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Childhood-onset systemic lupus erythematosus presenting with menorrhagia as the initial manifestation: a case report

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Case Report

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Abstract

Background: Childhood-onset systemic lupus erythematosus(cSLE) is a multisystem autoimmune disease that frequently presents with hematologic involvement. Among adolescent patients with cSLE, menorrhagia is a rare clinical manifestation. When concurrent by severe anemia and thrombocytopenia, it can be life-threatening and carries a high risk of misdiagnosis or delayed diagnosis.

Case Presentation: A 13-year-old girl was admitted to hospital presenting with chest tightness, anemia, and severe thrombocytopenia, accompanied by pallor and scattered petechiae. Complete blood count showed a platelet count of 10×10^9/L and a hemoglobin level of 61 g/L. Medical history revealed significant menstrual abnormalities: markedly increased menstrual blood loss with prolonged menses.

Serological testing detected antinuclear antibodies at a titer of 1:3200, with positive anti-Smith (anti-Sm) antibodies. Laboratory results showed an indirect bilirubin level of 50.9 μmol/L, an LDH level of 1201 U/L, a reticulocyte count of 12.51%, and a positive direct antiglobulin test (DAT). Urinalysis revealed abnormal proteinuria; renal biopsy confirmed mild mesangial proliferative lupus nephritis (Class II). The patient presented with headache, dizziness and vomiting, brain MRI and cerebrospinal fluid examination suggested neuropsychiatric lupus, which was further supported by abnormal electroencephalography. The final diagnosis was systemic lupus erythematosus complicated by lupus nephritis, neuropsychiatric lupus, severe thrombocytopenia, acute abnormal uterine bleeding, and Evans syndrome (ES). The patient received sequential combination immunomodulatory treatment with hydroxychloroquine, oral prednisone, intravenous immunoglobulin, mycophenolate mofetil, and belimumab, together with supportive care. Short-acting oral contraceptives and GnRH analogs were subsequently administered to control uterine bleeding. By approximately two weeks after admission, platelet count rose to 248×10^9/L, urinalysis tested negative for proteinuria, neurological symptoms improved, and uterine bleeding resolved. At one-year follow-up, platelet-count and hemoglobin levels remained normal, 24-hour urinary protein excretion decreased to 0.17 g/24h, anti-dsDNA antibodies turned negative, and no relapse occurred.

Conclusion: For adolescent females who present with menorrhagia as the initial manifestation accompanied by severe thrombocytopenia and anemia, routine antinuclear antibody screening is recommended to screen for cSLE, even in the absence of typical cutaneous lupus lesions. Hemolytic markers should be systematically evaluated when hemolysis is suspected, even in the presence of confirmed blood loss. Comprehensive treatment can significantly improve outcomes.

1. Introduction

Systemic lupus erythematosus (SLE) is a complex multisystem autoimmune disease with highly heterogeneous clinical manifestations.[1] Compared with adult-onset SLE (aSLE), childhood-onset systemic lupus erythematosus (cSLE) is associated with more organ involvement, higher disease activity, a more severe clinical course, and elevated rates of disability and mortality.[2] cSLE often presents

initially with hematologic involvement, including autoimmune hemolytic anemia (AIHA) and immune thrombocytopenia (ITP). When these two cytopenias occur simultaneously or sequentially, the condition is called Evans syndrome.[3]

In adolescent females, menorrhagia is often initially attributed to pubertal dysfunctional uterine bleeding (DUB) or simple iron deficiency anemia and is easily overlooked by clinicians. However, when menorrhagia is accompanied by severe thrombocytopenia, it may also be an initial signal of hematologic involvement in SLE. Such patients often lack typical lupus signs, making it highly susceptible to misdiagnosis as an isolated hematologic disease, and consequently resulting in delayed diagnosis.

Here we report a case of a 13-year-old girl diagnosed with cSLE complicated with ES, whose initial clinical presentation was menorrhagia. We summarize and analyze the clinical course and management of this case, highlighting the diagnostic challenges associated with an atypical initial presentations, severe hematological involvement, and concurrent multisystem manifestations of cSLE, thereby aiming to improve clinicians' ability to recognize atypical presentations of the disease.

2. Case Presentation

2.1 Present Illness

Three days before admission, a 13-year-old girl developed dizziness and chest tightness, which worsened with activity, accompanied by pallor and scattered pinpoint petechiae over the body, with no ecchymosis observed. She denied fever, photosensitivity, facial rash, oral ulcers, joint swelling or pain, and alopecia. Physical examination demonstrated normal body temperature, pulse, and respiratory rate were within normal limits, blood pressure was elevated at 132/77 mmHg. The patient presented with severe anemic, and no characteristic lupus skin lesions (e.g., malar rash or discoid rash) or oral ulcers were observed. Cardiopulmonary and neurological examinations revealed no obvious abnormalities, and no hepatosplenomegaly was noted.

2.2 Past medical history and menstrual history

The patient was previously healthy with no significant relevant family history. Menarche occurred at the age of 12, and menstrual cycle was regular at 7/28 days with normal flow. Upon admission, she presented with abnormal heavy menstrual bleeding that had persisted for 13 days.

2.3 Laboratory and paraclinical tests

Hematological examination demonstrated a white blood cell count 5.26×10^9/L; a neutrophil count 3.46×10^9/L; a hemoglobin 61 g/L; a platelet count 10×10^9/L. The reticulocyte percentage was 12.51%, and peripheral blood smear examination revealed 2.2% schistocytes. Coagulation function tests showed no obvious abnormalities. Serum ferritin was 499.02 ng/mL, and the direct antiglobulin test (DAT) was positive. Flow cytometric immunophenotyping revealed no abnormal immunophenotypic cell

populations within the tested antibody panel, providing no evidence of an overt hematological malignancy.

Immunology test showed anti-U1-snRNP antibody positive (16.7), anti-Sm antibody positive (21.8), and antinuclear antibody positive (nucleolar pattern 1:3200). Anti-dsDNA antibody 17.97 IU/mL. Complement C4 0.088 g/L, complement C3 0.939 g/L. Antiphospholipid antibody testing, including lupus anticoagulant, anticardiolipin antibodies, and anti-β_2-glycoprotein I antibodies, was negative. Antineutrophil cytoplasmic antibodies (ANCA) were also negative.

Analysis of liver and kidney function at admission demonstrated total bilirubin 66.2 μmol/L, direct bilirubin 15.3 μmol/L, indirect bilirubin 50.9 μmol/L, LDH 1201 U/L, and a gradual decreases in bilirubin and LDH levels were found following therapy. Urinalysis revealed proteinuria (+), without hematuria or urinary red blood cell casts. The 24-hour urinary protein excretion was 0.333 g, and serum creatinine remained within the normal range. Given the newly diagnosed systemic lupus erythematosus and the presence of proteinuria, renal biopsy was performed to evaluate possible renal involvement. Renal biopsy revealed mild mesangial proliferative lupus nephritis (ISN/RPS class II), with an activity index of 0/24 and a chronicity index of 1/24 (Fig. 1).

(B) Transmission electron microscopy shows mesangial electron-dense deposits (red arrows).

Regarding neurological findings: brain MRI revealed bilateral frontal white matter hyperintensities, appearing as scattered punctate T1 isointense and T2 hyperintense signals, with hyperintensity on FLAIR (Fig. 2). These lesions were classified as grade 1 on the modified Fazekas scale. Lumbar puncture revealed a cerebrospinal fluid (CSF) opening pressure of 220 mmH_2O. CSF cell counts and protein levels were within normal limits, and microbiological investigations did not identify an infectious pathogen. Electroencephalography (EEG) demonstrated diffuse slow-wave abnormalities.

In addition, chest CT demonstrated no pleural effusion, slight thickening of the pulmonary arteries, and minimal pericardial effusion. Echocardiography reveals mild left ventricular hypertrophy and trace pericardial effusion.

2.4 Diagnosis

According to the 2019 revised EULAR/ACR classification criteria for systemic lupus erythematosus[4], the patient met the criterion of positive antinuclear antibodies (≥ 1:80). Based on the applicable weighted clinical and immunological criteria, the patient fulfilled the classification criteria for SLE with a total score of ≥ 10 points; therefore, SLE was diagnosed.The Systemic Lupus Erythematosus Disease Activity Index 2000 (SLEDAI-2K) score was retrospectively calculated as 16[5], indicating high disease activity. Other main diagnoses encompassed lupus encephalopathy, mild mesangial proliferative lupus nephritis (class II), severe acute abnormal uterine bleeding, anemia, and severe thrombocytopenia.

2.5 Treatment and Prognosis

Given the severe hematological manifestations and concurrent renal and neurological involvement, combination immunomodulatory treatment was initiated. Hydroxychloroquine (200 mg once daily) was introduced on day 3, followed by oral prednisone (60 mg/day) and intravenous immunoglobulin (IVIG; 400 mg/kg/day for five consecutive days) on day 5. Mycophenolate mofetil (MMF; 250 mg twice daily) was added on day 9, and intravenous belimumab (480 mg, 10 mg/kg) was initiated on day 13 to address severe hematological involvement and concomitant lupus nephritis.

Supportive treatment included platelet and packed red blood cell transfusions. Short-acting oral contraceptives for menorrhagia management but discontinued promptly once SLE was diagnosed due to heightened thrombotic risk; and GnRH agonists for recurrent uterine bleeding. Other interventions included etamsylate and tranexamic acid for hemostasis. Mannitol was also administered during the management of neurological symptoms.

Two weeks after admission, menstruation ceased, the platelet count rose to 248×10^9/L, anemia improved (Fig. 3), urinalysis became negative for protein, headache and dizziness subsided, and vomiting ceased. Serum bilirubin, urinary bilirubin, and LDH gradually returned to normal. Following discharge, prednisone was gradually tapered, while hydroxychloroquine, MMF, and belimumab were continued. Belimumab was administered at 480 mg every two weeks for the first three infusions, followed by 480 mg every four weeks thereafter.

At the 1-year follow-up, the menstruation cycles remained regular, the hematological parameters were stable. Renal function was normal, and 24-hour urinary protein quantification gradually decreased to 0.17 g/24h at the latest follow-up. No neurological symptoms recurred. Complement C4 and C3 levels remained normal, and anti-dsDNA antibodies converted to negative. Repeat testing of the ANA profile yielded results consistent with prior measurements. No significant abnormalities were detected in hemolysis-related biomarkers. No disease relapse, drug-related adverse reactions, or infectious events were reported during the entire follow-up. A summary of the key laboratory data is demonstrated in Fig. 3 and Fig. 4.

Note

Dashed colored lines (green: D3, blue: D5, purple: D9,orange:D13) mark key treatment nodes.

3. Discussion

3.1 Hematologic Involvement

The hematologic system is one of the most frequently affected organ systems in cSLE. Severe hematologic manifestations occur in approximately 45.5% of cSLE patients and often represent the initial clinical presentation—preceding involvement of other organ systems, indicating a higher disease activity and poorer prognosis.[6] ES represents a rare but life-threatening complication of cSLE, with an estimated prevalence of 1.3%. As demonstrated by Lube et al., cSLE patients presenting with ES exhibit

higher rates of hemorrhage (91%) and active disease (82%), whereas classic cutaneous features such as malar rash and musculoskeletal involvement are less common, thereby increasing the diagnostic difficulty.[7]

This patient presented with menorrhagia but no cutaneous lupus rash or arthralgia/arthritis, easily misdiagnosed as abnormal uterine bleeding. Although heavy DUB may account for anemia, it fails to explain the severe thrombocytopenia and biochemical evidence of hemolysis in this case. Laboratory findings supported the coexistence of AIHA and severe thrombocytopenia (Table 1), consistent with ES.[3] Bone marrow flow cytometric immunophenotyping revealed no abnormal immunophenotypic cell populations within the tested antibody panel, providing no evidence of an overt hematological malignancy. Together with the exclusion of primary marrow disorders, these findings suggest that the cytopenia was predominantly attributable to peripheral immune-mediated cellular destruction. Nevertheless, the presence of 2.2% schistocytes raised concern for a possible concurrent microangiopathic process. Although antiphospholipid antibody testing was negative, ADAMTS13 activity was not assessed, and thrombotic microangiopathy could not be definitively excluded.

Previous studies have demonstrated that autoimmune cytopenias can result from Fcγ receptor-dependent phagocytosis of antibody-opsonized blood cells within the reticuloendothelial system, particularly the spleen, as well as from complement-mediated hemolysis [3 8] .Such extensive peripheral immune destruction reflects a systemic breakdown of immune tolerance, consistent with published evidence that ES stems from systemic immune dysregulation rather than an isolated hematologic disorder[3]. Severe hematologic involvement, particularly ES, is more likely to be a prodromal manifestation of systemic autoimmune disease. This hypothesis is supported by Granel et al., who reported that 45% of ANA-positive children presenting with ES subsequently developed SLE, at a significantly accelerated rate compared with those presenting with isolated ITP[9].

Accordingly, in adolescents presenting with unexplained menorrhagia or severe cytopenia—particularly accompanied by laboratory evidence of hemolysis—management should not be restricted to gynecologic symptomatic control. ANA and autoantibody testing is warranted for early recognition and targeted intervention of underlying systemic autoimmune disease.

Table 1
Hematological findings supporting the diagnosis of Evans syndrome

Laboratory parameter	Result	Interpretation
Platelet count	10×10^9/L	Severe thrombocytopenia
Hemoglobin	Hb 61 g/L	Severe anemia
Reticulocyte count	12.51% (> 4%)	Increased erythropoietic response
Indirect bilirubin	50.9 μmol/L	Supports hemolysis
LDH	1201 U/L	Supports hemolysis
DAT	Positive	Supports immune-mediated hemolysis
Schistocytes	2.2%	Raises concern for microangiopathic hemolysis

3.2 Subclinical Renal Involvement

The patient presented with low-grade proteinuria (0.333 g/24 h), without hematuria or urinary red blood cell casts. However, renal biopsy revealed mild mesangial proliferative lupus nephritis (ISN/RPS class II), confirming renal involvement despite limited urinary abnormalities. Drawing on the mechanistic framework proposed by Dai et al.[10], mesangial electron-dense deposits are consistent with immune complex deposition, which may activate complement and local inflammatory pathways, promoting mesangial cell proliferation and extracellular matrix expansion. These mechanisms are compatible with the mild mesangial changes observed in this patient. Although the renal biopsy report described suspected thrombus-like material within a few renal arterioles, its significance remained uncertain, and no definitive diagnosis of thrombotic microangiopathy was established. The positive anti-dsDNA antibodies, reduced complement C4 levels, and characteristic renal histopathological findings collectively supported the diagnosis of immune complex-mediated lupus nephritis, despite the presence of only low-grade proteinuria. Both the KDIGO 2024 and the ACR 2024 guidelines support individualized kidney biopsy decisions based on proteinuria, kidney function, and the overall clinical context[11 12]. Previous studies have also demonstrated that histologically confirmed lupus nephritis may occur in patients with low-level proteinuria [13]. These findings highlights the importance of careful renal assessment and longitudinal monitoring in pediatric SLE, even when urinary abnormalities are mild.

3.3 Neurological Involvement

The patient presented with headache, dizziness, and vomiting. Brain MRI showed scattered punctate white-matter hyperintensities in bilateral frontal lobes, and EEG revealed diffuse slow waves. CSF analysis demonstrated normal cell counts and protein levels, with no infectious pathogens identified. Based on the overall clinical presentation and neurological findings, lupus encephalopathy (NPSLE) was clinically diagnosed, although these findings were nonspecific[14]. The pathophysiology of NPSLE is multifactorial, potentially involving blood–brain barrier disruption, autoantibody-mediated neuronal injury, complement activation, and neuroinflammation [15]. Severe anemia may have contributed to impaired

cerebral oxygen delivery, while the presence of schistocytes warranted consideration of microangiopathic processes. Additionally, blood pressure during neurological episodes was undocumented, and cerebral venous sinus thrombosis was not specifically excluded by dedicated venous imaging. This case underscores the importance of integrating clinical manifestations with neuroimaging, electrophysiological findings, and CSF analysis when evaluating neurological involvement in pediatric SLE.

3.4 Treatment Strategies

The management of SLE aims to suppress disease activity, prevent organ damage, and improve long-term prognosis, with individualized therapeutic strategies based on disease severity.[16] Given the presence of severe hematologic manifestations and concurrent subclinical renal and neurological involvement, early combined immunomodulatory therapy was initiated. Oral prednisone was prescribed for anti-inflammation therapy, and IVIG was administered to modulate immunity and raise platelet counts. This is consistent with the higher IVIG utilization observed in patients ES-complicated cSLE, as reported by Lube et al[6]. Hydroxychloroquine was used as background therapy, while MMF and belimumab were incorporated to achieve broader disease control. These agents have complementary immunomodulatory mechanisms, including suppression of inflammation, inhibition of lymphocyte proliferation, and modulation of B-cell activity. Evidence from pediatric clinical trials supports belimumab as an add-on treatment for active cSLE, with a favorable safety profile[17]. In the present case, belimumab was introduced primarily to address severe hematological involvement and concomitant lupus nephritis. The favorable clinical response observed following combination therapy supports the clinical rationale for an individualized, multimodal treatment approach, although the relative contributions of individual agents remain difficult to establish from this single case.

4.Conclusion

Adolescent females with cSLE complicated by ES may exhibit atypical initial presentations, including menorrhagia and severe thrombocytopenia, often preceding or occurring independently of classic mucocutaneous musculoskeletal manifestations. Clinicians should maintain a high index of suspicion for the rare overlap between cSLE and ES in adolescents presenting with unexplained severe cytopenia and menorrhagia, and should promptly initiate comprehensive immunological and hematological evaluations. Notably, acute massive hemorrhage may mask hemolytic markers, underscoring the necessity of a systematic assessment of hemolysis-related parameters to avoid diagnostic delay or misdiagnosis. Early initiation of immunosuppressive therapy is critical to reverse cytopenia and mitigate the risk of irreversible organ damage. The one-year follow-up of this case supports the efficacy and safety of combined immunomodulatory therapy, contributing to the limited literature on this atypical pediatric presentation and highlighting the importance of comprehensive menstrual history assessment in the diagnostic evaluation.

5. Patient Perspective

The girl and her family expressed satisfaction with the treatment outcomes. After treatment, her menstruation returned to normal, with complete resolution of previous symptoms including headache, vomiting, and other discomforts. She has resumed normal schooling and daily life. The patient and family expressed gratitude to the medical team and are willing to cooperate with long-term follow-up.

Declarations

Ethical approval and informed consent: This study was approved by Ethics Committee of the Sichuan Provincial People's Hospital.

Written informed consent for publication of this case report and any accompanying images was obtained from the patient's parent/guardian. A copy of the consent form is available for review by the Editor of this journal.

Availability of data and materials All data generated or analysed during this study are included in this published article and its supplementary information files. The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Conflicts of interest: All authors declare no conflicts of interest.

Author contributions: PC: data curation, writing – original draft, XZ: Writing – review & editing, YZ: Writing – review & editing, JD: Writing – review & editing, BT: Writing – review & editing, Quality control, Project administration.

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Figures

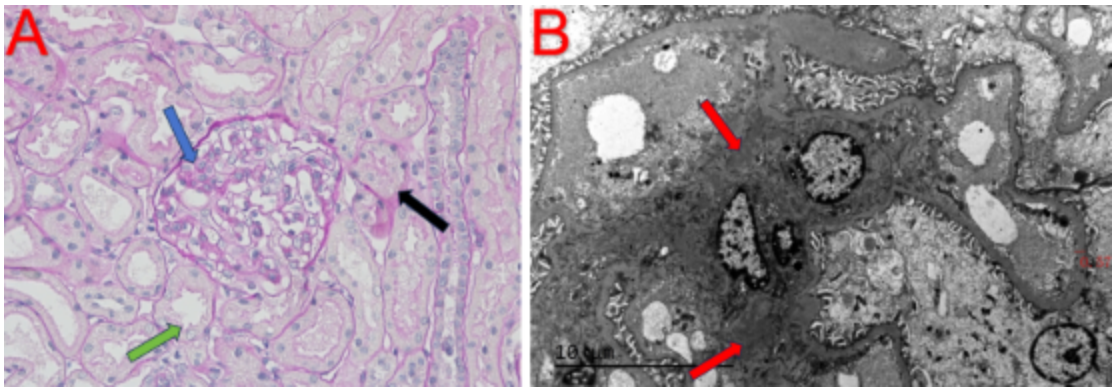


Figure 1

Renal pathological findings. (A) Periodic acid–Schiff (PAS) staining: mild segmental proliferation of mesangial cells and matrix (blue arrow); loss of brush border(green arrow); granular degeneration of tubular epithelial cells (black arrow).

(B) Transmission electron microscopy shows mesangial electron-dense deposits (red arrows).

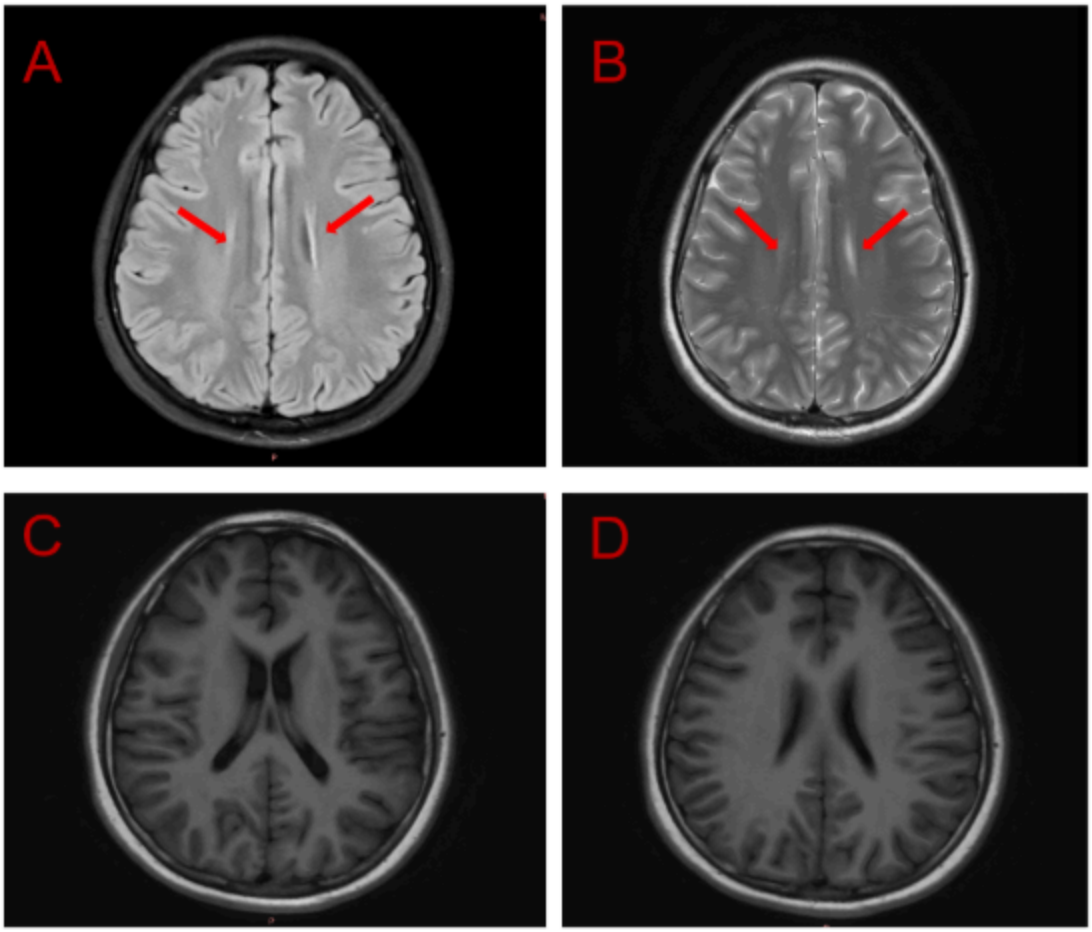


Figure 2

Brain MRI findings of the patient：Axial T2-FLAIR (A) and T2-FSE (B) images demonstrate bilateral frontal white matter hyperintensities, appearing as scattered punctate lesions (indicated by red arrows)； Corresponding axial T1-FLAIR images (C and D) show no corresponding hyperintensity; the lesions are T1 isointense and no abnormal enhancement.

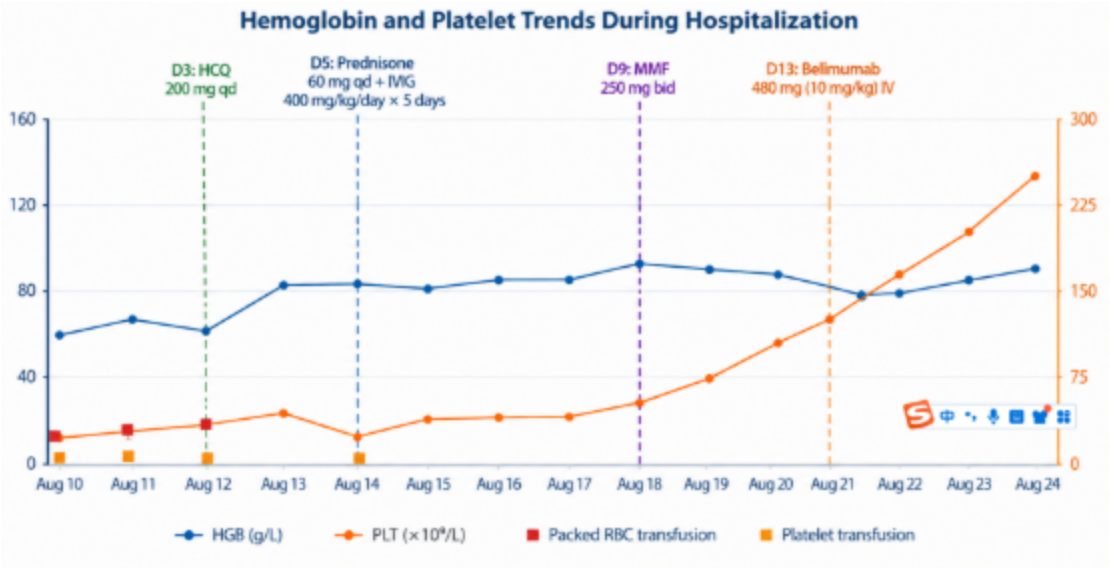


Figure 3

Key nodes and trends of hemoglobin and platelets

Note: Dashed colored lines (green: D3, blue: D5, purple: D9,orange:D13) mark key treatment nodes.

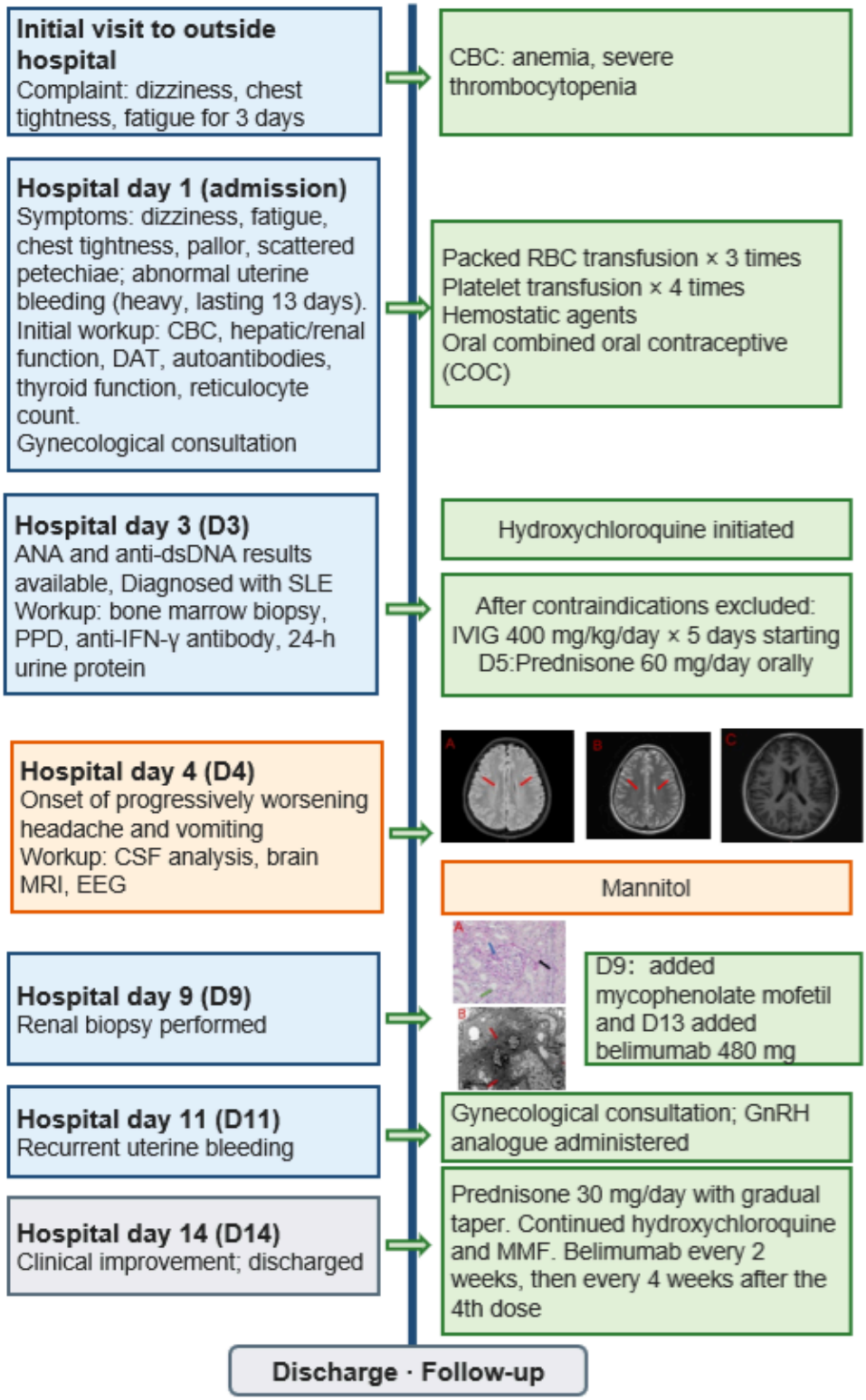


Figure 4

Timeline of the patient’s clinical course. CBC-complete blood count, DAT-direct antiglobulin test, CSF – cerebrospinal fluid; EEG – electroencephalogramy; MRI – magnetic resonance imaging; PTA – prior to admission; SLE – systemic lupus erythematosus; IVIG-intravenous Immunoglobulin; GnRH-gonadotropin releasing Hormone.