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Case Report: Intracranial large artery embolism caused by carotid thrombosis formed by neck massager

carotid thrombosis formed by neck massager

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Abstract

BACKGROUND

There are few reported cases of intracranial large artery embolism caused by carotid thrombosis formed by a neck massager. The authors report a case of carotid thrombosis formed by a neck massager.

CASE SUMMARY

A 49-year-old woman presented with left limb weakness and dysarthria after a history of neck massage for one month. Neurological examination showed left central facial paralysis and left hemiparesis with a 12-point National Institutes of Health Stroke Scale (NIHSS) score. Brain magnetic resonance imaging revealed restricted diffusion on diffusion-weighted imaging in the right parietal and temporal lobes. Computed tomography angiography (CTA) indicated M3 segment embolism of the right middle cerebral artery. Neck CTA revealed thrombosis of the bilateral common carotid arteries. Carotid ultrasound showed thrombosis in the bilateral common carotid arteries (approximately 2 cm below the proximal end of the carotid sinus), and contrast-enhanced ultrasound did not suggest enhancement. No hypertension, diabetes, heart disease, vasculitis, or thrombophilia was found after admission. After one week of

treatment with aspirin 200 mg and atorvastatin 40 mg, a carotid ultrasound reexamination showed that the thrombosis had significantly reduced.

CONCLUSION

Neck massages may cause carotid artery thrombosis.

Key Words: Neck massager; carotid thrombosis formed; Intracranial large artery embolism; stroke; endothelial damage.

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Core Tip: It has been reported that a stroke caused by neck massage is mainly related to arterial dissection due to the tearing of the inner vessel. The authors report a rare case of intracranial large artery embolism caused by carotid thrombosis formed by a neck massager. Combined with the analysis of the cases indexed in PubMed, only one case of a free thrombus in the carotid artery has been reported, although the etiology is unclear. We found that an intracranial large artery embolism was caused by a carotid thrombosis formed by a neck massager, which was treated effectively by antiplatelet therapy. In the future, it may be necessary to study further and propose stricter quality management standards for massagers.

INTRODUCTION

The probability of a stroke caused by a neck massage is 1/300–1/400,000, which is mostly related to arterial dissection due to the tearing of the vascular inner layer (mainly involving the vertebral artery, followed by the carotid artery) (1-3). Cerebral infarction caused by arterial embolism after a massage is rare (4,5).

A neck massager stimulates the neck according to the principle of pulsed microcurrents to promote blood circulation and alleviate fatigue. In this report, we describe an intracranial large artery embolism caused by carotid thrombosis formed by a neck massager.

CASE PRESENTATION

Chief complaints

Left lower limb weakness for more than five hours.

History of present illness

More than five hours prior, left lower limb weakness suddenly occurred in a 49-year-old female patient; the left upper limb could move autonomously, but the lower limb could only shift on the bed surface. She had slurred speech, no nausea or vomiting, limb twitching, and urinary incontinence. She was given thrombolytic therapy with alteplase (50 mg) at 11:50 am in another hospital, and her symptoms did not worsen after thrombolytic treatment. In our hospital, the patient received intensive statin treatment (atorvastatin 40 mg).The next day, the patient was transferred to the neurology ward; however, the left limb weakness worsened. The NIHSS score was 12, and the mRS was 4. After four days of treatment, the left limb weakness improved. At discharge, the patient had improved; her NIHSS score was 2, and her mRS score was 1.

History of past illness

The patient had no history of hypertension, diabetes, hyperlipidemia, heart disease, smoking, or drinking. Four months prior, the patient started to use a neck physiotherapy instrument for 1 month (**Figure 1N**) (twice a day, 15 minutes each time) because of neck muscle tension.

Personal and family history

The patient denied any family history of malignant tumors.

Physical examination

Vital signs: body temperature: 37.1°C; blood pressure: 122/82 mmHg; heart rate: 82 beats per min; and respiratory rate: 18 breaths per min. The cardiopulmonary examination was normal. Her cognition seemed normal; her speech was a little vague; the neck showed soft passivity; the Kernig sign was negative; the pupils were symmetrical and 3 mm in diameter; the light reflex was normal; eye movement was normal; there was no nystagmus; bilateral hearing was symmetrical; bilateral nasolabial folds were symmetrical; and the tongue could be stuck out on request in the center.

Left lower limb muscle strength level 1, left upper limb muscle strength level 5, right lower and upper limb muscle strength level 5, and the tendon reflexes of the extremities were normal; the muscle tone of the extremities was normal; Babinski sign positive on the left, negative on the right; and bilateral acupuncture sensations and deep sensations were symmetrical. The bilateral finger-nose touch, rotation, heel, knee, and tibia tests were stable and accurate, and the closed eyes sign could not be combined. Her initial National Institutes of Health Stroke Scale (NIHSS) score was 4, the modified Rankin Scale (mRS) score was 4, and her water swallow test grade was 2.

Laboratory examinations

Laboratory results were normal except for the D dimer 4410.0 μg/L. In addition, her thrombophilia genes were investigated, and the results suggested two risk factors: a 2KB heterozygous mutation upstream of PAI-1 (NM_000602.5) and a homozygous missense mutation in MTHFR (NM_005957.5).

Imaging examinations

Computed tomography(CT) of the head showed no brain hemorrhage. Computed tomography angiography (CTA) revealed occlusion of the M3 segment of the right middle cerebral artery (MCA) (**Figure 1A**).

Brain magnetic resonance imaging (MRI) showed limited diffusion on diffusion-weighted imaging of the right parietal and temporal lobes (**Figure 1B**). CTA (**Figure 1C–D**) and ultrasonography (**Figure 1E–F**) showed bilateral common carotid artery (CCA) thrombosis. Color Doppler carotid angiography (**Figure 1G–H**) and high-resolution enhanced MRI (**Figure 1I**) suggested bilateral CCA local thrombosis. On the eighth (**Figure 1J–K**) and 21st (**Figure 1L–M**) days, carotid ultrasound was repeated, and the thrombosis was reduced in size.

FINAL DIAGNOSIS

Intracranial large artery embolism caused by carotid thrombosis formed

TREATMENT

Oral aspirin 200 mg and atorvastatin 40 mg daily at discharge were prescribed. After discharge, the aspirin was reduced to 100 mg once a day and the atorvastatin to 20 mg once a day.

OUTCOME AND FOLLOW-UP

After six months of follow-up, there was no stroke recurrence, her NIHSS score was 1, and her mRS score was 1. No symptoms were aggravated, no complications occurred, and no adverse events were recorded. The patient is sfied with the treatment and can now continue with normal life.

DISCUSSION

To our knowledge, this report is the first to document a stroke caused by carotid thrombosis due to a neck massager. The brain MRI of the patient showed multiple acute infarcts in the right cerebral cortex, similar to arterial embolization. The cranial CTA showed that the M3 segment of the upper trunk of the right MCA was occluded. Carotid ultrasound showed symmetrical thrombosis of the bilateral common carotid arteries. At the same time, other etiologies, such as cardiogenic, immune, infectious, and

hematological diseases, were ruled out. After antiplatelet therapy, dynamic reexamination with carotid artery ultrasound showed the thrombus had significantly reduced and eliminated 21 days after stroke onset. The authors believe that the neck massager destroyed the vascular endothelial cells in this patient, thus forming a carotid artery thrombosis and causing an intracranial arterial embolism.

Cerebral infarction caused by neck massage is often secondary to vascular dissection (1-3). Only one case of a free thrombus in the carotid artery has been reported, and the specific etiology is unclear (4). Another case documented vertebral artery dissection after repetitive use of a handheld massage gun (5). The literature has suggested that massage can make the blood in the carotid artery swirl (6). Long-term changes in blood flow and acute injury of the artery may lead to changes in the structure of the endothelial cells. The lipids exposed after plaque rupture activate platelets and the coagulation system to form thrombi, reducing the blood supply to the brain. In addition, endothelial damage may impact plaques on the arterial wall, causing them to break and fall off. Plaque shedding may lead to distal vascular embolism, which may cause vasospasm, intimal hyperplasia, and accelerated arteriosclerosis, resulting in intracranial arterial embolism (7).

The massager used by the patient in this report is based on the principle of pulsed microcurrents to stimulate local muscles and acupoints by fixing electrodes in the bilateral anterior cervical regions. Carotid and contrast-enhanced ultrasound in this patient suggested that the intima was intact, and the evidence for plaque rupture was insufficient. The possibility of endothelial damage was considered, which may be related to her mutations in MTHFR (8).

The treatment of carotid artery dissection may include antiplatelet therapy, anticoagulation therapy, or surgical treatment according to the incidence, clinical symptoms, and vascular conditions (9). Emergency endovascular treatment may be selected for patients with acute intracranial large artery occlusion and hypoperfusion (10). However, the intracranial occlusive segment of this patient was the M3 segment of the MCA, and there was no indication for emergency treatment. Carotid artery

thrombosis is usually caused by atherosclerosis or carotid artery dissection; however, there is no clear guidance on drug treatment (11). The patient's clinical symptoms improved after antiplatelet therapy, and the thrombus disappeared 21 days later, indicating that antiplatelet treatment was effective.

This patient had a stroke after starting to use a neck massager for one month. Bilateral symmetrical thrombus signals occurred at the location of the common carotid artery stimulated by the electric pulse, and the local intima was intact. The thrombus disappeared 21 days after antiplatelet treatment. The development of the thrombus was considered to be related to local endothelial injury after electric pulse stimulation. Different electric pulse frequencies may have varying effects on endothelial cells, and further research is needed to explore a more suitable safety frequency for users and establish a better approach to evaluating the safety of massagers.

The limitation of this case is that although the mechanism of intimal injury is proposed through ultrasound findings, there is no further pathology confirmation. In the future, more research is needed to find a safe massage power and formulate stricter quality management standards.

CONCLUSION

Neck massages may cause carotid artery thrombosis.

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