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Original Article

Clinical Course of Acute Pancreatitis in Chronic Kidney Disease Patients in a Single Kidney Center (PGTi) in Karachi

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Abstract

Introduction: The aim of this study was to assess the clinical course, etiology and complications of acute pancreatitis among chronic kidney disease (CKD) patients in a tertiary care renal center in Karachi.

Methods: We retrospectively evaluated the clinical course of CKD patients who presented to our emergency room with signs and symptoms of acute pancreatitis over a period of two years.

Results: During the study period, 247 CKD patients presented to our emergency room with symptoms suggestive of acute pancreatitis. Only 43 patients (17.4%) had more than a threefold increase in serum amylase and/or lipase levels fulfilling the diagnostic criteria of acute pancreatitis. They included 25 pre-dialysis CKD patients (58.13%) and 18 end stage renal failure patients (41.86%) on regular hemodialysis (HD). Among the 25 pre-dialysis CKD patients, 17 patients developed acute kidney injury (AKI), ten of whom required temporary HD. Twelve of those patients (70%) returned back to their baseline renal functions after 3-4 weeks. Gallstones were the cause of pancreatitis in seven patients (16.3%) while no cause was identified in 29 patients (67.4%). Nine patients (20.9%) developed multi-organ failure and 12 patients (27.9%) required admission to the intensive care unit (ICU). All patients survived except for one patient (2.3%) who died in the ICU. patients with less than threefold increase in serum amylase and lipase levels responded well to conservative management and had a favorable clinical course.

Conclusion: In severe acute pancreatitis the mortality rate can be as high as 40-58% especially in association with comorbid conditions. In this series of CKD patients however, the overall mortality rate was 2.3%, probably due to the predominance of milder forms of pancreatitis.

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Introduction

Acute pancreatitis is a condition characterized by abdominal pain, nausea and vomiting with elevation of pancreatic enzymes in serum [1]. The disease occurs with similar frequency among various age groups, but the cause of the condition and the likelihood of death vary according to age, gender, race and other risk factors. The most important risk factors in adults are gallstones and excessive alcohol usage [1, 2]. Other causes of pancreatitis are metabolic derangements (e.g. hypertriglyceridemia, hypercalcemia), viral infection, medications (e.g. azathioprine and thiazides), pancreatic duct obstruction (e.g. tumors) and trauma. In children, systemic diseases are a common cause. In 20% of cases no cause is found and these cases are labeled as idiopathic [1].

Severe acute pancreatitis is associated with systemic complications like organ dysfunction and local complications like necrosis, abscess and pseudo cyst formation. Mild acute pancreatitis is associated with minimal organ dysfunction and lacks the features of severe acute pancreatitis [1, 3]. The pathogenesis of pancreatitis is due to inappropriate activation of the pro-enzyme trypsinogen into the activated enzyme trypsin as well as the formation of highly toxic lysolecithin. This results in pancreatic injury and inflammation, which can extend beyond the organ itself causing multiple organ failure and death. The diagnosis depends on typical clinical features together with a three-fold elevation of pancreatic enzymes. Amylase once elevated returns to normal in about 48 to 72 hours. However, increased amylase level is found in certain non pancreatic conditions like visceral perforation, small bowel ischemia or obstruction and ectopic pregnancy. As lipase is predominately secreted by the pancreas it is more superior and sensitive in

Table 1: Ultrasound scan findings at presentation of CKD patients ultimately diagnosed to have acute pancreatitis

Ultrasound findings at presentation	Number of patients (%)
Normal ultrasound	29 (67.4%)
Gall stones	7 (16.27%)
Multiple polyps in gall bladder	4 (9.3%)
Edematous wall of gall bladder	3 (7%)

diagnosing pancreatitis as compared to amylase [4]. Abdominal imaging techniques that are helpful in confirming acute pancreatitis or excluding other intra abdominal conditions include ultrasound, CT scan and MRI. Endoscopic ultrasonography and ERCP are more accurate tests for diagnosing and ruling out biliary causes of pancreatitis. The aim of this study was to assess the etiological factors, severity and the outcome of acute pancreatitis in chronic kidney disease (CKD) patients.

Methods

We retrospectively reviewed the medical records of all CKD patients who presented to the Emergency Room of the Kidney Center (PGTi), Karachi, Pakistan, between January 2006 and April 2008 with signs and symptoms suggestive of acute pancreatitis, irrespective of age, gender, and comorbidity. Data was collected through a data collecting form which included age, gender, symptoms at presentation, number of days the patient was kept on nil per oral, the highest amylase and lipase levels, need for admission to the intensive care unit (ICU), number of days spent in the ICU, risk factors for development of acute pancreatitis, local and systemic complications, ultrasound scan findings, amount of fluid given, requirement of hemodialysis (HD), analgesics and antibiotics used. The study included patients in various CKD stages. All patients presenting with symptoms and signs of acute pancreatitis had the following baseline investigations: complete blood picture, urea, creatinine, electrolytes, calcium, glucose, albumin serum amylase, lipase levels and abdominal ultrasound scan. Abdominal CT scan was not done due to the risk of inducing contrast induced nephropathy in CKD patients. Chronic dialysis was defined as regular thrice weekly HD for more than six weeks duration. End stage renal failure (ESRF) was defined as requirement of chronic dialysis. Acute pancreatitis was defined as an episode of acute abdominal pain and discomfort with more than threefold increase in serum amylase and/or lipase level.

Statistical package for social science (SPSS) version 17.0 was used for data analysis. Results are presented as mean ± standard deviation for quantitative variables and percentages for qualitative variables.

Results

A total of 247 CKD patients presented to our emergency room during the study period with symptoms suggestive of acute pancreatitis. They included 140 males and 107 females, with a mean age of 34 ± 6 years (range 18-75). Only 43/247 patients (17.4%) fulfilled the diagnostic criteria of acute pancreatitis with more than threefold increase in serum amylase/lipase levels. The presenting complaints of patients ultimately diagnosed to have acute pancreatitis are shown in Figure-1. Among those 43 patients diagnosed as acute pancreatitis, 23 were males (53.5%) and 20 were females (46.5%). Twenty-three patients were diabetic (53.4%), nine patients were hypertensive (20.9%) and two patients were alcoholic (4.6%). Eighteen patients had ESRF and were maintained on regular HD while 25 patients had pre-dialysis CKD. Among the 25 pre-dialysis CKD patients diagnosed with acute pancreatitis, 17 (68%) developed acute kidney injury (AKI) as a complication of acute pancreatitis; ten of whom required temporary HD while seven patients were managed conservatively. Among the 17 patients who developed AKI, 12 patients (70%) returned to their baseline kidney function over a period of 3-4 weeks, and all patients who required dialysis regained dialysis independence.

Table-1 shows the ultrasound findings at presentation of CKD patients diagnosed with acute pancreatitis. Thirty patients (69.8%) required various forms of analgesics while 13 patients (30.2%) had mild symptoms and required no analgesics. Thirty-four patients (79%) were given empirical antibiotics. Twenty-six patients (60.5%) developed different systemic complications during their hospital stay (Figure-2). Twelve patients (27.9%) required ICU admission, three of whom remained in the ICU for more than a month. All patients survived except for one patient (2.3%) who developed severe multi organ failure and died in the ICU. Patients with less than threefold increase in serum amylase and lipase level were treated conservatively; resolution of symptoms and signs with

Figure 1: Symptoms at presentation to the emergency room of CKD patients ultimately diagnosed to have acute pancreatitis

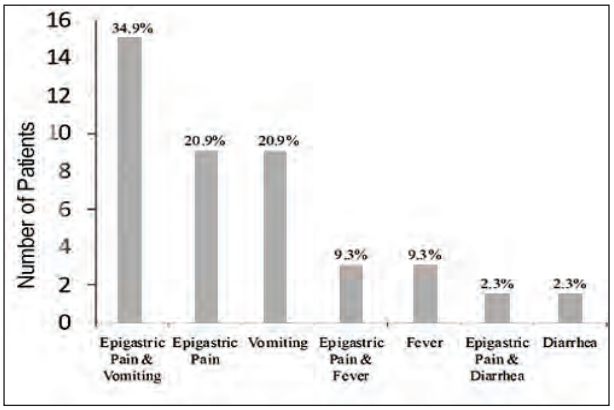
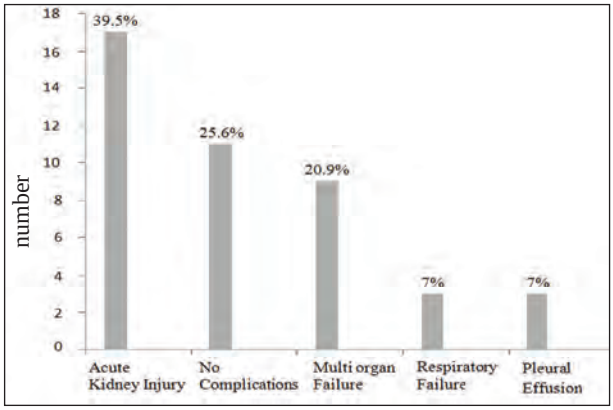


Figure 2: Systemic complications of acute pancreatitis among the studied CKD patients



normalization of enzyme level was observed in all of them.

Discussion

An apparent increase in the incidence of acute pancreatitis was noticed in the past 40 years in different parts of the world. For example, the incidence in Scotland has risen from 9.4 per 100,000 per year in 1968-1980 to 41.9 per 100 000 per year in 1995 [5]. There is a likelihood of increasing incidence of acute pancreatitis in our part of the world, though the exact incidence is not known. Gallstones were the commonest cause of acute pancreatitis in our study, which is in accordance to different studies from other parts of the world [1]. The mortality in patients with acute pancreatitis is associated with the number of failing organs and the severity and reversibility of the organ dysfunction. In our study, 17 out of 43 CKD patients developed acute kidney injury; ten of whom required temporary HD. Seventy percent of those patients eventually recovered their baseline kidney function. Early diagnosis and proper management is vital in this setting.

Elevated serum pancreatic enzymes (amylase and lipase) are the main tools of diagnosing acute pancreatitis. In patients with renal insufficiency, concentration of gastrointestinal hormones such as cholecystokinin, serum gastric inhibitory peptide, and glucagons are significantly increased in relation to degree of renal impairment. During long term HD these increased hormone concentration do not return to normal. This increase in hormone concentrations causes hypersecretion of pancreatic enzyme, predominantly trypsin. In the absence of acute pancreatitis, plasma concentration of amylase and lipase are frequently increased in uremic patients and patients on chronic dialysis, both HD and peritoneal dialysis. A greater than threefold increase in serum amylase and lipase activity together with acute onset abdominal pain are generally considered diagnostic of acute pancreatitis in CKD patients [6]. Among the 247 CKD patients included in this study, only 43 (17.4%) patients fulfilled this criterion. Another group of patients had less than threefold increase in serum enzyme level although they presented with typical clinical features of acute pancreatitis. Their signs and symptoms subsided and enzymes level normalized with conservative treatment. It is possible that they had a mild form of pancreatitis or other gastrointestinal conditions that responded to conservative management.

The prevalence of acute renal failure alone in severe acute pancreatitis is found to be between 5-20%, while the incidence of acute renal failure along with other complications like respiratory failure, sepsis, and pleural effusion is reported between 14-28% in different studies from various parts of the world [7-9]. In our study the incidence of acute renal failure alone was 25.6% while 20.9% of patients developed multiple complications including acute renal failure. This is in accordance with other studies [3, 7-9]. The rationale for antibiotic prophylaxis in acute pancreatitis is based on the fact that mortality for infected pancreatic necrosis is higher than that for sterile necrosis. Randomized controlled trials have shown positive effects for the prevention of infected pancreatic necrosis and even a reduction in mortality, but these effects were not confirmed in all studies [10]. In our study, antibiotics were used empirically in 80% of cases. Chronic diseases, local complications and the presence of organ failure significantly increase the mortality of acute pancreatitis. In this study, 60.5% of patients developed systemic complications, but the overall mortality was only 2.3%. This figure is low compared to a reported mortality rate of 40 – 58% in other studies [11-13], and

may have resulted from the predominance of milder forms of pancreatitis.

Conclusion

The etiology of acute pancreatitis in this group of patients is in accordance to that described in other studies. Despite high morbidity, mortality rate was lower than described in other studies, which is probably due to the predominance of milder forms of pancreatitis.

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