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JCIMCR OPEN ACCESS Journal of Clinical Images and Medical Case Reports

ISSN 2766-7820

Case Report

Open Access, Volume 5

Brucellosis presenting as mononeuritis multiplex

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Received: Jan 11, 2024

Accepted: Jan 29, 2024

Published: Feb 05, 2024

Archived: www.jcimcr.org

DOI: www.doi.org/10.52768/2766-7820/2836

Abstract

Background and aim: Brucella infection is common and causes central and peripheral nervous system disorders. The peripheral nerve involvement is usually a symmetrical sensorimotor polyneuropathy. Guillain-Barre Syndrome and carpal tunnel syndrome have also been described in brucellosis. There is no description of mononeuritis multiplex in this infection.

Methods: We describe a case of an Iraqi man who presented with symptoms of a systemic infection. Nerve conduction studies confirm mononeuritis multiplex. Blood and CSF studies confirm an infection with Brucella.

Results: The nerve conduction studies reveal severe bilateral median and ulnar nerve entrapment in the forearm and hands as well as sensorimotor polyneuropathy in the lower extremities.

Interpretation: We describe a case of acute brucellosis presenting with the electrophysiological findings of mononeuritis multiplex.

Introduction

Brucella infection is very common in the Mediterranean countries. Neurological manifestations of brucellosis have been described over the past two decades [1]. Many central and peripheral nervous system pathologies have been described in patients with acute and chronic Brucella infection [2]. To our knowledge, mononeuritis multiplex has not been described previously in brucellosis.

Case report

We describe a case of a 58-year-old Iraqi man previously healthy, not hypertensive or diabetic, with no previous history of any systemic or neurological disorders, and no family history of neurological diseases, presented with back pain, weight loss, headache, and dizziness for the past three months. His vital signs were normal. He was afebrile. He had a normal cardiovascular and lung examination, a soft abdomen, and normal peripheral pulses.

The neurological examination revealed mild proximal and distal muscle weakness in the lower extremities, partially because of low back pain. Patellar reflexes were normal. He had absent Achilles deep tendon reflexes bilaterally. Normal mental status, orientation, memory, speech, and cranial nerves including fundoscopy. No cerebellar signs. Negative Romberg and tandem walking. Mild decreased sensation for light touch and vibration in the feet.

The patient underwent an MRI of the brain, cervical, and dorsal spine, which were normal. Chest x-ray was normal. Routine hematology was normal with a WBC count of 5600 cells/cc, and chemistry studies were normal. Brucella titers were elevated with the direct antibody >1:640 and brucella indirect antibody >1:1280.

CSF studies were performed revealing brucella direct antibody <1:20 and brucella indirect antibody >1:20; CSF WBC was 24 cells/cc, protein 0.41 mg% and glucose 52 mg%.

The patient underwent a nerve conduction studies of the median, ulnar, peroneal, posterior tibial and sural nerves in his four limbs (Table 1). The results showed evidence for a mild generalized, symmetrical, sensorimotor axonal polyneuropathy affecting the motor and sensory fibers in the lower extremities. Furthermore, nerve conduction studies revealed severe bilateral carpal tunnel syndrome and severe entrapment of the left ulnar nerve at the olecranon fossa. These electrophysiological findings are suggestive of mononeuritis multiplex (Table 1).

Table 1: Nerve conduction study parameters of the median, ulnar, posterior tibial, peroneal and sural nerves. Latency in milliseconds, amplitude in millivolts or microvolts, and conduction velocities in meters/second.

	Latency (ms)	Amp (mV)	CV (m/s)
Median MNC			
Wrist (L)	5.9	9	
Wrist-Elbow (L)	11.6	7	39
Wrist (R)	5.3	9.5	
Wrist-Elbow (R)	11.2	8.6	37
Median SNC			
Wrist (L)	5.1	6	29
Wrist (R)	5.2	14	29
Ulnar MNC			
Wrist (L)	3.7	9.7	
Wrist-Below Elbow (L)	8.2	9.3	42
Below-Above Elbow (L)	12.4	7.9	29
Wrist (R)	3.5	12.3	
Wrist-Below Elbow (R)	7.8	11.8	47
Below-Above Elbow (R)	11.1	10.7	42
Ulnar SNC			
Wrist (L)	3	4	40
Wrist (R)	3.1	17	39
Peroneal MNC			
Ankle (L)	3.4	2.1	
Ankle-Fibula (L)	13.7	1.8	41
Ankle (R)	3.7	0.9	
Ankle-Fibula (R)	14.6	0.8	39
Tibial MNC			
Ankle (L)	4.7	3	
Knee (L)	15.4	2.9	41
Ankle (R)	4.6	2.9	
Knee (R)	15.1	2.3	42
Sural SNC			
Ankle (L)	3.4	2	41
Ankle (R)	3.4	2	45

Interpretation

The case described above is of a previously healthy man with no risk factors for any peripheral nervous system disorder presenting with the clinical and serological studies confirming acute brucellosis. He had mild weakness of his lower extremities limited by the back pain. His nerve conduction studies show multiple severe nerve entrapments in the median and ulnar nerves of both upper extremities as well as a polyneuropathy of the lower extremities.

The review of the literature confirms disorders of the central and peripheral nervous system in acute and chronic brucellosis. Central nervous system disorders in brucella infection occur in around 5% of confirmed cases [1]. The pathology involves occlusive vascular disease, meningitis, encephalitis, white matter disease, myelitis, and even psychiatric conditions [1]. These symptoms usually subside after a prolonged treatment with the conventional anti-bacterial therapy [2].

Furthermore, peripheral nervous system involvement in brucellosis has been extensively described in the literature. These disorders occur in around 41% of confirmed cases of neurobrucellosis [3]. Most of the pathologies of the peripheral nervous system associated with brucella infection involve sensory and motor axons of the peripheral nerves presenting clinically and electrophysiologically as a classical sensorimotor axonal polyneuropathy. These cases usually reveal elevated CSF white blood cell count and protein level with a positive serology for brucella either B.melitensis or B.abortus [1,2].

Isolated plexus or isolated peripheral nerve involvement has also been reported in brucella infections such as acute lumbosacral neuritis, brachial plexusitis, or sciatic neuritis [1,2]. Involvement of many roots in brucella infection presents with the clinical picture of polyradiculitis whereby the CSF protein is usually elevated and the MRI lumbosacral spine reveals enhancement of the cauda equina. These cases can be independent of other reported cases of cauda equine involvement in patients with brucella infection of the lumbosacral discs with discitis and inflammation of the cauda equina [2].

Mononeuropathies secondary to infection of isolated nerves are very rare in brucella infection and have been reported as infectious carpal tunnel syndrome. These patients improved clinically and electrophysiologically with the treatment of the brucella rather than decompression of the carpal tunnel [4].

Brucellosis has also been the etiological trigger of acute inflammatory demyelinating polyneuropathy. The described cases develop the full-fledged picture of the Guillain-Barre syndrome days or weeks after the diagnosis of the brucellosis. The Guillain-Barre syndrome described in these cases could be the classical demyelinating type or even the axonal type (AMSAN). These cases have the classical abnormal CSF and ganglioside antibody profile. These patients do not improve only with treatment of the brucellosis but require the conventional treatment of GBS by plasma exchange or IVIG [5].

Recently, a case report describes subacute motor polyradiculopathy associated with brucella infection. The pathology involved only the motor fibers sparing the sensory fibers confirmed by electro-diagnostic studies. EMG on this patient revealed also fibrillation potential and small motor units and the author labeled this as a pseudomyopathic pattern associated with a polyradiculopathy in a case of brucellosis [3].

The value of the case we are presenting is that the patient had no risk factor for mononeuropathy or polyneuropathy and presented with only systemic symptoms of an underlying infection. On the other hand, the nerve conduction study revealed

acute severe multifocal nerve entrapment in the median and ulnar nerves in both upper extremities without the patient complaining of symptoms related to these nerve pathologies. The electrophysiology also revealed pathology in the motor and sensory fibers of the lower extremities compatible with sensorimotor polyneuropathy. Our case suggests that the diagnosis of mononeuritis multiplex can be associated with systemic brucellosis. The fact that the patients CSF protein was normal confirms that the pathology is a mononeuritis multiplex and not a radiculitis. The association is further strengthened with the absence of commensurate symptoms of nerve involvement as well as elevated CSF white blood cell count and protein level and positive CSF serology of Brucella. These patients should not undergo surgical decompression of the entrapment but rather be treated with the classical anti-brucella therapy.

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