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CASE REPORT

Acute Pancreatitis as Initial Presentation of Cocaine-Induced Vasculitis: A Case Report

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ABSTRACT

Context Levamisole-contaminated cocaine is an increasingly reported cause of vasculitis and immunologic abnormalities in cocaine abusers. The systemic effects of vasculitis are commonly seen in the dermatologic, hematologic and renal systems but rarely the gastrointestinal system. **Case report** We present an atypical case of cocaine-induced vasculitis presenting initially as an acute pancreatitis and then rapidly progressing to involve multi-organ systems over the next couple of weeks. **Conclusion** Internists should recognize that acute pancreatitis can present as an atypical and rare initial systemic manifestation of cocaine-induced vasculitis.

INTRODUCTION

Acute pancreatitis as an initial presentation of Wegener's granulomatosis has been cited in only a few case reports. Since this presentation is rare, the diagnostic process is difficult and sometimes may lead to fatal outcomes [1]. Levamisole-contaminated cocaine is an increasingly reported cause of vasculitis and approximately 70% of cocaine in the United States is said to be contaminated with levamisole [2].

Levamisole contaminated cocaine has been known to cause agranulocytosis, leukoencephalopathy, or cutaneous vasculitis. Cocaine toxicity has also been associated with intestinal ulcers, ischemic colitis and infarction but pancreatitis from direct cocaine toxicity is extremely rare. We report such a case.

CASE REPORT

A twenty-two- year old male with no past medical history, presented with mid abdominal pain radiating to the chest with shortness of breath. Patient was sent to the emergency room for a chest computed tomography (CT) scan after a routine laboratory test at an urgent care center showed elevated D-dimer levels. Patient denied any fever, chills, recent travel or prior history of thromboembolic disease. Patient was adopted and does not know his family history. His blood pressure was 139/74 mmHg, heart rate 67 beats/minute; respiratory rate 18 cycles/minute, oxygen saturation was 98% on room air. Clinical examination showed mild epigastric tenderness, otherwise unremarkable. His chest CT scan was negative for pulmonary embolism but showed some stranding at the tail of the pancreas suspicious for acute pancreatitis (Figure 1). Routine laboratory tests showed white blood cell count of 14.4/µL, elevated lipase(754U/L), creatinine (0.79 mg/dL), ESR(1mm/h), C-reactive protein (0.08 mg/dL), alanine aminotransferase (23 U/L), aspartate aminotransferase(71 U/L), alkaline phosphatase(139 U/L), y-glutamyl transferase (209 U/L) reference range 1-54 U/L. Patient was kept nil per oral, intravenous normal saline and pain medications were commenced. Patient admitted to drinking some whiskey daily. A right upper quadrant ultrasound did not show any evidence of gallstones but revealed a fatty liver. His lipid panel and triglycerides were normal. A urine toxicology screen was positive for cocaine and marijuana. His ethanol blood level was normal.

On day 2-3 of admission, patient was noticed to have elevated creatinine (3.71 mg/dL) with an initial normal baseline creatinine and thrombocytopenia (59/µL). Subcutaneous heparin used for deep vein thrombosis prophylaxis was stopped due to suspected heparin induced thrombocytopenia and a platelet factor-4 antibody test was requested. The patient subsequently became oliguric with a urine output of (200 mL in 24 h) despite adequate fluid hydration (8 L/12 h). A renal ultrasound done did not show any hydronephrosis or stones. Attending physicians in the departments of gastroenterology, nephrology and hematology were consulted for better subspecialty care and my patient was closely monitored at the intermediate care-unit. A follow up magnetic resonance cholangiopancreatography (MRCP) was done which showed heterogeneous pancreas suggesting inflammation (Figure 2); otherwise his biliary anatomy was normal. My patient was thought

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to have an atypical form of pancreatitis since his clinical condition was fairly stable but his laboratory indices were worsening. An immunoglobulin g4 test was done to rule out auto-immune pancreatitis which came back negative. Other differential diagnoses entertained were hemolytic uremic syndrome/thrombotic thrombocytopenic purpura (HUS/TTP) or disseminated intravascular coagulopathy (DIC) but the definite diagnosis was still inconclusive. The patient's lab tests showed thrombocytopenia, anemia, negative platelet factor-4, elevated direct bilirubin, low haptoglobin, high lactate dehydrogenase, elevated D-dimer and positive fibrin degradation products. Schistocytes were seen on blood microscopy. An ADAMS-13 level was negative which ruled out TTP. Patient eventually became anuric and developed azotemia. A diagnosis of acute renal failure with a likely vasculitic process was made. Urinalysis showed muddy brown casts, proteinuria and white blood cells. Fractional excretion of sodium was 3.57. Work up for glomerulonephritis which included tests for human immunodeficiency virus (HIV), hepatitis, anti-neutrophil cytoplasmic antibody (ANCA) ,anti-nuclear antibody (ANA) ,anti-glomerular basement membrane antibody, anti-cardiolipin antibody ,scleroderma antibody ,complement C3/C4 were all negative. Hemodialysis was subsequently initiated and a kidney biopsy was eventually done which revealed pauci-immune necrotizing glomerulonephritis with features of glomerular thrombotic microangiopathy consistent with the patient's ingestion of levamisole (Figures 3, 4). Patient was commenced on high dose intravenous solumedrol and rituximab. He was transfused with nine units of packed red blood cells and he also had nine sessions of plasmapheresis. Patient's renal function and hematological indices improved over two months of treatment and he was discharged in stable clinical condition with no abdominal pain.

DISCUSSION

My patient had an ANCA negative pauci-immune necrotizing glomerulonephritis (thought to be induced by levamisole contaminated cocaine). Initially presented as an acute pancreatitis; this is atypical and rare making the diagnostic process challenging. Pauci-immune necrotizing glomerulonephritis is the most frequent cause of rapidly progressive glomerulonephritis and in 60% of cases. It is associated with positive anti-neutrophil cytoplasmic antibodies (ANCA). It is either the renal manifestation of Wegener's granulomatosis, microscopic polyangiitis, Churg-Strauss syndrome, or a renal-limited vasculitis. Immune conditions like systemic lupus erythematosus, polyarteritis nodosa and Wegener's granulomatosis have been reported to rarely cause pancreatitis although few case reports cite Wegener's granulomatosis affecting the gastrointestinal system with an initial presentation of pancreatitis which may be rapidly progressive and fatal [3]. Although my patient consumed alcohol which may be etiologically tied to pancreatitis, his clinical presentation was very atypical and as his vasculitic picture became clearer, we had to go searching for another etiology of his pancreatitis.

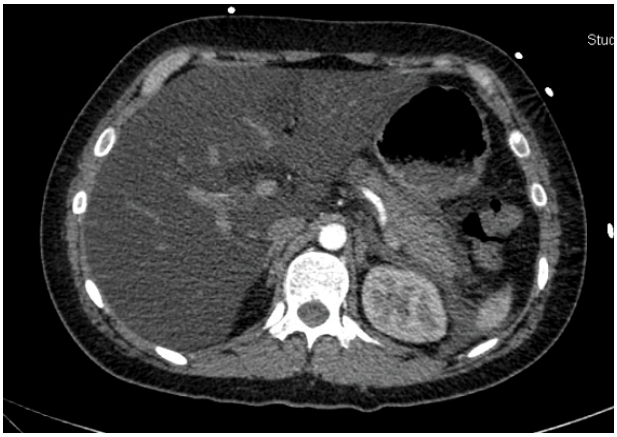

Figure 1. Abdominal view of CTA of the chest showing stranding adjacent to the body and tail of the pancreas with associated fluid suggestive of acute pancreatitis.

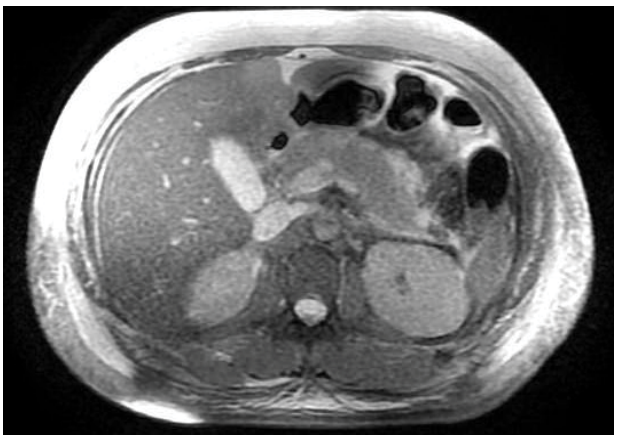

Figure 2. MRCP showing findings of thickened, heterogeneous appearance to the pancreas with adjacent fluid at the tail and body suggestive of acute pancreatitis.

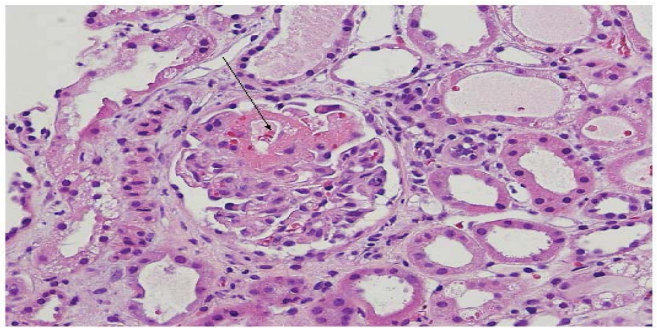

Figure 3. Segmental glomerular necrsosis necrosis

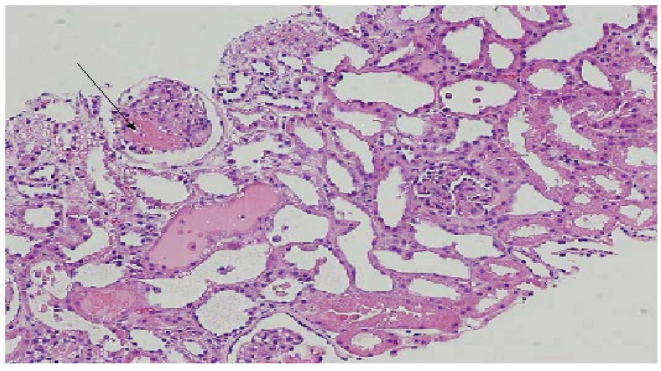

Figure 4. Severe acute tubular necrosis. Note segmental glomerular necrosis (arrow). H&E

CONCLUSION

First, internists should recognize that acute pancreatitis can present as an atypical and rare initial systemic manifestation of cocaine-induced vasculitis. Second, vasculitic disease of the pancreas, even though rare, should be considered when other causes of pancreatitis have been excluded as some cases have been reportedly fatal.

Conflict of Interest

Authors declare to have no conflict of interest.

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