Case 12235

Eurorad

Blunt common carotid arterial injury resulted in dissection and myointimal hyperplasia

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Section: Interventional radiology
Area of Interest: Arteries / Aorta Cardiovascular system Contrast agents Interventional vascular Trauma
Procedure: Contrast agent-intravenous
Procedure: Diagnostic procedure
Procedure: Education
Imaging Technique: CT
Imaging Technique: CT-Angiography
Imaging Technique: Image manipulation / Reconstruction
Imaging Technique: MR
Imaging Technique: MR-Angiography
Special Focus: Biological effects Dissection Haemodynamics / Flow dynamics Case Type: Clinical Cases
Authors: E. Squillaci PhD, M. Antonicoli MD, S. Abrignani MD, E. Vasili MD, L. Neroni MD, I. Coco MD, G. Simonetti PhD
Patient: 47 years, male

Clinical History:

A 47-year-old female patient came to the emergency department due to a syncope lasting for 20 minutes.
Physical examination revealed tachycardia, hypertension (190/120 mmHg), mild strength deficit of left inferior limb with hypertone. These symptoms resolved within 3 hours.
Blood tests did not indicate any significant alteration.
The patient was hospitalized for further investigations.

Imaging Findings:

A Computed Tomography (CT) examination of the head in basal conditions didn't show any evidence of haemorrhage or ischaemic lesions.
A colour Doppler ultrasound examination of epiaortic arteries revealed an isoechoic left CCA wall thickening that arose from both intima and media layers that determined a not significant stenosis.
A CT angiography (CTA) of epiaortic and intracranial arteries showed a concentric hypodense thickening (2.5 mm) of the left common carotid artery (CCA) wall without contrast enhancement, extended from its origin to bifurcation (108 mm length), with occlusion of the ipsilateral external carotid artery. Contralateral CCA, bifurcation, external, internal carotid arteries and bilateral vertebral and cerebral arteries had a normal course, calibre and patency.
A brain Magnetic Resonance (MR), performed in basal conditions and after the administration of gadolinium, showed only bilateral small sub-cortical hyperintensities in T2 and the study with angiographic sequences confirmed

the CTA findings.

Discussion:

Blunt injury of CCA is an extremely rare vascular trauma which represents only 2.5% of all blunt cerebrovascular injuries [1]. Most frequent causes are motor vehicle accidents (MVA) [2, 3].
Dissections, occlusions, and transections represent common expressions of blunt carotid injuries (BCI) [4-8], a more rare event is lesion evolution into myo-intimal hyperplasia [9, 10].

Risk factors for BCI include skull and cervical fractures, Horner syndrome and seat belt sign [3, 11, 12]. Particularly, the last one is found in 5.8% of confirmed cerebrovascular injuries [13].
Early recognition of BCI is fundamental to avoid complications, such as ischaemic stroke [14], however, clinical manifestation may be delayed for even more than 10 years [10, 15].

Colour Doppler examination is a first-level not invasive technique with the advantages of both morphologically characterize CCA wall and to determinate stenosis hemodynamic significance[17]. In this case, images showed that wall thickening originated from intima and media as for hyperplasia and hypertrophy of these layers, not determining significant stenosis.
CTA is useful to establish wall density, thickening extension and any signs of arterial wall complications [16]. Our findings were compatible with a stable lesion, without contrast-enhancement suggesting no acute dissections, inflammation or haemorrhage. [16]
Magnetic resonance (MR) with high contrast resolution was also performed to evaluate whether the carotid injury could have caused ischaemic lesions not seen on CT.
The only finding of subcortical hyperintensities, associated with clinical presentation and side deficit, suggested a hypertensive-related rather than carotid-related TIA.
Vasculitis was excluded because there was no other clinical evidence such as fever, carotid pain, or rising of inflammatory biochemical markers [18].
Left CCA lesion did not have the typical string-of-beads-pattern as fibromuscolar dysplasia [19] and post-surgical myointimal hyperplasia were excluded by clinical history.
The patient initially did not report any history of trauma but, after asking for it specifically, she remembered that two years before she was involved in a MVA. She also reported an area of erythema in the left side of her neck the next day, corresponding to the seatbelt.
Therefore we hypothesized that neck compression caused by the seatbelt trauma might have caused an asymptomatic circumferential arterial dissection, with the formation of a sub-intimal haematoma and inflammation, cronically organized into myointimal hyperplasia.
Mortality in BCI is up to 23% with a high proportion of permanent neurological deficits [20].
Considering stability of myointimal hyperplasia, the patient was discharged after 3 days with anti-platelet therapy (ASA 200 mg/die) and diagnostic monitoring.
BCI is an enigmatic pathology that can rarely result in myo-intimal hyperplasia.
CTA, colour Doppler and MR imaging are safe and accurate techniques for the diagnosis.

Differential Diagnosis List: Left Common Carotid Artery BCI evolved into myointimal hyperplasia., Takayasu arteritis, Post-endoarterectomy myointimal hyperplasia, Fibromuscolar displasya

Final Diagnosis: Left Common Carotid Artery BCI evolved into myointimal hyperplasia.

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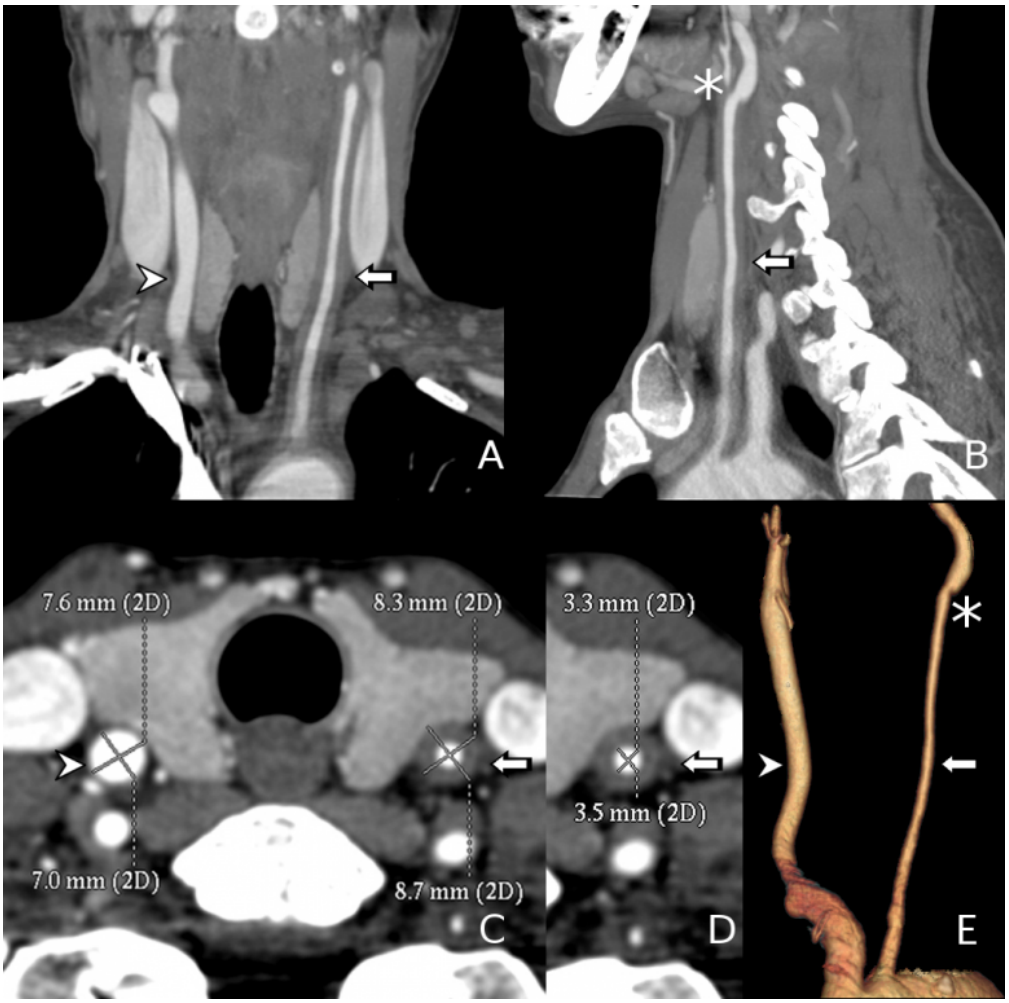
Figure 1

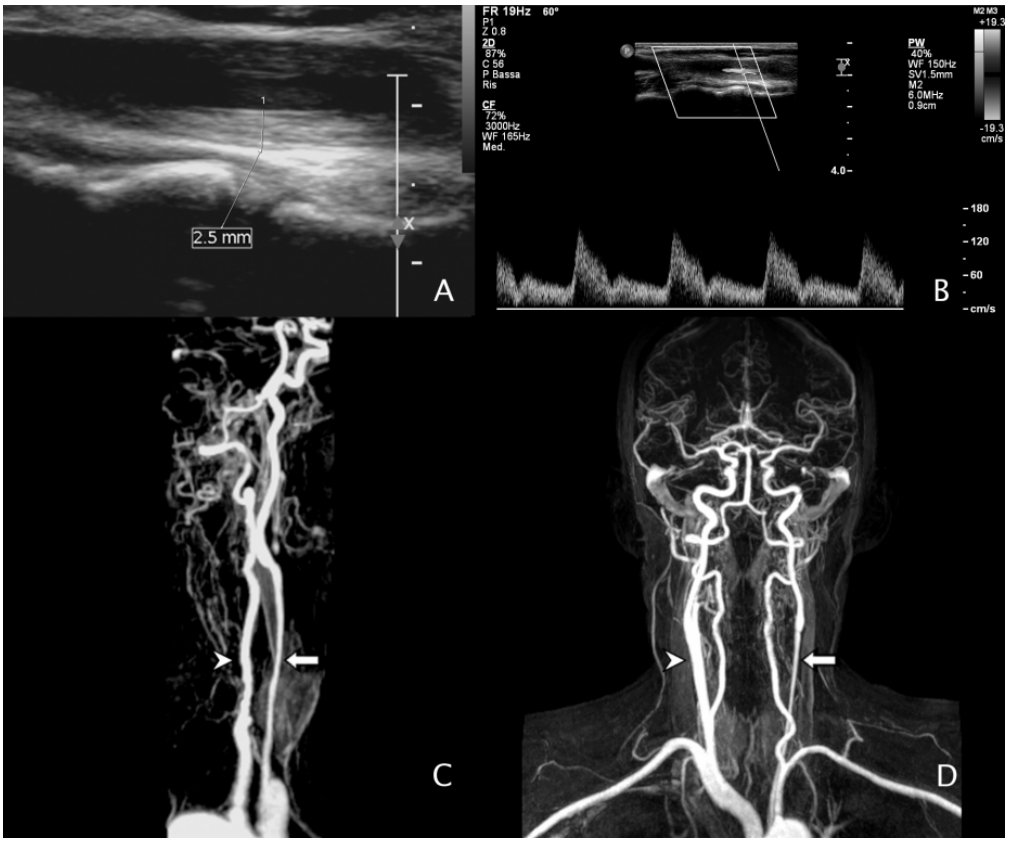
a



Description: CTA in multiplanar (A, B), axial (C, D) and VR (E) reconstructions showing left CCA concentric all-lenght thickening (arrows), ipsilateral ICA regular patency with occluded ECA (*). Regular course, calibre and patency of contralateral CCA (arrowheads). **Origin:** Department of Diagnostic Imaging, Molecular Imaging, Interventional Radiology and Radiation Therapy, University Hospital Tor Vergata, Rome, Italy.

Figure 2

a



Description: B-mode ultrasound (A) showing left CCA thickening, with haemodynamically not significant flow alteration at colour Doppler (B). AngioRM (C, D) showing narrowing in left CCA (arrows) with normal contralateral carotid arteries (arrowheads). **Origin:** Department of Diagnostic Imaging, Molecular Imaging, Interventional Radiology and Radiation Therapy, University Hospital Tor Vergata, Rome, Italy.