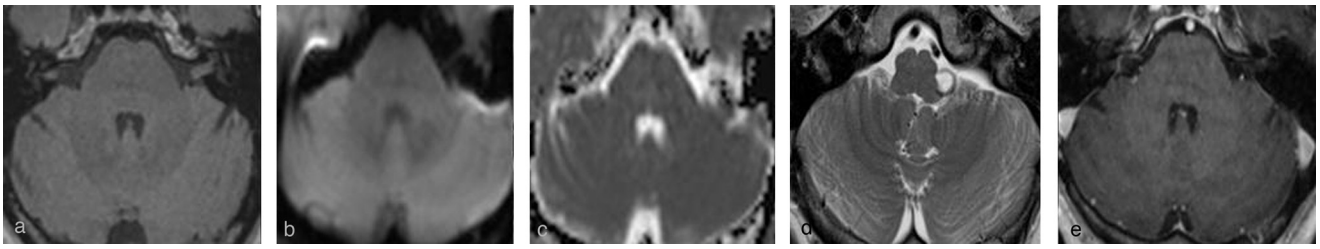

Figure 2 Follow-up axial FLAIR (a) and T2-weighted (d) MR images a month later show the disappearance of the pathological hyperintensities in the pons, medulla oblongata and cerebellum, and normal signals on diffusion (b) and ADC (c) images, with no contrast enhancement (e).

on lesion distribution, the presence (or predominance) of vasogenic (rather than cytotoxic) edema and the patient's clinical history.

Treatment consists of blood pressure normalization and control of the underlying disorder. However, there are recurrent forms of PRES (due to insufficient blood pressure control or continued immunosuppressive treatment) [1,5].

Conclusion

PRES may manifest at atypical locations as in the present case. Clinical history and follow-up imaging studies are crucial for making the correct diagnosis.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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Giant cell arteritis with severe bilateral involvement of the intracranial arteries

Introduction

Giant cell arteritis (GCA) is a vasculitis characterized by granulomatous inflammation in the wall of medium-size and large arteries and preferentially affects extracranial branches of the carotid artery [1]. Cerebrovascular events in active GCA are rarely due to the arteritic involvement of intracranial arteries [2].

Case report

A 68-year-old man with a history of polymyalgia rheumatica for 4 years and hypertension was admitted to our Stroke unit because of sudden right hemiparesis. On admission, the patient reported an unusual headache 2 weeks before with scalp hyperesthesia during 6 days and right amaurosis fugax a few days ago.

Cerebral brain magnetic resonance imaging (MRI) showed multiple, bihemispheric and recent small cerebral infarcts, located in the perfusion areas between the middle and anterior cerebral arteries territories (Fig. 1A). MR and CT angiography revealed pre-occlusive stenoses of both internal carotid arteries (ICAs) located in the siphons (Fig. 1B) and multifocal low-grade stenoses of both vertebral arteries. These stenoses were confirmed by cerebral angiography (Fig. 2A), which also revealed narrowing and dilatations of the external right carotid artery (Fig. 2B). There was no biological inflammatory syndrome but analysis of the cerebro-spinal fluid revealed a hyper-proteinorachia (1.14 g/L) without cells.

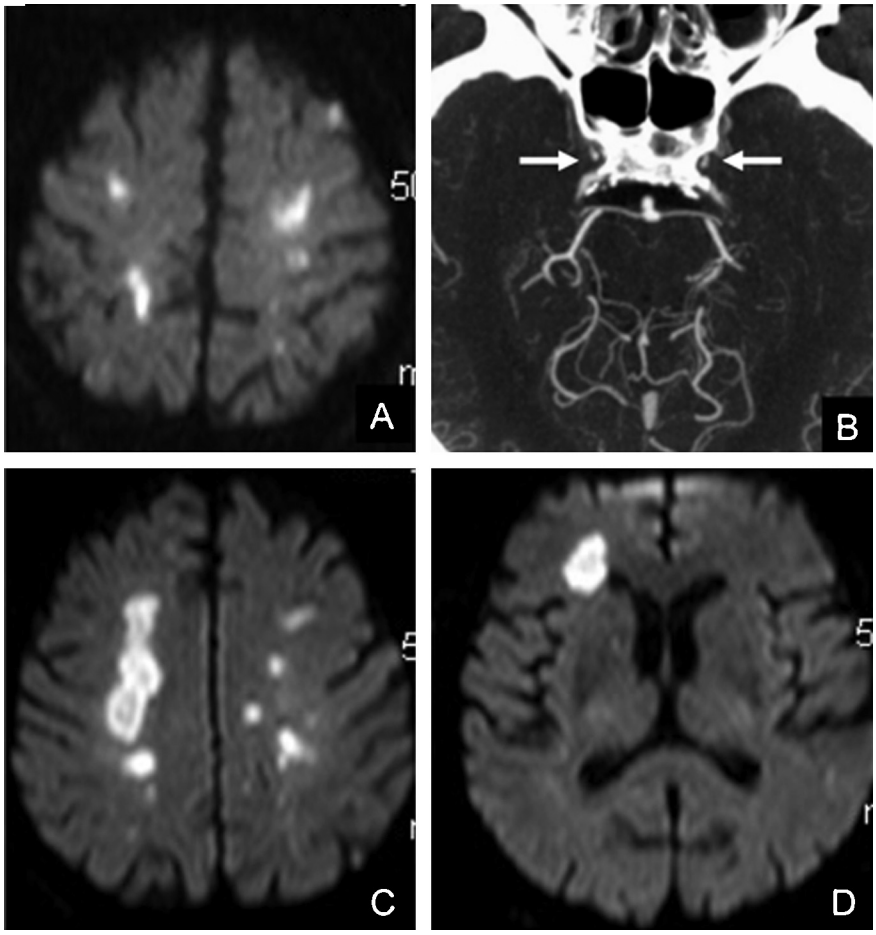

Figure 1 A. Axial diffusion-weighted cerebral MRI demonstrates acute multiple, bihemispheric and small cerebral infarcts. B. CT angiography reveals pre-occlusive stenoses of both internal carotid arteries located in the siphons (arrows). C and D. New axial diffusion-weighted cerebral MRI, 2 weeks after the first one, shows new and larger ischemic lesions.

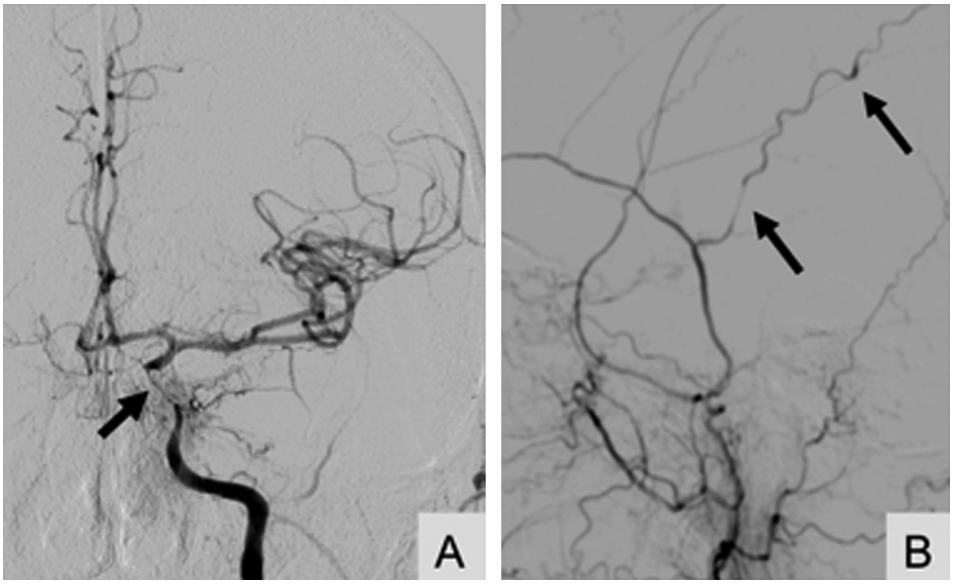

Figure 2 A. Digital subtraction angiogram of the left internal carotid artery confirms the pre-occlusive stenosis (arrow). B. Digital subtraction angiogram of the right external carotid artery reveals segmental narrowing and dilatations (arrows).