CASE REPORT

HORIZONTAL CONJUGATE GAZE PALSY IN EOSINOPHILIC MENINGITIS

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Abstract. Two cases of eosinophilic meningitis who presented with headache and strabismus are reported. Pertinent physical examination revealed unilateral horizontal conjugate gaze palsy with absence of doll's eye maneuver and hemiparesis. The etiologic agent of eosinophilic meningitis is presumed to be *Angiostrongylus cantonensis* and the infected location that produce horizontal conjugate gaze palsy was a pontine lesion.

Unilateral horizontal conjugate gaze palsy is usually due to contralateral frontal or ipsilateral pontine damage. The most common etiologic pathology is stroke. To our knowledge, it has never been reported in eosinophilic meningitis. This report demonstrates two cases of eosinophilic meningitis with unilateral horizontal conjugate gaze palsy.

Case 1: A 31-year-old male farmer was admitted to Srinagarind Hospital, Khon Kaen, Thailand in November 1995 because of headache and strabismus. He had been in good health in the past. One week previously, he experienced severe bilateral throbbing headache. Three days before admission, he found that his eyes looked toward the left side. He gave a long history of having eaten raw Pila snails and had eaten them 2 weeks before this illness.

On physical examination, he was asthenic and alert man with body temperature of 37.0°C. Pertinent examination revealed a deviation of both eyes toward the left side which could not move to the opposite site (Fig 1). Neck stiffness, doll's eye maneuver and motor weakness were not detected. The rest of the general and neurological examination, including vertical gaze, were normal.

Complete blood count revealed hematocrit of 40%, white blood cell 9,100 cells/mm^3 wiht 60% polymorhonuclear cells, 39% lymphocytes and 1% eosinophils. CT scan of the brain was within normal limits. Cerebrospinal fluid (CSF) analysis showed an opening pressure of 100 mm of water, protein of 34 mg/dl, glucose of 51 mg/dl, white blood cells 46 cells/mm^3 with 9% polymorphonuclear cells, 43% lymphocytes and 48% eosinophils. Gram stain, India ink preparation, cryptococcal antigen and culture were negative.

The patient was treated with paracetamol. Headache disappeared within 2 days. Two weeks later, the left eye could adduct but the right eye still had lateral rectus muscle palsy (Fig 2).

Case 2: A 49-year-old, previously healthy woman was admitted to Srinagarind Hospital in March 1998 with the chief complaint of severe headache and strabismus for 6 days. She gave a long history of having eaten raw Pila snails and had eaten them 2 weeks before this illness.

Physical examination revealed a distressed woman with normal body temperature. She had a normal physical and neurological examination, except a deviation of both eyes toward the right side. She could not move the eyes to the left side and doll's eye maneuver was absent.

Complete blood count showed hematocrit 37%, white blood cell 13,600 cells/mm^3 with 63% PMNs, 26% lymphocytes, 6% monocytes, 4% eosinophils and 1% basophils. MRI of the brain was within normal limits. CSF analysis revealed a normal opening pressure, white blood cell of 490 cells/mm^3 with 90% lymphocytes and 10% eosinophils, protein of 56 mg/dl and glucose of 37 mg/dl (blood glucose 72 mg/dl). Stains of the CSF for bacteria, India ink and acid fast bacilli, cryptococcal antigen and culture were all negative.

The patient was treated with prednisolone 60 mg/d for 2 weeks. Headache was disappeared within 5 days. On follow-up 2 weeks later, both eyes could partially move to the left side.

The differentiation of frontal lesion from pontine lesion that produces horizontal gaze galsy is that with a frontal lesion, the patient generally has a hemiparesis and the eyes look away from the hemiparesis side but can be brought to the other

Fig 1–Both eyes looked toward the left side in primary gaze.

Fig 2–Two weeks later, the left eye could adduct but the right eye had lateral rectus muscle palsy.

side with an oculocephalic maneuver. In contrast, in a pontine lesion affecting the abducens nucleus and/or the paramedian pontine reticular formation (PPRF), the eyes look toward the hemiparesis side (although hemiparesis is an inconstant finding) and there is loss of oculocephalic maneuver (Masdeu and Brazis, 1990).

The major causative agent of eosinophilic meningitis in Thailand is the rat lung worm, *Angiostrongylus cantonensis* (Punyagupta *et al,* 1970). Infective third-stage larvae develop in slugs and snails. Humans are infected primarily in the central nervous system by this parasite after ingestion of an infected intermediate host. The pathologic abnormalities are found in the brain, including brainstem, and spinal cord (Sonakul, 1978). The pathologic mechanisms are directed invasion of motile worms, inflammatory responses to foreign bodies and possible toxicity of worm excretory products (Koo *et al,* 1988). The common presenting symptoms of eosinophilic meningitis are severe headache, neck stiffness, nausea and vomiting (Punyagupta *et al,* 1975; Yii, 1976; Kuberski and Wallace, 1979; Schmutzhard *et al,* 1988). About 5% of patients have eye muscle paralysis and usually present with unilateral or bilateral lateral rectus muscle palsy.

Eosinophilic meningitis in these two cases was presumptively caused by *Angiostrongylus cantonensis* and the location of horizontal gaze palsy is pontine lesion. From our patients, eosinophilic meningitis must be considered as one of the etiologic possibilities of horizontal gaze palsy.

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