SURGICAL MANAGEMENT OF NON BILHARZIAL INFLAMMATORY BOWEL DISEASE

An Essay

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Master Degree of General Surgery

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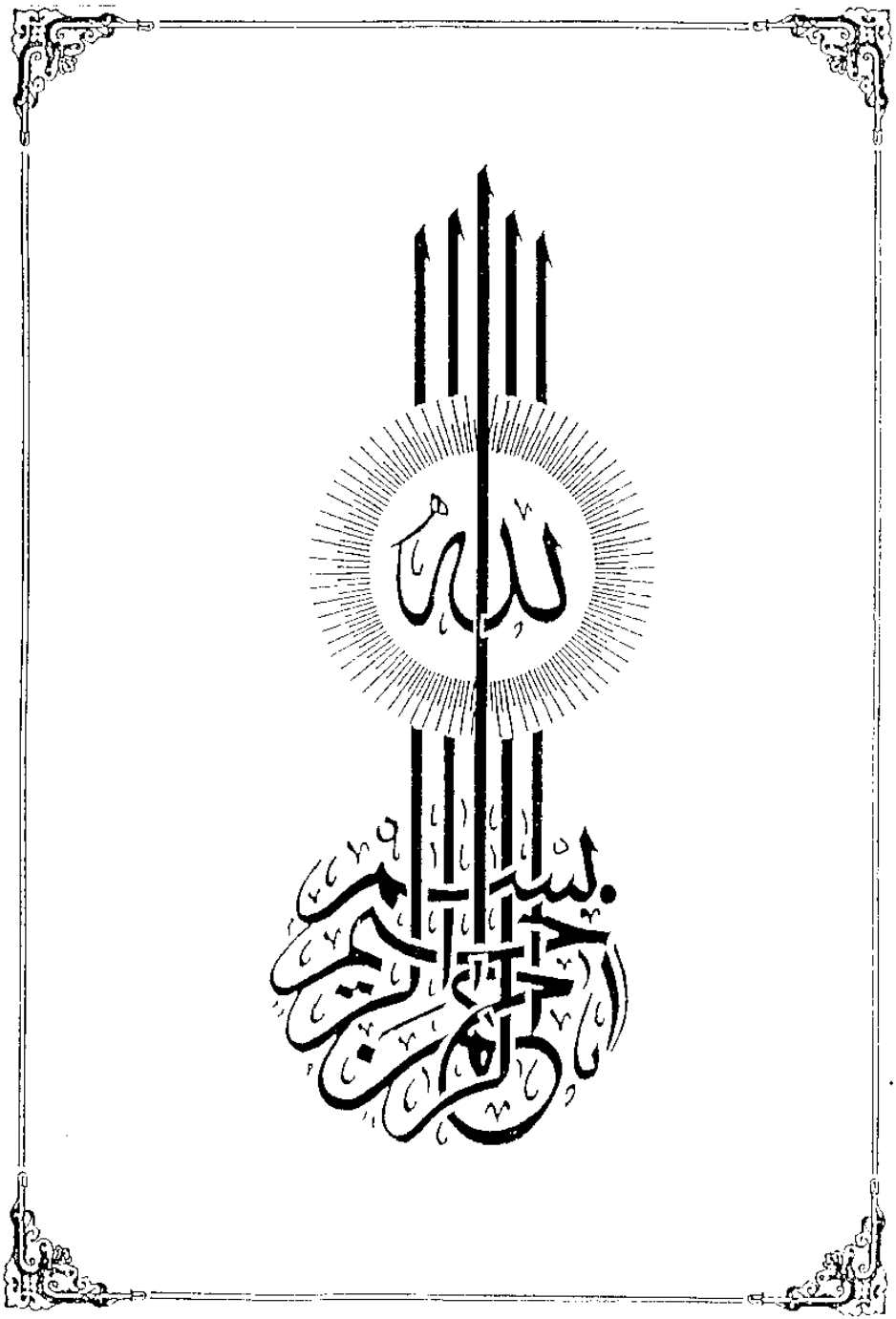

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INTRODUCTION

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Inflammatory bowel disease is a common problem in our country especially that caused by parasitic infestations such as amoebiasis, Bilharziasis; etc. However, non parasitic colitis is not a rare problem and usually its diagnosis is difficult because of the existence of the more common and more simply diagnosed parastic colitis.

Ulcerative colitis and Crohn's disease are the most common and most challenging types of colitis both in diagnosis and management.

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.

CLASSIFICATION

CLASSIFICATION OF INFLAMMATORY BOWEL DISEASE

According to **Joseph B Krisner** (1985) inflammatory bowel disease (IBD) could be classified into:

Idiopathic

Ulcerative colitis

Ulcerative proctitis

Crohn's disease

Ileojujenitis

Intermediate colitis

Bacterial

Shigella

Salmonella

Gonococcal

Syphilitic

Tuberculosis

Pathogenic E. coli

Staphylococcal entero colitis

Yersinia entro colitis

Campylobacter ssp. jejuni

Pseudomonas entero colitis

Parasitic

Amoebiasis

Schistosomiasis

Balantidiasis

Crypto-sporidiosis

Fungal

Histoplasmosis

Viral

Cytomegalvorus

Herpesvirus proctitis

Nor walk virus, other virus, "viroids"

Irradiation induced colitis

Drug, Chemical Food-related

Antibiotics

Cytotoxic drugs (5 FU)

Heavy metals (Mercury)

Milk protein allergy

Soybean colitis

Cathartic colitis

Soaps, detergents

Hydrogen peroxide.

Miscellanesus

Ischemic colitis

Solitary ulcer, rectum

Eosinophilic entero colitis

Collagenous colitis

Uremic colitis

Diverticulitis

Allergic proctitis

Metabolic e.g. scleroderma

Leukemia associated colitis

Inflammatory bowel disease include a wide range of etiologically identified and idiopathic disorders as well as miscellaneous entities.

The "non specific inflammatory bowel disease" include ulcerative colitis and proctitis and Crohn's disease of the gastrointestinal tract, their designation as non specific reflects the continuing mystery as to their origin.

Ulcerative colitis and Crohn's disese share many clinical and laboratory features but could be differentiated on the basis of composite clinical, endoscopic radiologic and pathologic findings.

PATHOLOGY

PATHOLOGY OF ULCERATIVE COLITIS

A- Definition:

Ulcerative colitis may be defined as an acute, subacute or chronic inflammation of the colon and rectum (proctocolitis) of unknown etiology or pathogenesis with the following features.

(1) a variable course, unpredictable prognosis and many local and systemic complications.

(2) rectal bleeding, diarrhea, cramping abdominal pain, fever, anorexia and weight loss.

(3) proctosigmoidoscopic and roentogen features that are usually diagnostic.

It is predominantly a diffuse muscosal disease with continuous involvement without skip areas and is characterized pathologically by crypt abcesses, ulcerations, increased capillary formation and vascular congestion (Farmer et al 1985).

B- Aetiology:

The search for a single cause of ulcerative colitis may be unrealistic, several aetiological factors may have in common initial injury to colonic mucosa in biological predisposed individuals. (Schwartz, 1983).

1. Viral Etiology:

Interest in a possible viral cause for IBD is supported by the clinical similarity of ulcerative colitis to a viral colitis Lymphopathia venereum (Monif & Hood 1970).

AT the present time, there appears to be no conclusive evidence of a caustive virus in the etiology of IBD. While accepting the validity of these considerations. The possible involvement of viral agents (e.g., "Slow virus" or "Viroid") can not be excluded (Prusiner 1982).

2. Bacterial infection:

Bacteriologic interest in ulcerative colitis originally involved streptococcus faecalis, Entamoeba histolytica, hemolytic E. coli, proteus, Mycoplasma (Kirsner & Palmer 1954).

Attention currently is directed to intestinal anaerobes, variant cell wall deficient bacteria and mycobacteria.

However, no pathogenic organism exclusive to ulcerative colitis has been identified inspite of the fact that agglutinating antibodies against E. coli antigens

are more frequent and in high titres in patients with ulcerative colitis than in matched controls **(Heddle & Shearman 1979)**, but no correlation exists between the E. coli agglutinin and the site, extent and severity of IBD.

3. Psychogenic Consideration:

Stress may contribute to illness by altering host immune responses (Keller et al 1981). Such considerations provide a plausible basis for the participation of psychogenic stress in IBD **(Almy 1980)**, but the fundamental question of an initiating role remains unresolved.

As percieved presently, emotional stress or nervous tension does not directly cause ulcerative colitis. Undoubtedly, however, they can "non specifically" precipitate or intensify IBD.

4. Genetic Aspects:

Multiple occurrences of IBD in the same family are identified in from 20% to 40% of IBD. First degree relatives (parents, siblings) are more vulnerable than second degree relatives (aunts, uncles). Genetically influenced susceptibility to IBD is further supported by the association of IBD with ankylosing spondylitis 10 to 20 times more often than in general population **(Mc Connell, 1972)**. Also, among patients with ankylosing

spondylitis, the prevalence of IBD in many series was higher than could be anticipitated by chance (Costello, et al. 1980).

However, ulcerative colitis is not genetically transmitted disorder and no genetic marker of ulcerative colitis have been revealed as yet.

5. Immune Mechanisms:

The possible involvement of immune mechanisms in IBD was proposed by Kirsner and Palmar in 1954. On the basis of suggestive clinical observations: (1) The association of IBD with immunologically mediated conditions such as iritis, thrombocytopenic purpura, systemic lupus erythematosus and Hashimoto's thyroiditis; (2) The increased frequency of IBD among children and adolescents with potentially hightened immunologic responsiveness; (3) The immune related concomitants of IBD (e.g.; erythema nodosum, pyoderma gangrenosum, cutaneous vasculitis, autoimmune hemolytic anaemia); (4) The favourable therapeutic responses to corticosteroids.

Truelove (1961) and his collegues conducted a number of studies to examine the hypothesis of milk allergy. In a small series of patients Truelove reported

that removal of milk and milk products from the diet was followed by clinical remission, while their reintroduction into the diet seemed to lead to an aggravation of symptoms in the course of their next few days or weeks.

Despite this suggestive theory evidence, the theory that ulcerative colitis represents an immunologic reaction to certain food has still to be proved, for it has yet to be shown that withholding of certain food substances can cure all patients of their disease (Goligher, 1980).

Shorter and his colleagues have accumulated impressive evidence that a class of circulating lymphocytes in patients with ulcerative colitis exert specific cytotoxicity against the colonic epithelial cells in vitro (Shorter, et al., 1968) and that the effector cells may be FC-receptor bearing cells (Stobo et al. 1976).

Circulating anticolon-antibodies in the sera of these patients may have the binding capacity for a class of peripheral lymphocytes, especially FC-receptor bearing cells, which in turn may cause antibody dependant cell mediated cytotoxicity in the colon (Hibi et al. 1982).