



Original Research Article

Pseudothalamic pattern of sensory loss in lateral medullary syndrome- A clinicoanatomic correlation

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ARTICLE INFO

Article history:

Received 23-09-2020

Accepted 21-10-2020

Available online 30-11-2020

Keywords:

Pseudothalamic pattern

Stopford classification

Lateral medullary syndrome

Central post

ABSTRACT

Context: Lateral medullary infarction is typically associated with loss of pain and temperature over ipsilateral face and contralateral body. However, sensory loss in contralateral face and body has been described in lateral medullary syndrome- the pseudothalamic pattern or type IV sensory loss of Stopford classification.

Aims: This article aims at identifying lateral medullary syndromes associated with sensory impairment over contralateral face and body, and reviewing the neuroanatomical substrate leading to the same.

Settings and Design: This was a prospective study conducted at Department of Neurology, at a tertiary level teaching hospital, Kerala, India, over a period of 5 years, among patients with acute ischemic stroke.

Methods and Material: We studied all patients with acute ischemic stroke, who were admitted to our department, over a five-year period. Patients presenting with lateral medullary syndrome, with a pseudothalamic pattern of sensory loss were shortlisted and were assessed by investigators independently. All patients underwent MRI brain, which was assessed by investigators 1 and 2, independently. The demographic profile, risk factors, clinical features, neuroimaging findings and outcomes were analysed.

Results: A total of 1492 patients with acute ischemic strokes were identified, of which 6 with lateral medullary syndrome with a pseudothalamic pattern of sensory loss were included in the study. Of the 6 patients, two had central post stroke pain, which was refractory to treatment.

Conclusions: The type IV sensory loss of Stopford classification or the pseudothalamic pattern of sensory loss is a less described pattern in lateral medullary syndrome. The central post stroke pain (CPSP) which may develop in these patients is difficult to treat.

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1. Introduction

The loss of pain and temperature sensation in ipsilateral face and contralateral body (crossed pattern) in lateral medullary syndrome is well recognized and has been referred to as type I sensory loss of Stopford classification.^{1,2} It occurs due to far lateral infarctions of the medulla. Sensory loss on the contralateral face, arm, trunk and leg (pure sensory stroke pattern) in lateral medullary- type IV sensory loss of Stopford classification, is less recognized.^{3,4} Previous studies have shown that that painful sensory symptoms can develop after stroke in patients with a non-thalamic stroke,

of which a major percentage occurs after later medullary strokes.⁵ This is referred to as the central post stroke pain (CPSP). It has been defined as a “chronic, spontaneously irritating sensation”.⁵

This article aims at identifying lateral medullary syndromes associated with pseudothalamic pattern of sensory loss, with review of the neural circuitry responsible, to aid in recognition and reporting. We also assessed the development of CPSP in these patients.

2. Materials and Methods

This was a prospective study conducted at the department of Neurology, at a tertiary level teaching hospital,

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in middle Travancore, Kerala, over a period of five years, from August 2015 to August 2020. We identified 1492 consecutive patients with acute ischemic strokes. Subjects presenting with acute onset of symptoms of lateral medullary syndrome, with sensory impairment over contralateral face and body, with 1.5 T MRI brain showing infarction in the lateral medulla, were included in the study group and were independently assessed by investigators 1 and 2. Pain perception was tested with the use of a pinprick and temperature with a cold tuning fork. Facial sensation was compared with the sensation of an intact limb. All the patients underwent a standard protocol of investigations, including MRI brain, 12-lead ECG and transthoracic echocardiography. The neuroimaging findings were assessed by investigators 1 and 2, independently. The demographic profile, vascular risk factors such as systemic hypertension, diabetes mellitus, dyslipidemia, and history of heart disease / chronic kidney disease were recorded. Functional outcome was assessed at discharge and at the end of three months. Statistical analysis was done using SPSSv 21.

3. Results

A total of 46 patients with infarction in the lateral medulla were identified. On the date of the examination, impaired sensation of pinprick and impaired appreciation of cold tuning fork was noted in the contralateral face, body/limb in six patients. This corresponds to type IV sensory loss of Stopford classification, also referred to as pseudothalamic pattern. Of the six patients, four were males two were females. Five out of the six patients (83.3%) were hypertensive, four were diabetic (66.6%) and 3 had dyslipidemia (50%). One had documented atrial fibrillation (16.6%).

The main clinical symptoms at presentation were vertigo, vomiting and unsteadiness and it was seen in all the six patients (100%). On examination, Horner's sign was noted in 5 patients (83.3%), nystagmus in 5 patients (83.3%) and palatal palsy in 3 patients (50%). A sensory symptom was elicited on history only in two patients (33.3%) and they described it as a pricking type of sensation over the face. (Table 1) Sensation over face was compared with the sensation of an intact limb, since it may be impaired bilaterally in some patients with lateral medullary syndrome. All the six patients included had impaired pain and temperature sensation over contralateral face, body and limb. MRI brain revealed diffusion restriction involving mid-lateral medulla in all these patients (Figure 1). In one of our patients, in addition to the medullary infarction, a cerebellar infarct was also noted. (Figure 1C).

On follow up, 3 months later, 2 patients (33.3%) had developed severe pain over the contralateral face and body/limbs, referred to as central post stroke pain (CPSP). They had severe allodynia. Both these patients developed this

symptom around 4 weeks after the stroke. Both these patients were started on Amitriptyline and on follow up 3 months later, the symptom was still persisting. In the rest of the four patients, there were no sensory symptoms on review at 3 months.

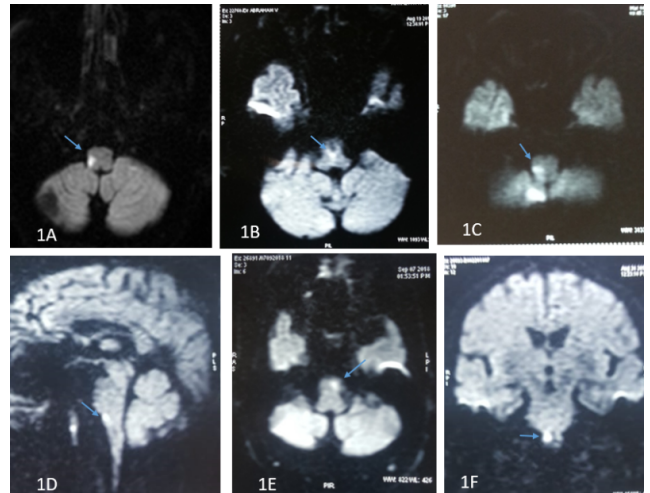


Fig. 1: Diffusion weighted MRI brain sequences of patients 1-6: 1A, B, C-axial sequence showing infarct in the right lateral medulla, the right ventromedial medulla, the right lateral medulla and cerebellum, respectively 1D-sagittal sequence showing infarct in the lateral medulla, 1E-axial sequence showing infarct in the left lateral medulla, 1F- coronal sequence showing infarct in the right lateral medulla.

4. Discussion

Lateral medullary syndrome is associated with various patterns of sensory disturbances, including ipsilateral, contralateral or bilateral facial sensory impairment, with varying sensory disturbances over the contralateral face, arm, trunk and legs.^{1,6,7} The contralateral loss of pain and temperature over the trunk is due to involvement of the spinothalamic tract in the lateral medulla. Owing to the specific somatotopic organisation of the spinothalamic tract, with the cervical afferent fibres being arranged medially and the lumbosacral afferent fibres arranged laterally, the sensory loss may be partial (involving either upper or lower part of the body).⁴

In the classical pattern of sensory loss, i.e., loss of sensation over the ipsilateral face and contralateral body, the descending trigeminal tract and the spinothalamic tract are involved. This is referred to type I sensory loss of Stopford classification. It is seen in far lateral lesions of the medulla.¹ In type II sensory loss of Stopford classification, the contralateral face and contralateral upper part of the body is affected. It is seen in more medial lesions. Here, the crossed ventral trigeminothalamic tract, carrying pain and heat sensation from the contralateral side

Table 1: Showing the clinical features of the patients included in the study

Case No:	1	2	3	4	5	6
Age						
Sex	F	F	M	M	M	M
Vertigo	+	+	+	+	+	+
Vomiting	+	+	+	+	+	+
Ataxia	+	+	+	+	+	+
Horner's	-	+	+	+	+	+
Nystagmus	-	+	+	+	+	+
Palatal palsy	-	-	+	+	+	-
Subjective sensory symptoms	+	-	+	-	-	-
Objective loss of pain & temperature over contralateral face and body	+	+	+	+	+	+
Central post stroke pain	-	+	-	+	-	-

F- female, M- male

of the face, near the medial part of the spinothalamic tract gets involved. Since the medial part of the spinothalamic tract is involved, the cervical afferent fibres which ascend medially are affected, hence the sensory loss is confined to contralateral upper body.⁴ Type III sensory loss occurs in large mediolateral lesions, here both the descending and ascending trigeminal tracts and the spinothalamic tracts are affected, resulting in sensory loss over bilateral face and contralateral body (bilateral type).^{2,4} In type IV sensory loss, there is loss of sensation over the contralateral face and contralateral body, due to involvement of the crossed ventral trigeminothalamic tract and spinothalamic tract.⁴ Vaudens et al have first described this pattern and they noted that retro-olivary area in the ventromedial tegmentum was affected in these patients. In our study, type 4 sensory loss was seen in 6 out of 46 patients (13.1%). This is comparable to the study by Kim et al, where 7 out of 41 patients with lateral medullary infarction has type 4 sensory loss (17.1%).¹ In another study on pure lateral medullary infarction, around 25 % of patients had type 4 sensory loss.³

Previous reports suggest that that painful sensory symptoms can develop after stroke in patients with a non-thalamic stroke.⁵ Lateral medullary strokes contribute to a significant portion of nonthalamic central poststroke pain (CPSP) syndrome. Previous studies have noted that CPSP after the spinothalamic tract injury in lateral medullary syndromes is due to hypersensitivity phenomenon-uninhibited firing of central neurons through excessive feedback mechanism following partial damage.⁸ One mechanism proposed is that since the spinothalamic tract is near the spinoreticulothalamic tract, deafferentation of the spinothalamic tract renders the reticulothalamic system responsive to stimulation, thus provoking painful sensations.^{9,10} In our study 2 patients developed CPSP, which was not very responsive to treatment, as observed by MacGowan et al.⁵ They developed this symptom around 4 weeks after the stroke. The delayed onset of CPSP may be due to the time required for the partial recovery of the fibres

of the injured spinothalamic tract, through sprouting and receptor hypersensitivity.¹

5. Conclusion

The type IV sensory loss of Stopford classification or the pseudothalamic pattern of sensory loss is a less described pattern in lateral medullary syndrome. The central post stroke pain (CPSP) which may develop in these patients is difficult to treat.

6. Source of Funding

No financial support was received for the work within this manuscript.

7. Conflict of Interest

The authors declare they have no conflict of interest.

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Cite this article: Sheetal S, Thomas R. Pseudothalamic pattern of sensory loss in lateral medullary syndrome- A clinicoanatomic correlation. *IP Indian J Neurosci* 2020;6(4):272-275.