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LETTER TO THE EDITOR

Impact of a gluten-free diet on patients with celiac disease, nonceliac gluten sensitivity, and asymptomatic controls. A need for healthier gluten-free foods[☆]



El impacto de la dieta libre de gluten en pacientes con enfermedad celíaca, sensibilidad al gluten no celíaca y controles asintomáticos. La necesidad de alimentos libres de gluten más sanos

Dear Editors,

We read the recently published editorial “Impact of a gluten-free diet on patients with celiac disease, nonceliac gluten sensitivity, and asymptomatic controls” with great interest.¹ In that editorial, Dr. Coss-Adame emphasizes the value of the prospective study by Remes-Troche et al.,² for being the first investigation showing that a gluten-free diet (GFD) can determine metabolic abnormalities, not only in patients with celiac disease (CD), but also in those with non-celiac gluten sensitivity (NCGS), a condition that is generally considered free from complications.

The study demonstrated that both patients with CD and those with NCGS have a statistically similar increased risk of developing metabolic syndrome (MS), non-alcoholic fatty liver disease (NAFLD), and obesity, after only 6 months on a GFD. For example, the increase in the presence of MS was 20% in patients with CD and 15% in patients with NCGS. It is noteworthy that those numbers could be higher, as follow-up continues, given that the highest rates of metabolic alterations in patients with CD on a GFD have been found to present after 5–10 years on the diet.³

In addition to joining Coss-Adame in congratulating Remes-Troche et al. on their original demonstration, we wish to comment on the association between the development of metabolic abnormalities and the implementation of a GFD, in patients with CD and patients with NCGS.

In celiac patients, the exclusion of gluten from the diet prompts the healing of the intestinal mucosa, although some gastrointestinal tract defects can persist.⁴ Thus, increasing intestinal absorption capacity may be responsible for the development of metabolic disorders. However, patients with NCGS should have normal intestinal absorption, most likely meaning the development of metabolic abnormalities in that group of patients is associated with other mechanisms. It could be speculated that the GFD itself was responsible for the development of metabolic disorders.⁵ There is evidence that the GFD is an unbalanced diet, with multiple nutritional deficiencies and a high glycemic index. With respect to their gluten-containing equivalents, gluten-free foods are deficient in proteins, fiber, folate, iron, potassium, and zinc, whereas they have an excess of fat, carbohydrates, sugars, and sodium. Growing evidence indicates that the GFD needs to be improved by lowering fats and calories and including more adequate levels of fibers, proteins, electrolytes, vitamins, and other micronutrients.⁵ A concomitant factor for the development of metabolic alterations in NCGS after a GFD could derive from the remission of dyspeptic symptoms, possibly resulting in excessive food intake.

No matter what the mechanisms leading to metabolic alterations from a GFD are, it is important to underline that MS, NAFLD, and obesity are established risk factors for cardiovascular disease and diabetes.

In conclusion, we agree with the suggestion of Coss-Adame to inform patients about the metabolic risks associated with a GFD, prescribe an appropriate diet to suit individual needs, and provide preventive cardiovascular surveillance for patients on a GFD.

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Conflict of interest

The authors declare that they have no conflict of interest.

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