Original article

Clinical features, biological characteristics and prognostic factors in patients with pancreatic cancer

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Abstract

Background: Pancreatic cancer is a rapidly progressive disease with a low 5-year survival rate, an increasing incidence, and substantial difficulty in diagnosis at the early stages. Early identification of suspected pancreatic cancer and referral for imaging and histopathologic sampling are therefor extremely important. The objectives of this study were: 1) to characterize clinical features and biological characteristics; and 2) To evaluate overall survival and identify prognostic factors associated with mortality.

Patients and Methods: This was a prospective descriptive study conducted in patients with suspected pancreatic cancer who underwent endoscopic ultrasound-guided fine-needle biopsy (EUS-FNB) at the Endoscopy Department, Cho Ray Hospital.

Results: A total of 89 patients were diagnosed with pancreatic cancer. Abdominal pain was the most frequent presenting symptom (92.1%). Hyperglycemia at the time of diagnosis was observed in 57.3% of patients. Elevated CA19-9 and CEA levels were found in 80.9% and 44.9% of cases, respectively. The mean overall survival from diagnosis was 4.44 ± 2.62 months. Patients receiving chemotherapy had significantly longer overall survival compared with those receiving non-specific treatment ($P < 0.001$). Tumor size, vascular invasion, and distant metastasis were identified as independent prognostic factors for mortality ($P < 0.05$).

Conclusions: The clinical presentation of pancreatic cancer is generally nonspecific, with abdominal pain being the most common symptom. Tumor size, vascular invasion, and distant metastasis are independent prognostic factors in patients with pancreatic cancer.

Keywords: *pancreatic cancer; clinical features, biological features of pancreatic cancer; mortality prognosis in pancreatic cancer.*

1. INTRODUCTION

Pancreatic cancer is one of the most aggressive and lethal malignancies, characterized by rapid progression and poor outcomes. Although its incidence is relatively low compared with other cancers, pancreatic cancer carries a disproportionately high mortality rate. At the time of diagnosis, most patients present with advanced diseases, frequently accompanied by local invasion or distant metastasis. According to Rawla P et al., pancreatic cancer has a 1-year survival rate of approximately 24% and a 5-year survival rate of only 9% [1].

Conversely, early-stage pancreatic cancer (tumor size ≤ 2 cm, without local invasion) that is appropriately treated is associated with markedly improved 5-year survival rates, reaching 68.7% for stage IA and 85.8% for stage 0 [2]. These findings underscore the pivotal role of early diagnosis in improving treatment outcomes and prolonging survival in patients with pancreatic cancer.

However, the early detection of pancreatic cancer remains a major clinical challenge. The etiology of the disease has not yet been fully elucidated, and early-stage pancreatic cancer is often characterized by subtle, nonspecific clinical manifestations that are easily mistaken for other benign gastrointestinal disorders. Consequently, the proportion of patients diagnosed at an early stage remains very low. In a large pooled analysis by Yoshida T et al. involving 2.490 patients with pancreatic cancer, only 0.8% of patients had tumor size less than 10 mm at the time of diagnosis [3].

In this context, identifying clinical and biological characteristics that may indicate early-stage pancreatic cancer, as well as determining prognostic factors associated with patient survival, has become an urgent priority. Such data not only improve the effectiveness of early diagnosis but also help guide appropriate treatment strategies, facilitate

personalized care, and ultimately enhance patient prognosis.

Based on these considerations, this study was conducted to:

1. Characterize clinical features and biological characteristics in patients with pancreatic cancer.

2. To evaluate overall survival and identify prognostic factors associated with mortality in patients with pancreatic cancer.

2. PATIENTS AND METHODS

2.1. Study Population

Patients with suspected pancreatic cancer who underwent endoscopic ultrasound–guided fine-needle biopsy (EUS-FNB) at the Endoscopy Unit, Cho Ray Hospital, between April 2024 and January 2025 were enrolled in this study.

2.2. Study Design and Methods

Study design: This was a prospective, descriptive study.

Exclusion criteria: Patients were excluded if they had previously undergone partial pancreatectomy; had hemodynamic instability or severe cardiopulmonary disease precluding EUS; had upper gastrointestinal conditions preventing complete pancreatic evaluation; had uncorrected coagulopathy or were receiving antithrombotic therapy; or declined participation.

Study equipment and materials:

Clinical and paraclinical data, including hematological and biochemical tests, computed tomography (CT), and magnetic resonance imaging (MRI).

Endoscopic ultrasound systems: Olympus EVIS EXERA III 190, EU-ME2, equipped with a linear-array echoendoscope (GF-UCT180).

EUS-FNB needles were available at the Department of Endoscopy, including 19-gauge, 20-gauge, and 22-gauge biopsy needles.

Study procedure:

All patients underwent clinical evaluation, laboratory testing, and imaging studies, and indications for EUS-FNB were confirmed by the Endoscopy Unit. Written informed consent was obtained prior to the procedure, and patients fasted for at least 8 hours.

EUS-guided fine-needle biopsy was performed to obtain tissue samples, which were submitted for histopathological analysis. Patients were monitored for at least 2 hours after the procedure for immediate adverse events. Inpatients were returned to their wards and outpatients were discharged if clinically stable.

Procedure-related complications were assessed at 24 hours by direct clinical evaluation for inpatients or telephone follow-up for outpatients. Patients with histologically confirmed pancreatic cancer were included in the final analysis. Overall survival was monitored starting from the first patient who underwent EUS-FNB, with follow-up completed 6 months after the last patient underwent the procedure.

2.3. Statistical Analysis

Statistical analyses were performed using SPSS version 25.0 (IBM SPSS Statistics for Windows, IBM Corp., Armonk, NY, USA). Categorical variables were presented as frequencies and percentages. The normality of continuous variables was assessed using the Kolmogorov–Smirnov test. Normally distributed continuous variables were expressed as mean ± standard deviation (SD), whereas non-normally distributed variables were presented as median values.

The association between two continuous variables was evaluated using correlation analysis and expressed as the correlation coefficient (r). Pearson's correlation coefficient was used for normally distributed variables, while Spearman's rank correlation coefficient (rho) was applied for variables with non-normal distribution. Corresponding p values were reported.

Survival analysis was performed using the Kaplan–Meier method. Prognostic factors associated with overall survival were identified using the Cox proportional hazards regression model. A two-sided p value < 0.05 was considered statistically significant.

2.4. Ethical Considerations

The study protocol was approved by the Institutional Review Board/Ethics Committee for Biomedical Research of the University of Medicine and Pharmacy, Hue University (approval number: H2024/012).

3. RESULTS

A total of 89 patients with pancreatic cancer were diagnosed and followed during the study period. The cohort comprised 52.8% men and 47.2% women, with a mean age of 63.7 ± 10.5 years.

3.1. Clinical and biological characteristics of patients with pancreatic cancer

Table 1. Medical history and risk factors of patients with pancreatic cancer

Medical history and risk factors	n	%
Heavy smoking	38	42.7
Heavy alcohol consumption	17	19.1
History of chronic pancreatitis	8	9.0
History of diabetes mellitus	18	20.2
Other comorbidities	29	32.9
Patients without identified medical history or risk factors (female)	39 (29)	43.8 (32.6)

Among the patients, 42.7% had a history of heavy smoking, 19.1% reported heavy alcohol consumption, 20.2% had a history of diabetes mellitus, and 9.0% had a history of chronic pancreatitis. Other comorbidities were reported in 32.9% of patients, most commonly hypertension; one patient had a history of uterine fibroid surgery and one had ureteral calculi. Notably, 39 patients (43.8%) had no identifiable medical history or risk factors, including 29 women and 10 men.

Table 2. Clinical characteristics of patients with pancreatic cancer

Clinical factors	n	%
Abdominal pain	82	92.1
Nausea	10	11.2
Fatigue	60	67.4
Anorexia	60	67.4
Weight loss	59	66.3
Jaundice	19	21.3
Other symptoms	15	16.9
Mean duration of abdominal pain (weeks)	6.91 ± 4.79	
Mean weight loss (kg) Median (minimum–maximum)	4.21 ± 4.26 3.00 (0.00 – 15.00)	

Abdominal pain was the most common presenting symptom, reported in 92.1% of patients, with a mean duration of 6.91 ± 4.79 weeks. Other frequent symptoms included fatigue, anorexia, and weight loss, with a mean weight loss of 4.21 ± 4.26 kg; the maximum recorded weight loss was 15 kg over 12 weeks. In addition, 16.9% of patients presented with other symptoms, most commonly pruritus and dark-colored urine, and fever was observed in 3 patients.

Table 3. Baseline blood biochemical parameters

Blood biochemical characteristics	n	%
Glucose		
Elevated fasting glucose (> 110 mg/dL)	51	57.3
Normal fasting glucose	38	42.7
Albumin		
Hypoalbuminemia (< 3.5 g/dL)	8	9.0
Normal (≥ 3.5 g/dL)	81	91.0
Protein		
Decreased total protein (< 6 g/dL)	9	10.1
Normal total protein (≥ 6 g/dL)	80	89.9
AST		
Elevated (> 48 U/L)	33	37.1
Normal (≤ 48 U/L)	56	62.9
ALT		
Elevated (> 49 U/L)	26	29.2
Normal (≤ 49 U/L)	63	70.8
Bilirubin total		
Increased (> 1 mg/dL)	34	38.2
Normal (≤ 1 mg/dL)	55	61.8
Bilirubin direct		
Increased (> 0.2 mg/dL)	66	74.2
Normal (≤ 0.2 mg/dL)	23	25.8

At diagnosis, 57.3% of patients had hyperglycemia, whereas hypoalbuminemia and decreased total protein levels were observed in 9.0% and 10.1%, respectively. Elevated AST and ALT levels were present in 37.1% and 29.2% of patients, and increased direct bilirubin was observed in 74.2%.

Table 4. Baseline tumor marker levels

Tumor markers	n	%
CEA		
Elevated (≥ 5 ng/mL)	40	44.9
Normal (< 5 ng/mL)	49	55.1
Mean ± SD	103.88 ± 590.63	
Median (minimum–maximum)	4.50 0.50-5172.40	
CA 19-9		
Elevated (≥ 35IU/ml)	72	80.9
Normal (< 35IU/ml)	17	19.1
Mean ± SD	4555.76 ± 10901.41	

Median (minimum–maximum)	320.40 0.60 – 52866.60

Elevated CEA levels were observed in 44.9% of patients, with a median value of 4.5 ng/mL, whereas elevated CA 19-9 levels were present in 80.9% of patients, with a median value of 320.4 IU/mL.

3.2. Endoscopic ultrasound characteristics of pancreatic cancer

Table 5. Tumor characteristics, metastasis, and vascular invasion on endoscopic ultrasound

Endoscopic ultrasound characteristics		n	%
Lymph node metastasis	Lymph node	31	34.8
	No lymph node metastasis	58	65.2
Vascular invasion	Vascular invasion	38	42.7
	No vascular invasion	51	57.3
Distant metastasis	Liver metastasis	12	13.5
	No distant metastasis	75	84.3
	Peritoneal metastasis	2	2.2
Mean tumor size ± SD (mm)		36.82 ± 9.67	
Median (minimum–maximum)		35.00 (16.00 - 68.00)	

EUS identified lymph node metastasis in 34.8% of patients, vascular invasion in 42.7%, and distant metastasis in 15.7%, most commonly to the liver; peritoneal metastasis was observed in 2 cases. The mean tumor size on EUS was 36.82 ± 9.67 mm.

3.3. Survival time and associated factors in patients with pancreatic cancer

Table 6. Survival and treatment outcomes from the time of diagnosis

Survival status	n	%
Alive	7	7.9
Deceased	78	87.6
Lost to follow-up	4	4.5
Total	89	100.0
Mean of survival time ± SD	4.44 ± 2.62	
Median (minimum–maximum)	4.00 (1 - 13)	
Treatment modality		
Chemotherapy	41	46.1
Surgery	2	2.2
No specific treatment	42	47.2
Lost to follow-up	4	4.5
Total	89	100.0

Patients were followed from the time of diagnosis until the end of the study. The follow-up period extended from the first EUS-FNB procedure to 6 months after the last EUS-FNB case. Overall, 87.6% of patients died during follow-up, 7.9% were alive, and 4 patients were lost to follow-up after 1 month. The mean overall survival from diagnosis was 4.44 ± 2.62 months, ranging from 1 to 13 months. Among patients with available follow-up data, 46.1%received chemotherapy, 47.2%received no specific treatment, and 2.2% underwent surgery.

Table 7. Cumulative overall survival time

Time	Overall survival rate, %
3 months	61.5
6 months	21.3
12 months	2.7

The overall survival rates were 61.5% at 3 months and 21.3% at 6 months; only 2.7% of patients survived beyond 12 months.

Table 8. Association of clinical, biochemical, and tumor-related factors with overall survival

Factors	Overall survival	
	r	P- value
Age	0.033	0.763
Weight loss	0.054	0.623
Time of weight loss	-0.009	0.934
Protein	**0.282**	**0.009**
Albumin	**0.304**	**0.005**
CA19-9	**-0.279**	**0.010**
CEA	-0.169	0.123
Tumor size on EUS	**-0.408**	**<0.001**

Overall survival was weakly positively correlated with total protein levels and moderately positively correlated with albumin levels. Conversely, overall survival showed a weak inverse correlation with CA 19-9 levels and a moderate inverse correlation with tumor size on EUS. No significant correlations were observed between overall survival and age, weight loss or CEA levels.

Table 9. Overall survival according to treatment modality

Treatment modality	n	Deceased	OS (3 months)	P- value
Chemotherapy	41	36	80.5%	< 0.001
Surgery	2	0	NA	
Non-specific treatment	42	42	40.5%	

Patients receiving chemotherapy had significantly longer overall survival than those receiving non-specific treatment ($p < 0.05$).

Table 10: Univariable and multivariable Cox regression analyses of prognostic factors for mortality.

Independent variables	Univariable analysis		Multivariable analysis	
	HR (95% CI)	P-value	HR (95% CI)	P- value
CEA (+1UI/mL)	1.059 (1.022 - 1.097)	0.001	1.001 (0.999 - 1.003)	0.135
CA19.9 (+1UI/mL)	1.003 (1.001 - 1.005)	0.001	1.038 (0.988 - 1.090)	0.441
Protein	0.608 (0.436 - 0.848)	0.003	0.878 (0.579 - 1.330)	0.539
Albumin	0.602 (0.381 - 0.951)	0.030	0.848 (0.470 - 1.530)	0.584
Tumor size on EUS	1.040 (1.017 - 1.063)	0.001	1.037 (1.011 - 1.064)	**0.005**
Vascular invasion	1.854 (1.176 - 2.921)	0.008	1.733 (1.064 - 2.822)	**0.027**
Distant metastasis	4.054 (2.137 - 7.690)	< 0.001	3.410 (1.646 - 7.065)	**0.001**

Univariable Cox regression analysis identified seven factors - CEA, CA 19-9, total protein, albumin, tumor size on EUS, vascular invasion, and distant metastasis - as potential predictors of mortality. In multivariable Cox regression analysis, tumor size on EUS, vascular invasion, and distant metastasis remained independent predictors of mortality.

4. DISCUSSION

In this study, 89 patients were diagnosed with pancreatic cancer, with a male predominance (52.8%) and a mean age of 63.74 ± 10.54 years. These findings are consistent with previously published data. Lee BK et al. reported a mean age of 67.4 years, with males accounting for 52.7% of cases [4]. Likewise, a meta-analysis by Pausawasdi N et al. demonstrated a male predominance in most studies on solid pancreatic tumors, with a pooled mean age of 65.4 years [5]. Overall, the epidemiological characteristics of our cohort are in line with global trends in pancreatic cancer.

Regarding medical history and risk factors, Table 1 shows that 42.7% of patients were smokers, 19.1% had a history of heavy alcohol consumption, 20.2% had diabetes mellitus, and 9.0% had chronic pancreatitis. These factors have been well established as being closely associated with the development and progression of pancreatic cancer. Among them, cigarette smoking was considered the most important and well-substantiated risk factor, accounting for approximately 20 - 35% of pancreatic cancer cases [6]. Lee BK et al. reported smoking and heavy alcohol use in 30.9% and 20.9% of patients with pancreatic tumors, respectively [4]. Epidemiological studies estimate that 11 - 32% of pancreatic cancer cases were attributable to smoking, with risk increasing according to smoking intensity and duration [7]. According to Bosetti C et al., smokers had a 2-3-fold higher risk of pancreatic cancer compared with non-smokers [8].

Alcohol consumption also contributes to pancreatic carcinogenesis, primarily through chronic inflammation. Alcohol and its metabolites promote carcinogenesis via chronic inflammatory pathways and genomic instability, with excessive alcohol intake accounting for 60 - 90% of chronic pancreatitis cases [9]. According to Rawla et al., consumption of ≥60 g of alcohol per day represented a definite risk factor for pancreatic cancer, particularly among individuals who also smoke [1].

Chronic pancreatitis is a well-recognized risk factor for pancreatic cancer. Persistent inflammation and pancreatic fibrosis create a procarcinogenic microenvironment that facilitates genetic mutations and disordered cellular differentiation. The risk of pancreatic cancer is approximately 0.4% after a single episode of acute pancreatitis but increases up to 9-fold in patients with chronic pancreatitis [10]. This risk was significantly higher than that of the general population, particularly among heavy smokers (> 60 pack-years), highlighting the synergistic effects of these risk factors in pancreatic carcinogenesis [10].

Type 2 diabetes mellitus was also associated with an increased risk of pancreatic cancer, with an approximately 2.4-fold higher risk compared with the general population [7]. The risk was markedly elevated during the first year after diabetes onset (14-15-fold) and subsequently declines to approximately 3-fold after three years [7]. Accordingly, diabetes may represent an early manifestation of pancreatic cancer. While diabetes was often considered secondary to tumor-related pancreatic destruction or ductal obstruction, emerging evidence suggests a role for metabolic dysregulation, such as insulin resistance, supported by the normalization of glucose levels following tumor resection in some patients [7].

Current evidence on pancreatic cancer risk factors remains limited, with identifiable risk factors present in only approximately 40% of cases [9]. In this study, a

substantial proportion of patients - particularly women - had no recognized or typical risk factors. As shown in Table 1, 39 patients (43.8%) had no documented risk factors, including 29 women and 10 men. These findings suggest that, beyond established risk factors, additional and incompletely understood mechanisms may contribute to pancreatic carcinogenesis. Potential contributors include genetic susceptibility and somatic mutations, environmental exposures, dietary factors, endocrine influences, and female sex hormones. A more comprehensive understanding of pancreatic cancer pathogenesis is therefore needed, along with focused investigations into non-classical risk factors.

Regarding clinical presentation, abdominal pain was the most common symptom, reported in 92.1% of patients, with a mean duration of 6.91 ± 4.79 weeks (Table 2). Fatigue, anorexia, and weight loss were also frequent, whereas jaundice occurred in 21.3% of cases. Pancreatic cancer is often clinically silent at early stages, with symptoms varying according to tumor size, location, and extent of invasion. Tumors of the pancreatic head typically present earlier and are more commonly associated with jaundice, abdominal pain, and weight loss than tumors of the body or tail. Porta et al. reported abdominal pain in 79% of patients and cholestasis in 59%, with obstructive jaundice significantly more frequent in pancreatic head tumors ($p < 0.001$) [11]. Abdominal pain is the most prevalent symptom and may occur even in tumors < 2 cm, typically preceding diagnosis by 1–2 months and characterized by progressive, visceral epigastric pain radiating to the back [12]. Overall, our findings were consistent with prior reportsConsistent with the observed association between pancreatic cancer and glucose metabolism disorders, Table 3 shows that 57.3% of patients had elevated blood glucose levels, whereas only 20.2% had a documented history of diabetes mellitus (Table 1). This marked discrepancy suggests that a substantial proportion of patients developed new-onset hyperglycemia during the disease course, further supporting a link between pancreatic cancer and impaired glucose tolerance.

Whether diabetes mellitus represents a cause or a consequence of pancreatic cancer remains controversial, with accumulating evidence supporting a bidirectional relationship. Diabetes is considered an independent risk factor for pancreatic cancer, potentially mediated by chronic insulin resistance, which promotes pancreatic cell proliferation and carcinogenesis. A meta-analysis of 23 cohort studies demonstrated a 52% increased risk of pancreatic cancer among individuals with diabetes compared with non-diabetic controls [13]. Conversely, approximately 80% of patients with pancreatic cancer have impaired glucose tolerance or diabetes at diagnosis, supporting the hypothesis that diabetes is more often a consequence of the malignancy than a causal factor [9].

The marked discrepancy between current hyperglycemia (57.3%) and a prior history of diabetes (20.2%) suggests that a substantial proportion of patients developed tumor-related secondary diabetes. Pancreatic cancer–associated diabetes should be suspected in older patients with late-onset diabetes and without typical metabolic risk factors. These findings support a bidirectional relationship between pancreatic cancer and diabetes, in which new-onset diabetes may represent both an early clinical indicator and a consequence of the malignancy.

As shown in Table 4, CEA levels were elevated in 44.9% of patients, whereas CA 19-9 was elevated in 80.9%. Although CA 19-9 is the most extensively investigated biomarker for pancreatic cancer, its diagnostic performance remains limited. Elevated CA 19-9 levels are not specific to pancreatic malignancy and may also occur in benign hepatobiliary and pancreatic conditions, including acute cholangitis, cirrhosis, pancreatitis, and obstructive jaundice [14]. Furthermore, CA 19-9 demonstrates suboptimal sensitivity and may be undetectable in Lewis antigen–negative individuals, even in advanced disease [14]. With a reported sensitivity of 75.5%, specificity of 77.6%, and a low positive predictive value (0.5%–0.9%), CA 19-9 was not suitable as a screening biomarker for pancreatic cancer [15].

Nevertheless, accumulating evidence supports the role of CA 19-9 in prognostication and post-treatment surveillance. Zhang L et al. demonstrated that CA 19-9 was useful for predicting prognosis and detecting recurrence after pancreatic resection, with sensitivities of 79%–81% and specificities of 82%–90% [14]. These findings suggest that CA 19-9 is more valuable as a prognostic and monitoring marker than as an independent diagnostic test.

CEA is a classical tumor marker widely used in several malignancies, particularly colorectal cancer, and has recently gained attention in pancreatic cancer. In our study, elevated CEA levels were observed in 44.9% of patients, comparable to the findings reported by Truong Xuan Long et al., in which 46.9% of pancreatic cancer patients had elevated CEA levels [16].

Despite limited sensitivity and specificity, CA

19-9 and CEA remain useful for prognostication and treatment monitoring in pancreatic cancer, and their combined assessment or inclusion in multi-marker panels may enhance diagnostic and prognostic accuracy in the absence of reliable liquid biomarkers.

Patients were followed from diagnosis until death or study completion. Overall mortality was 87.6%, with 7.9% of patients alive at the end of follow-up and 4.5% lost to follow-up. Survival ranged from 1 to 13 months, with a mean survival of 4.44 months (Table 6). As shown in Table 7, only 21.3% of patients survived beyond 6 months and 2.7% beyond 12 months, reflecting the highly aggressive nature of pancreatic cancer.

As shown in Table 8, tumor size demonstrated a moderate, significant inverse correlation with survival ($r = -0.408$, $p < 0.001$), indicating shorter survival in patients with larger tumors. This finding was consistent with the pathophysiology of pancreatic cancer, as larger tumor size reflects more advanced local disease, increased vascular and adjacent organ invasion, and a higher risk of distant metastasis. Similar associations have been reported in previous studies, including that by Ayoub I I et al., who showed that larger tumor size was associated with higher TNM stage, greater tumor aggressiveness, and increased vascular and lymphatic invasion [17].

Consistent with our findings, a study by Takahashi C et al. involving 11,707 patients with pancreatic cancer demonstrated a strong association between tumor size and survival, with patients harboring tumors ≤2 cm showing significantly longer median survival than those with tumors >2 cm (30.6 vs. 20.5 months; $p < 0.001$) [18]. These results further confirm tumor size as a valuable prognostic indicator in pancreatic cancer.

We observed a weak positive correlation between survival and serum total protein and albumin levels ($r = 0.282$ and 0.304, respectively), suggesting that better nutritional status is associated with longer survival. In contrast, a weak inverse correlation was found between survival and CA 19-9 levels ($r = -0.279$, $p = 0.01$), indicating shorter survival with higher CA 19-9 concentrations. However, the modest strength of this association underscores the limited reliability of CA 19-9 as an individual prognostic marker, likely due to its susceptibility to confounding factors such as biliary obstruction, pancreatitis, and biological tumor heterogeneity.

No statistically significant correlations were observed between survival and age, CEA levels, degree of weight loss, or duration of weight loss (Table 8). These findings suggest that common clinical parameters such as age and weight loss are insufficient to independently predict survival in pancreatic cancer, despite often being considered indicators of advanced disease. Our results were consistent with those of Zhang J et al. in a cohort of 276 patients with unresectable pancreatic cancer, in which metabolic syndrome and distant metastasis were identified as independent prognostic factors, whereas age, sex, and tumor location were not [19].

As shown in Table 9, overall survival differed significantly between patients receiving chemotherapy and those receiving non-specific treatment, with statistical significance ($p = 0.001$). Rebekah R. White et al. reported a median survival of 13 months in patients treated with radiotherapy combined with 5-FU, compared with 6 months in untreated patients [20]. These findings underscore the important role of chemotherapy in improving survival outcomes in pancreatic cancer, particularly given that most patients are diagnosed at advanced stages and are not candidates for curative surgery.

Results in Table 10 showed that univariate Cox regression identified seven variables - CEA, CA19-9, total protein, serum albumin, tumor size on EUS, vascular invasion, and distant metastasis- as factors associated with mortality risk. In contrast, multivariate Cox regression demonstrated that only three variables - tumor size on EUS, vascular invasion, and distant metastasis - remained independent prognostic factors for mortality. These findings indicate that although biochemical markers such as CEA, CA19-9, total protein, and albumin were associated with prognosis in univariate analysis, their effects were attenuated after adjustment for tumor-related and disease-stage factors. This observation is consistent with previous reports, including the study by Zhang J et al. in 276 patients with unresectable pancreatic cancer, which identified metabolic syndrome and distant metastasis as independent prognostic factors [19]. These results highlight the essential contribution of imaging-based evaluation, with endoscopic ultrasound playing a central role in risk assessment and in shaping treatment planning, surveillance, and prognostic estimation.

Consequently, identifying pancreatic cancer at an early stage - when the tumor remains limited in size, without vascular involvement or distant spread - and implementing an appropriate therapeutic approach may significantly influence patient survival.

5. CONCLUSION

Smoking and heavy alcohol consumption were the most prevalent identifiable risk factors. Clinical manifestations of pancreatic cancer were generally nonspecific, with abdominal pain being the most common presenting symptom. Tumor size, vascular invasion, and distant metastasis were identified as independent prognostic factors in patients with pancreatic cancer.

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