STUDY OF DERMATOGENIC ENTEROPATHY IN CASES OF PSORIASIS

THESIS

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By

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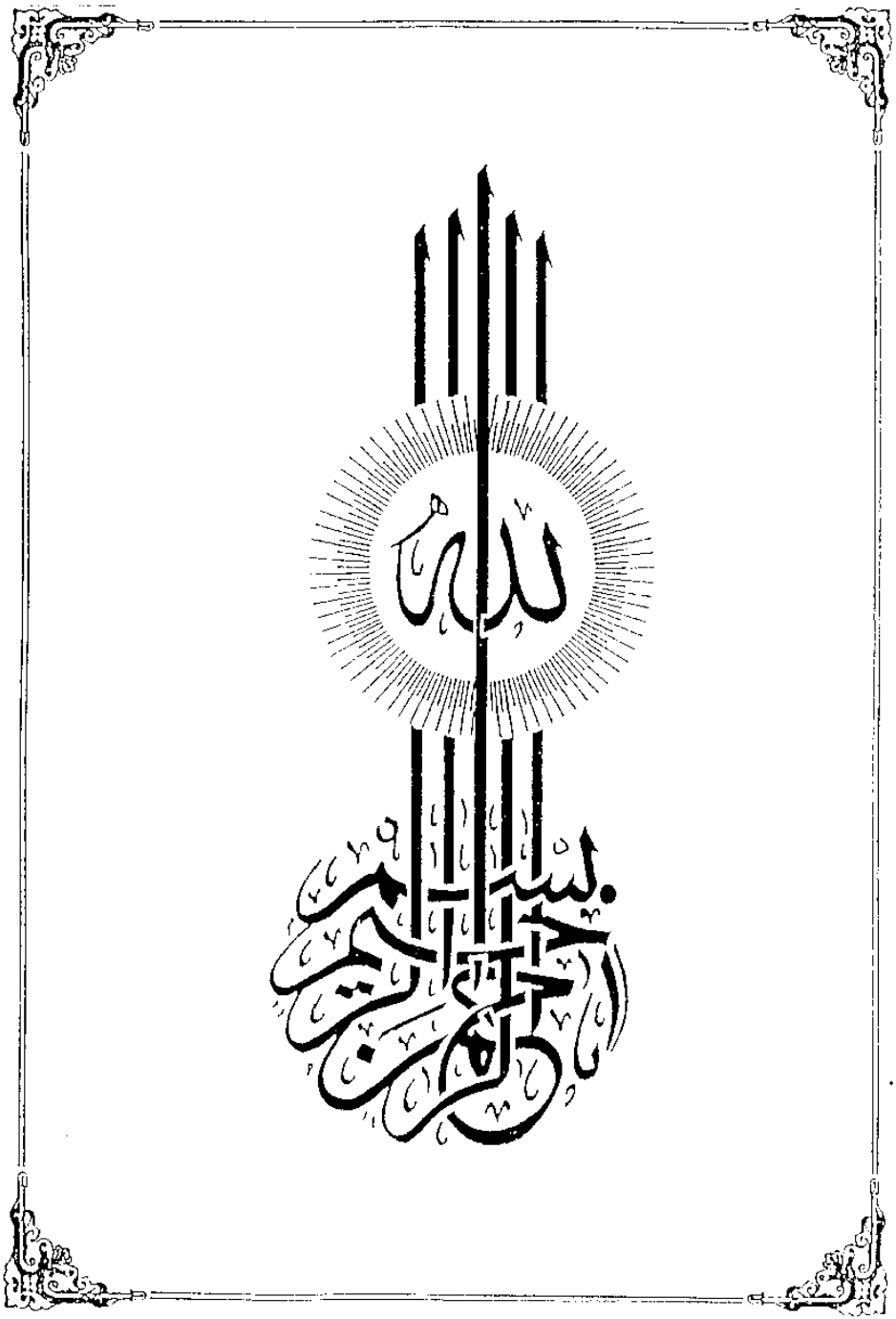
TO MY MOTHER WITH MY

ALL LOVE AND BEST WISHES



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C O N T E N T S

INTRODUCTION

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The skin and the mucosae of the alimentry tract are in direct continuity with each other at the lips and anal orifice, and may be affected jointly in a number of diseases. As a result malabsorption ... and morphological abnormalities of the small intestine have been described in association with no less than eight skin diseases namely dermatitis herpatiformis (Marks, et al., 1966; Brow et al., 1971; Weinstein et al., 1974), psoriasis (Shuster et al., 1967), rosacea (Watson et al., 1965), eczema (Marks, and Shuster,1970), subcorneal pustular dermatosis, ichthyosis, acrodermatitis anteropathica and Brocq's erythrodermia (Trier, J.S., 1971). Evidence of altered intestinal function is not convincing in some of the eight skin diseases. Available reports are often conflicting and controdictory and in some instances, the number of documented cases of this relationship is so small that coincidental occurrence of skin and intestinal diseases cannot be excluded (Jrier, 1971).

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Many metabolic and other systemic consequences have been noticed in association with skin diseases. One of these consequences is the incidence of malabsorption, which was first reported by (Shuster and Marks, 1965). Severity of malabsorption was related to the degree of the skin surface involved, also malabsorption responded to topical treatment of the rash (Shuster and Marks, 1965). This relation was also suspected in some other skin conditions e.g. psoriasis but was not definitely proven by comprehensive studies to prove or disprove such an association.

In the present work , a study of the small intestinal absorption in cases of psoriasis is done using the D-xylose absorption test and the lactose tolerance test to screen the functional ability of the small intestine.

REVIEW OF LITERATURE

Dermatitis Herpetiformis

Dermatitis herpetiformis is an intensely pruritic vesicular skin disease of unknown cause which responds dermatically to sulphapyridine or sulfones.

It is well established that most patients with DH have a flat proximal intestinal mucosal lesion (Marks et al., 1966; Brow et al., 1971), which reverts towards normal on a gluten free diet (Weinstein et al., 1974). Hence these patients are asymptomatic because their flat mucosa is confined to the duodenum and proximal jejunum (Brow, et al., 1971). Some patients with DH do not have diffuse flat proximal intestinal mucosa on random biopsy. When multiple biopsies are taken, they are found to have either patchy proximal intestinal lesions or in some instances, completely normal intestinal mucosa (Brow, J.R., Parker. F., Weinstein W.M., 1971). Studies were carried out on those patients with DH and morphologically normal small intestinal mucosa (Weinstein, 1974). This study revealed that they had a latent form of coeliac sprue. This latent form of the disease was unmasked by the administration of a high gluten diet to 2 subjects with DH who had previously had morphologically normal small intestinal mucosa.

The administration of this diet was continued 22 to 30 weeks. Moderately abnormal and severe " flat "

mucosal lesions were induced in both patients with DH, but not in the normal volunteer, Yet neither patient with DH developed clinical or laboratory evidence of malabsorption on completion of the high gluten diet, one of the two patients with DH was studied on a normal gluten containing diet, and his intestinal mucosa reverted to normal by 19 weeks. Latent celiac sprue may be defined as a condition in whitch the intestinal mucosa becomes unequivocally abnormal in response to increased dietary gluten. The diagnosis of celiac sprue is made by the small bowel biopsy finding of a severly abnormal or flat mucosa near the duodenojejunal junction and by an unequivocal clinical and biochemical response to a gluten-free diet. Those patients with coeliac sprue, who have few or no symptoms before treatment should also have a biopsy taken aftertreatment with the gluten free diet to document reversion of the flat mucosa towards normal. The patchy distribution of the lesions incluced with the high gluten diet is similar to that described in some DH patients studied on a regular diet (Weinstein, 1974).

In DH there is cutaneous deposition O IgA , and there is circulating immune complexes and an increased incidence of histocompatability antigen HLA-B8, (there is also increase incidence of this HLA-B 8 in cases of

coeliac disease), as well as an association with immunologic disorders, e.g. thyroid disease, rheumatoid arthritis, chronic liver disease, cutaneous vasculitis, addisons disease, systemic lupus erythromatosis and ulcerative colitis. Circulating immune complexes have been detected using three different methods in many patients with celiac disease and dermatitis herpetiformis. These complexes contain either IgM and IgG alone or both IgM and IgG (Mohammed et al., 1976). The antigenic moiety of the complexes is unknown while gluten is known to be responsible for pathologic characteristics in the small bowel, the lesions in the skin and other organs may be a consequence of tissue damage induced by gluten. This is supported by the presence of antinuclear antibodies against single-stranded DNA (Moorthy et al., 1978). Certainly the diagnosis of celiac disease may not always be straight forward. Some patients may not have the characteristic flat mucosa on a single biopsy, as the lesion may be patchy (Scott and Losowsky, 1975), some patients with mucosal abnormality may not be severe and be detected only on more sensitive, techniques than routine assessment of villous architecture (Robinson et al., 1971; Fry et al., 1972; Stavens et al., 1975). One technique is interepithelial

lymphocyte counting (Ferguson and Murray, 1971; Fry et al., 1972) and although an increased count may be specific for coeliac disease, it has been used as an index of mucosal abnormality in DH (Fry et al., 1972; Fry et al., 1974). In the diagnosis of dermatitis herpetiformis it has been claimed that the usual criteria of clinical and histological features of the rash and response to Dapsone may not be completely reliable and that the best criterion is the demonstration of deposits of IgA at the papillary tips in clinically uninvolved skin (Fry et al., 1974). Another link between the two conditions is an increased incidence of histocompatability antigen HLA-B8. In coeliac disease the reported incidence of HLA-B8 is 66 - 87 % compared with 17 - 30 % in controls (Flachuk et al., 1972 ; Albert et al., 1973; McNeish et al., 1973; Stokes et al., 1973; Scott et al., 1974 a). In DH the initial reports (Katz et al., 1972; Gebhard et al., 1973; White et al., 1973) showed that the incidence of HLA-B8 was significantly increased compared with controls (58-68 % compared with 24 - 31 %), but less so than in coeliac disease. Two recent reports, however, suggest a similar incidence (Seah et al., 1976; Reunala et al., 1976).