Contrast-induced encephalopathy possibly secondary to endothelial damage after successful mechanical thrombectomy☆

Encefalopatía por contraste secundaria a posible daño endotelial tras trombectomía mecánica exitosa

Dear Editor:

The implementation of acute stroke protocols[1] is leading to more extensive use of neurointerventional techniques. Patients with increasingly complex conditions are eligible for this type of treatment, giving rise to new clinical scenarios. We present a case of encephalopathy with onset following successful mechanical thrombectomy associated with imaging evidence of blood—brain barrier (BBB) disruption, which fully resolved both clinically and radiologically. Our patient was an 82-year-old woman with history of hypertension, diabetes, dyslipidaemia, anticoagulation therapy with acenocoumarol for atrial fibrillation, and stage 3B-4 chronic kidney disease due to diabetic nephropathy. The patient consulted due to impaired language production and right hemiparesis. At the time of arrival at hospital, she presented arterial pressure of 150/92, National Institute of Health Stroke Scale (NIHSS) of 17, International Normalised Ratio (INR) of 1.89, and the previously known kidney failure. A computed tomography (CT) scan (Fig. 1A—C) revealed an extensive ischaemic penumbra in the territory of the left middle cerebral artery (MCA), with no signs of established ischaemia with M1 occlusion. The patient underwent primary thrombectomy as fibrinolysis was contraindicated (INR > 1.7); complete recanalisation (TICI grade 3) was achieved after one pass of a Penumbra Ace® distal aspiration catheter (Fig. 1D—E). Her condition did not improve, with arterial pressure increasing to 226/100. A brain CT scan performed 2 hours after recanalisation (Fig. 1F—G) showed contrast extravasation in the left hemisphere, which led us to a diagnosis of encephalopathy due to BBB rupture secondary to hypertensive crisis and contrast toxicity (iohexol at 350 mg/mL). She subsequently developed secondarily generalised focal seizures in the left hemisphere, which did not resolve after administration of 10 mg of diazepam and 750 mg of intravenous phenytoin; she was therefore administered analgesia/sedation and levetiracetam. A brain CT scan performed at 24 hours (Fig. 1H) revealed disappearance of the contrast and signs of oedema in the left hemisphere, with no established infarction. Several electroencephalograms ruled out epileptic activity and therefore phenytoin was suspended. The patient was extubated without incident. A brain CT scan performed 8 days after onset showed no evidence of stroke. The patient was discharged without symptoms (NIHSS: 0) with apixaban adjusted to her renal function prescribed for secondary prevention; levetiracetam was suspended. A brain magnetic resonance imaging study showed no residual ischaemic lesion. At 3 months, she presented a score of 0 on both the NIHSS and the modified Rankin Scale. Multiple reported cases of encephalopathy due to disruption of the BBB may be included on the spectrum of posterior reversible encephalopathy[2] and hyperperfusion syndrome.[3] Although they share some characteristics, our case presents peculiarities, especially its manifestation after thrombectomy with complete recanalisation. Thrombectomy may have been the cause, as it produces endothelial damage,[4] which would trigger disruption of the BBB and subsequent contrast extravasation, which is exacerbated in patients with kidney disease. This type of damage would involve the effect of reperfusion and the toxic effect of contrast, in some patients (especially those with kidney failure). This would occur in previously hypoperfused areas where arterial pressure alterations would play a fundamental role, as in the case of hyperperfusion syndrome after carotid revascularisation.[3] Some studies postulate that contrast extravasation following neurointervention may be related with an increase in intracranial bleeding.[5,6] A case of hyperperfusion after thrombectomy with poor progression has also been described.[7] Reported cases of hyperperfusion after rtPA are more numerous.[8] Some cases have been reported of contrast-induced encephalopathy[9] after such angiographic procedures as cardiac catheterisation,[10] characterised by disruption of the BBB with contrast extravasation to the extravascular space; this is generally reversible within 72 to 96 hours. We have not found similar reports to our case in the literature; our most striking finding is the complete recovery observed, with no residual ischaemia despite the patient's age and the initial clinical severity. This complication, despite being infrequent, should be considered in interventional procedures, especially in those patients at greater risk of BBB damage: risk factors include older age, haemodynamic instability, and kidney disease (especially diabetic nephropathy). It is important to consider this entity in cases of clinical deterioration following neurointervention, once haemorrhagic complications have been ruled out. The measures to be taken include controlling blood pressure, eliminating associated factors, and adding antiepileptics when necessary; these may be suspended in the medium term if there is no structural lesion.[2] Clinical and radiological recovery may be expected within 72—96 hours.

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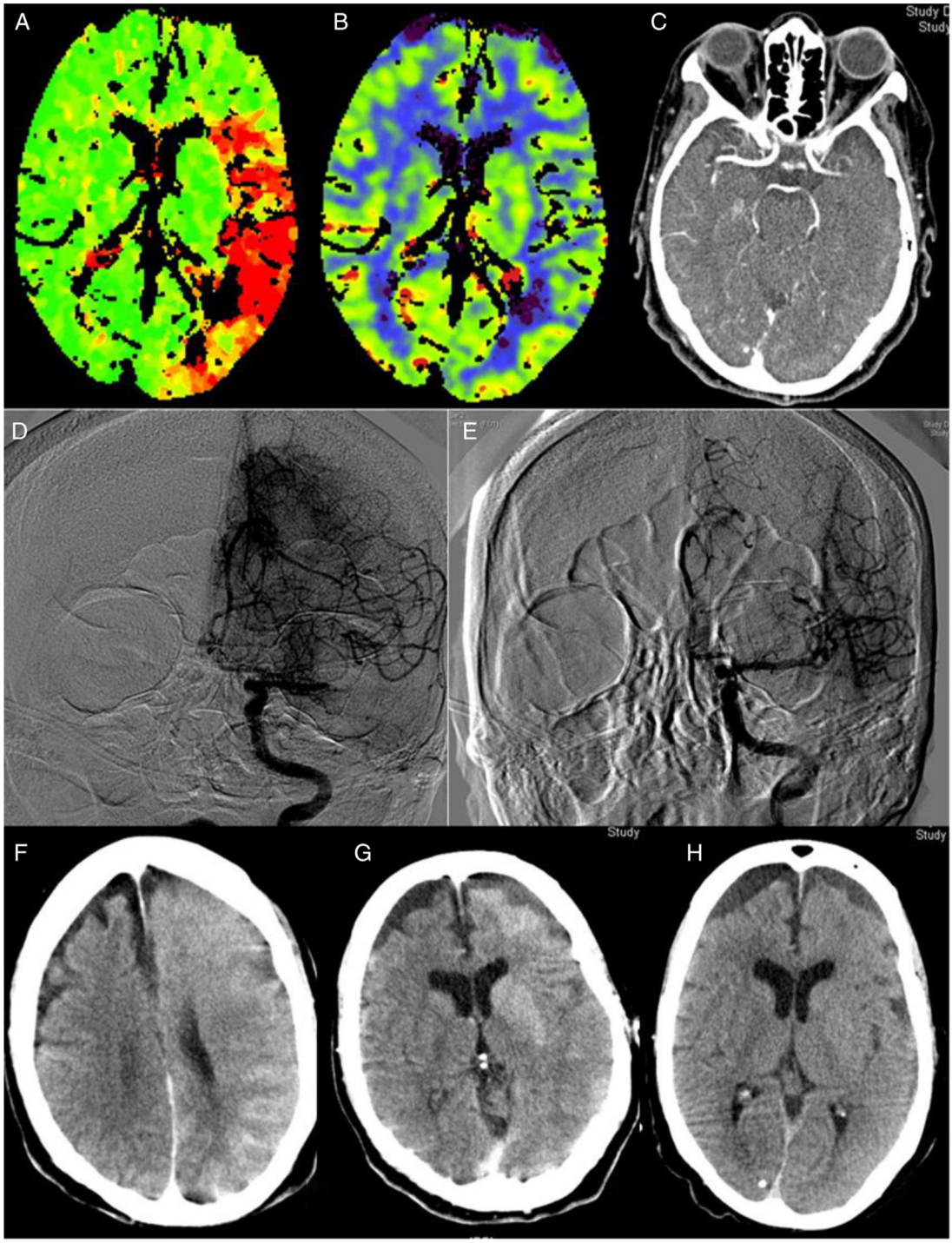

Fig. 1 Perfusion CT (A and B): increased time to peak (A) in an extensive area of the left MCA territory, with no established ischaemia on the cerebral blood volume map (B). CT angiography (C): occlusion at the left proximal M1 segment. Angiography: occlusion at the left proximal M1 segment (D) with complete recanalisation after one pass of a Penumbra Ace® distal aspiration catheter (E). CT scan after thrombectomy (F and G): generalised contrast extravasation in the left hemisphere. CT scan at 24 hours (H): signs of oedema in the left hemisphere with disappearance of the accumulated contrast and no signs of established ischaemia.

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Why does depression worsen progression and treatment response in the most frequent neurological disorders? Implications for clinical practice☆

¿Por qué la depresión empeora el curso y la respuesta al tratamiento en los trastornos neurológicos más frecuentes? Implicaciones en la práctica clínica

Dear Editor:

It was with great interest that we read the recently published study by Robles Bayón and Gude Sampedro[1] on the prevalence of behavioural and psychiatric symptoms, particularly anxiety and depression, in patients from a cognitive neurology clinic. Approximately one in every 2 to 3 patients with the most frequent neurological disorders presents depression at some point over the course of the disease.[2] However, numerous studies suggest that depression may also precede neurological disease and even affect its progression, including the response to pharmacological treatment.

In the case of epilepsy, 3 population studies have shown that patients with primary depression are 2 to 7 times more likely to develop epilepsy, and that those with epilepsy are 3 to 5 times more likely to develop depression.[3] Likewise, presenting depression (alone or combined with other psychiatric disorders) before epilepsy onset influences disease severity and treatment: patients with depression are twice as likely to develop drug-resistant epilepsy,[4] and are at greater risk of presenting seizures at one year[5]; they even show poorer response to surgical treatment for refractory temporal lobe epilepsy.[6]

High depression scores may be expected in patients with migraine, although an inverse correlation has also been observed. In a cohort study including nearly 1200 patients with migraine, patients with severe headache, and controls, presence of major depression during a 2-year follow-up period predicted the first-onset migraine (OR = 3.4; 95% CI, 1.4-8.7), but not other types of headache.[7]

Regarding cerebrovascular accidents, a meta-analysis of 28 prospective studies of a total of 320 000 individuals, including 8478 patients with stroke, showed that a history of depression was associated with increased risk of stroke (adjusted hazard ratio [HR] = 1.45; 95% CI, 1.29-1.63).[8]

A similar bidirectional relationship has also been observed in dementia. In a meta-analysis of 20 studies including approximately 100 000 individuals, 19 studies reported increased risk of Alzheimer disease among individuals with a history of depression; the risk also increased

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