

Fecal calprotectin levels in patients with cystic fibrosis

Mehmet Ağilli¹, İbrahim Aydın², Fevzi Nuri Aydın³

Departments of Biochemistry, ¹Ağrı Military Hospital, Ağrı, ²Sarıkamuş Military Hospital, Sarıkamuş, and ³Şırnak Military Hospital, Şırnak, Turkey.

E-mail: mehmetağilli@yahoo.com

To the Editor,

In a recent issue of The Turkish Journal of Pediatrics, we read with great interest the article by Fallahi et al.¹ about fecal calprotectin levels in patients with cystic fibrosis (CF). However, we think that some points should be discussed.

In the Abstract, the authors stated that “after the intervention, the fecal calprotectin levels decreased in 29 patients (21 patients in the drug group, and only 8 patients in the placebo group; $p < 0.001$)”, while accepting the upper limit as $50 \mu\text{g/g}$. It would have been better to say “calprotectin levels lower than $50 \mu\text{g/g}$ were obtained in 29 patients” instead of “decreased in 29 patients” because calprotectin levels of 16 patients were already lower than $50 \mu\text{g/g}$ before the intervention, and it is not stated in the paper whether these patients were included in the 29 patients. Thus, this phrase adds a falsely higher value to the probiotic powder effect than was actually present. Furthermore, the text and data Table are inconsistent because there is an obvious mistake in the Table. After the “Intervention” section of the Table, fecal calprotectin levels were shown inversely; $<50 \mu\text{g/g}$ and $>50 \mu\text{g/g}$ must be reversed.

The authors considered steatorrhea as a sign of pancreatic insufficiency (PI), but not sufficient to diagnose PI itself. Assessments of exocrine pancreatic function, such as fecal fat quantification and ^{13}C -triglyceride breath test, are the methods of choice for the diagnosis of PI². In addition, the authors should specify whether the patients received pancreatic enzyme replacement therapy (PERT). It is very important to point out because the inflammation, as defined by an elevated calprotectin in this study, can be a result of CF or due to PERT. It is known that high doses of PERT cause fibrosing colonopathy, an inflammatory condition³. Also, C-reactive protein, erythrocyte sedimentation rate or leukocyte count could be assessed to demonstrate the efficacy of calprotectin on the inflammatory status and to exclude possible infectious diseases.

The authors used probiotic drugs which did not contain maltodextrin, but they used maltodextrin as placebo. Nickerson and McDonald⁴ demonstrated that the ubiquitous dietary component maltodextrin enhances *Escherichia coli* adhesion, and suggested that specific polysaccharides may promote dysbiosis of gut microbes. Therefore, maltodextrin usage as placebo would not be acceptable because it is not an ingredient of probiotic drugs and because of its possible side effects, which could cause an inflammatory state in the gut. A significant increase in calprotectin levels was found in the placebo group after the intervention. This result suggests a possible maltodextrin side effect in the gut; we wonder if there is an explanation for this increment.

In conclusion, there have been no definitive studies reported on the diagnostic value of fecal calprotectin in CF. Single measurement of fecal calprotectin may not be sufficiently accurate to rule out inflammation, and other markers may be required⁵. Explanation of these concerns will certainly provide clearer information for the readers.

REFERENCES

1. Fallahi G, Motamed F, Yousefi A, et al. The effect of probiotics on fecal calprotectin in patients with cystic fibrosis. *Turk J Pediatr* 2013; 55: 475-478.
2. Domínguez-Muñoz JE. Pancreatic exocrine insufficiency: diagnosis and treatment. *J Gastroenterol Hepatol* 2011; 26: 12-16.
3. FitzSimmons SC, Burkhart GA, Borowitz D, et al. High-dose pancreatic-enzyme supplements and fibrosing colonopathy in children with cystic fibrosis. *N Engl J Med* 1997; 336: 1283-1289.
4. Nickerson KP, McDonald C. Crohn's disease-associated adherent-invasive *Escherichia coli* adhesion is enhanced by exposure to the ubiquitous dietary polysaccharide maltodextrin. *PloS One* 2012; 7: e52132.
5. Werlin SL, Benuri-Silbiger I, Kerem E, et al. Evidence of intestinal inflammation in patients with cystic fibrosis. *J Pediatr Gastroenterol Nutr* 2010; 51: 304-308.