Pediatric Rheumatology

CASE REPORT

Open Access

Efficacy of combined JAK1/2 inhibition and cyclosporine in paediatric-onset Still's disease with lung involvement: a case report

Serena Palmeri[1,2], Maddalena Lancieri[2], Jacopo Ferro[3], Pietro Salvati[4], Martino Cheli[5], Riccardo Papa[1], Silvia Rosina[1], Stefano Volpi[1,2], Valerio Gaetano Vellone[3,6], Angelo Ravelli[2,7], Marco Gattorno[1] and Roberta Caorsi[1*]

Abstract

Background Paediatric-onset Still's disease is a severe autoinflammatory disorder marked by high fever, systemic inflammation, rash, organomegaly, and arthritis. Pulmonary complications, once anecdotally reported, have been increasingly observed in paediatric Still's disease. A more severe lung involvement, Still-associated lung disease, has emerged in the last ten years, including interstitial lung disease, pulmonary hypertension, and pulmonary alveolar proteinosis (PAP). This complication carries significant morbidity and mortality. Its mechanism remains poorly understood; current biologic therapies targeting interleukin (IL)-1 and IL-6 often prove insufficient, supporting the need for innovative approaches or combined treatment.

Case presentation A 10-year-old girl with Still's disease developed PAP and pulmonary hypertension. After several treatment failures and ongoing oxygen dependency, she responded favourably to a combination of baricitinib and cyclosporine. This allowed for gradual corticosteroid tapering and eventual discontinuation, achieving sustained disease control without further oxygen supplementation. Pulmonary assessment, echocardiography, cardiac catheterization, bronchoalveolar lavage, and lung biopsy were performed, along with immunological profiling, HLA typing, and genetic analysis. The type I IFN signature was closely monitored, with normalization observed after reaching the therapeutic dose of baricitinib.

Conclusion This case provides evidence of the long-term efficacy and safety of baricitinib combined with cyclosporine in managing Still-LD, offering a promising option for this severe complication.

Keywords Still disease, Systemic juvenile idiopathic arthritis, sJIA lung disease, JAK inhibitor, Cyclosporine, Case report, Baricitinib

*Correspondence:
Roberta Caorsi
robertacaorsi@gaslini.org
[1]Rheumatology and Autoinflammatory Diseases Unit, IRCCS Istituto Giannina Gaslini, Genoa, Italy
[2]Department of Neurosciences, Rehabilitation, Ophthalmology, Genetics, Maternal and Child Health (DINOGMI), University of Genoa, Genoa, Italy
[3]Pathology Unit, IRCCS Istituto Giannina Gaslini, Genoa, Italy
[4]Pulmonary Disease and Respiratory Endoscopy Unit, IRCCS Giannina Gaslini, Genoa, Italy
[5]Cardiology Unit, IRCCS Istituto Giannina Gaslini, Genoa, Italy
[6]Department of Integrated Surgical and Diagnostic Sciences (DISC), University of Genoa, Genoa, Italy
[7]Direzione Scientifica, IRCCS Istituto Giannina Gaslini, Genoa, Italy

Background

Paediatric-onset Still's disease, formerly defined as Systemic Juvenile Idiopathic Arthritis (sJIA), is characterized by systemic inflammation, fever, organomegaly, cutaneous rash, serositis and adenopathy. It can be associated with arthralgia or arthritis [1]. Although pulmonary involvement in both paediatric and adult Still's disease was previously considered rare and typically limited to reversible pleural effusions or mild parenchymal changes, it has emerged as a potentially severe and life-threatening manifestation, encompassing interstitial lung disease (ILD), pulmonary hypertension, and pulmonary alveolar proteinosis (PAP) [1–4]. This complication was initially observed in patients with severe disease, recurrent macrophage activation syndrome (MAS), high exposure to anti-interleukin (IL)-1/IL-6 [5], particularly in those developing delayed hypersensitivity reactions resembling drug reaction with eosinophilia and systemic symptoms (DRESS) [3]. However, the pathogenetic mechanism leading to this complication has not yet been clarified [2, 3, 5].

We report the case of a ten-years-old girl of European origin, with Still's disease complicated by lung involvement, improving with a combination of JAK1/2 inhibitor and cyclosporine.

Case report

The patient was referred to our institution at age six for severe Still disease, poorly responsive to first- and second-line therapies and recently complicated by pulmonary involvement requiring oxygen supplementation.

The disease onset occurred at nine months with daily fever, polyarticular arthritis, diarrhoea, hepatosplenomegaly, as well as elevated inflammatory markers, ferritin, transaminases, anaemia and leukocytosis. She tested negative for ANA, ANCA, ASMA, autoimmune hepatitis- and celiac disease- associated antibodies.

She experienced injection-site reaction after the first dose of anakinra (1 mg/kg) and anaphylaxis after the second monthly dose of the anti-IL-6 receptor antibody tocilizumab (10 mg/kg). The disease was successfully controlled with canakinumab (4 mg/kg every 4 weeks) combined with methotrexate (MTX) for three years, with the latter gradually tapered and discontinued at age five. One month after MTX discontinuation, she presented recurrent fever episodes and, a few months later, daily fever associated with diffuse lymphadenopathies, hepatosplenomegaly, diarrhoea, headache, and elevation of inflammatory markers (CRP 7 mg/dL, ESR 36 mm/h), including ferritin (880 ng/mL). Positron Emission Tomography (PET) scan revealed increased uptake involving the bilateral axillary and external iliac lymph-nodes. Bone marrow aspiration and excisional biopsy of the axillary lymph-node ruled out a malignancy; oral steroid treatment was therefore started with benefit. Steroid tapering was attempted twice, unsuccessfully, despite the reintroduction of MTX after 6 months.

At the age of six, due to the occurrence of persistently low peripheral blood oxygen levels by pulse oximetry (90%), requiring oxygen supplementation, the patient was referred to our centre.

At first admission in our hospital, the patient's therapy included canakinumab (4 mg/kg monthly), subcutaneous methotrexate (MTX, 7.5 mg/week) and oral prednisone (0.5 mg/kg/day). Persistent oxygen saturation below 90% was associated with clinical signs of chronic oxygen deprivation (grade 2 finger clubbing of both hands and feet). Spirometry was normal, while Diffusing Capacity of the Lung for Carbon Monoxide (DLCO) test could not be performed for inadequate cooperation. During admission, she developed persistent fever, and signs of macrophage activation syndrome (MAS): elevated ferritin (1825 ng/mL), triglycerides (311 mg/dL), AST (140 U/L); reduction of fibrinogen (126 mg/dL) and platelets (123000/uL). She did not display hypereosinophilia nor skin rash. The ongoing treatment with canakinumab and methotrexate was discontinued and she received high-dose intravenous methylprednisolone and oral cyclosporine (Fig. 1), resulting in prompt clinical and laboratory improvement. Chest CT scan demonstrated focal inter- and intra-lobular thickening of the lung, with regional areas of honeycombing and lung hyperexpansion (Fig. 2A, B), as well as diffuse lymphadenopathies.

Bronchoalveolar lavage (BAL) revealed increased cellularity with an enriched population of macrophages and epithelioid histiocytes, many with features of large foamy macrophages.

Lung parenchymal biopsy showed alveolar proteinosis with complex pneumopathy and arteriopathy (Fig. 2C–F).

Echocardiography and invasive cardiac catheterization demonstrated the presence of severe pulmonary hypertension (Supplementary materials); sildenafil was therefore initiated, achieving stable oxygen saturation without need of oxygen supplementation. Systemic hypertension required treatment with amlodipine and later bisoprolol. Due to previous biological drug failure and positive type I interferon (IFN) signature, detected during the diagnostic work-up performed at admission (Fig. 1), treatment with the JAK inhibitor baricitinib (4 mg/day) was associated with the ongoing treatment with prednisone and cyclosporine, allowing a good control of the clinical picture and the progressive tapering of corticosteroids (Fig. 1). After 4 months of treatment, in light of the persistently mild elevation of CRP and the still positive interferon signature, baricitinib was increased to 6 mg/day. Six months after the first admission, while on treatment with baricitinib 6 mg/day, cyclosporine 3 mg/kg/day, prednisone

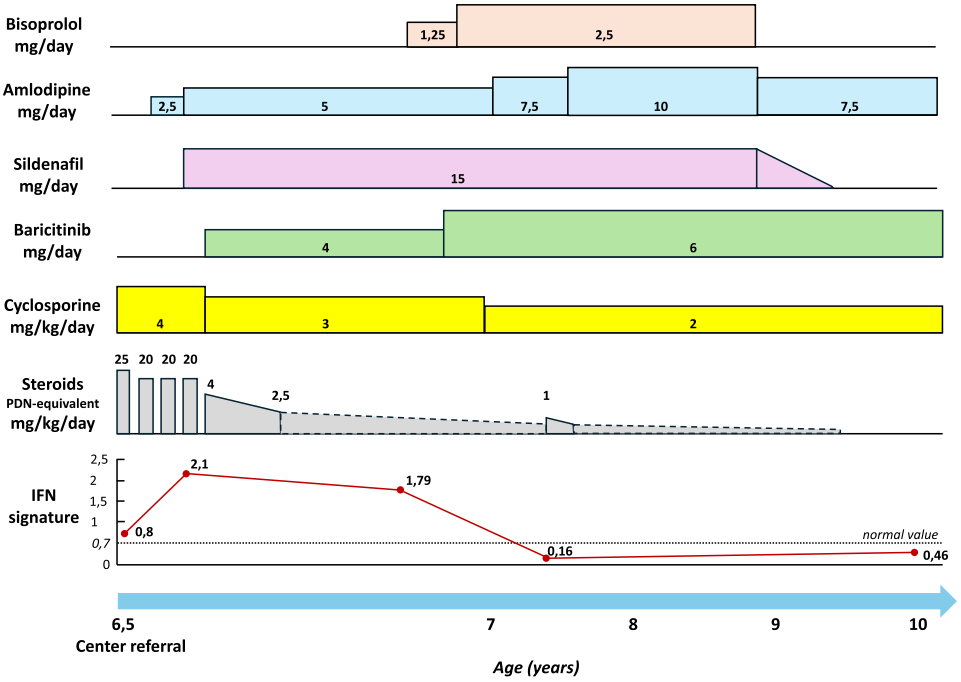

Fig. 1 Timeline of Administered Therapies. Longitudinal representation of the therapies administered to the patient at our Centre following the onset of sJIA-associated lung disease, along with their respective dosages. The x-axis represents the patient's age.The connected dot plot shows the trend of the type I interferon signature in the patient. The dashed line represents the normal value. IFN-signature methods have been reported in Supplementary Methods. IFN: interferon; PDN: prednisone

1 mg/kg/day, sildenafil 15 mg/day, amlodipine and bisoprolol, the patient was in good clinical conditions, with normal spirometry (Supplementary Figure S1); the echocardiography showed a reduction in pulmonary hypertension (sPAP 31 + 5 mmHg).

Of note, the patient tested positive for the HLA-DRB1*15 haplotype, reported to be associated with an increased risk of developing delayed hypersensitivity reaction and related pulmonary disease, and the HLA-DRB1*11 haplotype, associated with increased risk of developing pediatric Still disease [6, 7]. A large panel for Inborn Errors of Immunity was negative (Supplementary Table S1).

Subsequent follow-up confirmed persistent disease control with occasional articular exacerbations. At age 8, she developed transient SARS-CoV-2–related respiratory failure, resolved with brief oxygen support and no complications. After 3 years of baricitinib and cyclosporine, steroidal treatment was withdrawn and, at age 9, with stable spirometry, DLCO and 6MWT, sildenafil was tapered and discontinued. At the time of writing, 3.5 years after lung disease onset, the patient is stable on cyclosporine (2 mg/kg/day), baricitinib (6 mg/day), amlodipine and bisoprolol, with no disease reactivation, normal inflammatory markers and IFN signature. Chest CT confirmed complete resolution of acute features of pneumopathy with a marked improvement in honeycombing (Fig. 2A, B).

Discussion

This manuscript presents the successful long-term management of a case of pediatric Still-LD with the association of JAK inhibitor and cyclosporine. The underlying mechanisms and the best management of this condition remain poorly understood.

Interleukin-18 has been implicated in Still-LD pathogenesis. High IL-18 levels in serum [2, 8] and BAL [2], along with anecdotal improvement with anti-IL-18 therapy, suggest a role in pulmonary inflammation [9, 10]. In our case, IL-18 dosage was unavailable at JAK inhibitor initiation, but it was normal at last follow-up, along with CXCL9, CXCL10, and IFN-γ (Supplementary Table S2). A positive type I IFN signature during active disease further supports an underlying immune dysregulation contributing to pulmonary and systemic pathology (Fig. 1).

Certain factors have been associated with the risk of developing Still-associated lung disease, including early onset of Still's disease, MAS, anaphylaxis to biologic therapies, and specific HLA subtypes [4].

Over the past two decades, advances in Still management have significantly improved survival, especially in case of recurrent MAS. Still-LD may represent a complication in patients with severe systemic disease who, in past decades, may not have survived long enough for pulmonary involvement to manifest [1].

Biologic agents targeting IL-1 and IL-6, commonly used in Still, have been associated with hypersensitivity

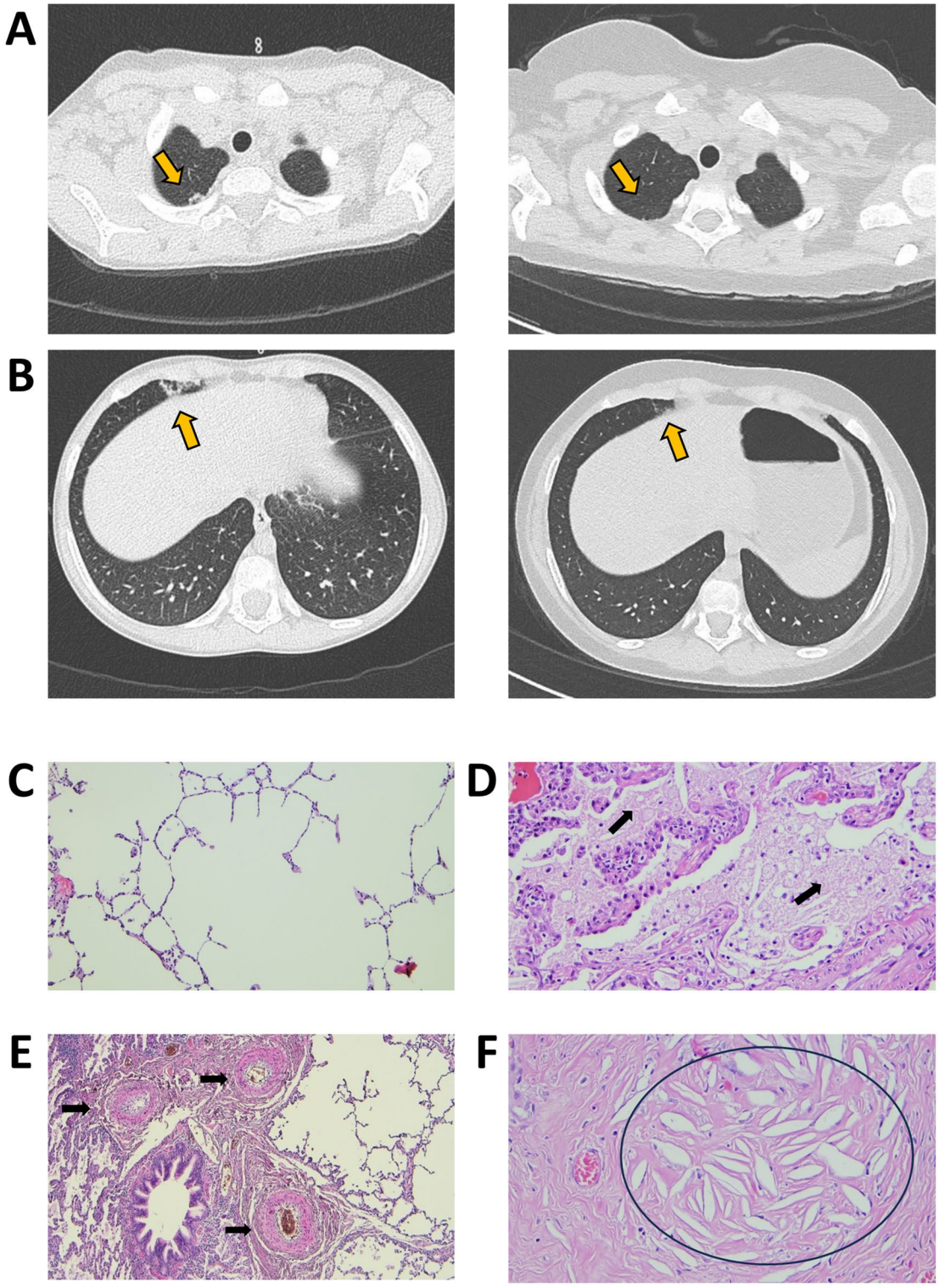

Fig. 2 (See legend on next page.)

reactions resembling DRESS syndrome, as well as with the development of lung manifestations [7, 11, 12]. Additionally, the rise in Still-LD cases over the past decade appears to parallel the growing use of these medications [4, 12].

Our patient carries the HLA-DRB1*15 allele, associated with DRESS-like reactions and lung disease [7], and

(See figure on previous page.)

Fig. 2 Chest CT scan (A-B) from admission to the last follow-up and histological examination of the lung (C-F) in the reported patient. In panel A, the image on the left shows thickening of the interlobular and intralobular septa with early signs of honeycombing in the right upper lobe at the time of admission, while the image on the right shows the normalization of the findings at the end of the follow-up. Similarly, panel B shows the same normalization of findings in the middle lobe. Panels C-F show histological examination of the lung. C) lung parenchyma showed multifocal dilatation of the alveolar spaces with rupture of septa, indicating a condition of emphysema. Hematoxylin-eosin 20x magnification. D) areas characterised by alveolar spaces filled with eosinophilic, granular proteinaceous material, and foamy histiocytes were observed (arrows), image referable to alveolar proteinosis. Hematoxylin-eosin 20x magnification. E) proximal and distal arterioles with hypertrophy of the muscular tunica and intimal hyperplasia were observed (arrows). Hematoxylin-eosin 4x magnification. F) in addition to alveolar proteinosis, a deposition of cholesterin needles with sclerohyalinosis were also observed (circle). Hematoxylin-eosin 40x magnification

developed hypersensitivity reactions to both anakinra and tocilizumab prior to lung involvement. Although hypereosinophilia was not observed during active disease (while on high-dose steroid therapy) or follow-up, its presence at earlier stages cannot be excluded and may have contributed to lung disease development.

The role of immunosuppressive therapy in Still-LD remains debated. The increasing availability of biologics has likely reduced the use of traditional immunosuppressants. Emerging evidence suggests that T lymphocytes play a pathogenic role in lung involvement [2, 5]; accordingly, insufficient immunosuppressive therapy in the management of systemic disease may increase the risk of pulmonary complications. Interestingly, our patient presented clear signs of lung disease approximately one year after the initial discontinuation of methotrexate, and after the the reactivation of the systemic features of the disease, without response to the reintroduction of methotrexate. Following withdrawal of the ongoing treatment, the introduction of cyclosporine, together with induction corticosteroid therapy and subsequent initiation of a JAK inhibitor, led to resolution of the lung disease. This clinical course supports a potential role for immunosuppressive therapy, particularly T-cell–targeted immunosuppression, in the management of this complication.

With the inherent limitations of a single-case description, we observed sustained improvement of the clinical picture and good long-term safety with the combination of cyclosporine and baricitinib: the COVID-19 infection had an uncomplicated course, and no reactivation of herpesvirus infections—a possible complication of the combined treatment—was observed. Recently, eight cases of pediatric Still-LD treated with different JAK inhibitors have been described, with variable response: four out of eight patients achieved complete symptoms control, including two who remained stable after steroid discontinuation. However, in three patients this treatment was not effective in disease control, even if a progression of the lung involvement was reported only in one case [13]. A case series of nine patients revealed benefit with tofacitinib and, consequently, JAK1 and JAK3 blockade [14]. Other isolated reports support the efficacy of JAK inhibitors in pediatric Still [15–18]. The data currently available are not sufficient to demonstrate superior efficacy of any specific JAK inhibitor in pediatric Still's disease. We decided to treat our patient with baricitinib because it has been shown to be effective in patients with monogenic interferonopathies [19], it was already approved for the treatment of rheumatoid arthritis and a clinical trial in pediatric juvenile idiopathic arthritis was ongoing at the time of treatment initiation.

In this case, monitoring the interferon signature helped guide modulation of JAK inhibitor therapy; in particular, its increase, together with a rise in CRP four months after treatment initiation, and in the absence of concomitant infectious events, allowed us to successfully increase the baricitinib dosage.

The emerging role of JAK inhibitors in Still-LD is particularly noteworthy, as they may modulate IL-18 expression by interfering with transcriptional regulation mediated by STAT and NFκB, thus disrupting the crosstalk between type I IFN, TLR signals, and IFN-γ-driven inflammation [13, 20]. Furthermore, blocking the JAK pathway has been shown to reduce TNF-induced caspase-1 expression and IL-18 bioactivity in rheumatoid arthritis synovial fibroblasts [21]. However, the lack of response to this drug reported in some patients may underline a different pathogenetic mechanism in such cases [13].

Notably, at the onset of pulmonary involvement, our patient presented with an acute clinical picture dominated by hypoxemia and histological evidence of PAP on lung biopsy, despite only focal abnormalities on imaging. In addition to the immunosuppressive therapy administered, supportive measures were crucial, including the screening and timely treatment of pulmonary hypertension, as well as management of systemic arterial blood pressure.

In conclusion, combining JAK inhibitors with T-lymphocyte–targeting immunosuppressants such as cyclosporine may offer an effective treatment strategy in paediatric-onset Still disease with lung involvement; further studies are needed to clarify their role and identify possible biomarkers of therapeutic response.

Abbreviations

6MWT	Six-minute walking test
BAL	Bronchoalveolar lavage
CRP	C-reactive protein
CT	Computed tomography
CXCL9	C-X-C motif chemokine ligand 9
CXCL10	C-X-C motif chemokine ligand 10

DLCO	Diffusing capacity of the lung for carbon monoxide
DRESS	Drug reaction with eosinophilia and systemic symptoms
HLA	Human leukocyte antigen
IFN-γ	Interferon-gamma
IL-1	Interleukin-1
IL-1β	Interleukin-1 beta
IL-6	Interleukin-6
IL-18	Interleukin-18
ILD	Interstitial lung disease
JAK	Janus kinase
JIA	Juvenile idiopathic arthritis
MAS	Macrophage activation syndrome
MTX	Methotrexate
PAP	Pulmonary alveolar proteinosis
PET	Positron emission tomography
sJIA	Systemic juvenile idiopathic arthritis
sJIA-LD	Systemic juvenile idiopathic arthritis-associated lung disease
sPAP	Systolic pulmonary artery pressure

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12969-026-01198-4.

Supplementary material 1

Acknowledgements
not applicable.

Author contributions
S.P. and M.L. drafted the manuscript. J.F. conducted the histological analysis and authored the section on histological findings. S.P., P.S., M.C., R.P., S.V., S.R., V.G.V., R.C., A.R. and M.G. managed patient follow-up and reviewed the manuscript draft. R.C. conceived the idea for the manuscript. All authors reviewed and approved the last version of the manuscript.

Funding
not applicable.

Data availability
Additional data are available upon request by contacting the corresponding author.

Declarations

Ethics approval and consent to participate
This research was conducted in accordance with the Declaration of Helsinki. Off-label baricitinib therapy was approved by the Ethics Committee of IRCCS Istituto Giannina Gaslini and Liguria Ethics Committee, Genoa (Italy). Written informed consent was obtained from the parents.

Consent for publication
Written informed consent was obtained from the parents.

CARE statement
The structure of this case report follows the CARE (Case Report) checklist, ensuring comprehensive reporting of the patient's clinical details and outcomes.

Competing interests
R.C., S.V and R.P received consultancy and speaker fee from SOBI. MG. received consultancy and speaker fee from Boheringer, Fresinius-Kabi, Kiniksa, Novartis and SOBI. S.P., M.L., J.F., P.S., M.C., S.R., V.G.V. declared no conflict of interest for this work. A.R. received consultancy and speaker fee from AbbVie, Angelini, BMS, Pfizer, Hoffman LaRoche, Novartis, Pfizer, Reckitt Benckiser.

Received: 8 November 2025 / Accepted: 18 February 2026
Published online: 13 March 2026

References

1. Matteis AD, Bindoli S, Benedetti FD, Carmona L, Fautrel B, Mitrovic S. Systemic juvenile idiopathic arthritis and adult-onset Still's disease are the same disease: evidence from systematic reviews and meta-analyses informing the 2023 EULAR/PReS recommendations for the diagnosis and management of Still's disease. Ann Rheum Dis. 2024, Dec, 1;83(12):1748–61.
2. Schulert GS, Yasin S, Carey B, Chalk C, Do T, Schapiro AH, et al. Systemic juvenile idiopathic arthritis-associated lung disease: characterization and risk factors. Arthritis Rheumatol. 2019;71(11):1943–54.
3. Saper VE, Chen G, Deutsch GH, Guillerman RP, Birgmeier J, Jagadeesh K, et al. Emergent high fatality lung disease in systemic juvenile arthritis. Ann Rheum Dis. 2019, Dec, 1;78(12):1722–31.
4. Towe C, Grom AA, Schulert GS. Diagnosis and management of the systemic juvenile idiopathic arthritis patient with emerging lung disease. Pediatr Drugs. 2023, Nov, 1;25(6):649–58.
5. Kimura Y, Weiss JE, Haroldson KL, Lee T, Punaro M, et al. Childhood arthritis rheumatology research alliance carra net investigators. Pulmonary hypertension and other potentially fatal pulmonary complications in systemic juvenile idiopathic arthritis. Arthritis Care Res (Hoboken). 2013, May;65(5):745–52. https://doi.org/10.1002/acr.21889.
6. Ombrello MJ, Remmers EF, Tachmazidou I, Grom A, Foell D, Haas JP, et al. HLA-DRB1*11 and variants of the MHC class II locus are strong risk factors for systemic juvenile idiopathic arthritis. Proc. Natl. Acad. Sci. U. S. A. 2015, Dec, 29;112(52):15970–75.
7. Saper VE, Ombrello MJ, Tremoulet AH, Montero-Martin G, Prahalad S, Canna S, et al. Drug hypersensitivity Consortium; INCHARGE Consortium. Severe delayed hypersensitivity reactions to IL-1 and IL-6 inhibitors link to common HLA-DRB1*15 alleles. Ann Rheum Dis. 2022, Mar;81(3):406–15.
8. Chen G, Deutsch GH, Schulert GS, Zheng H, Jang S, Trapnell B, et al. Identification of distinct inflammatory programs and biomarkers in systemic juvenile idiopathic arthritis and related lung disease by serum proteome analysis. Arthritis Rheumatol. 2022;74(7):1271–83.
9. Yasin S, Solomon K, Canna SW, Girard-Guyonvarc'h C, Gabay C, Schiffrin E, et al. IL-18 as therapeutic target in a patient with resistant systemic juvenile idiopathic arthritis and recurrent macrophage activation syndrome. Rheumatology. 2020, Feb, 1;59(2):442–45.
10. Rood JE, Rezk A, Pogoriler J, Finn LS, Burnham JM, Josephson MB, et al. Improvement of refractory systemic juvenile idiopathic arthritis-associated lung disease with single-agent blockade of IL-1β and IL-18. J Clin Immunol. 2023, Jan, 1;43(1):101–08.
11. Saper VE, Tian L, Verstegen RHJ, Conrad CK, Cidon M, Hopper RK, et al. Interleukin (IL)-1/IL-6-Inhibitor-associated drug reaction with eosinophilia and systemic symptoms (DReSS) in systemic inflammatory illnesses. J Allergy Clin Immunol Pract. 2024, Nov, 1;12(11):2996–3013.e7.
12. Binstadt BA, Nigrovic PA. The conundrum of lung disease and Drug hypersensitivity-like reactions in systemic juvenile idiopathic arthritis. Arthritis Rheumatol. 2022;74(7):1122–31.
13. Côte G, Quartier P, Belot A, Melki I, Hentgen V, Merlin E. The place of JAK inhibitors in systemic juvenile idiopathic arthritis with lung disease: French experience. Rheumatology. 2024, Oct, 24;keae589.
14. Swanson C, Khan EK, Cooper A, Ibarra M, Tam-Williams J. Pediatric systemic juvenile idiopathic arthritis-related lung disease: description of clinical cohort and review of management. Pediatr Pulmonol. 2024;59(3):724–33.
15. Zekre F, Duncan A, Laurent A, Tusseau M, Pescarmona R, Collardeau-Frachon S, et al. Rescue of pap-mas in systemic JIA using janus kinase inhibitors, case report and systematic review. J Clin Med. 2023, Apr, 4;12(7):2702.
16. Bader-Meunier B, Hadchouel A, Berteloot L, Polivka L, Béziat V, Casanova JL, et al. Effectiveness and safety of ruxolitinib for the treatment of refractory systemic idiopathic juvenile arthritis like associated with interstitial lung disease: a case report. Ann Rheum Dis. 2022, Feb, 1;81(2):e20.
17. Jørgensen SE, Christiansen M, Høst C, Glerup M, Mahler B, Lausten MM, et al. Systemic juvenile idiopathic arthritis and recurrent macrophage activation syndrome due to a CASP1 variant causing inflammasome hyperactivation. Rheumatology. 2020, Oct, 1;59(10):3099–105.
18. de Jesus Aa, Hou Y, Brooks S, Malle L, Biancotto A, Huang Y, et al. Distinct interferon signatures and cytokine patterns define additional systemic autoinflammatory diseases. J Clin Invest. 2020, Apr, 1;130(4):1669–82.
19. Sanchez GAM, Reinhardt A, Ramsey S, Wittkowski H, Hashkes PJ, Berkun Y, et al. JAK1/2 inhibition with baricitinib in the treatment of autoinflammatory interferonopathies. J Clin Invest. 2018;128(7):3041–52. https://doi.org/10.1172/JCI98814.

20. Verweyen EL, Schulert GS. Interfering with interferons: targeting the JAK-STAT pathway in complications of systemic juvenile idiopathic arthritis (SJIA). Rheumatology. 2022, Mar, 1;61(3):926–35.
21. Marotte H, Tsou PS, Fedorova T, Pinney AJ, Lewis B, Koch AE. Blocking the janus-activated kinase pathway reduces tumor necrosis factor alpha-induced interleukin-18 bioactivity by caspase-1 inhibition. Arthritis Res Ther. 2014, Apr, 24;16(2):R102.

Publisher's Note