SURGICAL MANAGEMENT
QF
INFLAMMATORY BOWEL DISEASE

ESSAY

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in

General Surgery

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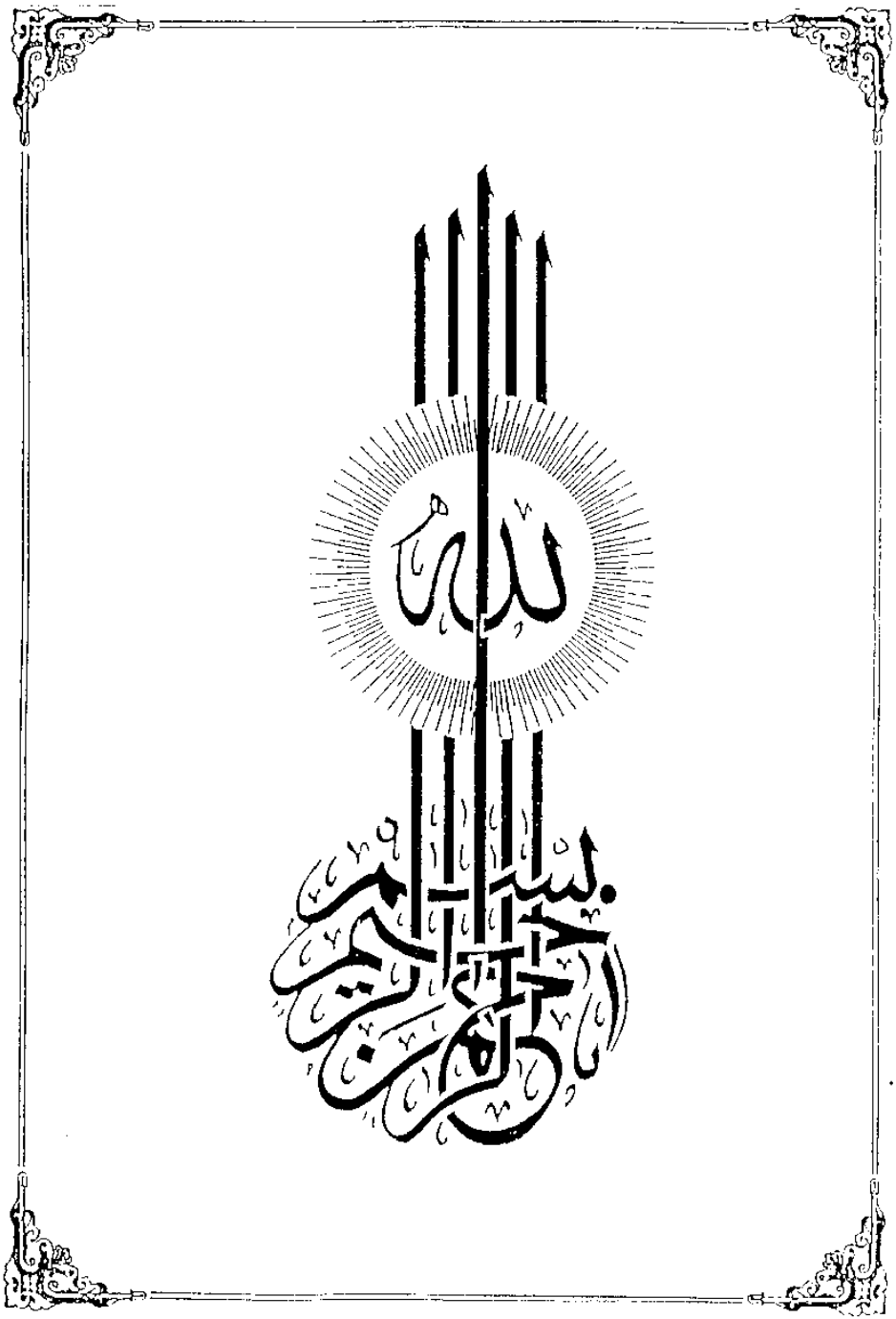

To

My Wife AFAF,

My Daughter DALIA.

ACKNOWLEDGEMENT

First and foremost, I feel always indebted to God, the kind and Marciful.

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Introduction

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Inflammatory bowel disease is general term to desegnate any number of clinicopathologic disorders in childern and adult who present with similar clinical manifestations (fever, diarrhoea with or without blood, distention).

Only through a meticulous diagnostic evaluation including a careful history and physical examination, Laboratory and roentgenographic studies and endoscopic visualization usually with biopsies one can arrive at a reasonably specific interpretation. There are various clues such as : age, extent of disease and anatomic localization that are important in differential diagosis. Some points that should be stressed at the onest are that many gaps still exist in our understanding of aetiology and pathogeneses of these diseases.

Inflammation alone is an inadequate characterization of the tissue reaction since ulceration, necrosis, vascular changes, fibrosis and atrophy are other important morphologic components regardless of the specific diagnostic terminology.

These overlapping features are a constant source of potential confusion to the pathologist who examine these cases. Some specific macro-and micro-scopic findings are important clues (micro-organisms, granulomas, vasculitis, skip areas) when correlated with the appropriate clinical setting.

Pathology

PATHOLOGY OF CROHN'S DISEASE

A. *Definition* :-

It is a chronic granulomatous inflammatory disorder that may involve any segment of the gastrointestinal tract from mouth to anus. The distribution is approximately small bowel 25%, colon 20% and ileocolic 55%. 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.

B. *Aetiology* :

According to O'Donoghue (1981).

Although the cause of Crohn's Disease remains unknown the 1970s have seen a veritable explosion in the interest paid to a number of possible aetiological factors, some of which deserve closer attention.

1) *Trans-missble agent* :

There are currently two popular theories :

I) *Viral* :

Gitnick, Arthur & Shibata (1976), Isolated cytopathic agents from each of four Crohn's disease specimens they studied. E.M appearance

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 out, does not imply a causal relationship.

II) *Cell Wall Deficient (CWD) Bacteria* :-

Cell wall deficient (CWD) bacteria, by their nature, are able to squeeze through a 0.2 u filter. This Knowledge has led to an intensive search in very recent times for such organisms. Parent & Mitchell (1978) isolated CWD basteria from homogenised tissues of all eight Crohn's disease patients they studied. These workers, in a search for mycobacteria, isolated a strain of Mycobacterium Kansasii from a mesenteric lymph node of one of 27 Crohn's disease patients. How ever, skin testing with an extract prepared from this mycobacterium showed that only 20% of a control population gave a positive result, almost 50% of patients with Crohn's disease. Burnham and his colleagues, using hypertonic media, isolated organisms that appeared under the electron microscope to be cell wall deficient from 22 fo 27 Crohn's cultures, 7 of 13 ulcerative colitis cultures, and just one of 11 control samples.

2) *Genetic Factors* :-

The problem of genes versus the environment is reviewed by Lewkonia & Mc Connell (1976) who list four possible explanations for familial aggregation of cases :

a) Random chance,
b) the effect of shared environment,
c) the effects of shared genes, and
d) the effects of shared genes controlling the response to shared environmental influences.

It would appear that genetic factors must be invoked to explain the frequency which familial inflammatory bowel disease is noted and the rarity of its occurrence in the spouse of a patient, while environmental factors must be invoked to explain the increasing incidence of the disease in recent years.

3) *Immunological Aspects* :-

In 1963, Pearlmann & Broberger showed that the circulating leucocytes from patients with inflammatory bowel disease were cytotoxic for human colonic cells grown in tissue culture. This work was confirmed by Watson and colleagues, who showed that the likely effector cell was the lymphocyte (Watson, Quigley &

Bolt, 1966b).

In 1970, workers in the Mayo Clinic made the fascinating observation that normal lymphocytes could be made cytotoxic for colonic epithelial cells by incubating them with serum from patients with inflammatory bowel disease (Shorter et al, 1970). Stobo and colleagues have suggested that the non-T, non-B killer cell is the effector lymphocyte in this system (Stobo et al, 1976), and Thayer (1976) has postulated that immune complexes are the triggering mechanism. Although it may be difficult to implicate these mechanisms in the aetiology of Crohn's disease because of the problems mentioned above they may, nevertheless, be of importance in the pathogenesis of the disease and further work in this area of cell cytotoxicity might lead to a more rational form of treatment.

4) *Diet* :

Thornton, Emmett & Heaton (1979) analysed the pre-illness diet of 30 newly diagnosed patients with Crohn's disease and prepared them with 30 well-matched controls. They found that the patients had a diet that was high in refined sugar and in raw fruit and vegetables and they suggested that this may favour the development of the disease. In a second study, the same group of

workers showed that patients placed on a fibre-rich, unrefined carbohydrate diet in addition to conventional treatment faired significantly better than matched patients who were given similar treatment but no dietary instruction (Heaton, Thornton and Emmett, 1979).

C. *Pathological Picture* :

1) *Macroscopic Description* :-

a). *External Appearance* :

In Crohn's disease the overall length of the lowel is practically constant. In the involved region of the bowel, fat tends to grow over the serosa (Marshak, 1975).

Oedema and congestion of the outer surface of the bowel were observed during operation in less than half the cases. One or several zones of contracture (stricutre formation) were observed in many of the cases due to localized thickenings of the bowel wall. Diseased segments are beefy, dull purple red, thickened to two or three times of the normal diameter and covered with strands and patches of thick, greyish white exudate. The Thickened bowel wall is very firm, rubbery, and virtually incompressible. The involved

segments are often adherent to adjacent bowel loops or other viscera, or several loops may be matted together in a bulky conglomerate mass (Hawk, 1967).

b). *Internal Appearance* :

The most important feature of this disease is the presence of fissured ulcers both longitudinal and transverse, in a net-work like manner, implying the classical *cobblestone* appearance. In a minority of cases the longitudinal ulcers were large, paralleling the axis of the bowel. The ulcer bases were carpeted with purulent discharge (Lockhart Mummary, 1964). In a moderate number of cases the ulcerations were smaller, confined to the mucous membrane, with irregular contour, and always in the space between the principal lesions. The mucous membrane in between the ulcers is usually normal, but sometimes it is oedematous, congested and haemorrhagic.

Digitiform polypoid lesions may present, due to cicatracial process.

Fistulae may be seen which may be external or internal (Stienberg 1974).