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Editorial

Body composition and metabolic features in Crohn's disease: an update

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Introduction

Crohn's disease (CD) is an inflammatory bowel disease (IBD) potentially affecting the whole gut, with an incidence that is nowadays increasing in both Europe and the United States[1-2]. Since CD is a chronic intestinal disease, it can frequently lead to an impairment of nutritional status that ranges from 65% to 75% in CD and from 18% to 62% in ulcerative colitis (UC), the other major form of IBD[3]. The simultaneous occurrence of decreased nutrient intake and malabsorption and increased energy expenditure could be considered as factors responsible for the high risk of malnutrition in CD.

The importance of preventing and monitoring the nutritional and metabolic alterations in CD patients is strongly supported by several factors. Among them it is relevant to underline the association between malnutrition and a compromising of gut barrier function, the improvement of patients' quality of life due to nutritional status improvement and to reduce post-operative risk in well-nourished patients requiring surgical intervention[4]. In addition, an accurate evaluation of body composition and energy metabolism in these patients is warranted because of the increased interest for enteral nutrition as a safe and effective therapeutic approach[5]. Since the aetiology of CD is largely debated, glucocorticoids still represent the most potent treatment at least in the active phase of the disease activity. Thus, severe side-effects in patients requiring long-term systemic steroid therapy to avoid relapses, are likely to occur[6]. The catabolic effect of steroids on body composition and energy metabolism include bone demineralisation, growth delay, fat mass increase and insulin resistance.

The assessment of body composition can be performed by different techniques, ranging from simple ones as anthropometry and bioimpedance analysis, to sophisticated measurements such as dual X-ray absorptiometry, isotopic dilution, neutron activation analysis and computed tomography which allow an accurate determination of the various body weight components: fat mass (FM), fat-free mass (FFM), total body water and bone mass.

The energy requirements of a subject can be easily measured by indirect calorimetry using an open-circuit ventilated hood system under strictly standardised conditions. Briefly, after voiding, the subject enters a quite room, with the air temperature (24-26 °C) and the humidity level (35-40%) kept constant. The subject is then placed in a semi-supine position on a bed and remain awake and motionless for at least 30 minutes before and throughout the measurement. The system should be calibrated immediately before each measurement with standard gases of known concentration. Basal metabolic rate (BMR) and substrate oxidation rates are calculated from oxygen consumption, carbon dioxide production and nitrogen urinary excretion. The measurement of daily energy expenditure by means of a calorimetric chamber or the double-labelled water technique is possible only in few research units worldwide, and along with the assessment of total energy intake and energy loss with the urine and stools, allows to compute the energy balance of an individual.

A body weight reduction represents a typical feature of CD patients in different phases

of disease activity[7] and weight loss is primarily due to a decrease in FM in spite of a loss of FFM, this latter representing the metabolically active component of the organism. Besides a reduced energy intake or increased nutrient malabsorption, weight loss in CD patients could be also partially due to increased energy requirements, essentially related to the inflammatory signs of the disease. The reports in the literature on this point are controversial, probably as a consequence of the differentcharacteristics of patient populations examined, that ranged from patients with inactive to severe disease or undergone parenteral nutrition regimen. Moreover, CD patients in a remission phase of the disease activity and not receiving steroid therapy or nutritional support, showed a preferential lipid utilisation in basal conditions and an increased value of BMR normalised by either FFM or body weight[8]. These metabolic features have also been described in steroid-treated patients[9]. It has been recently shown that, inactive ileal CD patients had an increased diet-induced thermogenesis after a standard test meal (50.2 kJ/kg body weight) than control subjects[10]. This finding, along with the increased lipid oxidation, could be of relevance in explaining the difficulty of these patients in gaining weight, and could suggest that a diet relatively rich in lipids may favour the attainment of a good energy balance.

As far as bone mineralisation is concerned, a low bone mineral density was found to be a typical feature of newly-diagnosed CD patients, but not of UC patients, indicating a difference in the metabolic pathways between the two diseases[11]. In this connection, our group reported that, while CD patients showed the above-mentioned peculiar metabolic characteristics, patients affected by UC did not differ in any of the variables examined from controls[8,12]. This can probably be due to the different disease localisation and extent, as CD is a systemic disease while UC is more limited to the colonic mucosa. The data present in the literature suggest that the peculiar metabolic characteristics shown by CD patients are independent of disease localisation[13]. It could be hypothesised that the cause of increased lipid oxidation rate with the consequent reduction in fat mass may be searched for in the pathogenic mechanism of the disease (i.e. increased production of inflammatory mediators). In addition, although no differences in the variables examined were detected between CD subgroups, patients with ileal and ileo-colonic localisation presented a greater reduction of body weight compared to controls than patients with colonic disease[13].

Finally, a not impaired whole body glucose uptake and oxidation was found in CD patients in a remission phase of the disease activity and not undergoing steroid treatment. This was probably due to the good preservation of FFM and to low blood and tissue cytokine concentration[14] found in inactive patients.

In conclusion, CD patients are at high risk of developing a nutritional status impairment up to real emaciation. Since malnutrition by itself correlates with a decreased function of the intestinal mucosa and since an appropriate nutritional treatment has been reported as effective as steroids in maintain remission in CD patients, the assessment of nutritional status and energy requirements plays an important role in the management and follow-up of these patients.

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