

# CASE REPORT

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## CORTICAL STROKE CAUSING GRADED FACIAL SENSORY INVOLVEMENT

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**ABSTRACT:** Cortical stroke produces unusual clinical findings. Cortical sensory representation is believed to be unilateral and contralateral. Here we report a patient with graded sensory loss due to cortical stroke. Stimulation studies reveal sensory facial representation of upper face to be bilateral, while that of lower face is unilateral and contralateral. The corollary of these studies explain our findings, the upper part of the face due to its bilateral representation is least effected, while the lower face due to its unilateral and contralateral representation is most severely involved in cortical stroke.

**KEYWORDS:** Cortical Stroke, facial sensory loss, somatotopic representation, unilateral, bilateral.

**INTRODUCTION:** Cortical stroke causing sensory involvement confined to face is unusual. Isolated facial sensory findings have been described in brain stem strokes, classically in Lateral medullary syndrome though uncommon.<sup>(1)</sup> Very rarely cases had been reported where by stroke has caused sensory symptoms confined to face.<sup>(2,3)</sup> Here a patient with an unusual sensory loss involving the hemiface alone, that too with a graded severity, least involved in the upper face and severe in the lower, due to a cortical stroke is reported.

**CASE:** 52 year old non-hypertensive, non-diabetic, a habitual smoker and alcoholic developed vague head sensation was admitted in the local hospital. The next day he developed right sided facial weakness, numbness of the right side of face. The numbness was described as altered sensation of touch, which was mild over the forehead with total numbness and inability to feel any sensation in the lower face. He did not have vertigo, vomiting, ataxia, nasal twang or nasal regurgitation.

Examination showed central facial palsy on the right, impaired corneal reflex, sensory impairment of the right side, was able to appreciate touch, pain and temperature on the upper face (corresponding to approximately V1), reported as the touch felt different from the opposite side of the face and other body parts. But on the lower face, he was not able to feel any sensation and reported that area (corresponding to V3) felt totally numb. His motor power was normal with normally elicited reflexes in the limbs, without ataxia or sensory signs.

- His systemic exam including cardiovascular exam was normal.
- His metabolic workup showed euglycemia and hypercholesterolemia.
- CT scan head was normal. MRI showed diffusion restriction in the pre and post central gyri suggestive of acute cortical infarcts. MRA was normal.
- Stroke workup with ECG and echocardiogram were normal.
- He was treated with anti-platelets and statins.
- Over the days his facial weakness and sensory signs improved.

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**DISCUSSION:** Sensory symptoms caused by stroke can be of various types. The common ones are hemisensory symptoms or sensory symptoms confined to acral areas producing cheiro-oral, cheiro-paedal or cheiro-oro-paedal syndromes.<sup>(3,4,5&6)</sup> Strokes can also produce atypical sensory loss like pseudomyelopathic, pseudoradicular or cortical pattern of sensory disturbance depending on the topography of the lesion.<sup>(5)</sup> Still rare are the sensory symptoms involving the non-acral parts-the discrete areas of the contralateral proximal arm, shoulder, trunk and upper thigh.<sup>(7)</sup>

Sensory symptoms in which face alone getting involved is described in lateral medullary syndrome, rarely where ipsilateral, contralateral or bilateral is reported.<sup>(1)</sup> Two case reports, one in which lower part of the face involving part of mandibular division, but not encompassing it fully was involved, where the lesion was localized to a lacune in the ventroposteromedial nucleus of thalamus and another in which numbness in the tip of tongue and lower lip caused by thalamic haemorrhage.

Afferent corneal reflex is lost in peripheral or pontomedullary trigeminal pathways, however there is a suprasegmental modulation of the reflex, as somatotopic representation of the cornea to painful stimuli is localized in the primary somato sensory cortex, in the middle of the superior inferior extent of contralateral S1, corresponding to contralateral face.<sup>(8)</sup>

In this case it was a cortical stroke responsible for the deficit. Both motor cortex and to a lesser extent sensory cortex (pre and post-central gyri) were involved.

Kim has analysed the pattern of sensory pattern abnormality in cortical stroke.<sup>(9)</sup> It differed depending on the site of lesion. He described three types of sensory abnormalities.- predominantly cortical sensations were involved when the lesion was in the upper parietal lobe and posterior central gyrus, anterior parietal artery distribution, while primary modalities were involved in parietal operculum and insular cortex, posterior parietal artery territory with angular branch involvement. In the third group where the sensory was only subjective paresthesia without any objective deficit, then lesion was very small in the parietal operculum and insular cortex. Here in our patient, we had two types of sensory involvement, pertaining to different parts of the parietal lobe and arterial territories, the upper facial symptom (V1) was only subjective paresthesia, while in the lower face (V3) all modalities were effected. To explain this finding of mild and severe sensory involvement, i.e, differential severity of involvement, mild involvement of parietal operculum and insular cortex which accounts for the subjective paresthesia over the forehead or V1 region and severe involvement of the same area causing loss of all modalities of sensation, should occur, which is not possible.

Based on activation foci, segmentally arranged somatotopy was noted in the post central gyrus-primary somatosensory cortex.<sup>(10)</sup> Activation of primary somatosensory cortex displayed a laminar response resembling the trigeminal nerve divisions. V2 more rostral, V1 caudal and V3 medial. But here the lesion was a very small one involving the post central gyrus, which again cannot explain the large area which needs to be involved in the sensory homunculus, accounting for the present sensory symptom of hemi facial sensory involvement.

Iannetti et al<sup>(11)</sup> described the effect of mechanical stimulation of different trigeminal divisions within the primary and secondary human somatosensory cortex. The signals differed between V1 and V3 stimulation. On mechanical stimulation of the V1 and V3 divisions, the fMRI signals in the primary somatosensory cortex, S1 and secondary S11 in the contralateral

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hemisphere increased. Within S1 and S11 the foci activated by stimulation of the two trigeminal divisions largely overlapped. But ipsilateral representation differed. When V3 stimulation activated contralateral cortex alone, V1 activated S1 and S11 bilaterally, showing distinct cortical representation within the facial territories.

Thus, in this interesting case of sensory involvement of the hemiface alone, where the forehead corresponding to V1 showed only sensory symptom of paresthesia and subjective altered touch alone with total numbness and loss of all modalities of sensation in the lower face corresponding to V3, it could be that the upper face have bilateral representation and the lower unilateral, one, contralaterally for sensations, by which the mild involvement of upper face and severe involvement of lower face can be explained. This is in par with that of motor facial involvement where central facial palsy produces predominantly contralateral lower facial weakness, but the explanation accounting for the same differs.<sup>(12)</sup>

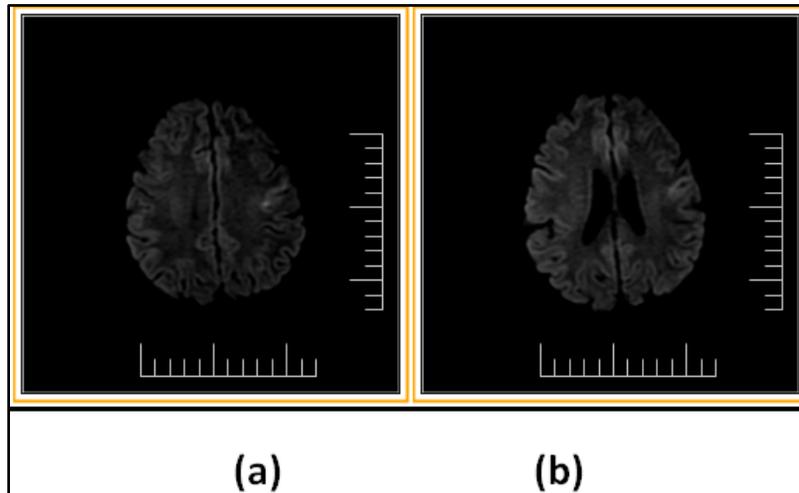
It is from strokes and the dysfunction caused by it; in addition to the stimulatory experiments by Penfield and Jasper the cortical functions were best studied. Here, from this case, we find that sensory representation for upper face is bilateral while for the lower part, it is unilateral and contralateral.

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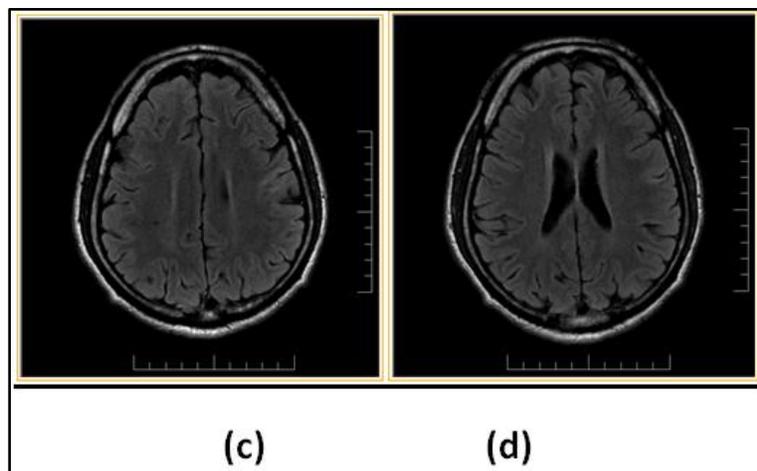
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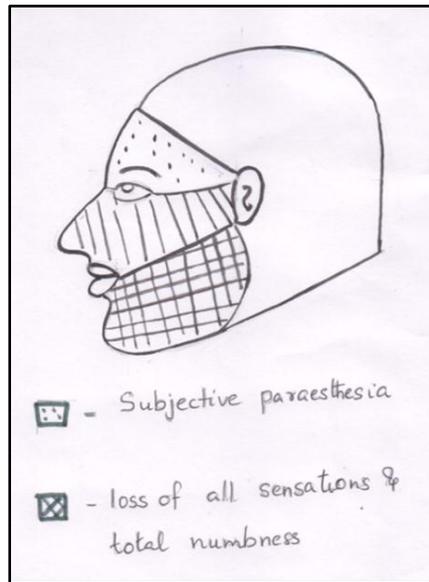


**Fig. (a) & (b): Diffusion Weighted images showing cortical hyperintensity in the left Peri-rolandic region.**



**Fig. (c) & (d): T2 FLAIR images showing cortical hyperintensity in the left Peri-rolandic region**

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