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Brain Stem Infarction Presenting as Isolated Trigeminal Sensory Neuropathy (Case Report)

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Objective: We present a patient with isolated trigeminal sensory neuropathy secondary to the involvement of brain stem.
Material and method: A 49 year-old man who has numbness on the right side of his face and oral cavity was investigated. Cranial MRI revealed ischemic focus at right inferior dorsolateral pontine region. The only risk factor was protein S deficiency. The symptoms of the patient disappeared almost completely within a month.
Conclusion: When an isolated trigeminal sensory neuropathy is diagnosed, one should think of brain stem involvement and investigate the risk factors for occlusive vessel diseases.

Key Words: Trigeminal sensory neuropathy, Brain stem infarction, Protein S deficiency

İzole Trigeminal Sensoriyel Nöropati ile Presente Olan Beyin Sapı İnfarktı (Olgu Sunumu)

Amaç: Beyin sapı tutulumuna bağlı izole trigeminal sensoriyel nöropatisi olan bir olgu sunulmaktadır.
Gereç ve yöntem: Yüzünün sağ yarısı ve ağız içinde uyuşma nedeni ile incelenen 49 yaşındaki erkek hastanın kranial MRG'sinde sağ inferior dorsolateral pontin bölgede iskemik odak saptandı. Hastadaki tek risk faktörü protein S eksikliği idi. Hastanın yakınmaları bir ay içinde tamamen düzeldi.
Sonuç: İzole trigeminal sensoriyel nöropati tanısı konduğunda beyin sapı tutulumuna bağlı olabileceği düşünülmeli ve tıkayıcı damar hastalığı için risk faktörleri araştırılmalıdır.

Anahtar Kelimeler: Trigeminal sensoriyel nöropati, Beyin sapı infarktı, Protein S eksikliği

The brain stem contains relatively tight packaging of numerous ascending and descending tracts, as well as cranial nerve nuclei. Thus, even small lesions within this vital structure can produce very significant neurologic deficits. With the widespread usage of magnetic resonance imaging (MRI), brain stem lesions have become easily diagnosed entities. MRI guides the clinician to correlate neurologic findings with anatomic location of lesion. Minor neurologic findings related to brain stem involvement are uncommon. Isolated trigeminal sensory neuropathy secondary to the involvement of brain stem is an extremely rare condition. [1-4]

CASE

A forty nine years old male patient was admitted with numbness of right hemifacial region and oral cavity. The personal and familial medical history was uneventful. Neurologic examination revealed sensation on the right side of the face was decreased to the light touch and pinprick in all distribution of the trigeminal nerve. The corneal reflex was intact bilaterally. Strength and bulk of the masseter and temporalis were symmetrical. Motor deficit or any other abnormal neurological finding was not detected. Cranial MRI revealed ischemic focus at right inferior dorsolateral pontine region (iso-hypointense at T1 weighted, hyperintense at T2 weighted images and FLAIR sequence). There was no contrast enhancement (Fig. 1). Cervical and cranial magnetic resonance angiographies were normal. Doppler ultrasonography of the carotid and vertebral arteries showed no abnormality. A mild increase in cholesterol level was derived from routine blood analyses. Laboratory tests (anticardiolipin antibodies, antinuclear antibody, complement C3 and C4 levels) in terms of vasculitis were normal. Protein S was below normal limit, and it was evaluated as the most important risk factor. Antiplatelet treatment (acetylsalicylic acid 300mg/day) was started and the patient recovered almost completely within a month.

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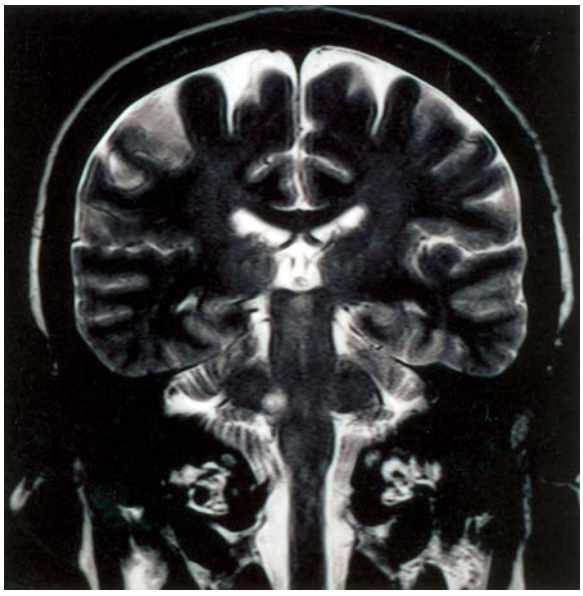
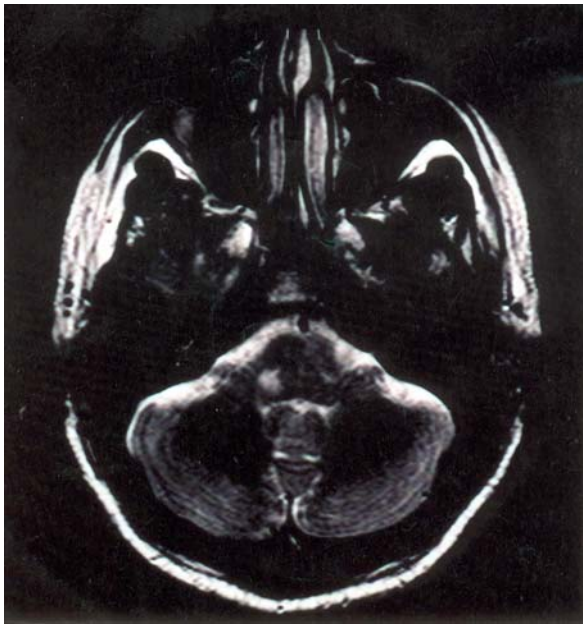
Fig 1. Cranial MRI findings of the patient: a small infarction at right inferior dorsolateral pontine region. Coronal T2 weighted **(A)** and axial FLAIR sequence **(B)** images.

A



B



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DISCUSSION

Trigeminal nerve is a mixed nerve with motor and sensory components. It provides sensory innervation to most of the facial and cranial regions such as mouth, mucous membranes of paranasal sinuses, cornea and conjunctiva. The cell bodies of the sensory part of the nerve are localized in the Gasser ganglion, which is the largest ganglion of the human body. Proximal axons of ganglion constitute the sensory root and pass into mid pons while sending shorter upward and longer downward branches. Branches coursing upwards carry sensory impulses arising from touch and light pressure and form synapses with the axons of the main sensory nucleus. Proprioceptive afferent impulses coming from facial and masseter musculature terminate in mesencephalic nucleus. Nerve fibers carrying nosciceptive impulses extend inferiorly and terminate in spinal nucleus. The axons of the spinal nucleus extend to upper cervical segments. This nucleus divides into three parts from rostral to caudal as pars oralis, pars interpolaris and pars caudalis. The nuclei of trigeminal nerve are in crosswise linkage with thalamus, via trigeminothalamic tract. Peripheral neurons stemming from Gasser ganglion divide into 3 branches. The first (ophthalmic) branch leaves the cranium through superior orbital fissure. The second (maxillary) and the third (mandibular) branches leave the cranium via foramen rotundum and foramen ovale respectively. [5,6]

The findings of our case suggest that the involvement of brain stem should be investigated in a patient with isolated trigeminal sensory neuropathy using MRI. The clinical data point to the involvement of main sensory nucleus and upper segments of the spinal nucleus of trigeminal nerve.

The prognoses of cases with isolated trigeminal sensory neuropathy are usually good.[1-4] Likewise, in our case clinical symptoms are resolved within a month. In our case, protein S deficiency was the only risk factor. Hence, our findings suggest that protein S levels should also be investigated in these cases.
The submission of this case was considered worthwhile in that isolated trigeminal sensory neuropathy secondary to the infarct of the brain stem is a rarely encountered entity.

REFERENCES

1. Ishii K, Tamaoka A, Shoji S. Dorsolateral pontine segmental infarction presenting as isolated trigeminal sensory neuropathy. J Neurol Neurosurg Psychiatry 1998; 65:702.

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2. Pedraza L, Campero M. Trigeminal sensory neuropathy: anatomico-physiological correlation. J Neurol Neurosug Psychiatry 2000; 68:390.
3. Holtzman RN, Zablozki V, Yong WC, Leeds NE. Lateral pontine tegmental hemorrhage presenting as isolated trigeminal sensory neuropathy. Neurology 1987; 37: 704-6.
4. Nakamura K, Yamamoto T, Yamashita M. Small medullary infarction presenting as isolated trigeminal sensory neuropathy. J Neurol Neurosurg Psychiatry 1996; 61:138.
5. Adams RD, Victor M, Ropper AH. Principles of Neurology 7th ed. New York: Mc Graw-Hill, 2001.
6. Hughess RAC. Disease of the fifth cranial nerve. In: Dyck PJ, Thomas PK, Lambert EH, et al (eds): Pheripheral Neuropathy, 3rd ed. Philadelphia, Saunders, 1993: 801-817.

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