Pressure measurement in pancreatic duct and biliary duct system in dogs with acute pancreatitis.

Fujiwara, H

(Citation)
The Kobe journal of the medical sciences,37(2):47-55

(Issue Date)
1991

(Resource Type)
departmental bulletin paper

(Version)
Version of Record

(URL)
https://hdl.handle.net/20.500.14094/E0034084

Kobe J. Med. Sci. 37, 47/55, April 1991

PRESSURE MEASUREMENT IN PANCREATIC DUCT AND BILIARY DUCT SYSTEM IN DOGS WITH ACUTE PANCREATITIS.

HIDETOSHI FUJIWARA

First Division, Department of Surgery,
Kobe University School of Medicine

INDEXING WORDS

pancreatic ductal pressure; acute pancreatitis; sphincter of Oddi

SYNOPSIS

We investigated the intraductal pressures of the pancreatico-biliary system in dogs with acute pancreatitis. Intraductal pressures were measured by the method of perfusion manometry. After the perfusion with saline (the common bile duct (CBD: 0.8ml/min, the pancreatic duct (PD: 0.2ml/min)), two parameters, the residual pressure (RP) and the pressure decay time (DT), were measured before and after the induction of acute pancreatitis.

The RP of the PD was elevated after inducing severe pancreatitis. There were statistically significant difference of the RP of the PD between severe and moderate pancreatitis. In addition the DT of the PD was also prolonged in severe pancreatitis. There were significant difference of the DT of the PD between moderate and severe pancreatitis. On the other hand the RP of the CBD was also elevated in severe pancreatitis. There were statistically significant difference of the RP of the CBD between severe and moderate pancreatitis. However there was no significant difference between them concernig the DT of the CBD.

Received for publication : February 21, 1991
Author's name in Japanese 藤原英利

The histological examination of periampullary area in severe pancreatitis showed measurable inflammatory changes (papillitis). However there was no such finding in moderate pancreatitis. Our results suggest that pressure measurement in the PD is useful for predicting the severity of acute pancreatitis because the pressure of both the PD and the CBD were elevated in severe pancreatitis but not in moderate pancreatitis and this difference is mainly due to the difference of the influence of pancreatitis to the periampullary region.

INTRODUCTION

In acute pancreatitis, especially hemorrhagic and necrotizing pancreatitis the mortality is very high in spite of vigorous treatments.[1,2,4,6] At present the cause of acute pancreatitis is not known yet, but the result is thought to be autodigestion of the pancreas per se by its own enzymes.[7]

On the other hand, it is well known that the pressure of the bile duct increases in acute pancreatitis and drainage of the bile duct is performed as one of the treatments.[7] One of the causes of increase in the bile duct pressure is thought to be the influence of pancreatitis on the periampullary area.[9] In this concept it is considered that as the pressure of the pancreatic duct might be increased in acute pancreatitis due to the influence of inflammation on periampullary area and this increasing might augment the autodigestion of the pancreas due to reflux of pancreatic juice to acinar cells.

In this study we observed the changes of the pressures in the pancreatic duct and the biliary system in severe (hemorrhagic and necrotizing) pancreatitis and moderate (edematous) pancreatitis in dogs.

MATERIALS AND METHODS

Operative procedures:

Adult mongrel dogs of either sex, weighing 10kg to 15kg, were used for the studies. Food was withheld from dogs for 16 hours preoperatively but water was given freely and dogs were anesthetized with sodium pentobarbiturate (25mg/kg) for

induction and maintenance.

A midline abdominal incision was made in all dogs. The cystic duct and the end of the dorsal pancreatic duct were carefully isolated. A plastic catheter (atom tube 6fr) was introduced through the cystic duct into the common bile duct. Another plastic tube (atom tube 3fr) was inserted into the dorsal pancreatic duct(Fig. 1).

Pressure measurement of pancreatic duct and biliary tract:

Both catheters were attached to external transducers (Nihon Koden TP400T, Japan) and connected to a multichannel direct polygraph (Nihon Koden W625G, Japan). After measurement of the basal pressure, saline was perfused into the common bile duct at a constant flow rate of 0.8ml/min and then at a constant flow rate of 0.2ml/min into the pancreatic duct. In order to determine the characteristics of these systems, two parameters were measured: the pressure decay time (DT) and the residual pressure (RP). These parameters were measured in normal pancreas and at 12 hours,1, 2, 3, 4, 5 and 7 days after induction of acute pancreatitis.

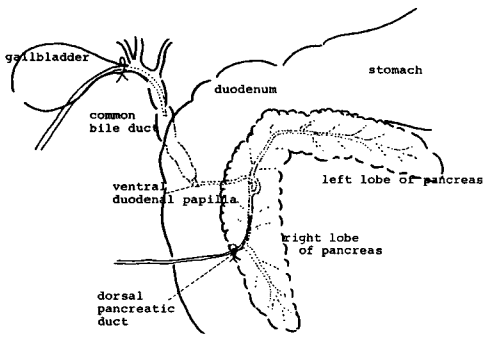

Fig. 1
Anatomy of the pancreatic duct and the common bile duct in dogs. A plastic tube (Atom tube 6 Fr) was introduced through the cystic duct into the common bile duct and another plastic tube (Atom tube 3 Fr) was inserted into the dorsal pancreatic duct for pressure measeurements in the common bile duct and the pancreatic duct respectively.

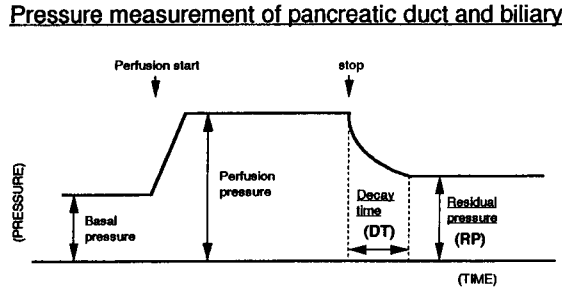

Fig. 2
Analysis of the manometric tracing.

The DT is defined as the time required to get a constant pressure after stopping the perfusion and the RP is defined as the constant pressure after stopping the perfusion (Fig. 2). These two parameters correlate with the resistance in the terminal of the ducts.

Production of acute pancreatitis:

Acute pancreatitis was produced by a retrograde injection of deoxycholic acid (DCA) or trypsin through the indwelling catheter in the pancreatic duct. Severe hemorrhagic and necrotizing pancreatitis was produced by 20% DCA solution (0.2ml/kg) (Fig. 3) and moderate edematous pancreatitis was produced by 2,000 U/ml trypsin (Mochida, Japan) solution (2ml/kg)(Fig. 4).

Histological examination:

For histological examination of pancreas, dogs were sacrificed on the first, the fifth and the seventh days after producing pancreatitis. The tissue was fixed in Zamboni's solution, paraffin embedded and staind with hematoxylin and eosin.

Statistics:

All values were expressed as the mean ± one standard deviation. Statistical analysis were performed with Student's t-test. A probabillty value that was less than 0.05 was considered statistically significant.

RESULTS

Alteration of the pancreatic duct pressure:

In normal pancreas the RP was 39.5 ±4.7 cmH_2O. After severe pancreatitis was induced, the RP markedly increased reaching a peak 98.6±67.0 cmH_2O on the first day and then gradually decreased to a level of 53.0±32.5 cmH_2O on the 7th day. However, it remained in the significant high level even on the 7th day. In contrast the RP slightly increased but almost remained in the normal range in moderate pancreatitis. There was a significant difference between severe and moderate pancreatitis on the second and the 3rd day.($p<0.05$) (Fig. 5).

The pancreatic DT was 15.7±9.5 sec in control. In severe pancreatitis the DT was immediately delayed to 56.7±30.5 sec on the first day and remained in the significant high level till the 3rd day and it returned to the normal range on the 5th day. In moderate pancreatitis there was no change in the DT. There was a significant difference between severe and moderate pancreatitis on the second ($p<0.01$) and the 3rd day ($p<0.05$) (Fig. 6).

Alteration of the common bile duct pressure:

The RP of the CBD was 9.3 ± 1.9 cmH_2O in control. After severe pancreatitis was induced the RP increased with pressure reaching a peak 20.1 ± 10.4 cmH_2O on the first day. And it remained in the significant high level for 3 days. On the other hand the RP in moderate pancreatitis slightly increased but was not significant compared to the control. Significant differences between severe and moderate pancreatitis were observed on the first ($p<0.05$), the second ($p<0.05$) and the 3rd day. ($p<0.01$) (Fig. 7).

The DT of the CBD was 8.3 ± 4.7 sec in controls. In severe pancreatitis DT was prolonged to 13.5 ±3.4 sec on the first day. After the 3rd day the DT began to return to the normal level. In moderate pancreatitis the DT remained within normal limits. However no significant difference was observed between severe and moderate pancreatitis throughout the experiments (Fig. 8).

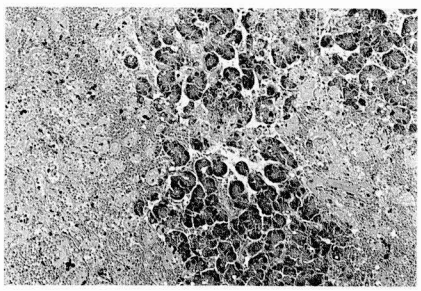

Fig. 3
Canine pancreas twenty-four hours after the infusion of 20% DCA(severe pancreatitis). The photomicrograph shows widely spreaded bleeding and necrosis of the pancreas (H & E : X 10).

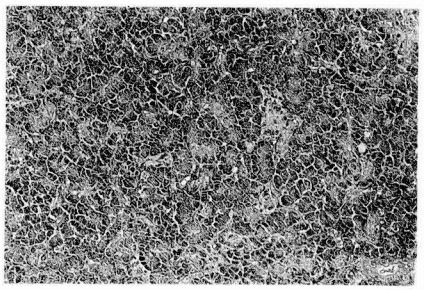

Fig. 4
Canine pancreas twenty-four hours after the infusion of 2000U/ml trypsin solution(moderate pancreatitis). The photomicrograph shows edema and bleeding of the interlobules of the pancreas (H & E : X 10).

The histological examination of peri-ampullary area of the canine pancreas 3 days after the infusion of 20% DCA was performed. There are marked edema of the muscular layer of the duodenum, destruction of the acinar cells and observable inflammation in the periampullary region (papillitis) due to severe pancreatitis (Fig. 9A and B). But no such inflammation occurred in moderate pancreatitis.

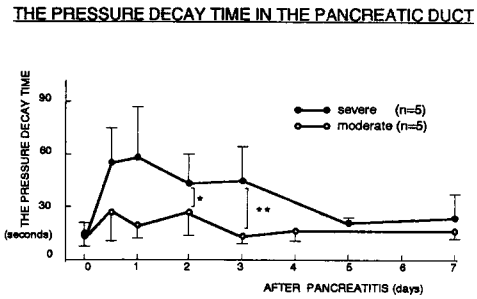

Fig. 5
The residual pressure in the pancreatic duct in severe (DCA induced) pancreatitis and moderate (trypsin induced) pancreatitis. (mean ± SD). " * " indicates significantly higher pressure levels in severe pancreatitis compared with moderate pancreatitis. * $p<0.05$.

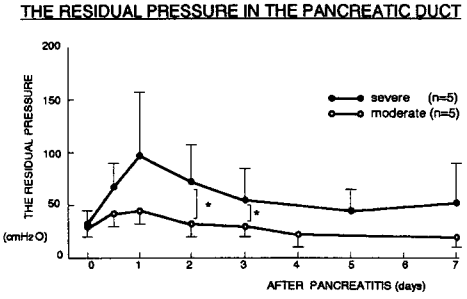

Fig. 6
The pressure decay time in the pancreatic duct in severe (DCA induced) pancreatitis and moderate (trypsin induced) pancreatitis. (mean ± SD). " * " indicates significantly longer time levels in severe pancreatitis compared to moderate pancreatitis. * $p<0.05$, * * $p<0.01$

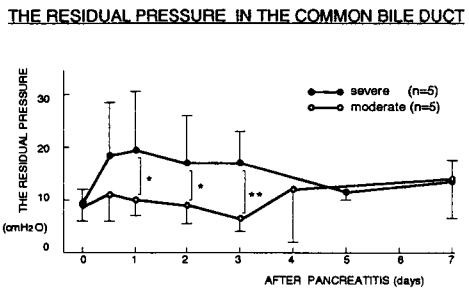

Fig. 7
The residual pressure in the common bile duct in severe (DCA induced) pancreatitis and moderate (trypsin induced) pancreatitis. (mean ± SD). " * " indicates significantly higher pressure levels in severe pancreatitis compared with moderate pancreatitis. * $p<0.05$, * * $p<0.01$

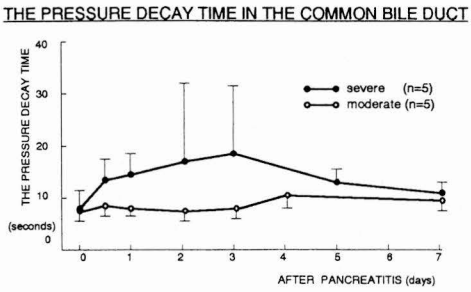

Fig. 8
The pressure decay time in the common bile duct in severe (DCA induced) pancreatitis and moderate (Trypsin induced) pancreatitis. (mean ± SD).

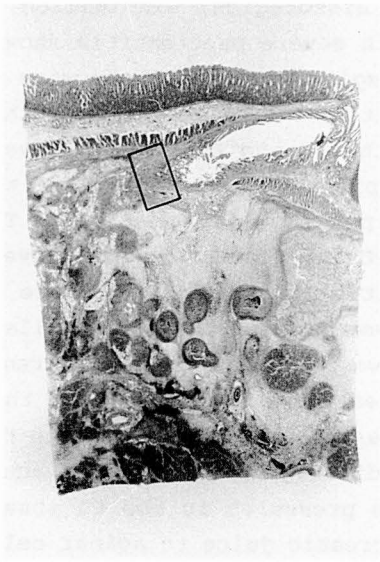

Fig. 9A
Periampullary area of the canine pancreas 3 days after the infusion of 20% DCA. There are marked edema of the muscular layer of the duodenum and destruction of the acinar cells. (H &E : X 4).

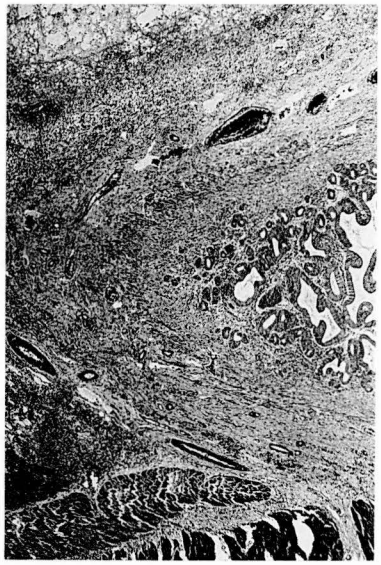

Fig. 9B
Photomicrograph is an enlargement of the area outlined in figure 9A. It shows marked edema of the muscular layer and infiltration of inflammation cells into the interstitum(H & E : X 20).

DISCUSSION

In this report we observed the influence of periampullary inflammation caused by acute pancreatitis on the intraductal pressures in the pancreatico-biliary system in dogs. We used two parameters: the residual pressure (RP) and the pressure decay time (DT). These two parameters are thought to reflex the resistence in the end of the duct and the function of the papilla of Vater.[5] We observed measurable elevation of the pressures in the PD and the CBD (increasing of the RP and prolongation of the DT) in severe pancreatitis. However, there were no observable changes in the case of moderate pancreatitis. Stolte et al[8] reported the pathological changes in the papilla of Vater in rat with acute pancreatitis and that this changes were the cause and the consequence of pancreatitis. Histological examination of the periampullary region in dogs with severe pancreatitis showed marked edema of the periampullary region. However there were no such findings in moderate pancreatitis. It seemes likely that the elevation of the pressures in the PD and the CBD in severe pancreatitis is mainly due to papillary dysfunction by the influence of inflammation on the periampullary region. The difference of the pressures in the PD and the CBD between severe pancreatitis and moderate pancreatitis is thought to be due to the difference of influence of inflammation on the periampullary area. But the reason why DT of CBD was no significant difference between moderate and severe pancreatitis is unknown at this present and under investigation. The pressure measurement in the pancreatic duct is worthwhile in predicting the severity of acute pancreatitis. The elevation of the pressures in the PD itself makes a chance to reflux of the pancreatic juice to acinar cells and accelerates the autodigestion of the pancreas. Placements of stents and drainage in the PD in acute pancreatitis by using endoscopic technique has been already reported.[3] Our preliminary data showed that drainage of the pancreatic duct after inducing severe pancreatitis made an improvement of pancreatitis judging from the histological findings of pancreas. It is possible that drainage in the PD will be a useful treatment for acute pancreatitis. The effectiveness of the pancreatic duct drainage on the treatment of acute severe pancreatitis is under

investigation.

ACKNOWLEGEMENT

The author is indebted to Professer Saitoh Y., Dr.Kuroda Y., Dr.Suzuki Y., Dr.Fujino Y. and Dr. Kawamura T., First Division, Department of Sugery, for support and encourgement in this work.

REFERENCE

1. Aldridge M.C., Ornstein M., Glazer G.Dudley H.A.F.; Br J. Surg. 1985, 72:796/800. Pancreatic resection for severe acute pancreatitis.
2. Imrie C.W., Benjamin I.S.,Ferguson J.C.,Mckay A.J.,Mackenzie I.,O'neill J.,Blumgart L.H.; Br. J. Surg. 1978, 65:337/341. A single-centre double-blind trial of Trasylol therapy in primary acute pancreatitis.
3. Kozarek R. A., Patterson D.J., Ball T. J., Traverso L.W.; Ann. Surg.1989, 209; 261/266. Endoscopic placement of pancreatic stents and drain in the management of pancreatitis.
4. Lawson D.W., Daggett W. M., Civetta J. M.,Corry R.J.,Bartlett M.K.; Ann. Surg. 1970, 172:605/626. Surgical treatment of acute necrotizing pancreatitis.
5. Matsushiro T., Nagashima H., Yamamoto K., Hariu T.; SURGERY 1984, 95:96/101. Perfusion cholangiomanometory with papillary stimulation by dilute hydrochoric acid.
6. Ranson J.H.C., Spencer F.C.; Ann. Surg. 1978, 187:565/575 the role of peritoneal lavage in severe acute pancreatitis.
7. Rinderknecht H.; Dig Dis Sci 1986, 31:314/320. Activation of pancreatic zymogens. Normal activation, premature intrapancreatic activation, protective mechanisms against inappropriate activation.
8. Stolte M., Waltschew A. ; Hepato-gastroenterol 1986, 33: 163/169. The papilla of Vater and Chronic pancreatitis.
9. Tooli J., Roberts Thomson I. C., Dent: J.; Br. J. Surg. 1985, 72:859/863. Sphincter of Oddi motility disorder in patients with idiopatic recurrent pancreatitis.