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Original Article

Internal Medicine Section

Usefulness of Endoscopic Ultrasound for the Detection of Asymptomatic Pancreatic Morphological Changes in Patients with Alcoholic Liver Disease: A Cross-sectional Study

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ABSTRACT

Introduction: Although alcoholism is a common aetiological link between liver and pancreatic disorders, the frequency of coinciding pancreatic disease in patients with alcohol-related liver disease is not well understood. The present study mentions about the use of Endoscopic Ultrasound (EUS) with standardised criteria to assess asymptomatic or overt pancreatic pathology in patients with alcoholic liver disease.

Aim: To determine the prevalence of asymptomatic Alcoholic Chronic Pancreatitis (ACP) in Indian patients affected by Alcoholic Liver Cirrhosis (ALC) using EUS.

Materials and Methods: The present observational cross-sectional study was conducted on patients attending the Outpatient Department of Gastroenterology at Santokba Durlabhji Memorial Hospital Cum Medical Research Institute, Jaipur, Rajasthan, India. A total of 35 patients were screened with alcoholic liver disease for pancreatic abnormalities using EUS. The patients were recruited for the study over a period of 12 months, from September 2013 to August 2014. The prevalence of chronic pancreatitis (CP) was determined, and abnormalities were graded according to the Rosemont criteria. The severity of alcoholic liver disease, as indexed by Child Pugh Scoring (CPS) and Model for End-stage Liver Disease (MELD), was evaluated for any association with endosonographic findings of CP. The influence of various clinico-demographic factors on alcoholic liver and pancreatic diseases was analysed using the PSS Inc. Chicago, IL program.

Results: A total of 35 male patients, aged 28 to 65 years with a mean age of 46.97 years, fulfilled the inclusion criteria. EUS revealed a prevalence of CP in 20% of patients. Three patients showed changes suggestive of CP, of which two had ALC and one had Alcoholic Hepatitis (AH). Four patients were prioritised in categorised as indeterminate for CP, with three having cirrhosis and one having Alcoholic Fatty Liver (ALF). No significant influence of clinico-demographic profiles on the final outcome parameters was observed. The patient with endoscopic changes had a MELD score of 12.85±5.11 compared to patients without changes of ACP (19.1±5.71) (p=0.013). ALC was found to be inversely related to EUS changes of ACP.

Conclusion: The EUS is an effective screening tool for evaluating pancreatic abnormalities in patients with alcoholic liver disease. The present study provides a comprehensive review of previous findings in light of the varying facts and figures. As there is still a lack of experimental animal models for ALC and ACP, studies of this kind may shed light on hidden links in disease pathology.

Keywords: Alcoholic chronic pancreatitis, Alcoholic hepatitis, Chronic liver disease

INTRODUCTION

Chronic alcoholism is presumed to be a common factor in pancreatic and liver disorders. However, hepatic and pancreatic tissues demonstrate nutritional, hormonal, environmental, or genetic differences that determine the distinct responses of these two organs to alcohol-induced injury. Recent studies from various parts of the world have presented divergent and even conflicting data regarding the prevalence and co-existence of Alcoholic Chronic Pancreatitis (ACP) and Alcoholic Liver Cirrhosis (ALC) among chronic alcoholic individuals [1-3]. It has been estimated that less than 5% of alcoholic subjects develop ACP, while the prevalence of ALC among alcoholics is approximately 2%, excluding cases of hepatitis B or C [4]. The frequency of co-existence between ACP and ALC in alcoholic patients is speculated to range from 20% to 47% in different studies [1-3,5]. The rate of co-existence has been determined based on data acquired from hospitals, autopsy findings, histopathological studies of liver and pancreatic tissues, or other sophisticated diagnostic methods such as Ultrasound (US), Computed Tomography (CT), Endoscopic Retrograde Cholangiopancreatography (ERCP), and Endoscopic Ultrasound (EUS) [6].

The EUS has evolved as the gold standard screening tool, with the ability to detect subtle changes in the early stages of chronic pancreatitis (CP). It can identify both ductal and parenchymal alterations with higher sensitivity (80%) and specificity (86%) compared to US (58%), ERCP (74%), and CT (75%) in diagnosing ACP. The visualisation of the entire pancreas using EUS has been reported to range from 57% in early studies [6-8] to nearly 100% for experienced endosonographers. EUS has become the preferred investigative method due to its higher diagnostic yield and lower complication rate, especially for early pancreatic pathologies. Among the various studies [1,3] conducted worldwide to evaluate subclinical changes of ACP in patients with ALC, only a few studies [9,10] included EUS as a screening method. Furthermore, most of the studies [1,3] have been conducted in Western populations with different socio-demographic profiles. Therefore, the present study aimed to determine the prevalence of asymptomatic ACP in Indian patients affected by ALC using EUS.

MATERIALS AND METHODS

The present observational cross-sectional study was conducted on patients attending the OPD of Gastroenterology at Santokba

Durlabhji Memorial Hospital Cum Medical Research Institute, Jaipur Rajasthan, India. The patients were recruited for the study over a period of 12 months, from September 2013 to August 2014, following the development of proper inclusion and exclusion criteria. The study received approval from the Institutional Review Board (Certificate No. 4002) and included 35 patients with Chronic Liver Disease (CLD).

Sample size calculation: The sample size was calculated using the Cochran formula, considering a prevalence rate of 4.8% for alcohol-related liver disease [3].

Inclusion criteria: In this study, we included patients with a history of significant alcohol intake for at least five years, consuming a quantity greater than 40 grams daily.

Exclusion criteria: During the recruitment of the study group, the following patients were excluded: those with clinical, laboratory, or objective evidence of pancreatic disease on imaging; those with a past or family history of pancreatic disease; and those with a history of abdominal trauma, diabetes mellitus, or gallstone disease. Patients with CLD due to non-alcoholic causes (such as Hepatitis B, Hepatitis C, and Autoimmune) and those with poor general condition and high-risk oesophageal varices were also excluded.

Study Procedure

Patients were classified as alcohol-dependent based on the CAGE questionnaire [11], and scoring systems such as CPS and MELD were used [12,13]. The patients were grouped into three categories: alcoholic fatty liver (FL), alcoholic hepatitis (AH), and alcoholic liver cirrhosis (ALC).

The diagnostic evaluation of CLD involved clinical history, examination, laboratory tests, imaging, and endoscopy to classify the patients into FL, AH, or cirrhosis. Clinical and demographic data were recorded using a predesigned proforma. A battery of laboratory tests was performed, including Complete Blood Count (CBC), Liver Function Test (LFT), Prothrombin Time with International Normalised Ratio (PT with INR), Fasting Blood Sugar (FBS), Ultrasonography (USG), Hepatitis-B Surface Antigen (HBsAg), Hepatitis C Virus (HCV) Antibody (Ab), and Antinuclear Antibody (ANA). The diagnosis of subcategories of alcoholic liver disease was based on the standard criteria accepted in the recent American Association for the Study of Liver Diseases (AASLD) practice guidelines for ALD 2019 [14].

After obtaining informed consent, EUS was performed under conscious sedation with premedication using intravenous pentazocine or midazolam, along with hyoscine-N-butyl. The procedure was conducted using a UM3 system (Olympus America, Inc., Melville, N.Y.) by an experienced single endoscopist (DA). With the patient in the left lateral decubitus position, the head of the pancreas was imaged with the tip of the transducer initially positioned at the inferior angle of the duodenum. The endoscope was gradually pulled back into the stomach, where the body and tail of the pancreas were imaged. To optimise transducer contact, a small amount of water was instilled into the stomach during scanning. The EUS findings were interpreted using the EUS criteria of the Rosemont classification [Table/Fig-1] [15].

Consensus-based parenchymal features of CP	Consensus-based ductal features of CP	EUS diagnosis of CP on the basis of consensus criteria [15]
Major criteria	**Major criteria**	**I. Consistent with CP**
Major A Hyperechoic foci with shadowing (Echogenic structures ≥2 mm in length and width that shadow)	**Major A** MPD calculi (Echogenic structure (s) within MPD with acoustic shadowing)	A. 1 major A feature (+) ≥3 minor features B. 1 major A feature (+) major B feature C. 2 major A features
Lobularity (Well-circumscribed, ≥5 mm structures with enhancing rim and relatively echo-poor centre)		
Major B		**II. Suggestive of CP**
Lobularity with honeycombing (Contiguous ≥3 lobules)		A. 1 major A feature (+) <3 minor features B. 1 major B feature (+) ≥3 minor features C. ≥5 minor features (any)
Minor criteria	**Minor criteria**	**III. Indeterminate for CP**
Lobularity without honeycombing hyperchoic foci without shadowing (Echogenic structures foci ≥2 mm in both length and width with no shadowing)	Irregular MPD contour (Uneven or irregular outline and ectatic course) Dilated side branches (3 or more tubular anechoic structure search measuring ≥1 mm in width,budding from the MPD)	A. 3 to 4 minor features, no major features B. major B feature alone or with <3 minor features
Cysts		**IV. Normal**
Anechoic, rounded/ elliptical structures with or without septations	MPD dilation (≥3.5-mm body or O1.5-mm tail)	≤2 minor features, no major features
Stranding (Hyperechoic lines of ≥3 mm in length in at least 2 different directions with respect to the imaged plane)	Hyperechoic MPD margin (Echogenic, distinct structure greater than 50% of entire MPD in the body and tail)	

[Table/Fig-1]: Rosemont classification for diagnosing and grading endoscopic chronic pancreatitis.

STATISTICAL ANALYSIS

Statistical analysis was performed with quantitative data expressed as measures of central location (mean) and measures of dispersion (standard deviation), and qualitative data expressed as percentages and ratios. Chi-square test and Student's t-test were used to compare the ratios and means of the groups, respectively. A p-value less than 0.05 was considered statistically significant.

RESULTS

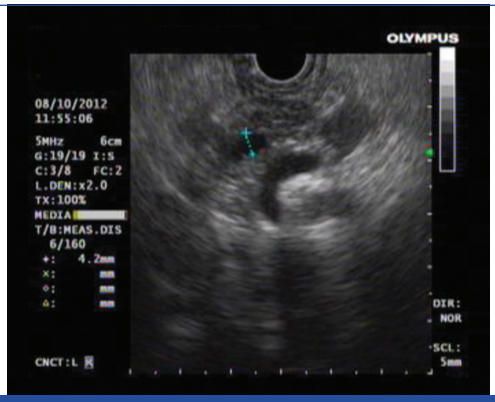
The study group consisted exclusively of male patients, with ages ranging from 28 to 65 years and a mean age of 46.97 years. The mean duration of alcohol intake was 18.11 years. The quantity of alcohol consumed per day varied among individuals, ranging from 40 to 240 grams. 54% of the patients were classified as alcohol-dependent based on the CAGE questionnaire. The patients were grouped into three categories, with 4 patients (11.4%) diagnosed with alcoholic FL, 7 patients (20%) diagnosed with AH, and 24 patients (68.6%) diagnosed with ALC. The Child-Pugh-Turcotte (CPT) scoring system was only performed for patients with ALC, of which the majority (14 patients, 58%) were classified as class C, 9 patients (37.5%) as class B, and 1 patient (4.5%) as class A. The MELD scoring system reflected that 65.7% of patients had scores ranging from 10 to 19, 8.5% had scores below 10, 20% had scores ranging from 20 to 29, and 5.8% had scores ranging from 30 to 39. The clinical, demographic, haematological, and biochemical profiles of the patients are shown in [Table/Fig-2].

Characteristics	Evidence of CP by EUS (n=7) (Mean±SD)	No evidence of CP by EUS (n=28) (Mean±SD)	p-value
Age (years)	50.14±11.72	46.17±9.88	0.366
Alcohol consumption (years)	25.00±12.89	17.00±8.77	0.06
Alcohol/day (grams)	1.28±53.98	98.66±38.09	0.09
CAGE score	2.14±1.21	1.64±1.09	0.298
Haemoglobin (gm/dL)	10.57±3.10	10.17±2.66	0.738
Serum creatinine (mg/dL)	0.72±0.17	0.99±0.59	0.249
Random blood sugar (mg/dL)	109±9.46	105±26.38	0.740
Aspartate transaminase (IU/L)	99.14±26.91	15.8±215.88	0.480
Alanine transaminase (IU/L)	61.42±31.47	10.9±226.97	0.580
Serum bilirubin (mg/dL)	3.11±4.25	2.518±100.52	0.570
Alkaline phosphatase (IU/L)	184±107.74	171±72.01	0.689
Albumin/Globulin ratio	0.42±0.53	0.14±0.35	0.096
PT (INR)	1.44±0.42	1.76±0.59	0.181
MELD	12.85±5.11	19.10±5.71	0.013

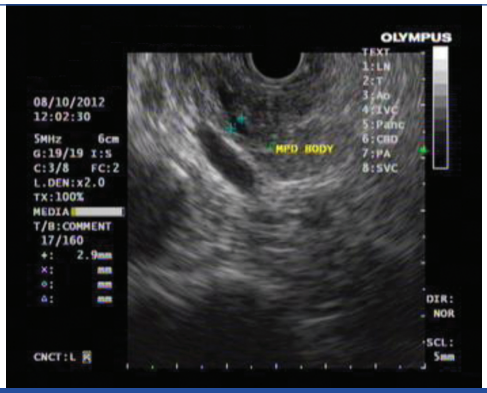
[Table/Fig-2]: Clinico-demographic and laboratory parameters of the study population (N=35).

Upper gastrointestinal endoscopy showed a normal study of the pancreas in six patients and low-risk oesophageal varices in 22 patients. Portal gastropathy and duodenopathy were accompanied by oesophageal varices in 11 patients (31.4%). There were no incidences of gastric varices observed during endoscopy.

The EUS revealed a prevalence of morphological changes in the pancreas in 7 patients (20%) with a 95% Confidence Interval (CI) of 10-35.8%, as evaluated according to the Rosemont classification [15]. The proportion of patients showing ductal and parenchymal changes on EUS were 11.4% and 20%, respectively [Table/Fig-3,4]. None of the patients fulfilled the criteria, based on EUS findings, for a diagnosis "consistent with Chronic Pancreatitis (CP)". Three patients were recognised as having changes "suggestive of CP". Out of these, two patients had ALC and one patient had AH. Four patients were categorised as "indeterminate for CP". Among these, three patients had cirrhosis and one patient had alcoholic Fatty Liver (FL).



[Table/Fig-3]: Showing mildly dilated Major Pancreatic Duct (MPD) (4.4 mm in head; 4.2 mm in neck and 2.6 mm in body) with irregular outlines.



[Table/Fig-4]: Showing lobularity and hyperechoic areas in uncinate process.

Patients diagnosed with CP were statistically compared in terms of various clinical, demographic, and laboratory parameters with those without CP [Table/Fig-2]. The only statistically significant difference was seen in the MELD score, which was found to be inversely related to EUS changes of ACP. The patients with endoscopic CP had a MELD score of 12.85±5.11, compared to patients without changes of ACP (19.1±5.71) (p=0.013).

DISCUSSION

Prolonged alcohol ingestion is an established aetiology for both chronic liver and pancreatic diseases. The average duration for the development of CP is 6 to 8 years, whereas liver cirrhosis usually occurs after a latency of 8 to 10 years [16]. These data indicate a more diverse and greater extent of repair mechanism in hepatic tissue compared to the pancreas. Despite the presence of a common aetiological factor, alcoholism, there is discordance in terms of the frequency of coincidence of both disorders, as claimed by various epidemiological studies [2,16,17]. This could be explained by the different methodologies used for assessing the outcome parameters related to the disorders, the additional influence of demographic and sociocultural factors, and the presence of confounders such as hepatitis B or C virus. In fact, more than one-third of patients with ALC exhibit antibodies against HCV [18]. Contrary to a few previous studies [19-22], the current study provides more reliable data due to the exclusion of confounding factors (hepatitis B or C virus, non alcoholic liver or pancreatic diseases, or surgery). A large number of surveys depict a positive association between ALC and ACP, although a few studies reveal an inverse association [21]. This could be seen that in cases of liver cirrhosis, modification of pancreatic secretion to high volume and low protein might confer protection to the pancreas by decreasing the precipitation of protein and calcium [19].

Contrary to earlier studies that used ERCP [23,24] and other functional imaging for the evaluation of the pancreas in ALD patients, this study used EUS for the same purpose with the advantage of better sensitivity, a lower complication rate, and the ability to detect changes even at inception. To the best of authors knowledge, the application of EUS in pancreatic evaluation is a pioneering effort in India. The use of EUS over ERCP can also reduce the bias of including cases of ERCP-induced pancreatitis [25]. However, EUS has some limitations, such as being operator-dependent, and the diagnosis of CP is based on subjective criteria associated with variability. Two distinct echogenic patterns are seen in early CP: One with predominantly ductal dilatation and a hyperechogenic duct wall, and the other with mainly parenchymal changes associated with a dilated MPD and normal duct wall [6].

There was also disagreement regarding the predetermined threshold of elemental lesions used for the EUS diagnosis of CP. Clearly, the higher the threshold, the higher the specificity at the compromise of sensitivity (low sensitivity). The Rosemont Classification resolved the discrepancies regarding the diagnosis of CP by defining an appropriate threshold [15]. The criteria classify CP based on the presence of different elemental features into three categories:

1) Endosonographic diagnosis "consistent with CP" is achieved by the presence of: a) one major A feature and more than three minor features; b) one major A and one major B; or c) two major A features.
2) Endosonographic diagnosis "suggestive of CP" is achieved by the presence of: a) one major A and fewer than three minor features; b) major B and fewer than three minor features; or c) any five or more minor features.
3) Endosonographic finding "indeterminate for CP" is achieved by: a) more than two minor features and fewer than five minor features without major features; or b) major B feature alone or with fewer than three minor features. Less than two minor features are interpreted as "normal" results. This last category excludes features such as cysts, dilated MPD and side branches, and hyperechoic non shadowing foci.

The present study also evaluated the association of various clinico-demographic factors with subclinical or overt CP in patients with CLD. The skewed data with regard to gender also reflects the sociocultural taboo for the consumption of alcohol in Indian females. Studies from the West had a significant proportion of female patients, which removes the gender bias seen in the present study [3]. The mean age of the present study cohort is 46.97 years, which is nearly similar to another Indian study [21] but lower compared to Western studies by approximately one decade [3]. This could be explained by social trends of alcohol consumption in India and the influence of nutritional and other environmental factors.

The present study compared different parameters in groups with changes of CP and a normal pancreas. The study did not find any statistically significant difference in age, duration and quantity of alcohol intake, and CAGE score in both groups, but the MELD score was significantly lower in patients with CP. The lower MELD score can be supported by the results of previous studies showing an inverse relation between ALD and ACP. A study by Hastier P et al., used both ERCP and EUS to assess pancreatic findings in patients with ALC [26]. The authors observed changes of ACP in 14 out of 72 (19.7%)

patients by ERCP, and out of these 14 patients, 13 (92.8%) showed ductal and parenchymal changes on EUS. One drawback of the study was the lack of use of standardised Rosemont criteria for classifying EUS changes. Additionally, contrary to the present study, they only included cirrhotic patients, resulting in a biased association with ACP.

Various studies have been conducted in the recent past, which are based on ERCP [21,23,24,27]. Kochhar R et al., found ductal changes in 43.47% of their 46 patients [28]. The results of Singhvi A et al., were very close to ours, but this wide variation is difficult to explain [21]. Factors other than alcohol, such as genetics, environmental, and dietary differences, may be the probable reason. The present study did not find any correlation between EUS findings and the stage of alcoholic liver disease. Admittedly, not all of our patients had histological confirmation of the exact status of liver disease. A few autopsy-based studies, like the one by Renner IG et al., found changes of CP in 20% and 18% of patients with CLD, which included 77% cirrhosis and 23% sclerosing hyaline necrosis based on histopathology [29]. The result of the present study was comparable to the study by Renner IG et al., and correlates well, as both studies include patients with ALD and are based on a very sensitive and specific method for the detection of CP [29].

The present study is a fair attempt to review the previous findings amidst the plethora of controversial facts and figures. As there is still a lack of an experimental animal model of the diseases, a study of this kind may shed light on hidden links in disease pathogenesis.

Limitation(s)

The present study population comprised exclusively male patients, so authors were unable to derive any gender-based association with CP.

CONCLUSION(S)

The present study concluded that asymptomatic pancreatic changes on EUS are frequent in patients with alcoholic liver disease. However, only a minority of these changes are clinically relevant and produce symptoms. It would be interesting to follow-up with such patients for signs and symptoms of pancreatic disease. We believe that more data and long-term follow-up, along with structural and functional documentation of lesions detected at EUS, is needed to understand their significance.

REFERENCES

[1] Durbec JP, Sarles H. Multicenter survey of the etiology of pancreatic diseases: Relationship between the relative risk of developing chronic pancreatitis and alcohol, protein and lipid consumption. Digestion. 1978;18(5-6):337-50.
[2] Veena AB, Rajesh G, Varghese J, Sundaram KR, Balakrishnan V. Alcoholic chronic pancreatitis and alcoholic liver cirrhosis: Differences in alcohol use habits and patterns in Indian subjects. Pancreas. 2012;41(5):703-06.
[3] Niu X, Zhu L, Xu Y, Zhang M, Hao Y, Ma L, et al. Global prevalence, incidence, and outcomes of alcohol related liver diseases: A systematic review and meta-analysis. BMC Public Health. 2023;23(1):01-21.
[4] Forsmark CE, Feldman M, Friedman LS, Sleisenger LJ. Fordtran's gastrointestinal and liver disease: Pathophysiology, diagnosis, management. 11th ed. Philadelphia: PA, Saunders, an imprint of Elsevier Inc; 2020.
[5] Tandon BN, Sood S. Tropical Hepatogastroenterology. 1st edition. Elsevier, A Division of Reed Elsevier India Private Limited; 2008.
[6] Stevens T, Parsi MA. Endoscopic ultrasound for the diagnosis of chronic pancreatitis. World J Gastroenterol. 2010;16(23):2841-50.
[7] Irisawa A, Katakura K, Ohira H, Sato A, Bhutani MS, Hernandez LV, et al. Usefulness of endoscopic ultrasound to diagnose the severity of chronic pancreatitis. J Gastroenterol. 2007;42(Suppl 17):90-94.
[8] Iglesias-García J, Lariño-Noia J, Lindkvist B, Domínguez-Muñoz JE. Endoscopic ultrasound in the diagnosis of chronic pancreatitis. Rev Esp Enferm Dig. 2015;107(4):221-28.
[9] Fung BM, Abadir AP, Eskandari A, Levy MJ, Tabibian JH. Endoscopic ultrasound in chronic liver disease. World J Hepatol. 2020;12(6):262-76.
[10] Mainenti PP, Romano F, Pizzuti L, Segreto S, Storto G, Mannelli L, et al. Non-invasive diagnostic imaging of colorectal liver metastases. World J Radiol. 2015;7(7):157-69.
[11] Williams N. The CAGE questionnaire. Occup Med (Lond). 2014;64(6):473-74.
[12] Valerin MA, Napeñas JJ, Brennan MT, Fox PC, Lockhart PB. Modified Child-Pugh score as a marker for postoperative bleeding from invasive dental procedures. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2007;104(1):56-60.
[13] Bayona Molano MDP, Barrera Gutierrez JC, Landinez G, Mejia A, Haskal ZJ. Updates on the model for end-stage liver disease score and impact on the liver transplant waiting list: A narrative review. J Vasc Interv Radiol. 2023;34(3):337-43.
[14] Crabb DW, Im GY, Szabo G, Mellinger JL, Lucey MR. Diagnosis and treatment of alcohol-associated liver diseases: 2019 practice guidance from the American Association for the study of liver diseases. Hepatology. 2020;71(1):306-33.
[15] 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(7):1251-61.
[16] Rivelsrud M, Paur I, Sygnestveit K, Nilsen RM, Tangvik RJ. Nutritional treatment is associated with longer survival in patients with pancreatic disease and concomitant risk of malnutrition. Clin Nutr. 2021;40(4):2128-37.
[17] Beger HG, Warshaw AL, Hruban RH, Buchler MW, Lerch MM, Neoptolemos JP, et al. The pancreas: An integrated textbook of basic science, medicine, and surgery. 3rd ed. Oxford: John Wiley & Sons; April 2018.
[18] Novo-Veleiro I, Alvela-Suárez L, Chamorro AJ, Gonzalez-Sarmiento R, Laso FJ, Marcos M. Alcoholic liver disease and hepatitis C virus infection. World J Gastroenterol. 2016;22(4):1411-20.
[19] Rabih SA, Agudo RG, Huidobro ML, Ferrús MY, Carro PG, Pérez FR, et al. Exocrine pancreatic insufficiency and chronic pancreatitis in chronic alcoholic liver disease: Coincidence or shared toxicity? Pancreas. 2014;43(5):730-34.
[20] Lu M, Sun Y, Feldman R, Saul M, Althouse A, Arteel G, et al. Coexistent alcohol-related cirrhosis and chronic pancreatitis have a comparable phenotype to either disease alone: A comparative retrospective analysis. World J Hepatol. 2023;15(3):431-40.
[21] 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(6):1069-77.
[22] Issa Y, Kempeneers MA, Van Santvoort HC, Bollen TL, Bipat S, Boermeester MA. Diagnostic performance of imaging modalities in chronic pancreatitis: A systematic review and meta-analysis. Eur Radiol. 2017;27(9):3820-44.
[23] Freeman ML. Complications of endoscopic retrograde cholangiopancreatography: Avoidance and management. Gastrointest Endosc Clin N Am. 2012;22(3):567-86.
[24] Hastier P, Harris AG, Caroli-Bosc FX, Dumas R, Delmont JP. Comparison of post-ERCP biological pancreatitis in chronic alcoholic and control nonalcoholic group. Digest Dis Sci. 1996;41:549.
[25] Yamamiya A, Irisawa A, Abe Y, Arisaka T, Ohnishi T, Hoshi K, et al. Diagnosing chronic pancreatitis by endoscopic ultrasound assessing the association between ultrasound and pathological findings: A narrative review. DEN Open. 2023;3(1):e164.
[26] 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(6):705-09.
[27] Shaikh DS, Vartak S, Vartak U. Histomorphological changes in pancreas and liver among chronic alcoholics-an autopsy study. J Clin Diagn Res. 2022;16(11):EC27-EC31.
[28] Kochhar R, Kumar Sethy P, Sood A, Nagi B, Singh K. Concurrent pancreatic ductal changes in alcoholic liver disease. J Gastroenterol Hepatol. 2003;18(9):1067-70.
[29] Renner IG, Savage WT, 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(7):593-99.

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