Comments on “Role of intestinal microbiota in the development of multiple sclerosis”

Comentario del artículo «Papel de la microbiota intestinal en el desarrollo de la esclerosis múltiple»

Dear Editor,

It was with great interest that we read the review article by Drs Castillo-Álvarez and Marzo-Sola on the role of intestinal microbiota in the development of multiple sclerosis. Their study constitutes a valuable contribution to the literature addressing the role of gut microbiota in multiple functions of the human body, especially immune response. However, we feel that the authors have not mentioned some of the topics addressed in other studies conducted by Spanish researchers and published in international journals. With a view to enriching the discussion and reflections on the role of microbiota in multiple sclerosis, and more specifically to complementing, if possible, the outstanding article by Castillo-Álvarez and Marzo-Sola, we will address the omitted subjects, particularly the association between Candida species and multiple sclerosis.

The human microbiota has been reported to host some yeast species of the Candida genus. Furthermore, several studies have suggested a link between cow milk and multiple sclerosis, and it is a well-known fact that yeasts of the Candida genus can be isolated from some dairy products. Likewise, some rare cases of acute zonal occult outer retinopathy associated with multiple sclerosis and infections due to Candida species have been described in the literature. These observations led us to hypothesise that Candida species may be associated with multiple sclerosis. In that study, we obtained blood samples from 80 patients with multiple sclerosis and 240 age- and sex-matched controls. According to our results, presence of Candida antigens was significantly associated with multiple sclerosis. More specifically, odds ratios (95% CI) were as follows: 2.8 (0.3–23.1; P=.337) for Candida famata, 1.5 (0.7–3.4; P=.290) for Candida albicans, 7.3 (3.2–16.6; P=.001) for Candida parapsilosis, and 3.0 (1.5–6.1; P=.002) for Candida glabrata. Results were similar after excluding those patients receiving immunosuppressants. These findings led us to conclude that presence of Candida antigens was associated with increased risk of multiple sclerosis. Although the significance and scope of our results is difficult to determine, they lay the foundations for further research into this topic.

In any case, we wish to congratulate Castillo-Álvarez and Marzo-Sola on their excellent study and look forward to meeting them at a medical conference to further discuss these topics in person.

References


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Focal cortical dysplasia in a three years old patient with epilepsy partialis continua\textsuperscript{a,\textdaggerdbl,\textdaggerddagger}

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.\textsuperscript{1,\textdagger}\textsuperscript{1} 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.\textsuperscript{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.\textsuperscript{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-isooimmune 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

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\textsuperscript{\textdaggerdbl,\textdaggerddagger} Clinical case presented in a moderated poster session at the 35th Annual Meeting of the Spanish Society of Paediatric Neurology, held in Granada, Spain.