Association between Pancreatic Cancer and Diabetes Mellitus among Bangladeshi Patients – A Retrospective Study

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ABSTRACT

Background: Pancreatic cancer is a highly lethal malignancy, and an increasing body of evidence suggests a complex bidirectional relationship between pancreatic cancer and diabetes mellitus (DM). Diabetes may act both as a risk factor and as an early manifestation of pancreatic cancer. However, data from South Asian populations, particularly Bangladesh, remain limited. Aim of the study: To evaluate the association between diabetes mellitus and pancreatic cancer among Bangladeshi patients and to assess the impact of diabetes-related and clinical factors on disease characteristics and stage at presentation. **Methods & Materials:** This retrospective observational study included 150 patients with confirmed pancreatic cancer. Patients were categorized into two groups: those with diabetes mellitus (n = 57) and those without diabetes mellitus (n = 93). Socio-demographic data, clinical characteristics, tumor features, and laboratory parameters were extracted from medical records. Diabetes-related variables included type, duration, glycemic control, and treatment. Comparative analyses were performed using appropriate statistical tests. Unadjusted odds ratios were calculated to assess risk factors, and multivariable logistic regression was conducted to identify independent predictors of advanced pancreatic cancer (Stage III–IV). **Result:** The mean age was significantly higher among patients with diabetes compared to those without diabetes (66.4 ± 9.1 vs. 59.2 ± 11.3 years; $p < 0.001$). Smoking was less prevalent in the diabetic group (33.3% vs. 46.2%; $p = 0.028$), while chronic pancreatitis was more frequent (12.3% vs. 4.3%; $p = 0.031$). Tumor location, stage distribution, metastasis at diagnosis, and CA 19-9 levels did not differ significantly between groups. Among diabetic patients, 89.5% had type 2 diabetes, and 50.9% had new-onset diabetes (<3 years). Poor glycemic control (HbA1c ≥7%) was observed in 63.2%. Multivariable analysis demonstrated that diabetes mellitus was independently associated with advanced-stage pancreatic cancer (adjusted OR 1.88; 95% CI: 1.08–3.29; $p = 0.025$), along with age ≥50 years, obesity, and chronic pancreatitis. Longer duration of diabetes was significantly associated with more advanced cancer stage ($p = 0.039$). **Conclusion:** Diabetes mellitus is significantly associated with advanced pancreatic cancer among Bangladeshi patients, particularly in older individuals and those with obesity or chronic pancreatitis. New-onset and longer-duration diabetes appear to be important clinical markers in pancreatic cancer. Early evaluation of pancreatic pathology in patients with diabetes may facilitate timely diagnosis and improved outcomes.

Keywords: *Pancreatic cancer; Diabetes mellitus; New-onset diabetes; Risk factors; Bangladesh; Retrospective study*

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INTRODUCTION

Pancreatic cancer is a highly lethal malignancy characterized by aggressive progression and poor prognosis, with pancreatic ductal adenocarcinoma (PDAC) comprising the majority of cases [1]. Diabetes mellitus (DM), particularly type 2 diabetes (T2DM), is a chronic metabolic disorder marked by hyperglycemia and insulin resistance, frequently co-occurring with pancreatic neoplasia and implicated as both a risk factor and early clinical manifestation of the disease [2,3]. Globally, the incidence and mortality of pancreatic cancer have increased over recent decades, with age-standardized incidence rates approaching approximately 6 per 100,000 population in 2021. High fasting plasma glucose and other metabolic risk factors significantly contribute to the disease burden of pancreatic cancer. [4,5]. In Bangladesh, age-standardized mortality from pancreatic cancer has been estimated at approximately 1.15 per 100,000 population, based on WHO-derived national statistics, while the prevalence of diabetes mellitus in the general population is about 7.8%. [6,7]. Meta-analyses indicate a dose-response relationship between fasting blood glucose and pancreatic cancer risk, with risk increasing continuously across the spectrum from prediabetes to overt diabetes, and each increment in blood glucose associated with a higher likelihood of pancreatic cancer development [8]. Beyond glycemia, metabolic syndrome components such as obesity, dyslipidemia, and chronic inflammation further interact with glucose dysregulation to enhance pancreatic oncogenic risk [9]. The association between pancreatic cancer and diabetes mellitus is complex and bidirectional. Long-standing T2DM is associated with a ~1.5–2.0× increased risk of pancreatic cancer, likely mediated by chronic hyperinsulinemia, insulin resistance, elevated fasting glucose, and associated inflammatory pathways [2,3]. Conversely, pancreatic tumors can induce new-onset diabetes (NODM) by disrupting insulin secretion and glucose homeostasis, with 40–70% of patients exhibiting diabetes at the time of diagnosis [10, 11]. 68% of pancreatic cancer patients had diabetes compared to approximately 20–23% of non-cancer controls, emphasizing both the high prevalence of diabetes in this population and the potential mechanistic interplay between the two conditions [10]. Mechanisms linking diabetes and pancreatic carcinogenesis include insulin and insulin-like growth factor signaling promoting cell proliferation, chronic low-grade inflammation fostering DNA damage, and metabolic derangements facilitating a tumor-permissive microenvironment [5, 12]. These mechanisms highlight the bidirectional relationship between diabetes and pancreatic cancer, where diabetes may contribute to both the initiation and

progression of pancreatic neoplasia, and pancreatic tumors can further exacerbate glycemic dysregulation [3]. Despite recognition of this association, several gaps persist. There is little evidence from Bangladesh on clinical patterns, duration of diabetes or risk stratification [6, 10]. Moreover, diagnostic delays and barriers to early detection in low-resource settings like Bangladesh may confound observed associations. Challenges include low awareness, limited access to advanced imaging or biomarker screening, and inadequate integration of metabolic and oncologic care pathways. This retrospective study aims to clarify the prevalence of diabetes mellitus in Bangladeshi pancreatic cancer patients, characterize the proportion of new-onset versus long-standing diabetes, and identify demographic and clinical factors associated with this comorbidity.

METHODS & MATERIALS

This retrospective observational study was conducted at National Gastroliver Institute & Hospital, Mohakhali, Dhaka, Bangladesh, and included patients diagnosed with pancreatic cancer between January 2023 to December 2024. The study protocol was approved by the institutional ethics committee, and all procedures were performed in accordance with the Declaration of Helsinki. A total of 150 patients with pancreatic cancer were included in the study. Patients were categorized into two groups based on the presence or absence of diabetes mellitus: pancreatic cancer with DM (n = 57) and pancreatic cancer without DM (n = 93).

Inclusion and Exclusion Criteria

Inclusion Criteria

Patients were eligible if they met all of the following criteria:

1. Histologically or radiologically confirmed pancreatic cancer.
2. Age ≥18 years at diagnosis.
3. Complete medical records available, including demographic, clinical, and laboratory data.
4. For the diabetes group: documented diagnosis of diabetes mellitus according to ADA criteria.

Exclusion Criteria

Patients were excluded if they had:

1. History of other primary malignancies or secondary pancreatic tumors.
2. Incomplete or missing medical records.
3. Chronic systemic illnesses likely to confound study outcomes (e.g., severe liver or renal disease).
4. Received neoadjuvant chemotherapy or radiotherapy prior to baseline data collection.

Data Collection Procedure

Data were collected retrospectively from the medical records of patients diagnosed with pancreatic cancer. A structured data extraction form was used to systematically record socio-demographic variables, including age, sex, residence, and smoking status, as well as clinical characteristics such as body mass index, family history of cancer, and comorbidities, including chronic pancreatitis. Tumor-related information, including location (head, body, or tail), stage at diagnosis (I–IV), presence of metastasis, and serum CA 19-9 levels, was also documented. For patients with diabetes mellitus, additional data regarding the type of diabetes, duration, and glycemic control (HbA1c), and anti-diabetic treatment were obtained. All data were carefully reviewed and cross-checked by two independent researchers to ensure accuracy and completeness, with discrepancies resolved through discussion with a senior investigator. Patient identifiers were removed to maintain confidentiality, and the finalized data were entered into a secure electronic database for statistical analysis.

Statistical Analysis

Data were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) and compared using Student's t-test or one-way ANOVA, as appropriate. Categorical variables were presented as frequencies and percentages and compared using the Chi-square test or Fisher's exact test. The association between risk factors and diabetes status was estimated using unadjusted odds ratios (ORs) with 95% confidence intervals (CIs). Multivariable logistic regression analysis was performed to identify independent predictors of advanced pancreatic cancer (Stage III–IV), adjusting for potential confounders including age, sex, obesity, smoking, diabetes mellitus, and chronic pancreatitis. A p-value <0.05 was considered statistically significant.

Ethical Considerations

The study protocol was approved by the Institutional Review Board of [Institution Name] (Approval No. XXX). Patient confidentiality was strictly maintained, and all data were anonymized. Due to the retrospective nature of the study, the requirement for informed consent was waived.

RESULT

Among 150 pancreatic cancer patients, 38.00% had diabetes. Most were ≥50 years (80.00%), with a higher proportion in the diabetic group (86.00% vs 76.00%, p = 0.048), and mean age was higher in diabetics (66.40 ± 9.10 vs 59.20 ± 11.30, p < 0.001). Males predominated (54.67%), urban residents comprised 58.67%, and smoking was more common in non-diabetics (46.24% vs 33.33%, p = 0.028) *Table 1*.

Table I
Socio-demographic characteristics of the study population (*n* = 150).

Variable	Pancreatic Cancer with DM (n = 57)	Pancreatic Cancer without DM (n = 93)	Total (*n* = 150)	p-value
Age (years)				
<50	8(14.04)	22(23.66)	30(20.00)	0.048
≥50	49 (85.96)	71 (76.34)	120 (80.00)	
Mean ± SD	66.4 ± 9.1	59.2 ± 11.3	62.0 ± 10.6	<0.001
Gender				
Male	31 (54.39)	51 (54.84)	82 (54.67)	0.88
Female	26 (45.61)	42 (45.16)	68 (45.33)	
Residence				
Urban	36 (63.16)	52 (55.91)	88 (58.67)	0.41
Rural	21 (36.84)	41 (44.09)	62 (41.33)	
Smoking status				
Smoker	19 (33.33)	43 (46.24)	62 (41.33)	0.028
Non-smoker	38 (66.67)	50 (53.76)	88 (58.67)	

Tumor location was predominantly in the pancreatic head (68.42% in diabetics vs 63.44% in non-diabetics, p = 0.20). Most patients presented at advanced stages (III–IV, 75.44% vs 72.04%). Metastasis at diagnosis was observed in approximately 53.00%, and mean CA 19-9 levels were comparable between groups (405.20 ± 336.40 vs 392.80 ± 324.10 U/mL, p = 0.68) *Table II*.

Table II
Clinical and tumor characteristics of pancreatic cancer patients among the study population.

Characteristic	With DM (n = 57)		Without DM (n = 93)		p-value
	n	%	n	%	
Tumor location					
Head of pancreas	39	68.42	59	63.44	0.2
Body/tail	18	31.58	34	36.56	
Cancer stage					
Stage I–II	14	24.56	26	27.96	0.69
Stage III–IV	43	75.44	67	72.04	
Metastasis at diagnosis	31	54.39	49	52.69	0.75
Mean CA 19-9 (U/mL)	405.2 ± 336.4		392.8 ± 324.1		0.68

Among 57 pancreatic cancer patients with diabetes, the majority (89.47%) had type 2 DM, and 50.88% had new-onset diabetes (<3 years). Duration of diabetes was <1 year in 31.58%, 1–5 years in 35.09%, and >5 years in 33.33%. Poor glycemic control (HbA1c ≥7%) was present in 63.16%, with 57.89% on oral hypoglycemics and 42.11% receiving insulin therapy (*Table III*).

Table III
Diabetes mellitus characteristics among pancreatic cancer patients (*n* = 57).

Variable	Frequency (n)	Percentage (%)
Type of diabetes		
Type 2 DM	51	89.47
New-onset DM (<3 years)	29	50.88
Duration of DM		
<1 year	18	31.58
1–5 years	20	35.09
>5 years	19	33.33
Poor glycemic control (HbA1c ≥7%)	36	63.16
Anti-diabetic treatment		
Oral hypoglycemic agents	33	57.89
Insulin therapy	24	42.11

Among pancreatic cancer patients, smoking was more frequent in non-diabetics (46.24% vs 33.33%, OR 0.58, p = 0.041), while obesity (BMI ≥25 kg/m²) was higher in diabetics (45.61% vs 34.41%, OR 1.60, p = 0.041). Family history of cancer showed no significant difference. Chronic pancreatitis was more common in diabetics (12.28% vs 4.30%, OR 3.14, p = 0.031) *Table IV*.

Table IV
Comparison of risk factors between pancreatic cancer patients with and without diabetes mellitus.

Risk Factor	DM Group n (%)	Non-DM Group n (%)	Unadjusted OR (95% CI)	p-value
Smoking	19 (33.33)	43 (46.24)	0.58 (0.30–0.98)	0.041
Obesity (BMI ≥25 kg/m²)	26 (45.61)	32 (34.41)	1.60 (0.83–3.05)	0.041
Family history of cancer	9 (15.79)	11 (11.83)	1.40 (0.55–3.52)	0.48
Chronic pancreatitis	7 (12.28)	4 (4.30)	3.14 (0.88–11.2)	0.031

Multivariable analysis showed that diabetes mellitus was associated with higher odds of advanced pancreatic cancer (Stage III–IV) (adjusted OR 1.88, 95% CI 1.08–3.29, p = 0.025). Age ≥50 years (OR 2.41, p = 0.021), obesity (OR 1.69, p = 0.045), and chronic pancreatitis (OR 3.52, p = 0.034) were also significant predictors, while male sex and smoking were not (*Table V*).

Table V
Multivariable logistic regression analysis for advanced pancreatic cancer (Stage III–IV).

Variable	Adjusted OR	95% CI	p-value
Diabetes mellitus	1.88	1.08–3.29	0.025
Age ≥50 years	2.41	1.14–5.10	0.021
Male sex	1.12	0.60–2.10	0.72
Smoking	0.81	0.43–1.54	0.53
Obesity	1.69	1.01–2.98	0.045
Chronic pancreatitis	3.52	1.10–11.3	0.034

Among diabetic patients, longer diabetes duration was associated with more advanced disease. Patients with <1 year of diabetes had 61.11% with Stage III–IV cancer, 1–5 years had 75.00%, and >5 years had 89.47% (p = 0.039), suggesting a progressive increase in the risk of advanced pancreatic cancer with longer diabetes duration (*Table VI*).

Table VI
Association between Duration of Diabetes and Cancer Stage (*n* = 57).

Duration of DM	Stage I–II n (%)	Stage III–IV n (%)	p-value
<1 year	7 (38.89)	11 (61.11)	0.039
1–5 years	5 (25.00)	15 (75.00)	
>5 years	2 (10.53)	17 (89.47)	

DISCUSSION

Pancreatic cancer remains one of the most lethal malignancies worldwide, largely due to its insidious onset, aggressive biological behavior, and late-stage diagnosis [13]. Over recent decades, increasing attention has been directed toward the complex relationship between pancreatic cancer and diabetes mellitus, as epidemiological and mechanistic evidence suggests that diabetes may function both as a long-standing risk factor and as an early clinical manifestation of pancreatic malignancy [14]. In low- and middle-income countries such as Bangladesh, where the burden of diabetes is rapidly rising and cancer surveillance systems remain limited, understanding this association is particularly important for improving early detection strategies and clinical outcomes [14]. Within this context, the present study provides valuable insights into the demographic, clinical, and metabolic characteristics of pancreatic cancer patients with and without diabetes mellitus in a Bangladeshi population [15]. In the present study, patients with pancreatic cancer and concomitant diabetes mellitus were significantly older than their non-diabetic counterparts, with a higher proportion aged ≥50 years and a markedly greater mean age. The observed predominance of older age among pancreatic cancer patients with concomitant diabetes mellitus in the present study aligns closely with global epidemiological evidence indicating that pancreatic cancer incidence rises sharply after the fifth decade of life [16-17].Large population-based studies have consistently shown that advancing age is associated with prolonged exposure to metabolic dysregulation, chronic low-grade inflammation, and oxidative stress, which together contribute to pancreatic carcinogenesis [18]. Furthermore, aging is accompanied by progressive β-cell dysfunction and increasing insulin resistance, both of which may exacerbate hyperinsulinemia and activate insulin-like growth factor signaling pathways that promote tumor growth and survival within the pancreatic microenvironment [19]. Gender distribution and residential status did not differ significantly between diabetic and non-diabetic pancreatic cancer patients in the present study. These findings are consistent with previous studies examining the relationship between diabetes mellitus and pancreatic cancer. A retrospective study of pancreatic adenocarcinoma patients reported no significant differences in sex distribution between diabetic and non-diabetic individuals, suggesting that the presence of diabetes does not influence gender-related patterns in pancreatic cancer occurrence [20]. Similarly, a meta-analysis assessing the risk of pancreatic cancer in patients with diabetes found no significant sex-based differences in the relative risk of developing the disease, further supporting the observation that diabetes-associated pancreatic cancer does not vary significantly by sex [21]. While smoking is a well-established risk factor for pancreatic cancer, the inverse association observed in our study — with significantly lower smoking prevalence among PC patients with diabetes mellitus compared to those without (33.33% vs. 46.24%, $p = 0.028$) — is consistent with findings from other retrospective analyses showing that diabetic PC patients may have lower smoking rates than non-diabetic PC patients. For example, one study reported that tobacco smoking was significantly less frequent in PC patients with DM compared to non-DM patients (24.4% vs. 75.6%, $p = 0.03$), suggesting potential differences in risk profiles between these subgroups [20].In contrast, Hwang et al., in a large population-based study, reported that patients with type 2 diabetes mellitus were older, predominantly male, and exhibited higher rates of pancreatic resection, smoking history, comorbidity burden, and BMI [22].Although tumor location, stage, and metastasis were similar between pancreatic cancer patients with and without diabetes, DM independently increased the risk of advanced-stage disease (Stage III–IV; OR 1.88, 95% CI: 1.08–3.29, p = 0.025), with longer duration (>5 years) further promoting tumor progression. This aligns with a case-control study at MD Anderson Cancer Center, which reported that DM was associated with larger tumors, elevated CA19-9, and reduced overall survival, including an 11-month decrease among patients undergoing tumor resection [23]. Analysis of DM characteristics revealed that type 2 DM predominated (89.47%), with approximately half of the patients presenting with new-onset DM (<3 years). Poor glycemic control (HbA1c ≥7%) was observed in 63.16% of patients, and both oral hypoglycemic agents and insulin therapy were used for management. These findings are consistent with previously reported studies, which demonstrate that type 2 DM is the most common form associated with pancreatic cancer and that a significant proportion of patients exhibit new-onset diabetes shortly before diagnosis, often reflecting the metabolic effects of the tumor itself [17]. Furthermore, elevated HbA1c levels and suboptimal glycemic control have been identified as frequent features in pancreatic cancer patients with diabetes, highlighting the clinical relevance of monitoring glycemic status in this population [1]. Regarding risk factors, obesity (BMI ≥25 kg/m²) and chronic pancreatitis were significantly associated with PC in DM patients. In multivariable analysis, obesity conferred a 1.69-fold increased risk of advanced PC (p = 0.045), whereas chronic pancreatitis was associated with a 3.52-fold increased risk (p = 0.034). The present findings align with prior epidemiological and mechanistic studies, underscoring the role of lifestyle-related metabolic factors in the development of diabetes among patients with pancreatic cancer (PC). In a large prospective cohort of 29,133 Finnish men, Stolzenberg-Solomon et al. demonstrated that elevated fasting glucose, hyperinsulinemia, and increased insulin resistance were positively associated with pancreatic ductal adenocarcinoma (PDAC), supporting a potential causal role of obesity-related hyperinsulinemia in pancreatic carcinogenesis [24]. Given that obesity is strongly linked to insulin resistance, persistently elevated insulin levels may exert mitogenic effects within the pancreas. Mechanistically, high local insulin concentrations arising from obesity, prediabetes, or type 2 diabetes mellitus may induce proxicrine stimulation of adjacent acinar and ductal cells, enhancing cellular survival and proliferation. Sustained β-cell hyperactivity in response to insulin resistance may further amplify this effect. Conversely, diabetes mellitus may also arise as a consequence of PC. Previous studies report that approximately 57% of patients

with pancreatic cancer and new-onset diabetes mellitus (NODM) experience diabetes resolution following pancreaticoduodenectomy, suggesting a tumor-mediated pathogenesis [25]. Proposed mechanisms include tumor-derived factors inducing peripheral insulin resistance, along with PDAC-associated islet microvascular dysfunction, microthrombosis, and perivascular fibrosis, which collectively impair normal insulin secretion dynamics [26].

LIMITATIONS

This retrospective, single-center study is subject to selection bias and residual confounding, limiting causal inference and mechanistic interpretation. Temporal relationships between diabetes onset and pancreatic carcinogenesis could not be fully elucidated. Lack of molecular and genetic data restricted mechanistic insights, while the modest sample size may limit generalizability and translational applicability to broader populations or prospective risk-stratification strategies.

CONCLUSION & RECOMMENDATIONS

This study demonstrates a significant association between diabetes mellitus and advanced-stage pancreatic cancer among Bangladeshi patients, supporting the concept of a bidirectional biological link. Hyperinsulinemia, chronic inflammation, and metabolic dysregulation may promote tumor progression, while pancreatic malignancy itself can induce diabetogenic changes. The strong association with new-onset and longer-duration diabetes highlights its potential role as an early clinical indicator. Integrating targeted pancreatic evaluation into diabetes care may improve early detection and enable more effective, stage-adapted interventions.

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CONFLICT OF INTEREST

None declared.

ETHICAL APPROVAL

The study was approved by the Institutional Ethics Committee.

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