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CASE REPORT

Baricitinib Combination Therapy Demonstrates Significant Improvement in Cardiac Conduction Defects in Rapidly Progressive Systemic Sclerosis: A Case Report

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Objective: To evaluate the efficacy of baricitinib in combination therapy for managing refractory, rapidly progressive systemic sclerosis (SSc) with severe cardiac conduction defects and interstitial lung disease (ILD).

Methods: A 48-year-old male patient with SSc complicated by significant cardiac enlargement, third-degree atrioventricular block, heart failure, progressive ILD, and partial intestinal obstruction was included in the study. Prior treatments with mycophenolate mofetil (MMF), tacrolimus, and cyclophosphamide (CTX) had shown limited efficacy. The patient subsequently received a combination regimen of glucocorticoids, intravenous immunoglobulins, CTX, and baricitinib (4 mg daily).

Results: The patient exhibited significant clinical improvements, including a reduction in cardiac size, restoration of sinus rhythm, and resolution of heart failure symptoms. ILD and skin sclerosis showed substantial regression. Pulmonary function tests indicated significant recovery in lung capacity and diffusion capacity. Additionally, gastrointestinal symptoms such as abdominal pain and bloating were completely resolved.

Conclusion: This case highlights the potential of baricitinib as an adjunctive therapy for refractory SSc with multiorgan involvement. The observed improvements in cardiac conduction defects, ILD, and skin fibrosis suggest that JAK inhibitors may offer a promising therapeutic avenue for severe SSc cases resistant to conventional treatments.

Keywords: systemic sclerosis, cardiac conduction defects, interstitial lung disease, baricitinib, case report

Introduction

Systemic sclerosis (SSc) is a chronic autoimmune disorder characterized by progressive skin fibrosis and multiorgan dysfunction, commonly involving the lungs, heart, gastrointestinal tract, and kidneys.[1] Cardiac involvement, though frequently clinically silent in early stages, can manifest as arrhythmias, heart failure, or myocardial injury and is associated with a poor prognosis.[2] Target therapy for primary cardiac disease in SSc is so far not well established. Inhibitor of IL-1 and IL-6 are under investigation.[3] We report a case of rapidly progressive SSc complicated by severe cardiac enlargement, third-degree atrioventricular block, decompensated heart failure, and progressive interstitial lung disease (ILD).

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Case Description

A 48-year-old male presented in December, 2022, with fever, cough, dyspnea, fatigue, and Raynaud's phenomenon (oxygen saturation: 90%). Chest high-resolution CT (HRCT) demonstrated multiple ground-glass opacities (GGOs) in both lungs, predominantly involving the lower lobes and affecting approximately 25% of the lung parenchyma. The bronchi appeared patent with normal course, and air bronchograms were visible, consistent with viral pneumonia (Figure 1A). Initially diagnosed with COVID-19, he received nirmatrelvir/ritonavir (Paxlovid) and human albumin infusion. Despite treatment, symptoms persisted, prompting admission to a tertiary hospital for piperacillin-tazobactam and Intravenous immunoglobulin (IVIG) (70 g cumulative).

In April, 2023, diagnostic workup revealed: Positive ANA (1:320, cytoplasmic granular pattern; quantitative 204.09 U/mL); Strong anti-SS-A antibodies (+++) and Ro52 (++); Elevated KL-6 (2516 U/mL); Lung biopsy confirming usual interstitial pneumonia (UIP). The patient was diagnosed with SSc-associated ILD and initiated on methylprednisolone (16 mg qd), mycophenolate mofetil (MMF) (1g bid), tacrolimus (1g bid), and nintedanib. While initial response was favorable, by December 2023 he developed progressive cutaneous sclerosis (face, forearms, hands), worsened Raynaud's phenomenon, exertional dyspnea, and severe gastrointestinal symptoms (nausea, abdominal pain, distention). Oral Cyclophosphamide (CYC) was attempted but discontinued due to intolerance (nausea and vomiting).

In February 2024, the patient was admitted to our Rheumatology and Immunology department with a 15-month history of recurrent cough and dyspnea, accompanied by 4 months of progressive skin induration and abdominal distension/pain. He had no history of smoking, alcohol use, or family autoimmune disorders. Physical Examination: Vital signs: Temp 36.5°C, HR 74 bpm, RR 19/min, BP 132/74 mmHg. Cutaneous findings: Severe sclerodactyly (face, forearms, hands, abdomen), modified Rodnan Skin Score (mRSS) was 23, active Raynaud's phenomenon. Pulmonary: Bilateral basal Velcro crackles; Cardiovascular: Regular rhythm, no murmurs; Abdomen: non-tender, no rebound. Laboratory tests showed: Complete blood count: Hemoglobin (Hgb) 144.00 g/L (reference: 110–170), platelet count (PLT) 464.00×10^9/L (reference: 100–300), white blood cell count (WBC) 12.55×10^9/L (reference: 4–10 L); Biochemistry: Albumin 39.2 g/L (reference: 40–50), ALT 44 U/L (reference: 9–50), AST 63 U/L (reference: 15–40),

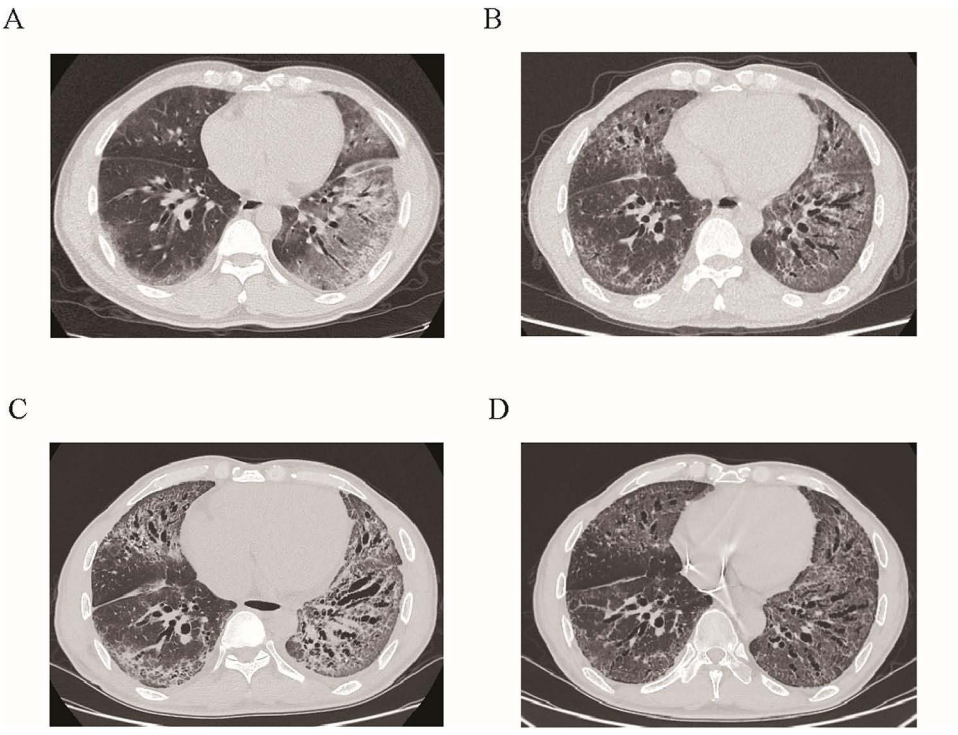

Figure 1 Chest-enhanced CT before and after treatment (**A**–**D**). (**A**) Bilateral pulmonary exudative lesions. (**B**) Diffuse interstitial changes and increased inflammatory lesions in both lungs. (**C**) Extensive progression of interstitial fibrosis and inflammation. (**D**) Decreased interstitial fibrosis and inflammation.

creatine kinase (CK) 1155 U/L (reference: 50–310), CK-MB 78 U/L (reference: 0–24), NT-proBNP 120 ng/L (reference: 0–73), troponin I 0.032 μg/L (reference: 0–0.034); IL-6 12.38 pg/mL (reference: ≤ 5.04). Autoantibodies: weakly positive ANA (immunofluorescence) at 1:100 with a cytoplasmic granular pattern; anti-SS-A52 and anti-SS-A60 positive; anti-SCL-70, anti-PM-SCL and anti-centromere antibody (ACA) negative. All anti-myositis antibodies (including anti-Jo-1, anti-MDA5, anti-TIF1, SAE1/2, Mi-2, SRP and so on) negative. Pulmonary function tests: 1. Moderate restrictive ventilatory impairment, with MVV (Maximal Voluntary Ventilation) at 72.2% of predicted; 2. moderate diffusion dysfunction, moderate reduction in total lung capacity (Table 1); Echocardiogram: Enlarged left atrium and ventricle, with reduced left ventricular diastolic function (Table 2); On follow-up, chest HRCT revealed increased reticular markings and multiple patchy areas of consolidation in both lungs, with approximately 60% of the lung parenchyma involved. Subpleural sparing was evident, accompanied by bronchial traction and dilatation, suggestive of interstitial pneumonia (Figure 1B). Abdominal MRI: No significant abnormalities; the bronchoalveolar lavage fluid Next Generation Sequencing (NGS) results revealed no pathogenic microorganisms. These findings fulfilled the 2013 ACR/EULAR classification criteria for SSc.[4] The final diagnosis was SSc, ILD, and heart failure. Despite elevated CK levels, dermatomyositis was excluded due to: absence of significant limb weakness and negative myositis-specific antibodies. Treatment initiated: CYC 1.2 g monthly, methylprednisolone 80 mg daily for 3 days; and pirfenidone (antifibrotic therapy). In April 9, 2024, the patient pulmonary function tests indicated moderate restrictive ventilatory impairment (MVV 75.4% of predicted), moderate diffusion dysfunction, severe reduction in total lung capacity (Table 1). ECG showing thrid-degree avtrioventricular block, junctional escape rhythm with complete right bundle block (Figure 2A).

In April 2024, the patient was hospitalized for persistent palpitations and chest tightness lasting three weeks. Physical examination revealed progressive cutaneous fibrosis involving the face, upper extremities, and abdomen. Arterial blood

Table 1 Pulmonary Function Test

	2024-03-04	2024-04-09	2024-05-21	2024-06-17	2024-07-23
MVV (L/min)	72.20%	75.40%	-	70.20%	98.50%
FVC (L)	3.14	2.66	2.29	2.36	2.85
FEV1 (L)	2.98	2.44	2.26	2.31	2.80
FEV1%FVC (%)	95.01	91.7	98.92	97.82	98.19
PEF (L/s)	10.98	8.57	6.67	8.08	9.69
MEF75 (L/s)	10.98	8.57	6.67	8.06	9.69
MEF50 (L/s)	8.43	6.86	5.33	6.97	8.68
MEF25 (L/s)	2.11	1.68	2.84	3.96	3.11
MEF75/25 (L/s)	6.06	4.68	4.74	6.32	6.78
TLC (L)	4.61	4.07	4.42	4.45	5.53
RV (L)	1.08	1.69	2.4	2.25	2.88
VC MAX (l)	3.14	2.66	2.29	2.36	2.85
RV%TLC-SB (%)	40.27	41.41	54.31	50.60	33.07
FRC (L)	2.92	2.72	3.28	3.27	4.20
DLCO (mmol/min/kpa)	4.64	4.77	5.45	4.46	9.13
DLCO/VA (mmol/min/kpa/L)	1.04	1.21	1.27	1.03	1.69

Abbreviations: MVV, Maximal Voluntary Ventilation; FVC, Forced Vital Capacity; FEV1, Forced Expiratory Volume in 1 second.

Table 2 Ultrasonic Cardiogram

	2022-11-01	2023-02-10	2023-07-18	2024-03-01	2024-04-23	2024-05-27	2024-06-17	2024-08-20
LA (mm)	33	26	35	39	40	34	34	23
RV (mm)	21	19	21	24	21	20	20	25
LV (mm)	50	51	52	53	54	47	47	40
RA (mm)	45*42	44*44	42*41	44*40	49*41	47*43	48*41	44*40
LVEF (%)	61%	64%	64%	60%	64%	66%	60%	63%

Abbreviations: LA, Left Atrium; RV, Right Ventricle; LV, Left Ventricle; RA, Right Atrium; LVEF, Left Ventricular Ejection Fraction.

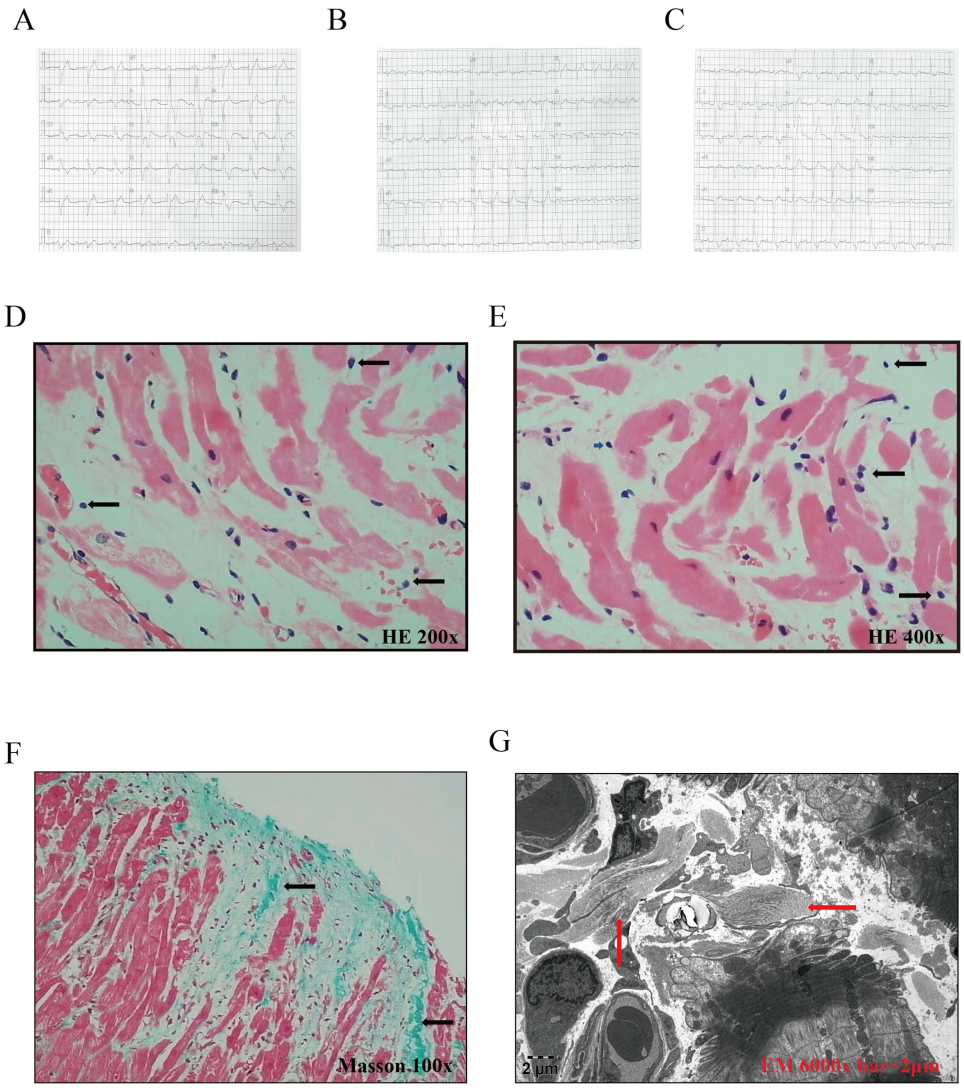

Figure 2 ECG and transcatheter endomyocardial biopsy findings (**A**–**G**). (**A**) ECG showing third-degree avtrioventricular block, junctional escape rhythm with complete right bundle block. (**B**) ECG showing dual-chamber pacing (DDD mode), the sensing and pacing functions were normal. DDD pacing mode means the pacemaker senses the electrical signal from both the atrium and ventricle, and paces both atrium and ventricle in atrial-ventricle sequence. (**C**) ECG showing sinus rhythm with ventricular pacing in VAT mode. VAT stands for the pacing mode of pacemaker. V represents the ventricle, A represents the atrium, and T represents trigger. So VAT pacing mode means: after sensing the electrical signal from the atrium (A), the pacemaker will trigger (T) an electrical impulse to pace the ventricle (V). (**D**-**F**) Transcatheter endomyocardial biopsy showing interstitial fibrosis with lymphocytic myocarditis (**D** and **E**) black arrows indicate lymphocyte infiltration, (**F**) black arrow indicates Masson staining, collagen fibers are green). (**G**) Electron microscopy demonstrating focal collagen deposition in the myocardial interstitium (red arrow indicates intercellular collagen fiber bundles in cardiomyocytes).

gas analysis showed: elevated PCO_2: 6.08 kPa (reference: 4.65–5.98), reduced PO_2: 10.48 kPa (reference: 10.64–13.3), and oxygen saturation of 95.9% (reference: 95–98%). CK 906 U/L, NT-proBNP 274 ng/L, and D-dimer 1180 μg/L. Subsequent chest HRCT showed diffuse GGOs with widespread interlobular septal thickening. Compared with the previous scan, both the extent and number of lesions had progressed, involving roughly 80% of the lung parenchyma. Bronchiectasis was more pronounced, and small bilateral pleural effusions were present (Figure 1C). Echocardiography showed enlargement of the left atrium and ventricle, mild pulmonary hypertension, impaired left ventricular diastolic function, and minimal pericardial effusion (Table 2). A transcatheter endomyocardial biopsy showed interstitial fibrosis with lymphocytic myocarditis (Figure 2D–F) (D and E black arrows indicate lymphocyte infiltration, F black arrow indicates Masson staining, collagen fibers are green). Electron microscopy confirmed focal collagen deposition within the myocardial interstitium (Figure 2G) (G red arrow indicates intercellular collagen fiber bundles in cardiomyocytes). After excluding viral myocarditis, CYC-induced cardiotoxicity, and other potential etiologies, these findings were consistent with SSc-related cardiac involvement. The final diagnosis included: SSc; SSc-related cardiac involvement: third-degree

atrioventricular block, complete right bundle branch block, left atrial and ventricular enlargement, and heart failure with preserved ejection fraction (NYHA class III); SSc-related ILD; Incomplete intestinal obstruction. The patient successfully underwent permanent dual-chamber pacemaker implantation. Given evidence of active SSc affecting multiple organ systems (ILD, cardiac dysfunction, and gastrointestinal dysmotility), we initiated the following combined immunosuppressive and antifibrotic regimen: methylprednisolone (500 mg daily for 3 days, followed by a tapering oral dose 48 mg qd); IVIG (0.4 g/kg/day, administered for 5 days per month, and continued 6 months); CYC was administered according to the National Institutes of Health (NIH) protocol (1.2 g monthly); baricitinib (4 mg qd).

In August, 2024, Laboratory tests showed: complete blood count (Hgb 152.00 g/L, PLT 365.00×10^9/L, WBC 13.27×10^9/L); Albumin 40.4 g/L, CK 72 U/L, CK-MB 54 U/L, ALT 32 U/L, CREA 54.5 umol/L, eGFR 116.8 mL/mim/1.73 m2, and NT-proBNP 27 pg/mL. A later Chest HRCT demonstrated marked reduction in GGOs compared with the prior study, with approximately 50% of the lung parenchyma affected. Interlobular septal thickening and bronchial traction dilatation had improved (Figure 1D). Pulmonary function tests showed mild restrictive ventilatory impairment, with MVV at 98.5% of the predicted value, normal diffusion capacity, and mild reduction in total lung capacity (Table 1). ECG showing dual-chamber pacing (DDD mode), the sensing and pacing functions were normal (Figure 2B). Echocardiography showed mild pulmonary hypertension (PH), LVEF 60%, left ventricular diastolic function was impaired (Table 2). Gastroscopy performed revealed: chronic superficial gastritis with erosion, duodenal bulb ulcer (S2). Colonoscopy showed no abnormalities in the colonic mucosa. By September 2024, the ECG revealed sinus rhythm with ventricular pacing in VAT mode (Figure 2C). The patient subsequently regained spontaneous sinus rhythm with marked improvement in cardiac function. The patient achieved complete resolution of cutaneous manifestations, with the mRSS improving from 23 at baseline to 0 at the 12-month follow-up. The treatment regimen was well tolerated, with no occurrence of severe infections or clinically significant laboratory abnormalities during the observation period. The patient continued to receive combination therapy with baricitinib (2 mg qd), methylprednisolone (4 mg qd), and CYC.

Discussion

This case describes a case of refractory SSc with multiple complications, including rapidly progressive ILD, severe cardiac involvement, and incomplete intestinal obstruction. The patient initially developed ILD, Raynaud's phenomenon, and skin thickening following COVID-19 infection. Treatment with glucocorticoids, MMF, tacrolimus and IVIG temporarily improved ILD but failed to prevent relapse, which manifested as worsening ILD, advanced skin sclerosis, and new-onset severe cardiac and gastrointestinal involvement. Switching to CYC 2.4 g alone yielded suboptimal outcomes. Ultimately, combination therapy with baricitinib, methylprednisolone, IVIG, and CYC resulted in marked improvement.

Cardiac Involvement in SSc

SSc-related cardiac involvement is relatively uncommon; however, once the heart is affected, multiple structures—including the coronary arteries, myocardium, conduction system, pericardium, and valves—may be involved, leading to myocardial ischemia, heart failure, arrhythmias, pericardial effusion, and valvular dysfunction. Cardiac involvement in SSc often portends a poor prognosis, with significantly increased mortality in patients presenting with myocardial disease or conduction abnormalities.[5,6] The primary pathological changes in SSc-associated myocardial involvement include myocardial fibrosis and myocarditis. The underlying mechanisms involve microvascular ischemia, chronic inflammation, and progressive myocardial fibrosis, ultimately resulting in systolic and diastolic dysfunction and various types of arrhythmias.[7] Clinically, patients may present with dyspnea, chest tightness, palpitations, or edema; progression to heart failure requires prompt treatment. Currently, only a few case reports have documented the use of MMF, CYC, tocilizumab, and rituximab for SSc-related cardiac involvement.[8–11] High-dose corticosteroids, CYC, and MMF remain the mainstays of therapy, especially in severe organ involvement such as interstitial lung disease and myocarditis.[12] However, clinical response varies greatly among individuals, and drug toxicity is not negligible. In our study, echocardiography showed enlargement of the left atrium and ventricle, impaired left ventricular diastolic function. Endomyocardial biopsy revealed interstitial fibrosis with lymphocytic myocarditis. Electron microscopy confirmed focal collagen deposition in the myocardial interstitium. This patient was diagnosed with SSc-related cardiac involvement: third-degree atrioventricular block, complete right bundle branch block, left atrial and ventricular enlargement, and heart failure with preserved ejection fraction (NYHA class

III). Due to the unavailability of tocilizumab in our hospital and the patient's severe ILD, we selected baricitinib as the alternative therapy. In this case, cardiac involvement continued to worsen despite MMF and CYC therapy; only after the addition of baricitinib, methylprednisolone, and IVIG for 3 months did the heart size diminish substantially. By the fourth month of treatment, the patient regained spontaneous sinus rhythm with significant improvement in cardiac abnormalities, achieving NYHA class I cardiac function.

Treatment of Progressive ILD

Progressive ILD, occurring in approximately 25–30% of cases, often progresses to severe restrictive disease within five years. First-line therapies such as MMF and CYC were ineffective in this patient. Significant improvement followed the addition of baricitinib (4 mg daily), a Janus kinase (JAK) inhibitor with anti-inflammatory and antifibrotic properties. Evidence suggests JAK inhibitors may be effective in refractory ILD.[13], complementing therapies like tocilizumab[14] and rituximab.[11] JAK inhibitor was promising in the improvement of the sclerosis and early radiological abnormalities in SSc-ILD patients.[15] This case highlights the need for aggressive treatment of refractory ILD, with JAK inhibitors emerging as potential alternatives when standard therapies fail.

The patient's intestinal obstruction and gastrointestinal symptoms, initially resistant to prokinetic agents, resolved with IVIG and baricitinib combination therapy. Weight loss reversed, and skin thickening improved from severe to mild.

JAK inhibitors (eg, baricitinib) exert immunomodulatory effects by blocking the JAK-STAT signaling pathways of multiple cytokines—including IL-6, IFN-γ, and GM-CSF—which are key mediators in various autoimmune and inflammatory diseases.[16] Study in animal models and SSc patients suggest that JAK inhibitors can reduce fibroblast activation and inhibit excessive extracellular matrix production, thereby alleviating both inflammation and fibrosis.[17] To date, no reports have addressed the use of JAK inhibitors in severe SSc-related cardiac involvement.

In this case, the addition of baricitinib demonstrated marked improvements in cardiac function, cardiac structure, and ILD. These findings suggest that baricitinib may exert synergistic effects by simultaneously modulating anti-inflammatory and antifibrotic pathways, particularly attenuating inflammatory myocardial injury. The observed outcomes further indicate that JAK inhibitor-based combination therapy may hold broader therapeutic potential for addressing both fibrotic and inflammatory manifestations in SSc. Further studies are warranted to validate these findings and explore the broader applicability of JAK inhibitors in SSc management. No severe infections or significant laboratory abnormalities were observed during the one-year follow-up. The limitations of this study include the lack of a control group, the single-case design, and limited follow-up period, which restrict generalizability. Future multicenter studies with extended observation are needed to validate these findings.

Conclusion

This case highlights the potential of baricitinib combination therapy as an adjunctive therapy for refractory SSc with multiorgan involvement. The observed improvements in cardiac conduction defects, ILD, and skin fibrosis suggest that JAK inhibitors may offer a promising therapeutic avenue for severe SSc cases resistant to conventional treatments.

Ethics Approval and Informed Consent

This study was approved by the Ethics Committee of First Affiliated Hospital, Jinan University. The patient provided written informed consent. The consent for publication has been obtained from the patient. Institutional approval was not required for the publication of the patient's case details.

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Disclosure

The authors declare that they have no competing interests in this work.

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