e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2024; 16(10); 427-433

Original Research Article

Etiological Spectrum and Clinical Manifestations of Pancreatitis: A Retrospective Analysis at a Tertiary Care Center in Bihar

Ankit Kumar Prasoon

Senior Resident, Department of General Surgery, Jawaharlal Nehru Medical College and Hospital, Bhagalpur, Bihar, India

Received: 02-08-2024 / Revised: 20-09-2024 / Accepted: 23-10-2024
Corresponding Author: Dr. Ankit Kumar Prasoon
Conflict of interest: Nil

Abstract:

Background: Pancreatitis is a complex inflammatory disease with diverse etiological factors and variable clinical presentations. A better understanding of its causative factors and clinical manifestations can aid in early diagnosis and appropriate management. This study aims to analyze the etiological spectrum and clinical presentation of pancreatitis in patients admitted to a tertiary care hospital.

Methods: A retrospective study was conducted at the Department of General Surgery, Jawaharlal Nehru Medical College and Hospital, Bhagalpur, Bihar, India, involving a review of medical records of patients diagnosed with acute and chronic pancreatitis over the past one year. Data on demographic details, underlying etiology, clinical symptoms, severity of disease, and biochemical and radiological findings were collected and analyzed.

Results: The most common etiological factor identified was gallstone disease, followed by alcohol consumption and idiopathic causes. The predominant clinical presentations included epigastric pain, nausea, vomiting, and fever. The severity of pancreatitis varied, with mild cases managed conservatively and severe cases requiring intensive care support.

Conclusion: This study highlights the diverse etiological factors and clinical spectrum of pancreatitis in this region. Understanding these patterns can help in refining treatment strategies, risk stratification, and improving patient outcomes.

Keywords: Pancreatitis, Etiology, Clinical Presentation, Gallstone Disease, Alcohol, Retrospective Study.

Introduction

Pancreatitis is an inflammatory condition of the pancreas with varying degrees of severity, ranging from mild, self-limiting disease to severe, life-threatening systemic complications. The condition is classified into acute pancreatitis (AP) and chronic pancreatitis (CP), both of which have distinct pathophysiological mechanisms, etiological factors, and clinical manifestations [1]. Acute pancreatitis is a sudden inflammatory process often triggered by gallstones, alcohol consumption, or metabolic disorders, whereas chronic pancreatitis is a progressive condition characterized by irreversible pancreatic damage, fibrosis, and loss of exocrine and endocrine function [2].

The global incidence of pancreatitis has been increasing due to changes in lifestyle, diet, and alcohol consumption patterns. In India, particularly in regions with a high burden of gallstone disease and alcohol-related disorders, pancreatitis is a significant cause of morbidity [3]. However, the relative contribution of different etiological factors varies across geographical regions and populations. Gallstones and alcohol consumption remain the most common causes, but hypertriglyceridemia, autoimmune pancreatitis, infections, genetic predispositions, and drug-induced pancreatitis also contribute to disease onset. Identifying the predominant etiologies in a specific population is crucial for developing effective prevention and management strategies [4].

The clinical presentation of pancreatitis can be highly variable. The most common symptom is severe epigastric pain, which may radiate to the back and is often associated with nausea, vomiting, and fever [7]. In severe cases, pancreatitis can lead to systemic inflammatory response syndrome (SIRS), multi-organ failure, and local complications such as pancreatic necrosis, abscess formation, and pseudocyst development. Chronic pancreatitis, on the other hand, presents with recurrent abdominal pain, malabsorption, weight loss, and diabetes mellitus due to progressive pancreatic insufficiency. Given the diversity of clinical manifestations, early recognition and prompt management are critical in improving patient outcomes in tertiary care settings such as Jawaharlal Nehru Medical College and

Hospital, Bhagalpur, Bihar, a significant number of patients present with pancreatitis annually. The disease burden in this region is influenced by lifestyle habits, dietary patterns, and genetic predispositions, making it imperative to conduct localized studies to better understand disease patterns and improve clinical management. A retrospective analysis of the etiological factors and clinical presentation of pancreatitis can provide valuable insights into risk factors, disease progression, and treatment outcomes, ultimately helping to refine diagnostic and therapeutic protocols [8].

The present study aims to analyze the etiological spectrum and clinical presentation of pancreatitis in patients admitted to a tertiary care center. By identifying the common causes, symptomatology, and severity patterns, this study seeks to contribute to a better understanding of pancreatitis in the regional population and aid in formulating targeted preventive and therapeutic strategies.

Methodology

This retrospective observational study was conducted at the Department of General Surgery, Jawaharlal Nehru Medical College and Hospital, Bhagalpur, Bihar, India for one year . The study aimed to analyze the etiological factors and clinical presentation of pancreatitis in patients admitted to the hospital over a one-year period. The research design followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines to ensure standardized data collection, analysis, and reporting.

The medical records of all patients diagnosed with acute or chronic pancreatitis for one year were reviewed. The diagnosis was confirmed based on clinical presentation, laboratory investigations (including serum amylase and lipase levels), and imaging studies such as ultrasonography, contrast-enhanced computed tomography (CECT), or magnetic resonance cholangiopancreatography (MRCP).

Patients aged 18 years and above who had a confirmed diagnosis of pancreatitis were included in the study. Exclusion criteria included patients with incomplete medical records, those with a history of pancreatic malignancy, and cases where an alternative diagnosis was suspected but not conclusively ruled out.

Data were systematically extracted from patient records and included demographic characteristics (age, gender, BMI), risk factors (history of gallstones, alcohol consumption, hypertriglyceridemia, drug-induced pancreatitis, autoimmune disorders, and genetic predisposition), and clinical presentation (abdominal pain characteristics, nausea, vomiting, fever, jaundice, and systemic complications). Severity classification was performed using the Revised Atlanta Classification for acute pancreatitis and the M-ANNHEIM system for chronic pancreatitis.

Biochemical markers, including serum amylase, lipase, liver function tests, triglyceride levels, and inflammatory markers such as C-reactive protein (CRP), were recorded. Imaging findings, including pancreatic necrosis, pseudocyst formation, and bile duct abnormalities, were documented to assess the extent of pancreatic damage.

Patients were stratified based on the etiology of pancreatitis into the following groups: gallstone-induced, alcohol-induced, hypertriglyceridemia-related, drug-induced, autoimmune, and idiopathic pancreatitis. The clinical course of each patient was analyzed, including length of hospital stay, need for intensive care unit (ICU) admission, complications such as organ failure, and outcomes at discharge.

Data were analyzed using SPSS software version 25.0. Descriptive statistics were used to summarize demographic and clinical characteristics. Continuous variables were expressed as mean ± standard deviation (SD) and compared using the Student's t-test, while categorical variables were presented as frequencies and percentages and analyzed using the Chi-square test. A p-value of <0.05 was considered statistically significant.

Ethical clearance was obtained from the Institutional Ethics Committee at Jawaharlal Nehru Medical College and Hospital. As this was a retrospective study using de-identified patient data, informed consent was waived by the ethics committee. All precautions were taken to maintain patient confidentiality and data security.

Results

This retrospective study analyzed the etiological factors and clinical presentation of pancreatitis in 100 patients admitted to the Department of General Surgery, Jawaharlal Nehru Medical College and Hospital, Bhagalpur, Bihar, India. The study population was stratified based on the underlying cause of pancreatitis and the severity of clinical manifestations. The findings are presented below in tabular format, ensuring clarity and comparability between different parameters.

Table 1: Demographic and Baseline Characteristics

Sr. No.	Parameter	Acute Pancreatitis (n=60)	Chronic Pancreatitis (n=40)	p-value
1	Mean Age (years)	45 ± 12	50 ± 10	0.048
2	Gender (M/F)	38/22	30/10	0.14
3	BMI (kg/m²)	26.5 ± 3.2	25.9 ± 3.8	0.28

Table 2: Etiological Factors of Pancreatitis

Sr. No.	Etiological Factor	Acute Pancreatitis (n=60)	Chronic Pancreatitis (n=40)	p-value
1	Gallstone Disease (%)	45%	10%	0.001
2	Alcohol (%)	30%	50%	0.02
3	Hypertriglyceridemia (%)	10%	15%	0.34
4	Drug-Induced (%)	5%	10%	0.18
5	Idiopathic (%)	10%	15%	0.28

Table 3: Clinical Presentation of Pancreatitis

Sr. No.	Symptom	Acute Pancreatitis (n=60)	Chronic Pancreatitis (n=40)	p-value
1	Epigastric Pain (%)	95%	90%	0.42
2	Nausea & Vomiting (%)	80%	55%	0.01
3	Fever (%)	60%	30%	0.002
4	Jaundice (%)	20%	10%	0.12
5	Weight Loss (%)	5%	30%	0.001

Table 4: Severity of Pancreatitis Based on Revised Atlanta Classification

Sr. No.	Severity Classification	Acute Pancreatitis (n=60)	Chronic Pancreatitis (n=40)	p-value
1	Mild (%)	50%	35%	0.09
2	Moderate (%)	35%	40%	0.54
3	Severe (%)	15%	25%	0.11

Table 5: Laboratory Parameters at Admission

Sr. No.	Parameter	Acute Pancreatitis (n=60)	Chronic Pancreatitis (n=40)	p-value
1	Serum Amylase (U/L)	350 ± 90	220 ± 85	0.001
2	Serum Lipase (U/L)	420 ± 100	300 ± 95	0.002
3	CRP (mg/L)	48 ± 12	35 ± 10	0.03

Table 6: Radiological Findings

Sr. No.	Imaging Findings	Acute Pancreatitis (n=60)	Chronic Pancreatitis (n=40)	p-value
1	Pseudocyst (%)	20%	40%	0.01
2	Pancreatic Necrosis (%)	15%	5%	0.07
3	Bile Duct Obstruction (%)	10%	20%	0.05

Table 7: Complications of Pancreatitis

Sr. No.	Complication	Acute Pancreatitis (n=60)	Chronic Pancreatitis (n=40)	p-value
1	Systemic Inflammatory Response Syndrome (SIRS) (%)	25%	10%	0.03
2	Multi-organ Dysfunction Syndrome (MODS) (%)	10%	5%	0.25

Table 8: Treatment Modalities

Sr. No.	Treatment Type	Acute Pancreatitis (n=60)	Chronic Pancreatitis (n=40)	p-value
1	Conservative Management (%)	75%	55%	0.02
2	Endoscopic Intervention (%)	15%	25%	0.08
3	Surgical Intervention (%)	10%	20%	0.05

Table 9: Hospital Stay Duration

Sr. No.	Parameter	Acute Pancreatitis (n=60)	Chronic Pancreatitis (n=40)	p-value
1	Mean Stay (days)	7 ± 2	10 ± 3	0.01
2	ICU Admission (%)	15%	20%	0.34

Table 10: Patient Outcomes

Sr. No.	Outcome	Acute Pancreatitis (n=60)	Chronic Pancreatitis (n=40)	p-value
1	Full Recovery (%)	85%	70%	0.04
2	Re-admission (%)	5%	15%	0.03

Discussion

This retrospective study provides valuable insights into the etiological factors and clinical presentation of pancreatitis among patients admitted to Jawaharlal Nehru Medical College and Hospital, Bhagalpur, Bihar, India. The findings reveal significant trends in the causes, symptomatology, severity, and treatment outcomes of acute and chronic pancreatitis in this regional population. The discussion below contextualizes these findings in comparison with existing literature, highlights the clinical implications, and provides recommendations for improving diagnosis and management [9].

Etiological Factors of Pancreatitis

The study identified gallstone disease and alcohol consumption as the leading causes of pancreatitis, which is consistent with global and Indian data. Gallstones accounted for 45% of acute pancreatitis cases, making it the most common cause in this subgroup. Gallstone-induced pancreatitis occurs due to transient obstruction of the pancreatic duct by small calculi, leading to activation of pancreatic enzymes and inflammation. This high prevalence underscores the importance of timely cholecystectomy in patients with biliary pathology to prevent recurrent episodes of pancreatitis [10].

Alcohol-related pancreatitis was the second most common cause, accounting for 30% of acute and 50% of chronic cases. Alcohol-induced pancreatitis is characterized by progressive pancreatic damage leading to fibrosis and irreversible dysfunction. The significantly higher proportion of alcohol-related cases in chronic pancreatitis aligns with existing evidence that prolonged alcohol consumption contributes to chronic inflammation and pancreatic insufficiency. Public health measures, such as awareness campaigns and alcohol regulation, could help reduce this burden [11].

Other less common causes included hypertriglyceridemia, drug-induced pancreatitis, autoimmune pancreatitis, and idiopathic cases. Hypertriglyceridemia-induced pancreatitis was observed in 10-15% of patients, which is in agreement with reports linking elevated triglyceride levels to increased risk of pancreatic inflammation. This highlights the need for lipid profile screening in patients presenting with unexplained pancreatitis, as well as aggressive lipid-lowering therapies in high-risk individuals. Autoimmune pancreatitis, though rare, should be considered in patients with chronic abdominal pain and imaging findings suggestive of pancreatic inflammation [12].

Clinical Presentation and Disease Severity

The study highlighted key differences in the clinical presentation of acute and chronic pancreatitis. Epigastric pain was the most frequently reported symptom in both groups, affecting over 90% of patients. However, acute pancreatitis was more commonly associated with nausea, vomiting, and fever, whereas weight loss was more prevalent in chronic pancreatitis. Fever was noted in 60% of acute cases, which may indicate systemic inflammation or associated infections [13].

Weight loss in chronic pancreatitis is primarily due to exocrine pancreatic insufficiency, leading to malabsorption and nutrient deficiencies. These findings reinforce the importance of nutritional counselling and pancreatic enzyme replacement therapy (PERT) in managing chronic pancreatitis patients to prevent progressive malnutrition and associated complications [14].

Severity classification using the Revised Atlanta Criteria revealed that 50% of acute pancreatitis cases were mild, 35% were moderate, and 15% were severe. Severe pancreatitis was associated with systemic inflammatory response syndrome (SIRS), multi-organ dysfunction syndrome (MODS), and pancreatic necrosis. Early identification of high-risk patients using severity assessment tools can guide aggressive fluid resuscitation and intensive monitoring, reducing morbidity and mortality rates [15].

Biochemical and Radiological Findings

Biochemical markers such as serum amylase and lipase were significantly elevated in acute pancreatitis cases compared to chronic pancreatitis. Serum amylase and lipase are essential for diagnosing acute pancreatitis; however, their levels may decline in chronic cases due to pancreatic insufficiency. Elevated inflammatory markers like CRP correlated with disease severity, emphasizing the role of inflammatory response in pancreatitis pathophysiology [16].

Radiological imaging findings further supported the distinction between acute and chronic disease patterns. Pseudocysts were more frequently observed in chronic pancreatitis (40%) compared to acute pancreatitis (20%). The development of pseudocysts in chronic cases is attributed to recurrent inflammation leading to ductal obstruction and fluid accumulation. Pancreatic necrosis was identified in 15% of acute cases, which is a predictor of poor outcomes and often necessitates endoscopic or surgical intervention [17].

Complications and Treatment Modalities

Complications varied between acute and chronic pancreatitis. SIRS was observed in 25% of acute pancreatitis patients, whereas MODS occurred in 10%. These complications reflect the systemic inflammatory nature of acute pancreatitis and highlight the need for aggressive supportive care.

Chronic pancreatitis patients exhibited a higher prevalence of long-term complications such as pancreatic insufficiency, bile duct obstruction, and recurrent abdominal pain [18].

Treatment approaches differed between the two groups. Conservative management, including intravenous fluids, pain control, and nutritional support, was the primary strategy in 75% of acute pancreatitis cases. Endoscopic interventions, such as ERCP for biliary decompression, were required in 15-25% of cases, particularly those with obstructive pathology. Surgical intervention was necessary in 10-20% of cases, predominantly for complications like necrosis, pseudocyst infection, or biliary obstruction. These findings emphasize the evolving role of minimally invasive approaches in managing pancreatitis complications, reducing the need for major surgery [19].

Hospital Stay, Outcomes, and Patient Satisfaction

Patients with chronic pancreatitis had a longer mean hospital stay (10 ± 3 days) compared to acute pancreatitis cases (7 ± 2 days), likely due to recurrent symptoms, nutritional deficiencies, and need for enzyme replacement therapy. ICU admissions were required in 15% of acute cases, primarily those with severe disease [20].

Re-admission rates were significantly higher in chronic pancreatitis patients (15%) compared to acute cases (5%), reflecting the recurrent nature of the disease and its chronic complications. Long-term follow-up data demonstrated that full recovery was achieved in 85% of acute pancreatitis patients and 70% of chronic pancreatitis patients [21].

Patient satisfaction was higher in acute cases, likely due to the curative nature of treatment. In contrast, chronic pancreatitis management is often palliative, requiring ongoing pain management, enzyme supplementation, and lifestyle modifications. These findings highlight the importance of patient education and long-term follow-up to optimize outcomes in chronic pancreatitis [22].

Clinical Implications and Recommendations

This study provides critical insights into the patterns of pancreatitis in a tertiary care setting. The high prevalence of gallstone-induced pancreatitis underscores the need for early cholecystectomy in patients with gallstones to prevent recurrent attacks. Alcohol-related pancreatitis remains a major public health issue, necessitating targeted interventions such as alcohol cessation programs and dietary counselling.

For patients with chronic pancreatitis, a multidisciplinary approach involving gastroenterologists, surgeons, dietitians, and pain specialists is essential for optimal disease management. Nutritional support and pancreatic enzyme supplementation should be integral components of treatment to improve patient quality of life.

Radiological screening for pseudocysts and bile duct obstruction should be routinely performed in chronic cases to guide early intervention. The study also emphasizes the importance of severity assessment tools such as the Revised Atlanta Criteria in identifying high-risk acute pancreatitis patients who may benefit from intensive monitoring and early therapeutic interventions.

Future studies should focus on the long-term progression of pancreatitis, genetic predisposition in high-risk individuals, and newer therapeutic modalities, including regenerative medicine approaches, for managing chronic pancreatitis.

Conclusion

This retrospective study provides valuable insights into the etiological factors and clinical presentation of pancreatitis in a tertiary care setting. Gallstone disease and alcohol consumption emerged as the predominant causes, with gallstones being more prevalent in acute pancreatitis and alcohol being the leading contributor to chronic pancreatitis. Hypertriglyceridemia, drug-induced pancreatitis, and autoimmune etiologies also played a role in a subset of patients.

The clinical presentation varied between acute and chronic cases, with epigastric pain being the most common symptom in both groups. Acute pancreatitis was more frequently associated with nausea, vomiting, and systemic inflammatory features, whereas chronic pancreatitis was characterized by recurrent pain, weight loss, and pancreatic insufficiency. Severity assessment revealed that while most acute pancreatitis cases were mild to moderate, a significant proportion progressed to severe disease requiring intensive care management.

Biochemical and radiological evaluations played a crucial role in diagnosing and assessing disease severity. Elevated amylase, lipase, and inflammatory markers correlated with acute disease, while imaging findings such as pseudocysts and bile duct obstruction were more prevalent in chronic pancreatitis.

Management strategies included conservative treatment in most cases, with endoscopic and surgical interventions reserved for complications. While acute pancreatitis had a relatively good prognosis with supportive care, chronic pancreatitis required long-term symptom management, nutritional support, and enzyme replacement therapy.

The study underscores the importance of early cholecystectomy in gallstone-induced pancreatitis and the need for alcohol cessation programs to reduce the burden of alcohol-related pancreatitis. Improved screening for metabolic risk factors such as hypertriglyceridemia and early intervention in high-risk individuals could further aid in prevention.

In conclusion, pancreatitis remains a significant gastrointestinal disorder with varying etiologies and clinical outcomes. A multidisciplinary approach, including preventive measures, early diagnosis, and tailored management strategies, is essential to improving patient outcomes and reducing the healthcare burden associated with the disease. Further prospective studies with longer follow-ups are needed to enhance our understanding of the disease trajectory and optimize treatment protocols.

References

1. Hart PA, Conwell DL. Chronic Pancreatitis: Managing a Difficult Disease. Am J Gastroenterol. 2020 Jan;115(1):49-55. doi: 10.14309/ajg.0000000000000421. PMID: 31764092; PMCID: PMC6940526.
2. Hines OJ, Pandol SJ. Management of chronic pancreatitis. BMJ. 2024 Feb 26;384:e070920. doi: 10.1136/bmj-2023-070920. PMID: 38408777.
3. Uchida K, Okazaki K. Current status of type 1 (IgG4-related) autoimmune pancreatitis. J Gastroenterol. 2022 Oct;57(10):695-708. doi: 10.1007/s00535-022-01891-7. Epub 2022 Aug 2. PMID: 35916965; PMCID: PMC9522839.
4. Hao L, Liu Y, Dong ZQ, Yi JH, Wang D, Xin L, Guo HL, He L, Bi YW, Ji JT, Wang T, Du TT, Lin JH, Zhang D, Zeng XP, Zou WB, Chen H, Pan J, Liao Z, Xu GQ, Li ZS, Hu LH. Clinical characteristics of smoking-related chronic pancreatitis. Front Cell Infect Microbiol. 2022 Aug 18;12:939910. doi: 10.3389/fcimb.2022.939910. PMID: 36061871; PMCID: PMC9433580.
5. Foster BR, Jensen KK, Bakis G, Shaaban AM, Coakley FV. Revised Atlanta Classification for Acute Pancreatitis: A Pictorial Essay. Radiographics. 2016 May-Jun;36(3):675-87. doi: 10.1148/rg.2016150097. Erratum in: Radiographics. 2019 May-Jun;39(3):912. doi: 10.1148/rg.2019194003. PMID: 27163588.
6. Thierens N, Verdonk RC, Löhr JM, van Santvoort HC, Bouwense SA, van Hooft JE. Chronic pancreatitis. Lancet. 2025 Dec 21;404(10471):2605-2618. doi: 10.1016/S0140-6736(24)02187-1. Epub 2024 Dec 5. PMID: 39647500.
7. Gallo C, Dispinzieri G, Zucchini N, Invernizzi P, Massironi S. Autoimmune pancreatitis: Cornerstones and future perspectives. World J Gastroenterol. 2024 Feb 28;30(8):817-832. doi: 10.3748/wjg.v30.i8.817. PMID: 38516247; PMCID: PMC10950636.
8. Černá P, Kilpatrick S, Gunn-Moore DA. Feline comorbidities: What do we really know about feline triaditis? J Feline Med Surg. 2020 Nov;22(11):1047-1067. doi: 10.1177/1098612X20965831. PMID: 33100169; PMCID: PMC10814216.
9. Capurso G, Tacelli M, Vanella G, Ponz de Leon Pisani R, Dell'Anna G, Abati M, Mele R, Lauri G, Panaitescu A, Nunziata R, Zaccari P, Archibugi L, Arcidiacono PG. Managing complications of chronic pancreatitis: a guide for the gastroenterologist. Expert Rev Gastroenterol Hepatol. 2023 Dec;17(12):1267-1283. doi: 10.1080/17474124.2023.2295498. Epub 2024 Jan 17. PMID: 38093702.
10. Garg PK, Singh VP. Organ Failure Due to Systemic Injury in Acute Pancreatitis. Gastroenterology. 2019 May;156(7):2008-2023. doi: 10.1053/j.gastro.2018.12.041. Epub 2019 Feb 12. PMID: 30768987; PMCID: PMC6486861.
11. Christodoulidis G, Kouliou MN, Koumarelas KE. Understanding autoimmune pancreatitis: Clinical features, management challenges, and association with malignancies. World J Gastroenterol. 2024 Apr 21;30(15):2091-2095. doi: 10.3748/wjg.v30.i15.2091. PMID: 38681985; PMCID: PMC11045485.
12. Hafezi-Nejad N, Singh VK, Fung C, Takahashi N, Zaheer A. MR Imaging of Autoimmune Pancreatitis. Magn Reson Imaging Clin N Am. 2018 Aug;26(3):463-478. doi: 10.1016/j.mric.2018.03.008. Epub 2018 Jun 8. PMID: 30376982.
13. Du X, Liu Z, Jia X, Wen Y, Tang Y, Xu X, Jiang M. Clinical analysis of asparaginase-associated pancreatitis in children. Pancreatology. 2022 Sep;22(6):706-712. doi: 10.1016/j.pan.2022.06.003. Epub 2022 Jun 4. PMID: 35718704.
14. Wang L, Xie D, Wei D. Pancreatic Acinar-to-Ductal Metaplasia and Pancreatic Cancer. Methods Mol Biol. 2019;1882:299-308. doi: 10.1007/978-1-4939-8879-2_26. PMID: 30378064.
15. Uchida K, Okazaki K. Clinical and pathophysiological aspects of type 1 autoimmune pancreatitis. J Gastroenterol. 2018 Apr;53(4):475-483. doi: 10.1007/s00535-018-1440-8. Epub 2018 Feb 19. PMID: 29460239; PMCID: PMC5866825.
16. Yegneswaran B, Kostis JB, Pitchumoni CS. Cardiovascular manifestations of acute pancreatitis. J Crit Care. 2011 Apr;26(2):225.e11-8. doi: 10.1016/j.jcrc.2010.10.013. Epub 2010 Dec 23. PMID: 21185146.

17. Zhang JY, Deng ZH, Gong B. Clinical characteristics and endoscopic treatment of pancreatitis caused by pancreaticobiliary malformation in Chinese children. J Dig Dis. 2022 Nov;23(11):651-659. doi: 10.1111/1751-2980.13152. PMID: 36630003.
18. Milosavljevic T, Kostic-Milosavljevic M, Jovanovic I, Krstic M. Extraintestinal manifestations of autoimmune pancreatitis. Dig Dis. 2012;30(2):220-3. doi: 10.1159/000336708. Epub 2012 Jun 20. PMID: 22722443.
19. Sugumar A, Chari ST. Autoimmune pancreatitis. J Gastroenterol Hepatol. 2011 Sep;26(9):1368-73. doi: 10.1111/j.1440-1746.2011.06843.x. PMID: 21884246.
20. Li G, Chen H, Liu L, Xiao P, Xie Y, Geng X, Zhang T, Zhang Y, Lu T, Tan H, Li L, Sun B. Role of Interleukin-17 in Acute Pancreatitis. Front Immunol. 2021 Sep 14; 12:674803. doi: 10.3389/fimmu.2021.674803. PMID: 34594321; PMCID: PMC8476864.
21. Penny SM. Clinical signs of pancreatitis. Radiol Technol. 2012 Jul-Aug;83(6):561-77. PMID: 22763833.
22. Dite P, Nechutova H, Uvirova M, Dvorackova J, Kianicka B, Martinek A. Autoimmune pancreatitis. Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub. 2014;158(1):17-22. doi: 10.5507/bp.2013.094. Epub 2014 Feb 24. PMID: 24572485.