A 45 Year Old with Idiopathic Pancreatitis

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ABSTRACT
In 2013, there were over 38,000 deaths related to pancreatic cancer in the United States, which resulted in pancreatic cancer being the fourth most common cause of cancer-related deaths in the US.1 Studies have reported that patients with hereditary pancreatitis were 87 times more likely to develop pancreatic cancer at the age of 55.2 Patients with hereditary pancreatitis are also susceptible to dependency on scheduled analgesics such as opioids. Here, we report the case of a patient diagnosed with idiopathic pancreatitis with a strong family history of pancreatitis.

INTRODUCTION
Chronic pancreatitis is a disease in which continuous or episodic pancreatic inflammation irreversibly destroys local and contiguous tissue. Though associated with many etiologies, it is most often attributed to alcohol abuse, ductal obstruction, autoimmune disease, or idiopathic origin.3 Our understanding of idiopathic pancreatitis, which represents one fourth of all patients with chronic pancreatitis, has been enhanced over the past decade by studies exploring the role of genetics. Mutations and gene locations have been identified which are associated with increased risk of the disease (e.g., SPINK1, PRSS1).4 The long-term management of patients with idiopathic and hereditary pancreatitis (HP) presents a daunting challenge for healthcare providers. Patients with idiopathic and hereditary pancreatitis often have an unpredictable course and experience significant comorbidity.

CASE PRESENTATION
A forty-five-year-old Hispanic male with type 2 diabetes mellitus and a seventeen-year history of chronic pancreatitis presented to the emergency department with a one-day history of epigastric pain, nausea, and vomiting. The patient also reported diarrhea of one month in duration. The epigastric pain was described as constant, sharp, and with radiation to the back and hip. There was no antecedent trauma, change in medications, heavy alcohol use, or any other inciting events. Furthermore, there was no fever, cough, palpitations, or acid reflux symptoms. The patient had experienced similar episodes in the past resulting in multiple hospitalizations. The patient was taking gabapentin and metformin at home and was allergic to ketorolac. The patient’s family history consisted of pancreatitis among his twin brother, father, and paternal grandfather. The father and grandfather both had died of pancreatic cancer. On physical examination there was epigastric tenderness to palpation, there was no abdominal distention, and no Murphy or Psoas signs. The body temperature was 36.9°Celsius, the pulse was 78 beats per minute, the arterial blood pressure ranged 146-160/62-90 mmHg, and the respiratory rate was 20 breaths per minute.

The patient was admitted to the hospital, his oral intake was stopped, and he was given IV hydration, as well as treatment with analgesics and anti-emetics. The laboratory work-up included a complete blood count, complete metabolic panel, serum lipase, glycated hemoglobin, cardiac enzymes, C-reactive protein, gram & leukocyte stains of the stool, fecal occult blood testing, stool ova & parasite and bacterial cultures, urinalysis, and C. difficile toxintests. Peptic ulcer disease, perforated ulcer, acute coronary syndrome, and infection were ruled out as etiologies of the patient’s abdominal pain and diarrhea.

The patient’s symptoms continued with no improvement of his nausea and vomiting. Unable to achieve adequate pain management, he developed insomnia and worsened anxiety. He was treated with alprazolam with minimal relief.

The abdominal ultrasound revealed a round mass at the junction of the pancreatic body and tail. A fine needle aspirate showed a typical ductal cells suspicious, but not diagnostic, of malignancy. The patient subsequently required a patient-controlled hydromorphone pump and a celiac plexus block for pain control. After the celiac plexus block, the patient was maintained on a liquid diet and then advanced to an 1800-Calorie diabetic/low fat diet.

After a 37-day stay at the hospital, the patient was eventually discharged to his home with ketorolac, hydroxyzine, and pantoprazole. He was instructed to to follow up with his gastroenterologist in one-to-two weeks after hospital discharge.

DISCUSSION
For patients with idiopathic pancreatitis, hereditary pancreatitis (HP) should be in the differential since patients with HP present in the same way as those with sporadic pancreatitis but at an earlier age. The diagnosis of HP typically relies upon clinical features: a positive family history spanning two generations with at least two others diagnosed with pancreatitis.5 Although HP is inherited in an autosomal dominant fashion, obtaining genetic studies appears not to be cost effective because the clinical management is similar to that for non-hereditary pancreatitis.

Continued on page 11
The clinical management involves pain management, counseling for tobacco and alcohol cessation to decrease the disease progression to pancreatic cancer. The benefit of diagnosing hereditary pancreatitis is the ability to detect pancreatic cancer at an earlier stage. The annual incidence to death ratio for patients with pancreatic cancer is high at 0.92 because it is always detected at a late stage.

CONCLUSION
The diagnosis of HP, even if made clinically, should always be in the differential diagnosis for patients presenting with recurrent pancreatitis. It is important for healthcare providers to provide patient education focusing on the progression of the hereditary pancreatitis to pancreatic cancer, the autosomal dominant pattern of inheritance, the importance of screening for pancreatic cancer, and for conscious control of analgesics.

REFERENCES


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1 in 5 persons living with HIV do not know it.

- People accessing health care are NOT routinely tested for HIV.

- Persons unaware of their HIV infection are unable to benefit from care.

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