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Case report

Autoimmune duodenitis and protein-losing enteropathy: An uncommon presentation of systemic lupus erythematosus

Andrea Isabel Ancasi-Medina,[1*] Helena C. Calle-Herrera,[1] Cristhian A. Vizcarra-Vizcarra,[2] Lily E. Montesinos-Valencia,[3] Zaima Luque-Quilla,[4] Yolanda A. Llerena-Concha.[5]

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[1] Medical Student, School of Medicine, Universidad Católica de Santa María, Arequipa, Peru.
[2] Nephrologist, School of Medicine, Universidad Católica de Santa María, Arequipa, Peru.
[3] Nephrologist, School of Medicine, Universidad Católica de Santa María, Hospital Nacional Carlos Alberto Seguín Escobedo, Arequipa, Peru.
[4] Pathologist, Hospital Regional Honorio Delgado, Arequipa, Peru.
[5] Pathologist, School of Medicine, Universidad Católica de Santa María, Hospital Regional Honorio Delgado, Arequipa, Peru.

***Correspondence:** Andrea Isabel Ancasi-Medina.
andrea.ancasi@estudiante.ucsm.edu.pe

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Abstract

Introduction: Systemic lupus erythematosus (SLE) is an autoimmune disease that occurs more frequently in young women and affects various organs. However, gastrointestinal involvement is uncommon and may present as autoimmune duodenitis or protein-losing enteropathy, and their coexistence as the first manifestation of the disease is even more unusual. **Case presentation:** We present the case of a 22-year-old male patient with polyserositis, abdominal pain, diarrhea, generalized edema, hypoalbuminemia, and microcytic anemia. Endoscopy showed erosive duodenitis, and histology revealed nonspecific chronic inflammation. Immunological studies were positive for antinuclear antibodies (1:180) and other autoimmune markers, confirming the diagnosis of SLE with atypical gastrointestinal manifestations in the form of protein-losing enteropathy and autoimmune duodenitis. The patient received methylprednisolone treatment and had a favorable clinical course. **Discussion:** SLE has atypical gastrointestinal manifestations. Protein-losing enteropathy has a low prevalence, and its diagnosis is based on clinical findings and biopsy results. Duodenitis is more nonspecific, but finding both manifestations together in addition to an SLE diagnosis is rare. **Conclusion:** SLE may initially present with unusual gastrointestinal manifestations such as autoimmune duodenitis and protein-losing enteropathy in patients with generalized edema, severe hypoalbuminemia without proteinuria, and persistent anemia.

Keywords

Systemic lupus erythematosus, duodenitis, protein-losing enteropathies.

INTRODUCTION

Systemic lupus erythematosus (SLE) is an autoimmune disease that occurs more frequently in women of childbearing age and is rare in men. It affects multiple organs, including the kidneys, joints, and skin[1,2]. However, uncommon manifestations such as gastrointestinal involvement may also occur. Gastrointestinal manifestations are present in 40%–60% of patients during the course of the disease[3] and in 18% of patients at disease onset[4]. The most common manifestations are autoimmune hepatitis and lupus enteritis[4]. Autoimmune duodenitis and protein-losing enteropathy have also been reported[5].

SLE-associated autoimmune duodenitis presents with malabsorption symptoms and an increased number of intraepithelial lymphocytes[6]. In contrast, protein-losing enteropathy (PLE) is characterized by edema and hypoalbuminemia in the absence of nephrotic-range proteinuria, secondary to protein loss through the gastrointestinal tract[7,8].

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We report the case of a man with an unusual presentation of SLE characterized by autoimmune duodenitis and protein-losing enteropathy, given the uncommon presentation and low frequency of this association. The patient provided informed consent, and the CARE guidelines for clinical case reports were followed.

CASE PRESENTATION

A 22-year-old man with a history of pulmonary tuberculosis at 15 years of age and multiple hospitalizations (2022, 2024, and 2025) for abdominal pain, diarrhea, and iron-deficiency anemia of undetermined etiology presented with the current illness. He developed asthenia, nausea, vomiting, bloody stools, and progressive abdominal and lower-extremity swelling that extended to the thighs. He also experienced a 7-kg weight loss over the preceding three months. Three days before admission, he developed dyspnea on moderate exertion and therefore presented to the emergency department.

Physical examination revealed a blood pressure of 90/55 mm Hg, a heart rate of 104 beats per minute (bpm), and an oxygen saturation of 95%. The skin and conjunctivae were pale. Cardiac examination revealed a regular rhythm with normal heart sounds and no murmurs. Vesicular breath sounds were present bilaterally. The abdomen was globular and mildly distended, with limited depressibility and no tenderness on palpation. Shifting dullness was present. There was ++/+++ edema of the lower extremities.

Laboratory studies revealed severe microcytic hypochromic anemia, severe hypoalbuminemia, and non-significant proteinuria. Thoracentesis showed a pleural fluid-to-serum protein ratio of 0.4 and a pleural fluid-to-serum lactate dehydrogenase (LDH) ratio of 0.31, consistent with a transudate. Sputum testing for Koch's bacilli was negative. Abdominal ultrasonography showed kidneys of normal size and hepatomegaly.

Protein-losing enteropathy was suspected. Therefore, upper gastrointestinal endoscopy was performed and showed erosive duodenitis, with an autoimmune rather than infectious etiology considered likely. Histopathology revealed nonspecific chronic duodenitis. Colonoscopy showed erosive colitis, with an infectious rather than inflammatory etiology considered likely. Histopathology revealed nonspecific chronic ileitis and colitis, with negative findings for granulomas and inflammatory bowel disease. Autoimmune testing was subsequently performed and yielded positive results. The patient's laboratory findings are presented in **Table 1**. Because of hospital limitations, additional studies, such as abdominal computed tomography or magnetic resonance enterography, could not be performed.

During hospitalization, the patient developed hemoptysis and respiratory failure (06/18/25). Given the positive autoimmune markers, polyserositis, and duodenitis, SLE was suspected. Methylprednisolone pulse therapy was initiated on 06/20/25, with a favorable clinical course, followed by prednisone at 50 mg/day. Subsequently, the patient showed improvement, with increases in hemoglobin and albumin levels after immunosuppressive treatment (**Table 1**). Unfortunately, the patient died of superimposed pneumonia in November 2025.

DISCUSSION

SLE is an autoimmune disease that occurs predominantly in women. Its most important clinical manifestations include skin rash, alopecia, serositis, arthritis, pleuritis, and lupus nephritis[9]. However, its clinical presentation can be heterogeneous, which may make diagnosis challenging. Uncommon manifestations of SLE include lupus hepatitis, interstitial lung disease, lupus retinopathy, aseptic meningitis, lupus enteritis, protein-losing enteropathy (PLE), and duodenitis[6,10]. We present the case of a 22-year-old man with a positive ANA titer of 1:180 and gastrointestinal manifestations, including autoimmune duodenitis and protein-losing enteropathy.

Gastrointestinal manifestations of SLE are uncommon and may represent the initial presentation of the disease. Associated PLE has a low prevalence (1.9%–19%)[7,11] and may constitute the main clinical manifestation[12]. Therefore, early recognition is based on nonspecific findings such as generalized edema, ascites, and severe hypoalbuminemia without nephrotic-range proteinuria, as observed in this patient. In addition, persistent iron-deficiency anemia also supported the suspicion of PLE. Upper gastrointestinal endoscopy showed erosive duodenitis, and biopsy specimens revealed nonspecific chronic inflammation (**Figure 1**), findings that have also been described in other patients with autoimmune gastrointestinal involvement[12,13]. This may result from mucosal ulceration, non-necrotizing vasculitis of mesenteric or intestinal vessels, intestinal lymphangiectasia, and increased capillary permeability. These processes lead to intravascular cellular activation, complement activation, and production of proinflammatory cytokines[8].

Some specific tests, such as fecal alpha-1-antitrypsin measurement or radionuclide imaging with radiolabeled albumin[12], may aid in the diagnosis. In the present report, the diagnosis was established by excluding other causes based on the clinical presentation and favorable response to immunosuppressive therapy.

Duodenitis, in turn, may result from various conditions, including infections, nonsteroidal anti-inflammatory drugs,

Table 1. Patient's laboratory findings

Test	Reference range	14/05/25	19/06/25	23/06/25	19/07/25	27/08/25	02/09/25	11/09/25	06/10/25
Hemoglobin (g/dL)	13.5-16.0	7.9	7.6	9.2	9.3	9.5	9.7	-	10.7
White blood cells (/µL)	4000-10.000	9200	7840	9680	9880	12.050	7900	7900	7900
Neutrophils (/µL)	2000-7500	4876		9099	7904	6868	6241	-	6873
Lymphocytes (/µL)	1000-4800	3772		387	1679	3494	1027	-	790
Platelets (/µL)	150.000-450.000	498.000	417.000	593.000	361.000	622.000	483.000	453.000	213.000
Mean corpuscular volume (fL)	80.0-100.0	-	-	-	72.2	-	-	-	79.9
Mean corpuscular hemoglobin (pg)	27.0-33.0	-	-	-	21.0	-	-	-	24.4
Mean corpuscular hemoglobin concentration (g/dL)	32.0-36.0	-	-	-	29.1	-	-	-	30.5
Vitamin B12 (pg/mL)	200-900	191.6	-	-		-	-	-	-
Iron (µg/dL)	50-170	19	26	-	27	-	-	-	54
Folic acid (ng/mL)	3-17	7.1	14.97			-	-	-	
Glucose (mg/dL)	70-110	83	79	84	69	132	57	57	74
Creatinine (mg/dL)	0.4-1.5	0.84	0.56	0.63	0.55	0.90	0.69	0.61	0.74
Urea (mg/%)	15.00-40.00	47	36	47	45	33	51	51	26
Aspartate aminotransferase (U/L)	11-39	25	33	38	35	18	-	-	29
Alanine aminotransferase (U/L)	0-45	12	14	26	38	18	-	-	10
Lactate dehydrogenase (U/L)	200-480	288	271	270	407	323	-	-	-
Total bilirubin (mg/dL)	0.2-1.2	0.85	0.52	0.57	0.83	0.52	-	-	0.30
Direct bilirubin (mg/dL)	0.0-0.4	0.38	0.16	0.26	0.38	0.17	-	-	0.13
Indirect bilirubin (mg/dL)	0.2-0.9	0.47	0.36	0.31	0.45	0.35	-	-	0.17
Total protein (g/dL)	6.00-8.00	2.7	3.0	3.9	5.1	3.8	3.9	-	4.1
Albumin (g/dL)	3.5-5.5	1.9	2.1	3.0	3.7	2.5	2.6	-	2.7
Globulin (g/dL)	2.3-3.5	0.80	0.9	0.90	1.40	1.30	1.30	-	1.40
Cholesterol (mg/dL)	0-200	68	69	128	48	-	-	-	89
Triglycerides (mg/dL)	0-160	109	-	106	113	-	-	-	130
Uric acid (mg/dL)	2.5-7.0	3.9	3.0	-	5.3	-	-	-	2.0
Antinuclear antibodies	Negative (<1:80)	1:80	-	-	-	-	-	-	-
C3	75.0-135.0 mg/dL	69.9	-	-	-	-	-	-	-
C4	9.0-35.0 mg/dL	32.1	-	-	-	-	-	-	-
Antineutrophil cytoplasmic antibodies	Negative	Negative	-	-	-	-	-	-	-
Anti-SS-A 52 kDa (Ro52) antibodies	0–20 U/mL	23.0	-	-	-	-	-	-	-
Antihistone antibodies	Negative (<1.0 U/mL)	-	-	-	-	-	-	-	-
24-hour urinary protein (mg/24 h)	24.0-141.0	36.25	156.80	-	243.20	-	-	338.30	-

Table prepared by the authors.

alcohol, chronic bile reflux, and lupus, depending on the histologic pattern[14]. Duodenal biopsy in this patient showed mild, nonspecific chronic duodenitis with mild mucosal atrophy (**Figure 1A** and **1B**), a finding compatible with autoimmune duodenitis. This is a rare condition characterized by immune-mediated atrophy of the duodenal mucosa and often also involving the esophagus, stomach, and colon[14]. Anti-enterocyte antibodies are an immunologic marker of autoimmune duodenitis and are present in 85% of patients. Their presence therefore contributes to the diagnosis, although they may also be detected less frequently in celiac disease (7.7%)[15,16]. In this case, antibody testing was not performed because of the patient's financial limitations. Mild, nonspecific chronic ileitis was also identified. No features suggestive of intestinal tuberculosis, Crohn's disease, or celiac disease were found (**Figure 1C** and **D**).

Other differential diagnoses that may also present with villous atrophy on histopathologic examination include celiac disease and Crohn's disease. Celiac disease is an autoimmune disorder that affects the small intestine, and gastrointestinal symptoms occur after gluten ingestion. Histopathologically, it is characterized by intraepithelial lymphocytic infiltration, with more than 40 lymphocytes per 100 epithelial cells)[15]. This finding was not reported in the present case. Crohn's disease, in contrast, is a chronic inflammatory bowel disease that presents with abdominal pain, diarrhea, and hematochezia. It is characterized by patchy lesions throughout the gastrointestinal tract, predominantly involving the ileocolon and small intestine, as well as transmural inflammation. The latter may be confirmed by magnetic resonance enterograph[17]. Biopsy showed no granulomas or findings compatible with Crohn's disease.

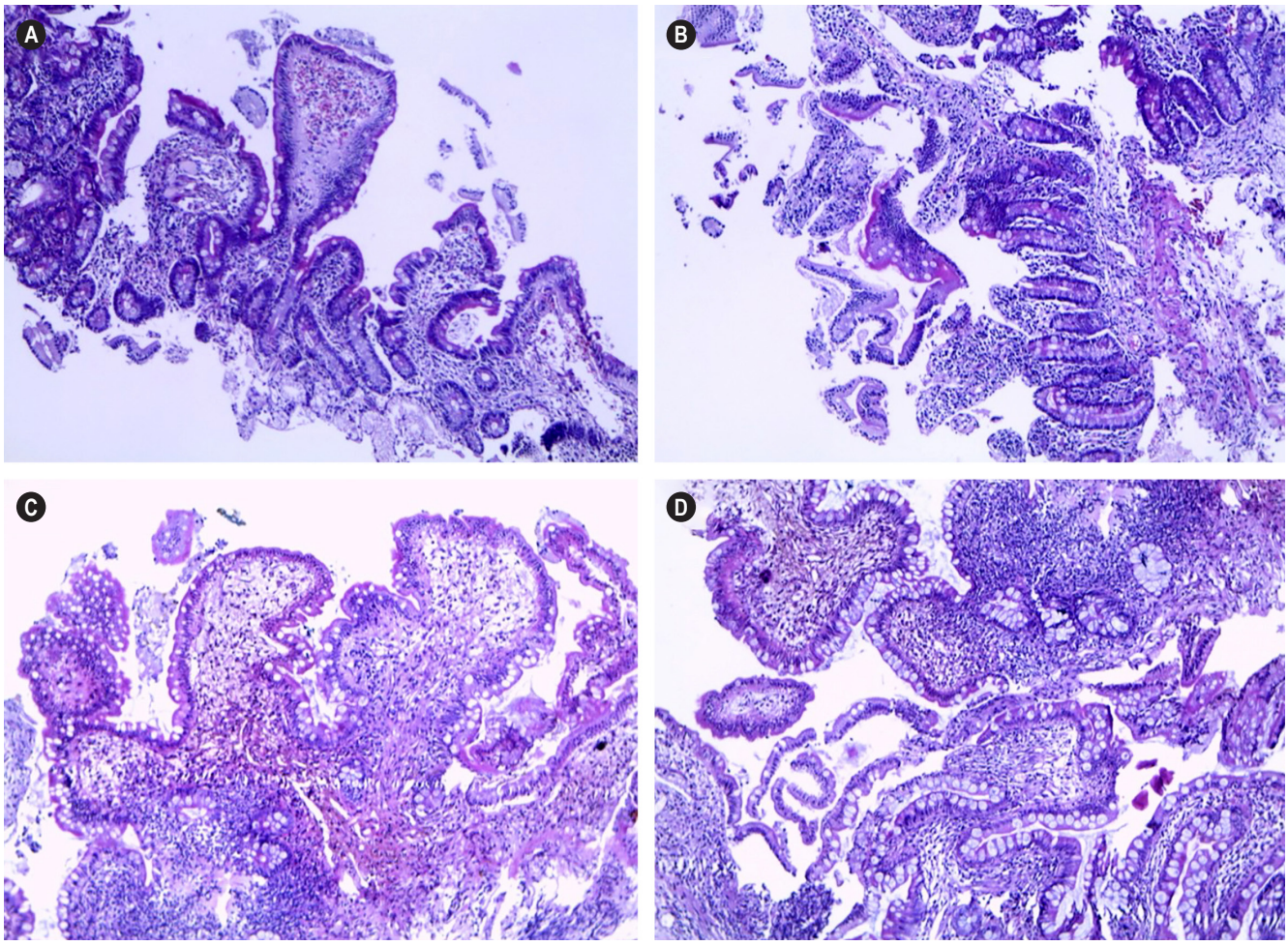


Figure 1. Duodenal and ileal biopsy specimens stained with hematoxylin and eosin, 100×. **A** and **B.** Mild, nonspecific chronic duodenitis and mild mucosal atrophy. Fewer than 30 lymphocytes per 100 enterocytes are present. **C** and **D.** Mild, nonspecific chronic ileitis. There is no villous atrophy. No features suggestive of intestinal tuberculosis, Crohn's disease, or celiac disease are identified. Images courtesy of the authors.

Table 2. Cases reported in PubMed (2020–2025) in which protein-losing enteropathy was the initial manifestation of systemic lupus erythematosus

Author and year	Country	Sex and age	Clinical presentation	Laboratory findings	Endoscopy/ colonoscopy	Pathology	Diagnosis	Treatment
Thanh C, 2025[21]	Vietnam	Female, 15 years	Bilateral eyelid and lower-extremity edema, ascites, headache	ANA: 1:100 anti-DNA: positive; lupus anticoagulant: positive; C3: 4.8 g/dL; C4: 0.7 g/dL	Gastritis	Gastric biopsy: nonspecific inflammation. Ileal biopsy: nonspecific chronic ileitis	Protein-losing enteropathy	Methylprednisolone, prednisolone, hydroxychloroquine, warfarin
Abu Jheasha A, 2024[5]	Palestine	Female, 22 years	Epigastric pain, diarrhea, vomiting, ascites, mild lower-extremity edema, arthralgia	ANA: positive; anti-DNA: positive; C3: 26 mg/dL C4: 2.9 mg/dL	Diffuse edema of the gastric and duodenal mucosa	Not reported	Protein-losing enteropathy	Methylprednisolone, hydroxychloroquine
Ramesh R, 2023[20]	Sri Lanka	Female, 38 years	Generalized ascending edema, ascites, mild exertional dyspnea, arthralgia, alopecia	ANA: 1:1000; anti-DNA: negative; C3: 30 mg/dL C4:10 mg/dL	Erythematous, diffusely appearing gastric mucosa. Granular-appearing terminal ileal mucosa with shortened villi	Ileal biopsy: mucosa with hyperplastic lymphoid follicles. No features of inflammatory bowel disease or vasculitis	Protein-losing enteropathy	Prednisolone, hydroxychloroquine
Abdalla E, 2023[19]	Sudan	Male, 33 years	Ascites, jaundice, fever, dyspnea, weight loss, abdominal pain	ANA: 1:320; anti-Sm: positive	Gastritis	Not reported	Protein-losing enteropathy	Prednisone, azathioprine, hydroxychloroquine
Prabasara K Jr, 2021[12]	Sri Lanka	Female, 39 years	Anasarca, ascites, alopecia	ANA: positive; ENA: negative	Normal	Duodenal biopsy: mild lymphoplasmacytic infiltrates. Jejunal biopsy: lymphoid follicle in the lamina propria of the jejunum	Protein-losing enteropathy	Prednisolone, hydroxychloroquine, azathioprine
Quintero E, 2021[22]	United States	Female, 28 years	Watery diarrhea, intermittent abdominal pain, vomiting, ascites	ANA: 1:1280; anti-DNA: positive;	Esophagitis, gastritis	Duodenal biopsy: normal. Colonic biopsy: normal	Protein-losing enteropathy	Methylprednisolone, prednisone

Table prepared by the authors.

Although lupus may involve the intestine in a patchy distribution, transmural involvement is more characteristic of Crohn's disease. Lupus enteritis is a form of small-bowel vasculitis that predominantly affects the jejunum and ileum, with radiologic or histologic evidence of mesenteric arteriolar vasculitis and a favorable response to corticosteroids and belimumab[18]. These findings were also absent in the present case.

In this report, a substantial improvement in hemoglobin and albumin levels was observed after corticosteroid therapy. Although Crohn's disease is also treated with corticosteroids, no compatible histologic findings were identified, and the clinical course was not typical of this disease. Given the favorable response to prednisone and the lack of availability of other immunosuppressive agents in our setting, corticosteroid therapy alone was continued. Despite this, the patient subsequently died from associated infectious complications.

Finally, this case was unusual because the patient was a young man, considering that SLE occurs predominantly in

women[1]. Furthermore, its presentation as autoimmune duodenitis associated with PLE makes this a rarely described case in the literature. Therefore, a bibliometric search was conducted in PubMed using the terms "(protein-losing enteropathy) AND (Systemic lupus erythematosus)" and "(autoimmune duodenitis) AND (Systemic lupus erythematosus)." Six similar reports were identified (**Table 2**). Of these, 83.33% involved young women, only one case involved a young man[19], and none described an association between autoimmune duodenitis and PLE.

Regarding the clinical presentation, the most frequently reported symptoms were generalized edema, ascites, diarrhea, and abdominal pain, whereas arthralgia[5,20] and alopecia[12,20] were reported least frequently. Because our patient did not present with the latter symptoms, the diagnosis of SLE was more difficult to establish.

Regarding treatment, all of the reports reviewed used systemic corticosteroid regimens, such as prednisolone or methylprednisolone, together with hydroxychloroquine, azathioprine, and anticoagulants in some cases[12,19,21].

CONCLUSIONS

SLE is an autoimmune disease that is uncommon in men and may initially present with unusual gastrointestinal manifestations that complicate diagnosis, particularly in the absence of classic signs. This report describes an uncommon presentation of SLE characterized by autoimmune duodenitis and protein-losing enteropathy, an association that has rarely been documented as a simultaneous presentation in the literature. SLE should be suspected in the presence of generalized edema, severe hypoalbuminemia without proteinuria, persistent anemia, and nonspecific inflammatory endoscopic and histologic findings suggestive of possible gastrointestinal involvement secondary to SLE, together with positive autoimmune markers and a response to corticosteroid therapy.

Ethical considerations

The case report respected the patient's privacy and confidentiality. The patient signed informed consent, and the report was conducted in accordance with the principles of the Declaration of Helsinki.

Informed consent

Written informed consent was obtained from the patient, and the confidentiality of his data was maintained.

Conflicts of interest

The authors declare that they have no conflicts of interest regarding the publication of this manuscript.

Use of artificial intelligence

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Author contributions

- Study conception and design: Andrea Isabel Ancasi-Medina, Helena C. Calle-Herrera, and Cristhian A. Vizcarra-Vizcarra
- Data acquisition: Andrea Isabel Ancasi-Medina, Helena C. Calle-Herrera, Cristhian A. Vizcarra-Vizcarra, Lily E. Montesinos-Valencia, Zaima Luque-Quilla, and Yolanda A. Llerena-Concha.
- Data analysis and interpretation: Andrea Isabel Ancasi-Medina, Helena C. Calle-Herrera, Cristhian A. Vizcarra-Vizcarra, Lily E. Montesinos-Valencia, Zaima Luque-Quilla, and Yolanda A. Llerena-Concha.
- Manuscript drafting: Andrea Isabel Ancasi-Medina, Helena C. Calle-Herrera, and Cristhian A. Vizcarra-Vizcarra.
- Critical revision of the manuscript: Andrea Isabel Ancasi-Medina, Helena C. Calle-Herrera, Cristhian A. Vizcarra-Vizcarra, Lily E. Montesinos-Valencia, Zaima Luque-Quilla, and Yolanda A. Llerena-Concha.

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