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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.

 $\textbf{Keywords} \hbox{: Brucellosis; Guillain-Barr\'e syndrome; Neurobrucellosis}$

Introduction

Guillain-Barre 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 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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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 third |
| 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) | Difempioine + Devyoyoline + Centerniaine /2 |
| | | | 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.

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.

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