TISSUE ENZYMATIC CHARACTERISTIC OF INTESTINAL BILHARZIAL POLYPOSIS

A THESIS

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BY

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بسم الله الرحمن الرحيم

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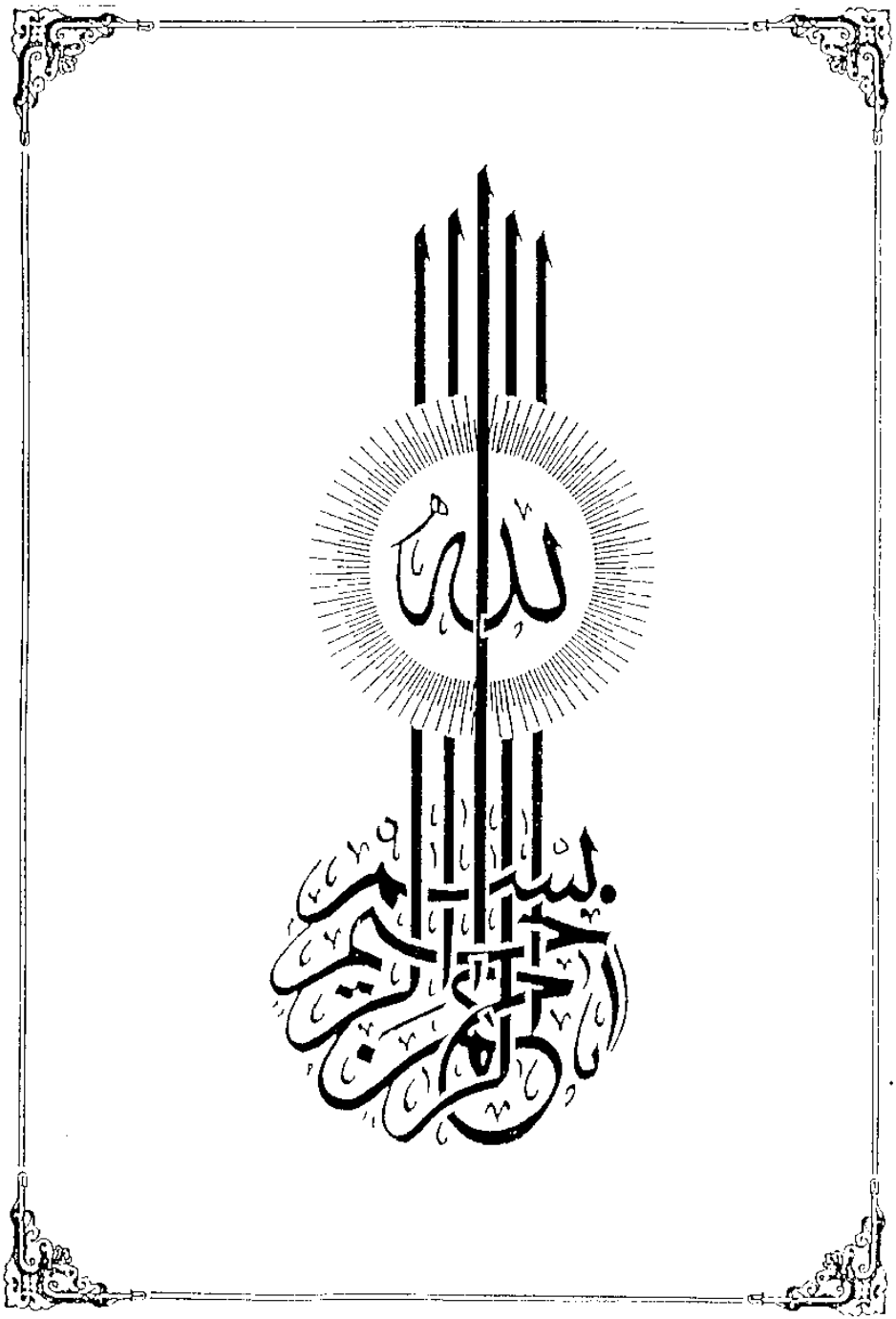

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The total activities of glucose-6 phosphate dehydrogenase (G.6-P.D) lactate dehydrogenase pyruvate kinase have been shown to be higher in concer colon than in normal colonic mucosa (Vatn et al., 1982). Tubular adenomas of the large intestine which were believed at a time to be precancerous have been reported to exhibit similar enzymatic changes.

In our country intestinal schistosomiasis may cause polyposis. Whether bilharzial polyps exhibit enzymatic patterns different from normal mucosa and/or other possible bilharzial lesions is not yet defined.

The aim of this work is to study the above mentioned enzymes in homogenates of intestinal bilharzial polyps in correlation with their histopathological picture and its repercussion on the possibility of malignant transformation.

REVIEW OF LITERATURE

ANATOMY OF RECTO-SIGMOID COLON

According to Boyed et al. (1958), Ellis (1960), Last (1972), and Morson and Dawson (1972), the following description is given:

I. The sigmoid (pelvic) colon:

It is variable in length varying from 5-30 inches, average 15 inches. It extends from the iliac colon at the pelvic brim to the commencement of the rectum in front of the third piece of the sacrum. It is completely ivested in the peritoneum and hangs free on a mesentery, the sigmoid (pelvic) mesocolon. There is no change in the gut wall between terminal sigmoid colon and upper rectum; the distinction is only of peritoneal attachment. Where there is mesentery the gut is called sigmoid. Where the mesentery ceases gut is called rectum.

II. The rectum:

It is about 5 inches long. It extends from the front of the third piece of the sacrum at the termination of the pelvic mesocolon to a point at the level of the apex of the prostate gland 1 to 1 1/2 inches in front of the coccyx, at which it bends sharply backwards to become continuous with the anal canal. The rectum is moulded to the concavity of the sacrum and cocyx. At its junction with the anal canal it is dilated to form the ampulla. The peritoneum covers the front and sides of the upper third of the rectum and the front of the middle third. The lower is devoid of peritoneum.

III. Anal canal:

It is about 1 1/2 inches long. It is a short canal connecting the rectum to the exterior acting mainly as a sphincter controlling the defaecation process. It is a slit-like canal when empty, but during the passage of the faeces it may become greatly distended. Laterally it is separated from the fat of the ischio-rectal fossa by the pevator ani muscle and the external sphineter. Anteriorly, in the male, the canal is separated from the membranous urethra by the perineal body, while in the female it is related to the perineal body and the lower part of the vagina, posteriorly, it is related in both sexes to the ano-coccygeal body. For the greater part of its length the anal canal is surrounded by sphincteric muscles which control the mechanism of defaecation, the internal involvuntery and external voluntary sphincter.

and is follicular in shape. The mucosa shows tiny ulcerations due to extrusion of ova.

Pathogenesis:

Sorour (1928), stated that when the deposited ova get impacted in tissues, they disintegrate liberating toxic proteins which evoke a severe inflammatory response. Hashem (1962), Owed punctate haemorrhages and ulcers seen in the mucosa to ova extrusion, while the deep minute haemorrhages of the mucosa are due to rupture of the capillary wall due to engaging of the spine of ova in it, which is followed by contraction of the vessel and rupture of its wall, discharging ova out. Elwi (1967), stressed that these early lesions occur in the large bowel when ova deposition is mild in nautre. Elwi, 1976, emphasized that ova deposited in the submucosa and/or digest their way enzymatically causing no significant reaction so long they are not impacted in tissues.

Pathogenesis of schistosome egg granuloma:

The pathogenesis of schistosome egg granuloma which is the main pathological feature of schistosomiasis remained vague until. Warren (1972) stated that the egg after being laid by the worm, the egg embryonates and within a few days begins to secrete soluble substances which pass through the pores in egg shell. These soluble egg antigens sensetize the host, resulting in the development of thymic lymphocytic memory cells. Following sensetization, the further antigenic secretions stimulate the memory cells to release their lymphokines which have an effect on the migration of both

macrophages and eosinophils. Thus lymphocytes, macrophages and eosinophils gather about the egg nidus in the tissues, resulting in the formation of granuloma. According to Von Lichtenberg (1978) the egg granuloma is first elicited by cell mediated immunity via T-lymphocytes but subsequently its size and cellular composition must be under the influence of changing subpopulations of both T and B cells.

II. Lat lesions in intestinal schistosomiasis:

Elwi (1976), stated that the late lesions showed as sandy patches, polypi, ulcers, pericolic masses, fistulas, strictures, rectal prolapse and bilharzial intestinal obstruction.

Schistosomal polypi:

According to Dimmette and Sproat (1955) they affect the rectum in 60.6%, sigmoid colon in 12% rectosigmoid colon in 27.2%. S.mansoni eggs were found in 52.5% and S. haematobium eggs in 30.2%, While a combination of eggs of both species was found in 17.6% of the polyps. Cheever et al. (1978), found that the rectosigmoid colon contained polyps in 67% of all the affected cases they studied.

Pathogenesis of Schistosomal Polyps:

Many mechanisms have been postulated by different pathologists in order to explain how polyps are formed in case of intestinal schistosomiasis. Dimmette et al. (1956), postulated that the ova which are deposited in the veins of submucosa pass through the walls of the veins into the submucosa then to the muscularis mucosa and

mucosa. Some ova are extruded to the lumen while others fail to find on exit and stay in the submucosa. These ova excite infammatory reaction with granulomatous formation and necrosis. Healing by fibrous tissue follows. The muscularis mucosae hypertrophies, these offer a good barrier to the escape of the ova. Such imprisoned ova degenerate and excite more fibrous tissue prodution with foreign body reactio, with repetition of this process a nodule is formed. This nodule elevates the hypertrophied muscularis mucosa and mucosa proper to form the earliest detectable polyp. As more ova are deposited, the inflammatory process progress with qubsequent fibrosis. On the other hand Hashem (1962), and El Roby (1976) emphasized that the ova are deposited in the superficial layer of the submucosa (supramuscularis mucosa), where the connective tissue is loose and allows the accumulation of a large amount of schistosomal granulation tissue. This exuberent tissue-raises the covering epithelium which itself may be the seat of adenomatoid hyperplasia and intense mucoid activity and a papilloma is formed. This may be flat or pedunculated according to the distribution of the ova eliciting the reaction; whether the ova are evenly destributed in a patch of submucosa or not.

Bogliolo (1967), stated that the formation of polyps represent an excessive hyperplastic neoformation disproportionate to the small number of eggs or worms encountered and to the usually slight inflammatory reaction. Polyp formation should be considered as a manifestation of an allergic reaction. In favour of this is also the high flourescent antibody titre and postive immunodiffusion reaction against schistosomiasis reported in these cases by (Ata et al., 1976). As the

submucosal gronulomatous process progresses, the mucularis mucosa becomes shredded, the mucosa is more elevated and it undergoes also, an adenomatoid hyperplasia. As the polyp becomes older, the adenomatoid hyperplasia decreases while fibrosis of the core increases Cheever et al. (1978), reported that involvement of the muscularis mucosae at the base of the polyp together with its oblitration by granulomatous lesions or by calcific ova in a dense fibrous tissue was almost always present. They added that polyps result from tissue destruction at the site of oviposition, the subsequent tissue repair did not restore the defect. It is postulated that large polypi result from peristalitic action on smaller polypi concurrent with continued oviposition and that the polypi occurred at sites where ova were concentrated regardless the species, yet S. mansoni ova burdens were more capable of producing polypi.

Coufer (1971), stated that the majority of schistosomal intestinal polyposis are multiple (could be single), and affect mainly the rectum and sigmoid colon.

The schistosomal polyps vary in size from 2 to 20 mm in diamter and from 2-24 mm in length. They have a red colour but ulceration may change the colour into blackish grey as a result of the associated haemorrhage. Small polyps are sessile but as they increase in size a pedicle could be seen and moreover, small polypoid projections arise from the polyp proper which acquire a mulberry configuration, upon section, the majority of the polyps are friable and gritty. The

mucosal covering of schistosomal polyps particularly those of the rectum may ulcerate as a result of mechanical abrasion produced by the faecal contents of the intestine. Secondary infection and necrosis of the whole polyp may occur and as a result it may casted off leaving a small ulcer with a rounded form, sharp edges and granular floor (Elwi, 1976).

Microscopi picture:

Dimmette et al. (1956), stated that, the typical bilharzial polyp is composed of a fibrous tissue stalk projection from the submucosa towards the lumen of the bowel and covered with mucosa. The mucosa consists of distorted glands showing varied degree of mucoid activity and mucinous degeneration. Areas of adenomatoid hyperplasia with nuclear alteration which resembl those usually classified as "precancerous" in adenomatous polyp, were found in 1.3% of schistosomal polyps of the large intestine. The mucosa is frequently interrupted by focal areas of ulceration. Such ulcers could be small or large. In large ulcers the mucosa is replaced by granulation tissue, with infiltration in the neighbourhood by mononuclear cells, eosinophils and few polymorphonuclear leukocytes. The muscularis mucosa is shredded and even hardly seen in some cases. The supporting tissue of the polyp and the submucosa show either diffuse or follicular bilharzial reaction is usually seen in young and new polyps, while in older polyps,marked fibrosis is present. Schistosoma ova are found in great numbers. They could be viable, non viable or calcified. The viable ova are usually seen enclosed in young fibrous tissue