FEASIBILITY AND UTILITY FOR LONG-TERM MONITORING OF FECAL LACTOFERRIN IN PATIENTS WITH ULCERATIVE COLITIS



Open-Minded

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Introduction

It is known that approx. one third of patients with ulcerative colitis in remission present with acute symptoms of irritable bowel syndrome. Hence, to distinguish IBS symptoms from active disease on an every-day basis has a crucial impact on treatment decision and might save patients from relevant side effects. Fecal biomarkers like lactoferrin and calprotectin have been proposed as potential tools for long-term monitoring of IBD patients for identifying presence of intestinal inflammation and for predicting a flare. However, information on the weekly variation in levels due to lifestyle, medication and die changes over an extended period of time (>6months) is lacking.



Aims

In this study, we evaluated lactoferrin levels of UC patients in stool samples collected twice weekly for a target period of 1 year.

Methods

Ulcerative colitis (UC) patients in sustained remission sampled twice weekly in their home environment and sent the samples to the reference lab in a preprepared fashion. Fecal lactoferrin was determined quantitatively using an ELISA (IBD-SCAN; ≥7.25µg/g). Baseline and assessments of an acute flare included CAI, endoscopy and biopsy for Mayo and Riley histopathology scores. Clinical symptoms were assessed using the CAI (< 5 defining clinical remission) on a monthly basis and in the event of a clinical flare reported by the patients.





Results

In this poster we report the first preliminary data for long-term monitoring of lactoferrin in IBD. A total of 642 fecal specimens were collected over a mean period of 33.5 weeks (range 18 to 40) in 10 UC patients (8 male; mean age: 56.2 ± 13.1) with Mayo scores ≤ 1 . Baseline data, medical history and maintenance medications are shown in table 1. Mean lactoferrin for each patient ranged from 2 to 12 with standard deviation (SD) ranging from 3 to 6 (figure 1). During the reported monitoring interval, 20% of patients experienced symptoms fulfilling the clinical criteria of an acute flare with CAI 6 and 9 (figure 2). However, fecal lactoferrin did not show a relevant increase for both patients indicating no intestinal inflammation (figure 3 + 4). Endoscopy and histological examination ruled out acute inflammation and supported symptoms of acute IBS in both cases. The escalation of the anti-inflammatory treatment could be prevented.

Conclusion

Fecal lactoferrin levels had low variability over time for UC patients in remission and shows promise as an aid for differentiating active IBD from IBS symptoms for optimizing treatment.

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