

Brain tuberculoma (*Mycobacterium africanum*): high index of suspicion helps in avoiding biopsy/surgery

A. Botturi · E. Prodi · A. Silvani · P. Gaviani ·
G. Vanoli · A. Carbone · A. Salmaggi

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Abstract Partial seizures can be due to a growing cerebral lesion, which may be tumoral or inflammatory/infectious in nature. The differential diagnosis is obviously important; increasing immigration to Europe from Africa is leading to an increase of infectious disease involving also the central nervous system. The authors report imaging the a case of a brain tuberculoma due to *Mycobacterium africanum* mimicking brain tumor, in which diagnosis was possible by inoculum in guinea-pig of material obtained by mediastinal biopsy of enlarged lymph nodes. Specific treatment led to marked reduction in the size of the brain lesion.

Keywords Cerebral tuberculoma · *Mycobacterium africanum* · MRI · Chest CT · Granulomatous epithelioid-cell lymphadenitis

We report the case of a young man from Guinea, who developed in the summer of 2007 (at age 22) partial motor seizures involving the right upper limb. His family history, physiological and remote pathological anamnesis were

reported as uneventful; he had left his country of origin in 2002, without going back to Guinea since that year.

At first admission in our hospital, in July 2008, neurological examination was normal. General physical examination was also normal. Neuroimaging showed a cerebral lesion in the left posterior frontal lobe (hyperintense in T2 and hypointense in T1-weighted images) with irregular contrast enhancement and no mass effect (Fig. 1a–c). Also numerous small area of hyperintensity in T2-weighted images were detected in the white matter of both the cerebral hemispheres, without contrast enhancement.

Microcalcifications were present at CT scan. Cerebrospinal fluid analysis was normal (0.3 elements/mm³, normal total protein and glucose), with negative IEF, and normal ACE levels. PCR for *Mycobacterium tuberculosis* was normal. CSF culture was normal. HIV antibodies and antibodies anti-schistosoma, strongyloides, amoeba, toxocara were absent. Tuberculin skin test was weakly positive. Chest CT showed multiple adenopathies with bilateral hilar involvement and also some enlarged axillar lymph nodes (Fig. 2a). Spinal MRI was normal. Total body PET-TC (FDG) performed in September 2008 showed high metabolic activity at the level of the chest adenopathies.

On September 12, 2008, the patient underwent biopsy of a paratracheal lymph node via mediastinoscopy; the histological diagnosis was of granulomatous epithelioid-cell lymphadenitis (Fig. 3). Culture of the bioptic specimen was negative, but the inoculum in guinea-pig led to development of *Mycobacterium africanum*. The patient was then started on a multi-drug regimen with isoniazid, rifampicin, ethambutol, and pirazine for 2 months, followed by two-drug therapy. Repeat MRI after 2 months of antitubercular treatment showed a marked reduction of the size of the cerebral lesion (Fig. 1d–f) and chest CT showed reduction

A. Botturi (✉) · E. Prodi · A. Silvani · P. Gaviani ·
A. Salmaggi
Fondazione IRCCS Istituto Neurologico Besta, Milan, Italy
e-mail: andrea.botturi@istituto-besta.it

G. Vanoli
Medicina Nucleare, e centro PET, Fondazione S. Raffaele del
Monte Tabor, Milan, Italy

A. Carbone
Anatomia Patologica, Fondazione IRCCS Istituto Tumori,
Milan, Italy

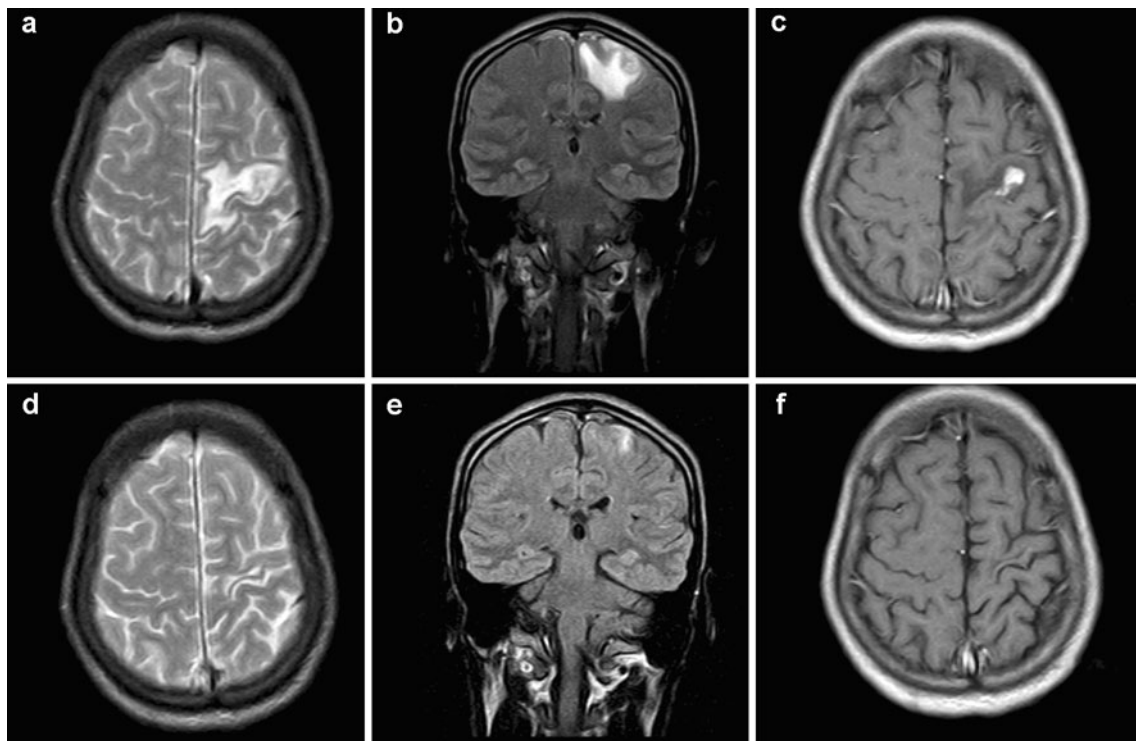


Fig. 1 Brain magnetic resonance imaging (MRI) before (a–c) and after (d–f) antitubercular therapy. MRI shows a cortico-subcortical lesion at the level of the left precentral gyrus with dishomogeneous low signal on T2 (a) and FLAIR (b) images and intense and homogeneous contrast enhancement on T1 images after gadolinium administration (c). Surrounding parenchyma shows high signal

intensity on long TR images (a, b) and low signal on T1 images (a), due to the presence of vasogenic edema. No significant mass effect is present. After antitubercular therapy, MRI demonstrates minimal residual high intensity signal on T2 (d) and FLAIR (e) images. There is no contrast enhancement after gadolinium administration (f)

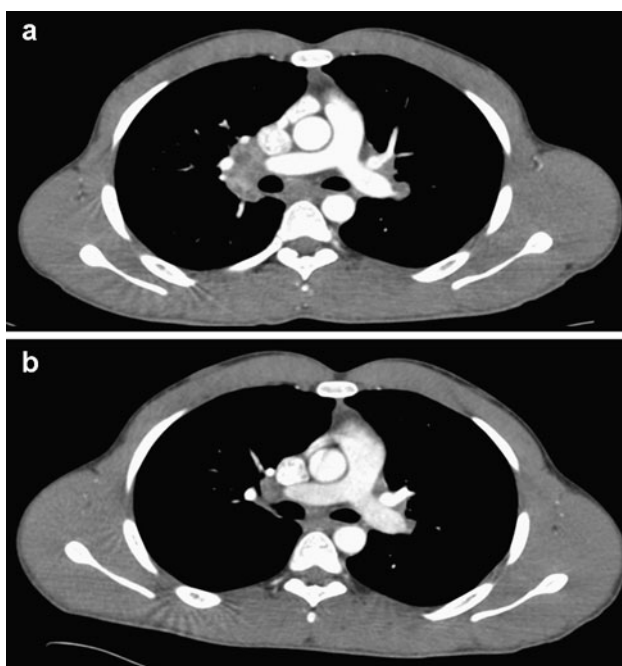


Fig. 2 Thorax contrast enhanced computed tomography (CECT) demonstrating enlarged mediastinal lymph nodes (a). CECT performed after antitubercular therapy shows reduction of lymphadenopathy (b)

of the thoracic adenopathies (Fig. 2b). *M. africanum* [1] is endemic in equatorial areas in Africa; its multi-organ tropism closely mirrors that of *M. tuberculosis*, but the course of the disease is generally less aggressive.

In this case, cerebral involvement was the first clue to diagnosis [2, 3]. The negativity of CSF examination in our case is noteworthy; nucleic acid amplification (NAA) tests have emerged as promising alternatives in the diagnosis and management of infectious diseases [4]. However, NAA is not sensitive as a culture of mycobacteria. Moreover, the yield of CSF culture for mycobacteria may be lower than that of tissue-derived material [5]. Many factors are implicated in negative NAA test, especially in extrapulmonary specimens, for example the presence of polymerase chain reaction inhibitors [6].

The identification and evaluation of new NAA targets for specific detection of MT may decrease the rate of false negative cases, however, the possibility of false negative results should be kept in mind when a clinical suspicion is present. With increasing immigration to Italy and Europe from Africa, cases such as this may become more frequent and neurologists should be aware of these presentations in order to avoid unnecessary and potentially harmful cerebral surgery/biopsy [7, 8].

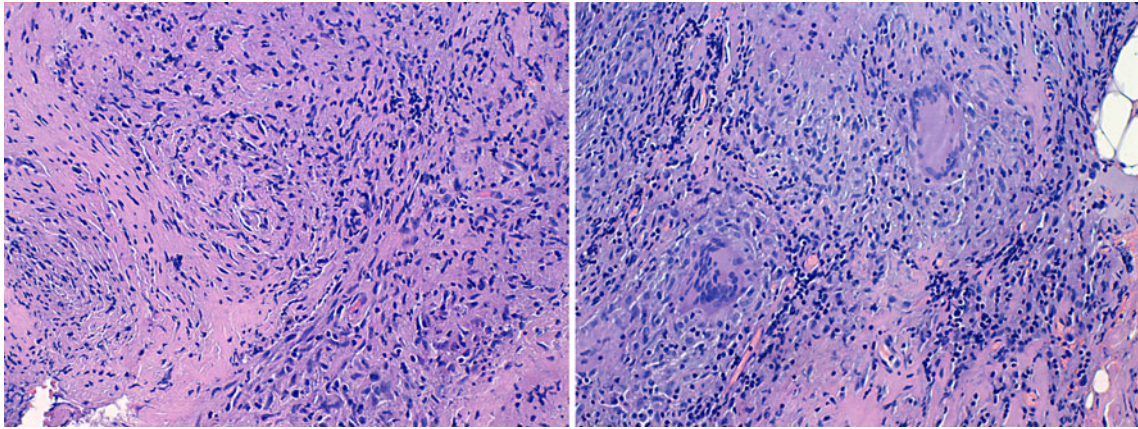


Fig. 3 Granulomatous epithelioid-cell lymphadenitis. H&E, $\times 10$

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