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Acute pancreatitis secondary to acute undiagnosed lupus: A case report

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ABSTRACT

Introduction: Pancreatitis is the acute inflammation of the pancreas associated with mortality, commonly presenting to the emergency department with significant abdominal pain, nausea and vomiting, and in severe cases, acute respiratory distress syndrome (ARDS).

Case Report: We present a rare case of pancreatitis secondary to acute systemic lupus erythematosus in a young patient with no past medical history and undiagnosed systemic lupus erythematosus (SLE). The patient had no classic exposures or ingestions of common causes of pancreatitis, and underwent an extensive workup with prolonged hospitalization until the antibody investigations revealed acute SLE.

Conclusion: This case illustrates the importance of keeping autoimmunity as a potential cause of acute pancreatitis particularly in a patient with no other classic cause of pancreatitis. Despite SLE being described as a rare and uncommon cause of pancreatitis in patients with known SLE diagnosis, this case sheds significant light on pancreatitis being a first-time presentation in a patient with no past diagnosis of SLE. Awareness of this etiology may help prevent extended hospitalizations, decrease complications, and improve mortality in respective patients with undifferentiated acute pancreatitis.

Keywords: Autoimmune, Lupus, Pancreatitis, Rheumatology

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INTRODUCTION

Pancreatitis is the inflammation of pancreatic tissue, which may be acute or chronic in nature. An inflamed pancreas may release endogenous digestive enzymes such as proteases, which autodigest the pancreas and break down local surrounding tissue. In complex or untreated cases, pancreatitis may lead to increased systemic inflammatory markers, hypoxemia, ARDS, with the potential of high mortality. Diagnostic criteria for pancreatitis include two out of the three findings: Classic epigastric burning abdominal pain radiating to the back, elevated lipase >3× the upper limit of normal, and characteristic imaging findings on either computed tomography or ultrasound [1]. The current mainstay treatment of acute pancreatitis is volume resuscitation with intravenous (IV) fluids, electrolyte management, and addressing the underlying cause of the pancreatitis [2]. The common underlying causes of pancreatitis include gallstones, alcohol use, hypertriglyceridemia, abdominal trauma, hypercalcemia, and drugs [3]. Although autoimmune causes of pancreatitis have been

described in literature, cases pertaining to acute SLE, in particular in undiagnosed patients have been rare. With the mortality being up to 30% [4], addressing the underlying cause of pancreatitis is and should be a priority to prevent recurrence of pancreatitis episodes.

CASE REPORT

A 24-year-old male with no past medical history presented to the emergency department for four days of newly experienced epigastric pain with anorexia and non-bloody and non-bilious emesis. On presentation he denied history of new medications, supplements, alcohol or drug use, gallstones, fevers or chills. Physical examination on arrival was notable for an uncomfortable appearing patient with normal body habitus, mild diffuse alopecia capitis, focal epigastric and right upper quadrant tenderness. Physical exam otherwise non-peritonitic, without any rashes, joint swelling, or other findings suggestive of SLE. Vitals on presentation: blood pressure 145/87, pulse 77, temperature 98.1, respiratory rate of 18, SpO_2 of 100%. Serum laboratory investigations in the emergency department revealed the following pertinent and significant findings: White blood cell count of 3.29 thousands per microliter (K/uL) (Reference [Ref]: 4.80–10.80 K/uL), Hemoglobin 10.6 grams per deciliter (g/dL) (Ref: 14.0–18.0 g/dL), Sodium 134 millimoles per liter (mmol/L) (Ref: 136–145 mmol/L), Potassium 4.5 mmol/L (Ref: 3.5–5.1 mmol/L), BUN 17.0 milligrams per deciliter (mg/dL) (Ref: 6.0–23.0 mg/dL), Creatinine 0.77 mg/dL (Ref: 0.70–1.20 mg/dL), Glucose 94 mg/dL (Ref: 74–109), Anion Gap 7.0 milliequivalents per liter (mEq/L) (Ref: 8.0–16.0 mEq/L), and a lipase level of 961 Units per liter (U/L) (Ref: 13–60 U/L). All other initial laboratory investigations within normal limits or insignificant. Patient was resuscitated with three liters of lactated ringers, received a total of 8 mg of intravenous ondansetron, 20 mg of intravenous famotidine, and 4 mg of intravenous morphine, and eventually admitted to Medicine with the diagnosis of pancreatitis of unknown etiology for further workup and management.

Initial ultrasound of the right upper quadrant revealed a normal gallbladder, without any stones, gallbladder wall thickening, or common bile duct dilatation (Figure 1A and B). While admitted, patient underwent a computed tomography of abdomen and pelvis with intravenous contrast, revealing inflammatory changes in the right pericolic gutter, and moderate complex fluid in the abdomen surrounding the pancreas (Figure 2). No other acute findings were radiographically noted. Magnetic resonance imaging of the abdomen was performed to evaluate for soft tissue lesions potentially not evident on tomography, revealing worsening extensive peripancreatic edema extending into the pelvis bilaterally; otherwise without any radiologic evidence for pancreatic mass or intra-abdominal mass (Figure 3).

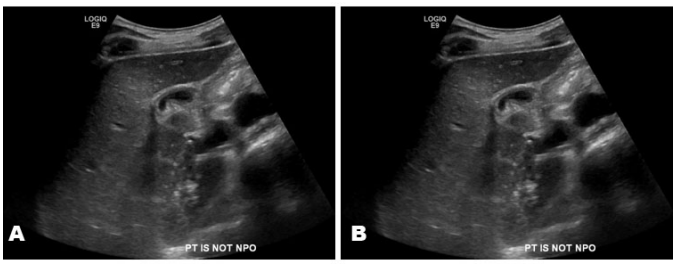

Figure 1: (A) Right upper quadrant ultrasound—Transverse view of the gallbladder—The gallbladder appears normal. No stones are identified. No biliary ductal dilatation or gallbladder wall thickening is present. (B) Right upper quadrant ultrasound—Longitudinal view of the gallbladder—The gallbladder appears normal. No stones are identified. No biliary ductal dilatation or gallbladder wall thickening is present.

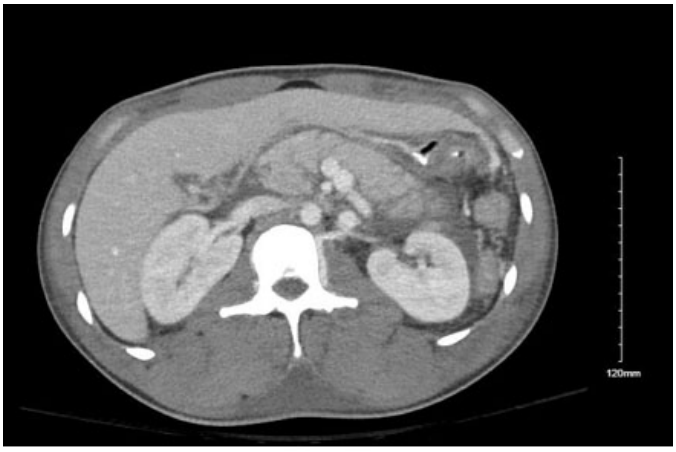

Figure 2: CT abdomen pelvis with contrast—Inflammatory changes in the right pericolic gutter. Moderate complex fluid in the abdomen and pelvis. There is fluid in the left pericolic gutter and around the pancreas.

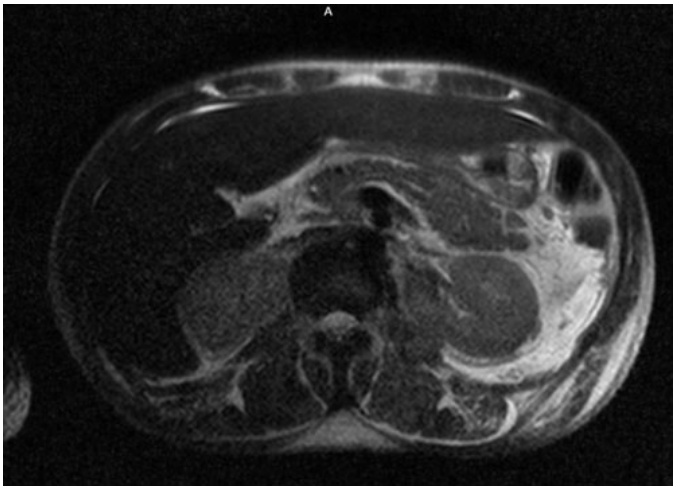

Figure 3: MRI abdomen without contrast—No evidence for pancreatic mass or intra-abdominal mass. Extensive peri-pancreatic edema as well as retroperitoneal edema extending into the pelvis bilaterally most likely related to acute pancreatitis.

Patient continued to receive intravenous boluses and maintenance fluids per standard of care for acute

pancreatitis, along with adjunct therapies including pro re nata (PRN) ondansetron and morphine.

Despite this, the patient's epigastric pain, and nausea and vomiting persisted. Furthermore, the patient's hospital course was complicated with progressive elevation of creatine phosphokinase, serum creatinine, hepatic transaminases, and serum lipase. The peak values were seen on day eight of hospitalization, with the following laboratory findings: creatinine phosphokinase 10,717 U/L (Ref: 20–200 U/L), serum creatinine 1.64 mg/dL (Ref: 0.70–1.20), BUN 49 mg/dL (Ref: 6.0–23.0 mg/dL), ALT 330 U/L (Ref: 10–45 U/L), AST 1472 U/L (Ref: 10–40 U/L), ALK Phos 322 U/L (Ref: 40–120 U/L), total bilirubin 10.40 mg/dL (Ref: 0.2–1.2 mg/dL), and lipase 2597 U/L (Ref: 13–60 U/L).

Throughout the first eight days of hospitalization, patient underwent an extensive panel of investigations including; urine and hematologic toxicology screen, hepatitis panel, lipid panel, acetaminophen level, salicylate level, ethanol level, malaria and parasitic panel, urine eosinophils, respiratory viral panel, syphilis screen, blood smear, Epstein–Barr panel, cytomegalovirus (CMV) panel, ceruloplasmin level, thyroid panel, hemochromatosis panel, cardiolipin, hemoglobin electrophoresis, measles mumps rubella panel, tuberculosis quantiferon; all returning either within normal or unremarkable limits.

On day nine of hospitalization, Anti-dsDNA antibody returned with a value of >1000 International units per mL (IU/mL) (Ref: normal ≤29 IU/mL). Antinuclear antibody 1:1280 (Ref: normal <1:80) in a homogenous pattern, Anti-Smith AB of 1.4 AI (immunoassay) (Ref: normal <0.9 AI), Chromatic antibody >8.0 AI (Ref: normal <0.9 AI).

Following the return of positive systemic lupus erythematosus serologies, Rheumatology consult was placed, and the patient was started on high dose methylprednisolone 1 mg/kg/day for three days. After the three day treatment with methylprednisolone, patient had the following improved serum laboratory results: Creatinine phosphokinase 537 U/L (Ref: 20–200 U/L), creatinine 1.04 mg/dL (Ref: 0.70–1.20), BUN 43.0 (Ref: 6.0–23.0), ALT 93 U/L (Ref: 10–45 U/L), AST 161 U/L (Ref: 10–40 U/L), ALK phos 292 U/L (Ref: 40–120 U/L), total bilirubin 2.44 mg/dL (Ref: 0.2–1.2 mg/dL), and lipase 250 U/L (Ref: 13–60 U/L). The patient made a full recovery and was discharged after a total of 25 days with a prescription for prednisone (5 mg twice a day), hydroxychloroquine (200 mg twice a day), and azathioprine (50 mg twice a day) maintenance therapy. The patient continues to have regular outpatient rheumatology follow up.

DISCUSSION

Systemic lupus erythematosus is a worldwide multisystemic autoimmune condition due to an impaired adaptive and innate immune system. The onset of disease process requires an interplay of a genetic predisposition, an impaired immune system, which may progress with environmental factors. The pathophysiology entails the immune system failing to clear apoptotic cells from the human body, which then cause the nuclear material of the cells and antigens to be exposed onto the cell surface [5]. The normal functioning T-cells and B cells will read these nuclear materials as foreign, and produce antibodies against them. These antibodies, being made against cells of the human body, become known as autoantibodies [6]. The human immune system thereby starts attacking various cells of its own.

Systemic lupus erythematosus is a challenging disease to diagnose as it can present in various organs, each with its own respective clinical feature. However, with rheumatologic advancements, many criteria have been developed to aid in the diagnosis. Antinuclear antibodies (ANA) is the most commonly found antibody in SLE patients, with a sensitivity of 97.8% [7]. Antinuclear antibodies are a subset of antibodies that bind to nuclear components such as proteins, RNA, histones and other intrinsic nuclear antigens [6]. These antinuclear antibodies are a feature of autoimmune connective tissue disease and can indicate the presence of autoimmune disease [8]. The American College of Rheumatology and the European Alliance of Associations for Rheumatology formalized the latest criteria for diagnosis in 2019; an ANA titer >1:80 with additional clinical criteria that total at least 10 points [8]. Furthermore the clinical diagnostic criteria also include whether there is the presence of fever, hematologic shifts, neuropsychiatric symptoms, mucocutaneous involvements, serosal involvements, musculoskeletal involvements, renal involvements, the presence of antiphospholipid antibodies, specific patterns of complement proteins, and of course, the SLE-specific antibodies such as Anti-DsDNA antibody or anti-smith antibody.

Systemic lupus erythematosus can carry significant morbidity for a patient, primarily due to it's potential to involve multiple organs and systems. Cutaneous findings, arthritis, and arthralgias are commonly seen. Renal involvement can be seen in 50% of patients, and has been noted as the strongest predictor of mortality [9]. Gastrointestinal complications have also been well-documented, including mesenteric vasculitis causing abdominal pain in 40% of patients [10]. Furthermore due to the immunocompromised impact and cardiovascular involvement, patients with SLE have up to three times higher mortality compared to the general population [11].

The initial management of SLE is directed toward preventing organ damage and reducing the disease activity to minimal levels. Treatment comprises various immune suppressants, immunomodulators, along with symptomatic pain control [12]. Patient education is given to avoid the variety of environmental triggers. Corticosteroids are effective for acute flares, with higher doses being used to treat major organ involvement, such

as lupus nephritis. High dose corticosteroid therapy as indicated for organ-threatening disease entails 1000 mg/day of methylprednisolone for three days [13]. Low dose corticosteroids are standardly used for maintenance therapy to prevent flares [12]. However, long-term corticosteroid use carries its own morbidity, and has been associated with irreversible organ damage, hence why adjunctive and alternative regimens have been utilized. As per the EULAR guidelines, hydroxychloroquine is the first-line immunomodulating treatment for SLE management recommended to all patients unless contraindicated [14]. Hydroxychloroquine decreases the number of flare-ups, while also being shown to reduce mortality by up to 50% compared to patients with SLE not on antimalarial therapy [15]. Cytotoxic immunosuppressants such as cyclophosphamide, mycophenolate, and azathioprine have also been shown to be very effective second-line agents for long-term prevention of flares [14]. For severe disease, biological agents such as rituximab, belimumab, and anifrolumab have also been approved by the food and drug association [14].

Pancreatitis is defined as either the inflammation or the fibrosis of the pancreas based on its acute or chronic presentation. Pancreatitis is one of the most frequent gastrointestinal reasons for hospital admissions accounting for up to $2.5 billion of healthcare costs [16]. With incidence of 13–45/100,000 for acute and 5–12/100,000 [17, 18], pancreatitis led to 275,000 hospitalizations in 2009, a two-fold increase since 1988 [19]. Acute pancreatitis can arise from alcohol use, gallstones, hyperlipidemia, hypercalcemia, drugs, infections, trauma, malignancies, autoimmune disorders, endoscopic retrograde cholangiopancreatography (ERCP), or it can be idiopathic. These inciting events can cause the inflammation and hemorrhage of pancreatic tissue which in turn can activate certain of its own endogenous enzymes and result in it's own autodigestion. This autodigestion can lead to the hemorrhagic necrosis of the pancreas and the fat necrosis of the peripancreatic fat [3]. Chronic pancreatitis is the fibrosis of the pancreatic parenchyma resulting from recurrent episodes of acute pancreatitis. It is most commonly caused by alcoholism in adults [20].

Both acute and chronic pancreatites typically present as epigastric pain that radiates to the back. Acute cases are associated with nausea, vomiting, and elevated serum lipase and amylase levels. On the contrary, chronic pancreatitis results in its own insufficiency, therefore presenting with decreased lipase and amylase levels, and downstream fat-soluble vitamin malabsorption secondary to loss of endogenous digestive enzymes [21].

The overall mortality rate of acute pancreatitis is currently up to 3%, with 30% for severe disease, translating to about 1.60 deaths in a 100,000 person population [4, 22]. Mortality odds increased with age, male gender, comorbidities such as renal disease, cardiovascular disease, hematologic abnormalities, hepatic disease, and sepsis [4]. The largest influential factor on mortality is the quick and accurate identification of pancreatitis within the first 48–72 hours of hospital presentation [1].

Management of pancreatitis is very crucial to prevent the progression of the disease and its complications. It is important to risk-stratify patients based on laboratory results, including the BUN/Creatinine, complete blood count, calcium, triglycerides, and lactate, to assess for end-organ damage and measurement of the systemic inflammatory response syndrome (SIRS) score.

With risk stratification, the severity of pancreatitis can be broken down into three categories: mild, moderate, and severe. Mild pancreatitis is classified as the lack of organ failure or systemic involvement [1]. Typically in mild pancreatitis, an average of 3.2 liters of fluid is sequestered and third spaced [23]. Moderate pancreatitis is classified as transient organ failure that lasts less than 48 hours, with some degree of systemic involvement [1]. In the moderate group, an average of 6.4 liters of fluid is sequestered [23]. About 20% of moderate pancreatitis cases progress to severe pancreatitis, which is classified as persistent multi-organ failure lasting greater than 48 hours [1]. Severe cases have a mortality rate of up to 30% [4]. In these patients, an average of 7.5 liters of fluid are sequestered [23]. The sequestration of fluid occurs secondary to inflammatory cytokines which cause localized and systemic vascular endothelial dysfunction [3]. This classification helps providers decide whether the patient is indicated for intensive care unit monitoring.

The initial management in the emergency department for acute pancreatitis is supportive with fluid resuscitation, pain control, and proper nutrition [2]. Fluid resuscitation with normal saline or lactated Ringer's solution has been shown to reduce morbidity and mortality as it counteracts the hypovolemia that could result in hypotension, pancreatic hypoperfusion, acute tubular necrosis, ischemic pain, and resultant lactic acidosis [24]. The current evidence regarding the volume of fluids for resuscitation entail a 10 mL/kg bolus for patients who are hypotensive, followed by 1.5 mL/kg/hour of maintenance fluids [25]. Furthermore, surgical causes of acute pancreatitis such as gallstone pancreatitis typically require operative intervention. In all cases, pain control can be achieved via the use of analgesics such as morphine, fentanyl, hydromorphone, and NSAIDs. Antibiotics may be provided as up to 25% of patients with moderate and severe pancreatitis develop extra-pancreatic infection such as urinary tract infection, pneumonia, and sepsis [26].

In the United States, up to 178 per 100,000 people have systemic lupus erythematosus [27]. There are a very limited number of cases reported with pancreatitis as the initial presentation of undiagnosed systemic lupus erythematosus. Literature reports have estimated that the annual incidence of SLE-related pancreatitis to be 1.1 out of 1000 individuals living with SLE [28]; however, limited literature exists to describe acute pancreatitis in the patient with undiagnosed SLE. With the mortality rate of pancreatitis being up to 30% [4], it is critical to identify

the underlying cause, in this case, SLE, to promptly treat the underlying autoimmunity with corticosteroids and biologics. It's important to highlight that corticosteroids and biologics are not standard of care for acute pancreatitis, as the majority of acute pancreatitis presentations are not due to autoimmunity. Performing a detailed physical exam, particularly in patients with unexplained underlying causes of pancreatitis, is important to aid in the respective diagnosis of SLE-induced pancreatitis. Clinicians being aware that undiagnosed SLE may lead to an acute pancreatitis presentation in the emergency department will help decrease patient mortality, morbidity, as well as hospital burden. In our own patient, a comprehensive and exhaustive serial workup eventually led to the diagnosis of acute pancreatitis secondary to SLE, allowing us to provide the appropriate treatment for a good patient outcome.

CONCLUSION

This case illustrates how a 24-year-old male with no past medical history or comorbid risk factors presented to the emergency department with severe pancreatitis and had an extensive hospital course until he was ultimately diagnosed with systemic lupus erythematosus. The patient's clinical symptoms of pancreatitis, along with respective laboratory values of pancreatitis and end-organ damage improved shortly after treatment with three days of high dose methylprednisolone of 1 mg/kg/day.

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Author Contributions

Shayan Azizi – Design of the work, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Aishvarya Jain – Design of the work, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Jorge O Gutierrez – Design of the work, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Fatih B Kaner – Design of the work, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Rajan Khanna – Conception of the work, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Guarantor of Submission

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Conflict of Interest

Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.