



Advances in Modern Biomedicine

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Article

The Relationship of Blood Glucose with Early Morbidity, Mortality, and Other Prognostic Factors in Pancreatic Cancer

Bahri Ozer¹, Ferdi Bolat¹, Fatih Keyif¹, Songul Peltek Ozer², Mustafa Sit¹, İbrahim Karagöz³, Gulali Aktas^{4,*}

¹Department of General Surgery, Abant İzzet Baysal University Hospital, Bolu, Turkey

²Department of Pathology, İzzet Baysal Training and Research Hospital, Bolu, Turkey

³Department of Anesthesiology and Reanimation, Abant İzzet Baysal University Hospital, Bolu, Turkey

⁴Department of Internal Medicine, Abant İzzet Baysal University Hospital, Bolu, Turkey

*Corresponding author: Gulali Aktas, draliaktas@yahoo.com

Abstract

Aim: Pancreatic cancer remains a major global health issue due to its high morbidity rate and aggressive course. The association between diabetes and pancreatic cancer exhibits a bidirectional nature. Elevated levels of blood glucose and insulin resistance might create a favorable microenvironment for cancer development. In our study, we aimed to investigate the relationship between preoperative blood glucose levels and morbidity, mortality, and prognostic factors in pancreatic cancer.

Methods: Patients were classified into two groups: those with preoperative elevated glucose levels (>126 mg/dL) and those with normal glucose levels (≤ 125 mg/dL). The parameters analyzed included gender, age, postoperative drain amylase levels, length of hospital stay, morbidity, mortality, tumor size, and the number of metastatic lymph nodes.

Results: A total of 86 patients were included in the study. In predicting early postoperative mortality, age, tumor size, lymph node count, and preoperative glucose levels were not statistically significant ($p=0.16, 0.84, 0.81, 0.43$, respectively). However, morbidity, drain amylase levels, and hospital stay duration were statistically significant ($p=0.001, 0.005, 0.004$, respectively). ROC analysis revealed that hospital stay duration of more than 11 days had a sensitivity of 92% and a specificity of 58% for predicting mortality.

Conclusion: Present study showed that hospitalization duration and postoperative amylase levels were strongly associated with patient outcome in subjects with pancreas cancer. Proactive identification and management of these factors are essential to enhance postoperative outcomes and reduce mortality risks in this patient population.

Keywords

Pancreatic cancer, Blood glucose, Diabetes, Hospitalization, Mortality

Article history

Received: 30 April 2025

Revised: 27 May 2025

Accepted: 4 June 2025

Available Online: 18 June 2025

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1. Introduction

Pancreatic cancer remains a major global health issue due to its high morbidity rate and aggressive course. The most common subtype, ductal adenocarcinoma, has a strong association with diabetes. Despite advances in diagnostic tools, late-stage diagnosis limits the effectiveness of surgical and other therapeutic interventions. This necessitates the investigation of factors that may contribute to early diagnosis and prognosis assessment. Besides genetic and environmental factors, metabolic diseases also have a crucial role in the development of pancreatic cancer. Specifically, diabetes is strongly associated with pancreatic cancer and is considered a significant factor in both the onset and progression of the disease [1].

Pancreatic cancer, predominantly manifesting as ductal adenocarcinoma of the pancreas, is among the most deadly malignancies worldwide. It accounts for approximately 85% of all pancreatic cancer cases [2]. Characterized by aggressive tumor biology and a dense stromal microenvironment, ductal adenocarcinoma of the pancreas often eludes early detection, leading to a dismal prognosis. Pancreatic cancer is the seventh leading cause of cancer-related deaths worldwide, with its incidence rates varying significantly across different regions. In Europe, for instance, it is the seventh most common cause of mortality among all oncological diseases [3]. Risk factors include advanced age, smoking, obesity, type 2 diabetes, and a family history of the disease. Notably, approximately 5–10% of cases are attributed to inherited genetic mutations. The clinical presentation of pancreatic cancer is often nonspecific, symptoms such as abdominal pain, weight loss, jaundice, and new-onset diabetes typically manifest in the advanced stages of the disease. As a result, many patients are diagnosed when the disease is already unresectable [4]. Diagnostic modalities include imaging techniques like ultrasound, CT scan and magnetic resonance imaging, along with endoscopic ultrasound and biopsy for histopathological confirmation. Surgical removal is currently the only potentially curative treatment; however, only a minority of patients present with resectable disease [5]. Adjuvant therapies, including chemotherapy regimens such as FOLFIRINOX and gemcitabine-based protocols, have shown modest improvements in survival. Despite these advancements, the overall 5-year survival rate remains low, underscoring the need for continued research into early detection and novel therapeutic strategies [6]. Pancreatic cancer poses significant clinical challenges due to its aggressive nature, late-stage presentation, and limited treatment options. Ongoing research efforts aim to enhance early detection methods and develop more effective therapies to improve patient outcomes [7,8].

Blood glucose levels, encompassing both fasting glucose and glycated hemoglobin (HbA1c), have been identified as significant prognostic factors in cancer outcomes. Elevated blood glucose levels, even in non-diabetic individuals, have been associated with increased cancer mortality. A literature review highlighted that slight increases in fasting or casual glucose levels were linked to higher cancer mortality, particularly in men, and that impaired glucose tolerance or prediabetes states were associated with increased mortality, especially from stomach, liver, and pancreatic cancers [9]. Hyperglycemia has been suggested as an independent predictor of poor prognosis in various cancers. In patients with advanced pancreatic cancer, elevated blood glucose levels were linked to reduced overall survival, with a median survival of 7.5 months compared to 8.8 months in normoglycemic patients [10]. Similarly, chronic hyperglycemia has been linked to adverse outcomes in small cell lung cancer, with higher HbA1c levels correlating with increased risk of locoregional recurrence and distant metastasis [11]. Conversely, optimal glycemic control may improve cancer prognosis. Studies have suggested that maintaining blood glucose levels within target ranges can enhance survival outcomes in cancer patients. For instance, in patients with non-small cell lung cancer, better glycemic control was associated with improved overall survival [12]. Blood glucose levels play a crucial role in cancer prognosis. Elevated glucose levels, even in non-diabetic individuals, are associated with increased cancer mortality, while optimal glycemic control may improve survival outcomes. These findings highlight the critical role of monitoring and managing blood glucose levels in cancer patients, as this may contribute to improved treatment outcomes and overall survival. Conditions that characterized with high blood glucose levels, such as diabetes mellitus [13], metabolic syndrome [14], prediabetes [15], and obesity [16], are all associated with some degree of inflammation. Malignant conditions are also associated with inflammation [17]. Hence, outcome of the pancreas cancer and blood glucose levels could be linked in some common pathway.

The association between pancreatic cancer and diabetes mellitus exhibits a bidirectional nature. Epidemiological studies have shown that the relative risk of pancreatic cancer is approximately twice as high in populations with prevalent diabetes [18]. Elevated serum glucose levels and insulin resistance may create a favorable microenvironment for cancer development. Additionally, there is strong evidence suggesting that newly diagnosed diabetes could be an early indicator of pancreatic cancer [19,20]. In patients with pancreatic cancer, pre-existing type 2 diabetes was associated with a higher incidence of cachexia, greater weight loss, and decreased survival probability, regardless of initial body weight or tumor progression [21]. However, the impact of diabetes on disease progression, early morbidity, and mortality in patients diagnosed with pancreatic cancer remain unclear.

In present study, we aimed to investigate the relationship between preoperative blood glucose levels and morbidity, mortality, and prognostic factors in pancreatic cancer.

2. Methods

In present study, patients diagnosed with pancreatic cancer at Bolu Abant Izzet Baysal University Faculty of Medicine Hospital between January 2016 and December 2024, were retrospectively analyzed. Patients with pancreatic cancer that undergone surgery were included to the study. Patients who underwent pancreatic surgery for non-neoplastic or benign conditions were excluded from the study. We also excluded subjects that require palliative surgery, subjects with end stage kidney disease, cirrhosis and pregnancy. Approval was obtained from the Bolu Abant Izzet Baysal University Clinical Research Ethics Committee (approval no: 2025/111). Medical data were accessed through the hospital automation system. Demographic data, laboratory parameters at admission, treatment approaches, and surgical methods were recorded. For blood glucose levels, a fasting measurement taken after at least 8 hours was considered valid. Patients were classified into two groups: those with preoperative elevated glucose levels (>126 mg/dL) and those with normal glucose levels (≤ 125 mg/dL), according to the American Diabetes Association criteria [22]. Patients in diabetic group were diagnosed with type 2 DM just before surgery according to the preoperative tests results. These subjects were on no anti-diabetic medications. The parameters analyzed included gender, age, postoperative drain amylase levels, length of hospital stay, morbidity, mortality, tumor size, and the number of metastatic lymph nodes. These parameters were compared between the normal glucose and elevated glucose groups. Mortality was defined as death occurring within the first 30 days postoperatively. Additionally, patients were divided into two groups (survivors and deceased), and study data were compared between these groups.

Statistical analyses were conducted using IBM SPSS Statistics for Windows, Version 20.0 (Armonk, NY, USA). Descriptive statistics were employed to summarize the data. The Kolmogorov-Smirnov tests were used to assess normality. For data following a normal distribution, independent samples t-test was employed, while the Mann-Whitney U was used for non-normally distributed data. Receiver Operating Characteristic (ROC) curves were generated, and the Area Under the Curve (AUC) was calculated for variables showing statistical significance. A p-value of <0.05 was considered indicative of statistical significance.

3. Results

A total of 86 patients were enrolled to the study, with a mean age of 68 years (range: 38-88), with 48 males and 38 females. In the preoperative elevated glucose group, there were 25 males and 18 females, with a mean age of 67 (± 9.5) years. They were diagnosed with type 2 DM just before surgery according to the preoperative tests results. These subjects were on no anti-diabetic medications. The tumor size was 30 mm (2-80), the number of metastatic lymph nodes was 0 (0-18), postoperative drain amylase levels were 142 (3-158400), hospital stay duration was 21 days (3-59), morbidity was observed in 12 patients, and mortality in 11 patients.

In the preoperative normal glucose group, there were 23 males and 20 females, with a mean age of 70 (± 13). The tumor size was 30 mm (10-100), the number of metastatic lymph nodes was 2 (0-13), postoperative drain amylase levels were 249 (20-44818), hospital stay duration was 19 days (8-62), morbidity was observed in 11 patients, and mortality in 8 patients. When comparing gender, age, tumor size, metastatic lymph node count, drain amylase levels, hospital stay, morbidity, and mortality between the groups, no statistically significant differences were found ($p=0.66, 0.64, 0.70, 0.26, 0.20, 0.82, 0.80, 0.43$) (Table 1).

Table 1. Results of elevated glucose and normal glucose groups.

Variables	Preoperative Elevated Glucose Group	Preoperative Normal Glucose Group	p
<i>Mean \pm SD or Median (min.–max.)</i>			
Age(years)	67 (± 9.5)	70 (± 13)	0.64
Gender (n)	Female	18	0.66
	Male	25	
Tumor size (mm)	30 (2-80)	30 (10-100)	0.70
Metastatic lymph node (n)	0 (0-18)	2 (0-13)	0.26
Postop. drain amylase (U/l)	142 (3-158400)	249 (20-44818)	0.20
Hospital stay (days)	21 (3-59)	19 (8-62)	0.82
Morbidity (n)	12	11	0.80
Mortality (n)	11	8	0.43

In predicting early postoperative mortality, age, tumor size, lymph node count, and preoperative glucose levels were not statistically significant ($p=0.16, 0.84, 0.81, 0.43$). However, morbidity, drain amylase levels, and hospital stay duration were statistically significant ($p=0.001, 0.005, 0.004$) (Table 2). ROC analysis revealed that a hospitalstay duration of more than 11 days had a sensitivity of 92% and a specificity of 58% for predicting mortality (Figure 1).

Table 2. Relationship of mortality with parameters.

Variables	Exitus	Survived	p
<i>Mean \pm SD or Median (min.–max.)</i>			
Age (years)	70 (± 10)	66 (± 11)	0.16
Tumor size(mm)	33 (± 20)	22 (± 17)	0.84
Metastatic lymph node (n)	1 (0-6)	1 (0-18)	0.81
Preop. glucose (mg/dl)	132 (74-426)	116 (63-397)	0.43
Morbidity (n)	present	4	0.001
	absent	63	
Postop. drain amylase (U/l)	4108 (± 10986)	3785 (± 19969)	0.005
Hospital stay (days)	17 (± 16)	24 (± 12)	0.004

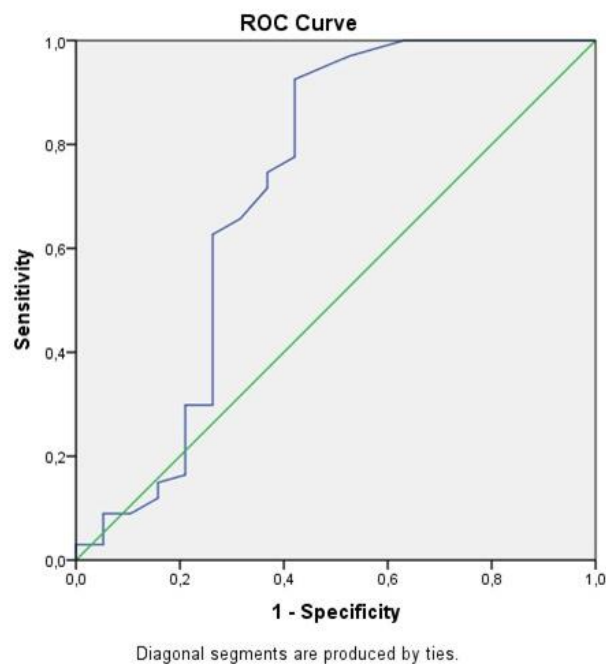


Figure 1. The sensitivity and specificity of hospital stay in detecting mortality

4. Discussion

Present study showed that hospitalization duration and postoperative amylase levels were strongly associated with patient outcome in subjects with pancreas cancer.

Pancreatic cancer is closely associated with alterations in glucose metabolism, often leading to elevated plasma glucose levels. This relationship is bidirectional: while long-standing diabetes can increase the risk of developing pancreatic cancer, the onset of pancreatic cancer can also induce diabetes or exacerbate existing glucose metabolism disorders [23].

Epidemiological studies have shown that individuals with type 2 diabetes mellitus face a 1.5- to 2-fold higher risk of developing pancreatic cancer compared to those without diabetes. Chronic hyperglycemia and hyperinsulinemia associated with diabetes are believed to promote pancreatic tumorigenesis. A meta-analysis of prospective studies found a linear relationship between fasting blood glucose levels and pancreatic cancer risk, indicating that even modest increases in glucose levels can elevate cancer risk [24,25].

Conversely, pancreatic cancer can lead to impaired glucose metabolism. Studies have shown that elevated glucose levels, indicative of prediabetes, can be presented up to three years before a pancreatic cancer diagnosis. This suggests that new-onset hyperglycemia or diabetes in adults may be an early manifestation of pancreatic cancer [26].

Hyperglycemia may also influence the efficacy of pancreatic cancer treatments. Research suggests that elevated glucose availability may enhance the sensitivity of pancreatic cancer cells to chemotherapy. In hyperglycemic conditions, the expression of GCLC, a key enzyme in glutathione biosynthesis, is diminished, leading to enhanced oxidative stress and increased susceptibility of cancer cells to chemotherapeutic agents [27].

The intricate link between pancreatic cancer and glucose metabolism has significant clinical implications. Monitoring changes in plasma glucose levels, particularly new-onset hyperglycemia or worsening glycemic control in adults, could aid in the early detection of pancreatic cancer. Moreover, understanding a patient's glycemic status may help tailor therapeutic strategies, potentially improving treatment efficacy and outcomes [28].

These evidence suggests that the relationship between plasma glucose levels and pancreatic cancer is complex and multifaceted, involving both the influence of diabetes on cancer risk and the impact of pancreatic cancer on glucose metabolism. Ongoing research continues to elucidate these connections, aiming to enhance early detection and treatment approaches for this aggressive malignancy.

Elevated amylase levels have been observed in patients with pancreatic cancer, but their diagnostic and prognostic significance is complex. Amylase, an enzyme mainly secreted by the pancreas and salivary glands, is essential for carbohydrate digestion. In pancreatic cancer, amylase levels may fluctuate depending on disease progression and tumor characteristics [29]. Elevated levels of amylase may be caused by the obstruction of the pancreatic ductus. Obstruction of the pancreatic ducts by a tumor can lead to increased amylase levels in the blood [30]. However, elevated amylase is not specific to pancreatic cancer and can also result from other conditions such as pancreatitis or salivary gland disorders. Therefore, while elevated amylase may raise suspicion, it is not definitive for diagnosing pancreatic cancer. In some cases, especially with significant pancreatic damage or advanced disease, amylase levels may remain normal or even decrease. This variability limits the utility of amylase as a standalone diagnostic marker for pancreatic cancer.

Research has explored the relationship between amylase levels and patient outcomes in pancreatic cancer. A study involving 351 patients with metastatic pancreatic cancer found that elevated plasma amylase was an independent adverse prognostic factor. Patients with higher amylase levels had poorer survival rates compared to those with normal levels. This suggests that amylase could serve as a prognostic marker in advanced stages of the disease [31]. Another large-scale study with 101,765 participants from the general population indicated that both extremely low and extremely high pancreatic amylase levels were associated with a two- to threefold increased risk of developing pancreatic cancer and chronic pancreatitis. This finding highlights the potential of amylase levels as a risk indicator, although it does not establish a direct causal relationship [29]. While amylase levels can provide insights into pancreatic function, their variability and lack of specificity limit their effectiveness as a sole diagnostic tool for pancreatic cancer. However, in conjunction with other diagnostic methods, such as imaging studies, biopsy, and additional biomarkers like CA 19-9, amylase measurements can contribute to a more comprehensive assessment. Moreover, monitoring amylase levels in patients with known pancreatic cancer may offer prognostic information, aiding in treatment planning and management. These data suggests that elevated amylase levels in pancreatic cancer patients can reflect underlying pancreatic pathology and may have prognostic value, particularly in advanced disease stages. However, due to their nonspecific nature, amylase measurements should be interpreted within the broader context of clinical findings and diagnostic investigations.

Prolonged hospitalization following pancreatic cancer surgery has been linked to increased postoperative mortality. Several studies have identified factors contributing to extended hospital stays and their association with higher mortality rates. A comprehensive study analyzing data from 22,523 patients who underwent pancreatic resection found that in-hospital mortality exhibited a bimodal distribution, with higher rates observed in both younger patients (under 40 years) and older patients (75 years and above). Independent predictors of in-hospital mortality included nonelective surgery, advanced age, and undergoing procedures other than distal pancreatectomy. These factors were also associated with prolonged hospital stays, suggesting that patient demographics and the urgency and type of surgical intervention significantly influence postoperative outcomes [32]. Another study focusing on postoperative outcomes identified additional risk factors for prolonged hospitalization and increased mortality, such as preoperative therapy, higher comorbidity scores, lower income, and more extensive surgical procedures. The study emphasized that patients aged 70 and above were particularly susceptible to higher 30-day mortality rates and extended hospital stays. This reports emphasize the need for thorough preoperative assessment and careful patient selection to reduce the risks associated

with extended hospital stays [33]. Understanding the factors that contribute to prolonged hospital stays and increased mortality after pancreatic cancer surgery is crucial for improving patient outcomes. By identifying high-risk patients through comprehensive preoperative evaluations, including assessments of age, comorbidities, and socioeconomic factors, healthcare providers can tailor perioperative care plans to address individual risks. Implementing strategies such as enhanced recovery after surgery protocols, optimizing nutritional status, and ensuring meticulous surgical planning may help reduce hospital stay durations and associated mortality rates.

Several reports have explored the association between the length of postoperative hospital stay and survival outcomes in patients with pancreatic cancer. While direct evidence linking the length of hospital stay to survival outcomes is limited, various factors related to postoperative recovery can influence survival rates. Postoperative complications and survival is linked to the length of hospitalization. Studies have highlighted that major postoperative complications, such as organ failure and infections, can adversely affect survival in pancreatic cancer patients. For example, a multicenter cohort study reported that postoperative infections were significantly associated with poorer overall survival, with median survival times of 21.9 months in patients who developed infections compared to 33.0 months in those who did not [34]. Similarly, another study reported that major complications, including organ failure, poor survival, with organ failure directly affecting disease-free interval and overall survival [35]. Length of hospital stay therefore could be assessed as an indicator for prognosis. While not directly studied, the length of postoperative hospital stay can serve as an indirect indicator of recovery and the presence of complications. Prolonged hospital stays may reflect complications such as delayed gastric emptying, infections, or the need for additional interventions, all of which can influence survival outcomes. Another issue related to hospital stay after pancreas cancer surgery is the importance of postoperative management. Effective postoperative management, including early detection and treatment of complications, timely initiation of adjuvant therapies, and comprehensive surveillance, is crucial for improving survival in pancreatic cancer patients. A study emphasized that postoperative infection could indirectly worsen prognosis by preventing timely adjuvant therapy [35]. While the direct correlation between postoperative hospital stay and survival in pancreatic cancer patients requires further research, it is evident that postoperative complications and the quality of postoperative care significantly influence survival outcomes. Therefore, strategies aimed at minimizing complications and optimizing postoperative care are essential for improving survival rates in these patients. Accordingly, we found association between hospital stay and the outcome of the patients with pancreatic cancer after surgery.

Present study failed to show significant association between blood glucose level and postoperative outcome of the patients with pancreatic cancer. The association between plasma fasting glucose and postoperative outcomes in pancreatic cancer patients is still the focus of ongoing research. While some studies suggest a potential association, the overall evidence remains inconclusive. Association between plasma fasting glucose and survival is being studied in the literature in different settings. A meta-analysis involving 48,424 patients found no significant association between blood glucose levels, either preoperative or postoperative, and overall survival in individuals with pancreatic cancer. The reported hazard ratios were 1.10 (95% CI: 0.89–1.35) for preoperative and 1.19 (95% CI: 0.92–1.54) for postoperative glucose levels, suggesting minimal influence on survival outcomes. Similar findings were observed for fasting blood glucose (FBG) and glycated hemoglobin (HbA1c) levels. However, postoperative hyperglycemia was associated with an increased risk of postoperative complications, with an odds ratio of 3.06 (95% CI: 1.88–4.97) [36]. In contrast, a retrospective study of 225 patients found that elevated preoperative blood glucose levels were linked to decreased overall survival and a higher incidence of postoperative complications. Patients with elevated preoperative blood glucose had a median survival of 14.2 months compared to 20.5 months in those with normal levels. The hazard ratio for overall survival was 1.68 (95% CI: 1.15–2.45), suggesting a potential prognostic role for preoperative blood glucose levels [37]. While certain studies indicate that preoperative blood glucose levels may influence survival and complication rates in pancreatic cancer patients, the evidence is not uniformly consistent. The variability in findings underscores the need for further research to elucidate the role of blood glucose management in the postoperative care of patients with pancreatic cancer.

The present study found no significant association between preoperative blood glucose levels and tumor size in patients with pancreatic cancer. This observation challenges the intuitive notion that larger tumors would exert a greater impact on glucose metabolism. This finding suggests that the mechanisms underlying hyperglycemia in pancreatic cancer are more complex and not solely dependent on tumor burden. Several studies have explored the relationship between blood glucose levels and pancreatic cancer characteristics. For instance, a retrospective study by Wang et al. (2025) involving 225 patients found no significant difference in median tumor size between patients with normal and high preoperative blood glucose levels [37]. Subsequently, this finding was confirmed by other works in the medical literature [38]. This indicates that hyperglycemia may not be directly correlated with tumor size. The lack of association may be attributed to factors such as tumor location and its impact on pancreatic endocrine function. Tumors located in the pancreatic tail, where insulin-producing islet cells are more concentrated, might disrupt glucose metabolism more significantly than larger tumors in other regions. Additionally, pancreatic tumors may induce systemic insulin resistance or impair insulin secretion through inflammatory cytokines and tumor-derived factors, leading to hyperglycemia independent of tumor size [37,39]. Clinically, this finding underscores the importance of monitoring and managing blood glucose levels in pancreatic cancer patients, regardless of tumor size. Elevated preoperative blood glucose levels have been linked to

poorer overall survival and a higher risk of postoperative complications, underscoring their potential prognostic value [37,40]. Therefore, addressing hyperglycemia as a separate clinical concern is crucial in the comprehensive management of pancreatic cancer patients.

Present study didn't show an association between blood glucose and the number of lymph node metastasis in the study cohort. The relationship between preoperative blood glucose levels and lymph node metastasis in pancreatic cancer has garnered interest due to the known associations between hyperglycemia, diabetes, and cancer progression. However, recent evidence indicates that elevated blood glucose levels were not significantly associated with lymph node metastasis in pancreatic cancer patients. A retrospective study involving 225 patients with pancreatic cancer found no significant difference in lymph node involvement between patients with normal and high preoperative blood glucose levels. Specifically, lymph node metastasis was present in 43.8% of the normal glucose group and 30.6% of the high glucose group ($P=0.058$), suggesting that hyperglycemia does not correlate with increased lymphatic spread in this context [37]. This finding contrasts with observations in other malignancies. For instance, in gastric cancer, diabetes mellitus has been associated with more advanced lymph node metastasis, with diabetic patients exhibiting higher rates of N3 station metastasis compared to non-diabetic patients [41]. Similarly, in papillary thyroid carcinoma, higher fasting serum glucose levels have been linked to increased likelihood of lymph node metastasis [42]. The lack of association in pancreatic cancer may be attributed to the unique tumor biology and microenvironment of the pancreas. Pancreatic tumors often induce systemic metabolic alterations, such as insulin resistance and impaired insulin secretion, which may not directly influence lymphatic dissemination. Additionally, the anatomical and physiological characteristics of the pancreas might modulate the impact of hyperglycemia on tumor spread differently than in other organs [43]. These insights suggest that while managing blood glucose levels is crucial for overall patient health and may influence postoperative outcomes, preoperative hyperglycemia should not be regarded as a reliable predictor of lymph node metastasis in this population [38]. Therefore, lymphatic involvement should be assessed using established pathological and imaging criteria, independent of glycemic status. Further research is warranted to elucidate the mechanisms underlying the distinct interactions between glucose metabolism and tumor progression in pancreatic cancer, which may differ from those in other malignancies.

The relationship between blood glucose levels and pancreatic cancer survival is complex and has been the subject of various studies with differing conclusions. Some research indicates that elevated blood glucose levels, even in the absence of a diabetes diagnosis, are associated with poorer outcomes in pancreatic cancer patients. For instance, a study involving 697 patients with advanced pancreatic cancer found that those with hyperglycemia (fasting blood glucose ≥ 7.0 mmol/L) had a median survival of 7.5 months, compared to 8.8 months for normoglycemic patients. Hyperglycemia was linked to increased mortality, with a hazard ratio of 1.38 [10]. Another study observed that high preoperative blood glucose levels were associated with a significantly shorter median overall survival (10.0 months) compared to patients with normal glucose levels (23.0 months). This suggests that preoperative hyperglycemia may be an independent predictor of poor prognosis in pancreatic cancer patients [37]. However, not all studies have found a significant association between blood glucose levels and pancreatic cancer survival. A meta-analysis encompassing 10 studies with a total of 48,424 patients concluded that preoperative and postoperative blood glucose levels, as well as HbA1c levels, were not significantly associated with overall survival in pancreatic cancer patients. The analysis did find that postoperative blood glucose levels could predict postoperative complications, but not survival [36]. Similarly, we noted that blood glucose levels were not associated with survival of the pancreas cancer patients in the present work. The impact of blood glucose levels on pancreatic cancer survival remains a topic of ongoing research. While some studies suggest that hyperglycemia may be associated with poorer outcomes, others have not found a significant link. These discrepancies may be due to differences in study design, patient populations, and definitions of hyperglycemia. Further research is needed to clarify the relationship and to determine whether managing blood glucose levels can improve survival outcomes in pancreatic cancer patients.

The limitations of our study include the fact that it was a single-center study and the number of patients was not very large.

5. Conclusion

Prolonged hospitalization after pancreatic cancer surgery is associated with increased mortality, influenced by factors like patient age, comorbidities, socioeconomic status, and the nature of the surgical procedure. Hence we suggest that proactive identification and management of these factors are essential to enhance postoperative outcomes and reduce mortality risks in this patient population.

Abbreviations

No Abbreviations

Author contributions

BO, SPO, FB: Conceptualization, Data curation, Methodology, Investigation, Writing—original draft, Formal analysis. BO, FK, MS, IK, GA, SPO: Conceptualization, Methodology, Writing—review & editing, Supervision. Both authors approved the final version of the manuscript.

Conflicts of interest

The authors have no conflicts of interest.

Ethical approval

The study was approved by the institutional ethics committee of the Abant İzzet Baysal University (approval date: 04.03.2025 and approval number: 111).

Consent to participate

All participants have given informed consent to participate in the study.

Consent to publication

Not applicable.

Availability of data and materials

The data used and/or analyzed during the current study are available upon reasonable request to the corresponding author.

Funding

Not applicable.

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