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Acute Pancreatitis and Hepatic Necrosis in the Acute Afferent Loop Syndrome

A Histopathological Study in the Rat

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ABSTRACT

The morphological changes in the liver and pancreas during the first 12 hours in the acute afferent loop syndrome were studied in rats with a Billroth II gastric resection. Two essentially different types of changes were found in the pancreas. One was a coagulative necrosis without an inflammatory reaction, which has been found after instillation of bile, bile salts and the detergent sodium lauryl sulphate into the pancreatic ducts. The other type of pancreatitis was an intense, acute purulent inflammation often with bacteria visible in the histological sections. In the liver, large areas of necrosis were often encountered, sometimes mixed with polymorphonuclear leukocytes and bacteria, in a few cases combined with thrombi in small portal veins. The changes in the pancreas occurred very rapidly; only 4 hours after occlusion of the afferent loop there were signs of pancreatitis in some cases and 12 hours after occlusion there was an acute pancreatitis in all cases.

INTRODUCTION

The acute afferent loop syndrome is defined as meaning an occlusion of the afferent loop after a Billroth II gastric resection. The prognosis of this condition in man is poor if the diagnosis is not made in good time and the operation performed. In a series of 105 cases reported in the literature, 36 were untreated, and all died (2). Sixty-nine were operated upon, and 18 of these died. Studies on the acute afferent loop syndrome (ALS) in dogs showed that pancreatitis was regularly found on histological examination of the pancreas after death (3). In man too acute pancreatitis has been reported to occur in this condition (2, 4, 6). Perforation of the afferent loop is known to occur within 24 hours.

No investigation has been performed, however, on the time sequence in the development of the morphological changes in the liver and pancreas after occlusion of the afferent loop in rats with a Billroth II gastric resection. This problem is studied in this report.

MATERIAL AND METHODS

88 male albino rats of the same strain (Sprague-Dawley) were used. They comprised part of the material of a study presented by Bengtsson et al. (1). All rats underwent gastrojejunostomy and division of the pylorus. About 2 months later the afferent loop was closed by a ligature near to the gastrojejunostomy. After various intervals of time the animals were killed. The whole pancreas was then excised, together with the afferent loop. At the same time part of one lobe of the liver was excised. Four groups of animals were treated in this way ½, 4, 8 and 12 hours, respectively, after the occlusion. Two groups of sham-operations were also performed. These involved exactly the same procedure, i.e. with gastroenterostomy in a first session and laparotomy in a second session, with dissection of the afferent loop and application of a ligature around the loop without it being tied. The animals in the control groups were killed ½ hour and 4 hours, respectively, after the sham procedures. The methods have been described in detail by Bengtsson et al. (1).

Histological methods

Immediately after excision the specimens were fixed in 10% formaldehyde. The paraffin-embedded tissue blocks were cut at several levels to ensure a thorough examination of the gland. The sections were stained with hematoxylin-eosin.

RESULTS

Most of the control rats as well as those killed half an hour after ligation of the afferent loop showed normal pancreatic and liver tissue. In some cases there was a moderate acute interstitial inflammation with polymorphonuclear leukocytes in the pancreas (see also Table I).

With increasing time after ligation of the loop, the acute interstitial inflammation of the pancreas became more severe. Polymorphonuclears appeared in pancreatic ducts (Fig. 1), often in the neighbourhood of the common hepatic duct (Fig. 2). Bacteria were found in ducts and in the interstitial tissue.

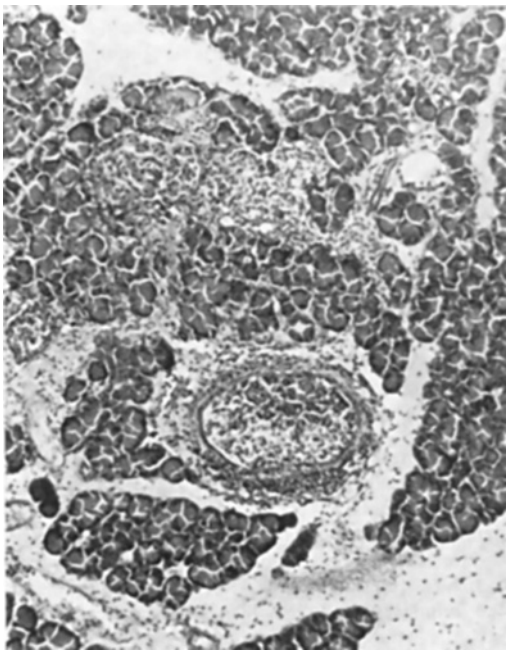

Fig. 1. Pancreas. Acute interstitial inflammation, polymorphonuclears in pancreatic ducts and a small abscess. ALS: 12 hours' occlusion. Hematoxylin-eosin, ×95.

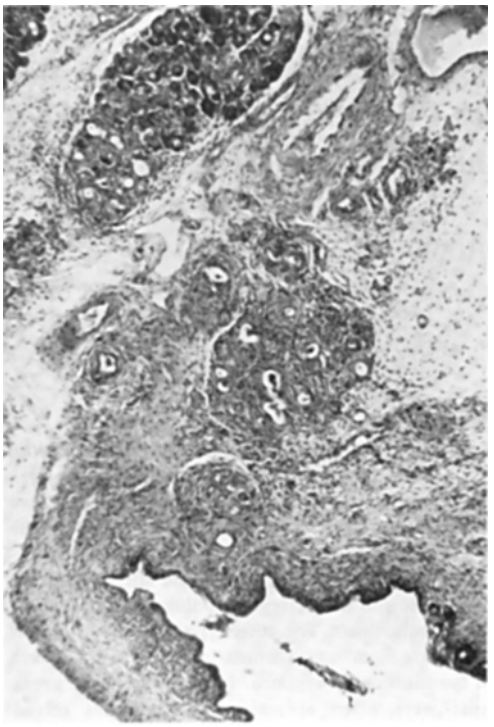

Fig. 2. Pancreas. Acute interstitial inflammation with polymorphonuclears appearing near the common hepatic duct and in small pancreatic ducts. ALS: 4 hours' occlusion. Hematoxylin-eosin, ×70.

The intense acute, purulent inflammation was also observed within the acinar tissue, with more or less total disappearance of acinar cells so that microabscesses often arose. In a few animals a quite different picture was observed in addition, namely small areas of coagulative necrosis without inflammatory cells (Fig. 3). Haemorrhages were never pronounced. Small bleedings were observed in half of the rats killed 12 hours after ligation of the loop.

In the livers, polymorphonuclears were a common finding in the portal tract (Table I). In a few rats large areas of necrosis, often in the form of abscesses, were found. They sometimes contained bacteria (Fig. 4). Thrombi in small portal veins within the liver were found in a few cases.

Table I. *The morphological changes in the pancreas and liver in the control groups and in the groups with acute afferent loop syndrome (ALS)*

	Pancreas					Liver				
	Interst. infl.	Microabscess	Coag. necr. without infl.	Polymorph. in ducts	Bacteria	Abscess	Polymorph in portal tracts	Thrombi	Bacteria	No. of rats
Controls, ½ hr	11	2				1	3			16
Controls, 4 hrs	3									4
ALS, ½ hr	11					1	7			16
ALS, 4 hrs	18	3	5	2	1	6	8			24
ALS, 8 hrs	7	4	3	5	3	6	7	4	4	10
ALS, 12 hrs	17	15		15	16	12	16	3	1	18

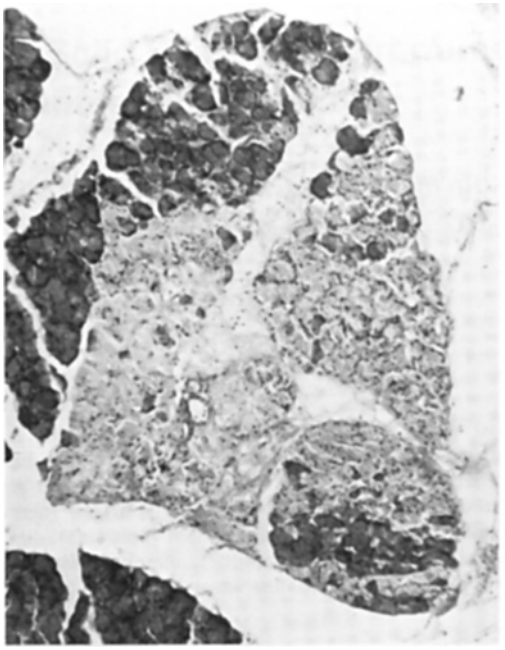

Fig. 3. Pancreas. An area of coagulative necrosis without inflammatory cells. ALS: 4 hours' occlusion. Hematoxylin-eosin, ×90.

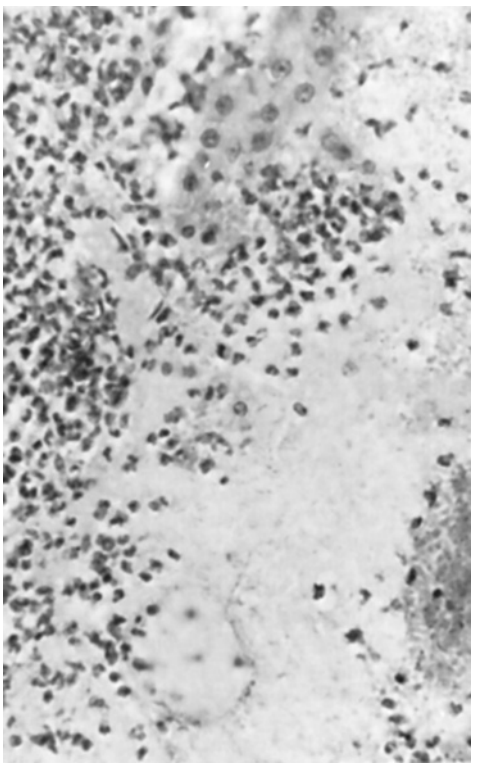

Fig. 4. Liver. Abscess containing bacteria. ALS: 8 hours' occlusion. Hematoxylin-eosin, ×300.

DISCUSSION

The morphological changes in the pancreas appeared to be of two essentially different types. One was a coagulative necrosis without an inflammatory reaction. This type has been found in pancreatitis induced by instillation of bile, bile salts and also the detergent sodium lauryl sulphate into the pancreatic ducts (5, 7), and is considered to be due to the toxic effects of the substances mentioned on the acinar cells. In our experiment bile may have entered the pancreatic ducts owing to the increased pressure in the closed afferent loop. Development of a typical acute haemorrhagic pancreatitis following this type of necrosis is well known.

The other type of pancreatitis was characterized by an acute inflammation with a total predominance of polymorphonuclear leukocytes. The inflammation was mainly interstitial, but polymorphonuclears and also bacteria in the ducts were a common finding. Slight acute interstitial inflammation was also found in the control series, but no bacteria were found. In the groups with a closed afferent loop bacteria were encountered also in the interstitial tissue in a few cases. The liver lesions also often contained bacteria (Table I). It seems probable, therefore, that the hepatic necrosis and the pancreatitis were often due to infection, probably in some cases ascending from the duodenum via the ducts, perhaps sometimes via the blood and lymphatic vessels, or as regards the pancreas, possibly also interstitially *per continuitatem*. Infection is no usual cause of acute pancreatitis, though it may be a secondary complication of acute haemorrhagic necrotizing pancreatitis. The present early finding of polymorphonuclears and bacteria in ducts in the absence of coagulative necrosis of acinar cells seems, however, to be an argument for a bacterial etiology. The contents of the afferent loop were examined as regards bacterial flora (1), and it was found that this occluded loop was highly suitable for bacterial growth—this was specially valid for anaerobic bacterial culture.

As has been shown by Dahlgren (2), the pressure in the closed afferent loop can rise up to 100 mmHg. It seems probable, therefore, that part of the contents of the closed loop can ascend into the liver and pancreas as a consequence of this high pressure. It is thus easy to understand that both bile and

bacteria can enter the pancreas and give rise to both types of morphological changes in the pancreas and the changes in the liver described here. In this study we have shown that the changes in the pancreas develop very rapidly—after only 4 hours of occlusion there were signs of acute pancreatitis both in the form of microabscesses and interstitial inflammation in some cases, and after 12 hours there was an extensive pancreatitis in all cases. The observations further substantiate the view that the acute afferent loop syndrome is a life-threatening condition which must be diagnosed and treated very early in order to avoid severe changes in the liver and pancreas.

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