EDITORIAL

Pancreatic Cancer Masquerading as Acute Pancreatitis: A Pernicious Predator

Pancreatic adenocarcinoma is recognized as one of the most lethal malignancies by both medical professionals and lay people alike. This tumor classically presents with painless jaundice, but when located in the pancreatic body and tail, abdominal and/or back pain coupled with weight loss are typical. Rarely, the initial manifestation is acute pancreatitis, and limited data suggest that this may occur in approximately 1%–7% of patients.[1,2] Importantly, we recognize that following acute pancreatitis, the incidence of pancreatic cancer is higher than that in the general population, especially in the first several years. It is unclear whether pancreatic cancer in the setting of acute pancreatitis is found at an earlier stage.[3] Limited observations suggest that risk factors for pancreatic cancer presenting with acute pancreatitis include older age, new-onset diabetes, and new-onset chronic pancreatitis.[4]

It is thus with great interest that we read the report from Luong et al in this issue of the journal.[5] Using data from a large integrated health-care system, this retrospective cohort study examined patients hospitalized for acute pancreatitis over a 10-year period who were subsequently diagnosed with pancreatic cancer. Patients were identified from records of abdominal cross-sectional imaging, both computed tomography (CT) and magnetic resonance imaging, who had 1 of these studies obtained up to 3 years prior to hospitalization and through the date of discharge. A natural language-processing algorithm was used to identify both parenchymal and ductal abnormalities. From their cohort, ultimately 9490 patients were included for analysis. Ninety-five percent of patients had at least 1 cross-sectional abdominal imaging study which included the pancreas within the previous 3 years. Fifty-two percent of patients had a CT scan at the time of acute pancreatitis. Not surprisingly, biliary tract disease was the most common cause of acute pancreatitis followed by alcohol use. Ninety-five patients (1%) had pancreatic cancer diagnosed within 3 years of the diagnosis of acute pancreatitis. Almost half of the patients had stage IV disease at the time of cancer diagnosis while 80% of them were ductal adenocarcinoma. Pancreatic head lesions were present in two-thirds of patients. The incidence of pancreatic cancer was highest in the first year following presentation (78%), and the incidence rate was much higher among older age patients, especially those older than 65 years. When examining risk factors for diagnosis, main duct dilatation was the highest risk factor with a hazard ratio of 6.6 (4.2–10.5). Other independent associated risk factors included unknown etiology of pancreatitis, male gender, and an underweight body mass index. Curiously, they did not identify smoking, history of chronic pancreatitis, or diabetes as risk factors, well established as predisposing factors.

It would have been very interesting to note the degree of inflammation for those patients who had imaging at pancreatitis diagnosis and whether that would have obscured the diagnosis. These data were not available based on their retrospective review. However, this has significant implications for management as marked inflammation can of course obscure a mass lesion but typically not ductal dilatation.

What are the implications of the study and how do we use the results? First, the main takeaways should be that any "older" patient with acute pancreatitis in whom the cause cannot be identified by routine diagnostic testing should undergo additional investigation to exclude pancreatic cancer. While not generally recommended, almost all patients presenting with acute pancreatitis have abdominal CT scanning performed in the emergency department. When scanning is performed, we must be certain a high-quality scan with intravenous contrast is performed. Ductal dilatation, especially if there is a focal cutoff (not discussed in this paper), should raise a significant "red flag." Subsequent magnetic resonance cholangiopancreatography or even endoscopic ultrasonography should be strongly considered here. Patients with apparent idiopathic pancreatitis who do not have cross-sectional imaging performed at diagnosis should also undergo high-quality cross-sectional abdominal imaging. Third, their length of stay was relatively short (~4 days), and, coupled with prior observations, most patients with pancreatic cancer presenting with acute pancreatitis have mild disease. In my experience, patients may present with abdominal pain and biochemical pancreatitis and yet have no inflammatory component. The tip-off is ductal dilatation, especially focal, and/or age. The carcinoembryonic antigen 19-9 concentration may be helpful when markedly elevated as lower-level elevations can be observed with biliary pancreatitis. In addition, since cancer is present, these patients do not typically return to baseline but have persistent smoldering symptoms, and this should also be a clue. Lastly, given the prevalence of cholelithiasis and alcohol use, one must be certain based on history and laboratory studies that these are indeed the cause, and if not, one must pursue other etiologies including cancer in the appropriate setting. We must always be on the lookout for pancreatic cancer in any older patient with apparent "idiopathic" disease and any focal pancreatic ductal dilatation as a pernicious predator may be lurking.

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