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Muscle weakness with a hidden cause

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SUMMARY

Idiopathic inflammatory myopathies (IIM) represent a diverse group of systemic autoimmune disorders characterised by skeletal muscle weakness with variable clinical manifestations.

Dermatomyositis (DM) is a heterogeneous IIM and is associated with progressive muscle weakness, with characteristic skin features, and with an increased risk of malignancy. Here, we report a previously healthy man in his early 40s who presented to the emergency department with a 14-day history of symmetric progressive proximal muscle weakness. Both upper arms were painful and swollen, and they displayed marked weakness in arm elevation. Climbing stairs was no longer possible due to muscle weakness and pain. A facial and truncal rash as well as periungual erythema were noted. Laboratory analysis revealed highly elevated creatine kinase. Based on these findings, the diagnosis of DM was confirmed. A comprehensive search for malignancy revealed a non-palpable pure seminoma. While DM as a paraneoplastic phenomenon of seminoma is extremely rare, our case underlines the importance of a thorough tumour search.

BACKGROUND

The association of idiopathic inflammatory myopathies (IIM), particularly dermatomyositis (DM), involves an increased risk of an underlying malignancy.¹ Meta-analyses have demonstrated a standardised incidence ratio (SIR) of 4.79² and 4.66,³ respectively, for overall cancer in patients with DM. The International Myositis Assessment and Clinical Studies Group (IMACS) has published evidence-based and consensus-based recommendations for tumour screening in IIM patients.⁴ Screening mode and frequency are based on an evaluation of autoantibody profiles, clinical features and IIM subtypes. Clinical risk factors for malignancy include male gender, dysphagia, acute onset, relapse despite immunosuppressive treatment or refractory myositis, as well as cutaneous ulcerations and/or necrosis.^{4,5} Serological risk factors are also well-established. Specifically, two myositis-specific antibodies (MSA)—anti-transcriptional intermediary factor 1 (anti-TIF1) and anti-nuclear matrix protein-2 antibodies (NXP-2)—show a strong association with paraneoplastic DM.^{6,7}

In cases of clinically suspected IIM, testing with a panel of MSA may provide guidance for estimation of type and risk of malignancy. Positive MSAs are found in many patients with IIM and allow an association with specific clinical phenotypes and dermatological findings, an estimate of the severity of myositis as well as a risk assessment for

the development of interstitial lung disease (ILD) and even overall mortality.^{8–10} However, DM can present as seronegative as well (table 1).

DM is often associated with adenocarcinomas and squamous cell carcinomas. These primarily include ovarian, lung and colorectal carcinoma, oropharyngeal and cervical cancer, malignant melanoma, non-melanoma skin cancer and lymphatic haematopoietic cancer.¹¹ The incidence of germ cell tumours and, even more so, of pure seminomas in association with DM appears to be very low,¹² as only a few cases have been reported so far.^{13,14}

CASE PRESENTATION

A previously healthy patient in his early 40s presented to the emergency department with symmetric progressive myalgia and weakness, primarily affecting the proximal limb muscles. Prior to the illness, he was in good general health, engaged in regular physical activity, and had no history of smoking, alcohol consumption, or use of any medications or illicit drugs. Symptom onset occurred over a 2-week period, beginning with rapidly progressive muscle pain and weakness, followed by fatigue and night sweats. Symptomatic treatment with nonsteroidal anti-inflammatory drugs (NSAIDs), initiated under the suspicion of a viral infection, did not alleviate the muscle pain. At presentation, the patient was no longer able to rise independently from a sitting to a standing position or walk on flat ground.

Notable findings on examination included circumferential swelling of the upper arms and a distinctive rash on the right inguinal side. Vital signs were within normal limits. During the clinical examination, there was a marked reduction in muscle strength, with abnormal objective muscle strength testing (Medical Research Council (MRC) scale grade of 2/5) in the shoulder girdle muscles, particularly affecting the deltoids. Strength was preserved in the distal muscles of both the upper and lower extremities, and sensory function was intact.

The patient presented with swelling on both eyelids as well as periorbital oedema with a concomitant erythematous rash over the nasal bridge and eyelids. Both upper arms showed diffuse subcutaneous swelling. Gottron's papules on the right metacarpophalangeal joints, as well as periungual erythema with small haemorrhages, were noted. On the right inguinal side, a poorly demarcated, papulo-erythematous skin lesion extending to the dermatomal border was observed (figure 1).

Laboratory testing revealed elevated creatine kinase (CK 10 226 U/L; upper limit of normal



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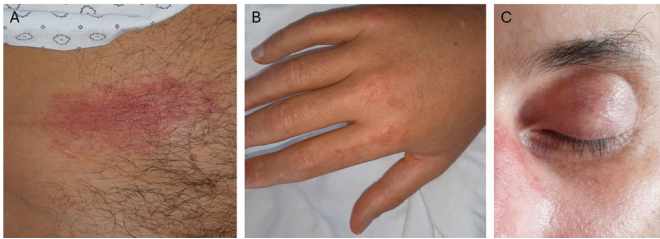


Figure 1 Clinical image at the time of initial presentation. (A) Erythematous and poorly demarcated rash located on the right inguinal region. It shows subtle papule and plaque formation and discrete scaling. (B) Subtle presence of Gottron’s papules over the metacarpophalangeal joints. (C) Mild periorbital oedema with erythema over the nasal bridge.

(ULN) 170 U/L), elevated lactate dehydrogenase (LDH 766 U/L; ULN 240 U/L), and transaminases (aspartate aminotransferase (AST) 433 U/L; ULN 50 U/L and alanine aminotransferase (ALT) 158 U/L; ULN 45 U/L). CRP (11 mg/L; ULN 5 mg/L) and an erythrocyte sedimentation rate (ESR 30 mm/h; ULN 8 mm/h) were slightly elevated, while leucocyte counts were normal. A mildly elevated high-sensitivity (hs) troponin T level (80 ng/L; ULN 14 ng/L) did not increase in a 1-hour and 3-hour follow-up. Echocardiography and ECG were unremarkable, and the patient had no signs of acute coronary syndrome. We therefore did not suspect heart disease, especially since active muscle diseases can be associated with elevated hs troponin T levels. Nevertheless,

we chose to closely monitor his troponin T levels but did not detect further elevations in the subsequent follow-up period. Based on these clinical and laboratory findings, a diagnosis of DM was suspected. A subsequent screen for myositis-specific autoantibodies (MSA) was positive for anti-NXP-2 antibodies. Furthermore, an MRI of the right shoulder and plexus was remarkable for ubiquitous subcutaneous and muscle oedema. An MRI-correlated biopsy of the right deltoid muscle revealed T-lymphocytic myositis with accompanying fasciitis and perifasciitis. The cellular infiltrate particularly consisted of CD3-positive lymphocytes and CD68-positive macrophages, compatible with a mixed inflammatory infiltrate in DM (figure 2). Initial serological results (ANA titres, ANCA) remained negative. Intravenous methyl-prednisolone (500 mg for 4 days) was initiated as a first-line treatment, followed by prednisone (60 mg/day with gradual reduction), in combination with azathioprine (75 mg/day). This treatment regimen initially led to a favourable clinical response, characterised by a rapid reduction in myalgias and CK levels and by improving muscle strength. At the initiation of immunosuppression, we performed thiopurine methyltransferase (TPMT) activity testing, which is relevant for azathioprine dose adjustment. A heterozygous mutation was detected, necessitating a dose reduction of 50% for azathioprine. After a thorough clinical examination, which was unremarkable regarding a potential malignancy, a CT of the thorax and abdomen was performed. It revealed two polypoid-tubular, sharply demarcated masses in the retroperitoneum inferior to

Table 1 IIM: frequency of disease-specific autoantibodies (abs) and risk of associated malignancy, according to ^{9 10} and ²¹			
	Frequency in adults with IIM	Risk of malignancy	Mainly clinical manifestation
Anti-synthetase-syndrome	Antibody detection mandatory		
Anti-Jo1 abs	~20%		~70% to 96% ILD, 94% arthritis
Anti-PL7 abs	<5%		~100% ILD
Anti-PL12 abs	<5%		~90% to 100% ILD, MCTD, arthritis
Anti-EJ, anti-OJ, anti-KS, anti-ZO, anti-HA abs	<5%		ILD and severe muscle weakness
Dermatomyositis	~50% seronegative		
Anti-Mi2 abs	~5% to 10%	Reports vary	Skin manifestation
Anti-MDA-5 abs	<5%	No known association	ILD, unfavourable prognosis
Anti-SAE1/2 abs	<5%	Reports vary	Skin manifestation
Anti-NXP-2 abs	<5%	~70%	Acute and severe muscle weakness, high CK-values, oedema, kidney failure, calcinosis
AntiTIF1γ abs	~20%	~50% to 75%	Skin manifestation, rarely ILD
necrotising myopathy			
Anti-SRP abs	~20% to 30%	No known association	~30% ILD, severe weakness, high CK values, carditis
Anti-HMGCR abs	~10% to 20%	Reports vary	~60% previous statin-exposure
IBM			
Anti-cN1A abs	~30% in IBM	No known association	40% of inclusion body myositis
Overlap disease			
Anti-PM/Scl abs	~8%	No known association	Polymyositis-scleroderma overlap
Anti-U1RNP abs	~10%	No known association	Association with MCTD
Anti-Ro52 abs	~25%	No known association	Association with SLE, Sjögren syndrome, SSc, MCTD
Anti-Ku abs	<1%	No known association	Polymyositis-scleroderma overlap
anti-CN1A, cytosolic 5'-nucleotidase 1A; anti-Jo-1, histidyl-tRNA-synthetase; anti-Ku, Ku autoantigen, DNA repair complex; anti-MDA-5, melanoma differentiation-associated protein 5; anti-Mi-2, chromatin-remodelling complex; anti-NXP-2, nuclear matrix protein-2; anti-PL-7, threonyl-tRNA-synthetase; anti-PL-12, alanyl-tRNA-synthetase; anti-PM/Scl, polymyositis/scleroderma complex; anti-Ro52, also known as TRIM21; anti-SAE1/2, small ubiquitin-like modifier-activating enzyme; anti-SRP, signal recognition particle; anti-TIF-1γ, transcription intermediary factor 1-gamma; anti-U1RNP, U1 ribonucleoprotein; IBM, inclusion body myositis; IIM, idiopathic inflammatory myopathies; ILD, interstitial lung disease; MCTD, mixed connective tissue disease; SLE, systemic lupus erythematosus; SSc, systemic sclerosis.			

the renal hilum. Subsequently, a CT-guided biopsy of one of the retroperitoneal masses resulted in the diagnosis of a metastatic malignant germ cell tumour (figure 3). The profile with negative markers of alpha-fetoprotein (AFP < 1.0U/L) and beta-human chorionic gonadotropin (beta-HCG < 10µg/L) was consistent with a pure seminoma, tumour stage IIIB (cTx cN2 cM0 S2). The patient did not complain about local urogenital symptoms, and no testicular induration or swelling was palpable. However, an additional ultrasound showed an oval, hypoechoic mass in the left testis. Ten days after the initial presentation, intended chemotherapy following the platinum, etoposide and bleomycin (PEB) protocol for three cycles was initiated, followed by orchiectomy and contralateral testicular biopsy.

During the first cycle of chemotherapy, DM with immobilising myalgias and rising CK levels flared up, necessitating a temporary administration of intravenous immunoglobulins (IVIGs). The patient also developed new-onset dysphagia, which had not been present previously. Due to a favourable clinical course with a declining CK level and improving muscle weakness, glucocorticoids were tapered and eventually stopped after the third cycle of chemotherapy. Azathioprine was discontinued previously due to leucopenia.

OUTCOME AND FOLLOW-UP

Two months after completion of curative chemotherapy, fluorine-18 fluorodeoxyglucose positron emission tomography/computed tomography (18F-FDG-PET/CT) imaging showed an overall good response, characterised by regression of the retroperitoneal lymph node metastases with only minimal residual FDG avidity. No FDG uptake was detected in the previously MRI-abnormal deltoid muscle. LDH, beta-HCG and AFP levels were within the normal range. The serum parameters CK, LDH, AST and ALT were normalised. The patient’s overall status improved significantly, allowing him to return to normal daily and work-related activities. After 1 year of follow-up, no increase in tumour markers or suspicious lymph nodes on CT were detected. The patient remained asymptomatic, with no clinical evidence of DM

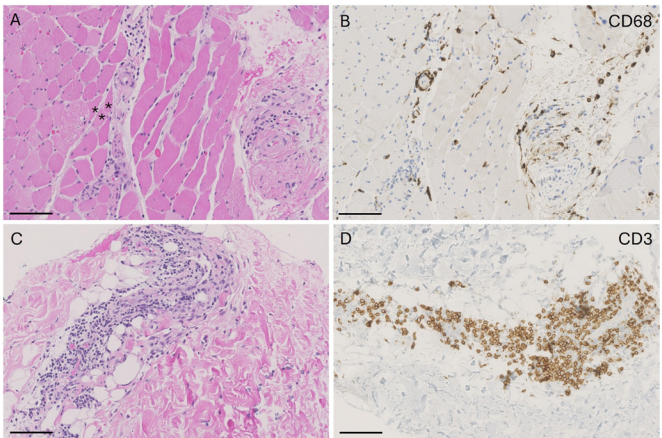


Figure 2 Muscle biopsy. (A) Striated muscle with perifascicular atrophic fibres (*) and loose oedematous endomysium, infiltrated by lymphocytes and (B) CD68-positive macrophages. (C) The adjacent fascia and fibrofatty perifascia are infiltrated by (D) CD3-positive T-lymphocytes. (A,C) H&E. (B,D) Immunohistochemistry with anti-CD3 and anti-CD68 antibodies, respectively, visualised by 3,3'-diaminobenzidine (DAB) chromogen, on 5-µm thin formalin-fixed paraffin-embedded (FFPE) tissue slices. Scale bars: 100 µm.

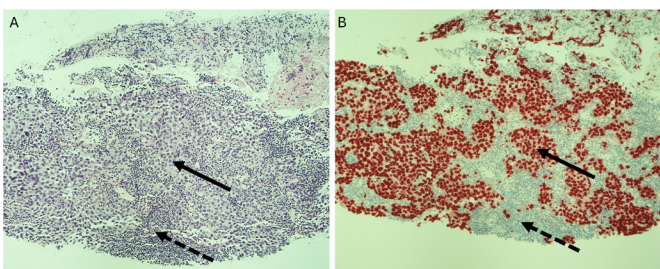


Figure 3 Biopsy of a retroperitoneal lymph node showing infiltration of an undifferentiated tumour (A; H&E). Immunohistochemically positive for SALL4 (B), corresponding to a germ cell tumour (arrow). Additional immunohistochemistry identified this germ cell tumour as a metastasis of a seminoma (positive for OCT3/4; negative for pancytokeratin and CD30). Between the tumour cell nests, there is a lymphocyte-rich and plasma cell-rich infiltrate (dashed arrow). Scale bars: 200 µm.

recurrence. Further follow-up will be conducted according to the European society for medical oncology (ESMO) guidelines recommendations for testicular cancer.¹⁵

DISCUSSION

In this case report, we identified an uncommon coincidence of a pure seminoma and paraneoplastic DM with the presence of anti-NXP-2 antibody. The rarity of this constellation is underscored by the low incidence of both individual diagnoses. Both diseases have different age distributions. Seminoma has a peak incidence in young adulthood, between the ages of 15 and 40 years,¹⁵ whereas the incidence of paraneoplastic DM increases with each decade of life, reaching its peak in individuals over the age of 80 years.¹⁶

DM can occur before, after or simultaneously with an underlying malignancy.¹¹ Here, DM was detected simultaneously with the seminoma. Retrospectively, mild symptoms had first appeared more than 6 months before hospitalisation. At initial presentation, however, the symptoms were pronounced and severe. Considering the metastatic stage of the seminoma at the time of diagnosis, it is plausible that the tumour had been present for a minimum of 6 months prior to detection.

High risk factors for IIM-related cancer	
IIM subtype	- Dermatomyositis
MSA	- anti TIF1-γ antibodies - anti NXP2 antibodies
Clinical features	- Age >40 years at IIM onset - Persistent high disease activity despite therapy - Dysphagia (moderate to severe) - Cutaneous necrosis
Total	

Figure 4 Risk factors for IIM-related cancer and proposed screening (adapted from,⁴ with permission). In case of ≥2 risk factors, the probability of IIM-related cancer is considered high: screening at the time of diagnosis is recommended, including comprehensive history, physical examination, laboratory exam with blood count, liver function test, serum erythrocyte sedimentation rate (ERS), serum C-reactive protein (CRP), serum prostate-specific antigen (PSA) and cancer-antigen 125 (CA-125), urinalysis and CT scan of the neck, thorax, abdomen and pelvis, among others. In selected cases, 18F-FDG PET/CT and upper and lower gastrointestinal endoscopy may be included. IIM, Idiopathic inflammatory myopathies.

The underlying pathophysiological mechanism explaining both the paraneoplastic phenomenon itself and its temporal association with malignancy is not fully understood.¹⁷ The reasons for varying likelihoods of different tumours being associated with DM remain unclear. Studies investigating these processes are difficult to conduct due to low case numbers. Paraneoplastic syndromes, in general, appear to be an uncommon phenomenon in urogenital tumours, particularly in testicular cancer.¹⁸ This further emphasises the rarity of our case.

According to the European Alliance of Associations for Rheumatology/American College of Rheumatology classification criteria for adult and juvenile IIM, the diagnosis of IIM is supported by clinical, laboratory and histopathological findings.¹⁹ Clinical findings, particularly the presence of pathognomonic skin manifestations, allowed for a classification of 'definitive IIM', specifically DM. A muscle biopsy is not mandatory for diagnosis if sufficient classification criteria are met. Nevertheless, an MRI-guided muscle biopsy was performed in order to exclude other myopathies, confirm the diagnosis and assess the severity of inflammation.

Our patient met several high-risk criteria for underlying malignancy: the detection of anti-NXP-2 antibodies, an age over 40 years and high disease activity. In such a case, comprehensive cancer screening is recommended (figure 4, adapted from⁴ with permission). Due to the rapidly progressive and severe clinical presentation, we initially opted for a CT scan of the thorax and abdomen to promptly facilitate further diagnostic steps, including biopsy sampling.

Consistent with the observed phenotype of anti-NXP-2 antibodies,²⁰ oedema, muscle weakness, myalgias, dysphagia, classic DM rashes and rapid, severe muscle involvement occurred at the onset of the disease. However, in contrast to other antibodies, there is no association between anti-NXP-2 and ILD.²¹ Cancer screening CT revealed no signs of interstitial pneumopathy, and the patient did not report dyspnoea. We nevertheless performed pulmonary function testing to conclusively rule out ILD before initiating chemotherapy due to the use of bleomycin.

Regarding the onset of dysphagia, there was a temporal correlation with the start of chemotherapy. In addition to exacerbating myopathy and leading to immobility, chemotherapy also triggered significant swallowing dysfunction. Myopathy in arms and legs was clinically more severe and accompanied by higher CK levels than at first presentation. This suggests that chemotherapy triggered the same paraneoplastic mechanisms, and a subclinical involvement of the pharyngeal and laryngeal muscles may have been present earlier. Dysphagia in patients with DM seems to be an under-recognised clinical feature²² and appears to correlate with the presence of specific antibodies, particularly anti-NXP-2 antibodies²³ and anti-transcription intermediary factor 1 (TIF1)- γ -antibodies²⁴ in paraneoplastic IIM.

According to guidelines, glucocorticoids with an initial dose (prednisone equivalent) of 1 mg/kg body weight per day, combined with azathioprine or methotrexate, are sufficient in most cases on the new diagnosis of DM.¹⁰ As other cases with severe clinical courses benefitted from high-dose glucocorticoid regimens, we opted for the same approach, which later necessitated a second-line therapy with IVIG due to symptom exacerbation under first-line therapy.²⁵

Learning points

- In idiopathic inflammatory myopathies (IIM), a comprehensive clinical evaluation—including diagnostic investigations such as laboratory parameters and antibody profiling—is essential for accurate diagnosis.
- Both clinical and laboratory findings can provide valuable insights into the risk of an underlying malignancy.
- Cancer screening should be guided by risk stratification to avoid missing a malignancy and to ensure appropriate causal treatment.
- A thorough search for rarely associated malignancies is warranted.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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