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Case Report Open Access

Alcoholic Pancreatitis with Chronic Liver Disease- An Uncommon Dual Phenomenon

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1. Abstract

1.1. Introduction

Alcohol-related chronic pancreatitis and chronic liver disease are two serious, long-term consequences of excessive alcohol consumption. Both conditions are characterized by progressive tissue damage, fibrosis, and impaired organ function, and are associated with significant health burdens. Symptoms can overlap and may include abdominal pain, weight loss, and nausea, while later stages of liver disease may cause jaundice. Rarely both these conditions condition can be seen in same patient but usually there is independent hepatic involvement or of pancreas.

1.2. Case Report

We report a case of fifty-five-year-old male, a chronic alcoholic for last twenty years and takes around 60-80 gm/day. His last drink was one month back. He presented with right lumbar pain for last one month and acute onset epigastric pain for last two days. The epigastric pain was associated with severe vomiting and radiation to back. Patient developed paralytic ileus for five days, after that it recovered. On evaluation, contrast enhanced computed tomography scan revealed right renal mass lesion suspected of renal cell carcinoma, along with acute pancreatitis with CTSI score of 4/10 and early cirrhotic changes in liver. His endoscopy was normal and fibroscan score was 21 Kpa which was suggestive of cirrhotic pattern. He is being worked for renal mass lesion by urologist.

1.3. Conclusion

Alcohol effects both liver and pancreas independently but simultaneous involvement is not common. The reasons for same are not known but in every alcoholic the impact on hepatic, pancreatic or any other organ should be meticulously worked up.

2. Introduction

Alcohol-related chronic pancreatitis occurs due to chronic inflammation and damage to the pancreas resulting from long-term heavy alcohol use. Alcohol and its byproducts can damage pancreatic cells, and it's also believed to increase the pancreas's susceptibility to damage from other agents. This can lead to recurrent acute attacks that gradually cause irreversible damage. Impaired production of digestive enzymes and hormones like insulin, leads to malabsorption, weight loss, diarrhea, and diabetes. Alcohol-induced hepatic injury progresses from fatty liver to chronic hepatitis and cirrhosis but rarely reaches to hepatocellular carcinoma (H.C.C) because many patients succumb to their illness before reaching to HCC stage. Alcohol metabolism generates free radicals, causing oxidative stress that damages liver cells. This leads to inflammation, fat accumulation, and the development of fibrosis, which eventually progress to cirrhosis. The liver loses its ability to perform essential functions like detoxifying blood and producing vital proteins. In beginning disease can remain silent, but can later include jaundice, fatigue, ascites and coagulopathy. Both ALD and pancreatitis share common underlying mechanisms, such as cell damage, activation of stellate cells and fibrosis. Oxidant stress, a key factor in liver damage, also plays a role in pancreatic damage. The risk of both diseases increases with the amount and duration of alcohol consumption. Individuals with alcoholic chronic pancreatitis often have a higher average daily alcohol intake than those with chronic pancreatitis alone. The simultaneous presence of both conditions is complex, and a greater understanding is needed to explain why some individuals develop both while others don't. The most critical step is stopping alcohol consumption to prevent further damage. Treatment for both pancreatitis and liver disease involves supportive measures like intravenous fluids, pain management, and nutritional support. Depending on the severity, other treatments can include medications, surgical interventions, or even a liver or pancreatic transplant. Smoking cessation is also an important strategy, as heavy alcohol users often smoke, and this can exacerbate pancreatic damage.

3. Case Report

We report a case of fifty-five-year-old male, moderately built with BMI of 23, a chronic alcoholic for last twenty years and takes

around 60-80 gm/day. His last drink was one month back. He was not a known case of any chronic illness like diabetes mellitus, hypertension or thyroid disorder. He presented with right lumbar pain for last one month for which he sought advice from some private practitioner who gave symptomatic treatment and got ultrasonogram abdomen done which revealed some mass lesion in right renal area. Later on he developed acute onset epigastric pain for last two days. The epigastric pain was associated with severe vomiting and radiation to back. Patient developed paralytic ileus for five days, after that it recovered. The biochemical evaluation revealed low hemoglobin of 10.5 gm% with normocytic normochromic picture and mild thrombocytopenia. The lipid profile was on lower side. The rest parameters including blood sugar, thyroid, viral screen was normal except for slightly low level of serum calcium and serum Vitamin D3 level. The electrocardiogram and chest x-ray were normal and repeat ultrasonogram at this stage re-confirmed right renal mass lesion with dilated gut loops, hence pancreas could not be visualized. The contrast enhanced computed tomography scan showed pancreas to be diffusely bulky in size and showed heterogenous enhancement with peri-pancreatic fluid & fat stranding, along with multiple calcific foci seen in pancreatic head region. There was thickening of left anterior renal fascia & lateral conal fascia with mild ascites in perihepatic space and in pelvis. Liver was normal in size but showed irregular nodular contour with caudate lobe hypertrophy. Spleen was borderline enlarged in size, measuring 123 mm. A large ill- defined heterogeneously enhancing lesion measuring 75 x 85 x 64 mm with few tiny foci of calcification and non-enhancing areas of necrosis seen involving mid pole of right kidney. This lesion was surrounded by few vascular channel/collaterals? neovascularization. The lesion was also extending into right renal vein with its dilatation- suggestive of renal vein thrombosis. On delayed scan lesion showed splaying of right pelvi-calyceal system in right kidney. The final impression was right renal mass lesions suspected of renal cell carcinoma, along with acute pancreatitis with CTSI score of 4/10 and early cirrhotic changes in liver. His endoscopy was normal and fibroscan score was 21 Kpa which was suggestive of cirrhotic pattern. He is being worked for renal mass lesion by urologist.

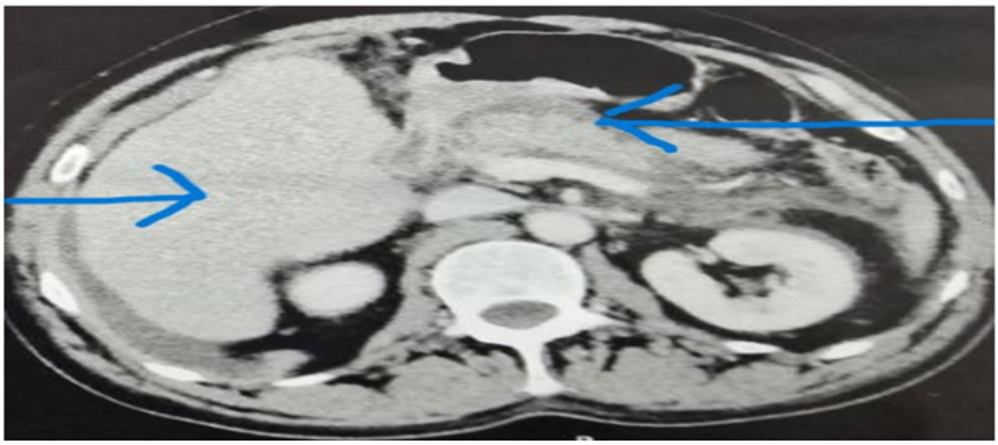

Figure 1: CECT scan abdomen showing irregular margins of liver (small blue arrow) and acute pancreatitis changes (large blue arrow).

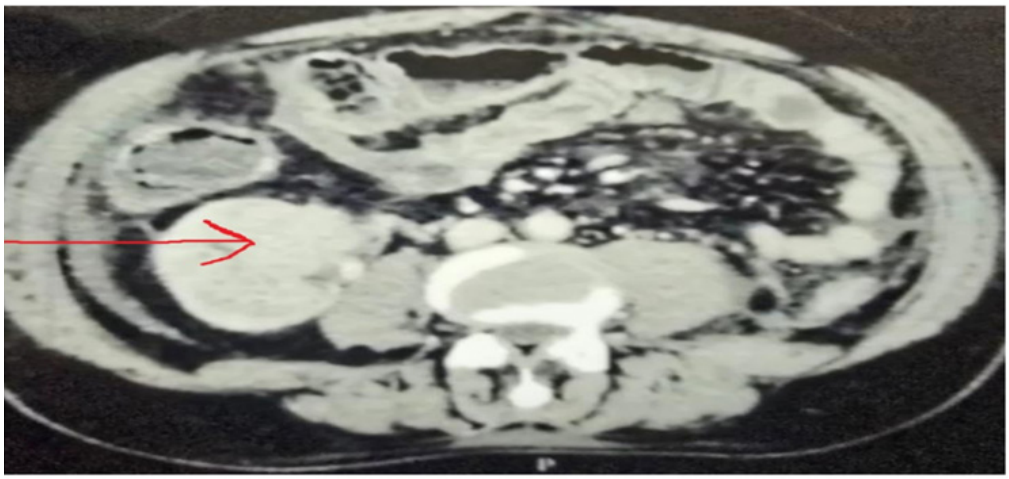

Figure 2: CECT scan abdomen showing right renal Mass suggestive of renal cell carcinoma (red arrow).

4. Discussion

Alcoholism is the most widespread substance abuse not yet affected territories. Its health consequences are varied, including a wide range of mental and neurological disorders and parenchymal organ damage disease. Patterns of alcohol abuse and organ damage vary greatly among individual alcoholics. Alcoholic liver cirrhosis (ALC) and alcoholic chronic pancreatitis (ACP) are the most frequent organ manifestations. While both are serious and potentially fatal, empirical experience showing that liver transplantation is almost never compromised by alcoholic pancreatitis has

indicated that concomitant and equal involvement of both these organs is not a rule. Pathogenetic hypotheses consider orchestration of genetic, metabolic and nutritional factors [1]. Individual sensitivity to alcohol results not only in variable addiction but, also, in variable organ damage. In this population, males predominated in both organ diseases, especially among those with a primary diagnosis of chronic pancreatitis. Average daily alcohol consumption is always difficult to estimate when it involves different periods of drinking with individually fluctuating patterns. Alcohol is the cause of deaths among 13.5% of individuals aged 20 to 39 years worldwide [2]. Studies suggest the incidence of alcohol-associated liver disease (ALD) is increasing disproportionally among adolescents and young adults (AYAs), especially young females [3-5]. Alcohol overconsumption has detrimental effects on several organs, with the liver and pancreas being particularly vulnerable. The liver sustains the highest degree of tissue injury because it is the primary site of ethanol metabolism. In addition to direct metabolic stress, alcohol causes tissue inflammation, resulting in maladaptive fibrotic response. Alcohol is also a main cause of acute and chronic pancreatitis and can affect other organs, such as the heart and stomach [6]. Alcohol-induced pancreatitis often manifests as a spectrum, ranging from discrete episodes of acute pancreatitis (AP) to chronic irreversible changes. Although higher doses of alcohol increase the risk, only about 2.5%–3% of heavy drinkers develop pancreatitis, suggesting alcohol alone is not sufficient to cause AP. Instead, ethanol sensitizes the pancreas to injury, requiring additional genetic or environmental factors for pancreatitis to occur. As reported in many other series, chronic alcoholic pancreatitis is associated with histological changes in liver in significant proportion of patients [7]. ALD is associated with 9-fold higher odds of prevalent pancreatitis compared to the general population. The hospitalization rate for AP following ALD diagnosis is 6-fold higher. About 10% of patients with ALD have or develop AP, suggesting that assessing history of pancreatitis and its sequelae might be relevant for patients with ALD [8]. The coexistence of ALD and pancreatitis is seen in clinical practice. However, the existing literature on this subject is largely lacking, and the available estimates show great variation, ranging from 1.4% to 70% for pancreatitis prevalence in alcohol-related cirrhosis [9]. In this nationwide cohort study comprising over 37,000 patients with ALD and more than 350,000 matched comparators from the general population, we observed a 6.3-fold increased rate of AP over a mean follow-up of 7 years, with follow-up extending to 51 years. Notably, 7% of ALD patients had a history of either acute or chronic pancreatitis, which is nine times more likely compared to the general population [8]. In our case also there was involvement of both liver and pancreas, as evidenced by first episode of acute pancreatitis in background of early cirrhotic changes in liver. One more thing in contrast to previous reports in literature was renal mass suggestive of renal cell carcinoma because it is well documented that alcohol decreases the risk of renal cell carcinoma [10]. The treating health care team should not only focus on hepatic aspect in alcoholics but also to other organs like pancreas, heart, lungs, brain etc. The alcoholic patient merits a multidisciplinary approach for taking care of overall impact on human body as a whole. It will re-affirm the habit of complete abstinence from alcohol because till date patient and their family members assess severity of disease on basis of hepatic damage only but once they understand the gravity of chances of damage on other organs, will lead to development of fear of continuing with alcohol intake.

5. Conclusion

Alcohol effects both liver and pancreas independently but simultaneous involvement is not common. The reasons for same are not known but in every alcoholic the impact on hepatic, pancreatic or any other organ should be meticulously worked up.

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