Summary

Inflammatory bowel disease (IBD) is an idiopathic disease, probably involving an immune reaction of the body to its own intestinal tract. The two major types of IBD are ulcerative colitis (US) and Crohn`s (CD) disease, which are chronic inflammatory diseases of the gastrointestinal tract. As the name suggests, ulcerative colitis is limited to the colon; Crohn`s disease can involve any segment of the gastrointestinal tract from the mouth to the anus.

Inflammatory bowel disease (IBD) is the most common chronic inflammatory condition after rheumatoid arthritis. Advances in the understanding of immunological basis and the pathogenesis of inflammatory bowel disease have encouraged the development of many new diagnostic methods and novel strategies for the treatment of inflammatory bowel disease.

The aetiology of inflammatory bowel disease is presently unknown but is likely multifactorial. The currently held paradigm involves the complex interaction of four elements: genetic susceptibility, host immunity, environmental factors and commensal enteric bacteria. The most recent hypothesis on the pathogenesis of IBD states that individuals, who have a genetic predisposition, when confronted with unidentified aggressors from their natural environment, develop a loss of tolerance to luminal bacterial antigens (i.e. dysregulation of the enteric immune response) and initiate an uncontrolled inflammatory reaction targeted at the bowel wall.

Crohn`s disease is associated with Th1-type T cell–mediated inflammation. This produces an excess IL-12, IL-17, IL-23, IFN-γ and macrophages derived cytokines, also there is overproduction of IL-1B, IL-2, IL-6 and IL-8. In comparison, ulcerative colitis is associated with Th2-type T cell–mediated inflammation with excess IL-4, IL-5, IL-10 and IL-13 production. Also HLA class II antigens, Nuclear factor kappa B (NF-κB)

and Tumor necrosis factor alpha (TNF-α) are involved in the pathogenesis of IBD.

Inflammatory bowel disease (IBD) is not just a disorder of one organ system, but rather a multi-systemic disease. Patients with IBD commonly present with a wide range of systemic and local complications. Patients can present with these instead of the classical bowel symptoms.

Since the exact aetiology of IBD is not known, the diagnosis of IBD is confirmed by history, clinical evaluation and a combination of laboratory findings (serological markers [e.g. ASCA and P-ANCA] and faecal markers [e.g. calprotectin and lactoferrin]), endoscopic {Eosophagogastroduodenoscopy (EGD), colonscopy, endosonography, Double Balloon Enteroscopy (DBE), and Wireless Capsule Endoscopy (WCE), }pathologic examination of tissue, histological, and imaging examination (contrast studies, MRI, CT, doppler US, white blood cell scan, and PET scan).

Most patients can be adequately treated using a combination of aminosalicylates, antibiotics and corticosteroids to achieve sustained response or remission, though many patients will require immunomodulators. The developments of novel biologic therapies particularly anti TNF- α antibody (infliximab), has been very beneficial. In the near future more humanized anti-TNF antibodies will become available with more convenient route of administration (SC) and lower immunogenicity. In additional to conventional therapy, probiotics, helminth ova therapy**,** leukocyte filtration, bone-marrow and stem-cell transplantation, nutritional support, alternative therapy, growth factors, and other novel therapies play an important role in management of many patients suffering from IBD.