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CASE REPORT

A Rare Case Report of Dermatomyositis Accompanied by Blisters

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Abstract: We report a patient with dermatomyositis who developed blisters. The patient was a female, 51 years old. She came to our hospital because of edematous purplish erythema on the face, neck, trunk, and extremities that itched for 1 month. Histopathology of the lesions showed: squamous epithelial tissue, hyperkeratosis of the epidermis, mild thickening of the stratum spinosum, liquefaction and degeneration of the basal cells, perivascular lymphocytes in the dermis, plasma cell infiltration; myofibers of varying thicknesses, disappearance of transverse striations, and lymphocytic infiltration of interstitial muscles. Diagnosis: dermatomyositis. Water blisters appeared on the skin lesions of the patient with dermatomyositis. Given that the probability of dermatomyositis being accompanied by a tumor is approximately 10% - 20%, this is considered a serious condition by modern medicine. Therefore, we conducted a series of examinations, including immunohistochemistry, to determine the source of the blisters.

Keywords: dermatomyositis, blister, tumour

Introduction

Dermatomyositis (DM) is an inflammatory myopathy characterized by symmetrical proximal extensor muscle inflammation in the limbs. Specific skin lesions include Heliotrope sign and Gottron papules, and the probability of concurrent malignant tumors is relatively high.[1] The former is more commonly seen as purplish red patches on the eyelids and periorbital skin that may be accompanied by edema, while the latter mainly presents as papules at the swollen areas of the finger joints followed by lichenoid changes. The skin lesions of DM patients often show purple-red skin heterochromia-like changes, accompanied by severe itching and nail plate nutritional deficiency.[2] In addition, DM can also present with centripetal lancinating erythema, skin erosion and ulcers, etc., and may only have skin manifestations without muscle symptoms.

The main pathological manifestations of dermatomyositis on the skin include vacuolar degeneration of the basal cell layer of the epidermis, inflammatory infiltration in the dermis, and interface dermatitis. The main pathological manifestations of the muscles include degeneration and necrosis of muscle fibers, inflammatory cell infiltration, atrophy around muscle bundles, regeneration and fibrosis, and vascular pathology may present with microvascular damage, complement deposition, and calcium deposition.[3] Dermatomyositis is often accompanied by complications in other systems[4] For instance, if it affects the lungs, pulmonary fibrosis or pleurisy may occur; if it affects the heart, arrhythmia or conduction blockage may happen. If the striated muscles in the pharynx and the upper part of the esophagus are affected, it can cause difficulty in swallowing, choking when drinking water, nasal reflux, etc. The dilation of the lower part of the esophagus and the weakened peristalsis of the small intestine can lead to acid reflux, esophagitis, difficulty in swallowing, upper abdominal distension and pain, and absorption disorders. Moreover, it can also affect the kidneys, causing kidney failure. Modern medicine mainly employs therapies such as glucocorticoids and immunosuppressants for the treatment of dermatomyositisIn recent years, the application of biological agents has become quite widespread.[5]

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However, in clinical practice, patients with dermatomyositis presenting with blisters are extremely rare. Recently, our department admitted a patient with blistering dermatomyositis, and the following report is presented:

Case Report

Medical History

Female patient, 51 years old. One month ago, red patches and papules appeared on the face and neck. The patient felt itching and had difficulty in swallowing and breathing. Later, the number of rashes increased and the area expanded. The local traditional Chinese medicine prescribed oral administration of traditional Chinese medicine decoction and topical application of ointment (the specific ingredients are unknown). Later, the rashes worsened, involving the face, chest, abdomen, back, as well as causing weakness in the limbs and muscle pain. The patient experienced symptoms of difficulty in swallowing five days before the visit. She was admitted to the hospital for treatment on May 8, 2022. She also had thirst, restlessness, poor sleep, yellow urine, and dry stool. There was no fever, no cough or expectoration, no chest tightness or chest pain, no abdominal pain or diarrhea, no weight loss, and no history of contact with known allergens.She had a 20-year history of coronary heart disease and denied other medical history.

Physical Examination

The patient has tenderness in the deltoid muscle of the upper arm, weakness in the muscles of the limbs, with muscle strength at grade 4, weakened. The patient's tongue muscles show no tenderness. No oral ulcers are present on the patient's oral mucosa. The patient's elbow and knee joints exhibit redness and swelling, with no deformity. No abnormalities were found in the superficial lymph nodes throughout the body. No other abnormalities were observed. Dermatological examination: Facial area shows edematous purple-red patches centered on the eyelids, merging into patches (Figure 1A), diffuse edematous purple-red patches on the upper part of the front chest and abdomen, the rash is slightly thick, distributed symmetrically on the limbs, covered with fine and silvery-white scales (Figure 1B). Point-like pigmentation and hypopigmentation spots can be seen on the skin in the posterior neck and posterior lumbar region (Figure 1C), with skin hypopigmentation. Edematous purple-red patches are seen on both sides of the lower arms near the wrist creases, with many scattered water blisters, the larger ones reaching 0.4cm * 0.8cm in size (Figure 2), the blister fluid is clear, the blister wall is loose, and Nikolsky's sign is negative.

Laboratory Examination

Pemphigus antibody test results: Anti-BP180 antibody IgG determination (BP180) 36.6 RU/ML ↑, all other results negative. Chest CT shows a small amount of chronic inflammation in the right lung, small nodules in the left main bronchus, small micro-nodules in the lower lobe of the left lung, decreased liver density, tuberculosis infection T-cell spot test: (+), tuberculosis antibacterial antibody (-). Creatine kinase 728 U/L ↑, creatine kinase-MB isoenzyme 38 U/L ↑, lactate dehydrogenase 421 U/L ↑, α-hydroxybutyrate dehydrogenase 317 U/L ↑. Anti-nuclear antibody (ANA) positive (1:320), immunoglobulin G (IgG) 16.3 g/L ↑, troponin T 82.76 ng/L ↑, B-type natriuretic peptide precursor 81 pg/mL ↑.

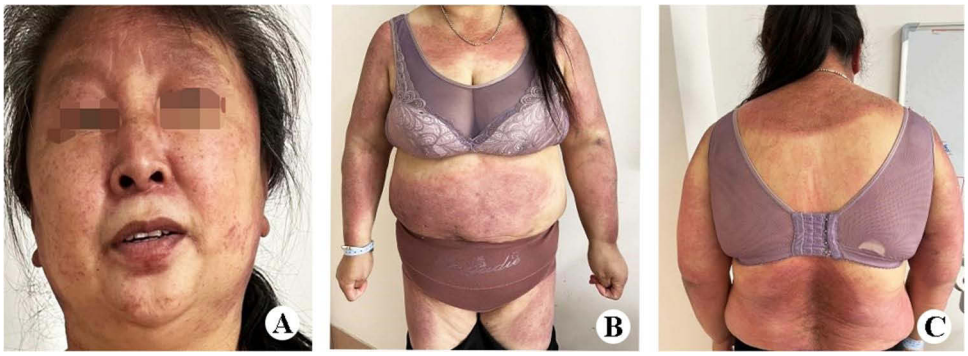

Figure 1 (**A**) shows a generalized edematous purplish rash centered on the eyelid; (**B**) presents a dense distribution of edematous purplish rashes on the front chest, abdomen, and limbs, accompanied by desquamation; (**C**) shows a confluent mass of dense edematous purplish rashes on the back of the neck and waist, with heterochromic changes, slightly swollen skin lesions, infiltration, and mild desquamation.

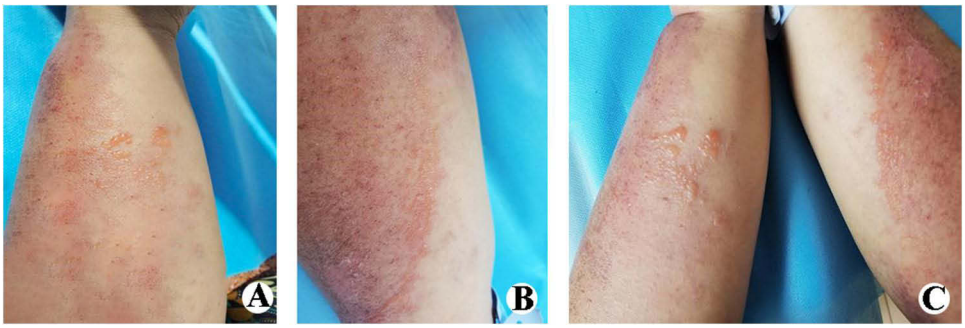

Figure 2 (**A**) shows scattered blisters on the left forearm; (**B**) shows scattered blisters along the lesion border on the right forearm; (**C**) shows blisters on both forearms, with clear fluid and thick walls.

Alanine aminotransferase (ALT) 44 U/L ↑, aspartate aminotransferase (AST) 91 U/L ↑, alkaline phosphatase (ALP) 27 U/L ↓, total protein (ALB) 62.2 g/L ↓, albumin (ALB) 32.9 g/L ↓, triglycerides (TG) 3.61 mmol/L ↑, alpha-fetoprotein, carcinoembryonic antigen and other test results showed no abnormalities. Muscle biopsy suggests uneven muscle fiber size, disappearance of striations, lymphocyte infiltration between muscle fibers (Figure 3). Skin pathology of the forearm shows squamous epithelial tissue, overcorrosion on the surface, mild thickening of the spinous layer, liquefaction and degeneration of basal cells, lymphocytes and plasma cells infiltration in the dermis (Figure 4). Based on the patient's skin lesions, subjective symptoms, test results and histopathological results, the diagnosis is: dermatomyositis.

Treatment

Administer oral desloratadine citrate tablets at a dose of 8.8 mg each time, once daily. Administer oral triamcinolone acetonide tablets at a dose of 4 mg each time, once daily. Apply topical fluticasone propionate cream and zinc oxide borax ointment alternately to the affected skin areas, avoiding the blisters. Wet the blistered areas with compound Phellodendron liquid and apply it. All the above treatments were carried out simultaneously for a total of 21 days. The patient's blisters disappeared, the color of the dermatomyositis skin lesions became lighter, and muscle strength slightly recovered.

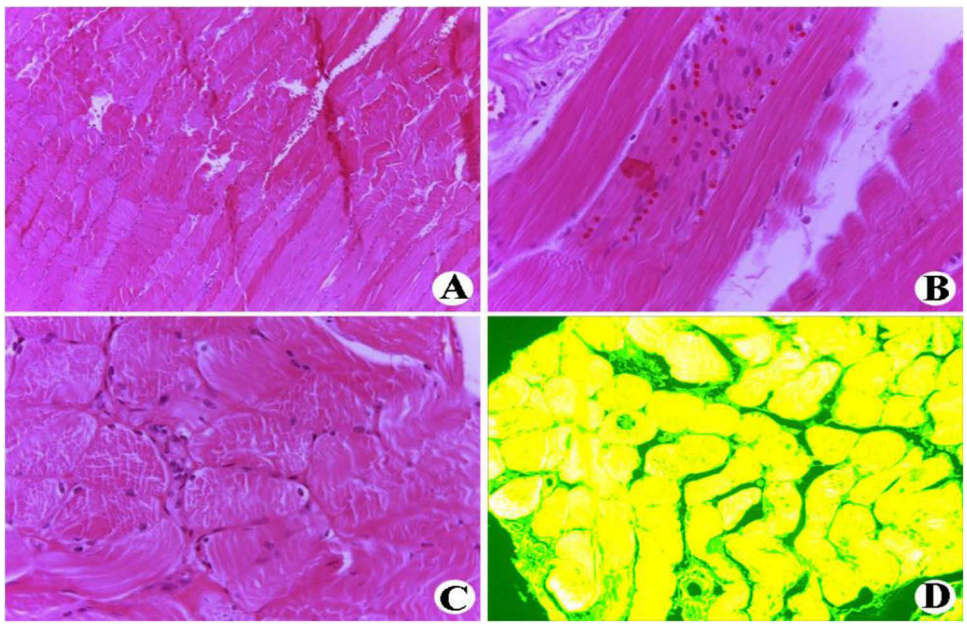

Figure 3 HE staining ×100: Obvious uneven thickness of muscle fibers, disappearance of striations, and lymphocyte infiltration can be seen between the muscles (**A**); HE staining ×400: In addition to A, the number of muscle cell nuclei increases (**B** and **C**); HE ×400: Direct immunofluorescence negative staining (**D**).

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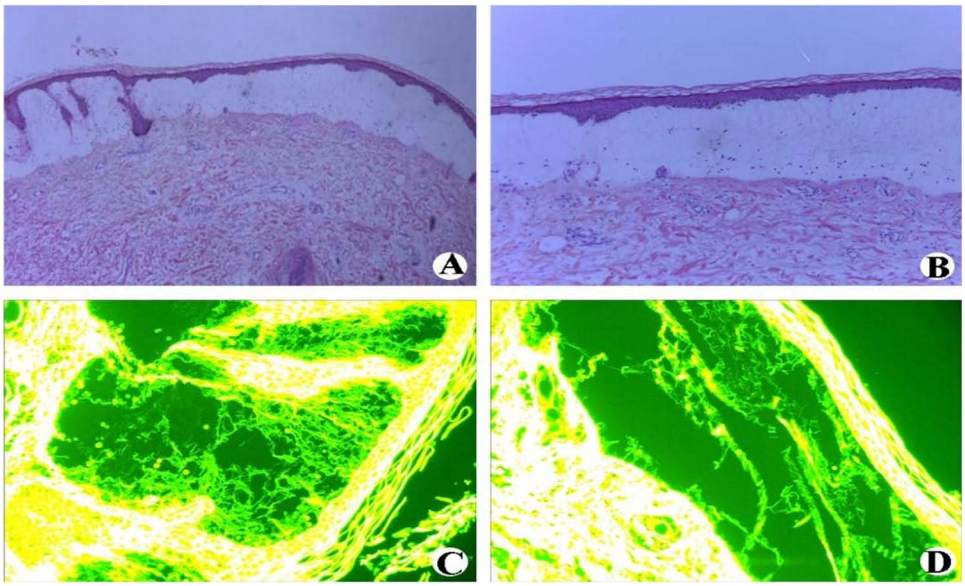

Figure 4 HE×40 (**A**), HE×100 (**B**): There are blisters under the epidermis, squamous epithelium, excessive surface keratinization, slight thickening of the spinous layer, liquefaction and degeneration of basal cells, and infiltration of lymphocytes and plasma cells around the dermal blood vessels. There are a few red blood cells in the blisters; HE×100: Direct immunofluorescence is negative, no fluorescence deposition is observed in the intercellular and basement membrane regions (**C** and **D**), and the flocculent substances are the contents of the blisters.

Discussion

DM is a type of inflammatory myopathy characterized by symmetrical proximal extensor muscles of the limbs. The occurrence of blisters in DM is extremely rare in clinical practice. The first report of DM with blistering skin lesions was made in 1903.[6] Modern medicine believes that the mechanism of blister formation in DM is not yet clear. Most experts believe that the blisters are subepidermal blisters, and their formation is related to factors such as subepidermal edema, mucin deposition, and cell-mediated epithelial cell damage.[7–9] In this case, when the patient came to our hospital, blisters had already appeared on the forearm. As a dermatologist, I first differentiated it from bullous diseases. It was necessary to first confirm whether the blisters were associated with dermatomyositis or were independent skin lesions. Immunofluorescence is of great significance for the diagnosis of such diseases. For DM skin lesions, immunofluorescence examination shows that in the muscle tissue, granular or linear fluorescent substances can be seen in the myofibril, and in the skin lesion, granular or linear fluorescent substances can be seen in the basement membrane.[10] Autoimmune bullous diseases such as pemphigus and bullous pemphigoid show IgG deposition, C3 deposition, and sometimes IgA and IgM deposition in the immune fluorescence.[11]

Two intact masses of non-organized tissues were completely removed from the right deltoid muscle of the patient. Under He staining, it was observed that the muscle fibers were uneven in thickness, the number of muscle cell nuclei increased, the striations disappeared, and lymphocytes were infiltrating between the muscle cells. Direct immunofluorescence showed negative results. This supported the diagnosis of dermatomyositis. A spindle-shaped skin tissue sample was taken from the blister on the right forearm. Under He staining, subepidermal blisters were visible, thus ruling out pemphigus. Squamous epithelial tissue with excessive surface keratinization, mild thickening of the spinous layer, liquefactive degeneration of basal cells, lymphocyte and plasma cell infiltration around blood vessels in the dermis, a few red blood cells in the blister, and direct immunofluorescence showed negative results. No fluorescence deposition was observed between cells and the basement membrane zone.

Bullous pemphigoid (BP)[12] presents clinically as tense blisters and large blisters on normal-looking skin, erythema or urticarial plaques. Nikolsky's sign is negative. Pathologically, there are subepidermal blisters or fissures, with eosinophils and neutrophils infiltrating the superficial dermis and the blister fluid. Direct immunofluorescence shows linear deposition of IgG and C3 in the basement membrane zone, and a few IgA and IgE linear deposits. ELISA detects increased levels of pathogenic anti-BP180 and anti-BP230 antibodies in the serum. Linear IgA bullous dermatosis[13] is an

autoimmune skin disease characterized by peripheral blistering ring or semi-ring-shaped skin lesions, resembling a "pearl string" pattern. The histopathology is not specific, and the immunopathology shows linear deposition of IgA in the basement membrane zone. Therefore, bullous pemphigoid and linear IgA bullous dermatosis, which are blistering and bulla-forming diseases, are excluded. The diagnosis of dermatomyositis is supported, and the blisters are confirmed to be formed as a complication of dermatomyositis, mainly due to high epidermal edema and excessive epidermal tension causing subepidermal blisters. In addition, it should be differentiated from contact dermatitis. Contact dermatitis can also produce erythematous blisters. Carefully inquire about whether the location of the blisters has ever been exposed to irritants, and carefully inquire about the medication history of external drugs to rule out contact dermatitis.

Dermatomyositis is characterized by microvascular injury and capillary depletion in both muscle and skin. Studies suggest that interactions between VEGFA and its receptors (VEGFR-1, VEGFR-2, VEGFR-3), as well as with ADAMTS1 and neuropilin-1 (NRP1), may be involved in dermal microvascular remodeling in inflammatory diseases. Blister formation in dermatomyositis may result from increased vascular permeability due to endothelial dysfunction. Tissue hypoxia stimulates vascular morphogenesis; similarly, in dermatomyositis, inflammation and vascular damage may induce local hypoxia, thereby triggering a VEGF-driven response that could influence the dynamics of inflammation and blister formation.[14]

It is reported that approximately 8% of DM patients develop or progress to cancer, especially within one year after being diagnosed with dermatomyositis, and the risk of developing malignant tumors significantly increases.[10] Among adult DM patients, the risk of developing malignant tumors is 4.66 times that of the general population. Among middle-aged and elderly patients with dermatomyositis, about 6% to 60% have concurrent malignant tumors, and the probability of middle-aged and elderly women with vesicular DM developing malignant tumors is approximately twice that of men.[10] Patients with dermatomyositis who develop vesicles are highly prone to concurrent interstitial pneumonia and malignant tumors.[15] Therefore, most scholars at home and abroad believe that the prognosis of vesicular DM is poor and complete and detailed screening as well as close observation and follow-up are necessary. Therefore, for dermatomyositis patients in clinical practice, early diagnosis and treatment should be carried out as soon as possible. For patients with vesicular skin lesions, comprehensive examinations are necessary to rule out malignant tumors and interstitial pneumonia. This is very important.

This patient presented to the hospital with coronary heart disease and denied any other chronic diseases. Post-admission lung CT showed a small amount of chronic inflammation in the right lung, a small nodule in the left main bronchus, small micro-nodules in the lower lobe of the left lung, decreased liver density, a positive T-cell spot test for tuberculosis infection, which was consistent with a diagnosis of pulmonary tuberculosis. There was a small amount of inflammation. Subsequent follow-up should be closely monitored. The possibility of concurrent interstitial pneumonia cannot be ruled out. The risk of active pulmonary tuberculosis in DM patients is still unclear. Currently, there are only a few case series reports on the risk of active tuberculosis in DM patients at home and abroad, but studies have shown that the risk of active tuberculosis in DM patients is significantly higher than that in non-DM patients.[16] The most important risk factors for developing active tuberculosis include male gender, DM comorbidity, and the use of corticosteroids and azathioprine in DM patients.[17] In conclusion, the risk of active tuberculosis in DM patients is higher. The patient has been treated for half a month, and the blisters have completely subsided, the edematous purplish-red patches have largely disappeared, muscle tone has decreased, muscle strength in the limbs has partially recovered, and the patient has been discharged. Due to the relatively high probability of dermatomyositis combined with malignant tumors when blisters occur, the patient is advised to undergo comprehensive examinations regularly after discharge. Currently, this patient is under follow-up.

Conclusion

Dermatomyositis with blistering has a higher probability of being associated with tumors. Dermatologists encountering patients with similar clinical presentations should conduct thorough examinations to confirm the source of the blisters. Concurrently, comprehensive auxiliary tests should be performed, including tumor markers, cardiac, renal, gastrointestinal, and respiratory system evaluations, to prevent missed or misdiagnosis that could delay treatment. Finally, symptomatic treatment should be administered as part of a comprehensive management approach. Due to the unique nature of this condition, regular follow-up is essential. Following discharge, the patient continued oral medication therapy. Due to financial constraints and personal circumstances, the patient did not return for follow-up appointments. Upon telephone

contact, the patient reported complete resolution of skin lesions six months post-discharge, though muscle strength had not yet returned to normal levels. The patient remains under ongoing follow-up.

Ethical Informed Consent Statement

Written informed consent has been provided by the patient to have the case details and any accompanying images published. This case has been approved by the Shenyang Seventh People's Hospital and can be made public.

Disclosure

Tianming Ma and Xiaoqing Xiang are co-first authors, they have contributed equally to this work. The authors report no conflicts of interest in this work.

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