International Journal of Research in Medical Sciences
Ramineni HB et al. Int J Res Med Sci. 2015 Jan;3(1):379-380
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: 10.5455/2320-6012.ijrms20150178

Case Report

Bilateral thalamic infarcts presenting oculomotor nerve palsy: case report

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Received: 11 December 2014
Accepted: 22 December 2014

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ABSTRACT

Bilateral thalamic infarctions are rare; oculomotor nerve palsy can be the result of direct or indirect damage to the oculomotor nerve. We report a case of oculomotor nerve palsy associated with changes in visual pattern and speech disturbances. There is no loss of consciousness. A computed tomography scan of brain showed acute bilateral thalamic infarct. Oculomotor nerve palsies with pupillary involvement warrant thorough investigation and there is no treatment to re-establish function of the weak nerve other than the body's own healing process.

Keywords: Oculomotor nerve palsy, Speech disturbance, Visual disturbances

INTRODUCTION

Bilateral thalamic infarction is uncommon. These paired infarctions have a differential diagnosis that includes metabolic and toxic processes, infection, vascular lesions, and neoplasia.[1] Bilateral thalamic infarction are observed in 0.6% of ischemic stroke patients[2] and have been documented to cause memory deficits reminding of those found after lesions of the medial temporal lobe.[3] In addition, symptoms of cognitive, emotional and behavioral which are of frontal type have been reported after thalamic infarction.[4] Oculomotor nerve palsies are well-recognized ocular motility abnormality. The prevalence of third, fourth, and sixth nerve palsies has been documented in large population studies, which have found a higher proportion of sixth nerve palsies followed by third and then fourth nerve palsies.[5]

CASE REPORT

A 40 year-old woman was admitted to the hospital because of double vision and weakness of upper eyelids and speech disturbance since 10 days. The patient reported the sub-acute onset of right upper lid ptosis, vertical binocular diplopia. On admission her temperature was 37.5°C, blood pressure was 120/80 mmHg and heart rate was 84 beats/min; the remaining findings of the physical examination were within normal limits. The heart sounds were normal, with no murmur, gallop, or rub. The pulmonary examination revealed no jugular venous distension and the breath sounds were equal bilaterally. The electrocardiogram was in normal sinus rhythm. On neurological examination, there was no loss of motor function and the deep tendon reflexes were present. The ocular examination showed bilateral third nerve palsy with non-reactive mydriasis. Brain imaging (computed tomography) showed bilateral thalamic infarction (Figure 1).Considering the history, clinical examination and laboratory findings, and patient was diagnosed as a case of oculomotor nerve palsy. The patient was treated with Ciprofloxacin-eye drops, Monocef IV 1 gm, syrup. Glycerol and oral methylcobalamin. Unfortunately, there is no treatment to reestablish function of the weak nerve other than the body's own healing. The patient was discharged and reviewed again after 2 weeks and 8 weeks. Lid position,

extra-ocular movements and speech were found to be normal.

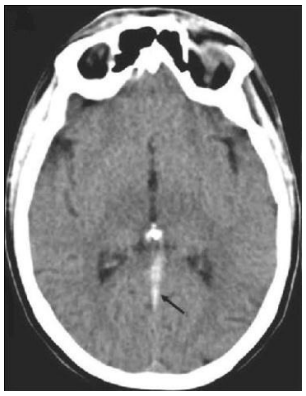

Figure 1: Computed tomography showing bilateral thalamic infarction.

DISCUSSION

We report the case of a patient who presented with bilateral thalamic infarcts with oculomotor nerve palsy. Bilateral thalamic infarcts are rare occurrences, accounting for 22 to 35% of all thalamic infarcts.[6] There are four major thalamic vascular territories, each with a predilection for supplying particular groups of nuclei: the tuberothalamic, the inferolateral, the posterior choroidal and the paramedian vessels. The paramedian arteries arise from a short portion of the posterior cerebral arteries situated between the basilar bifurcation and the junction with the posterior communicating artery.[7] The thalamus contain nuclei that integrate cortical function and serve as pathway of communication across the cerebral cortex and midbrain. Medial and lateral geniculate nuclei are involved with visual and auditory function. The thalamus is also responsible for regulating consciousness; sleep and alertness, when the thalamus is infarcted, patients may have symptoms including vertical gaze palsy, memory impairment, confusion and coma.[8] The patient's presenting symptoms include abnormal eye movements, cognitive and behavioral impairment. Abnormal eye movements result from infarction in the midbrain nucleus which is usually associated when the decrease in the level of consciousness resolves, cognitive and behavioral changes become more apparent: disorientation. Behavioral disturbances are explained by discontinuance of the thalamocortical fibres, especially when the dorsomedian nucleus, which massively projects onto the frontal cortex, is involved.[9] The treatment options are use of Ciprofloxacin-eye drops, Monocef IV 1 gm, Syp. Glycerol and oral methylcobalamin. The patient responded well with subsidizing of diplopia, drooping of lids and speech disturbance.

CONCLUSION

Patients with artery of bilateral thalamic infarcts with oculomotor nerve palsy may present with unusual clinical findings that can confound the diagnosis and potentially delay treatment. Careful evaluation of the patient's history, clinical presentation together with imaging findings facilitates in making the correct diagnosis.

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

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DOI: 10.5455/2320-6012.ijrms20150178
Cite this article as: Ramineni HB, Mukiri S, Jammula P, Suryadevara V. Bilateral thalamic infarcts presenting oculomotor nerve palsy: case report. Int J Res Med Sci 2015;3:379-80.