Introduction

Inflammatory bowel disease (IBD) is characterized by a chronic relapsing intestinal inflammation. The causes of IBD are unknown but genetic, environmental, immunological, and microbial factors may be involved. Results from studies on animals suggest that the intestinal flora participates in the initiation and perpetuation of IBD, ***(Mallant-Hent et al., 2006)***.

Ulcerative colitis (UC) and Crohn's disease (CD) are caused by abnormal activation of the immune system in the intestines. The immune system is composed of immune cells and the proteins that these cells produce. These cells and proteins serve to defend the body against harmful bacteria, viruses, fungi, and other foreign invaders. Activation of the immune system causes inflammation within the tissues where the activation occurs. Inflammation is, in fact, an important mechanism of defense used by the immune system, ***(Iltanen et al., 2006)***.

Normally, the immune system is activated only when the body is exposed to harmful invaders. In patients with Crohn's disease and ulcerative colitis, however, the immune system is abnormally and chronically activated in the absence of any known invader. The continued abnormal activation of the immune systems causes chronic inflammation and ulceration. The susceptibility to abnormal activation of the immune system is genetically inherited. First degree relatives (brothers, sisters, children, and parents) of patients with IBD are thus more likely to develop these diseases, ***(Wejman et al., 2006)***.

Nowadays the incidence of Crohn's disease and ulcerative colitis is increasing but diagnosis of inflammatory bowel disease and the differentiation between Crohn disease and ulcerative colitis is still based on morphological changes identified at endoscopy, radiology, and histopathology. In 5-15% of cases this differentiation is not possible (diagnosed with indeterminate colitis), ***(Mokrowiecka et al., 2007)***.

Many Crohn's disease (CD) patients develop complications (fistulae and abscesses), and require surgery, often repeatedly and at variable instances. Identifying serological markers that determine their early or repeated manifestation can enable implementing more aggressive preventive strategies, ***(Amre et al., 2006)***.

Several antibodies have been associated with Crohn's disease and are associated with distinct clinical phenotypes. Antibodies directed against Saccharomyces cerevisiae (ASCA), perinuclear components of neutrophils (pANCA), and porin protein C of Escherichia coli (anti-OmpC) are reported to be associated with disease phenotype and may be of diagnostic importance in IBD, ***(Papp et al., 2007)***.