

Systemic Lupus Erythematosus-Associated Autoimmune Myelofibrosis Mimicking TAFRO Syndrome: A Case Report and Literature Review

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Abstract

Systemic lupus erythematosus (SLE) is a multisystem autoimmune disorder with diverse hematologic manifestations, rarely complicated by autoimmune myelofibrosis (AIMF), a non-clonal and potentially reversible form of marrow fibrosis. Differentiating SLE-associated AIMF from TAFRO syndrome can be difficult because both conditions share overlapping features, including cytopenias, effusions, renal dysfunction, systemic inflammation, and reticulin fibrosis. We describe a 33-year-old woman who presented with recurrent low-grade fever, pedal edema, ascites, and dyspnea. Laboratory evaluation revealed pancytopenia, Coombs-positive hemolytic anemia, proteinuric renal disease, elevated inflammatory markers, antinuclear antibodies (ANA) positivity (1:2560, speckled pattern), high anti-dsDNA titers, and low complement C3 levels. Bone marrow biopsy demonstrated grade III reticulin fibrosis with increased megakaryocytes without significant atypia, while testing for myeloproliferative driver mutations, including *JAK2*, *CALR*, and *MPL*, was negative. Although the patient fulfilled several clinical criteria suggestive of TAFRO syndrome, lymph node biopsy showed only reactive hyperplasia without features of idiopathic multicentric Castleman disease, supporting a diagnosis of SLE-associated AIMF. She was treated with intravenous methylprednisolone pulses followed by oral prednisolone, hydroxychloroquine, intravenous immunoglobulin, and rituximab, resulting in marked clinical and hematologic improvement, with remission achieved within six weeks. This case highlights the importance of careful serologic, histopathologic, and clinicopathologic correlation in distinguishing AIMF from TAFRO-like syndromes, as prompt recognition and immunosuppressive therapy can lead to reversal of marrow fibrosis and favorable outcomes.

Categories: Hematology, Rheumatology, Internal Medicine

Keywords: autoimmune myelofibrosis, bone-marrow fibrosis, cytopenia, rituximab, systemic lupus erythematosus, tafro syndrome

Introduction

Systemic lupus erythematosus (SLE) is a multisystem autoimmune disorder with diverse clinical manifestations, ranging from mucocutaneous and musculoskeletal involvement to hematologic and renal disease [1]. Hematological abnormalities are common and are most often caused by peripheral immune-mediated destruction. However, in rare cases, bone marrow stromal involvement may result in autoimmune myelofibrosis (AIMF) [2,3].

AIMF represents a non-clonal form of myelofibrosis characterized by reticulin or collagen fibrosis in the marrow, cytopenias, and the absence of myeloproliferative driver mutations such as *JAK2*, *CALR*, or *MPL* [4,5]. Unlike primary myelofibrosis, AIMF is usually reversible with corticosteroids and other immunosuppressants.

According to the 2021 international consensus, terminology and diagnostic criteria have evolved, making it important to distinguish TAFRO syndrome from iMCD-TAFRO and a TAFRO-like phenotype. The 2021 international definition of iMCD-TAFRO requires relevant clinical findings, lymph node histopathology compatible with idiopathic multicentric Castleman disease, and the exclusion of alternative causes, including systemic autoimmune diseases [6,7]. In contrast, a “TAFRO-like phenotype” is a descriptive term for a similar presentation attributable to another underlying disorder, without implying a diagnosis of iMCD-TAFRO.

SLE can produce overlapping features, including fever, cytopenias, effusions, renal involvement, and marrow fibrosis, creating a diagnostic challenge [8,9]. We describe a young woman with SLE-associated AIMF who presented with TAFRO-like clinical features and review recent literature highlighting the findings that help distinguish this presentation from iMCD-TAFRO and clonal myelofibrosis.

Case Presentation

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A 33-year-old woman presented with a two-year history of intermittent low-grade fever, fatigue, pedal edema, and progressive exertional dyspnea. She was initially misdiagnosed with rheumatoid arthritis and received methotrexate without benefit.

On examination, she had pallor, hypertension, bilateral pedal edema, ascites, and bilateral pleural effusions. Laboratory tests showed pancytopenia (Hb 5.6 g/dL, WBC $3.67 \times 10^9/L$, platelets $7 \times 10^9/L$), elevated lactate dehydrogenase (LDH), and a positive direct Coombs test. Urinalysis revealed proteinuria of 2 g/day and microscopic hematuria (Table 1).

Investigation	Result	Reference range
Hemoglobin (Hb)	5.6 g/dL	13-17 g/dL
Total leukocyte count (WBC)	3670 /mm ³	4,000-11,000 /mm ³
Platelet count	7000 /μL	150,000-450,000 /μL
Reticulocyte count (%)	7.21	-
Total bilirubin	0.4 mg/dL	0.1-1.2 mg/dL
Direct bilirubin	0.2 mg/dL	0.1-0.39 mg/dL
Total protein	6.47 g/dL	6.5-7.8 g/dL
Albumin	1.75 g/dL	3.5-5.2 g/dL
Globulin	4.72 g/dL	2-3.5 g/dL
Lactate dehydrogenase (LDH)	744 U/L	<248 U/L
Urea	39 mg/dL	17-43 mg/dL
Creatinine	0.8 mg/dL	0.6-1.1 mg/dL
Direct Coombs test	Positive (IgG 3+)	-
24-hour urinary protein	1737 mg/24 h	Normal: <150 mg/24 h
Antinuclear antibodies (ANA)	4+, nuclear homogeneous pattern	-
Complement C3	17 mg/dL	90-180 mg/dL
Complement C4	<6 mg/dL	10-40 mg/dL
Ferritin	1344	13-150 ng/mL
Anti-double-stranded DNA (anti-dsDNA) antibodies	537 ELiA U/mL	Positive: >15
Urine protein	3+ proteinuria	Negative to trace
Urine RBCs	2+ RBCs	Absent/0-2 RBCs per HPF
Interleukin-6 (IL-6)	10.7	Normal: <7 pg/mL

TABLE 1: Baseline investigations.

The autoimmune profile demonstrated antinuclear antibodies (ANA) positivity (4+, nuclear homogeneous pattern), high anti-dsDNA titers, low C3 and C4 levels, and negative ANCA. Imaging revealed hepatosplenomegaly, ascites, and generalized lymphadenopathy. Bone marrow biopsy showed hypercellularity, increased megakaryocytes without atypia, and grade III reticulin fibrosis (Figure 1). Testing for *JAK2*, *CALR*, and *MPL* mutations was negative. Next-generation sequencing for myeloid mutations did not reveal any pathogenic variants (Table 2). Lymph node biopsy revealed reactive hyperplasia without features of Castleman disease. A renal biopsy could not be performed because of thrombocytopenia.

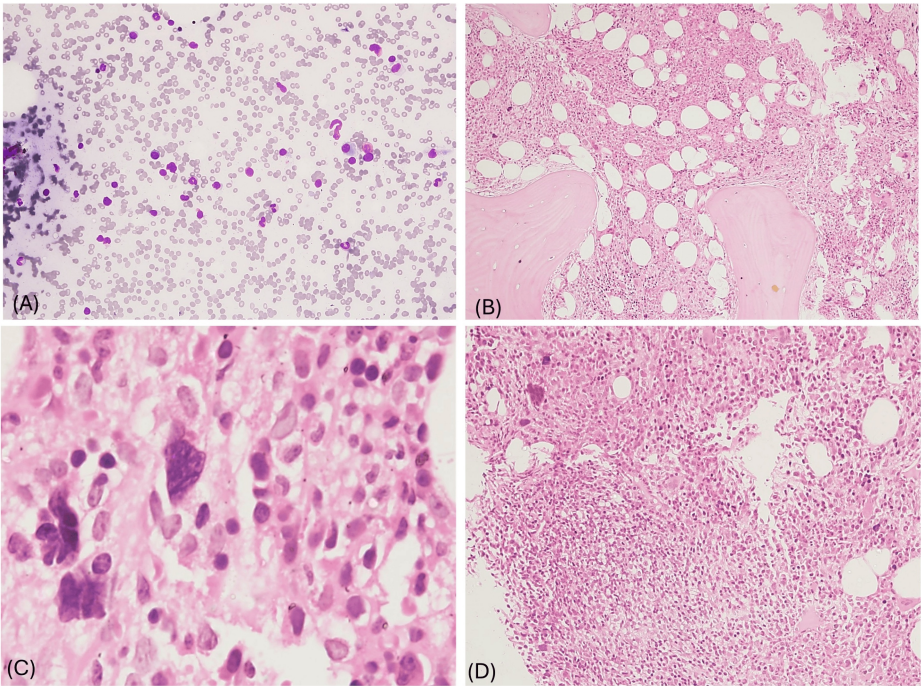


FIGURE 1: Bone marrow aspiration and biopsy.

(A) Paucicellular bone marrow aspirate showing scattered lymphocytes and plasma cells (40x; May-Grünwald-Giemsa stain). Bone marrow biopsy showing (B) hypercellular marrow spaces with streaks of fibrosis and an increased number of megakaryocytes (40x; H&E stain), (C) loose clustering and nuclear hyperchromasia of megakaryocytes (100x; H&E stain), and (D) small lymphoid aggregates composed of mature-appearing lymphocytes (40x; H&E stain).

Parameter	Finding
Batch	MYELOID_16_07_25
Investigation	Next-generation sequencing (myeloid mutation panel)
NGS result	No pathogenic mutations detected.
Genes covered (75-gene panel)	GNB1, CSF3R, MPL, NRAS, DNMT3A, CXCR4, SF3B1, IDH1, MYD88, GATA2, PDGFRA, KIT, TET2, FBXW7, TERT, PDGFRB, NPM1, DDX41, IKZF1, SAMD9, SAMD9L, CUX1, BRAF, EZH2, FGFR1, RAD21, JAK2, CDKN2A, ABL1, NOTCH1, ANKRD26, PTEN, SMC3, HRAS, WT1, MAP2K1, IDH2, PRPF8, TP53, NF1, SUZ12, STAT5B, STAT3, PPM1D, SRSF2, SETBP1, ELANE, CALR, JAK3, CEBPA, ASXL1, GNAS, RUNX1, U2AF1, PIGA, ZRSR2, BCOR, KDM6A, UBA1, GATA1, ATRX, STAG2, BCORL1, PHF6, EED, ETNK1, KMT2A, CBL, ETV6, KRAS, NFE2, SH2B3, PTPN11, FLT3, RB1
Technical details	Mean amplicon coverage >620x; coverage uniformity of 95.6%; sequencing performed on the Illumina MiSeq platform.

TABLE 2: Next-generation sequencing myeloid mutation panel.

A differential diagnosis of TAFRO syndrome versus SLE with AIMF was considered. The presence of lupus-specific antibodies favored SLE with AIMF. The patient was treated with IV methylprednisolone pulses (1 g/day × 3 days), followed by oral prednisolone, hydroxychloroquine, IV immunoglobulin (IVIG), and rituximab (375 mg/m² weekly × 4). Clinical improvement was noted within six weeks, with platelet counts rising to 100 × 10⁹/L and resolution of effusions.

Discussion

AIMF represents an uncommon but recognized hematological manifestation of SLE, with an estimated prevalence of less than 2% among lupus patients [1]. It is characterized by non-clonal reticulin or collagen deposition in the bone marrow, leading to cytopenias, in contrast to primary myelofibrosis, which shows clonal driver mutations such as JAK2, CALR, or MPL and atypical megakaryocytic proliferation [3,4]. The

pathogenesis of AIMF is believed to involve autoimmune-mediated cytokine activation, particularly involving IL-1, IL-6, and transforming growth factor- β , which stimulate fibroblast proliferation and excessive reticulin deposition [10].

AIMF may occur early or late in the course of SLE and is often reversible with timely immunosuppression. Case series have shown that corticosteroids remain the mainstay of therapy, frequently combined with disease-modifying agents such as hydroxychloroquine, mycophenolate mofetil, or rituximab for refractory cytopenias [4,10]. Early recognition is crucial, as prompt therapy can lead to reversal of marrow fibrosis and normalization of blood counts [11].

TAFRO syndrome, first described by Kawabata H et al., is defined by the constellation of thrombocytopenia, anasarca, fever or a hyperinflammatory state, reticulin fibrosis or renal dysfunction, and organomegaly [12]. The 2021 international consensus by Nishimura Y et al. further refined these criteria and validated a definition for idiopathic multicentric Castleman disease with TAFRO phenotype (iMCD-TAFRO), emphasizing the need to exclude autoimmune diseases, infections, and malignancies before diagnosis [6].

Because SLE and TAFRO share overlapping features, including cytopenias, anasarca, renal dysfunction, and hyperinflammatory states, the differential diagnosis can be challenging. Recent reports describe lupus patients presenting with TAFRO-like features, including fever, thrombocytopenia, anasarca, and marrow fibrosis, who were initially misclassified as having TAFRO syndrome [9,10]. However, the presence of lupus-specific serologies (ANA and anti-dsDNA positivity), hypocomplementemia, and a positive Coombs test usually favors SLE with AIMF over primary TAFRO syndrome [8,9].

In our patient, all major TAFRO criteria, thrombocytopenia, anasarca, fever, reticulin fibrosis, and organomegaly, were present. Nevertheless, strong lupus serologies, hypocomplementemia, and the absence of Castleman-like lymph node histopathology supported the diagnosis of SLE with AIMF rather than iMCD-TAFRO. Similar cases have been described by Rama Krishna Reddy MM et al. and Koduri PR et al., in which AIMF associated with lupus responded well to corticosteroids, emphasizing its reversibility [4,10].

Therapeutically, AIMF and TAFRO require different approaches. AIMF responds to immunosuppressive therapy, particularly corticosteroids, hydroxychloroquine, and rituximab, whereas TAFRO syndrome generally necessitates IL-6 blockade (tocilizumab or siltuximab) and cyclosporine [8]. Distinguishing between these two entities, therefore, has crucial prognostic and therapeutic implications, as cytokine-targeted therapy used for TAFRO is not effective in AIMF.

In the literature, 45 cases of SLE-associated AIMF had been reported prior to the 2018 review by Del Porto F et al. [13]. Here, we describe an additional case and compile eight further reports published after 2018, summarizing their clinical characteristics, marrow findings, therapy, and outcomes (Table 3) [5, 14-20]. Most showed non-clonal reticulin fibrosis with positive ANA/anti-dsDNA and responded well to corticosteroid-based immunosuppression, confirming the reversible nature of AIMF in SLE.

Author (year)	Age/sex	Clinical presentation	Bone marrow findings	Autoimmune profile	Treatment	Outcome
Guavita D et al. (2021) [5]	33/F	Lupus nephritis, dialysis dependence, fever, pancytopenia	Hypocellular marrow; Thiele grade I-II fibrosis; JAK2 negative	Positive Coombs test; anti-dsDNA negative	Methylprednisolone followed by rituximab	Refractory disease; death
Wibowo T et al. (2020) [14]	66/F	Pancytopenia, fatigue	Hypocellular marrow with grade 2 fibrosis	ANA+, anti-dsDNA+, low C3/C4	Prednisolone with tapering	Complete hematologic and marrow recovery
Mbonu I et al. (2019) [15]	20/F	Severe anemia, leukopenia, fever, lymphadenopathy, bone pain	Increased reticulin fibrosis; no blasts; JAK2, CALR, MPL, and BCR::ABL1 negative	ANA+, anti-dsDNA+, anti-Sm+, SSA+, RF+, low C3/C4	Intravenous methylprednisolone followed by hydroxychloroquine	Marked clinical and hematologic improvement
Abdulla MC and Alungal J (2021) [16]	44/F	Ecchymosis, gingival bleeding, thrombocytopenia, acquired hemophilia A	Hypocellular marrow with grade 1 fibrosis; JAK2, CALR, and MPL negative	ANA+, anti-dsDNA++, low C3/C4	Intravenous methylprednisolone followed by prednisolone and hydroxychloroquine	Resolution of cytopenias and fibrosis
Kapur SV and Oswal JS (2021) [17]	16/M	Fever, polyarthralgia, pancytopenia	Hypercellular marrow with MF-2 fibrosis and megakaryocytic hyperplasia	ANA+, anti-dsDNA+, anti-Sm+, anti-RNP+, low C3/C4	Pulse methylprednisolone followed by prednisolone, hydroxychloroquine, and MMF	Rapid hematologic recovery
Bulut GD et al. (2024) [18]	31/F	Refractory pancytopenia	Hypercellular marrow; MF grade 2-3	ANA+, SSA+, anti-dsDNA+, low complement levels	Corticosteroids, IVIG, rituximab	Sustained complete remission (>3 years)
Bensalek F et al. (2024) [19]	34/F	Gingival bleeding, pancytopenia	Grade 1-2 reticulin fibrosis with trilineage hypoplasia; JAK2 negative	Anti-dsDNA+, low C3, ANA negative	Pulse corticosteroids followed by hydroxychloroquine	Complete hematologic recovery
Soltani H and Dehghan A (2025) [20]	34/F	Arthralgia, petechiae, thrombocytopenia	Grade 3 reticulin fibrosis with megakaryocytic hyperplasia	ANA+, anti-dsDNA+, anti-Sm+, low C3/C4	Hydroxychloroquine, tofacitinib, IVIG, prednisolone	Complete resolution of marrow fibrosis after 10 months

TABLE 3: Reported cases of autoimmune myelofibrosis (AIMF) associated with systemic lupus erythematosus (SLE) after 2018.

ANA: Antinuclear antibodies; anti-dsDNA: Anti-double-stranded DNA antibodies; anti-Sm: Anti-Smith antibodies; anti-RNP: Anti-ribonucleoprotein antibodies; SSA: Sjögren's syndrome-related antigen A; RF: Rheumatoid factor; C3: Complement component 3; C4: Complement component 4; IVIG: Intravenous immunoglobulin; MMF: Mycophenolate mofetil; MF: Myelofibrosis; F: Female; M: Male.

Conclusions

SLE-associated autoimmune myelofibrosis should be considered in patients presenting with TAFRO-like manifestations, particularly when lupus-specific autoantibodies and hypocomplementemia are present. Careful exclusion of clonal myeloproliferative neoplasms and Castleman disease is essential, as AIMF is a reversible immune-mediated condition that responds well to timely immunosuppressive therapy. This case highlights the significant clinical overlap between autoimmune and hyperinflammatory syndromes, which may lead to diagnostic uncertainty and delays in treatment. Recognition of subtle lupus-specific clinical and serologic clues, along with detailed bone marrow and lymph node evaluation, is critical for establishing the correct diagnosis. Early initiation of corticosteroids and targeted immunosuppressive therapy can result in rapid hematologic recovery and improved overall outcomes. Greater awareness of this uncommon presentation may help clinicians avoid unnecessary investigations and ensure prompt, potentially life-saving treatment.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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