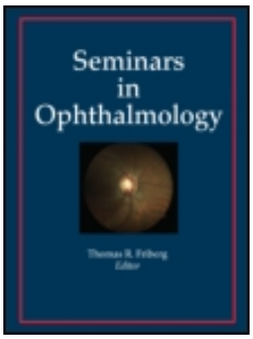

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CASE REPORT

Chronic Meningitis Complicating Intracranial Hypertension in Neurobrucellosis: A Case Report

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ABSTRACT

In neurobrucellosis, even though meningitis is encountered frequently, chronic intracranial hypertension is a rare manifestation. Early diagnosis and treatment is very important for the prevention of permanent visual loss secondary to poststasis optic atrophy in these cases. We report a case that presented with permanent visual loss secondary to intracranial hypertension in neurobrucellosis. Our goal is to draw attention to the consideration of neurobrucellosis in cases with papilla stasis, even in the absence of neurological findings in endemic areas.

Keywords: Chronic meningitis, intracranial hypertension, neurobrucellosis

INTRODUCTION

Brucellosis is a zoonozis caused by a gram-negative bacilli of the genus brucella. It is an important health problem and still endemic in Mediterranean and Middle Eastern countries. Brucellosis is a multisystem disease characterized by fever, frequent bone and joint diseases such as arthritis, sacroiliitis, spondylitis, and osteomyelitis, or respiratory, gastrointestinal, or cardiac involvement. Although central nervous system involvement is rare, the incidence is reported as 1.7–10% in different studies.[1,2] During the course of neurobrucellosis, meningitis and meningoencephalitis are encountered frequently; however, chronic intracranial hypertension is a rare manifestation.[1,2] We report an unusual case of neurobrucellosis admitting with intracranial hypertension secondary to chronic meningitis.

CASE REPORT

A 27-year-old female patient consulted our clinic complaining of blurred vision, headache, and diplopia. She had been admitted to the hospital with nausea, vomiting, headache, and blurred vision four weeks earlier. The pupillary response to light was poor bilaterally. She had left esotropia in the primary position with limited bilateral abduction. Her visual acuity was counting fingers at 10 cm in right eye and 30 cm in left eye. A color vision test and visual field test could not be performed because of the low vision. Bilateral papilla stasis was found with fundus examination. There was no neck stiffness, Kernig's sign, or other neurological deficits. Cranial magnetic resonance imaging (MRI) with Gadolinium was normal. Total blood count, liver and renal function tests were unremarkable. Erythrocyte sedimantation rate was

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6 mm/h. The thyroid hormone levels and blood chemistry were within normal range.

First lumbar puncture (LP) yielded clear cerebrospinal fluid (CSF) with increased opening pressure (850 mmH2O), lymphocytic pleocytosis (85/mm^3 lymphocyte), and elevated protein levels (178 mg/dl, normal 15–45 mg/dl). While CSF glucose level was 33 mg/dl, blood glucose level was 100 mg/dl. Blood and CSF cultures were negative. Brucellosis was diagnosed by Wright agglutination test with titer of 1:640 in blood and 1/80 in CSF. Combined treatment with rifampin (600 mg × 1/d), doxycyclin (100 mg × 2/d), and ceftriaxone (1gr × 2/d) was administered, and acetozolamid (250 mg × 4/d) was added for intracranial hypertension.

The patient was referred to a neurosurgen for the ventriculoperitoneal shunt. However, the procedure could not be performed due to high protein and cell count in CSF. CSF pressure did not decrease with the medical treatment and there was not enough improvement in vision; optic nerve sheath fenestration (ONSF) was suggested to the patient, but she did not accept this procedure. Hence, normal CSF pressure was maintained with the evacuative LPs. Approximately six evacuative LPs were done every five days during the hospitalization, which lasted about two months. The patient's clinical condition gradually improved. Intracranial hypertension and papilla stasis returned to normal within six weeks. She was discharged with rifampin, doxycyclin, and cotrimoxazole.

Three months later, she had orthotropia without abduction deficit and she was counting fingers at 2 meters bilaterally. Fundoscopic examination showed poststasis optic atrophy bilaterally.

DISCUSSION

Neurobrucellosis presents as meningoencephalitis in the acute form and polyradiculopathy, myelitis, meningitis, cerebellar syndrome, or cranial nerve involvement in the chronic form. CNS involvement can be due to direct effects of bacilli, cytokines, or endotoxins.[2] Brucella organisms are capable of prolonged intracellular survival in the phagocytes. When the host immunity decreases, the organisms proliferate and lead to various clinical manifestations. Brucella melitensis can be found in CSF or other tissue fluids and results in chronic meningitis. In the present case, the brucella organism could not be isolated from blood and CSF cultures, so the diagnosis was made by serological tests.

Neurobrucellosis has been categorized as acute meningoencephalitis, chronic meningitis with increased intracranial pressure, meningovascular involvement, CNS demyelinization and peripheral neuropathy, according to the clinical presentation.[3] Chronic meningitis with increased intracranial pressure has been rarely encountered as an initial manifestation of neurobrucellosis.[4,5] Papilledema, headache, nausea, vomiting, and blurred vision are the main presenting features of this condition. Fever, meningeal irritation, and focal neurologic deficit are not seen in these patients.[3] In addition, inflammatory changes or white matter and vascular patologies may be demonstrated with cranial MRI.[6,7] Similarly, in the present case, intracranial hypertension associated with chronic meningitis was recognized as an initial manifestation. Likewise, headache, nausea, vomiting, and blurred vision were the main presenting clinical symptoms without fever, meningeal irritation, and focal neurologic deficit.

Persistent visual loss rarely can occur due to optic atrophy secondary to intracranial hypertension in neurobrucellosis.[3] Although it has been reported in more than 50% of cases, usually complete resolution without vision loss occurs after appropriate antibiotic therapy.[8] Mclean et al. reported complete recovery after antibiotherapy in neurobrucellosis cases, except in one case where permanent visual loss was caused by optic atrophy secondary to papilledema.[8] Lack of recovery of severe visual impairment was considered to be due to late admission to the hospital and diagnosis of neurobrucellosis in the present case.

Surgical treatment must be considered in intracranial hypertension if visual function deteriorates despite medical therapy. Surgical indications are deterioration of visual field defects, visual acuities below 0.5, and intractable headache resistant to medical treatment. A lumboperitoneal shunt can be performed if headache is the prominent sign and ONSF should be chosen if visual field defects deteriorate in clinical practice.[9] ONSF can be suggested even for pseudotumor cerebri with progressive visual loss due to chronic atrophic papilledema.[10] In our case, ONSF was offered but she did not accept surgical treatment. Since a ventriculoperitoneal shunt could not be applied due to high protein and cell count in CSF, evacuate LPs were made in order to maintain CSF pressure in the normal range.

In conclusion, in endemic areas of brucellosis, the possibility of neurobrucellosis must be considered in patients with papilla stasis, even without focal neurological deficits or other common symptoms. Neurobrucellosis may cause permanent visual loss owing to increased intracranial pressure and poststasis optic atrophy. In order to prevent these complications, contemplating neurobrucellosis is crucial for early diagnosis and treatment.

DECLARATION OF INTEREST

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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