Case Report

DOI 10.3349/ymj.2010.51.6.978
pISSN: 0513-5796, eISSN: 1976-2437

Yonsei Med J 51(6):978-979, 2010

YMJ

Claude's Syndrome Associated with Neurocysticercosis

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Received: December 6, 2008
Revised: February 27, 2009
Accepted: March 15, 2009
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· The authors have no financial conflicts of interest.

Claude's syndrome is a distinctive brainstem syndrome characterized by ipsilateral third cranial nerve palsy with contralateral hemiataxia and is due to an intrinsic or extrinsic lesion in the midbrain. We report a case of Claude's syndrome caused by neurocysticercosis infection. A 68 year-old Asian man was admitted to our hospital because of ataxia, left ptosis, and diplopia. Brain magnetic resonance imaging (MRI) showed a cystic lesion in the midbrain, which was surrounded by ring enhancement and peripheral edema. Neurocysticercosis infection was diagnosed by the cerebral spinal fluid study. The patient was treated with albendazole and steroids. A follow-up brain MRI three months later demonstrated the disappearance of a surrounding brain edema and rim enhancement. The most common cause of Claude's syndrome is cerebrovascular disease and malignancy. However, there is no report caused by neurocysticercosis infection. Therefore, if we encounter Claude's syndrome, we should consider neurocysticercosis infection as one of the etiologic factors.

Key Words: Neurocysticercosis, Claude's syndrome, ataxia

INTRODUCTION

There are various clinical manifestations of neurocysticercosis (NCC) that depend on not only the topography, number, and size of the lesions, but also the status of the host's immune response to the parasite infection.[1] Only a few reports have described third cranial nerve palsy in patients with NCC involving, especially, the midbrain.[2-4] Meanwhile, Claude's syndrome is a distinctive brainstem syndrome characterized by ipsilateral third cranial nerve palsy with contralateral hemiataxia and is due to an intrinsic or extrinsic lesion in the midbrain.[5] We report a case of Claude's syndrome caused by NCC infection.

CASE REPORT

A 68 year-old man was admitted to our hospital because of ataxia, left ptosis, and diplopia started two days prior. His previous medical history was unremarkable. He did not travel overseas before this event. The left eye pupil was 1.5 mm larger than the right side with impaired pupillary light reflex. Extraocular movements were normal in the right eye. In contrast, there was limited movement (adduction, eleva-

tion, depression) in the left eye and fourth cranial nerve function was considered normal in the left eye because intorsion of the left eye was preserved. The gait was ataxic and dysdiadochokinesia was present on examination of the right arm.

Brain magnetic resonance imaging (MRI) showed a cystic lesion in the midbrain, which was surrounded by ring enhancement and peripheral edema (Fig. 1A and B). Enzyme-linked immunosorbent assay for an anticysticercal antibody in serum was negative, but it was positive in cerebral spinal fluid of 0.38 (normal range < 0.18). A lumbar puncture yielded clear cerebral spinal fluid under normal opening pressure, and cytochemical analysis showed 3 mononuclear cells per mm^3, 90 mg/dL proteins, and normal glucose contents. Cerebral spinal fluid cytospin test for malignancy was negative.

The patient was treated with oral albendazole 15 mg/kg/day in two divided doses for two weeks, and intravenous methylprednisolone 1g/day for six days. Paresis of the third cranial nerve and ataxia resolved completely within one week of treatment. A follow-up brain MRI three months later demonstrated the disappearance of surrounding brain edema and ring enhancement (Fig. 1C and D).

DISCUSSION

NCC is a common parasite affecting the central nervous system. Brain parenchyma involvement is the most common and usually presents with seizures. Other clinical manifestations may occur, depending upon the localization and viability of the parasite. There have been several reports that NCC occurred within the intrinsic midbrain, manifested by isolated third cranial nerve palsy with poor prognosis,[2] isolated bilateral ptosis,[4] accompanying hydrocephalus,[6] and recurrent third cranial nerve palsy.[7,8] However, none of these patients presented with ipsilateral third cranial nerve palsy accompanying contralateral hemiataxia, that is, Claude's syndrome. The Claude described a house painter who developed right third cranial nerve palsy with contralateral gait ataxia.[9] The pathological examination revealed a paramedian mesencephalic infarction on the right involving the superior cerebellar peduncles, the medial half of the red nucleus, and some reports describe the syndrome with Claude's original red nucleus involvement.[5,10] In this case, the lesion of NCC involved the left superior cerebellar peduncle but not the red nucleus. This finding supports the report that suggests the lesion of the superior cerebellar peduncle just below and medial to the red nucleus can be a cause of Claude's syndrome.[11] The most common cause of Claude's syndrome is cerebrovascular disease and malignancy.[11] There is no report caused by NCC infection. Therefore, we should consider NCC infection as an etiologic factor if we encounter Claude's syndrome.

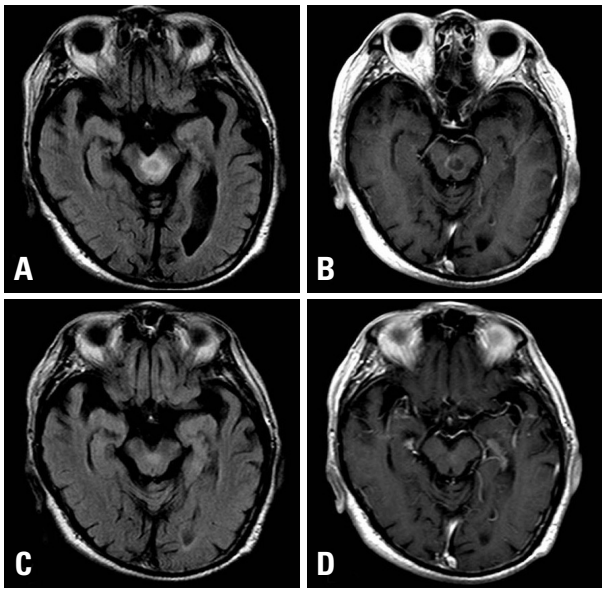

Fig. 1. Brain MRI. (A) Initial FLAIR image shows high signal intensity on left midbrain and perilesional edema. (B) Initial contrast enhanced T1-weighted image shows cystic lesion with peripheral ring enhancement. The lesion involves dorsal midbrain tegmentum, left third cranial nerve fascicle, and superior cerebellar peduncle, but not the red nucleus. The lesion is located posterior and inferior to the red nucleus. (C and D) Follow up MRI after three months, reveals disappearance of perilesional edema and ring enhancement.

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