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EDITORIAL

Prediction of disease course in inflammatory bowel diseases

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Abstract

Clinical presentation at diagnosis and disease course of both Crohn's disease (CD) and ulcerative colitis are heterogeneous and variable over time. Since most patients have a relapsing course and most CD patients develop complications (e.g. stricture and/or perforation), much emphasis has been placed in the recent years on the determination of important predictive factors. The identification of these factors may eventually lead to a more personalized, tailored therapy. In this TOPIC HIGHLIGHT series, we provide an update on the available literature regarding important clinical, endoscopic, fecal, serological/routine laboratory and genetic factors. Our aim is to assist clinicians in the everyday practical decision-making when choosing the treatment strategy for their patients suffering from inflammatory bowel diseases.

Key words: Inflammatory bowel disease; Crohn's disease; Ulcerative colitis; Disease course; Predictive markers; Clinical; Serology; Genetics

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In this TOPIC HIGHLIGHT series, we provide an update on the available literature regarding important clinical, endoscopic, fecal, serological/routine laboratory and genetic factors (Table 1) that may be used for the prediction of the disease course in inflammatory bowel diseases (IBD)[1]. Our aim is to assist clinicians in the everyday practical decision-making when choosing the treatment strategy for their patients suffering from IBD.

We focus first on clinical and endoscopic variables by Louis *et al*[2] and Allez *et al*[3] that were found to be important medium and long-term markers in the prediction of complicated disease course in IBD. The phenotypic classification of Crohn's disease (CD) plays an important role in determining the treatment, and may assist in predicting the likely clinical course of disease. Using the Vienna classification system, it has been shown in clinic-based cohorts that there can be a significant change in disease behavior over time, whereas disease location remains relatively stable[4,5]. In addition, factors, e.g. young age at onset or pediatric presentation, early steroid use, ileal disease, presence of perianal disease, mucosal healing or smoking were identified as an important risk factor for developing disabling disease or major abdominal surgery[6-10]. Fewer factors are available in ulcerative colitis. One of the important factors is disease extent, identified by previous studies and also a recent 10-year population-based inception cohort study[7].

A second group of markers that will be discussed in details are biomarkers. Biomarkers can be divided into long-term markers (e.g. genetics and serology) influencing the clinical phenotype and short-term markers that may be used for the prediction of flares. Genetic (e.g. nucleotide oligomerization domain, NOD2)/caspase activation recruitment domain (CARD15) and serology markers, discussed by Vermeire *et al*[8] and Dubinsky *et al*[9], may be important in the prediction of long term evolution of disease phenotype and need for surgery in CD or they might also be used to predict the efficacy and/or side effects of medical therapy. However, various geographical differences may exist at least for genetic

Table 1 Predictors for long and short term disease course in inflammatory bowel diseases

Markers
Disabling/complicated disease in CD
Young age at onset (pediatric or < 40 yr)
Small bowel disease
Stricturing
Perianal disease
Weight loss > 5 kg
Steroid need for the first flare at diagnosis
Early immunosupression and/or biological therapy (protective?)
NOD2/CARD15
rs1363670 near IL12B
ASCA, glycans
Surgery in CD/colectomy in UC
Young age at onset (pediatric or < 40 yr)
Small bowel disease
Stricturing or penetratingat diagnosis
Early immunosupression and/or biological therapy (protective?)
Deep ulcers at endoscopy
ASCA, glycans
Extensive disease in UC
Smoking (protective) in UC
Clinical relapse
CRP, ESR
Calprotectin
Complete or partial mucosal healing (protective)

NOD2/CARD15: Nucleotide oligomerization domain2/caspase activation recruitment domain15; ASCA: Anti-*Saccharomyces cerevisiae* antibodies; CRP: C reactive protein; ESR: Erythrocyte sedimentation rate; UC: Ulcerative colitis; CD: Crohn's disease.

markers. Finally, we focus on the predictive power of serum, fecal and mucosal markers, which were used by Scaldaferri *et al*[10] to identify relapses and/or disease progression.

The search for clinical, laboratory, and molecular markers to define and predict disease outcome in common diseases has moved rapidly forward with the help of modern technologies and vigorous collection of clinical data. However, many questions remain unanswered. We believe that this issue will be of strong interest not only for IBD specialists, but also for gastroenterologists, since our ultimate goal is to identify individual patient profiles, including clinical, laboratory and molecular markers, which will hopefully allow us to choose the most appropriate management in terms of therapy and intensity of follow-up.

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