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Case reports

Subacute cutaneous lupus erythematosus, and positive anti-MDA5 antibody in clinically amyopathic dermatomyositis with rapidly progressive interstitial lung disease: A case report and literature review

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ABSTRACT

Background: Clinically amyopathic dermatomyositis (CADM) is a subtype of DM with characteristic cutaneous lesion with normal creatinine kinase levels. Presence of anti-melanoma differentiation-associated gene 5 (MDA5) antibody is found to be associated with rapidly progressive interstitial lung disease (RP-ILD). *Aim of the work*: to report a CADM patient with positive anti-MDA5 antibody and RP-ILD with clinical features of systemic lupus erythematosus (SLE) who clinically responded to a combination of cyclophosphamide and other conventional immunosuppressant. *Case presentation*: A 44-year-old Indian woman presented with classical cutaneous lesions of DM with normal creatinine kinase levels amongst other clinical features. She was initially diagnosed with SLE before developing RP-ILD and a positive MDA5 antibody. Anti-nuclear antibody, anti-dsDNA and anti-Sm antibody were negative. Serum ferritin level was very high (1599 ng/mL) as compared to C-reactive protein (23.4 mg/L). Anti-Ro-52 and anti-PM-Scl 75 were positive. High resolution computed tomography (HRCT) of the lungs revealed features consistent with ILD. Histology of her skin biopsy was consistent with subacute cutaneous lupus erythematosus (SCLE). Her diagnosis was revised to CADM with overlapping SCLE. She responded to a combination of hydroxychloroquine, cyclosporine-A, mycophenolate mofetil, pulse methylprednisolone and pulse cyclophosphamide 750 mg/month for 6 months. Her cutaneous lesions gradually improved with normalization of serum ferritin level. Repeated HRCT showed no further progression of the pulmonary fibrosis. *Conclusion*: CADM with positive anti-MDA5 antibody associated with RP-ILD is rare with a high mortality rate. Early recognition and prompt treatment with a combination of immunosuppressant may improve the outcome of this complex disease.

1. Introduction

Dermatomyositis (DM) is a rare chronic inflammatory autoimmune disease characterized by classical heliotrope rash, poikiloderma (Shawl's sign), and Gottron's papules with proximal myopathy and raised creatinine kinase (CK) [1]. Clinically amyopathic dermatomyositis (CADM) is a subtype of DM characterized by distinct cutaneous lesions, without abnormally raised CK and profound muscle weakness [2].

Subacute cutaneous lupus erythematosus (SCLE) is one of the subtypes of cutaneous lupus which is highly photosensitive and characterized by papulosquamous (psoriasiform) or annular lesions of similar distribution but with sparing of the face [3,4]. Nevertheless, cutaneous lesions in both SCLE and DM, clinically and histopathologically may be indistinguishable [5].

Interstitial lung disease (ILD) has been known to be a complication of both DM and CADM [6,7]. Rapidly progressive ILD (RP-ILD) however, is more commonly found in CADM patient with positive anti-melanoma differentiation-associated gene 5 (anti-MDA5) antibody [8,9] which is usually associated with a fatal outcome [10].

Corticosteroids is still the mainstay of treatment and combination with conventional immuno-suppressants such as anti-

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malarial, methotrexate, cyclosporine, cyclophosphamide, mycophenolate and biological agents resulted in varying degrees of responses. Anti-CD20 antagonist such as rituximab has been successfully used in refractory cutaneous lesions with RP-ILD especially in anti-MDA5 antibody positive patients with variable outcome [11].

We report a CADM patient with positive anti-MDA5 antibody and RP-ILD with clinical features of systemic lupus erythematosus (SLE) who clinically responded to a combination of cyclophosphamide and other conventional immunosuppressant.

2. Case report

A 44-year-old Indian woman was diagnosed and treated as pneumonia in April 2019 when she presented with dry cough and fever for one month. Septic workout including screening for tuberculosis was negative. Few small solitary axillary nodes were palpable and following biopsy reactive lymphadenitis was found histologically. Plain chest radiograph revealed bilateral peripheral upper zone mild reticular shadowing suggestive of fibrosis. High resolution computed tomography (HRCT) chest showed patchy ground-glass appearance in both lung fields (Fig. 1). She had a few painful erythematous papules which was appear like vasculitic rash on both her palms which progressively worsened and became generalized. She subsequently developed alopecia, polyarthritis, oral ulcer, and mild muscle weakness with intermittent fever over the next month. However, she denied respiratory symptoms, Raynaud's phenomenon, or proximal myopathy. SLE was then diagnosed based on clinical manifestations and hypocomplementemia fulfilling the 2012 classification criteria for SLE [12] and she was given methylprednisolone and hydroxychloroquine. She was making progress, albeit rather gradually. Her skin lesions showed improvement but her alopecia worsened. In December 2019, she was hospitalized and treated for a chest infection. A month later, she developed high grade fever, vomiting, worsening alopecia, myalgia, fatigability, non-productive cough with mild dyspnea on exertion. Her skin lesions, however, remained the same.

Physical examination revealed generalized violaceous scaly erythematous rashes over her whole body, denser over the photo-exposed areas (Shawl's and V-shape sign), and upper limbs. There were heliotrope rash, Gottron's papules, cutaneous ulceration, scaly maculopapular rashes on both palms and periungual edema and telangiectasia observed. (Fig. 2) Mild synovitis over the small joints of both hands, and diffuse alopecia but no discoid or scarring on the scalp were observed. Fine crackles were heard at the bases of both lungs. Other system findings were unremarkable.

The initial laboratory investigations revealed low hemoglobin, 11.9 g/L (normal 11.5–16.5 g/L), thrombocytopenia, 135 $\times$ 10^9/L (normal 150–450 $\times$ 10^9/L), normal white cell and differential counts, erythrocyte sedimentation rate (ESR), 30 mm/hour and C-reactive protein (CRP), 23.4 mg/L. The aspartate and alanine transaminases were initially raised (162 U/L and 159 U/L respectively) and subsequently normalized. The ANA, anti-dsDNA, anti-citrulinated cyclic peptide antibody, rheumatoid factor and extractable nuclear antigen (anti-Jo-1, anti-nRNP/Sm, anti-Sm, anti-Ro, anti-La, anti-Scl-70, anti-PM-Scl 100, anti-centromere protein B, anti-PCNA) were all negative. Anti-Ro-52 and anti-PM-Scl 75 were positive. The C4 level was low, 0.11 g/L (normal 0.15–0.45 g/L) with normal C3 levels. The ILD markers, KL-6 and surfactant protein (SP-A) were not done due to unavailability. Lactate dehydrogenase (LDH) was also elevated (597 U/L) and normalized during the intensive treatment period. The renal profile and urinalysis were unremarkable. CK was 44 U/L (normal 24–173 U/L). Tumor markers (alpha-feto protein, cancer antigen (CA) 15.3, CA 19.9, CA 125, carcinoembryonic antigen (CEA), and quantitative human chorionic gonadotropin (HCG) were also negative. However, ultrasound of the abdomen was not performed, and Papanicolaou (Pap) smear was not consented. Screening for tuberculosis was also negative. Pulmonary function test was not done due to unavailability.

Myositis specific and associated antibodies (MSA and MAS) tests were performed qualitatively, eight months from the initial presentation. These revealed positive anti-MDA5/CADM140 antibodies. Quantitative measurement, however, was not available. Serum ferritin was 1599 ng/mL (normal 14.0–233.1 ng/mL) at this stage. At this juncture, a diagnosis of CADM with positive anti-MDA5/CADM140 overlapped with SCLE complicated by ILD was made.

Skin biopsy histology revealed leukocytoclastic vasculitis with lichenoid reaction and positive immunoglobulins in immunofluorescent study (IgG, IgA, IgM and C3) which was consistent with SCLE (Fig. 3).

Cyclosporine A 75 mg twice daily (5 mg/kg/day), mycophenolate mofetil 1 gm twice daily and oral methylprednisolone 28 mg daily were added. The latter was given upon discharge and completion of intravenous cyclophosphamide. Intravenous methylprednisolone 500 mg daily for 2 days was given whilst she was hospitalized, followed by pulse intravenous cyclophosphamide 750 mg (500 mg/m^2 body surface area), given monthly for 6 months without serious adverse events. There was no evidence of renal impairment, hemorrhagic cystitis and marrow suppression throughout the intensive treatment period (close monitoring engaged). The serum ferritin reduced to below 300 ng/mL and the CRP remained normal. Clinical signs and symptoms (cutaneous lesions, alopecia, and general wellbeing) markedly improved.

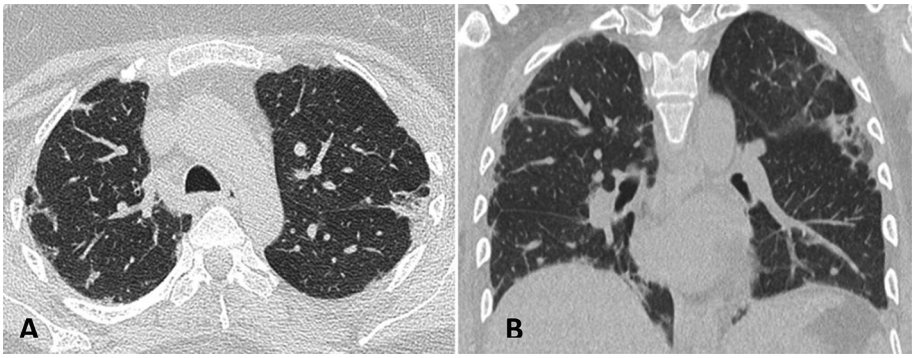

Fig. 1. High resolution computerized tomography (HRCT) chest axial view (A) coronal anterior view (B) showed patchy ground-glass appearance in both lung fields, more extensive in the periphery of the left lung and characteristic predominance of abnormalities in subpleural and basal regions.

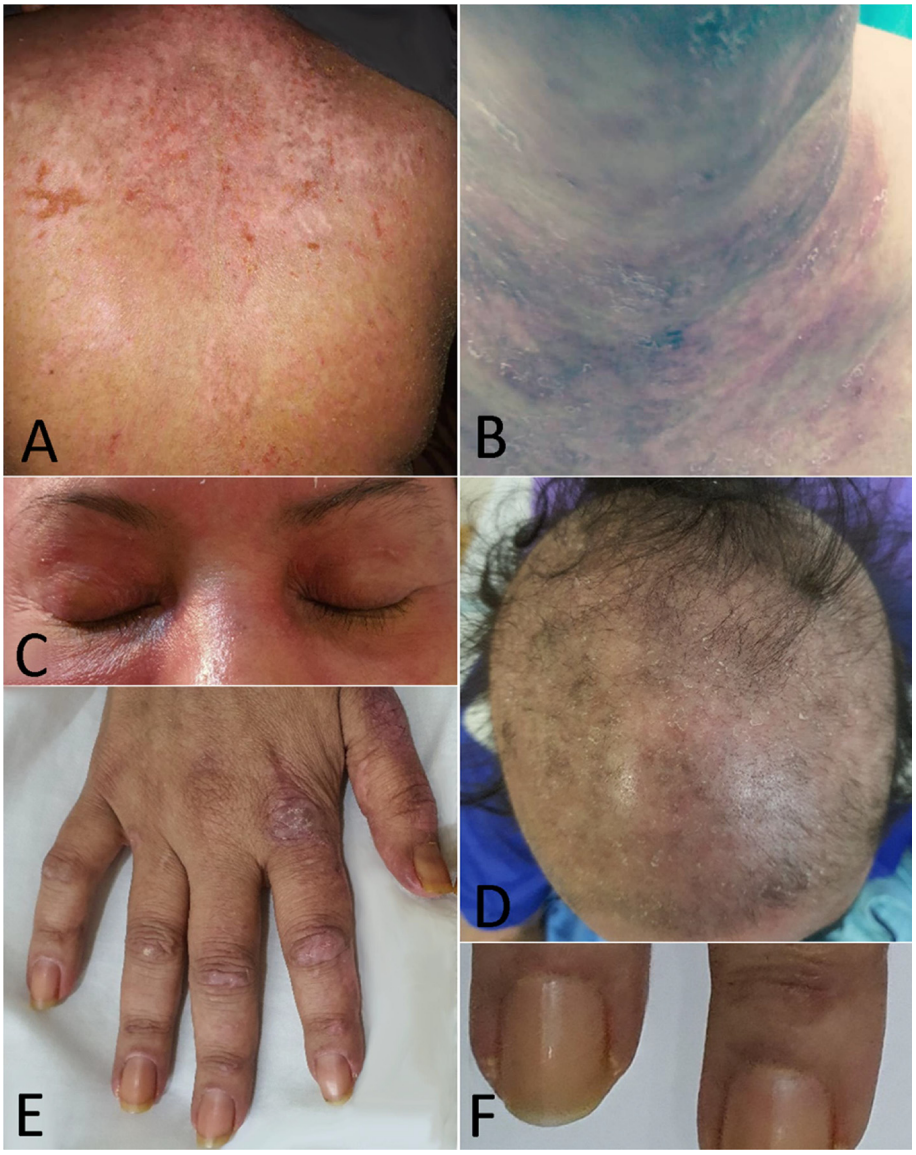

Fig. 2. Clinical characteristics of the patient. (A)Shawl and (B) V-shape signs with poikilodermic scaly erythematous rash, (C) Heliotrope rash, (D) Alopecia with erythematous scaly rash of the scalp. (E) and (F) Gottron's papules, periungual telangiectasia and edema.

Repeated HRCT after 5th cycle of intravenous cyclophosphamide showed improvement of the peripheral subpleural line (Fig. 4).

3. Discussion

Anti-MDA5 antibody is the subset of MSAs autoantibodies which is specifically found in idiopathic inflammatory myositis (IIM). Higher frequency of this antibody was observed in Asian population [13] with high mortality rate [14,15]. Anti-MDA5 antibody is significantly associated with RP-ILD, and characterized by hand swelling, arthritis, spectrum of cutaneous lesions [16,17] and more common in CADM [14]. Cutaneous lupus may exhibit similar lesions but lacks classical features of CADM. Heterogeneous clinical phenotypes and imaging findings of CADM/DM with different outcomes has been previously described [7–9,11,18–23] (Table 1).

The histopathological findings in SCLE and DM may be indistinguishable when a dermal deposit of mucin is present in both conditions. Thus, DM is frequently considered among the differential diagnosis of SCLE [5]. The presence of anti-Ro-52 antibody in earlier assessment of this case lead to a clear association with SCLE despite negative ANA in accordance to a previous study. [24]

Inverse relationship between hyperferritinaemia and CRP is a predictor of RP-ILD and the former has been considered as poor prognostic factor with levels $\geq$ 828 ng/mL in patient with positive anti-MDA5 antibody [25,26] The level of serum ferritin in this patient was markedly high as compared to CRP levels and improved with the treatment regime given.

This patient presented with the above mentioned characteristic cutaneous manifestations which mimics both SCLE and DM. RP-ILD was only diagnosed a year after her first presentation with characteristic CADM cutaneous lesions and a positive anti-MDA5 antibody. Though the initial HRCT chest revealed bilateral ground-glass appearance, the diagnosis was delayed as the myositis serological panel was performed later. This serological panel testing is not routinely performed in ILD cases without classical cutaneous manifestation as it is not widely available in Malaysia.

Indeed there are various causes of ILD and many may present with an indistinguishable radiological features. The typical findings of ILD are reticular opacities and honey combing which are sited

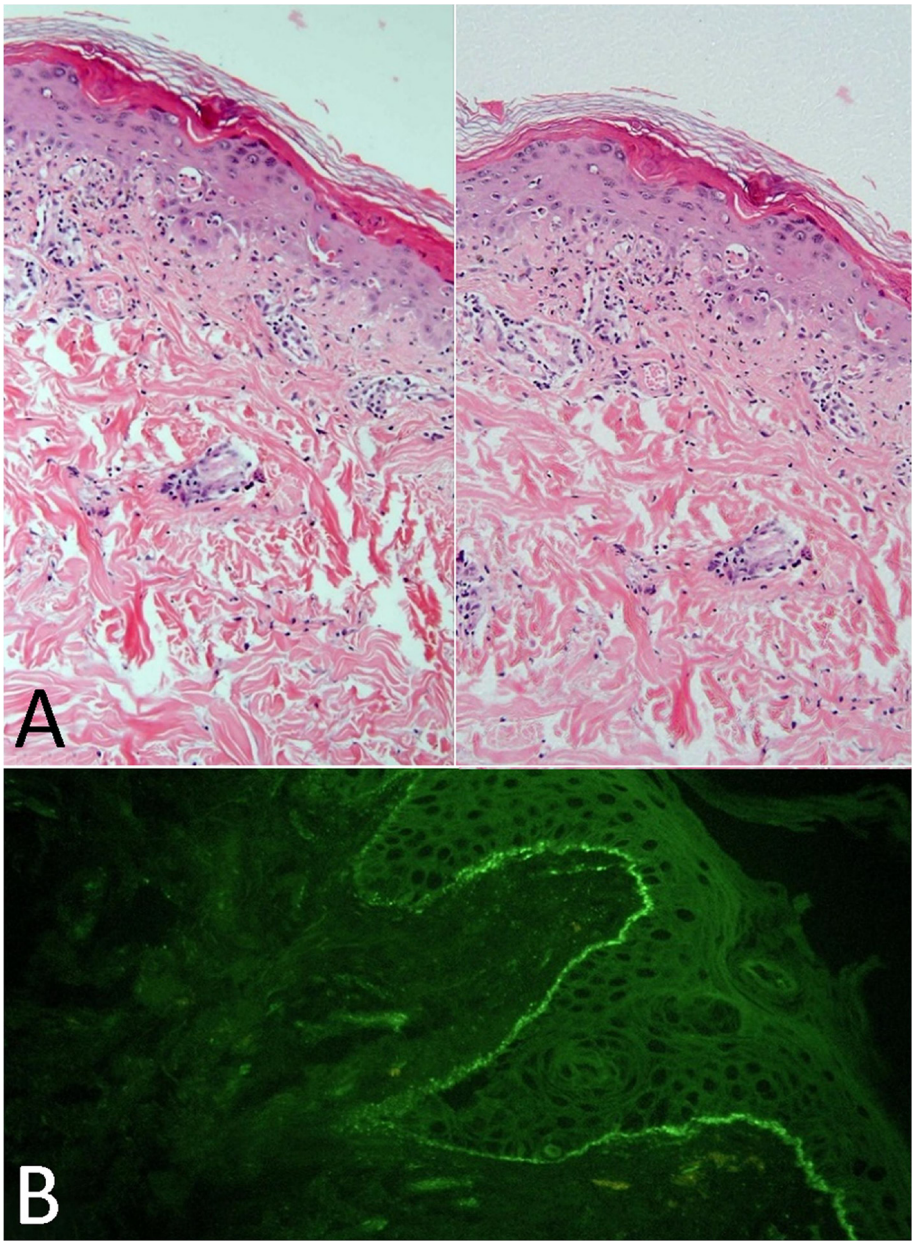

Fig. 3. Histopathological examination from skin biopsy showing (A) Leucocytoclastic vasculitis and lichenoid reaction consistent with subacute lupus erythematosus. (B) Immunofluorescence studies showing granular positivity at the dermo-epidermal junction and blood vessels for IgG, IgA, IgM and C3.

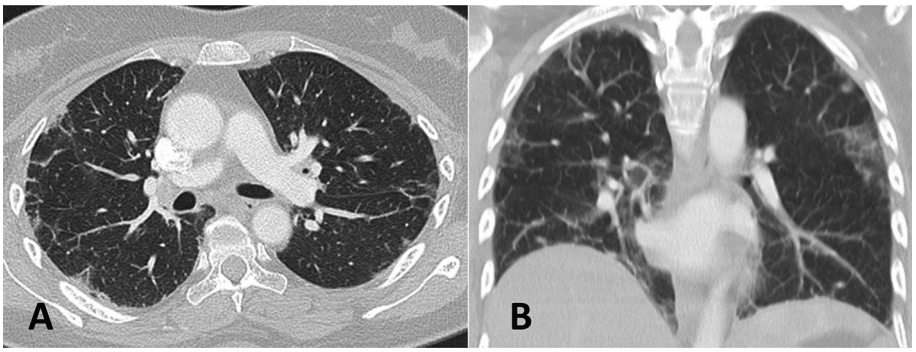

Fig. 4. High resolution computerized tomography (HRCT) chest axial view (A) coronal anterior view (B) three months later post treatment. There is marked improvement in the opacity at the periphery of the left lung. The coronal reformatted image shows resolution of the ground glass opacities with some residual fibrosis at the base of the right lung.

Table 1
Summary of clinical presentation of dermatomyositis (DM)/clinically amyopathic dermatomyositis (CADM) with positive anti-MDA5-antibody and rapidly progressive interstitial lung disease (RP-ILD) case reports.

Study	Country	Sex; Age	Clinical features of CADM/DM	HRCT	CK (U/L)	Ferritin (ng/mL)	Treatment	Outcome
Gonzalez et al [7]	Spain	F; 54	Polyarthritis, dyspnea	Peripheral GGO	Normal	327.7	MP, CYC, CyA	Survived
Aoyama et al [8]	Japan	M; 47	Unresolved cough, fever	Random focal areas GGO	673	1122	MP, CYC, TAC	Died
Huang and Mao [9]	China	M; 39	Right orbital heliotrope, progressive dyspnea	Patchy GGO (middle/upper), interstitial lesion, bilateral PE	Normal	6735	MP, HCQ, IVIg	Died
Koichi et al. [11]	Japan	F; 71	Heliotrope rash; Gottron's papules; buttock ulcers; palmar papules	Peripheral GGO (lower)	211	1782.8	MP, P, TAC, CYC, IVIg, RTX	Survived
Sato et al [18]	Japan	M; 59	Fever, Gottron's papules, periungual erythema, Shawl sign, scaling erythema (knees/elbows)	Consolidation, traction bronchiectasis	303	n.d.	MP, P, CYC, CyA, TAC, AZA	Survived
DeBacker et al [19]	Belgium	M; 55	Fatigue, polyarthritis, Gottron's, mechanics hand, ear ulcer, fever, weight loss, cough, dyspnea	Pulmonary infiltrates (subpleural/peribronchial), atelectasis, honey-combing	14	1669	MP, CYC	Died
Ogawa et al [20]	Japan	M; 48	Fever, anorexia, arthralgia/myopathy, dyspnea, heliotrope, Gottron's/palmar papules, mechanics hands, erythema	Reticular shadows, GGO (middle/lower)	278	781	MP, P, CyA, CYC, RTX	Survived
Yashiro et al. [21]	Japan	F; 51	Palmar/ periungual erythema, proximal myopathy, Gottron's	Consolidation, GGO (all lung)	275	975	MP, P, CYC	Survived
Pacot et al. [22]	France	F; 59 M; 51	Facial erythema, arthralgias, erosion Dyspnea (Case2)	- IPF (case2)	4100 normal	n.d	MP, CYC Case2: MP, CYC, PLPH, Lung transplant, CyA, MMF, TAC	Died Survived
Sakamoto et al [23]	Japan	2F,1M; 68–72	Fatigue, dyspnea	Consolidation, traction bronchiectasis	183/140/105	1486/235/1428	MP, P, CYC, CyA, TAC	Died
This study	Malaysia	F; 44	Fever, cough, dyspnea, polyarthritis, alopecia, oral ulcer, heliotrope rash, Gottron's/palmar papules, periungual telangiectasia	Peripheral GCO, basal fibrosis	44	1599	MP, P, CYC, CyA, MMF	Survived

DM: dermatomyositis, CADM: clinically amyopathic dermatomyositis, F: female, M: male, CK: creatine kinase, GGO: ground-glass opacity, PE: pleural effusion, IPF: interstitial pulmonary fibrosis, MP: methyl prednisolone, P: prednisolone, CyA: cyclosporine, TAC: taclorimus, CYC: cyclophosphamide, RTX: rituximab, IVIg: intravenous immunoglobulin, MMF: mycophenolate mofetil, AZA: azathioprine, PLPH: plasmapheresis, n.d.: not documented.

peripherally and basally. The latter is usually a feature of end stage parenchymal fibrosis which are seen in the posterior and basal lung zones, in the subpleural region and associated with poor prognosis. It is an important monitoring tool and indicator of the severity as well as the progress of the disease and treatment responses. The CT findings correlate strongly with the lung function tests early in the disease process rather than the histological findings. Clinical history and diagnostic tool such as pulmonary function test may not be able to determine the significant serial HRCT findings. ILD is an unpredictable disease. Some patients remain stable for long periods, in some patients a progressive decline in respiratory status will be seen, and in a third group acute exacerbations with high mortality risk may be seen [27]. This patient showed marked improvement clinically and radiologically after treatment.

HRCT findings of ground glass opacities and honeycombing are also seen in asymptomatic elderly patients which are mostly associated with smokers. So these findings may not always represent clinically relevant disease. Nevertheless, the presence of honey combing in a CT study is required for diagnosis of ILD. However, indistinguishable features between honey combing and traction bronchiectasis may subject patient to unnecessary surgical biopsy. Changes on CT imaging alone would not be an indication of reversibility of the disease. Ground glass opacities, which typically associated with inflammation, need not represent reversible lung disease, in fact may be due to microscopic fibrosis. Irregular reticulation normally associated with fibrosis can sometimes be reversible – so this component should resolve on serial HRCT after appropriate treatment. A structured multi-disciplinary approach comprising of a radiologist, pulmonologist and the clinician in-charge is the recommended method in dealing with complex ILD cases [28]. However, a dedicated ILD multi-disciplinary team is not available in our hospital setting. Therefore, clinical information remains an important and useful element, coupled with auxiliary investigations such as serology and histology to determine the ILD etiology. This is the case in this patient where the diagnosis was eventually clinched based on characteristic cutaneous lesions and a supportive serological profile (anti-MDA5 antibody positive). Clinical and radiological improvement with combination immunesuppressants supports the diagnosis of anti-MDA5 antibody positive connective tissue disease as the etiology of this patient's ILD.

In most reported cases previously, patients presented with respiratory failure within a short duration, requiring ventilatory support with a high mortality rate despite various treatment intervention. Variable responses were observed using steroids, cyclosporine, mycophenolate, cyclophosphamide, plasma exchange, intravenous immunoglobulin and rituximab at this stage [29,30]. Quantitative measurement of the level of anti-MDA5 antibody is important in monitoring the progress of RP-ILD [31] which is not available in our setting. In contrast, the progress of ILD devel-

opment in this patient was very gradual presumably secondary to early institution of immunosuppressant.

In conclusion, diagnosis and treatment of anti-MDA5 antibody positive with RP-ILD is daunting and challenging in CADM patient especially with the co-existence of SCLE which is rarely described. In favorable settings, MSA testing should be considered in patients with ILD despite the absence of cutaneous manifestations. This is because, early intervention with immunosuppressant with or without biologic may halt the progression of the ILD.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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