

# Dissecting Aneurysm of the Basilar Trunk in a Young Man

**A**19-YEAR-OLD Lebanese man was admitted to a hospital because of status epilepticus. Following seizure control, he was noted to be quadriparetic, and a magnetic resonance image of the brain revealed a pontine lesion that was hyperintense on T2-weighted images (**Figure 1**) and hypointense on T1-weighted images, suggestive of an infarct. He was referred to our hospital for further evaluation and management. His medical history revealed 2 prior episodes of "aseptic" meningitis associated with generalized tonic-clonic seizures within a period of 1 year. He was empirically treated with acyclovir and valproic acid. On initial examination at our medical cen-

ter, the patient was afebrile and had normal heart sounds, normal results of chest and abdominal examinations, and no evidence of lymphadenopathy or meningeal signs. Neurologically, he was awake and oriented, with quadripareisis, anarthria, and dysphagia. The deep tendon reflexes were hyperactive all over, including the jaw jerk, a bilateral positive Babinski sign, and ankle clonus. The patient also had bowel and bladder dysfunction. A review of the magnetic resonance image of the brain revealed an abnormal configuration of the basilar trunk on a T2-weighted coronal section (**Figure 2**). He underwent 4-vessel cerebral angiography, which showed a lobulated, fusiform aneurysmal formation at the mid portion of the basilar trunk, measuring  $3 \times 4$  mm, with

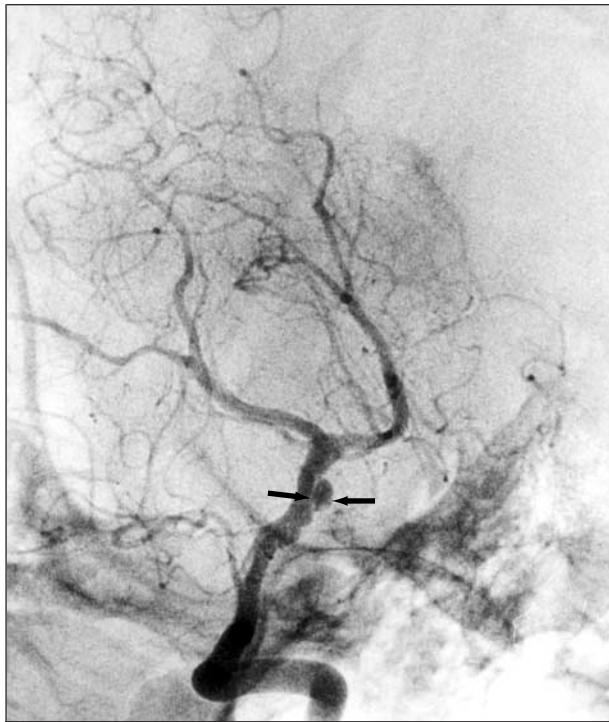
evidence of dissection (**Figure 3**). Complete blood cell count, results of blood chemical analysis, levels of antinuclear antibodies, anticardiolipin antibodies, lupus anticoagulant, and angiotensin-converting enzyme, and a coagulopathy profile were normal. Cerebrospinal fluid (CSF) studies showed lymphocytosis (white blood cell count, 5/mm; 82% lymphocytes, 16% segmented, and 2% eosinophils), a high protein level (2 g/L), and a low glucose level (46 mg/dL [2.5 mmol/L]). The corresponding serum glucose level was 150 mg/dL (8.3 mmol/L). Cerebrospinal fluid cultures for bacteria and mycobacteria were negative, and the polymerase chain reaction for tuberculosis in the CSF was negative. *Treponema pallidum* hemagglutination antibody and



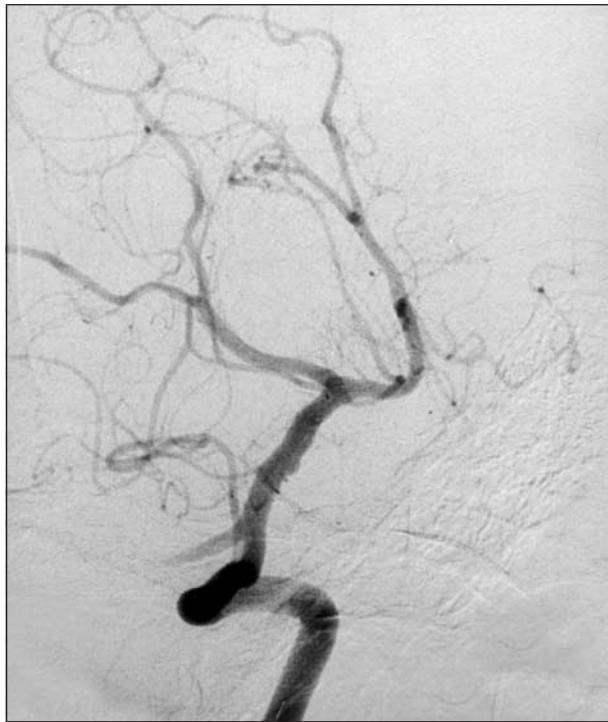
**Figure 1.** Magnetic resonance image (axial section, T2-weighted sequence) obtained at initial examination shows signal hyperintensity in the pontine area (arrow) and bilateral temporal lobes.



**Figure 2.** Magnetic resonance image (coronal section, T2-weighted sequence) obtained at initial examination demonstrates a lobulated aneurysmal dilatation of the basilar trunk (arrow).



**Figure 3.** Left vertebral angiogram obtained at the initial examination shows a lobulated aneurysm of the basilar trunk (right arrow) that is dissecting beyond the basilar artery wall (left arrow).



**Figure 4.** Left vertebral angiogram obtained at 6 months shows regression of the aneurysm.

VDRL tests were negative. *Brucella* titers, using a serum agglutinin test, were positive in the blood (direct, <1/20; indirect, 1/320) and the CSF (direct, <1/20; indirect, 1/160). The patient received a course of rifampin, doxycycline, and streptomycin. Soon after the initiation of treatment, he started showing signs of neurologic recovery. Six months later, the patient improved markedly. He recovered his speech and his sphincter control, regained motor function in the upper extremities, and started to walk with assistance. Repeated 4-vessel cerebral angiography revealed regression of the basilar aneurysm (**Figure 4**). Nine months later, the *Brucella* (direct and indirect) titers in the blood were both less than 1/20.

#### COMMENT

Brucellosis is one of the most endemic zoonotic diseases worldwide, including the Middle East. *Brucella melitensis* causes brucellosis in humans. *Brucella suis* has a preference for swine as animal hosts and *B. abortus* affects mainly cattle. Neurobrucellosis is very rare; it develops in less than 5% of systemic brucellosis cases.<sup>1</sup> Central nervous system involvement of the affected individuals can be categorized

into 1 of 6 groups: meningoencephalitis,<sup>2,3</sup> meningo-vascular involvement resulting in stroke or hemorrhage due to assumed rupture of mycotic aneurysm,<sup>4,6</sup> myelitis,<sup>2,3</sup> polyradiculitis,<sup>2,3</sup> neuritis involving the peripheral or cranial nerves,<sup>2,3</sup> or papilledema and increased intracranial pressure.<sup>5</sup> The diagnosis is based mainly on the constellation of the clinical signs and symptoms, serum analysis, and results of CSF serologic tests and cultures.

A good clinical outcome on medical treatment further supports the diagnosis. Treatment consists of a combination of 2 or 3 of the following medications: rifampin, doxycycline, streptomycin, or trimethoprim-sulfamethoxazole.

Herein, we report, to our knowledge, the first case of neurobrucellosis occurring with chronic recurrent meningoencephalitis, complicated by the occurrence of a lobulated dissecting aneurysmal formation of the basilar trunk. The aneurysm was first suspected on a magnetic resonance image of the coronal section at the level of the basilar artery and later confirmed by cerebral angiography. Although neurosyphilis may exhibit a similar clinical picture,<sup>7</sup> serum VDRL and treponema pallidum hemagglu-

tination antibody tests were negative. Furthermore, the patient did not receive the recommended treatment for tertiary syphilis. The *Brucella* titers revealed high indirect (immunoglobulin G) titers in both blood and CSF, indicating active infection with *Brucella* species that is not species-specific. The low direct (immunoglobulin M) titers suggest that the exposure to *Brucella* infection was not recent. The anti-*Brucella* treatment resulted in improvement of the neurologic status of the patient and the marked resolution of the aneurysm seen on the follow-up angiogram. Hence, it was assumed to be mycotic. The good clinical response as well as the normalization of the results of serologic tests for *Brucella* after treatment confirmed the diagnosis.

**Rosette Jabbour, MD**  
American University of Beirut  
Medical Center  
PO Box 113-6044  
Hamra 11032090  
Beirut, Lebanon  
(e-mail: rj04@aub.edu.lb)  
**Riad Khalifeh, MD**  
**Aghiad Al Kutoubi, MD**  
**Samir Atweh, MD**  
Beirut

**Author contributions:** Study concept and design (Drs Jabbour and Khalifeh); acquisition of data (Dr Jabbour); analysis and interpretation of data (Drs Kutoubi and Atweh); drafting of the manuscript (Dr Jabbour); critical revision of the manuscript for important intellectual content (Drs Khalifeh, Kutoubi, and Atweh); study supervision (Drs Khalifeh, Kutoubi, and Atweh).

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### ARCHIVES OF OPHTHALMOLOGY

#### High- and Low-Risk Profiles for the Development of Multiple Sclerosis Within 10 Years After Optic Neuritis: Experience of the Optic Neuritis Treatment Trial

*Optic Neuritis Study Group*

**Objective:** To identify factors associated with a high and low risk of developing multiple sclerosis after an initial episode of optic neuritis.

**Methods:** Three hundred eighty-eight patients who experienced acute optic neuritis between July 1, 1988, and June 30, 1991, were followed up prospectively for the development of multiple sclerosis. Consenting patients were reassessed after 10 to 13 years.

**Results:** The 10-year risk of multiple sclerosis was 38% (95% confidence interval, 33%-43%). Patients (160) who had 1 or more typical lesions on the baseline magnetic resonance imaging (MRI) scan of the brain had a 56% risk; those with no lesions (191) had a 22% risk ( $P < .001$ , log rank test). Among the patients who had no lesions on MRI, male gender and optic disc swelling were associated with a lower risk of multiple sclerosis, as was the presence of the following atypical features for optic neuritis: no light perception vision; absence of pain; and ophthalmoscopic findings of severe optic disc edema, peripapillary hemorrhages, or retinal exudates.

**Conclusions:** The 10-year risk of multiple sclerosis following an initial episode of acute optic neuritis is significantly higher if there is a single brain MRI lesion; higher numbers of lesions do not appreciably increase that risk. However, even when brain lesions are seen on MRI, more than 40% of the patients will not develop clinical multiple sclerosis after 10 years. In the absence of MRI lesions, certain demographic and clinical features seem to predict a very low likelihood of developing multiple sclerosis. This natural history information is a critical input for estimating a patient's 10-year multiple sclerosis risk and for weighing the benefit of initiating prophylactic treatment at the time of optic neuritis or other initial demyelinating events in the central nervous system. (2003;121:944-949)

*Corresponding author and reprints:* Roy W. Beck, MD, PhD, Optic Neuritis Study Group Coordinating Center, Jaeb Center for Health Research, 15310 Amberly Dr, Suite 350, Tampa, FL 33647 (e-mail: ontt@jaeb.org).