



## Neurobrucellosis: An Unusual Location of Brucellosis

Rym Abid\*, Olfa Smaoui, Imene Oueslati, Souha Hannachi, Riadh Battih and Bessem Louzir

Department of Internal Medicine, Military Hospital of Tunis, Tunisia

### Abstract

Man, 33 years old, a farmer, who had since two months an alteration of the general state and paresthesia type burn in lower and upper limbs, associated to weakness of the four limbs with progressive worsening, then appearance of instability to walking. Physical examination was objectified tetraparesia predominant in distal and posterior cord syndrome as well as static and kinetic cerebellar syndrome. The lumbar puncture had objectified hyper proteinorachie without other abnormalities. Cerebro-spinal cord MRI had shown a cerebellar and thalamus impairment. The serology of brucellosis in blood was positive. The diagnosis of neurological brucellosis had been retained. The patient was put under a triple therapy of antibiotics associated with corticotherapy with a good clinical improvement.

**Keywords:** Neurobrucellosis; Serology; Cerebellitis; Paralysis

### Introduction

Brucellosis is an anthroponozoonotic infection caused by bacteria of the genus *Brucella*. Its extension is worldwide with predominance in the Mediterranean basin and the developing countries. The clinical presentations of human brucellosis are polymorphic [1]. Neurological involvement remains rare but serious. The diagnosis of neurobrucellosis is based on the association of general and neurological clinical manifestations, brain imaging, serology of brucellosis and or positive blood cultures and a favorable response under specific treatment [1,2]. The purpose of our work is to recall this localization of brucellosis and to insist on the need for rapid and prolonged therapeutic management.

### Case Presentation

He was a 33-year-old man with no particular pathological background. He was a farmer from the North West of Tunisia. The patient had consulted for neurological signs appeared since 2 months. He complained of paresthesia type burns with muscle weakness of the four limbs and instability in walking appeared since two months. The patient had an impairment of general condition with a weight loss of 30 kg in two months and anorexia, without fever or night sweats.

The patient had not fever, with cardiopulmonary, abdominal and osteoarticular examination without abnormalities. Neurologically, he was aware and well oriented in time and space. The cranial nerves were intact. He had muscular atrophy of the four limbs with tetraparesia predominant in lower limbs. The patellar and ankle reflexes were absent on both sides with an impairment of the superficial sensitivity predominant in lower limbs. A static and kinetic cerebellar syndrome was objectified by a Romberg sign, incoordination in the finger-nose test, an ataxic walk with enlargement of the support polygon and difficulty in walking at a straight line, worsening all when closing the eyes. The electromyogram confirmed the neurogenic involvement of the four limbs with a severe axonal sensitive-motor neuropathy predominant in the lower limbs. A cerebro-spinal MRI showed diffuse lesions in hypointense T1 and hyperintense T2 Flair at the posterior fossa and involvement of the cerebellar white matter with enhancement after gadolinium injection (Figures 1 and 2). In the two thalami, a lesion was individualized in hypointense T1 and hyperintense T2 Flair, enhancing after gadolinium injection (Figures 3 and 4). The biological assessments were without abnormalities, in particular no biological inflammatory syndrome and no abnormalities in the blood count. The study of cerebro-spinal fluid showed a clear appearance with normal cytology (WBC= 2 / mm<sup>3</sup>), normal glucorrachia, hyper proteinorachie at 1.29 g/l and negative culture. Given the epidemiological context, a serology of brucellosis was requested, and was positive: Bengal Rose Test +++ and Wright serology in blood was positive at 1/360. The blood cultures were negatives. The diagnosis of neurobrucellosis was retained with polyradiculoneuritis and cerebellitis. The patient has received a triple antibiotic therapy based on Rifampicin (15 mg/kg/day), Doxycycline 200 mg/

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#### \*Correspondence:

Rym Abid, Department of Internal Medicine, 1008 Montfleury, Military Hospital of Tunis, Tunisia, Tel: 21899471066;

E-mail: abid\_rym@yahoo.fr

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**Figure 1:** Cerebro-spinal MRI: diffuses lesions in hyposignal T1 of the cerebellar white matter.



**Figure 3:** Cerebro-spinal MRI: lesions in hyposignal T1 of the thalami.



**Figure 2:** Cerebro-spinal MRI: diffuses lesions hyperintense T2 Flair of the cerebellar white matter.



**Figure 4:** Cerebro-spinal MRI: lesions hyperintense T2 Flair.

day and Sulfamethoxazole-trimethoprim (40 mg/kg/day), associated with oral corticosteroid therapy (prednisone) at a dose of 1 mg/kg/day for two weeks and then at a regressive dose of 10 mg/week. He has received also Pregabalin for neurogenic pain. The evolution was marked by progressive clinical improvement with improvement of walking after two weeks and of cerebellar syndrome after 2 months, as well as a decrease in Wright's serology rate and a regression of brain lesions in control imaging. The total duration of treatment was 12 months. The patient had kept memory problems as sequels.

## Discussion

Brucellosis is the most common zoonotic infection in the world. Its incidence varies from one country to another. It is still a public health problem in the Mediterranean basin [1]. The mode of transmission of this infection is generally digestive by the consumption of unpasteurized dairy products or cutaneo-mucus by contact with the flock. *Brucella melitensis* is the species most frequently responsible for human brucellosis [2,3].

Neurological involvement in brucellosis is rare but serious. It was first described by Hughes in 1896. The neurological manifestations can be seen at different stages of the disease: in the acute phase of brucellosis and in the sub-acute or chronic phase [1]. Physiologically, the involvement of the nervous system could be related either the intracellular development of the bacterium or the immune response mechanisms of the body against infection [4].

The neurological involvement of brucellosis is polymorphic and can affect both the central and peripheral nervous system. Several clinical presentations are possible. Meningitis and encephalitis are the most common sites in brucellosis. However, it may be myelitis, polyradiculoneuritis or sometimes diffuse involvement. Cerebellitis is

an unusual complication of brucellosis and is poorly described in the literature [1,5,6]. In our case, it was the combination of a peripheral polyradiculoneuropathy and a cerebellitis.

Neurobrucellosis must be evoked in association with an orienting epidemiological context, lesions of the nervous system not explained by another neurological pathology with confirmation of systemic progressive brucellosis, either by serology or blood cultures [5,7]. In fact, blood cultures are positive in 24% of cases of neurobrucellosis [1]. Cerebro-spinal fluid study assists in the diagnosis of neurobrucellosis if meningitis or meningoencephalitis is suspected. It typically shows lymphocytic meningitis [5,7]. Cerebrospinal fluid culture is positive for *Brucella* in 14% of neurobrucellosis [1].

At radiological explorations, neurobrucellosis can be manifested essentially by three types of lesions: inflammatory lesions, abnormalities of the white matter signal and ischemic vascular lesions [5,8].

The management of neurobrucellosis should be fast and effective because of the severity of the pathology. Treatment should include active antibiotics on *Brucella* and with good neuromeningeal diffusion for a long period. There is no consensus on the choice of antibiotic, the dose and the duration of treatment of neurobrucellosis, but the treatment is usually based on a combination of two or three antibiotics among doxycycline, rifampicin, trimethoprim-sulfamethoxazole, streptomycin or ceftriaxone [1,9,10]. The duration of treatment is variable ranging from 2 to 15 months, but duration of at least 6 months is recommended [1,5,11]. The indication of corticosteroids in the therapeutic management of brucellosis in combination with antibiotics is not well codified. Corticosteroid therapy is recommended in order to act on inflammatory process. Some authors indicate corticosteroid therapy systematically in front of a neurobrucellosis while others prescribe it in case of severe

forms: arachnoiditis, cranial nerve damage, myelopathy, intracranial hyperpressure, optic neuritis or papillary syndrome [4,5,9].

The evolution of neurobrucellosis is variable depending on the type of injury. Paralysis related to brucellosis usually regress completely with the administration of antibiotics, while the central nervous system is associated with permanent neurologic deficits. However, mortality during neurobrucellosis remains low [11,12].

## Conclusion

Neurological manifestations of brucellosis are polymorphic and of varying severity. The neurological involvement must be suspected on clinical and or radiological abnormalities, in an epidemiological context suggestive of brucellosis. Therapeutic management must be rapid but it is not clearly codified.

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