only 2 exposed cases of lipomas of the corpus callosum (an incidence of 0.03%).

We completely agree with the view expressed by the authors when they mention that intracranial lipomas can cause epileptic seizures, so a higher prevalence of lipomas should be considered in the epileptic population. On the other hand, anticonvulsant therapy may be necessary in these cases, especially in symptomatic patients.

Finally, we consider that, in addition to diagnosing the cause of death, a forensic pathologist is required to explain the pathophysiological mechanisms of the disease, as well as any possible aetiologies and risk factors involved. This is the philosophy that we attempt to apply during our daily work.

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Reply to the letter to the editor by Lucena Romero et al. on the article “Epileptic seizure and lipoma of corpus callosum: Cause or incidental finding” *

Respuesta a la carta al editor de Lucena Romero et al en relación con el artículo “Crisis epiléptica y lipoma del cuerpo calloso: causa o hallazgo”

Dear Sir,

We wish to thank Dr. Lucena et al. for their interest in the review “Epileptic seizures and lipoma of the corpus callosum: Cause or finding”, in which they have contributed their experience with intracranial lipomas. We would also like to clarify some of the comments made.

The incidence reported by the authors (0.03%) is similar to that reported by other authors in autopsy cases. 1

The authors describe two clinical cases with discovery of lipoma of the corpus callosum in the midline. The first case 2 describes a patient who died due to bronchoaspiration after an episode of generalised tonic-clonic epileptic seizure. The clinical history mentioned that the patient had suffered two previous episodes of epileptic seizures, which led to an aetiological study being conducted. This pointed to the existence of an intracranial lipoma (“brain fat”), which was confirmed during the autopsy by the finding of two lipomas located in the genu of the corpus callosum. Intracranial lipomas can be the cause of epileptic seizures. The prevalence of lipomas in the epileptic population is higher than that among the general population. 3 For this reason, the finding of an intracranial lipoma in epileptic patients does not necessarily indicate a causal association, but it should invite us to consider other possible aetiologies. In addition, we should conduct a study to assess whether there is adequate correlation between the location of the mass and the clinical-EEG characteristics of the epileptic seizures, thus allowing us to consider a causal association. 4 According to the authors, it was not possible to access the test results (especially the EEG) and no information was provided on the semiology of the epileptic seizures (primarily generalised or focal with secondary generalisation?); consequently, the association between lipomas and epileptic seizures should be taken with particular caution in this case, as it is not possible to ensure that lipomas are a causal factor. The second patient did not have a history of epileptic seizures, although she did suffer headache and psychomotor retardation, as well as the phenotypic traits characteristic of a congenital malformation. As we mentioned before, headache is the symptom most frequently reported in association with lipomas of the corpus callosum in adults 5; whereas it is psychomotor retardation in children. 6 We agree with the authors that, in the second case, the presence of lipoma of the corpus callosum might be in the context of a congenital malformation of the midline.

In conclusion, intracranial lipomas can be regarded as the cause for epileptic seizures as long as there is adequate correlation between the location of the mass and the clinical-EEG characteristics of the epileptic seizures, and if other alternative aetiologies have been excluded.

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