Extensive and Progressive Cerebral Infarction Associated with Mycoplasma pneumoniae Infection. A Case Report and Literature Review

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Case Report

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Abstract

Backgrounds

Mycoplasma pneumoniae (MP) is one of the most common respiratory pathogens causing respiratory infection in children, especially in those above 5 years old. Although rare, cerebral infarction is the most severe neurological complication of MP infection and could be fatal.

Case presentation

Here, we report a case of extensive and progressive acute cerebral infarction associated with MP infection, which not only received medical treatment but also underwent a decompressive craniectomy. Computed tomography angiography (CTA) and magnetic resonance angiography (MRA) revealed occlusion of the left internal carotid artery, left anterior cerebral artery, and middle cerebral artery. In order to better understand the relationships between MP infection and cerebral infarction both on clinical and radiological perspectives, literature of cerebral infarction associated with MP infection were searched and reviewed.

Conclusions

Cerebral infarction is a rare complication of MP infection, which can result in neurological sequelae or even death. Clinicians should pay attention to neurological signs or symptoms after MP infection. CT or MR even CTA or MRA should be considered to make timely assessment and diagnosis, especially in severe and refractory cases.

Introduction

Mycoplasma pneumoniae (MP) is one of the most common respiratory pathogens causing respiratory infection in children, especially in those above 5 years old. While most MP infections usually present with a mild clinical course, pneumonia is the most prominent clinical manifestation in about 3–10% of affected people [1]. MP infection accounts for 10–40% of all hospitalized children with pneumonia [2] and 2–12% of adults with community-acquired pneumonia [3]. It was reported that 0.1% of MP infections and up to 7% of MP respiratory tract infections develop neurologic complications [4]. While Meningoencephalitis is the most frequently reported central nervous system (CNS) complication of MP infection, several other typical neurological disorders have also been reported, such as transverse myelitis, myeloradiculopathy, poliomyelitis-like syndrome, Guillain-Barre syndrome, cranial neuropathy, cerebellar ataxia, focal encephalitis and cerebral infarction. Among these neurological complications, cerebral infarction, although rare, is the most severe and sometimes fatal.

Herein, we report a case of extensive and progressive acute cerebral infarction associated with MP infection, which not only received medical treatment but also underwent a decompressive craniectomy. In addition, all published cerebral infarction cases associated with MP infection were searched and reviewed to help better understand the relationships between MP infection and cerebral infarction both on clinical and radiological perspectives.

Case presentation

An 8-year-old girl was transferred to our hospital due to abruptly developed right hemiparesis. She had developed symptoms of cough 7 days ago and fever 6 days ago. Her symptoms worsened, although symptomatic treatment was given at a local clinic for 3 days. Then she was admitted to the local hospital and diagnosed with pneumonia. Antibiotics (Cefotaxime and Piperacillin and Tazobactam) were prescribed from the day of admission, but her symptom did not show any improvement. On the day of her transfer, she had exhibited shortness of breath, headache, weakness in right limbs for one day and dysarthria for half a day. Past medical and family history of this patient was unremarkable.

On admission, she had a temperature of 36.8℃, respiratory rate of 44 breaths/min, heart rate of 110 beats/min, and blood pressure of 96/62 mmHg. Her oxygen saturation was 94% while receiving 4 L/min of oxygen through a nasal cannula. Her breath sounds were coarse and lowered in the right lung with inspiratory crackles auscultated on both lung fields. Her heartbeat was regular without murmurs. On neurologic examination, her pupils were isochoric with insensitive light reflexes, and her right nasolabial fold becomes shallow. Neck rigidity was not seen. She had limited movements (right upper limb muscle power, grade 2; right lower extremity muscle power, grade2; left upper limb muscle power, grade 5; and left lower extremity muscle power, grade 5). Pathologic reflex of Babinski sign was present on the right. Her previous chest CT scan conducted in local hospital showed consolidation and pleural effusion in the right lung field. Because of newly developed neurologic symptoms, a brain computed tomography (CT) was immediately conducted on admission to our hospital. Hypoattenuation in the left frontal lobe was showed (Fig. 1a) and an acute cerebral infarction was suspected. Then she was immediately transferred to the intensive care unit.

The laboratory examinations conducted on the day of admission revealed a hemoglobin level of 11.5g/dl, white blood cell (WBC) counts of 4720/μL comprising 86.2% neutrophils, and platelet count of 172,000/μL. C-reactive protein level was 130.43mg/L. Procalcitonin level was 1.71ng/mL. Her liver function tests revealed a slightly decreased albumin level of 34.7 g/L, an elevated aspartate aminotransferase (AST) level of 112 U/L, and an alanine aminotransferase (ALT) level of 39 U/L. An increase in serum lactate dehydrogenase level of 929 U/L was also observed. The coagulation tests revealed increased fibrinogen level of 5.91 g/L and fibrin degradation products (FDP) level of 17.1 mg/L, and an elevated D-dimer level of 5.11 mg/L, which changed to 5.01g/L, 67.7mg/L, 19.24mg/L respectively. Other serum biochemistry results were all within the normal limits. The ultrasonic cardiogram and carotid duplex ultrasound findings were normal, while later thrombus in the left femoral vein was found. On the day of her admission, ceftizoxime and mannitol were infused, and continuous positive airway pressure (CPAP) ventilator support was given. She had recurrent fever, and the maximum temperature was 40.3 ℃. On the 2nd day of hospitalization, she became drowsy and had anisocoria. A repeat brain CT scan was conducted and showed an increasing hypo-density lesion in the left frontal lobe (Fig. 1b). And chest CT showed complete atelectasis in the lower lobe of the right lung, segmental consolidation in the middle lobe of the right lung and scattered patchy opacities in the left lung (Fig. 1c). To avert impending herniation, she received the left frontal and parietal decompressive

craniectomy. After craniectomy, this girl underwent a CT angiography (CTA), which showed occlusion of the left internal carotid artery, left anterior cerebral artery, and middle cerebral artery (Fig. 2a).

The pathological examinations showed the serum Mycoplasma antibody level was above 1: 320 and the polymerase chain reaction (PCR) of M. pneumoniae in sputum was 1.44 x 10^8. Besides, PCR of human metapneumovirus (HMPV) was also positive in sputum. Sputum tests for other respiratory pathogens such as respiratory syncytial virus, adenovirus, influenza A and B virus, parainfluenza 1,2,3 virus, bocavirus, metapneumovirus and Bordetella pertussis were all negative. The bacteriological cultures of blood, sputum, pleural effusion and cerebrospinal fluid (CSF) were sterile. Then, she was diagnosed with severe mycoplasma pneumoniae (SMPP), and the antibiotic was changed to azithromycin for 7 days. Mannitol and glycerol fructose were administered intravenously to decrease intracranial pressure. Phenobarbital was also prescribed to prevent seizure. Levofloxacin was later added due to no improvement of body temperature. On the 8th day of her admission, she underwent a bronchoalveolar lavage. Methylprednisolone (40mg bid) was added for anti-inflammatory therapy on the 12th day of hospitalization.

On the 7th, 15th and 30th day of hospitalization, occlusion of the left internal carotid artery, left anterior cerebral artery, and middle cerebral artery still existed, proved by magnetic resonance angiography (MRA) or repeat CTA (Fig. 2b-d). On the 49th day of hospitalization, her respiratory and neurologic status improved, but right-side weakness and dysarthria still existed. For active rehabilitation physiotherapy, she was transferred to the rehabilitation department. Two months later, although dysarthria improved and aided walking was capable, she still had right side weakness and slight impairment of brain function. Three months later, Chest CT re-examination showed a significant absorption in the lesions of the right lung, and only patchy of hyper-density remained in the lower lobe of the right lung (Fig. 3a). Large patches of hypo-intensity on T1WI and hyperintensity on T2WI were seen in the left frontotemporal lobe and basal ganglia, suggesting the formation of softening foci. MRA still revealed occlusion of the left anterior cerebral artery and middle cerebral artery (Fig. 3b).

Discussion

Mycoplasma pneumoniae is a common pathogen causing community acquired pneumonia in children. Although MP infection is asymptomatic and self-limited in most cases, it may present with extrapulmonary manifestations in severe cases, in which the CNS is a common site of involvement, with the prevalence ranging from 1.0–4.8% [5]. Cerebral infarction is a rare and severe neurological manifestation of M pneumoniae infection and only a few cases have been reported.

To better understand the relationships between MP infection and cerebral infarction both on clinical and radiological perspectives, we searched PubMed for studies published in English or in Chinese until 14/02/2024, using specific search terms (Fig. 4). Reference lists of all included articles and relevant review articles were also hand-searched. The inclusion criteria of studies or case reports were as follows: (1) MP infection proven by serology, culture or PCR; (2) cerebral infarction proven by CT or magnetic resonance imaging (MRI); (3) eligible data on age, sex, neurological symptoms and follow-up. A flow diagram for studies screening, eligibility assessment and inclusion was prepared according to Preferred Reporting Items for Systematic reviews and Meta Analyses (PRISMA) guidelines (Fig. 4) [6]. A total of 34 studies met the inclusion criteria (Fig. 4) among 59 identified studies through database searching, reporting a total of 39 patients who had a cerebral infarction associated with MP infection. Demographic characteristics and clinical manifestations were summarized in Table 1. Eighteen (46%) patients were female. The median age was 8 years old (range: 3 to 43 years), and 28 (72%) patients aged less than 18 years old. The median interval between the onset of respiratory symptoms and cerebral infarction was 9 days (range: 3 days to 10 months). Limb weakness was present in 30 of 39 (77%) patients, facial paralysis in 12 of 39(31%) patients, aphasia or dysarthria in 15 of 39 (38%) patients, decreased awareness or irritability in 10 of 39 (26%) patients, and visual impairment in 3 of 39 (8%) patients. CSF analysis revealed MP PCR or/and antibody were positive in 4/24 (17%) patients. In 7 of 35 (20%) patients, prothrombin time and/or activated partial thromboplastin time prolonged, and in 17 of 35 (49%) patients, fibrinogen, fibrin degradation products and/or elevated D-dimer level increased. positive anticardiolipin IgM antibodies, antinuclear antibodies and or antiphospholipid antibodies were reported in 4 of 30 (13%) patients. On chest radiograph, consolidation was found in 19 of 35 (54%) patients, pleural effusion in 12 of 35 (34%) patients, and atelectasis in 2 of 35 (6%) patients. Brain CT or MRI were all performed in 39 (100%) patients, in which 34 (87%) patients underwent an intracranial vessels evaluation by CTA or MRA. In the total 34 cases, 25 (74%) had occlusion in the middle cerebral artery (MCA), 8 (24%) in the internal carotid artery, 6 (18%) in the vertebral artery, and 3 (9%) in the basilar artery. Twenty-nine of 34 (82%) patients had occlusion in the anterior circulation, 9 of 34 (26%) in the posterior circulation, while 4 of 34 (12%) in both the anterior and posterior circulation. During follow-up, 4/39 (10%) patients died, 20/39 (51%) patients had neurological sequelae and 15/39 (39%) patients had complete neurological recovery. Past medical history or family history of 35/39 (90%) patients were remarkable, while 2 patients had sickle cell trait, 1 patient had Steven-Johnson syndrome and 1 patient had Down syndrome.

Table 1
Characteristics of patients with cerebral infarction after MP infection in the literature and current study.

Reference	Year	Case	Age	Gender	Onset	Neurological Symptoms	CSF Analysis	Hypercoagulability	Autoimmune Antibody	Chest Ra
Mark I. Pensler et al.[18]	1980	1	21y	M	7d	right-sided hemiplegia	(-)	(-)	/	diffuse i
Mulder LJ et al.[19]	1987	1	30y	F	7d	right-sided hemiplegia, aphasia	(-)	FDP	(-)	diffuse i
Dowd AB et al.[20]	1987	1	31y	M	21d	left-sided hemiplegia	/	/	(-)	/
Pongsakdi Visudhiphan et al.[21]	1992	1	12y	F	10d	right-sided hemiplegia, right-sided facial palsy	(-)	/	(-)	consolic pleural f
Michael Fu et al.[22]	1998	1	5y	F	10d	right-sided hemiplegia, aphasia	(-)	fibrinogen, FDP, D-dimer	(-)	consolic
Claudio S. Padovan et al.[23]	2001	1	36y	F	8d	left-sided hemiplegia, left facial palsy	MP-PCR, antibody (+)	APTT, D-dimer level	antinuclear antibodies, anticardiolipin antibodies	diffuse i
Philippe Ovetchkine et al.[24]	2001	1	8y	M	/	left-sided hemiplegia	(-)	(-)	(-)	/
Charalampos Antachopoulos et al.[25]	2002	1	8y	M	14d	right-sided hemiplegia	(-)	(-)	(-)	normal
S. Sotgiu et al.[26]	2003	1	36y	F	14d	right-sided hemiplegia, aphasia, lowered consciousness	/	(-)	/	normal
Salvatore Leonardi et al.[27]	2005	2	6y/5y	M/F	3d/14d	lowered consciousness/ hypotonia and foot drop gait	MP antibody (+)/ MP antibody (+)	(-)/(-)	(-)/(-)	interstiti infiltrati
Chun-Yi Lee et al.[28]	2009	1	4y	M	9d	irritability and dysarthria	(-)	fibrinogen, D-dimer, FDP	(-)	consolic pleural f
Ju Seok Ryu et al.[29]	2009	1	13y	M	/	left-sided hemiplegia, dysarthria, visual impairment	(-)	(-)	(-)	normal
Wei Wang et al.[30]	2009	3	9y/4y/5y	F/M/F	11d/7d/6d	left-sided hemiplegia/ right-sided facial palsy/ left-sided hemiplegia, left-sided facial palsy	//(-)	PT, fibrinogen/ D-dimer↑/ D-dimer↑, PT	(-)/(-)/(-)	consolic pleural f consolic pleural e consolic
Joe Senda et al.[31]	2010	1	21y	M	/	right-sided hemiplegia, aphasia, lowered consciousness	(-)	PT, APTT, FDP, thrombin-antithrombin III-complex, D-dimer, cold agglutinin	antiphospholipid antibodies, anticardiolipin antibodies	consolic

Reference	Year	Case	Age	Gender	Onset	Neurological Symptoms	CSF Analysis	Hypercoagulability	Autoimmune Antibody	Chest Ra
Min Kong et al. [32]	2012	1	11Y	F	5d	right-sided hemiplegia, aphasia	(-)	APTT, PT, D-dimer	/	consolic pleural f
Manyong Lee et al.[33]	2013	1	33y	M	10m	quadriplegia, dysarthria	(-)	(-)	(-)	consolic necrosis fluid
Alejandro V. Garcia et al.[34]	2013	1	13y	F	6d	left-sided hemiplegia, ataxia.	/	(-)	/	bilateral consolic
Gun-Ha Kim et al.[35]	2013	1	3y	F	7d	left-sided hemiplegia and left-sided facial palsy	(-)	FDP	antinuclear antibodies	infiltrati
Piero Pavone et al.[36]	2014	1	4y	F	9d	lowered consciousness	(-)	(-)	(-)	/
Fahad A. Bashiri et al.[37]	2015	1	10y	M	14d	right-sided hemiplegia and right-sided facial palsy	/	(-)	(-)	/
Yunguang Bao et al.[38]	2016	1	8y	M	14d	visual impairment	(-)	(-)	(-)	consolic pleural f
Ben Kang et al. [39]	2016	1	5y	F	6d	left-sided hemiplegia and left-sided facial palsy	(-)	fibrinogen, D-dimer	(-)	infiltrati fluid
A. Garcia Tirado et al.[40]	2016	1	6y	M	2d	visual impairment	/	(-)	(-)	bilateral thickeni
Yu Hyeon Choi et al.[41]	2017	1	5y	M	10d	dysarthria, limited eye movement, irritability	(-)	D-dimer	(-)	consolic atelecta
Pournamy Sarathchandran et al.[42]	2018	1	39y	M	11d	right-sided hemiplegia, right-sided facial palsy, dysarthria	(-)	(-)	(-)	normal
Xingnan Jin et al.[43]	2018	1	7y	M	13d	lowered consciousness, left-sided facial palsy, aphasia	/	D-dimer	/	consolic atelecta hydroth
Mansi Oberoi et al.[44]	2021	1	37y	M	21d	right-sided hemiplegia, aphasia	/	/	/	normal
Yefeng Wang et al.[45]	2021	1	5y	M	21d	paralysis, lowered consciousness, aphasia	/	D-dimer	anticardiolipin antibody	consolic pleural f
Guodong Ding et al.[46]	2021	1	5y	F	9d	left-sided hemiplegia, dysarthria, dysphagia	MP-PCR(+)	PT, D-dimer	(-)	consolic pleural f
Ahmad J. Abdulsalam et al.[47]	2021	1	6y	F	10d	right-sided hemiplegia, right-sided facial palsy, lowered consciousness, aphasia	(-)	(-)	(-)	infiltrati

Reference	Year	Case	Age	Gender	Onset	Neurological Symptoms	CSF Analysis	Hypercoagulability	Autoimmune Antibody	Chest Ra
Sanjay K. Yadava et al. [48]	2021	1	43y	M	15d	left-sided hemiplegia	(-)	/	/	infiltrati
P Vinodhini et al.[49]	2022	1	4y	F	6d	right-sided hemiplegia, right-sided facial palsy	/	APTT	(-)	consolic pleural f
Shasthara Paneyala et al. [50]	2022	1	22y	F	5d	left-sided hemiplegia and left-sided facial palsy, lowered consciousness	/	D-dimer	/	pleural f
Chunjiao Han et al.[17]	2022	3	7y/9y/3y	M/F/M	14d/6d/5d	paralysis	/	D-dimer	/	consolic consolic consolic

Neurological complications of MP infection are divided into two subtypes according to the occurring time after the onset of respiratory symptoms, para-infectious type (less than 3 days) and post-infectious type (up to 2–3 weeks) [7]. The mean interval between the onset of respiratory symptom and neurological manifestation was 9.6 days (range: 2 to 14 days) [8]. In our case, the interval between the onset of respiratory symptoms and the development of cerebral infarction is 7 days. As shown in the literature review, the main symptoms of cerebral infarction involve hypotonia (muscle weakness), altered consciousness, and cranial nerve function impairment (facial palsy, aphasia or dysarthria, eye movement disturbance or visual impairment). In our case, the girl presented with right hemiparesis, dysarthria and facial palsy. Both CTA and MRA proved that the patient had an occlusion in the left internal carotid artery, left anterior cerebral artery, and middle cerebral artery, which are the most common location as presented in the literature review. As far as we know, this is the second case of acute cerebral infarction associated with MP infection, which not only received medical treatment but also underwent a decompressive craniectomy due to increasing intracranial hypertension. And more interestingly, in our case, PCR of human metapneumovirus (HMPV) was also positive in sputum, which indicated a mixture infection of MP and HMPV. To the best of our knowledge, this has not been described in MP associated cerebral infarction thus far. Stroke in children is rare, with an annual incidence of 2–4/100,000 in the United States [9]. Based on the research done by the International Pediatric Stroke Study (IPSS), at least 24% of cases of Acute ischemic stroke (AIS) were related to infection, which is the second most common cause of AIS [10]. The most common infectious agent is Varicella zoster virus, but other pathogens including Mycoplasma pneumoniae, Chlamydia pneumoniae, Parvovirus B19, Borrelia burgdorferi, influenza A virus, Human immunodeficiency virus, and mumps virus infection have been identified as potential risk factors for arterial ischemic stroke during childhood [11]. HMPV has not been reported yet, therefore, further investigations should be done to figure out whether virus infection like HMPV would increase the possibility of developing AIS post MP infection.

Although it still hasn't been figured out why some cases of MP infection can cause cerebral infection, several mechanisms have been postulated, which includes direct neurological invasion, hypercoagulability or thrombotic states, and vasculitis. The IgM antibody or PCR of MP in the cerebrospinal fluid in several reported cases, which were included in our literature review, have suggested the possibility that MP can directly invade the bloodstream and subsequently infect the neurological system. Direct CNS invasion of MP is usually assessed by brain tissue culture or CSF analysis of MP-specific antibodies or PCR. Hypercoagulability is another possible mechanism. It was reported that surface proteins and chemical mediators produced by MP infection were supposed to play a pivotal role in causing cerebral infarction [12]. Besides, an elevation of fibrin, D-dimer or fibrinogen were reported in 17 patients, which also could be seen in our patient. D-dimer is a specific degradation product of cross-linked fibrin that reflects blood hypercoagulability, intravascular thrombosis and secondary fibrinolysis [13]. It was reported that D dimer can be used as an indicator to evaluate the severity of MP pneumonia and could be considered as a risk factor for developing extrapulmonary complications when D dimer > 3.55 mg/L [14]. Furthermore, MP infection can cause micro vascular endothelial injury and release cytokines such as tumor necrosis factor [15] and interleukin-1 due to severe inflammation and the altered endothelial anticoagulant state [16]. Thus, a hypercoagulable state combined with impaired endothelial anticoagulant function would facilitate subsequent embolism and lead to cerebral artery occlusion. MP infection can promote the body to produce auto-antibodies such as anticardiolipin antibodies (ACA), β2-glycoprotein antibodies or lupus anticoagulant antibodies, and then form immune complexes, resulting in the injury of respiratory tract and other organs outside the lungs [17]. Therefore, vasculitis is another mechanism causing cerebral infarction.

Angiography is the gold standard for diagnosing cerebral infarction caused by cranial artery embolism. However, due to the invasiveness and potential risks of angiography, its clinical application in children is limited. CTA is a non-invasive method, and its tendency to replace DSA as the gold standard to assess cranial blood vessels is becoming increasingly apparent. For children suspected with cerebral infarction, the combination of MRI and MRA is preferred to help

identify the responsible artery, and diffusion-weighted imaging (DWI) sequence can help indicate the position of acute infarction. Cerebral infarction is prone to severe sequelae and even life threatening if not timely diagnosed and treated. Therefore, it's of vital significance to find risk factors which indicate severe extra-pulmonary complication like cerebral infarction based on clinical manifestations, biochemical examinations and chest radiography. A retrospective case-control analysis [17] performed on 48 children with MP pneumonia found that pulmonary consolidation (≥2/3 lobe), and pleural effusion were independent risk factors for embolism in children with MPP. In our literature review, 19/35 (54%) patients were found to have consolidation and 12/35 (34%) patients have pleural fluid. However, detailed information of the chest radiological manifestations of these patients were not reported, so we could not make further analysis based on the information provided.

Conclusions

To summarize, we reported a case of an extensive and progressive cerebral infarction associated with MP infection which underwent a decompressive craniectomy due to increasing intracranial hypertension. To better understand the relationships between MP infection and cerebral infarction both on clinical and radiological perspectives, literature of cerebral infarction associated with MP infection were searched and reviewed. As illustrated in our literature review, although rare, cerebral infarction can sometimes happen, and result in neurological sequelae or even death. Clinicians should pay attention to neurological signs or symptoms after MP infection. CT or MR even CTA or MRA should be considered to make timely assessment and diagnosis, especially in severe and refractory cases. Further investigations should be done to analyze risk factors which indicate severe extra-pulmonary complication like cerebral infarction based on clinical manifestations, biochemical examinations and chest radiography.

Abbreviations

MP Mycoplasma pneumoniae

CTA Computed tomography angiography

MRA Magnetic resonance angiography

CNS Central nervous system

WBC White blood cell

AST Aspartate aminotransferase

ALT Alanine aminotransferase

FDP Fibrin degradation products

CPAP Continuous positive airway pressure

PCR Polymerase chain reaction

HMPV Human metapneumovirus

CSF Cerebrospinal fluid

SMPP Severe mycoplasma pneumoniae

AIS Acute ischemic stroke

ACA Anticardiolipin antibodies ()

Declarations

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Author contributions

Shijia Ni: Conceptualization; Writing-original draft.

Siyi Che: Investigation; Software; Writing-original draft.

Jinhua Cai: Conceptualization; Methodology; Writing-review & editing.

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Availability of data and materials

The raw data supporting the conclusions of this article are available upon reasonable request.

Ethics approval and consent to participate

Informed consent was approved by the patient's parents. Ethical approval was waived by the Institutional Ethics Committee of Children's Hospital of Chongqing Medical University.

Consent for publication

Consent for publication was approved by the patient's parents.

Competing interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Figures

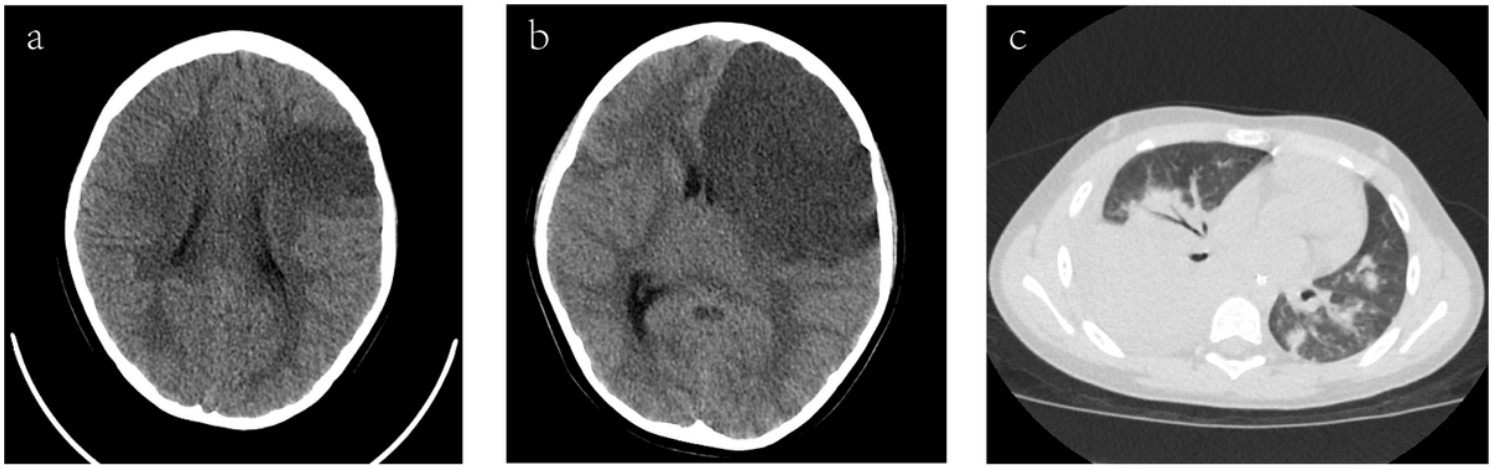


Figure 1

Emergency brain and chest CT findings after admission.

(a) The first brain CT scan immediately after admission to the hospital, showed patchy hypo-density in the left frontal lobe; (b) Re-examined brain CT 2 days after admission, showed increasing swelling of the left frontal lobe with expansion of the hypo-density opacity; (c) Chest CT showed complete atelectasis in

the lower lobe of the right lung, segmental consolidation in the middle lobe of the right lung, and scattered patchy opacities in the left lung.

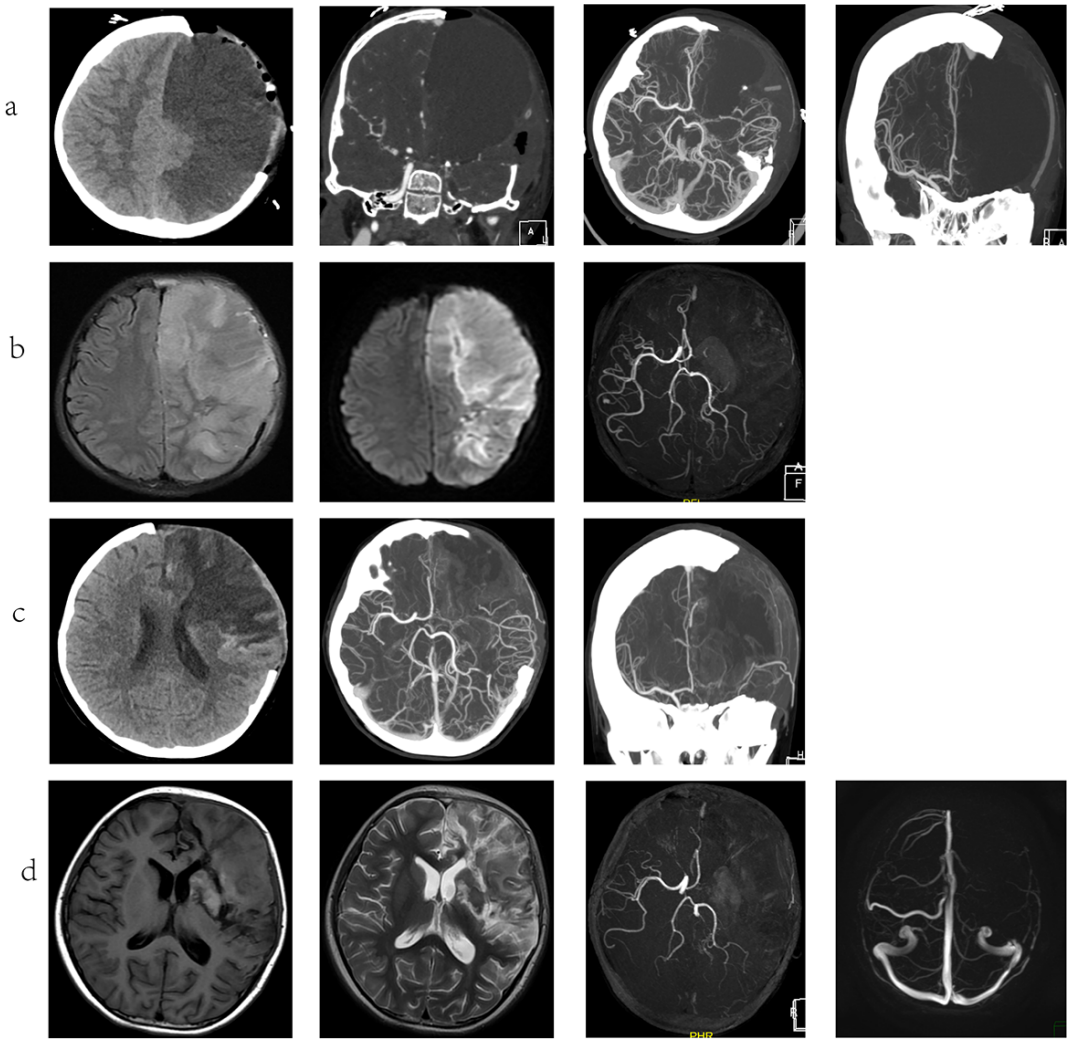


Figure 2

Computed tomography angiography (CTA) and magnetic resonance angiography (MRA) during hospitalization.

(a) On the 1st day post decompressive craniectomy, CT showed extensive hypo-density in the left cerebral hemisphere, and CTA showed occlusion of the left internal carotid artery, left anterior cerebral artery, and middle cerebral artery; (b) MRI+MRA examination on the 7th day post operation showed obvious swelling of the left cerebral hemisphere with large patches of hyperintensity on T2WI, obvious diffuse lesions on DWI, and the left anterior cerebral artery and middle cerebral artery were not visualized; (c)Cranial CTA was re-examined on the 15th day post operation showed reduced swelling of the left cerebral hemisphere and reduced extent of cerebral infarction, but the left internal carotid artery, left anterior cerebral artery, and middle cerebral artery were still not visible; (d)MRI+MRA examination on the 30th day post operation showed patchy T1WI hypo-intensity and T2WI high-intensity in the left frontotemporal lobe and left basal ganglia, slightly higher T1WI and slightly lower T2WI signal in cortex and basal ganglia, indicating partial necrosis. Left anterior cerebral artery and middle cerebral artery of MRA were still not visualized, and the left frontal and parietal vein vessels were reduced.

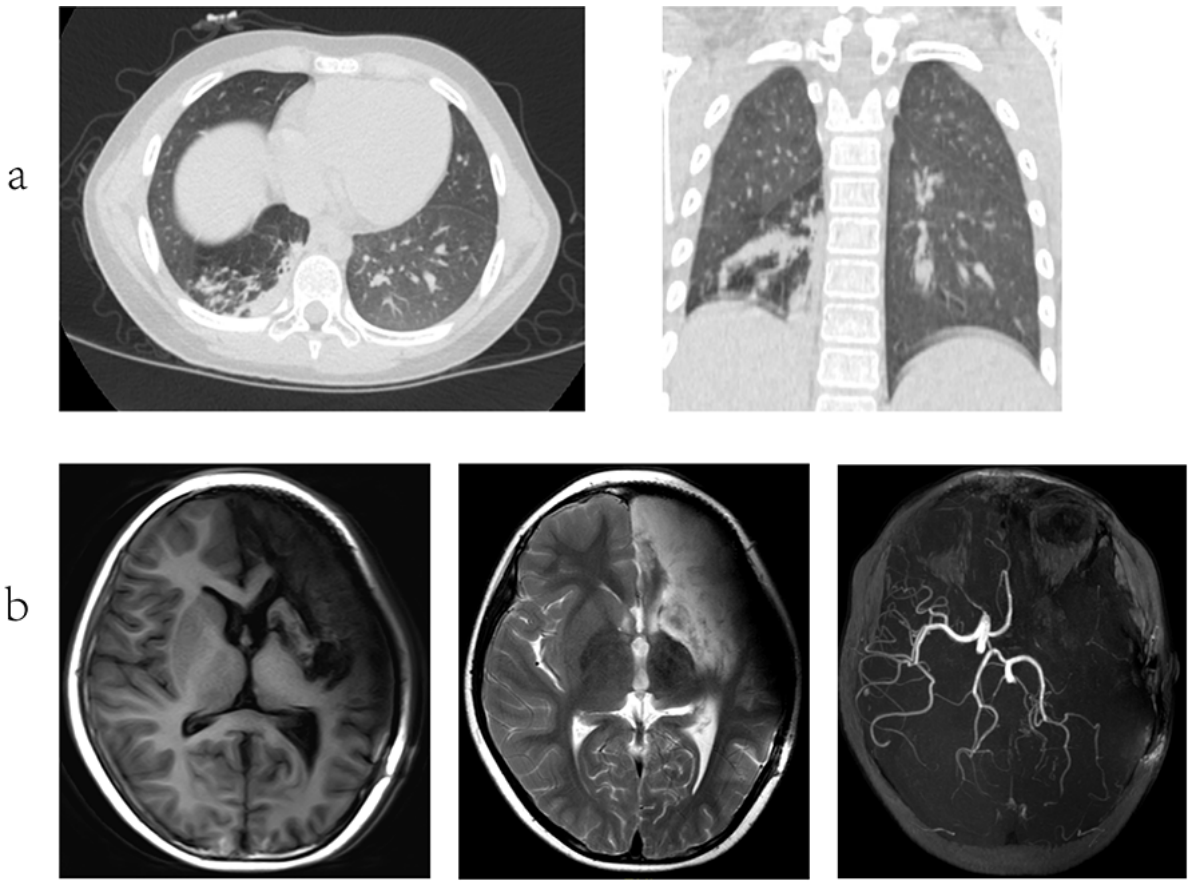
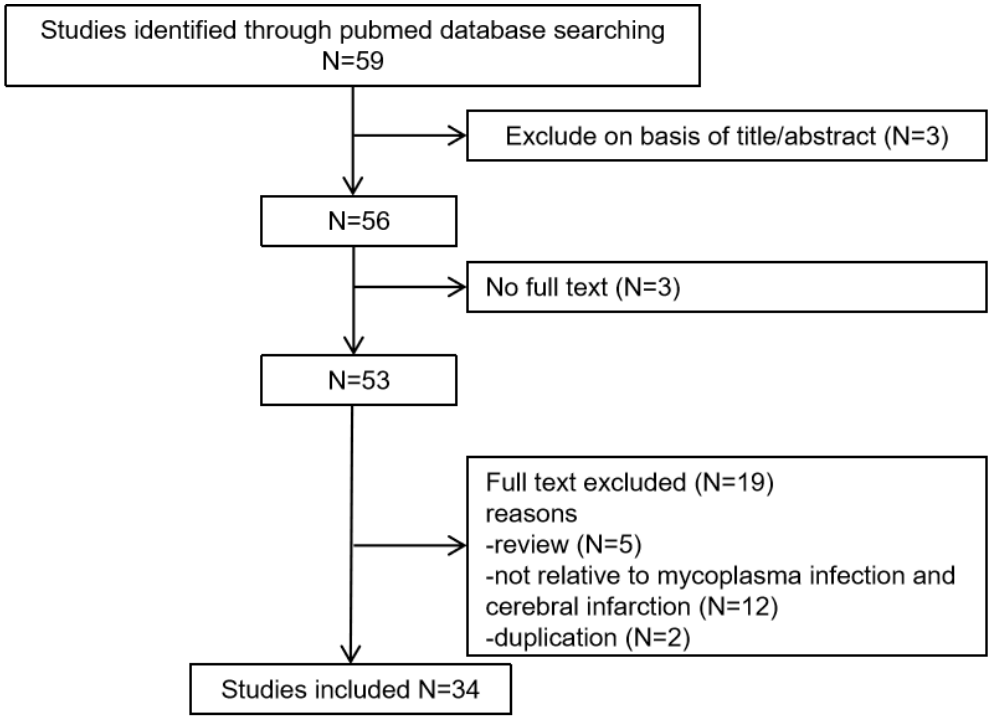


Figure 3

Chest CT and brain MRI+MRA follow-up after three month.

(a) Chest CT showed a significant reduction in the lesions of the right lung, and a small number of patchy hyper-density remained in the lower lobe of the right lung; (b)Brain MRI+MRA revealed large patches of T1WI hypo-intensity and T2WI hyper-intensity in the left frontotemporal lobe and basal ganglia, most of which were liquid signals, indicating the formation of softening foci. The left anterior cerebral artery and middle cerebral artery of MRA were still occluded.



Pubmed searching strategy
#1 (("mycoplasma pneumonia"[Title/Abstract]) OR ("mycoplasma pneumoniae"[Title/Abstract])) OR ("mycoplasma infection"[Title/Abstract])
#2 ((((("cerebral infarction"[Title/Abstract]) OR ("stroke"[Title/Abstract])) OR ("cerebral artery occlusion"[Title/Abstract])) OR ("cerebral embolism"[Title/Abstract])) OR ("cerebral thrombosis"[Title/Abstract])
#3 #1 AND#2

Figure 4

PRISMA diagram and electronic search strategy.