ORIGINAL ARTICLE
Turk J Surg

Risk factors for visceral artery pseudoaneurysm in chronic pancreatitis: A retrospective analysis

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ABSTRACT

Objective: Chronic pancreatitis (CP) leads to enduring abdominal pain and functional insufficiency, alongside notable risks posed by vascular complications. Pseudoaneurysms (PSA) are common in CP, necessitate careful management due to potential life-threatening hemorrhage. Literature suggests a 5-10% incidence of gastrointestinal bleeding in CP, often related to PSA affecting nearby arteries. Our study aims to evaluate the prevalence and outcomes of vascular complications in CP, aiding in improved management strategies.

Material and Methods: This retrospective observational study was conducted on the patients diagnosed with CP at a tertiary care center in Northeast India from April 2018 to December 2023. Demographic data and risk factors such as smoking and alcohol use were collected from medical records. The diagnosis and etiological assessment followed the M-ANNHEIM criteria, employing contrast-enhanced computed tomography.

Results: In our study of 86 patients with CP, predominantly male (68.6%), the median age at presentation was 37.4 years. Arterial PSAs were identified in 11 patients (12.79%), with a median onset of 18.2 months from symptom onset. Univariate analysis revealed that male sex (p=0.015), alcohol abuse (p=0.001), smoking (p=0.035), pseudocyst formation (p=0.008), and absence of parenchymal calcification (p=0.002) were significantly associated with PSA development. Interestingly, inflammatory head mass was more prevalent in patients without PSA (49.3% vs. 9.1%, p=0.02), suggesting a potential protective effect. On multivariate analysis, independent predictors of PSA formation included an alcohol abuse [odds ratio (OR): 10.75, 95% confidence interval (CI): 0.967-119.53, p=0.05], a pseudocyst presence (OR: 27.41, 95% CI: 1.591-472.39, p=0.02), and a bulky pancreatic head (OR: 12.72, 95% CI: 2.97-54.51, p=0.0006), while parenchymal calcification remained inversely associated (OR: 0.1279, 95% CI: 0.016-1.02, p=0.05).

Conclusion: Arterial PSA formation in CP is independently associated with alcohol abuse, pseudocysts, and inflammatory head mass, while parenchymal calcification appears protective. Endovascular coiling has emerged as a promising intervention, demonstrating effective management of PSA and successful prevention of hemorrhagic complications.

Keywords: Chronic pancreatitis, vascular complications, pseudoaneurysm, coil embolization

INTRODUCTION

Chronic pancreatitis (CP) is a progressive inflammatory disorder characterized by irreversible damage to the pancreatic parenchyma, leading to persistent abdominal pain and impaired endocrine and exocrine function. While the etiology of CP is multifactorial, involving factors such as alcohol abuse, genetic predisposition, and idiopathic factors, its clinical course is often complicated by the development of vascular complications (1).

Vascular complications in CP encompass a wide range of pathologies, including pseudoaneurysms (PSA), arterial thrombosis, and venous occlusions, presenting significant diagnostic and therapeutic challenges (2). Among these, visceral artery PSA is the most common arterial complication, with potentially life threatening consequences due to risk of severe hemorrhage (3). PSAs are more frequently associated with CP than with acute pancreatitis (AP), posing considerable risk of morbidity and mortality (4).

Previous studies have indicated that vascular complications, notably gastrointestinal bleeding, occur in 5-10% of patients with CP, with a significant portion attributed to bleeding from PSAs. PSAs commonly occur in the arteries near the pancreas,

Cite this article as: Anand U, Yadav S, Kodali R, Parasar K, Kumar R, Priyadarshi RN, et al. Risk factors for visceral artery pseudoaneurysm in chronic pancreatitis: a retrospective analysis. *Turk J Surg.* [Epub Ahead of Print]

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Received: 18.06.2025
Accepted: 28.07.2025
Epub: 11.08.2025

DOI: 10.47717/turkjsurg.2025.2025-6-10

Available at www.turkjsurg.com

such as the gastroduodenal artery (GDA), splenic artery (SA), and superior mesenteric artery. Studies have suggested that PSAs associated with CP necessitate surgical intervention more frequently than those associated with AP (3,4).

Despite advances in diagnostic techniques and therapeutic interventions, the understanding of vascular complications in CP remains limited, particularly regarding their prevalence, risk factors, and clinical outcomes. Much of the existing literature consists of case reports and series highlighting the need for comprehensive studies to elucidate the epidemiology and impact of these complications. In this context, our study aims to investigate the vascular complications of CP through a retrospective analysis of patients managed at a tertiary care center. By examining a large cohort of CP patients, we aim to delineate the prevalence, clinical characteristics, and outcomes of vascular complications, providing valuable insights into their management and prognostic implications.

MATERIAL and METHODS

A retrospective review was conducted on all patients aged 18-75 years diagnosed with CP at a tertiary care center in Northeastern India between April 2018 and December 2022. Exclusion criteria included patients with a PSA attributable to causes other than trauma and those with prior vascular conditions. Patients >75 years old were excluded to reduce heterogeneity and potential bias from age-related comorbidities, that could influence vascular complications and outcomes. Ethical approval was obtained from the Institutional Ethics Committee of All India Institute of Medical Sciences (protocol code-IEC/2022/994, date: 14.12.2022), and the study adhered to the principles outlined in the Declaration of Helsinki as well as local and national regulations. Informed consent was taken from all participants involved in the study. The medical records were thoroughly reviewed, considering the demographic details and risk factors associated with CP, such as smoking and alcohol abuse. Alcohol abuse is defined as chronic intake >80 g/day for men and >60 g/day for women for >5 years. Contrast-enhanced computed tomography (CECT) with the pancreatic protocol has been done for the diagnosis of CP and etiological evaluation has been done according to M-ANNHEIM criteria. Notable features such as pancreatic calcifications, ductal lesions, and pseudocysts were documented. Ductal lesions included main pancreatic duct strictures, dilatation >5 mm, intraductal calculi or hypoechoic ductal nodules seen on CECT. PSA were identified via abdominal computed tomography angiography (CTA), characterised by a hyper-attenuating contrast-enhanced sac adjacent to an artery, with evidence of communication or contrast leakage from a defined vascular territory on digital subtraction angiography (DSA) (5).

Patients who presented with gastrointestinal or intra-abdominal bleeding received immediate resuscitation with intravenous fluids and/or blood transfusions as necessary. Treatment strategies were based on the patient's hemodynamic status. For hemodynamically stable patients, a CTA was conducted to assess PSA. If PSAs were detected, patients underwent DSA followed by endovascular embolization (6). If endovascular embolization was unsuccessful, patients were considered for percutaneous thrombin injection. For hemodynamically stable patients with PSAs amenable to direct percutaneous thrombin injection (i.e., large PSA with narrow neck), the procedure was performed. In cases where patients were hemodynamically unstable, they underwent surgical intervention. The size of PSAs, determined by the maximum diameter in any plane, (and their location) was documented. Patients were followed up every three months for one year to monitor their progress.

Statistical Analysis

The median and the interquartile range (IQR) were used to represent continuous data, whereas the total number of participants and proportion were used to represent categorical data. Baseline bivariate comparisons were performed after the group was stratified according to the occurrence of vascular events. When appropriate, Fisher's exact test or the chi-square test was used to test the categorical variables. After assessing if continuous variables were normal with the Shapiro-Wilk test, the Mann-Whitney U test was used. Risk factors for vascular events in CP patients were identified using a multivariable Cox proportional hazard model. Univariate analysis was performed to assess potential risk factors, with those significant in the univariate analysis included in the final multivariable model. Hazard ratios and 95% confidence intervals (CIs) were calculated. Statistical analysis was conducted using IBM SPSS, version 27.0, with a two-sided p-value of less than 0.05 considered statistically significant.

RESULTS

Table 1 presents the distribution of vessel involvement in cases of arterial PSA. Out of 86 patients, 11 were diagnosed with arterial PSA, representing a prevalence of 12.79%. SA was the most commonly involved vessel, accounting for 45.45% of cases, followed by the GDA and left gastric artery at 18.1% and 27.3% respectively. The proper hepatic artery was involved in 9.09% of cases.

Table 1. Site of arterial pseudoaneurysm (n=11)

Vessel involved	Number of cases (percentage)
Splenic artery	5 (45.45%)
Gastroduodenal artery	2 (18.18%)
Proper hepatic artery	1 (9.09%)
Left gastric artery	3 (27.27%)

Values are presented as number (%).

Table 2 presents the clinical presentation of patients with arterial PSA. The majority of cases were symptomatic (63.6%), with gastrointestinal bleeding being the most common symptom (43%). Hemorrhagic shock was observed in 28.5% of cases, while abdominal pain and decreased hemoglobin levels were reported in 14.2% of cases each. Notably, 36.36% of patients remained asymptomatic despite the presence of arterial PSAs.

Table 2. Clinical presentation of patients presented with arterial pseudoaneurysms (n=11)

Clinical manifestation	Number of cases (percentage)
Asymptomatic	4 (36.36%)
Gastrointestinal bleeding	3 (42.8%)
Haemorrhagic shock	2 (28.5%)
Abdominal pain	1 (14.2%)
Decreased haemoglobin	1 (14.2%)

Values are presented as number (%).

Table 3 compares demographic and clinical characteristics between two groups with and without PSA. The median age at presentation was 37 years (IQR 30-45) in the PSA group and 34 years (IQR 28-42) in the non-PSA group. All patients in the PSA group were male (100%), compared to 64% in the non-PSA group (p=0.015). The median duration of symptoms was significantly shorter in the PSA group (18.2 months, IQR 10-26) than in the non-PSA group (32.4 months, IQR 18-48). Regarding etiology, alcohol abuse was markedly higher in the PSA group (90.9% vs. 22.7%, p=0.001). Smoking and pseudocysts were more common in the PSA group (36.4% vs. 12%, p=0.035) than in the non-PSA group (36.4% vs. 5.3%, p=0.008). In contrast, inflammatory pancreatic head mass and parenchymal calcification were more

Table 3. Comparison of demographic and clinical profiles between patients with and without pseudoaneurysm in chronic pancreatitis

Variables	Pseudoaneurysm (n=11)	No pseudoaneurysm (n=75)	p-value
Age at presentation (years) Median (IQR)	37.7 (30-45)	34 (28-42)	0.265
Sex (male) (percentage)	11 (100%)	48 (64%)	0.015
Duration of symptoms (months), Median (IQR)	18.2 (10-26)	32.4 (18-48)	0.032
Alcohol abuse (percentage)	10 (90.9%)	17 (22.7%)	0.001
Smoker (percentage)	4 (36.4%)	9 (12%)	0.035
Pseudo cyst (percentage)	4 (36.4%)	4 (5.3%)	0.008
Inflammatory head mass (percentage)	1 (9.1%)	37 (49.3%)	0.02
Parenchymal calcification (percentage)	4 (36.4%)	60 (80%)	0.002

Values are presented as number (%), median (IQR), IQR: Interquartile range.

Table 4. Association of various risk factors with pseudoaneurysm formation (n=11)

Variables	Univariate analysis		Multivariate analysis	
	Odds (95% CI)	p-value	Odds (95% CI)	p-value
Male sex	13 (0.74-230)	0.068	-	-
Alcohol intake	34.1 (4.07-286)	0.001	10.75 (0.967-119.53)	**0.05**
Smoking	4.19 (1.02-17.2)	0.002	2.167 (0.277-16.93)	0.461
Pseudocyst	10.1 (2.07-49.7)	0.001	27.41 (1.591-472.39)	**0.02**
Inflammatory head mass	0.103 (0.0125-0.843)	0.012	12.72 (2.97-54.51)	**0.0006**
Parenchymal calcification	0.002 (0.0369-0.552)	0.002	0.1279 (0.016-1.02)	**0.05**

Values are presented as median (IQR), IQR: Interquartile range, CI: Confidence interval.

prevalent in the PSA-non-PSA group (36.4% vs. 80%, p=0.002) compared to the non-PSA group (9.1% vs. 49.3%, p=0.02).

Of the 11 patients diagnosed with PSA, the majority (n=8) were successfully managed with endovascular coiling, achieving a 100% technical success rate without procedural complications. Two patients, whose PSA were anatomically favourable for direct access and demonstrated narrow necks, underwent percutaneous thrombin injection under image guidance. Both procedures were technically successful, with complete thrombosis confirmed on follow-up imaging. One patient, who presented in a state of hemodynamic instability and was not a candidate for radiological intervention, underwent emergency surgical ligation of the bleeding vessel. All patients were monitored post-intervention in a high-dependency setting based on their clinical status. There were no recorded incidents of rebleeding, reintervention, or procedure-related morbidity during the one-year follow-up period. The median length of hospital stay among the 11 PSA patients was 7 days (IQR: 5-10 days), reflecting favorable short-term recovery following timely intervention.

Table 4 presents the univariate and multivariate analyses assessing of risk factors associated with PSA development. On univariate analysis, significant associations were observed with male sex (p=0.068), alcohol intake (p=0.001), smoking (p=0.002), presence of pseudocyst (p=0.001), bulky pancreatic head (p=0.012), and absence of parenchymal calcification (p=0.002). In multivariate logistic regression, alcohol intake [odds ratio (OR): 10.75; 95% CI: 0.967-119.53; p=0.05], pseudocyst (OR: 27.41; 95% CI: 1.591-472.39; p=0.02), and bulky pancreatic head (OR: 12.72; 95% CI: 2.97-54.51; p=0.0006) remained independent predictors of PSA formation. Conversely, parenchymal calcification demonstrated a protective effect (OR: 0.1279; 95% CI: 0.016-1.02; p=0.05).

DISCUSSION

The management of vascular complications in CP remains a significant challenge due to the complex interplay between chronic inflammation, fibrosis, and the vasculature surrounding the pancreas. Among these complications, arterial PSAs stand out due to their high mortality risk if not promptly diagnosed and treated. Our study aimed to examine the incidence, risk factors, clinical presentation, and management outcomes of PSAs in patients with CP, focusing on a cohort from a tertiary care center in Northeastern India.

In our study, we observed an incidence of arterial PSAs in 12.79% of CP patients, which aligns with the broader literature that reports an incidence range of 4-10% (7-9). This relatively high incidence may be attributed to the advanced state of CP in the study population, reflecting the progressive nature of the disease in this region. The frequent involvement of the SA, observed in 45.45% of PSA cases in our study, can be explained by its anatomical proximity to the pancreas. The repetitive inflammation and fibrosis associated with CP can erode the arterial wall, leading to PSA formation. The SA tortuous course and close association with the pancreatic tail make it particularly vulnerable. Our study identified several key risk factors associated with the development of arterial PSAs in CP patients. Alcohol abuse emerged as a significant risk factor, with a strong association observed between heavy alcohol consumption and PSA formation. This is consistent with existing knowledge that alcohol-induced pancreatitis tends to be more severe, leading to more pronounced fibrosis and inflammatory changes, which in turn increase the likelihood of vascular involvement. Mallick et al. (9) highlighted alcoholic pancreatitis as a leading cause of arterial PSA. They reported a significant association between chronic alcohol consumption, pancreatitis, and developing arterial PSAs, particularly involving the SA. The study by Maatman et al. (10) also highlighted the relationship between alcoholic pancreatitis and arterial PSAs, emphasizing a significant link between chronic alcohol, pancreatitis, and the development of arterial PSAs. Olesen et al. (7) proposed three complication clusters for CP: Inflammatory, fibrotic, and pancreatic insufficiency. The inflammatory cluster encompasses complications like pseudocysts, venous thrombosis, pseudoaneurysms, ascites, and upper gastrointestinal bleeding (7).

Smoking, another modifiable risk factor, was also significantly associated with PSA development. Smoking exacerbates pancreatic inflammation and impairs the healing process, thereby increasing the risk of complications such as PSA. This finding underscores the importance of smoking cessation as part of the management strategy for CP patients. Smoking exacerbates pancreatic fibrosis through the activation of pancreatic stellate cells and upregulation of interleukin-22 in mouse models (11). In our study, smoking was significantly associated with arterial PSA formation in univariate analysis but did not retain significance in the multivariate model. This suggests that the observed association may be confounded by other variables, such as alcohol use or pseudocyst formation. Previous studies have variably reported smoking as a contributory factor in CP-related complications; however, its independent role in vascular sequelae such as pseudoaneurysm formation remains inconclusive (12-15). Our findings align with studies where smoking did not emerge as an independent predictor, highlighting the need for further prospective research to clarify its role.

The presence of pancreatic pseudocysts was another significant risk factor identified in our study. Pseudocysts, which result from the encapsulation of pancreatic fluid collections by fibrous tissue, can exert pressure on adjacent vessels, potentially leading to erosion and pseudoaneurysm formation. This finding

is particularly important because it highlights the need for careful monitoring of patients with pseudocysts for early signs of vascular complications. Interestingly, parenchymal calcification, which is typically considered a marker of chronic pancreatic damage, was found to have a protective effect against the development of PSAs. This counterintuitive finding could be due to the stabilization of the pancreas and surrounding structures by the calcified tissue, reducing the likelihood of vessel erosion. However, further research is needed to confirm this hypothesis and fully understand the protective mechanisms involved.

The clinical presentation of PSAs in our study varied widely, with some patients presenting with acute gastrointestinal bleeding or hemorrhagic shock, while others remained asymptomatic despite the presence of sizable aneurysms. This variability underscores the importance of high clinical suspicion and routine surveillance imaging in CP patients, especially those with known risk factors (16). Endovascular coiling emerged as the primary treatment modality for PSAs in our cohort, with excellent outcomes. This minimally invasive approach offers several advantages, including the ability to precisely target the PSA, reduce the risk of rebleeding, and avoid the morbidity associated with open surgical procedures (17-21). Our findings are consistent with other studies that have demonstrated the efficacy of endovascular techniques in managing PSAs, particularly in hemodynamically stable patients. However, in cases where endovascular coiling was not feasible or failed, alternative approaches such as percutaneous thrombin injection were employed. This technique has been increasingly recognized as a viable option for treating select cases of PSAs, particularly when the anatomy is not favorable for coiling. The success of these interventions in our study highlights the importance of a multidisciplinary approach involving interventional radiologists, gastroenterologists, and surgeons to optimize outcomes for CP patients with vascular complications.

Study Limitations

Despite the valuable insights provided by our study, several limitations must be acknowledged. The retrospective design introduces potential bias due to reliance on the accuracy and completeness of medical records. The relatively small sample size restricts the generalizability of our findings, particularly concerning the prevalence and risk factors of arterial PSAs in diverse populations. Moreover, the absence of detailed data on inflammatory markers and other potential risk contributors limits our understanding of PSA pathophysiology. The observed protective effect of parenchymal calcification warrants further exploration in larger, prospective studies. Additionally, although endovascular coiling demonstrated favorable outcomes, data on its long-term durability and recurrence risk remain limited. Future research should include broader clinical and laboratory parameters and longer follow-up to refine risk stratification and treatment strategies.

CONCLUSION

In conclusion, arterial PSA formation in CP is independently associated with alcohol abuse, inflammatory head mass, and pseudocyst formation, while parenchymal calcification may offer a protective effect. These findings underscore the importance of focused surveillance in high-risk patients and support endovascular coiling as the treatment of choice, while emphasizing the need for prospective studies to optimize long-term management.

Ethics

Ethics Committee Approval: Ethical approval was obtained from the Institutional Ethics Committee of All India Institute of Medical Sciences (protocol code-IEC/2022/994, date: 14.12.2022), and the study adhered to the principles outlined in the Declaration of Helsinki as well as local and national regulations.

Informed Consent: Informed consent was taken from all participants involved in the study.

Footnotes

Author Contributions

Concept - U.A., K.P.; Design - U.A., K.P.; Data Collection or Processing - S.Y., R.N.P., B.N.S., K.K.; Analysis or Interpretation - S.Y., R.K., B.N.S., K.K.; Literature Search - U.A., Ra.K., K.P., R.K., R.N.P., K.K.; Writing - U.A., S.Y., R.K., K.P., Ra.K., R.N.P., B.N.S. K.K.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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