Is Neurobrucellosis the Pandora’s Box of Modern Medicine?

To the Editor—The recent article by Guven et al [1], titled “Neurobrucellosis: Clinical and Diagnostic Features,” sheds light on many important yet vexing issues in the clinical presentation, diagnosis, and management of a relatively common zoonotic infection. The relevance of discussing this issue is understated by the lack of consensus in the diagnosis and management of neurobrucellosis, in combination with unchanged diagnostic and therapeutic approaches for brucellosis being practiced for decades. We would like to express our views regarding certain aspects in the diagnosis and management of neurobrucellosis.

The widely followed diagnostic criteria overemphasizes the role of abnormal cerebrospinal fluid (CSF) (positive Brucella agglutination titer in any titer, isolation of Brucella from CSF, and abnormal CSF parameters) in the diagnosis of neurobrucellosis [2]. However, in resource-poor settings such as ours, facilities for determination of CSF Brucella agglutination titers are sparse. Due to the long
time period required and low yield of isolation of the fastidious organism from CSF, diagnostic utility of CSF analysis in making a timely diagnosis is low [3]. Because neurobrucellosis is an endemic problem in our country [4], we strongly advocate the proposal for considering the diagnosis of neurobrucellosis in individuals with consistent clinical features even in the absence of positive CSF Brucella agglutination results. One of the most important differentials for neurobrucellosis in our settings is tuberculosis. Clinical features and neuroimaging findings in brucellosis and tuberculosis may be so similar that a definitive diagnosis is made only after pathological analysis. However, among the clinical features found to be significantly associated with neurobrucellosis by Guven et al [1] (headache, blurred vision, loss of vision, hearing loss, and confusion), hearing loss due to vestibulocochlear nerve involvement seems to be unique for brucellosis [5]. Hence, electrophysiological studies (brainstem auditory-evoked potentials) should be performed more commonly to detect subclinical vestibulocochlear nerve involvement, as it may assist in pointing toward the diagnosis of neurobrucellosis. In addition to the neuroimaging findings described by Guven et al [1], neurobrucellosis is also well documented to produce deep gray matter involvement [6] and extensive white matter lesions mimicking demyelinating disorders such as multiple sclerosis [7]. Even though ceftriaxone-based regimens have been reported to be more successful with a shorter duration of therapy [8], in view of the variable sensitivity profile of Brucella species to third-generation cephalosporins, we still advocate aminoglycoside antibiotic–based (unless contraindicated) combination polymicrobial therapy for neurobrucellosis [9] to minimize the relapse rates.

The authors also did not mention the role of corticosteroids in severe neurobrucellosis. Despite the lack of definitive guidelines and evidence from randomized trials, short-course steroid therapy [10] (intravenous methylprednisolone for 3 days followed by oral prednisolone 1 mg/kg/day tapered over 4–6 weeks) has been found to be effective in minimizing the residual deficits in those with arachnoiditis, optic neuritis, and multiple sclerosis–like presentation (diffuse central nervous system involvement).

Note
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