ULCERATIVE COLITIS

ESSAY

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(GENERAL SURGERY)

By

ADEL ABDEL WAHED ABDEL MONEIM
M.B., B.Ch.

Supervised By

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

Prof. Dr. KHAIRY SABER
Chairman of General Surgrey Dept.
Maadi Armed Forces Hospital

Faculty of Medicine

Ain Shams University

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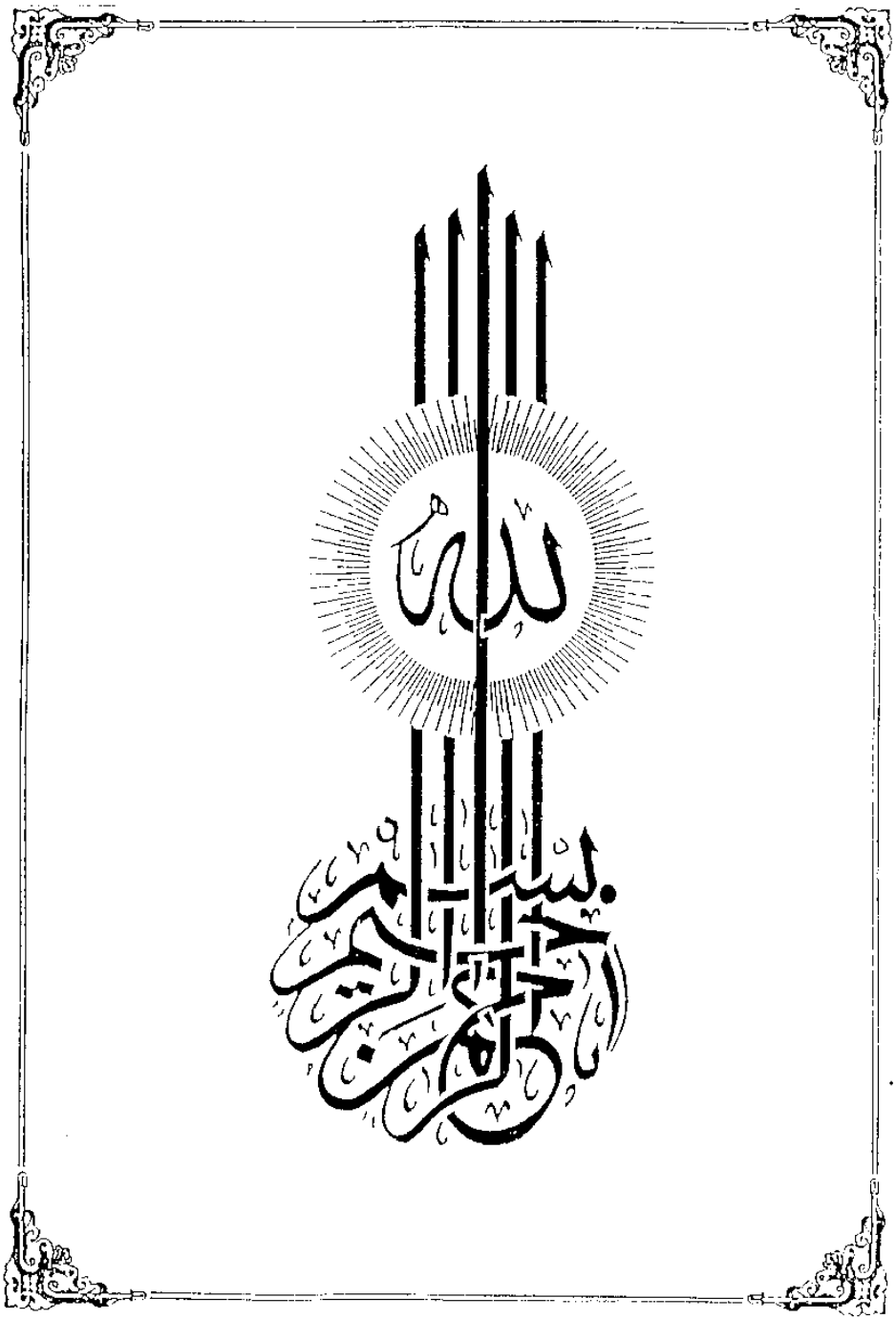

C O N T E N T S

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INTRODUCTION

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Ulcerative colitis is an inflammatory disease of unknown origin that varies in severity and clinical course. It occurs most often in patients 30 - 40 years old. It appears to have a genetic component and to involve the immunological system.

The disease may be classified by either the severity of an attack, the clinical course or by anatomic localization of inflammation.

The aim of this work is to present an up todate study of different items of ulcerative colitis.

E P I D E M I O L O G Y

EPIDEMIOLOGY

Ulcerative colitis is predominant in young adults particularly women (Cullinan and MacDougall, 1957; Edwards and Truelove, 1963; Watts et al., 1966 c,d; Lawrence, 1983). The disease is twice as common in England as in other European countries (Melrose, 1955, 1956). It is found worldwide but it is more common in Western countries (Lawrence, 1983).

The disease is two or three times as frequent amongest Jews as among non-Jews.(Weiner and Lewis, 1960). It is commoner among officers than among enlisted men (Acheson and Nefzger, 1963). The age at onset of ulcerative colitis has a bimodal distribution with the first peak between ages 15 - 30 years and a second lower peak in the sixth to eighth decade (Garland et al., 1981; Lawrence, 1983). There is no correlation between the incidence of ulcerative colitis and ABO and MN blood groups but a positive correlation was found with Rhesus type c.c. (Boyed et al., 1961; Thayer and Bove, 1965). There is a familial predisposition to ulcerative colitis (Sloan et al., 1950; Barker, 1962; Kirsner and Spencer, 1963; Lawrence, 1983). However, at least 90% of patients have no family history of the disease (Goligher, 1979).

AETIOLOGY

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The cause of ulcerative colitis is unknown but several theories have been advanced, of which the main ones have been infective, nutritional, psychosomatic and immunological (Truelove and Emanoel, 1973; Goligher, 1979).

The cause of ulcerative colitis was attributed to infection by Bargen (1924). Extensive studies into the infective hypothesis have failed to demonstrate a pathogenic organism as the agent directly responsible (Truelove, 1973; Goligher, 1979). It is possible that bacterial allergens may be involved in some of the immunological disturbances occuring in the disease (Truelove and Emanoel, 1973).

The psychosomatic theory was suggested as patients with ulcerative colitis are often highly strung and their attacks frequently coincide with periods of increased stress (Murray, 1930). Psychotherapy can produce a variable clinical improvement in patients with ulcerative colitis (Paulley, 1956). Various investigations have considered that the subjects who develop ulcerative colitis have a characteristic personality and that these personality factors are present long before the onset of

the colitis (Engel, 1966). However, in the most recent full-scale psychological study, most of the subjects with ulcerative colitis were assessed as psychologically normal, while an emotional disturbance shortly before the onset of the illness was not usual (Feldman et al., 1967). Similarly, an epidemiological study of social and psychological factors failed to elicit any strong evidence in support of the psychosomatic theory (Mendeloff et al., 1970).

Attempts to identify a nutritional cause have likewise proved fruitless (Truelove and Emanoel, 1973).

The most popular theory at the moment is that ulcerative colitis represents an immunological response to antigens which may be alimentary "foods, chemicals or drugs", bacterial or autogenous (Andreson, 1925). Clinical observations have also suggested the possibility that the disease may be an example of food allergy, at any rate in some of the subjects. Cow's milk is the item of diet which has been incriminated most often (Andreson, 1942; Rider et al., 1960; Truelove, 1961). The patients with ulceratie colitis frequently have high titres of circulating antibodies to cow's milk proteins (Taylor and Truelove, 1961) and a controlled dietary trial has shown that a minority of patients benefit from a milk-free diet (Wright

and Truelove, 1965). However, the beneficial effects of a milk-free diet may be unconnected with immunological processes and may be due to the removal of lactose from the diet, which in turn is beneficial to those subjects who suffer from associated hypolactasia (Pena and Truelove, 1973). Despite this suggestive evidence, the theory that ulcerative colitis represents an immunological 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, 1979).

The possibility that ulcerative colitis is an auto-immune disease has received much attention in recent years as circulating autoantibodies to colon were demonstrated in the sera of patients with ulcerative colitis (Broberger and Perlmann, 1959). Unfortunately, these autoantibodies do not correlate with the severity or the course of the disease, which immediately suggests that they may be a response to the damaged epithelium rather than a prime cause of the damage (Wright and Truelove, 1966).

Cellular rather than humoral hypersensitivity is anoth is supported by the demonstration of a cyto-toxic action of leukocytes from patients with ulcerative colitis against human colonic

epithelium growing in tissue culture (Broberger and Perlmann, 1963).

Another immunological feature of potential importance is the cross-reaction which has been observed between colonic epithelium and some of the colonic bacteria, notably E. coli 014 (Perlmann et al., 1967). This raises the possibility that an intestinal infection might set up immunological processes which themselves would damage the colonic epithelium (Truelove and Emanoel, 1973).

Multiple factors interact in the pathogenesis of ulcerative colitis such as external agents, host responses and genetic immunologic influences. Accordingly, ulcerative colitis and Crohn's disease are different manifestations of a single disease process. The host becomes sensitized to the antigens of the inciting external agent or agents e.g. microbial, viral or dietary. Once immunologic priming of the gut is established, any insult that increases mucosal permeability to those antigens can precipitate an inflammatory reaction in the bowel wall. The types of antigens and many other factors determine the nature of the inflammatory process e.g. Crohn's disease or ulcerative colitis (Lawrence, 1983).

PATHOLOGY