

A Case of Cerebral Infarction Associated with *Mycoplasma pneumoniae* Infection^{*}

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ABSTRACT

A 4-year-old boy developed right hemiplegia and aphasia during *Mycoplasma pneumoniae* (*M. pneumoniae*) infection. Computed tomography of the brain showed a large cerebral infarction of the left temporal area. Carotid angiography revealed an obstruction of a branch of the left middle cerebral artery. The possible pathogenesis of central nervous system (CNS) symptoms due to *M. pneumoniae* infection is discussed.

INTRODUCTION

Clinical manifestations of *Mycoplasma pneumoniae* infection include pneumonia, rashes, hematologic abnormalities, myocarditis, pericarditis, and arthritis. In addition, many CNS manifestations have been reported. In this paper, we describe a case of cerebral infarction associated with *M. pneumoniae* infection.

CASE REPORT

A 4-year-old boy was referred to us because of right hemiplegia and aphasia. He had been in good health until 9 days prior to admission when he started to have pyrexia and cough. A diagnosis of pneumonia was made and he was treated with Lincomycin. Five days later, he became aphasic, and paralysis of the right lower and upper extremities was noted. On the next day, swelling with tenderness of the left parotid and right submandibular glands appeared, and a coincidental infection with mumps virus was diagnosed.

On admission, physical examination revealed an irritable boy with clear consciousness. There was flaccid paralysis of the right lower and upper extremities. Tendon reflexes, including Achilles's and patellar reflexes, were absent

on the right. Right abdominal wall and right cremasteric reflexes were also absent. All reflexes on the left side were normal. There were no pathological reflexes, nuchal rigidity or Kernig's sign. The pupillary light reflexes were normal bilaterally. No signs of cranial nerve neuropathy were noted. There were no rashes on the skin. Breath sounds were normal and there were no rales audible. Hepatosplenomegaly was absent.

Red blood cell, white blood cell and platelet counts were normal, as were bleeding time, prothrombin time and partial thromboplastin time. Blood sedimentation rate was 86 mm/h. The cold agglutination titer on day 6 of his illness was 1 : 256 and on the day 9 1 : 1024. The serum *M. pneumoniae* passive hemagglutination titer was 1 : 640 on day 6 of the illness and the *M. pneumoniae* complement fixation (CF) titer was 1 : 512 on day 18.

On the day 6 of his illness the cell count in the cerebrospinal fluid (CSF) was 24/3 mm³, and the *M. pneumoniae* CF titer in the CSF was 1 : 1. On day 10, the second CSF examination showed pleocytosis, 63/3 mm³ with mononuclear cells 61/3 mm³, and the *M. pneumoniae* CF titer was 1 : 2. Protein and sugar concentrations of CSF were within normal lim-

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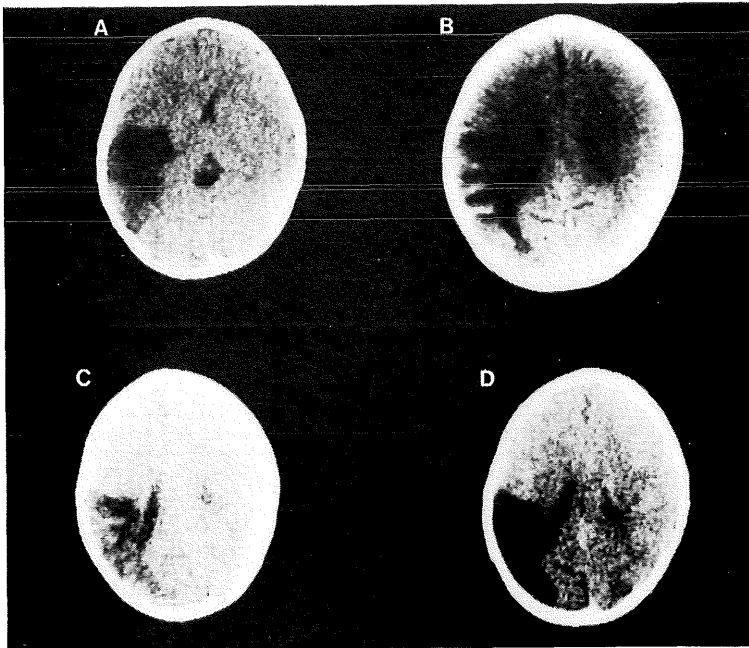


Fig. 1. Computed tomography of brain on 5th day (A) of his illness, 1 month later (B), 3 months later (C), and 1 year later (D)



Fig. 2. Carotid angiography showing obstruction of a branch of the left middle cerebral artery

its.

Computed tomography of the brain demonstrated a large low density area in the left temporal lobe (Fig. 1). Carotid angiography showed an obstruction of a branch of the left middle cerebral artery. The left posterior

parietal artery was not visualized (Fig. 2). Electroencephalography showed low voltage and decreased frequency of theta waves in the temporal and occipital areas, comparable to those of the computed tomographic and angiographic abnormalities.

The patient was treated with Lincomycin intravenously for seven days, then Doxycycline orally for ten days. Glycerol and dexamethasone were administered for ten days for CNS symptoms. The swelling of his salivary glands disappeared on the 7th day of his illness. Resolution of aphasia occurred rapidly. On the 20th day, he regained ability to walk and to move his right arm. Computed tomography of the brain showed a decrease in the low density area of the left temporal lobe. A fungiform area was noted in the low density area, suggesting recovery of vessels.

DISCUSSION

The concurrence of pneumonia and a rise in specific antibody titer is best explained by *M. pneumoniae* infection in our patient. The clinical manifestations of *M. pneumoniae* infection

are numerous, and polyradiculitis, encephalitis, aseptic meningitis, acute cerebellar ataxia, cranial nerve neuropathy, psychosis and transverse myelitis have been reported^{4,5,8,10}. Hemiplegia is frequent in children with multifocal encephalitis due to *M. pneumoniae* infection⁹. However, isolated hemiplegia without changes in mental status is extremely rare.

Several possibilities on the pathogenesis of CNS involvement secondary to *M. pneumoniae* infection have been suggested. *Mycoplasma neurolyticum* produces a neurotoxin causing degeneration of brain tissue in mice¹¹ and some strains of *Mycoplasma gallisepticum* induces cerebral polyarteritis in turkeys¹². However, no neurotoxin has been demonstrated for *M. pneumoniae*. In one case of CNS disease associated with *M. pneumoniae* infection, *M. pneumoniae* has been isolated from the CSF⁸. However, numerous attempts to isolate *M. pneumoniae* have been unsuccessful, suggesting that invasion of the CNS by *M. pneumoniae* is not necessary for the appearance of CNS complications⁸. An autoimmune process in which antibodies against *M. pneumoniae* are directed against brain tissue has also been suggested¹¹. The tendency to hypercoagulability in many cases of *M. pneumoniae* infection may cause thrombosis of the vessels in some region of the CNS^{2,6}.

The precise mechanisms in the cerebral infarction of our patient remain unclear. Parker et al.⁷ reported the first case of cerebral infarction with hemiplegia and suggested the possibility of local vasculitis secondary to *M. pneumoniae* infection. A local vasculitis and hypercoagulability due to *M. pneumoniae* might play a role in the obstruction of brain vessels.

Finally, we could not clarify the cause of pleocytosis observed in our patient. It might be due to coincidental infection with mumps virus. The possibility that *M. pneumoniae* infection was in part responsible for the CNS symptoms could not be excluded.

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