Case Report

Pseudotumor—like Presentation of Neurobrucellosis

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Abstract

Brucellosis is bacterial zoonoses. In endemic areas brucellosis can present with clinical features of nearly any neurological illness. Meningitic presentation is most common, with patient presenting with either acute or chronic meningitis. Pseudotumor—like presentation is also documented and accounts for only 4% of cases of neurobrucellosis. Here we are documenting a case of neurobrucellosis with presentation similar to pseudotumor cerebri but with abnormal CSF. This highlights the fact that being a potentially treatable condition brucellosis should always come in the differential diagnosis of neurotuberculosis especially if there are atypical features e.g. pseudotumor presentation with abnormal CSF. ©

INTRODUCTION

We are documenting a case of neurobrucellosis with presentation similar to idiopathic intracranial hypertension but with abnormal CSF. This is also to highlight certain features that are of relevance to physicians in tropical and developing countries.

CASE HISTORY

A 30-year-old well-educated, affluent lady living in a major metropolitan city had continuous headache with early morning exacerbations and vomiting of one-month duration. She had no history of fever, altered sensorium or seizures. She had been with her family for a boat trip across Singapore, Thailand and Malaysia for 3 weeks prior to the onset of this illness. She was on treatment for hypothyroidism for the past 3 years. Examination revealed bilateral papilloedema, with no other focal neuro-deficit, and no signs of meningeal irritation. With evidence of raised intracranial pressure and no focal neurodeficit, a possibility of benign intracranial hypertension was considered, especially in view of the association with hypothyroidism. This possibility became further stronger when investigations revealed normal counts, erythrocyte sedimentation rate (ESR) of 15 mm in 1st hour, a normal magnetic resonance imaging (MRI) brain and venogram, with usual perimetry analysis showing enlargement of bilateral blind spots and a left inferior nasal scotoma. Visual acuity was normal. Accordingly a cerebrospinal fluid (CSF) tap and manometry was done. While the pressure was found to be 370 mm of CSF and revealed cells (all lymphocytes) 32 to 35 /mm³, protein 60 mg%, sugar 38 mg% (corresponding blood sugar 61 mg%). This led us to look for other diagnosis. Staining, culture and CSF PCR for M. tuberculosis were negative, Lyme's serology was negative, C-reactive protein and antinuclear antibodies were also negative. Serology for brucella revealed IgM titers of 130 and IgG of 295 U/l. Treatment was started with doxycycline, trimethoprim-sulfamethaxazole, and rifampicin on follow-up at four weeks. On follow up after four weeks, the papilloedema resolved and the repeat CSF study was normal along with normal CSF manometry.

DISCUSSION

Brucellosis is a bacterial zoonosis, caused by four pathogenetically important species. Prevalence studies in animals in India reveal 6.5% infection rates in sheep and goats (Haryana, 1968). Cows and buffaloes in rural areas were found to be free from infection, while those in organized farms were found infected1. The most important vehicle for transmission is raw milk, rather than occupational exposure. Milk products especially soft cheese, ice cream, raw meat, bone marrow from infected animals, and raw vegetables, or water supplies contaminated by feces or urine of infected animals may also be responsible. Aerosol transmission is described in endemic areas having animals.2

The clinical presentation of neurobrucellosis is diverse and can imitate any neurological illness. However it usually lies in one of the following categories—meningoencephalitis, meningomyelitis, papilloedema without localizing features (pseudotumor—like presentation), meningo cavernous presentation, CNS demyelination, peripheral neuritis, compressive myelopathy and radiculopathy secondary to brucella spondylitis. Meningitic presentation is most common, with patient presenting with either acute or chronic (> 4
weeks duration) meningitis. This is because meninges appear to be the site of initial neurological involvement. Pseudotumor presentation is also documented and accounts for only 4% of cases of neurobrucellosis. Hence brucella should come high on the list of differentials being a potentially treatable condition. CSF is abnormal in almost all patients with neurobrucellosis with lymphocytic pleocytosis of varying degree. CSF and serum antibodies to brucella antigen can be detected in several ways. Culture of the organism is most definitive but positive in only 30% at best.

In view of high rates of development of resistance, monotherapy has been abandoned. The antibiotics of use are the tetracyclines (doxycycline 100 mg twice daily), aminoglycosides dosage (streptomycin / amikacin / gentamicin), rifampicin (450 to 900 mg once daily) and trimethoprim-sulfamethoxazole (TMP-SMX: two tablets every twelve hours). For most patients the most practical and safe combination is triple drug therapy of doxycycline, rifampicin, and TMP-SMX for 8 to 12 weeks. Children under the age of eight and pregnant ladies are better kept on TMP-SMX with rifampicin for 12 weeks.

To summarize, the four points highlighted that are of relevance to physicians are: (1) brucellosis is not necessarily a rural disease; (2) raw milk and milk products rather than occupational exposure, is the most common mode of transmission; (3) being a potentially treatable condition, brucellosis should always come in the differential of neurotuberculosis especially if there are atypical features e.g. pseudotumor presentation with abnormal CSF; (4) polytherapy is the rule for period of eight to twelve weeks.

**References**