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Transient striatal involvement with frequent seizures and fast recovery associated with *Mycoplasma pneumoniae* infection

Mycoplasma pneumoniae infeksiyonu ile ilişkili geçici striatal tutulum görülen, sık nöbet geçiren ve hızlı iyileşme gösteren bir olgu

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ABSTRACT

Mycoplasma pneumoniae **is a pathogen of atypical pneumonia and can cause various kinds of extrapulmonary manifestations. Central nervous system is the most affected organ during or after the course of *Mycoplasma pneumoniae* infections. Presentation with recurrent seizures, neuropsychiatric symptoms and transient basal ganglia involvement have been rarely described. Here we report a case with recurrent seizures and neuropsychiatric presentation including hallucinations and transient basal ganglia involvement with rapid clinical improvement.**

Key words: Basal ganglia involvement, children, seizure, *M. pneumoniae* infection

ÖZET

Mycoplasma pneumoniae **atipik pnömoni etkenidir ve akciğerler dışındaki organ ve sistemleri de etkileyebilir; en sık ekstrapulmoner etkilenim sinir sisteminde görülür. *Mycoplasma pneumoniae* infeksiyonu ile ilişkili sık nöbetler, nöropsikiyatrik semptomlar ve geçici basal ganglion tutulumu ender olarak bildirilmiştir. Burada yineleyen nöbetleri, halüsinasyon gibi nöropsikiyatrik bulguları ve geçici bazal ganglion tutulumu olan, klinik bulguları kısa sürede düzelen bir olgu sunulmuştur.**

Anahtar kelimeler: Bazal ganglion tutulumu, çocuklar, nöbet, *M. pneumoniae* infeksiyonu

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INTRODUCTION

Mycoplasma pneumoniae is an important pathogen which causes nervous system disorders during or after the course of a respiratory tract infection. Central nervous system involvement occurs in 0.1% of all *M. pneumonia* infections [(1)]. Central nervous system complications include encephalitis, acute disseminated encephalomyelitis, transverse myelitis, cranial nerve palsies, stroke, acute and chronic inflammatory polyneuropathies, ocular myasthenia gravis and cerebellitis [(2)]. Acute bilateral striatal necrosis is a symmetrical degeneration of the caudate nucleus and putamen and sometimes the globus pallidus, substantia nigra and tegmental nuclei. Although *M. pneumonia* infections may be a cause of acute bilateral striatal necrosis, transient basal ganglia involvement associated with *M. pneumonia* infections has been rarely reported [(3-8)]. On the other hand, frequent recurrent seizures with hallucinations as a manifesta-

tion of *M. pneumonia* infections are also rare. Here we report a case with seizures and transient striatal involvement associated with *M. pneumonia* infection.

CASE REPORT

A previously healthy four-year-old male was admitted to our hospital with a one week history of fever and coughing and one day history of headache. Six days ago, he was diagnosed as upper respiratory tract infection and was prescribed azithromycin by his family physician. On admission, vital signs were normal. Oropharynx and right tympanic membrane were hyperemic. Remainder of the physical and neurologic examinations was normal. During follow up in the pediatric emergency department, he had a simple febrile seizure. Abnormal laboratory findings at admission included a leukocyte count of 21.000/ mm^3, and C-reactive protein of 105 mg/L. He was hospitalized and intravenous ceftriaxone was started. He had three additional generalized tonic-clonic seizures on the first day of hospitalization which were controlled with phenytoin and valproic acid. On the second day of his hospitalization, the patient became confused and began to have intermittent hallucinations. Neurologic examination of the patient revealed a Glasgow Coma Scale of 11 with increased deep tendon reflexes and bilateral Babinski sign. There was neither neck stiffness nor Kernig sign. A lumbar puncture was performed because of ongoing hallucinations and revealed 90 leukocytes, a protein level of 20 mg/dl (15-45 mg/dl) and a glucose level of 60 mg/dl (simultaneous blood glucose 100 mg/dl). Meningoencephalitis was suggested and acyclovir was added to ceftriaxone treatment. Electroencephalograms were normal and brain magnetic resonance imaging showed increased patchy T2 signal of bilateral basal ganglia (corpus striatum) (Fig. 1). Results of PCRs performed for adenovirus, herpes simplex virus, varicella zoster virus and enterovirus were unremarkable. Serum *M. pneumoniae* immunoglobulin M and G (34 RU/ml) were positive. Acyclovir treatment was stopped and chlarityromycin was started at a dose of 15 mg/kg/day. Metabolic examinations including blood gases, serum ammonia, lactic acid and pyruvic acid analyses were normal. Antiganglioside antibodies were not detected. On the fifth day of hospitalization the patient's neurologic status began to recover and he was completely normal after the first week of hospitalization. A control magnetic resonance imaging one week later after the first imaging showed no abnormality (Fig. 2). A control serologic examination for *M. pneumonia* performed four weeks after discharge showed increased titers of IgG (64 RU/ml) and *M. pneumonia* IgM.

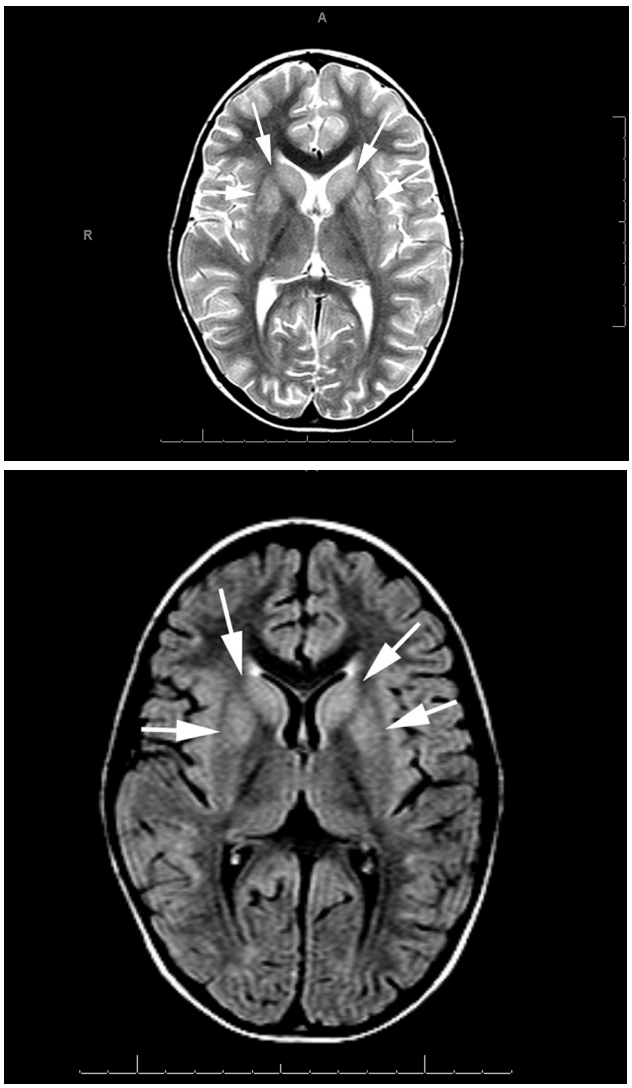

Figure 1. T2 weighted (a) and Fluid attenuated inversion recovery (FLAIR) sequences (b), transverse images show bilateral increased signal of basal ganglia (arrows).

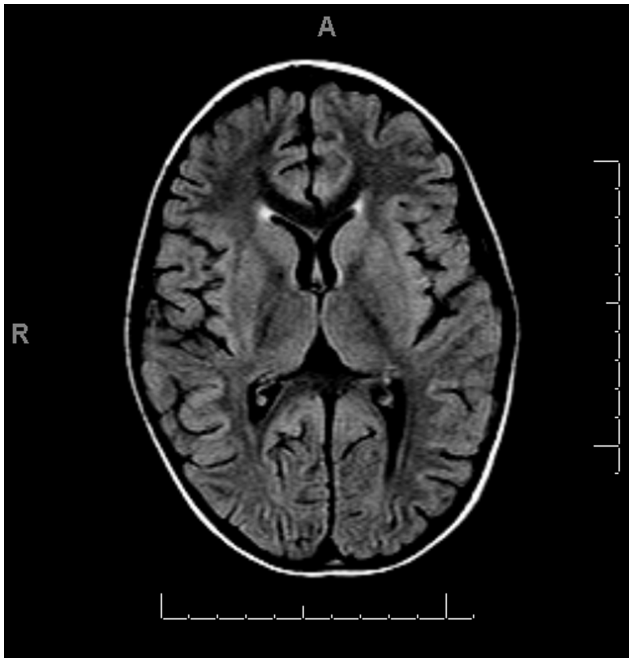

Figure 2. Control MRI shows no abnormal signal of basal ganglia on FLAIR image.

DISCUSSION

Mycoplasma Pneumoniae which is known to cause atypical pneumonia is an important infectious pathogen in pediatric population. Extrapulmonary manifestations of *M. pneumoniae* are of major clinical significance and central nervous system is the most affected organ during or after the course of *M. pneumoniae* infections. Cytokine production or a direct type, immune-mediated or indirect type mechanisms, and vascular occlusion are three hypotheses to explain central nervous system involvement [9]. Acute bilateral striatal necrosis is usually associated with either endogenous or exogenous toxins and with poor neurodevelopmental outcomes. Bilateral striatal lesions in childhood occur infrequently. Bilateral high T2- and low T1- signal changes involve a wide range of etiologies including viral infections, mitochondrial disorders, hypoglycemia and exposure to exogenous and endogenous toxins [10]. Bilateral acute striatal necrosis has also been described in cases with *M. Pneumoniae* infections. Some of the cases recovered completely during the follow up, but some of the cases exhibited neurologic disorders like intractable dystonia, overactive urinary bladder due to loss of dopaminergic inhibition, chorea and coordination difficulties [3-8]. Acute phase of bilateral striatal necrosis lasts a few days to weeks with subsequent clinical improvement and persistence of the striatal lesions in the neuroradiological follow up. Rapid improvement and the complete resolution of the cerebral lesions have also been reported [7-8]. To best of our knowledge, only two cases of basal ganglia and thalamic involvement associated with *M. pneumoniae* infection have been reported who showed rapid clinical and radiological recovery [7-8]. The first case was a two-year-old boy who presented with abrupt onset of ataxia, irritability and lethargy. Brain magnetic resonance imaging showed bilateral basal ganglia, and thalamic hyperintensity. Eleven days after the onset of symptoms, the patient fully recovered with pulse steroid and intravenous immunoglobuline treatments. One month later, a control magnetic resonance imaging showed complete resolution of the lesions. The patient had also increased anti-GM1 ganglioside IgM antibodies [7]. The second case was a previously healthy 5-year-old boy who presented with an atypical pneumonia. He rapidly developed encephalitis revealed by a generalized status epilepticus. After transient improvement, he became confused and mutic, with dystonic postures of his limbs. CT scan and MRI showed abnormal signals in the whole basal ganglia, typical of bilateral striatal necrosis. Serologic tests for *Mycoplasma pneumoniae* were positive. The child recovered almost completely [8]. Our case showed a more rapid recovery and did not need an immunomodulatory treatment. Radiologic resolution also occurred in one week and antiganglioside antibody and metabolic test results were unreemaarkable. Transient basal ganglia involvement along with rapid clinical improvement and cerebrospinal fluid pleocytosis of our case suggested an episode of benign encephalitis.

Seizures are common manifestations of *M. pneumoniae* encephalitis [11]. During the acute phase of encephalitis, it was shown that 53.5% of patients had seizures. The most common seizure type is primary

focal seizures associated with secondary generalized tonic-clonic seizures [12]. Recurrent seizures and neuropsychiatric presentation including hallucinations have been rarely described. Type of refractory seizures include generalized tonic-clonic seizures, generalized nonconvulsive seizures, focal seizures with secondary generalization, neuropsychiatric symptoms or expressive aphasia [13]. Arkilo et al described three patients with refractory seizures and hallucinations whose seizures were controlled by immunomodulatory treatments including corticosteroids and immunoglobulins [13]. In our case seizures were of generalized tonic-clonic type, which were controlled with phenytoin and valproic acid without need for immunomodulatory treatment.

In conclusion, besides severe neurologic manifestations, *M. pneumoniae* may cause transient striatal involvement with frequent seizures, and fast neurologic recovery without need for immunomodulatory treatment is possible.

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