

Can ultrasound predict the severity of acute pancreatitis early by observing acute fluid collection?

Yan Luo¹, Chao Xin Yuan¹, Yu Lan Peng¹, Pei Lin Wei¹, Zhao Da Zhang², Jun Ming Jiang³, Lin Dai¹ and Yun Kai Hu¹

Subject headings pancreatitis/classification; acute fluid collection; pancreatitis/ultrasonography; C reactive protein; prognosis

Luo Y, Yuan CX, Peng YL, Wei PL, Zhang ZD, Jiang JM, Dai L, Hu YK. Can ultrasound predict the severity of acute pancreatitis early by observing acute fluid collection. *World J Gastroenterol*, 2001;7(2):293-295

INTRODUCTION

The spectrum of acute pancreatitis (AP) ranges from a mild spontaneously resolved disorder to severe disease with a mortality up to 20%-48.4%^[1-3]. sAP is defined as the AP with organ failure and/or local complications which developed from acute fluid collection (AFC) including necrosis, abscess, pseudocyst formation into or around the pancreas^[4]. sAP is only about 15%-25% but almost all of mortality and morbidity of AP is concentrated in it^[5]. Early diagnosis and assessment of severity in AP are still far from ideal. The role of clinical assessment is reliable but for its subjectivity the value in the early detection of severity is limited^[1,6]. C-reactive protein (CRP) is a promising laboratory marker of severity, even though it is not specific for AP^[1]. Single or multiple biochemical criteria and different scoring systems have been used as prognostic indicators, but none has been proven satisfactory in clinical practice^[1,6].

The aim of this study is to investigate the prognostic significance of evaluating the severity of AP by observing AFC using ultrasonography.

¹Ultrasound Department of First Affiliated Hospital of West China University of Medical Science, Chengdu 610041, Sichuan Province, China

²Surgery Department of First Affiliated Hospital of West China University of Medical Science, Chengdu 610041, Sichuan Province, China

³Traditional Chinese Medicine, Department of First Affiliated Hospital of West China University of Medical Science, Chengdu 610041, Sichuan Province, China

Dr. Yan Luo, graduated from West China University of Medical Sciences as a postgraduate in 1990, now chief and lecturer of ultrasound department, specialized in ultrasound diagnosis in abdominal and vascular diseases, having 10 papers published.

Supported by Clinical Key Subject Fund of the Health Administration, No.97050234.

Correspondence to: Dr Yan Luo, Ultrasound Department of First Affiliated Hospital of West China University of Medical Science, 37# Guxue Street, Chengdu 610041, China

Tel.0086-28-5422304, Fax. 0086-28-5422987

Email. luoyan@mail.sc.cninfo.net, luoluohai@sina.com.cn

Received 2000-07-12 Accepted 2000-08-26

SUBJECTS AND METHODS

General information

A total of 627 patients who underwent ultrasound examination for AP between 1996-01/1998-12 in the 1st Affiliated Hospital of West China University of Medical Sciences were analyzed retrospectively. Among them 293 were male and 334 female, aged from 4 to 82 years, with an average of 43.02 years; 502 had mild acute pancreatitis (mAP), and 125 severe acute pancreatitis (sAP). All examinations were performed by attending doctors in the ultrasound department using AI Performa or Dasonics 2D Gateway ultrasound machines with 3.5-5MHz probes.

The primary or first ultrasound examination

Usually on the d1 or d 2 after patient admission to our hospital, routine upper abdominal scanning was performed, with particular observation of the size and the echogenicity of pancreas, the pancreatic and bile ducts. Additional attention was made to peripancreatic AFC, including the lesser sac, anterior pararenal spaces, posterior pararenal spaces, peritoneal cavity, and even thoracic cavity.

Follow-up ultrasound examinations

Continued observation of the pancreas with further attention of those spaces mentioned above, as well as the presence or absence of AFC, the quality and the nature of the fluid (i.e., necrosis, abscess or pseudocyst). If necessary, an ultrasound-guided needle aspiration was performed in order to determine the nature of the fluid and to relieve any pressure build-up. Typically repeated scans were performed 3-7 days following the initial scan. However, this interval was dependent upon the severity of AP. For analysis, the patients were classified into four groups according to the fluid collection: no AFC, 1 site AFC, 2 sites AFC, more than 2 sites AFC.

Treatment

All patients were treated by the methods, including traditional Chinese therapy, aggressive intravenous fluid resuscitation, intensive monitoring of vital functions, supportive therapy. Operative intervention was used only when local complication occurred despite maximal conservative therapy.

Clinical parameter

Age, sex, etiology, hospital stay, local

complication, number of operation, mortality. sAP and mAP were diagnosed according to the clinical features, symptoms, physical examination, laboratory tests and image findings.

Data collection

The size of the pancreas, the number of spaces where there were AFC, as well as any regression or resolution in AFC were recorded. If fluid had resolved, the amount of time necessary for complete resolution and any complications associated with AP as well as the amount of time necessary to develop such complications were monitored. Meanwhile, the response to therapy (either medical or surgical), mortality, and length of hospital stay were noted.

Statistical analysis

Continuous variables were analyzed by *t* test. Discrete variables were analyzed by the χ^2 test and rank sum test using SPSS. $P < 0.05$ was considered significant.

RESULTS

The number and distribution of AFC and the clinical outcome are summarized in Table 1.

Table 1 AFC and clinical outcome

	0 site	1 site	2 site	>2 sites	Total
Mild AP	256(51)	91(18.1)	75(14.1)	80(15.9)	502(100) ^a P
Severe AP	10 (8)	13(10.4)	18(14.4)	84(67.2)	125(100) ^a P
<i>n</i>	263	101	91	144	599
Mean hospital stay	12.2±6.14	15.8±9.89	19.4±10.13	28.4±19.68	17.8 ±14.37
	^b P: ^b P ^b P	^b P: ^b P ^b P	^c P: ^c P ^c P	^d P: ^d P ^d P	^b P
Local complication	0	22	16	40	78
Operation	0	2	5	33	40
Death	2	2	1	15	20

n: the number of mean hospital stay, exclude dead patients and the patients who discharged themselves without complete cure. Value in parentheses are percentage.

^a $P < 0.00001$, $\chi^2 = 147.78$, DF = 3.

To analyze the mean hospital stay using Kruskal-wallis test, ^b $P < 0.00001$, $\chi^2 = 161.47$, DF = 3; furthermore using Wilcoxon test (1 test) to analyze the mean hospital stay between every two group: ^b $P < 0.00001$, $1 = 4.09$; ^c $P < 0.00001$, $1 = 6.69$; ^d $P < 0.00001$; ^e $P = 0.0206$, $1 = 2.32$; ^f $P < 0.00001$, $1 = 8.02$; ^g $P < 0.00001$, $1 = 5.07$

In the local complication, all 22 local complications in one site were necrosis or pseudocyst formation in sac with or without infection; 56 local complication in two and more than two sites were sac necrosis formation with another space necrosis.

The operation meant all kind drainage for local complication during AP, did not include cholecystectomy after AP.

In dead cases, 4 cases with 0 and 1 site AFC died within 48 h of admission for multisystem organ failure with an average of 28 h; 16 cases with two and more than two sites AFC died within 2-35 days with an average of 18.6 d. Three patients died in

wk 1, 4 died in wk 2, and 9 died in wk 3.

The prognostic significance of the severity of AP using AFC number are shown in Tables 2 and 3.

Table 2 To prognosticate the severity of AP using 2 and more than 2 sites AFC

	Clinical sAP	Diagnosis mAP	Total
US			
2 & >2 sites	102	155	257
< 2 sites	23	347	370
Total	125	502	627

Sensitivity 81.6%, speciality 69.2%, accuracy 70.2%.

Table 3 To prognosticate the severity of AP using more than 2 sites AFC

	Clinical sAP	Diagnosis mAP	Total
US			
>2 sites	84	80	164
2 & < 2 sites	41	422	463
Total	125	502	627

Sensitivity 67.2%, speciality 84.1%, accuracy 80.7%.

DISCUSSION

Acute pancreatitis is an acute inflammatory process of pancreas, with variable involvement of other regional tissue or remote organ system, some inflammatory cytokine related to it and any high pressure of the pancreatic duct can lead to accumulation of pancreatic juice into pancreas or spill into the peripancreatic area^[7-20]. As pancreas is situated in the anterior portion of the retroperitoneum and behind the lesser sac, the fluid can accumulate in the lesser sac and the vast retroperitoneal space (especially in anterior pararenal space). The retroperitoneal space is bounded by the posterior parietal peritoneum in front and the transverse fascia behind, extending from the diaphragm to the upper brim of the pelvis. As soon as the pancreatic fluid and exudate enter the retroperitoneal space, they spread freely under tension, extending downward through the lateral part of the retroperitoneal space toward the iliac fossa and upward to the undersurface of the diaphragm and mesenteries involved^[21-24].

AFC is quite common in acute pancreatitis^[25], while more than 50% can resolve spontaneously, only part of them will develop into local complications, which include necrosis, abscess and pseudocyst formation into or around the pancreas^[7]. Once local complications ensue, the onset of severe acute pancreatitis is established^[4].

sAP is only about 15%-25% but almost all of mortality and morbidity of AP is concentrated in it^[26-35]. The principle benefit of early correct assessment of severity in AP is that patients who require intensive monitoring and therapy are detected at once. The role of clinical assessment is reliable but for its subjectivity the value in the early detection of severity is limited^[1,6]. C-reactive

protein (CRP) is a promising laboratory marker of severity, even though it is not specific for AP^[1]. Multiple-factor biochemical criteria and different scoring system, such as Ranson's early prognostic signs and Acute Physiology and Chronic Health Evaluation (APACHE) have been used as prognostic indicators, but none has been proven ideal in clinical practice^[1,6].

Contrast-enhanced CT is proven to be able to predict the severity of AP by scoring the degree of pancreatic tissue necrosis^[36-39], yet the main and widely accepted indication for dynamic CT in AP is clinical suspicion of life threatening local complication and to plan invasive intervention 2-3 weeks after admission for its costliness and X-ray exposure. Therefore, the candidate with AP for contrast-enhanced CT was about 14%^[1], and 11.5% patients receiving contrast-enhanced CT in our study was considered to be adequate. Preliminary experiences with magnetic resonance imaging have not identified advantages compared with contrast-enhanced CT^[40].

Our data showed that the distribution of AFC is different in mAP and sAP, 96.4% (256/266) patients without AFC had mAP while 51.2% (84/164) patients in more than 2 sites AFC group had sAP. The number of AFC was related to the severity of AP, the hospital stay of patients, the emergence of local complication, and the operation possibility and mortality which is similar to other trials^[37]. In other words, 69.1% patients in mAP had no AFC or 1 site AFC while 67.2% patients in sAP had more than 2 sites AFC, and 2 sites AFC in both mAP & sAP were 14.1%, this seemed to imply that 2 sites AFC is the watershed for mAP and sAP in ultrasonography, when there are 2 sites AFC in AP patients, further examinations should be made and more close attention should be paid. The sensitivity, specificity and accuracy were 81.6%, 69.2% and 70.2% respectively if using 2 and more than 2 sites AFC to distinguish mAP and sAP. The sensitivity, specificity and accuracy were 67.2%, 84.2% and 80.1%, respectively if using more than 2 sites AFC.

In conclusion, the number of AFC is related to the severity of AP, the hospital stay of patients, the emergence of local complication, the operation possibility and mortality. Our preliminary experiences showed that ultrasound can be used as an early and elementary prognostic indicator for severity of AP by observing AFC.

REFERENCES

- 1 Kempainen E, Sainio V, Haapiainen R, Kivisaari L, Kivilaakso E, Puolakkainen P. Early localization of necrosis by contrast enhanced computed tomography can predict outcome in severe acute pancreatitis. *Br J Surg*, 1996;83:924-929
- 2 Mann DV, Hershman MJ, Hittinger R, Glazer G. Multicentre audit of death from acute pancreatitis. *Br J Surg*, 1994;81:890-893

- 3 Hwang TL, Chiu CT, Chen HM, Chen SC, Jeng LB, Jan YY, Wang CS, Chen MF. Surgical results for severe acute pancreatitis-comparison of the different surgical procedures. *Hepatogastroenterology*, 1995;42:1026-1029
- 4 Bradley EL III. A clinically based classification system for acute pancreatitis: summary of the International Symposium on Acute Pancreatitis, Atlanta, Ga, September 11 Through 13, 1992. *Arch Surg*, 1993;128:586-590
- 5 Lucarotti ME, Virjee J, Alderson D. Patient selection and timing of dynamic computed tomography in acute pancreatitis. *Br J Surg*, 1993;80:1393-1395
- 6 Ranson JHC. Diagnostic standards for acute pancreatitis. *World J Surg*, 1997;21:136-142
- 7 Kawamura DM. *Abdomen and Superficial Structures*, second edition. Lippincott, 1997;7:249
- 8 Wang XP, Xu JY, Yuan YZ. PLA-2 and acute pancreatitis. *Xin Xiaohuabingxue Zazhi*, 1993;1:112-113
- 9 Wang YJ, Li N, Li JS. The detection and clinical significance of acute pancreatitis related protein. *Xin Xiaohuabingxue Zazhi*, 1996;4(Suppl 5):87-88
- 10 Wang XP, Wang GL, Wu XN, Yuan YZ, Xu JY. The significance of blood supply obstruction in acute pancreatitis. *Xin Xiaohuabingxue Zazhi*, 1997;5:55-56
- 11 Xu XF. The significance of LPO and SOD in acute pancreatitis. *Xin Xiaohuabingxue Zazhi*, 1997;5:473
- 12 Lu XD, Liu GS, Chen YR. Experimental and clinical research of tumor necrosis factor alpha on acute pancreatitis. *Xiaohuabingxue Zazhi*, 1997;5:534
- 13 Zhao LG, Chen Q. Decisive factor and severity assessment in severe acute pancreatitis. *Xin Xiaohuabingxue Zazhi*, 1997;5:591-592
- 14 Tu WF, Li JS. Inflammatory mediators and cytokines in pancreatitis-associated systemic inflammatory response syndrome. *Xin Xiaohuabingxue Zazhi*, 1997;5:595-598
- 15 Xu GH, Shi BJ, Liu HY. Analysis of prognostic factors in 178 patients with acute pancreatitis. *Xin Xiaohuabingxue Zazhi*, 1997;5:723-724
- 16 Chen GC, Chen YX, Zhao YP. The relationship between severe acute pancreatitis and other organs failure. *Xin Xiaohuabingxue Zazhi*, 1997;5:741-742
- 17 Xu JY. The research of severe acute pancreatitis. *Xin Xiaohuabingxue Zazhi*, 1996;5(Suppl 6):1-2
- 18 Lu L, Zhou Y, Fu YC. Significance of changes of inflammatory mediators in patients with acute pancreatitis. *Huaren Xiaohua Zazhi*, 1998;6:80-81
- 19 Wang ZF, Pan CE, Liu SG. Role of inflammatory mediators in acute pancreatitis. *Huaren Xiaohua Zazhi*, 1998;6:170-171
- 20 Wu XN. Current concept of pathogenesis of severe acute pancreatitis. *World J Gastroenterol*, 2000;6:32-36
- 21 Wu HG, Jing YZ, Zheng GQ, Peng QF. Retroperitoneal extension type acute necrotic pancreatitis. *Chin Med J*, 1981;94:171-174
- 22 Parekh D. Pancreatic ascites and effusion: risk factors for failure of conservative therapy and the role of octreotide. *Arch Surg*, 1992;127:707-712
- 23 Cohen JM, Weinreb JC, Maravilla KR. Fluid Collections in the intraperitoneal and extraperitoneal spaces: Comparison of MR and CT. *Radiology*, 1985;155:705-708
- 24 Love L, Meyers MA, Churchill RJ, Reynes CJ, Moncada R, Gibson D. Computed tomography of extraperitoneal spaces. *AJR*, 1981;136:781-789
- 25 King LR, Siegel MJ, Balfe DM. Acute pancreatitis in children: CT findings of intra and extrapancreatic fluid collections. *Radiology*, 1995;195:196-200
- 26 Li XX, Chen XL. The significance of 88 cases severe acute pancreatitis using non operative method. *Shijie Huaren Xiaohua Zazhi*, 1999;7:719-720
- 27 Ruan LB, Wang HG, Yang JZ, Tian GS. Case report of severe acute pancreatitis with left iliac artery thrombosis. *Shijie Huaren Xiaohua Zazhi*, 2000;8:207
- 28 Pan XZ. The early diagnosis and therapy of severe acute pancreatitis. *Xin Xiaohuabingxue Zazhi*, 1993;1:49-51
- 29 Pei DS. The rare complication of acute pancreatitis. *Xin Xiaohuabingxue Zazhi*, 1993;1:119-120
- 30 Huang DY, Huang DX. The surgical therapy and prognosis of 80 cases severe acute pancreatitis. *Xin Xiaohuabingxue Zazhi*, 1993;1:140-141
- 31 Cheng RX. Thirty-five cases severe acute pancreatitis. *Xin Xiaohuabingxue Zazhi*, 1995;3(Suppl 3):129
- 32 Xi XM, Zhu ZY, Wang XL, Dong QM, Shao JJ. Study on operative timing and procedures in acute necrotic pancreatitis. *Xin Xiaohuabingxue Zazhi*, 97;5:387-388
- 33 Zeng JG. The treatment of 119 cases acute pancreatitis. *Huaren Xiaohua Zazhi*, 1998;6(Suppl 7):393
- 34 Yuan QW, Shun L. The clinical analysis of 156 cases acute pancreatitis accompany with liver damage. *Huaren Xiaohua Zazhi*, 1998;6(Suppl 7):402
- 35 Gong ZY, Tang YQ, Han TQ. The complications and operation of 109 cases severe acute pancreatitis. *Shijie Huaren Xiaohua Zazhi*, 2000;8:1072-1074
- 36 Vesentini S, Bassi C, Talamini G, Cavallini G, Campedelli A, Pederzoli P. Prospective comparison of C reactive protein level, Ranson score and contrast enhanced computed tomography in the prediction of septic complications of acute pancreatitis. *Br J Surg*, 1993;80:755-757
- 37 Balthazar EJ, Robinson DL, Megibow AJ, Ranson JHC. Acute pancreatitis: value of CT in establishing prognosis. *Radiology*, 1990;174:331-336
- 38 Balthazar EJ, Ranson JHC, Naidich DP, Megibow AJ, Caccavale R, Cooper MM. Acute pancreatitis: prognostic value of CT. *Radiology*, 1985;156:767-772
- 39 Saifuddin A, Ward J, Ridgway J, Chalmers AG. Comparison of MR and CT scanning in severe acute pancreatitis: initial experiences. *Clin Radiol*, 1993;48:111-116
- 40 Luo Y, Yuan CX, Jiang JM, Dai L, Peng YL, Li YZ. The clinical significance of ultrasoning acute fluid collection in acute pancreatitis. *Shijie Huaren Xiaohua Zazhi*, 2000;8:1077-1078