CASO CLÍNICO/CASE REPORT

Chronicle of a Foretold Intersection: *Mycoplasma pneumoniae* and Brown-Séquard Syndrome

Crónica de uma Intersecção Anunciada: *Mycoplasma pneumoniae* e a Síndrome de Brown-Séquard

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Abstract

Mycoplasma pneumoniae is a frequent cause of community-acquired respiratory infection in children but can also produce neurological complications. We report a 9-year-old child who developed neck pain followed by right hemiparesis and contralateral sensory loss, compatible with a Brown-Séquard-like presentation. Brain imaging was normal; spinal magnetic resonance imaging (MRI) showed a focal, right-lateral, C2–C4 T2-hyperintense lesion without contrast enhancement, consistent with myelitis. Respiratory multiplex PCR and serology supported recent *M. pneumoniae* infection. The patient improved substantially after corticosteroids, targeted antibiotics and rehabilitation, with near-complete functional recovery and no recurrence of disease. Serum MOG-IgG and AQP4-IgG were negative, and neuroimaging did not reveal features typical of MOGAD or NMOSD, helping exclude primary demyelinating disease. This case highlights *M. pneumoniae* myelitis presenting as a Brown-Séquard syndrome in childhood and underscores the importance of a structured differential diagnosis to guide timely and etiology-specific management.

Resumo

O *Mycoplasma pneumoniae* é uma causa frequente de infeção respiratória adquirida na comunidade em idade pediátrica, podendo também causar complicações neurológicas. Apresenta-se o caso de uma criança de 9 anos que desenvolveu cervicalgia, seguida de hemiparesia direita e perda sensitiva contralateral, compatíveis com uma apresentação tipo síndrome de Brown-Séquard. A imagiologia cerebral foi normal; a ressonância magnética medular revelou uma lesão focal lateral direita entre C2–C4, hiperintensa em T2, sem realce pós-contraste, compatível com mielite. A PCR multiplex respiratória e a serologia identificaram infeção recente por *M. pneumoniae*. A criança apresentou melhoria substancial após corticoterapia, antibioterapia dirigida e reabilitação, com recuperação funcional quase completa e sem recidiva. Os anticorpos MOG-IgG e AQP4-IgG séricos foram negativos e a neuroimagem não mostrou características de MOGAD ou NMOSD, permitindo excluir doença desmielinizante primária. Este caso reforça a importância do diagnóstico diferencial estruturado para uma abordagem etiológica e atempada.

Introduction

Brown–Séquard syndrome (BSS) is a rare form of spinal cord injury resulting from hemisection of the cord, clinically characterized by ipsilateral loss of motor function, vibration and proprioception, together with contralateral impairment of pain and temperature sensation.[1] Although its true incidence is unknown, BSS occurs more frequently in adults and is typically associated with penetrating trauma or motor vehicle accidents.[1] Despite trauma being its most common cause, several infectious etiologies have also been reported.[1]

Mycoplasma pneumoniae (*M. pneumoniae*) is an atypical pathogen classically associated with community-acquired respiratory infections in school-aged children and adolescents.[2,3] Beyond respiratory disease, *M. pneumoniae* can lead to neurological involvement with a broad clinical spectrum, including meningoencephalitis, acute disseminated encephalomyelitis (ADEM), myelitis, cerebellar ataxia, Guillain–Barré syndrome, cerebellar infarction and peripheral neuropathy.[2-5] Encephalitis is the most common neurological manifestation and, in approximately one-third of cases, may occur in the absence of respiratory symptoms.[2,4,5]

The pathophysiology remains incompletely understood.[3,4] Neurological complications may develop without any preceding respiratory symptoms and when present, such symptoms are often non-specific.[3-6] Although serology remains the diagnostic standard, it requires paired acute and convalescent samples, frequently confirming infection only retrospectively.[3,4] Detection of the organism in upper airway samples may simply represent carriage, particularly in healthy children or during community outbreaks.[4,5] Moreover, *M. pneumoniae* DNA is rarely identified in cerebrospinal fluid, even in patients with strong clinical suspicion of central nervous system involvement.[3,5,6]

Case Report

A previously healthy 9-year-old girl initially presented with a productive cough and rhinorrhoea on Day 1. On Day 3, she developed cervical pain radiating to the right upper limb, followed on Day 4 by subjective weakness in the right upper and lower limbs. By Day 5, she reported paresthesia and loss of sensation in the left limbs and experienced urinary incontinence. Six days after symptom onset, she attended the Emergency Department of the hospital in her area of residence. Neurological examination demonstrated right-sided hemiparesis without facial involvement. Brain computed tomography (CT) and magnetic resonance imaging (MRI) were normal, and she was transferred to a tertiary center.

On admission, she exhibited right-sided hemiparesis, with grade 2 distal strength in the upper limb and grade 2 proximal strength in the lower limb. Deep tendon reflexes were hypokinetic in the right superior limb (brachioradialis, biceps and triceps), while patellar and Achilles reflexes were brisk on the same side. Plantar responses were bilaterally equivocal. A left-sided sensory level at D2 was noted for pain, as well as vibratory hypoesthesia with a high dorsal level, but on the right side; a right-sided steppage gait was also evident. Overall, findings suggested a cervical right-sided pyramidal syndrome involving both the upper and lower limbs, with impaired proprioception on the right, hypoalgesia on the left, and early sphincter dysfunction, consistent with a Brown–Séquard-like presentation.

Given suspected cervical BSS, vertebro-medullary MRI was done and revealed a focal T2-hyperintense lesion in the right lateral cord at C2–C3 extending to C4 (most pronounced at C3), without post-contrast enhancement, suggestive of an inflammatory/demyelinating etiology (myelitis) (**Fig. 1**). Cerebrospinal fluid analysis was unremarkable. Treatment was initiated with intravenous methylprednisolone (1 g/day), a single intravenous dose of ceftriaxone (4 g), and intravenous ciprofloxacin (1 g/day for 7 days, followed by 800 mg/day for an additional 7 days). Respiratory multiplex PCR identified *M. pneumoniae* and serology testing confirmed a recent infection; ceftriaxone was discontinued.

After 12 days of corticosteroids (including 7 days of

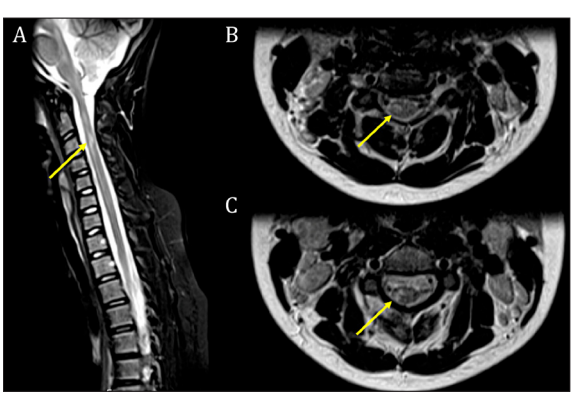

Figure 1. Spinal cord MRI revealing a hyperintense lesion on T2-weighted images (arrows), extending from C2 to C4 (A, sagittal T2) with clear right lateralization on axial sections (B and C, FLAIR).

intravenous methylprednisolone), 14 days of intravenous ciprofloxacin and one week of physiotherapy, she showed marked improvement in motor function and gait, with complete resolution of sphincter symptoms, enabling discharge. Serum myelin oligodendrocyte glycoprotein (MOG-IgG) and aquaporin-4 (AQP4-IgG) antibodies were negative. Cerebrospinal fluid oligoclonal bands showed a mirror pattern. Corticosteroids were gradually tapered over 1 month, and follow-up was continued in rehabilitation and demyelinating disease clinics.

Over 10 months of physiotherapy, recovery was remarkable. One-year follow-up MRI demonstrated resolution of the previously observed right-sided T2-hyperintense C2–C4 lesion, with no residual signal abnormality or new lesions. At the most recent pediatric neurology evaluation, strength had recovered to grade 4 in the right wrist, hand and thigh, with persistent right-sided hyperreflexia. Overall, the clinical course was favorable, with near-complete functional recovery and no recurrence.

Discussion

Beyond its well-known respiratory manifestations, *M. pneumoniae* can also cause neurological complications, particularly in children. These may include encephalitis, acute disseminated encephalomyelitis (ADEM), transverse myelitis and other post-infectious immune-mediated syndromes.[2-5] The underlying pathophysiology is not fully understood, but both direct central nervous system invasion and immune-mediated mechanisms have been proposed.[3,4] In our case, the distinguishing feature was the topography of the spinal cord lesion, resulting in a BSS-like presentation, an exceptionally rare manifestation of *M. pneumoniae* myelitis. Identification of *M. pneumoniae* through multiplex PCR in respiratory samples, supported by serology, together with substantial improvement following targeted antibiotic therapy and corticosteroids, strongly supports a causal relationship.

A crucial step in the diagnostic work-up was the exclusion of primary demyelinating disorders. MOG-IgG and AQP4-IgG were negative, and neuroimaging lacked features consistent with myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD), and neuromyelitis optica spectrum disorder (NMOSD), according to contemporary diagnostic criteria. Multiple sclerosis (MS) was also considered unlikely, given the absence of characteristic MRI findings or a relapsing clinical course,[7-10] so far.

This case underscores the importance of including *M. pneumoniae* infection in the differential diagnosis of acute myelitis, even when the clinical presentation is atypical, such as a BSS. A comprehensive and structured diagnostic approach is essential to exclude primary demyelinating disease and to ensure timely, etiology-specific treatment, which can promote excellent neurological recovery and minimize long-term sequelae. ■

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CFT: Conceptualization; data collection and clinical research; data analysis; manuscript writing; submission and management of the editorial process.

MC: Data collection and clinical research; data analysis; manuscript writing.

JP and JA: Data analysis and interpretation; critical review of the manuscript.

FP: Conceptualization; clinical supervision; analysis and validation of conclusions; critical review of the manuscript.

All authors approved the final version to be published.

CFT: Conceptualização; Colheita e investigação clínica; análise dos dados; redação do manuscrito; submissão e gestão do processo editorial.

MC: Colheita e investigação clínica; análise dos dados; redação do manuscrito.

JP e JA: Análise e interpretação dos dados; revisão crítica do manuscrito.

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