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Clinical Case Report

A case presentation of a patient diagnosed with acute cerebral infarction and myasthenia gravis

Wang Zhongyu[1], Zhang Sumei[2,*], and Bai Jiangyan[2]

[1] Chongqing Medical University, Chongqing 400016, China
[2] Department of Neurology, Taiyuan Central Hospital, Shanxi Province, 030009, China

*Correspondence: fenfan8536@163.com

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Abstract

A 78-year-old male patient presented to Taiyuan Central Hospital with complaints of speech impairment, intermittent right eyelid drooping, and occasional diplopia. The patient, who had semantic dementia, exhibited progressively worsening symptoms. He had a medical history of coronary heart disease and hypertension. Physical examination revealed impaired short-term memory and arithmetic ability, ptosis of the right upper eyelid severe enough to cover the cornea, restricted outward movement of the left eye, and decreased bilateral knee and ankle reflexes. No other abnormalities were detected. Magnetic resonance imaging (MRI) showed cerebral infarction in the right temporal lobe. Based on these findings, differential diagnoses included myasthenia gravis and paraneoplastic neurological syndromes. Repetitive nerve stimulation and neostigmine tests supported the diagnosis of myasthenia gravis. Clinically, the coexistence of acute cerebral infarction and myasthenia gravis is rare. Due to the overlap of symptoms, the diagnosis requires a comprehensive evaluation of relevant symptoms and tests.

Keywords: Acute cerebral infarction, myasthenia gravis, coronary artery disease, hypertension, autoimmune neuromuscular disease

1. Introduction

Acute cerebral infarction is a severe neurological condition characterized by necrosis of brain tissue due to hypoxia and ischemic processes following a sudden interruption of blood flow to the brain. Cerebral infarctions can result from large artery occlusions or small vessel diseases. Global stroke statistics indicate that cerebral infarctions account for 85% of all stroke cases, with a significant proportion of these patients developing permanent neurological damage

The most common risk factors for acute cerebral infarction include hypertension, coronary artery disease, diabetes, hyperlipidemia, smoking and alcohol consumption, obesity, and a sedentary lifestyle. Additionally, stroke due to cardiac embolism is frequently observed, particularly in patients with atrial fibrillation. Clinical manifestations vary depending on the location of the occluded vessel and generally present as focal neurological deficits. For instance, middle cerebral artery (MCA) occlusion leads to motor and speech impairments, whereas posterior cerebral artery (PCA) occlusion results in visual field loss and cognitive changes.

Recent studies suggest that autoimmune processes, vascular dysfunction, inflammation, and blood-brain barrier interactions may play a role in the link between myasthenia gravis and acute cerebral infarction. Myasthenia gravis is an acquired autoimmune neuromuscular disease that arises due to impaired nerve transmission at the neuromuscular junction. In this disorder, autoantibodies targeting postsynaptic acetylcholine

receptors (AChR) or key synaptic proteins such as muscle specific kinase (MuSK) lead to impaired muscle contraction. Myasthenia gravis has a variable course and is characterized by ptosis and diplopia in ocular muscles, dysphagia, dysarthria, dyspnea in bulbar muscles, and limb muscle weakness (Gwathney & Burns, 2015).

Pathophysiologically, while the neuromuscular transmission defect in myasthenia gravis results in muscle weakness and fatigue, acute cerebral infarction leads to motor dysfunction due to cerebral hypoxia and neuronal impairment. Interestingly, recent studies suggest that autoimmune processes may influence vascular functions and cerebral circulation, thereby increasing the risk of cerebral infarction in myasthenia gravis patients. Autoimmune processes are believed to have inflammatory effects on the vascular system. Systemic inflammation can lead to vascular endothelial damage and increased vascular permeability. Consequently, vascular dysfunction and atherosclerotic processes may accelerate, increasing the risk of cerebrovascular events. Additionally, the presence of antiphospholipid antibodies in some myasthenia gravis patients may contribute to a hypercoagulable state, further elevating the risk of cerebral infarction.

Cardiovascular risk factors are frequently observed in myasthenia gravis patients. Conditions such as hypertension, hyperlipidemia, and atherosclerosis are common risk factors for both myasthenia gravis and acute cerebral infarction, potentially increasing the likelihood of their coexistence, particularly in elderly patients.

In the advanced stages of myasthenia gravis, respiratory muscle weakness can lead to hypoxia. Hypoxia may alter cerebral blood flow and impose stress on the cerebrovascular system. This can disrupt the function of cerebral blood vessels, particularly in severe myasthenia gravis cases, thereby increasing the risk of ischemic stroke. Certain medications used in myasthenia gravis treatment have also been associated with cerebrovascular events. For example, long-term corticosteroid use can induce hypertension, hyperglycemia, and dyslipidemia, all of which may trigger cerebrovascular complications. Similarly, immunosuppressive therapies can increase the risk of systemic infections, further influencing vascular dysfunction mechanisms.

Paraneoplastic neurological syndromes (PNS) should also be considered in the differential diagnosis, as certain malignancies can present with myasthenia gravis like symptoms (Sasi et al., 2022). PNS is a group of disorders resulting from an autoimmune response triggered by the immune system's reaction to cancer cells, causing damage to the nervous system. Notably, autoimmune processes may compromise the blood-brain barrier in cases of myasthenia gravis associated with thymoma, potentially triggering cerebral vascular inflammation and thrombosis. Paraneoplastic antibody tests are crucial for diagnosis. Additionally, imaging modalities such as chest CT and abdominal ultrasound should be utilized to rule out other malignancy sources. Due to the rarity of the coexistence of myasthenia gravis and cerebral infarction, a comprehensive neurological and immunological evaluation is essential in the diagnostic process.

Myasthenia gravis and acute cerebral infarction are distinct diseases with different pathophysiological mechanisms. Myasthenia gravis is an autoimmune disease that disrupts neuromuscular transmission, whereas acute cerebral infarction is an ischemic condition resulting from vascular occlusion in the brain. The simultaneous occurrence of these two diseases is extremely rare, and no definitive statistical prevalence data are available on this association.

The potential risk of cerebral infarction in myasthenia gravis patients should be carefully considered. Regular screening and vascular protection strategies should be implemented for myasthenia gravis patients. In those with a history of cerebrovascular disease or risk factors, early diagnosis through neurological examination and imaging methods is crucial. Although the coexistence of myasthenia gravis and acute cerebral infarction is rare, a multidisciplinary approach is essential when these two conditions occur together.

2. Case presentation

2.1 Medical history

A 78-year-old male patient presented with a complaint of sudden-onset speech disorder on July 15, 2024. The patient showed symptoms such as difficulty finding words during speech, impaired fluency in sentence formation, and sometimes using meaningless words. The patient, who was previously diagnosed with semantic dementia, was reported to

have cognitive disorders that had been stable until recently, but had become more pronounced in recent weeks. He also had complaints of drooping right eyelid (ptosis) and occasional double vision (diplopia). There was no history of difficulty swallowing, upper or lower extremity weakness, sensory loss, dizziness, loss of consciousness, or seizures. The patient was able to control his urine and stool. He had no previous diagnosis of myasthenia gravis or any other neuromuscular disease.

The patient's medical history included coronary artery disease and hypertension. He was treated for coronary artery disease four years ago and has been taking aspirin, atorvastatin, bisoprolol, diltiazem and isosorbide mononitrate since then. He is receiving antihypertensive treatment due to hypertension and his highest blood pressure measurement was recorded as 160/110 mmHg. It was known that the patient was allergic to penicillin.

2.2 Physical Examination

Table 1: The content of physical examination

Vital signs	Body temperature	36.5℃
	Pulse	73 beats/minute (rhythmic)
	Respiratory rate	20/minute
	Blood pressure	134/78 mmHg in the right arm, 128/85 mmHg in the left arm
General status	Conscious, cooperative, oriented but there is comprehension disorder and difficulty in choosing words during speech. Sentences may become meaningless.	
Cranial nerve examination	Eyelid significant ptosis in the right eyelid, severe drooping to the point of completely covering the cornea	
	Eye movements	Eyelid significant ptosis in the right eyelid, severe drooping to the point of completely covering the cornea
	Pupillary reflexes	Pupils are equal, respond normally to light and near
	Conjugation disorder	None
	Double vision	Occasional double vision is described, but there is no loss of fixed fixation
	Facial muscles	No facial asymmetry, frontal muscle functions are preserved
	Dysarthria	Mild moderate, impaired sentence fluency
	Tongue movement	Normal
	Swallowing function	No dysphagia
Cognitive function	Memory	Significant short term memory impairment
	Arithmetic ability	Significant slowing down and errors in simple calculations
	Insight and reasoning	Weak, difficulty understanding abstract concepts
	Orientation	Time and space orientation preserved
Muscle strength and motor system	Muscle strength in upper and lower extremities 5/5 (normal) No movement limitation or asymmetric motor loss Babinski and Chaddock reflexes: negative	
Reflexes	Knee reflex	Bilaterally decreased (hyporeflexia)
	Ankle reflex	Bilaterally decreased (hyporeflexia)
	Pathological reflex	None
	Deep tendon reflexes	Mild loss of vibration perception
Sensory examination	vibration perception	Mild loss
	Pain and temperature sensation	Preserved
	Deep sensation	Preserved
Coordination examination	Gait and coordination	No wide-based gait or imbalance
	Romberg test	Negative
	Finger-toe and heel-knee tests	Normal

2.3 Past medical history

The patient had been followed up for coronary artery disease (CAD) and hypertension (HT) for the last four years. Although the degree of hypertension varied, the highest measured blood pressure was recorded as 160/110 mmHg, classifying the patient as stage 2 hypertension. Although there was no clear information on whether the patient's hypertension was under control, it was assessed that he was at high risk for cerebrovascular disease.

There was no specific data on whether cardiac symptoms (angina, exertional dyspnea, etc.) had progressed due to coronary artery disease. However, the patient's current diagnosis of myasthenia gravis and the presence of cardiovascular risk factors that may be associated with stroke are important in understanding the development of cerebrovascular events.

The patient's long term use of aspirin (100 mg/day) and atorvastatin (10 mg/day) indicates that he was receiving treatment aimed at preventing thromboembolic events. However, additional use of bisoprolol (beta blocker), diltiazem (calcium channel blocker) and isosorbide mononitrate (nitrous vasodilator) are important treatments for the management of coronary artery disease and possible cardiac ischemia.

It is also known that the patient is allergic to penicillin. However, the details of the patient's penicillin allergy (anaphylaxis, skin rash, angioedema, etc.) are not specified. Such allergic reactions may be important, because immunosuppressive treatments or alternative antibiotic use in cases of infection may be required in patients with myasthenia gravis.

There is no information about the patient's history of hyperlipidemia or diabetes. Still, these factors should also be evaluated for stroke in an individual with comorbidities such as CAD and HT. The use of atorvastatin in hyperlipidemia supports this possibility, but evaluating lipid profile and HbA1c levels is important for clinical management.

This medical history requires a multidisciplinary approach to the patient for acute cerebral infarction and myasthenia gravis. In particular, stabilizing cardiovascular risk factors and managing neuromuscular complications associated with myasthenia gravis should be carefully planned.

3. Diagnosis and treatment plan

The patient's current clinical condition indicates that he has significant risk factors for hypertension, coronary artery disease, and cerebrovascular disease. Brain magnetic resonance imaging (MRI) confirmed acute cerebral infarction in the right temporal lobe (see **Fig. 1**). However, the right eyelid ptosis, left eye outward movement restriction, speech disorder, and dysphagia detected in the neurological examination cannot be fully explained by cerebral infarction alone. This situation has raised the possibility of myasthenia gravis, one of the neuromuscular junction pathologies.

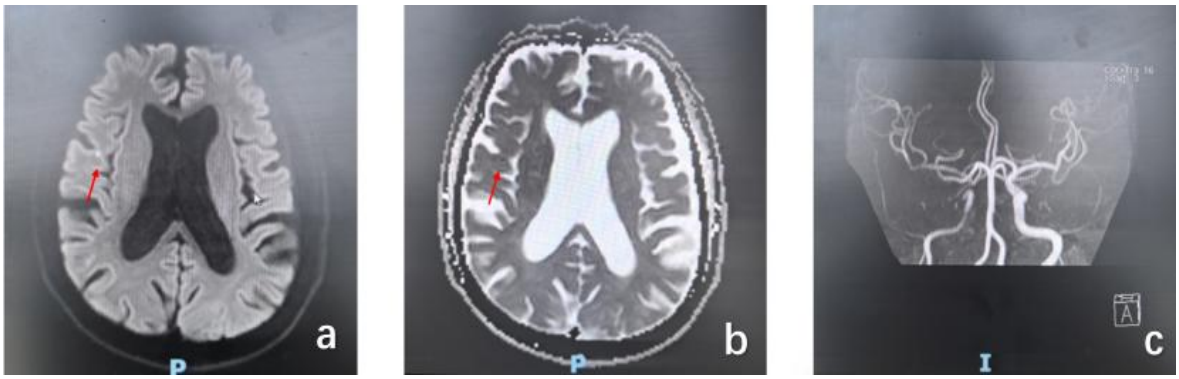

Figure 1. (a) DWI shows a small punctate lesion in the right temporal lobe. (b) ADC value decreases. (c) MRA shows intracranial arteriosclerosis; The intracranial segment of the right internal carotid artery and the middle cerebral artery appear shallower.

3.1 Diagnostic findings

Table 2: Diagnosis of acute cerebral infarction

Brain MRI (Diffusion Weighted Imaging - DWI)	Acute ischemic lesion was detected in the right temporal lobe
MR Angiography (MRA)	No intracranial large vessel occlusion was observed
Carotid Doppler Ultrasonography	Significant atherosclerotic plaques were detected in the carotid arteries

Table 3: Myasthenia gravis diagnosis

Serological Tests	Acetylcholine receptor antibodies (AChR-Ab)	Positive
	Muscle specific kinase (MuSK-Ab)	Negative
Electrophysiological Tests	Repetitive nerve stimulation test	Positive (Fatigue monitoring in muscle response)
	Single fiber electromyography (SF-EMG)	Increased jitter was detected
Neostigmine Test	Significant improvement in ptosis and muscle strength was observed after application	
Thymoma Scan Chest Computed Tomography (CT)	No mediastinal mass was detected	

3.2 Cardiovascular findings

Table 4: Heart and blood vessels.

Coronary angiography	50% stenosis was detected in the left anterior descending artery and 40% stenosis was detected in the right coronary artery
Echocardiography	Aortic stenosis (mild-moderate), ejection fraction 50%, left ventricular hypertrophy were observed
Blood Pressure	Blood pressure measurements: 140/85 mmHg (controlled), but occasionally increases to 160/100 mmHg
Lipid Profile	LDL 110 mg/dL, HDL 45 mg/dL, Triglyceride 180 mg/dL
Hyperhomocysteinemia	Homocysteine level 16 µmol/L (Reference range <10 µmol/L)

Table 5: Definitive diagnoses

Neurological system	1. Acute cerebral infarction (right temporal lobe)
	2. Myasthenia gravis (AChR-antibody positive, ocular and bulbar involvement form)
Cardiovascular system	3. Coronary artery disease (moderate atherosclerosis, compatible with angina pectoris)
	4. NYHA Class III Heart Failure (associated with left ventricular dysfunction)
	5. Aortic stenosis (mild moderate, does not require surgery as long as monitored)
	6. Stage 3 hypertension (very high risk category)
	7. Carotid artery atherosclerosis (compatible with moderate stenosis)
	8. Hyperhomocysteinemia (cerebrovascular risk increasing factor)

3.3 Treatment plan

Table 6: Treatment point

Basic Goals	Prevent ischemic events Slow the progression of atherosclerosis Improve cerebral perfusion
A. Antiplatelet Therapy	Aspirin 100 mg/day (If there is no contraindication, continue for life) Clopidogrel 75 mg/day (Dual antiplatelet therapy [DAPT] should be continued for at least 3 months, then re evaluated) If the risk of bleeding is high, aspirin monotherapy can be switched after 3 months
B. Lipid Lowering Therapy	Rosuvastatin 20 mg/day (Increase from the current dose of 10 mg to 20 mg, adjust to LDL target <70 mg/dL) Creatine kinase (CK) and liver enzymes should be monitored regularly If the patient experiences statin-induced muscle pain (myopathy), ezetimibe (10 mg/day) may be considered additionally
C. Blood Pressure Control (Target: <130/80 mmHg)	Losartan 50 mg/day (ARB, preferred over ACE inhibitors to reduce the risk of cerebral infarction) Amlodipine 5 mg/day (Calcium channel blocker, effective in preventing recurrent stroke) Spironolactone 25 mg/day (Can be added if resistant hypertension or heart failure is present) Indapamide 1.5 mg/day (Mild diuretic preferred in elderly hypertensive patients)
D. Management of Hyperhomocysteinemia	Vitamin B12 1000 mcg/day (sublingual or intramuscular) Folic acid 5 mg/day Dietary adjustment Green leafy vegetables, whole grains and legumes that lower homocysteine levels should be consumed

4. Conclusion

The coexistence of acute cerebral infarction and myasthenia gravis is a very rare clinical condition. While both diseases can cause significant neurological and systemic effects on their own, when they occur together, the diagnostic process becomes more complicated and the differential diagnosis requires careful clinical evaluation.

In this case, the symptoms such as speech disorder, ptosis, limited eye movements, dysphagia, and muscle fatigue in the patient created a picture that cannot be explained by acute cerebral infarction alone. As a result of detailed neurological examination, electrophysiological tests and serological examinations performed considering the possibility of myasthenia gravis, the patient was also diagnosed with myasthenia gravis

The simultaneous presence of more than one serious disease in an elderly patient requires a special approach in terms of both diagnostic and therapeutic aspects. The coexistence of two diseases based on different mechanisms, such as acute cerebral infarction and myasthenia gravis, may cause the symptoms to mask or exacerbate each other. In this case, focusing on the management of only one disease may lead to a worsening of the patient's clinical course. Therefore, a multidisciplinary approach is of great importance.

For a correct diagnosis and an effective treatment plan, the neurological symptoms should be carefully evaluated. In the presence of atypical symptoms, auxiliary diagnostic methods (MRI, electrophysiology, antibody tests, etc.) should be applied comprehensively. The patient's cardiovascular risk factors should be actively managed. In patients with myasthenia gravis, certain cardiac drugs (e.g. beta blockers) should be carefully selected as they may worsen the course of the disease.

Treatment should be individualized. Myasthenia gravis and cerebral infarction treatment should be planned individually, taking into account the patient's other systemic diseases. In conclusion, the coexistence of acute cerebral infarction and myasthenia gravis is a condition that complicates clinical management, and in such cases, early diagnosis, multidisciplinary follow up, and personalized treatment planning are of vital importance. Long term monitoring of the patient's neurological, cardiovascular and general health status is critical in improving quality of life.

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