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Central and Peripheral Nervous System Demyelination Following Mycoplasma Pneumonia Infection With Review of Literature

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Objective

Describe the rare occurrence of a combined central and peripheral nervous system demyelination in a serologically positive *Mycoplasma pneumoniae* (*M. pneumoniae*) patient.

Case Presentation

- A previously healthy 18-year-old white male presented with a dull headache, upper thoracic and lower cervical area pain, as well as progressive upper and lower extremity weakness. Two weeks prior to this event, he experienced a non-productive cough, which was persistent and lasted for one week. Upon physical examination, there was decreased sensitivity from the level of T3 down. Proximal and distal motor weakness was evident on muscle testing. The upper extremities displayed no movement against gravity. Patellar and ankle reflexes were absent.
- Initial laboratory results showed a complete blood count and complete metabolic panel within normal limits, with the exception of 93% neutrophils and 6% lymphocytes. A cerebral spinal fluid (CSF) analysis on admission showed 32 mg/dl protein (normal 12 to 60 mg/dl), 67 mg/dl glucose (normal 40 to 85 mg/dl), and zero white blood cells. A CSF specimen for bacterial culture was negative. Legionella, West Nile Virus, Lyme disease, Syphilis, and anti-nuclear antibody profile laboratory tests were negative.
- The patient's neurologic status rapidly deteriorated with worsening lower extremity weakness. Plasmapheresis therapy was initiated for suspected Guillain-Barré syndrome. On the third day of hospitalization, the patient was completely areflexic with quadriparesis. He underwent intubation and mechanical ventilation with eventual tracheostomy. His hospitalization was complicated by a spontaneous pneumothorax which required a temporary chest tube. Serology revealed a positive IgM antibody to *M. pneumoniae* with negative IgG, indicative of recent/current infection. Results of an electromyogram (EMG) were consistent with a predominantly axonal demyelinating peripheral neuropathy. A cervical MRI displayed multiple areas of pathologic enhancement from C4 through T1, compatible with a transverse myelitis. Neurologic and clinical improvement of our patient's combined polyradiculopathy and transverse myelitis occurred after five treatments of plasmapheresis, seven days of intravenous dexamethasone, and ten days of azithromycin. However, his left leg remained paralyzed. After a nineteen day hospitalization, the patient was transferred to a long term acute care center for rehabilitation.

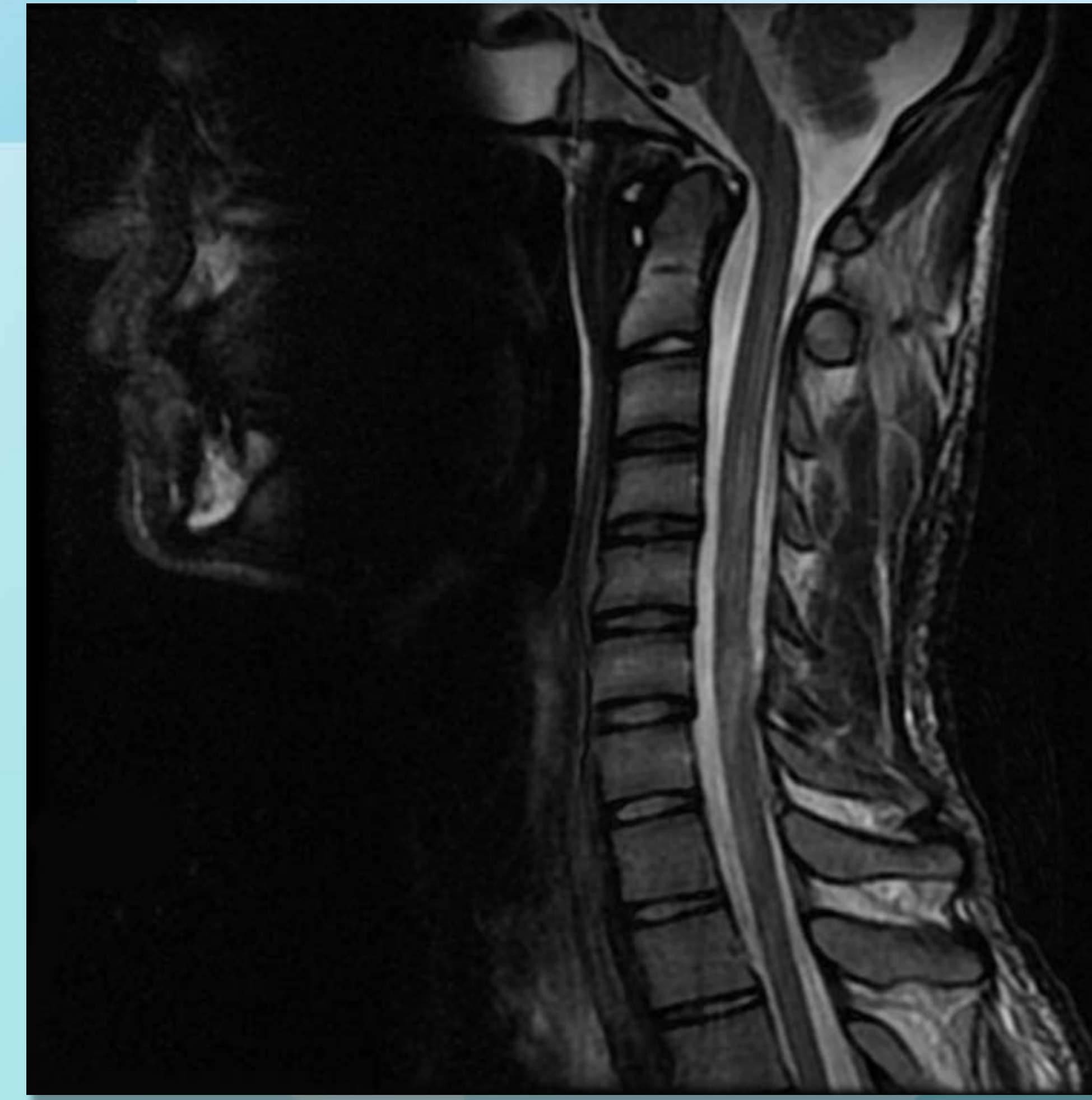


Figure 1.
Pathologic enhancement of the spinal cord from C4 to T1

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Discussion

- *M. pneumoniae* is associated with a wide spectrum of neurologic complications. These clinical manifestations range in severity and have been documented primarily through case reports within the medical literature. Neurologic diseases affected by this pathogen include encephalitis, meningoencephalitis, transverse myelitis, aseptic meningitis, cerebellar ataxia, cerebellar infarction, optic neuritis, cranial nerve palsies, polyradiculitis, peripheral neuropathy, Guillain-Barré syndrome, and psychosis [2-7]. Central and peripheral nervous system involvement occur in approximately 0.01-4.8% of patients infected with *M. pneumoniae* [2]. However, the overall incidence of neurologic complications may be lower than expected, given that only a minority of *M. pneumoniae* patients have a confirmed infection and are hospitalized [8,9].
- The pathogenesis of central nervous system disease secondary to *M. pneumoniae* infection remains unknown. There are three major categories that attempt to identify the pathogenesis of neurologic complications associated with *M. pneumoniae* infection. The first pathomechanism is classified as a direct-type which involves local site inflammation through the action of cytokines [13,14]. The next category refers to an indirect-type of mechanism that causes inflammation through autoimmunity, immune complexes, and allergies [13]. The third mechanism encompasses vascular occlusions which predispose the patient to either a thrombosis or vasculitis [15].
- Serology is commonly used for diagnosing *M. pneumoniae*. While the sensitivity and specificity of serology is high, this method of testing can be time consuming and interfered with by cross-reactions [3,16]. Polymerase chain reaction (PCR) testing is another diagnostic approach. PCR is beneficial because it does not require viable microorganisms and it can analyze histological specimens, fluid, and serum with rapid results. The disadvantage of PCR is that it has the tendency to overestimate the incidence of *M. pneumoniae* [3,17].
- The treatment of choice for neurologic manifestations in a confirmed *M. pneumoniae* patient remains undefined. The variability in treatments stem from the ambiguity of the bacteria's pathogenesis, small number of affected patients, and lack of controlled clinical trials. A combination of antibiotics, corticosteroids, and plasmapheresis is commonly utilized in this patient population.

Conclusion

M. pneumoniae remains a common respiratory pathogen that is capable of causing a wide-spectrum of extrapulmonary complications. Neurologic disease occurs more frequently with severe complications in comparison to other nonpulmonary manifestations of *M. pneumoniae*. Timely diagnosis and treatment is hindered by the lack of a clearly defined pathogenesis, sensitivity and specificity of available diagnostic techniques, and clinical trials to assess therapeutic treatment options. Recognition of central and peripheral nervous system demyelination in association with respiratory tract symptoms should prompt serological and PCR investigation for *M. pneumoniae*.