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Fatty Pancreas and Pancreatic Cancer: Commentary

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Description

Infiltration of fat into the pancreatic parenchyma without a large alcohol intake is what is known as fatty pancreas (FP). Several investigations using abdominal ultrasound imaging have found that the prevalence of FP varies from 16 to 35 percent. Since FP was first observed in the early 1930s, it was previously believed to be a benign incidental finding in imaging investigations carried out for other causes, and its clinical implications were not properly examined for many years. However, there has been growing evidence in recent years linking FP to a number of comorbid disorders. Most significantly, it has links to non-alcoholic fatty liver disease, obesity, diabetes, and the metabolic syndrome (NAFLD). Additionally, there is some contradictory information on its connection to PC. So, the objective of the current investigation was to determine whether there was a relationship between FP and PC [1].

Since it was thought to be a benign incidental finding for many years, the clinical repercussions of this disorder were really ignored. Over the past few years, there has been an increase in information regarding the clinical significance of FP, including its correlation with NAFLD and non-cancerous illnesses such diabetes mellitus, the metabolic syndrome, and cardiovascular diseases. Additionally, new research has demonstrated that FP is a risk factor for acute pancreatitis and has identified a link to a pre-cancerous pancreatic mucinous tumour. The evidence for the relationship with pancreatic cancer, however, is currently weak. In the present investigation, risk factors for PC were retrospectively assessed in a sizable cohort of patients who underwent EUS. Further research has revealed a direct link between FP and the onset of ductal adenocarcinoma and pancreatic intra-epithelial neoplasia. Furthermore, it has been demonstrated that FP worsens the prognosis of PC since it encourages the spread of pancreatic cancer. This association's postulated pathogenesis is the result of two patho-mechanisms [2].

The first is related to obesity, which is connected to oxidative stress and adipokine imbalance, which results in a low-grade chronic systemic inflammatory state that predisposes for recurrent pancreatitis, which is regarded as a significant risk factor for pancreatic cancer. The second one deals with how pancreatic fatty infiltration might contribute to the growth of the disease by causing steato-pancreatitis, which in turn causes damage to pancreatic cells, fibrosis, and ultimately cancer. Recent research has also demonstrated the importance of gut microbiota in the onset of PC and the progression of tumours. It was found that patients with PC had lower levels of gut microbial diversity, which was accompanied by an increase in potentially harmful pathogens like Enterobacteriaceae and a decrease in some beneficial probiotics like Bifidobacterium and butyrate-producing bacteria. This suggests that improving the microbiota of PC patients may be beneficial [3].

Approximately 34% of PC patients had pancreatic atrophy, according to a recent study, even though the authors themselves hypothesised that pancreatic atrophy would be a precursor to PC. In our investigation, we lacked information on the existence of pancreatic atrophy before PC was diagnosed. To accurately identify the effects of pancreatic atrophy in the absence of other concurrent causes at the time of diagnosis, additional research is necessary [4,5]. PC and dyslipidemia are related. We assume that it is not a coincidental association, but its coexistence with FP may provide an explanation. Additionally, PC was negatively correlated with male gender in our population (0.47, 95 % CI 0.24-0.89); this result is in contrast to the literature, which has shown that PC is slightly more common in males than in females. However, in our cohort, the average age in the PC group was significantly higher than in the non-PC group; consequently, the higher female-to-male ratio in the PC group may be explained by the fact that, in accordance with the Global Burden of Disease Study, females were more likely than males to develop PC.

Conflicts of Interest

The authors declare no conflict of interest.

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