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Case Report

Bilateral thalamic infarcts because of the occlusion of the artery of Percheron in a patient with patent foramen ovale: report of a case and review of literature

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Abstract: Objectives: To investigate the aetiology, clinical features and imaging characteristics of bilateral thalamus infarction caused by artery of Percheron (AOP) occlusion. Methods: To summarise the aetiology, clinical manifestations, imaging, treatment and prognosis of one case and bilateral thalamus infarction caused by AOP occlusion diagnosed in our department. Relevant reports in the country and abroad in recent 5 years were reviewed, and clinical and imaging features were further discussed. Results: Many risk factors exist for cerebral vascular disease, such as patent foramen ovale. Cerebral vascular disease is caused by embolism and the onset of stroke. Thalamic stroke is clinically characterised by a wide range of alterations of consciousness varying from confusion to coma. Thalamic stroke has a typical triad of symptoms: Altered consciousness, vertical gaze palsy and memory impairment. Thalamic stroke exhibits the characteristic of image change. The clinical symptoms of patients with ischaemic cerebrovascular disease after treatment significantly improved. Conclusions: The sudden disturbance of consciousness and abnormal behaviour of patients with the exclusion of other diseases should be considered in the possibility of the disease. This condition requires early imaging examination, such as craniocerebral magnetic resonance imaging, digital subtraction angiography, cardiac ultrasound and transcranial Doppler, to confirm the diagnosis. AOP occlusion is rare. However, AOP occlusion needs these auxiliary examinations for correct diagnosis, narrow evaluation of all the possible causes and long-term anticoagulant therapy.

Keywords: Artery of Percheron, thalamus infarction, patent foramen ovale

Introduction

Acute thalamic infarction accounts for approximately 11%-14% of acute ischaemic stroke in the posterior circulation [1]. Relatively, bilateral thalamus infarction accounts for 22%-35% of thalamic infarctions [1]. Thalamic infarction caused by artery of Percheron (AOP) occlusion is a special type of bilateral thalamus infarction. AOP occlusion accounted for 0.4% of the first stroke [2]. The major causes of AOP occlusion are cardiac embolism and small blood vessel-related diseases, and the most common cause of cardiac embolism is patent foramen ovale (PFO) [3]. Digital subtraction angiography (DSA) is rare. Onset of AOP occlusion is generally sudden; this disease is complicated and dangerous and easily misdiagnosed. The details of the image should be given considerable attention to help identify the disease and diagnosis. In addition, examination of the images is beneficial to understand the epidemiology, pathogenesis, aetiology and risk factors, clinical manifestations, imaging features, treatment and prognosis. Bilateral thalamus infarction case induced by AOP occlusion in typical PFO in our department was reviewed. We also reviewed the related literature at home and abroad in the recent 5 years (**Table 1**).

Case report

A 37-year-old man was presented to the emergency department in January 20, 2016. The man was admitted to the hospital for 3 h because of speech and behavioural disorder. He had no past history of substance abuse, head injury, trauma or seizure activity, heart disease,

Table 1. Case reports in bilateral lesions of the thalamus which are typical

Information	Risk factors	Clinical manifestations	Imaging	Treatment and prognosis
5-year-old boy	Experienced an upper respiratory tract infection.	Altered personality, headache, somnolence, and agitated behavior.	Axial T2-weighted and DWI revealed hyperintense signal in both thalami.	Clinical picture resolved completely after the second week of treatment.
16-year-old boy	CNS infection	Sudden onset of drowsiness and agitation.	MR films confirmed artery of Percheron occlusion was made.	Ischaemic stroke as per standard guidelines and antiinflammatory; his level of consciousness improved, and he was vertical gaze palsy.
27-year-old an man	PFO	Deeply stuporous	MR performed occlusion of the artery of Percheron.	Medical treatment with oral anticoagulation; hyperphagia, anosognosia and emotional lability with depressive symptoms.
35-year-old woman	Pregnancy	A sudden onset and persistent loss of consciousness.	MRI showed a symmetrical and bilateral thalamic infarction without evidence of other ischemic lesions.	A short-term anticoagulant treatment followed by aspirin for long-term prevention; clinical conditions remained stable, and she did not present any neurological deficit.
38-year-old man	Unknown	Hypophonia, memory dysfunction, time disorientation and apathy.	Bilateral thalamic infarction involving the medial group of thalamic nuclei MRI.	Aspirin therapy and so on; aphasia tended to be improved whereas the hypophonia persists and remains at the same level.
49-year-old man	Diabetic	Sudden and severe dizziness and gait instability; consciousness gradually deteriorated and finally he became confused.	MRI showed that abnormal signal intensity was found in the paramedian thalami and upper brainstem.	Aspirin, simvastatin and enalapril; the patient was conscious and had grade 2 right-sided pyramidal weakness, and his left oculomotor nerve palsy was unchanged.
67-year-old woman	Age, extensive atheroma plaques.	Suddenly lost consciousness	MRI showed a recent bilateral paramedian thalamic infarction.	Aspirin therapy, intubated and so on; a persistent attention deficit but was able to live independently.
70-year-old gentleman	Age, hypertension, smoker and drank alcohol.	Reduced level of consciousness such as disorientation, confusion, hypersomnolence, deep coma.	Bilateral thalamus and mid brain infarction involving Artery of Percheron.	Ischaemic stroke as per standard guidelines; woke-up, impulsivity with low safety awareness, poor midline awareness, inability to maintain his position and unsteady with gait.
70-year-old man	Age, hypertension, type 2 diabetes mellitus, hyperlipidemia and gout.	Acute disturbance of consciousness	MRI performed and demonstrated hyperintensities in the bilateral thalami and rostral mesencephalon.	Anticoagulant therapy; consciousness gradually improved.
72-year-old woman	Age, hypertension,dyslipidemia, bilateral breast cancer, and lower limb deep vein thrombos.	Impaired consciousness and snoring	Cerebral MRI revealed recent bilateral and symmetric thalamic stroke with extension to the highest part of the cerebral peduncles, a pattern characteristic of AOP occlusion.	Aspirin therapy; persistent hypersomnia and bilateral ptosis.
83-year-old woman	Age, hypertension and hypothyroidism.	Lost her urine and bitten the tongue; bilateral mydriasis and rolling movements of the upper left limb.	MRI showed bilateral thalamic stroke with extension to the right peduncle.	Sedated, intubated, aspirin therapy; a nursing home living.

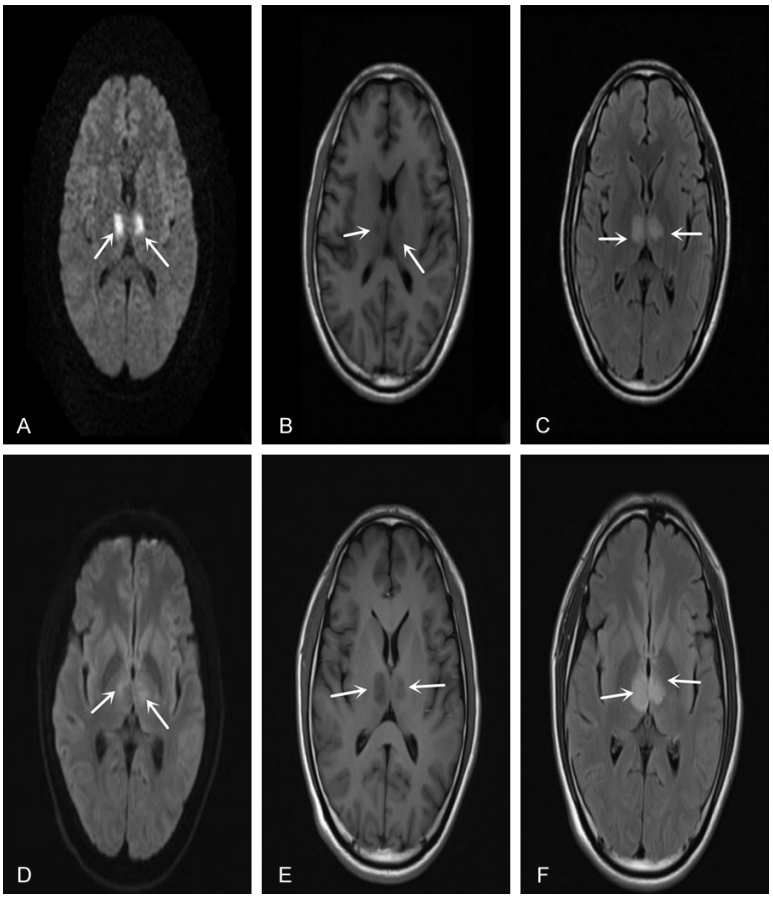

Figure 1. MRI and DWI imaging of the patient's head. A-C. Demonstrated areas of hyperintense signal in both thalamic on axial diffusion-weighted images were found in the brain. DWI showed symmetrical paramedian thalamic high signal intensity. D-F. After 1 week of treatment, the review of MRI showed that the DWI signal of bilateral thalamic lesions was reduced, which is shown by the arrow.

hypertension, diabetes mellitus, infectious disease. During examination, the patient showed the following initial vital signs: Temperature of 36.5°C, pulse of 52 beats/min, respiratory rate of 18 breaths/min and blood pressure of 109/72 mm Hg. Neurological examination showed persistent sleepiness responsive to intense vocal or painful stimuli. No sensitive or motor focal signs were detected. Deep tendon reflexes were present and symmetrical with no evident cranial nerve involvement. We were not able to establish the presence of eye movement impairment because he was not completely cooperative. However, we verified that pupils were isochoric and tended to be meiotic. Pupillary light reflex was present. The left arm and leg showed strength of four on the Medical Research Council scale. His abdominal reflexes are present, and the neck is soft. His Kerning's sign is negative. The results of the routine blood collection, comprehensive complete blood count, electrolytes, glycaemia and hepatic and renal function tests were found to be within normal limits. Urinary examination excluded illicit drug abuse. Electrocardiography and arterial blood gas analysis turned out to be normal. Computed tomography (CT) brain scan did not show any evidence of acute intracranial haemorrhage. CT brain scan showed low density shadow in the bilateral basal ganglia. The patient suffered from cerebral infarction. Magnetic resonance imaging (MRI) showed bilateral paramedian thalamic high signal intensity on T2-weighted and fluid attenuated inversio recovery (FLAIR) (**Figure 1B**, **1C**). At the same level, Diffusion-weighted imaging (DWI) and showed high signal intensity (**Figure 1A**). His blood pressure was 140 mm Hg. Cerebrospinal fluid (CSF) protein was mildly elevated at 0.63 g/L. CSF white blood cells were normal at $<1\times10^6$/L. No evidence of subarachnoid haemorrhage was observed. Biochemical, bacterial and fungal culture and acid and ink staining of CSF were normal. Magnetic resonance angiography (MRA) and magnetic resonance venography (MRV) of the head were performed. The left side of the foetal origin of the posterior cerebral artery and the right vertebral artery throughout the lumen are fine. The left transverse sinus and sigmoid sinus were compared with the small lateral sinus (**Figure 2**). The patient's electroencephalogram examination (EEG) was normal. Clearly, the patient was diagnosed with cerebral infarction. The patient was administered with antiplatelet aggregation to regulate lipid plaque, improve circulation, improve brain metabolism and for symptomatic treatment after his admission. After 1 week of treatment, the review of MRI showed that the DWI signal of bilateral thalamic lesions was re-

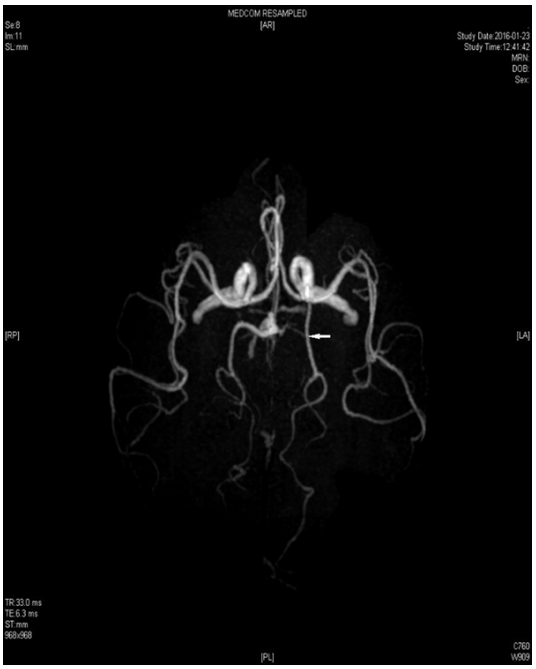

Figure 2. MRA imaging of the patient's head. The left embryonic cerebral artery is shown in the left side, and shown by the arrow.

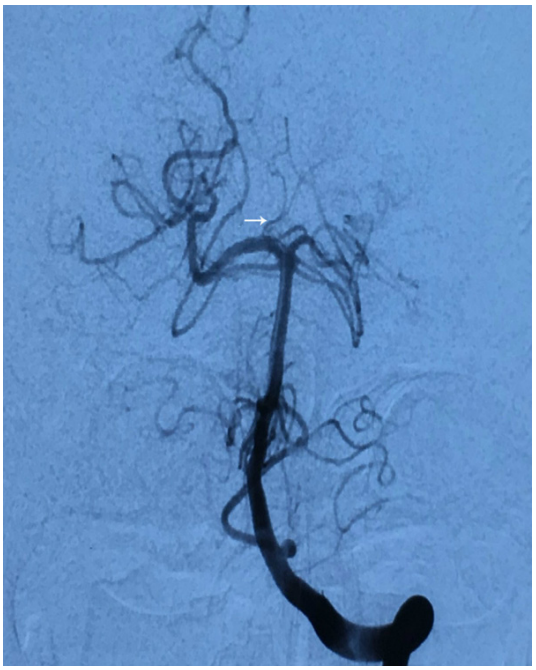

Figure 3. DSA imaging results of the patients. The Percheron artery, which is shown by the arrow, sends out the bilateral thalamus.

duced (**Figure 1D**). DSA showed that the posterior communicating artery openly supplies blood to the left posterior cerebral artery (PCA). The P1 segment of the left PCA was undeveloped. However, the distal segment of PCA is developing. A stenotic AOP originating from the right P1 segment was also visualized (**Figure 3**). To further investigate the cause of stroke in this young patient, colour Doppler ultrasound examination and transcranial Doppler were performed indicating that the patient has PFO (**Figure 4**). We consider that the PFO induced by the left to right shunt embolus induced the AOP occlusion, which caused the bilateral thalamic infarction. We recommend that the patient should be treated with cardiac surgery. However, the patient did not agree with the surgery. The patient was discharged 2 weeks later with full consciousness and verbal fluency. However, the patient's binocular vision impairment still exists. At follow-up 3 months later, he had completed the PFO occlusion. He recovered well after the operation. However, he had persistent hypersomnia and bilateral ptosis but was able to live independently.

Discussion

The patient is a young male. The patient denied that he had risk factors of cerebral vascular disease and stroke. The patient's nervous system performance showed mental confusion and verbal confusion as the first symptom. Symptom duration admission of urgent related laboratory tests suggests hypoglycaemia, hyperosmolar coma, toxic ketosis acid and CO poisoning. We combined the patient's MRI, MRA, DSA, cardiac ultrasound and transcranial Doppler (TCD) foaming test results showing that the patient has bilateral thalamus infarction. The pathogenesis for PFO induced the right to left shunt emboli causing Percheron artery occlusion.

The thalamic nuclei are composed of five major functional classes as follows: The reticular and intralaminar nuclei that take overarousal and nociception; The sensory nuclei in all major domains; The effector nuclei concerned with motor function and aspects of language; The associative nuclei that participate in high-level cognitive functions; And the limbic nuclei concerned with mood and motivation. Vascular lesions destroy these nuclei in different combinations and produce sensorimotor and behavioural syndromes depending on which nuclei are involved [4]. The thalamic arterial blood supply increases from perforated vessels with a complex distribution. The paramedian arter-

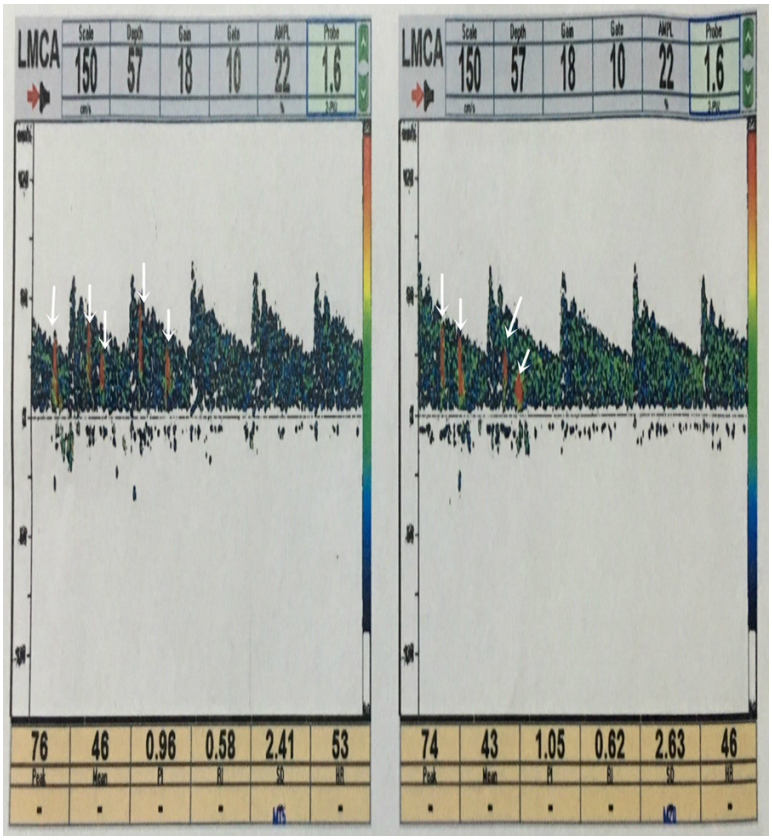

Figure 4. TCD foam experimental results. Monitoring of blood vessels: The left middle cerebral artery. Patients with calm breathing can be seen with the appearance of five micro bubbles. Valsalva action 15 s after the four micro bubbles appear.

ies provide blood supply to the paramedian thalamus, usually emerging directly from the first segment of the posterior cerebral arteries (P1 segment) on both sides. In some human brains, these conditions originate from a single pedicle known as the AOP [4]. AOP arises from the P1 section of the PCA. Variations of paramedian thalamic-mesencephalic arterial supply according to Percheron are as follows. Type A is the most common variation. Many small perforated arteries are increasing from the P1 segment of PCA. Type B: AOP is a single perforated blood vessel increasing from one P1 segment. Type C: Arcade of perforated branches increasing from artery bridging of P1 segment of both PCAs [5]. Type B is rare accounting for 22% of the variation in form as confirmed in DSA. However, this ischaemic presentation still remains uncommon. At present, a mutation rate of AOP and occlusion of the incidence of large sample epidemiological investigation were not observed [6, 7].

Aetiology and risk factors

AOP occlusion is most frequently caused by microangiopathy and cardioembolism from plaque build-up or lipohyalinosis [8, 9]. The proximity of the AOP to the basilar apex may explain the high frequency of cardioembolism as a cause of arterial occlusion, thereby resulting in bilateral thalamic infarcts. Increased occlusion risk includes systemic or internal cerebral hypertension, diabetes mellitus and atrial fibrillation. Build-up of microatheromatous plaques near the mouth of the paramedian artery can contribute to microangiopathy [3, 10], and thus, AOP occlusion [11]. Individual reports show that small artery disease is the most common cause of arterial occlusion leading to bilateral thalamic infarcts [12]. In this case, the patient is a young man with previous arterial occlusion. According to the results of the auxiliary examination, the patient was suffering from PFO. The PFO disease occurs when a small cardiogenic emboli after blockage causes blood to clot along the arterial wall, thereby resulting in an infarct. PFO is a cardiac septal legacy of tiny abnormal channel and the risk of stroke. The unexplained ischaemic stroke risk factors will increase the cryptogenic stroke risk; 10%-35% of the general population underwent transthoracic echocardiography [13]. Necessary imaging examination, such as TCD foaming experiments and transthoracic echocardiography, can improve the positive rate of detection of PFO disease and treatment of cryptogenic stroke and prevention to provide important information. AOP undergoes DSA. The aetiology of the basis of cardiogenic embolism is PFO, which is scheduled for PFO percutaneous transcatheter closure to reduce stroke recurrence rate and complication occurrence rate of risk. Therefore, improving the relevant auxiliary examination and actively determining the cause are very important. Moreover,

the best treatment and preventive measures should be developed to reduce the disability and recurrence rates of the disease.

Clinical presentation

The clinical manifestations of Percheron arterial occlusion are complex and diverse. The incidence is easy to be misdiagnosed as other diseases, such as metabolic encephalopathy, toxic encephalopathy, cerebral deep vein thrombosis, intracranial infection and intracranial tumours [14]. AOP embolism causes bilateral thalamic ischaemic stroke that has four characteristics [2]: 1. In acute onset, the level of consciousness changes with the initial symptoms, which can include coma or drowsiness for several hours or days after the gradual improvement of consciousness, sleep quality and indifference [15]; 2. Some patients manifest abnormal mental behaviour, and were sent to a hospital for treatment [16]. Symptoms, such as decline in memory, can be gradually eased after conscious state is changed because of the ascending reticular activation system of the thalamus and the nucleus of the thalamus [17]; 3. Some patients have ocular movement disorder and clinical symptoms of pupillary abnormalities of the cranial nerve damage considering the involvement of the midbrain periaqueductal gray [18]; 4. Other common symptoms may lead to language dysfunction, such as a reduction in spontaneous speech, low tone, constitutive sound difficulty and difficulty in uttering names. This condition is related to the involvement of the ventral anterior nucleus of thalamus [19]. A report [11] showed that one patient with a particular form of onset showed twitching of limbs and other similar epileptic seizures. Physicians considered this condition as paramedian bilateral thalamus infarction. Cases of limb movement disorder of individuals have been reported in the literature; possible lesions that involve the end of midbrain by cone beam CT are considered [12]. The patients underwent DSA examination, thereby confirming that stenotic AOP originated from the right P1 segment (as visualized). The P1 segment also issued small branches to supply blood to the bilateral and central thalamus. The occlusion resulted in bilateral thalamus infarction, thalamic reticular activation system injury and kernel anomaly caused by disturbance of consciousness and behaviour. Binocular visual difficulties exist in patients simultaneously, considering the involvement of the midbrain. This condition is probably due to illness of foci in patients with cranial nerve damage, as shown by MRI results.

Imaging and diagnostics

The disease is difficult to diagnose by simply relying on clinical manifestations; Thus, the diagnosis should be combined with neuroimaging examination before analysis [2]. Brain CT scan, especially high-resolution CT scanning, is the easiest and most economical way to determine suspected thalamus infarction. However, this method needs to be visible 24 h after the onset of the disease. The brain CT scan of this patient shows bilateral thalamic low density dot, which may be AOP lesions. However, the positive rate of CT scan is very low and easily leads to misdiagnosis or delays diagnosis. Brain MRI + DWI examination helps in the early diagnosis of the disease. The brain MRI + DWI examinations of this disease showed abnormal signal next to the bilateral thalamus middle zone, long elongated oval T1 and T2 signals; FLAIR + DWI showed corresponding round high signal image [16]. The result of brain MRI examination of our patient is similar to the above findings. After 10 days of treatment, brain MRI showed DWI signal reduction in bilateral thalamic lesions. Lazzaro et al. [20] reported that the brain “V word sign” is significantly important for AOP occlusion diagnosis. Thus, to improve brain MRI especially DWI for the timely detection of the disease is necessary. AOP occlusion can be determined by brain MRI examination. However, AOP occlusion is difficult to be diagnosed because the AOP is relatively small. The occlusion of AOP may be considered for arterial-to-arterial embolization or carrier atherosclerotic occlusion. Diagnosis of the occlusion of AOP is important to clarify the intracranial stenosis. DSA is a “gold standard” to check the blood vessels, thereby confirming the presence of AOP. The condition of our patient improved after DSA examination, revealing that AOP originated from the P1 segment of the right posterior cerebral artery and sends out a branch supplying to the central portion beside the thalamus, as confirmed by some domestic foreign literature reports in recent years [10]. Weidauer [21] first reported the presence of AOP by DSA examination. However, only a few studies have reported this method. The following reason may explain why this method is not

popular. AOPs are small and many, and awareness of AOP is insufficient. DSA is invasive and costly. Most patients refuse to avail of this examination. Doctors lack the understanding of DSA. Hospital medical equipment is not complete, and doctors need to improve imaging techniques.

Differential diagnosis

Clinically, the disease is similar to a variety of other diseases. Timely identification of bilateral thalamic lesions is needed to avoid misdiagnosis and delayed treatment. The major diseases identified include: 1. Vascular diseases, such as the basilar artery syndrome. The infarction area includes the hypothalamus, midbrain, upper pons, cerebellum, medial temporal lobe side, which was dominated by superior cerebellar artery, and the posterior cerebral artery. Ischaemic infarction manifestations and radiographic signs can confirm the diagnosis [3]. Deep vein thrombosis mainly occurs in adult women, especially during postpartum depression, pregnancy, oral contraceptives or coagulation dysfunction. Most patients exhibited headache, typically characterized by bilateral hypothalamic lesions involving the basal ganglia. Brain CT scan shows abnormal high density in vein; MRT1 prompted high signal sinus, MRV or DSA can show the diseased vein [8]. The brain CT, MRV and DSA findings in this case do not have the above imaging features; 2. Toxic encephalopathy caused by formaldehyde, alcohol, carbon monoxide, ammonia, cyanide and other drug abuse may also be associated with the acute onset of consciousness. These patients have a history of exposure or corresponding medical history. Poison can be screened out from the patient by routine laboratory tests. Imaging is mainly involved in basal ganglia [22]. The patient denied exposure to the above-mentioned poisons. Laboratory tests showed no evidence of poisoning. Thus, such diseases can be excluded; 3. Infectious diseases, such as viral meningitis encephalitis (such as encephalitis encephalitis) and toxoplasmosis, may be involved [3]. The disease may be involved in the bilateral thalamus and the tissue surrounding hypothalamus. Patients showed a history of infection; The CSF, EEG, brain biopsy and other related examinations can be used to diagnose the disease [4]. In the present case, the patient has no history of infection. Cerebrospinal fluid examinations showed slightly increased protein content, and pressure, cell count, sugar and chloride were normal. In addition, no signs of infection, such as diseases, can be identified; 4. Lastly, metabolic diseases, such as hypothyroidism, Fahr disease, osmotic demyelination disease, Wilson's disease, hepatic encephalopathy, vitamin B1 deficiency and encephalopathy, also exist [23]. Wernicke encephalopathy is a major cause of vitamin B1 deficiency, secondary to chronic alcoholism, gastrointestinal and haematological malignancies, dialysis and vomiting during pregnancy. The typical clinical manifestations are ataxia, altered consciousness and eye movement abnormalities, but these clinical manifestations have variability [24]. Diagnosis of these diseases has the following indicators: thyroid dysfunction, cirrhosis, and alcohol addiction, combined with patient laboratory tests of thyroid hormone, calcium-phosphate, serum copper and ceruloplasmin, vitamin levels, as well as the typical imaging findings [11]. In this case, the patient denied a history of the above-mentioned diseases. Improving the relevant laboratory tests on admission showed no obvious abnormalities. Thus, such diseases were excluded.

Treatment and prognosis

Bilateral thalamic infarcts is a special case of ischaemic stroke disease; If the patient's admission time is in the intravenous thrombolysis time window, you can actively use intravenous thrombolysis after discharge of thrombolytic contraindications [24]. Kostanian et al. [25] have reported one case of acute ischaemic stroke patient who have good neurological function after intravenous thrombolytic therapy. Patients who do not comply with intravenous thrombolysis may take measures in accordance with acute ischaemic cerebrovascular disease treatment guidelines, such as antiplatelet aggregation, lipid plaque stabilization, improved circulation, removing of free radicals and protection of the brain. The patients cannot give a specific time of onset on admission, and cannot accurately determine whether in intravenous thrombolysis time window or not. Additionally, early brain CT did not clearly show lesions because of the lack of understanding of the disease. Doctors did not administer thrombolysis therapy. After administration of ischaemic cerebrovascular disease treatment, the patient's condition improved. To find the cause of

the disease for the prevention of recurrent stroke is critical. Clinically, AOP occlusion leading to bilateral thalamus infarction caused by embolism, which is from PFO, is rarely reported. The patients' disease was confirmed to be caused by PFO through improving the relevant auxiliary examination. Clinicians should strengthen the understanding of the disease, which should be considered when patients show similar clinical manifestations of the disease. They should also promptly improve relevant auxiliary examination by ruling out other diseases to further clarify the diagnosis. During the disease treatment time, doctors should actively look for the cause, especially for young patients with no obvious risk factors for cerebrovascular disease, to prevent stroke recurrence. The majority of patients obtained good clinical prognosis after active treatment.

Disclosure of conflict of interest

None.

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