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Dermatomyositis as a Paraneoplastic Syndrome: Focus on Ovarian Cancer-Diagnostic and Therapeutic Perspectives

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Abstract

Dermatomyositis (DM) is a chronic autoimmune disease characterized by distinctive skin and muscle inflammation with a well-established association with internal malignancy. This review provides a comprehensive overview of the strong paraneoplastic link between DM and ovarian cancer, which represents the most overrepresented malignancy in affected women. We synthesize the epidemiology and key risk factors, emphasizing the heightened vigilance required for women over 40, a population in whom both conditions frequently cluster. The article details the proposed pathophysiological mechanisms, including autoimmune cross-reactivity and shared antigens, and highlights the utility of emerging biomarkers such as anti-TIF1-γ for cancer risk stratification. A practical, evidence-based approach to cancer screening is outlined, alongside therapeutic considerations that underscore the principle that effective treatment of the underlying cancer often leads to the resolution of DM symptoms. We emphasize the critical role of a multidisciplinary team and conclude with future directions for research, including the use of artificial intelligence in risk prediction and the need for more tailored, prospective studies.

Abbreviations: DM: Dermatomyositis; ILD: Interstitial Lung Disease; IIM: Idiopathic Inflammatory Myositis; PM: Polymyositis; HLA: Human Leukocyte Antigen; TIF1-γ: Transcriptional Intermediary Factor 1-gamma; NXP-2: Nuclear Matrix Protein 2; CCAR1: Cell Cycle and Apoptosis Regulator 1; SAE: Small Ubiquitin-like Modifier Activating Enzyme; CAM: Cancer-Associated Myositis; ADM: Adult Dermatomyositis; JDM: Juvenile Dermatomyositis; US FDA: United States Food and Drug Administration; PARP: Poly ADP Ribose Polymerase; VEGF: Vascular Endothelial Growth Factor; TKIs: Tyrosine Kinase Inhibitors; FRα: Folate Receptor Alpha; IVIG: Intravenous Immunoglobulin; AI: Artificial Intelligence; BRCA: Breast Cancer gene;

Keywords: Dermatomyositis; Ovarian Cancer; Paraneoplastic Syndrome; Autoantibodies; Cancer Screening.

Introduction

Dermatomyositis (DM) is a chronic inflammatory condition affecting both the skin and skeletal muscles and is widely regarded as an autoimmune disorder. Cutaneous manifestations typically include distinctive papules over the digits, erythematous lesions on the elbows and knees, a heliotrope rash around the eyes, periungual telangiectasias, and dystrophic cuticles [1]. Muscle involvement usually manifests as proximal muscle

weakness initially, with or without myalgias or tenderness. An amyopathic variant with minimal to no muscle inflammation has been described [2]. There is a well-established association of DM with an increased risk of internal malignancy [3]. Other important clinical features of DM include the presence of interstitial lung disease (ILD) [4-6]. The disease is an uncommon condition, with an estimated incidence of 0.5–0.89 cases per 100,000 individuals, and shows a female-to-male ratio of approximately 2:1 [7]. A well-recognized association exists between DM and malignancy, with ovarian cancer (13.3–26%) and breast cancer (13.5%) being the most frequently reported malignancies among affected women [8].

The association between dermatomyositis (DM) and malignancy has been well documented across both Caucasian and Asian populations. Population-based cohort studies from Denmark, Sweden, and Finland [9-10] show that DM and, to a lesser extent, polymyositis (PM) is linked to a higher risk of cancers, particularly of the ovary, lung, pancreas, stomach, urinary bladder, and hematologic malignancies such as non-Hodgkin's and Hodgkin's lymphoma [9]. Most are diagnosed within the first year after idiopathic inflammatory myositis (IIM) onset, underscoring the need for comprehensive clinical and laboratory screening soon after diagnosis. This review provides an overview of diagnostic and treatment strategies for cancer-associated dermatomyositis (DM), focusing on its link with ovarian cancer. It highlights the importance of early detection, evidence-based cancer screening, and how malignancy affects treatment decisions, emphasizing a multidisciplinary approach to improve patient outcomes.

Ovarian Cancer in Dermatomyositis: Epidemiology and Risk Factors

Dermatomyositis (DM) demonstrates a strong association with malignancy, with ovarian cancer found in 8.3% of patients with dermatomyositis [10]. This represents a significantly elevated risk compared to the general population, establishing ovarian cancer as the most overrepresented malignancy diagnosed in women with dermatomyositis. The demographic distribution shows a complex pattern where dermatomyositis affects women more frequently than men, with a bimodal age distribution typically showing peaks in childhood and middle age. However, when associated with malignancy, particularly ovarian cancer, the diagnosis tends to cluster in the perimenopausal and postmenopausal periods, aligning with the general epidemiology of ovarian cancer. The incidence of ovarian carcinoma, especially, is higher in patients with dermatomyositis than in the general population, making this association clinically significant for women diagnosed with DM, particularly those over 40 years of age [11].

The temporal relationship between dermatomyositis and ovarian cancer diagnosis is critically important for understanding age-related risks. Cancer is most commonly diagnosed simultaneously with or during the first year after the diagnosis of dermatomyositis, although there continues to be an elevated risk of malignancy even after 5 years [12]. This temporal clustering is particularly relevant as women over 40 face an increased baseline risk of ovarian cancer due to several factors. These include hormonal changes associated with perimenopause and menopause, as well as the accumulation of lifetime ovulatory cycles. Additionally, the clinical effects of genetic predispositions, such as BRCA mutations, typically begin to manifest in this age group [13]. Finally, longer-term environmental exposure to potential carcinogenic factors may also contribute to the elevated risk in this population [14]. This convergence of factors underscores the importance of heightened vigilance for occult malignancy in newly diagnosed dermatomyositis patients within this age group. Given the strong association between dermatomyositis and ovarian cancer, systematic screening approaches become crucial for women diagnosed with DM. Malignancies are usually identified through a history, physical exam, basic labs, and/or age-appropriate screening tests. In women, a transvaginal ultrasound and CA125 may be helpful to identify ovarian cancer.

The relationship between dermatomyositis and ovarian cancer suggests shared genetic susceptibilities, though the specific mechanisms remain incompletely understood [15]. Potential genetic contributors include HLA Associations, such as certain HLA alleles associated with autoimmune conditions may predispose to both DM and malignancy. Also, Immune system dysregulation leads to both autoimmunity and impaired tumor surveillance. Tumor Suppressor Gene Mutations, e.g, P53 mutations found in both DM-associated muscle pathology and ovarian carcinomas, and BRCA1/BRCA2 mutations may contribute to both DNA repair defects and autoimmune phenomena. Anti-p155/140 (anti-TIF1γ) antibodies are strongly associated with malignancy in adult DM. These antibodies may serve as biomarkers for cancer risk stratification [16]. Among environmental contributors, there are viral infections (particularly Epstein-Barr virus and cytomegalovirus) that may trigger both autoimmune responses and oncogenic processes. There also exists molecular mimicry between viral proteins and self-antigens. Occupational and environmental carcinogens may contribute to both inflammatory myopathy and ovarian cancer development. Silica exposure has been associated with autoimmune diseases and certain malignancies. Hormonal factors, including estrogen exposure patterns, may influence both DM development and ovarian cancer risk, and reproductive factors, including nulliparity, late menopause, and hormone replacement therapy.

Screening protocols for ovarian cancer in dermatomyositis emphasize early and repeated evaluation, especially within the first 3 years of disease onset, and should be intensified in the presence of high-risk features such as anti-TIF1γ positivity, older age, and rapid disease onset. CA-125 and transvaginal ultrasound are commonly used, but their sensitivity is limited; cross-sectional imaging (CT or PET/CT) is recommended for higher-risk patients or if initial screening is negative but suspicion remains high. Risk stratification is essential to guide the intensity and frequency of screening. A summary of these screening protocols for ovarian cancer in dermatomyositis is provided in (Table 1).

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Risk Stratification	Screening Modality	Frequency/Timing	Notes	References
All DM patients (esp. women >40, within 3 years of onset)	Pelvic examination, transvaginal ultrasound, CA-125 measurement	At diagnosis, repeat annually for 3–5 years if initial negative	CA-125 sensitivity is limited; combine with imaging for a higher yield	[1-6]
High-risk (older age, anti-TIF1γ+, rapid onset, cutaneous necrosis/vasculitis, dysphagia)	Enhanced imaging (CT abdomen/pelvis or PET/CT), gynecologic evaluation, CA-125	At diagnosis, consider repeat at 6–12 months for 3 years	PET/CT or CT may detect occult malignancy; high-risk features warrant more intensive screening	[1-4, 6-8]
Moderate-risk (no high-risk features, but age >45 or male sex)	Transvaginal ultrasound, CA-125, routine labs, consider CT abdomen/pelvis	At diagnosis, repeat annually for 3 years	Imaging and CA-125 may be considered even if not high risk	[1-6,9]
Low-risk (young, no risk factors, negative serology)	Age-appropriate cancer screening per general population guidelines	As per general population	No additional screening beyond standard recommendations	[1-4,9]
All DM patients	Comprehensive history, physical exam, review of systems	At diagnosis and every follow-up	Vigilance for new symptoms; repeat screening if new risk factors or symptoms emerge	[1-4,8,10]

Pathophysiological Mechanisms Linking DM and Ovarian Cancer

The connection between dermatomyositis (DM) and ovarian cancer is increasingly recognized as a manifestation of paraneoplastic syndromes, in which autoimmune mechanisms are triggered by the presence of a malignancy. One of the primary mechanisms proposed is autoimmune cross-reactivity, wherein the immune system mistakenly identifies antigens expressed by tumor cells as foreign, leading to an immune attack that extends to healthy tissues, including skin and muscle. This process is supported by findings that ovarian tumors can express myositis-specific autoantigens, such as TIF1-γ and NXP-2, which are also targeted in idiopathic dermatomyositis [17]. The immune response generated against these tumor-associated antigens can result in a systemic inflammatory reaction, culminating in muscle inflammation and characteristic skin eruptions. Paraneoplastic inflammation further amplifies this response. Tumor cells in ovarian cancer may release cytokines and chemokines that enhance immune activation and recruit inflammatory cells to muscle tissue, thereby contributing to the myositis phenotype [18]. This inflammatory microenvironment not only promotes tumor progression but also sustains chronic immune-mediated damage to muscle fibers. The release of damage-associated molecular patterns (DAMPs) from necrotic tumor or muscle tissue can act as additional stimuli for dendritic cell activation, perpetuating a cycle of autoimmunity [19].

Several models have been proposed to explain how cancer, particularly ovarian cancer, induces dermatomyositis. The most widely accepted is the "shared antigen hypothesis," suggesting that antigens shared between tumor cells and regenerating muscle cells lead to epitope spreading and sustained immune targeting [20]. Another model involves molecular mimicry, where structural similarities between tumor and muscle antigens result in the activation of autoreactive T-cells [21]. Additionally, the concept of immune checkpoint dysregulation has gained attention, wherein tumors may modulate immune checkpoints like PD-1/PD-L1, disrupting peripheral tolerance and facilitating autoimmune responses seen in paraneoplastic DM [22]. These proposed mechanisms highlight the intricate relationship between oncogenesis and immune dysregulation and underline the importance of recognizing dermatomyositis as a potential early marker for underlying ovarian malignancy.

Autoantibodies and Biomarkers for Cancer Risk Stratification

In the past, cancer-associated myositis (CAM) lacked reliable biomarkers for early detection. Recent discoveries, however, have identified specific autoantibodies that may signal an elevated cancer risk in patients with dermatomyositis (DM), particularly adult-onset cases. Two of the most studied markers are anti-TIF1-γ (previously known as anti-p140/155) and anti-NXP2, both of which are now recognized for their associations with malignancy in inflammatory myopathies [23]. These autoantibodies are thought to be produced in response to tumor-driven disruptions in cellular homeostasis, resulting from the expression of tumor-associated antigens. Their stability in serum and extended half-life make them attractive candidates for use in early detection and prognostication [24].

a. Anti-TIF1-γ (Transcriptional Intermediary Factor 1-gamma) is strongly associated with malignancy in adult-onset DM. Most cases of cancer associated with anti-TIF1-γ antibodies arise within three years before or after the diagnosis of DM, although reports suggest they may precede cancer detection by up to five years [25]. Their clinical utility lies not only in diagnosis but also in prognosis, with pooled sensitivity and specificity rates of 52% and 92%, respectively [26,27]. Notably, higher antibody titers often correlate with more aggressive disease and poor outcomes. Successful treatment of the associated malignancy is frequently followed by a decrease or disappearance of the autoantibody and improvement in myositis symptoms.

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b. Anti–CCAR1 (Cell Cycle and Apoptosis Regulator 1) has recently emerged as a potential co-marker that may refine cancer risk stratification. Studies suggest that while anti-TIF1-γ alone elevates cancer risk two to fourfold, its co-expression with anti–CCAR1 may have a mitigating effect on this risk. The prognostic significance of anti–CCAR1 appears to be cancer-type specific; for example, low anti–CCAR1 expression has been linked to improved survival in liver cancer, while the opposite is observed in ovarian cancer [28].

c. Anti-NXP2 (Nuclear Matrix Protein 2) is another marker found in adult and juvenile DM. It has been associated with features such as calcinosis, facial rash, periorbital swelling, dysphagia, and severe muscular involvement [29]. Evidence regarding its correlation with malignancy is mixed. While some studies support a connection with solid tumors, others found no statistically significant relationship between anti-NXP2 status and cancer incidence [30,31].

d. Anti-SAE (Small Ubiquitin-like Modifier Activating Enzyme) antibodies are primarily found in juvenile DM but have also been detected in adults. Although their presence has been linked to cancer in a few reports, the current body of evidence is insufficient to establish a definitive association. These antibodies have been more frequently observed in patients with adenocarcinomas of the cervix, gastrointestinal tract, and lungs [32].

e. Clinical Implications and Cancer Screening Strategy

Given the known link between dermatomyositis and cancer, early and thorough malignancy screening is essential. In women presenting with DM, ovarian cancer should be considered, and diagnostic evaluation should include serum Ca-125 levels and abdominal imaging [33]. In many cases, treatment of the underlying malignancy results in resolution of dermatomyositis. Recurrence of DM symptoms after remission may suggest tumor relapse.

Cancer Screening Strategies in Women with Dermatomyositis

Cancer is often diagnosed at the same time as or within the first year following a diagnosis of dermatomyositis; however, the risk of developing malignancy remains elevated even beyond five years [34]. The strategy for detecting ovarian cancer in patients with dermatomyositis is based on stratifying the risk of malignancy. Classifying patients into low/moderate and high-risk categories using a combination of factors such as age at DM onset, disease type: adult dermatomyositis (ADM) is more frequently associated with a higher risk of cancer compared to juvenile dermatomyositis (JDM), antibody profile e.g., anti-TIF1-γ, anti-NXP2 linked to high risk of malignancy and specific clinical features [35-37]. For instance, it was observed that malignancy associated with positive anti-TIF1 antibodies occurs exclusively within the three years before or after the onset of dermatomyositis, with the highest risk in individuals aged 39 years or older. This stratification allows for more personalized and effective care, as it supports effective monitoring, guides treatment decisions, and improves prognostic accuracy. Low/moderate risk patients typically undergo basic screening that includes the detection of risk factors, assessment of symptoms, and clinical examination, which may involve a pelvic exam to check for ovarian enlargement or ascites [38]. High-risk patients require a more thorough approach in their evaluations, which may include: pelvic and transvaginal ultrasound, CA-125 blood testing, CT scan/MRI of the abdomen and pelvis. If malignancy is clinically suspected, a whole-body PET scan can be considered; this can guide further investigations like biopsies and targeted treatments [39]. Patients who receive a negative result on their initial cancer screening should continue to be closely monitored.

Guidelines for ovarian cancer surveillance in women with dermatomyositis recommend a comprehensive strategy involving regular pelvic exams, CA-125 blood tests, and transvaginal ultrasounds every 6 to 12 months for at least 2 years after diagnosis. Extending monitoring for up to 5 years may also be beneficial, as the risk of malignancy can persist beyond the initial years [40]. Women diagnosed with dermatomyositis alongside ovarian cancer should undergo genetic testing for BRCA mutations, particularly if there is a family history of ovarian cancer. Genetic testing is an important component in caring for these patients to ensure they have appropriate risk reduction surveillance if they have positive testing [41]. While early detection of ovarian cancer can be challenging, maintaining a high level of clinical suspicion is important, considering all available screening options. When choosing cancer screening methods, it is essential to evaluate factors such as their accuracy, feasibility, and cost-effectiveness. Conventional cancer screening methods like imaging examinations and tumor marker tests have some limitations caused by factors such as medical costs, potential side effects from imaging, and low patient compliance with screening [42]. Tailored treatment plans that may include surgery, chemotherapy, and management of dermatomyositis symptoms play a vital role in monitoring the response of ovarian cancer to treatment and detecting recurrence, placing special emphasis on any deterioration in dermatomyositis symptoms as a possible indicator.

Therapeutic Considerations

Treatment of Ovarian Cancer

The most common form of ovarian cancer is epithelial, which often presents at an advanced stage due to its non-specific symptoms and lack of effective screening methods. The current standard of care is cytoreductive surgery followed by platinum-based chemotherapy [43]. However, the high rate of relapse within two years due to platinum resistance has led to the development of novel therapeutic strategies, including targeted therapy and immunotherapy [44].

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Targeted therapies function by binding to specific molecular markers on cancer cells to inhibit their proliferation and survival. These include PARP inhibitors (olaparib, niraparib, and rucaparib), which promote apoptosis by blocking DNA repair, and VEGF inhibitors like bevacizumab, which starve tumors by preventing new blood vessel growth [45]. Other targeted agents, such as certain tyrosine kinase inhibitors (TKIs) and folate receptor α (FRα)-targeting drugs like mirvetuximab soravtansine, are also being explored. Immunotherapy, a form of personalized medicine, aims to leverage the patient's own immune system to fight the cancer. This can involve cancer vaccines or adoptive cell therapy, where a patient's immune cells are modified and reinfused to promote a robust anti-tumor response.

Prognostic effects of ovarian cancer treatment on dermatomyositis

While treating the underlying malignancy is crucial for long-term remission of dermatomyositis (DM), acute symptom flare-ups can occur after treatment initiation [46]. This phenomenon, sometimes likened to a "tumor lysis-like syndrome," is hypothesized to be caused by a massive release of tumor antigens that trigger an acute exacerbation of the autoimmune response [47]. The risk of this reaction may be particularly high in patients with a *BRCA* gene mutation [48]. Therefore, a high index of suspicion for DM symptoms (rash, fatigue, and muscle weakness) should be maintained when initiating treatment for ovarian cancer [49,50].

Treatment of dermatomyositis

The management of dermatomyositis (DM) as a paraneoplastic syndrome is fundamentally different from its idiopathic counterpart, as the most effective long-term treatment is to address the underlying malignancy. While immunosuppressive therapies, such as corticosteroids and intravenous immunoglobulin (IVIG), are used to control the acute inflammatory symptoms of DM, they are often insufficient or ineffective until the source of the paraneoplastic process is removed or controlled. Therefore, the therapeutic strategy is centered on a coordinated, multidisciplinary approach that prioritizes the treatment of ovarian cancer with standard-of-care oncologic interventions like surgery and chemotherapy [51-53]. In many cases, effective treatment of the malignancy leads to a rapid and complete remission of the dermatomyositis symptoms, underscoring the direct causal link between the two conditions.

Multidisciplinary Approach

The successful diagnosis and management of dermatomyositis (DM) as a paraneoplastic syndrome for ovarian cancer requires a highly coordinated, multidisciplinary approach. This collaborative model brings together specialized expertise from rheumatology, dermatology, oncology, and gynecology to ensure comprehensive patient care. The rheumatologist and dermatologist are often the first to diagnose the myositis and cutaneous manifestations of DM, initiating the search for an underlying malignancy. They then work closely with gynecologic oncologists to guide diagnostic work-up and treatment of the cancer. This collaborative framework is essential for managing the complexities of both the autoimmune and oncological diseases, as treating the cancer often leads to remission of the DM symptoms [54].

The patient is at the center of this model, and personalized care and shared decision-making are crucial to its success. Given the high stress and physical burden of a dual diagnosis, clinicians must educate patients on the diagnostic process, treatment options, and long-term prognosis, empowering them to make informed choices. This includes a candid discussion of the benefits and risks of aggressive cancer screening and treatment, balanced with the management of debilitating DM symptoms. Formal tumor boards and specialized autoimmune-neoplasia clinics play a vital role in this collaborative effort. These forums allow experts from different disciplines to review complex cases, share insights, and develop an integrated treatment plan that addresses both the dermatomyositis and the ovarian cancer simultaneously [55]. These specialized clinics, although rare, represent a best-practice model by streamlining the patient's journey, reducing diagnostic delays, and ensuring that all aspects of care are aligned.

Future Directions

The field of dermatomyositis as a paraneoplastic syndrome is poised for significant advancements driven by a deeper understanding of its molecular basis. Research is focused on identifying emerging biomarkers and genomic predictors that can more accurately predict an individual patient's risk of malignancy [56]. The presence of specific autoantibodies, such as anti-TIF1-γ, already serves as a strong indicator, but future research aims to uncover more nuanced serological and genomic markers that could facilitate earlier and more precise cancer screening.

The integration of artificial intelligence (AI) offers a promising path toward personalized cancer risk prediction in DM. AI models could analyze vast clinical datasets, including a patient's demographics, symptoms, laboratory findings, and genetic information, to generate a dynamic risk score. This would allow clinicians to tailor cancer screening protocols to each patient's unique profile, avoiding unnecessary procedures for low-risk individuals while ensuring high-risk patients receive timely and thorough evaluations [57].

A major need in the field is for more prospective studies and cancer screening guidelines specifically tailored to dermatomyositis patients. Most current evidence is derived from retrospective studies and case reports, which limits our ability to establish clear, evidence-based guidelines. Future prospective cohort studies will be essential to validate new biomarkers and imaging modalities, ultimately leading to formalized screening recommendations that optimize the balance between early cancer detection and patient burden [58]. This will be a critical step toward standardizing care and improving patient outcomes on a global scale.

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Conclusion

The relationship between dermatomyositis and ovarian cancer is a critical example of a paraneoplastic syndrome that necessitates a high degree of clinical suspicion. This review has synthesized the current evidence, demonstrating that DM can serve as an early warning sign of an underlying malignancy. The strong temporal and demographic associations, combined with the presence of specific autoantibodies, provide a clear framework for identifying at-risk patients. While DM symptoms can be managed with immunosuppressive therapies, a durable remission is most often achieved through effective oncologic treatment of the ovarian cancer. The complexities of this dual diagnosis underscore the importance of a coordinated, multidisciplinary approach involving rheumatology, dermatology, gynecology, and oncology. As research continues to advance with emerging biomarkers and technologies, future efforts should focus on developing personalized risk-stratification tools and prospective screening guidelines to optimize patient care and improve long-term outcomes.

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