

A typical example of cerebral watershed infarct

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Abstract

Watershed infarcts (WI) evolve in hemodynamic risk zones. Clinical picture of WI can be associated to partial epileptic seizures. Diffusion weighted brain magnetic resonance imaging (MRI) allows a clear diagnosis. WI pathogenesis involves either embolic or hemodynamic mechanism. A 69-year old patient presented with sub-acute occurrence of right hemiparesis and partial epileptic seizures of the right arm. Carotid ultrasounds demonstrated occlusion of the right extra-cranial internal carotid artery (ICA) and tight stenosis of the contralateral ICA. Brain Diffusion-Weighted magnetic resonance revealed acute ischemic lesions within the watershed area of the left hemisphere. Our case supports the hypothesis of impaired washout of emboli in low-perfusion brain areas as the mechanism underlying cortical WI.

Introduction

Cerebral watershed (or border zone) infarcts (WI) involve the junction of the distal fields of two non-anastomosing arterial systems, which are hemodynamic risk zones. There are two types of WI: cortical WI, occurring at the junction between cortical territories of the anterior, middle and posterior cerebral arteries; and internal WI, occurring in the white matter between the deep and superficial perfusion systems of the middle cerebral artery.¹ Clinical picture of watershed infarctions is often progressive or fluctuating; symptoms may be mild and are therefore probably underestimated.¹ Another characteristic feature is occurrence of early-onset partial seizures, which in cortical WI occur more often than in other forms of stroke.² Pathogenesis remains not completely understood and involves either embolic or hemodynamic mechanism.¹ Due to its sub-acute onset and to the association to partial clonic seizures, WI diagnosis can be challenging for physicians. Nevertheless, WIs account for up to 10% of all cerebral infarcts³ and they must be kept under

consideration in order to avoid inappropriate treatment in the acute phase. In this phase, diffusion weighted brain MRI (DWI) allows a clearer diagnosis than cranial computed tomography (CT).⁴

Case Report

We report on the case of a 69-year-old man who presented to the emergency department of our hospital four days after the sub-acute occurrence of homonymous right hemianopia and right hemiparesis. The symptoms had been worsening progressively over the last four days. The patient's medical history disclosed smoking habit (78 pack years beginning at an age of 16 years). One year earlier, a non-small cell lung carcinoma was diagnosed and treated consequently with 6 cycles of palliative chemotherapy (carboplatin and gemcitabin). The patient wasn't on antiplatelet therapy. Neurological examination on hospital admission revealed paresis of the right upper limb and a severe motor deficit of the right lower limb with intact sensibility. Eyes and head deviation towards the left side and right hemi-inattention were present. Brain CT scan showed a hypodense focal lesion in the left prerolandic area. The patient was admitted with a diagnosis of ischemic stroke. And was started on secondary prevention therapy according to international guidelines for ischemic stroke treatment^{5,6} with acetylsalicylic acid 300 mg daily.

A few hours after hospital admission, the patient developed partial epileptic seizures of the right upper limb, which required IV phenytoin infusion.

Carotid ultrasounds demonstrated occlusion of the right extra-cranial carotid artery and tight stenosis of the contralateral internal carotid artery (Figure 1A, B).

Brain Diffusion-weighted magnetic resonance (DWI) revealed multiple hypertintense lesions extending within the cortical watershed area of the left hemisphere, suggesting a large watershed infarct (Figure 1C, D, E).

Over the following days, the patient had a nearly full clinical recovery, with a residual slight motor deficit of the distal right upper limb at discharge. Surgical or percutaneous treatment of left carotid stenosis was excluded in consideration of his advanced neoplastic disease; he continued adequate medical treatment with antiplatelet therapy acetylsalicylic acid 300 mg daily and statin. He experienced no further seizures and he was discharged on antiepileptic therapy with phenytoin.

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Discussion

We report on a typical neurological picture of cortical WI, which represents a complex clinical entity. Our patient sought for medical help only four days after symptoms onset, due to slow progression of the neurological deficit. This was associated to partial epileptic seizures of the upper limb, which are common in cortical WI and display a good response to antiepileptic treatment. Another typical feature displayed by the present case was the benign clinical course of the ischemic event, which is often seen in cortical WI.⁷ Sub-acute onset and association to partial seizures could make the diagnosis of stroke quite challenging. DWI proved to be the most useful imaging technique in order to make a right diagnosis in the acute phase of the disease and to understand its pathogenesis.⁴

Based on the fact that WI may occur during marked systemic hypoperfusion, the supposed pathological mechanism is hemodynamic failure. This theory is supported by the typical localization of WI in low-perfusion cerebral areas, also called *distal* or *border zone* fields. Moreover, syncope at WI onset, which is occasionally reported, might support the hemodynamic hypothesis. As opposed to the hemodynamic hypothesis, either cardiac or artery-to artery or microembolism have been suggested as a possible cause of WI. Various neuropathological and imaging studies found multiple embolic occlusions in the terminal vascular field distal to atherosclerotic carotid plaques,^{8,9,10} in particular in the cor-

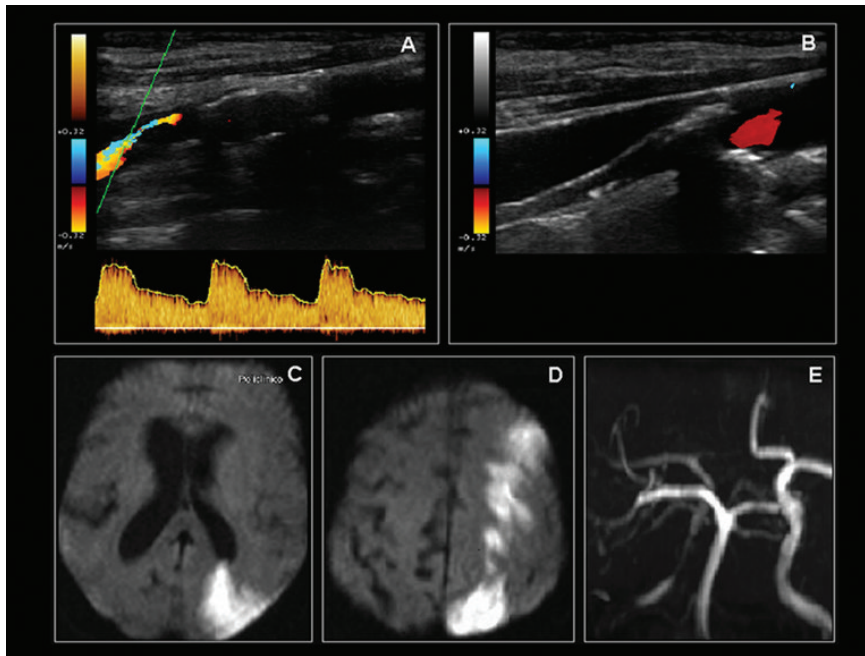


Figure 1. Duplex scan showing (A) tight stenosis of the left extra-cranial internal carotid artery and (B) occlusion of the right internal carotid artery. Diffusion weighted brain-magnetic resonance imaging (MRI) showing watershed infarcts within the left hemisphere (C,D); MR angiography showing hypo-perfusion of the right intra-cranial internal carotid artery (E).

tical form of WI.⁷ Anyway, a combination of both theories is likely to be the most realistic mechanism, in terms of impaired washout of emboli in case of decreased perfusion.^{11,12}

Our patient presented with a cortical WI distal to a tight stenosis of the left internal carotid artery associated to occlusion of the right one. Hence, hypoperfusion could definitely play a substantial role in the genesis of the reported case. Nevertheless, we suggest that this case could support the hypothesis of an impaired washout of emboli in low-perfusion brain areas.^{11,12} Indeed, if hypoperfusion was to be the dominant mechanism of the ischemic lesion, watershed area distal to the occluded carotid artery should to have been at even higher risk. On the other hand, our case keeps in line with the concept that embolic more than hemodynamic mechanism plays a crucial role in the pathogenesis of cortical WI.¹² Carotid reperfusion would have been an option in this case, although medical therapy alone was the preferred choice due to the high risk of the procedure in this patient and his global prognosis, due to his underlying disease.

Conclusions

WI are a frequent clinical entity, accounting for up to 10% of all cerebral infarcts,³ although they are often under-recognised due to slow progression of the presenting symptoms and to the frequent association to seizures.¹ Our case is a very illustrative example of cortical WI with typical onset, course and imaging appearance. We suggest that it supports the hypothesis of impaired washout of emboli in low-perfusion brain areas in cortical WI; treatment should be chosen accordingly.

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