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Fecal calprotectin in coeliac disease

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Abstract

We would like to share with the readers the results of our experience in 50 celiac disease (CD) patients, enrolled between September 2012 and April 2013, who were referred to our third-level CD Unit. The fecal calprotectin (FC) concentration of 50 adults with newly diagnosed CD was compared to that of a control group of 50 healthy subjects. FC level was determined by enzyme linked immunosorbent assay with diagnostic cut-off of 75 μg/g. In addition, we tried to correlate the FC level with symptoms, histological severity of CD (Marsh grade) and level of tissue transglutaminase antibodies (aTg) in CD patients. Finally, FC level was increased in five CD patients and in four controls (10% *vs* 8%, *P* = NS); mean FC concentration of patients and controls were 57.7 (SD ± 29.1) and 45.1 (SD ± 38.4) respectively. Furthermore, no significant correlation was seen between FC levels and symptoms/Marsh grade/aTg. The five CD patients did not show inflammatory lesions (*e.g.,* ulcers, erosions) at upper endoscopy. The four healthy controls with positive FC were followed-up for further six months; in this observational period they did not show clinical signs of any underlying disease. On these bases, we think that FC is not able to investigate the subclinical inflammatory changes of active CD and FC should be considered a useless tool in the diagnostic work-up of uncomplicated CD but it should be accompanied by aTg when ruling out organic disease in patients with irritable bowel syndrome.

Key words: Coeliac disease; Calprotectin; Bowel inflammation; Small bowel

Core tip: High levels of fecal calprotectin (FC) have been found in several gastrointestinal diseases but only few and discordant reports investigated the role of FC in celiac disease (CD). So, we would like to share with the readers the results of our experience in 50 CD patients, who were referred to our third-level CD Unit. We think that FC is not able to investigate the subclinical inflammatory changes of active CD and FC should be considered a useless tool in the diagnostic work-up of uncomplicated CD but it should be accompanied by aTg when ruling out organic disease in patients with IBS.

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TO THE EDITOR

In recent years many studies have explored the role of fecal calprotectin (FC) in detecting the presence of bowel inflammation[1]. High levels of FC have been found in several gastrointestinal diseases but only few and discordant reports investigated the role of FC in celiac disease (CD). In effect, a first report by Montalto *et al*[2] focused on FC concentrations in 28 untreated CD patients compared with a control group of healthy subjects. The authors

reported that FC level of CD patients do not differ significantly from those in controls. On the contrary, the paediatric study by Balamtekın *et al*[3] found that FC level was higher in children with newly diagnosed CD compared to that of CD patients under gluten free diet and healthy controls. Similar results were also found by Ertekin *et al*[4].

So, we would like to share with the readers the results of our experience in 50 CD patients, enrolled between September 2012 and April 2013, who were referred to our third-level CD Unit. The FC concentration of 50 adults with newly diagnosed CD was compared to that of a control group of 50 healthy subjects. FC level was determined by enzyme linked immunosorbent assay with diagnostic cut-off of 75 μg/g. In addition, we tried to correlate the FC level with symptoms, histological severity of CD (Marsh grade) and level of tissue transglutaminase antibodies (aTg) in CD patients. Finally, FC level was increased in five CD patients and in four controls (10% *vs* 8%; P = NS); mean FC concentration of patients and controls were 57.7 (SD ± 29.1) and 45.1 (SD ± 38.4) respectively. Furthermore, no significant correlation was seen between FC levels and symptoms/Marsh grade/aTg. The five CD patients did not show inflammatory lesions (*e.g.*, ulcers, erosions) at upper endoscopy. The four healthy controls with positive FC were followed-up for further six months; in this observational period they did not show clinical signs of any underlying disease. On these bases, we think that FC is not able to investigate the subclinical inflammatory changes of active CD and FC should be considered a useless tool in the diagnostic work-up of uncomplicated CD but it should be accompanied by aTg when ruling out organic disease in patients with irritable bowel syndrome.

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