

Edematous dermatomyositis as a rare cause of isolated bilateral arm swelling in a patient with urothelial carcinoma: case report and literature review

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Case Report

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Abstract

Background

Edematous dermatomyositis (DM) is a rare manifestation of idiopathic inflammatory myopathy and may initially resemble vascular causes of upper-limb swelling. Early recognition is clinically important because severe cases require prompt immunosuppressive treatment and because adult-onset DM, particularly in the presence of anti-transcription intermediary factor 1-gamma (anti-TIF1- γ) antibodies, has a close association with malignancy.

Case presentation:

An 86-year-old Caucasian woman with recently treated high-grade, muscle-invasive urothelial carcinoma of the bladder presented with a 2.5-week history of progressive, severe bilateral upper-extremity edema, proximal muscle weakness, and a violaceous eruption involving the periorbital region, face, anterior neck, and upper chest, accompanied by Gottron papules. Duplex ultrasonography excluded upper-extremity deep vein thrombosis and superficial venous thrombosis. Contrast-enhanced computed tomography of the chest and abdomen excluded central venous obstruction, thoracic or abdominal tumor recurrence, and metastasis. Electromyography and bilateral shoulder/upper-arm magnetic resonance imaging were compatible with active myositis, and myositis serology was positive for anti-TIF1- γ antibodies. During hospitalization, muscle weakness and dysphagia progressed rapidly. After insufficient clinical response to prednisolone and intravenous immunoglobulin, rituximab was administered as salvage therapy. Despite treatment, weakness and dysphagia progressed, and the patient died 6 weeks after admission.

Conclusions

Edematous dermatomyositis should be considered in the differential diagnosis of otherwise unexplained bilateral upper-limb edema after upper-extremity venous thrombosis and superior vena cava syndrome have been excluded, particularly in the presence of proximal muscle weakness, dysphagia, or dermatomyositis-specific cutaneous findings. The diagnosis of DM should prompt structured oncologic reassessment and surveillance, especially in patients with anti-TIF1- γ positivity or recent malignancy.

Background

Idiopathic inflammatory myopathies (IIMs) are rare immune-mediated disorders characterized by skeletal muscle inflammation and variable extramuscular involvement. Contemporary classification distinguishes several major clinicopathological and serological entities, including dermatomyositis (DM), antisynthetase syndrome, immune-mediated necrotizing myopathy, inclusion-body myositis, and overlap myositis [1–3]. DM occupies a distinct position within this spectrum because characteristic cutaneous manifestations are central to its recognition and may precede or accompany muscle disease [4]. Adult DM is rare, with population-based estimates placing its incidence in the range of approximately 5.5–9.6 cases per 1,000,000 person-years, although reported rates vary according to case definition, study period, and ascertainment method [5, 6].

Classic DM typically presents with symmetric proximal muscle weakness, skeletal-muscle enzyme elevation, myopathic findings on electromyography (EMG) or magnetic resonance imaging (MRI), and, when biopsy is

performed, characteristic inflammatory myopathic changes. The cutaneous phenotype is diagnostically important and includes Gottron papules or Gottron sign, heliotrope rash, photo-distributed erythema of the anterior neck and upper chest, shawl-sign erythema, periungual capillary abnormalities, and poikiloderma [2, 7–10]. Adult-onset DM is strongly associated with malignancy. Population-based studies have shown an increased cancer risk in patients with DM, particularly around the time of myositis onset [11, 12]. Anti-transcription intermediary factor 1-gamma (anti-TIF1- γ) antibodies are among the most consistently described serological markers of cancer-associated DM in adults, and international recommendations now incorporate antibody status into risk-stratified cancer screening strategies [13, 14].

Urothelial carcinoma of the urinary bladder is one of the most common malignancies worldwide. Global cancer statistics for 2022 estimated more than 600,000 new bladder cancer cases and more than 220,000 bladder cancer-related deaths worldwide [15]. Urothelial carcinoma represents the predominant histological subtype of bladder cancer in developed countries [16]. Although DM has been reported in association with several solid tumors, its occurrence in patients with urothelial carcinoma is uncommon and remains supported mainly by case reports and small literature syntheses [11, 17–19].

Subcutaneous edema is an uncommon manifestation of DM and may reflect a severe inflammatory phenotype, possibly related to microangiopathy, increased vascular permeability, fasciitis, and edema of inflamed muscle. Reported patterns include facial edema, generalized edema, and limb edema; however, isolated massive swelling of both upper extremities is exceptionally rare and has mainly been described in individual case reports [20–24]. The present case illustrates this unusual intersection of cancer-associated DM and an atypical edematous presentation dominated by isolated bilateral upper-limb swelling.

Case presentation

An 86-year-old Caucasian woman presented with 2.5 weeks of progressive painless edema of both arms extending from the shoulders to the fingertips, marked proximal arm weakness, and a red-violaceous eruption of the face and upper chest that had not responded to zinc-containing topical care; fever and arthralgia were absent. Her relevant oncologic history comprised high-grade muscle-invasive urothelial carcinoma of the urinary bladder diagnosed 1 year earlier. Three months after diagnosis, she underwent radical cystectomy with anterior exenteration and bilateral ureterocutaneostomy (pT3 pN0 [0/7], L1, V0, Pn0, R1 due to carcinoma in situ at the left ureteral margin, G3). Programmed death-ligand 1 (PD-L1) negativity precluded adjuvant nivolumab; after shared decision-making, she declined cisplatin/gemcitabine and opted for surveillance. Postoperative computed tomography (CT) at 3 months and bilateral ureterorenoscopy with renal pelvic biopsies and ureteral stent exchange at 6 months showed no evidence of malignancy. A left-sided deep vein thrombosis (DVT) diagnosed 1 month postoperatively had been treated with apixaban, initially supporting a vascular cause of the edema. The clinically relevant oncologic history, symptom onset, diagnostic work-up, and treatment are summarized in Table 1.

Table 1
Clinical timeline

Time relative to admission	Event	Therapeutic intervention
12 months before	High-grade muscle-invasive urothelial bladder carcinoma diagnosed.	—
9 months before	Pathology: pT3 pN0 (0/7), L1, V0, Pn0, R1 (carcinoma in situ at the left ureteral margin), G3.	Radical cystectomy, anterior exenteration, and bilateral ureterocutaneostomy.
8 months before	Tumor board: surveillance; nivolumab not indicated (PD-L1 negative). Left-sided DVT diagnosed.	Active surveillance; apixaban.
6 months before	Surveillance CT: no tumor recurrence.	—
3 months before	Bilateral ureterorenoscopy and renal-pelvis biopsies: no malignancy.	Ureteral stent exchange.
2 weeks before	Progressive bilateral arm edema and red-violaceous rash began.	—
Day 0 (admission)	Massive bilateral arm edema, symmetric proximal upper-limb weakness, and elevated troponin T suggesting subclinical cardiac involvement.	—
Day 2	Duplex ultrasonography: no upper-limb venous thrombosis. Contrast-enhanced CT: no central venous obstruction, recurrence, or metastases. Coronary angiography: no relevant stenosis.	—
Day 4	Neurologic examination, left-deltoid EMG, and shoulder/upper-arm MRI supported active myositis.	—
Day 5	Active DM with progressive weakness and edema.	Prednisolone 50 mg/day orally.
Day 10	Right deltoid muscle biopsy; new dysphagia developed.	Nasogastric tube placement.
Days 11–17	Persistent severe disease.	Prednisolone 100 mg/day intravenously.
Days 11–14	Persistent severe disease; progressive dysphagia.	IVIG (20 g/day for 4 days).
Day 18	Aspiration pneumonia developed.	Prednisolone 70 mg/day orally; piperacillin/tazobactam for 7 days.
Day 19	Inadequate response to glucocorticoids and IVIG; persistent weakness and dysphagia.	Rituximab 1000 mg intravenously.
Day 24	Transfer to palliative care ward.	—
Day 44	Death	—
Note. CT = computed tomography; DVT = deep vein thrombosis; EMG = electromyography; IVIG = intra-		

venous immunoglobulin; MRI = magnetic resonance imaging; PD-L1 = programmed death-ligand 1.

On admission, she was afebrile and hemodynamically stable (blood pressure 128/63 mm Hg, heart rate 81/min, oxygen saturation 100%, temperature 36.9°C, Glasgow Coma Scale score 15), with a body mass index of 21.0 kg/m² (51 kg, 1.56 m). Examination showed massive isolated bilateral upper-limb edema (Fig. 1A), without facial, truncal, or lower-extremity edema; peripheral perfusion and sensation were preserved. Proximal weakness prevented arm elevation above the horizontal and drinking movements, whereas distal strength was intact and dysphagia was absent initially. The cutaneous phenotype included heliotrope discoloration of the periorbital region and forehead, V-sign erythema of the neck and upper chest (Fig. 1B), and erythema with Gottron papules over the metacarpophalangeal and interphalangeal joints (Fig. 1C).

Given the combination of malignancy, prior thrombosis, and acute bilateral arm edema, the initial diagnostic priority was exclusion of upper-extremity venous thrombosis, superior vena cava syndrome, or another central venous outflow obstruction [25, 26]. The absence of head or neck edema, venous distension, and neurovascular deficits, together with DM-specific skin lesions and proximal myopathic weakness, shifted the working diagnosis toward edematous inflammatory myopathy.

Bilateral duplex ultrasonography, including the subclavian veins, excluded superficial and deep upper-limb venous thrombosis. Contrast-enhanced CT of the chest and abdomen showed no lymphadenopathy, suspicious pulmonary lesion, local recurrence, metastasis, thrombosis, or central venous obstruction. Possible mild cardiac involvement was suspected because N-terminal pro-B-type natriuretic peptide (NT-proBNP) was 638 pg/mL and high-sensitivity troponin T (hs-TnT) ranged from 91.7 to 111 pg/mL; however, electrocardiography was normal, transthoracic echocardiography showed preserved biventricular function without relevant valve disease or effusion, and coronary angiography showed only nonobstructive coronary sclerosis.

Neurologic examination showed intact cranial, facial, and oropharyngeal function. Shoulder elevation and deltoid strength were Medical Research Council (MRC) grade 2 bilaterally, external rotation grade 3, and biceps strength grade 2; triceps, distal upper-limb and lower-limb strength, and sensation were normal. Electromyography (EMG) of the left deltoid muscle revealed fibrillation potentials and low-amplitude, polyphasic, partially shortened motor-unit potentials, consistent with active myositis. Magnetic resonance imaging (MRI) demonstrated diffuse shoulder and upper-arm muscle edema (Fig. 2A), which was also visible on CT (Fig. 2B).

Laboratory testing showed albumin 27 g/L; antinuclear antibodies (ANA) were positive at 1:320 with a fine granular pattern, and dense fine speckled 70 (DFS-70) antibodies were positive at 1:160. EUROLINE immunoblotting for myositis-specific and myositis-associated antibodies was strongly positive only for anti-TIF1- γ . Deltoid muscle biopsy showed perimysial and fascia-adjacent inflammatory infiltrates composed predominantly of macrophages and cluster of differentiation 45 (CD45)-positive leukocytes, with partial cluster of differentiation 8 (CD8) positivity, marked capillary terminal complement complex (C5b-9) deposition, and no sarcolemmal complement or major histocompatibility complex (MHC) class II expression (not shown) (Fig. 3).

The final (integrated) diagnosis was severe edematous cancer-associated DM with dysphagia and possible mild cardiac involvement. The diagnosis was supported by typical DM skin findings, proximal myopathic weakness, elevated creatine kinase (CK), concordant EMG, MRI, and biopsy findings, and anti-TIF1- γ positivity; recent high-grade urothelial carcinoma supported a cancer-associated phenotype despite the absence of CT-detectable recurrence or metastasis [2, 8–10, 13, 14, 17–19].

Oral prednisolone 50 mg/day was started on day 5 after admission, but only modest cutaneous improvement occurred while edema, weakness, and dysphagia progressed. From day 11, treatment was escalated to

intravenous prednisolone 100 mg/day and intravenous immunoglobulin (IVIG) 20 g on four consecutive days for severe glucocorticoid-refractory DM with dysphagia [10, 27]. Dysphagia rapidly became severe enough to require nasogastric feeding; flexible otolaryngologic examination showed mild symmetric arytenoid and aryepiglottic-fold edema, consistent with the systemic edematous process. Due to aspiration pneumonia, the patient received antibiotic therapy with piperacillin/tazobactam for 7 days. Because the clinical response remained insufficient, a single 1,000-mg intravenous dose of rituximab was administered on day 19 as individualized rescue therapy.

The CK trajectory is shown in Fig. 4. CK peaked at 2503 U/L on admission and declined to 200 U/L by day 22 after corticosteroid treatment and subsequent escalation; nevertheless, arm edema, weakness, and dysphagia continued to worsen, illustrating a dissociation between biochemical and functional response. A positron emission tomography/computed tomography (PET/CT) scan to evaluate for occult residual urothelial carcinoma was planned but could not be performed because of rapid clinical deterioration. The patient was subsequently transferred to hospice for palliative care and died 6 weeks after the diagnosis of cancer-associated DM was established.

On day 12 after admission, the patient's experience of her illness was explored in a brief interview; the findings are presented in Additional file 1: Supplementary methods and patient perspective.

Discussion and conclusions

The current report appears to be the first well-documented example of an edematous phenotype of DM associated with urothelial carcinoma, and, more specifically, the first in which isolated bilateral arm swelling was the dominant initial manifestation.

We searched PubMed from inception through July 6, 2026, using predefined Medical Subject Headings (MeSH) and title/abstract terms for DM and urothelial malignancy. The full search strategy is provided in Additional file 1: Supplementary methods.

Our structured literature review identified only 29 verified cases of DM associated with urothelial carcinoma between 1991 and 2026. The clinical, demographic, and serologic characteristics of the published cases are summarized in Table 2. Additionally, none of the previously published cases was clearly characterized as edematous DM and none provided information for possible cardiac involvement. Age was available for 24 patients, with a median of 68 years and a range of 42 to 79 years; sex was reported for 25 patients, 21 of whom were male. Most tumors originated in the urinary bladder. Our patient, an 86-year-old woman, was therefore both demographically atypical and older than every previously reported patient for whom age was available.

Table 2

Clinical, demographic, and serologic characteristics of published DM cases associated with urothelial carcinoma

No.	Author [reference]	Year	Age/sex	Tumor/site	DM-tumor relationship	Myositis antibodies
1	Almog [41]	1991	76/male	Bladder TCC; local recurrence	DM developed 14 years after initial diagnosis; TCC recurred 4 months after DM diagnosis	Not reported
2	Marchal Escalona [42]	1992	Not reported/female	Infiltrating bladder tumor; TCC	DM as a paraneoplastic manifestation	Not reported
3	Mallon [43]	1999	75/male	Bladder TCC	DM paralleled the tumor course and recurred with tumor recurrence	Not reported
4	Talanin [44]	1999	62/male	Bladder carcinoma/TCC; metastatic	Associated with active malignancy	ANA 1:80; anti-SMA 1:40; RPR 1:4
5	Russ [45]	1999	Not reported	Bladder TCC	Gross hematuria and amyopathic DM prompted evaluation for malignancy	Not reported
6	Federman [46]	2000	68/male	Bladder TCC; subsequent liver metastases	DM recurred 1 year after initial tumor therapy and signaled progression	Not reported
7	Robinson [47]	2001	61/male	Poorly differentiated bladder TCC; metastatic	Associated with active malignancy	Not reported
8	Apaydin [48]	2002	63/male	Bladder TCC; localized	Associated with bladder carcinoma	Not reported
9	Rankin [49]	2002	64/male	Rapidly progressive bladder TCC; locally advanced	DM in the setting of rapid tumor progression	Not reported
10	García-Donoso [50]	2003	Not reported	Bladder TCC; recurrence	DM associated with tumor recurrence	Not reported
11	Hafejee [51]	2005	75/male	Bladder carcinoma/TCC; localized	DM secondary to bladder carcinoma	Not reported
12	Choi [52]	2006	42/male	Ureteral carcinoma (upper urinary tract)	DM prompted evaluation for an occult malignancy	Not reported

Note. ANA = antinuclear antibody; anti-Jo-1 = anti-histidyl-tRNA synthetase antibody; anti-Mi-1/Mi-2 = antibodies against Mi-1/Mi-2 antigens; anti-SMA = anti-smooth muscle antibody; BCG = Bacillus Calmette-Guérin; DM = dermatomyositis; ICI = immune checkpoint inhibitor; RPR = rapid plasma reagin test; TCC = transitional cell carcinoma.

No.	Author [reference]	Year	Age/sex	Tumor/site	DM-tumor relationship	Myositis antibodies
13	Sabio [53]	2006	79/male	Bladder TCC; localized	Paraneoplastic DM associated with bladder carcinoma	Not reported
14	Sharma [54]	2007	69/male	High-grade papillary urothelial carcinoma of the bladder	DM developed during treatment of bladder carcinoma	Not reported
15	Szwebel [55]	2008	Not reported	Tumor of the upper urinary tract; exact site and histologic subtype not provided	Paraneoplastic dermatomyositis sine dermatitis	Not reported
16	Xu [19]	2013	60/male	Grade III bladder TCC; localized	DM developed during the course of bladder carcinoma	Not reported
17	Requena [29], Case 2	2014	74/male	Bladder carcinoma; tumor recurrence	DM synchronous with tumor recurrence	ANA positive, 1:160; myositis-specific antibodies not reported by individual case
18	Requena [29], Case 3	2014	68/male	Bladder carcinoma	DM developed 18 months after tumor diagnosis	ANA negative; anti-Jo-1 negative; anti-Mi-1/Mi-2 negative
19	Requena [29], Case 10	2014	69/male	Bladder carcinoma; tumor recurrence	DM developed 15 months before tumor recurrence	ANA negative; anti-Jo-1 negative
20	Fitzpatrick [30]	2014	67/female	Bladder TCC; metastatic recurrence after remission	Recurrent DM signaled metastatic recurrence	Not reported
21	Bientinesi [56]	2016	Not reported	Panurothelial TCC	Paraneoplastic DM	Not reported
22	Kase [57]	2018	65/male	High-grade invasive	DM prompted further evaluation for malignancy	ANA negative;

Note. ANA = antinuclear antibody; anti-Jo-1 = anti-histidyl-tRNA synthetase antibody; anti-Mi-1/Mi-2 = antibodies against Mi-1/Mi-2 antigens; anti-SMA = anti-smooth muscle antibody; BCG = Bacillus Calmette-Guérin; DM = dermatomyositis; ICI = immune checkpoint inhibitor; RPR = rapid plasma reagin test; TCC = transitional cell carcinoma.

No.	Author [reference]	Year	Age/sex	Tumor/site	DM-tumor relationship	Myositis antibodies
				urothelial carcinoma of the bladder		anti-Jo-1 negative
23	Sabaté-Ortega [17]	2023	75/male	High-grade urothelial carcinoma; iliac recurrence after cystectomy	DM associated with distant recurrent disease	anti-TIF1-γ weakly positive
24	López Guerra [35], Case 2	2023	75/female	Stage IV urothelial carcinoma (pT3 pN2 M1)	DM developed parallel with tumor progression	anti-TIF1-γ positive (high titer)
25	López Guerra [35], Case 3	2023	73/male	Stage IIIB urothelial carcinoma; progression with nodal and bone involvement; pembrolizumab	DM symptoms developed 1 week after the second pembrolizumab infusion; skin lesions may retrospectively have preceded ICI therapy	anti-TIF1-γ positive (high titer)
26	Dimond [36]	2024	71/male	Bladder TCC; intravesical BCG therapy	Early symptoms preceded BCG; overt clinical manifestations developed afterward	anti-TIF1-γ positive
27	Kampoli [58]	2024	66/female	Urothelial carcinoma of the left renal pelvis; local para-aortic recurrence	DM developed 3 years after nephrectomy/ureterectomy and led to detection of local recurrence	Not reported
28	Torniai [37]	2025	50/male	Sarcomatoid urothelial carcinoma of the bladder; metastatic	DM developed 6 months before tumor diagnosis; flare paralleled progression	anti-TIF1-γ positive
29	Marwaha [18]	2026	65/male	Metastatic urothelial carcinoma of the bladder	DM developed concurrently with metastatic progression	Not reported in abstract
Note. ANA = antinuclear antibody; anti-Jo-1 = anti-histidyl-tRNA synthetase antibody; anti-Mi-1/Mi-2 = antibodies against Mi-1/Mi-2 antigens; anti-SMA = anti-smooth muscle antibody; BCG = Bacillus Calmette-Guérin; DM = dermatomyositis; ICI = immune checkpoint inhibitor; RPR = rapid plasma reagin test; TCC = transitional cell carcinoma.						

The relationship between adult DM and malignancy is well established, with the highest cancer risk clustering around the onset of inflammatory myopathy [11, 12, 28]. In the population-based study by Hill et al., 198 of 618 (32%) patients with DM had a malignancy with 58% of these cancers diagnosed after DM onset. The overall cancer risk after DM was increased threefold (standardized incidence ratio 3.0; 95% confidence interval 2.5–3.6). In descending order, the site-specific standardized incidence ratios were highest for ovarian cancer (10.5), lung cancer (5.9), pancreatic cancer (3.8), non-Hodgkin lymphoma (3.6), gastric cancer (3.5), and colorectal cancer

(2.5) [11]. This distribution suggests that the association between urothelial carcinoma and DM, as observed in our patient, is uncommon. Bladder cancer was comparatively uncommon in the DM cohort, with only three cases after DM diagnosis and no statistically significant excess risk. In contrast, Hill et al. observed nine bladder cancers among patients with polymyositis, corresponding to a significantly increased standardized incidence ratio of 2.4 (95% confidence interval 1.3–4.7) [11]. Thus, bladder cancer was not among the dominant tumor types in unselected DM cohorts and showed a clearer epidemiologic association with polymyositis. Thus, the evidence linking urothelial carcinoma specifically to DM remains largely case based [17–19]. Within the urothelial literature, the temporal pattern is heterogeneous: DM has prompted the initial cancer work-up, preceded the recognition of recurrence, emerged during active disease, or flared in parallel with tumor progression [17, 18, 29, 30]. In our patient, DM developed after radical cystectomy, while CT and repeated endoscopic assessment had shown no overt recurrence. Even so, the pT3 high-grade tumor, the R1 carcinoma-in-situ margin, the short interval between oncologic treatment and myositis onset, and strong anti-TIF1- γ positivity support a cancer-associated phenotype. Because occult active disease could neither be proven nor conclusively excluded, cancer-associated DM is more precise than definitively paraneoplastic DM.

Anti-TIF1- γ positivity provides an additional link to malignancy. This autoantibody is among the most consistently reported serologic markers of cancer-associated DM and is incorporated into contemporary risk-stratified screening recommendations [13, 14, 31, 32]. Only five of the 29 independently verifiable urothelial carcinoma-associated cases documented anti-TIF1- γ antibodies, all in reports from 2023 onward. The present case represents a sixth serologically characterized example. The true proportion cannot be estimated because most older reports predated routine myositis-specific antibody testing. Previous case series also suggest that anti-TIF1- γ -positive disease is associated with prominent dysphagia and cutaneous involvement, malignancy, and adverse outcomes [31–33]. Our patient exhibited all these clinical characteristics, including prominent cutaneous involvement, progressive dysphagia, an associated malignancy, and a fatal outcome. A report of small-cell neuroendocrine carcinoma of the urinary bladder further demonstrated tumor-cell expression of transcription intermediary factor 1-gamma (TIF1- γ), although the mechanistic significance remains uncertain [34]. Importantly, our patient had received neither immune checkpoint inhibitor (ICI) therapy nor intravesical *Bacillus Calmette-Guérin* (BCG). Her presentation therefore differs from the high-titer anti-TIF1- γ -positive cases that developed during ICI therapy, from the etiologically ambiguous BCG-associated case, and from the report in which preexisting paraneoplastic DM complicated subsequent pembrolizumab treatment [35–37]. The absence of such a treatment trigger strengthens the interpretation of a tumor-associated autoimmune process, although it does not establish whether microscopic residual carcinoma was present.

The edematous phenotype is the most distinctive clinical feature. Edema is uncommon in DM and has more often been described as generalized swelling, facial edema, or involvement of several limb regions. By 2014, only 19 cases had been assembled in the literature, and edema was proposed as a marker of severe disease that may require early, aggressive therapy [38]. In a later series of edematous myositis, dysphagia was frequent, capillary C5b-9 deposition occurred in most biopsies, and perimysial edema was prominent [24]. Generalized edema remains rare even in contemporary reports [21, 22]. Bilateral upper-limb edema has occasionally heralded aggressive DM, but published patients often had additional lower-extremity or generalized edema, and the serologic phenotype was not necessarily anti-TIF1- γ -positive [20, 39]. In the present patient, swelling extended from both shoulders to the fingertips while the face, trunk, and lower extremities remained externally unaffected. Within the compiled urothelial carcinoma literature, this distribution appears unique. The muscle-biopsy findings support a vasculopathic DM phenotype. Comparable findings have been reported in anti-TIF1- γ -positive DM

associated with urothelial cancer, including perimysial inflammation, myovasculopathy, capillary loss, and C5b-9 deposition [36]. These parallels support the hypothesis that complement-mediated microangiopathy and increased vascular permeability contributed directly to the massive subcutaneous edema.

This presentation creates a substantial diagnostic pitfall. In an older patient with cancer and a previous venous thrombosis, massive arm swelling appropriately prompts urgent exclusion of upper-extremity deep venous thrombosis, central venous obstruction, superior vena cava syndrome, and tumor-related lymphatic obstruction [25, 26]. Bilateral ultrasonography and contrast-enhanced imaging ruled out these diagnoses. The absence of facial or neck swelling, venous collateralization, lower-extremity edema, and severe hypoalbuminemia also argued against a generalized hemodynamic or oncotic mechanism. The decisive clues were found at the bedside: proximal arm weakness with preserved distal hand strength, Gottron papules, heliotrope discoloration, and V-area erythema. Elevated CK and concordant EMG, MRI, and biopsy findings then established the myopathic component. Clinically, unexplained bilateral arm edema should trigger a focused dermatologic and neuromuscular examination once common vascular causes have been excluded.

A clinically important feature was the dissociation between biochemical and functional response. CK declined from 2503 U/L to approximately 200 U/L after corticosteroid treatment and subsequent escalation, yet the edema, weakness, and dysphagia continued to worsen. Enzyme normalization therefore did not indicate disease control. Serial assessment should prioritize muscle strength, swallowing safety, respiratory function, nutritional status, edema, skin activity, and functional independence. IVIG was justified by the severe dysphagia and by randomized evidence supporting its efficacy in active DM [27]. Rituximab was subsequently used as individualized rescue therapy after inadequate response to glucocorticoids and IVIG. The fatal course despite this escalation underscores that edematous cancer-associated anti-TIF1- γ -positive DM can be fulminant and treatment refractory.

Management of cancer-associated DM requires simultaneous consideration of immunosuppression and tumor control. Several urothelial reports describe improvement after cancer-directed treatment or recurrence of DM in parallel with tumor relapse, suggesting that myositis activity may function as a clinical marker of tumor burden in selected patients [17, 18, 29, 30]. The relative priority of oncologic and immunomodulatory treatment remains uncertain when severe weakness or dysphagia progresses rapidly [40]. In our patient, no macroscopic target for tumor-directed therapy was identified. Planned PET/CT assessment for occult malignancy could not be completed because of rapid deterioration, leaving the therapeutic strategy focused on inflammatory disease. This scenario is clinically important: a patient may have a highly suggestive cancer-associated serologic and clinical phenotype despite negative conventional imaging. Where the patient's condition permits, enhanced and repeated malignancy screening is appropriate, particularly in older adults with anti-TIF1- γ positivity [14].

This case and literature review have several limitations. The evidence on urothelial carcinoma-associated DM is based mainly on isolated case reports and small series, with considerable heterogeneity in diagnostic criteria, tumor staging, antibody testing, histopathology, treatment sequence, and follow-up. Many older reports predate routine myositis-specific antibody testing, and publication bias may favor severe, recurrent, or therapeutically unusual presentations. In the present case, occult residual or recurrent carcinoma could not be definitively confirmed or excluded because PET/CT was not completed. The rapidly fatal course also limited longitudinal assessment of myositis activity and tumor status.

This case appears to represent the first clearly documented edematous phenotype of cancer-associated, anti-TIF1- γ -positive DM linked to urothelial carcinoma, with massive isolated bilateral arm swelling as the predominant clinical manifestation. Edematous DM should therefore be included in the differential diagnosis of otherwise unexplained bilateral upper-extremity swelling after venous thrombosis and central venous obstruction have been excluded, particularly when proximal muscle weakness or DM-specific cutaneous findings are present. This case also represents the oldest patient with reported age in the compiled series (Table 2). The clinical course was fulminant, with progressive weakness and dysphagia despite improvement in CK levels. In older adults, especially those with anti-TIF1- γ antibodies or a recent malignancy, recognition of this phenotype should prompt urgent immunomodulatory treatment, early and repeated swallowing assessment, and structured evaluation for occult or recurrent cancer.

Abbreviations

ANA

Antinuclear antibody

anti-Jo-1

Anti-histidyl-tRNA synthetase antibody

anti-Mi-1/Mi-2

Antibodies against Mi-1/Mi-2 antigens

anti-SMA

Anti-smooth muscle antibody

anti-TIF1- γ

Anti-transcription intermediary factor 1-gamma antibody

BCG

Bacillus Calmette-Guérin

C5b-9

Terminal complement complex (membrane attack complex)

CD8

Cluster of differentiation 8

CD45

Cluster of differentiation 45

CK

Creatine kinase

CT

Computed tomography

DFS-70

Dense fine speckled 70

DM

Dermatomyositis

DVT

Deep vein thrombosis

EMG

Electromyography

hs-TnT

High-sensitivity troponin T
ICI
Immune checkpoint inhibitor
IIM
Idiopathic inflammatory myopathy
i.v.
Intravenous
IVIG
Intravenous immunoglobulin
MeSH
Medical Subject Headings
MHC
Major histocompatibility complex
MRC
Medical Research Council
MRI
Magnetic resonance imaging
NT-proBNP
N-terminal pro-B-type natriuretic peptide
p.o.
Oral administration
PD-L1
Programmed death-ligand 1
PET/CT
Positron emission tomography/computed tomography
RPR
Rapid plasma reagin test
STIR
Short-tau inversion-recovery magnetic resonance imaging
TCC
Transitional cell carcinoma
TIF1- γ
Transcription intermediary factor 1-gamma

Declarations

Ethics approval and consent to participate

Ethics committee approval was not required for this single-patient case report under the institutional regulations of Brandenburg University Hospital.

Consent for publication

Written informed consent for publication (including clinical data, images and the manuscript) was obtained from the patient before submission.

Competing interests

The authors declare that they have no competing interests.

Use of generative artificial intelligence

The authors used ChatGPT (OpenAI) for translation and language editing. All output was reviewed and revised by the authors, who take full responsibility for the final manuscript.

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Author Contribution

O.P. was the principal author, curated the clinical data, prepared Figures 1, 2, and 4 and Tables 1 and 2, and drafted the manuscript. W.S. performed the neuropathological evaluation of the muscle biopsy and provided the histopathological images presented in Figure 3. D.P. supervised the clinical work and critically revised the manuscript. All authors reviewed and approved the final manuscript.

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Availability of data and materials

All data supporting the findings of this case report are included in the article. Additional data are available from the corresponding author upon reasonable request.

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Figures



Figure 1

Clinical manifestations of edematous dermatomyositis. **(A)** Severe isolated bilateral upper-limb edema without lower-limb involvement. **(B)** V-shaped erythema of the upper chest and anterior neck (V-sign). **(C)** Erythema of the dorsal hand with Gottron papules over the metacarpophalangeal and interphalangeal joints.

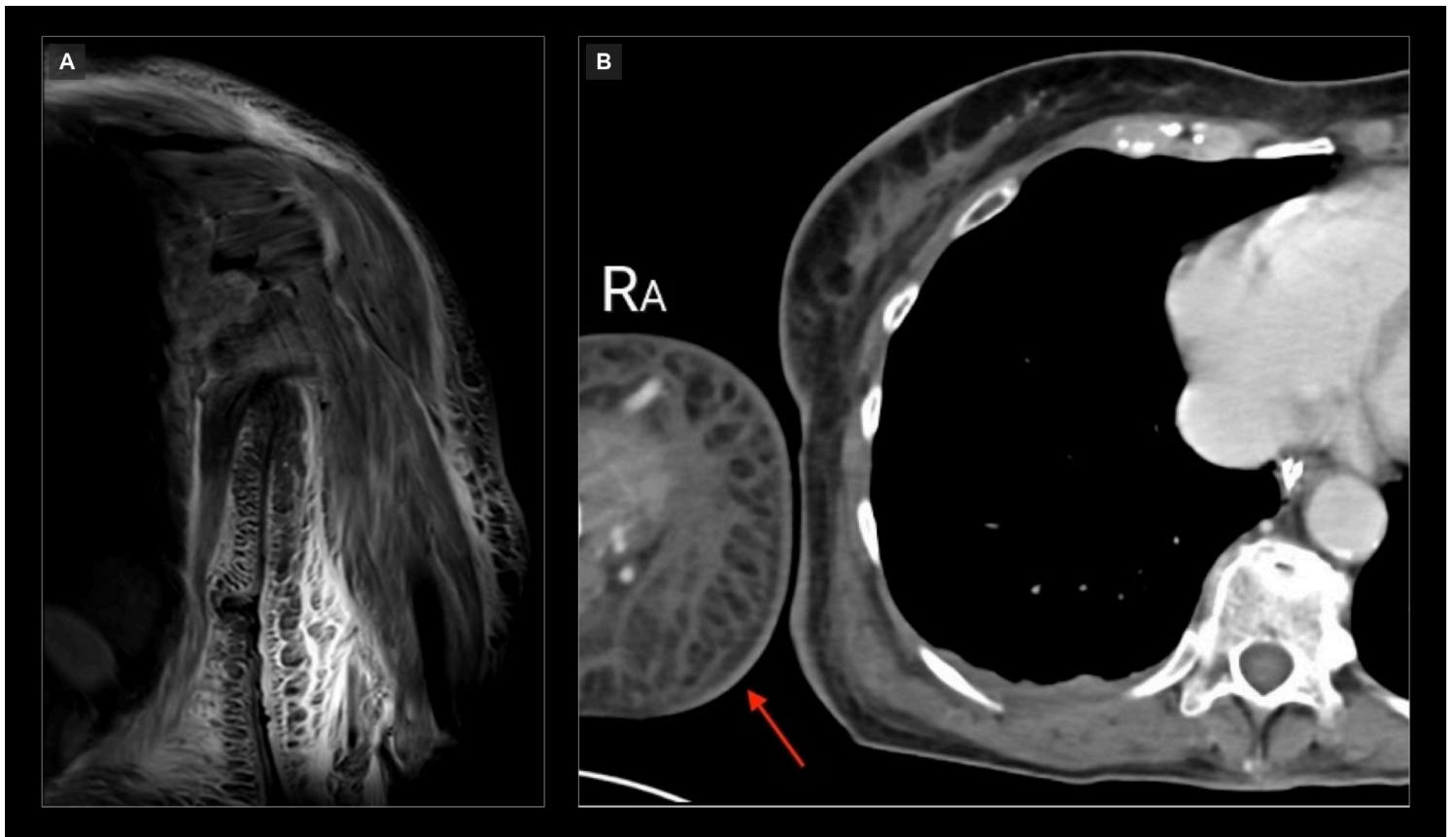


Figure 2

Imaging findings of upper-limb edema. **(A)** Short-tau inversion-recovery (STIR) magnetic resonance imaging of the left arm shows marked edema of the proximal muscles and subcutaneous tissue. **(B)** Axial computed tomography of the right upper arm demonstrates diffuse subcutaneous edema (red arrow).

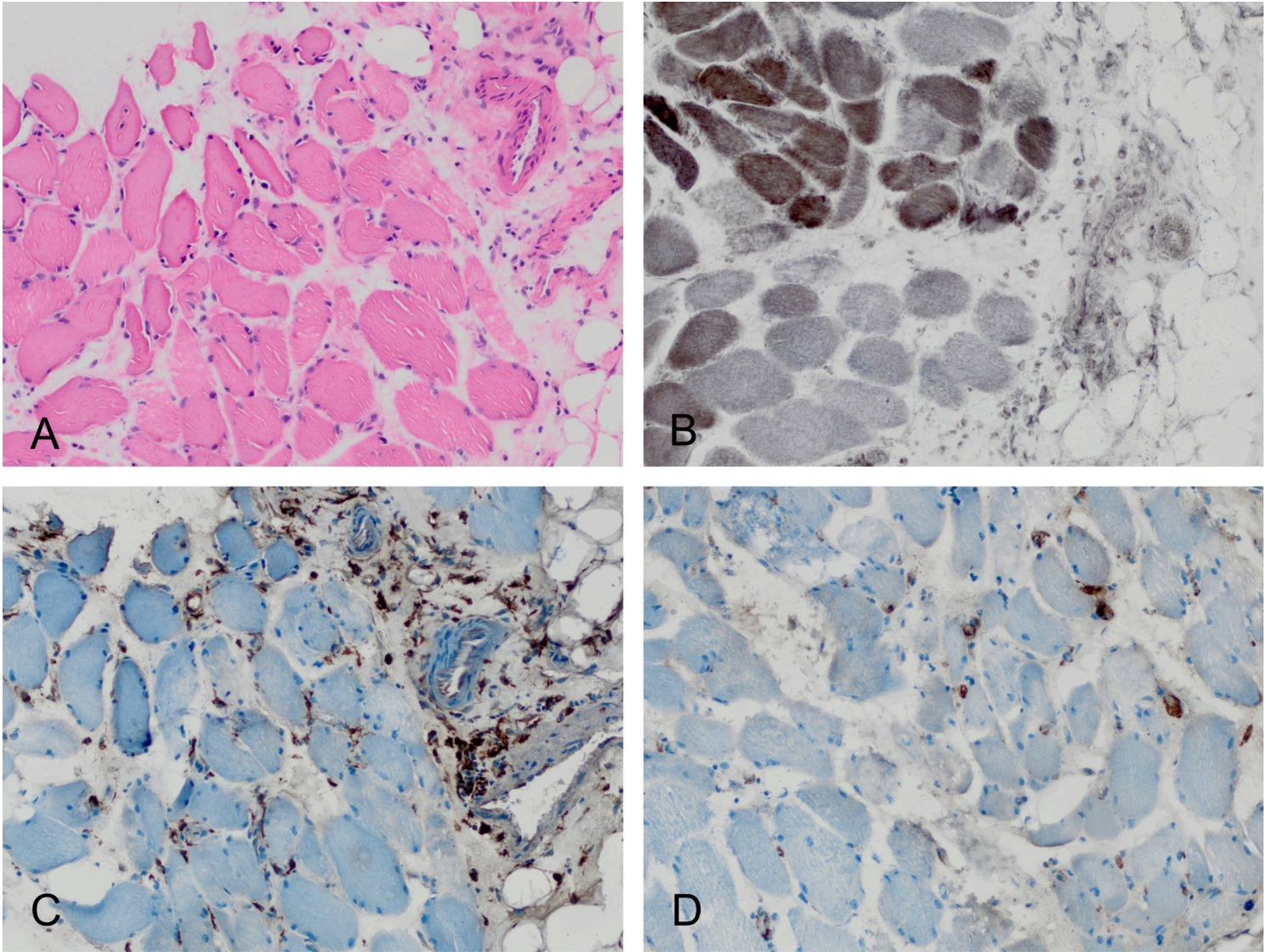


Figure 3

Neuropathologic evaluation of the right deltoid muscle biopsy. **(A)** Hematoxylin and eosin staining shows perimysial lymphomonocytic inflammatory infiltrates extending partly into the endomysium, with numerous atrophic myofibers adjacent to the perimysium (original magnification $\times 200$). **(B)** Cytochrome c oxidase-succinate dehydrogenase staining highlights perifascicular cytochrome c oxidase pallor. **(C)** Immunohistochemistry for cluster of differentiation 68 demonstrates prominent macrophage infiltration in the perimysium extending into endomysial areas (original magnification $\times 200$). **(D)** Marked capillary C5b-9 deposition is present without sarcolemmal complement expression (original magnification $\times 200$). In the clinical context, these findings were consistent with anti-TIF1- γ -associated cancer-associated DM.

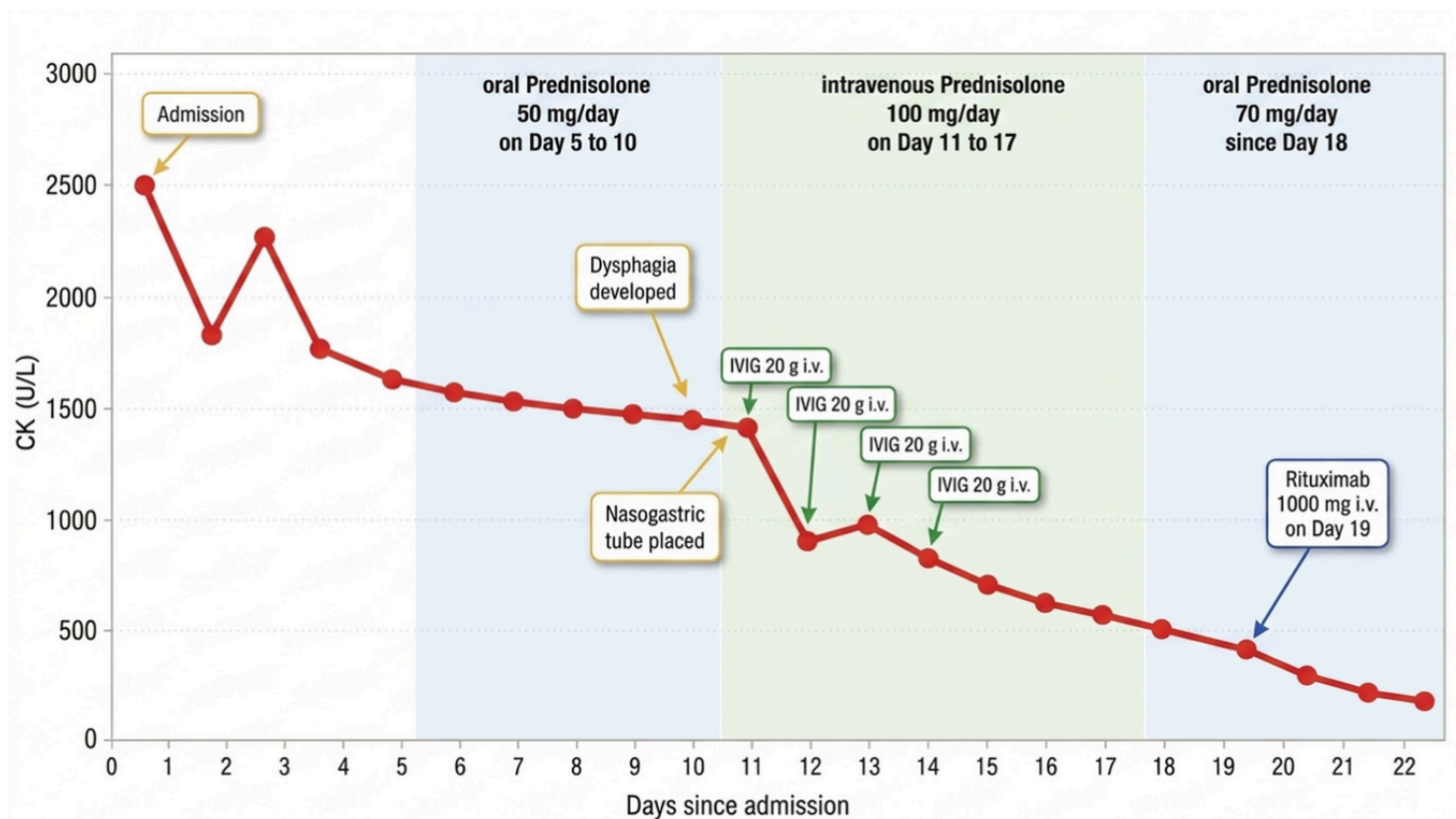


Figure 4

CK trajectory during hospitalization. The red line shows the CK course together with important clinical and therapeutic events. Abbreviations: i.v., intravenous; p.o., oral administration; U/L, units per liter.

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