RESEARCH ARTICLE

Analysis of patients with acute pancreatitis in chronic renal failure under haemodialysis

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Abstract

Objective: To evaluate acute pancreatitis in patients with end-stage renal disease on haemodialysis, and to compare it with patients without end-stage renal disease.

Method: The retrospective, cross-sectional study was conducted at Adana City Training and Research Hospital, Turkiye, and comprised data from September 2017 to December 2023 of patients presenting with acute pancreatitis with or without end-stage renal disease. Those with end-stage renal disease who were on haemodialysis were placed in group A, while patients without chronic kidney disease represented control group B. Demographic and laboratory data, the severity of pancreatitis, the aetiology of acute pancreatitis, accompanying diseases, the duration of hospitalisation and the prognosis of the patients in both groups were evaluated and compared. Data was analysed using SPSS 25.

Results: Of the 178 patients, 54(30.33%) were in group A with mean age 56.0±14.5 years (range: 20-85 years) among whom 26(48.1%) were males. There were 124(69.66%) patients in group B with mean age 52.5±16.2 years (19-88 years), and there were 59(47.6%) males. Acute pancreatitis was more severe, and the number of idiopathic cases was higher in group A compared to group B ($p<0.05$). Significant differences were also noted for aetiological causes, comorbidities, intensive care unit admission, length of hospital stays, and mortality between the groups($p<0.05$).

Conclusion: In patients with end-stage renal disease on haemodialysis, acute pancreatitis was more severe in all respects compared to those without chronic kidney disease.

Keywords: Acute pancreatitis, Chronic renal failure, Haemodialysis, Survival. (JPMA 76: 167; 2026)

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Introduction

In clinical practice of gastroenterology, acute pancreatitis (AP) is a frequently encountered disease that requires emergency hospitalisation.[1] Besides, there are studies reporting increased incidence of AP in end stage renal disease (ESRD) cases on dialysis, especially peritoneal dialysis.[2-6] However, studies about the prevalence of AP in chronic renal failure on haemodialysis (HD) are inconclusive.[3-5,7,8] There are limited studies on the prognosis of AP in ESRD on routine HD.[9]

AP must be kept in mind in patients who have chronic renal failure on dialysis treatment, presenting with acute abdominal pain.[10] However, in patients with renal failure, increased levels of amylase and lipase are observed in more than half of the patients. Elevated amylase and lipase levels may also result from diseases other than AP.[11] Therefore, a correct interpretation of high amylase and lipase levels in patients with ESRD is crucial.

In patients undergoing dialysis, early diagnosis, timely and appropriate treatment, and accurate prognosis determination for AP are critical.[12] AP in the context of chronic renal failure is more complex.[13]

The current study was planned to evaluate AP in patients with ESRD receiving routine HD, and to compare their outcomes with AP patients without chronic kidney disease (CKD).

Materials and Methods

The retrospective, cross-sectional study was conducted at Adana City Training and Research Hospital, Turkiye, and comprised data from September 2017 to December 2023 of adult patients presenting with AP with or without ESRD. Those with ESRD who were on HD were placed in group A, while patients without CKD represented control group B. After approval from the ethics review committee of the University of Health Sciences, Turkiye, the sample size was calculated using G*Power version 3.1.9.7.[14] Given the lack of similar studies, the sample size was calculated for alpha (α) 0.05, effect size 0.55 and power 0.95. Group A comprised data of all patients meeting the inclusion criteria, while group B was raised using simple random sampling technique. Patients with lack of data, those under peritoneal dialysis, and patients having chronic renal diseases but not on HD were excluded, and so were those with chronic pancreatitis and patients with malignancy.

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Diagnosis of AP was made if two out of three of the following criteria exist: abdominal pain (typical pain of AP is epigastric pain, spreading to back), increased amylase and lipase levels (>3 times the upper limit of normal value), and findings consistent with AP on computed tomography (CT), magnetic resonance imaging (MRI) or transabdominal ultrasonography.[15]

All AP patients were classified into three severity groups as per the of the Atlanta guidelines.[15] AP severity was considered mild if no signs of organ failure, and no local or systemic complications existed. The severity was moderate if transient organ failure (relieving in 48 hours) and/or local or systemic complications without persistent organ failure (>48 hours) existed. AP was considered severe if persistent one or more organ failure existed. Besides, Ranson score was calculated in group B.[16]

In addition, data was noted regarding patients' age, gender, laboratory details, aetiology of pancreatitis, accompanying diseases, duration of hospitalisation (inwards or intensive care unit [ICU]), prognosis, time to first attack from the beginning of HD, and patients' first hospitalisation for AP.

Data was analysed using SPSS 25. Continuous variables were expressed as mean±standard deviation (SD) or median with interquartile range (IQR), while categorical variables were expressed as frequencies and percentages. Distribution of variables were analysed with Kolmogorov-Smirnov test. Chi-square test or Fisher exact test was used to compare categorical variables. Continuous variables were compared using student t test if the variables were distributed normally, or Mann Whitney U test if the variables were not distributed normally. P<0.05 was considered significant.

Results

Of the 178 patients, 54(30.33%) were in group A with mean age 56.0±14.5 years (range: 20-85 years) among whom 26(48.1%) were males. There were 124(69.66%) patients in group B with mean age 52.5±16.2 years (19-88 years), and there were 59(47.6%) males. In group A, 46(85.2%) patients had a single pancreatitis attack, 5(9.3%) had two attacks, and 3(5.6%) had three AP attacks. In group B, 95(76.6%) patients had a single attack, 21(14.6%) had two attacks, 5(4%) had three attack, and 3(2.4%) had four attacks. There was no significant difference between the groups (p=0.338). AP was more severe, and the number of idiopathic cases was higher in group A compared to group B (p<0.05).

The frequency of diabetes mellitus (DM), hypertension (HT), coronary artery disease (CAD) and congestive heart failure (CHF) was significantly higher in group A compared to group B (p<0.05).

Table: Demographic data, severity of pancreatitis, aetiology of pancreatitis, accompanying diseases, hospitalisation duration and prognosis of patients in the study and control groups.

	Study Group (n=54) (n(%))	**Control group** (n=124) (n(%)	***p*-value**
Mean Age (years)	56.0±14.5 (20-85)	52.5±16.2 (19-88)	0.019**
Gender (Male)	26 (48.1)	59 (47.6)	0.944*
Severity (Atlanta)			
Mild	46 (85.2)	117(94.4)	0.050*
Moderate	6 (11.1)	3 (2.4)	
Severe	2 (3.7)	4 (3.2)	
Aetiology			
Biliary	26 (48.1)	85 (68.5)	0.005*
Hyperlipidaemia	6 (11.1)	19 (15.3)	
Other	5 (9.3)	4 (3.2)	
Idiopathic	17 (31.5)	16 (12.9)	
Accompanying Diseases			
DM	24 (44.4)	27 (21.8)	0.002*
HT	35 (64.8)	24 (19.4)	<0.001*
CAD	15 (27.8)	13 (10.5)	0.004*
CHF	4 (7.4)	2 (1.6)	0.049*
ICU hospitalization	15 (27.8)	8 (6.5)	<0.001**
Duration of ICU (days)	6±8.2 (1-30)	5.5±7.3 (1-23)	0.678**
Duration of total Hospital stay (days)	6±7.7 (1-40)	4±5.4 (1-40)	<0.001**
Prognosis			
Dead	3 (5.6)	1 (0.8)	0.049*
Alive	51 (94.4%)	123 (99.2%)	

*Chi-Square Test; ** Mann Whitney U test; DM: Diabetes mellitus, HT: Hypertension, CAD: Coronary artery disease, CHF: Congestive heart failure, ICU: Intensive care unit, ESRD: End-stage renal disease, HD: Haemodialysis.

Median time from the beginning of HD to first attack of AP in group A was 36 months (IQR: 58).

In group A, 15(27.8%) patients needed ICU admission compared to 8(6.5%) in group B (p<0.001), but the duration of ICU stay was not significant between the groups (p>0.05). Total length of hospitalisation in group A was significantly higher compared to group B (p<0.001).

In group A, 7(13%) patients and 19(15.3%) in group B needed endoscopic retrograde cholangiopancreatography (ERCP) (p=0.682).

In group A, 3(%) patients died compared to 1(%) death in group period (p<0.05) (Table).

Discussions

The current evaluated patients with ESRD under HD who presented with AP. There are limited studies regarding the incidence, aetiology and clinical course of AP in such patients.[3-6]

The pathogenesis of AP in ESRD is not fully unknown.[8,17] However, several theories have been proposed. Gastrointestinal hormones (i.e. gastrin, cholecystokinin, glucagon) are mainly metabolised and excreted by the kidney, and in chronic renal failure, these hormones' levels are increased, resulting in increased secretion of pancreatin and leading to over-secretion of pancreatic enzymes, disorders in pancreatic function and damages. The ischaemia-reperfusion process due to hypotension during HD may contribute to pancreatic injury. Contrast agents may lead to pancreatic ischaemia and necrosis by increasing plasma viscosity and low blood flow.[8] Use of contrast agents may cause necrotising pancreatitis in chronic patients under HD. The development of hypercalcaemia in patients with chronic renal disease can cause pancreatic vascular sclerosis, affecting local blood flow to the pancreas, causing pancreatic ischaemia and necrosis.[8,17]

In the general population, aetiologies of pancreatitis are generally biliary stones, alcohol, and hyperlipidaemia.[1] In the current study, there was significant difference according to aetiological factors between the groups (p<0.05). The most common observed aetiological factors were biliary disease in group A. Idiopathic AP in group A was also higher than group B. In some studies, HD is a risk factor for developing AP,8 but development of AP may not be related to a single factor. The aetiology of AP in patients with ESRD may differ from patients without ESRD.[13] Idiopathic pancreatitis may be associated with the cumulative effect of various pathogenic factors in patients with CKD, including renal insufficiency, uraemia, hypercalcaemia in conjunction with secondary hyperparathyroidism, hyperlipidaemia, and drug use, in addition to being related to dialysis.[7,8,10] These factors may have a role in patients with ESRD in the current study.

In patients with renal failure, accompanying comorbid conditions, such as DM, HT, and CAD, are common.[3,18] In the current, the frequency of accompanying diseases, like DM, HT, CAD and CHF, in group A were higher than patients in group B (p<0.05). Studies showed that DM was a precipitating factor for pancreatitis, and DM worsened prognosis in patients with renal failure.[5,19] DM may have been a precipitating factor for the current patients, worsening the prognosis. Comorbid diseases may lead to a higher ICU hospitalisation rate, longer duration of hospital stay, more severe disease, and increased mortality.

There are a few studies regarding mean time from the beginning of HD to the first attack of pancreatitis.[5,7] Lankisch et al. reported mean time interval between first HD and first attack of pancreatitis as 21 months.[7] In the current study, the median time interval from first HD to first attack of pancreatitis was 36 months (IQR:58).

Diagnosis of AP in chronic renal failure is complex. In these patients, abdominal pain, nausea and vomiting are the most common symptoms, while abdominal tenderness and decreased bowel sounds are frequent physical examination findings.[7,20,21] Symptoms and signs of AP in ESRD are similar to non-ESRD.[20,22] Increase in serum amylase and lipase levels is the main laboratory parameter in AP diagnosis. However, renal failure is one of leading causes of elevated amylase and lipase levels, and, therefore, correct interpretation of elevated amylase and lipase levels in ESRD is very important.[11,12] About 60-80% of patients with renal failure may show increased amylase and lipase levels up to three times of the upper limit of normal levels.[11] Besides, since many diseases, such as infection, salivary gland disease, malignancy and shock, may increase amylase and lipase levels, it may not be easy to diagnose AP.[11,12] Serial enzyme follow-up and selection of proper imaging modalities are important.[11] Ultrasonography alone may not be enough to detect the signs of AP. Use of CT and MRI may be more suitable. But in patients with renal failure, contrast agents are less frequently used than patients without renal failure.[10] In patients with renal failure, late admission to hospital and/or failure to use appropriate imaging method may cause the diagnosis to be missed in these patients.[4,10,15] In patients with sudden abdominal pain whom amylase and lipase levels are higher than three times of the upper limit of normal, AP must be considered at first.[4] Besides, in patients healed by conservative therapy but still having elevated amylase and lipase levels lower than three times of the upper limit of normal, mild AP may be considered rather than hyperamylasaemia associated with renal failure.[1,12]

The treatment of pancreatitis in CKD is similar to that of patients without end-stage renal failure.[22] However, appropriate hydration and analgesia, along with antibiotic therapy, when necessary, are frequently employed treatment methods. Close monitoring of patients for volume overload, and adjusting analgesia and antibiotic doses in accordance with renal function is crucial. In patients with concomitant choledocholithiasis, ERCP may be needed. In the current study 13% patients in group A and 15.3% in group B needed ERCP (p=0.682). No complication related to ERCP in patients with ESRD was observed.

Limited data exists regarding the effects of ESRD on AP. Most patients tend to have mild AP[7,12] but clinical course of AP is not always predicted even in mild cases. Improvements in the treatment of AP and intensive care exist, but severe pancreatitis remains a potentially lethal disease with high mortality rate.[23,24] Prognosis depends on the development of organ failure or pancreatic or peripancreatic necrosis-related secondary infections1. In the current study, most patients in group A had low Atlanta scores, and low Atlanta and Ranson scores (1.2±1.2) were observed in group B, too. There was significant difference between the groups for Atlanta scores (p=0.05).

The initial assessment of AP severity in ESRD patients undergoing HD can be useful in predicting and preventing complications and mortality as well as in decision related to patients' follow-up in either ICU or the ward. The AP prognosis is often predicted by using, scores such as Atlanta, Ranson, Glaskow-Imrie, Bedside Index of Severity in Acute Pancreatitis Score (BISAPS) and Marshall scores.[25,26] Since calculation of these scores depends on urea and creatinine levels, they are not suitable for use in patients with chronic renal failure. Thus, Ranson scores were noted in group B alone in the current study. In some studies, Red cell distribution with (RDW) has been shown as a prognostic parameter in AP.[27-29] In the current study, there were higher RDW values in patients with ESRD than in patients without ESRD (p<0.05). To determine prognosis in patients with ESRD under HD, new scoring systems and new parameters are needed.

In the present study, ICU admission, and total duration of hospital stay were higher in group A(p<0.001). However, there was no significant difference in the length of ICU hospitalisation between the groups.

A recent study reported that in CKD patients with AP had more hospital admissions, higher hospital costs, and increased mortality than the general population.[3,9,13] A study reported that mortality of AP in patients with ESRD increased almost twofold (p<0.01).[9] In the current study, three patients in group A died; one had postsurgical issues (Whipple operation), one had biliary AP, and the last one had idiopathic AP. In group B, only one patient died of AP caused by hyperlipidaemia (p<0.05). Since one patient had undergone Whipple operation, mortality per se may not be related to AP. Mortality and morbidity in AP have not been studied systematically in patients with ESRD.[9,13] Studies including more patients may reveal the difference accurately.

The current study has limitations as it was conducted at a single centre, had a retrospective design, and the habits of the patients (alcohol intake, etc.) were not explored.

Conclusion

The most common causes of AP in patients with ESRD under HD were biliary disease, and most of them had mild AP. Renal disease and accompanying diseases may trigger and affect AP prognosis in these patients. In the current group A, the rate of ICU hospitalisation, and total length of hospitalization stay were higher than group B. Mortality rates were higher in group A patients due to AP severity and the presence of comorbid conditions.

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References

1. Boxhoorn L, Voermans RP, Bouwense SA, Bruno MJ, Verdonk RC, Boermeester MA, et al. Acute pancreatitis. Lancet. 2020;396:726-34. doi:10.1016/S0140-6736(20)31310-6
2. Avram MM. High prevalence of pancreatic disease in chronic renal failure. Nephron. 1977;18:68-71. doi:10.1159/000180768
3. Hou SW, Lee YK, Hsu CY, Lee CC, Su YC. Increased risk of acute pancreatitis in patients with chronic hemodialysis: a 4-year follow-up study. PLoS One. 2013;8:e71801. doi:10.1371/journal.pone.0071801
4. Bruno MJ, van Westerloo DJ, van Dorp WT, Dekker W, Ferwerda J, Tytgat GN, et al. Acute pancreatitis in peritoneal dialysis and haemodialysis: risk, clinical course, outcome, and possible aetiology. Gut. 2000;46:385-9. doi:10.1136/gut.46.3.385
5. Chen HJ, Wang JJ, Tsay WI, Her SH, Lin CH, Chien CC. Epidemiology and outcome of acute pancreatitis in end-stage renal disease dialysis patients: a 10-year national cohort study. Nephrol Dial Transplant. 2017;32:1731-6. doi:10.1093/ndt/gfw400
6. Rutsky EA, Robards M, Van Dyke JA, Rostand SG. Acute pancreatitis in patients with end-stage renal disease without transplantation. Arch Intern Med. 1986;146:1741-5.
7. Lankisch PG, Weber-Dany B, Maisonneuve P, Lowenfels AB. Frequency and severity of acute pancreatitis in chronic dialysis patients. Nephrol Dial Transplant. 2008;23:1401-5. doi:10.1093/ndt/gfm769
8. Wang H, Rong J, Song C, Zhao Q, Zhao R, Xie Y, et al. Hemodialysis and risk of acute pancreatitis: a systematic review and meta-analysis. Pancreatology. 2021;21:89-94. doi:10.1016/j.pan.2020.11.004
9. Cheungpasitporn W, Thongprayoon C, Ungprasert P, Wijarnpreecha K, Raimondo M, Kroner PT. Acute pancreatitis in end-stage renal disease patients in the USA: a nationwide, propensity score-matched analysis. Eur J Gastroenterol Hepatol. 2019;31:968-72. doi:10.1097/MEG.0000000000001449
10. Van Dyke JA, Rutsky EA, Stanley RJ. Acute pancreatitis associated with end-stage renal disease. Radiology. 1986;160:403-5. doi:10.1148/ radiology.160.2.3726119
11. Aatif T, AR. Pancreatic enzymes in end-stage renal disease patients on maintenance hemodialysis. Indian J Med Biochem. 2019;23:343-6.
12. Nasir K, Ahamd A. Clinical course of acute pancreatitis in chronic kidney disease patients in a single kidney center (PGTi) in Karachi. Arab J Nephrol Transplant. 2012;5:87-90.
13. Kroner PT, Mareth K, Raimondo M, Lee DD, Alsaad A, Aslam N, et al. Acute pancreatitis in advanced chronic kidney disease and kidney transplant recipients: results of a US nationwide analysis. Mayo Clin Proc Innov Qual Outcomes. 2019;3:160-8. doi:10.1016/j.mayocpiqo.2019.03.006
14. Faul F, Erdfelder E, Lang AG, Buchner A. G*Power 3: a flexible statistical power analysis program for the social, behavioral, and biomedical sciences. Behav Res Methods. 2007;39:175-91. doi:10.3758/bf0319 3146
15. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, et al. Classification of acute pancreatitis—2012: revision of the Atlanta classification and definitions by international consensus. Gut.

2013;62:102-11. doi:10.1136/gutjnl-2012-302779
16. Basit H, Ruan GJ, Mukherjee S. Ranson criteria. StatPearls. Treasure Island (FL): StatPearls Publishing; 2024.
17. Kishino T, Nakamura K, Mori H, Yamaguchi Y, Takahashi S, Ishida H, et al. Acute pancreatitis during haemodialysis. Nephrol Dial Transplant. 2005;20:2012-3. doi:10.1093/ndt/gfh949
18. Tonelli M, Wiebe N, Guthrie B, James MT, Quan H, Fortin M, et al. Comorbidity as a driver of adverse outcomes in people with chronic kidney disease. Kidney Int. 2015;88:859-66. doi:10.1038/ki.2015.228
19. Shen HN, Lu CL, Li CY. Epidemiology of first-attack acute pancreatitis in Taiwan from 2000 through 2009: a nationwide population-based study. Pancreas. 2012;41:696-702. doi:10.1097/MPA.0b013e 31823 db941
20. Golay V, Roychowdhary A. Acute pancreatitis in chronic kidney disease—a common but often misunderstood combination. Ren Fail. 2012;34:1338-40. doi:10.3109/0886022X.2012.718951
21. Padilla B, Pollak VE, Pesce A, Kant KS, Gilinsky NH, Deddens JA. Pancreatitis in patients with end-stage renal disease. Medicine (Baltimore). 1994;73:8-20. doi:10.1097/00005792-199401000-00002
22. Barbara M, Tsen A, Rosenkranz L. Acute pancreatitis in chronic dialysis patients. Pancreas. 2018;47:946-51. doi:10.1097/MPA.00000 00000001119
23. Zerem E. Treatment of severe acute pancreatitis and its complications. World J Gastroenterol. 2014;20:13879-92. doi:10.3748/wjg.v20.i38. 13879
24. Feng YC, Wang M, Zhu F, Qin RY. Study on acute recent stage pancreatitis. World J Gastroenterol. 2014;20:16138-45. doi:10.3748/ wjg.v20.i43.16138
25. Harshit Kumar A, Singh Griwan M. A comparison of APACHE II, BISAP, Ranson's score and modified CTSI in predicting the severity of acute pancreatitis based on the 2012 revised Atlanta classification. Gastroenterol Rep (Oxf). 2018;6:127-31. doi:10.1093/gastro/gox029
26. Çağın YF, SY. Akut pankreatit şiddetini değerlendirmede kullanılan yöntemler. Guncel Gastroenteroloji. 2015;2015:275-82.
27. Yalcin MS, Tas A, Kara B, Olmez S, Saritas B. New predictor of acute necrotizing pancreatitis: red cell distribution width. Adv Clin Exp Med. 2018;27:225-8. doi:10.17219/acem/67590
28. Zhou H, Mei X, He X, Lan T, Guo S. Severity stratification and prognostic prediction of patients with acute pancreatitis at early phase: a retrospective study. Medicine (Baltimore). 2019;98:e15275. doi:10.1097/MD.0000000000015275
29. Goyal H, Awad H, Hu ZD. Prognostic value of admission red blood cell distribution width in acute pancreatitis: a systematic review. Ann Transl Med. 2017;5:342. doi:10.21037/atm.2017.06.61

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BS: Writing, data collection, interpretation and concept.
MM: Data collection, interpretation, writing, drafting and concept.
MH & FD: Data collection, interpretation, writing and drafting.
DDC: Data collection, interpretation, writing, drafting and statistical analysis.
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