Neurobrucellosis case presenting with headache and diplopia: An endemic problem

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BACKGORUND AND AIM

Neurologic involvement occurs approximately in 10 percent of brucellosis cases. Manifestations include meningitis (acute or chronic), encephalitis, brain abscess, myelitis, radiculitis, and/or neuritis. We would like to emphasize crucial points that physicians must be familiar with during neurobrucellosis diagnosis.

CASE

A 30-year-old female patient applied to the emergency department with a headache that had lasted for a month and increased for the last three days. The headache was unilateral and localized to the vertex. The headache was constant and throbbing. The pain was severe and accompanied by nausea. She had a buzzing in her ear. There was no accompanying autonomic finding. Analgesics response for headache was limited. The headache had been accompanied by a double vision for the last two days. The patient's vital signs were stable, and the fever was 36.5. Her neurological examination revealed esotropia and papilledema in the left eye. Another neurological examination was normal. There was no neck stiffness. In laboratory examination, there was any feature except for iron lack and erythrocyturia. Brain MRI showed that the optic nerve sheaths were dilated and had dural enhancement. The color of CSF (Cerebrospinal fluid) was clear and odorless. CSF pressure was measured at 45 cmH2O. Cell count was counted normally. CSF glucose was 28.1 mg/dL (low), and protein was normal. There was the growth of Brucella spp. in CSF culture. Rose bengal test was positive, and brucella antibody titer was 1/5120. There was no growth in the blood culture. She was diagnosed with neurobrucellosis. Ceftriaxone 2x2000 mg, rifampicin 1x600 mg, doxycycline 2x100 mg, acetazolamide 2x1000 mg were started. After 3 months, the patient's clinic improved, and her treatment was completed. Other complications related to brucellosis were not observed.

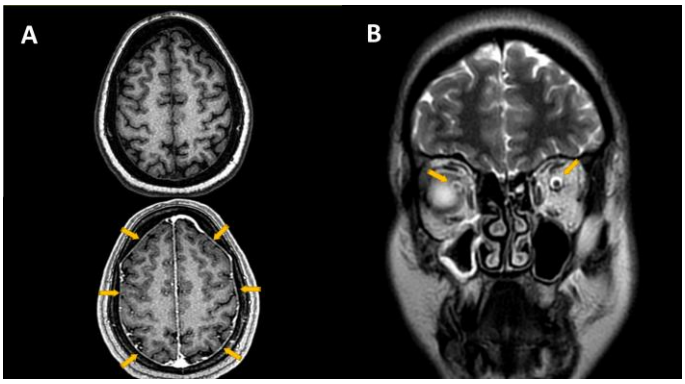

Figure 1: A; *Axial section of T1 weighted of brain MRI shows dural enhancement,* ***B;*** *Coronal section of T2 weighted of brain MRI shows the optic nerve sheaths are dilated.*

CONCLUSION

Brucellosis typically presents with insidious onset of fever, malaise, night sweats, and arthralgias. The fever pattern is variable. Additional symptoms may include weight loss, arthralgia, low back pain, headache, dizziness, anorexia, dyspepsia, abdominal pain, cough, and depression. Brucellosis can affect any organ system, such as neurologic, cardiovascular, pulmonary, ocular, and dermatologic. Ocular involvement is rare. The absence of fever and nuchal rigidity, normal infection parameters, and CSF cell count were the confounding factors in our patient. However, progressive headache with severe and acute onset and accompanying neurological symptoms should alert the physician regarding secondary headache. Dural enhancement and the dilated optic nerve sheaths in neuroimaging; indicate meningeal involvement and increased intracranial pressure. Also, the fundoscopic examination can detect papilledema and may support increased intracranial pressure diagnosis. In such cases, CSF examination is needed for a differential diagnosis. Pleocytosis, mild to moderately elevated protein levels, and hypoglycorrhachia in CSF examination must warn physicians of bacterial pathogens. Considering endemic infectious pathogens, additional examinations may guide the physician. For example, adenosine deaminase level may be a useful adjunctive test for diagnosing central nervous system brucellosis. Early diagnosis and treatment significantly prevent some dangerous complications of brucellosis, such as encephalitis and brain abscess.