Letters to the Editor

Chronic Fatigue Syndrome and Non-celiac Gluten Sensitivity. Association or Cause?

Síndrome de fatiga crónica y sensibilidad al gluten no celiaca. ¿Asociación o causa?

To the editor:

In their letter to the editor Response to: Fibromyalgia and chronic fatigue caused by non-celiac gluten sensitivity, Qanneta et al. pose a conceptual problem between the association of chronic fatigue syndrome (CFS) with non-celiac gluten sensitivity (SGNC) from the prevalent conception of considering chronic fatigue as a distinct disease. From this perspective, being chronic fatigue the central disease associated with other processes, they consider NCGS as a comorbid condition and not as an underlying cause.

From my point of view fibromyalgia and chronic fatigue (FM/CFS) are not defined diseases but just syndromic terms that reflect reality. In the same way that diagnosing unexplained fever is no more than describing a clinical condition for which the cause of the fever is unknown, diagnosing FM/CFS is to describe a symptomatic set of unknown cause. Considering FM/CFS as defined diseases may even have negative effects, since the diagnosis seems to mean that the doctor knows what is happening to the patient, when I believe it is accepting a good deal of ignorance regarding the cause in the case of the physician and resignation in the case of the patient. I work based on the model considering FM/FC as a syndromic description. In this model, various more or less complex causes, which are probably interrelated, and that we are beginning to understand can produce the same clinical syndrome. Qanneta et al. proposes classifying CFS as pure and secondary.1 I believe all CFS are secondary. NCGS of course does not explain all cases of FM/CFS, but it is an explanation for some of the patients. If the treatment of NCGS solves the FM/CFS syndrome, it may be reasonably said that the NCGS was the cause, especially if symptomatic recurrence occurs after ingesting gluten. There are other causes of FM/CFS different from NGCS which may not be detected using complementary explorations. For example, I have had patients in which the withdrawal of statins and psychotropic drugs formally resolved the symptoms of patients diagnosed with fibromyalgia. Even if the clinical manifestations have been resolved, one cannot say that “then the patient had no fibromyalgia,” as I do not think that the case presented had FM/CFS-like symptoms but in reality had no FM/CFS, as stated in their letter.

Qanneta et al. refer poor experiences with GFD regarding systemic symptoms of patients with chronic fatigue. However we have observed a clear response of systemic symptoms, including mental fatigue, in fibromyalgia patients when treating NCGS. More than 30% of patients achieved remission of fibromyalgia or regained a normal life or went back to work or suspended treatment. The following case, treated during the preparation of this manuscript, illustrates this relationship: a 23 year old woman with 3 years since the onset of FM/CFS with severe fatigue that limited daily life activities, mental fatigue, generalized pain, headache, diarrhea and oral ulcers. She had been evaluated by gastroenterology, rheumatology, internal medicine and cardiology, with multiple studies that had ruled out pathology, including celiac disease. HLA typing showed DQ8 and the presence of duodenal biopsy showed type 1 Marsh enteropathy and intraepithelial lymphocytosis (29 CD3 lymphocytes per 100 enterocytes). After one year of follow-up, after starting diet without gluten or dairy, she presented complete remission of all her symptoms, with a normal life, practicing sports and without medication. She chose not to eat triggering foodstuffs.

There may be several explanations for the observed different experience with the treatment of NCGS such as patient selection, time of treatment or the treatment modality of NGCS.

In conclusion, we agree that the problem of FM/CFS is complex and requires multifactorial treatment, but there is a clear difference in perspective from which to address the problem. I prefer to dispense with the prevalent concept of FM/CFS as a disease, avoid the use of psychotropic drugs and seek causes that not show their faces in laboratory testing we usually perform.

Reference


Carlos Isasi Zaragozá

Servicio de Reumatología, Hospital Puerta de Hierro, Majadahonda, Spain

E-mail address: cisasi.hpth@salud.madrid.org