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Pancreatic Carcinoma with HCV- An Uncommon Association

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1. Abstract

1.1. Introduction

HCV infection is associated with an increased risk of pancreatic cancer, though this association becomes less strong when potential risk factors like diabetes, smoking, and alcohol intake are accounted for. The exact mechanisms are not fully understood, but may involve chronic inflammation, viral integration, or common origins of the cells in the liver and pancreas. While further research is needed, antiviral treatment for HCV may potentially reduce this risk

1.2. Case Report

A sixty-four-year-old male having non-significant past history, was detected to be suffering from chronic hepatitis C virus (HCV) infection on routine screening. On detailed evaluation, his complete hemogram, renal function tests, thyroid profile, lipid profile, blood sugar were normal. The anti HCV antibody was positive but anti -HIV antibody and HbsAg were negative. The HCV RNA quantitative load was 2.4 x 106 I.U./ml with mild hyperbilirubinemia and transaminitis. The ultrasound abdomen revealed mild hepatomegaly and fibroscan score was 6.7 Kpa, suggestive of non-cirrhotic stage. He was treated with 12 weeks treatment with sofosbuvir 400 mg & Daclatasvir 60 mg. He completed his course and 12 weeks SVR was achieved. At this point of time, he complained of pain abdomen for last few days, hence was subjected to fresh ultrasonogram abdomen which now revealed a mass lesion in pancreas with multiple lesions in liver. Thus, for confirmation, CECT scan abdomen was done which confirmed it to be carcinoma pancreas with metastatic lesions in liver.

1.3. Conclusion

Our case report is an uncommon presentation in which carcinoma pancreas developed after successful treatment of chronic HCV, despite their being no other risk factors like alcohol, obesity and diabetes mellitus. There is any strong association between the two or is a co-incidental finding, is further area of research but it merits vigil in HCV patients for future development of the same.

2. Introduction

HCV infection is associated with an increased risk of pancreatic cancer, though this association becomes less strong when potential risk factors like diabetes, smoking, and alcohol intake are accounted for. The exact mechanisms are not fully understood, but may involve chronic inflammation, viral integration, or common origins of the cells in the liver and pancreas. Meta-analyses of studies show that people with HCV infection have a greater likelihood of developing pancreatic cancer compared to those without the infection. The elevated risk of pancreatic cancer is observed to be attenuated when studies adjust for other known risk factors for pancreatic cancer, such as diabetes, smoking, and alcohol intake. The precise way in which HCV contributes to pancreatic cancer is not fully understood. The possible explanations include chronic inflammation induced by HCV; the virus might migrate between the liver and pancreas due to the close anatomical relationship of these organs. Hepatocytes (liver cells) and pancreatic cells share a common developmental origin, and viral infection could potentially disrupt this process.

3. Case Report

A sixty-four-year-old male having non-significant past history, was detected to be suffering from chronic hepatitis C virus (HCV) infection on routine screening. The general physical and systemic examination was essentially normal. On detailed evaluation, his complete hemogram, renal function tests, thyroid profile, lipid profile, auto immune profile, blood sugar were normal. The anti HCV antibody was positive but anti -HIV antibody and HbsAg were negative. The HCV RNA quantitative load was 2.4 x 106 I.U./ml with mild hyperbilirubinemia and transaminitis. The ultrasound abdomen revealed mild hepatomegaly and fibroscan score was 6.7 Kpa, suggestive of non-cirrhotic stage. He was treated with 12 weeks treatment with sofosbuvir 400 mg & Daclatasvir 60 mg. He completed his course and 12 weeks SVR was achieved. At this point of time, he complained of pain abdomen for last few days,

hence was subjected to fresh ultrasonogram abdomen which now revealed a mass lesion in pancreas with multiple small lesions in liver. Thus, for confirmation, CECT scan abdomen was done which showed in pancreas a hypo enhancing lesion measuring 37x30 mm involving body of pancreas. The lesion was closely abutting gastric wall anteriorly; perilesional fat stranding was seen. It completely encased portal confluence, splenic artery and vein causing com-

plete obliteration of splenic vein. Area of contact with SMA was less than 90 degrees. The distal pancreas was atrophic with dilated main pancreatic duct which measured 5 mm. Multiple small perilesional, peri-portal, gastro-hepatic and para-aortic nodes were seen. The common bile duct was dilated to 7 mm with mild dilatation of intra hepatic bile ducts. Liver showed multiple hypo-enhancing lesions largest measuring 24 x 22 mm in sixth segment.



Figure 1: Showing Carcinoma Pancreas in HCV Patient.

4. Discussion

Hepatitis C virus (HCV) may increase the risk of hepatopancreatic biliary tumours other than hepatocellular carcinoma (HCC). Previous case control studies indicated a possible association between HCV and intrahepatic cholangiocarcinoma (ICC). Little is known about the association between HCV and extrahepatic cholangiocarcinoma (ECC) or pancreatic cancer. HCV has been found to be related to many extra hepatic malignancies like pancreatic cancer. Limited data is available about directly acting antiviral (DAA) role in extrahepatic malignancy risk. In a retrospective case control study, which included 76 pancreatic cancer patients and 100 age and sex matched controls. Untreated HCV patients showed 3.5fold increase in pancreatic cancer risk than HCV negative controls (p value 0.028). Patients who were treated from CHC by DAAs and achieved sustained virological response had no significant increase in pancreatic cancer risk. Patients with liver cirrhosis showed 7.1fold increase in pancreatic cancer risk (p value 0.032). [1] One more study showed that the risk of pancreatic cancer was slightly elevated (1.23; 1.02, 1.49), but was attenuated after adjusting for alcohol use, pancreatitis, and other variables. [2] People with HIV

and people with HCV were each at 2.8 times higher risk of developing pancreatic cancer compared to people who tested negative for these viruses, while people with HBV/HCV co-infection were at 2.9 times higher risk of developing pancreatic cancer. [3] HCV infection was associated with increased risk of pancreatic cancer, but this association was attenuated among studies that adjusted their results for potential risk factors for pancreatic cancer. [4] one another study conducted in Sweden also showed that HCV infection might be associated with an increased risk of pancreatic cancer but further studies are needed to verify such association. [5] Our model treatment centre is high flow centre under National viral hepatitis control program and uptill now 25,000 HCV patients have been treated free of cost but this is the first case which has reported with pancreatic cancer. Further prospective cohort studies are recommended to confirm the association and clarify the causal link between HCV and pancreatic cancer. Evaluating whether antiviral treatment for HCV can decrease the risk of pancreatic cancer is an area of future study. Developing cost-effective strategies for pancreatic cancer surveillance in patients with chronic viral hepatitis is also needed.

5. Conclusion

Our case report is an uncommon presentation in which carcinoma pancreas developed after successful treatment of chronic HCV, despite their being no other risk factors like alcohol, obesity and diabetes mellitus. There is any strong association between the two or is a co-incidental finding, is further area of research but it merits vigil in HCV patients for future development of the same.

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