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Successful Stenting of a Symptomatic Carotid Artery Diaphragm

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Abstract: Carotid diaphragm (CD) is an atypical form of fibromuscular dysplasia (FMD), and an underestimated cause of recurrent ischemic stroke (IS). A 30-year-old woman with right hemiplegia caused by CD, treated successfully by percutaneous stenting, with favourable outcomes. CD is a rare cause of recurrent IS which can be detected by non-invasive imaging. Endovascular treatment appears to be an attractive therapeutic option in stroke secondary prevention.
Keywords: carotid diaphragm, ischemic stroke, arteriography, percutaneous stenting.

INTRODUCTION

The CD is a rare and unknown cause of ischaemic stroke (IS) [1]. Described for the first time in 1967 by Ehrenfeld [2], Historically, diaphragms had been known as a fibromuscular hyperplasia, atypical fibromuscular dysplasia, atypical fibromuscular hyperplasia, septal fibro- muscular dysplasia, septa, diaphragms, webs and pseudo-valvular folds [3].

In most cases, the diagnosis of CD was based on angiography and it appears as a translucent endoluminal filling defect [4], on the posterolateral side of the carotid bulb, just beyond the carotid bifurcation [5]. The CD is associated with a high risk of thrombo-embolic recurrent events. in case of CD; a radical treatment is possible and the diagnosis of this entity must be considered In every recurrent stroke case.

We report a case of a CD treated successfully by stenting in stroke secondary prevention.

CASE REPORT

A 30-year-old woman was admitted after a sudden onset right hemiplegia, and dysarthria. She had no risk factors for atherosclerosis. Cerebral scan showed a large infarction in the territory of the left middle cerebral artery (Fig-1).

A duplex color-ultrasound imaging was performed revealing endoluminal lesion which lead to suspect a CD (Fig-2), although turbulence at low speed was evident downstream of the spur.

The angio Computed tomography scan (CT) of the supra-aortic trunks and the Willis polygon demonstrated an image of endoluminal subtraction of the left carotid bulb, larger than high, non-stenosing according to the criteria of "North American Symptomatic Carotid Endarterectomy Trial" (NASCET) (Fig-3). It first evoked a carotid diaphragm. No other stroke's etiology has been found.

2 weeks later, selective carotid and cerebral angiography confirmed the diagnosis of a non-stenosing carotid diaphragm more associative to blood stagnation in the spur formed by the diaphragm and the wall of the artery (Fig-4).

The clinical evolution was favourable with partial recovery. The involvement of the carotid diaphragm in IS was established, and the indication of left carotid stenting in secondary prevention was decided. aspirin was started, to which was added Clopidogrel 75 mg 10 days before the gesture, the effectiveness of the anti-aggregation was tested the day before the intervention. Stenting was Practiced 3 weeks after the stroke by right arterial femoral route with a long introducer 6 French/90 cm which was placed in the left primary carotid artery. The left carotid diaphragm was crossed with a 0.014-inch Transend® microguide, and a 9 mm diameter, 40 mm long Carotid Wallstent® self-expanding stent was deployed.

The arteriographic control showed the persistence of the filling defect as well, another self-expanding conical stent was set up with a satisfying result (Fig-5). The gesture was simple, with no neurological complications per-post-procedure.

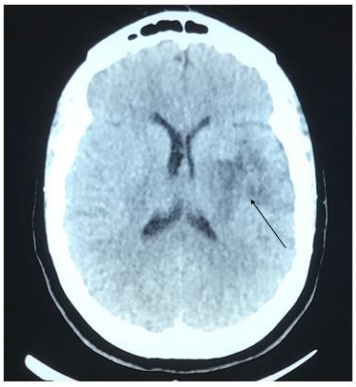

Fig-1 : Cerebral scan showing a large infarction in the territory of the left middle cerebral artery

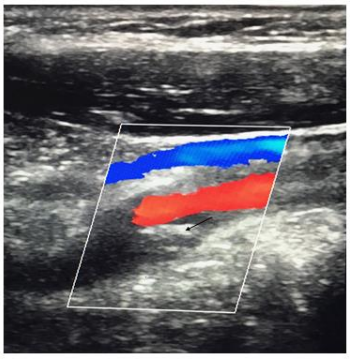

Fig-2: Color duplex ultrasound demonstrated a diaphragm (black arrow) in the left CCA

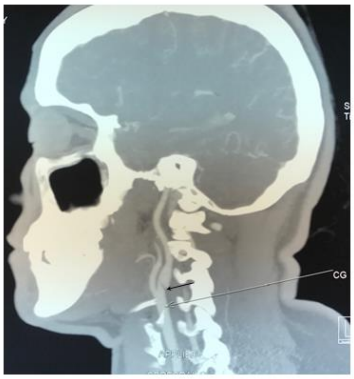

Fig-3: Angio CT demonstrating a non stenosing substraction image of the posterior wall of the carotid (black arrow)

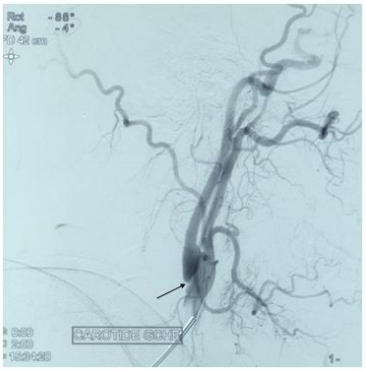

Fig-4: digital subtraction angiography showing a non-stenosing carotid diaphragm associative to a blood stagnation in the spur formed by the diaphragm and the wall of the artery (black arrow)

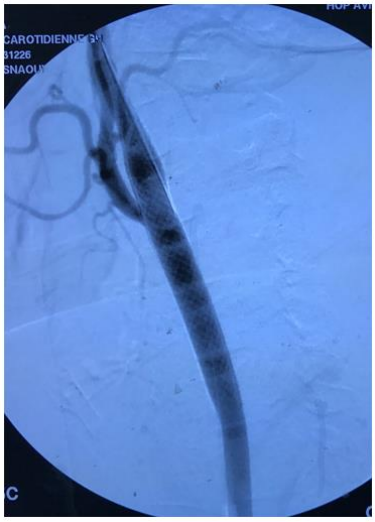

Fig-5 : digital subtraction angiography showing a succesful carotid stenting with disappearance of the web and the blood stagnation

DISCUSSION

CD is a rare cause of recurrent ischemic stroke, peculiarly in middle-aged patients. Literature concerning CD is poor, with only few cases since its first description in 1967 by Ehrenfeld [2].

In the literature the prevalence is variable. Choi et al reported a prevalence of 1% in a series of 576 cervical angiographies [6]. A higher prevalence has been reported among the Afro-Caribbean population [5]. The average age of diagnosis was from 45 years to 50 years in the series of Joux [3, 6, 7], however the diagnosis at a later age was reported by other authors [6]. In our case the discovery of the disease was made at a relatively younger age.

Conventionally the CD is defined angiographically as a linear filling defect and a thin tansluscent endoluminal web that does not change with the head positions, localized in the posterior wall of the carotid bulb.

Computed tomography seems to be the method of choice for detecting the CD and it appears as a protruding spur in the lumen of the vessels. However, the typical appearance of diaphragm can be temporarily masked by the presence of a thrombus [6, 8]. MRI show an irregular, non-characteristic filling[3, 9].

Ultrasound-Doppler has a low sensitivity for Detection of carotid diaphragms. It can detect indirect signs of stenosing lesions, which is rarely the case [3, 8]. The presence of calcifications or atherosclerosis on cervical or cerebral arteries excludes the diagnosis of CD [5].

In our case the diagnosis was strongly suspected by the Doppler and confirmed by the angio computed tomography scan (CT) of the supra-aortic trunks and arteriography.

The CD, even if non stenosing, as in our case, is associated with a high risk of recidive in the same territory. This risk is estimated at 20% over 30 months [1, 6, 8], which justifies an etiological treatment in stroke secondary prevention.

Antiplatelet therapy alone does not appear to be associated with a decreased risk of recurrence in symptomatic patients [9] although it remains a reasonable therapeutic option in asymptomatic patients.

Surgery (endarterectomy or complete excision of the dysplasic segment) has been proposed by several authors with good long term results [1]. Joux *et al*. reported a prevalence of IS which decreased from 20% to 0% under surgical treatment compared to antiplatets alone [6, 8]. Surgery also has the advantage of providing histological confirmation of the disease [1, 6, 8].

Another author suggests stenting [3], which is less invasive, because it is a pathology of the young adults and therefore no technical difficulty (no risk in endovascular navigation in the absence of atheromatous lesions) [1].

In the absence of studies comparing surgery to endovascular therapy, the choice of technique must take in consideration the field, the anatomical particularities, the operator and patient's choice.

CONCLUSION

The non-stenosing CD is a rare cause of recurrence IS of the young adults to know, its diagnosis can be suspected by the Doppler, and confirmed by angiography or angio-CT or angio-MRI.

The high risk of IS recurrence seems to justify radical treatment either by surgery or endovascular therapy with favourable long term results.

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