CASE REPORT

Acute Necrotizing Pancreatitis in the Setting of CMV Viremia and AIDS: A Case Report and Review of Literature from 1980 to 2012

Stephen Osiro[1], Raza Muhammad[1], Jose Ruiz[2], Anatole Sladen[3], Njambi Mathenge[4], Kevin J Tiwari[4]

Departments of [1]Medicine, [2]Pathology and [3]Radiology, Woodhull Medical Center. Brooklyn, NY, USA. [4]St. George's University School of Medicine. Grenada, West Indies

ABSTRACT

Context Cytomegalovirus (CMV)-induced pancreatitis in patients with AIDS is a known entity with poor prognosis. **Case report** We report a case of a 43-year-old woman with AIDS and CMV viremia who was evaluated for hypotension and found to have severe necrotizing pancreatitis. The authors have also conducted a MEDLINE search for CMV-induced pancreatitis from 1980 to 2012 and reviewed the pertinent results. **Discussion** Until mid-1990s in the United States, pancreatitis due to CMV was mainly diagnosed at autopsy in AIDS patients. However, presumably due to the advent of antiretroviral therapy, there has since been a significant decline in the number of reported cases among these individuals. Rather, our review revealed that the occurrence of CMV-induced pancreatitis has since been described in a variety of clinical settings, ranging from patients on corticosteroid therapy to immunocompetent persons. **Conclusions** Clinicians need a high index of suspicion to timely diagnose CMV-induced pancreatitis as patients often present with non-specific signs and symptoms. As it occurred in our case, early intervention is crucial and may alter the outcome in such patients.

INTRODUCTION

Acute pancreatitis affects about 170 persons per million in the United States annually, and is often due to gallstones or alcoholism [1]. It may also be caused by hypertriglyceridemia, biliary tract procedures, and drugs [2]. Pancreatitis due to opportunistic infections remains a concern to clinicians since they tend to have atypical presentations and may cause severe necrosis with increased morbidity and mortality [3].

Cytomegalovirus (CMV) is estimated to be present in 40% to 70% of the world's population and 50% to 95% of HIV-infected individuals [4, 5]. In developed countries, more than 40% of the people are seropositive for CMV by the age of 40 years, with up to 90% seropositivity reported in Japan [3]. Although rare, CMV-induced pancreatitis is currently well-established in the literature and primarily occurs in immunocompromised individuals, such as those with AIDS [6], lupus [7, 8], and dermatomyositis [9]. It has also been reported in patients receiving corticosteroid

therapy and/or organ transplants [3, 10, 11, 12]. Based on reports by some authors, CMV can cause pancreatitis in immunocompetent patients as well [13, 14].

Most reports of pancreatitis due to CMV were fatal [3, 11, 15]. Favorable outcomes therefore depend not only on early clinical suspicion, but also timely management. In this article, we report an AIDS patient who developed acute necrotizing pancreatitis in the presence of severe CMV viremia and also review the pertinent literature from 1980 to 2012.

CASE REPORT

A 43-year-old immigrant female with AIDS first came to our emergency department complaining of intermittent post-prandial abdominal pain for 3 months. The pain was localized mainly around her epigastrium, and was associated with nausea, occasional vomiting, and watery diarrhea. She had developed food intolerance over this period, and as a result, had lost 32 lbs (14.5 kg)

Although the patient had been infected with HIV for two years, she was never on antiretroviral therapy. Her vision in both eyes was also diminished with near total blindness of the left eye. She was a non-smoker and denied alcohol use. Her past surgical history was significant for cholecystectomy. In the emergency department, she was febrile (101.4°F; 38.6°C) and tachycardic (heart rate: 139 beats/min). Chest X-ray
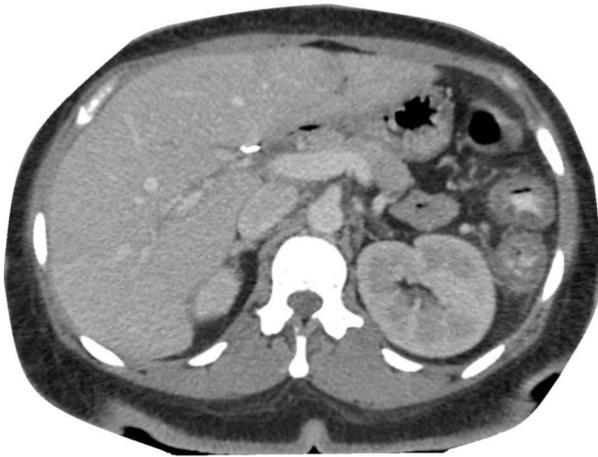
Figure 1. CT scan of the abdomen in our patient two weeks before developing acute pancreatitis.

was normal, but computed tomography (CT) scan of the abdomen showed non-specific pancolitis with no sign of acute pancreatitis (Figure 1). Her serum amylase and lipase were normal.

Further investigations revealed a low CD4 count of 14.2 μL^{-1} (reference range: 544-1,894 μL^{-1}) and significant cytomegalovirus viremia (CMV DNA, QN, PCR: 38,017 copies/mL; reference range: 0-200 copies/mL). Based on the symptoms and CT findings, the patient was admitted and managed for acute colitis during which she was also started on prophylactic trimethoprim-sulfamethoxazole and azithromycin. Notably, she became afebrile by day 1 and her abdominal pain and diarrhea were resolved by day 6. However, she was tachycardic throughout her hospital stay and was only discharged home at her insistence. No antiviral treatment for CMV viremia was given during her in-patient stay. It was only at discharge that she was prescribed oral valganciclovir. Four days later, she was seen at our follow-up HIV clinic where ritonavir, darunavir, emcitrabine, and tenofovir were also started.

At the GI clinic 11 days post-discharge, the patient appeared toxic, weak, and complained of mild, diffuse abdominal pain and dizziness. She was found to be
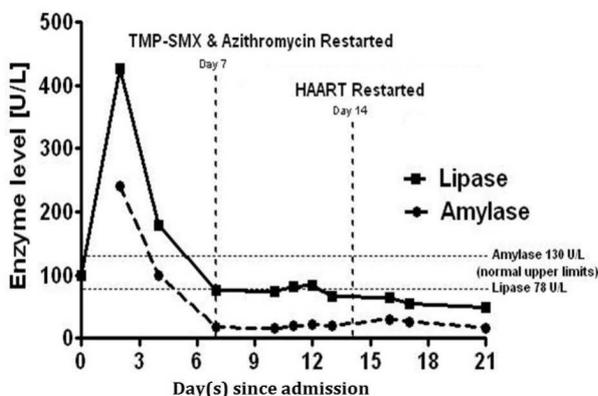
Figure 2. A graph showing changes in the levels of serum amylase and lipase in our patient from admission to discharge, correlating with the duration of her CMV-induced pancreatitis and its resolution.

hypotensive (blood pressure: 85/45 mmHg and tachycardic (heart rate: 134 beats/min). On day 2 after her readmission, she spiked a fever of 102.8°F (39.3°C) and had several episodes of non-bilious vomiting. Upright abdominal radiograph showed no free air or bowel obstruction. Her serum lipase increased from 99 U/L to 427 U/L (reference range: 8-78 U/L) within 48 hours (Figure 2), while her serum amylase was only elevated on day 2 of readmission (240 U/L; reference range: 40-130 U/L). Abdominal ultrasound (US) was negative for biliary duct disease. CT scan of the abdomen revealed enlarged and nonenhancing body and tail of the pancreas consistent with pancreatic necrosis (Figure 3). Cerebrospinal fluid analysis was positive for active shedding of CMV virus
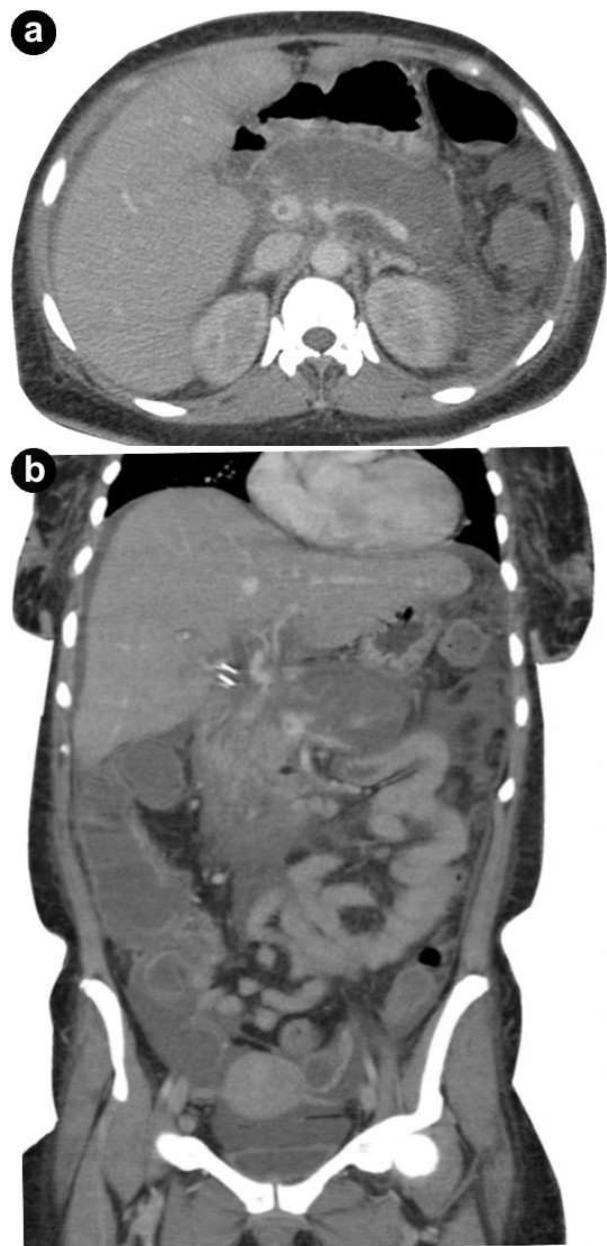
Figure 3. Coronal (**a.**) and sagittal (**b.**) CT scan of the abdomen showing enlarged and non-enhancing body and tail of the pancreas consistent with pancreatic necrosis.
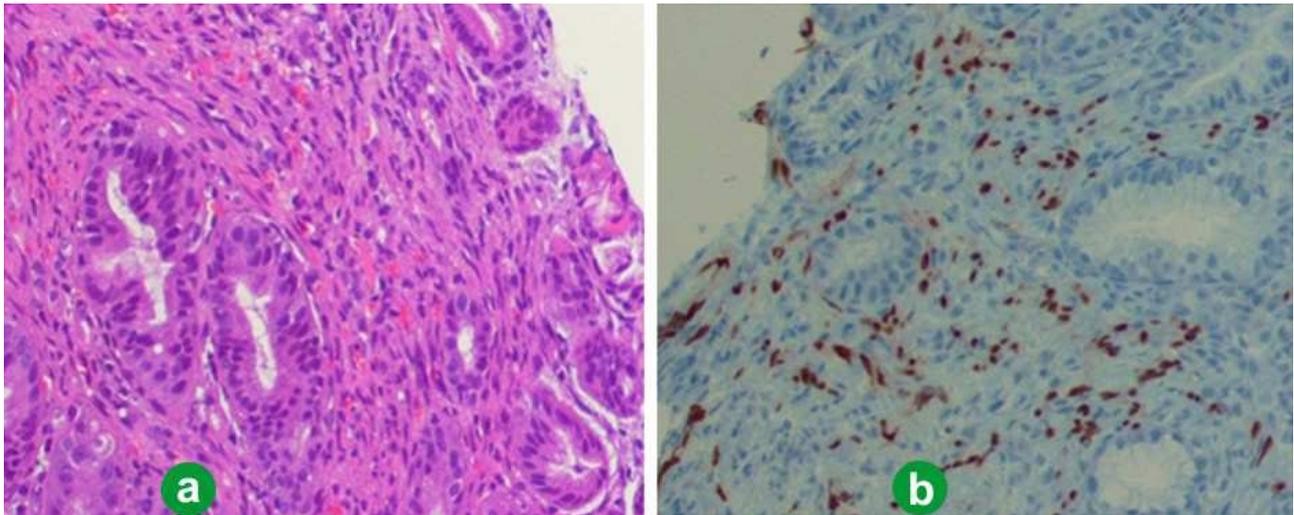
Figure 4. a. Gastric biopsy showing foci with spindle cells and proliferation of irregular jagged vascular channels lined by inconspicuous endothelial cells compatible with Kaposi's sarcoma. **B.** Immunohistochemistry studies showing human herpes virus type 8 (HHV-8).

(12,811 copies/mL) without signs of meningitis. The patient's serum anti-CMV IgG antibodies were elevated (118.8 EU/mL; reference range: 0-8 EU/mL), while anti-CMV IgM antibodies were negative. No other virus was detected in the blood or cerebrospinal fluid.

The patient was presumably diagnosed with CMV-induced acute pancreatitis and all her medications temporarily stopped. She was given intravenous (i.v.) fluids, analgesics, doripenem, and a 3-week course of intravenous ganciclovir. One week later, her pancreatitis resolved and she was restarted on trimethoprim-sulfamethoxazole and azithromycin. On hospital day 14, her antiretroviral therapy medications were also resumed. Upper endoscopy was performed which revealed multiple 0.1 to 2 cm firm nodules in the stomach and duodenum. The biopsy specimen was consistent with Kaposi's sarcoma (Figure 4). Ophthalmology examination revealed bilateral CMV retinitis (right eye more than left eye), with an old branch retinal artery occlusion of the left eye. After 4 weeks of in-patient management, a repeat abdominal CT showed resolved pancreatitis with dilated pancreatic duct (Figure 5). The patient was discharged home without any clinical evidence of recurrent pancreatitis.

DISCUSSION

CMV is a herpetic virus most often transmitted by blood transfusion, or intimate genital or oral contact. After subclinical primary infection, it establishes latency in the salivary gland cells, endothelial vascular tissue, renal epithelial tissue, T lymphocytes, and polymorphonuclear cells [16]. It is mainly considered an opportunistic infection and may reactivate spontaneously in the setting of an altered or suppressed immune system. The most susceptible individuals are therefore the elderly, transplant recipients, diabetics, and patients with HIV/AIDS [17]. Interestingly, recent literature indicates that severe CMV infections, such as

myocarditis, encephalitis, colitis, pneumonia, cholangitis, sinusitis, esophagitis, hepatitis, and thrombocytopenia can also occur in immunocompetent persons [13,18].

CMV as a cause of pancreatitis is rare, although pancreatic involvement has been reported in up to 10% of autopsy of patients who had CMV infection [19]. In this review, we looked into all the published case reports of CMV-induced pancreatitis in the literature from 1980 to 2012. In total, we reviewed 31 cases.

Figure 5. CT scan of the abdomen showing the relatively normal pancreas in our patient 3 weeks after resolution of her symptoms. Note the dilated pancreatic duct, a common sequelae of acute pancreatitis.

Pertinent features of CMV-induced pancreatitis become evident when all these cases are analyzed (Table 1). For instance, it mainly occurs in males. In addition, fever seems to be the most common initial complaint among the patients. However, fever alone is a non-specific symptom as patients may also present with epigastric pain, diarrhea, dysphagia, weight loss, anorexia, or no symptoms at all. In the early 1990s, presumably before the advent of antiretroviral therapy medications, CMV-induced pancreatitis was predominantly diagnosed at autopsy among AIDS patients, where histopathology showed CMV inclusions around the areas of pancreatic necrosis [19, 20]. Nevertheless, clinical diagnosis is currently attainable with CT or US-guided biopsy of the pancreas. Moreover, it is apparent that most cases of pancreatitis due to CMV tend to have concomitant necrosis, localized mainly in the tail [21].

Pathogenesis of CMV-Induced Pancreatitis

The exact mechanism by which CMV causes pancreatitis has not been fully understood. However, several studies indicate that it is potentially a three-step process, initiated by CMV-induced ischemic damage of the pancreatic tissue. According to Varani and Landini [18], CMV can infect endothelial cells and cause arteritis. It is believed that the resultant ischemia not only attracts and sequesters neutrophils, but also activates the proteolytic enzymes, such as trypsin, within the pancreas. Such enzymes and the inflammatory mediators released by the inflamed pancreas then autodigest the cellular membranes and cause edema, fat necrosis as well as parenchymal cell necrosis [1]. The vascular damage and necrosis may then predispose to secondary bacterial infections which further propagate the cascade.

In our case, we speculate that CMV could have been involved in two ways. First, the virus could have caused subclinical pancreatitis, as was evident by the intermittent episodes of abdominal pain and vomiting in the presence of normal CT scans and serum biomarkers. However, with the administration of trimethoprim-sulfamethoxazole, a synergy of pancreatic acinar damage ensued, leading to overt pancreatitis. This premise is difficult to prove, since our patient had tolerated trimethoprim-sulfamethoxazole for two weeks before the onset of her abdominal pain, and continued to do so one week later. On the other hand, it can be argued that her post-pancreatitis tolerance of the medication was because the CMV viral load had been drastically reduced by the administration of i.v. ganciclovir. Nevertheless, the endoscopic finding of gastric Kaposi sarcoma strongly indicates that our patient's prior abdominal pain was less likely due to pancreatitis, but rather due to the cancerous lesions.
Our alternative theory is that CMV was exclusively responsible for the pancreatitis as a result of the severe immunosuppression in our patient. There is perhaps a

critical CD4 count and a threshold CMV viral load beyond which the subclinical CMV infection of the pancreas changes into severe necrotizing pancreatitis due to the cytotoxic effects of numerous CMV inclusions. We strongly believe that the latter was the case with our patient, as several autopsy studies show that the pancreatic changes in patients with CMV-induced pancreatitis are of chronic nature, suggesting a more indolent and prolonged inflammation [6, 22].

Differential Diagnosis

Acute pancreatitis has multiple causes, including gallstones, excessive alcoholism, hypertriglyceridemia, and drugs. Our patient denied alcohol use and had normal levels of triglycerides. Bile duct disease as the cause of her necrotizing pancreatitis was unlikely due to the negative sonography.
The drugs that are known to cause pancreatitis in AIDS patients include didanosine, pentamidine and trimethoprim-sulfamethoxazole. Although the mechanism by which these drugs cause pancreatic damage is unknown, it is thought that it could be either due to an allergic reaction or the generation of a toxic metabolite. Studies show that the cumulative dose of such drugs is the strongest risk factor [23]. The latter is probably the reason why most cases of drug-induced pancreatitis are often found in patients with renal impairments [24].
As previously stated, our patient was on trimethoprim-sulfamethoxazole for two weeks before the onset of her symptoms, well within the known window during which this medication causes organ toxicity. However, it is also important to note that she had no prior history of sulfa drug allergy or renal impairment that could have caused excessive plasma accumulation of the drug. In addition, while our patient had been on antiretroviral therapy medications for one week before the pancreatitis, the drugs she was taking and the short duration made them less likely as the cause.
Thus, although there was no CT-guided needle aspiration for confirmation, our patient's rapid response to i.v. ganciclovir greatly supported our diagnosis. In addition, the occurrence of a confirmed bilateral CMV retinitis further reinforced our suspicion that the pancreatitis was not an isolated finding, but was rather in tandem with other inflammatory changes taking place all over her body. We strongly suspect that the reason why oral valganciclovir given to our patient the week before could not avert her development of CMV pancreatitis was probably down to her poor oral intake in the setting of acute GI symptoms and intestinal Kaposi's sarcoma. The authors concede, though, that the definite diagnosis of CMV pancreatitis requires positive pathology findings. It is therefore difficult to ascertain whether her improvement was exclusively due to the initiation of i.v. ganciclovir therapy, or the discontinuation of the antiretroviral therapy medications, or both.

Table 1. Published case reports of CMV-induced pancreatitis in the literature from 1980 to 2012.

Author/Year	Age/Sex	Main symptom(s)	Clinical setting	Location	Necrosis on CT scan	Concomitant CMV infection	Diagnosis
Osiro *et al.*, 2012 (this report)	43F	Diarrhea, fever, epigastric pain	AIDS	Body, tail	Yes	Retinitis, colitis, esophagitis	Clinical (Suspicion) [a]
Perdan-Pirkmajer *et al.*, 2011 [7]	33M	Epigastric pain, anorexia	Systemic lupus erythematosus	-	No	-	Clinical (Suspicion) [a]
Terada, 2011 [3]	76M	Abdominal pain, fever	Vasculitis	-	Yes	Interstitial pneumonitis	Autopsy
Kamalkumar *et al.*, 2009 [17]	38M	Epigastric pain, fever, vomiting	Renal transplant	-	No	Cholangitis, ampullary papillitis	Clinical (Biopsy)
Tomonari *et al.*, 2006 [10]	31M	-	Acute myelogenous leukemia, blood transplant	-	-	-	Clinical (Suspicion) [a]
Oku *et al.*, 2005 [13]	55M	Jaundice	-	-	No	Cholangitis	Clinical
Oruğ *et al.*, 2004 [21]	29M	Abdominal pain	Renal transplant	Head, body, tail	Yes	-	Clinical (Suspicion) [a]
Klassen *et al.*, 2000 [25]	31M	-	Pancreatic transplant	-	-	Esophagitis, gastritis, duodenitis	Clinical (Biopsy)
	41F	Fever	Pancreatic transplant	-	-	-	Clinical (Biopsy)
	28F	-	Pancreatic transplant	-	-	-	Clinical (Biopsy)
	40M	Low grade fever, diarrhea	Pancreatic transplant	-	-	-	Clinical (Biopsy)
Ikura *et al.*, 2000 [8]	29F	-	Systemic lupus erythematosus	-	-	-	Clinical (Biopsy)
Colebunders *et al.*, 1994 [29]	-	-	HIV/AIDS	-	-	-	Clinical
	-	-	HIV/AIDS	-	-	-	Clinical
Cella and Gupta, 1992 [27]	45M	Fever, anorexia	HIV/AIDS	-	-	-	Clinical (Suspicion) [a]
Yasumoto *et al.*, 1992 [12]	27F	Abdominal pain, thirst, muscle weakness, anuria	-	-	-	-	Clinical (Suspicion) [a]
González-Reimers *et al.*, 1992 [30]	50F	Disorientation, mental obtundation, vomiting	HIV/AIDS	Head	Yes	Gastritis, pneumonitis	Clinical, Autopsy
Bäckman *et al.*, 1991 [31]	27F	Fever	-	Tail	Yes	-	Clinical (Suspicion) [a]
	44M	Fever		Head	Yes	-	Clinical (Suspicion) [a]
	35F	Fever		-	Yes	-	Clinical (Suspicion) [a]
	40M	Nausea, vomiting		Tail	Yes	-	(Suspicion) [a]
	32M	Fever, abdominal pain, diarrhea		Tail	Yes	-	Clinical (Suspicion) [a]
Wilcox *et al.*, 1990 [11]	-	-	HIV/AIDS	-	-	-	Autopsy
	-	-	HIV/AIDS	-	-	-	Autopsy
Bigio and Haque, 1989 [6]	-	Fever, abdominal pain	Chronic myelogenous leukemia	-	-	Acalculous cholecystitis	Clinical, Autopsy
Joe *et al.*, 1989 [15]	31M	Diarrhea, epigastric pain	HIV/AIDS	Head	No	-	Clinical (Biopsy)
Hinnant *et al.*, 1989 [32]	44M	Right upper quadrant pain, diarrhea	HIV/AIDS	-	-	Retinitis	Clinical
Wolf *et al.*, 1989 [22]	65M	Left arm weakness	HIV/AIDS	-	Yes	Pneumonitis, adrenal necrosis	Autopsy
	33M	Shortness of breath, diarrhea	HIV/AIDS	-	-	Pneumonitis	Clinical
Iwasaki *et al.*, 1987 [19]	50F	Dysphagia	-	Tail	Yes	-	Autopsy
Peterson *et al.*, 1980 [33]	-	Fever	-	-	-	-	Autopsy

[a] "Clinical suspicion" indicates reported non-biopsy confirmation of an active CMV infection through PCR, CMV serology, complement fixation CMV titer, or culture of pancreatic ductal washings in the setting of a clinically-diagnosed acute pancreatitis.
" - " indicates unknown status
AIDS: acquired immunodeficiency syndrome

Role of Serum Biomarkers and/or Radiology in the Diagnosis of CMV-induced Pancreatitis

The diagnosis of acute pancreatitis is primarily clinical, and is usually established by the detection of an elevated serum lipase (and sometimes amylase) at the onset of symptoms [25, 26]. We report no variation in the rise of these serum biomarkers in cases of CMV-induced pancreatitis compared to other causes.

Radiological findings in acute pancreatitis vary markedly, with one or more abnormalities reportedly seen in over 50% of the patients [7, 27]. However, the findings are often inconsistent and non-specific [15]. Some of the radiographic tests can also be severely limited in the setting of an acute abdomen. For instance, in our case, the gallbladder and pancreas were not conclusively identified on abdominal ultrasound. In addition, our first CT scan showed no inflammatory changes to the pancreas and only a repeat scan two weeks later revealed pancreatic necrosis. In lieu of this, we propose that when CMV-induced pancreatitis is suspected in a patient with the viremia, the diagnosis should be based on clinical aexamination (with or without an elevation in serum biomarkers), even if the radiological exams are normal. Whether serial CT scans could be beneficial in such cases is subject to debate, given the increased risk of radiation exposure.

Management Options

The ability of ganciclovir to alter the course of CMV infections in patients with AIDS is well-documented in the literature [18]. Thus, our patient was treated with conservative management in the form of nothing by mouth, hydration, morphine, antibiotics, and an intravenous gancyclovir for 4 weeks. By hospital day 3, she had marked improvement of her symptoms and after one week of therapy, the pancreatitis resolved.

A report by Orug *et al.* [21] indicated that early CT scanning and debridement improves the prognosis of patients with acute necrotizing pancreatitis. Nevertheless, although we agree that dynamic CT scans can help detect complications in such patients [28], the decision to debride the necrotic pancreatic tissue should be on a case-by-case basis. Surgical intervention was not considered necessary in our patient due to her rapid recovery on conservative therapy alone.

CONCLUSION

The authors have reported a case of CMV-induced pancreatitis in an AIDS patient and reviewed the published literature on this condition. Although it has been far less-reported in the United States among these patients over the last decade compared to the early 1990s, there remains the possibility of its recurrence due to globalization and immigration. In addition, our review revealed that CMV-induced pancreatitis could be more prevalent in immunosuppressed individuals as well as those with autoimmune disorders than previously thought. A high index of clinical suspicion is therefore needed to timely diagnose it since the resultant pancreatic necrosis often carries poor prognosis. On the other hand, as was in our case, timely intervention may significantly alter the outcome and lead to recovery.

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