Cryptococcal Meningoencephalitis Presenting as Cerebellitis
- A case report -

T.G. Lee, S.H. Park¹, H.B. Lee¹, B.W. Yoon, K.H. Chang², J.K. Roh.

Department of Neurology and Radiology², Seoul National University College of Medicine
Department of Neurology, Boramae City Hospital¹

= Abstract = We herein report an elderly patient with cryptococcosis in the nervous system who presented with cerebellar dysfunction and showed cerebellar parenchymal lesion with adjacent focal meningeal enhancement on the brain CT and the serial brain MRIs. The cerebellar lesion suggests cryptococcal inflammatory reaction or a cerebellar infarction from the arteritis of a branch of the left superior cerebellar artery. This clinical and neuroimaging finding of cerebellitis has not been reported in cryptococcosis. We think cryptococcosis would be included in the differential diagnosis of cerebellitis.

Key words: brain MRI, cerebellitis, cryptococcosis

INTRODUCTION

Cryptococcal infection is the most common fungal infection of the central nervous system both in AIDS patients (Dismukes, 1988) and the general population. Early diagnosis is important because it is potentially treatable with antifungal therapy. The laboratory diagnosis usually depends on the sensitive and specific cryptococcal antigen determination in the cerebrospinal fluid (CSF) and serum. However, in some patients with early meningitis, the cryptococcal antigen is negative in the CSF (Harrison and McAllister, 1991). In addition, computerized tomography (CT) of the brain is usually normal or shows nonspecific abnormalities of contrast-enhanced mass lesions (Garcia et al., 1985; Fujita et al., 1981), ring-enhancing lesions (Zuger et al., 1986), hydrocephalus or brain edema (Tijia et al., 1985). Since 1988 (Jarvik et al.), there have been several MRI reports on cryptococcosis in the nervous system with limited number of the patients.

Cerebellitis has been reported rarely in the viral (Silverstein et al., 1972; Goldstein et al., 1963), rickettsial (Silipapajacul et al., 1991), and salmonella (Misra et al., 1985) infections, but not in the cryptococcosis. We report an elderly non-AIDS patient with CNS cryptococcosis, whose serial magnetic resonance imagings (MRIs) revealed cerebellitis with adjacent meningeal enhancement on the cerebellum.
CASE DESCRIPTION

A 67-year-old farmer with headache, vomiting and clumsiness of the hands was admitted to Borame City Hospital in July, 1994. He had enjoyed his good health until one month prior to the admission, when he developed headache insidiously. His headache continued day after day, and was followed by nausea and vomiting which progressively worsened thereafter. However, he did not complain of febrile sensation or nuchal rigidity.

On admission, physical examination was unremarkable. His mental status was normal. Neurological examination revealed mild nuchal rigidity as well as bilateral cerebellar dysfunction, ataxic gait, scanning slurred speech, terminal dysmetria, intention tremor, clumsiness of the hands without weakness. The brain MRI at 2.0 Tesla showed predominantly unilateral high signal lesions in the cerebellum on the T2-weighted images (Fig. 1-A), and focal meningeal enhancement on the adjacent cerebellum on Gd-DTPA enhancement (Fig. 1-B). This cerebellar parenchymal lesion on the T2-weighted images did not show enhancement on the Gd-DTPA-enhanced T1-weighted images. This lesion suggested a cerebellar infarction in the vascular territory of the left superior cerebellar branch. The sagittal T1-weighted images disclosed cerebellar swelling with the fourth ventricular collapse (Fig. 1-B). There were enhancing high signals in the bilateral basal ganglia (Fig. 1-C). Brain CT before the antifungal therapy also revealed meningeal enhancement around the cerebellum, as well as slight low density lesion in the cerebellar parenchyme, which was smaller than that of the brain MRI.

CSF studies revealed opening pressure of upper normal range (16 cm H2O), lymphocytic pleocytosis (white blood cells, 600/mm³; lymphocyte, 94%), increased protein level (101 mg/dl), normal glucose level (92 mg/dl) about 60% of the serum level, all negative results for malignant cells, carcinoembryonic antigen, lactate dehydrogenase, bacterial smear and culture, acid-fast bacilli and fungal smears, viral (Epstein Bar, Varicella Zoster, adn Herpes virus), rickettsial, and Mycoplasma pneumoniae antibodies; Widal test, VDRL, and negative ELISA for the cysticercosis, sparganosis and paragonimiasis which are endemic in the country. Cryptococcal and bacterial antigen determination were negative on the first CSF study. Leukocytosis and increased sedimentation rate were persistent on the repeated examinations. Rheumatoid factor, FTA-ABS, anti-HIV, anti-phospholipid, and anti-nuclear antibodies were negative in the serum. Cerebral angiography and chest X-ray findings were normal.

To control cerebellar swelling, mannitol and dexamethasone (4 mg per 6 hours) were administered intravenously. After two weeks, prior to the antifungal therapy, follow-up MRI of the brain showed markedly decreased size of the cerebellar parenchymal lesion with decreased meningeal enhancement and cerebellar swelling (Fig. 1). During the admission, progressive was neck stiffness and intermittent fever with chills. His mental status progressively deteriorated to fluctuating confusion after a brief period of clinical improvement due to the effect of dexamethasone and mannitol.

Repeated CSF examinations demonstrated decreased glucose level, high titer of the cryptococcal antigen (positive up to 1:10,000 dilution), positive India-ink preparation and the growth of cryptococci on the Saboraud agar. Combined antifungal therapy with amphotericin-B and 5-fluorocytosine for three months resulted in only modest improvement of the clinical course and gradual decrease in the antibody titers of the CSF.

DISCUSSION

Very unusual is cerebellar dysfunction as the initial clinical manifestation of CNS cryptococcosis in our patient. In addition, the first brain MRI (Fig. 1) revealed asymmetric cerebellar high signal lesions, localized meningeal enhancement and the cerebellar swelling. These decreased on the follow-up MRI (Fig. 2) in response to the administration of mannitol and corticosteroid. However, the clinical effect of the administration was transient for about a week. We think these clinical and MRI findings are compatible with cerebellitis.

We could exclude other concurrent infection.
Fig. 1. (Initial MRI)
1-A: Asymmetric high signal lesion in the cerebellum on T-weighted image.
1-B: Meningeal enhancement around the cerebellum and the collapse of the fourth ventricle due to cerebellar swelling on T-weighted image.
1-C: Bilateral enhancing high signal lesions in the basal ganglia on T-weighted image.

Fig. 2. (Follow-up MRI). Remarkably decreased meningeal enhancement and cerebellar swelling on T-weighted image.
connective tissue disorders, and the brain tumor on the basis of the clinical feature and course, intensive laboratory evaluation, and the neuroimaging studies. It is unclear whether the cerebellar lesion is due to cryptococcal inflammation or vasculitis of a branch of the left superior cerebellar artery. Cerebral angiography of the vertebro-basilar system was normal. The insidious onset of progressive cerebellar dysfunction as well as normal angiography may imply inflammatory origin rather than vasculitis.

In the majority of autopsy cases of cryptococcal meningoencephalitis, involvement of the nervous system presents as a meningoencephalitis, usually localized in the cerebral and cerebellar hemispheres. Changes in the brain are usually in proximity to the meningeal reaction, but they may extend deep into the parenchyma as in our patient (Francesco Scaravilli, 1992).

We think cryptococcosis would be included in the differential diagnosis of the cerebellitis evolving subacutely or chronically. Although probably unusual, this finding may help provide early diagnosis and treatment especially when other laboratory findings are not conclusive in early phase, and consequently the prognosis could be better than that of delayed treatment as in our case.

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