

Case Report

Guillain Barre Syndrome Associated with Brucellosis: A Case Report and Review of the Literature

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Abstract

Introduction: Guillain-Barré syndrome (GBS) ranks as the most frequent cause of acute flaccid paralysis in the world. It is an autoimmune polyradiculoneuropathy, usually preceded by an acute infection. Rarely, brucellosis may induce a GBS.

Objective: To evaluate the clinical and microbiological diagnostic properties of Brucella-induced GBS.

Case Report: A 54-year-old woman, with no past medical history, was followed in infectious disease department. She was diagnosed with brucellosis. She had received antibiotic therapy (Rifampicin 600 mg/day and Doxycycline 200 mg/day). After 4 days of treatment, she was referred to our department because of rapidly progressive, ascending, symmetric weakness and bilateral paralysis of muscles of the face. On admission, she was alert. The deep tendon reflexes (DTRs) were absent in all extremities. Muscle strength was 3/5 in the upper extremities and 2/5 in the lower extremities. Proprioception in the lower extremities was impaired, but she did not have any sensory problems. Our patient also presented a facial diplegia. Physical examination was normal, except for splenomegaly. A lumbar puncture showed an albumin-cytologic dissociation in the CSF. Nerve-conduction studies were suggestive of demyelinating polyradiculoneuropathy. Coombs Wright titration was 1/160.

Discussion: With a diagnosis GBS preceded by brucellosis, our patient was given an antibiotic therapy (Rifampicin 600 mg/day and Doxycycline 200 mg/day). During hospitalization, she had four plasma exchange sessions and a motor rehabilitation. In a follow-up after 4 weeks, our patient presented a partial recovery, and she was able to walk without support.

Conclusion: This case demonstrates that brucellosis can present with a rare neurologic manifestation including GBS. Molecular mimicry seems to be responsible for this complication, through the synthesis of autoantibodies against myelin gangliosides. Thus, brucellosis should be ruled out in all patients who develop acute flaccid paralysis, especially in those who live in endemic areas.

Keywords: Brucellosis; Guillain-Barré syndrome; Neurobrucellosis

Introduction

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Case Report

A 54-year-old woman, with no past medical history, was followed in infectious disease department. She was diagnosed with brucellosis. She did not have immediate contact with contaminated animals but reported consumption of unpasteurized dairy products. She had received antibiotic therapy (Rifampicin 600 mg/day and Doxycycline 200 mg/day). After 4 days of treatment, she was referred to our department because of rapidly progressive, ascending, symmetric weakness and bilateral paralysis of muscles of the face. The patient did not complain of breathing problems, swallowing disorders or vesicosphincteric disorders. On admission, she was alert. The deep tendon reflexes (DTRs) were absent in all extremities. Muscle strength was 3/5 in the upper extremities and 2/5 in the lower extremities. Proprioception in the lower extremities was impaired, but she did not have any sensory problems. Our patient also presented a facial diplegia. Physical examination was normal, except for splenomegaly. A lumbar puncture showed a raised protein level of 0.7 g/l and 2 white blood cells (WBC). Glucose level was normal. CSF culture did not identify any bacterial growth. Nerve-conduction studies were suggestive of demyelinating polyradiculoneuropathy.

Serology of the human immunodeficiency virus (HIV) and Campylobacter jejuni was negative. Coombs Wright titration was 1/160. An abdominal ultrasound confirmed the clinical finding of splenomegaly.

With a diagnosis GBS preceded by brucellosis, our patient was given an antibiotic therapy (Rifampicin 600 mg/day and Doxycycline 200 mg/day). During hospitalization, she had four plasma exchange sessions and a motor rehabilitation. In a follow-up after 4 weeks, our patient presented a partial recovery, and she was able to walk without support.

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Page 2 of 3

Discussion

Our case demonstrates the development of post infectious polyradiculoneuropathy. The acute progressive, ascending, symmetric weakness and areflexia were compatible with a diagnosis of GBS. The electrodiagnostic findings and the albuminocytologic dissociation in the CSF supported our diagnosis.

Guillain-Barré syndrome is an acute inflammatory polyradiculoneuropathy. It is preceded, in most of cases, by a gastrointestinal or respiratory infection [1]. Brucella-induced GBS has been rarely reported. In fact, the nervous system may be involved in diffuse brucellosis. Although rare, neurobrucellosis is still a problem throughout the world and particularly in the Mediterranean Basin. The involvement of nervous system can be categorized into central or peripheral and may be either acute or chronic [2]. The peripheral form, that of polyradiculoneuropathy, was found to occur in around 7% of cases [3]. The occurrence of polyradiculopathy during the acute phase of illness may be due to the immunologic and inflammatory reactions of the host to the presence of these bacteria. In fact, in an experimental animal model, the ganglioside-like molecules expressed on the surface of Brucella induced autoantibodies against myelin gangliosides, resulting in acute paralysis and GBS signs [4]. That's why serological tests for brucellosis should be performed in cases of flaccid limb weakness in endemic areas. The cultures in this case are most often negative and serology using different tests: Wright-agglutination test, the rose Bengal test, the coombs test, the mercapto-ethanol test and the ELISA, can confirm the diagnosis. In our study, as in the literature, the treatment of Brucella induced GBS is the combination of two or three suitable antibiotics for long periods, with intravenous immunoglobulin or plasma exchange sessions.

There have been a few case reports of GBS associated with brucellosis in the literature. Ten patients were reported. In our study, as in the literature, symptoms referable to this complication included a progressive, ascending, symmetric weakness, hypotonia, areflexia with or without sensory problems. Wright-agglutination test, the rose Bengal test, the coombs test, the mercapto-ethanol test or the ELISA were used to confirm the diagnosis. An appropriate antibiotic therapy was prescribed to most of patients for a minimum of four months, associated with intravenous immunoglobulin or plasma exchange sessions. In the literature and in our case, brucella-induced GBS resolved with proper and early management. Data about GBS associated with brucellosis found in the literature are presented in Table 1 [5-12].

Authors	Age/Sex	Country/Year	The Clinical Presentation	Diagnosis-Tools	Treatment
Garcia [5]	Three patients	Espagne (1989)	Acute progressive, ascending, symmetric weakness	Coombs Wright titration	Rifampicine+ Doxycycline
			Paresthesia	Nerve-conduction studies	Plasma exchange sessions
			Areflexia		A partial recovery of 2 patient. death of the thir
Al-Eissa [6]	9-year-old A woman	Arabie saoudite (1996)	Acute progressive, ascending, symmetric weakness	Coombs Wright titration	Rifampicine+ Doxycycline
			Protracted paroxysms of severe hypertension	Nerve-conduction studies	Plasma exchange sessions
					A partial recovery after 2 months
Akdeniz [7]	60-year-old A woman	Turkie (1997)	Flaccid paralysis	CSF culture Brucella melitensis	Rifampicine+Doxycycline (6 months) + Streptomycine (1,5 month)
			Hyporéflexia	The Wright agglutination test	A partial recovery after one year
				The Rose Bengal test	
				Nerve-conduction studies	
Kochar [8]	32-year-old A man	India (2000)	Proximal polyradiculopathy	CSF culture Brucella melitensis	Rifampicine + Doxycycline + Streptomycine
			Hypotonia	The Wright agglutination test	Plasma exchange sessions
			Hyporéflexia	ELISA	A partial recovery after 5 months
				Nerve-conduction studies	
Namiduru [9]	14 years A woman	Turkie (2003)	Acute progressive, ascending, symmetric weakness	CSF culture Brucella melitensis	Rifampicine+ cotrimoxazole
				The Wright agglutination test	Plasma exchange sessions
				Nerve-conduction studies	A partial recovery after 2 months
Barzegar [10]	9-year-old Homme	Iran (2009)	Progressive weakness in the lower extremities	The Wright agglutination test	Plasma exchange sessions
			Paresthésia	2-mercaptoethanol titer : negative	A partial recovery after 2 months
				Nerve-conduction studies	
Montalvo R [11]	40-year-old Homme	Peru (2010)	Progressive, ascending, symmetric weakness	The Wright agglutination test	Rifampicine + Doxycycline (4 mois)
			Hypotonia	Rose bengale test	Plasma exchange sessions
			Aréflexia	Nerve-conduction studies	A partial recovery after 3 months
Farhang babmahmoodi [12]	26-year-old A woman	Iran (2011)	Breathing problems	Coombs Wright titration = 1/160 Rifampicine+Doxycycline +Gentamicine mois)	
			Progressive, ascending, symmetric weakness		
			Hypotonia	Mercapto-étanol Test ≥ 1/40	Intravenous immunoglobulin
			Aréflexia	IgG GM1 (+)	A partial recovery after 1.5 month
			Proprioception in the lower extremities was impaired	Nerve-conduction	

Table 1: Clinical presentation, diagnostic tools and treatment of patients presenting a Guillain-Barré syndrome associated with brucellosis.

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Conclusion

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Page 3 of 3