Recurrence in Whipple's disease. Response to a reply☆

La recurrencia en la enfermedad de Whipple. Contestación a réplica

Dear Editor:

We read with considerable interest the reply and manuscript on Whipple's disease (WD) published by Benito-León et al. and cited in the reference section of our letter.[1,2] We would like to thank these authors for their input. They emphasise the isolated presentation in the central nervous system and the relapsing-recurring course that may characterise Whipple's disease with neurological involvement. We agree that when WD presents as isolated encephalitis, diagnosis is difficult due to symptoms being polymorphic. Of all the symptoms, the one most suggestive of WD is oculomasticatory myorhythmia. However, the presence of any of the listed disorders due to an unknown cause requires an examination to rule out or confirm WD. As our colleagues rightly point out, there is a specific treatment for WD that can resolve all of its symptoms. Although it is a bacterial infection, the best way of curing it is still being researched, and relapses do occur. Benito-León indicates that with early diagnosis, sequelae and deterioration will be much more controllable. We could not agree more with that statement. In the case we presented, a diagnosis of WD was finally established for a patient who had been suffering from multiple recurrences and systemic remissions for 20 years.[3] With better understanding and exchange of information, underdiagnosis of WD will become less common.

Why do recurrences appear, and what areas are being researched?

One line of research focuses on the analysis of *Tropheryma whipplei* (TW) bacteria. A group of specialists from different European countries researched the bacterial genotype in 39 patients with WD and in 10 healthy carriers. The digestive tract was infected in 25 patients, and 3 of those patients suffered from relapses with nervous system involvement. There were no digestive symptoms in 7 patients; 5 cases had endocarditis; 1 had spondylodiscitis; and 1 had isolated neurological infection. These observations reveal that TW DNA has a high level of genetic variability and is not correlated with the array of affected organs, relapse severity, or the bacterial pathogenesis, even when we include the carrier group.[4]

The second hypothesis has to do with a host's inability to control an infection. In a study of 145 patients with WD and 166 controls, Moos et al. analysed the behaviour of macrophages and lymphocytes. They concluded that macrophages in ill patients were incompetent to degrade TW.[5] Other publications on macrophage inefficiency in the immune system have already been mentioned.

Numerous recurrences were observed in our patient, most of which coincided with decreases in dosage or discontinuation of the antibiotic during the very long follow-up time. We therefore concur with Benito-León; it is best to continue antibiotic treatment during an extended period of time to avoid relapses, until such time as we dispose of strategies for controlling abnormal macrophage function in the host.

References

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R.O. Domínguez[a,*], I. Davolos[b]

[a] *Departamentos de Neurología, Facultad de Medicina, Universidad de Buenos Aires, Buenos Aires, Argentina*
[b] *Medicina Interna, Hospital Sirio Libanés, Facultad de Medicina, Universidad de Buenos Aires, Buenos Aires, Argentina*

* Corresponding author.
E-mail address: dominguezraulo@yahoo.com.ar (R.O. Domínguez).

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Cerebral death is not a synonym of whole brain death☆

Muerte cerebral no es un término sinónimo de muerte encefálica

Dear Editor:

It was with great interest that we read the original article by Iriarte et al.[1] regarding university students' concepts of death defined according to neurological criteria. We agree with the authors' view that both health professionals and society at large should possess a basic knowledge of these concepts, and the medical and legal implications which they entail. However, we would like to call attention to the term '*muerte cerebral*' or 'cerebral death' which the authors use throughout the manuscript. It is true that there is a lack of uniformity among the different definitions of brain death. It is also true that while firmly established standards for diagnosing brain death do exist, the standards vary greatly

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