulla, where the spinal trigeminal tract and its nucleus lie, seems to have been spared.

Learning points

- ▶ Lateral medullary infarctions (LMIs) may have atypical presentations.
- ▶ In some cases, such as the one presented in this study, the classical crossed sensory symptomatology may be absent. If the rest of the symptoms are highly suggestive of an atypical pattern described above, the lack of the typical pattern of involvement should not prevent one from diagnosing an LMI.
- ▶ A sensory deficit in the contralateral side of the face is attributed to a lesion that involves the ventral trigeminothalamic tract. This lesion is typically more ventrally and medially located in the medulla. When the physician observes that the ipsilateral side of the face remains unaffected, the dorsolateral part of the medulla and the spinal trigeminal tract and its nucleus have probably been spared.

ventral trigeminothalamic tract accounts for this atypical presentation. The prognosis of LMI is generally good; the majority of patients, including our patient, were able to recover and resume their previous activities.¹²

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