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Case Report

Pancreatic Atrophy/ Insufficiency Incidentally Found in a Patient Newly Diagnosed with Crohn's Disease

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Abstract

Pancreatic atrophy is most commonly a consequence of cystic fibrosis, recurrent pancreatitis, or of anatomic variation of the organ. Recently, cases of pancreatic atrophy associated with inflammatory bowel disease have been reported, with the question of which entity being a precedent of the other remaining unanswered. We present a case of pancreatic atrophy/ insufficiencies incidentally detected in a patient ultimately diagnosed with Crohn's disease and review the literature of the association between these two entities.

ABBREVIATIONS

IBD: Inflammatory Bowel Disease; VTE: Venous Thrombo embolism

INTRODUCTION

Pancreatic atrophy/insufficiency is a rare finding in the pediatric population, and only recently has there been a purported association between this finding and inflammatory bowel disease (IBD). We present an unusual case of a pediatric patient who, in the process of being diagnosed with what was ultimately Crohn's disease, was simultaneously suffering from pancreatic insufficiency due to pancreatic atrophy.

CASE PRESENTATION

An anxious fourteen-year-old male with no significant past medical history presented to the emergency room with a one day history of severe left-sided chest pain, which was characterized as sharp and worsened with deep inspiration. The pain was a seven out of ten in intensity with no alleviating factors and did not radiate to his neck, shoulder, or back

The patient also complained of worsening left calf pain over the few days prior to presentation to the emergency department. He had been febrile for a few days prior, and had been experiencing intermittent episodes of shortness of breath without any cough, congestion, or night sweats. He denied any recent trauma. The only gastrointestinal complaint elicited was a history of two weeks of non-bloody diarrhea approximately two months prior to presentation, which resolved spontaneously. Additionally, he reported losing twenty pounds over the last few months prior to presentation.

In the emergency room the patient's vital signs were: temperature 102.6° Fahrenheit, heart rate 149 beats per minute, blood pressure 97/52 mmHg, and respiratory rate of 16-20 breaths per minute, with oxygen saturation ranging from 99-100% on pulse oximetry on two liters O_2 via nasal cannula. His body mass index was 34.1.

The only positive findings on physical exam were audible crackle in his left lower lung field and a swollen, warm, and tender calf.

Notable laboratory findings were a white blood cell count of 19 thousand per cubic milliliter (nl: 4.8-10.8thousand per cubic milliliter), a microcytic anemia based on a hemoglobin of 8 grams

per deciliter (nl: 11.1-15.7 grams per deciliter), mean corpuscular volume of 72.8 mcm^3 (nl: 77-87 mcm^3), and areticulocyte proportion of 2.6%. His erythrocyte sedimentation rate was 88 millimeters per hour and his D-dimer level was elevated at 1230 nano grams per deciliter (nl: 0-230 nanograms per deciliter) in addition to having a prolonged prothrombin time and elevated INR.

Radiographic work up included a chest x-ray which was normal and computed tomography of the chest was which revealed left lower lobe consolidation and left hilar lymphadenopathy. Venous duplex sonogram of the left leg revealed thromboses in the popliteal vein, anterior and posterior tibial veins and the peroneal vein. An electrocardiogram showed sinus tachycardia without ST elevations or T-wave abnormalities. The patient was started on enoxaparin for anticoagulation and intravenous antibiotics for treatment of presumptive left lower lobe pneumonia.

A complete gastroenterology and hematology workup was initiated to investigate the causes of the anemia and the deep vein thrombosis. Iron studies confirmed iron deficiency, but the stool guaiac was negative for blood. Fecal calprotectin was elevated at 160.1 micrograms per gram (abnl >= 120.1 micrograms per gram). Stool lactoferrin was positive as well. Serum albumin levels were decreased at 2.4 grams per deciliter (nl: 3.1-4.8 grams per deciliter).Magnetic resonance enterography of the abdomen was negative except for an incidental finding of an atrophic pancreas (Figure 1).

Evaluation of pancreatic function revealed pancreatic insufficiency, with a pancreatic elastase-1 level less than 15 microgram per gram (nl: >200 microgram per gram), 1-25 $(OH)_2$ Vitamin D level was low at less than 8picograms per milliliter (nl 24-86 picograms per milliliter), and a vitamin K level of 50 picograms per deciliter (nl: 80-1160 picograms per deciliter). Additionally, studies for fecal fat were positive.

The patient's thrombophilia workup was negative. However, after discharge in follow up his Factor VIII level and erythrocyte sedimentation level were both elevated at 209% (nl 60-125%) and 101 millimeters per hour (nl: 0-10 millimeters per hour), respectively. In light of these persistently elevated inflammatory markers, esophago gastro duodenoscopy and colonoscopy were performed which revealed erythematous mucosa in the esophagus, gastritis in the stomach, and diffuse superficial ulcerations, exudates, friability, and aphthous ulcers in the ascending colon, which were histological confirmed to be Crohn's disease.

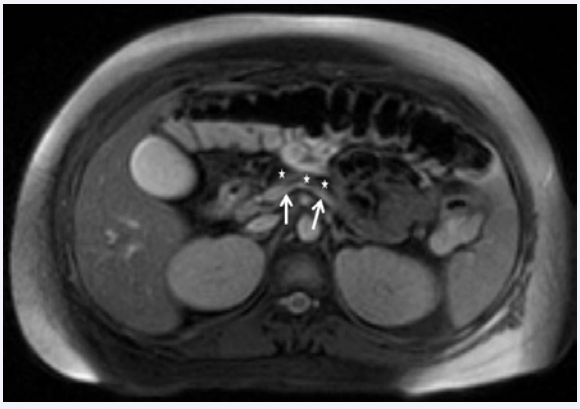

Figure 1 Axial MRI image demonstrates minimal to no pancreatic tissue in the expected location of the pancreas (stars) anterior to the splenic vein (arrows).

Once the underlying diagnosis of Crohn's disease was made, the patient was started on a corticosteroid taper and 5-aminosalicylic acid treatment, the latter of which he continues to take presently. Subcutaneous enoxaparin was continued for six months post-discharge from the hospital and discontinued when his inflammatory markers normalized. The microcytic anemia detected on admission has responded to iron supplementation, and he continues to take pancreatic enzyme supplements. He remains euglycemic to date.

DISCUSSION

Pancreatic atrophy in pediatric patients is most commonly the result of underlying cystic fibrosis (the test for which was negative in our patient) or recurrent pancreatitis, or a consequence of pancreas divisum or another anatomic variant, none of which was present in our asymptomatic patient [1]. Additionally, given the level of pancreatic parenchyma loss in our patient, symptoms relatable to pancreatitis, chronic or recurrent, primary or secondary, should have been present, which were not.

Pancreatic insufficiency in patients with IBD has been described in the literature and is thought to be a possible form of extra-intestinal IBD, especially in patients with a marked increase in stool frequency, or an independent autoimmune phenomenon in the context of IBD, though it may also be idiopathic [2-5].It is often difficult to determine which entity precedes the other, as their symptoms overlap, and the severity of pancreatic insufficiency may relate to the severity of the IBD [6].

Studies have shown that symptomatic chronic pancreatitis in pediatric patients may be indicative of underlying IBD [7]. Pediatric patients who have pancreatic disease in the setting of IBD can often present with elevated pancreatic enzymes, which initiates the investigation into their underlying etiology [5]. The elevated enzymes may occur in the setting of a completely asymptomatic patient and may be indicative of both underlying pancreatic and bowel pathology [5]. Our patient, however, did not manifest any elevation in pancreatic enzymes, most probably due to the profound loss of pancreatic parenchyma. His pancreatic disease was diagnosed via investigation of the gland's function after radiographic detection of its atrophy while remaining asymptomatic. This presentation is unique in the medical literature.

Venous thrombo embolism [VTE] occurs three times as often in patients with IBD than in the general pediatric population, with three times as many males experiencing this than females [8-9]. The increased clotting tendency in this cohort of patients has not been explained: in addition to not having an increased rate of genetic hyper coagulability states, no single abnormality in levels of pro coagulant, anticoagulant or fibrinolytic components in the blood, nor any individual endothelial, platelet, inflammatory or immune factor has been isolated as the cause of this phenomenon

[8].To date, no pediatric inpatient or outpatient guidelines exist regarding VTE prophylaxis for IBD patients[10].

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