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*RESEARCH ARTICLE*

# STUDY OF SERUM CALCIUM AND SERUM AMYLASE IN PROGNOSIS OF ACUTE PANCREATITIS AND ITS MANAGEMENT

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## Abstract

**Background**: Acute pancreatitis is one of the most common acute non-surgical emergenciesgetting admitted under general surgery unit. These patients generally at the time of presentation are very fragile and need adequate resuscitation which if failed can lead to SIRS which can eventually lead to MODS. Rise in serum amylase and fall in serum calcium are important diagnostic tools and their relationship can help in predicting better prognostic results in patients with acute pancreatitis

**Objective**: This study is done to:1.to study serum amylase levels in patients of acute pancreatitis at the admission and at 48 hours2.to study serum calcium levels at the time of admission3.to find out relationship, if any, between serum calcium and serum amylase in relation to prognosis compared by hospital stay in patients of acute pancreatitis4.to study total hospital stay in patients of acute pancreatitis

**Methods**: this hospital based retrospective open case study includes patients admitted in a tertiary hospital at Navi Mumbai from May 2007 to September 2009. Due clearance was taken for the study by ethics committee of the medical college. The patients admitted from opd or emergency room were explained about their involvement in the study and due consent was taken. Initial assessment and resuscitation were done by on call surgery team. Patient was then shifted to ward or surgical icu based on patients' condition on admission. complete blood profile and radiological assessment were done. patients progress was monitored from the time of admission till discharge. Patients on admission serum calcium and serum amylase were sent. Amylase was repeated at 48 hours from admission. Their values are relationship with each other were compared

Results: In our study of 60 patients,55 patients had serum amylase level above normal limits. At 48 hrs, in 32 patients, serum amylase level fell to normal limits.in rest 21patients, levels were reduced but was still above normal limits.in 2 patients, serum amylase were further elevated.

In this study, 16 patients had calcium level less than 8.4. 1 patient had calcium level more than 10.4. Rest patients had serum calcium level within normal range

**Conclusion**: In our study in 60 patients of acute pancreatitis, serum amylase levels were raised at admission and was an important diagnostic tool for diagnosis of acute pancreatitis. The serum amylase levels significantly dropped after 48 hours. Serum calcium levels fluctuated marginally within normal limits and dropped just below normal in some cases. however, serum calcium was not important as a diagnostic tool

Serum amylase levels at admission corelated well with hospital stay and relationship was direct and linear. higher serum amylase levels were associated with longer hospital stay and lower levels with lesser stay. Serum amylase levels declined in most cases at 48 hours.The serum calcium levels also corelated well with hospital stay and relationship was linear and inverse. lower levels were associated with longer hospital stay and higher / normal levels with shorter hospital stay.



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## Introduction:-

**Materials and Methods**

Study Design and Setting

This retrospective study was conducted at our tertiary care hospital over a period of 2 years and 4months from month of May 2007 to September 2009. This study received ethical approval from the institutional review board.

Study Population

All patients who presented to the surgical outpatient department and emergency room with acute abdomen which was later diagnosed as acute pancreatitis. Male and female patients were included in this study. Age restrictions were not imposed.

## Data Collection.

The medical records of the eligible patients were reviewed, and data were extracted using a standardized collection form. The following parameters were collected.

1. Demographics: Age, sex, residence (rural/urban), personal history including diet and addictions, previous similar complaints
2. Clinical presentation: Chief complaints, duration of symptoms, and physical examination findings
3. Diagnostic investigations: Ultrasonography, Blood samples, x ray abdomen and chest, CT SCAN of abdomen and pelvis, MRCP
4. Management details: Initial Resuscitation,Conservative measures, medical therapy, physiotherapy &rehabilitation
5. Follow-up outcomes: Symptom resolution, recurrence, complications, and patient satisfaction.

## Statistical Analysis.

The collected data were coded, entered, and analysed using the appropriate statistical software. Descriptive statistics were used to determine and analyse serum calcium, serum amylase and their relationship to prognosis in patients with acute pancreatitis. Categorical variables are expressed as frequencies and percentages, while continuous variables are presented as means with standard deviations or medians with interquartile ranges based on the distribution pattern.

## Results.

Demographic Characteristics

A total of 60 patients with acute pancreatitis wereanalysed during the 2 year 4 month-month study period. Among these, 39 (65%) were males and 21 (35%) were females, with a female-to-male ratio of approximately 1.8:1. The age distribution of patients ranged from 23to 72 years. The majority of patients (66.6%) were aged 20-50 years age group.

**Table 1: Age and Sex Distribution of Patients with acute pancreatitis**

| Age Group (years) | Female | Male | Total | Percentage (%) |
|---|---|---|---|---|
| 0-9 | 0 | 0 | 0 | 0 |
| 10-19 | 0 | 1 | 1 | 1.6 |
| 20-29 | 5 | 5 | 10 | 16.66 |
| 30-39 | 4 | 14 | 18 | 30 |
| 40-49 | 5 | 10 | 15 | 25 |
| 50 &above | 7 | 9 | 16 | 26.6 |
| Total | 21 | 39 | 60 | 100.0 |

Distribution of acute pancreatitis Of the 60 cases, 42(70%) were diagnosed with acute oedematous pancreatitis, while 18(30%) had acute necrotising pancreatitis.

Clinical Presentation

The most common presenting complaint was acute pain in abdomen radiating to back, reported by 51 patients (85%), followed by vomiting/nausea in 36 patients (60%) and fever in 24 (40%) Some patients present with multiple symptoms. The duration of symptoms ranges from a couple of hours to couple of weeks.

**Table 2: Clinical Presentation of acute pancreatitis**

| Clinical Feature | Number of Cases | Percentage (%) |
|---|---|---|
| Epigastric pain | 51 | 85 |
| Vomiting / nausea | 36 | 60 |
| fever | 24 | 40 |

## Diagnostic Approaches.

All patients underwent thorough clinical examination, followed by selective diagnostic investigations based on clinical presentation and suspicion. Ultrasonography was performed in 60 patients (100%), CECT was performed at 48 hrs from admission in 48 patients (80%). MRCP was performed in 20 patients (33%) in whom common bile duct and pancreatic duct obstruction pathology was suspected.

**Table 3: Diagnostic Modalities Utilized**

| Diagnostic Modality | Number of Cases | Percentage (%) |
|---|---|---|
| Clinical Examination | 60 | 100.0 |
| Ultrasonography | 100 | 100 |
| CECT AP | 48 | 80 |
| MRCP | 20 | 33% |

**Management Strategies.**

The management approach varies based on the diagnosis, severity of symptoms, patient preferences, and risk assessment. Conservative management was employed in 59 patients (98%). Conservative therapy included medical management withAnalgesics,anti-emetics, PPI therapy, chest physiotherapy,spirometry, early mobilisation and early initiation of enteral feeding. Surgical interventions were performed in 1 patientout of 60 who had acute haemorrhagic pancreatitis in which laparotomy was done with packing and drain placement.21 out of 60 patient were admitted to Surgical ICU as they had signs of MODS. 27 out of 60 were Admitted in Surgical HDU in whom deterioration was suspected. 12 were admitted directly to wards who were clinically and pathologically stable.

**Correlation Of Clinical And Radiological Diagnosis.**

The accuracy of the clinical diagnosis compared with the radiological diagnosis was analysed. The overall sensitivity of clinical diagnosis was 80%. Sonography was the diagnostic modality of choice on admission followed by CT scan at 48 hrs from admission.

Acute oedematouspancreatitis was present in 36 out of 60 cases and acute necrotising pancreatitis was present in 24 cases out of 60.

**Table 4:: Sensitivity of Clinical Diagnosis in Correlation with radiology**

| Diagnosis | Clinical Diagnosis | Final radiological Diagnosis | Difference | Sensitivity (%) |
|---|---|---|---|---|
| Acute pancreatitis | 42 | 60 | 18 | 70 |
| Total | 60 | 60 | - | - |

**Follow-up Outcomes**

Follow-up data were available for 21 patients (35%) with a mean follow-up duration of 4.2 ± 1.8 months. Among the patients with acute oedematouspancreatitis,12(57.2%)) reported complete symptomatic relief, while 9 (42.8%) showed mild symptoms which were intermittent requiring additional interventions like EUS and pseudocyst formation which required drainage due to compression symptoms or required cystogastrostomy for quality-of-life improvement.

## Discussion.

## 1.Serum Amylase At Admission

The association between elevated serum amylase levels and acute pancreatitis first described by Moynihan 70 in 1929 has been used as the corner stone of the diagnosis.

27 patients (45%) had serum amylase levels between 300 to 500 U/l. 22 patients (36.7%) had serum amylase levels between 80 to 300 U/l. only one patient had serum amylase levels greater than 1000 U/I

The diagnostic levels of serum amylase for acute pancreatitis in studies by Elman et and Grossman et all have been reported as 5 times above normal

Kumar et all54) reported only 10% of their cases having serum amylase of more than 1000 U/l whereas in many foreign studies (34.35.48.54) majority of patients have serum amylase levels of more than 1000 U/I

5 patients (8.3%) of our series had normal serum amylase levels.

Levin et all reported 15% of patients with acute pancreatitis with normal serum amylase levels.

The major difficulty with serum amylase is that there are many other non-pancreatic causes of hyperamylasaemia that make interpretation of this marker difficult at times.

Hyperamylasaemia in all other conditions rarely attains a value five times above normal although this commonly occurs in acute pancreatitis

In USA, values of serum amylase two to four times normal are considered positive as per studies by Imrie et al

**2.Serum Amylase After 48 Hours**

Serum amylase after 48 hours was found to be useful prognostic indicator.

In our study, 2 patients (3.3%) had followed up levels of serum amylase more than the initial values. Both the patients had gall stone induced acute pancreatitis, prolonged hospital stay and required surgical intervention. In 32 patients (53.3%), serum amylase normalized in 48 hours. In the remaining 26 patients (43.4%), serum amylase declined from initial levels but did not touch normal values.

Brokus et al 24), Foster et al, Pollock et alk) and Veith et alto) have all reported that the persistence of elevated serum amylase levels may be an ominous prognostic sign.

**3.Serum Calcium**

An important biochemical abnormality which occurs in acute pancreatitis is the loss of intravascular albumin. As a direct consequence of this, the circulating protein bound calcium falls, causing a drop in total blood calcium.

The prognostic value of serum calcium level in acute pancreatitis has been reported by Banks et al, Brokus et al 24, Shader et al Although the maximum decrease in serum calcium may not occur until the fifth or seventh day of illness, a serum calcium level below 8 mg/dl during first 48 hours of treatment is associated with a significant increased incidence of death or major complications as reported by Blamey et al21) and Ranson et al

In our study, the mean value of serum calcium was 8.8 md/dl. As seen from our study patients (26.7%) had serum calcium levels less than 8.4 mg/dl, however none of them developed any signs and symptoms related to hypocalcaemia. Of the remaining, only one PATIENT (1.7%) had serum calcium levels exceeding 10.4 mg/dl and rest had normal serum calcium levels.

**4. Hospital Stay**

Prognosis parameters studied in our series was hospital stay vis-à-vis serum amylase and serum calcium levels. 46.7% of patients required hospitalization for 7-14 days. In our study the mean hospital stay was 12 days. Mann(s) et al reported mean hospitalization of 18 days. The average length of stay in hospital varies from 10 to 17 days as per studies of Andersson), Appelros(3), Banks(7), Bollen (25), Eland(5), Fagenholz(7), Floyd, Frey/43), Goldacre (46), Mofidi(69) and Yadav (20), The average length of hospital stay for uncomplicated pancreatitis is 5-14 days and average length of hospital stay for complicated pancreatitis can range as high as 40 to 65 days as per patient care guidelines of The Society for Surgery of the Alimentary Tract (102)

Hospital stay well co-related with serum calcium levels and relationship of inverse proportion was observed as was observed by Izquierdo (51), Condon (30), Lindkvist(63) O'Farrell), Jorgensen (53), Nilsson (72), Pezzilli(9) and Connor(31) in their studies.
Average serum calcium for patients staying less than 7 days in hospital was 9mg/dl, for 7-14 days was 8.9mg/dl, 15-21 days was 8.4mg/dl and for more than 21 days was 8.6mg/dl.
Hospital stay well co-related with serum amylase levels and relationship of direct proportion as reported by Khan and Parekh (55); Yaadav et al (107) and also observed in studiesof Steinberg(93), Lin (62), Clavien et al (29), Winslet et al (205), Sotoudehmanesh(91), Gislason (45), Andersen, McCullough (68), Fagenholz(38) and de Carvalho (33)
Average serum amylase for patients staying less than 7 days in hospital was 1210/1, for 7-14 days was 337U/I, 15-21 days was 517U/I and for more than 21 days was 451U/I.

## Conclusion

In our study of 60 patients of acute pancreatitis, the serum amylase levels at admission were raised and were an important diagnostic tool. The serum amylase declined after 48 hours. The serum calcium levels fluctuated marginally within normal limits and dropped just below normal in some cases. However, serum calcium levels were not important as a diagnostic tool.
The values of serum amylase at admission correlated well with hospital stay and the relationship was direct and linear. Higher serum amylase levels were associated with longer hospital stay and lower levels with lesser stay. The serum amylase levels after 48 hours showed decline in most cases.
The serum calcium levels also correlated well with hospital stay and the relationship was linear and inverse. Low/Borderline low levels of serum calcium were associated with longer hospital stay and higher/normal levels with lesser stay.

## References.

1. Andersen AM, Novovic 5. Ersboll AK, Hansen MB: Mortality in alcohol and biliary acute pancreatitis. Pancreas 2008, 36(4):432-434,
2. Andersson R, Andersson P, Haraldsen P, Drewsen G, Eckerwall G: Incidence. management and recurrence rate of acute pancreatitis. Scandinavican Journal of Gastroenterology 2004, 39:891-894.
3. Appelros S, Borgstrom A: Incidence, aetiology and mortality rate of acute pancreatitis over 10 years in a defined urban population in Sweden. The British journal of surgery 1999, 86(4):465-470.
4. Bais, R. Am. Jnl. Of Clin. Path 2002; 78: 184-8
5. Balthazar EJ, Freeny PC, van Sonnenberg E. Imaging and intervention in acute pancreatitis. Radiology 1994; 193:297-306.
6. Balthazar EJ: Acute pancreatitis: Assessment of severity with clinical evaluation. Radiology 2002; 223:603-613.
7. Banks PA, Freeman ML: Practice guidelines in acute pancreatitis. Am J Gastroenterol 2006, 101(10):2379-2400.
8. Bapat RD, M B Agarwal, M S Kamdar, B C Mehta, S Rao, P N Rao Consumptive coagulopathy and fibrinolysis in acute pancreatitis. J Postgrad Med 1982; 28(4):214-217
9. Bapat R D; Jadhav R N; Mohite J D; Rohandia O S Modified Partington's procedure for pancreatojejunostomy in chronic pancreatitis. Indian journal of gastroenterology: official journal of the Indian Society of Gastroenterology 1997;16(2):54-5.
10. Bapat R D; Naik A: Shah SH; Relekar R G; Pancreatic calculus causing obstructive jaundice.
Indian journal of gastroenterology: official journal of the Indian Society of Gastroenterology 1991;10(1):27-8.
11. Bapat R D; Nazareth H M; Kulkarni A G; Shah A B Prognostic marker in acute pancreatitis--a prospective study-1986. Indian journal of gastroenterology: official journal of the Indian Society of Gastroenterology 1986;5(2):113-5.
12. Bapat R D; Pipalia D H; Naik SR; Ratnam V J; Plumber ST; Bhandarkar S D;Chronic calcific pancreatitis (C.C.P.) in western Maharashtra (experience of 55 cases). Journal of postgraduate medicine 1987;33(4):201-5.
13. Bapat R D; Upadhye A S; Mathur S K; Karmarkar S J; Pipalia D; Plumber S T; Gaitonde S G; Pancreatic pseudocysts (report of two cases). Journal of postgraduate medicine 1986;32(3):163-5.
14. Bapat R D; Upadhye A S; Nazareth H M; Hande A M; Pseudocyst of pancreas-a diagnostic conundrum (a case report). Journal of postgraduate medicine 1987;33(2):91-3.
15. Bapat R.D., S. P. Thorat, N. N. Rege, A. S. Naik, U. M. Thatte, A. Joshi, S. A. Dahanukar; EmblicaOfficinalis: A Novel Therapy for Acute Pancreatitis - An Experimental Study (1995) Hindawi Publishing Corporation,

http://dx.doi.org/10.1155/1995/51310 http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2443754, HPB surgery: a world journal of hepatic, pancreatic and biliary surgery 1995;9(1):25-30.
16. Bapat RD, Kantharia CV, Prabhu RY, Dalvi AN, Raut A, and Supe AN: Spectrum and outcome of pancreatic trauma: Tropical gastroenterology: official journal of the Digestive Diseases Foundation 28(3):105-8, 2007
17. Bapat RD, Prabhu RY, Irpatgire R, Naranje B, Kantharia CV, and Supe AN; Influence of timing on performance of laparoscopic cholecystectomy for acute biliary pancreatitis. Tropical gastroenterology: official journal of the Digestive Diseases Foundation 30(2):113-5, 2009
18. Bapat RD, Satish R R, Kantharia CV. Choice Of Surgical Procedures for Chronic Pancreatitis. Bombay Hospital Journal. January 2001. http://www.bhj.org/journal/2001_4301_jan/reviews_175.htm.
19. Baron TH, Morgan DE. Acute necrotizing pancreatitis. N Engl J Med 1999;340:1412-7.
20. Baurer P.J. Anal biochem 1981; 110; 61-72
21. Beeler M.F. and Catrou P.G. "Disorders of calcium Metabolism" in Interpretations in clinical A.C.S.P. Press Chicago 1983; 34-44
22. Beger HG, Rau B, Mayer J, Pralle U. Natural course of acute pancreatitis. World J Surg 1997;21:130-5.
23. Blamey 5 L, Imrie C W and O'Neill J (1984); Prognostic factors in acute pancreatitis. Gut 25; 1340-1346
24. Bockus H L, Kaiser M H and Roth JLA (1953) Clinical features of acute inflammation of the pancreas. Archives of internal medicine 96: 308-321
25. Bollen TL, van Santvoort HC, Besselink MG, van Leeuwen MS, Horvath KD, Freeny PC, Gooszen HG: The Atlanta Classification of acute pancreatitis revisited. The British journal of surgery 2008, 95(1):6-21.
26. Calleja GA, Barkin JS. Acute pancreatitis. Med Clin North Am 1993;77:1037-56.
27. Case RM, Argent BE: Pancreatic duct cell secretion: Control and mechanisms of transport. In: Go VLW, DiMagno EP, Gardner JD, et al ed. The Pancreas: Biology, Pathobiology, and Disease, 2nd ed. New York: Raven Press; 1993:301-350.
28. Case RM: Pancreatic exocrine secretion: Mechanisms and control.
In: Beger HG, Warshaw AW, Buchler MW, etal ed. The Pancreas, Oxford: Blackwell Science; 1998:63-100.
29. Clavien PA, Robert J, Meyer P, et al. Acute pancreatitis and amylasaemia. Ann Surg 1989;210(5):614-20.
30. Condon JR, Ives D, Knight MJ, Day J. The aetiology of hypocalcaemia in acute pancreatitis. Br J Surg. 1975 Feb;62(2):115-8
31. Connor SJ, Lienert AR, Brown LA, Bagshaw PF: Closing the audit loop is necessary to achieve compliance with evidence-based guidelines in the management of acute pancreatitis. N Z Med J 2008, 121(1275):19-25.
32. Corfield AP, Cooper MJ, Williamson RC. Acute pancreatitis: Aetiologies and serology of a lethal disease of increasing incidence. Gut 1985;26:724-9.
33. de Carvalho FR, dos Santos JS, Elias Junior J, Kemp R, Sankarankutty AK, Fukumori OY, Souza MC, de Castro-e-Silva O: The influence of treatment on profile of acute biliary pancreatitis. Acta Cir Bras 2008, 23(Suppl 1):143-150.
34. Dragonetti GC, Licht H, Rubin W. Pancreatitis. Evaluation and treatment, elevations in serum amylase. Prim Care 1996;23:525-34,
35. Eland IA, Sturkenboom MJ, Wilson JH, Stricker BH: Incidence and mortality of acute pancreatitis between 1985 and 1995. Scandinavian journal of gastroenterology 2000, 35(10):1110-1116.
36. Elman R., Arneson N., Graham E: Value of blood amylase in the diagnosis of pancreatic disease; a clinical study. Archives of surgery 1929: 19:943-67
37. Fagenholz PJ, Castillo CF, Harris NS, Pelletier AJ, Camargo CA Jr: Increasing United States hospital admissions for acute pancreatitis, 1988-2003. Annals of epidemiology 2007, 17(7):491-497.
38. Fagenholz PJ, Fernandez-del Castillo C, Harris NS, Pelletier Al, Camargo CA Jr: Direct medical costs of acute pancreatitis hospitalizations in the United States.Pancreas 2007, 35(4):302-307.
39. Farell C.E. "Electrolytes" in Clinical Chemistry Theory. Analysis and Correlation. The C V Mosby Company. Kaplan L.A., Pesce A.J. (Ed). 1984; Chap 55; 1054
40. Floyd A, Pedersen L, Nielsen GL, Thorladcius-Ussing O, Sorensen HT: Secular trends in incidence and 30-day case fatality of acute pancreatitis in North Jutland County, Denmark: a register-based study from 1981-2000. Scandinavian journal of gastroenterology 2002, 37(12):1461-1465
41. Foo.Y.A. and Brosalki, S.B. Ann. Clin. Biochem. 2006; 23: 624-37
42. Foster P. D and Ziffren SE (1962). Severe acute pancreatitis. Archives of surgery 85; 252-259.
43. Frey CF, Zhou H, Harvey DJ, White RH: The incidence and case-fatality rates of acute biliary, alcoholic, and idiopathic pancreatitis in California, 1994-2001. Pancreas 2006, 33(4):336-344.
44. Gamaste VV. Diagnostic tests for acute pancreatitis. Gastroenterologist 1994;2:119-30.

45. Gislason H, Horn A, Hoem D, Andren-Sandberg A, Imsland AK, Soreide O, Viste A: Acute pancreatitis in Bergen, Norway. A study on incidence, aetiology and severity. Scand J Surg 2004, 93(1):29-33.
46. Goldacre MJ, Roberts SE: Hospital admission for acute pancreatitis in an English population, 1963-98: database study of incidence and mortality. Bmj 2004, 328(7454):1466-1469.
47. Golub R, Siddiqi F, Pohl D: Aetiology of Acute pancreatitis: A meta-analysis. J GastrointestSurg 1998; 2:496-503.
48. Grossman M S; Prolonged hyperamylasaemia following acute pancreatitis. American Journal of Digestive diseases, 1964, 9; 618-624
49. Imrie C.W. and Shearer M G: Diagnosis and management of severe acute pancreatitis in Recent advances in Surgery No 10, Editor R C G Russell, Churchill Livingston, 1986
50. Isenmann R, Runzi M, Kron M, et al: Prognosis of patients with predicted severe acute pancreatitis: A placebo-controlled, double-blind trial. Gastroenterology 2004; 126:997-1004.
51. Izquierdo R, Bermes E Jr, Sandberg L, Saxe A, Oslapas R, Prinz RA.; Serum calcium metabolism in acute experimental pancreatitis. Surgery. 1985 Dec;98(6):1031-7
52. J.F.Ziva, and P.R. Pannali, "Plasma enzymes in diagnosis" in clinical chemistry in diagnosis and treatment. Lloyd London 2009 chapter XV: 341-2
53. Jorgensen T: Treatment of gallstone patients. Copenhagen: National Institute of Public Health, Denmark, and Danish Institute for Health Technology Assessment; 2000
54. Kaushik S P, Kumar A & Wig JD. Surgical management of acute pancreatitis and its complications. Indian Journal of Surgery, April 1988, 104-108
55. Khan AA, Parekh D, Young C, et al: Improved prediction of outcome in patients with severe acute pancreatitis Arch Surg 2002; 137:1136-114
56. Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: a severity of disease classification system. Crit Care Med 1985;13:818-29.
57. Koehler D F & Eckfeldt JH: Diagnostic value of routine amylase assay of hyperamylasaemia serum Gastroenterology 1982, 82:887-890
58. Kornfeld S: Trafficking of lysosomal enzymes in normal and disease states. J Clin Invest 1986; 77:1-6.
59. Le Moine O, Devaster JM, Deviere J, Thiry P, Cremer M, Ooms HA. Markers of acute alcoholic pancreatitis. Dig Dis Sci 1994;39: 2634-8.
60. Lerch MM, Saluja AK, Runzi M, et al: Pancreatic duct obstruction triggers acute necrotizing pancreatitis in the opossum. Gastroenterology 1993; 104:853-861
61. Levine JS; Hyperamylasemia in Decision making gastroenterology. D C Decker, 1985, 94-95
62. Lin XZ, Wang SS, Tsai YT, et al. Serum amylase, isoamylase, and lipase in the acute abdomen. Their prognostic value for acute pancreatitis. J Clin Gastroenterol 1989;11(1):47-52.
63. Lindkvist B, Appelros S, Manjer J, Borgstrom A: Trends of acute pancreatitis in a Swedish population. Clin Gastroenterol Hepatol 2004, 2(9):831-837.
64. Liu CL, Lo CM, Fan ST. Acute biliary pancreatitis: diagnosis and management. World J Surg 1997; 21:149-54.
65. Mann M J and Hershman MJ: Multicentre audit of death from acute pancreatitis. British Journal of Surgery 1994, 81; 890-893
66. Marshall JB. Aetiology and treatment of Acute pancreatitis. A review with an emphasis on new developments. Arch Intern Med 1993;153:1185-98.
67. Mayer AD, McMahon MJ, Corfield AP, Cooper MJ, Williamson RC, Dickson AP, et al. Acute pancreatitis. N Engl J Med 1985; 312:399-404.
68. McCullough L, Sutherland F, Preshaw R, Kim S: Gallstone pancreatitis: hospital stay. HPB (Oxford) 2003, 5(2):96-99.
69. Mofidi R, Madhavan KK, Garden OJ, Parks RW: An audit of the management of patients with acute pancreatitis against national standards of practice. The British journal of surgery 2007, 94(7):844-848.*93
70. Moynihan B. (1925). Acute pancreatitis. Annals of Surgery 81; 132-142
71. Nathens et al. Management of the critically ill patient with severe acute pancreatitis. Critical Care Medicine 2004, 32, 2524-36.
72. Nilsson E, Fored CM, Granath F, Blomqvist P: Cholecystectomy in Sweden 1987-99: a nationwide study of mortality and preoperative admissions. Scandinavian journal of gastroenterology 2005, 40(12):1478-1485.
73. Norman S. Williams, Christopher J.S. Bulstrode, P. Ronan O'Connell, SatyajitBattacharya: The pancreas. Bailey & Love's Short Practice of Surgery (2008), 64:1130-1153

74. O'Farrell A, Allwright S, Toomey D, Bedford D, Conlon K: Hospital admission for acute pancreatitis in the Irish population, 1997 2004: could the increase be due to an increase in alcohol-related pancreatitis? J Public Health (Oxf) 2007, 29(4):398-404.
75. Opie EL: The aetiology of acute haemorrhagic pancreatitis. Bull Johns Hopkins Hosp 1901; 12:182-192.
76. Owyang C, Logsdon CD: New insights into neurohumoral regulation of pancreatic secretion. Gastroenterology 2004; 127:957-964.
77. Palade G: Intracellular aspects of the process of protein secretion. Science 1975; 189:347-358.
78. Pancreatitis UKWPOA: UK guidelines for the management of acute pancreatitis. Gut 2005, 54(Suppl 3):1-9
79. Pezzilli R, Uomo G, Gabbrielli A, Zerbi A, Frulloni L, De Rai P, Castoldi L, Cavallini G, Di Carlo V: A prospective multicentre survey on the treatment of acute pancreatitis in Italy. Dig Liver Dis 2007, 39(9):838-846.
80. Pitchumoni CS, Bordalo O. Evaluation of hypotheses on pathogenesis of alcoholic pancreatitis. Am J Gastroenterol 1996;91:637-47.
81. Pollock A V (1959). Acute pancreatitis: Analysis of 100 patients. British medical journal Aug 1996:6-14
82. Powell et al. Antibiotic prophylaxis in the initial management of severe acute pancreatitis. British Journal of Surgery 1998, 85, 582-7.
83. Ranson JH C and Pasternack B.S (1977). Statistical methods for quantifying the severity of clinical acute pancreatitis. Journal of Surgical Research, 22; 79-91.
84. Ranson JH. Diagnostic standards for acute pancreatitis. World J Surg 1997;21:136-42.
85. Ranson JH. Risk factors in acute pancreatitis. HospPract [Off Ed] 1985;20:69-73.
86. Ranson JHC, Rifkind KM, Roses DF, et al: Prognostic signs and the role of operative management in acute pancreatitis. SurgGynecolObstet 1974; 139:69-81.
87. Ranson JHC: Etiological and prognostic factors in human acute pancreatitis: A review. Am J Gastroenterol 77:633, 1982.
88. Rau et al. Acute pancreatitis. Critical Care Medicine 2001, 29, 1556-62
89.Shader A E and Paxton R (1966) Fatal pancreatitis. American Journal of Surgery. 111:369-373
90. Sheehy TW. Acute alcoholic pancreatitis. Continuing Education: March 1980 March:87-88.92-3,97-100,105,107,109
91 .Sotoudehmanesh R. Khatibian M. Kolahdoozan 5. Ainechi S. Malboosbaf R. Nourale M: The incidence and severity of acute pancreatitis after ERCP. Am J Gastroenterol 2007, 102(5):978-983.
92. Steinberg W, Tenner S. Acute pancreatitis and carcinoma of pancreas. N Engl J Med 1994;1198-207.
93. Steinberg WM, Goldstein SS, Davis ND, et al. Diagnostic and prognostic assays in Acute Pancreatitis. Ann Intern Med 1985;102(5):576-580
94. Tenner S, Banks PA. Acute pancreatitis. World J Surg 1997;21:143-8.
95. Thomson HJ. Causes of Acute pancreatitis. JR CollSurgEdinb 1985:30: 104-11.
96. Tietz NW. Diagnosis of pancreatitis by enzyme tests-old problems, new techniques. Clin Chem Acta 1997;257:85-98.
97. Townsend: Sabiston Textbook of Surgery, 18th ed. (2008); Vol 2 Sec10 Ch55: 1589-1623
98. Tran DD. Evaluation of severity in patients with acute pancreatitis. Am J Gastroenterol 1992:87:604-8.
99. Tuesta MA. Severity of acute pancreatitis. Am J Gastroenterol 2002;17:22-6.
100. Uhl et al. IAP guidelines for the surgical management of acute pancreatitis.Pancreatology 2002, 2, 565-73.
101. UK guidelines for the management of acute pancreatitis. Working Party of the British Society of Gastroenterology, Association of Surgeons of Great Britain and
Ireland, Pancreatic Society of Great Britain and Ireland, Association of Upper Gi Surgeons of Great Britain and Ireland. Gut 2005; 54(Suppl 3):1-9.
102. Veith FJ and Filler R M (1963): Significance of prolonged elevation of serum amylase. Annals of surgery 158; 20-25
103. Webster PD, Spainhour JB. Management of acute pancreatitis. HospPract 1974;59-66.*12

104. Werner et al. Management of acute pancreatitis: from surgery to interventional intensive care. Gut 2005, 54, 426-36.
105. Winslet M, Hall C, London NJ, et al. Relation of diagnostic serum amylase levels to severity of acute pancreatitis Gut 1992;33(7):982-6.
106. www.ssat.com/cgi-bin/acupanc6.cgi-Acute pancreatitis:- Patient care guidelines of The Society for Surgery of the Alimentary Tract
107. Yadav D, Lowenfels AB: Trends in the epidemiology of the first attack of acute pancreatitis: a systematic review. Pancreas 2006, 33(4):323-330.