Acute pancreatitis caused by Systemic Lupus Erythematosus activity: A case report and literature review

Eduardo Velasque Marques[a], Luiz Alberto Cerqueira Batista Filho[b, c*], Alexandre Toledo Maciel[d]

[a] Hospital Santa Marcelina de Itaquera, São Paulo, Brazil
[b] Research Department, Imed Group Brasil, São Paulo, Brazil
[c] Internal Medicine, Hospital Santa Marcelina de Cidade Tiradentes, São Paulo, Brazil
[d] Research Department, Imed Group Brasil, São Paulo, Brazil

*** Corresponding author**
luiz.cerqueira@imedgroup.com.br

Citation
Velasque Marques E, Cerqueira Batista Filho LA, Toledo Maciel A. Acute pancreatitis caused by Systemic Lupus Erythematosus activity: A case report and literature review. *Medwave* 2023;23(7):e2684

DOI
10.5867/medwave.2023.07.2684

Submission date
Dec 16, 2022
Acceptance date
Jul 18, 2023
Publication date
Aug 3, 2023

Keywords
Pancreatitis, systemic lupus erythematosus, immunosuppressives, abdominal pain, gastrointestinal manifestation

Postal address
Av. Angélica, 2530 - 9° andar - Bela Vista, São Paulo, Brazil

Abstract

A 32-year-old woman with systemic lupus erythematosus came to the rheumatology outpatient clinic reporting abdominal pain for a week, along with fever, arthralgias, myalgias, alopecia, asthenia and dyspnea on exertion over the last two months. She was hypotensive and tachycardic, requiring admission to the intensive care unit. She was diagnosed with lupus-related acute pancreatitis, an unusual complication occurring in less than 1% of cases. Most cases are mild and self-limited; however, severe and life-threatening events with multiple organ failure are possible. This article is a case report of lupus-related critical acute pancreatitis, and a literature review.

Main messages

- Acute pancreatitis is a rare complication of systemic lupus erythematosus (SLE), with a prevalence between 0.7% and 4%, even though abdominal pain is a relatively common symptom in patients with lupus.
- Most cases are mild and self-limited; however, patients may present with severe disease with multiple organ dysfunctions.
- SLE-related pancreatitis has a higher mortality rate than non-lupus, presumably due to immunosuppression and other comorbidities such as nephritis.T herapy with corticosteroids should be initiated as first-line treatment, improving prognosis.

Introduction

Systemic Lupus Erythematosus (SLE) is a multisystem autoimmune inflammatory disorder characterized by a wide range of single or combined manifestations associated with autoantibodies. To confirm the diagnosis of SLE, the patient must have at least four of the 17 Systemic Lupus International Collaborating Clinics (SLICC) criteria, with at least one clinical and one immunological criterion or a renal biopsy evidencing a typical lupus nephritis pattern associated with positive Antinuclear Factor (ANA) and/or anti-DNAds [1].

Acute pancreatitis in adult SLE was first reported by Reifenstein *et al.* in 1939 as a complex event of unknown etiology and fatal outcome [2,3,4]. It is a recognized, albeit rare, complication and should be part of the differential diagnoses when assessing abdominal pain in patients with SLE [5,6,4,,9].

The gastrointestinal (GI) tract is one of the most commonly affected systems, with GI symptoms ocurring in 50% of patients with SLE [5]. Abdominal pain has a prevalence of 19.2% to 23.5% [5,6]. The annual incidence of SLE-related pancreatitis is estimated to be 0.4 to 1.1 per thousand patients, occurring in 0.5 to 0.9% of patients with SLE, qualifying it as rare [9,12,13]. The prevalence of pancreatitis in patients with SLE varies between 0.7% and 4% [14,15]. However, this prevalence may be underestimated, as subclinical cases with asymptomatic pancreatic enzyme elevation go undiagnosed and unreported. Up to 30.5% of asymptomatic SLE patients are estimated to have hyperamylasemia [4,5,6,16,7].

It is important to be aware of SLE atypical presentations given it is a heterogeneous disease. This work aims to show a rare and severe complication of SLE, which, thanks to early diagnosis and rapid treatment, had an outcome beyond expectations.

Case report

The patient gave her informed consent before this article was written. A 32-year-old woman was admitted to the emergency department of Hospital Santa Marcelina, referred from her first consultation at the rheumatology outpatient clinic. She had a wasting syndrome, with a reported weight loss of approximately 20 kg, in addition to non-thermometered afternoon fever, arthralgia, myalgia, alopecia, asthenia, and dyspnea on exertion over the last two months. During the evaluation, she reported having episodes of dysuria and foamy urine for one month and self-limited diarrheal episodes for one day.

The patient's history included diagnosed epilepsy, moderate distal erosive esophagitis (Los Angeles grade C), hiatal hernia, mild enanthematous pangastritis, and reported being allergic to paracetamol. She made sporadic use of clonazepam, omeprazole 40 mg a day, and sertraline 100 mg a day. Her last menstruation had been two months ago. She denied using oral contraceptives. Regarding family history, the patient reported that her mother has a diagnosis of SLE.

On physical examination, she was in fair general condition, emaciated, pale, dehydrated, with an axillary temperature of 37.8°C. On inspection, she had a malar rash, alopecia, and eyebrow rarefaction; she had borderline hemodynamics with a heart rate of 120 beats per minute, capillary refill time of three seconds and blood pressure of 80/50 mmHg. She also exhibited pain on palpation of the upper abdomen and a palpable liver three cm from the right costal margin.

She had laboratory tests performed two months ago: hemoglobin 9.9 g/dL, hematocrit 29.6%, platelets 143 000/mm³, leukocytes 1560/mm³, 770 of them segmented and 620 lymphocytes, creatinine 0.40 mg/dL, sodium 139 mEq/L, potassium 3.5 mEq/L, C-reactive protein 0. 8 mg/L, amylase 69 U/L, alkaline phosphatase 61 U/L, INR of 1.12, aspartate aminotransferase 31 U/L, alanine aminotransferase 29 U/L, gamma-glutamyl transpeptidase 117 U/L, total bilirubin 0.2 (direct 0.1 / indirect 0.1), urine density 1.005, pH 8.0, 1000 leukocytes and 1000 red blood cells, thyroid-stimulating hormone (TSH) 1.85 mU/L, thyroxine (T4) 0.92 mU/L, and non-reactive Venereal Disease Research Laboratory Test (VDRL).

She was admitted to the Santa Marcelina Hospital due to abdominal pain and hemodynamic instability. Initial laboratory tests showed: hemoglobin 6.7g/dL, hematocrit 20.1%, normocytic and normochromic with mild anisocytosis and poikilocytosis, leukocytes 2930/mm³, 5% rods, 85% neutrophils, 8% lymphocytes and 2% monocytes, platelets 140 000/mm³, INR of 1.31, recombinant tissue plasminogen activator (RTPA) of 1.59, C-reactive protein 22.5 mg/L (normal values up to 5 mg/L), urea 18 mg/dL, creatinine 0.36 mg/dL, total bilirubin

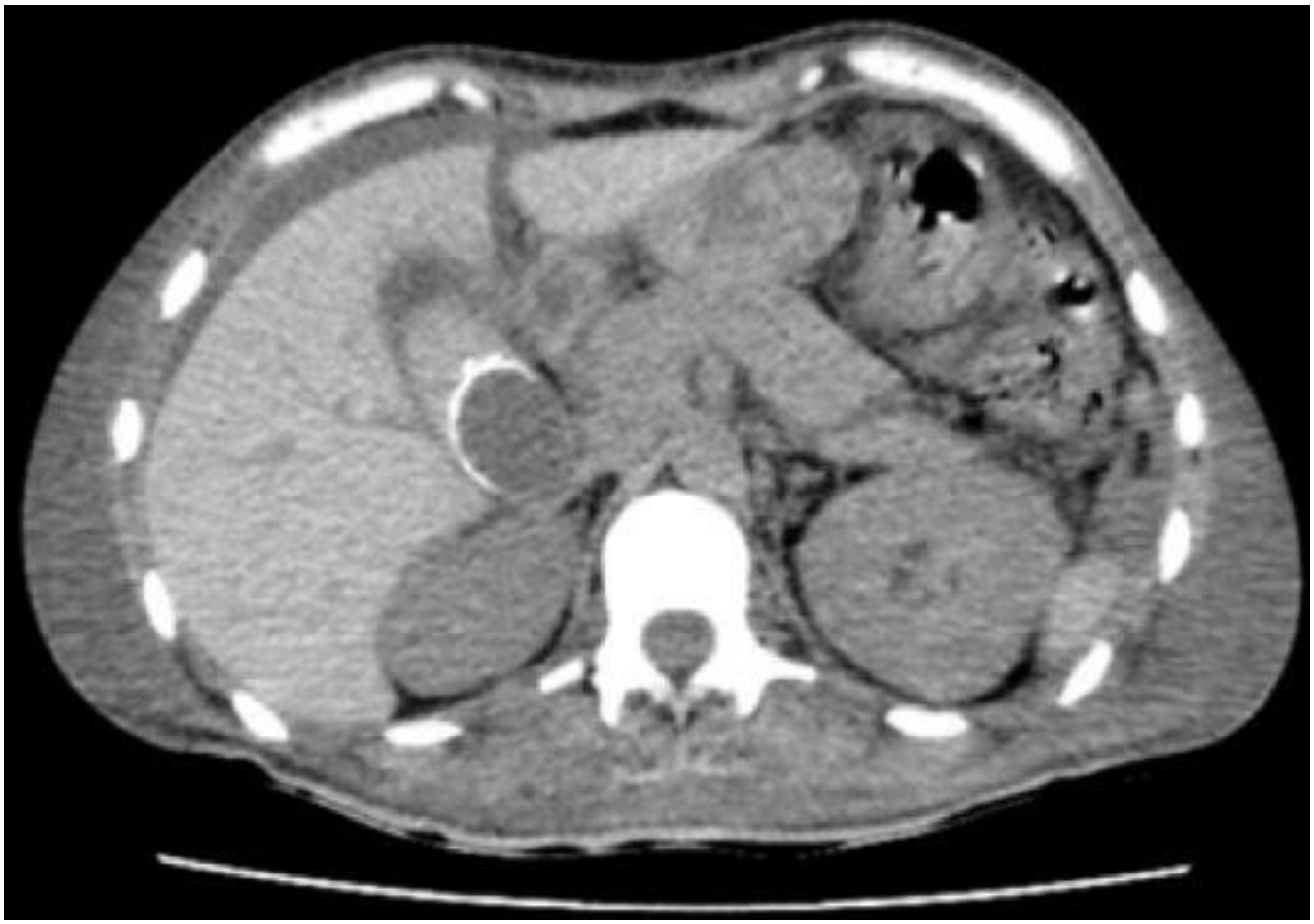
0.41 (direct 0.3 / indirect 0.11), alanine aminotransferase 103 U /L, aspartate aminotransferase 423 U/L. Arterial blood gas with pH 7.42, CO_2 partial pressure 32 mmHg, HCO_3- 20.8 mmol/L, base excess -3.4 mmol/L, O_2 partial pressure 92 mmHg, oxygen saturation 97%, sodium concentration 130 mmol/L, potassium 3 mmol/L, calcium ion concentration 1.02 mmol/L, glucose concentration 82 mg/dL, lactate concentration 1 mmol/L.

Initially, the diagnostic hypotheses of urinary or abdominal sepsis and active SLE were suggested, and an open sepsis protocol was carried out with the laboratory tests already described, cultures, chest X-rays, and early empirical antibiotic therapy. The patient underwent eight days of antibiotic therapy with ceftriaxone and metronidazole, as there was no history of a recent hospitalization or other risk factors for multidrug-resistant germs. Therapy was suspended after negative blood and urine culture results, in addition to laboratory and radiographic results not suggestive of infection. A β-HCG was also requested to investigate amenorrhea, with a result < 0.1, in addition to serology for HIV, HTLV, Hepatitis B, and C. All serology results obtained were non-reactive. She recieved a unit of red blood cells since her hemoglobin level was below 7.0 g/dL.

On the 4th day after admission, a diagnosis of acute lithiasic pancreatitis was suggested due to an amylase of 204 U/L and lipase of 238 U/L. Fasting and treatment with hydrocortisone were carried out due to the possibility of active lupus.

To evaluate her diffuse abdominal pain on palpation and hepatomegaly, computed tomography (CT) of the abdomen and pelvis and ultrasonography (US) of the abdomen was performed. The CT scan showed the presence of a single gallbladder stone and pancreas edema (Figure 1). Ultrasonography, performed on the 4th day after admission, showed no changes in the pancreas, liver, intrahepatic and extrahepatic bile ducts, a gallbladder with thin and smooth walls, and a single 25 mm stone. Despite the presence of cholelithiasis with a single large stone, there were no anatomical alterations of the bile duct or the Wirsung duct, making it unlikely that this finding was the cause of pancreatitis, as such findings suggest a chronic calculus cholecystopathy.

Figure 1. Contrasted CT scan of the abdomen showing single gallbladder stone, as well as pancreatic edema without dilation of the Wirsung duct.



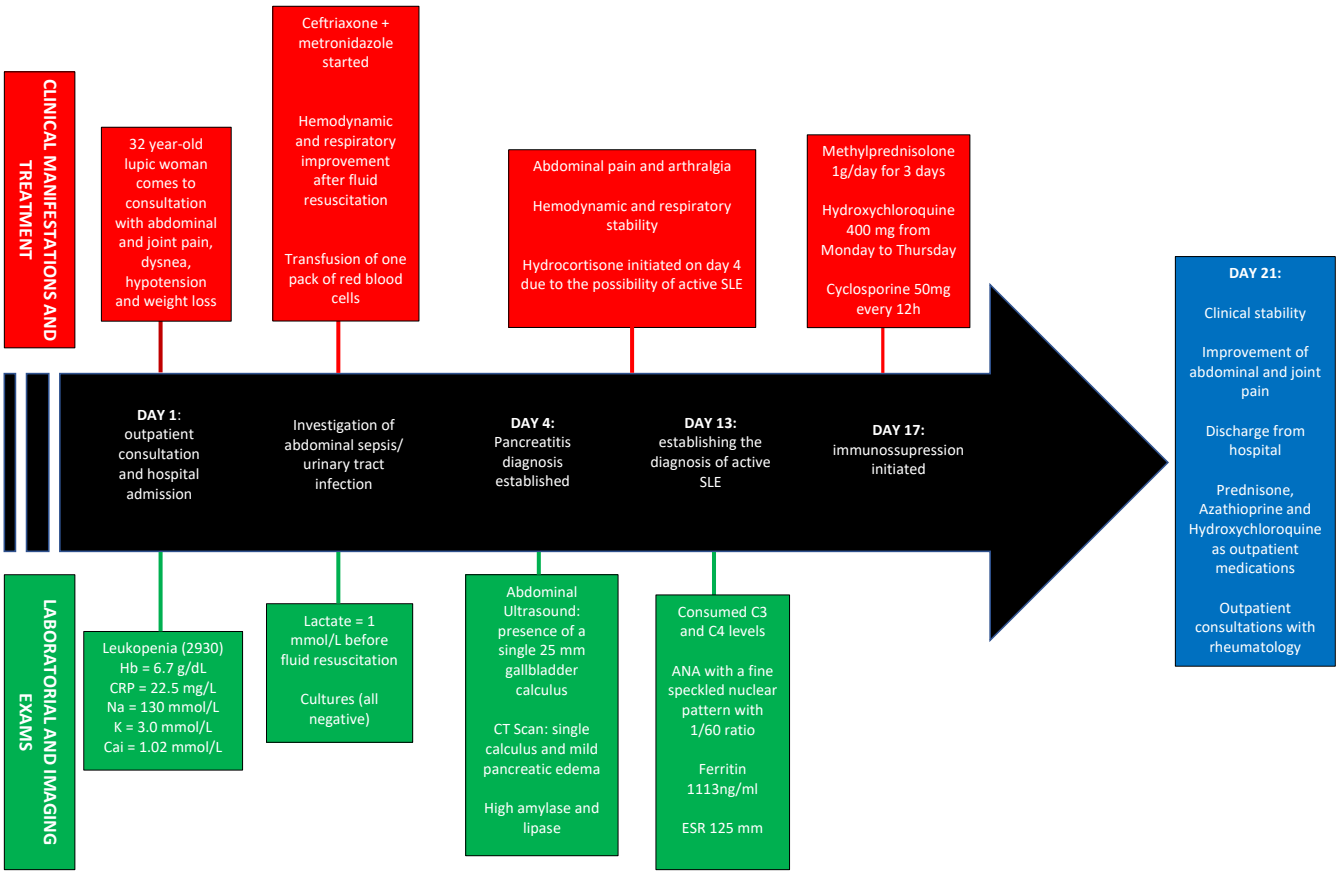
Source: Santa Marcelina Hospital (2021).

Thirteen days after admission, the patient was diagnosed with active SLE due to clinical examination (alopecia and arthralgia/arthritis) and suggestive laboratory results: lymphopenia with thrombocytopenia, presence of ANA with fine speckled nuclear pattern in a ratio of 1/160, evidence of consumed complement (C3 of 18 mg/dL and C4 of 2 mg/dL) presence of 24-hour proteinuria higher than 500 mg and erythrocyte sedimentation rate of 125 mm. Other requested tests were anti-DNA and Rheumatoid Factor, both non-reactive. For the anemia, the following tests were performed with their respective results: reticulocyte count 4.4%, ferremia 44 ug/dL, ferritin 1113 ng/mL, transferrin saturation 38%, total iron binding capacity (TIBC) 116 ug/dL, vitamin B_{12} 1533 pg/mL, folic acid 2.4 ng/mL, protein electrophoresis without alterations, negative direct and indirect Coombs. Transthoracic echocardiogram with aorta diameter of 25 mm, left atrium diameter of 29 mm, interventricular septum thickness of 8 mm, posterior wall thickness 8 mm, and left ventricular ejection fraction of 65%.

Due to inconclusive results in investigations for isolated diagnoses of hematological alterations and acute pancreatitis, the patient's entire clinical condition was attributed active SLE.

On the 17th day after admission, pulse therapy was started to control active lupus disease with methylprednisolone one g/day for three days. On the 19th day of hospitalization, pulse therapy was completed, and maintenance treatment with hydroxychloroquine 400 mg from Monday to Thursday was started. On the 20th day, one mg/kg/day of prednisone was reintroduced, along with cyclosporine 50 mg every 12h; hydroxychloroquine 400 mg was maintained Monday through Thursday. On the 21st day, the patient was discharged early due to significant clinical improvement, with prescribed azathioprine 50 mg every 12 hours and hydroxychloroquine 400 mg on alternate days, in addition to prednisone one mg/kg/day and scheduled outpatient follow-up in 13 days (Figure 2). Considering a second approach to cholelithiasis, she was also referred for outpatient follow-up with the general surgery team to schedule an elective cholecystectomy.

Figure 2. Timeline of the case report.



SLE (systemic lupus erythematosus), Hb (hemoglobin), CRP (C-reactive protein), Na (sodium), K (potassium), Cai (ionized calcium), ESR (erythrocyte sedimentation rate), CT (computed tomography).
Source: Prepared by the authors based on data from the study.

During an outpatient consultation 55 days after discharge, the patient reported improvement in joint pain and persistence of epigastric pain after eating fatty foods with associated diarrhea in the last week (four to five bowel movements per day, with no signs of invasiveness). She also mentioned amenorrhea since her last hospitalization (four months ago) and fatigue on exertion, which does not prevent her from performing basic daily activities. She was still taking sertraline, prednisone 40 mg (1mg/kg), omeprazole, complex B vitamins, folic acid, thiamine 300 mg daily, and vitamins A and D. On physical examination, she was in fair general condition, pale +/4+, thinner 3+/4+, blood pressure 100/60 mmHg, weight 34 Kg (from 30. 4 kg at her last visit), heart rate 105 bpm, excavated and mildly tense abdomen, painful on palpation of the right hypochondrium and epigastrium, murphy's sign present. She underwent tests one month after discharge with the following results: urea 25 mg/dL, creatinine 0.34 mg/dL, sodium 139 mEq/L, potassium 3.6 mEq/L, hemoglobin 8.6 g/dL, normocytic and normochromic, leukocytes 8870/mm³ being 1064 lymphocytes, platelets 705 000/mm³. A urine test revealed 16 000 leukocytes and 32 000 erythrocytes, 24-hour proteinuria of 300 mg, and creatine phosphokinase of 15U/L. She continued to receive prednisone 40mg/day, azathioprine 100mg/day, hydroxychloroquine on alternate days, thiamine, complex B vitamins, folic acid, and vitamin D. Influenza vaccination was requested for her next appointment, and a new appointment was scheduled in three months with new control tests.

In an outpatient consultation 132 days after discharge, the patient reported improvement in her abdominal pain and better food tolerance, and dyspnea improvement, which was still present with moderate exertion. The current regimen for SLE was hydroxychloroquine 200 mg/day, prednisone 20 mg/day (dose changed two months before), and azathioprine 100 mg/day. She was still taking sertraline 100 mg/day; omeprazole; B complex vitamins; folic acid; thiamine 300 mg; vitamins A and D. On physical examination: fair general condition, discolored +/4+, weight 46.5 Kg (from 30. 4 kg at her last visit), excavated abdomen, somewhat tense, painful on palpation of the right hypochondrium and epigastrium, and livedo reticularis in the upper limbs. Laboratory tests carried out 121 days after discharge showed: glucose 81 mg/dl, urea 19 mg/dL, creatinine 0.63 mg/dL, sodium 140 mEq/L, potassium 3.9 mEq/L, albumin 3.7 g/dL, total bilirubin 0.3 mg/dL, alkaline phosphatase 111 U/L, alanine aminotransferase 23 U/L, aspartate aminotransferase 28 U/L, gamma-glutamyl transpeptidase 79 U/L, lactic dehydrogenase 171 U/L, C3 54 mg/dL, C4 2 mg/dL, C-reactive protein 33.2 mg/L, hemoglobin 10.7 g/dL, 4560 leukocytes, 375 000 platelets, creatine phosphokinase 35 U/L. Urine tests: proteinuria 1+ and hemoglobinuria 2+. The patient, despite having been using prednisone 20 mg/day for two months, she should have been using 40 mg/day, so we prescribed prednisone 30 mg/day for 30 days with a later reduction to 20mg/day, maintaining azathioprine and hydroxychloroquine at the current dose, laboratory tests were requested, and a return visit was scheduled in three months.

Discussion

Definitive conclusions cannot be drawn from an individual case. However, it is valid to point out similarities and differences between what is presented here and what is reported in the literature. In addition, the scientific community is more likely to report unusual and severe cases with fatal outcomes than cases with complete recovery. Pancreatitis is infrequent among SLE patients, affecting only eight SLE patients on a 10-year basis resulting in only 25 hospital admissions of SLE patients with pancreatitis among 2947 admissions of SLE patients (0.85%) over 20 years [8]. Until 2010, only approximately 160 cases of SLE-related pancreatitis were reported in adult and pediatric patients [9–13]. The frequency of SLE-related pancreatitis may be underestimated in some studies due to undiagnosed cases, as patients presenting milder symptoms, such as mild abdominal pain, are often treated as dyspeptic syndrome. Furthermore, pancreatitis may resolve spontaneously or with corticosteroid therapy prescribed for active SLE [8,14]. SLE-related pancreatitis has a higher mortality rate than non-lupus, presumably due to immunosuppression and other comorbidities such as lupus nephritis [15,16]. Among patients with SLE, 84% present with disease activity in the context of acute pancreatitis [16]. trauma, toxic-metabolic disorders (such as alcohol abuse, hypertriglyceridemia, and hypercalcemia), structural abnormalities of the pancreas, and drug-induced pancreatitis should be ruled out [8,17,18].

In cases of SLE-related pancreatitis, several pathogenic mechanisms have been postulated: Organ ischemia secondary to hypovolemia or severe hypotension, atherosclerotic disease, vasculitis, pancreatic artery micro-thrombosis related to antiphospholipid antibody syndrome, intimal thickening/proliferation, presence of anti-pancreatic antibodies, organ inflammation due to T-cell infiltration, immunocomplex deposit, and complement activation, as well as viral infection due to the patient's chronic immunosuppressed state [8–10,16–21]. In an American cohort study of 1811 patients, in which 63 patients (3.5%) had SLE-related pancreatitis, an association was observed between hypertriglyceridemia greater than 200 mg/dL and the development of the condition [22].

Gastrointestinal manifestations of SLE include nausea, vomiting, anorexia, and abdominal pain, and the most frequent causes of acute abdomen in patients with SLE are mesenteric vasculitis, hepatobiliary disease, pancreatitis, and gastroenteritis [23]. The clinical manifestations of pancreatitis in SLE and non-SLE patients are indistinguishable, with 88% presenting with abdominal pain [16]. Other clinical features in the described acute pancreatitis cases associated with increased lupus activity were fever and mucocutaneous involvement [14,24,25].

Every SLE patient with an acute abdomen should be considered for immediate surgical intervention, like any other patient. Once the need for urgent surgical intervention has been ruled out, an investigation should proceed, looking for the same etiologies that affect non-lupus patients, as mentioned above. Only when all the most common etiologies are excluded, and

no cause is identified can the diagnosis of lupus pancreatitis be established and treatment initiated [8,10,15,16,19]. In the case presented, pancreatitis was diagnosed by excluding the most common causes in non-lupus patients, investigating toxic-metabolic and hepatobiliary etiologies. The absence of dilatation of the biliary tree, observed on tomography, in addition to the absence of characteristic findings on ultrasonography and normal liver function, led to a thorough investigation. Importantly, tomographic findings have a diagnostic accuracy of 70% to 90% in cases of acute pancreatitis, regardless of severity, and generally correlate with the severity of the disease [10].

After admission, supportive treatment and antibiotic therapy was started, believing it to be an infectious process that led to abdominal sepsis. After negative cultures and no improvement in the clinical picture, it was necessary to look for differential diagnoses. The clinical picture led to laboratory and imaging tests. Tests were performed for the diagnosis of SLE, for which the patient was already being monitored in the outpatient clinic, and for the diagnosis of the disease affecting her during hospitalization.

As treatment, pulse therapy with methylprednisolone was performed, with subsequent use of azathioprine to control the disease, a treatment that proved effective during the patient's follow-up period, which corroborates the hypothesis that these drugs are not responsible for the pathology. From the beginning of pulse therapy during hospitalization, the patient showed significant improvement in a very short period of time, making it unnecessary to prolong hospitalization. The patient evolved with weight gain without further complications after a long outpatient follow-up.

In patients who do not respond to treatment with high doses of corticosteroids, alternative treatments with high doses of immunoglobulins, cytotoxic agents, and/or plasmapheresis can be performed [17–19].

Conclusions

In suspected cases of acute pancreatitis, although SLE is a rare etiology, it should be considered in any patient with suspected SLE and abdominal pain because of the potentially fatal outcome (18-27% mortality) whenever symptoms are associated with elevated serum amylase/lipase levels, imaging tests, and autoantibody tests [26,27]. Attention should also be paid to the possibility that pancreatitis may be the initial manifestation of SLE, as it is frequently associated with increased lupus disease activity [8,19,28].

Once the most frequent etiologies, such as obstructive mechanisms or metabolic toxicity, have been excluded, pulse therapy with corticosteroids should be initiated as first-line treatment. Despite suspicions of its involvement in the disease, this therapy has shown much clinical evidence of improving prognosis [17]. In this case, the mechanism probably involved in the pathogenesis was the lupus disease itself, with no evidence of coagulation alterations or hypertriglyceridemia.

In summary, mortality in patients with SLE-related pancreatitis can be reduced by early diagnosis and corticosteroid therapy, in addition to supportive measures.

Notes

Contributor roles

EV: contributed to the conception and design of the work, in addition to the acquisition, analysis, and interpretation of the data. LACBF: critically reviewed the intellectual content. ATM: approved the final version for publication.

Competing interests

The authors declare no conflicts of interest.

Funding

The authors did not receive any financial support.

Ethics

The patient authorized the presentation of her case by signing the Informed Consent Form. This work complied with the guidelines provided in Resolution 466/2012 of the Brazilian National Health Council.

Provenance and peer review

Not commissioned. Externally peer-reviewed by three reviewers, double-blind.

Language of submission

English.

References

1. Petri M, Orbai A-M, Alarcón GS, Gordon C, Merrill JT, Fortin PR, et al. Derivation and validation of the Systemic Lupus International Collaborating Clinics classification criteria for systemic lupus erythematosus. Arthritis Rheum. 2012;64: 2677–86. https://doi.org/10.1002/art.34473
2. Reifenstein EC, Reifenstein GH. A variable symptom complex of undetermined etiology with fatal termination: including conditions described as visceral eythema group (Osler), disseminated lupus erythematosus, atypical verrucous endocardidis (Libman-Sacks), fever of unknown origin (Christian) and diffuse peripheral vascular disease (Baehr and others). Arch Intern Med. 1939;63: 552–74. https://doi.org/10.1001/archinte.1939.00180200122010
3. Jaspersen D. Gastrointestinale Manifestationen des systemischen Lupus erythematodes. Symptomatik, Diagnostik und Differentialdiagnostik. Fortschr Med. 1992;110: 167–169.
4. Izzedine H, Caramella C, Ratziu V, Deray G. Chronic calcifying pancreatitis and systemic lupus erythematous. Pancreas. 2005;31: 289–90. https://doi.org/10.1097/01.mpa.0000175162.34704.6b
5. García-Carrasco M, Mendoza-Pinto C, Autrán-Limón MA, Herrera Robles E, Méndez Martínez S, Etchegaray Morales I,

et al. Prevalence of functional gastrointestinal disorders in adults with systemic lupus erythematosus. Lupus. 2018;27: 788–793. https://doi.org/10.1177/0961203317747718
6. Fawzy M, Edrees A, Okasha H, El Ashmaui A, Ragab G. Gastrointestinal manifestations in systemic lupus erythematosus. Lupus. 2016;25: 1456–1462. https://doi.org/10.1177/0961203316642308
7. Singh R, Saunders B, Scopelitis E. Pancreatitis leading to thrombotic thrombocytopenic purpura in systemic lupus erythematosus: a case report and review of literature. Lupus. 2003;12: 136–9. https://doi.org/10.1191/0961203303lu258cr
8. Derk CT, DeHoratius RJ. Systemic lupus erythematosus and acute pancreatitis: a case series. Clin Rheumatol. 2004;23: 147–51. https://doi.org/10.1007/s10067-003-0793-3
9. Wang F, Wang N-S, Zhao B-H, Tang L-Q. Acute pancreatitis as an initial symptom of systemic lupus erythematosus: a case report and review of the literature. World J Gastroenterol. 2005;11: 4766–8. https://doi.org/10.3748/wjg.v11.i30.4766
10. Duncan HV, Achara G. A rare initial manifestation of systemic lupus erythematosus--acute pancreatitis: case report and review of the literature. J Am Board Fam Pract. 2003;16: 334–8. https://doi.org/10.3122/jabfm.16.4.334
11. Tian XP, Zhang X. Gastrointestinal involvement in systemic lupus erythematosus: insight into pathogenesis, diagnosis and treatment. World J Gastroenterol. 2010;16: 2971–7. https://doi.org/10.3748/wjg.v16.i24.2971
12. Penalva JC, Martínez J, Pascual E, Palanca VM, Lluis F, Peiró F, et al. Chronic pancreatitis associated with systemic lupus erythematosus in a young girl. Pancreas. 2003;27: 275–7. https://doi.org/10.1097/00006676-200310000-00016
13. Swol-Ben J, Bruns CJ, Müller-Ladner U, Hofstädter F, Link J, Hechenrieder C, et al. Leukoencephalopathy and chronic pancreatitis as concomitant manifestations of systemic lupus erythematosus related to anticardiolipin antibodies. Rheumatol Int. 2004;24: 177–81. https://doi.org/10.1007/s00296-003-0366-4
14. Limwattana S, Dissaneewate P, Kritsaneepaiboon S, Dendumrongsup T, Vachvanichsanong P. Systemic lupus erythematosus-related pancreatitis in children. Clin Rheumatol. 2013;32: 913–8. https://doi.org/10.1007/s10067-013-2242-2
15. Pascual-Ramos V, Duarte-Rojo A, Villa AR, Hernández-Cruz B, Alarcón-Segovia D, Alcocer-Varela J, et al. Systemic lupus erythematosus as a cause and prognostic factor of acute pancreatitis. J Rheumatol. 2004;31: 707–12.
16. Breuer GS, Baer A, Dahan D, Nesher G. Lupus-associated pancreatitis. Autoimmun Rev. 2006;5: 314–8. https://doi.org/10.1016/j.autrev.2005.11.004
17. Essaadouni L, Samar E, Krati K. Pancreatitis as initial manifestation of systemic lupus erythematosus. Lupus. 2010;19: 884–7. https://doi.org/10.1177/0961203309356456
18. Ergas D, Toledo S, Sthoeger D, Sthoeger ZM. Chronic relapsing lupus pancreatitis. Isr Med Assoc J. 2007;9: 44–5.
19. Nesher G, Breuer GS, Temprano K, Moore TL, Dahan D, Baer A, et al. Lupus-associated pancreatitis. Semin Arthritis Rheum. 2006;35: 260–7. https://doi.org/10.1016/j.semarthrit.2005.08.003
20. Lariño Noia J, Macías García F, Seijo Ríos S, Iglesias García J, Domínguez Muñoz JE. Pancreatitis and systemic lupus erythematosus. Rev Esp Enferm Dig. 2009;101: 571–9. https://doi.org/10.4321/s1130-01082009000800009
21. Kobayashi S, Yoshida M, Kitahara T, Abe Y, Tsuchida A, Nojima Y. Autoimmune pancreatitis as the initial presentation of systemic lupus erythematosus. Lupus. 2007;16: 133–6. https://doi.org/10.1177/0961203306073137
22. Makol A, Petri M. Pancreatitis in systemic lupus erythematosus: frequency and associated factors - a review of the Hopkins Lupus Cohort. J Rheumatol. 2010;37: 341–5. https://doi.org/10.3899/jrheum.090829
23. Vergara-Fernandez O, Zeron-Medina J, Mendez-Probst C, Salgado-Nesme N, Borja-Cacho D, Sanchez-Guerrero J, et al. Acute abdominal pain in patients with systemic lupus erythematosus. J Gastrointest Surg. 2009;13: 1351–7. https://doi.org/10.1007/s11605-009-0897-4
24. Duval A, Lamare L, Jian R, Pouchot J. Hépatopancréatite inaugurale d'un lupus érythémateux systémique. Gastroentérologie Clinique et Biologique. 2008;32: 417–420. https://doi.org/10.1016/j.gcb.2008.01.026
25. Carducci M, Calcaterra R, Mussi A, Franco G, Morrone A. Acute pancreatitis as initial manifestation of systemic lupus erythematosus and subacute cutaneous lupus erythematosus: report of two cases. Lupus. 2008;17: 695–7. https://doi.org/10.1177/0961203308090429
26. Richer O, Ulinski T, Lemelle I, Ranchin B, Loirat C, Piette JC, et al. Abdominal manifestations in childhood-onset systemic lupus erythematosus. Ann Rheum Dis. 2007;66: 174–8. https://doi.org/10.1136/ard.2005.050070
27. Perrin L, Giurgea I, Baudet-Bonneville V, Deschênes G, Bensman A, Ulinski T. Acute pancreatitis in paediatric systemic lupus erythematosus. Acta Paediatr. 2006;95: 121–4. https://doi.org/10.1080/08035250500325090
28. Campos CF, Scrignoli JA, de Almeida LP, Ferreira BL, Ribeir SL, de Lima DS, et al. Pancreatite aguda e rutura espontânea de pseudocisto pancreático em paciente com lúpus eritematoso sistêmico. Acta Reumatol Port. 2010;35: 236–240.

Pancreatitis aguda por actividad del lupus eritematoso sistémico: Reporte de caso y revisión de la literatura

Resumen

Mujer de 32 años con lupus eritematoso sistémico acude a consulta externa de reumatología por dolor abdominal de una semana de evolución, además de fiebre, artralgias, mialgias, alopecia, astenia y disnea de esfuerzo de 2 meses de evolución. También presentó hipotensión y taquicardia, por lo que requirió ingreso en la unidad de cuidados intensivos. Le diagnosticaron pancreatitis aguda relacionada con el lupus, que es una complicación inusual que ocurre en menos del 1% de los pacientes. La mayoría de los casos son leves y autolimitados, sin embargo, es posible que se presenten eventos graves y potencialmente mortales, con disfunción multiorgánica. Este artículo es un reporte de caso de una pancreatitis aguda crítica relacionada con lupus y una revisión de la literatura.