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Acute Pancreatitis Following Consumption of Cassava

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Abstract

Cassava is a low-protein, high-carb meal that has been linked in the past to tropical chronic pancreatitis in underdeveloped nations. Although it has recently been shown not to be the exclusive cause of chronic pancreatitis, it is still possible that excessive levels of cyanogens play a function as a cofactor in pancreatitis development. A common risk factor for both acute and chronic pancreatitis is alcohol consumption. However, the comparatively low incidence of pancreatitis among persistent drinkers suggests that other factors, like as genetics, food, and other toxins, are important in the onset of both acute and chronic pancreatitis. We describe a case of acute pancreatitis in a patient with a history of chronic alcohol use who had a lot of home-cooked cassava but no alcohol at the time. We suggest that eating a lot of cassava caused a sensitive pancreas to experience an acute pancreatitis episode.

Keywords: Cyanogens, Pancreatitis, Cassava.

Introduction

The third most significant source of carbohydrates in the tropics is cassava, commonly referred to as yucca or manioc. It is an edible starchy tuberous root that is especially well-liked by people in Southeast Asia, Africa, and Latin America. Communities that regularly consume cassava are aware that some preparation, such as soaking, cooking, or fermentation, is necessary to keep people healthy. Linamarin and lotaustalin, two cyanogenic glucosides that are converted to hydrogen cyanide, are found in raw cassava roots and leaves. Cassava poisoning has been linked to neuropathy, goiter, and acute cyanide toxicity. Due to the earlier discovery of tropical calcific pancreatitis (TCP) in people who were malnourished and resided in developing areas that consume cassava as a primary food source, it was previously believed that cassava was linked to TCP.

But as more information accumulates, cassava is no longer considered to be a significant risk factor for tropical calcific pancreatitis. Human case-control studies revealed cassava was not a risk factor, and rats given cassava for an extended period of time did not develop pancreatitis or diabetes. It is hotly contested whether reliance on high-carb, low-protein food sources, such cassava, causes a macronutrient shortage that leads to chronic pancreatitis. However, some data suggests that consumption of cassava may contribute to micronutrient deficiencies that result in antioxidant deficits, which may contribute to the onset of chronic pancreatitis.

Alcohol is to blame for 45% of cases of chronic pancreatitis and 30% of cases of acute pancreatitis. Acute pancreatitis affects 10% of chronic alcoholics, while chronic pancreatitis affects only 5–10% of alcoholics. Both illnesses' etiologies are still hotly contested. According to studies, some toxins, such as alcohol and possibly cyanide, such as pancreatic secretory trypsin inhibitor (PSTI) or serine protease inhibitor kazal type 1 (SPINK1) genes, may increase a patient's risk of getting pancreatitis.

We describe a patient with a history of chronic alcohol use who developed severe pancreatitis after chowing down on home-cooked cassava but without temporal alcohol consumption.

Case presentation

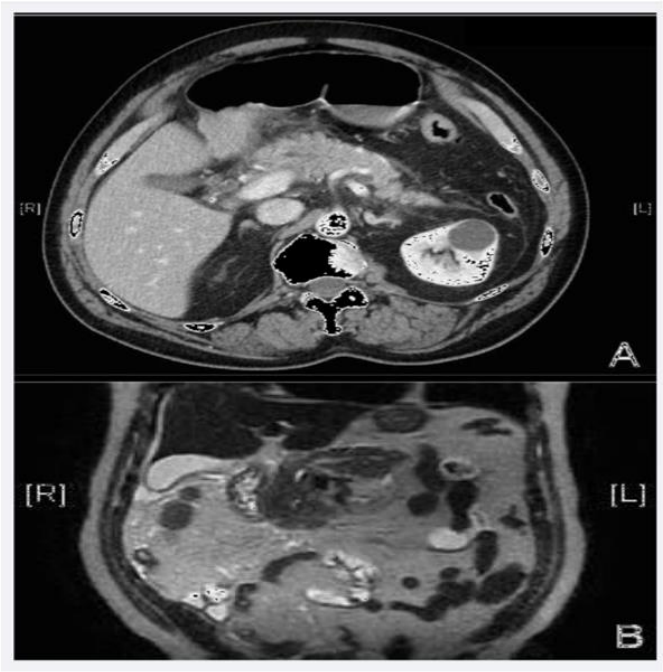
The symptoms of acute pancreatitis in a 50-year-old man with hypertension and persistent alcohol consumption (180 ml of whisky per night) included painful epigastric discomfort, stomach bloating, nausea, and three episodes of bilious non-bloody vomiting. Aspartate aminotransferase (AST) was 25 IU/L, alanine aminotransferase (ALT) was 34 IU/L, total bilirubin was 1.5 mg/dL, and indirect bilirubin was 0.9 mg/dL, according to the results of the tests. Triglycerides are 76 mg/dL and total cholesterol is 188 mg/dL. IgG subclass 4 levels of ANA negative screen of 7 mg/dL. No cholelithiasis could be seen on abdominal ultrasonography. An abdominal and pelvic computed tomography (CT) scan revealed peripancreatic inflammatory alterations that were consistent with acute pancreatitis and slight protrusion of the pancreatic and biliary channels. According to magnetic resonance cholangiopancreatography (MRCP), there were no signs of pancreatic or biliary ductal strictures or stones, only peripancreatic inflammation.

The patient reported consuming about 400 grams of home-cooked cassava on the day of admission while at a friend's party. On the day of the presentation, alcohol was not consumed. He claimed not to regularly consume cassava. Cassava consumption in the past has been trouble-free. He denied that his drinking had increased recently. There was no history of cholelithiasis, biliary colic, or pancreatitis in the past. Olmesartan 40 mg/hydrochlorothiazide 12.5 mg daily for hypertension was one of the medications, along with aspirin 75 mg. Each night, he consumes about 180 ml of whisky. He used to smoke one pack a day for 11 years before giving it up 20 years ago. He measured 28.7 kg/m2 for his body mass index.

In addition to receiving more than 10 liters of intravenous fluids, the patient also received morphine for pain relief. To lower his blood pressure, losartan 50 mg was begun instead of olmesartan 40 mg/hydrochlorothiazide 12.5 mg. His amylase and lipase levels fell to 189 U/L and 43 U/L, respectively, after 48 hours. He was sent home after switching to an oral diet.

An endoscopic ultrasonography six weeks after discharge showed a heterogenous pancreas consistent with chronic pancreatitis. The peripancreatic inflammation had subsided, and the previously noted pancreatic cyst had shrunk to a size of.78 cm by.92 cm, according to interval changes. Following up, the patient disclosed that he continued to consume 180ml of whisky every day,

avoided eating cassava, and noticed no return of his abdominal pain.



(A)Axial contrast-enhanced CT image demonstrates peripancreatic inflammatory stranding, compatible with acute pancreatitis. There is mild prominence of the pancreatic duct. A left renal cyst is incidentally noted. (B) - Coronal T2-weighted MR image shows peripancreatic inflammatory stranding, prominence of the pancreatic duct, and trace right upper quadrant ascites. No evidence of cholelithiasis or choledocholithiasis was seen.

Discussion

Acute pancreatitis can be brought on by cholelithiasis or alcohol in up to 60–75% of cases. By blocking the pancreatic duct with gallstones and causing a buildup of pancreatic enzymes, cholelithiasis results in acute pancreatitis. The pancreatic glands are subjected to oxidative stress from ethanol and its metabolites, which leads to premature activation of zymogens in acinar cells. Other less common causes of acute pancreatitis include drug-induced pancreatitis, infection, hypertriglyceridemia, trauma, autoimmune pancreatitis, and idiopathic.

The patient's continuous alcohol usage and prescription for low dose hydrochlorothiazide 12.5mg daily were two of his main risk factors for acute pancreatitis. Since there were no gallstones detected on any of the confirmatory imaging examinations, cholelithiasis was ruled out as the etiology of the acute pancreatitis.

Given that the triglyceride levels, ANA screen, and IgG subclass 4 values were all within the normal range, hypertriglyceridemia and autoimmune acute pancreatitis were also ruled out. Numerous studies have demonstrated that drinking alcohol and using tobacco together may more than double the chance of developing acute pancreatitis. The same study, however, showed that people who had given up smoking for more than 20 years reduced their risk levels to those of never-smokers, so eliminating smoking as a risk factor in this patient.

In our search of the literature, we were unable to find any earlier studies linking ingestion of cassava to the development of acute pancreatitis. On the other hand, there were numerous publications on its conceivable connection to chronic pancreatitis. Due to its geographic relationship with endemic areas, it was formerly thought that starvation and chronic cyanide toxicity of cassava were important risk factors for tropical calcific pancreatitis. Through the use of case control studies, rat models, and a sizable population of TCP patients who do not consume cassava, numerous research conducted more recently have failed to establish cassava as the only causal agent of TCP. The development of pancreatic injury may be influenced by increased cyanogenic glucosides and high cassava consumption, which are linked to nutritional shortages and low antioxidant levels. Combining cassava with other pancreatic toxins raises the possibility that it could harm the pancreas. The quantity and frequency of alcohol consumption affect the

risk of developing alcoholic acute pancreatitis. Alcohol sensitizes the pancreas to important micropathological events, such as the disorganization of cellular organelles, according to animal models. However, only 10% of all chronic users experience acute pancreatitis, indicating that environmental, dietary, and genetic factors may likely play a significant role in the onset of alcoholic acute pancreatitis. More specifically, alcohol contributes to the onset of chronic pancreatitis. According to several studies, alcohol is the main cause of 45%–90% of chronic pancreatitis. Pancreatologists continue to disagree over the timing of alcoholic acute pancreatitis and alcoholic chronic pancreatitis. According to published research, certain occurrences of acute pancreatitis affect healthy pancreas, while others affect pancreas with previous chronic conditions, as in the case of our patient. This shows that acute pancreatitis may be at risk in the latter grouping due to underlying chronic pancreatitis. Few empirical studies support the idea that chronic pancreatitis progresses as a result of recurring acute pancreatitis episodes. Therefore, we suggest that the chronic pancreatitis of our patient—likely brought on by long-term alcohol use—predisposed his pancreas to additional harm from other poisons, such cassava.

Conclusion

Many studies have been conducted on the effects of cassava consumption as an etiology of tropical calcified pancreatitis, and the results are largely negative. However, its part in depleting antioxidants raises the possibility that it may act as an immediate pancreatic damage trigger. Similar to how alcohol is well established to be a significant risk factor for acute pancreatitis, the fact that only a tiny percentage of chronic alcohol users experience acute pancreatitis suggests that additional cofactors are required. We suggest that acute high cassava consumption, when combined with chronic pancreatitis brought on by ethanol consumption, may be an additional risk factor for acute pancreatitis in our chronic alcoholic patient who temporarily experienced acute pancreatitis after devouring 400 grams of home-cooked cassava.

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