Introduction

INTRODUCTION

The term inflammatory bowel disease (IBD) is reserved for conditions characterised by the presence of idiopathic intes tinal inflammation. Conditions such as infective or ischaemic enteritis are covered in Chapters 6, 74 and 77 . Crohn’s disease (CD) may a ff ect any portion of the gastrointestinal tract from mouth to anus, most typically the distal ileum, the anal canal and the lar ge bowel, whereas ulcerative colitis (UC) is confined to the large intestine is characterised primarily by mucosal inflammation, whereas CD most typically involves transmural inflammation. On occasion there may be di ffi culty distinguishing UC from CD in the colon. This occurs in approximately 10% of patients with colitis; in such instances the term indeterminate colitis (IC) may be used. The incidence and prevalence of IBD is highest in Europe and North America, where it a ff ects around 3 in 1000 people. The overall incidence is steadily rising w orldwide, linked to improved public hygiene, dietary changes and industrialisa tion. Both UC and CD occur in individuals who may have a genetic predisposition and who are exposed to environmental factors that trigger abnormal immune responses that lead to intestinal inflammation. Microscopic colitis includes two main subtypes: lymphocytic colitis and collagenous colitis (CC). The aetiolog y is uncertain but may reflect inappropriate immune responses to alterations in the gut microenvironment conse quent to oral drug ingestion, particularly non-steroidal anti inflammatory drugs. UC is characterised by mucosal inflammation of the large bowel, always involving the rectum (proctitis) and extending to involve varying degrees of mor e proximal colon (colitis). When the entire colon and rectum are involved (pancolitis), some patients may also have a degree of ‘backwash ileitis’, in which there is secondary inflammation in the terminal ileum. Burrill Bernard Crohn , 1884–1983, gastroenterologist, Mount Sinai Hospital, New Y ork, NY , USA. - . UC - UC is a chronic condition that tends to be relapsing and remitting. Early relapse and persistent disease within the first 2 years of diagnosis are both predictors of a severe disease course. The extent of disease may also change after initial diagnosis; half of patients with UC a ff ecting the rectum or rectosigmoid progress to develop more proximal disease. - Histological hallmarks of UC typically include atrophy - and distortion of the crypts, irregularity of the mucosal villi, marked infiltration of plasma cells within the deep lamina pro - pria (basal plasmacytosis) and m ucus depletion, but none of these is pathognomonic; the diagnosis ultimately depends on clinical correlation, disease course and elimination of other potential causes, especially infection (Figure 75.1). Pseudo - polyposis occurs in almost one-quarter of cases. Stricturing in

The principles of medical management • The role of surgery

in acute and elective settings • The management of postoperative complications and • long-term outcomes Figure 75.1 Mucosal biopsy in ulcerative colitis illustrating in /f_l amma

tory in /f_i ltrate and crypt abscess formation.

UC is very unusual (unlike in CD) and should prompt urgent assessment because of the possibility of coexisting carcinoma. A small proportion of patients develop irregular mucosal swell ings (dysplasia-associated lesions or mass [DALMs]), which are highly predictive of coexisting carcinoma. CD (see Crohn’s disease (regional enteritis)) is char acterised by discontinuous transmural inflammation of the bow el caused by transmural inflammation of any part of the gastrointestinal tract from mouth to an us, but most commonly the ileocaecal region, colon and anus. There is often a degree of rectal sparing when the colon is involved. The transmural inflammation may be patchy (rather than di ff use) and crypt distortion is commonly seen. Histology typically demonstrates discontinuous segments of disease or ‘skip lesions’, involve ment of the terminal ileum and the presence of granulomas with a tendency for more marked inflammation in the proxi mal colon (Figure 75.2). Clinical correlation of histopathology with endoscopic and radiological findings is key to clinc the diagnosis of CD. Stricturing in the colon, while usually benign in CD, may mask an underlying neoplasm. When endoscopic and histological appearances do not cat egorically confirm either UC or CD, and the term IC is used, the clinical phenotype may help define the diagnosis, especially if there ar e features of small bowel or perianal disease sug gestive of CD. Patients with IC may la ter come to a definitive diagnosis of UC or CD, depending on the disease course.

Figure 75.2 Photomicrographs of Crohn’s disease illustrating mucosal ulceration and transmural in /f_l ammation (arrows) non-caseating granulomas (b) (courtesy of Professor Kieran Sheahan, St Vincent’s University Hospital, Dublin, Ireland).

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