Neuroendocrinology Letters Volume 44 No. 2 2023
ISSN: 0172-780X; ISSN-L: 0172-780X; Electronic/Online ISSN: 2354-4716
Web of Knowledge / Web of Science: Neuroendocrinol Lett
Pub Med / Medline: Neuro Endocrinol Lett

Left middle cerebral artery occlusion associated with mycoplasma pneumonia in a child: A case report

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Submitted: 2022-09-04 Accepted: 2023-03-07 Published online: 2023-03-07

Key words: **mycoplasma pneumoniae; cerebral infarction; middle cerebral artery infarction; stroke**

Neuroendocrinol Lett 2023; **44**(2):63–67 **PMID:** 37182227 NEL440123C02

Abstract

Cerebral infarction is a rare and severe manifestation of central nervous system damage caused by mycoplasma pneumoniae infection. We report that a 16-year-old girl was hospitalized with cough, expectoration and fever for 5 days and shortness of breath for 1 day. At the time of admission, the chest computed tomography showed double lung fields infiltration and pleural effusion. The detection of mycoplasma pneumoniae antibodies (IgG and IgM) were positive. The right limb movement of the patient was found incapacitated on the seventh day of hospitalization. Computed tomography, magnetic resonance imaging and magnetic resonance angiography of the head demonstrated the acute cerebral infarction after mycoplasma pneumoniae infection. Early anti-infective therapy, microcirculation improvement and rehabilitation treatment improved the prognosis of this child. Craniocerebral imaging examinations and laboratory tests are helpful for diagnosis. Early detection and treatment can improve the prognosis of patients.

CASE REPORT

INTRODUCTION

Mycoplasma pneumoniae is a common cause of upper and lower respiratory tract infections, most commonly in children and young people (Kumar *et al.* 2019). Previous knowledge of mycoplasma pneumonia was limited to the fact that it was the cause of benign respiratory disease. In recent years, extrapulmonary complications of mycoplasma pneumoniae infection, including the central nervous system symptoms, were more and more recognized by people. These include encephalitis, meningoencephalitis, aseptic meningitis, transverse myelomyelitis, acute disseminated encephalomyelitis, Guillain-Barre syndrome, cerebral infarction and so on (Tsiodras *et al.* 2005; Daxboeck, 2006).

Given the risk of mycoplasma pneumoniae associated stroke and the limitations of its understanding, we reported that a 16-year-old girl was hospitalized with left middle cerebral artery occlusion after mycoplasma pneumonia, thereby improving clinicians' understanding of this disease and providing experience for the diagnosis and treatment of mycoplasma pneumoniae associated stroke.

CASE REPORT

A 16-year-old girl was admitted to the emergency ward with a cough and fever. Five days ago, she developed a cough, yellow phlegm and fever

without any obvious inducement. Her body temperature was up to 39.1 °C, and she did not have chills. She took roxithromycin, acetaminophen and other drugs orally. Four days ago, she received an intravenous infusion of cephalosporin and azithromycin at a local clinic. Shortness of breath appeared one day ago, and she was admitted to the local hospital. The enhanced computed tomography (CT) of the chest suggested pneumonia and pleural effusion, and she was recommended to be transferred to our hospital.

She is a junior high school student, slim, healthy, likes sports, and has no special genetic or family history. She was fully conscious and normal in her orientation at the time of physical examination. Vital signs: Body Temperature 38.7 °C, Heart Rate 114 beats/min, Respiratory Rate 26 breaths/min, and Blood Pressure 94/53 mmHg. The strength of the girl's limbs was normal and no positive signs were found in the neurological examination. Arterial blood gas measurement revealed a pH of 7.44, a PO_2 of 63 mmHg, a PCO_2 of 36 mmHg, and an SO_2 of 92.7%. Her breathing was thick and her lungs could be heard wet and dry rales. On admission, echocardiography (ECG) indicated sinus tachycardia and the chest CT indicated double lung field infiltration and pleural effusion (Figure 1). The results of the laboratory examinations on the day of admission are shown in Table 1.

On the second day of admission, the patient's temperature fluctuated between 39.1 and 39.6 °C. The patients were treated with meropenem and doxycycline combined with abidol hydrochloride. On the third day of admission, the patient developed restlessness, delirium and cyanosis, and the oxygen partial pressure dropped to 53 mmHg. The patient was transferred to the Intensive Care Unit and given ventilator-assisted treatment. Later, the patient retreated from heat, regained consciousness, the limb movements were normal, and the blood oxygen saturation increased to 98%. It appeared that the patient's symptoms gradually improved, with fibrinogen 2.69 g/L and platelet 121×10^9/L, but D-dimer 27.4 ug/mL. The blood culture and sputum smear were negative many times. Two-timed cardiac ultrasonography only indicated mild tricuspid regurgitation and a small amount of pericardial effusion. Her homocysteine mass spectrometry was 16.36 umol/L (reference range, 0-10 umol/L), the blood lipids were normal. However, on the seventh day of admission, the patient suddenly appeared motor aphasia and right limb paralysis, with right upper limb muscle strength level 1 and right lower limb muscle strength level 2. Her head CT examination indicated a large patch of decreased density, uneven density, and unclear boundary in the left frontotemporal parietal lobe, insula and basal ganglia region, with the narrowed cerebral sulcus, the compressed left ventricle, and the intracranial midline that shifted to the right. Further craniocerebral magnetic resonance imaging (MRI) examination showed large patchy, slightly longer T1 and T2 abnormal signals on the left side of both hemispheres, with high diffusion-weighted imaging (DWI) signals (Figure 2). The magnetic resonance angiography (MRA) of the head showed the left middle cerebral artery occlusion (Figure 3). Therefore, we considered that the patient had an acute left large area cerebral infarction.

Under this circumstance, we continued to improve the microcirculation, reduce brain edema, give thrombolytic therapy and gradually improve the symptoms of patients. At the time of discharge, the fibrinogen, prothrombin time, prothrombin activity level, prothrombin time, and C-reactive protein of the patients had returned to normal. D-dimer was 7.97 ug/mL, which was significantly lower than that at admission (17.49 ug/mL). Her blood routine had returned to normal. Only hemoglobin (100 g/L) and red blood cell counts (3.06×10^{12}/L) remained below normal.

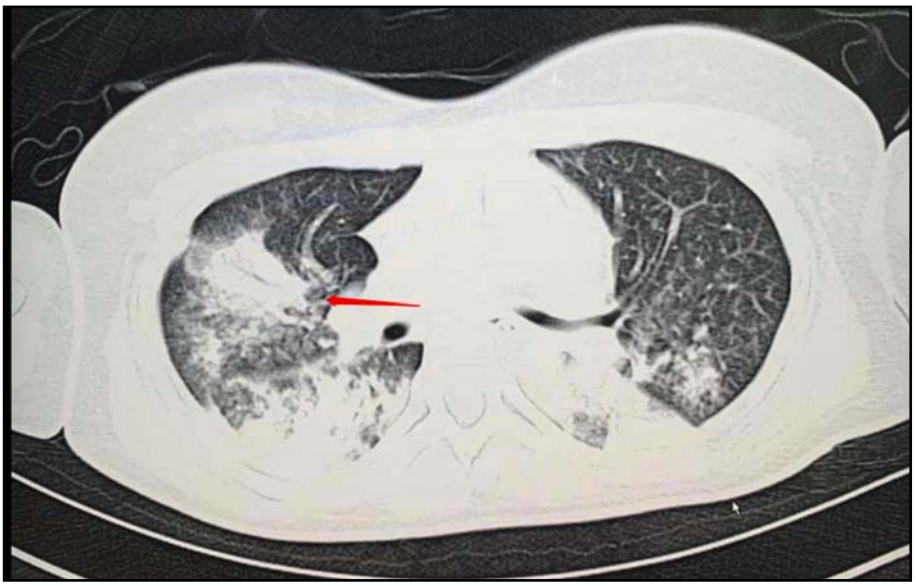

Fig. 1. The chest CT on the day of admission showed double pneumonia. CT: computed tomography

Tab. 1. The laboratory results of the patient on the day of admission

Indexes	Value	Reference values
Red blood cell count	3.96×10^{12}/L	$3.8\text{-}5.2\times10^{12}$/L
Hemoglobin	109g/L	110-150 g/L
White blood cell count	4.99×10^{9}/L	$3.5\text{-}9.5\times10^{9}$/L
Neutrophil ratio	88.81%	50-70%
Lymphocyte ratio	7.0%	20-40%
Platelet count	85×10^{9}/L	100-300 10^{9}/L
C-reactive protein	> 200 mg/L	0-10 mg/L
Procalcitonin	0.52 ng/mL	<0.046 ng/mL
Albumin	33.0 g/L	35-55 g/L
Glutamate aminotransferase	44.0 IU/L	13-35 IU/L
Creatine kinase	1061.0 IU/L	24-195 IU/L
Lactate dehydrogenase	574.0 IU/L	110-240 IU/L
Fibrinogen	4.66 g/L	2-4 g/L
D-dimer	17.49 ug/mL	0-1 ug/mL
Prothrombin activity	76.60%	75-100%
Thrombin time	13.9 s	14-21 s
Prothrombin time	14.3 s	10-14 s

One week after discharge, the patient's condition was stable, without fever or chest weakness, and she went to the rehabilitation department of our hospital for rehabilitation treatment. D-dimer was 2.21 ug/mL, procalcitonin was 0.09 ng/ml, slightly above normal, and C-reactive protein was 3.12 mg/L. After 20 days of rehabilitation, the patient was discharged from the hospital. Two weeks after discharge, the patient was hospitalized again in the rehabilitation department. At this time, D-dimer, blood coagulation and blood routine test were completely normal. She had been in rehabilitation treatment for a total of four months. She visited the superior hospital during the rehabilitation period in our hospital, and there were no abnormalities in protein S, protein C and other immune-related vasculitides. Reexamination of craniocerebral MRA indicated improvement of middle cerebral artery occlusion. When she was discharged from the hospital for the last time, she was able to walk alone, and her right limb strength was level 4. She could hold things in her right hand with a little awkward, but her speech was still a little clumsy.

DISCUSSION

Mycoplasma pneumoniae is one of the common causes of upper and lower respiratory tract infections in children (Kumar *et al.* 2019). It accounts for 35% of outpatient pneumonia cases and 3%-18% of inpatients (Mirijello *et al.* 2020; Luby, 1991). The clinical course of mycoplasma pneumoniae infection is usually mild, and pneumonia is the most prominent clinical manifestation. In 0.1% of cases of mycoplasma pneumoniae infection, several neurological diseases are reported to be extrapulmonary, including meningitis, transverse myelitis, epilepsy, acute disseminated encephalomyelitis, Guillain-Barre syndrome and stroke (Tsiodras *et al.* 2005; Daxboeck, 2006; Kang *et al.* 2016). In general, all types of neurological diseases associated with mycoplasma pneumoniae are more common in young people between the ages of 6 and 21, who are more likely to be infected with mycoplasma pneumoniae infection (Yildirim *et al.* 2020; Rodman *et al.* 2018; Inchley *et al.* 2017). However, data on mycoplasma pneumoniae-associated stroke are limited. In this report, we reported a case of 16-year-old girl with acute stroke after mycoplasma pneumoniae infection, which was demonstrated by CT, MRI and MRA of the head.

In recent years, the cause of stroke in children has been more and more recognized. Cerebral infarction in children is a relatively rare disease compared to adults. Zhao *et al.* previously reported the clinical and imaging characteristics of cerebral infarction in children (Zhao *et al.* 2019). They analyzed the etiology of 54 children with cerebral infarction and finally concluded that infection accounted for 54%, vascular disease accounted for 40%, and trauma accounted for 26%, which were the top three causes of cerebral infarction in children (Zhao *et al.* 2019). However, the relationship between mycoplasma pneumoniae infection and stroke is not completely clear, and few reports

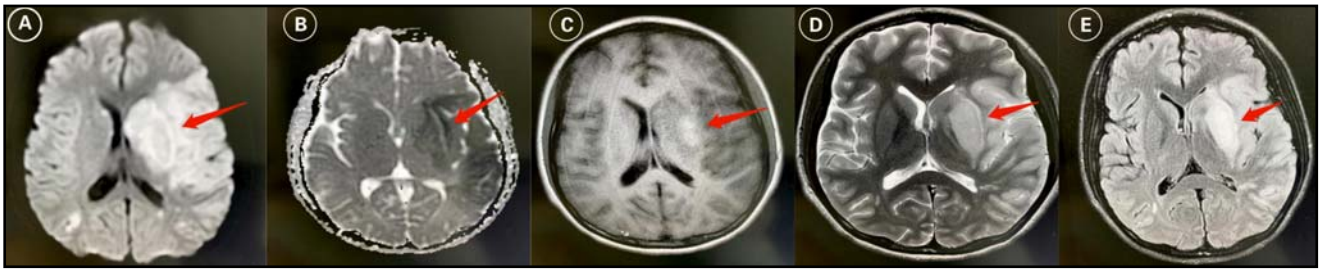

Fig. 2. The MRI of the head was performed on the 8th day of admission. Axial MRI diffract-weighted imaging showed large patchy limited dispersion in the left cerebral hemisphere in both hemispheres (A), the corresponding part shows a low apparent diffusion coefficient on the apparent diffusion map (B), and the corresponding part showed a low apparent diffusion coefficient on the apparent diffusion map, large flaky, spotted slightly longer T1 (C) and slightly longer T2 abnormal signals (D), Flair high signals (E). MRI: magnetic resonance imaging.

have been reported so far. According to the previous researches, the mechanisms of stroke associated with mycoplasma pneumoniae infection are associated with direct nervous system invasion, hypercoagulable or thrombotic states, vasculitis that may cause strokes and immune-mediated injury (Chiang *et al.* 2011; Kim *et al.* 2013; Leonardi *et al.* 2005). In our case report, the girl developed hemiplegia and motor aphasia on the basis of fever and cough respiratory tract infection. The imaging examinations confirmed cerebral infarction, and the patient's mycoplasma pneumoniae antibody test was positive. Besides, no abnormality was found in the two cardiac ultrasonography, and the blood culture tests were also negative. The patient was admitted to ICU for ventilator-assisted breathing because of pulmonary infection, so it was not suitable for angiography. But the cerebral MRI indicated that the void shadow of subequal cisterna did not disappear, which did not support moyamoya disease. In our case, although there was no evidence of direct nervous system involvement since the lumbar cerebrospinal fluid puncture was not performed at the time, the retrospective analysis showed that the fibrinogen was 4.66 g/L at admission, and the peak value of D-dimer reached 27.4 ug/mL before the onset of cerebral infarction, which was higher than normal. And C-reactive protein >200 mg/L and procalcitonin 0.52 ng/mL were significantly higher than normal. Therefore, we suspected that cerebral infarction was caused by a hypercoagulable state, thrombotic state, and vasculitis (Leonardi *et al.* 2005).

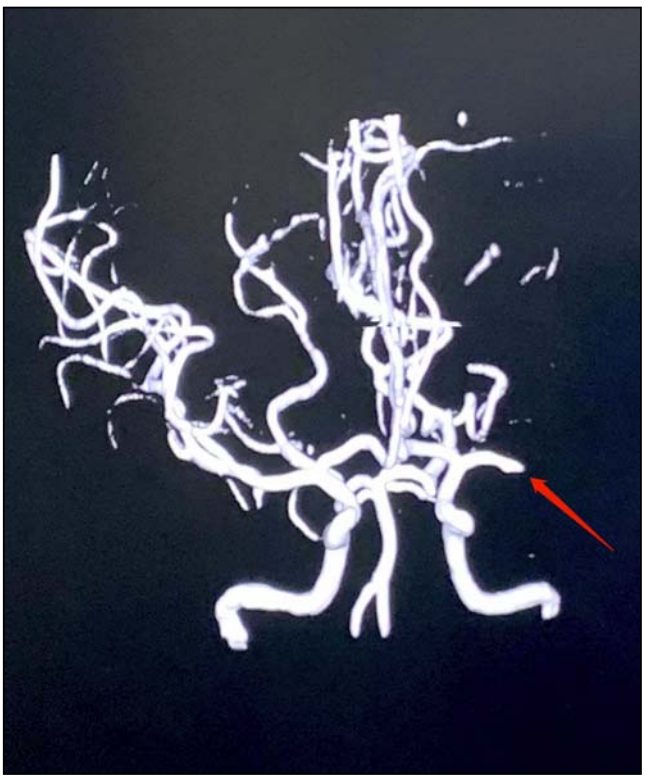

Fig. 3. The MRA of the head showed a small amount of blood flow signals passing through the proximal segment of the left middle cerebral artery, no obvious blood flow signals passing through the middle and distal segment, and no obvious signs of occlusion and stenosis were seen in the rest. MRA: magnetic resonance angiography.

Previous studies suggested that the average time from respiratory symptoms caused by mycoplasma pneumoniae infection to central nervous system symptoms was 8.8 days, and the left middle cerebral artery occlusion was the most common pathology of the central nervous system (Narita, 2009; Bitnun & Richardson, 2010). The girl we reported had a 12-day interval from cough, sputum and fever to awkward speech and poor movement on her right side. Cerebral MRI indicated multiple acute cerebral infarctions in both cerebral hemispheres, mainly distributed in the left middle cerebral artery, which was consistent with the above reports (Narita, 2009; Bitnun & Richardson, 2010). Therefore, we believe that the clumsy speech and poor right limb movement are related to mycoplasma pneumoniae infection.

There is no clear treatment plan for cerebral infarction associated with mycoplasma pneumoniae infection. Previously, Parker *et al.* reported that all children were treated with macrolide antibiotics (Parker *et al.* 1981), but Leonardi *et al.* believed that early use of macrolide antibiotics could not prevent the onset of cerebral infarction (Leonardi *et al.* 2005). In our case, cerebral infarction occurred after mycoplasma

pneumonia. Therefore, we used macrolide antibiotics early and took thrombolytic therapy in time after cerebral infarction. After active rehabilitation treatment, the prognosis of the patients was significantly improved. Therefore, we believe that for mycoplasma pneumoniae infection-related cerebral infarction, macrolide antibiotics and active thrombolysis treatment should be used as soon as possible, so as to improve the prognosis of patients.

The purpose of this report is to emphasize that infection is one of the important causes of stroke in children, especially after fever. When mycoplasma pneumoniae infection is particularly severe and difficult to treat, as clinicians, we should carefully handle the signs and symptoms of the nervous system. We should also pay attention to the examination of speech and physical movements in lethargic and lethargic patients. When neurological defects occur, we should highly suspect neurological diseases associated with mycoplasma pneumoniae. For patients with severe infection, the changes in fibrinogen and D-dimer should be closely observed. In conclusion, for patients with mycoplasma pneumoniae respiratory infection (including children), attention should be paid to the risk of cerebral ischemia and stroke in clinical treatment, especially when the infection is accompanied by elevated fibrinogen and D-dimer.

DECLARATION

Ethics approval and consent to participate

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This study is approved by relevant Ethics Committee. Written informed consent was obtained.

Consent for publication

The patient's parents have agreed to publish this case report and signed the informed consent form.

Competing interests

The authors declare no conflict of interest.

Funding

None.

Authors' contributions

Xiaoqian Jin conceived the project, carried out literature research and clinical studies, wrote the manuscript. XiuHua Zuo contributed to data acquisition and data analysis. All authors contributed to the data analysis and revised the manuscript.

Acknowledgments

None.

Availability of data and material

Data are contained within this paper.

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