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CASE REPORT

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New asymptomatic thrombosis caused by carotid web during the acute period of cerebral infarction

Yan Wang[1], Hai-Lei Li[2], Xiao-Hua Xu[1], Jin-Hao Ye[3] and Jian Li[4*]

Abstract

Background At present, the carotid web (CaW) as an important cause of cryptogenic ischemic stroke has gradually received clinical attention. CaW is associated with a high risk of stroke and patient is more likely to have recurrent stroke if the CaW is untreated. We report a patient who developed CaW related thrombosis during the acute period of cerebral infarction.

Case presentation A 49-year-old male patient with CaW in the left internal carotid artery was diagnosed by computed tomography angiography (CTA) and had two cerebral infarctions in two years. Within 72 h after thrombolysis for an acute cerebral infarction, acute thrombosis was identified between the web and the posterior wall of the carotid artery on carotid ultrasound. Emergent carotid endarterectomy (CEA) was performed to remove abnormal CaW structures and thrombosis to prevent stroke. The patient recovered well and was asymptomatic at 2 months follow-up.

Conclusion Carotid web related thromboembolism is a rare cause of stroke. Carotid ultrasound plays an important role in the diagnosis of asymptomatic thrombosis caused by carotid web. Carotid endarterectomy is effective for stroke prevention in patient with carotid web related thrombosis.

Keywords Carotid web, Asymptomatic thrombosis, Cerebral infarction, Carotid ultrasound, Carotid endarterectomy

Background

As a rare cause of acute cerebral infarction, carotid web (CaW) has attracted clinical attention since decades ago [1]. The histological definition of CaW is atypical fibromyodysplasia, the spine-like luminal protrusion of CaW is an important pathological basis for thrombosis [2]. Most CaWs are associated with a high risk of stroke, leading to recurrent symptoms of cerebral ischemia and infarction [1, 3].

Duplex ultrasound and computed tomography angiography (CTA) are recommended as the diagnostic imaging for CaW [2]. The characteristic of CaW on CTA is a shelf like filling defect protruding from the lumen of the carotid artery in the sagittal plane, and a linear defect

*Correspondence:
Jian Li
lijian@hku-szh.org
[1]Department of Ultrasound, The University of Hong Kong-Shenzhen Hospital, No.1, Haiyuan Road, Futian District, Shenzhen 518053, China
[2]Division of Vascular Surgery, Department of Surgery, The University of Hong Kong-Shenzhen Hospital, No.1, Haiyuan Road, Futian District, Shenzhen 518053, China
[3]Department of Neurology, The University of Hong Kong-Shenzhen Hospital, No.1, Haiyuan Road, Futian District, Shenzhen 518053, China
[4]Department of Radiology, The University of Hong Kong-Shenzhen Hospital, No.1, Haiyuan Road, Futian District, Shenzhen 518053, China

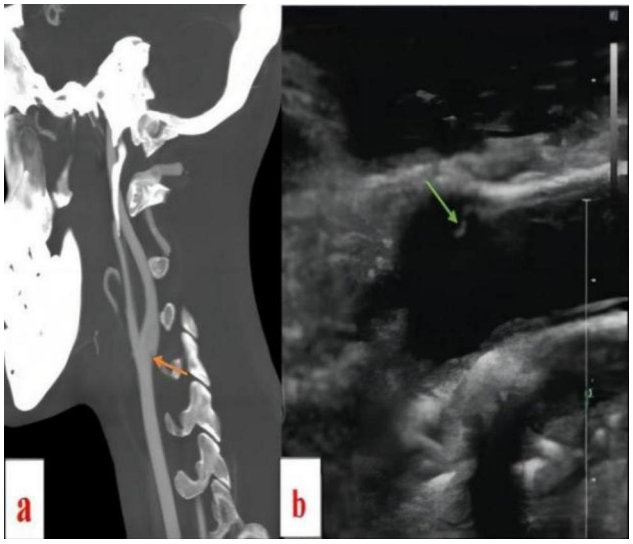

Fig. 1 **a** CTA showed CaW (orange arrow), **b** Carotid ultrasound showed CaW (green arrow)

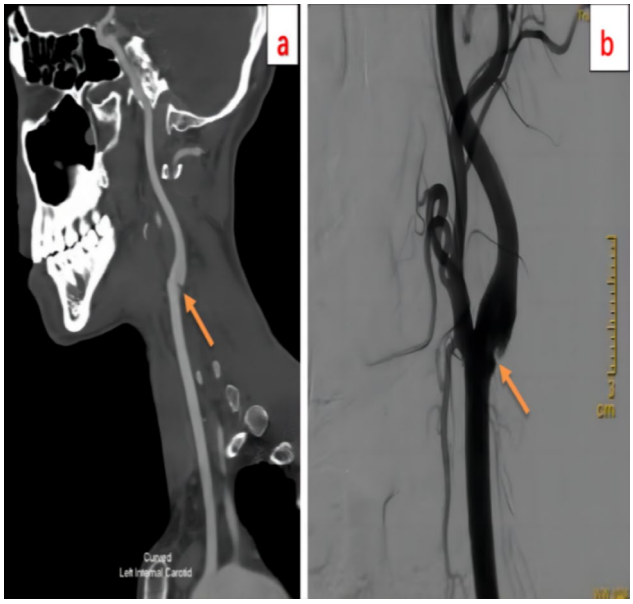

Fig. 2 **a**,**b** CTA and DSA showed CaW (orange arrow), no filling defect

of lumen division appears in the axial view, usually at the origin of the internal carotid artery (ICA), and most of them originate from the posterior lateral wall of the proximal ICA [4]. CaW was found in 2.5% of patients with large vessel occlusive acute ischemic stroke by CTA examination, most of them were female [5]. In this study, we report a patient with CaW associated stroke who developed asymptomatic thrombosis again in the acute period of cerebral infarction (within 72 h).

Case presentation

A 49-year-old male presented with aphasia and right limb weakness for four hours. He had a past medical history of stroke, and Caw had been identified on CTA (Fig. 1a) and carotid ultrasound (Fig. 1b) during previous hospitalization. He denied a past medical history of hypertension, diabetes or coronary arterial disease. Since the first ischemic stroke, the patient had been taking anti-platelet drugs and statins continuously to prevent recurrence of stroke .

On physical examination, he was unconscious, somnolence, aphasia. He had right-sided central facio-lingual palsy, and right limb muscle strength was grade 4 (Lovett scale). The National Institute of Health stroke scale (NIHSS) score was 8, and Glasgow Coma Scale (GCS) score was 4/2/6. Blood cell and coagulation tests were done in Accident and Emergency Department and the results were unremarkable. Head CT scan showed no significant abnormalities. Acute cerebral infarction was diagnosed based on the symptoms and intravenous thrombolytic therapy with rt-PA (0.9 mg/Kg) was given. The patient recovered well after thrombolysis, and the NIHSS score was improved to 1. About 30 min after thrombolysis, urgent CTA of head and neck was done and linear filling defect was identified at the origin of left internal carotid artery (ICA) (Fig. 2a). The distal M1 segment of left middle cerebral artery (MCA) was close to complete occlusion. Cerebral CT perfusion (CTP) showed the perfusion was delayed in the left cerebral hemisphere: mean transit time (MTT) and time to peak (TTP) were prolonged, cerebral blood flow (CBF) and cerebral blood volume (CBV) were slightly raised. About 75 min after thrombolysis, emergency cerebral digital subtraction angiography (DSA) was performed. During intraoperative angiography, we found the occluded segment of MCA was successfully recanalized, so cerebral artery thrombectomy was not performed. CaW was observed at the origin of the left internal carotid artery, contrast retention without filling defect was observed at the carotid bulb (Fig. 2b). After 24 h of thrombolysis, repeated CTP showed that the abnormal perfusion area was significantly reduced and was limited to the left basal ganglia. The patient was treated with aspirin, and his condition was stable. During this period, the patient's blood pressure was maintained at about 100/70mmHg for a long time without any antihypertensive drugs.

After 60 h of thrombolysis, carotid ultrasound found CaW in the posterior aspect of the left internal carotid bulb. Surprisingly, a slightly hyperecho mass of 6.1×3.9 mm was detected between web and posterior wall of the bulb, obvious eddy currents were detected around it. The CaW is a double layer of strong echo membranous structure protruding into the lumen, about 6.2 mm in length and 1.2 mm in thickness, consistent with the direction of blood flow. The angle between the web and the posterior wall was 35°. Left CaW with acute thrombosis in situ was highly suspected (Fig. 3).

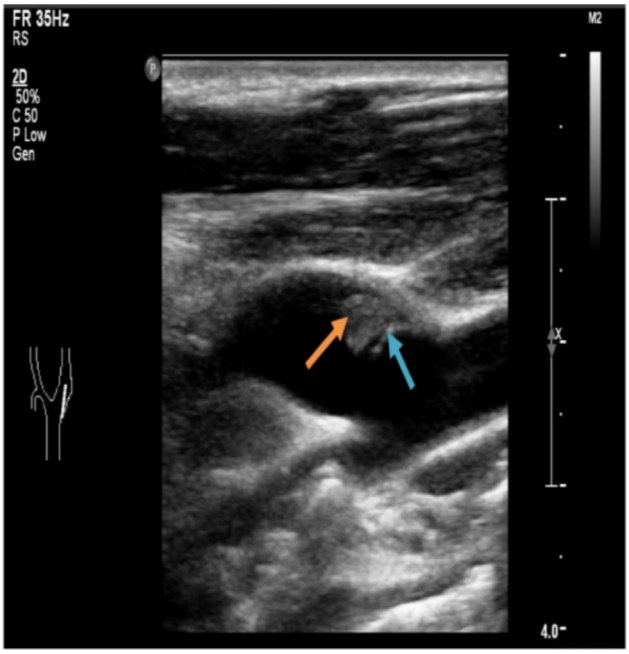

Fig. 3 Carotid ultrasound showed CaW (blue arrow) and a new thrombus (orange arrow)

Emergent carotid endarterectomy (CEA) was performed under general anesthesia. During operation, blood clot was identified between the carotid intima and posterior arterial wall (Fig. 4). The thrombus was removed and endarterectomy was performed. Pathology revealed mixed thrombus, a small amount of fibrinoid nets, red blood cells and inflammatory cells. Pathology showed that the thickness of the wall was uneven, some of the wall tissues were hyalinized, and some of the wall tissues were mucoid. The patient recovered well and discharged on postoperative Day 10. The patient was followed up at 2 months, he was asymptomatic without evidence of recurrent cerebral infarction on carotid ultrasonography.

Discussion

In this study, we reported a 49 years old male patient with recurrent cerebral infarction caused by CaW. Published studies reported that women and African Americans are significantly at high risk for CaW, especially in symptomatic CaW patients [6]. The patient with ischemic stroke caused by CaW usually has a younger age [7].A recently published epidemiological survey showed that the prevalence of CaW in transient ischemic attack/stroke, cryptogenic ischemic stroke, macrovascular occlusion stroke, carotid artery stenosis patients were 1.2%, 6.4%, 1.1%, and 4.4, respectively [8].The incidence of acute ischemic stroke (AIS) in patients diagnosed with CaW is as high as 89%, most of the AIS are ipsilateral, and the recurrence rate is also high [9].The incidence of CaW in young patients with cryptogenic stroke is as high as 25%, and

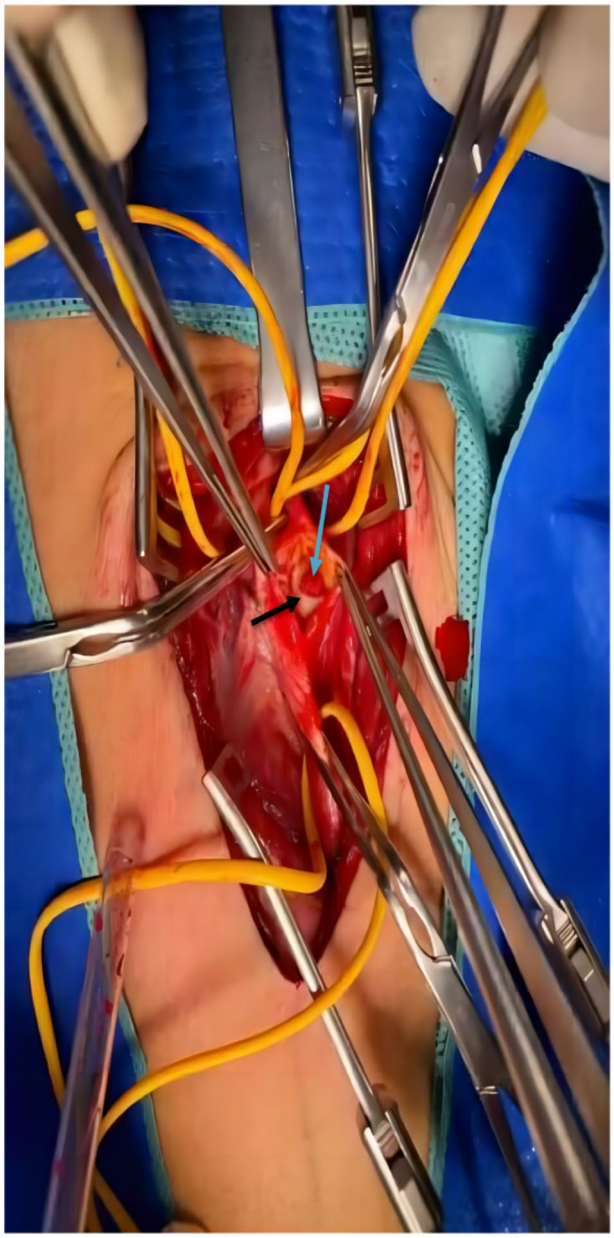

Fig. 4 CaW (black arrow) and red thrombus (blue arrow) were observed during CEA

most of the patient have proximal large vessel occlusion [6, 10]. The mechanism of stroke may be blood flow disorder, blood stasis, thrombosis, and embolism caused by abnormal CaW structure [4, 11]. Our patient is a typical cryptogenic ischemic stroke caused by ipsilateral carotid artery CaW, and the recurrent attack is probably due to the hemodynamic disorder caused by CaW, which leads to thrombosis formation and embolism to distal cerebral artery, resulting in cerebral infarction.

Previous reports have shown that CaW-induced embolic thrombosis of the great arteries is typical of mixed thrombosis [12].The pathological report of

thrombosis in this patient was also consistent with this finding. Studies have found that there is obvious blood stasis and low wall shear stress around the CaW reticular structure, which is a hemodynamic risk factor for thrombosis [13].The artificial CaW model further confirmed that the lower the blood flow velocity, the more prone to turbulence. In particular, when the stenosis rate is higher and the angle is smaller, the turbulence intensity (TI) will be higher, which is also the pathogenesis of most CaW ischemic strokes [14]. In addition, the angle of CaW in stroke patients is smaller (mostly acute angle≤90.1°), and with a significant longer length (≥3.1 mm) [15].The characteristic of this Caw on ultrasound (CaW length of 6.2 mm, with a small Angle of 35°), especially the obvious eddy around CaW detected by ultrasound supported this CaW was associated with a high-risk of stroke. In addition, the prolonged low basal blood pressure (about 100/70mmHg) leading to the insufficient carotid perfusion pressure in the acute phase of stroke maybe another important risk factor for the rapid recurrence of thrombosis.

At present, although the value of ultrasound alone in the diagnosis of CaW has been questioned, ultrasound still has irreplaceable advantages [16]. First of all, it is simple and convenient to operate, which can be evaluated and screened anytime and anywhere, and provide timely and effective guidance and suggestions for clinical practice. In this study, the new asymptomatic thrombosis can be timely and accurately detected, carotid ultrasound examination played a crucial role. Otherwise, this new thrombosis may cause another large artery embolic infarction, and the outcome is unpredictable. Secondly, ultrasound detection can quantitatively evaluate some basic morphological and hemodynamic characteristics of CaW, which is of more practical significance for stroke prevention. In addition, ultrasound examination technology is constantly innovating and developing. Recent studies have found that CaW display rate of CaW in longitudinal section of ultrasound is significantly higher than that in cross section, thus improving the diagnostic accuracy of CaW in ultrasound examination [17]. Furthermore, the slow flow sensitive ultrasonic microfluidic imaging (MFI) technology by duplex ultrasonography can significantly improve the diagnostic ability of CaW [18]. In the future, relevant clinical studies should be carried out to further verify the application value of carotid ultrasound in the prevention and control of long-term stroke for patients with CaW.

The treatment for CaW is still controversial. The conclusion of MR CLEAN trial has clearly warned that drug treatment alone may not be able to prevent the occurrence and recurrence of stroke caused by CaW [19]. For symptomatic patients with a carotid web in whom no other cause for stroke can be identified after detailed neurovascular work up, carotid endarterectomy or carotid artery stenting may be considered to prevent recurrent stroke [20]. At present, most studies tend to prefer CEA strategy for the prevention and control of the high recurrence rate of CaW stroke [21]. CEA may be appropriate as a treatment method along with prevention failure of antiplatelet agents in patients with CaW.

Although thrombosis caused by carotid web is rare, it has been reported by published study [12, 13, 22]. In this case, conservation antiplatelet therapy was not effective to prevent thrombosis and recurrence of stroke, and surgical intervention was indicated. CEA was arranged after thrombolysis when the patient's condition was stable. Thrombosis was identified on duplex ultrasound after thrombolysis and emergent CEA was performed to prevent recurrent cerebral infarction. The patient recovered well.

Conclusion

CaW is a rare cause of cerebral infarction. Routine duplex ultrasound examination is of great value for the diagnosis of CaW and prevention of stroke. Carotid endarterectomy is the treatment of choice in patients with CaW related thrombosis.

Declarations.

Abbreviations

CaW	Carotid web
CEA	Carotid endarterectomy
CAS	Carotid stenting
DSA	Digital subtraction angiography
CTA	Computed tomography angiography
CTP	CT perfusion
ICA	Internal carotid artery
MCA	Middle cerebral artery
MFI	Microfluidic imaging
AIS	Acute ischemic stroke

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Author contributions

HLLand JHY were involved in direct patient care, JHY performed the DSA and HLLwas involved in the operation of CEA.YW and XHX performed carotid ultrasonography. JLCompleted the CTA diagnosis report. YW and JL were responsible for manu script design and wrote the manuscript. All authors read and approved the final manuscript.

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Data availability

All data are available in the manuscript.

Declarations

Competing interests

The authors declare no competing interests.

Ethics approval and consent to participate

Not applicable.

Consent for publication
The patient in this study provided written informed permission for his medical history to be published in this case report.

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