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Clinical case

Insulin resistance syndrome in a patient with limited cutaneous systemic sclerosis and type 2 diabetes and its response to plasma exchange, immunosuppression, and the Minimed® 780G continuous subcutaneous insulin infusion hybrid system: A case report

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Abstract

Introduction: Type B insulin resistance syndrome (TBIRS) is an immune-mediated disorder characterized by severe acanthosis nigricans and difficult-to-control diabetes mellitus. This condition poses a complex clinical challenge.

Objective: To describe the case of a patient with clinical features compatible with Type B insulin resistance syndrome, highlighting the challenges in glycemic control and the response to immunosuppressive therapy combined with a hybrid continuous subcutaneous insulin infusion system.

Case presentation: A 57-year-old woman with a history of limited cutaneous systemic sclerosis and type 2 diabetes mellitus presented with severe insulin resistance, acanthosis nigricans, and markedly elevated blood glucose levels. Anti-insulin receptor antibodies were consistently negative. The patient received multiple treatments, including plasmapheresis, immunosuppressive therapy, and continuous subcutaneous insulin infusion using a MiniMed® 780G system, resulting in improved continuous glucose monitoring metrics and reduced daily insulin requirements.

Highlights

- A hybrid closed-loop system, corticosteroids, and plasma exchange may be useful.
- There is a diagnostic challenge in these cases, clinicians must be attentive.
- Need for more research to validate the effectiveness of different therapies.

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Discussion: This case suggests a possible association between systemic sclerosis and type B insulin resistance syndrome despite persistently negative anti-insulin receptor antibodies. A false-negative result may be explained by prior immunomodulatory treatment. The combination of immunosuppressive therapy and a hybrid closed-loop continuous subcutaneous insulin infusion system demonstrated favorable clinical outcomes.

Conclusion: This case report contributes to the understanding of type B insulin resistance syndrome and highlights the potential benefit of integrating immunosuppressive therapies with advanced insulin delivery technologies. Further studies are needed to define their role in the management of patients with severe insulin resistance.

Keywords: Hyperglycemia, acanthosis nigricans, cyclophosphamide, hyperinsulinemia, continuous subcutaneous insulin infusion system, CSII.

Síndrome de resistencia a la insulina en una paciente con esclerosis sistémica cutánea limitada y diabetes tipo 2, y su respuesta al intercambio plasmático, la inmunosupresión y el sistema híbrido de infusión continua de insulina subcutánea Minimed® 780G: reporte de caso

Resumen

Introducción: el síndrome de resistencia a la insulina (IRS) tipo B es una enfermedad de origen inmunomediado, caracterizada por acantosis nigricans severa y diabetes mellitus de difícil control. Esta condición representa un desafío clínico complejo.

Objetivo: describir el caso de una paciente compatible con el IRS tipo B, destacando los desafíos en el control glucémico, así como la respuesta al tratamiento inmunosupresor combinado con un sistema híbrido de infusión subcutánea continua de insulina.

Presentación del caso: mujer de 57 años con antecedentes de esclerosis sistémica cutánea limitada y diabetes mellitus tipo 2, quien presentó resistencia severa a la insulina, acantosis nigricans y niveles de glucosa marcadamente elevados. Los anticuerpos contra el receptor de insulina fueron consistentemente negativos. La paciente recibió múltiples tratamientos, incluyendo plasmaféresis, inmunosupresión e infusión subcutánea continua de insulina mediante un sistema MiniMed® 780G, logrando una mejoría en los parámetros del monitoreo continuo de glucosa y una reducción de la dosis diaria de insulina.

Discusión: este caso sugiere una posible asociación entre la esclerosis sistémica y el IRS tipo B, pese a la negatividad persistente de los anticuerpos contra el receptor de insulina. Un resultado falso negativo podría explicarse por los tratamientos inmunomoduladores previos. La combinación de inmunosupresión y un sistema híbrido de infusión subcutánea continua de insulina mostró resultados clínicos favorables.

Conclusión: Este reporte contribuye a la comprensión del IRS tipo B y resalta el potencial de integrar terapias inmunosupresoras con tecnologías avanzadas de administración de insulina. Se requieren estudios adicionales para definir su papel en el manejo de pacientes con resistencia extrema a la insulina.

Palabras clave: hiperglucemia, acantosis nigricans, ciclofosfamida, hiperinsulinemia, sistema de infusión subcutánea continua de insulina, ISCI.

Destacados

- Un sistema híbrido de asa cerrada, corticosteroides e intercambio plasmático pueden ser útiles.
- Hay un desafío diagnóstico en estos casos; los médicos deben estar atentos.
- Es necesaria más investigación para validar la efectividad de diferentes terapias.

Introduction

Insulin resistance syndrome (IRS) comprises a rare group of conditions characterized by insulin receptor dysfunction, hyperinsulinemia, hyperglycemia, and various associated manifestations, including weight loss, acanthosis nigricans, and hirsutism (1). This syndrome is classified into type A and type B, with the former arising from genetic abnormalities of the insulin receptor and the latter from autoantibodies against the receptor (2–3). Both types require high exogenous insulin doses to achieve glycemic control (1, 4).

In type A IRS, symptoms typically manifest early in life, and treatment focuses on a hypocaloric diet, physical activity, insulin administration, recombinant insulin-like growth factor 1 (rIGF-1), and insulin sensitizers (5).

As of 2020, there were 119 reported cases of type B IRS in the literature (6), primarily affecting females between the fifth and sixth decades of life, with approximately 52.2% of cases associated with systemic autoimmune diseases, most commonly systemic lupus erythematosus (SLE). Recommended treatments, based on case reports and case series, include various immunosuppressants, such as glucocorticoids, cyclophosphamide, cyclosporine, azathioprine, and intravenous immunoglobulin, as well as plasma exchange (3, 5, 6–8). Herein, we present a probable new case of IRS managed with plasma exchange, immunosuppression, and a hybrid continuous subcutaneous insulin infusion system.

Case report

In July 2022, a 57-year-old woman presented with progressive generalized weakness, blurred vision, dizziness, and markedly elevated blood glucose levels (>400 mg/dL). She had a history of limited cutaneous systemic sclerosis, diagnosed in 1992, and positive antinuclear antibodies with an anticentromere pattern. The patient did not report any other relevant medical history.

In 2010, she was diagnosed with type 2 diabetes mellitus, and her glycemic control progressively deteriorated despite conventional treatments, including diet and oral hypoglycemic agents. Concurrently, she developed severe acanthosis nigricans, necessitating increasing insulin doses. By 2014, her weight was 77 kg, with a body mass index (BMI) of 28 kg/m2, and she required insulin doses of up to 1200 IU/day. Type B IRS was suspected as a diagnostic consideration. Notably, anti-insulin receptor antibodies were consistently negative, while anti-insulin antibodies were positive on several occasions (as determined by radioimmunoassay). She underwent seven sessions of plasma exchange, methylprednisolone pulses, rituximab, and azathioprine on an outpatient basis, resulting in improved glycemic control, a reduction in daily insulin requirements to 140–200 IU, and an HbA1c level of 10.5%. However, glycemic control progressively worsened, prompting the initiation of continuous subcutaneous insulin infusion (CSII) therapy using the MiniMed® Paradigm® VEO device, later followed by transition to the MiniMed® 640G, albeit with limited improvement due to a persistently high coefficient of variation, time in range (TIR) of 50–60%, and time above range (TAR) of >25%, while still requiring an average insulin dose of 300 IU/day. In 2015, liraglutide was added to the treatment regimen but discontinued due to gastrointestinal intolerance. Concurrently, sodium-glucose cotransporter protein-2 (SGLT-2) inhibitors were maintained for several years. Between 2016 and June 2022, she experienced five hospitalizations due to disease relapses associated with increased insulin resistance, necessitating methylprednisolone pulses and multiple plasma exchange sessions. She transitioned from azathioprine to tacrolimus from 2015 to 2018, initially achieving an approximately 8% improvement in HbA1c levels, followed by a loss of efficacy.

Her most recent hospitalization, in June 2022, was prompted by weight loss, exacerbated glycemic control, and worsening acanthosis nigricans (figure 1), with an HbA1c level of 10.3%. She denied symptoms related to the cardiopulmonary, gastrointestinal, urological, or neurological systems. On examination, her blood pressure was 97/68 mmHg, her pulse rate was 82 bpm, and she exhibited velvety-like, hyperpigmented, hyperkeratotic plaques in intertriginous areas, as well as on the face, neck, and upper extremities.

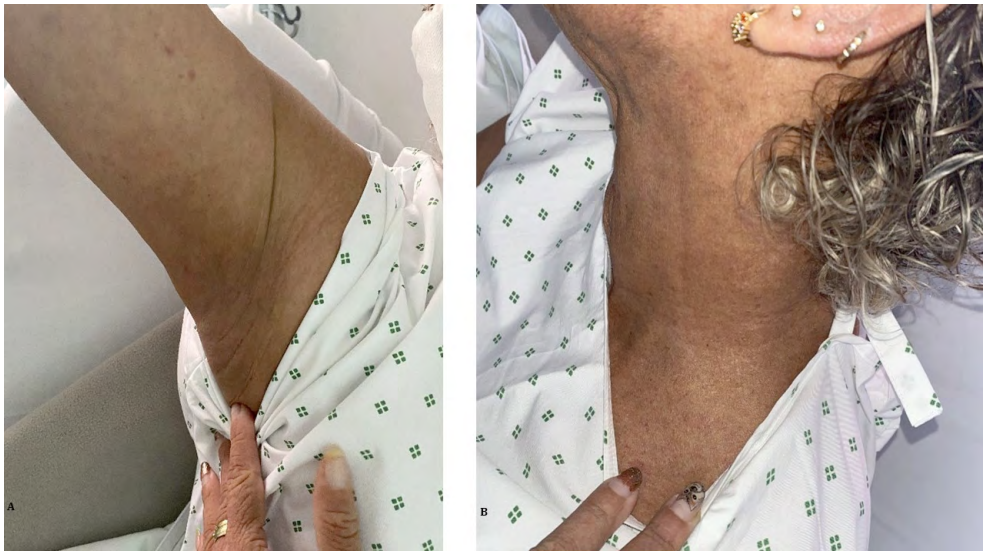

Figure 1. Extensive acanthosis nigricans in the axillary fold (A) and neck (B) of our patient as a marker of insulin resistance

Source: Author's own elaboration.

Table 1 outlines the diagnostic studies conducted during hospitalization. Severe diabetes decompensation necessitated high-dose insulin therapy via continuous intravenous insulin infusion, with an approximate daily dose of 300 IU. Five plasma exchange sessions, three methylprednisolone pulses (1 g each), and 750 mg of intravenous cyclophosphamide were administered, followed by monthly cyclophosphamide therapy for three months. Despite these interventions, glycemic control remained inadequate and variable. Consequently, the patient transitioned to the MiniMed® 780G CSII device. In the following days, her continuous glucose monitoring metrics significantly improved (figure 2A). Over the subsequent weeks, she achieved her target time in range (TIR) and coefficient of variability values, with a daily insulin dose of approximately 200 IU (figure 2B). The patient continues to receive oral prednisolone, which has been tapered to 10 mg daily.

Discussion

Type B IRS has been predominantly associated with various systemic autoimmune diseases, primarily SLE (9-10), and less frequently with systemic sclerosis (11-12). Herein, we report a case of a patient with limited cutaneous systemic sclerosis, characterized by the classic immunological marker of anticentromere antibodies, who developed insulin resistance associated with severe acanthosis nigricans. Interestingly, she tested negative for anti-insulin receptor antibodies, which diverges from the classic presentation of type B IRS (13).

According to existing literature, the diagnosis of type B IRS has been supported by the presence of anti-insulin receptor antibodies, which have been evaluated in research laboratories using techniques such as ELISA, immunoprecipitation, or Western blot, primarily due to the limited availability of standardized commercial assays (13-14). In our case, the patient tested negative for these antibodies using a radioimmunoassay at a commercial laboratory. Therefore, we believe this result may represent a false negative attributable to the technique itself and/or low antibody titers associated with prior treatments with rituximab and systemic steroids. Instead, she presented positive anti-insulin antibodies, which have been associated with both hyperglycemia and hypoglycemia in the context of Hirata's disease (15). However, she did not experience hypoglycemic episodes and did not meet the diagnostic criteria, as she had previously been exposed to exogenous insulin (14).

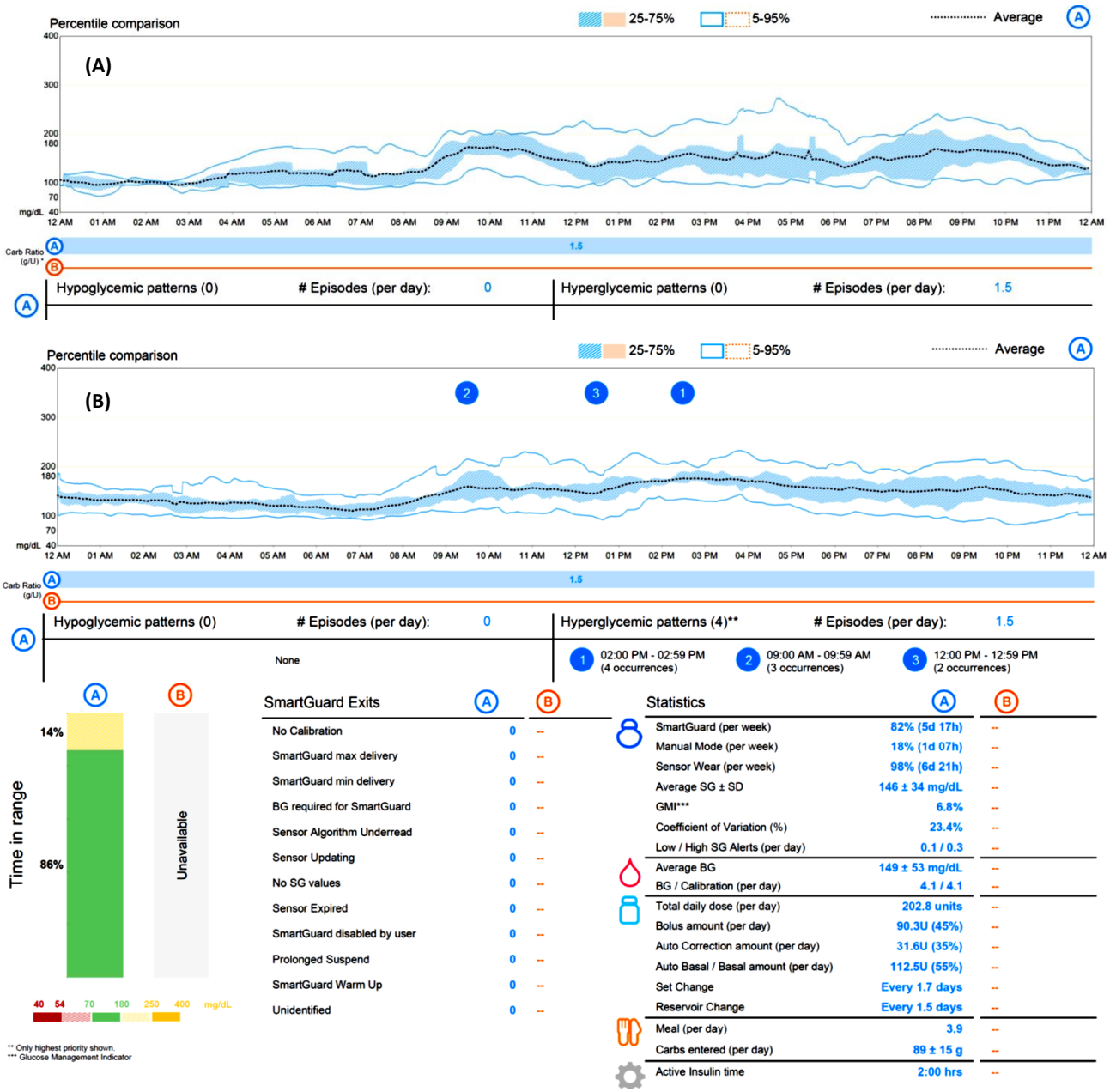

Figure 2. **(A)** Average CGM report in the first three days after plasma exchange and starting CSII MiniMed® 780G, without detecting episodes of hypoglycemia and with an average blood glucose of 145 ± 61 mg/dL. **(B)** Four weeks after plasmapheresis + cyclophosphamide + the new CSII MiniMed® 780G, TIR of 85% without hypoglycemia and 15% above the range was a good goal for the patient considering her previous difficulties in glycemic control. Additionally, in the general CSII data, it was observed that she had a good glucose control indicator (ICG) (6.8%), with a requirement for 202 IU of insulin per day.

Source: Author's own elaboration.

Table 1. Summary of paraclinical tests reported in June 2022

Laboratory tests	Result	Reference range	Laboratory tests	Result	Reference range
Peripherally blood			**Diabetes mellitus markers**		
White blood cell count (10ˆ3/µL)	18.28	3.98 – 10.04	Serum glucose (mg/dL)	348	74 – 106
Neutrophils (%)	75.5	34 – 71.1	Glycosylated hemoglobin (%)	11.2	<7
Lymphocytes (%)	17.70	19.3 – 51.7	Basal insulin (µUI/mL)	52	2.6 – 24.9
Hemoglobin (g/dL)	14.7	11.2 – 15.7	HOMA IR	44	<2
Platelet count (10ˆ3/µl)	244	182 – 369	Anti-insulin antibodies (%)	12.7 %	<8.2
TP (sec.)	9.6	9.4 – 12,5	Anti-insulin receptor antibodies	<1	<1
aPTT (sec.)	27.9	25.1 – 36.5			
Blood biochemistry			**Endocrinological tests**		
ALT (U/L)	36	0 – 31	Total testosterone (ng/mL)	0.9	0.03 – 0.41
AST (U/L)	26	0 – 32	TSH (µUI/mL)	1.5	0.27 – 4.2
Total bilirubin (mg/dL)	0.31	0 – 0.9	**Immunological tests**		
Alkaline phosphatase (U/L)	179	35 – 104	ANA (centromeric)	1:1280 dil.	
LDH (U/L)	206	135 – 214	ENA (SSA, SSB, Sm, RNP) (U/mL)	<1	<15
Total proteins (g/dL)	6.6	6.4 – 8.3	Anti-dsDNA (IU/ mL)	<0.1	<20
Albumin (g/dL)	4.1	3.5 – 5.2	C3 (mg/dL)	130	90 – 180
Ferritin (ng/mL)	449	13 – 150	C4 (mg/dL)	16	10 – 40
B12 vitamin (pg/mL)	612	197 – 771	Anti-CL antibodies IgG (GPL-U/mL)	1	<10
Uric acid (mg/dL)	4.6	2.4 – 5.7	Anti-CL antibodies IgM (MPL-U/mL)	0.9	<7
Creatinine (mg/dL)	0.65	0.51 – 0.95	Anti-β2GPI antibodies IgG (U/mL)	1.6	<5
Sodium (mmol/L)	137	136 – 145	Anti-β2GPI antibodies IgM (MU/mL)	9.4	<8

Potassium (mmol/L)	4.1	3.5 – 5.1	Lupus anticoagulant (seg.)	Negative	
Chlorine (mmol/L)	99	98 – 107	Anti-mitochondrial antibodies	1:80 dil.	
Calcium (mg/dL)	8.6	8.6 – 10	Anti-smooth muscle antibodies	1:80 dil.	
Magnesium (mg/dL)	2.1	1.59 – 2.56	Rheumatoid factor	40	<14
Phosphorus (mg/dL)	3.8	2.5 – 4.5	**Infectious tests**		
Total cholesterol (mg/dL)	202	0 – 200	HBsAg	0.34	<1
HDL (mg/dL)	100	< 100	Anti-HBc antibodies	2.3	>1
LDL (mg/dL)	36	> 65	Anti-HIV I and II antibodies	0.19	<1
Triglycerides (mg/dL)	463	0 – 200	RPR serology	Not reactive	

PT: prothrombin time; aPTT: activated partial thromboplastin time; ALT: alanine aminotransferase; AST: aspartate aminotransferase; LDH: lactate dehydrogenase; HDL: high-density lipoprotein; LDL: low-density lipoprotein; HOMA IR; homeostatic model assessment for insulin resistance; ANA: antinuclear antibodies; ENA: extractable nuclear antigen; dsDNA: double-stranded DNA; C3-C4: C3 and C4 complement; CL: cardiolipin; β2GPI: beta-2-glycoprotein I; HBsAg: hepatitis B surface antigen; HBc: total hepatitis B core; HIV: human immunodeficiency virus; RPR: rapid plasma reagin.

Source: Author's own elaboration.

Various glycemic control strategies have been explored for the management of type B IRS, including immunosuppression and alternative insulin regimens. The literature describes only two cases involving the use of CSII. Sjoholm *et al.* (16) reported the case of a 25-year-old patient who required plasma exchange and multiple immunosuppressive therapies, resulting in partial improvement. Osanami *et al.* (17) described a 66-year-old man who underwent plasma exchange, rituximab treatment, and MiniMed® 640G CSII, with positive outcomes. Beginning in 2014, our patient was managed with the MiniMed® Paradigm® VEO, coupled with continuous glucose monitoring (CGM), and was subsequently transitioned to the MiniMed® 640G, with limited success. She was later switched to the MiniMed® 780G hybrid system. Unlike its predecessor, this device automatically adjusts basal insulin infusion every five minutes based on continuous glucose monitoring values within a specific target range. Additionally, it delivers automatic correction boluses controlled by a hyperglycemia prediction algorithm (18). This transition resulted in acceptable time in range (TIR) values and a reduction in the daily insulin dose by approximately 100 IU compared with previous devices. Concurrently, the patient continued to receive plasma exchange and low-dose maintenance glucocorticoids. However, she did not achieve the remission criteria proposed in previous studies (19-20). This may be attributed to the patient's pre-existing type 2 diabetes mellitus before the onset of the insulin receptor antibody syndrome.

Given the rarity of immunologically mediated forms of IRS, conducting clinical trials to validate different therapies remains challenging. Apart from plasma exchange and immunosuppressants, proposed management strategies include

combination regimens (18) and, since the early 21st century, the use of SGLT-2 inhibitors (19). While immunosuppressive therapies and plasma exchange remain common treatments, the successful integration of a hybrid closed-loop CSII system with these interventions appears promising for managing severe insulin resistance.

In summary, this case report adds to the growing body of knowledge regarding insulin resistance syndrome (IRS), shedding light on innovative treatment strategies for complex and challenging cases. It underscores the vital role of individualized patient care and ongoing research in addressing rare and intricate metabolic disorders such as IRS. Despite extensive intervention, the precise etiology of the marked insulin resistance observed in our patient remains elusive. While this case report suggests the potential benefits of integrating a hybrid closed-loop continuous subcutaneous insulin infusion (CSII) system with traditional immunosuppressive therapy and plasma exchange, further comprehensive investigations are necessary to validate these findings. This case highlights the need for continued research and collaboration to identify optimal therapeutic approaches for IRS and related conditions.

Authors' contributions

Fabio Nelson Figueroa-Agudelo: Conceptualization, supervision; Andrés Octavio García-Trujillo: Conceptualization, supervision; Víctor Manuel Blanco-Pico: Methodology, formal analysis and investigation, supervision; Guillermo Edinson Guzmán-Gómez: Formal analysis and investigation, supervision; Jorge Wilmar Tejada-Marín: Writing - original draft preparation, supervision; Carlos Alberto Cañas: Writing - original draft preparation, supervision; and Karen Milena Feriz-Bonelo: writing - review and editing, supervision.

Ethical statement

For the preparation of this case, patient confidentiality was ensured. Identifying information was omitted, and anonymous language was used to protect the patient's privacy. In addition, informed consent was obtained from the patient, and authorization was granted by the institutional ethics committee for the publication of the case. Finally, the basic principles of medical ethics, including beneficence, non-maleficence, autonomy, and justice were upheld, ensuring that the case contributed to medical knowledge regarding the management of patients with similar conditions.

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Conflicts of interest

The authors declare that they have no known competing financial interests or personal relationships that could have influenced the work reported in this paper.

Artificial intelligence (AI) disclosure statement

The authors declare that artificial intelligence was not used in the preparation, analysis, or writing of this clinical case report.

Data availability statement

The authors declare that no data associated with this study are available in publicly accessible repositories. Any inquiries or requests regarding the article should be directed to the corresponding author.

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