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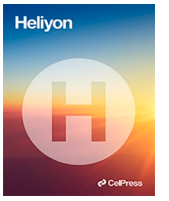

Case report

A woman with carotid atherosclerotic plaques suffered a massive cerebral infarction after carbon monoxide poisoning–A case report

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ABSTRACT

Cases of carbon monoxide (CO) poisoning complicated with massive cerebral infarction are rare. A chinese female patient with carotid atherosclerotic plaque was found unconscious during bathing. The patient was diagnosed as carbon monoxide poisoning complicated with massive acute cerebral infarction by carbon monoxide hemoglobin (COHb) detection and imaging examination. The patient regained consciousness after treatment with hyperbaric oxygen, antiplatelet, and atorvastatin. This case suggests that patients with carotid atherosclerotic plaque may be more susceptible to develop acute cerebral infarction when CO poisoning occurs.

1. Background

Carbon monoxide (CO) poisoning is one of the common diseases in the emergency department. Heart and brain damage are two common complications in patients with CO poisoning, while the most serious complication of CO poisoning is persistent neurological sequelae [1]. A study from professor Lin showed that patients with CO poisoning had a higher rate of stroke [2]. However, few studies have reported a relationship between CO poisoning and acute stroke, especially massive cerebral infarction. In this study, we reported a case of CO poisoning complicated with massive acute cerebral infarction in a patient with carotid atherosclerotic plaque and explored the possible causes. Written informed consent was obtained from the patient.

2. Case report

A 69-years-old woman from China took a bath in a bathroom equipped with a gas water heater. Unfortunately, she was found lying on the floor of the bathroom half an hour later and lost consciousness. Physical examination showed that the patient had right-sided gaze in both eyes, involuntary movement of both arms and legs, and increased muscle tension in the right limb. The blood pressure of the patient was 111/65 mmHg and SPO2 was 86 %. In addition, the patient has a history of hypertension, diabetes and carotid atherosclerotic plaque. She usually took medicines intermittently to control blood pressure and blood glucose, but never monitored her blood pressure and blood glucose. The patient was delivered to hospital by ambulance, and blood gas analysis indicated that the COHb

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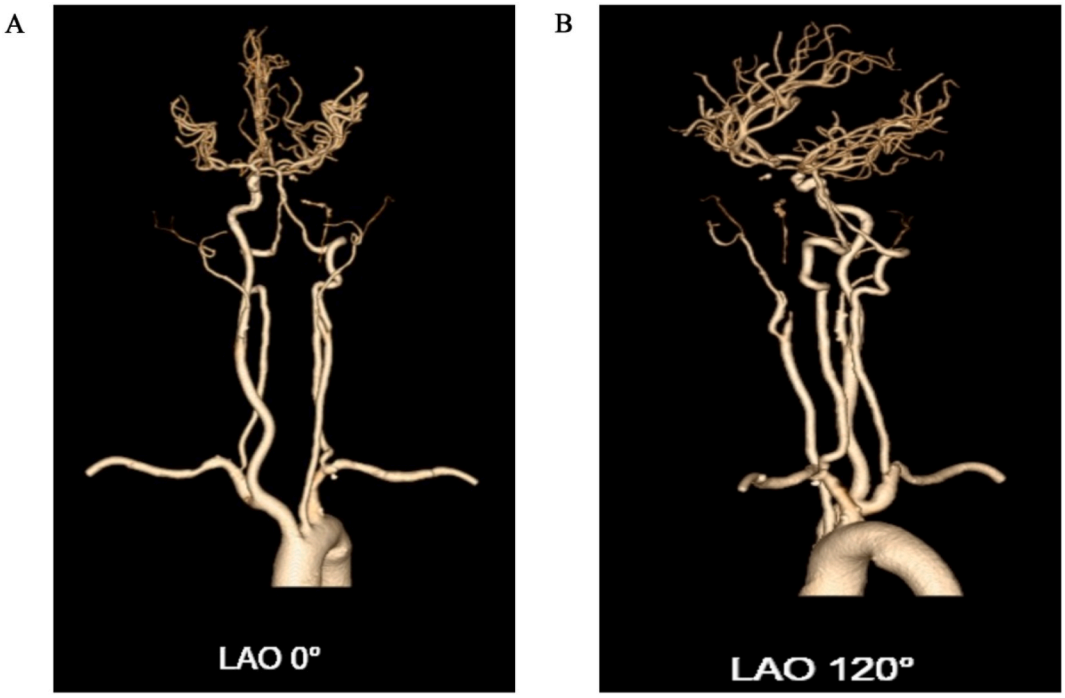

Fig. 1. The examination of CTA showed the occlusion of the left internal carotid artery.

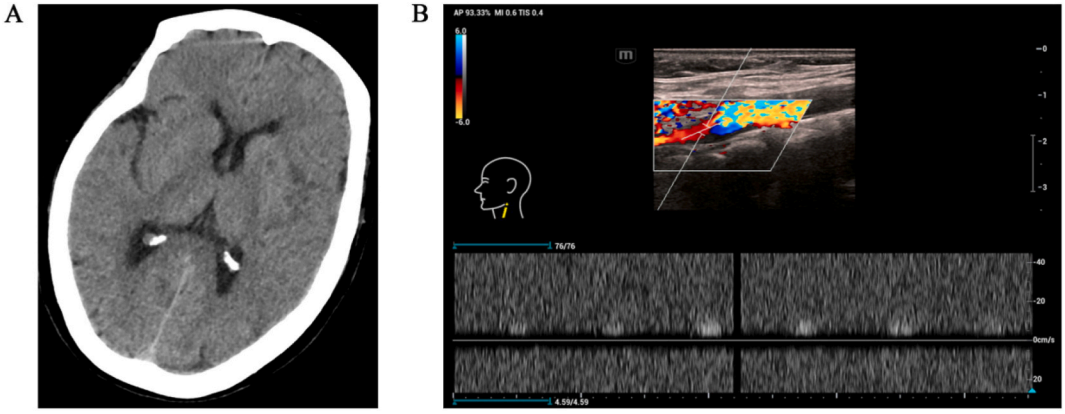

Fig. 2. (A) The examination of CT scan showed that multiple plaques and lacunar infarction were seen in the left temporal lobe, insula lobe, bilateral lateral ventricles and basal ganglia. (B) The examination of cervical vascular ultrasound revealed the proximal subtotal occlusion of the left internal carotid artery.

was 35 %. CTA tests were performed 2 hours after the patient was found unconscious, indicating the presence of atherosclerotic plaques in aortic arch and carotid artery and left internal carotid artery occlusion (Fig. 1). In addition, The CT scan showed that multiple plaques and lacunar infarction were seen in the left temporal lobe, insula lobe, bilateral lateral ventricles and basal ganglia (Fig. 2A). Then, the patient became conscious after treating with hyperbaric oxygen, but could not respond normally. The physical examination showed that the left limb muscle strength was grade 3, and the right limb muscle strength was grade 1. After admission, she was given treatment including hyperbaric oxygen (once a day for 10 days), plavix (75mg a day until discharge), rosuvastatin calcium (10mg a day until discharge) and others. The result of cervical vascular ultrasound revealed that bilateral carotid intima-media thickening with plaque formation and proximal subtotal occlusion of the left internal carotid artery (Fig. 2B). Moreover, The result of the head MRI examination revealed that there were significant abnormal signals in the right temporo-occipital lobe, bilateral basal ganglia, left lateral ventricle and left cerebral hemisphere cortex. Massive acute cerebral infarction was considered. Besides, there were abnormal signals in the left lateral ventricle and bilateral basal ganglia which were considered as CO toxic encephalopathy (Fig. 3). As a result, the muscle strength of the left limb recovered to grade 5 and the right limb muscle strength recovered to grade 3 after 11 days

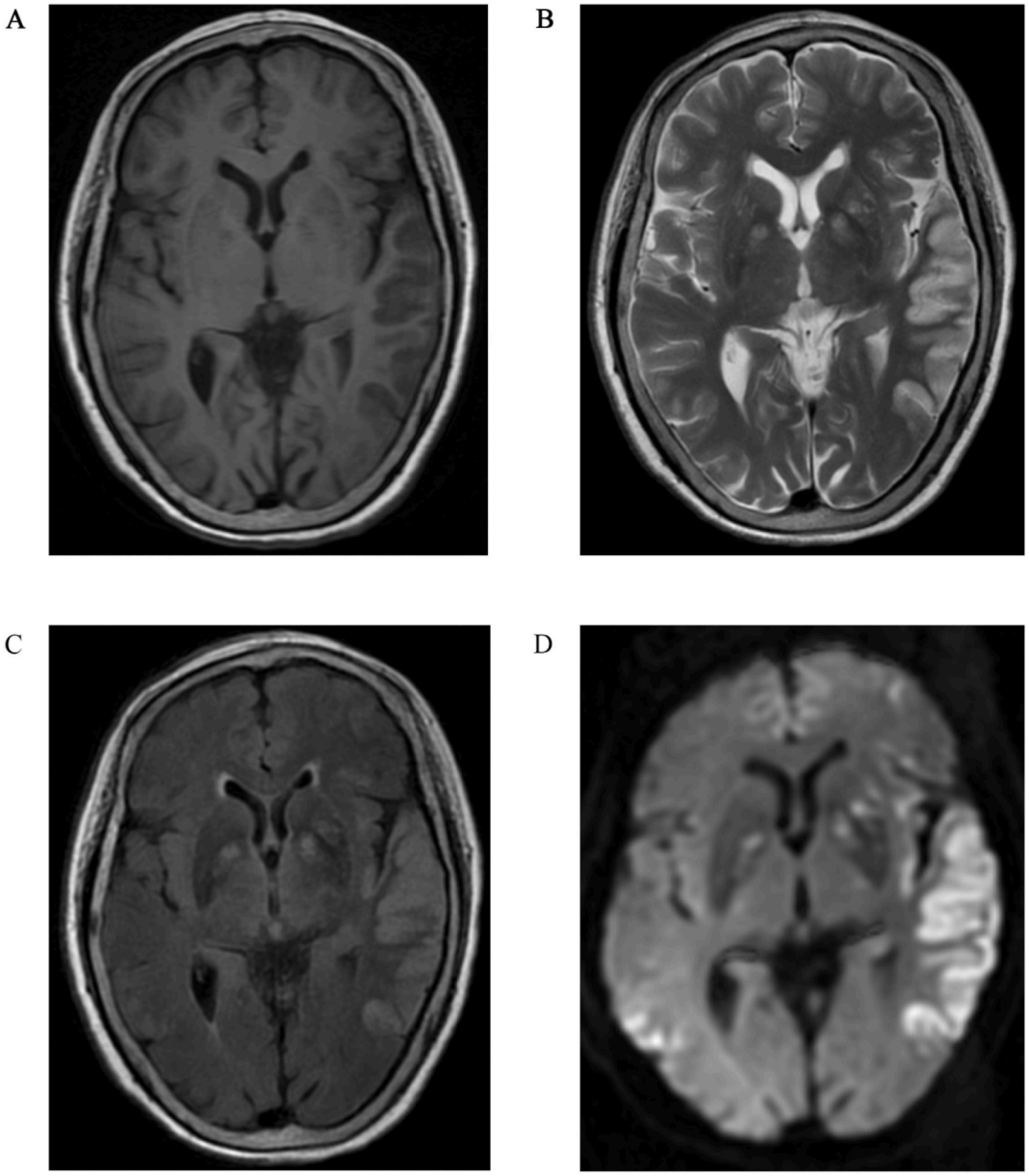

Fig. 3. (A) T1 showed a hypointense signal. (B) T2 showed high signal intensity. (3) FLAIR appeared as slightly hyperdense. (D) DWI showed diffusion-restricted hyperintensity.

of treatment. Finally, the patient was in stable condition and discharged. Follow-up was performed 2 months later. The patient continued to take plavix and rosuvastatin calcium after discharge, and the cognitive function and limb muscle strength of the patient were not significantly different from those at discharge.

3. Discussion

CO poisoning complicated with acute cerebral infarction is rare, and its mechanism is still unclear. There are several possible reasons. Firstly, according to previous studies, ischemia-sensitive basal ganglia, white matter, lateral ventricle and cerebral cortex are easily damaged by CO poisoning [3]. This is consistent with the MRI result of this patient. Therefore, we speculate that carbon monoxide poisoning causes cerebral hypoxia, further leads to cerebral vasospasm and cerebral thrombosis, and finally causes acute cerebral infarction.

Besides, CO poisoning can lead to vascular endothelial injury, hyperlipidemia, lipid peroxidation and arterial wall intima and subintima lipid deposits, which aggravates atherosclerotic plaques and further causes arterial stenosis. Carbon monoxide exposure

appears to accelerate plaque formation via increasing capillary permeability in animals on atherogenic high-cholesterol diets [4,5]. In addition, CO can increase the permeability of the vascular endothelium and cause a high degree of edema of the intimal cells, which further leads to stenosis of the arterial lumen [6]. Moreover, massive cerebral infarction often occurs after occlusion of large vessel including internal carotid artery [7]. Therefore, we concluded that on the basis of the presence of internal carotid artery plaque in this patient, the stimulation of CO led to the aggravation of arterial plaque and the edema of intimal cells which further resulted in internal carotid artery occlusion and eventually caused large-scale infarction. It may an another mechanism.

Finally, the asymmetric ischemic signals is common in cerebral infarction caused by the detachment of emboli, such as emboli originating from the heart, aortic arch, and carotid artery [8]. Atherosclerotic lesions of the thoracic aorta have been taken as one of an important factor of stroke and peripheral embolization [9]. Coincidentally, the above results were consistent with CTA and carotid ultrasound examination results of this patient. Thus, multiple cerebral infarction caused by plaque detachment from the aortic arch or carotid artery cannot be excluded. However, CO poisoning also can manifest as asymmetric cortical and subcortical lesions [6]. Therefore, the etiology of this patient has not been determined entirely. Stroke and carbon monoxide poisoning may occur coincidentally at the same time. However, considering the patient's past medical history, we believe that CO poisoning-induced internal carotid artery occlusion is the most likely cause of massive cerebral infarction.

4. Conclusion

CO poisoning complicated with acute cerebral infarction may involve a variety of mechanisms. CO aggravates atherosclerotic plaque and causes arterial occlusion, which may be an important regulatory pathway for acute cerebral infarction caused by CO poisoning. Conditions such as high blood pressure, diabetes, and hyperlipidemia often cause atherosclerotic plaques, which means that people with these conditions are more likely to develop acute cerebral infarction in the event of CO poisoning. Therefore, prevention of CO poisoning should be emphatically endorsed for everyone.

CRediT authorship contribution statement

Jierong Mo: Writing – review & editing. **Zhiquan Li:** Data curation. **Zhiyong Lin:** Formal analysis. **Peiyi Liu:** Investigation. **Weigan Xu:** Supervision. **Zuhua Huang:** Data curation. **Lichan Mo:** Data curation. **Lingyan Jiang:** Investigation. **Tianen Zhou:** Funding acquisition. **Jun Jiang:** Supervision, Funding acquisition.

Ethics statement

The study was reviewed and approved by Ethics Committee of The First People's Hospital of Foshan.

Data availability statement

The data of this study are available.

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Declaration of competing interest

All authors declare no conflict of interest.

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