بعض الدراسات على الكفاءة الوظيفية للبنكرياس
فى الجاموس والأبقار
٤ ـ دراسات مورفولوجيه مجهرية وميكروسكوبية

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شملت هذه الدراسة ، دراسة ميدانية على ٢٠٥ عينات من البنكرياس والكبد فى الحيوانات المذبوحة فى سلخانة مدينة أسيوط ثم دراسة تجريبية على الالتهاب الحاد للبنكرياس بحقن الكلوروفورم فى أنسجة البنكرياس وكذلك دراسة تجريبية على مرض البول السكرى بحقن الحيوانات بمادة الالوكزان .

وقد درست التغيرات المورفولوجيه ودونت النتائج ونوقشت .

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SOME STUDIES ON PANCREATIC FUNCTION IN CATTLE AND BUFFALOES

4- MACRO-AND MICRO-MORPHOLOGICAL STUDY

(With 8 Figures and 4 Table)

By

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SUMMARY

This part of the study concerns with the description of the gross and microscopic appearance of pancreas and liver of cattle and buffaloe after induction of experimental pancreatitis using local injection of chloroform and from animals after experimental diabetes using alloxan. As control specimens collected from slaughter house of Assiut were used.

The correlation between metabolic profile of carbohydrates (blood glucose and hepatic glycogen) and the morphological properties of the endocrine part of the pancreas was considered and discussed. Also the effect of damaged exocrine part of the pancreas on the behaviour of the enzyme parameters was evaluated.

INTRODUCTION

Many cattle and buffaloes have been delivered to the clinic of the faculty with apparent glucosuria, while others were suffering from acute abdominal pain without clear definite causative agents. Liver, kidney, intestine and pancreas may be responsible for such conditions.

In the avialable literature, neither decumented incidence of pancreatic diseases in cattle and buffaloes, nor the pathophysiological picture of these diseases could be found. Moreover,the literature lacks any information about the diabetogenic picture of alloxan in these animal

spieces.

From the morphological point of view, only the natural and experimental diseased conditions of the pancreas in canines were fully described and discussed, (COFFIN and THORDAL-CHRISTENSEN, 1953, ANDERSON and LOW, 1965, ANDERSON and STRAFUSS, 1971, and FREUDIGER, 1975).

The present study is dedicated in order to study, 1) the morphological pancreatic picture and the hepatic glycogen content of cattle and buffaloes in random samples obtained from the abbatoir, 2) the morphological changes and effects of the experimentally induced acute pancreatitis, 3) the alloxan diabetes in these animal spieces.

MATERIALS AND METHODS

In this study three groups of pancreas the liver materials were used. The first consisted of 100 cattle and 105 buffaloe specimens collected at slaughter house of Assiut. The second consisted of specimens taken from three male buffaloes and two male cattle after induction of acute pancreatitis. Acute pancreatitis was produced by the injection of 25 ml chloroform in different sites of the pancreatic parenchyma using surgical procedure adopted after BROBST et al.,(1970), specimens were taken 1,2,4,7 and 15 days after injection. The third group of material was obtained from five male cattle 1,2,4,7 and 15 days after a single intravenous injection of a 5% freshly prepared solution of alloxan in the jugular vein as described by JARRETT (1946), and LUKENS, (1948).

All liver and pancreas specimens were taken soon after slaughter of the animals and fixed in 10% neutral formalin. After good fixation paraffin sections were prepared and stained with Haematoxylin and Eosin. In addition frozen sections from unfixed liver tissue were subjected to a semiquantitative determination of glycogen content using PAS technique (McMANUS, 1948).

RESULTS

In slaughter house material, all the examined liver and pancrease specimens revealed a normal morphological picture both grossly and microscopically. The glycogenic granules in the hepatocytes were regularly distributed within the hepatic lobules.

In animals after experimentally induced acute pancreatitis all the examined liver specimens revealed no gross pathological changes except in one case. In this case there was slight thickening of the bile ducts. Likewise microscopic examination revealed no pathological alterations except in the forementioned case where slight cholangitis and vacuolar degeneration in the hepatocytes were detected. Moreover there was no signigicant difference in the glycogen distribution when compared with the first group of material collected from normally slaughtered animals.

The pancreas was smaller than normal and appeared to be nodular in appearance and firm in consistency. On microscopic examination obvious changes could be found. One and two days after injection,diffuse pancreatic necrosis was observed, (Fig. 1). Seven days after injection, absence of some pancreatic lobules was noticed, which were replaced in some areas by adipose tissue, while in others, proliferation of the interstitium was present. Fifteen days after injection, fibrosis of the pancreas was the main finding, (Fig. 2), which reached in some areas to complete pancreatic sclerosis, (Fig. 3).

In the alloxan injected animals, the liver was swollen, greyish brown to yellowish brown in colour and appeared to be softer than normal. In one case depressed, dark red spots were observed. These spots were irrigular in shape and on fingre pressure seemed to be spongy in texture. The spots varied in size and on cutting showed clotted blood. On microscopic examination proteinous dystrophic changes were observed in all cases examined. The dystrophic changes were observed in all cases examined. The dystrophic changes ranged from parenchymatous degeneration

to vacuolar degeneration, slight proliferation of the reticuloendothelial cells and histiocytic cellular infilterations in the portal triads could be also sometimes seen.

The grossly seen dark red spots appeared microanatomically as a network of blood filled spaces and had a continuation with the hepatic sinusoids. The hepatic parenchyma which surrounded these telangiectitic lesions showed pressure atorophy and dystrophic changes, (Fig. 4).

In regard to the distribution of the glycogen granules in the hepatic cells, marked decrease not only quantitatively but also, nearly the prepheral third of the hepatic lobules, showed no glycogen storage.

In the pancreas, no gross pathological alterations in all the examined cases could be detected, while by microscopic examination, changes in the islet cells could be proved. In comparison with the normal islets in buffaloes, (Fig. 5), in alloxan injected animals, two days after injection vacuolation of the cytoplasm and pyknosis of the nuclei was noticed, (Fig. 6). One week post alloxan injection, complete coagulation necrosis of the islet cells was observed, (Fig. 7). In some areas complete lysis of the cells happened and only remenants of the cellular debris could be observed. Fifteen days, post alloxan injection, well marked fibrosis in the islet places was noticed, (Fig. 8).

DISCUSSION

It is well known that, the islets; the endocrine part of the pancreas is scattered irrigularly throughout the exocrine part and comprise only a small fraction of the total volume of this organ. Significant difference between the histological appearance of the pancreas in specimens taken from normally slaughtered animals and that described in the literature could not be noticed.

It is also reported by SENGAR and SINGTH, 1971, MALIK and PARAKASH, 1972, that the general histology of the pancreas in cattle and buffaloes

is similar and resembles that described for various animals and man, however the islets of langerhans are spherical in outline and relatively small in cattle, whereas in buffaloes they are irrigular in outline and larger.

In the present work, acute pancreatitis was induced in cattle and buffaloe using chloroform injections in the pancreatic parenchyma. The avialable literature lacks any information about acute pancreatitis in these animal spieces, the present morphological results will be discussed with the clincopathological parometers described and published in the second and third parts of this study, (HASSAN et al., 1980),and also with the morphological findings described in other species.

Macroscopic and likewise the microscopic examinations of the liver showed no pathological alterations, except slight cholangitis and hepatocytic dystrophic changes in one animal. This could be assumed to liver fluke infection which suspected to be the cause of both the inflammatory changes in the bile ducts and the dystrophic changes in the hepatic cells.

The level of the liver enzymes (Table 1.) did not significantly differ from the physiological activities. These results correspond with the absence of any morphological changes in the liver after acute pancreatitis.

In regard to the pancreatic alterations, diffuse pancreatic necrosis followed by fibrosis till complete pancreatic sclerosis was observed. From the clinical point of view, the behaviour of the enzyme parameters, (Table 2), transient increased serum amylase level was followed by gradual decreased values till the 15th day post injection: The behaviour of amylase enzyme in such cases could be attributed to the inflammatory processes following chloroform injection which probably interferred with the normal release of pancreatic secretion in pancreatic duct system and absorption by blood. The behaviour of trypsin enzyme could be attributed

to the damage of the pancreatic acinar tissue and its complete sclerosis.

In the alloxan injected animals, proteinous dystrophic changes were observed nearly in all the examined liver specimens. The behaviour of the liver enzymes (Table 3) showed gradual increased S-GOT and S-GPT levels and this usually accompanies the metabolic liver disturbances. The phosphatases are slightly affected. These changes could be assumed to the hepatocytic dystrophic changes described in our findings.

The pancreas of the alloxan injected animals revealed vacuolation of the islet cells followed by their necrosis, lysis and subsequently their fibrous connective tissue replacement. The islet cell vacuolation was described by WEICHSELBAUM and STANGEL, (1902) as hydrobic degeneration, while TORESON, (1951) attributed similar cytoplasmic vacuolations to glycogenic infilterations, but VOLK and LAZARUS,(1956) described both of the forementioned lesions in experimental diabetes of dogs, and the process is further described as hydrobic degeneration by CHORS,(1962). Whatever, it is degenerative lesion, localized selectively in the islet cells and this may explain the interference with the normal process of synthesis, storage and release of insulin and the production of the diabetic state of the animals and the persistent hyperglycaemia described in the third part of this study;(HASSAN et al., 1980).

In our findings alloxan had no effect on the exocrine acinar pancreatic parenchyma and that explains perhaps the slight affection of the pancreatic enzymes, (Amylase, lipase and Trypsin, Table 4), which remains within the physiological levels.

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Table (I)

Changes In Liver Enzymes (GOT, GPT, Alkaline Phosphatase And Acid Phosphatase) Before And After Induced Pancreatitis.

Time of specimen No. of cases	Before Operation 5 cases	After 1 day 5 cases	After 2 days 4 cases	After 4 days 3 cases	After 7 days 2 cases	After 15 days One case
GPT U/Lit.	10.20 (8-13)	12.20 (9 -16)	10.30 (8 -13)	9.70 (8 -12)	11.50 (10_13)	10.00
GOT U/Lit.	37.40 (34-48)	39.80 (36-48)	43.80 (28-57)	31.30 (20-40)	39.30 (37-42)	40.00
Alkaline Phosphatase U/Lit.	6.80 (11-22)	18.60 (11-31)	16.50 (11-33)	22.00 (22-22)	11.00 (11-11)	22.00
Acid Phosphatase U/Lit.	2.60 (2.0-3.0)	3.00 (2.0-3.5)	2.50 (2.0-3.0)	2.50 (1.8- 3)	2.50 (2.5-2.5)	2.00

Table (2)

Changes In Pancreatic Enzyme-Lipase And Amylase-In Serum, And Trypsin In Duodenal Fluid And Faeces Before And After Induced Pancreatitis.

Time of specimen	Before oparation	After 1 day	After 2 days	After 4 days	After 7 days	After 15 days
No. of Cases	5 cases	5 cases	4 cases	3 cases	2 cases	One case
Amylase° U/100 ml.	67.60 (40-90)	149.40 (120-160)	120.00 (100-150)	110.00 (100-120)	108.00 (108-108)	100.00
Trypsin° in duodenal fluid U/Lit.	16.00	5.70	2.50	3.20	5.70	6.90
Trypsin* In faeces.	++++ve	+ve	-ve	+ve	+ve	++ve

° Quantitative determination.

* Qualitative determination.

Table (3)

Changes In Liver Enzymes (GOT, GPT, Alkaline Phosphatase And Acid Phsophatase) Before And After Injection With Diabetogenic Dose Of Alloxan.

Time of specimens	Before Injection	After 1 day	After 2 days	After 4 days	After 7 days	After 15 days
No. of cases	5 cases	5 cases	4 cases	3 cases	2 cases	One case
GPT U/Lit.	8.10 (6.5-10)	8.20 (7-10)	10.20 (10-10.5)	12.00 (10-14)	15.00 (15-15)	25.00
GOT U/Lit.	27.00 (24-37)	30.00 (24-44)	38.80 (28-47)	40.70 (30-48)	41.50 (35-48)	48.00
Alkaline Phosphatase U/Lit.	18.20 (11-22)	28.60 (22-33)	24.80 (22-33)	22.00 (11-33)	18.00 (13-33)	22.00
Acid Phosphatase U/Lit.	3.00 (2.5-4.0)	3.16 (2.8-3.5)	3.08 (2.5-4.0)	2.80 (2.5-3.0)	2.75 (2.5-3.0)	3.00

Table (4)

Chagnes In Pancreatic Enzyme (Lipase And Amylase) In Serum And Trypsin In Duodenal Fluid And Faeces Before And After I.V. Injection With Diabetogenic Dose of Alloxan.

Time of Specimens	Before Injection	After 1 day	After 2 days	After 4 days	After 7 days	After 15 days
No. of cases	5 cases	5 cases	4 cases	3 cases	2 cases	One case
Amylase U/100ml	78.00 (70-80)	72.20 (60-85)	68.20 (60-78)	67.30 (60-74)	69.50 (67-72)	48.00
Lipase ml of 0.05 M NaoH	0.74 (0.-0.9)	0.72 (0.6-0.9)	0.58 (0.5-0.7)	0.57 (0.5-0.6)	9.55 (0.5-0.6)	0.70
Trypsin in Duden-al fluid,U/Lit.	16.40	15.40	14.80	13.40	12.20	16.00
Trypsin In Faeces	++++ve	++++ve	++++ve	++++ve	++++ve	++++ve

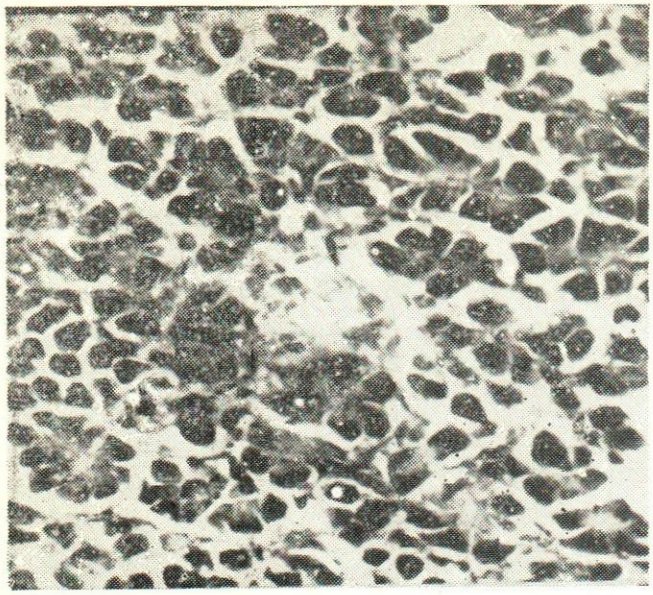

Fig. 1 : Showing diffuse pancreatic necrosis. (H & E. 25 x 12 5).

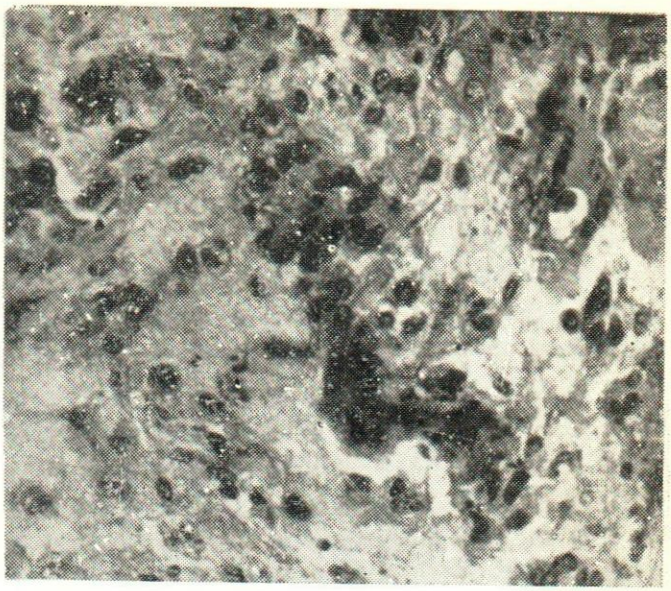

Fig. 2 : Showing incomplete pancreatic fibrosis. (H & E. 40 x 12.5).

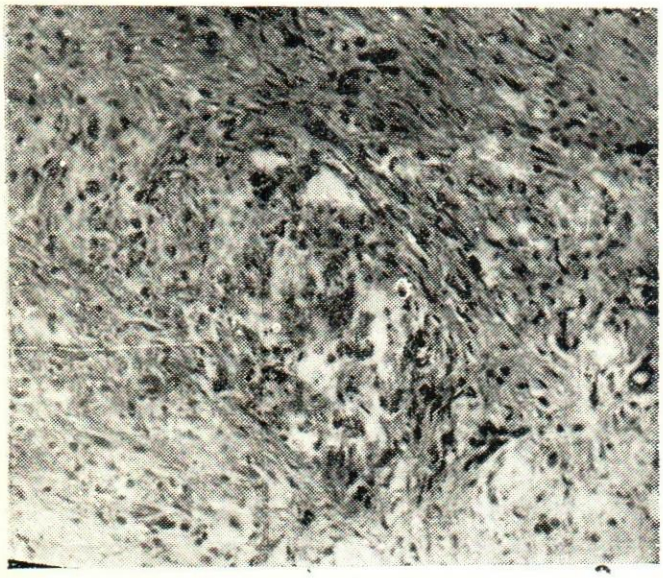

Fig. 3 : Showing complete pancreatic sclerosis. (H & E. 10 x 12.50).

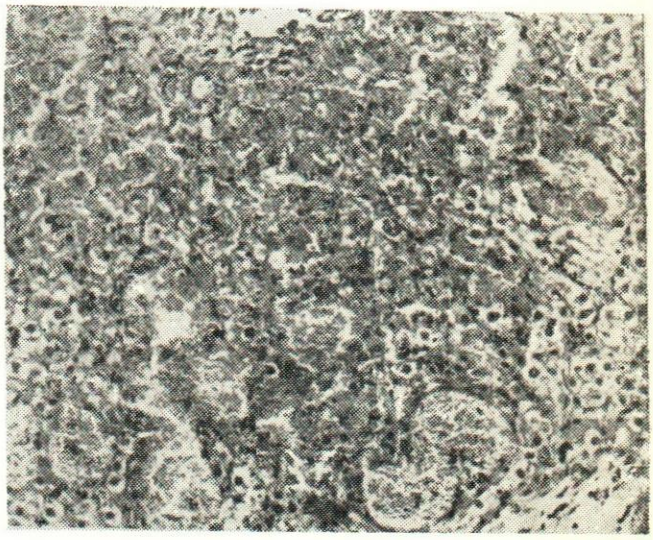

Fig. 4 : Showing liver telangiectitic lesions. H & E. 25 x 12.5).

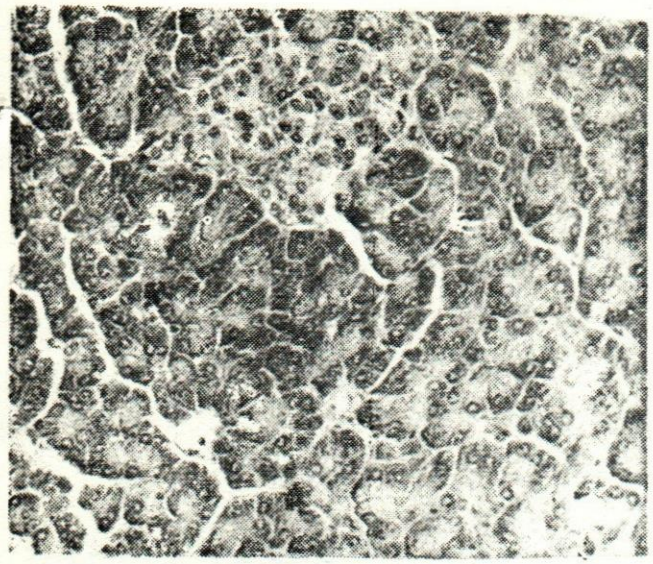

Fig 5 : Showing normal buffalo islet cells. (H & E. 25 x 12.5)

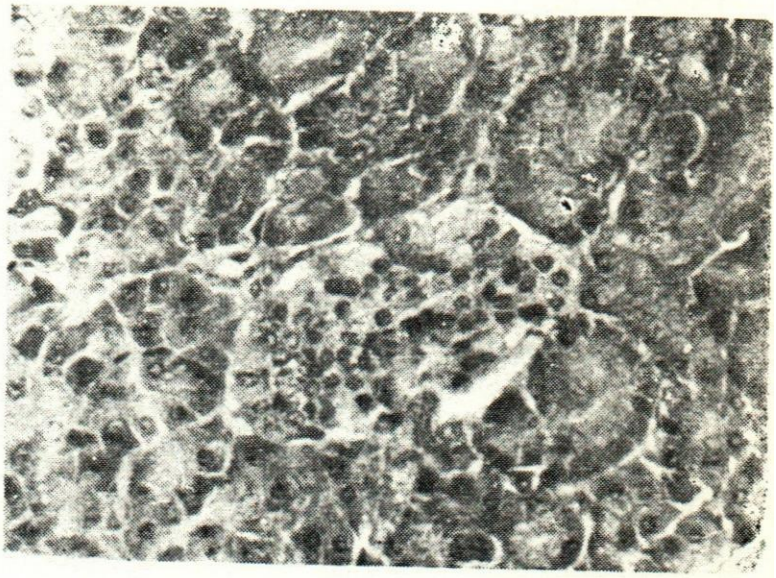

Fig. 6 : Showing islet cell vacuolation and their nuclear pyknosis. (H & E. 25 x 12.5).

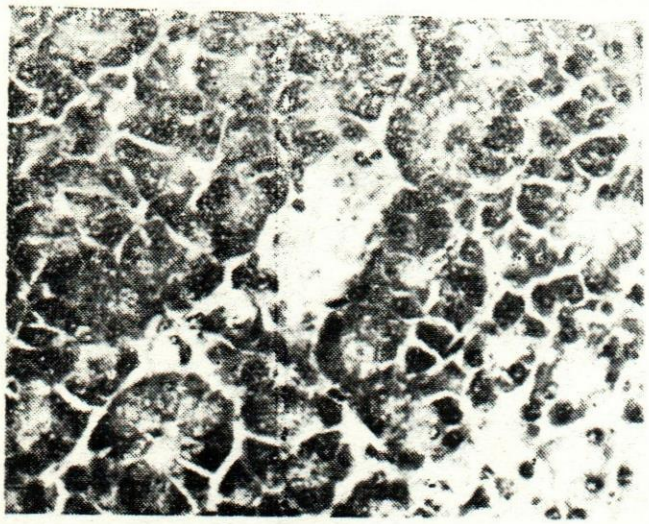

Fig. 7 : Showing islet cell coagulation necrosis. (H & E. 25 x 12.5)

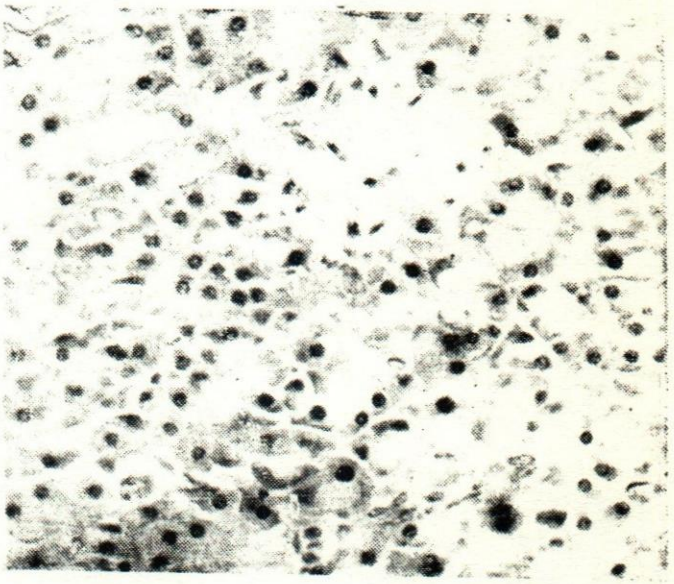

Fig. 8 : Showing fibrosis in islet cell places. (H & E. 25 x 12.5).