[b] *Departamento de Medicina, Facultad de Medicina, Universidad Complutense, Madrid, Spain*
[c] *Centro de Investigación Biomédica en Red sobre Enfermedades Neurodegenerativas (CIBERNED), Madrid, Spain*
[d] *Instituto de Investigación, Hospital 12 de Octubre (i+12), Madrid, Spain*

* Corresponding author.
E-mail address: jbenitol67@gmail.com (J. Benito-León).
2173-5808/

Focal cortical dysplasia in a three years old patient with epilepsia partialis continua☆,☆☆

Displasia cortical focal en paciente de 3 años con epilepsia parcial continua

Introduction

Epilepsia partialis continua (EPC) is characterised by spontaneous regular or irregular clonic movements of a specific part of the body and affecting facial muscles or distal muscles of the limbs especially, although they may also affect the chest or abdomen. These movements may be continuous or appear in recurrent intervals of a few seconds for at least 1 hour and up to several days or weeks, and they may be aggravated by activity or external stimuli.[1,2] Jerking movements affect agonist and antagonist muscles simultaneously. Although patients usually remain conscious, they often display marked postictal weakness. Epileptic activity in EPC is generated in the motor cortex or adjacent areas.[3,4]

In children, the most frequent cause of EPC is Rasmussen encephalitis; other causes include tumours, vascular lesions, infections, and metabolic disorders. In the past years, the number of patients with EPC due to cortical dysplasia (CD) has increased.[5,6] We present a case of focal CD manifesting as EPC.

Clinical case

Medical history and prodrome

Our patient was a 3-year-old boy with no medical history of interest except for non-isoimmune neonatal jaundice, which required no phototherapy. He displayed normal psychomotor development. According to his parents, 3 weeks before symptom onset, our patient had displayed behavioural changes (irritability with no apparent cause) and slept more hours than usual. One week before symptom onset he showed symptoms of viral infection and common cold and experienced nausea but was afebrile.

Symptoms

He was brought to hospital due to an episode of right-sided deviation of head and eyes and head nodding lasting a few seconds. On the same day, he experienced a similar episode while awake and another while asleep. During this last episode, he sat up with his upper limbs in flexion and displayed right-sided deviation of head and eyes and head clonus, which resolved spontaneously after a few seconds. After this episode, our patient fell onto his back and was drowsy. The frequency of these episodes increased in the following days.

Treatment and progression

We started treatment with valproic acid and subsequently added intravenous levetiracetam and phenytoin. However, seizures increased and became continuous. At this point, our patient received midazolam infusion and was put into a barbiturate-induced coma for seizure control. In addition to these measures, our patient began a ketogenic diet. He also received intravenous lacosamide, which led to a decrease in the number of seizures. The video-EEG trace, however, revealed epileptiform activity which was nearly continuous in the frontal region of the left hemisphere and maximal in the frontopolar region; signs of focal motor status epilepticus were seen in the oro-lingual-facial region and the right hand. These findings were compatible with EPC (Fig. 1). We conducted several complementary tests to determine the cause of EPC (serology test, tests for neurotropic viruses and oligoclonal bands in CSF, test for serum and CSF NMDA-receptor antibodies, metabolic study, karyotyping, and MRI on 2 occasions); test results were all normal. A subsequent 3-T MRI scan revealed findings suggestive of focal dysplasia at the bottom of the left superior frontal sulcus; the PET scan displayed hypermetabolism in that area (Fig. 2).

Given that our patient had drug-resistant epilepsy, we opted for emergency surgery. We performed left frontal lobectomy to remove the area displaying CD. The anatomical pathology study confirmed the presence of type IIa focal CD.

A follow-up MRI scan performed 6 months after surgery revealed no remnants of dysplastic tissue. The patient was

☆ Please cite this article as: Castaño de la Mota C, Conejo Moreno D, García Fernández M, García Peñas JJ. Displasia cortical focal en paciente de 3 años con epilepsia parcial continua. Neurología. 2017;32:478—480.

☆☆ Clinical case presented in a moderated poster session at the 35th Annual Meeting of the Spanish Society of Paediatric Neurology, held in Granada, Spain.

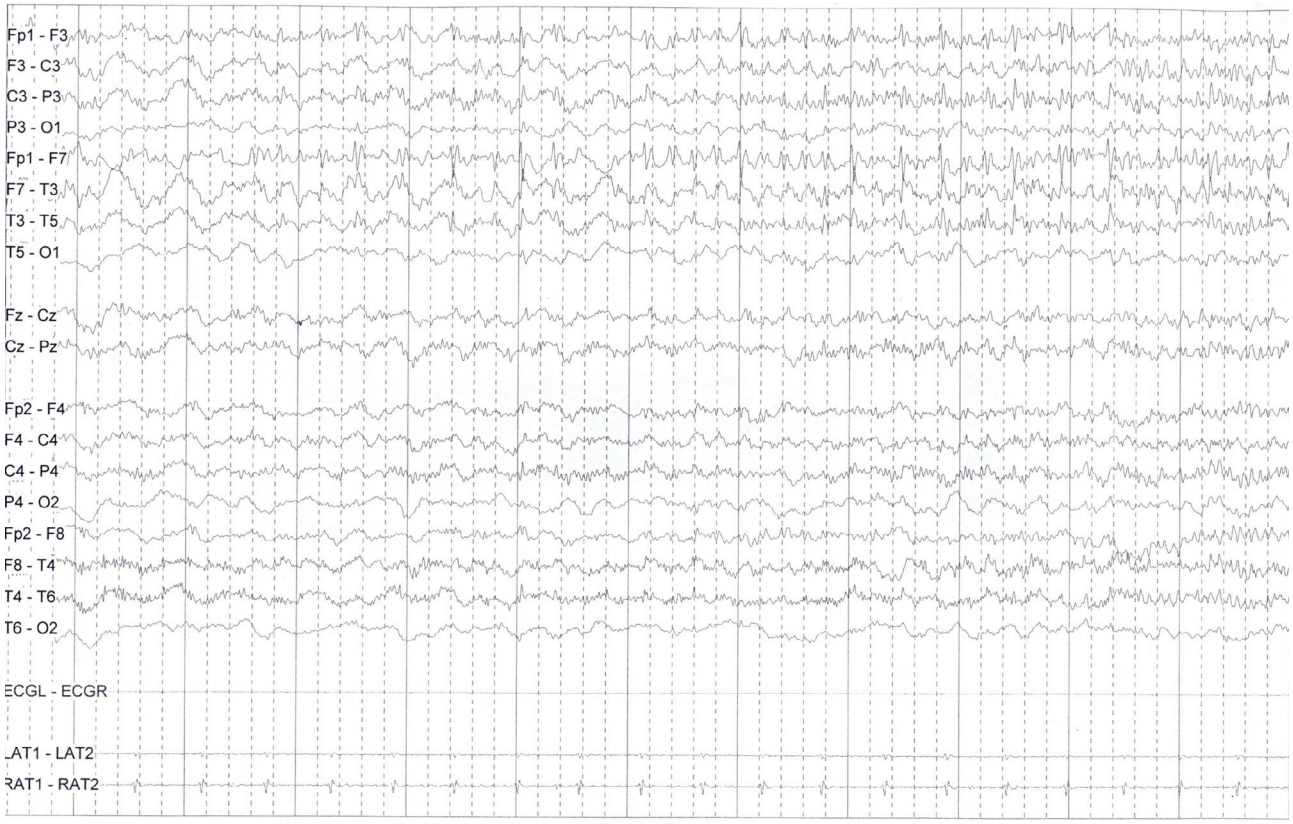

Figure 1 Video-EEG. Continuous epileptiform activity on the left frontal region in the form of 5-Hz rhythmic spikes (EEG activity recorded during barbiturate-induced coma).

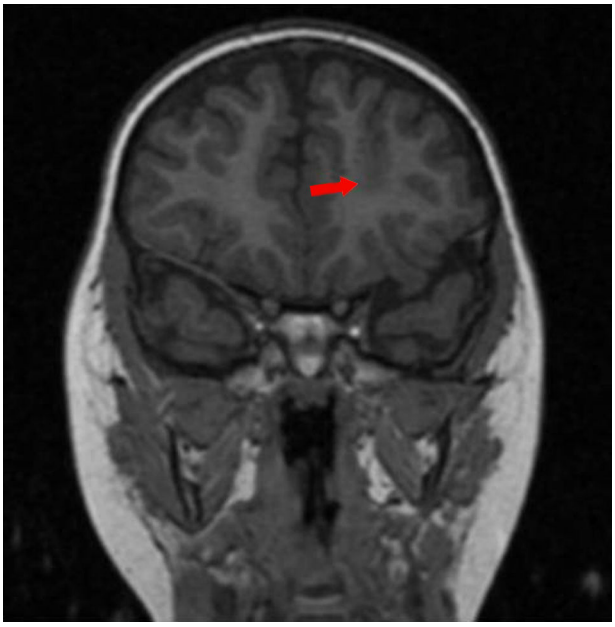

Figure 2 High-resolution (3-T) T1-weighted MRI scan (coronal section) revealing dysplasia at the bottom of the left superior frontal sulcus (loss of differentiation between grey and white matter).

seizure-free. At our most recent follow-up consultation, he was receiving a single antiepileptic agent.

Discussion

Improvements in neuroimaging and the development of 3-T MRI have opened up new possibilities for diagnosing some lesions, such as CD, which cannot be detected using conventional MRI.

As demonstrated by our patient, surgery during the acute phase may be an effective treatment approach in cases of drug-resistant epilepsy secondary to localised lesions.

References

1. Sinha S, Satishchandra P. Epilepsia partialis continua over last 14 years: experience from a tertiary care center from south India. Epilepsy Res. 2007;74:55—9.
2. Bien CG, Elger CE. Epilepsia partialis continua: semiology and differential diagnoses. Epileptic Disord. 2008;10:3—7.
3. Pandian JD, Thomas SV, Santoshkumar B, Radhakrishnan K, Sarma PS, Joseph S, et al. Epilepsia partialis continua: a clinical and electroencephalography study. Seizure. 2002;11:437—44.
4. Guerrini R. Physiology of epilepsia partialis continua and subcortical mechanisms of status epilepticus. Epilepsia. 2009;50:7—9.
5. Tezer FI, Celebi O, Ozgen B, Saygi S. A patient with two episodes of epilepsia partialis continua of the abdominal muscles caused by cortical dysplasia. Epileptic Disord. 2008;10:306—11.

6. Misawa S, Kuwabara S, Hirano S, Shibuya K, Arai K, Hattori T. Epilepsia partialis continua as an isolated manifestation of motor cortical dysplasia. J Neurol Sci. 2004;225:157—60.

C. Castaño de la Mota[a,*], D. Conejo Moreno[b], M. García Fernández[c], J.J. García Peñas[d]

[a] *Servicio de Pediatría y de Neuropediatría, Hospital Universitario Infanta Leonor, Madrid, Spain*
[b] *Servicio de Pediatría y de Neuropediatría, Hospital Universitario de Burgos, Burgos, Spain*
[c] *Unidad de Video-EEG, Hospital Infantil Universitario Niño Jesús, Madrid, Spain*
[d] *Servicio de Neuropediatría, Hospital Infantil Universitario Niño Jesús, Madrid, Spain*

[*] Corresponding author.
E-mail address: ccastano993@yahoo.es
(C. Castaño de la Mota).
2173-5808/

Acute occlusion of a giant aneurysm of the internal carotid artery: Recanalisation of the middle cerebral artery through the contralateral carotid artery☆

Oclusión aguda de aneurisma gigante de arteria carótida interna: Recanalización de la arteria cerebral media a través de la arteria carótida contralateral

Dear Editor,

Early recanalisation of an occluded vessel is essential for good outcomes in patients with acute stroke.[1] When access to the occlusion is impossible, the contralateral carotid artery may be approached via the anterior communicating artery (ACoA). We present the case of a patient with an occlusion in the distal segment of the internal carotid artery (ICA) (carotid-T occlusion) caused by an embolus in a giant aneurysm in the cavernous segment of the ICA which migrated cranially. Intracranial stenting was performed through the contralateral ICA and the ACoA.

Introduction

Occlusion of the cervical ICA resulting from thrombosis of a giant aneurysm in the cavernous segment of the ICA limits the viability of endovascular treatment. Several authors have used microcatheters to reach the emboli through ipsilateral vessels and administer intra-arterial fibrinolytic therapy.[2] The literature also reports some cases of anterior-to-posterior circulation approach[3] through the contralateral ICA[4] using Penumbra devices.

We present the first case of stent placement through the ACoA in a patient with acute stroke due to carotid-T occlusion caused by an embolus from a giant aneurysm in the cavernous segment of the ICA.

Clinical case

Our patient was a 53-year-old left-handed man with no relevant history who visited our hospital due to sudden loss of consciousness. After the patient recovered consciousness, he displayed severe left-sided hemiplegia, left facial palsy, and dysarthria (NIHSS score of 14). Code stroke was activated upon arrival at the emergency department; time from symptom onset to arrival at the emergency department was 90 minutes. A brain CT scan detected an expansive process in the temporal region involving the ICA and early signs of infarction in the territory of the right middle cerebral artery (MCA) (ASPECTS score of 6). CT angiography revealed complete occlusion of the origin of the ICA resulting from thrombosis of a giant aneurysm in the ICA; occlusion extended towards segments A1 and M1 of the anterior cerebral artery (ACA) and MCA, respectively (carotid-T occlusion) (Fig. 1A and B).

Angiography was performed with the Seldinger technique; we studied right intracranial circulation through the left ICA. Contrast injected into the left carotid artery was observed to flow into the ACoA towards the right A2 segment (Fig. 1C and D); we therefore decided to use a microcatheter to navigate through the ACoA in order to place a stent between right A1 and M1 to open the right distal ICA occlusion. To this end, we placed a 7F sheath introducer measuring 80 cm (Super Arrow-Flex®) in the left common carotid artery (CCA); the left cervical ICA was catheterised using a Navien microcatheter of 0.072″ inner diameter (Covidien®). After administering 10 mg abciximab intravenously, we placed 2 stents (Codman® Enterprise vascular reconstruction device) using a PROWLER SELECT Plus microcatheter (Codman®) and a Synchro-14 guidewire (Stryker®). An angiography (parenchymal phase) performed at the end of the procedure showed contrast passing through the branches of the right MCA (Fig. 1E-G). Recanalisation was achieved at 420 minutes after symptom onset. Following our hospital's protocol, our patient began dual antiplatelet

☆ Please cite this article as: Pérez Montilla ME, Bravo Rey IM, Bautista Rodríguez MD, Alvarado SV, Bravo-Rodríguez FA, Delgado Acosta F. Oclusión aguda de aneurisma gigante de arteria carótida interna: Recanalización de la arteria cerebral media a través de la arteria carótida contralateral. Neurología. 2017;32:480—484.