Caspian Journal of Internal Medicine 2026 (Spring); 17(2): 449-456
DOI: 10.22088/cjim.17.2.449

Original Article

Survival rate in patients with pancreatic cancer in Guilan province, Iran

Mohammad Almasi (MD) [1]
Niloofar Faraji (MSc) [1]
Farahnaz Joukar (PhD) [1]
Narges Eslami (MSc) [1]
Saman Maroufizadeh (PhD) [2]
Amineh Hojati (MD) [1*]
Fariborz Mansour-Ghanaei (MD) [1]

1. Gastrointestinal and Liver Diseases Research Center, Guilan University of Medical Sciences, Rasht, Iran
2. Department of Biostatistics, School of Health, Guilan University of Medical Sciences, Rasht, Iran

*** Correspondence:**
Amineh Hojati, Gastrointestinal and Liver Diseases Research Center, Guilan University of Medical Sciences, Rasht, Iran

Email: amineh.hojati.gi@gmail.com
Phone: +989113374813

Received: 20 Aug 2025
Revised: 28 Oct 2025
Accepted: 30 Oct 2025
Published: 10 March 2026

Abstract

Background: Pancreatic cancer is among the deadliest malignancies globally, marked by poor prognosis and limited survival. This research investigated the demographic and clinical determinants affecting overall survival in patients diagnosed with pancreatic cancer in Guilan Province, Iran.

Methods: A total of 50 pathologically and radiologically confirmed cases of pancreatic cancer were analyzed. Demographic, anthropometric, and clinical data were extracted from medical records. Survival probabilities were estimated using the Kaplan–Meier method, and intergroup differences were tested using the log-rank test. Cox proportional hazards modeling identified predictors of mortality, with significance defined at $p < 0.05$. Statistical analyses were conducted in SPSS (v16) and GraphPad Prism (v8).

Results: The majority of patients were male (60%) and over 60 years old (58%), while 46% had normal BMI and 40% had blood group O. Comorbidities were present in 42%, and 32% were diagnosed at stage IV. Mean and median survival were 39.6 and 9.0 months, respectively. One-, three-, and five-year survival rates were 45.2%, 31.3%, and 23.5%. Advanced tumor stage (III/IV) significantly predicted shorter survival ($p < 0.001$). Underweight individuals had higher mortality risk (HR = 2.79; 95% CI: 1.21–6.42; P = 0.016). In multivariate models, male gender, blood group O, and advanced disease remained independent negative prognostic indicators.

Conclusion: Male sex, advanced stage, low BMI, and blood group O were major determinants of poorer survival in pancreatic cancer. These results emphasize the need for individualized treatment and closer follow-up among high-risk groups.

Keywords: Pancreatic cancer, Survival determinants, Prognostic factors, Tumor stage.

Citation:
Almasi M, Faraji N, Joukar F, et al. Survival rate in patients with pancreatic cancer in Guilan province, Iran. Caspian J Intern Med 2026; 17(2): 449-456.

Pancreatic cancer is one of the most aggressive and lethal malignancies worldwide, characterized by late diagnosis, limited treatment options, and poor overall prognosis (1, 2). Despite representing only 2–3% of all new cancer cases, pancreatic cancer accounts for approximately 7% of all cancer-related deaths globally, underscoring its disproportionately high mortality rate (3, 4). In many countries, particularly those with aging populations and increasing prevalence of risk factors such as obesity and diabetes, the incidence of pancreatic cancer is on the rise, making it a growing public health concern (5,6). Survival rates for pancreatic cancer remain dismally low, with a 5-year survival rate of less than 10% in most populations (7, 8). The prognosis is often determined by a complex interplay of demographical and clinical factors. Several studies have reported that advanced age at diagnosis, male sex, and lower socioeconomic status are associated with worse survival outcomes (9, 10). Additionally, comorbid conditions such as diabetes mellitus, chronic pancreatitis, and obesity have been implicated not only in the development of pancreatic cancer but also in adversely affecting patient survival (11, 12).

Publisher: Babol University of Medical Sciences

Tumor-specific characteristics, such as histological subtype, tumor grade, size, lymph node involvement, and presence of distant metastases, further contribute to survival variability. Importantly, access to curative surgical resection, adjuvant chemotherapy, and timely diagnosis are among the strongest determinants of prolonged survival (13–16). Unlike other gastrointestinal cancers that have benefited from improved diagnostic and treatment modalities, pancreatic cancer remains stubbornly resistant to early detection and effective intervention (17–19). Despite ongoing advancements in imaging, molecular diagnostics, and targeted therapies, the heterogeneity of pancreatic cancer outcomes necessitates a deeper evaluation of these prognostic variables (20–22). Understanding the association between demographic and clinical characteristics and the survival rate of patients with pancreatic cancer is crucial for improving patient stratification, guiding treatment decisions, and identifying high-risk populations. In the current study, we evaluated the associations between various demographic and clinical factors with survival rates in patients diagnosed with pancreatic cancer, with the goal of contributing to more personalized and effective care strategies.

Methods

Study design and population: This cross-sectional, observational investigation was carried out on a group of 50 patients with a confirmed diagnosis of pancreatic cancer residing in Guilan Province, northern Iran. The study population was determined using Cochran's sample size estimation formula, based on the anticipated prevalence of pancreatic cancer within the regional population. A census sampling technique was adopted to include all eligible individuals meeting the inclusion criteria during the study period. Participants were required to be 18 years or older, have a definitive diagnosis of pancreatic malignancy confirmed through computed tomography (CT) imaging and fine-needle aspiration (FNA) cytology, and to have received their primary diagnosis and/or treatment within Guilan Province. Individuals were excluded if their clinical records were incomplete, if they had another concurrent primary cancer, if they underwent therapy outside the province, declined participation, or were expected to survive less than one month after diagnosis. Prior to enrollment, written informed consent was obtained from all participants in accordance with institutional and national ethical standards.

Data acquisition and variables: Data were systematically retrieved from hospital records and pathology reports using a structured checklist designed for this study. Collected information encompassed demographic details (age, sex, marital status, and ethnicity), anthropometric data (body mass index [BMI]), lifestyle habits (smoking, alcohol, or substance use), and clinical features (comorbidities, family cancer history, and disease stage).

BMI was classified according to World Health **Organization criteria**:

- Underweight: <18.5 kg/m²
- Normal: 18.5–24.9 kg/m²
- Overweight: 25–29.9 kg/m²
- Obese: ≥30 kg/m²

Blood type was recorded as A, B, AB, or O. Disease staging was based on radiological findings from CT scans, classified according to the American Joint Committee on Cancer (AJCC) system into stages I–IV. Other variables included the presence of chronic pancreatitis, tumor site (head vs. body/tail of the pancreas), and type of treatment administered categorized as no treatment, single-modality, or multimodal therapy (combination of surgery, chemotherapy, and/or radiotherapy).

Statistical procedures: Quantitative variables were expressed as means±standard deviation (SD) or medians with interquartile ranges (IQR) when appropriate. Categorical variables were reported as counts and percentages. The Kaplan–Meier estimator was used to calculate survival probabilities, and differences between groups were tested using the log-rank test. To assess prognostic factors influencing survival, both univariate and multivariate Cox proportional hazards regression analyses were conducted, yielding hazard ratios (HR) and 95% confidence intervals (CI). Variables with $p<0.1$ in univariate analysis were entered into the multivariate model. A two-tailed $p<0.05$ was considered statistically significant. All statistical analyses were performed using SPSS Version 16.0 (IBM Corp., Armonk, NY, USA), while GraphPad Prism Version 8.0.1 (GraphPad Software, San Diego, CA) was utilized for graphical visualization of survival curves.

Results

Demographic and clinical characteristics: Of the 50 enrolled patients, 58.0% were aged above 60 years, and 60.0% were males. All patients were married, and most (74.0%) belonged to the Gilak ethnic group. Based on BMI classification, 46.0% had normal weight, 28.0% were underweight, 10.0% overweight, and 16.0% obese. Regarding blood group distribution, type O was the most common (40.0%), followed by type A (28.0%), type B

(24.0%), and type AB (8.0%). Lifestyle assessment revealed that 30.0% of participants were smokers, 10.0% reported substance use, and 4.0% consumed alcohol. Comorbidities were found in 42.0% of participants, while 14.0% had a previous diagnosis of chronic pancreatitis. Furthermore, 44.0% had a family history of cancer, with 12.0% specifically reporting pancreatic cancer in first-degree relatives. Regarding disease stage, 14.0% were classified as stage I, 30.0% as stage II, 24.0% as stage III, and 32.0% as stage IV. Tumors most commonly originated in the head of the pancreas (78.0%). Concerning treatment, 22.0% received no therapy, 38.0% underwent a single treatment modality, and 40.0% received multimodal management (table 1).

Survival outcomes: The mean overall survival was 39.6 months (95% CI: 22.2–57.1), while the median survival was 9.0 months (95% CI: 1.8–16.2). Estimated one-, three-, and five-year survival rates were 45.2%, 31.3%, and 23.5%, respectively (figure 1). According to the log-rank test, survival time did not significantly differ by age group, although patients older than 60 years tended to have shorter survival ($p>0.05$). Underweight patients experienced significantly worse survival compared to those with normal BMI (P=0.038). Participants with blood type O had lower survival probabilities, but the difference did not reach statistical significance (P=0.068). Tumor stage was strongly related to prognosis: individuals with advanced disease (stage III/IV) had significantly shorter survival compared to those with early-stage tumors (stage I/II, $p<0.001$) (figure 2; table 1).

Univariate and multivariate regression analyses: In the univariate Cox model, older age (>60 years) was associated with a 1.79-fold increase in mortality risk (HR=1.79, 95% CI: 0.87–3.64, P=0.111). Underweight status significantly elevated mortality risk compared to normal BMI (HR=2.79, 95% CI: 1.21–6.42, P=0.016). Blood group O conferred an 85% higher risk of death (HR=1.85, 95% CI: 0.93–3.68, P=0.082). Patients with advanced disease (stage III/IV) showed a more than threefold higher mortality risk compared with early stages (HR=3.53, 95% CI: 1.62–7.70, $p<0.001$) (table 1).

After adjustment in the multivariate model, male gender remained an independent predictor of higher mortality (HR=3.38, 95% CI: 1.26–9.10, P=0.016). Blood group O was also independently associated with shorter survival (HR=2.86, 95% CI: 1.10–7.42, P=0.031). Although patients with a family history of cancer had an increased mortality risk (HR=2.46, 95% CI: 0.94–6.48, P=0.068), this association did not reach statistical significance. Interestingly, current smokers demonstrated a trend toward lower mortality (HR=0.36, 95% CI: 0.11–1.20, P=0.095). Among all variables, disease stage remained the strongest independent prognostic factor; patients in stage III/IV had a 3.32-fold higher risk of death compared with those in stages I/II (HR=3.32, 95% CI: 1.16–9.46, P=0.025) (table 2).

Table 1. Comparison of survival time in patients with pancreatic cancer across different levels of demographic and clinical variables using the Log-rank test.

Variable		Median (IQR, 95% CI)	P-value
Age	≥ 60	22.0 (0.0 – 52.6)	0.097
	< 60	7.0 (3.7 – 10.3)	
Gender	Male	7.0 (2.2 – 11.8)	0.179
	Female	20.0 (0.0 – 52.3)	
Ethnicity	Gilak	8.0 (2.8 – 13.2)	0.323
	Non-Gilak	22.0 (0.0 – 53.6)	
BMI (kg/m²)	Normal	38.0 (0.0 – 80.8)	0.038
	Underweight	5.0 (0.0 – 11.5)	
	Overweight/Obese	6.0 (0.0 – 17.7)	
Blood Group	A/B/AB	20.0 (0.0 – 40.9)	0.068
	O	8.0 (4.3 – 11.7)	
Smoking	No	10.0 (2.6 – 17.4)	0.975
	Yes	9.0 (0.0 – 19.2)	
Drug Use	No	8.0 (2.9 – 13.1)	0.139
	Yes	120.0 (NA – NA)	

Variable		Median (IQR, 95% CI)	P-value
Alcohol Use	No	9.0 (4.1 – 13.9)	0.457
	Yes	4.0 (NA – NA)	
Underlying Disease	No	5.0 (0.0 – 31.5)	0.936
	Yes	11.0 (3.5 – 18.5)	
Chronic Pancreatitis	No	8.0 (3.3 – 12.7)	0.422
	Yes	120.0 (NA – NA)	
Family History of Cancer	No	14.0 (0.0 – 31.3)	0.288
	Yes	8.0 (2.8 – 13.2)	
Disease Stage	I / II	38.0 (16.0 – 60.0)	< 0.001
	III / IV	5.0 (3.5 – 6.5)	
Tumor Location	Head	9.0 (3.6 – 14.4)	0.744
	Other	7.0 (0.0 – 20.9)	
Treatment Type	No Treatment	20.0 (NA – NA)	0.339
	Monotherapy	8.0 (0.9 – 15.1)	
	Multimodal Therapy	11.0 (0.0 – 29.5)	

Interquartile range (IQR); confidence interval (CI); body mass index (BMI). P-value according to the Log-rank test.

Table 2. Univariate and multivariate analyses of demographic and clinical factors associated with survival in patients with pancreatic cancer.

Variable		Univariate Analysis HR (95% CI)	P-value	Multivariate Analysis aHR (95% CI)	P-value
Age	≤ 60 (reference)	1	–	1	–
	> 60	1.79 (0.87–3.64)	0.111	0.53 (0.19–1.51)	0.234
Gender	Male	1.60 (0.79–3.24)	0.196	3.38 (1.26–9.10)	0.016
	Female (reference)	1	–	1	–
Ethnicity	Gilak (reference)	1	–	1	–
	Non-Gilak	0.66 (0.29–1.54)	0.339	1.12 (0.40–3.16)	0.833
BMI (kg/m²)	Normal (reference)	1	–	1	–
	Underweight	2.79 (1.21–6.42)	0.016	1.77 (0.57–5.46)	0.324
	Overweight/Obese	1.72 (0.73–4.05)	0.218	1.38 (0.48–3.93)	0.552
Blood Group	A/B/AB (reference)	1	–	1	–
	O	1.85 (0.93–3.68)	0.082	2.86 (1.10–7.42)	0.031
Smoking		1.01 (0.47–2.18)	0.976	0.36 (0.11–1.20)	0.095
Drug Use		0.25 (0.03–1.90)	0.181	0.27 (0.03–2.84)	0.275
Alcohol Use		1.69 (0.40–7.14)	0.475	0.92 (0.11–7.78)	0.941
Comorbidities		0.97 (0.50–1.91)	0.937	0.60 (0.25–1.43)	0.248
Chronic Pancreatitis		0.62 (0.19–2.06)	0.439	0.55 (0.14–2.12)	0.388
Family History of Cancer		1.43 (0.72–2.85)	0.305	2.46 (0.94–6.48)	0.068
Disease Stage	I/II (reference)	1	–	1	–
	III/IV	3.53 (1.62–7.70)	<0.001	3.32 (1.16–9.46)	0.025
Tumor Location	Head (reference)	1	–	1	–
	Other	1.14 (0.51–2.53)	0.751	0.73 (0.27–2.01)	0.544
Type of Treatment	No treatment (reference)	1	–	1	–
	Monotherapy	0.82 (0.32–2.15)	0.692	0.53 (0.14–2.05)	0.359
	Multimodal Therapy	0.53 (0.19–1.44)	0.212	0.30 (0.07–1.29)	0.106

Body mass index (BMI); hazard ratio (HR); confidence interval (CI)

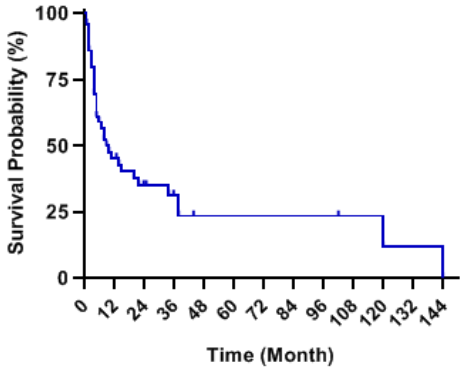

Figure 1. The survival probability of patients with pancreatic cancer was estimated and analyzed.

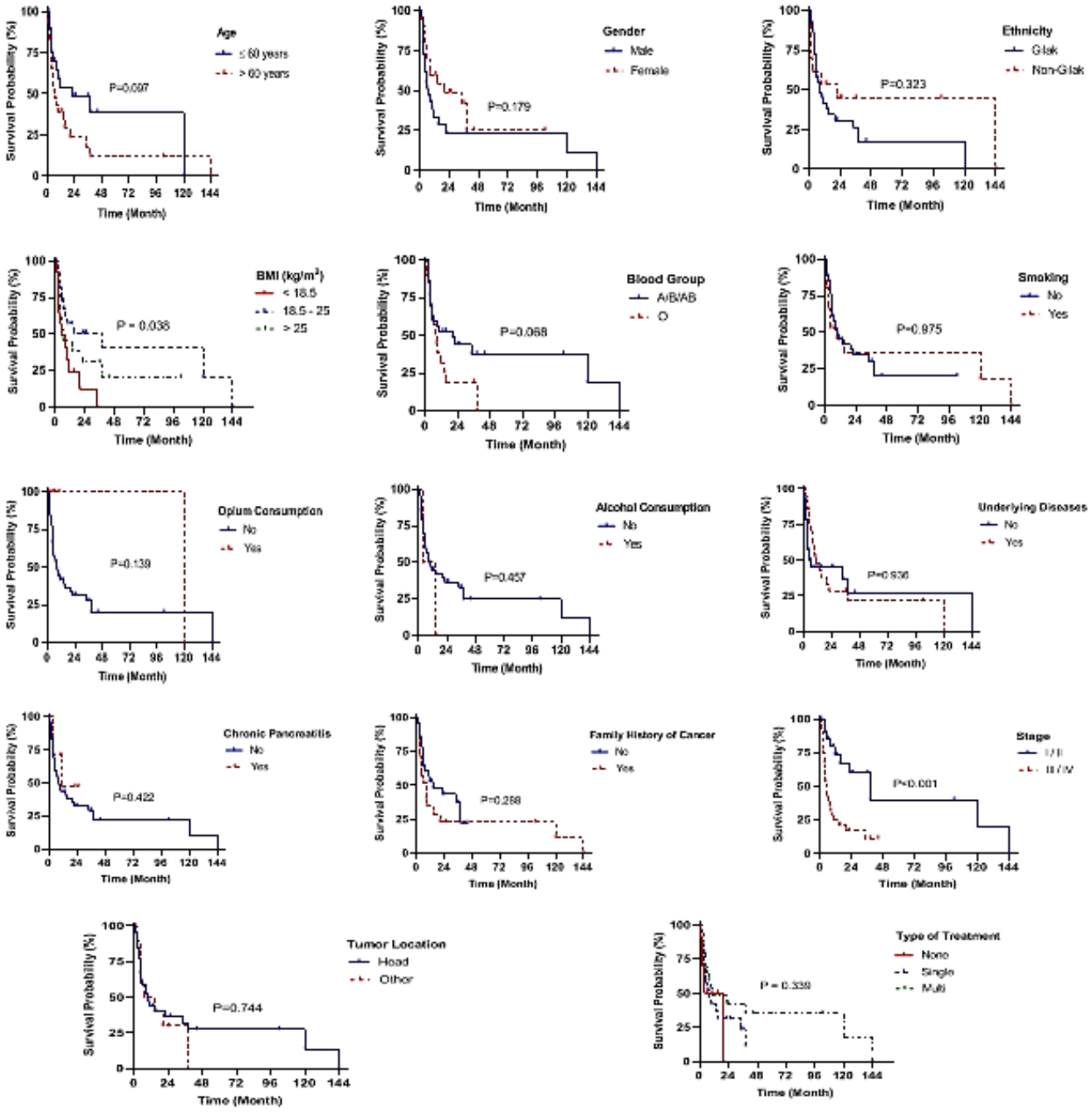

Figure 2. The survival probability of patients with pancreatic cancer according to the variables using log-rank test and Kaplan–Meier method.

Discussion

The exact causes of pancreatic cancer remain unclear (23), though risk factors like smoking, diabetes, obesity, alcohol use, chronic pancreatitis, non-O blood type, and family history are known contributors. While general population screening is ineffective, targeted approaches in high-risk groups are being explored (6, 24). This study, identified key demographic and clinical predictors of survival among patients with pancreatic cancer in Guilan Province, Iran. Understanding the demographic, clinical, and laboratory risk factors associated with pancreatic cancer is crucial for improving risk stratification, guiding personalized treatment decisions, and enhancing overall clinical management and patient outcomes. We observe that most patients with pancreatic cancer were over 60 ears and males. Pancreatic cancer incidence is higher in older adults and men. The risk increases with age because pancreatic carcinogenesis is a cumulative process influenced by long-term exposure to carcinogens, age-related genetic mutations, and declining immune surveillance (6). Higher pancreatic cancer incidence and poorer outcomes in men may be due to a combination of biological differences, such as genetic mutations, and gender-related factors like greater exposure to risk factors (e.g., smoking, obesity) and disparities in healthcare access or treatment responses (6, 25, 26). However, Gehrels et al. observed that females had worse overall survival compared to males with pancreatic cancer (10). The mean survival time of approximately 39.6 months and a median survival of nine months observed in our population, confirming the aggressive nature of pancreatic cancer and its dismal outcomes even with modern therapeutic advances. Farokhi et al. represented a case of primary squamous cell carcinoma of the pancreas achieving 13 months overall survival, nearly double the typical 7-month median (22). Another study by Bakasa and Viriri reported that survival rates for pancreatic ductal adenocarcinoma remain extremely low, with approximately 2% reaching five-year survival (27). Another study by Li et al. found the overall rate of 10.5% for 5-year survival rates in these patients (28). However, the reported five-years survival rates in our study was 23.5%, which is higher than worldwide rate of 11% for pancreatic patients (29). These differences may stem from variations in patient demographics, healthcare access, stage at diagnosis, treatment modalities, and genetic or environmental factors specific to the studied population. Nonetheless, even with relatively higher rates in our study, pancreatic cancer continues to demonstrate a dismal prognosis worldwide, underscoring the urgent need for improved early detection and more effective therapies. Consistent with existing literature, our study found that advanced tumor stage (III/IV) was a significant independent predictor of reduced survival. Patients with late-stage disease had over an increased risk of mortality compared to those diagnosed at earlier stages (30–32). While, early detection efforts remain paramount and may significantly alter survival trajectories, late presentation limits therapeutic options predominantly to palliative care, leading to poor survival outcomes (33). A study by Blackford et al. demonstrated that patients diagnosed at an earlier tumor stage tend to have significantly better survival outcomes compared to those diagnosed at more advanced stages. Individuals under surveillance were more likely to be identified at an earlier stage, with smaller tumors, and consequently showed improved long-term survival and overall prognosis (30). Nutritional status is another crucial predictor for cancer patients' survival. In the current study, underweight patients exhibited a nearly threefold increased mortality risk compared to those with normal BMI. Similarly, Poulia et al. demonstrated that poor nutritional status and related symptoms were associated with worse progression-free and overall survival in patients with pancreatic cancer (34). Related mechanisms, including cancer-associated cachexia, impaired immune function, poor tolerance to treatment, and decreased physical performance. These conditions are often exacerbated by gastrointestinal dysfunction, which promote muscle wasting and metabolic imbalance (35, 36). Together, they weaken the patient's overall condition, limit therapeutic options, and accelerate disease progression, highlighting the need for early nutritional assessment and intervention to improve outcomes and support quality of life in affected individuals.An intriguing finding of this study was the independent association between blood group O and increased mortality risk in pancreatic cancer patients. While previous evidence on ABO blood groups and pancreatic cancer risk has suggested that individuals with non-O blood groups had a higher risk of developing pancreatic cancer, and this association was more pronounced among secretors compared to non-secretors, while no interaction was observed between ABO and Lewis antigens (37, 38). Non-O blood groups may promote tumor growth and spread by influencing inflammation and immune responses through markers. ABO antigens, expressed on gastrointestinal cells, can alter infection susceptibility and the tumor microenvironment. Their differential expression in normal and cancerous tissues suggests a potential role as prognostic biomarkers (39, 40). Differences in study design, population traits, outcome measures, genetic backgrounds, environmental factors, and healthcare access may explain the discrepancy in findings. In the current study, no

significant association was observed between age, ethnicity, smoking, drug and alcohol use, comorbidities, chronic pancreatitis, tumor location, treatment type, and family history of cancer with survival rate in pancreatic cancer. This suggests that, within this population, these factors may not independently influence prognosis or may be overshadowed by more dominant prognostic determinants. Moreover, the absence of significant associations may be attributed to factors such as limited sample size, population variability, or the influence of unmeasured confounders, highlighting the challenges of accurately predicting survival in pancreatic cancer based solely on demographic or clinical parameters. Further studies on a larger scale, incorporating both retrospective and prospective designs, are warranted to validate these findings, identify additional prognostic markers, and enhance our understanding of the complex factors influencing survival outcomes in pancreatic cancer. Our findings confirmed the poor prognosis associated with pancreatic cancer and identifies advanced disease stage (III/IV), underweight status, male sex, and blood group O as significant independent predictors of increased mortality. These findings provide valuable insights into key prognostic factors that can guide personalized treatment strategies and improve patient management in pancreatic cancer.

Acknowledgments

This study was driven from gastroenterology fellowship thesis by Dr. Mohammad Almasi.

Funding: Self-funding.

Ethics approval: The research protocol received ethical approval from the Ethics Committee of Guilan University of Medical Sciences (approval ID: IR.GUMS.REC.1403.386).

Conflict of interests: The authors declare that they have no competing interests.

Authors' contribution: M.A contributed to conceptualization, validation, investigation, methodology, and was involved in both the original draft writing and manuscript review and editing. N.F and N.E participated in investigation, visualization, and also contributed to writing the original draft and reviewing and editing the manuscript. F.J was responsible for investigation, methodology, and contributed to the original draft. S.M contributed to validation, data curation, formal analysis, and methodology. A.H and F.M played a central role in conceptualization, data curation, resources, supervision, validation, visualization, and contributed to both the original draft and the review and editing process.

References

1. Zhao Z, Liu W. Pancreatic cancer: a review of risk factors, diagnosis, and treatment. Technol Cancer Res Treat 2020; 19: 1533033820962117.
2. Leiphrakpam PD, Chowdhury S, Zhang M, et al. Trends in the global incidence of pancreatic cancer and a brief review of its histologic and molecular subtypes. J Gastrointest Cancer 2025; 56: 71.
3. Luo W, Wang J, Chen H, et al. Epidemiology of pancreatic cancer: New version, new vision. Chin J Cancer Res 2023; 35: 438–50.
4. Zottl J, Sebesta CG, Tomosel E, Sebesta MC, Sebesta C. Unraveling the burden of pancreatic cancer in the 21st century: Trends in incidence, mortality, survival, and key contributing factors. Cancers (Basel) 2025; 17: 1607.
5. Shou S, Liu R, He J, et al. Current and projected incidence rates of pancreatic cancer in 43 countries: an analysis of the Cancer Incidence in Five Continents database. BMJ Open Gastroenterol 2025; 12: e001544.
6. Rawla P, Sunkara T, Gaduputi V. Epidemiology of pancreatic cancer: Global trends, etiology and risk factors. World J Oncol 2019; 10: 10–27.
7. Gyawali B, Booth CM. Treatment of metastatic pancreatic cancer: 25 years of innovation with little progress for patients. Lancet Oncol 2024; 25: 167–70.
8. Dreyer SB, Beer P, Hingorani SR, Biankin AV. Improving outcomes of patients with pancreatic cancer. Nat Rev Clin Oncol 2025; 1–18.
9. Qadir RMAB, Umair MB, Tariq UB, et al. Unraveling pancreatic cancer: Epidemiology, risk factors, and global trends. Cureus 2024; 16: e72816.
10. Gehrels AM, Wagner AD, Besselink MG, et al. Gender differences in tumor characteristics, treatment allocation and survival in stage I–III pancreatic cancer: a nationwide study. Eur J Cancer 2024; 206: 114117.
11. Ruze R, Song J, Yin X, et al. Mechanisms of obesity- and diabetes mellitus-related pancreatic carcinogenesis: a comprehensive and systematic review. Signal Transduct Target Ther 2023; 8: 139.
12. Allen T, Sheikhkoni N, Gonzalez AF, et al. A comprehensive diagnostic and treatment framework for pancreatic cancer patients with comorbidities. J Med Res Case Reports 2025; 7: 1-9.
13. Keefer J, Cooper M, Pacioles T, Arrington A. Long-term survival after metastatic pancreatic cancer. Marshall J Med 2024; 10: 3.
14. Blomstrand H, Olsson H, Green H, Björnsson B, Elander NO. Impact of resection margins and para-aortic lymph node metastases on recurrence patterns and prognosis in

resectable pancreatic cancer – a long-term population-based cohort study. HPB (Oxford) 2023; 25: 1531–44.
15. McGuigan A, Kelly P, Turkington RC, et al. Pancreatic cancer: A review of clinical diagnosis, epidemiology, treatment and outcomes. World J Gastroenterol 2018; 24: 4846.
16. Ma SJ, Oladeru OT, Miccio JA, et al. Association of timing of adjuvant therapy with survival in patients with resected stage I to II pancreatic cancer. JAMA Netw Open 2019; 2: e199126.
17. Jamal MH, Khan MDN. Developments in pancreatic cancer emerging therapies, diagnostic methods, and epidemiology. Pathol Res Pract 2025; 271: 156012.
18. Faraji N, Mashkoor NR, Emamifar A, et al. The cytotoxic effect of cobalt oxide nanoparticle conjugated by menthol on colorectal cancer cell line and evaluation of the expression of CASP8 and FEZF1-AS1. J Clust Sci 2025; 36: 39.
19. Faraji N, Almasi M, Mirmazloumi M, et al. Altered expression patterns of lncRNA MEG3 and LINC01611 in patients with colorectal cancer. Hum Gene 2025; 201470.
20. Pang XX, Xie L, Yao WJ, et al. Advancements of molecular imaging and radiomics in pancreatic carcinoma. World J Radiol 2023; 15: 10–19.
21. Hu X, Wang Z, Zhu Y, et al. Advancements in molecular imaging for the diagnosis and treatment of pancreatic ductal adenocarcinoma. Nanoscale Adv 2025; 7: 2887-2903.
22. Farokhi P, Sadeghi A, Moghaddas A, Heidarpour M, Dinari S. Primary squamous cell carcinoma, a rare pathological report of pancreatic cancer. Casp J Intern Med 2021; 12: 407–12.
23. Stoffel EM, Brand RE, Goggins M. Pancreatic cancer: Changing epidemiology and new approaches to risk assessment, early detection, and prevention. Gastroenterology 2023; 164: 752–65.
24. Pang Y, Holmes MV, Chen Z, Kartsonaki C. A review of lifestyle, metabolic risk factors, and blood-based biomarkers for early diagnosis of pancreatic ductal adenocarcinoma. J Gastroenterol Hepatol 2019; 34: 330–45.
25. Logan K, Pearson F, Kenny RPW, Pandanaboyana S, Sharp L. Are older patients less likely to be treated for pancreatic cancer? A systematic review and meta-analysis. Cancer Epidemiol 2022; 80: 102215.
26. Pijnappel EN, Schuurman M, Wagner AD, et al. Sex, gender and age differences in treatment allocation and survival of patients with metastatic pancreatic cancer: A nationwide study. Front Oncol 2022; 12: 839779.
27. Bakasa W, Viriri S. Pancreatic cancer survival prediction: A survey of the state-of-the-art. Comput Math Methods Med 2021; 2021: 1188414.
28. Li Q, Feng Z, Miao R, et al. Prognosis and survival analysis of patients with pancreatic cancer: retrospective experience of a single institution. World J Surg Oncol 2022; 20: 11.
29. Huang B, Huang H, Zhang S, et al. Artificial intelligence in pancreatic cancer. Theranostics. 2022; 12: 6931–54.
30. Blackford AL, Canto MI, Dbouk M, et al. Pancreatic cancer surveillance and survival of high-risk individuals. JAMA Oncol 2024; 10: 1087–96.
31. Keyl J, Kasper S, Wiesweg M, et al. Multimodal survival prediction in advanced pancreatic cancer using machine learning. ESMO Open 2022; 7: 100555.
32. Patel VR, Adamson AS, Liu JB, Welch HG. Increasing incidence and stable mortality of pancreatic cancer in young Americans. Ann Intern Med 2024; 178: 142–4.
33. Hawkes N. Cancer survival data emphasise importance of early diagnosis. BMJ 2019; 364: l408.
34. Poulia KA, Antoniadou D, Sarantis P, Karamouzis MV. Pancreatic cancer prognosis, malnutrition risk, and quality of life: A cross-sectional study. Nutrients 2022; 14: 442.
35. Kordes M, Larsson L, Engstrand L, Löhr JM. Pancreatic cancer cachexia: three dimensions of a complex syndrome. Br J Cancer 2021; 124: 1623–36.
36. Lee DU, Hastie DJ, Fan GH, et al. Effect of malnutrition on the postoperative outcomes of patients undergoing pancreatectomy for pancreatic cancer: Propensity score–matched analysis of 2011–2017 US hospitals. Nutr Clin Pract 2022; 37: 117–29.
37. Wolpin BM, Chan AT, Hartge P, et al. ABO blood group and the risk of pancreatic cancer. J Natl Cancer Inst 2009; 101: 424–31.
38. Kim J, Yuan C, Amundadottir LT, et al. Relationship between ABO Blood Group Alleles and Pancreatic Cancer Is Modulated by Secretor (FUT2) Genotype, but Not Lewis Antigen (FUT3) Genotype. Cancer Epidemiol biomarkers Prev a Publ Am Assoc Cancer Res cosponsored by Am Soc Prev Oncol 2023; 32: 1242–8.
39. Albadrani HM, Hamed M, Zakariyah A, et al. Differential expression of ABO in normal and tumor tissues: Implications for cancer biology and prognosis. J Taibah Univ Med Sci 2024; 19: 1132–42.
40. Hasani F, Hosseini SS. Breaking barriers: Unleashing the potential of ABO blood group antigen therapy in the battle against solid tumors. In: Adunyah SE, editor. Rijeka: Intech Open 2023; Available from: DOI: 10.5772/intechopen.1003886. Accessed 21 Jun, 2026.