



## Case Report

# Transient Ischemic Attack and Carotid Web with Atherosclerotic Plaque

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## Abstract

Carotid web, considered a non-atherosclerotic disease, has been increasingly identified as a risk factor for ischemic stroke and transient ischemic attack. The association of an atherosclerotic plaque with carotid web is very rarely described, especially in the absence of atherosclerotic risk factors. Here, we report a case of a 23-year-old woman presenting symptoms of transient ischemic attack, with ipsilateral carotid web associated to an atherosclerotic plaque with signs of intraplaque haemorrhage, successfully treated with carotid endarterectomy.

**Keywords:** Transient Ischemic Attack; Atherosclerotic Plaque; Carotid Web; Young.

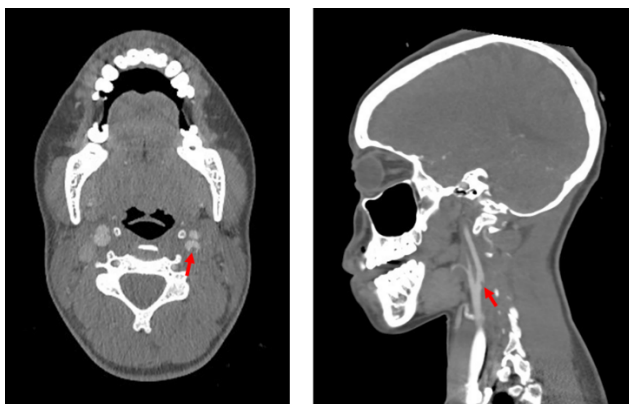
## Introduction

Carotid web (CaW) is a rare and underdiagnosed cause of ischemic stroke, more frequently found in young females [1] and in patients of African descent [2], with absence of vascular risk factors in the majority of patients [3]. It is defined on angiography as an intraluminal-filling defect mostly located in the posterior wall of the carotid bulb [4]. Histologically it is thought to be an intimal variant of fibromuscular dysplasia with intimal fibrosis and

hyperplasia [1,3,4]. We report a case of a young woman presenting symptoms of transient ischemic attack (TIA) in the left middle cerebral artery territory, in which computed tomography angiography (CTA) of head and neck revealed an ipsilateral CaW. Brain magnetic resonance imaging (MRI) with T1 weighted fat-saturated (T1FS) sequence of the cervical vessels detected an atherosclerotic plaque with signs of intraplaque haemorrhage associated to the CaW. The patient was subsequently treated with carotid endarterectomy and follow-up at four months showed no recurrent neurological symptoms.

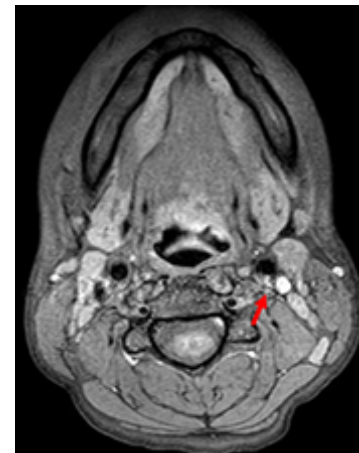
## Case Presentation

A 23-year-old woman of African descent, with no relevant medical history, presented at the emergency department after experiencing a transient right brachiofacial hypoesthesia lasting for approximately 5 minutes. Neurological examination at admission was normal. There were no prodromal symptoms, trauma or headache associated. The diagnosis of TIA was made and the patient was included in our TIA clinic. Her home medication consisted solely of a combined oral contraceptive pill. No other vascular risk factors were identified and family history was unremarkable. CTA of head and neck showed a shelf-like protrusion in the posterior wall of the left carotid artery bulb consistent with CaW (Figure 1). The right carotid artery showed no anomalies and in particular, no contralateral CaW was identified. Brain MRI revealed no recent ischemic lesion on diffusion weighted imaging (DWI). T1FS imaging of the cervical vessels showed the presence of a non-stenotic atherosclerotic plaque with signs of intraplaque haemorrhage along the posterior wall of the left carotid artery bulb associated to the CaW (Figure 2). Dual antiplatelet therapy (Aspirin and Clopidogrel) was initiated together with a statin and the combined oral contraceptive pill was discontinued. The patient was treated with carotid endarterectomy within a week and Aspirin monotherapy was continued after surgery. Intraoperative findings confirmed the presence of a CaW associated to an atherosclerotic plaque (Figure 3). Histological examination of this sample revealed intimal hyperplasia with infiltration of spumous macrophages (Figure 4). Follow-up at four months revealed no recurrent neurological symptoms.



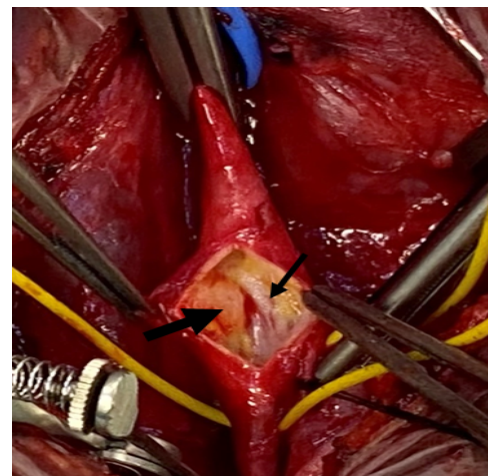
**Figure 1:** CTA of head and neck.

Presence of an intraluminal shelf-like protrusion in the posterior wall of the left carotid artery bulb on (A) axial and (B) sagittal images (red arrow), consistent with the diagnosis of CaW.



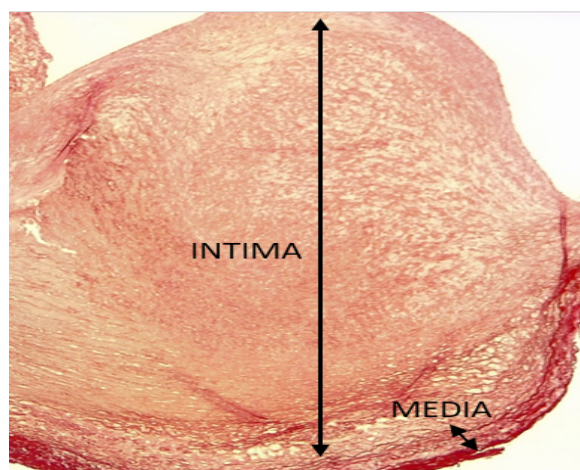
**Figure 2:** Brain MRI.

T1FS imaging of the cervical vessels showing a non-stenotic atherosclerotic plaque with signs of intraplaque haemorrhage along the posterior wall of the left carotid artery bulb (red arrow).



**Figure 3:** Intraoperative findings during carotid endarterectomy.

Intraoperative findings confirmed the presence of a Caw (thick black arrow) and an atherosclerotic plaque (thin black arrow).



**Figure 4:** Histological examination.

Orcein staining of the CaW sample revealed intimal hyperplasia with infiltration of spumous macrophages.

### Investigations

Further work-up with transthoracic echocardiography and cardiac monitoring showed no relevant abnormalities. No hypertension was detected. Tran's oesophageal echocardiography showed a patent foramen ovale (PFO) with a right-left shunt and passage of 5-10 bubbles, with no increased passage of number of bubbles after Valsalva manoeuvre. There was no atrial septal aneurysm associated. The shunt was considered small so no PFO closure was performed. Laboratory investigation for vascular risk factors showed normal results [total cholesterol (176 mg/dL), high-density lipoprotein cholesterol (77 mg/dL), low-density lipoprotein cholesterol (90 mg/dL), triglycerides (47 mg/dL) and fasting blood sugar level (79 mg/dL)]. There were no thrombophilic or autoimmune disorders detected (protein C, free protein S, antithrombin activity, activated protein C resistance, prothrombin G20210A mutation, homocysteine, lupus anticoagulant, anticardiolipin antibodies IgG/IgM, anti-beta-2-glycoprotein I IgG/IgM, antinuclear antibodies, anti-neutrophil cytoplasmic antibodies and rheumatoid factor).

### Discussion

CaW has been increasingly identified as a risk factor for ischemic stroke and TIA, and is considered a non-atherosclerotic disease [2]. Association of CaW to

atherosclerotic plaque is very rare and there are only a small number of case reports available [5,6]. As described in most series of CaW, our patient is female [1] and from African descent [2]. However she is much younger than the average patient presenting stroke or TIA in the presence of CaW [7,8]. In a retrospective review conducted by Hu and co-workers on patients presenting a TIA, the incidence of CaW at the symptomatic carotid bifurcation on CT angiography was 8.9%. The mean age was 58 years and most of the patients were female (n = 9/12;  $P < 0.015$ ) [7]. There were no other major risk factors identified in the majority of these patients and none showed a recent ischemic lesion on DWI [7], as was illustrated in our patient. In our case, we also found the presence of an atherosclerotic plaque associated with the CaW, which is a rare finding. To our knowledge only a few cases of atherosclerotic plaque associated with CaW have been published until today [5,6]. These cases show many differences with our patient. In both the patients were male and from Chinese descent, with age ranging from 50 to 60 years old, and presenting atherosclerotic risk factors [5,6]. Furthermore, our patient showed signs of intraplaque haemorrhage on T1FS imaging of the cervical vessels, which has not been described in the two cases mentioned above. This unique case shows that CaW associated to atherosclerotic plaque should be considered as a possible cause of TIA in the young adult, even in the absence of atherosclerotic risk factors. T1FS imaging of the cervical vessels allows identifying a potential intraplaque haemorrhage, with increased risk of ipsilateral stroke and therefore impacting treatment options, with urgent carotid endarterectomy being the preferred intervention compared to carotid stenting in this case.

### Conclusion

With this case report, we aim to raise awareness of carotid web possibly being associated to an atherosclerotic plaque, even in the young adult presenting symptoms of transient ischemic attack without recent ischemic lesion on DWI and in the absence of atherosclerotic risk factors. By adding T1 weighted fat-saturated imaging of the cervical vessels, we were able to identify an intraplaque haemorrhage, which is associated to an increased risk of ipsilateral stroke with potential implications on choice of treatment.

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**Ethics Statements:** patient signed informed consent for publication.

**Competing Interests:** none.

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