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Case report

Refractory SLE-Associated Immune Thrombocytopenia in a Resource-Limited Setting: A Case Report and Rationale for Early BAFF Inhibition

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Abstract
Severe immune thrombocytopenia (ITP) is a life-threatening haematological manifestation of systemic lupus erythematosus (SLE) that may prove refractory to standard therapies, particularly in resource-limited settings where access to advanced biologic agents is restricted. We report a case of refractory SLE-associated thrombocytopenia and discuss the rationale for early B-cell activating factor (BAFF) inhibition. A 50-year-old Libyan man with SLE presented with severe thrombocytopenia, autoimmune haemolytic anaemia, and mucocutaneous bleeding requiring intensive care admission. Initial laboratory investigations revealed a platelet count of 2 × 10^3/μL, haemoglobin of 5.1 g/dL, low complement levels, and a positive direct Coombs test. The patient received high-dose corticosteroids, intravenous immunoglobulin (IVIG), rituximab, mycophenolate mofetil, repeated blood product transfusions, and supportive care in accordance with current treatment recommendations. Platelet counts remained critically low for 27 days, with minimal response achieved on day 27 (>10 × 10^3/μL), partial response on day 31 (>20 × 10^3/μL), and a clinical response (>50 × 10^3/μL) by approximately day 45. Complete haematological response was not attained, and platelet counts remained below the normal range at final follow-up. The delayed and incomplete response highlights the challenges of managing refractory SLE-associated thrombocytopenia in settings with limited therapeutic options. Emerging evidence suggests that BAFF inhibition with belimumab may complement rituximab by targeting autoreactive plasma cell survival pathways and improving haematological outcomes. This case underscores the unmet need for accessible biologic therapies in resource-limited settings and generates the hypothesis that earlier introduction of BAFF inhibition, alone or in combination with rituximab, may improve outcomes in refractory SLE-associated thrombocytopenia. Prospective studies are warranted to evaluate this therapeutic strategy.
Keywords. Immune Thrombocytopenia, Case Report, SLE.

Introduction

Systemic lupus erythematosus (SLE) is a complex autoimmune disorder characterised by protean manifestations affecting multiple organ systems [1]. Although SLE disproportionately affects females (female: male ratio ≈ 9:1), certain male populations – such as those with Klinefelter syndrome (47, XXY) – carry an elevated risk [2]. Haematological manifestations are among the most prominent features of SLE; autoimmune cytopenias, most notably autoimmune haemolytic anaemia (AIHA) and thrombocytopenia, affect up to 40% of patients and represent a major contributor to disease-associated morbidity and mortality [3,4]. Thrombocytopenia in SLE is defined as a platelet count below 100,000/mm^3 and is stratified by severity: mild (>50,000/mm^3), moderate (20,000–50,000/mm^3), and severe (≤20,000/mm^3) [4].

Severe immune thrombocytopenia (ITP) results from antibody-mediated platelet destruction, driven by T-follicular helper cells, autoreactive B lymphocytes, and plasma cells – a process that can lead to profound and potentially fatal bleeding episodes [5]. The underlying pathogenesis of SLE involves activation of autoreactive immune cells by self-antigens, triggering a cascade of inflammatory mediators and survival signals – including IL-6, IL-17, TNF-α, B-cell activating factor (BAFF), and APRIL (A Proliferation-Inducing Ligand) – that perpetuate autoimmunity and tissue damage [6]. Standard treatment strategies aim to suppress immune dysregulation and typically include high-dose corticosteroids, intravenous immunoglobulin (IVIG), and B-cell depletion with rituximab. In refractory cases, cytotoxic agents such as cyclophosphamide or long-term immunosuppressants including azathioprine, mycophenolate mofetil, and cyclosporine are considered [7].

The 2023 EULAR recommendations further expand therapeutic options to include thrombopoietin receptor agonists (TPO-RAs), fostamatinib – a spleen tyrosine kinase inhibitor – and splenectomy for patients with refractory disease [7]. For consistent evaluation of response, we adopted the International Working Group (IWG) criteria for ITP: complete response (platelets ≥100 × 10^3/μL), response (≥30 × 10^3/μL with at least twofold increase from baseline), and no response (<30 × 10^3/μL or less than twofold increase) [8]. In this report, we additionally define minimal response (>10 × 10^3/μL) and clinical response (>50 × 10^3/μL) to capture the stepwise recovery observed. Despite these advances, access to newer biologic agents remains severely restricted in many parts of the world, creating a critical gap between guideline

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recommendations and real-world clinical practice. This case report describes a patient with severe refractory thrombocytopenia in the setting of SLE, details the stepwise interventions implemented at Benghazi Medical Centre, underscores the urgent need for broader access to biologic therapies, and generates the hypothesis that early BAFF inhibition combined with rituximab may improve outcomes in refractory cases.

Case presentation

A 50-year-old Libyan man, married but with a history of infertility, was first diagnosed with SLE in 2021 following presentation with oral ulcers, arthritis, positive antinuclear antibodies (ANA) and anti-double-stranded DNA (anti-dsDNA) antibodies, low complement levels (C3 and C4), and autoimmune haemolytic anaemia (AIHA). At that time, his AIHA proved resistant to standard therapy and was managed with high-dose intravenous methylprednisolone pulses, intravenous immunoglobulin (IVIG), two rituximab infusions, and supportive blood transfusions. He stabilised and was discharged on tapering prednisolone (20 mg daily) and hydroxychloroquine (200 mg twice daily). Unfortunately, he was subsequently lost to follow-up for nearly two years.

On 9 July 2025, the patient re-presented with a chronic cough and low-grade fever and was referred to Benghazi Medical Centre (BMC) with suspected SLE flare complicated by anaemia and severe thrombocytopenia. On arrival, he exhibited signs of active mucocutaneous bleeding, including epistaxis, gingival bleeding, ecchymoses, and melaena. On physical examination, the patient was alert and oriented, appearing pale but without jaundice or respiratory distress. Vital signs on admission were: heart rate 105 bpm, respiratory rate 18 breaths/min, blood pressure 129/79 mmHg, and oxygen saturation 98% on room air.

Cardiovascular and respiratory examinations were unremarkable. Abdominal examination revealed hepatosplenomegaly, with the spleen palpable just below the costal margin. Rectal examination was normal, and there were no signs of deep vein thrombosis. The patient was admitted to the intensive care unit (ICU), where he remained for 25 days. Laboratory investigations revealed marked anaemia and severe thrombocytopenia, accompanied by elevated erythrocyte sedimentation rate (ESR), raised C-reactive protein (CRP), elevated lactate dehydrogenase (LDH), and low complement levels. Lupus anticoagulant testing (LA1 and LA2) was performed to exclude antiphospholipid syndrome. Renal function, serum electrolytes, coagulation profile, electrocardiogram, and HbA1c were all within normal limits. Serological screening for hepatitis B, hepatitis C, and HIV was negative. Abdominal and pelvic ultrasound confirmed splenomegaly without additional abnormalities. The most significant laboratory findings are summarised in (Table 1).

Table 1. Most significant laboratory findings on admission.

Test	Result	Reference Range
Haemoglobin	5.1 g/dL	13–17.4 g/dL
RBC	1.11 × 10^6/µL	4.5–5.5 × 10^6/µL
MCV	115.3 fL	80–100 fL
Haematocrit	12.8% (↓)	40–52%
Platelets	2 × 10^3/µL	150–450 × 10^3/µL
WBC	7.09 × 10^3/µL	4–11 × 10^3/µL
Neutrophils	85.1% (↑)	50–70%
Lymphocytes	11.6% (↓)	20–40%
ESR	128 mm/h	1–10 mm/h

Table 2. Comprehensive Laboratory Profile in Autoimmune Evaluation

CRP (quantitative)	10 mg/L	0–5 mg/L
Albumin	4.7 g/dL	3.5–5.0 g/dL
Total Bilirubin	0.68 mg/dL	0.0–1.20 mg/dL
Direct Bilirubin	0.29 mg/dL	0–0.20 mg/dL
LDH	500 U/L	135–225 U/L
Direct Coombs Test	Positive	Negative
Anti-dsDNA	39.6 U/mL	< 100 U/mL
Anti-Smith IgG	0.2 U/mL	< 15 U/mL
Anti-Cardiolipin IgM	0.9 U/mL	< 7 U/mL
Anti-Cardiolipin IgG	1.4 U/mL	< 10 U/mL
ANA	1 U/mL	>1.1 U/mL
C3	0.7 g/dL	0.8–1.8 g/dL
C4	0.01 g/dL	0.1–0.4 g/dL
Lupus Anticoagulant LA1	45.5 sec	30.4–45.3 sec
Lupus Anticoagulant LA2	35.5 sec	27.7–33.5 sec
LA1/LA2 Ratio	1.28	0.0–1.40
Urinalysis	Blood +++; numerous RBCs; casts nil;	—

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	albumin nil	
Blood Group	A+	—
APTT	27.2 sec	28–40 sec
PT	11.4 sec	9.7–11.8 sec
INR	1.01	0.89–1.13
D-Dimer	1.4 µg/mL	0–0.5 µg/mL
Fibrinogen	3.26 g/L	1.7–4.2 g/L

Management and response

Treatment was initiated on 8 July 2025 (day 1) with intravenous methylprednisolone (500 mg daily) and IVIG (0.4g/kg daily), alongside repeated blood and platelet transfusions. Platelet counts fell as low as 1 × 10^3/µL on day 5, reflecting the severity of the refractory thrombocytopenia. On day 5 (12 July), the first rituximab infusion (1 g) was administered. Oral prednisolone (60 mg daily) replaced intravenous methylprednisolone from day 6. Despite these measures, platelet counts remained critically low (2–8 × 10^3/µL) through the end of week 2. On day 12 (19 July), therapy was escalated to intravenous dexamethasone (40 mg daily for 4 days) for its rapid immunosuppressive onset, and mycophenolate mofetil (MMF, 2 g/day) was commenced. A second rituximab infusion (1 g) was administered on day 19 (26 July), two weeks after the first. To evaluate the haematological response, we used the International Working Group (IWG) criteria for immune thrombocytopenia [8], which define complete response (platelets ≥100 × 10^3/µL), response (≥30 × 10^3/µL with at least twofold increase from baseline), and no response (<30 × 10^3/µL or less than twofold increase). In addition, we defined minimal response as >10 × 10^3/µL and clinical response as >50 × 10^3/µL to describe the stepwise recovery observed in this patient. Quantitative analysis of the platelet trajectory showed that counts remained at nadir levels (1-8 × 10^3/µL) for the first 27 days. Minimal response (>10 × 10^3/µL) was first confirmed on day 27 (3 August), with a manual platelet count of 10 × 10^3/µL. Partial response (>20 × 10^3/µL) was achieved by day 31 (7 August), when manual count confirmed 20 × 10^3/µL. The patient was discharged on 10 August. Platelet counts continued to rise on outpatient review, reaching 30 × 10^3/µL on 19 August (day 43) and 70 × 10^3/µL by approximately day 45, constituting a clinical response (>50 × 10^3/µL). This level was maintained at the final recorded outpatient visit on 27 October 2025 (day 111). Complete hematological response (>100 × 10^3/µL) was never attained. The overall recovery slope from admission to final follow-up was +0.62 × 10^3/µL per day. The complete treatment and platelet trajectory are summarized in Table 3 and visualized in (Figures 1 and 2). Quantitative recovery metrics are presented in (Table 4).

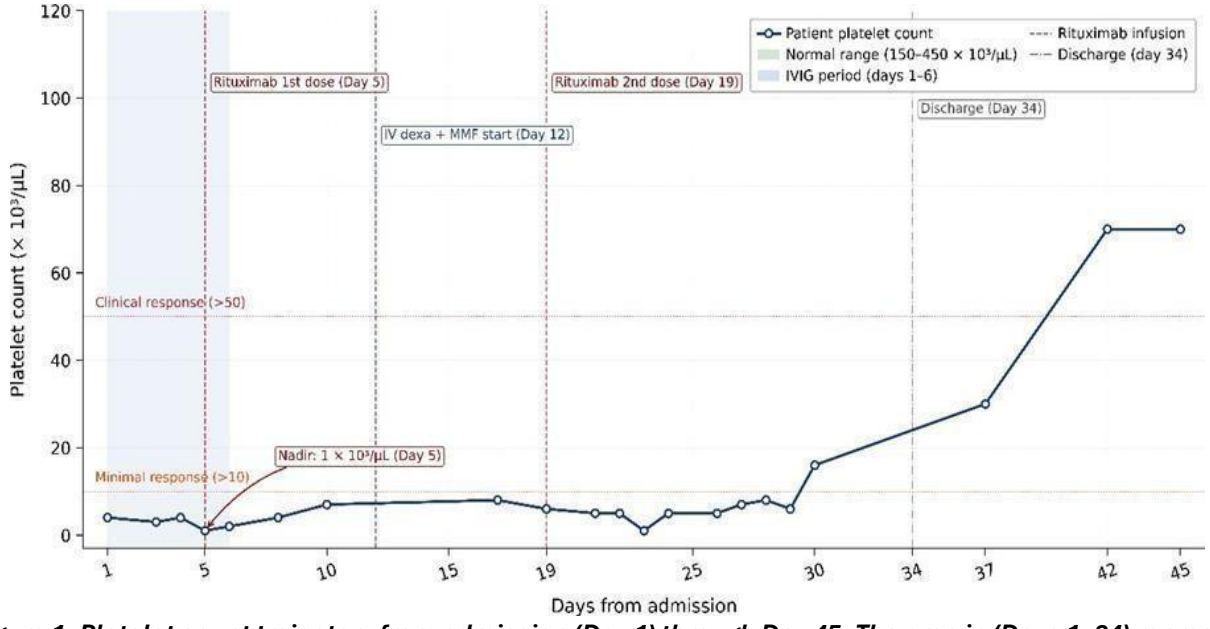

Figure 1. Platelet count trajectory from admission (Day 1) through Day 45. The area in (Days 1–34) represents inpatient ICU admission (Days 1–25) and ward stay (Days 2C–34); (Days 35–45) represents outpatient follow-up.

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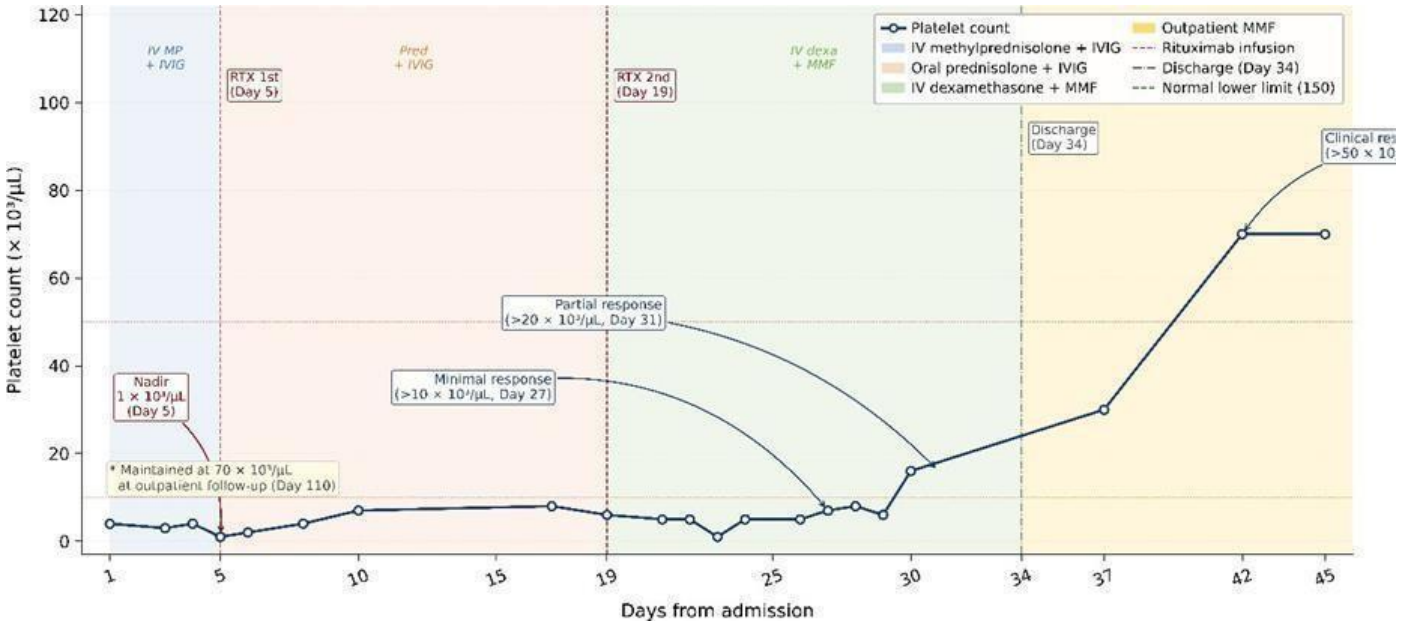

Figure 2. Platelet trajectory with treatment phases (days 1- 45)

Table 3. Chronological treatment timeline and platelet response. *Manual platelet count.

Date (Day)	Platelets ×10^3/μ L	Treatment / Events
08 Jul 2025 (Day 1)	4	IV methylprednisolone 500 mg; IVIG 0.4 g/kg; blood and platelet transfusions
10 Jul 2025 (Day 3)	3	IV methylprednisolone 500 mg; IVIG 0.4 g/kg; platelet transfusion
12 Jul 2025 (Day 5)	4→1	IV methylprednisolone 500 mg; IVIG 0.4 g/kg; Rituximab 1 g (1st infusion)
13 Jul 2025 (Day 6)	2	Oral prednisolone 60 mg; IVIG 0.4 g/kg
14 Jul 2025 (Day 7)	4	Oral prednisolone 60 mg
17 Jul 2025 (Day 10)	7	Oral prednisolone 60 mg
18 Jul 2025 (Day 11)	8	Oral prednisolone 60 mg (end of week 2 — insufficient response)
19 Jul 2025 (Day 12)	6	Switch to IV dexamethasone 40 mg; mycophenolate mofetil 2 g/day commenced
26 Jul 2025 (Day 19)	5	Rituximab 1 g (2nd infusion, 2 weeks after 1st); MMF 2 g/day
28 Jul 2025 (Day 21)	5	MMF 2 g/day continued
03 Aug 2025 (Day 27)	10*	MMF 2 g/day; manual platelet counts 10 × 10^3/μL (minimal response achieved)
07 Aug 2025 (Day 31)	20*	MMF 2 g/day; manual platelet counts 20 × 10^3/μL (partial response achieved)
10 Aug 2025 (Day 34)	—	Patient discharged; weekly outpatient follow-up commenced
19 Aug 2025 (Day 43)	30	Outpatient review — platelet count rising
27 Oct 2025 (Day 111)	70	Final recorded outpatient visit — clinical response confirmed

"The arrow (→) indicates a drop from 4 × 10^3/μL to 1 × 10^3/μL on the same day."

Table 4. Quantitative hematological recovery metrics.

Parameter	Definition	Value in This Case
Baseline platelets	Admission value	2 × 10^3/μL
Nadir platelet count	Lowest recorded value	1 × 10^3/μL (Day 5)
Time to minimal response	Days to > 10 × 10^3/μL	Day 27 (03 Aug 2025)
Time to partial response	Days to > 20 × 10^3/μL	Day 31 (07 Aug 2025)
Time to clinical response	Days to > 50 × 10^3/μL	Day 45 (approx. 22 Aug 2025)
Time to complete response	Days to > 100 × 10^3/μL	Not achieved
Peak platelet count	Highest recorded value	70 × 10^3/μL (Day 45 and Day 111)
Overall recovery slope	(Final − Baseline) ÷ days	+0.62 × 10^3/μL/day

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Discussion

This case illustrates the formidable clinical challenge posed by severe immune thrombocytopenia (ITP) as a haematological manifestation of SLE – a complication associated with significant morbidity and, in refractory cases, considerable mortality [9]. The diagnosis of immune-mediated haemolysis was supported by a positive direct Coombs test in conjunction with markedly elevated LDH, consistent with concurrent Evans syndrome. The patient's failure to respond adequately to an escalating sequence of established therapies – including high-dose corticosteroids, IVIG, rituximab, and transfusion support – is indicative of a refractory disease course, which carries a particularly poor prognosis [9].

Therapeutic decisions throughout this admission were guided by the 2023 EULAR recommendations for the management of severe autoimmune thrombocytopenia in SLE, which advocate high-dose intravenous glucocorticoids with or without IVIG as first-line therapy, followed by rituximab, cyclophosphamide, and long-term maintenance immunosuppression in non-responding patients [7]. Despite strict adherence to these recommendations, platelet counts remained persistently at nadir levels for the first 27 days, and mucocutaneous bleeding continued throughout the ICU admission, underscoring the refractory nature of this case. The unavailability of thrombopoietin receptor agonists (TPO-RAs) – such as romiplostim and eltrombopag – at Benghazi Medical Centre further narrowed the therapeutic options, representing a critical gap between international guidelines and local clinical reality.

Rituximab, an anti-CD20 monoclonal antibody that depletes circulating B cells, is widely used off-label in refractory ITP, with published response rates of approximately 45% [10]. In this patient, the modest and delayed rise in platelet counts – with minimal response not achieved until day 27 and clinical response only by day 45 – suggested a partial and attenuated therapeutic effect. Why did platelet counts continue to fall to a nadir of 1×10^3/μL on day 5 despite high-dose corticosteroids and IVIG? This reflects the overwhelming antibody-mediated platelet destruction characteristic of refractory Evans syndrome. IVIG typically requires 24–72 hours to saturate Fc receptors and modulate macrophage clearance, while corticosteroids require several days to suppress autoantibody production. During this critical window, pre-existing autoantibodies continued to drive rapid platelet clearance, explaining the paradox of ongoing decline despite initiation of therapy. Why did it take 27 days to achieve even a minimal response? The prolonged interval likely reflects the requirements for rituximab-mediated B-cell depletion to reduce autoantibody-producing cells, combined with the slow clearance of pre-formed anti-platelet antibodies (IgG half-life ~21 days). This timeline is consistent with published rituximab response kinetics in refractory ITP, where the median time to response ranges from 4–8 weeks. Furthermore, long-lived plasma cells, particularly within the spleen, are resistant to B-cell depletion and continue to produce pathogenic autoantibodies [11,12]. BAFF (B-cell activating factor) provides essential survival signals to these autoreactive plasma cells, thereby sustaining autoantibody production and perpetuating platelet destruction even in the setting of rituximab therapy [11,12].

Belimumab, a fully human monoclonal antibody that inhibits soluble BAFF, was approved by both the FDA and EMA in 2011, representing the first novel biologic agent licensed for SLE in over five decades [13,14]. Beyond its established role in controlling lupus disease activity [14], recent molecular studies have demonstrated that belimumab modulates signalling through BLNK, BANK1, and MEF2C pathways, impacting B-cell differentiation and interfering with interferon-mediated NF-κB signalling, thereby contributing to broader disease modification [15]. Belimumab offers the additional practical advantage of two available administration routes: intravenous infusion – given every two weeks for the first three doses, then every four weeks – or subcutaneous injection, which may be preferable for long-term outpatient use [16]. Notably, Levy et al. have documented that the breadth of belimumab's clinical programme – encompassing more than 7,200 patients across 75 countries over a decade – has prompted investigation of its use in hematological conditions beyond SLE, including immune thrombocytopenia, further supporting its potential role in the present case [14]. Emerging evidence supports a role for belimumab specifically in the management of refractory SLE-associated thrombocytopenia. Nakayama et al. reported two male patients with SLE-related severe thrombocytopenia who achieved remission following subcutaneous belimumab therapy, eliminating the need for further platelet transfusions [17]. Clinical improvement was observed within the first weeks of treatment and extended beyond hematological parameters to encompass other disease manifestations, including interstitial pneumonia in one patient and resolution of thoracoabdominal effusion and lymphadenopathy in the other. Normalization of autoantibody levels was documented by day 59 and day 70, respectively [17]. Furthermore, a phase IIb prospective study by Mahévas et al. evaluated the combination of rituximab and belimumab in adults with persistent or chronic ITP, reporting complete platelet recovery in 13 of 15 patients within 12 weeks [18]. Notably, the rituximab dosing protocol in that study – 1 g administered twice, two weeks apart – was identical to the regimen used in the present case; the addition of five belimumab infusions at 10 mg/kg may account for the markedly superior outcomes observed [18]. Taken together, these findings support the hypothesis that early addition of belimumab to rituximab could improve outcomes in refractory SLE-associated thrombocytopenia. However, because our patient did not receive belimumab, we cannot claim efficacy. Instead, this case generates a testable hypothesis for prospective studies: in resource-limited settings where treatment options are few, upfront combination of B-cell depletion and BAFF inhibition might accelerate platelet recovery and reduce transfusion dependence.

In the present case, profoundly low complement levels (C3 0.7 g/dL, C4 0.01 g/dL) throughout the admission confirmed ongoing lupus disease activity as the primary driver of hematological deterioration, despite the anti-dsDNA level of 39.6

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U/mL being within the normal reference range (<100 U/mL). This pattern—active hematological disease with normal anti-dsDNA—has been described in SLE, where platelet-directed autoantibodies may dominate over anti-dsDNA production. The ANA result of 1.0 U/mL was negative by the laboratory's reference range (>1.1 U/mL). However, the patient's SLE diagnosis was established in 2021 based on prior positive ANA, anti-dsDNA positivity, low complement levels, and autoimmune hemolytic anemia. ANA titers can fluctuate over time, and a negative ANA on a single measurement does not exclude active SLE—particularly when haematological manifestations are the dominant disease feature, as in this case. The necessity of a 25-day ICU stay, platelet nadir of 1 × 10^3/μL, reliance on frequent manual platelet monitoring, and dependence on repeated transfusions collectively underscore the severity of the clinical course. Although the patient achieved a response (platelets ≥30 × 10^3/μL with doubling from baseline) by IWG criteria on day 31, and later a clinical response (>50 × 10^3/μL) by day 45, his counts remained below the normal range (150 × 10^3/μL) at final follow-up, leaving him at continued risk of serious bleeding events. The absence of belimumab and TPO-RAs in this setting precluded their use, but the patient's protracted course and delayed partial response highlight an unmet clinical need. In comparable resource-limited centres, where salvage therapies are restricted to corticosteroids, IVIG, and rituximab alone, the key clinical question is whether early access to BAFF inhibition would shorten the time to platelet recovery. This case cannot answer that question, but it provides a rationale for future collaborative studies. Regrettably, belimumab is not currently accessible in Libya, and private importation is prohibitive due to cost. This case therefore serves not only as a clinical report but as a call to action: the absence of approved biologic therapies in resource-limited settings translates directly into avoidable patient suffering and potentially preventable mortality. In summary, this case does not prove that belimumab would have altered the outcome, but it accomplishes three objectives. First, it documents the real-world consequences of limited biologic access in a Libyan referral centre. Second, it identifies a pressing unmet need for affordable BAFF inhibitors and TPO-RAs. Third, it generates the specific hypothesis that early combination therapy (rituximab plus belimumab) may be superior to rituximab alone in refractory lupus-associated thrombocytopenia.

Conclusion

This case report documents a severe and refractory presentation of SLE-associated immune thrombocytopenia in a resource-limited setting, highlighting the critical disconnect between internationally endorsed treatment guidelines and the therapeutic options realistically available at referral centres in Libya. Despite adherence to the 2023 EULAR recommendations – including high-dose corticosteroids, IVIG, rituximab, and mycophenolate mofetil – the patient achieved only a partial and delayed haematological response, with a platelet nadir of 1 × 10^3/μL and counts remaining below the normal range at final follow-up. Using the International Working Group (IWG) criteria for immune thrombocytopenia [8], a response (platelets ≥30 × 10^3/μL with doubling from baseline) was reached on day 31, but complete response (≥100 × 10^3/μL) was never attained. The evidence reviewed in this report, together with the delayed, incomplete response observed here, generates the hypothesis that earlier introduction of belimumab – either alone or combined with rituximab – could improve outcomes in refractory SLE-associated thrombocytopenia. Prospective studies are needed to test this hypothesis, particularly in resource-limited settings where treatment escalation options are severely constrained. Similarly, the availability of TPO receptor agonists such as romiplostim or eltrombopag could provide an important adjunctive strategy to reduce transfusion dependence and bleeding risk in such patients. Beyond the individual case, these findings carry broader implications for health policy. Securing the availability and affordability of advanced biologic therapies at referral centres in Libya and comparable resource-limited settings is not merely a matter of clinical optimisation – it is a matter of patient safety. Coordinated efforts between health authorities, academic institutions, and international organisations are urgently needed to bridge this gap and ensure that patients with life-threatening autoimmune complications have access to the treatments they require. Future prospective studies evaluating the combination of rituximab and belimumab specifically in SLE-associated refractory thrombocytopenia are warranted, with particular attention to outcomes in resource-limited settings where standard salvage therapies are unavailable.

Ethical approval and informed consent

This case report was prepared in accordance with the CARE reporting guidelines. This case report was conducted in accordance with the ethical standards of the Declaration of Helsinki. Written informed consent was obtained from the patient for the publication of this case report and any accompanying data. Formal ethics committee review was not required for this single case report under the institutional policies of the University of Benghazi and Benghazi Medical Centre; however, the case was handled in full compliance with applicable institutional guidelines.

Conflict of interest

None declared.

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