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Beyond Gallstones and Alcohol: Acute Pancreatitis as a Sentinel Event of Primary HIV

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ABSTRACT

Acute Pancreatitis (AP) is an abrupt inflammation of the pancreas with diverse causes. Gallstones and alcohol account for approximately 70–80% of cases [1]. Other causes include metabolic disorders, medications, and, rarely, systemic infections. We present a 48-year-old woman with new-onset type 2 diabetes who was admitted for severe epigastric pain and acute kidney injury. She had no history of alcohol use or pancreatitis-inducing drugs. Laboratory tests showed leukocytosis with marked lymphopenia; serum amylase was ~8× normal. Given the lymphopenia, Human Immunodeficiency Virus (HIV) serology was performed and returned positive, indicating acute HIV-1 infection. Contrast CT confirmed interstitial pancreatitis (Balthazar grade C) without necrosis. The patient received aggressive IV fluids, electrolyte repletion, and early enteral nutrition, and was started on antiretroviral therapy according to World Health Organization (WHO) guidelines [8]. Her condition improved over two weeks with normalization of enzymes and renal function. This case underscores that acute (primary) HIV infection can rarely present as unexplained acute pancreatitis, and suggests HIV testing in idiopathic pancreatitis, especially when accompanied by immunologic abnormalities like lymphopenia [3].

Abréviation:

- Acute Pancreatitis (AP)
- Human Immunodeficiency Virus (HIV)
- World Health Organization (WHO)
- White Blood Cells (WBC)
- Intensive Care Unit (ICU)
- Antiretroviral Therapy (ART)
- Nucleoside/Nucleotide Reverse Transcriptase Inhibitors (NRTIs)
- Cluster Of Differentiation 4 (CD3)

Introduction

Acute pancreatitis is an acute inflammatory condition of the pancreas, ranging from mild edema to severe necrosis. It is a leading cause of gastrointestinal hospitalization. The most common etiologies are gallstone obstruction and chronic alcohol abuse, together accounting for about 70–80% of cases [1]. Other well-known causes include hypertriglyceridemia, hypercalcemia, certain medications, and anatomical factors. Early pancreatic enzyme activation leads to acinar injury and systemic inflammation [9]. Management is largely supportive (Fluid resuscitation, pain control, and nutritional support) along with treatment of the underlying cause. Although guidelines (e.g. the revised Atlanta Classification) stratify severity by organ failure and complications, the initial management principles remain consistent across etiologies.

By contrast, infections are an uncommon cause of AP. Viral etiologies (such as hepatitis, mumps, and others) have been reported but account for only a small fraction of cases [5]. Notably, a recent systematic review of viral pancreatitis found that primary HIV-1 infection was identified in ~3.8% of virus-associated pancreatitis cases [5]. In HIV-infected patients, pancreatitis may occur due to the virus itself, opportunistic infections, or as a drug-related complication. Bitar, *et al*. reported the first pediatric case of AP revealing acute HIV infection, emphasizing that acute HIV should be considered in the differential diagnosis of unexplained pancreatitis [3]. We describe an adult patient whose acute pancreatitis led to the diagnosis of primary HIV-1 infection.

Case Presentation:

A 48-year-old woman with recently diagnosed, untreated type 2 diabetes mellitus presented with a 2-month history of intermittent epigastric crampy pain that had worsened over the past week. The pain became more constant and severe, accompanied by postprandial vomiting and greenish diarrhea. She reported anorexia, weight loss, and profound fatigue. There was no fever. She denied alcohol use, smoking, or illicit drugs, and was not taking any new medications (No antiretrovirals, or known pancreatotoxic drugs). Family history was non-contributory.

On admission, the patient was hypotensive (BP 90/60 mmHg) and tachycardic (HR 110/min) but afebrile. She appeared ill and dehydrated. Abdominal examination revealed marked tenderness in the epigastrium without rebound or guarding. Initial labs showed leukocytosis (WBC 15,000/μL) with pronounced lymphopenia (absolute lymphocyte count 500/μL), hematocrit 34%, and platelets normal. Serum lipase was ~8 times the upper normal limit. Renal function was impaired (BUN 50 mg/dL, creatinine 2.0 mg/dL), and potassium was low. Liver enzymes and bilirubin were normal. Triglycerides and calcium were within normal limits. Given the combination of unexplained pancreatitis and lymphopenia, an HIV-1/2 test was ordered and returned positive; viral load was high, consistent with acute seroconversion.

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Abdominal ultrasound showed a normal gallbladder without gallstones and no intra-/extrahepatic bile duct dilatation. Contrast-enhanced CT (portal venous phase) demonstrated diffuse pancreatic enlargement with homogeneous enhancement and surrounding peripancreatic fat stranding, consistent with interstitial edematous pancreatitis (Balthazar grade C) (Figure 1). There were no peripancreatic fluid collections or areas of non-enhancement/necrosis (CT Severity Index = 2). No biliary pathology or other intra-abdominal cause was identified. On axial slices, the anteroposterior pancreatic thickness measured approximately 2.2 cm at the neck (Figure 2) and 3.5 cm at the body (Figure 3).

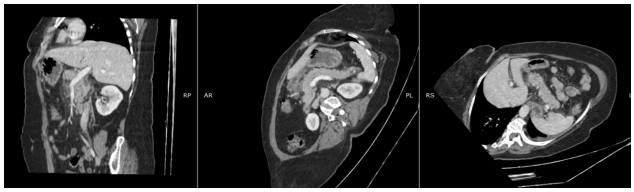

Figure 1: Contrast-enhanced CT (portal venous phase): interstitial edematous pancreatitis (Balthazar grade C) with homogeneous enhancement and peripancreatic fat stranding; no necrosis or fluid collection.

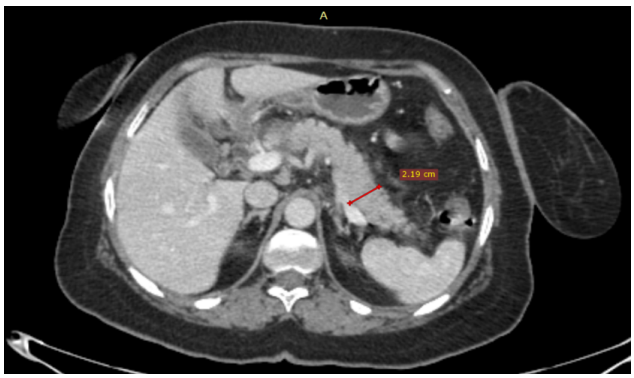

Figure 2: Axial contrast-enhanced CT at the pancreatic neck: anteroposterior thickness 2.19 cm with surrounding fat stranding; homogeneous enhancement and no biliary dilatation.

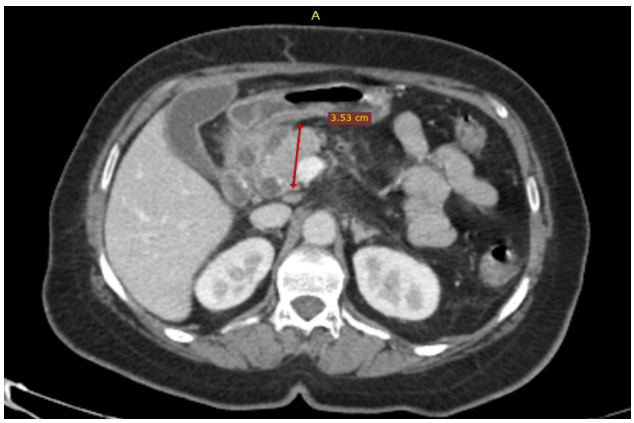

Figure 3: Axial contrast-enhanced CT at the pancreatic body: anteroposterior thickness 3.53 cm with peripancreatic inflammatory stranding; no focal non-enhancement/necrosis or walled-off collection.

The patient was admitted to the ICU for intensive management. She received aggressive IV crystalloid resuscitation and correction of electrolyte abnormalities. Epidural analgesia was used for pain control. Early enteral feeding was initiated via a nasojejunal tube. Continuous renal replacement therapy was started for uremia due to acute renal failure. An infectious disease consultation was obtained. In accordance with WHO recommendations to treat all HIV-infected patients [8], combination antiretroviral Therapy (ART) was initiated promptly after stabilization. A regimen of tenofovir disoproxil/emtricitabine plus dolutegravir was chosen, avoiding older NRTIs (e.g. didanosine, stavudine) associated with pancreatitis.

Over the next 10 days, her abdominal pain and inflammatory markers gradually improved. Renal function normalized, and pancreatic enzyme levels fell. She remained hemodynamically stable with no local or systemic complications (no pseudocyst or organ failure beyond the initial course). The patient was discharged on oral ART and insulin for her diabetes, with outpatient follow-up arranged.

Discussion

Pancreatitis in HIV-positive patients is relatively common compared to the general population [6], but its causes and course differ from typical pancreatitis. In high-prevalence settings, a significant proportion of AP cases are associated with HIV. For example, Anderson and Thomson found that 16.9% of patients admitted for AP in South Africa were HIV-positive [2]. In that cohort, women and Black Africans predominated. Alcohol was a much less frequent etiology among HIV-infected patients (24.5% vs 68.3% in HIV-negative patients), while antiretroviral use was implicated in 35.8% of HIV-associated AP cases. These findings underscore the importance of considering HIV in idiopathic pancreatitis, especially in areas with high HIV prevalence.

The pathophysiology of pancreatitis in HIV involves multiple mechanisms [9]. Direct cytopathic effects of HIV on pancreatic tissue have been proposed, as well as immune-mediated damage. Opportunistic infections (CMV, Mycobacterium, Toxoplasma, Pneumocystis) can invade the pancreas when immunosuppression is severe.

Drug toxicity is also a major factor: older Nucleoside Reverse-Transcriptase Inhibitors (didanosine, zalcitabine, stavudine, zidovudine) are known to cause mitochondrial toxicity leading to pancreatitis [7], especially with high cumulative doses. Modern ART regimens have a lower pancreatic toxicity profile, although protease inhibitors and hypertriglyceridemia can occasionally contribute. In the present patient, AP developed prior to ART initiation, implicating the HIV infection itself (or an unidentified opportunistic process) in pancreatic injury.

Clinically, primary HIV infection can present with nonspecific symptoms (Fever, rash, lymphadenopathy, cytopenias). Our patient's pronounced lymphopenia prompted HIV testing. Indeed, Bitar *et al*., stressed that acute HIV should be in the differential diagnosis of unexplained AP at all ages [3]. In practice, persistent lymphopenia or other hematologic abnormalities in AP should trigger HIV screening. Early diagnosis is critical, as it allows timely management of both HIV and pancreatitis.

Management of HIV-associated AP follows standard guidelines: aggressive early fluid resuscitation, pain control, and nutritional support. We provided intensive care-level support for organ dysfunction (including temporary dialysis). Concurrently, we treated the HIV infection: current guidelines recommend initiating ART in all HIV-infected patients regardless of CD4 count [2,8], and this was done promptly once the patient was stabilized. We selected a pancreatic-safe regimen, avoiding didanosine and stavudine. In published cases, identifying and discontinuing an offending agent (when present) is key; our patient had not yet been on ART, highlighting that even primary HIV can cause AP.

This case reinforces that acute pancreatitis can be the first manifestation of HIV infection. Physicians should maintain a high index of suspicion for HIV in any patient with idiopathic AP, especially with risk factors or laboratory clues. In addition to Bitar's recommendation [3], our experience and the literature support routine HIV testing in unexplained AP cases. This approach can uncover otherwise hidden HIV and improve patient outcomes.

Conclusion

We report a rare case of acute pancreatitis as the presenting sign of primary HIV-1 infection. This underscores the need for HIV testing in patients with unexplained acute pancreatitis. Early recognition allowed prompt initiation of ART and targeted supportive care. Clinicians should consider HIV infection in the differential diagnosis of idiopathic pancreatitis, especially when typical causes are absent and immune suppression is suggested by laboratory findings.

Conflict of Interest: There are no potential competing interests.

Author Confirm: All authors have approved the manuscript for submission and equally contributed for the case.

Ethical Consideration: Not required.

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