ORIGINAL RESEARCH PAPER

Volume-7 | Issue-4 | April-2018 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A CASE OF SENSORY STROKE

General Medicine

Dr Amandeep Singh Kaloti

Professor Medicine, Department of Medicine, PIMS, Jalandhar, Punjab.

ABSTRACT

Sensory stroke is caused in 5-7% of cases of a lacunar infarct. The term lacuna is used to describe a small infarct or a small cavity in the brain tissue that develops after the necrotic tissue of a deep infarct is resorbed. A lacuna is attributed to a arterial insufficiency in the distribution of a penetrating branch of a large cerebral artery. Well defined clinical syndromes with dicrete areas of infarctions are known to occur. Here I report a case of a 60 years old female who presented with tingling sensation and pain in the right half of the body. On investigation and doing CECT brain, she was found to have a lacunar infarct in the left thalamus region. She was started on treatment and was put on teicyclic antidepressants in addition to other treatment, to which she responded.

KEYWORDS

Lacunar infarcts, Sensory stroke, Thalamus.

Introduction:-

Pure sensory stroke occurs in 6-7% of the patients of lacunar infarctions. The most common site is the thalamus posteroventral region. The lesion responsible is microatheroma / lipohyalinosis. The clinical features most commonly associated with sensory stroke are Paresthesiae, numbness, mild sensory loss, thalamic pain. There may not be any sensory loss. It usually responds to tricyclic antidepressants or other neuropathic pain agents. The disease course is usually benign. The symptoms usually subside within a few days or weeks[1].

Case Report:-

This patient 60yrs of age female presented with the chief complaint of tingling sensation and pain in the right half of the body for the past 1 day. There was-

No h/o headache
No h/o any preferential weakness
No h/o any speech difficulty
No h/o ataxia
No h/o any cranial nerve involvement
No h/o any bowel or bladder disturbance
No h/o any involuntary movements
No h/o convulsions
No h/o fall / trauma / loss of conciousness

Patient was a known case of hypertension and had been on irregular treatment. Patient was also a case of dyslipidemia. During the hospital stay, the symptoms of the patient i.e., paresthesiae and pain changed on to predominantly pain in the right half of the body. There were no similar episodes in the past. Nothing significant was present in the family history. The personal history was also normal. Patient had attained menopause at 45 years of age. On examination, the patient was conscious, cooperative and well oriented. Patient was afebrile, pulse rate of 88/minute, regular, respiratory rate of 18/min, thoraco-abdominal in nature. The patient had a blood pressure of 160/100 mmHg in the right arm, in supine position. There was no pallor, icterus, cyanosis, clubbing, lymphadenopathy and edema. JVP was not raised. Arcus senilis, xanthomas and xanthelasmas were present. On CNS examination, it was found that the higher mental functions were normal, speech was normal, cranial nerves were normal, motor system examination was normal. Reflexes were normal except that plantar reflex was absent on the right side. On sensory system examination it was found that on the right side touch, pain, temperature, vibration, joint position sense were diminished. There was no neck stiffness. Other systems were found to normal. The investigations done showed a Hb of 11gm/dl. RFT, LFT, ECG, CXR were all normal. Lipid profile showed a total cholesterol level of 316 mg/dl, triglyceride level of 412 mg/dl, LDL of 220 mg/dl, HDL of 35 mg/dl, VLDL of 130 mg/dl. CECT brain done showed a lacunar infarct in the left thalamus. Blood sugar levels were normal. The patient was started on treatment for CVA and was also given the tricyclic antidepressant Amitriptyline. She responded to the treatment well and the pain subsided.

Discussion:-

The term lacunar is used to describe a small infact or a small cavity in the brain tissue that develops after the necrotic tissue of a deep infact is resorbed. A lacuna is attributed to arterial insufficiency in the distribution of a penetrating branch of a large cerebral artery. Well defined clinical syndromes with the discrete areas of infarction seen in the lacunar infarctions. Thus, the so called "classic lacunar syndrome". The risk factors for lacunar infaction are aging, hypertention, diabetes mellitus, dyslipidemia, smoking. Prevalence in common in blacks & hispanic population. The frequency increases with age. There is no significant sex difference[2].

The causes include-

- Lipohyalinosis nad firinoid necrosis
- Atheromas
- Post aneurysm haemorrhage
- Embolisms
- Atypical causes like polycyhtaemia, vasculitis, infections, connective tissue disorders, CNS lyme disease.

Patho physiology includes that the lacunar infrarctions are small deep infarcts with a maximum diameter of 1.5 cm & a volume of 0.2 to 3.4 cms. These infarctions occur in the vessels having a diameter of 100-400 microm. These lacunar infarcts are located in basal gaglia, putamen, thalamus, pons, centrum semiovale, white matter of internal capsule, cerebellum. Most occur in the

1) Lenticulo-striate branches of MCA
2) Anterior striate & Heubner arteries (ACA)
3) Anterior Choroidal
4) Paramedian branch of Basilar artery
5) Thalmoperforator branch of PCA.

Types of Lacunar Syndromes are :-

1) Pure motor hemiparesis
2) Pure sensory stroke
3) Sensorimotor stroke
4) Ataxic hemiparesis
5) Dysarthria – Clumsy Hand Syndrome

Others :-

- Himichorea – Hemiballism
- Dystonia
- Internuclear Ophthalmoplegia
- Classic Brainstem Syndromes[3].

Conclusion:-

Pure sensory strokes are characterised by subjective symptoms with no evidence of weakness, speech difficulties or other classic symptoms of cerebral infarction or haemorrhage. It may cause central pain. The disease course is usually benign and the symptoms subside within a few days to weeks. But these sensory strokes may easily be ignored.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Karl B Alstadhaug, Jan F Prytz: Pure sensory syndromes and post-stroke pain secondary to bilateral thalamic lacunar infarcts: a case report. J Med Case Resp, Vol 6; 2012, PMC3514397.
2. Wardlaw J M: What causes lacunar stroke? Journal of Neurology, Neurosurgery and Psychiatry, 2005; 76: 617-619.
3. G de Jong, F Kessels, J Lodder: Two types of lacunar infarcts. Stroke, 2002; 33: 2072-2076.