Inflammatory Bowel Disease

Historical Perspective, Epidemiology, and Risk Factors

Talha A. Malik, MD, MSPH[a,b,*]

KEYWORDS

• Inflammatory bowel disease • Crohn's disease • Ulcerative colitis
• Historical perspective • Epidemiology • Risk factors

KEY POINTS

- Inflammatory bowel disease describes 2 well-established but not entirely discrete disease entities, Crohn's disease (CD) and ulcerative colitis (UC), which are represented by an uncontrolled immune-mediated inflammatory response in genetically predisposed individuals to an unknown environmental trigger that interacts with the intestinal flora and primarily affects the alimentary tract.
- Approximately 1.5 million persons in North America have inflammatory bowel disease.
- Several potential risk factors of inflammatory bowel disease have been studied and include particular environmental triggers, intestinal immune mechanisms, heritable factors, gut flora, diet, mesenteric fat, medications, nicotine, infectious agents, immunization, hygiene, pregnancy, breastfeeding, stress, and lifestyle.
- There are data to suggest a higher mortality in CD compared with the general population; however, there is no definitive evidence to suggest higher mortality among patients with UC compared with the general population.
- Epidemiologic studies have expanded understanding of the occurrence, distribution, determinants, and mechanisms of inflammatory bowel disease and this allows clinicians to identify safer and more effective approaches to management and therapeutics.

INTRODUCTION

Inflammatory bowel disease (IBD) consists of 2 well-established but not entirely discrete disease entities, Crohn's disease (CD) and ulcerative colitis (UC). They together are a group of closely related but heterogeneous disease processes. The

Conflicts of interest: None.

[a] Department of Medicine-Gastroenterology, University of Alabama at Birmingham, 1808 7th Avenue South, BDB 391, Birmingham, AL 35294, USA; [b] Department of Epidemiology, University of Alabama at Birmingham, 1808 7th Avenue South, BDB 391, Birmingham, AL 35294, USA
* Department of Medicine-Gastroenterology, University of Alabama at Birmingham, 1808 7th Avenue South, BDB 391, Birmingham, AL 35294.
E-mail address: tmalik@uabmc.edu

Surg Clin N Am 95 (2015) 1105–1122
http://dx.doi.org/10.1016/j.suc.2015.07.006 **surgical.theclinics.com**
0039-6109/15/$ – see front matter

mechanism of IBD involves an uncontrolled immune-mediated inflammatory response in genetically predisposed individuals to an unknown environmental trigger that interacts with the gut microbiome (intestinal flora) and primarily affects the alimentary tract (Fig. 1).[1–6] It is estimated that more than 1.5 million Americans have IBD, approximately half represented in each of these 2 discrete IBD subgroups.[2]

HISTORICAL PERSPECTIVE

Background

It is thought that Alfred the Great, who is commonly considered to be the first King of England (849–899 CE), may have had CD.[7] However, it was not until 1913 that a discrete disease condition resembling what is now considered to be CD was identified, when Kennedy Dalziel, a British physician, described patients with transmural inflammation of the small and large intestines.[7,8] Subsequently in 1932, Dr Burrill Crohn, Dr Leon Ginzburg, and Dr Gordon Oppenheimer published articles describing a condition that caused inflammation of the terminal ileum and which they called regional or terminal ileitis. This disease entity later began to be referred as CD.[7–10]

UC was first described in ancient Greece by Hippocrates as a condition characterized by chronic diarrhea and bloody stools.[7,8] This condition was thought to be related to ulceration and inflammation of the large intestine. In the 1600s, Thomas Sydenham, a British physician, named this disease bloody flux. In 1859, Samuel Wilks, another British physician, identified UC as a discrete disease entity.[7,8]

The first breakthrough that established IBD as the prime intestinal autoimmune disease occurred in the 1950s when it was noted that symptoms in patients with both CD and UC responded to corticosteroids.[3] In the 1970s, traditional immune modulators, predominantly thiopurine analogues, began to be used and eventually became first-line steroid-sparing agents.[3] In 1997, Targan and colleagues[11] published findings of the so-called Crohn's Disease cA2 Study, which assessed the effectiveness of the biologic antibody against tumor necrosis factor (TNF) cA2 (infliximab) in induction of remission in luminal CD. This study began the era of biologics. During the first decade of the twenty-first century, biologics began to gradually emerge as the most effective

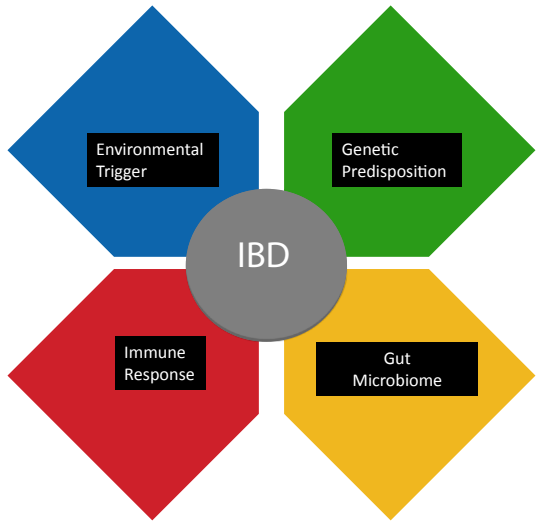

Fig. 1. IBD mechanism.