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Original Research

Assessing Pancreatic Morphology via Endosonography in Alcohol-Induced Chronic Liver Disease

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Abstract

Objectives: Alcohol consumption is a major risk factor for liver cirrhosis and chronic pancreatitis (CP). The interplay between pancreatic changes and alcoholic cirrhosis remains poorly understood due to limited diagnostic tools. Endosonography (EUS) offers high sensitivity for detecting pancreatic morphological changes, even in early fibrosis stages.

Methods: Between February 2010 and February 2017, 71 male patients diagnosed with alcoholic cirrhosis based on clinical, biochemical, and imaging findings were enrolled. Cirrhosis and pancreatitis from other causes were excluded. EUS, performed under midazolam and propofol sedation using a radial probe, classified pancreatic morphology per Rosemont criteria: normal, indeterminate for CP, suggestive of CP, or consistent with CP. Clinical data, including alcohol and smoking history, liver function, and portal hypertension markers, were recorded.

Results: EUS identified normal pancreatic morphology in 28 patients (39.4%), indeterminate findings in 18 (25.4%), and CP-consistent or suggestive changes in 25 (35.2%). Logistic regression revealed no significant association between pancreatic changes and age, smoking, alcohol intake, BMI, spleen size, INR, platelet count, diabetes mellitus (DM), or compensated cirrhosis. Kaplan-Meier analysis revealed no significant survival difference between patients with normal pancreatic morphology (median 3.9 years) and those with abnormal morphology (median 3.1 years; p=0.792). One patient (1.4%) with normal morphology developed pancreatic cancer after 3.3 years. Hepatic and extrahepatic malignancy incidence reached 18% over five years, with hepatocellular carcinoma (HCC) at 4.3%, yet no statistically significant association was found between pancreatic changes and malignancy development (p=0.639). Portal hypertension severity and mortality showed no correlation with pancreatic findings.

Conclusion: EUS proves valuable for assessing pancreatic changes in alcoholic cirrhosis, illuminating the complex relationship between alcohol consumption and pancreatic morphology.

Keywords: Alcoholic cirrhosis, endosonography, pancreatic morphology

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Alcohol is a well-established fibrogenic agent affecting both the liver and pancreas. Prolonged consumption triggers chronic hepatic and pancreatic fibrosis, mediated partly by stellate cell activation.[1] Despite these effects, the coexistence of alcoholic cirrhosis and chronic pancreatitis (CP) is infrequently observed in clinical settings.[2] Liver fibrosis progression can be effectively tracked using biopsy or stiffness markers for precise staging[3], and evidence suggests parallels with pancreatic fibrogenesis.[4] However, assessing pancreatic fibrosis is hindered by the risks of routine biopsies in CP, which include pancreatitis and severe complications. Although magnetic resonance imaging

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(MRI) has emerged as a method for detecting pancreatic fibrosis, no standardized staging criteria exist, unlike for liver fibrosis.[5,6]Consequently, pancreatic fibrosis often manifests only in advanced CP stages with overt symptoms.

Endosonography (EUS) provides a highly sensitive method for identifying early pancreatic fibrosis, detecting subtle anatomical changes due to its proximity to the pancreas and resistance to air or liquid artifacts.[7-11] This study systematically evaluates clinical, imaging, and laboratory findings in alcoholic cirrhosis patients, employing EUS to assess pancreatic morphology per Rosemont criteria and elucidating the intricate link between alcohol consumption and pancreatic changes.

Methods

Study Population and Design

Conducted in the Gastroenterology Department from February 2010 to February 2017, this study enrolled 71 male patients with alcoholic cirrhosis, diagnosed via medical history, laboratory assessments, and imaging. Alcohol consumption exceeding 80 g/day for at least 10 years was required, with other cirrhosis etiologies (e.g., chronic viral hepatitis, autoimmune hepatitis) excluded through comprehensive testing.[2] Detailed records of alcohol duration, quantity, type, and smoking history were collected. Patients underwent EUS initially for non-pancreatic indications, such as jaundice, cholestasis, biliary pathology (e.g., portal biliopathy, choledocholithiasis), abdominal masses, lymphadenopathy, gastric or esophageal lesions, or vascular assessments (e.g., esophageal varices).

Inclusion and Exclusion Criteria

Active alcohol consumers were included; those abstinent for ≥2 years were excluded. Patients with CP symptoms (e.g., epigastric pain, weight loss, steatorrhea), prior CP diagnosis, or risk factors (e.g., biliary disease, pancreatotoxic drugs, dyslipidemia, hyperparathyroidism, congenital anomalies) were excluded, as were those with gastrointestinal surgeries affecting EUS, acute pancreatitis history, absent cirrhosis signs, or alternative cirrhosis causes (e.g., HBV, HCV). Cirrhosis diagnosis relied on imaging (e.g., irregular liver margins, varices, splenomegaly), biochemical markers (AST/ALT >1, albumin <3.8 g/dL, platelets <200,000/mL), and clinical signs (e.g., ascites, variceal bleeding, encephalopathy).

EUS Procedure

Radial EUS was performed by an experienced endosonographer (2000 cases/year) under sedation with midazolam (up to 5 mg) and propofol, using a Hitachi Prerius Ultrasonography system with Pentax radial 360° and linear probes (7.5-10 MHz; Pentax FG36UX, New York, NY). The pancreas was assessed via the duodenum (head) and stomach (body/tail), with morphology classified per Rosemont criteria: normal, indeterminate for CP, suggestive of CP, or consistent with CP.[12] The Rosemont criteria were selected for their structured approach, enhancing diagnostic accuracy and interobserver reliability by evaluating parenchymal (e.g., hyperechoic foci, lobularity) and ductal features (e.g., stones, dilation), distinguishing major and minor changes.[13]

Statistical Analysis

Analyses used SPSS v23.0 (IBM Corp., Armonk, NY). Continuous variables were reported as mean±SD or median (range) based on Shapiro-Wilk normality tests, and categorical variables as frequencies and percentages. Group comparisons employed t-tests or Mann-Whitney U tests for continuous data, and chi-square or Fisher's exact tests for categorical data. Logistic regression identified predictors of pancreatic changes, reporting odds ratios (OR) and 95% confidence intervals (CI), with variables selected by clinical relevance and univariate results. Kaplan-Meier analysis with log-rank testing assessed survival differences ($p<0.05$).

Ethical Considerations

Participants provided informed consent after the study briefing. The research adhered to the Helsinki Declaration and was approved by the Izmir Katip Celebi University Non-interventional Clinical Research Ethics Committee (date: 21.02.2018; number: 74).

Results

This retrospective study evaluated 71 male patients with alcoholic cirrhosis, aged 54±8 years (range: 30–73). Demographic and clinical characteristics are summarized in Table 1. Chronic alcohol use averaged 25.5±8.4 years, with 87.3% active smokers, a mean BMI of 29.6±4.7, and esophageal or gastric varices in 62%.

The endosonographic assessment showed normal pancreatic morphology in 28 individuals (39.4%), indeterminate findings in 18 (25.4%), and features suggestive of or consistent with CP in 25 (35.2%). Severe abnormalities appeared in only 2 cases (2.8%), both asymptomatic for pancreatic disease. Detailed comparisons across Rosemont-classified groups are presented in Table 2, showing no notable differences in age, alcohol consumption, smoking, Child-Pugh scores, BMI, or DM (all $p>0.05$). Logistic regression (Table 3) found no link between pancreatic alterations and spleen size, INR, platelet count, varices presence ($p=0.184$), DM, or compensated cirrhosis.

Table 1. Patients characteristics

Features	n=71
Age	51.9±8.8
Age of starting alcohol use	26.2±6.9
Smoking, n (%)	62 (87.3)
Cigarettes (p*year)	33.9±18.3
Duration of alcohol consumption (years)	25.5±8.4
Amount of alcohol (g/day)	169±58
Alcohol amount in the last 10 years (g/day)	189±86
Albumin	3.4±0.7
T. bilirubin	1.9 (0.4-31)
Platelet	137 (43-427)
INR	1.3±0.2
CPS	6 (5-13)
Varices, n (%)	44 (62.0)
Spleen size (cm)	13 (8-18)
BMI, n (%)	29.6±4.7
Pancreas morphology with endosonography	
Consistent or suggestive chronic pancreatitis	25 (35.2)
Indetermine pancreas	18 (25.4)
Normal pancreas	28 (39.4)

INR: International Normalized Ratio; CPS: Child-Pugh Score; BMI: Body Mass Index.

Kaplan-Meier analysis (Fig. 1) revealed comparable median survival between normal (3.9 years) and abnormal morphology groups (3.1 years; p=0.792). By year three, four individuals underwent liver transplantation, unaffected by pancreatic findings. Malignancy data indicated one case (1.4%) of pancreatic cancer after 3.3 years in a patient with normal morphology, with HCC incidence at 1.7% (one year) and 4.3% (five years). Overall malignancy reached 3.0% at one year and 18.0% at five years, yet no relationship with pancreatic morphology emerged (p=0.639). Portal hypertension severity, assessed via spleen size and varices, showed no correlation with EUS-detected changes.

Discussion

This study aimed to assess pancreatic morphology in alcoholic cirrhosis using EUS, highlighting its utility in detecting changes linked to chronic alcohol consumption.[1] Alcohol induces fibrosis in both the liver and pancreas via stellate cell activation.[14] In the liver, this progresses from fatty liver to cirrhosis, detectable by biopsy or elastography[15], while pancreatic fibrosis, though mechanistically similar[16,17], is harder to stage due to biopsy risks. Historically, CP and cirrhosis coexistence was deemed rare due to diagnostic challenges without overt dysfunction[18,19], yet autopsy data report a 20% concurrence.[20] Prior studies noted CP in 19.7% and parenchymal changes in 25.3% of cirrhosis cases.[21] Our 35.2% prevalence of CP-consistent or suggestive features suggests that EUS enhances detection.

Although MRI and EUS elastography assess pancreatic fibrosis[5,6,22,23], no staging system exists. Radial EUS, validated histologically for ≥3 criteria[24], revealed pancreatic changes in 35.2% of our cohort. Recent advances in EUS elastography suggest potential for refining early detection and staging, supporting its evolving role.[25] Bhutani et al. reported 58% in asymptomatic and 89% in symptomatic alcoholics using a four-feature threshold, versus none in controls[26],

Table 2. Comparision of the groups according to endosonographic morphology

Patients characteristics	Normal (n=28)	Indeterminate for chronic pancreatitis (n=18)	Consistent or suggestive of chronic pancreatitis (n=25)	p
Age	53±9	49±7	53±10	0.328
Age of first alcohol intake	26±6	26±6	27±8	0.892
Alcohol intake duration (yr)	27±9	23±7	25±8	0.281
Alcohol intake (g/day)	171±67	168±40	167±59	0.967
Alcohol intake in recent 10 year (g/day)	168±68	199±72	206±107	0.239
Total alcohol intake (lt)	1658±930	1380±525	1545±668	0.514
Smoking rate (%)	23 (82.1)	18 (100)	21 (84.2)	0.367
Smoking (pack-year)	29 ±21	33±19	29±21	0.842
Child-Pugh Score, n (%)	7 (5-11)	7 (5-9)	8 (5-13)	0.296
A	7 (25.0)	7 (38.9)	8 (32.0)	
B	18 (64.3)	8 (44.2)	9 (36.0)	
C	3 (10.7)	3(16.7)	8 (32.0)	
BMI (kg/m^2)	29.3±7.1	30.9±2.9	29.1±4.7	0.796
Diabetes Mellitus, n (%)	6 (21.4)	5 (27.8)	7 (28.0)	0.828

Table 3. Logistic regression univariete analysis for pancreatic changes

Variable	p	OR	95.0% CI
Age	0.536	1.016	0.963 - 1.075
Smoking	0.398	0.500	0.101 - 2.477
Alcohol Consumption	0.101	0.994	0.988 - 1.001
BMI	0.841	0.981	0.812 - 1.185
Spleen Size	0.625	1.049	0.865 - 1.273
INR	0.228	0.266	0.031 - 2.286
Platelet	0.831	0.999	0.994 - 1.005
Total Bilirubin	0.616	1.018	0.949 - 1.092
Presence of Varices	0.184	2.024	0.715 - 5.725
Compensated Cirrhosis	0.328	0.618	0.235 - 1.622
Diabetes Mellitus	0.541	0.705	0.229-2.164

INR: International Normalized Ratio; BMI: Body Mass Index.

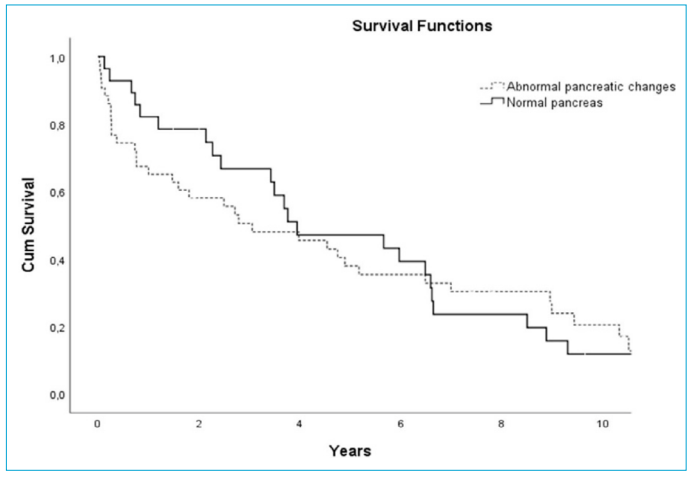

Figure 1. The survival of patients with abnormal pancreatic changes* (median: 3.1 years) and those with a normal pancreas (median: 3.9 years) was compared using endosonography (Log Rank, p=0.792).

**Abnormal pancreatic morphology includes findings consistent with, suggestive of, or indeterminate for chronic pancreatitis.*

while Hastier et al. found CP in 19% and parenchymal changes in 25.3%, stable over 22 months.[27] Our higher rate may reflect EUS's sensitivity with Rosemont criteria[13], contrasting with Bhutani's stricter criteria, potentially capturing earlier changes.

Our analysis found no association between pancreatic findings and alcohol volume, smoking, or DM, unlike Yusoff and Sahai's study of 1,157 patients, where heavy ethanol ingestion (OR 5.1, 95% CI 3.1-8.5), male sex (OR 1.8, 95% CI 1.3-2.55), clinical suspicion (OR 1.7, 95% CI 1.2-2.3), and heavy smoking (OR 1.7, 95% CI 1.2-2.4) predicted severe abnormalities.[28] This may reflect population or methodological variances. Our 35.2% aligns with Singhvi et al.'s meta-analysis (ACP in ALC: 16.2%, 95% CI 10.4-24.5)[29], supporting overlap, yet independent progression is evident, with no correlation to Child-Pugh scores (r=0.18, p=0.133).[30,31]

While pancreatic steatosis may precede hepatic steatosis[1,16], our 35.2% pancreatic fibrosis rate showed no link to liver fibrosis severity.[30] Literature suggests early stellate cell activation in pancreatic steatosis[16], yet unlike hepatic fibrosis, staging remains unclear[5], and Patel et al. indicate pancreatic fat does not reliably predict fibrosis progression, suggesting distinct pathways.[32] Nakamura et al.'s ERCP data show higher ACP in compensated cirrhosis (45.2%) versus decompensated (17.6%; OR 0.26, 95% CI 0.071-0.950; p<0.05), though ductal-specific[30], and CP may exacerbate portal hypertension.[33]

Survival (p=0.792) and malignancy risk (p=0.639) did not differ by morphology. Notably, one patient (1.4%) with normal findings developed pancreatic cancer after 3.3 years, aligning with evidence linking heavy alcohol use (median 169 g/day) to cancer risk, even without fibrosis.[34,35] Genkinger et al.'s 14-cohort analysis suggests a modest association, possibly via acetaldehyde or folate depletion.[36] This case underscores a potential oncogenic hazard.

Limitations include a small, male-only sample, retrospective design, and absent control group, limiting generalizability and causal inference. Cross-sectional imaging was limited to a subset of patients, precluding EUS comparison. This study sets the stage for longitudinal research on pancreatic change reversibility post-alcohol cessation.

In conclusion, EUS is valuable for evaluating pancreatic changes in alcoholic cirrhosis, revealing a complex alcohol-pancreas relationship. Our findings suggest non-parallel progression with liver fibrosis, necessitating further longitudinal studies.

Disclosures

Ethics Committee Approval: The study was approved by the Izmir Katip Celebi University Non-interventional Clinical Research Ethics Committee (date: 21.02.2018; number: 74).

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References

1. Apte MV, Phillips, PA, Fahmy RG, Darby SJ, Rodgers, SC, McCaughan GW, et al. Does alcohol directly stimulate pancreatic fibrogenesis? Gastroenterology 2000;118:780–94. [Crossref]
2. Gullo L, Casadei R, Campione O, Grigioni W, Marrano D. Alcoholic liver disease in alcoholic chronic pancreatitis: a prospective study. Ital J Gastroenterol 1995;27:69–72.
3. Ganne-Carrié N, Ziol M, de Ledinghen V, Douvin C, Marcellin P, Castera, L, et al. Accuracy of liver stiffness measurement for the diagnosis of cirrhosis in patients with chronic liver diseases. Hepatology 2006;44:1511.[Crossref]
4. Buchholz M, Kestler HA, Holzmann K, Ellenrieder V, Schneiderhan W, Siech M, et al. Transcriptome analysis of human hepatic and pancreatic stellate cells: organ-specific variations of a common transcriptional phenotype. J Mol Med 2005;83:795-805.[Crossref]
5. Winston CB, Mitchell DG, Cutwater EK, Erlich SM. Pancreatic signal intensity on T1-weighted fat saturation MR images: clinical correlation. J Magn Reson Imaging 1995;5:267–71. [Crossref]
6. Watanabe H, Kanematsu M, Tanaka K, Osada S, Tomita H, Hara A, et al. Fibrosis and postoperative fistula of the pancreas: correlation with MR imaging findings preliminary results. Radiology 2014;270:791–9. [Crossref]
7. Clain JE, Pearson RK. Diagnosis of chronic pancreatitis. is a gold standard necessary? Surg Clin North Am 1999;79:829–45. [Crossref]
8. Buscail L, Escourrou J, Moreau J, Delvaux M, Louvel D, Lapeyre F, et al. Endoscopic ultrasonography in chronic pancreatitis: a comparative prospective study with conventional ultrasonography, computed tomography, and ERCP. Pancreas. 1995;10:251–7. [Crossref]
9. Kahl S, Glasbrenner B, Leodolter A, Pross M, Schulz HU, Malfertheiner P. EUS in the diagnosis of early chronic pancreatitis: a prospective follow-up study. Gastrointest Endosc. 2002;55:507–11. [Crossref]
10. Amouyal G, Bellaïche G., Amouyal, P. The value of echo-endoscopy (EE) in the early diagnosis of alcoholic chronic pancreatitis (CP). Intermediate results of a prospective study [abstract]. Gastroenterol Clin Biol 1992;16:53. [Article in French]
11. Walsh TN, Rode J, Theis BA, Russel RC. Minimal change chronic pancreatitis. Gut 1992;33:1566–71. [Crossref]
12. Sahai AV. EUS and chronic pancreatitis. Gastrointest Endosc 2002;56(Suppl 4):S76–81. [Crossref]
13. Catalano MF, Sahai A, Levy M, Romagnuolo J, Wiersema M, Brugge W, et al. EUS-based criteria for the diagnosis of chronic pancreatitis: the Rosemont classification. Gastrointest Endosc 2009;69:1251–61. [Crossref]
14. Vera M, Nieto N. Hepatic stellate cells and alcoholic liver disease. Rev Esp Enferm Dig 2006;98:674. [Crossref]
15. Gianni E, Forte P, Galli V, Razzolini G, Bardazzi G, Annese V. Prospective evaluation of liver stiffness using transient elastography in alcoholic patients following abstinence. Alcohol Alcohol 2017;52:42–7. [Crossref]
16. Masamune A, Kikuta K, Satoh M, Satoh A, Shimosegawa T. Alcohol activates activator protein-1 and mitogen-activated protein kinases in rat pancreatic stellate cells. J Pharmacol Exp Ther 2002;302:36–42. [Crossref]
17. Apte MV, Pirola RC, Wilson JS. Battle-scarred pancreas: role of alcohol and pancreatic stellate cells in pancreatic fibrosis. J Gastroenterol Hepatol 2006;21(Suppl 3):S97–101. [Crossref]
18. Caradonna P, Costamagna G, Benedetti G, Gentiloni N, Gasbarrini GB. Chronic pancreatitis prevalence in liver cirrhosis. Morphological and functional study. Ital J Gastroenterol 1996;28:91–4.
19. Angelini G, Merigo F, Degani G, Camplani N, Bovo P, Pasini AF, et al. Association of chronic alcoholic liver and pancreatic disease: a prospective study. Am J Gastroenterol 1985;80:998–1003.
20. . Renner IG, Savage WT 3rd, Stace NH, Pantoja JL, Schultheis WM, Peters RL. Pancreatitis associated with alcoholic liver disease. A review of 1022 autopsy cases. Dig Dis Sci 1984;29:593–99. [Crossref]
21. Del Olmo Martinez ML, Meroño García E, Moreira Vicente V, Barba Bermejo M, Caro-Patón Gómez A. Pancreatic function and morphology in chronic alcoholism with and without cirrhosis. Rev Esp Enferm Dig 1992;82:225–9.[Article in Spanish]
22. Itoh Y, Itoh A, Kawashima H, Ohno E, Nakamura Y, Hiramatsu T., et al. Quantitative analysis of diagnosing pancreatic fibrosis using EUS-elastography (comparison with surgical specimens). J Gastroenterol 2014;49:1183–92. [Crossref]
23. Schrader H, Wiese M, Ellrichmann M, Belyaev O, Uhl W, Tannapfel A, et al. Diagnostic value of quantitative EUS elastography for malignant pancreatic tumors: relationship with pancreatic fibrosis. Ultraschall Med 2012;33:E196–201. [Crossref]
24. Bhutani MS, Arantes VN, Verma D, Moezzi J, Suryaprasad S, Kapadia AS, et al. Histopathologic correlation of endoscopic ultrasound findings of chronic pancreatitis in human autopsies. Pancreas 2009;38:820–4. [Crossref]
25. Tsuchiya T, Itoi T, Sofuni A, Tonozuka R, Mukai S, Yamamoto K, et al. Advances in endoscopic ultrasound elastography: clinical applications and future directions. Dig Endosc 2021;33:672–84.
26. Bhutani MS. Endoscopic ultrasonography: changes of chronic pancreatitis in asymptomatic and symptomatic alcoholic patients. J Ultrasound Med 1999;18:455–62. [Crossref]
27. Hastier P, Buckley MJ, Francois E, Peten EP, Dumas R, Caroli-Bosc FX, et al. A prospective study of pancreatic disease in patients with alcoholic cirrhosis: comparative diagnostic value of ERCP and EUS and long-term significance of isolated parenchymal abnormalities. Gastrointest Endosc 1999;49:705–9. [Crossref]
28. Yusoff IF, Sahai AV. A prospective, quantitative assessment of the effect of ethanol and other variables on the endosonographic appearance of the pancreas. Clin Gastroenterol Hepatol 2004;2:405–9. [Crossref]
29. Singhvi A, Abromitis R, Althouse AD, Bataller R, Arteel GE., Yadav D. Coexistence of alcohol-related pancreatitis and alcohol-related liver disease: a systematic review and meta-analysis. Pancreatology 2020;20:1069–77. [Crossref]

30. Nakamura Y, Kobayashi Y, Ishikawa A, Maruyama K, Higuchi S. Severe chronic pancreatitis and severe liver cirrhosis have different frequencies and are independent risk factors in male Japanese alcoholics. J Gastroenterol 2004;39:879–87. [Crossref]
31. Lu M, Sun Y, Feldman R, Saul M, Althouse, A., Arteel, G., Yadav, D. Coexistent alcohol-related cirrhosis and chronic pancreatitis have a comparable phenotype to either disease alone: A comparative retrospective analysis. World J Hepatol 2023;15:431. [Crossref]
32. Patel NS, Peterson MR, Brenner DA, Heba E, Sirlin, C., Loomba, R. Association between novel MRI-estimated pancreatic fat and liver histology-determined steatosis and fibrosis in non-alcoholic fatty liver disease. Aliment Pharmacol Ther 2013;37:630–9. [Crossref]
33. Takase M, Suda K, Suzuki F, Nakamura T, Futagawa S. A histopathologic study of localized portal hypertension as a consequence of chronic pancreatitis. Arch Pathol Lab Med 1997;121:612–4.
34. Gupta S, Wang F, Holly EA, Bracci PM. Risk of pancreatic cancer by alcohol dose, duration, and pattern of consumption, including binge drinking: a population-based study. Cancer Causes Control 2010;21:1047–59. [Crossref]
35. Lowenfels AB, Maisonneuve P. Epidemiology and risk factors for pancreatic cancer. Best Pract Res Clin Gastroenterol 2006;20:197–209. [Crossref]
36. Genkinger JM, Spiegelman D, Anderson KE, Bergkvist L, Bernstein L, Van Den Brandt PA, et al. Alcohol intake and pancreatic cancer risk: a pooled analysis of fourteen cohort studies. Cancer Epidemiol Biomarkers Prev 2009;18:765–76. [Crossref]