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CASE REPORT

Acute Pancreatitis Secondary to Hyperlipidemia After Exposure to Biktarvy and Alcohol in a HIV-Positive Patient:A Case Report

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Abstract: The etiology of acute pancreatitis is multifactorial. Hypertriglyceridemia is the third most common cause of acute pancreatitis. Biktarvy, a recently approved human immunodeficiency virus (HIV) treatment, is a single tablet regimen in combination with bictegravir, emtricitabine, and tenofovir alafenamide (BIC/FTC/TAF). A multitude of studies have identified a high risk of weight gain and hyperlipidemia, a trigger for acute pancreatitis, with Biktarvy use. This is the inaugural case of acquired immunodeficiency syndrome (AIDS) with acute pancreatitis as a secondary complication of Biktarvy and alcohol exposure, accompanied by cholelithiasis and diabetes. This case highlights the need for lipid monitoring in patients on Tenofovir Alafenamide (TAF)-based regimens.

Keywords: HIV, acute pancreatitis, hyperlipidemia, BIC/FTC/TAF

Introduction

The incidence rate of acute pancreatitis is relatively low in the general population, with an estimated annual incidence of 13 to 45 cases per 100,000 population.[1] However, it is much higher in people with HIV and acquired immunodeficiency syndrome (AIDS).[2] The most frequently present risk factors for acute pancreatitis, such as HIV, Nucleoside Reverse Transcriptase Inhibitors (NIRTs), Protease Inhibitors (PIs), Cluster of Differentiation 4+ T lymphocyte (CD4+T lymphocyte) count, hyperlipidemia, cholelithiasis, or alcohol in HIV/AIDS patients receiving highly active antiretroviral therapy.[3] Some studies have shown that TAF can cause weight gain and dyslipidemia.[4–7]With the application of new integrase inhibitors, ART-induced acute pancreatitis is reduced. However, reports of Dolutegravir (DTG) cause acute pancreatitis in nearly 2 years.[8,9] As dolutegravir and bictegravir belong to the same therapeutic class, a plausible hypothesis is that class-effect toxicity may be the underlying cause. This report delineates a probable case of acute pancreatitis that was likely related to BIC/FTC/TAF.

Case Presentation

A 42-year-old male patient with a medical history of HIV, obesity, type 2 diabetes mellitus, hyperlipidemia, cholelithiasis, and kidney stones presents with mild abdominal pain, absent nausea and vomiting. He reports that the pain began 20 hours prior and his gastrointestinal symptoms intensified after consuming food. The patient has been infected with HIV for 6 years, with no history of virological failure, and a favorable immune status (last CD4+T cell count of 275 cells/mm^3). His previous antiretroviral therapy (ART) regimen, which included TDF/EFV/3TC, was modified two years prior to the present study to Biktarvy, motivated by considerations of simplification. The patient has been diagnosed with diabetes mellitus and has been treated with metformin (500 mg twice per day) for a period of five years. The patient's haemoglobin A1c (HbA1c) level is 7.6%. Two years prior, the patient's biological investigations revealed the presence of hyperlipidemia, characterized by elevated triglyceride levels of 3.3 mmol/L (normal range <1.70), in conjunction with cholelithiasis and fatty liver. The patient's body mass index (BMI) was 26 kg/m^2. Following the transition to Biktarvy, the patient exhibited an 8-kilogram

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increase in weight, resulting in a body mass index of 29.8 kg/m^2. It is noteworthy that the patient does consume alcohol on occasion; the most recent instance was two days prior, during which he imbibed two glasses of white wine.

A physical examination was conducted at the time of admission, which revealed that the patient was overweight (body mass index 29.8 kg/m^2, weight 88 kg, height 1.74 m). The patient's temperature was 99.8°F (37.7°C), heart rate was 148 beats per minute, blood pressure was 148/95 mmHg, respiratory rate was 18 breaths per minute, and the abdomen was soft with mild pain in the upper abdomen. The skin was without jaundice, pruritus, or xanthomas.

Biological investigations demonstrated the presence of the following: hyperglycaemic (plasma glucose: 11.17 mmol/L, reference range <6.10), hyperlipidemia (Triglycerides, TG: 24.7 mmol/L, reference range <1.70), hypercholesterolemia (Total cholesterol, TC: 12.2 mmol/L, reference range <5.70), elevated blood lipase (214 U/L, reference range <60), normal amylase (97 U/L, reference range <135),Hyponatremia (129 mmol/L, reference range >137), elevated white blood cells ($14.13*10^{\wedge 9}$/L, reference range $3.5–9.5*10^{\wedge 9}$), elevated rapid C-reactive protein (94mg/L, reference range <6). The infectious serologies, including cytomegalovirus, Epstein-barr virus, were negative. A subsequent computed tomography (CT) scan revealed that the pancreas exhibited signs of swelling, and the surrounding fat space appeared indistinct. The most prominent swelling is observed in the pancreatic head. The presence of fluid exudate is evident, with distribution along the right colonic paracolic gutter (eg, Figure 1). Two gallbladder stones of notable size were identified, with the largest measuring approximately 16 millimeters in diameter (eg, Figure 2). A magnetic resonance cholangiopancreatography (MRCP) was performed, but no obvious pancreatic duct abnormality was identified (eg, Figure 3).

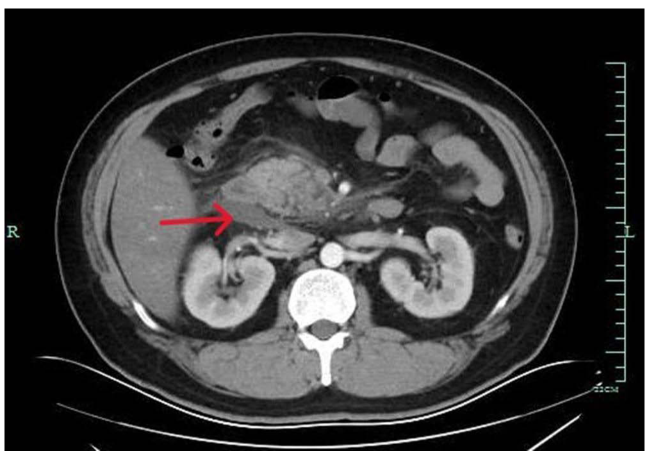

Figure 1 Abdominal Computed tomography (CT): the pancreas exhibited signs of swelling, and the surrounding fat space appeared indistinct. The most prominent swelling is observed in the pancreatic head. The presence of fluid exudate is evident, with distribution along the right colonic paracolic gutter (eg.the arrow mark).

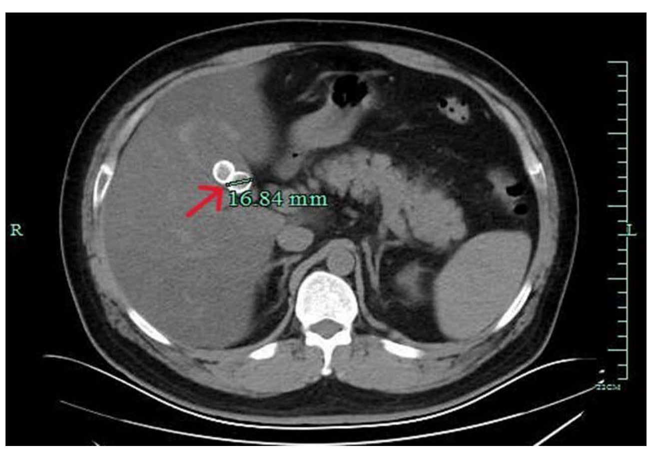

Figure 2 Abdominal CT:Two gallbladder stones of notable size were identified, with the largest measuring approximately 16 millimeters in diameter (eg.the arrow mark).

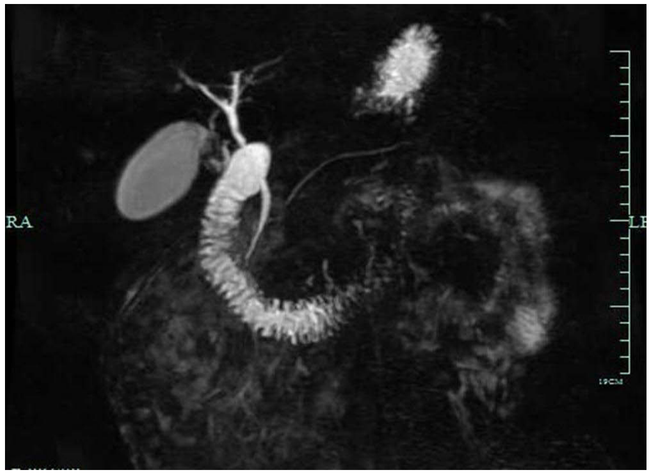

Figure 3 Magnetic Resonance Cholangiopancreatography (MRCP): no obvious pancreatic duct abnormality was found.

According to the 2012 Classification of acute pancreatitis—a revision of the Atlanta classification and definitions by international consensus—the patient satisfied all three of the diagnostic criteria for acute pancreatitis (eg, Box 1).[10] Consequently, the diagnosis of acute pancreatitis was made. According to the Revised Atlanta Classification of Severity of Acute Pancreatitis (eg, Box 2)[11] and the computed tomography severity index (CTSI) (eg, Box 3).[23] The CTSI score is calculated as the grading of acute pancreatitis plus the degree of pancreatic necrosis. A single accumulation of fluid is observed around the patient's pancreas. The acute pancreatitis is classified as grade D, which is scored 3 points. The absence of pancreatic necrosis is documented, resulting in a score of 0 points. The total score was 3 points. The patient exhibited no organ failure and was classified as mild pancreatitis.

Treatment

The standard treatment for acute pancreatitis resulting from hypertriglyceridemia involves the administration of fluids, the induction of fasting, the reduction of pancreatic secretion, the provision of nutritional support, and the management of

BOX 1 The Diagnostic Criteria for Acute Pancreatitis—Revised Atlanta Classification (2012)

Diagnostic criteria for acute pancreatitis require at least two out of the following three criteria to be met:
1. Abdominal pain suggestive of pancreatitis:Acute onset of persistent, severe upper abdominal pain, often radiating to the back
2. Serum enzyme elevation: Serum lipase or amylase level greater than three times the upper normal value
3. Characteristic imaging findings: Ultrasonography, computed tomography, or magnetic resonance imaging that shows irritation of the pancreas

BOX 2 Revised Atlanta Classification of Severity of Acute Pancreatitis

Mild acute pancreatitis requires neither the absence of organ failure (OF) nor local or systemic complications;
Moderate-severe acute pancreatitis is characterized by the presence of symptoms that are resolved within 48 hours (transitory OF) and/or the development of local or systemic complications without persistent OF.
Severe acute pancreatitis is characterized by the unrelenting (lasting > 48 hours) dysfunction of one or more organs

Notes: Local complications:interstitial edematous pancreatitis, necrotizing pancreatitis, pancreatic pseudocyst, necrotic collection, and pleural effusion.

BOX 3 CT Severity Index (CTSI)

A=Normal pancreas
B=Localized exudation, along with enlargement of the pancreas
C=Intrinsic pancreatic abnormalities, concomitant with or independent of inflammatory changes in the peri-pancreatic fat
D=peripancreatic fluid accumulation and cellulitis, typically located in the prerenal space
E=Fluid accumulation in the pancreatic or peripancreatic region, or the occurrence of fat necrosis in the peri-pancreatic area Score A-E:0–4 pancreatic necrosis score: none, 0; ≤ 30%, 2; ≥ 30%, 4; ≥50%,6.

Notes: The CT severity index (CTSI) has been demonstrated to be an effective scoring system for local pancreatic lesions. The severity of local lesions is determined by CT examination. The CTSI score is calculated as the grading of acute pancreatitis plus the degree of pancreatic necrosis. The following grading system is employed: mild pancreatitis, 0–3; moderate pancreatitis, 4–6; severe pancreatitis, 7–10.

pain. Etiologic treatment encompasses the administration of lipid-lowering agents, heparin, insulin, and plasma exchange.[12] Biktarvy is continued.

Insulin Drip

Insulin instillation for hypertriglyceridemia has been shown to be effective in lowering triglycerides below 500 mg/dL.[13] It also increases the levels of peripheral lipoprotein lipase, and decreases blood triglyceride levels. The dose was 0.1 to 0.3 U/kg/h by continuous intravenous pumping, and blood glucose was monitored every 30 minutes. Insulin accelerates the formation of chylomicrons and increases the metabolism of LDL.[12–14]

Heparin

Heparin can increase lipoprotein lipase, which helps break down triglycerides. It causes a short-term increase in lipoprotein lipase, but long-term use reduces the activity of this enzyme, leading to a net increase in triglyceride levels and also an increased risk of bleeding.

Plasmapheresis

Plasma exchange is the physical removal of triglycerides from the blood, resulting in a more rapid reduction of the level below 500 mg/dL.[12,15] The main advantage of plasma exchange over insulin in the correction of hypertriglyceridemia is that it is faster, but it is expensive and more suitable for severe patients.

Outcome and Follow-Up

Considering that although there are no typical symptoms of cholecystitis, the patient has an immune deficiency combined with diabetes, gallbladder stone 16 millimeters in diameter, which are high-risk factors. In case of an acute attack of cholecystitis, the infection progresses rapidly and the risk of complications is high. Therefore, an active intervention for surgical removal of the gallbladder is necessary. On hospital day 8, laparoscopic cholecystectomy was performed under general anesthesia. On hospital day 11, we rechecked blood parameters of the patient, plasma glucose (4.86 mmol/L, normal range <6.10), TG (2.75 mmol/L, normal range <1.70), TC (4.64 mmol/L, normal range <5.70), blood lipase (96 U/L, normal range <60), amylase (96U/L, normal range <135), white blood cells return to normal. CT showed that pancreatic exudation was less than before, and the patient's condition was improved, and he was discharged.

After being discharged from the hospital, the patient stopped drinking alcohol, maintained a regular diet and took lipid-lowering medications. Insulin was used to control blood sugar levels and keep them stableAfter 9 months, the patient was admitted to the hospital again due to abdominal pain. Computed tomography (CT) showed extensive exudate in the right retroperitoneal area secondary to pancreatic uncinate process inflammation, following cholecystectomy (eg, Figure 4). We checked the blood parameters of the patient: plasma glucose 9.3 mmol/L (normal range <6.10), triglycerides TG 14.96 mmol/L (normal range <1.70), total cholesterol TC 8.87 mmol/L (normal range <5.70), blood lipase (198 U/L, normal range <60),

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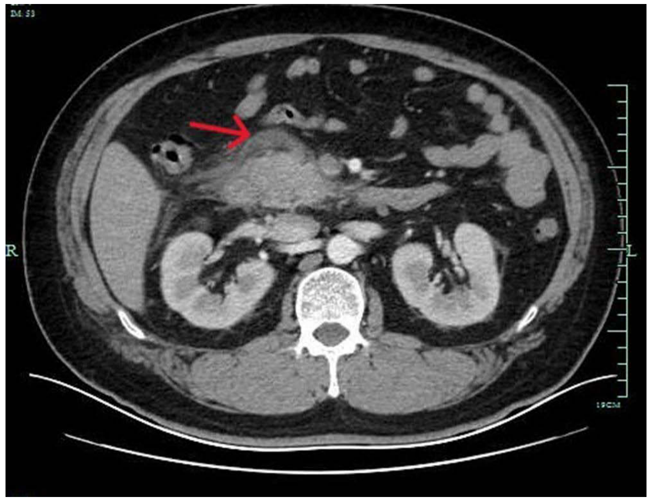

Figure 4 Abdominal Computed tomography (CT): extensive exudate in the right retroperitoneal area secondary to pancreatic uncinate process inflammation, following cholecystectomy (eg.the arrow mark).

amylase (152U/L, normal range <135). We consider the patient to have acute pancreatitis caused by hypertriglyceridemia, and provide fluid resuscitation, fasting, reduced pancreatic secretion, nutritional support, lipid-lowering drugs, and insulin. Finally, the patient improved and was discharged from the hospital. After eliminating the risk factors such as gallbladder-related issues, alcohol, and high blood sugar, the patient still experienced recurrent hyperlipidemia and triggered acute pancreatitis. We suppose that TAF may be pertinent to gaining weight and abnormal lipid metabolism, leading to hyperlipidemia, and subsequently triggered acute pancreatitis. We replaced the antiviral drugs with Ainuovirine, Tenofovir Disoproxil Fumarate and Lamivudine. After observing for one year, the patient's lipid levels remained stable and the pancreatitis did not recur.

Discussion

There is a wide range of factors that can affect the pancreas, from the direct lesion by the HIV itself, high viral load, low CD4 +T cell count, opportunistic infections, alcohol abuse, history of hepatobiliary or pancreatic disease, and ART.[16] From the literature, we found that HIV entry into pancreatic acinar cells mediates endoplasmic reticulum and oxidative stress, which triggers acinar necrosis.[17,18] The incidence rate of acute pancreatitis is relatively low in the general population; however, it is considerably higher in individuals with HIV and acquired immunodeficiency syndrome (AIDS).[24] Patients with hypertriglyceridemia-induced pancreatitis typically have a serum triglyceride level higher than 1000 mg per deciliter, have a more complex and severe course, and have higher mortality than common causes of acute pancreatitis.

Among antiretroviral therapies (ARTs), prior nucleoside reverse transcriptase inhibitor (NRTI) regimens have been associated with mitochondrial toxicity, resulting in mitochondrial dysfunction and subsequent pancreatic cell death. PI has been demonstrated to be associated with altered body fat distribution and metabolic disorders, including insulin resistance and hypertriglyceridemia.[16] Consequently, this results in the induction of acute pancreatitis. BIC/FTC/TAF in the Phase 3 study, grade 3 and 4 laboratory abnormalities were found, mainly elevated serum amylase, and no lead to pancreatitis.[19–21] Clotet B et al[8,9] reported integrase inhibitors may exert their effects on the anatomical structure of the pancreas (i.e. mitochondrial damage, induction of a prooxidative and/or proinflammatory state).[22] A screening of 6,423 patients who transitioned from TDF to TAF after a minimum of four weeks of use in the OPERA cohort was conducted to assess the lipid impact of switching from TDF to TAF in real-world clinical practice. This screening revealed significant increases in total cholesterol, low-density lipoprotein, and high-density lipoprotein levels subsequent to the transition from TDF to TAF.[2] We suppose that TAF may be pertinent to gaining weight and abnormal lipid metabolism, leading to hyperlipidemia, and subsequently triggered acute pancreatitis.

In our case, after switched from efavirenz to Biktarvy for antiviral treatment, the patient experienced weight gain, elevated blood lipids, and pancreatitis attacks following alcohol consumption. Following the cessation of alcohol consumption, modification of dietary habits, and regulation of blood sugar levels, the recurrence of pancreatitis was observed. In order to prevent the patient from experiencing another episode of pancreatitis, the antiviral treatment plan

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was modified. Following a year of observation, no further episodes of pancreatitis were observed, thereby validating the efficacy of the drug substitution plan implemented.

The etiology of drug-induced pancreatitis remains to be elucidated. Previous understandings of its etiology have been primarily derived from case-control studies, case reports, animal studies, and other experimental data. Despite the relatively low incidence of drug-induced pancreatitis, there has been an observed annual increase in cases coinciding with the continuous introduction of new pharmaceutical agents. The recognition of drug-induced pancreatitis as a distinct clinical entity is imperative, and clinicians must prioritize its management and treatment. Presently, there are significant gaps in the epidemiological research on drug-induced pancreatitis in many regions worldwide. Research on the pathogenesis of drug-induced pancreatitis has seen some progress, but further in-depth studies are necessary to further elucidate the mechanisms involved.

Conclusion

A multitude of factors contributed to the development of the patient's acute pancreatitis. The available evidence does not support a direct causal relationship between the drug and the development of pancreatitis. More evidence is still necessary to determine if pancreatic morbidity is directly related to drugs used in HAART therapy or to other comorbidities.

The utilization of BIC/FTC/TAF necessitates the monitoring of potential pancreatic injury development (via assessment of blood lipids, serum lipase, and amylase), prompt intervention, and timely adjustment of antiviral medications. This case underscores the necessity for clinicians to exercise caution when prescribing new integrase inhibitors for patients with multiple underlying comorbidities, including gallbladder stones, diabetes, obesity, dyslipidemia, or those at risk of metabolic diseases. In such cases, close monitoring of the patient's lipid levels is imperative, or alternatively, the avoidance of such medications should be considered.

Abbreviations

AIDS, acquired immunodeficiency syndrome; HIV, human immunodeficiency virus; Biktarvy (BIC/FTC/TAF), bictegravir, emtricitabine and tenofovir alafenamide; TAF, tenofovir alafenamide; NIRTs, nucleoside reverse transcriptase inhibitors; PIs, protease inhibitors; CD4+T, cluster of differentiation 4+ T lymphocyte; CT, computed tomography; ART, active antiretroviral therapy; DTG, dolutegravir; ART, antiretroviral therapy; TDF/EFV/3TC, tenofovir disoproxil fumarate/efavirenz /lamivudine; MRCP, magnetic resonance cholangiopancreatography; TG, triglycerides; TC, total cholesterol; AP, acute pancreatitis.

Data Sharing Statement

The raw data supporting the conclusions of this article will be made available from the corresponding author.

Ethics and Consent

This case report was approved by the Ethics Committee of Xixi Hospital of Hangzhou. Informed written consent was obtained from the patient for publication of this case report and accompanying images.

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Disclosure

All authors report no conflicts of interest in this work.

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