IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 18, Issue 5 Ser. 19 (May. 2019), PP 47-50
www.iosrjournals.org

A Retrospective, Comparative Study of Characteristics of Hypertriglyceridemic Pancreatitis and Biliary Pancreatitis in A Tertiary Care Hospital, Kurnool, Ap

Dr. Kranthi Kumar[1], Dr.Shankara Sharma Bondalapati [2*]
[1]*Post Graduate, Department of Gastroenterology, Kurnool Medical College and Govt General Hospital, Kurnool.*
[2*] *Professor and HOD, Department of Gastroenterology, Kurnool Medical College and Govt General Hospital, Kurnool.*
Corresponding Author: Dr.Shankara Sharma Bondalapati

Abstract: ***Introduction:*** *Acute pancreatitis (AP) is a common gastro intestinal emergency with an increasing incidence in India during the past decades. Because of its severity ranging from mild to severe or even fatal, or with systemic inflammatory conditions, AP continues to arouse the interest of clinical researchers.*
Materials and Methods: *Study was conducted from January 2018 to December 2018, 32 patients of HTG pancreatitis AP and 27 cases of biliary pancreatitis were enrolled into our hospital (Department of Gastroenterology, Kurnool Medical College and Govt General Hospital, Kurnool). The patients were identified using primary diagnosis through computer-based system. The data with medical charts were reviewed retrospectively and the relative clinical data were collected. The diagnosis of acute pancreatitis requires two of the these features: (1) abdominal pain consistent with acute pancreatitis (acute onset of a persistent, severe, epigastric pain often radiating to the back); (2) serum amylase activity at least three times greater than the upper limit of normal; and (3) characteristic findings of acute pancreatitis through contrast-enhanced computed tomography (CECT) or magnetic resonance imaging (MRI) or transabdominal ultrasonography. Serum triglyceride level more than 11.3 mmol/L (1000 mg/dL) and exclusion of other etiologies were recognized as the HTG etiology.*
Results: *In our research the patients in HTG pancreatitis groups were younger than biliary pancreatitis group (years versus years, Table 1). The ratio of male and female was close in HTG pancreatitis groups and biliary pancreatitis group (male 48.4% versus 48.8%, female 51.6% versus 51.6%). There were 2 patients (2/126, 1.6%) in HTP pancreatitis group and 4 patients in Biliary Pancreatitis Group (4/168, 2.4%) who underwent surgical drainage. There was no clear difference from the former reported research in these two types of AP. Maybe the reasons lied in the fact that biliary group had more serious infection and older age than HTG group. The sample of our research was not so big and our research is a retrospective research, so well designed prospective research is needed.*
Conclusion: *In conclusion, for the trend of the increasing proportion of HTG pancreatitis in total AP, we should pay more attention to the characteristics and treatment of HTG pancreatitis. The proportion of recurrent AP, the proportion of severe AP, and the comorbidity of DM were higher than biliary group. The proportion of the complications of GI bleeding, sepsis, and multiple organ dysfunction syndrome (MODS) in HTG group was less than biliary group. Apheresis could effectively reduce serum TG levels soon. There was no significance difference of the mortality between two groups. Also there is shortcoming of our research, the total sample is not big, and it is a retrospective research. The number of aphereses is not big (6 cases) and needs more research. We do not discuss the detail type of HTG.*
Key Words: *Acute pancreatitis, multiple organ dysfunction syndrome, Diabetes mellitus*

Date of Submission: 17-05-2019 Date of acceptance: 02-06-2019

I. Introduction

Acute pancreatitis (AP) is a common gastro intestinal emergency with an increasing incidence in India during the past decades. Because of its severity ranging from mild to severe or even fatal, or with systemic inflammatory conditions, AP continues to arouse the interest of clinical researchers.[1] Although AP in most patients is mild, moderate to severe as well as recurrent AP remains a tremendous burden on both public healthcare system and the patient's family. Of note, 10-20% of patients with AP developed severe disease with a mortality of up to 30%. The most common etiologies of AP are cholelithias is with obstruction of common bile duct or main pancreatic duct and alcohol abuse. Currently, a rapid increase in obesity nationwide and worldwide

is associated with an elevated incidence of metabolic diseases and changes in disease spectra. Hyperlipidemia, characterized by serum hypertriglyceridemia, has become the third leading cause of AP and contributed to 1-10% of patients with AP.[2] Moreover, mild to moderate hyperlipidemia can be regarded as an underlying phenomenon or comorbidity of pancreatitis. Previous cohort studies have indicated that serum triglyceride (TG) of ≥1000 mg/dL(11.3 mmol/L) is a high risk factor for patients with hypertriglyceridemic acute pancreatitis (HTGAP). Ithas also been considered necessary to diagnose HTGAP when the patient's fasting TG level is 5.65-11.3 mmol/L with chylous blood which occurs in about 20% of all patients with AP.[3] Clinical studies have demonstrated that HTGAP may contribute to increased severity and mortality, higher frequencies of comorbidities and systemic complications, longer length of hospitalization, and more frequent recurrence, than other subtypes of AP. Our emergency department has accepted many patients with AP and provided them with an early diagnosis and effective interventions. We thus conducted a retrospective study on AP patients who were admitted to our emergency department between 2012 and 2016, focusing on HTGAP and Biliary Pancreatitis.[4] Considering that notable dyslipidemias have a strong genetic component despite the important role that secondary dietary factors play in the clinical phenotype, we further per-formed gene mutation detection for 11 patients with HTGAP to determine the molecular genetic characteristics of this special subtype of AP.[5] In this study, we aimed to predict the severity and recurrence risk in HTGAP with the new-generation sequencing technology based upon clinical information of the patients.[6,7]

II. Materials And Methods

Between January 2018 to December 2018, 32 patients of HTG pancreatitis AP and 27 cases of biliary pancreatitis were enrolled into our hospital (Department of Gastroenterology, Kurnool Medical College and Govt General Hospital, Kurnool). The patients were identified using primary diagnosis through computer based system. The data with medical charts were reviewed retrospectively and the relative clinical data were collected. The diagnosis of acute pancreatitis requires two of the these features: (1) abdominal pain consistent with acute pancreatitis (acute onset of a persistent, severe, epigastric pain often radiating to the back); (2) serum amylase activity at least three times greater than the upper limit of normal; and (3) characteristic findings of acute pancreatitis through contrast-enhanced computed tomography (CECT) or magnetic resonance imaging (MRI) or transabdominal ultrasonography. Serum triglyceride level more than 11.3 mmol/L (1000 mg/dL) and exclusion of other etiologies were recognized as the HTG etiology.

The finding of stone in common bile duct through CT or Magnetic Resonance Cholangiopancreatography (MRCP) could help to define the biliary etiology of acute pancreatitis and also should exclude other etiologies of AP.

The severity of AP was classified into mild, moderate, or severe type according to the new Atlanta classification. Mild acute pancreatitis had no organ failure or local or systemic complications and usually resolves in the first week. Moderate type was defined by the presence of transient organ failure, local complications, or exacerbation of comorbid disease relieved in 24 h. Severe type was defined by persistent organ failure (>48 h). Local complications were peripancreatic fluid collections, pancreatic and peripancreatic necrosis, pseudocyst, and walled-off necrosis. Once admitted into hospital all patients were treated medically according to generally accepted principles including inhibiting oral intake, restoring fluid and electrolytes intravenously, and administration of prophylactic or treatment antibiotics. A proton pump inhibitor was given to inhibit gastric acid secretion and somatostatin to inhibit pancreatic excretion.

Serum triglyceride (TG) level was checked shortly after admission. If there was evidence of severe AP, pancreatic rest via deeper ligament of Treitz nasojejunal enteral feeding tube (preferred) or total parenteral nutrition (TPN) would be entertained.

Apheresis has been shown to remove TG from the serum of patients with HTG pancreatitis quickly. The premise of using apheresis is that it removes available TG in very low density lipoprotein (VLDL) and chylomicrons from serum and prevents generation of FFA which cause local or systemic effects. In most patients, TG levels decrease by ~65% and 85% after one or two sessions, respectively. There were 6 patients enrolled into apheresis treatment in HTG pancreatitis group and TG levels decreased more than 60% soon in our research. The candidates for apheresis were the patients with severe AP who continue to have TG levels above 1000 mg/dL after the first 24-48 hours.

Surgical intervention (necrosectomy with drainage or continuous postoperative lavage) was performed when infected pancreatic necrosis was clinically suspected or confirmed by positive bacteriologic results of CT-guided fine-needle aspirations. Endoscopic retrograde cholangiopancreatography (ERCP) was performed in 6 cases in biliary pancreatitis group. The indications included the following: (1) The patients had the clinical manifestation of cholangitis or biliary obstruction, (2) the patients' conditions aggravated, and (3) CT or MRCP showed that the stone of sludge exists in the common bile duct [4]. A papillotomy was done if stones and sludge were present in the common bile duct and some followed with endoscopic nasobiliary drainage.

Comorbidity was defined as a preexisting disease in addition to the acute pancreatitis. Comorbidity was diagnosed if the condition was an active problem and/or needs routine treatment prior to the onset of acute pancreatitis. The assessment of organ function and diagnosis of MODS was performed on the basis of the APACHEII multiple organ dysfunction score.

Statistical Analysis: Statistical evaluation was performed with SPSS 18.0 software. The significant differences of clinical characteristics of the HTG pancreatitis and biliary pancreatitis patients were tested with the $\chi 2$ test. The differences between the two groups were tested through independent samples-test. was considered statistically significant.

III. Results

Table 1: General clinical characteristics of patients with AP

Clinical Characteristic	HTG pancreatitis (N=32)	Biliary Pancreatitis (N=27)
Mean Age (Year, Range)	35.8±3.6	53±4.3
Male	15 (46.87)	13 (48.14)
Female	17 (53.125)	14 (51.85)
Medium Hospital stay in days (Range)	12.15±2.5	9.2±1.3
Surgical Drainage	3 (9.375)	5 (18.51)
Recurrence Rate	5 (15.625)	3 (11.11)
Hospital Deaths	2 (6.25)	2 (7.40)

Table 2: Comorbidity in acute pancreatitis

Comorbidity	HTG pancreatitis (N=32)	Biliary Pancreatitis (N=27)
Hypertension	4 (12.5%)	12 (44.44%)
Coronary Artery Disease	2 (6.25%)	10 (37.03%)
Diabetes Mellitus	13 (40.625%)	10 (37.03%)
Renal Insufficiency	-	9 (33.33%)
Cerebral vascular disease	-	8 (29.62%)

IV. Discussion

HTG is a metabolic disorder due to an elevated synthesis of TG-rich lipoproteins VLDL, a reduced catabolism of these particles, or a combination of both mechanisms. Normally, values of TG are below 2.2 mmol/L (200 mg/dL).[8] The role of HTG in causing AP is commonly accepted. According to the literature, HTG is the third cause for acute pancreatitis after gallstones and alcohol.[9] HTG is reported to account for more than 10% of all AP episodes. There is even some evidence that HTG pancreatitis is associated with a higher severity and a higher complication rate.[10]

Several mechanisms for the disease have been indicated including the hydrolysis of TGs forming free fatty acids inducing inflammation, chylomicrons inducing hyperviscosity leading to ischemia, and genetic predisposition.[11] In addition, cytokines seem to play an important role in acute pancreatitis including the systemic responses. It is generally accepted that TG levels of > 11.3 mmol/L (1000 mg/dL) trigger AP and induce serious complications. Therefore, rapid lowering of TG levels is a primary medical goal in preventing serious harm to the patient suffering from HTG.[12]

In our research the patients in HTG pancreatitis groups were younger than biliary pancreatitis group (years versus years, Table 1). The ratio of male and female was close in HTG pancreatitis groups and biliary pancreatitis group (male 48.4% versus 48.8%, female 51.6% versus 51.6%). There were 2 patients (2/126, 1.6%) in HTP pancreatitis group and 4 patients (4/168, 2.4%) in the Biliary Pancreatitis Group who underwent surgical drainage. There was no clear difference from the former reported research in these two types of AP.[13] Maybe the reasons lied in the fact that biliary group had more serious infection and older age than HTG group. The sample of our research was not so big and our research is a retrospective research, so well designed prospective research is needed.

The medium hospital stay of HTG pancreatitis groups was significantly longer than biliary pancreatitis group (days versus days). The reason was that the ratio of severe AP in HTG pancreatitis was higher than the biliary pancreatitis group (31.0% versus 26.2%).[14]

The recurrent proportion in HTG pancreatitis in our research was 15.625% (5/32) and higher than biliary group (11.11%, 3/27). After detailed disease history inquiry, all the 5 cases of recurrent AP in HTG pancreatitis group have improvement. Ten patients had stopped the lipid lowering drugs and all the others were high fat diet.[15]

V. Conclusion

In conclusion, for the trend of the increasing proportion of HTG pancreatitis in total AP, we should pay more attention to the characteristics and treatment of HTG pancreatitis. The proportion of recurrent AP, the proportion of severe AP, and the comorbidity of DM were higher HTG Group than biliary group. The proportion of the complications of GI bleeding, sepsis, and multiple organ dysfunction syndrome (MODS) in HTG group was less than biliary group. Apheresis could effectively reduce serum TG levels soon. There was no significance difference of the mortality between two groups. Also there is shortcoming of our research, the total sample is not big, and it is a retrospective research. The number of aphereses is not big (6 cases) and needs more research. We do not discuss the detail type of HTG.

References

[1]. W. Uhl, A. Warshaw, C. Imrie et al., "IAP guidelines for the surgical management of acute pancreatitis," Pancreatology, vol. 2, no. 6, pp. 565–573, 2002.
[2]. M. Arvanitakis, M. Delhaye, V. De Maertelaere et al., "Computed tomography and magnetic resonance imaging in the assessment of acute pancreatitis," Gastroenterology, vol. 126, no. 3, pp. 715–723, 2004.
[3]. A. V. Kyriakidis, P. Karydakis, N. Neofytou et al., "Plasmapheresis in the management of acute severe hyperlipidemic pancreatitis: report of 5 cases," Pancreatology, vol. 5, no. 2-3, pp. 201–204, 2005.
[4]. J. C. Marshall, D. J. Cook, N. V. Christou, G. R. Bernard, C. L. Sprung, and W. J. Sibbald, "Multiple organ dysfunction score: a reliable descriptor of a complex clinical outcome," Critical Care Medicine, vol. 23, no. 10, pp. 1638–1652, 1995.
[5]. S. J. D. O'Keefe, "A guide to enteral access procedures and enteral nutrition," Nature Reviews Gastroenterology and Hepatology, vol. 6, no. 4, pp. 207–215, 2009.
[6]. F. Yi, L. Ge, J. Zhao et al., "Meta-analysis: total parenteral nutrition versus total enteral nutrition in predicted severe acute pancreatitis," Internal Medicine, vol. 51, no. 6, pp. 523–530, 2012.
[7]. H. Al-Humoud, E. Alhumoud, and N. Al-Hilali, "Therapeutic plasma exchange for acute hyperlipidemic pancreatitis: a case series," Therapeutic Apheresis and Dialysis, vol. 12, no. 3, pp. 202–204, 2008.
[8]. J.-H. Chen, J.-H. Yeh, H.-W. Lai, and C.-S. Liao, "Therapeutic plasma exchange in patients with hyperlipidemic pancreatitis," World Journal of Gastroenterology, vol. 10, no. 15, pp. 2272–2274, 2004.
[9]. J. Gubenšek, J. Buturović-Ponikvar, A. Marn-Pernat et al., "Treatment of hyperlipidemic acute pancreatitis with plasma exchange: a single-center experience," Therapeutic Apheresis and Dialysis, vol. 13, no. 4, pp. 314–317, 2009.
[10]. J.-H. Yeh, J.-H. Chen, and H.-C. Chiu, "Plasmapheresis for hyperlipidemic pancreatitis," Journal of Clinical Apheresis, vol. 18, no. 4, pp. 181–185, 2003.
[11]. R. S. Kohli, W. Bleibel, A. Shetty, and U. Dhanjal, "Plasmapheresis in the treatment of hypertriglyceridemic pancreatitis with ARDS," Digestive Diseases and Sciences, vol. 51, no. 12, pp. 2287–2291, 2006.
[12]. M. A. Dettelbach, L. J. Deftos, and A. F. Stewart, "Intraperitoneal free fatty acids induce severe hypocalcemia in rats: a model for the hypocalcemia of pancreatitis," Journal of Bone and Mineral Research, vol. 5, no. 12, pp. 1249–1255, 1990.
[13]. S. Domschke, P. Malfertheiner, W. Uhl, M. Büchler, and W. Domschke, "Free fatty acids in serum of patients with acute necrotizing or edematous pancreatitis," International Journal of Pancreatology, vol. 13, no. 2, pp. 105–110, 1993.
[14]. L. Berglund, J. D. Brunzell, A. C. Goldberg et al., "Evaluation and treatment of hypertriglyceridemia: an endocrine society clinical practice guideline," Journal of Clinical Endocrinology and Metabolism, vol. 97, no. 9, pp. 2969–2989, 2012.
[15]. J. E. Lambert and E. J. Parks, "Postprandial metabolism of meal triglyceride in humans," Biochimica et Biophysica Acta, vol. 1821, no. 5, pp. 721–726, 2012.

Dr. Kranthi Kumar. "A Retrospective, Comparative Study Of Characteristics Of Hypertriglyceridemic Pancreatitis And Biliary Pancreatitis In A Tertiary Care Hospital, Kurnool, Ap." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 18, no. 5, 2019, pp 47-50.