



Paraneoplastic Dermatomyositis as the Initial Presentation of Endometrial Adenocarcinoma: First Reported Case in Latin America and Literature Review

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Abstract

Idiopathic inflammatory myopathies are a group of rare autoimmune diseases. A subset of these cases may occur in association with an underlying malignancy, a condition known as cancer-associated myositis (CAM), reported in approximately 13% of cases according to the Euromyositis registry. The malignancies most commonly linked to this syndrome include cancers of the lung, thyroid, breast, stomach, ovaries, cervix, and hepatocellular carcinoma. However, the association with endometrial adenocarcinoma has been only rarely described in the medical literature. In our review, we identified six previously reported cases (from Japan, Canada, and the United States). We present the first documented case in Latin America of a patient who developed this uncommon form of CAM, characterized by severe clinical presentation and favorable response following treatment directed at the neoplasm and administration of intravenous immunoglobulin.

Keywords: *Paraneoplastic dermatomyositis, inflammatory myopathies, Endometrial cancer, axonal motor polyneuropathy, Abnormal uterine bleeding.*

Introduction

Cancer-associated myositis (CAM) has been reported in approximately 13% of patients according to the Euromyositis Registry ^[1]. The rare association between endometrial adenocarcinoma and paraneoplastic inflammatory myopathy complicates its diagnostic consideration within the context of autoimmune disease. This report presents a clinical case that illustrates this relationship. It underscores the importance of a targeted medical history, in which symptoms such as metrorrhagia may be key to suspecting an underlying neoplastic process in the context of idiopathic inflammatory myopathy—particularly when it presents with atypical features or is initially difficult to manage.

Case Presentation

A 51-year-old female with a past history of colon cancer treated between 2019 and 2020 with chemotherapy and partial surgical resection presented with five months of abnormal uterine bleeding

associated with colicky pelvic pain and a 10-kg weight loss during that period (current weight 49 kg).

Upon admission, she appeared chronically ill, pale, and tachycardic, with proximal muscle weakness, loss of head control, impaired swallowing, bilateral and symmetric violaceous periorbital erythema with eyelid edema (“heliotrope rash”), poikiloderma in photo-exposed areas of the upper chest, and hyperkeratosis with fissuring of the hands (**Figure 1A, B, C**).

The initial approach focused on abnormal uterine bleeding with severe anemia requiring transfusional support. She had previously undergone outpatient pelvic ultrasonographic evaluation, which showed an endometrial thickening of 7.6 mm and multiple uterine fibroids. CA 19-9 levels were elevated. Given these findings, an endometrial biopsy was performed, revealing an invasive malignant neoplasm with papillary and solid patterns, extensive necrosis, and no lymphovascular invasion. Staging studies—including contrast-enhanced MRI of the abdomen and pelvis, upper GI endoscopy, and colonoscopy—showed no evidence of metastasis or dissemination.

Regarding proximal muscle weakness and an elevated creatine kinase (CK) level of 608 U/L at admission, together with skin findings highly suggestive of inflammatory myopathy—specifically dermatomyositis—complementary tests were ordered (**Table 1**).

MRI of the lower limbs (T2-weighted sequences) showed extensive edema of muscle fibers in the anterior and posterior compartments of both thighs and legs, with moderate atrophy and fatty infiltration. A muscle biopsy of the right thigh revealed myofibrillar degeneration and interstitial lymphohistiocytic inflammation involving the endomysium and perimysium (**Figure 2**). Based on these findings, a diagnosis of paraneoplastic dermatomyositis associated with endometrial carcinoma was made.

The Gynecologic Oncology team performed a hysterectomy with bilateral salpingo-oophorectomy. Immunohistochemistry

revealed endometrioid carcinoma with lymphovascular invasion, metastatic involvement of the left ovary, and a positive left obturator-iliac lymph node. The final staging of the endometrial adenocarcinoma was FIGO stage IIIC1.

Management of dermatomyositis included high-dose methylprednisolone 1000 mg IV daily for three days due to ominous clinical features (cephaloparesis) and high risk of respiratory compromise, followed by oral prednisolone 1 mg/kg/day. Due to clinical refractoriness, intravenous immunoglobulin (IVIG) was administered at 2 g/kg over five days (400 mg/kg/day), along with azathioprine 50 mg orally every 12 hours and methotrexate 15 mg orally weekly. The patient showed progressive clinical improvement in muscle strength and laboratory parameters. She was discharged after 49 days of hospitalization with scheduled outpatient follow-up.



Figure 1. A. Mild heliotrope erythema. B. Photosensitive rash and poikiloderma on the upper chest in a “V-sign” distribution. C. Hyperkeratosis and fissuring of the fingertips.

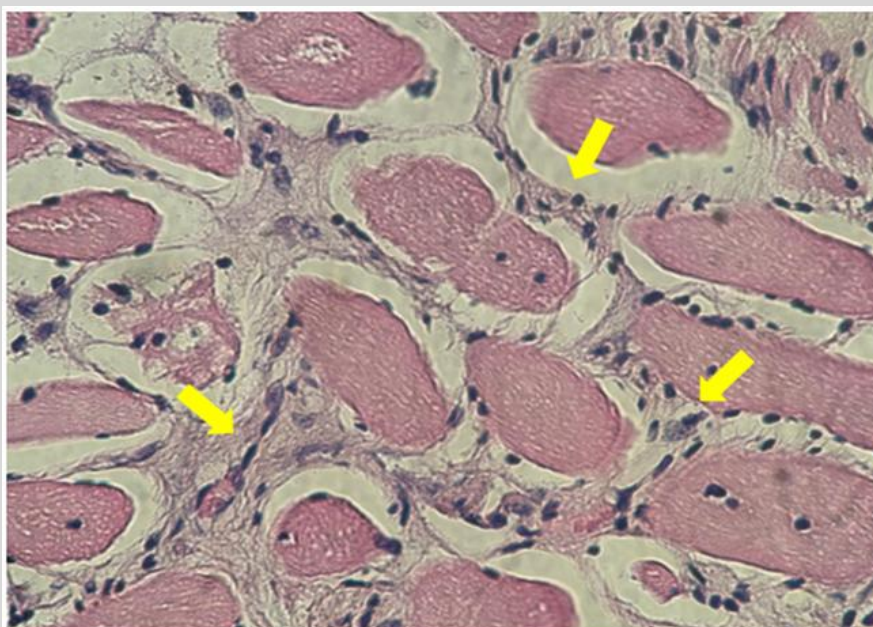


Figure 2: Light microscopy, 40x, H&E stain: Lymphocytic infiltration of the endomysium (yellow arrow).

Table 1: Laboratory Findings

Laboratory Test	Values	Reference Ranges
ANAs	1:320, fine granular pattern	< 1:80
Anti-dsDNA	Negative	< 1/10 dilutions
Anti-Ro	8.3	< 15 U/mL
Anti-La	3.3	< 15 U/mL
Anti-Sm	0.4	< 15 U/mL
Anti-RNP	2.2	< 15 U/mL
Anti-Jo1	3.3	< 15 U/mL
Anti-β2 Glycoprotein 1 IgM	1.0	< 5 U/mL
Anti-β2 Glycoprotein 1 IgG	1.6	< 5 U/mL
Anti-Cardiolipin IgM	1.0	< 5 U/mL
Anti-Cardiolipin IgG	2.2	< 5 U/mL
Lupus anticoagulant	1.08	< 1.2
RPR	Non-reactive	Non-reactive
HIV 1 and 2 antibodies	Negative	Negative
HBsAg	0.11	< 0.9
Anti-HCV	0.04	< 0.90
TSH	5.14	0.37–4.7 mIU/L
Free T4	2.08	0.7–1.8 ng/dL

Abbreviations: Anti-dsDNA: Anti-double stranded, RPR: Rapid plasma reagin. TSH: Thyroid-stimulating hormone, HBsAg: Hepatitis B surface antigen

Table 2: Comparison of reported cases of paraneoplastic dermatomyositis secondary to endometrial adenocarcinoma.

Characteristic	Current Case (Berrocal et al., 2025)	Lim et al., 2020 ⁸	Wada et al., 2014 ⁹	Kasuya et al., 2013 ¹⁰	Famularo et al., 2017 ¹¹	Khamooshi et al., 2022 ⁷
Patient age	51 years	58 years	46 years	52 years	65 years	64 years
Temporal relationship DM–Cancer	DM precedes cancer diagnosis	Recurrence 4 years after initial treatment and remission	Simultaneous diagnosis	DM precedes cancer by 2 months	DM leads to cancer discovery	DM during active cancer treatment
Histology	Endometrioid adenocarcinoma G2	Nodal recurrence of endometrial adenocarcinoma	Type 1 endometrioid adenocarcinoma	Endometrioid, TIF1γ+, Smad4–	Endometrial adenocarcinoma	Metastatic serous carcinoma (lung and liver)
Staging	FIGO IIIC1 (ovary and lymph node involvement)	Isolated nodal recurrence	FIGO IIIB (ovarian metastases)	No lymphadenectomy performed	Not reported	FIGO IV (lung/liver metastases)
Myositis-specific antibodies	Not reported	Anti-TIF1γ (++) ANA 1:640	Not reported	Anti-TIF1γ (anti-p155/140) positive	Not detected	Anti-TIF1γ and anti-Mi-2 positive
Cutaneous manifestations	Heliotrope rash, poikiloderma, fissured hands	Gottron papules, V-sign, periungual changes	Gottron papules and heliotrope rash	Severe palpebral rash, recurrent cutaneous flares	Heliotrope rash, neck and chest erythema	Pruritic erythematous rash on chest and extremities
Muscular manifestations	Severe weakness, dysphagia, cephaloparesis	None (amyopathic form)	None (amyopathic form)	Mild weakness, CK 863	None (amyopathic form)	Severe dysphagia, proximal weakness, CK 4500
Oncologic treatment	Complete oncologic surgery	Lymphadenectomy; radiotherapy discontinued	Surgery + chemotherapy (carboplatin/paclitaxel)	Hysterectomy without lymphadenectomy	Declined surgery	Surgery, chemotherapy, and radiotherapy; no clinical response
Immunologic treatment	Steroids, IVIG, azathioprine, methotrexate	Topicals, hydroxychloroquine; remission post-surgery	Not reported	Prednisone, partial improvement	Not administered	IVIG, IV steroids, mycophenolate

						; no clinical improvement
Outcome	Sustained clinical and CK improvement	Rapid cutaneous remission after surgery	Progressive improvement	Remission after surgery; recurrence with lung metastases	Lost to follow-up	Death from sepsis and multiorgan failure

Abbreviations: DM: Dermatomyositis. ANA: Antinuclear antibodies. Anti-TIF1 γ : Anti-Transcription Intermediary Factor 1 Gamma. CK: Creatine kinase. CT: Chemotherapy. RT: Radiotherapy. IVIG: Intravenous immunoglobulin. AZA: Azathioprine. MTX: Methotrexate. HCQ: Hydroxychloroquine.

Discussion

Inflammatory myopathies are a group of immune-mediated diseases characterized by autoimmune inflammatory changes in muscle tissue, usually manifesting as weakness (although amyopathic or hypomyopathic forms exist). Dermatomyositis (DM) is a subtype characterized by myositis associated with specific or highly suggestive cutaneous findings such as heliotrope rash, Gottron papules, anterior neck and “V-sign” erythema, shawl sign, and periungual abnormalities [2]. Other immune-mediated myopathies include immune-mediated necrotizing myopathy (IMNM), inclusion body myositis, antisynthetase syndrome, and overlap syndromes with other systemic autoimmune diseases [2]. Polymyositis has become increasingly uncommon as a diagnosis due to advances in myositis-specific antibody testing, but remains a consideration for cases not meeting criteria for other subtypes [3].

Cancer-associated myositis (CAM) is clinically defined as the coexistence of idiopathic inflammatory myopathy and a malignancy diagnosed within three years before or after the onset of myositis [4]. This association has been recognized since the first report of polymyositis with gastric cancer in 1916. DM is more strongly associated with malignancy than polymyositis, occurring in up to 13% of DM cases and affecting all ages and sexes [5]. In our case, studies to evaluate systemic autoimmune disease involvement were negative, supporting the diagnosis of CAM.

Cancer incidence peaks within the first year after myositis diagnosis and gradually decreases over the subsequent five years. In some patients, inflammatory myopathy is diagnosed during recurrence of a prior cancer. The malignancies most commonly associated with CAM include adenocarcinomas of the lung, thyroid, breast, stomach, ovaries, cervix, and hepatocellular carcinoma (approximately 70% of cases) [6]. Ovarian cancer is the most common gynecologic malignancy associated with DM and is strongly linked to anti-transcription intermediary factor 1 gamma (TIF1- γ) antibodies [6]. However, the association between endometrial cancer and DM is exceedingly rare.

We identified six previously reported cases of this association, all outside Latin America (Japan, Canada, and the United States) [7-12]. A descriptive observational study from the Mayo Clinic (1952-1982) found 10 cases of paraneoplastic DM (including one case of endometrial cancer), although insufficient details were available for comparison due to the age of the report [12]. Comparison with published cases highlights the wide clinical spectrum of DM associated with endometrial cancer, ranging from amyopathic forms to severe and refractory neuromuscular disease. Our case is notable for its neuromuscular severity, initial refractoriness to conventional immunosuppression, and need for IVIG.

Pathophysiologically, tumor cells and affected muscle cells have been shown to express similar antigens. This suggests that the link between cancer and inflammatory myopathy involves shared autoantigens, leading to immune responses targeting both tumor tissue and skeletal muscle [13].

Risk factors for malignancy in inflammatory myopathies include capillary damage on muscle biopsy, severe cutaneous disease (shawl sign, cutaneous necrosis), Gottron sign, cutaneous leukocytoclastic vasculitis, centropalpebral erythema, older age at onset, treatment resistance, dysphagia, elevated ALT, AST, total CK, and hypoalbuminemia [14]. Interestingly, interstitial lung disease and pruritus appear associated with a lower malignancy risk [15].

Autoantibodies with positive malignancy risk include TIF1- γ (anti-p155, anti-p155/140) and anti-nuclear matrix protein (NXP-2).

Those with negative malignancy risk include antisynthetase antibodies, anti-Mi-2, anti-SRP, and anti-MDA5 (with the last two offering lower protection), as well as systemic autoimmune disease-associated antibodies (anti-RNP, anti-PM-Scl, anti-Ku), although they confer higher risk of ILD in DM [13].

Immunomodulatory treatment in CAM is similar to that in non-cancer myositis [16]. Specific considerations include the need for multidisciplinary care, oncologic coordination, potential interactions between immunosuppressants and antineoplastic agents, and the possible long-term risk of cancer recurrence or secondary neoplasm driven by prolonged immunosuppression [17,18]. Myositis may improve rapidly after complete tumor resection. In one cohort of 43 CAM patients, surgical tumor removal resulted in significant CK and LDH reductions in 24 patients (55.8%) [19], though attributing improvement solely to surgery versus medical therapy is difficult.

Immunosuppressive therapy depends on muscular involvement severity. Systemic immunosuppression is not indicated when muscle involvement is clinically insignificant without CK elevation $>5\times$ normal. For mild-to-moderate disease, glucocorticoids are first-line therapy, typically combined with steroid-sparing agents (DMARDs) due to high relapse risk with glucocorticoid monotherapy. Severe muscle disease (diaphragmatic weakness, dysphagia, cephaloparesis, or inability to perform self-care) warrants IVIG 2 g/kg over 2-5 days (monthly if refractory) plus IV methylprednisolone 250-1000 mg daily for three days, followed by oral prednisone 1 mg/kg/day [20]. Tapering should begin after 4-6 weeks, continuing over 9-12 months, with subsequent gradual tapering of DMARDs after at least one year of steroid-free remission. Treatment monitoring should include clinical strength assessment, CK, and aldolase. Patients with pulmonary involvement require pulmonary function tests every 2-3 months after starting therapy.

Although treatment refractoriness has been proposed as a red flag for occult malignancy in myositis, a recent systematic review and meta-analysis of 69 studies did not support this association [21].

Prognosis is significantly worse in cancer-associated myositis compared with non-cancer DM/PM. In a 25-year retrospective study of 99 patients, CAM survival was 68.2% at 1 year and 31% at 5 years, versus 89.6% and 86.4% respectively in non-CAM cases ($P < 0.001$) [18]. In patients with malignancy, the cancer itself drives overall prognosis.

Conclusion

Paraneoplastic dermatomyositis associated with endometrial cancer represents an exceptionally rare clinical entity that requires a high index of suspicion, especially in patients with atypical muscular symptoms accompanied by gynecologic signs such as metrorrhagia. This case—the first reported in Latin America—demonstrates that early recognition of unusual clinical and paraclinical patterns, combined with thorough oncologic evaluation and intensive multidisciplinary treatment, can reverse severe neuromuscular complications and improve patient outcomes.

Declarations

Ethics approval and consent to participate

Taken

Funding Statement

None

Data Availability

All data available on corresponding author upon responsible request.

Conflict of interest

The authors declare that there is no Conflict interest.

Acknowledgement

Not applicable.

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