INFLAMMATORY BOWEL DISEASES

ESSAY

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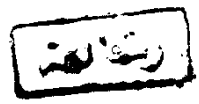

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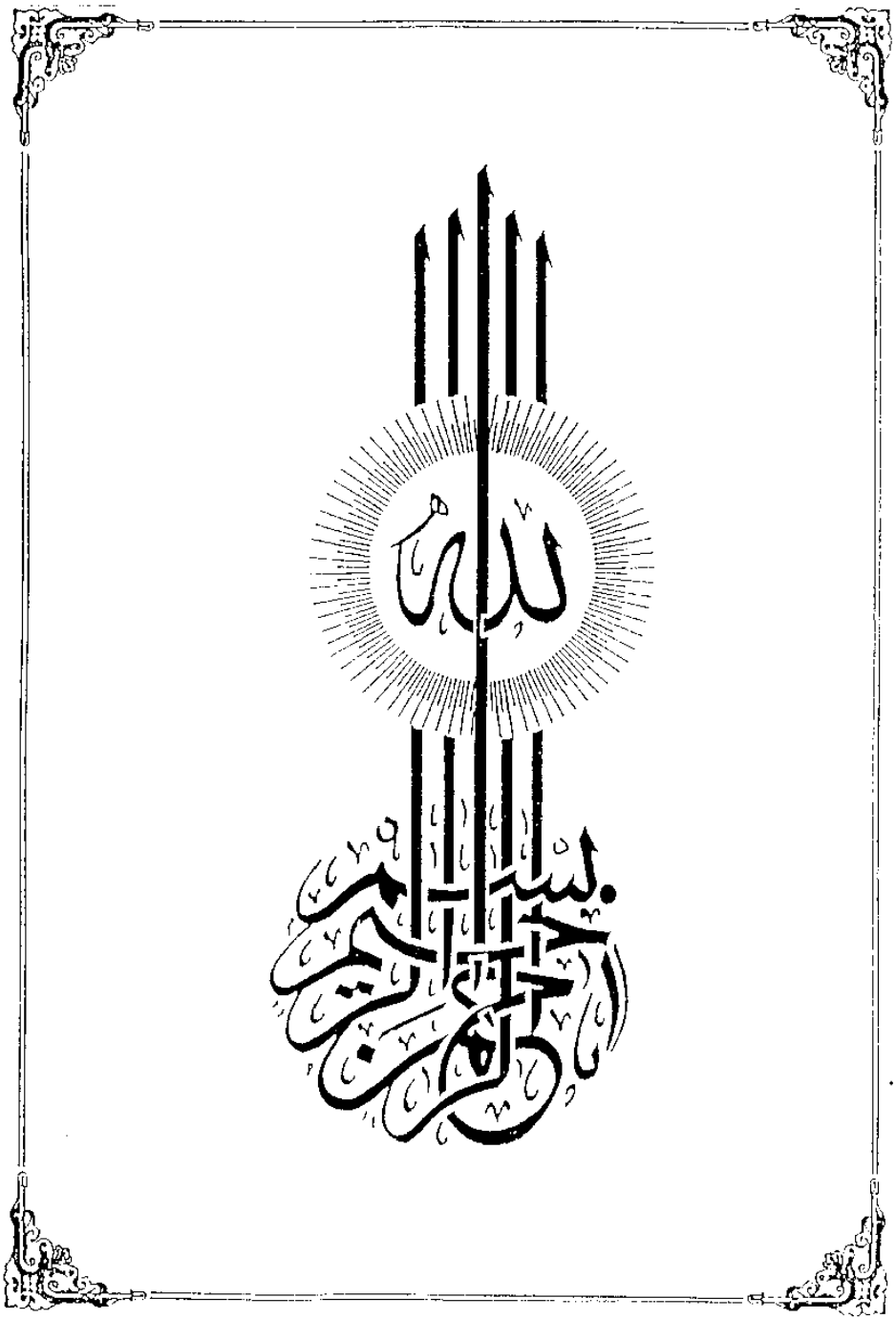

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INTRODUCTION

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Inflammatory bowel disease is a general term designated to a number of clinical pathologic disorders in children and adults who present with similar clinical manifestations which include fever, diarrhoea -with or without blood- and distention (Kirsner, 1978).

The increased incidence of different inflammatory lesions of the small and large intestine have drawn the attention of both physicians and surgens for further studies of their various aspects.

In addition to the well knwon granulomatous enterocolitis Chron's disease and ulcerative colities which are chornic diseases of unkown aetiology, other inflammatory lesions will be considered.

The development of fibrooptic colonoscopies and the ability to study the whole mucosal surface of the colon, have widened follow up and discovery of early malignancy.

Medical treatment alone is of value when the disease is mild, but it is now generally accepted that in severe or established cases the surgical treatment is the only solution.

This essay aims at studying the different items of ulcerative colitis and Crohn's disease, focusing on the main operative techniques for conserving continence in the surgical management. Also the other common inflammatory bowel lesions are considered in this essay.

Diverticulitis is not included in this essay as the primary state of the disease "diverticulosis" is considered as a bowel motility disorder and is not inflammatory from the start.

CLASSIFICATION

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I. Acute inflammatory lesions:
- Bacterial
  - Salmonella entritis
  - Shigella entritis
  - Campylobacter enteritis
  - Yersenia enteritis
  - pseudomembranous enterocolitis
  - Nectotising entrocolitis
- Parasitic
  - Acute amoebic dysentery
  - Bilharzial dysentery
- Miscellaneous
  - Ischemic colitis
  - Radiation colitis
  - Colitis associated with autoimmune defeciency syndromes
  - Mixed colitis
  - Non-specific colitis

II. Chronic inflammatory lesions
- Bacterial
  - TB enteritis
- Parasitic
  - Amoebic dysentery
  - Bilharzial dysentery
- Actinomycosis
- Granulomatous enteritis
  - Ulcerative colitis
  - Crohn's disease

CROHN'S DISEASE

Aetiology of Crohn's disease :

Crohn's disease is defined as "chronic granulomatous inflammatory disorder that may involve any segment of the GIT from mouth to anus. When it is confined to the colon it is called Crohn's colitis or granulomatous colitis, whereas involvement of the small bowel may be called regional enteritis or ileitis.

Incidence :

Although regional enteritis is the most common surgical disease of the small intestine, it is quite uncommon compared with other gastrointestinal diseases such as peptic ulcer and acute appendicitis (Schwartz et al.;, 1984).

The disease has highest incidence in United States, England and Scandinavia, and infrequent in central Europe, and only occasionally seen in Africa, Asia and South America (Schwartz et al., 1984).

The incidence is three times highr in Jews than in non jews. It is also higher in whites .

It is slightly higher in males and most frequent in young adults.

The 1970's have seen an explosion in the interst paid to a number of possible aetiologic factors :

1. **Viral aetiology** :

Gitnick, Arther & Shibata (1978), isolated cytopathic agents from four Crohn's disease specimens . They studied E.M. apperance and suggested a small RNA virus.

In 1977, Whorwell et al., isolated a reovirus like agent from 6 of 10 resected Crohn's disease specimens. This isolation as the authors point up, does not imply a causal relationship.

2. **Bacterial infection** :

Microbial possibilies in Crohn's disease have included T.B., lymphopathia venerium and various anaerobes. Currently, the focus is on mycobacterium Kanassii (Burnham et al., 1978), cell wall defecient psudomonas like bacteria (Parent and Mitchel, 1976) and Chlamydia trachomatis (Elliott et al.,m 1981). The gut microflora in Crohn's disease is characterized by bacterial overgrowth especially anaerobes including peptostreptococcus magnus (Bourgault et al., 1980).

3. **Genetic factors** :

The problem of genes versus the environment is reviewed by Lewkonia and McConnel (1970) who list 4 possible explanations for familial aggregation of cases :-

a) Random chance

b) The effect of shared environment

c) The effect of shared genes, and

d) The effect of shared genes controlling the response to shared environment.

It would appear that genetic factors msut be invoked to explain the frequency which famelial inflammatory bowel disease is noted and the rarity of its occurrence in the spouse of a patient.

Immunological aspects :

In 1980, Kemer & Alpert showed that circulating lymphocytes of patients with Crohn's disease are cytotoxic in vitro for autologus and allogenic colonic epithelial cells evidenced by:

1. Cytotoxic effect is specific for colonic epithelial cells and does not occur with gastric or intestinal epithelial cells.

2. The effect is rapid (2 hours) and does not require complement.

Dietary factors :

The alleged causal relationship between excessive intake of refined sugar and Crohn's disease lacks decisive evidence (Martini & Brandes, 1976).

Gross pathologic appearance of Crohn's disease:

The gross pathologic findings are very similar throughout the GIT i.e., discontinuous ulcerations stricture and fistula formation.

Samll intestine :

At operation, diseased segments are beefy, dull purple red, thickened to two or three times normal diameter, covered with strands and patches of thick grey white exudate. Mesentric fat tend to grow over the serosa so that it nearly encompasses the bowel in areas of maximal involvement. The thickned bowel wall is very firm, rubbery and virtually incompressible. Involved segments are often adherent to adjacent loops or other viscera or several loops may be matted together into a bulky conglomerate mass. Intermal fistulae are frequently present.

The mesentry of the segment is characteristically greatly thickned, dull and rubbery and contains lymph nodes.

The uninvolved proximal bowel is often dilated becasue of the considerable degree of obstruction in the diseased segment (Schwrtz, 1984).

The affected bowel wall is stenosed and immobile and the overlying mucosal surface shwos ulceration which are usually discontinuous with adjacent uninvolved mucosa resulting in a "Cobble stone" appearance produced by interconnecting ulcers surrounding mucosa with oedematous submucosa. (Morson & Dawson, 1979).

Though Crohn's disease may involve any segment of the GIT, yet isolated lesion in oesphagus, stomach or dudenum is rare (Schwartz, 1984).

Only about 35% of patients have disease limited to the small intestine and 20% are limitted to the colon, with almost half having small and large bowel involvement (Schwartz, 1984).

Three principle patterns of involvement of the small intestine that occur in 95% of all patients :

1) The most frequent site of involvement is the terminal ileum with minimal or no caecal abnormality, maximal disease of bowel and mesentry extends proximally for 15-25 cm beyond which the degree of involvement diminishes rapidly.

2) In neerly one half of patients both small and large bowel are involved. The disease is often continious, since terminal ileum is the most frequently involved segment of small intestine, and caecum and Rt. colon are the most frequent site of colonic involvement.

3) Jejunoileitis, extensively involves the distal one half of jejunum and proximal one half of ileum (Schwartz et al., 1984).

Large intestine, appendix and anus :

The site and extent of colonic involvement are highly variable. The gross appearance may closly resemble the appearance of Crohn's disease of the small gut. with marked thickning of the bowel wall and encroachment on the lumen, and with fissuring of the mucosa to produce a "cobblestone" effect on the mucosal surface.