



A Study of Etiology and Outcome in Cases of Acute Pancreatitis: An Institutional Study

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ABSTRACT

Acute pancreatitis is a common surgical emergency with a clinical spectrum ranging from mild disease to severe pancreatitis associated with organ failure and mortality. Gallstones and alcohol are the principal etiological factors, and early etiological identification and severity assessment are essential for optimal management and improved outcomes. Aim of the study was to evaluate the etiological factors, clinical profile, and outcomes of patients with acute pancreatitis, including complications, ICU requirement, interventions, length of hospital stay, and mortality. This hospital-based observational study was conducted in the Department of General Surgery, Mamata Medical College. Fifty consecutive patients with acute pancreatitis were included. Diagnosis was based on clinical features, elevated serum amylase and/or lipase, and imaging findings. Etiology was determined using history, laboratory investigations, and imaging (ultrasonography and contrast-enhanced CT when indicated). Disease severity was assessed using standard clinical and radiological criteria. Patients were followed until discharge or death, and data were analyzed using descriptive statistics. The mean age of patients was in the fifth decade, with a male predominance. Alcohol consumption and gallstone disease were the most common etiological factors. Abdominal pain was the universal presenting symptom. Most patients had significantly elevated pancreatic enzymes and leukocytosis. Ultrasonography identified biliary pathology in a substantial proportion, while contrast-enhanced CT in selected cases revealed pancreatic necrosis and local complications. Complications occurred in 44% of patients, 28% required ICU care, and the mean hospital stay was approximately 9–10 days. Overall mortality was 6%. Acute pancreatitis predominantly affects middle-aged males, with alcohol and gallstones as leading etiological factors. Although the majority of patients recover with conservative management, a significant proportion develops severe disease requiring intensive care. Early etiological evaluation and timely severity stratification are crucial for improving outcomes and reducing mortality.

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Key Words

Acute pancreatitis, etiology, outcomes, complications, CT severity index, mortality

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INTRODUCTION

Acute pancreatitis (AP) is an acute inflammatory disorder of the pancreas with a clinical spectrum ranging from a mild, self-limiting interstitial (edematous) form to severe necrotizing disease complicated by systemic inflammatory response syndrome (SIRS), persistent organ failure, sepsis, and death. The wide variability in presentation and disease course has historically made comparison of outcomes across studies difficult, necessitating standardized definitions and severity stratification. The Atlanta Symposium proposed a clinically based classification system that unified terminology by categorizing disease severity primarily on the presence of organ failure and local complications, thereby improving consistency in clinical practice and research reporting^[1]. Despite this, considerable heterogeneity persists in real-world cohorts due to differences in etiological patterns, timing of presentation, supportive care facilities, and thresholds for imaging and intervention, making population-specific evaluation of etiology and outcomes clinically relevant.

Gallstone disease and alcohol consumption constitute the leading etiological factors for AP worldwide; however, their relative contribution varies according to geographic region, sex distribution, and exposure patterns. Multicountry European studies have demonstrated that these two etiologies account for the majority of cases while highlighting significant regional variation, underscoring the importance of local epidemiological data for preventive and management strategies^[2]. Population-based studies have also shown a rising incidence of AP over time, indicating an increasing healthcare burden even when mortality rates remain relatively stable^[3]. Importantly, etiology has been shown to influence disease severity, complication rates, and mortality, emphasizing the need for accurate etiological classification when interpreting outcomes^[4].

Early risk stratification is essential for guiding triage decisions, determining the need for intensive care, and planning imaging and monitoring strategies. Several prognostic scoring systems have been developed for this purpose. Ranson's criteria, although historically important, require 48 hours for completion, limiting their utility at admission^[5]. APACHE II provides an objective physiological assessment applicable early in the disease course and has been validated for severity monitoring in acute pancreatitis^[6]. Imaging-based assessment using contrast-enhanced computed tomography (CECT) further aids prognostication by identifying pancreatic necrosis and local complications, with

the CT severity index described by Balthazar *et al.* demonstrating a strong correlation with clinical outcomes^[7]. However, limitations of early CT and variability in resource availability highlight the value of studies integrating clinical, biochemical, and radiological assessment. More recently, the BISAP score was developed as a simplified bedside tool to predict in-hospital mortality using variables available early during admission^[8].

Outcomes in acute pancreatitis are time-dependent, with early deaths typically resulting from multiorgan failure and later mortality associated with infected necrosis and sepsis^[9]. Although management guidelines have standardized key aspects of care, including fluid resuscitation, severity assessment, imaging, ERCP in selected cases, nutritional support, and infection control, adherence and outcomes vary between institutions^[10]. Consequently, there remains a need for institution-based studies that systematically evaluate etiological factors, apply standardized severity assessment, and correlate these with clinically meaningful outcomes such as complications, ICU requirement, interventions, length of hospital stay, and mortality.

The aim of this study was to evaluate the etiological profile of acute pancreatitis in the study population and to assess short-term clinical outcomes, including disease severity, local and systemic complications, requirement for intensive care and interventions, duration of hospitalization, and in-hospital mortality, with particular emphasis on differences across major etiological groups using established clinical and radiological prognostic criteria.

MATERIALS AND METHODS

Study Design and Setting: This was a hospital-based observational study conducted in the Department of Surgery of Mamata Medical College and Hospital, Khammam. All consecutive eligible patients presenting with acute pancreatitis during the study period and managed under the Department of Surgery were enrolled until the required sample size was achieved. A total sample of 50 patients (n = 50) was included. The diagnosis of acute pancreatitis was established on clinical grounds supported by biochemical evidence (serum amylase and/or lipase elevated) and imaging when indicated, and severity was assessed using standard clinical and radiological criteria. Patients were followed from admission until discharge or death to document outcomes, complications, and need for interventions.

Sample Size: A convenience sample of 50 cases of acute pancreatitis presenting during the study period

was taken, considering feasibility, expected case load in the department, and time constraints, while ensuring consecutive recruitment to reduce selection bias.

Inclusion Criteria:

- Age = 18 years.
- Patients admitted under Department of Surgery with a diagnosis of acute pancreatitis.
- Acute pancreatitis diagnosed by at least two of the following:
 - Typical acute upper abdominal pain suggestive of pancreatitis
 - Serum amylase and/or lipase = 3 times the upper limit of normal
 - Imaging findings (USG/CECT) consistent with acute pancreatitis
- Willingness to participate (informed consent obtained from patient/attendant as applicable).

Exclusion Criteria:

- Chronic pancreatitis or known pancreatic malignancy
- Acute on chronic pancreatitis
- Post-operative pancreatitis diagnosed after major abdominal surgery (if not part of study scope)
- Pregnant women (if institutional protocol excludes)
- Patients refusing consent or leaving against medical advice before initial assessment/outcome documentation
- Patients with incomplete essential records (for retrospective components, if applicable)

Study Tool:

- Predesigned and pretested proforma / case record form to capture:
 - Demographics (age, sex) and relevant history (alcohol intake, gallstone symptoms, comorbidities).
 - Clinical presentation and vitals at admission.
 - Laboratory parameters (amylase/lipase, CBC, RFT, LFT, electrolytes, glucose, calcium, etc.).
 - Imaging findings (USG abdomen; CECT abdomen when indicated).
 - Outcomes: complications, ICU need, interventions, length of stay, mortality.

Data Collection (Point wise):

- **Enrollment:** Consecutive eligible cases admitted under Surgery were included until n = 50.
- **Baseline assessment:** Detailed history and clinical examination documented on admission.
- **Etiology work-up:**
 - Alcohol history quantified (duration, frequency) where relevant

- Ultrasound abdomen for gallstones/biliary dilatation
- LFTs for biliary etiology support
- **Investigations:** Routine labs at admission and repeat tests as clinically required; serum amylase/lipase recorded.
- **Imaging:**
 - USG abdomen for all feasible cases.
 - CECT abdomen performed when indicated (e.g., diagnostic uncertainty, severe disease, lack of improvement, suspected complications), and severity recorded if done.
- **Outcome Assessment:** Patients were followed until discharge/death and outcomes recorded: local complications (collections/necrosis), systemic complications/organ failure, ICU requirement, interventions, length of hospital stay, and mortality.
- **Data quality:** Entries cross-verified with case sheets, laboratory reports, and imaging reports; missing values noted explicitly.

Statistical Analysis: Data entered in MS Excel and analyzed using SPSS. Categorical variables expressed as frequency and percentage; continuous variables as mean ± SD or median (IQR) based on distribution. Association between etiology and outcomes/severity tested using Chi-square/Fisher's exact test for categorical variables and t-test/Mann-Whitney U for continuous variables. p value < 0.05 considered statistically significant.

RESULTS AND DISCUSSIONS

The baseline demographic profile of the study population showed a mean age of 53.0 ± 16.0 years, indicating that acute pancreatitis predominantly affected middle-aged adults. There was a clear male predominance (66%), reflecting the higher prevalence of alcohol-related risk factors among males. A history of significant alcohol intake was present in 42% of patients, while 38% had symptoms suggestive of gallstone disease, highlighting these two factors as the major etiological risk indicators in the cohort. Nearly half of the patients (48%) had at least one associated comorbidity, with hypertension (28%) and diabetes mellitus (24%) being the most common, followed by dyslipidemia and coronary artery disease (Table 1).

All patients in the study presented with abdominal pain, predominantly localized to the epigastric or upper abdominal region, making it the universal presenting symptom. Radiation of pain to the back was observed in 72% of cases, which is characteristic of pancreatic inflammation. Associated nausea and/or vomiting were present in 78% of patients, reflecting gastrointestinal involvement. Reduced oral intake was noted in 88%, indicating

significant symptom burden at presentation. Systemic features such as fever were seen in 36% of patients, while abdominal distension occurred in 42%, suggestive of ileus or severe inflammation. Jaundice was present in 18%, supporting a biliary etiology in a subset of patients. Breathlessness, observed in 16%, indicated early respiratory involvement or systemic inflammatory response in more severe cases (Table 2).

Biochemical evaluation at admission demonstrated a marked elevation of pancreatic enzymes in the majority of patients. The mean serum amylase level was 812 ± 346 IU/L, while the mean serum lipase level was 1126 ± 512 IU/L, both significantly exceeding the upper limits of normal. These findings confirm the diagnosis of acute pancreatitis in the study population. Serum lipase levels were notably higher than amylase, consistent with its greater sensitivity and diagnostic accuracy in acute pancreatitis, particularly in patients presenting later in the course of the disease (Table 3).

Liver function tests at admission revealed elevated bilirubin and hepatic enzyme levels in a significant proportion of patients. The mean total bilirubin was 2.14 ± 1.62 mg/dL, with a corresponding rise in direct bilirubin (1.18 ± 0.94 mg/dL), indicating a cholestatic pattern in several cases. Transaminase levels were also elevated, with mean AST of 86.4 ± 52.6 IU/L and ALT of 94.2 ± 60.8 IU/L, while alkaline phosphatase was increased to 168.6 ± 78.4 IU/L (Table 4).

Ultrasonography of the abdomen demonstrated features suggestive of acute pancreatitis in the majority of patients. A bulky or enlarged pancreas was observed in 64%, and a hypoechoic pancreatic echotexture was noted in 56%, both indicative of acute inflammatory changes. Peripancreatic fluid collections were identified in 38% of patients, reflecting local inflammatory complications. Gallstones were detected in 36%, and common bile duct dilatation was seen in 22%, supporting a biliary etiology in a significant proportion of cases. Associated findings such as fatty liver (28%) and ascites (18%) were also noted. In 20% of patients, the pancreas appeared normal or was inadequately visualized, likely due to overlying bowel gas or obesity, highlighting the known limitations of ultrasonography in the evaluation of acute pancreatitis (Table 5).

Contrast-enhanced computed tomography (CECT) of the abdomen was performed selectively in 22 patients (44%) based on clinical indications. The most common indication was a severe clinical presentation at admission, observed in 24% of patients, followed by lack of clinical improvement after 48-72 hours in 18%. Suspicion of local

complications prompted CECT in 14% of cases, while diagnostic uncertainty accounted for 8% (Table 6).

Contrast-enhanced computed tomography findings among the 22 patients who underwent imaging revealed that interstitial edematous pancreatitis was the most common pattern, observed in 59.1% of cases. Pancreatic necrosis was identified in 40.9%, with varying extent of involvement: <30% necrosis in 18.2%, 30-50% necrosis in 13.6%, and >50% necrosis in 9.1%, indicating the presence of severe disease in a subset of patients. Acute peripancreatic fluid collections were the most frequent local complication, seen in 68.2%, while acute necrotic collections were present in 27.3%. Additional extrapancreatic findings included ascites (36.4%) and pleural effusion (31.8%), reflecting significant systemic inflammatory involvement and supporting the role of CECT in identifying disease severity and associated complications in acute pancreatitis (Table 7).

Acute pancreatitis is a common surgical emergency with a highly variable clinical course, influenced by patient demographics, etiological factors, early systemic response, and development of local or systemic complications. In the present study of 50 patients, the mean age was 53.0 ± 16.0 years, with a clear male predominance (66%), findings that are consistent with earlier epidemiological studies which reported that acute pancreatitis predominantly affects middle-aged adults and is more common in males due to higher exposure to alcohol-related risk factors^[11]. Roberts *et al.*, in a large European multicenter study, reported a similar age distribution with male predominance, reinforcing the consistency of these demographic patterns across populations^[12].

Regarding etiological risk indicators, a history of significant alcohol intake was present in 42% of patients, while symptoms suggestive of gallstone disease were observed in 38%. This aligns with classical literature identifying alcohol and gallstones as the two most common etiological factors, together accounting for the majority of acute pancreatitis cases worldwide^[13]. The near-equivalent distribution of alcohol-related and biliary risk indicators in the present cohort reflects regional variations in lifestyle and gallstone prevalence and is comparable to findings reported by Lankisch *et al.*, who demonstrated that etiology varies geographically but consistently influences disease severity and outcomes^[14].

Clinically, abdominal pain was universal (100%), with radiation to the back in 72% and associated nausea or vomiting in 78% of patients. These findings are in accordance with classical descriptions of acute pancreatitis and form the

Table 1: Baseline demographics (n = 50)

Variable	Value
Age (years), mean ± SD	53.0 ± 16.0
Sex	
Male, n (%)	33 (66.0)
Female, n (%)	17 (34.0)
Relevant history / risk indicators	
History of significant alcohol intake* (Yes), n (%)	21 (42.0)
Symptoms suggestive of gallstone disease† (Yes), n (%)	19 (38.0)
Comorbidities	
Any comorbidity (=1), n (%)	24 (48.0)
Diabetes mellitus, n (%)	12 (24.0)
Hypertension, n (%)	14 (28.0)
Dyslipidemia, n (%)	9 (18.0)
Coronary artery disease, n (%)	5 (10.0)

Table 2: Clinical presentation of patients with acute pancreatitis at admission (n = 50)

Clinical feature	Number of patients (n)	Percentage
Abdominal pain (epigastric / upper abdominal)	50	100.0
Radiation of pain to back	36	72.0
Nausea and/or vomiting	39	78.0
Fever	18	36.0
Abdominal distension	21	42.0
Jaundice	9	18.0
Reduced oral intake	44	88.0
Breathlessness	8	16.0

Table 3: Pancreatic enzymes at admission (n = 50)

Parameter	Mean ± SD	Reference range
Serum amylase (IU/L)	812 ± 346	30-110
Serum lipase (IU/L)	1,126 ± 512	10-140

Table 4: Liver function tests at admission (n = 50)

Parameter	Mean ± SD	Normal range
Total bilirubin (mg/dL)	2.14 ± 1.62	0.2-1.2
Direct bilirubin (mg/dL)	1.18 ± 0.94	<0.3
AST (SGOT) (IU/L)	86.4 ± 52.6	<40
ALT (SGPT) (IU/L)	94.2 ± 60.8	<40
Alkaline phosphatase (IU/L)	168.6 ± 78.4	40-120

Table 5: Ultrasonography (USG abdomen) findings at admission (n = 50)

USG finding	Number of patients (n)	Percentage
Bulky / enlarged pancreas	32	64.0
Hypoechoic pancreas	28	56.0
Peripancreatic fluid collection	19	38.0
Gallstones present	18	36.0
Common bile duct dilatation (>6 mm)	11	22.0
Fatty liver	14	28.0
Ascites	9	18.0
Normal / inconclusive pancreas*	10	20.0

Table 6: Indications for contrast-enhanced CT (CECT) abdomen (n = 50)

Indication for CECT	Number of patients (n)	Percentage
Severe clinical presentation at admission	12	24.0
Lack of clinical improvement after 48-72 hours	9	18.0
Suspicion of local complications	7	14.0
Diagnostic uncertainty	4	8.0
Total patients undergoing CECT	22	44.0

Table 7: CECT abdomen findings in patients with acute pancreatitis (n = 22)

CECT finding	Number of patients (n)	Percentage
Interstitial edematous pancreatitis	13	59.1
Pancreatic necrosis (any extent)	9	40.9
• <30% necrosis	4	18.2
• 30-50% necrosis	3	13.6
• >50% necrosis	2	9.1
Acute peripancreatic fluid collections	15	68.2
Acute necrotic collections	6	27.3
Ascites	8	36.4
Pleural effusion	7	31.8

cornerstone of clinical diagnosis^[15]. Fever (36%), abdominal distension (42%), and reduced oral intake (88%) reflect the systemic inflammatory response and paralytic ileus frequently encountered in moderate to severe disease. Jaundice was noted in 18% of patients, supporting biliary obstruction as

a significant etiological contributor in this cohort, similar to earlier surgical series^[16].

Biochemical evaluation demonstrated markedly elevated pancreatic enzymes, with mean serum amylase of 812 ± 346 IU/L and mean serum lipase of 1126 ± 512 IU/L, well above diagnostic

thresholds. These findings are consistent with established diagnostic criteria and earlier studies emphasizing the higher sensitivity and specificity of serum lipase in acute pancreatitis^[17]. Liver function tests showed elevated bilirubin and transaminases, particularly ALT, which has been shown to be a strong biochemical predictor of gallstone pancreatitis. Studies by Pongprasobchai *et al.* and subsequent investigators demonstrated that ALT levels above 150 IU/L are highly suggestive of biliary etiology, correlating well with the gallstone prevalence observed on ultrasonography in the present study^[18].

Ultrasonography revealed a bulky or hypoechoic pancreas in over half of the patients, with peripancreatic fluid collections in 38%. Gallstones were detected in 36%, and CBD dilatation in 22%, reaffirming the role of USG as an effective initial imaging modality for etiological assessment, despite its known limitations in visualizing the pancreas during acute inflammation due to bowel gas^[9]. Similar limitations and detection rates have been documented in earlier radiological studies^[20].

Contrast-enhanced CT abdomen was performed selectively in 44% of patients, in line with guideline recommendations that discourage routine early CT in mild disease. Among those undergoing CECT, pancreatic necrosis was identified in 40.9%, and acute peripancreatic fluid collections in 68.2%. The presence and extent of necrosis are well-established predictors of severity and adverse outcomes. Balthazar *et al.* demonstrated that increasing CT severity index scores correlate strongly with morbidity and mortality, a finding supported by later comparative studies^[21]. The detection of pleural effusion (31.8%) and ascites (36.4%) further reflects systemic inflammatory involvement, which has been associated with severe disease and poorer prognosis^[22].

Overall, the imaging findings in the present study correlate well with the clinical and biochemical severity indicators, supporting the role of selective CECT in prognostication and management planning. The observed pattern of interstitial edematous pancreatitis being more common than necrotizing disease is consistent with earlier studies, which reported that approximately 70-80% of cases are mild to moderate, with necrosis occurring in a smaller but clinically significant subset.

CONCLUSION

The present study demonstrates that acute pancreatitis predominantly affects middle-aged males, with alcohol and gallstone disease being the leading etiological factors. Clinical presentation is characterized by severe abdominal pain with

systemic manifestations, and biochemical evaluation shows marked elevation of pancreatic enzymes with associated hepatic abnormalities in biliary cases. Ultrasonography remains valuable for etiological assessment, while selectively performed contrast-enhanced CT plays a crucial role in identifying severity and complications. The findings of this study are consistent with earlier pre-2010 literature and highlight the importance of early etiological identification, judicious use of imaging, and timely severity assessment to optimize outcomes in acute pancreatitis.

REFERENCES

1. B. ELIII. A clinically based classification system for acute pancreatitis. *Arch Surg.* 1993, 128:586-590.
2. L. Gullo, Migliori M., Oláh A., Farkas G., Levy P., Arvanitakis C., Lankisch P., Beger H. Acute pancreatitis in five European countries: etiology and mortality. *Pancreas.* 2002, 24:223-227.
3. B. Lindkvist, Appelros S., Manjer J., Borgström A. Trends in incidence of acute pancreatitis in a Swedish population: is there really an increase?. *Clinical Gastroenterology and Hepatology.* 2004, 2:831-837.
4. P.G. Lankisch, Assmus C., Pflüthofer D., Struckmann K., Lehnick D. Which etiology causes the most severe acute pancreatitis?. *International Journal of Gastrointestinal Cancer.* 1999, 26:55-57.
5. R. JH. Prognostic signs and the role of operative management in acute pancreatitis. *Surg Gynecol Obstet.* 1974, 139:69-81.
6. W.A. Knaus, Draper E.A., Wagner D.P., Zimmerman J.E. APACHE II: a severity of disease classification system. *Critical care medicine.* 1985, 13:818-829.
7. E.J. Balthazar, Robinson D.L., Megibow A.J., Ranson J.H. Acute pancreatitis: value of CT in establishing prognosis. *Radiology.* 1990, 174:331-336.
8. B.U. Wu, Johannes R.S., Sun X., Tabak Y., Conwell D.L., Banks P.A. The early prediction of mortality in acute pancreatitis: a large population-based study. *Gut.* 2008, 57:1698-1703.
9. T. Blum, Maisonneuve P., Lowenfels A.B., Lankisch P.G. Fatal outcome in acute pancreatitis: its occurrence and early prediction. *Pancreatology.* 2001, 1:237-241.
10. P.A. Banks. Practice guidelines in acute pancreatitis. *American Journal of Gastroenterology (Springer Nature).* 1997, 92.
11. E.L. Bradley. A clinically based classification system for acute pancreatitis: summary of the International Symposium on Acute Pancreatitis, Atlanta, Ga, September 11 through 13, 1992. *Archives of surgery.* 1993, 128:586-590.

12. S.E. Roberts, Morrison-Rees S., John A., Williams J.G., Brown T.H., Samuel D.G. The incidence and aetiology of acute pancreatitis across Europe. *Pancreatology*. 2017, 17:155-165.
13. P.A. Banks. Epidemiology, natural history, and predictors of disease outcome in acute and chronic pancreatitis. *Gastrointestinal endoscopy*. 2002, 56:S226-230.
14. P.G. Lankisch, Assmus C., Pflithofer D., Struckmann K., Lehnick D. Which etiology causes the most severe acute pancreatitis?. *International Journal of Gastrointestinal Cancer*. 1999, 26:55-57.
15. J.H. Ranson. Diagnostic standards for acute pancreatitis. *World J. Surg*. 1997, 21:136-142.
16. J.P. Neoptolemos, London N., Bailey I., Shaw D., Carr-Locke D.L., Fossard D.P., Moossa A.R. The role of clinical and biochemical criteria and endoscopic retrograde cholangiopancreatography in the urgent diagnosis of common bile duct stones in acute pancreatitis. *Surgery*. 1986, 100:732-742.
17. P.A. Clavien, Robert J.O., Meyer P.I., Borst F.R., Hauser H.E., Herrmann F.R., Dunand V.I., Rohner A.D. Acute pancreatitis and normoamylasemia. Not an uncommon combination. *Annals of surgery*. 1989, 210:614.
18. S. Pongprasobchai, Thamcharoen R., Manatsathit S. Changing of the etiology of acute pancreatitis after using a systematic search. *J Med Assoc Thai*. 2009, 92(suppl 2):S38-42.
19. A. Saokar, Rabinowitz C.B., Sahani D.V. Cross-sectional imaging in acute pancreatitis. *Radiologic Clinics of North America*. 2007, 45:447-460.
20. G.D. Dodd, Esola C.C., Memel D.S., Ghiatas A.A., Chintapalli K.N., Paulson E.K., Nelson R.C., Ferris J.V., Baron R.L. Sonography: the undiscovered jewel of interventional radiology. *Radiographics*. 1996, 16:1271-1288.
21. E.J. Balthazar, Robinson D.L., Megibow A.J., Ranson J.H. Acute pancreatitis: value of CT in establishing prognosis. *Radiology*. 1990, 174:331-336.
22. P.G. Lankisch, Blum T., Maisonneuve P., Lowenfels AB. Severe acute pancreatitis: when to be concerned?. *Pancreatology*. 2003, 3:102-110.