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Case Report

Clinically amyopathic dermatomyositis manifested after the allogeneic haematopoietic stem cell transplantation: Case presentation and literature review

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ABSTRACT

Clinically amyopathic dermatomyositis (CADM) lacks muscle symptoms, associated with rapidly progressive interstitial lung disease. Anti-melanoma differentiation-associated gene 5 (MDA-5) antibody has been identified as a disease-labelling autoantibody. We report two cases of CADM manifested after the allogeneic haematopoietic stem cell transplantation (allo-HSCT)—Case 1: a 56-year-old man with acute leukaemia received the allo-HSCT and Case 2: a 45-year-old female patient with lymphoma received the allo-HSCT. She received donor lymphocyte infusion because of a post-transplant relapse. After allo-HSCT or donor lymphocyte infusion, Gottron papules emerged, and both patients were diagnosed as CADM based on dermatological findings coupled with the positivity of anti-MDA-5 antibody, accompanied by interstitial shadows consistent with ILD on chest computed tomography. Case 2 was initially diagnosed as a kind of chronic graft versus host disease. Their symptoms were improved by the combination of immunosuppressive agents with a concomitant decrease in anti-MDA-5 antibody levels. For Case 2, rituximab was subsequently started for relapse of lymphoma, resulting in a substantial decrease in the level of anti-MDA-5 antibody and improvement in rash and ILD. Our cases raise a possibility that CADM emerges after the HSCT, highlighting the importance of early diagnosis to avoid fated progression into ILD.

KEYWORDS: Interstitial lung disease; clinically amyopathic dermatomyositis; anti-MDA-5 antibody; chronic graft versus host disease (GvHD); allogeneic haematopoietic stem cell transplantation (allo-HSCT)

Introduction

Idiopathic inflammatory myopathies include heterogeneous autoimmune disorders characterised by chronic muscle inflammation with muscle weakness and myalgia. In particular, dermatomyositis (DM) is known to represent skin lesions such as heliotrope rashes and Gottron papules. To date, several definitions for amyopathic DM have been proposed, some of which are aimed to differentiate between patients with absolute absence of muscular involvement (amyopathic DM: ADM) and those with subclinical myositis (hypomyopathic DM: HDM). Both are defined as clinically amyopathic DM (CADM) [1]. Of note, patients with CADM occasionally represent polyarthritis, interstitial lung disease (ILD), myocarditis, and malignant tumour without skin or muscular involvement. In particular, rapidly progressive ILD (RPILD) is highlighted as a major fatal complication of CADM in a short period of time.

Anti-melanoma differentiation-associated gene 5 (MDA-5) antibody is one of the myositis-specific autoantibodies. It is highly exclusive in CADM characterised by unique specific skin symptoms and lack of skeletal muscle findings [2]. Anti-MDA-5 antibody-positive patients are frequently accompanied by RPILD and therefore distinctively show a poor prognosis as compared to those without anti-MDA-5 antibodies [3, 4].

Allogeneic haematopoietic stem cell transplantation (allo-HSCT) is a potent curative therapy for patients with haematological malignancies via the immune system of donor-driven graft-versus-leukaemia effect. On the other hand, acute graft versus host diseases (aGvHD and chronic graft versus host diseases (cGvHD) are known to be major complications of allo-HSCT. Acute GvHD is mainly related to exaggerated activation of T-cell and cytokine release, whereas dysregulation of B-cells is considerably involved in the pathogenesis of cGvHD. It is well documented that cGvHD may develop in some cases after the allo-HSCT, representing a variety of symptoms closely similar to those in autoimmune diseases [5]. However, there are very few reports of anti-MDA-5

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antibody-positive myositis emerged after the allo-HSCT [6, 7]. In this context, we here report two extremely rare cases in which CADM with RPILD was diagnosed by unique skin symptoms and lung findings after the allo-HSCT.

Case presentation

Case 1

A 56-year-old man was admitted with malaise, dry cough, shortness of breath at rest, and Gottron papules. About 3 years earlier, he received allo-HSCT from an HLA-haploidentical sibling donor for acute myeloid leukaemia (AML). HLA typing was as follows: patient A 26:01/26:01, patient B 40:01/40:06, patient C 07:02/08:01, DRB1 09:01/14:06, donor A 26:01/26:01, donor B 40:01/54:01, donor C 0102:07:02, and DRB1 14:05/14:06. Both the patient and donor had no history and family history of autoimmune diseases. The conditioning regimen consisted of a combination of busulfan (1.6 mg/kg), fludarabine (150 mg/m^2), and total body irradiation (TBI) (4 Gy). GvHD prophylaxis consisted of cyclophosphamide (CY), tacrolimus (TAC), and mycophenolate mofetil. The patient developed aGvHD (grade III: skin stage 2 and digestive stage 3) and cutaneous cGvHD (severe, regarding the consensus criteria for cGvHD [8]). He required additional immunosuppressive therapy with prednisolone (PSL), resulting in a gradual improvement of GvHD. He was free from immunosuppressants 19 months after the allo-HSCT.

About 33 months later, he complained of malaise, dry cough, and shortness of breath at rest. There were numerous papules in the skin adjacent to the back of the metacarpophalangeal and interphalangeal joints [Figure 1(a)]. Chest computed tomography (CT) scan revealed band-like opacities and consolidations in both lower lobes of the lung [Figure 1(b)]. Bronchoscopic findings via the bronchoalveolar lavage showed predominance in lymphocytes and macrophages without pathogenic microorganisms or malignant cells. Skin biopsy revealed liquefaction degeneration at the dermal–epidermal junction and perivascular lymphocytic infiltration in the dermis, consistent well with pathological findings of DM [Figure 1(c)]. Both antinuclear antibody (ANA) and anti-aminoacyl-tRNA synthetase (ARS) antibody were negative. On the other hand, anti-MDA-5 antibody was detected in sera with a considerable high titre (1350 index). Based on these findings, he was diagnosed with CADM accompanied by ILD. The combination therapy, including PSL (1 mg/kg/day) following pulse intravenous (IV) methylprednisolone (MP) (1000 mg/day, 3 days), TAC (3 mg/day), and IV CY (12.5 mg/kg/day, 6 times), was started. Symptoms of lung and skin were markedly improved with a considerable decrease in titres of anti-MDA-5 antibody over 3 months [Figure 1(d)]. He has been doing well without relapse of AML and CADM with ILD. The clinical and biological characteristics of Case 1 are summarised in Table 1.

Case 2

A 45-year-old woman was admitted with Gottron papules and periungual erythema. Four years earlier, she developed refractory primary mediastinal large B-cell lymphoma. She achieved complete remission with multi-drug chemotherapies but relapsed in a short period of time. Although high-dose chemotherapy with autologous HSCT was performed, she

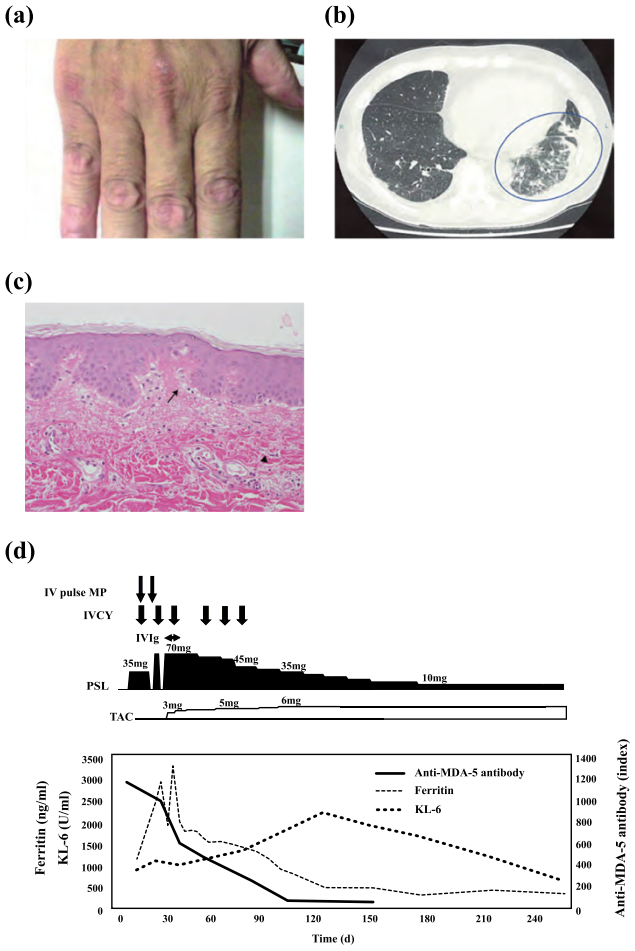

Figure 1. Clinical findings at diagnosis and clinical course in Case 1. (a) Numerous papules on the back of metacarpophalangeal and interphalangeal joints. (b) Chest CT scan revealed band-like opacity and consolidations (indicated by a circle). (c) Skin biopsy revealed liquefaction and vacuolar degeneration at the dermal–epidermal junction (indicated by an arrow), and perivascular lymphocytic infiltration in the dermis (indicated by an arrowhead) (haematoxylin and eosin ×20). (d) Treatment was started including IV pulse MP (1000 mg/day, 3 days), PSL (1 mg/kg/day), TAC (3 mg/day), and pulse IVCY (12.5 mg/kg/day, 6 times). The value for the titre of serum anti-MDA-5 antibody and ferritin concentration was drastically improved in parallel with amelioration of skin and lung symptoms. In addition, the titres of KL-6 were decreased slowly.

experienced a relapse in the mediastinum, iliac muscles, and bilateral adrenal glands 2 months after the transplantation. After the salvage chemotherapy and radiotherapy, partial remission was obtained. She received allo-HSCT from an HLA-matched sibling donor (HLA typing: A 02:06/11:01, B 15:01/40:02, C 03:03/07:02, and DRB1 04:03/11:01). Both the patient and donor had neither history nor family history of autoimmune diseases. A conditioning regimen consisted of fludarabine (125 mg/m^2), melphalan (140 mg/m^2), and TBI (4 Gy). GvHD prophylaxis consisted of cyclosporine (CsA) and short-term methotrexate. Engraftment was gained at Day 12, and complete donor chimerism was achieved at Day 31. She did not develop aGvHD. CT scan at Day 30 showed residual diseases in the mediastinum and adrenal glands, and the immunosuppressants were rapidly tapered. Regrettably, however, the residual lesions aggravated at around Day 200. After completed the irradiation to residual lesions, donor

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Table 1. Clinical and biological characteristics of patients.

	Case 1	Case 2
Sex	Male	Female
Diagnosis	AML	Primary mediastinal large B-cell lymphoma
Type of transplantation	HLA-haploidentical haematopoietic stem cell	Peripheral blood stem cell
Stem cell source	Sibling	Sibling
Conditioning regimen	Non-myeloablative	Non-myeloablative
Acute GvHD	Skin (stage2) Digestive(stages 2 and 3)	None
Chronic GvHD	Skin	None
Time from HSCT to CADM onset	33 months	8 months
ANA	Negative	1:80
ARS antibody	Negative	Negative
Treatment	PSL TAC IV pulse MP IVCY IVIg	PSL Cyclosporin Rituximab

Abbreviation: IVIg, intravenous immunoglobulin.

lymphocyte infusion (DLI) was performed on Days 229 and 245 (Day 229: 1×10^6 $CD3^+$ cells/kg, Day 245: 1×10^7 $CD3^+$ cells/kg, respectively). Cluster of Differentiation(CD) is cell surface molecules expressed on immune system cells, and CD3 is an antigenic molecule of the T-cell receptor. Nevertheless, she did not achieve complete remission of lymphoma.

Around Day 255, papules on the back of her metacarpophalangeal and interphalangeal joints and periungual erythema emerged [Figure 2(a)]. Because she was initially diagnosed as GvHD, she was treated with oral PSL (1 mg/kg/day) and CsA. Although skin symptoms were once improved, it was exacerbated during the course of tapering immunosuppressants around Day 390. Findings of skin biopsies showed liquefaction degeneration at the dermal–epidermal junction, perivascular lymphocytic infiltration in the dermis, and were consistent with pathological findings of DM [Figure 2(b)]. There were no muscle symptoms. Based on the positivity of anti-MDA-5 antibody (850 index), she was diagnosed with CADM. Chest CT revealed ground-glass opacities at both lower lobs, suggesting the involvement of ILD [Figure 2(c)]. Notably, ANA was positive, but anti-ARS antibody was negative.

In anticipation of therapeutic impact on both lymphoma and CADM with ILD, we started the treatment of rituximab (375 mg/m^2, once a week), and reciprocally, CsA and PSL were tapered. After four cycles of rituximab, her skin and pulmonary lesions were improved, and also the anti-MDA5 antibody titres were considerably decreased.

Regrettably, lymphoma was again exacerbated, and DLI was resumed (total six times, 5.0×10^6–4.0×10^7 $CD3^+$ cells/kg). As DLI may enhance autoimmunity, we concerned about the relapse of CADM. Therefore, four cycles of rituximab were used in combination with DLI. Although she remained free of skin and lung symptoms, she died of lymphoma on Day 716. The clinical and biological characteristics of Case 2 are summarised in Table 1.

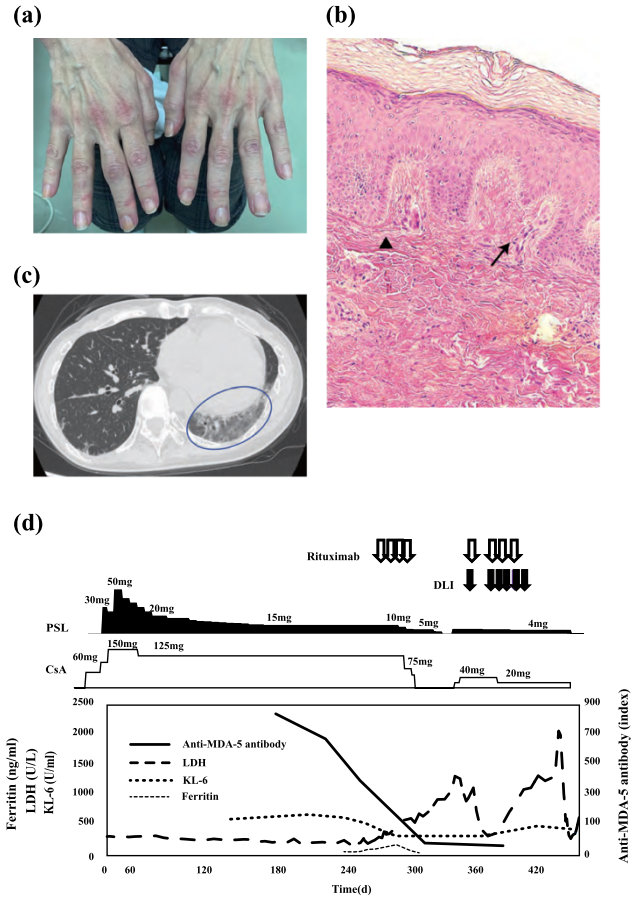

Figure 2. Clinical findings at diagnosis and clinical course in Case 2. (a) Papules on the back of her metacarpophalangeal and interphalangeal joints and periungual erythema. (b) Skin biopsy showed liquefaction degeneration at the dermal–epidermal junction (indicated by an arrow) and perivascular lymphocytic infiltration in the dermis (indicated by an arrowhead) (haematoxylin and eosin ×20). (c) Chest CT scan showing ground-glass opacity (indicated by a circle). (d) Because of the initial diagnosis of GvHD, we started the treatment with oral PSL (1 mg/kg/day) and CsA. Skin symptoms were aggravated during the course of tapering immunosuppressants. The patient was finally diagnosed as a CADM based on pathological findings and the positivity of anti-MDA-5 antibody. In anticipation of therapeutic impact on both lymphoma and CADM with ILD, we started the treatment of rituximab (375 mg/m^2, once a week) ~270 days after CADM onset. After four cycles of rituximab, titres of anti-MDA-5 antibody and KL-6 were considerably decreased, in parallel with amelioration of skin and lung symptoms. About 330 days after the CADM onset, the level of lactate dehydrogenase in sera was markedly increased, and lymphoma was again exacerbated. DLI was resumed (total six times, 5.0×10^6–4.0×10^7 $CD3^+$ cells/kg) with a combination of four cycles of rituximab. Although she remained free of skin and lung symptoms, she died of lymphoma ~450 days after CADM onset.

Discussion

We report two extremely rare cases of CADM followed by the allo-HSCT, which were closely reminiscent of cGvHD with PRILD. To treat CADM, robust immunosuppressive therapies from the early phase are highly recommended [9]. Recently, a prospective study of a three-drug combination, including high-dose steroid (1 mg/kg), calcineurin inhibitor, and IVCY for CADM, was reported, demonstrating an excellent 6-month survival rate of ~89% [10]. In accordance with this notion, early diagnosis, as well as initiation of treatment, resulted in a successful outcome in our Case 1.

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Table 2. Clinical and biological characteristics of previously reported seven patients with anti-MDA-5 antibodies after the allo-HSCT.

	Sex	Diagnosis	Type of transplantation	Acute GvHD	Chronic GvHD	DM-like skin findings	Presence of ILD	ANA	Anti-ARS antibody	Treatment	Outcome
Lepelletier et al. [6]	F	ALL	rPBSCT	Digestive	Digestive	Finger pad inflammation Digital ulcerations Palmar violaceous papules Nailfold telangiectasia	Yes	Positive in five of six patients	Positive in five of six patients	CsA IV pulse MP	Dead
	F	Sickle cells disease	CBT	None	None	Digital ulcerations Periungueal erythema Onychodystrophy Raynaud phenomenon	Yes			PSL CsA MMF IV pulse MP	Recovered
	F	MDS	rPBSCT	Skin Digestive Liver	Mouth	Finger pad inflammation Gottron papules Periungual erythema Facial and upper chest erythema	Yes			PSL	Dead
	M	Plasma cells leukaemia	rPBSCT	None	Skin Mouth Liver	None	Yes			PSL Cyclosporin IV pulse MP	Recovered
	M	MM	rPBSCT	Liver	Fascia Skin Eye Liver	None	No			–	–
	F	ALL	UR-PBSCT	Skin Digestive	Fascia Skin Eye Digestive	None	No			–	–
Gutierrez et al. [7]	F	AML	NR	NR	NR	Heliotrope patches Gottron papules Onychodystrophy	Yes	Positive 1:640	Negative	TAC Glucocorticoid Hydroxychloroquine	Recovered

Abbreviations: ALL, acute lymphoid leukaemia; MDS, myelodysplastic syndromes; MM, multiple myeloma; rPBSCT, related peripheral blood stem cell transplantation; CBT, cord blood transplantation; UR-PBSCT, unrelated peripheral blood stem cell transplantation; MMF, mycophenolate mofetil; NR, not reported.

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Importantly, potent immunosuppressive therapies after the allogeneic transplantation may shrink the anti-lymphoma effects of donor immunity and resultantly increase a recurrence risk of malignancies. To date, biologic agents, such as rituximab, abatacept, and tocilizumab, have been launched for the treatment of refractory cases of DM, and an excellent efficacy of B-cell removal with rituximab has been highlighted in conditions with considerable B-cell involvement [11]. In line with this notion, a previous report also showed an excellent efficacy of rituximab in anti-MDA-5 antibody-positive DM [12]. Rituximab is a monoclonal antibody that binds to CD20, a differentiation antigen expressing on the surface of human B lymphocytes, and therefore represents a subtle impact on donor T cells. In this context, rituximab was selected for the treatment of our Case 2, and expectedly, marked improvement in skin rash and ILD was observed.

It is well recognised that patients with anti-MDA-5 antibody-positive DM show a poor prognosis with a frequent combination of RPILD. However, underlying molecular pathophysiology still remains unknown. MDA-5 is one of the retinoic acid-inducible gene I-like receptors, which recognises picornavirus double-stranded RNA and subsequently induces antiviral responses by producing type I interferons and some of the inflammatory cytokines [13]. Intriguingly, a previous report showed a significant association between CADM and HLA-DRB1 01:01/04:05 in the Japanese population [14]. However, the mechanistic link between CADM and allo-HSCT has not yet been fully elucidated.

Although there are few reports of idiopathic autoimmune diseases after allo-HSCT [15], there are many reports of autoantibody-positive cGvHD with systemic lupus erythematosus, Sjögren syndrome, and systemic scleroderma-like symptoms [16–18]. In the literature, DM/polymyositis (PM) after all-HSCT is extremely rare [19–21]. Of the 12 cases of PM, eight were positive for ANA, four for anti-smooth muscle antibodies, and one for anti-mitochondrial antibodies. There were no myositis-specific autoantibodies except for one case with a U4/6 nuclear RNA synthetase complex [21]. DM is even less commonly reported, although positive ANA and anti-ARS antibodies have been reported in some cases [6, 7, 15, 20]. Of note, there were very few reports of anti-MDA-5 antibody-positive autoimmune myositis who were diagnosed after the allo-HSCT [6, 7]. In this context, we summarise the clinical characteristics of previously reported cases with anti-MDA-5 antibodies after the allo-HSCT in Table 2. In a retrospective analysis of 83 cases of chronic cutaneous and pulmonary GvHD, six cases were positive for anti-MDA-5 antibodies, four of which were associated with RPILD [6]. Two of them died, one of them died of PRILD, and the other did not regress from ILD and eventually died of infectious pneumonia [6]. Although specific skin rashes are clue to the diagnosis of DM, non-specific skin rash was also reported in a patient with anti-MDA-5 antibody [6]. It is therefore possible that some of the cases with RPILD after the allo-HSCT were not accurately diagnosed as an anti-MDA-5 antibody positive.

In summary, to avoid such a fatal complication as PRILD after the allo-HSCT, we do underscore the importance of identification of anti-MDA-5 antibody in patients with suspicion of skin or lung cGvHD.

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Conflict of interest

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Patient consent

The patients and their families wrote an informed consent to publish this report.

Ethical approval

Not applicable.

Author contributions

Y.T., M.D., and S.K. treated the patients and provided clinical data. R.M. wrote the manuscript. S.N. and H.M. wrote and edited the manuscript. T.N., S.T., T.H., K.T., K.M., Y.N., S.M., and T.F. reviewed or edited the manuscript and approved the final version of the manuscript for submission.

Data availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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