

Multiple Intracranial Tuberculomas in a Patient With Noonan Syndrome

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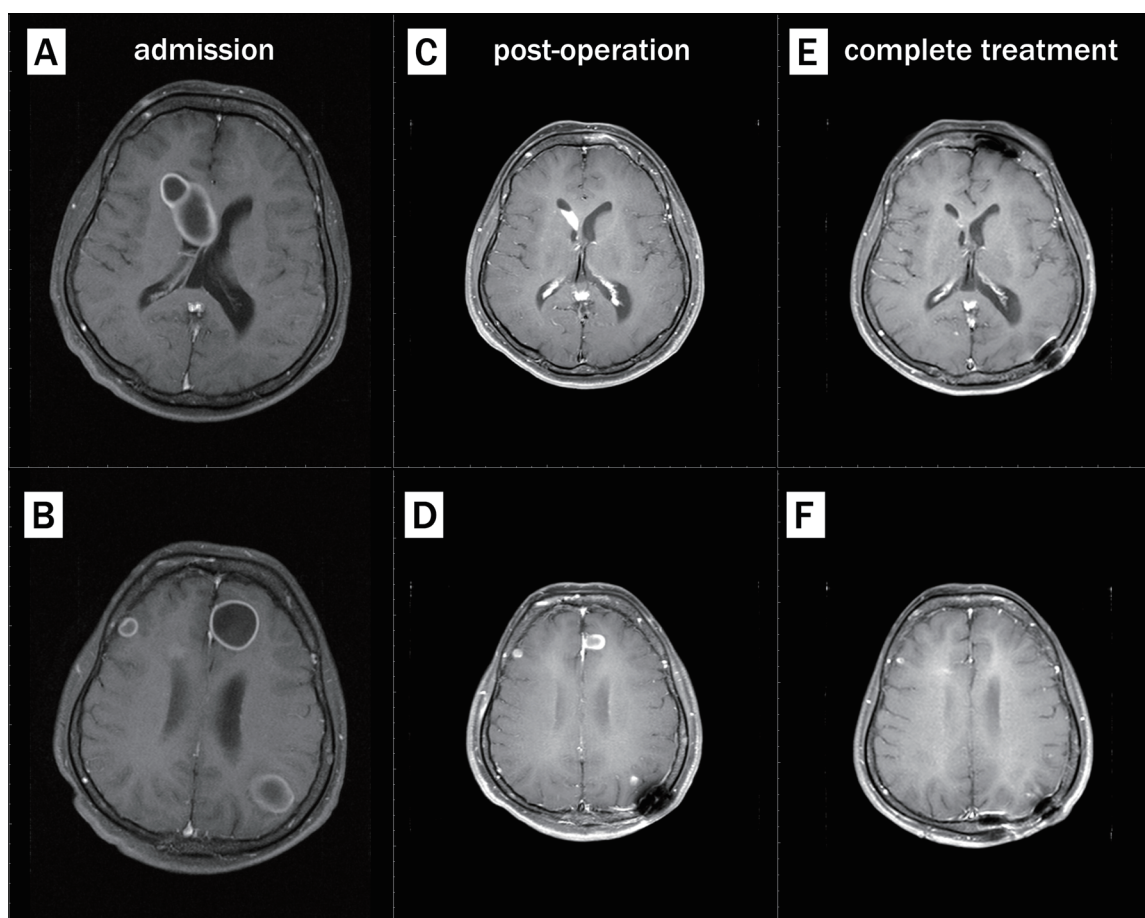


Figure 1. Magnetic resonance image (MRI) of the brain, postgadolinium T1-weighted image, axial views. (A) and (B), Admission MRI. (C) and (D), after surgical intervention. (E) and (F), three months after treatment completed. There are gadolinium-enhancing lesions in bilateral hemispheres. After surgery, the lesions markedly regressed. These lesions didn't recur after treatment completed.

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This 29-year-old female is a patient of Noonan syndrome presenting with short stature (145 cm). She was diagnosed as miliary pulmonary tuberculosis confirmed by acid-fast stain and culture of sputum. Four-combined anti-tubercular agents were administered. Human T-lymphotropic virus-1 and human immunodeficiency virus tests were negative. Acid-fast stain of sputum soon became negative and the lesions on chest X-ray resolved. Two months after starting anti-tubercular treatment, she developed intermittent bilateral temple headache without focal neurological signs or neck stiffness. Only mildly slow response and impaired attention were noted. Brain magnetic resonance imaging (MRI) revealed multiple brain abscesses associated with meningitis (Figure 1, A and B). The largest lesion (4 cm x 2 cm x 2 cm) was aspirated stereotactically. The aspirate was positive for acid-fast bacillus staining and mycobacterium polymerase chain reaction. Oral steroid was given in addition to previous anti-tubercular agent. In the following four months, she received serial stereotactic aspirations. After surgery, the lesions markedly regressed (Figure 1, C and D) and there was no neurological deficit. She received anti-tubercular agent for one year in total. Three months after treatment completed, there was no recurrence confirmed by brain MRI (Figure 1, E and F).

More than 80% of patients with Noonan syndrome have abnormalities of cardiovascular system⁽¹⁾, which may cause these patients susceptible to blood stream infection. But our patient didn't have cardiovascular abnormalities. Our patient has paradoxical development of intracranial tuberculomas during treatment of miliary pulmonary tuberculosis. This phenomenon has been observed and speculated as the result of an exaggerated immunological response⁽²⁻⁴⁾.

Tuberculomas can appear anywhere in the brain, but frontal, temporal and optochiasmatic lesions are associated with unfavorable outcome⁽³⁾. Biopsy is the gold standard in the diagnosis of intracranial tuberculomas. Surgical risk is lower in stereotactic brain biopsy compared with craniotomy^(5,6). Surgical intervention, with careful assessment, can be beneficial to the treatment of intracranial tuberculomas.

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