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Case Report

Acute Pancreatitis after Consuming 3.5 Lbs of Cassava

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Abstract

Cassava is a high carbohydrate, low protein food source that has been previously implicated to cause tropical chronic pancreatitis in developing countries Recently it has been disproven as a sole cause of chronic pancreatitis, however, its role as a possible cofactor (due to high levels of cyanogens) contributing to the development of pancreatitis cannot be ruled out. Alcohol is a well-known risk factor of both acute and chronic pancreatitis. However, the relatively small fraction of chronic alcohol users that develop pancreatitis suggest that additional factors, such as hereditary, diet, and other toxins, are necessary in the development of acute and chronic pancreatitis. We present a case of acute pancreatitis that occurred following gorging home-cooked cassava in a patient with a history of chronic alcohol use, but without temporal alcohol consumption. We propose that the gorging of cassava triggered an episode of acute pancreatitis in a sensitized pancreas due to chronic alcohol use.

ABBREVIATIONS

TCP: Tropical Calcific Pancreatitis; PSTI: Pancreatic Secretory Trypsin Inhibitor; SPINK1: Serine Protease Inhibitor Kazal type 1; AST: Aspartate aminoTransferase; ALT: Alanine aminoTransferase; CT: Computed Tomography; MRCP: Magnetic Resonance Cholangio Pancreatography.

INTRODUCTION

Cassava, also known as Yucca or Manioc, is the third most important source of carbohydrates in the tropics [1,2]. It is an edible starchy tuberous root that is most popular among the African, Southeast Asian, and Latin American populations. Societies that commonly consume cassava understand that some processing by soaking, cooking, or fermenting, is needed to prevent illness. Raw cassava roots and leaves contain linamarin, and lotaustalin, two cyanogenic glucosides that are converted to hydrogen cyanide [3,4]. Cassava toxicity has been associated with acute cyanide toxicity, goiter, and neuropathy [5]. Previously, cassava was felt to be associated with tropical calcific pancreatitis (TCP) due to the earlier observation of TCP in individuals who were malnourished and lived in developing areas that consume cassava as a major food source [5]. However, mounting evidence now deimplicates cassava as a major risk factor in the development of tropical calcific pancreatitis [5-7]: Case-control studies in humans showed cassava was not a risk factor [8] and rats fed cassava long-term did not produce diabetes or pancreatitis [9]. Macronutrient deficiency caused by reliance on high carbohydrate, low protein food sources, such as cassava, as an etiology of chronic pancreatitis is highly debated [6]. However, some evidence shows that micronutrient deficiency resulting in antioxidants deficit caused by cassava consumption may play a role in the development of chronic pancreatitis [8,10].

Alcohol is responsible for 30% of cases of acute pancreatitis in the United States [11], and 45% of chronic pancreatitis cases [12]. 10% of chronic alcohol users develop acute pancreatitis, and similarly, only 5-10% of alcoholics develop chronic pancreatitis. The mechanisms of both diseases are still highly debated. Studies have shown that diet and genetic variability in genes such as pancreatic secretory trypsin inhibitor (PSTI) or serine protease inhibitor kazal type 1 (SPINK1) may make patients more susceptible to developing pancreatitis from certain toxicities such as alcohol and possibly cyanide [13,14].

We present a case of acute pancreatitis that occurred following gorging home-cooked cassava in a patient with history of chronic alcohol use, but without temporal alcohol consumption.

CASE PRESENTATION

A 65 year old Hispanic man with hypertension, chronic alcohol use (3+ glasses of wine/night) presented with acute pancreatitis presenting with aching epigastric pain, abdominal bloating, nausea and 3 episodes of bilious non bloody vomiting. Labs revealed amylase 1420 U/L, lipase 1530 U/L, aspartate aminotransferase (AST) 27 IU/L, alanine aminotransferase

(ALT) 32 IU/L, Total bilirubin 1.4 mg/dL, indirect bilirubin .9 mg/dL. Total cholesterol 182mg/dL, triglycerides 72 mg/dL. Negative ANA screen IgG subclass 4 levels of 7mg/dL (Table 1). Abdominal ultrasound showed no cholelithiasis. Computed tomography (CT) scan of the abdomen and pelvis demonstrated peripandreatic inflammatory changes, which was compatible with acute pancreatitis and mild prominence of the pancreatic and biliary ducts. Magnetic resonance cholangiopancreatography (MRCP) showed peripancreatic inflammation without evidence of pancreatic or biliary ductal stricture or stones (Figure 1).

On the day of admission, the patient attended a friend's backyard barbecue where he reported ingestion of approximately 3-4lbs of home-cooked cassava (Figure 2). There was no alcohol consumption on the day of presentation. He reported no regular cassava consumption. Prior consumption of cassava was without incident. He denied any recent increase in alcohol consumption. There was no history of prior pancreatitis, biliary colic, or cholelithiasis. Medications included olmesartan 40mg/hydrochlorothiazide 12.5 mg daily for hypertension, and aspirin 81mg. He drinks approximately 3 glasses of wine per night. He is a past smoker of 1 pack per day for 11 years, and quit 30 years ago. He had a body mass index of 29.7 kg/m^2.

The patient received over 10 liters of intravenous fluids and received intravenous morphine for pain management. Olmesartan 40mg/hydrochlorothiazide 12.5 mg was discontinued and losartan 50mg was started to control his blood pressure. Within 48 hours, his amylase and lipase levels decreased to 189 U/L and 43 U/L respectively. He was transitioned to an oral diet and discharged home.

Six weeks following discharge, an endoscopic ultrasound revealed a heterogenous pancreas consistent with chronic pancreatitis. Interval changes demonstrated a resolution in

Table 1: Pertinent laboratory results obtained on admission, compared to normal laboratory values of the New York Presbyterian-Weill Cornell Hospital.

Laboratory test	Patient Results	Normal Values
White Blood Cell	15.0 x 10(3)/uL	3.4-11.2 x 10(3)/uL
Neutrophils %	89.8%	45%-75%
Amylase	1420 U/L	28-100 U/L
Lipase	1530 U/L	22-51 U/L
Aspartate aminotransferase	27 IU/L	
(AST)		
Alanine aminotransferase	32 IU/L	17-63 IU/L
(ALT)		
Total Bilirubin	1.4mg/dL	.3-1.2 mg/dL
Indirect Bilirubin	.9 mg/dL	.1-.8 mg/dL
Total Cholesterol	182 mg/dL	<= 200 mg/dL
Triglycerides	72 mg/dL	<= 150 mg/dL
ANA screen	negative	Negative
IgG subclass 4	7 mg/dL	7-89 mg/dL

Abbreviations: AST: Aspartate aminotransferase; ALT: Alanine aminotransferase.

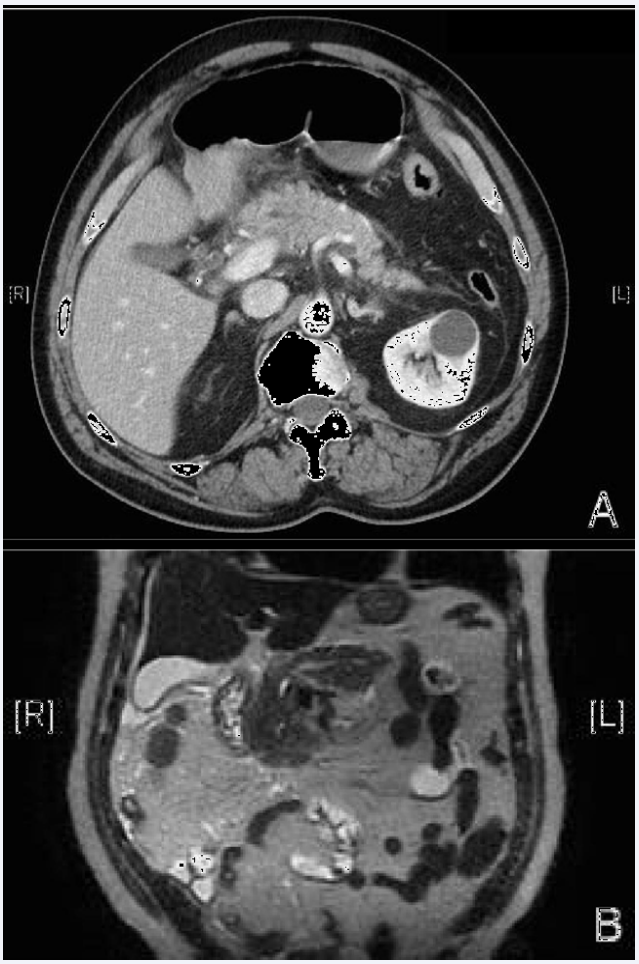

Figure 1 (A) - Axial contrast-enhanced CT image demonstrates peripancreatic inflammatory stranding, compatible with acute pancreatitis. There is mild prominence of the pancreatic duct. A left renal cyst is incidentally noted. (B) - Coronal T2-weighted MR image shows peripancreatic inflammatory stranding, prominence of the pancreatic duct, and trace right upper quadrant ascites. No evidence of cholelithiasis or choledocholithiasis was seen.

Figure 2 3.5 lbs of cooked cassava.

peripancreatic inflammation, and reduction in the previously seen pancreatic cyst to .78cm x .92cm. On follow up, patient reported that he continues to drink 3 glasses of wine daily, has abstained from consuming cassava, and reports no recurrences of abdominal pain.

DISCUSSION

Up to 60-75% of acute pancreatitis is caused by either cholelithiasis or alcohol. Cholelithiasis causes acute pancreatitis by the obstruction of the pancreatic duct by gallstones, leading to the accumulation of pancreatic enzymes. Ethanol and its metabolites cause oxidative stress on pancreatic glands, subsequently causing premature activation of zymogens within acinar cells [15]. Other less common causes of acute pancreatitis include drug-induced pancreatitis, infection, hypertriglyceridemia, trauma, autoimmune pancreatitis, and idiopathic [16].

In this patient, his major risk factors for acute pancreatitis included the chronic alcohol use and prescription of low dose hydrochlorothiazide 12.5mg daily. Cholelithiasis was excluded as a cause of acute pancreatitis given the lack of gallstones seen on several confirmatory imaging studies.

Hypertriglyceridemia and autoimmune acute pancreatitis were also excluded given triglyceride levels, ANA screen, and IgG subclass 4 levels all within normal range. Many studies have shown that smoking tobacco, in addition to alcohol use may increase the risk of acute pancreatitis by more than 2-fold. However, the same study demonstrated that patients who had stopped smoking for more than 2 decades reduced their risk levels to that of never-smokers, therefore excluding smoking as a risk factor in this patient [16,17].

In our literature review, we were unable to identify prior reports of association of cassava consumption to the occurrence of acute pancreatitis. However, there were many publications on its potential relationship with chronic pancreatitis. It was previously believed that the chronic cyanide toxicity of cassava in combination with malnutrition were significant risk factors of tropical calcific pancreatitis due to its geographic association in endemic areas [14]. More recently, multiple studies have failed to show cassava as the sole causative agent of TCP by use of case-control studies, rat models, and the large population of patients with TCP who do not consume cassava [8,9]. Increased cyanogenic glucosides and high cassava consumption is associated with micronutrient deficiencies and decreased levels of antioxidants, which may play a role in development of pancreatic damage [8]. It cannot be ruled out that cassava may cause pancreatic damage in combination with other pancreatic toxins. The risk of alcoholic acute pancreatitis is related to the amount and duration of alcohol use. Animal models have shown that alcohol sensitizes the pancreas to key micropathobiological processes, such as a disordering of cellular organelles [18]. However, only 10% of all chronic users develop acute pancreatitis, suggesting that other factors, such as environmental, diet, and genetics, play a major role in the development of alcoholic acute pancreatitis [18]. Alcohol plays a more definitive role in the development of chronic pancreatitis. Several studies suggest that alcohol is the primary etiology in 45%-90% of chronic pancreatitis [12,16]. The time-course of alcoholic acute pancreatitis and alcoholic chronic pancreatitis continues to be debated among pancreatologists [19]. Literature shows that some cases of acute pancreatitis occur in normal pancreata, while other cases occur in pancreata with preexisting chronic disease [20,21], as in the case of our patient. This suggests that in the latter subgroup, underlying chronic pancreatitis may be a risk factor for acute pancreatitis. Few empirical data show that recurrent episodes of acute pancreatitis lead to the progression of chronic pancreatitis [19,22]. Therefore, we propose that our patient's chronic pancreatitis, likely due to chronic alcohol use, predisposed his pancreas to further injury by other toxins such as cassava.

The effects of cassava consumption as an etiology of tropical calcified pancreatitis has been highly studied and mostly disproven [5-9]. However, its role in causing depletion of antioxidants, suggests a potential trigger for acute pancreatic damage [8,10]. Similarly, alcohol is known to be a major risk factor of acute pancreatitis, but its incidence in a small fraction of chronic alcohol users suggest that additional cofactors for acute pancreatitis are necessary [13,14,18]. In our chronic alcoholic patient who developed acute pancreatitis temporally after gorging of 3-4lbs of home-cooked cassava, we propose that acute high cassava may be an additional risk factor for acute pancreatitis when superimposed upon chronic pancreatitis resulting from ethanol ingestion.

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