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Intracranial High-Grade Stenosis and Hyperhomocysteinemia Presenting as Cortical Subarachnoid Hemorrhage Concomitant with Acute Ischemic Stroke in a Young Man

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Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Male, 33-year-old
Final Diagnosis: Cortical subarachnoid hemorrhage
Symptoms: Headache • weakness
Medication: —
Clinical Procedure: —
Specialty: Neurology

Objective: Rare co-existance of disease or pathology

Background: Cortical subarachnoid hemorrhage (cSAH) is a rare clinical presentation with different causes, but rarely happens along with acute ischemic stroke. Intracranial high-grade stenosis originated from brain has been regarded as an unusual cause of cSAH, especially in young adults.

Case Report: A case of 33-year-old male presented with mild headache and spontaneous left-sided body weakness. Initial brain computed tomography (CT) showed cSAH in the right superior frontal sulcus. Further neuroimaging examinations including magnetic resonance imaging (MRI), digital subtraction angiography (DSA), transesophageal echocardiogram (TEE); in addition, lumbar puncture and blood tests were performed. Diffusion-weighted imaging (DWI) showed an acute infarction in the right frontal lobe and corona radiata of the territory of middle cerebral artery (MCA). The MR angiography (MRA) displayed no flow signal in the right middle cerebral artery M1-segment, while the DSA displayed bloodstream slowness in the right MCA M1-segment which suggested high-grade stenosis of the right MCA. The abnormal laboratory data suggested hyperhomocysteinemia, and excluded causes of thrombosis, infection, or cancer. The mechanism of cSAH may come about in severe atherosclerotic stenosis of MCAs by the broken of expanded tenuous compensatory pial vessels. The patient had good recovered at follow-up.

Conclusions: This case demonstrates cSAH with acute ischemic stroke, which is an uncommon complication, in a young adult stroke patient; a high-grade atherosclerotic stenosis of the MCA was identified as the etiology.

MeSH Keywords: Cerebrovascular Disorders • Hyperhomocysteinemia • Neuroimaging • Stroke • Subarachnoid Hemorrhage • Young Adult

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Background

Cortical subarachnoid hemorrhage (cSAH) is an infrequent and critical subtype of non-aneurysmal SAH, with different causes, in which bleeding is found in one or a small number of brain cortex sulcus and does not spread into the basal cisterns, ventricles, sylvian fissure or interhemispheric fissure, and so on [1]. Because the etiologies of cSAH vary and the symptoms are atypical and diverse, it is easy to escape diagnosis, be misdiagnosed, or to treat mistakenly in the clinical course [2–4]. Diverse etiologies of spontaneous acute cSAH have been described, including the following: pial arteriovenous malformations, dural arteriovenous fistulas, arterial dissection, dural or cortical cerebral venous thrombosis, vasculitides, reversible cerebral vasoconstriction syndrome, posterior reversible encephalopathy syndrome, high-grade stenosis (such as severe atherosclerotic carotid disease), endocarditis, cerebral amyloid angiopathy, coagulation disorders, abscess, cavernomas, and primary and secondary brain tumors [5].

cSAH secondary to a high-grade internal carotid artery stenosis is a high-risk marker for cerebral ischemic stroke [6,7], but the exact mechanism is not clear. Etiology of ischemic stroke in young adults includes large artery atherosclerosis, cardioembolism, cerebral small vessel disease, other determined stroke etiologies (antiphospholipid syndrome, autoimmune diseases, cervical artery dissection (CeAD), Fabry disease, factor II/V diseases, protein C/S diseases, illicit drug use, intracranial dissection, malignancy, mitochondrial disorders, moyamoya disease, post-radiation, reversible cerebral vasoconstriction syndrome, vasculitis), and stroke of undetermined cause (cryptogenic stroke) [8]. We should be able to find the exact etiology of ischemic stroke in young adults.

Here, we report on the case of a 33-year-old young man with high-grade stenosis of the right middle cerebral artery (MCA) presenting with cSAH and acute ischemic stroke.

Case Report

Chief complaints

A 33-year-old male patient was admitted to our department on an emergency basis because of a sudden-onset left-sided body weakness with a mild headache for 12 hours.

History of present illness

The patient had a right temporoparietal headache for 12 hours at rest and had no nausea and vomiting. At the same time, he felt left-sided body weakness, but he could lift his arm and walk alone, without slurred speech, numbness, conscious disorder, dysphagia, blurred vision, fever, cough or chest pain. Those symptoms were constant, so he came to our hospital emergency room. His National Institutes of Health Stroke Scale (NIHSS) score was 2.

History of past illness

The patient had a normal medical history. There was no other history, such as head trauma or drug abuse. He smoked for 10 years, 20 cigarettes a day.

Physical examination

His temperature was 36.0°C and his heart rate was 68 beats per minute. His blood pressure was 123/83 mm Hg and oxygen saturation in room air was 99%. There was no obvious abnormality in other general medical examination. On neurological examination, there was no aphasia, agnosia, or apraxia. Motor examination revealed mild weakness (Medical Research Council [MRC] grade, 5⁻/5) of the left limbs. There were absent of a stiff neck and the Kerning sign. The remaining neurological examination findings presented normal.

Laboratory examinations

The routine hematological, urinary and biochemical test findings were entirely normal. The findings of coagulation function tests were normal. Electrocardiogram and chest x-ray were also normal. Serological tests revealed homocysteine (HCY) was 47.6 umol/L. The anti-phospholipid antibodies (including lupus anticoagulant and anti-cardiolipin antibodies) were negative. Antinuclear antibody (ANA), anti-double stranded DNA (anti-dsDNA), anti-Sjögren's syndrome A/B (anti-SSA/SSB), and perinuclear antineutrophil cytoplasmic antibody (P-ANCA) were negative.

Imaging examinations

A brain computed tomography (CT) that was done at the emergency room showed irregular high-density in the right superior frontal sulcus which was diagnosed with right cortical subarachnoid hemorrhage (Figure 1A–1C). Neck vascular ultrasound showed no atherosclerosis and stenosis. The results of a 24-hour dynamic electrocardiogram (ECG) and transesophageal echocardiogram (TEE) were normal. The patient underwent brain magnetic resonance imaging (MRI) and cerebrospinal fluid (CSF) test. Diffusion-weighted images (DWI) showed tiny hyperintensity in the right frontal lobe and corona radiata and hypointensity in the apparent diffusion coefficient (ADC), and he was diagnosed with acute ischemic stroke (Figure 1D–1I). The fluid-attenuated inversion recovery (FLAIR) image showed hyperintensity in the right frontal lobe (Figure 1J, 1K) which suggested it was an acute ischemic

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lesion. The FLAIR showed striped hyperintensity in the right superior frontal sulcus (Figure 1L) was diagnosed as cSAH. There was severe stenosis or occlusion in the right middle cerebral artery (MCA) on the magnetic resonance angiography MRA (Figure 1M). Rupture of intracranial aneurysms was excluded by the brain MRA. Susceptibility-weighted imaging (SWI) showed that there were microbleeds in the right frontal lobe, temporal lobe, basal ganglia and thalamus (Figure 2A–2D). Finally, the digital subtraction angiography (DSA) of the neck and head was performed on the 10th day. It showed that the right MCA M1 segment was severely stenosed with a degree of 90% (Figure 1N) and the right posterior circulation was normal (Figure 1O), and it excluded a few causes of hemorrhages such as vascular malformations (dural arteriovenous fistulas, pial arteriovenous malformations, reversible cerebral vasoconstriction syndrome, vasculitides, arterial dissection, cerebral venous thrombosis, and aneurysm. The patient's headache was mild and not thunderclap-like, and the results of MRA and DSA showed severely stenosed right MCA M1 segment, no "beading" in the ipsilateral of other arteries (distal MCA, anterior cerebral arteries [ACA], right internal carotid artery [RICA], right vertebral artery [RVA], and basilar artery [BA]) (Figure 1M–1O), so reversible cerebral vasoconstriction syndrome was excluded from the cause of cSAH. There were no basal collaterals or left ICA or M1 narrowing which did not suggest an early stage of moyamoya syndrome (Figure 1M–1O).

Further diagnostic work-up

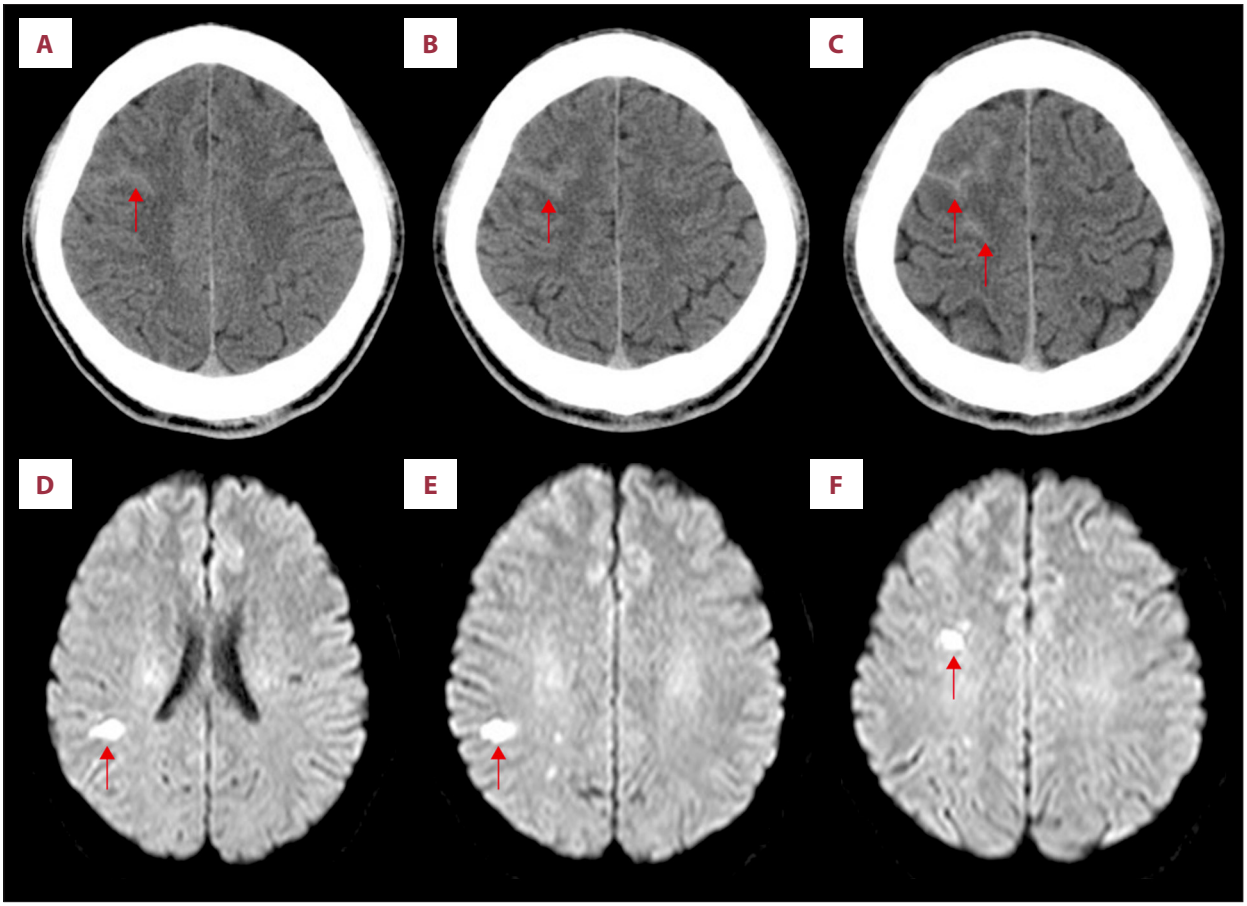
The angiotensin-converting-enzyme level was in the normal range. The results of hepatitis screening were negative. The CSF study was performed in the 4th day and the results revealed zero white blood cell, one red blood cell, CSF protein 0.28 g/L, and CSF glucose 3.3 mmol/L.

Final diagnosis

The final diagnosis of the presented case was cortical subarachnoid hemorrhage in combination with acute ischemic stroke, and hyperhomocysteinemia.

Treatment

Considering the brain CT and MRI findings, the patient was given: Shuxuetong injection to improve cerebrocirculation and cerebrometabolism, edaravone injection to eliminate free radicals, aspirin to antiplatelet aggregation, rosuvastatin to lower lipid and stabilize plaque, and folic acid, vitamin B6 and



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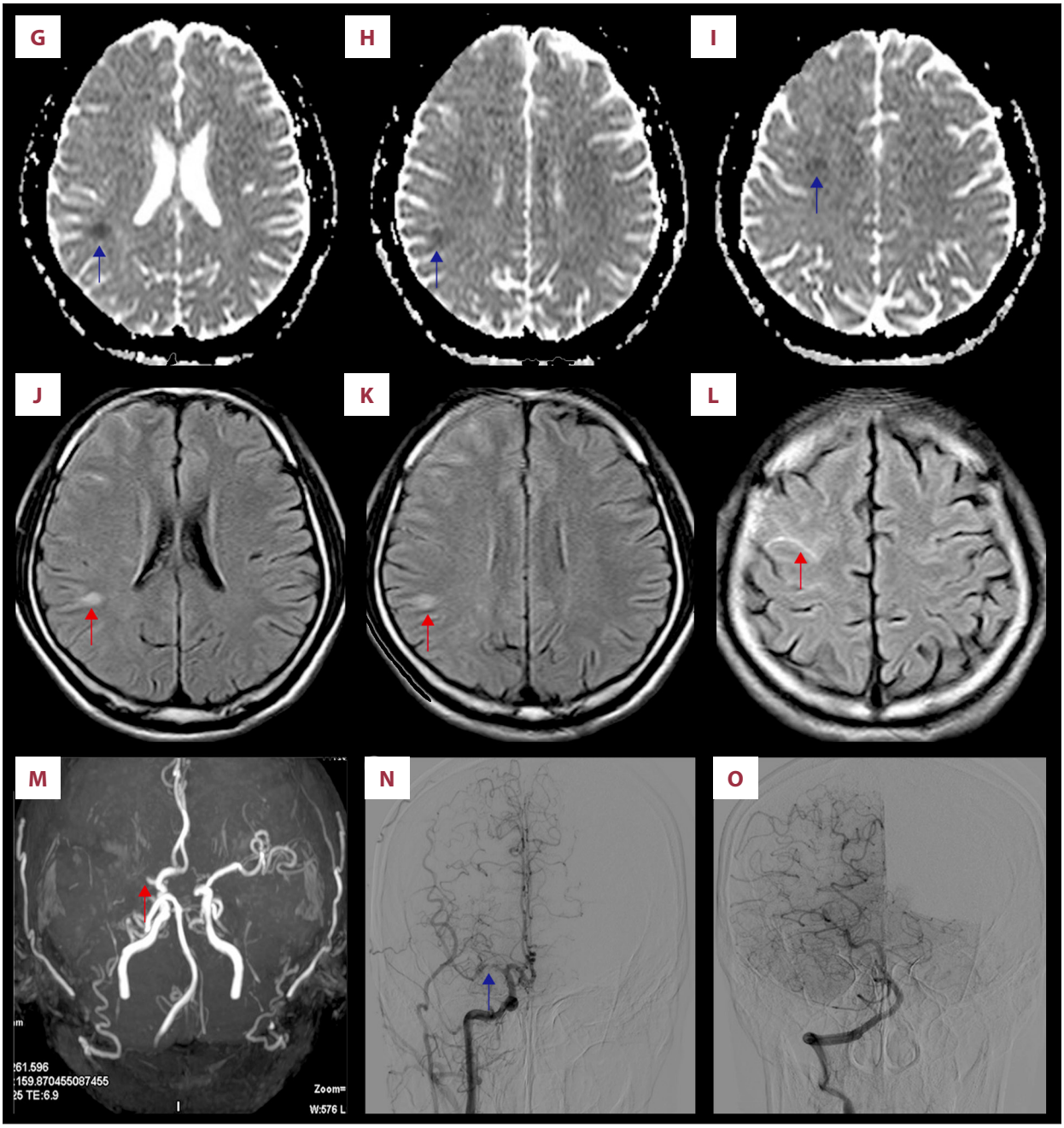

Figure 1. (**A–C**) Axial brain CT shows cortical subarachnoid hemorrhage in the right superior frontal sulcus (red arrow). (**D–F**) Axial diffusion-weighted image (DWI) shows acute ischemic infarction in the right frontal lobe and corona radiata (red arrow). (**G–I**) The apparent diffusion coefficient (ADC) shows acute ischemic infarction in the same location (right frontal lobe and corona radiata) (blue arrow). (**J, K**) Axial FLAIR shows acute ischemic infarction in the right frontal lobe (red arrow). (**L**) Axial FLAIR shows cSAH in the right superior frontal sulcus (red arrow). (**M**) Discloses the M1-segment of the right middle cerebral artery (MCA) occlusion (red arrow), a bilateral anterior cerebral artery from the right internal carotid artery (RICA), fetal posterior cerebral artery (PCA), and a hypoplastic left vertebral artery. (**N, O**) DSA shows severe stenosis in the M1-segment of the right MCA (**N**, blue arrow), and shows the normal cerebral arteries in the right posterior circulation (**O**).

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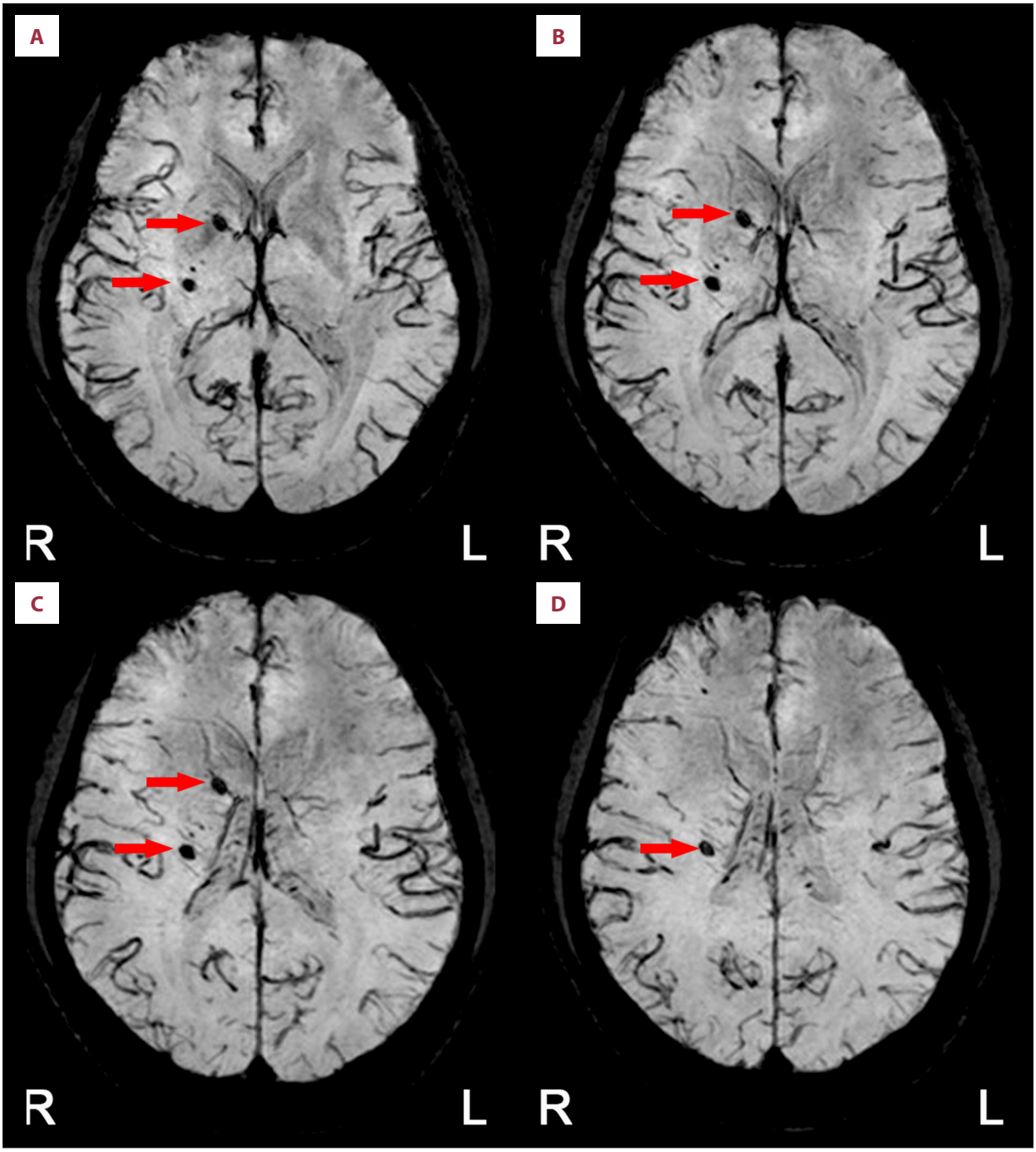

Figure 2. (**A–D**) Axial SWI shows microbleeds in the right frontal lobe, temporal lobe, basal ganglia and thalamus (red arrow).

vitamin B12 to reduce homocysteine concentrations. Upon completion of 2 weeks of therapy, the patient had fully recovered, and was discharged to hospital and continued to take aspirin, rosuvastatin, folic acid, vitamin B6, and vitamin B12.

Outcome and follow-up

The patient was asymptomatic at the 2-month and 8-month follow-up (NIHSS 0). Follow-up for MCA stenosis by brain MRA rule out stenosis due to spasm, while reversible cerebral vasoconstriction syndrome could be excluded from the cause of cSAH.

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Discussion

Cortical subarachnoid hemorrhage (cSAH) is a kind of cerebrovascular disease different from aneurysmal SAH; it accounts for 7.5% of nontraumatic SAH, but the prevalence is likely higher in the general population because it is poorly characterized or even hidden in clinical presentations [1,2]. cSAH has been associated with different vascular pathologies which involve pial arteriovenous malformations, dural arteriovenous fistula, arterial dissection, dural or cortical cerebral venous thrombosis, vasculitides, reversible cerebral vasoconstriction syndrome, posterior reversible encephalopathy syndrome, high-grade stenosis (such as severe atherosclerotic carotid disease), endocarditis, cerebral amyloid angiopathy, coagulation disorders, abscess, cavernomas, brain tumors, moyamoya disease, thrombocytopenia and others [1,2,6,9–13]. Reversible cerebral vasoconstriction syndrome or cerebral venous thrombosis is usually deduced to be the main cause of cSAH when patients are younger than 60 years of age [3,4,14–16]. Infective endocarditis has been identified as a cause of cSAH, with or without associated mycotic aneurysms [17], however, we have not encountered any patients with IE and cSAH on brain MRI.

When a thunderclap headache peaks at the beginning, presenting as explosive, severe, and unexpected, clinicians should at least be able to distinguish SAH, cerebral venous thrombosis, arterial dissection [15,18–23]. In our patient case, there was no evidence of cerebral venous thrombosis and arterial dissection. The results of infection, coagulation, and vasculitis screening tests were negative.

In addition, reversible cerebral vasoconstriction syndrome is characterized by thunderclap headache, with cSAH, reversible focal neurological deficits, or epileptic seizures [24–26]. The reversible arterial lesion can be diagnosed by MRA or ultrasound within 3 months. In our case, headache was not thunderclap and was slight, and the results of MRA and DSA showed the M1 segment of the right MCA was severely stenosed, not multifocal segmental cerebral artery vasoconstriction like “beading” in the ipsilateral other arteries (distal MCA, ACA, RICA, RVA, and BA), so we could exclude this headache from the cause of reversible cerebral vasoconstriction syndrome. According to the follow-up brain MRA imaging, we deduced that reversible cerebral vasoconstriction syndrome was not the cause of cSAH in combination with DSA, and the critical elements of the diagnosis of reversible cerebral vasoconstriction syndrome [27]. Although high-grade atherosclerotic stenosis of right MCA was the cause of cSAH, the patient did not undergo further extracranial-intracranial bypass or endovascular stenting treatment due to family financial difficulties.

A case of presumably acute symptomatic severe stenosis of right MCA and persistent focal deficits, combination with cSAH in a young adult has not been previously reported. IN our patient case, smoking and hyperhomocysteinemia were 2 risk factors for atherosclerosis. Hyperhomocysteinemia is an independent risk factor of cerebral apoplexy, whose detrimental effect is caused by ischemia and involves the increase of ROS and post-translational modifications of proteins [28]. There have been many studies that described mild hyperhomocysteinemia that produced an impact on arterial atherosclerotic modifications and then increased the risk for clinical presentations of stroke [29–34]. Acute ischemic infarction was diagnosed by MRI on the ipsilateral of cSAH, and both MRA and DSA suggested that the right MCA M1 segment was severely stenosed, which was suspected to be the main cause of ischemic stroke and cSAH in our case. The pathological mechanism is related to compensatory pial vasodilation and vulnerability. The mechanism of cSAH is presumed in cases of severe atherosclerotic stenosis of the middle cerebral arteries by the break of expanded tenuous compensatory pial vessels [5,7,35], combined with the failure of cerebrovascular autoregulatory mechanisms. The changes in local blood flow and perfusion pressure occur at the same time of the infarction, or a small amount of bleeding is caused by the rupture of blood vessels when an embolus reaches fragile collateral vessels.

The clinical presentations of cSAH are diverse due to diverse causes [36]. Because a small amount of hemorrhage is in the cortical sulcus of the brain, most cSAHs do not have the typical features of ruptured aneurysms, such as severe headache, neck stiffness, and disturbance of consciousness [3]. The clinical presentations of cSAH in patients under 60-year-old are headache including acute onset headache, gradual aggravation headache or mild headache, which was different than the typical thunderclap headache of aneurysmal SAH [4], and different from no headache or mild headache, transient aphasia, dysarthria, ataxia, limb weakness, or sensory disturbance of transient ischemic attack (TIA) [37]. A large proportion of cSAH patients are easily initially misdiagnosed as TIA, and thus neurologists should place high attention to potential cSAH.

The diagnosis of cSAH mainly depends on neuroimaging. The main findings of CT in cSAH are focal strip-like or linear high-density along the sulcus of the cerebral cortex with one convex surface or several adjacent ones, usually unilateral. cSAH is constrained to at least 1 or more than 2 sulci and generally includes the central sulcus, followed by the precentral sulcus and the intraparietal sulcus [38]. cSAH is easy to miss on non-enhanced brain CT whose sensitivity falls off quickly after 1- to 2-day onset of the disease, whereas MRI is used as a complementary diagnostic method [5,39]. cSAH is visible as focal linear hyperintensity along the sulcus in FLAIR sequence. SWI is of great value to diagnosing cSAH, and better than GRE T2 sequences in detecting microbleed and cortical hemosiderin [5]. cSAH is an unusual presentation of SAH as it is localized to

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the convexities of the brain and usually limited to adjacent sulci [4], and manifestations present as linear hypointensity in the subarachnoid space affecting ≥1 cortical sulci of the cerebral convexities on T2* GRE/SWI sequences with corresponding hyperintensity in the subarachnoid space on T1-weighted and/or FLAIR images [40]. Cerebral microbleeds are the deposition of blood component decomposition products leaked from cerebral small vessels in normal or near normal tissue, whose pathology is confirmed to be small perivascular hemosiderin deposits and whose severity can be evaluated on T2-GRE/SWI according to microbleed anatomic rating scale [41,42]. In our case, right frontotemporal lobe sulcus hemorrhage in CT was further confirmed by the FLAIR sequence. MRA and DSA indicated M1 segment stenosis in the right MCA, which was helpful for the etiology diagnosis of cSAH with acute cerebral infarction. Except for cSAH cases identified by brain CT, further brain MRI including FLAIR and SWI sequences should be performed to confirm the diagnosis and to actively explore for the possible causes.

The treatments of cSAH mainly include etiological treatment and symptomatic treatment [43]. However, the treatment principles for cSAH patients secondary to carotid intracranial or extracranial stenosis or occlusion with TIA or cerebral infarction are not clear. Whether antithrombotic medicine can be given as early as possible still needs to be further confirmed by large-scale evidence-based medicine research. There are no secondary prevention guidelines for young adults with stroke, but long-term treatment by antiplatelet is recommended after nearly any etiologies of stroke, except cervical artery dissection and a cardioembolic cause [8]. Therefore, antiplatelet, statin, lowering homocysteine and neuroprotective therapies were applied in our patient case and proven to be safe and effective after follow-up. Finally, it is important to identify the underlying causes of cSAH, not only for the need for early diagnosis and prompt exactitude therapy, but also for prognosis [36]. If cSAH is misdiagnosed, it may lead to potentially harmful treatments, then result in iatrogenic bleeding risk [2].

Conclusions

cSAH is an unusual complication of acute ischemic stroke in young adults. Because of diverse clinical presentations and etiologies, we should pay close attention to this uncommon cause of high-grade atherosclerotic stenosis of the cerebral arteries, and hyperhomocysteinemia as the cause of cSAH in the young ischemic stroke patients.

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Conflict of interest

None.

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