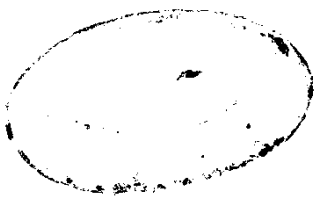
ULCERATIVE COLITIS

An Essay

Submitted for Partial Fulfillment of
Master Degree of *General Surgery*



Presented by

OSAMA ALI MOHAMED EL-ATRASH
M.B., B.Ch.

Supervised by

Prof. Dr. M. SAMIR ABOU ZEID
Professor of General Surgery
Faculty of Medicine, Ain Shams University

Dr. ALAA EL-DIN ABDALLA FARRAG
Lecturer of General Surgery
Faculty of Medicine, Ain Shams University

Faculty of Medicine

Ain Shams University

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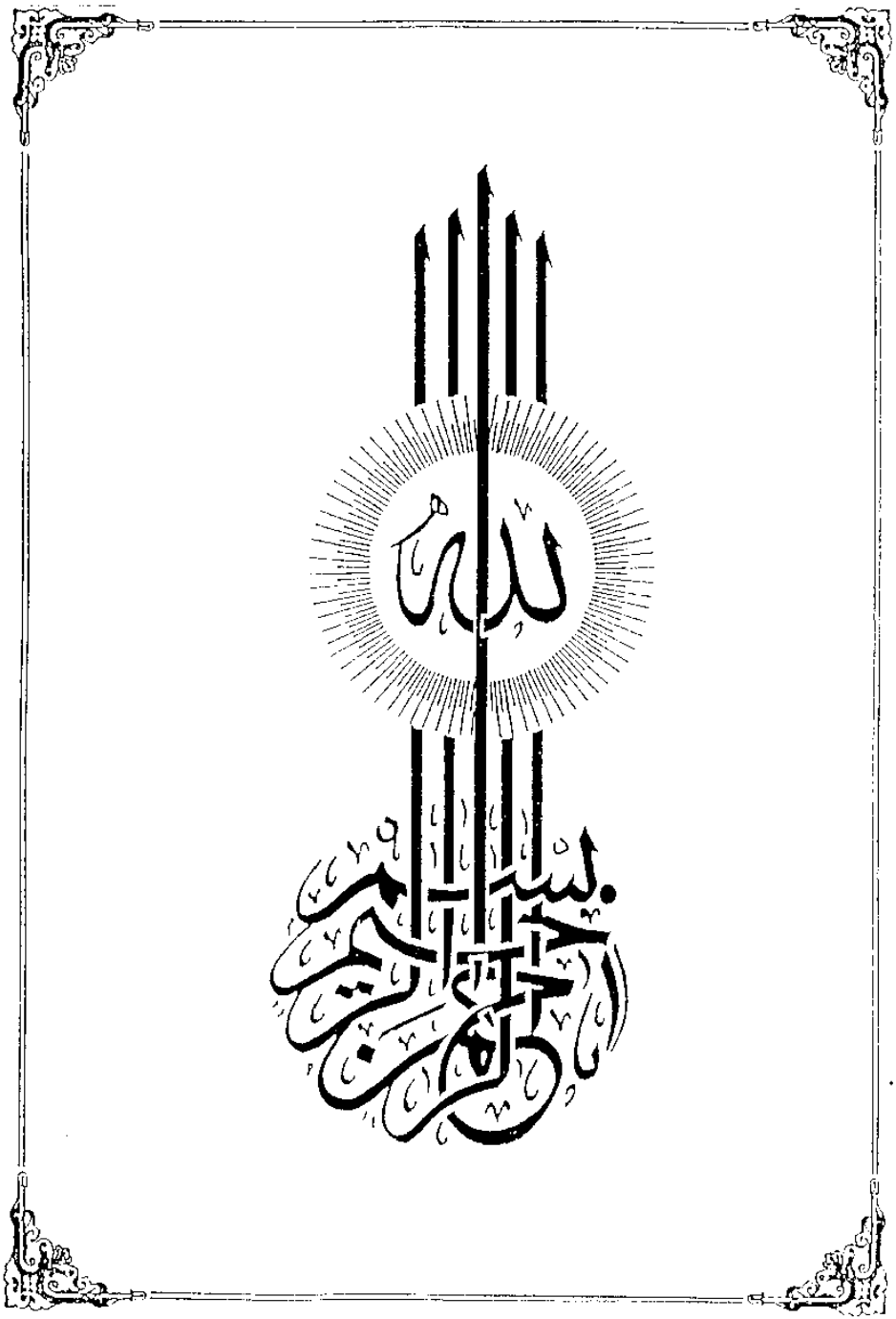
بسم الله الرحمن الرحيم

« سبحانك لا علم لنا إلا ما علمتنا
انك أنت العليم الحكيم »

قرآن كريم



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INTRODUCTION

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Ulcerative colitis is an inflammatory disease involving primarily mucosa and submucosa of the colon. It appears to have a genetic component and to involve the immunological system.

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.

The aim of this work is to present the different items of ulcerative colitis, focusing on the main operative techniques for conserving continence in the surgical management of ulcerative colitis.

EPIDEMIOLOGY

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Little is known about the epidemiology of ulcerative colitis. Since it was first separated from infective dysenteries, it has been shown to attack persons of all ages from early childhood to old age. Judging from the series of hospital cases from United States, both sexes are affected equally, and the mortality rates of the sexes are similar. ***(Sloan et al, 1950)***

In some European countries, however, women seem to be affected more commonly than men **(Rice-Oxley et al, 1950)**, and have a higher mortality. ***(Acheson et al, 1969)***

The age at onset of ulcerative colitis has a bimodal distribution with the first peak between ages 15-30 ys., and a second lower peak in the sixth to eighth decade. ***(Garland et al, 1981; Lawrence, 1983)***

Ulcerative colitis has been reported throughout the world from as far apart as India. ***(Tandonetal, 1965)***. It is possible that it is as common in these countries as in western Europe, but the true prevalance is masked by other infectious causes of diarrhea. ***(Moyberry, 1985)***

There is now strong statistical evidence that ulcerative colitis is two or three times as frequent amongst jews as among non jews. ***(Weiner and Lewis, 1960; Acheson and Nefzger, 1963)***

In Acheson and Nefzger's (1963), study of United States Army personnel, it was found that ulcerative colitis was commoner among officers than among enlisted men; white officers were

affected almost twice as frequently as white enlisted men, but there was no significant difference between rates in white and Negro enlisted men. There have been a number of reports of families in which several members have inflammatory bowel disease and it seems likely that there is a genetic predisposition to these conditions but the magnitude of the risk is uncertain. ***(Mayberry, 1985)***

It seems justifiable to assert that there is a genuine familial predisposition to ulcerative colitis. However, it is important to remember that at least 90% of the patients have no familial history of the disease. ***(Goligher, 1980)***

It has been confirmed that there is no correlation between the incidence of colitis and the ABO and MN blood groups or secratory status, but a positive correlation was found with Rhesus type cc. ***(Boyd et al, 1961; Thayer and Bove 1965)***

AETIOLOGY

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The search for a single cause of ulcerative colitis may be unrealistic. The colon can respond to injury (in the broad sense) in a limited number of ways. Several aetiological factors may have in common initial injury to colonic mucosa in biological predisposed individuals. ***(Schwartz, 1983)***

Several theories have been advanced, of which the main ones have been infective, psychosomatic and immunological. ***(Truelove and Emanol, 1973; Goligher, 1980)***

Extensive studies into the infective hypothesis have failed to implicate specific micro organisms. ***(Truelove, 1970; Goligher, 1980)***

Although most of the gastroenterologists believe that the cause of ulcerative colitis is unknown , a large number of reports by psychiatrists discuss it as an unquestionable psychosomatic illness.

Out of thirty four consecutive cases of ulcerative colitis studied psychiatrically twenty nine of the patients were regarded as "normal", five as abnormal with variable types of personalities ***(Feldman et al, 1967)***. Of these thirty four patients, seven had undergone fairly extensive psychatric care lasting from several months to eight years, but in none did it have an appreciable effect on the course of illness. Similarly, an epidemiological study of social and psychological factors failed to elicit any strong evidence in a support of the psychosomatic theory. ***(Mendelof et al, 1970)***

The generally favorable influence of adrenal corticosteroids on the course of chronic ulcerative colitis has led to a revival of the hypothesis that allergy or hypersensitivity plays a role in the genesis of the disease.

Truelove (1961), and his colleagues 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, whilst their reintroduction into the diet seemed to lead to an aggravation of symptoms in the course of their next few days or weeks. Taylors and Truelove (1961), estimated the serelogical reaction to purified cow's milk protein in patients with ulcerative colitis and in normal subjects and found that the colitics were more liable to have a high titre than was the normal.

Despite this suggestive theory evidence, the theory that ulcerative colitis represents an immunolgical reaction to certain foods 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)***

Although several investigators have described abnormalities of cellular and humoral immunity in patients with ulcerative colitis, the pathogenesis of ulcerative colitis has remained unresolved.

In the past two decades, circulating anti-colon antibodies have been detected in the sera of patients with ulcerative colitis. ***(Broberger and Perlmann, 1959; Marcussen, 1978)***

Shorter and his colleagues have accumulated impressive evidence that a class of circulating lymphocytes in patients with ulcerative colitis exert specific cytotoxicty 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)***. On the other hand, the presence of serum factors with apparently combining capacity and cytotoxic activity for lymphocytes (lymphocytophilic and lymphocytotoxic antibodies) has been reported in patients with ulcerative colitis. ***(Korsmeyer et al, 1974)***

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)***. Chapman and his colleagues found that about 17% of patients with ulcerative colitis possess a circulatory antibody to a colonic antigen. These antibodies were mainly of IgG or IgA class, and only 15% of the patients with anticolon antibodies had an IgM antibody. There was a markedly increasing frequency of circulating anticolon antibody in patients with primary sclerosing cholangitis in association with ulcerative colitis compared with patients with ulcerative colitis alone. ***(Chapman et al, 1986)***

The possibility that ulcerative colitis is an autoimmune disease has been suggested by the presence of a disease-specific protein(s) in the colonic mucosa of these patients, and formation of specific immune complex that might occur in vivo in the mucosa. Demonstration of complement on the basement membrane of colon epithelium of patients with ulcerative colitis is consistent with local immune complex formation. Extraintestinal manifestations associated with ulcerative colitis may also result from deposition of immune complex. ***(Thayer, 1976)***

However, further studies are necessary to explore if the protein(s) present in the colonic mucosa is derived from a bacterial protein that may be directly involved in the etiology of ulcerative colitis. Alternatively ,the protein(s) might be derived from colonic epithelial cells that became antigenic after a primary assault, or following incorporation with a bacterial protein. ***(Nagai and Das, 1981)***

Lawrence in (1983), postulated that external agents, host responses, genetic immunological infleunces interact in the pathogenesis of inflammatory bowel disease. According to this concept, ulcerative colitis and Crohn's disease are different manifestations of a single disease process. The host becomes sensitized to the antigens of inciting external agent or agents e.g. microbal, viral or dietary.