

Blue Toe Syndrome: A Rare Complication of Acute Pancreatitis

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ABSTRACT

Context Blue toe syndrome is an unusual complication of acute pancreatitis. It is characterized by tissue ischemia secondary to cholesterol crystal or atherothrombotic embolization leading to the occlusion of small vessels. Clinical presentation can range from a cyanotic toe to a diffuse multiorgan systemic disease that can mimic other systemic illnesses.

Case Report Here we describe a young male who developed this complication after acute alcoholic pancreatitis.

INTRODUCTION

Acute severe pancreatitis is a serious condition which can result in both local and systemic complications. Systemic complications are referable to the gastrointestinal, cardiovascular, renal, hepatic and neurological systems and to vascular complications [1]. Their occurrence adds to the morbidity and mortality. Blue toe syndrome is an unusual complication and is characterized by tissue ischemia secondary to cholesterol crystal or atherothrombotic embolization leading to the occlusion of small vessels [2].

We describe a young male who developed blue toe syndrome as a complication of acute pancreatitis.

CASE REPORT

A 25 year old male, occasional smoker and social drinker presented complaining of acute pain in the upper abdomen two days prior to admission. The pain developed 8-10 hours after consumption of liquor (200 mL of country made liquor) at a party. Initially, he was treated by a private practitioner with antacids but then, as there was no relief, he was referred to our hospital. The pain was dull, aching, radiating to the back and aggravated upon eating. He had one episode of vomiting on the day of admission. On examination, he was febrile and had tachycardia. The abdomen was mildly distended and there was tenderness in the epigastric area. Chest examination, fundus examination and other systemic examinations were unremarkable. Investigations revealed hemoglobin of 11 g/dL, a total leukocyte count of 18,000/mm³, blood sugar of 118 mg/dL, serum cholesterol was 218 mg/dL and triglycerides were 146 mg/dL. Serum electrolytes, renal functions and liver functions were normal. Serum amylase was 944 IU/L. Ultrasonography of the abdomen revealed a bulky, hyperechoic pancreas with a few hypoechoic areas suggestive of necrotizing pancreatitis and ascites. Ascitic fluid amylase was 2,779 IU/L. A CT scan of the abdomen was not done as the patient could not be shifted. He was kept fasting and treated with intravenous fluids and analgesics for pain relief. Antibiotics (ciprofloxacin and

metronidazole) and antacids (ranitidine) were given. He responded well to the conservative management. One week after hospitalization, a bilateral bluish discoloration of the toes was noted. It was not associated with any pain or tenderness. The possibility of systemic embolization was considered. Echocardiography, a carotid Doppler study, a coagulation profile and a disseminated intravascular coagulation workup were all normal. The patient was started on aspirin, pentoxifylline and heparin. The parts were kept warm and undue handling was avoided. However, the patient developed dry gangrene of the little left toe. His condition improved steadily over the next seven days; the pain decreased and he was accepting orally but the gangrene of the little left toe persisted with resolution of changes in the other affected toes. He was discharged on day 14 after admission.

DISCUSSION

Cholelithiasis, ethanol ingestion and idiopathic pancreatitis constitute 90% of all causes of acute pancreatitis. Our patient had an acute attack after ingestion of country made liquor, where the concentration of alcohol in various batches is likely to differ, thus making it difficult to estimate alcohol consumption in grams.

The sudden development of cyanotic lesions on the feet may be a result of athero-embolic disease or a number of other medical conditions [3]. A careful anamnesis and physical examination, basic laboratory tests, and noninvasive vascular assessment usually distinguish between medical and surgical causes [4]. In our patient, an embolism from the heart was ruled out. Significant atherosclerosis was not seen in this young male on carotid Doppler. Disseminated intravascular coagulation may complicate acute pancreatitis and may result in vascular occlusion, however, there was no evidence of disseminated intravascular coagulation in our patient either clinically or biochemically.

Vascular involvement with cholesterol embolism has been established as a cause of vascular occlusion in acute pancreatitis in

various studies and is known to result in cerebral infarction [5, 6, 7] and retinopathy [8].

The blue toe syndrome is characterized by tissue ischemia secondary to cholesterol crystal or atherothrombotic embolization leading to occlusion of small vessels. Embolization can occur spontaneously or from a variety of insults such as invasive vascular procedures, anticoagulation, or thrombolytic therapy [2]. Pathophysiologically chylomicrons and VLDL have been shown to develop calcium dependent agglutination by C-reactive proteins in acute pancreatitis, which could result in vascular occlusion and resultant infarction [9].

Clinical presentation can range from a cyanotic toe to a diffuse multiorgan systemic disease which can mimic other systemic illnesses [2]. The differential diagnosis can be divided into three categories: emboli from the cardiac and arterial system, acquired hypercoagulability disorders, and syndromes which lead to peripheral vascular pathology [3]. When an embolism is suspected, ultrasound examination of the heart, aorta, and periphery is indicated to rule out the source of the embolus. Angiography is avoided in order to avoid exacerbation of the cholesterol crystal embolization. Cholesterol crystal embolization evolves in 3 clinical forms: 1) the "paucisymptomatic" form, not diagnosed during the subject's lifetime and only recognized in autopsy studies, 2) a "benign" form such as the blue toe syndrome or cutaneous livedo, with a spontaneous resolution and mild prognosis, and 3) a "diffuse multisystemic" form with a very poor prognosis [10]. Our patient had developed changes in the toes and in all but one, these changes resolved spontaneously. Development of gangrene in blue toe syndrome is unusual but was seen in our patient.

Management is conservative and surgery is rarely indicated because the source of cholesterol crystal embolization is not certain [10]. Prevention is the most effective treatment because in 30% of patients, embolization is due to anticoagulant drugs,

recent fibrinolysis, percutaneous angioplasty, vascular surgery and/or diagnostics angiography. However, vascular occlusion due to cholesterol embolism can occur spontaneously during acute pancreatitis [10]. The medical treatment is mostly symptomatic: resting the affected part, keeping it warm, an appropriate dressing, anti-platelet drugs to minimize disease progression and hydration to ensure normal renal function. In diffuse and multi-visceral embolization, either colchicine or corticosteroids adjuvant therapy might be useful; prostanoid drugs are also a possible adjuvant treatment [10].

CONCLUSION

Blue toe syndrome is usually associated with invasive vascular procedures, anticoagulation or thrombolytic therapy. In our patient, this complication was seen after an attack of acute pancreatitis. The clinical course and the absence of any other risk factor for atheroembolic disease make cholesterol embolism the only possible mechanism responsible for blue toes in our patient.

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