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Human parvovirus B19-associated Evans syndrome with fulminant hematologic onset mimicking thrombotic thrombocytopenic purpura: a case report

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Case Report

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Abstract

Background

Human parvovirus B19 infection rarely presents with severe bicytopenia and multiorgan involvement. Acute presentations with fulminant immune-mediated cytopenia closely mimic severe thrombotic microangiopathies (TMAs) like thrombotic thrombocytopenic purpura (TTP), making early diagnosis highly challenging.

Case presentation

A 65-year-old male was admitted with hematemesis and melena. Suspecting thrombotic thrombocytopenic purpura or other thrombotic microangiopathies, emergency plasma exchange and continuous renal replacement therapy were immediately initiated. Crucially, pre-treatment samples for ADAMTS13 and metagenomic next-generation sequencing were collected prior to these interventions. Following rapid clinical deterioration requiring endotracheal intubation, intravenous methylprednisolone was initiated. Initial platelet and hemoglobin levels were 3×10^9/L and 90 g/L, which subsequently plummeted to nadirs of 1×10^9/L and 46 g/L. Immunohematological tests identified immune-mediated cytopenia: a weakly positive direct antiglobulin test for IgG, positive C3, and spherocytes. ADAMTS13 activity was normal. The pre-treatment metagenomic next-generation sequencing exclusively detected parvovirus B19 (21632 reads), shifting the diagnostic focus to B19-associated Evans syndrome. Following comprehensive therapy, the patient demonstrated a progressive regenerative erythropoietic response. He was successfully extubated, with platelets recovering to 174×10^9/L and hemoglobin to 86-87 g/L.

Conclusions

Parvovirus B19 should be actively considered in adults presenting with fulminant immune-mediated bicytopenia mimicking TTP. Procuring critical etiological and immunohematological samples prior to plasma exchange is essential to establish an accurate final diagnosis.

Background

Human parvovirus B19 is a non-enveloped, single-stranded DNA virus that exhibits a strong tropism for erythroid progenitor cells. In immunocompetent adults, the infection is typically asymptomatic or self-limiting, with common clinical manifestations including fever, rash, arthralgia, and transient hematological abnormalities[1]. However, a small subset of patients infected with human parvovirus B19 can develop severe anemia, thrombocytopenia, pancytopenia, and multisystem inflammatory or organ dysfunction[2]. Evans syndrome (ES) is a rare autoimmune cytopenia defined by the concurrent or sequential onset of warm autoimmune hemolytic anemia and immune thrombocytopenia; a minority of patients may also develop autoimmune neutropenia[3]. This syndrome can be primary or secondary to infections, autoimmune diseases, or lymphoproliferative disorders. The diagnosis of ES in adults relies

on a comprehensive assessment of evidence for immune thrombocytopenia and immune-mediated hemolysis, requiring the systematic exclusion of other conditions that can simultaneously cause anemia and thrombocytopenia[4].

However, when patients present acutely with anemia, profound thrombocytopenia, altered mental status, acute kidney injury, and multiorgan dysfunction, ES must be emergently differentiated from thrombotic thrombocytopenic purpura (TTP) and other thrombotic microangiopathies (TMAs). Given the rapid and potentially life-threatening progression of TTP, therapeutic plasma exchange and immunosuppressive therapy are typically initiated promptly in highly suspected cases, often before ADAMTS13 activity results are available. Therefore, alongside emergency interventions, samples for ADAMTS13, hemolysis, immunohematology, and etiology should ideally be collected prior to plasma exchange and blood product transfusions to ensure the reliability of subsequent differential diagnosis and etiological evaluation.

Human parvovirus B19-associated ES is relatively rare, and cases initially presenting with fulminant immune-mediated hemolytic anemia, profound thrombocytopenia, life-threatening gastrointestinal hemorrhage, and multiorgan dysfunction are exceedingly uncommon. These manifestations can highly overlap with TMAs such as TTP, posing significant challenges for early differential diagnosis and therapeutic decision-making. We herein report a case with clinical manifestations closely mimicking TTP but ultimately diagnosed as human parvovirus B19-associated ES, aiming to raise clinicians' awareness of this rare presentation and its differential diagnosis.

Case presentation

A 65-year-old male was admitted to our intensive care unit (ICU) on July 6, 2026, presenting with a 15-hour history of hematemesis and melena. Prior to the onset of symptoms, the patient had consumed overnight wood ear mushrooms. He subsequently developed nausea and vomiting, expelling a large volume of brown fluid, accompanied by profuse diaphoresis and melena, without other abnormal symptoms. He was immediately presented to a local hospital, where laboratory tests revealed a hemoglobin level of 90 g/L, a platelet count of 3×10^9/L, and a white blood cell count of 26.21×10^9/L, along with significant hepatic and renal impairment. After receiving fluid resuscitation, hemostatic therapy, and other supportive care, he was transferred to our hospital due to his critical condition. His past medical history included hypertension and lacunar infarction, with his blood pressure being controlled through regular, long-term oral administration of amlodipine besylate and valsartan. He had no history of hematological diseases, autoimmune disorders, malignancies, or immunosuppressive therapy. His family history was unremarkable.

Physical examination on admission revealed a temperature of 36.6°C, heart rate of 108 beats/min, respiratory rate of 18 breaths/min, and blood pressure of 139/80 mmHg. The patient was conscious but appeared lethargic and pale. Scattered petechiae were observed across the skin, and hemorrhagic blisters were present in the oral cavity. Bowel sounds were hypoactive, and the remainder of the physical

examination was unremarkable. Laboratory tests upon admission indicated a white blood cell count of 22.83×10^9/L, red blood cell count of 2.80×10^{12}/L, hemoglobin of 90 g/L, and platelet count of 3×10^9/L. Procalcitonin was 11 ng/mL, and C-reactive protein was 121.98 mg/L. Renal function tests showed a creatinine level of 483.1 μmol/L and blood urea nitrogen of 34.03 mmol/L; Hepatic panels revealed an alanine aminotransferase of 321 U/L, aspartate aminotransferase of 818 U/L, total bilirubin of 35.6 μmol/L, direct bilirubin of 8.5 μmol/L, and indirect bilirubin of 27.1 μmol/L. The coagulation profile demonstrated a prothrombin time (PT) of 16.8 seconds, activated partial thromboplastin time (APTT) of 32 seconds, prothrombin time activity of 61%, an international normalized ratio of 1.37, and a D-dimer level of 22320 μg/L. Arterial blood gas analysis showed a pH of 7.331, HCO_3^- of 15.4 mmol/L, base excess of -10.5 mmol/L, and partial pressure of carbon dioxide of 29.2 mmHg, with a normal partial pressure of oxygen. Computed tomography (CT) scans of the head, chest, and abdomen revealed lacunar infarction, encephalomalacia, emphysema, pneumonia, and pulmonary nodules. Given the presence of profound thrombocytopenia, anemia, active gastrointestinal bleeding, acute kidney injury, hepatic impairment, metabolic acidosis, and coagulopathy, a preliminary diagnosis of profound thrombocytopenia complicated by gastrointestinal hemorrhage and multiorgan dysfunction was established. Thrombotic microangiopathy (TMA) was highly suspected, with a primary focus on differentiating between thrombotic thrombocytopenic purpura (TTP) and hemolytic uremic syndrome (HUS). The patient's concurrent conditions included pneumonia, emphysema, hypertension, and an old lacunar infarction.

Given the patient's critical condition and the rapid, highly lethal progression of the initially suspected disease, emergency plasma exchange followed by continuous renal replacement therapy was initiated on July 6, immediately after collecting crucial pre-treatment samples for ADAMTS13 and plasma metagenomic next-generation sequencing (mNGS). Concurrently, comprehensive treatments including red blood cell and platelet transfusions, hemostasis, empiric antimicrobial therapy, and acid suppression were administered. In the early hours of July 7, the patient experienced recurrent hematemesis of dark red fluid and massive melena, leading to a further decline in hemoglobin and platelet levels. The platelet count dropped to a nadir of 1×10^9/L, and hemoglobin fell to a nadir of 46g/L. The patient progressively developed somnolence, oliguria to anuria, dyspnea, and hypoxemia. Subsequently, endotracheal intubation and mechanical ventilation were performed, and an intravenous bolus of methylprednisolone (40mg, Q12H) was administered. A multidisciplinary consultation involving the departments of gastroenterology, hematology, and rheumatology was also organized (Table 1).

Table 1
Immunohematological and Hemolysis Assessment for Differential Diagnosis

Diagnostic Category	Specific Evidence/ Assay	Key Finding	Clinical Interpretation
Evidence for AIHA / Evans Syndrome	DAT	IgG weak+, C3+	Indicates immune-mediated hemolysis
	Peripheral blood smear	Spherocytes present	Consistent with AIHA/ extravascular hemolysis
	Osmotic fragility	Increased	Immune-mediated spherocytosis (hereditary spherocytosis excluded)
	LDH	2488 U/L	Indicates severe hemolysis
	Reticulocyte count	Increased	Reflects compensatory marrow erythropoiesis
Exclusion of TMA	Peripheral blood smear	Schistocytes absent	Strong evidence against TMA
	ADAMTS13 activity	Within normal range	Excludes severe deficiency-mediated TTP
Exclusion of PNH and Others	Acidified serum test (Ham's)	Negative	Does not support PNH
	Sucrose hemolysis test	Negative	Does not support PNH

AIHA Autoimmune hemolytic anemia; DAT Direct antiglobulin test; TMA Thrombotic microangiopathy; TTP Thrombotic thrombocytopenic purpura; PNH Paroxysmal nocturnal hemoglobinuria.

Prior to the initial plasma exchange and blood product transfusion, the patient's plasma samples were collected and preserved. Given the high clinical suspicion of TTP and the unclear etiology at that time, these pre-treatment samples were prioritized for ADAMTS13 activity testing and mNGS. The remaining major hematological and immunological assays were performed using samples collected after the initiation of plasma exchange. Relevant test results were subsequently reported: peripheral blood smears revealed anisocytosis of mature erythrocytes, with the presence of spherocytes but no schistocytes; platelets were scarce, and the morphology of the residual platelets showed no obvious abnormalities. The direct antiglobulin test (DAT) was weakly positive for IgG and positive for C3, accompanied by increased erythrocyte osmotic fragility, an elevated early reticulocyte count, and a peak lactate dehydrogenase (LDH) level of 2488 U/L. Both the acidified serum test and sucrose hemolysis test were negative; the expressions of CD55 and CD59 on granulocytes and erythrocytes were normal. ADAMTS13 activity was within the normal range, and other immunological evaluations were unremarkable. By July 8, although some of the patient's clinical manifestations and hemolysis-related parameters had improved, the patient continued to exhibit profound thrombocytopenia, severe anemia,

active gastrointestinal hemorrhage, acute kidney injury, and hepatic impairment. Based on the aforementioned laboratory findings, autoimmune hemolytic anemia (AIHA) concurrent with immune thrombocytopenia was clinically considered. Consequently, the dosage of methylprednisolone was increased to an 80 mg IV bolus, Q8H.

On July 9, the initial plasma mNGS results were reported, exclusively detecting human parvovirus B19 with 21,632 reads and a relative abundance of 95.06%; no other pathogens were detected. Because this sample was collected prior to plasma exchange and blood product transfusion, and parvovirus B19 was the sole pathogen detected, the diagnostic focus shifted from the initially suspected TMA to infection-associated immune cytopenia. Combining evidence of profound immune thrombocytopenia, a positive DAT, the presence of spherocytes, increased erythrocyte osmotic fragility, and elevated reticulocyte counts and lactate dehydrogenase levels, the patient was diagnosed with autoimmune hemolytic anemia concurrent with immune thrombocytopenia, namely Evans syndrome. A repeat plasma sample was collected on July 10 for mNGS; the results reported on July 12 still detected only human parvovirus B19, with 4 reads and a relative abundance of 93.7%, and no other pathogens were identified. Based on the detection of solely parvovirus B19 in both pre- and post-treatment plasma mNGS, normal pre-treatment ADAMTS13 activity, and evidence of immune-mediated bicytopenia, a final diagnosis of human parvovirus B19-associated Evans syndrome was established (Table 2).

Table 2
Dynamic changes in mNGS reads and relative abundance for Parvovirus B19

Specimen of mNGS	Number of detected sequences (time of collection)	Relative abundance (time of collection)
Plasma1	21632 (2026-7-6)	95.06% (2026-7-6)
Plasma2	4 (2026-7-10)	93.7% (2026-7-10)

As the patient's clinical manifestations and laboratory parameters continued to improve, methylprednisolone was reduced to an 80 mg IV bolus, Q12H on July 11. By July 13, the patient's respiratory and hemodynamic status had stabilized, and he was successfully extubated; the platelet count increased to 174×10^9/L, and hemoglobin rose to 86 g/L and remained stable. On July 14, the patient was conscious with a platelet count maintained within the normal range. There was no recurrent active bleeding, urine output recovered, and renal function improved; consequently, methylprednisolone was further reduced to a 40 mg IV bolus, Q12H. Subsequently, the patient was transferred to the Department of Nephrology for further specialized renal care and supportive treatment. The patient received a total of 7 plasma-exchange sessions during his ICU stay, following a comprehensive clinical treatment timeline (Fig. 1). Following these targeted interventions, the dynamic changes in major laboratory parameters demonstrated a progressive recovery (Fig. 2). In-hospital follow-up indicated that on July 16, methylprednisolone was changed from an IV bolus to a diluted IV infusion, and the corticosteroid dose was gradually tapered based on clinical and laboratory findings. on July 22, the patient was switched to oral prednisone 40 mg QD. As of the latest in-hospital follow-up, the patient

remained in the Department of Nephrology. He was clinically stable and conscious, with resolution of skin petechiae and no recurrent bleeding, and rehabilitation had been initiated.

Discussion and conclusions

The primary characteristic of this case is that an elderly male with no explicit background of immunosuppression presented acutely with profound thrombocytopenia, severe anemia, life-threatening gastrointestinal hemorrhage, and multiorgan dysfunction[5]. The early concurrent presentation of altered mental status, acute kidney injury, and respiratory failure highly overlapped with TMAs such as TTP, posing significant challenges for early diagnosis and therapeutic decision-making[6]. As evidence including a positive DAT, the presence of spherocytes in the peripheral blood, and an elevated early reticulocyte count was gradually obtained, the diagnostic focus shifted from TMA to immune-mediated hemolytic anemia concurrent with immune thrombocytopenia, namely Evans syndrome; both pre- and post-treatment plasma mNGS detected only human parvovirus B19, with the pre-treatment sample showing a higher read count. This provided crucial etiological evidence for identifying potential secondary factors and establishing the final diagnosis of parvovirus B19-associated Evans syndrome. Thrombotic thrombocytopenic purpura is a rapidly progressive and potentially fatal hematological emergency. The concurrent presence of profound thrombocytopenia, anemia, altered mental status, and acute kidney injury provided sufficient clinical rationale for considering TTP in the early stages. For patients highly suspected of having immune-mediated TTP, plasma exchange and immunosuppressive therapy should typically be initiated as soon as possible before ADAMTS13 results are available, to avoid delaying intervention while waiting for test results [7, 8]. In this case, the sample for ADAMTS13 testing was collected prior to the initial plasma exchange and blood product transfusion, thereby avoiding the potential impact of exogenous plasma on the results. Therefore, its normal activity level holds reliable explanatory value and does not support immune-mediated TTP driven by severe ADAMTS13 deficiency. Combined with the absence of schistocytes on the peripheral blood smear, as well as a positive DAT and the presence of spherocytes, the diagnostic focus further shifted toward immune-mediated cytopenia. It should be noted that while normal ADAMTS13 activity primarily reduces the likelihood of immune-mediated TTP, it cannot independently rule out other types of TMAs. Furthermore, although prolonged PT and elevated D-dimer and fibrin degradation products (FDP) upon admission suggested coagulation system activation, fibrinogen did not persistently decrease, the APTT remained within the normal range, and PT recovered relatively quickly. Thus, the overall clinical picture does not support typical progressive disseminated intravascular coagulation (DIC).

Evans syndrome is an immune cytopenia characterized by the concurrent or sequential onset of warm autoimmune hemolytic anemia and immune thrombocytopenia. Its diagnosis necessitates the independent confirmation of immune-mediated hemolysis and immune thrombocytopenia, alongside the exclusion of thrombotic microangiopathies, medications, hematological malignancies, and other secondary etiologies[4]. In this case, the platelet count fell to a nadir of 1×10^9/L, and peripheral blood platelets were extremely scarce, though the morphology of residual platelets appeared unremarkable. Following corticosteroid and comprehensive therapy, the rapid recovery of the platelet count supported

immune-mediated thrombocytopenia. Concurrently, the DAT was weakly positive for IgG and positive for C3. The presence of spherocytes in the peripheral blood, increased erythrocyte osmotic fragility, and elevated levels of indirect bilirubin, lactate dehydrogenase, and reticulocytes collectively suggested peripheral immune-mediated erythrocyte destruction[3]. These manifestations are inconsistent with microangiopathic hemolysis, which is primarily characterized by a prominent increase in schistocytes. Taken together, the evidence robustly supports autoimmune hemolytic anemia (AIHA) concurrent with immune thrombocytopenia, establishing the diagnosis of Evans syndrome.

Human parvovirus B19 exhibits a pronounced tropism for erythroid progenitor cells, typically manifesting hematologically as suppressed erythropoiesis; in severe cases, this can lead to reticulocytopenia, transient aplastic crisis, or pure red cell aplasia[9]. However, B19 infection can also be accompanied by immune-mediated hemolytic anemia, immune thrombocytopenia, and pancytopenia, occasionally presenting as Evans syndrome. In this case, the prominent early elevation in the reticulocyte count, rather than the typical reticulocytopenia, may partially reflect a robust physiological compensatory hematopoietic response of the bone marrow following severe acute gastrointestinal hemorrhage. Conversely, combined with the positive DAT, the presence of spherocytes, and profound thrombocytopenia, this presentation is also highly consistent with infection-triggered, peripheral immune-mediated blood cell destruction. This pathological process—intertwining peripheral loss with intense compensatory bone marrow hyperplasia—masked or replaced the typical hematopoietic suppression associated with B19, indicating that the anemia was not primarily caused by the virus's direct inhibition of erythropoiesis[10]. B19 infection may induce autoantibody formation through mechanisms such as polyclonal B cell activation, molecular mimicry, complement activation, and abnormal autoantigen exposure. However, since these mechanisms were not directly verified in this case, B19 is more appropriately considered a probable immune trigger, rather than concluding that its specific pathogenic mechanism has been definitively proven.

The significant value of the etiological evidence in this case lies in the fact that the initial plasma mNGS sample was collected prior to plasma exchange and all blood product transfusions, detecting only human parvovirus B19 with 21632 reads and a relative abundance of 95.06%. This critical sampling timing excluded the possibility of detecting exogenous B19 nucleic acids introduced via blood transfusions, thereby endowing the result with high etiological explanatory value. After the patient underwent multiple sessions of plasma exchange and blood product transfusions, a repeat plasma mNGS sample collected on July 10 still exclusively detected B19, yielding 4 reads with a relative abundance of 93.7%. The repeated detection of the identical pathogen, even after the circulating plasma components had been profoundly altered by repeated exchanges and transfusions, further strengthened the correlation between B19 and the current illness. Concurrently, the marked reduction in mNGS reads synchronized temporally with the cessation of active bleeding, the recovery of the platelet count, and the improvement in reticulocytes and hemolysis-related parameters. This suggests a potential decrease in the circulating B19 nucleic acid burden during treatment. However, because mNGS read counts do not represent standardized quantitative viral loads, and the patient received multiple concurrent therapies, this change primarily serves as supportive evidence consistent with the clinical evolution. It cannot be

utilized to accurately quantify changes in viral load or to evaluate the specific antiviral efficacy of any single therapeutic intervention.

The laboratory diagnosis of human parvovirus B19 infection typically requires a combination of virus-specific IgM, IgG, and viral DNA testing. B19-IgM generally indicates a recent infection, whereas IgG can persist long-term post-infection; quantitative PCR is valuable for evaluating the level of viremia and its dynamic changes[11]. In this case, B19-IgM, IgG, and quantitative PCR assays were not performed; therefore, it is impossible to accurately determine the infection stage and viral load. Consequently, it is difficult to further differentiate among an acute infection, persistent DNAemia following a recent infection, or low-level nucleic acid residue from a past infection. However, the pre-treatment plasma mNGS yielded a high read count exclusively for B19, and the post-treatment follow-up still detected only this identical virus; furthermore, these findings demonstrate a clear temporal correlation and biological plausibility with the immune-mediated bicytopenia and clinical evolution. Synthesizing the etiological, hematological, and clinical evidence, establishing the diagnosis of human parvovirus B19-associated Evans syndrome is well-justified. The term "associated" not only reflects the potential triggering role of B19 in the current illness but also maintains a rigorous scientific boundary regarding the infection stage and specific pathogenic mechanisms, which remain incompletely understood.

In terms of treatment, there are currently no specific antiviral agents for human parvovirus B19 infection; therapeutic strategies largely depend on the patient's immune status, underlying conditions, and primary clinical manifestations. Intravenous immunoglobulin (IVIG) is primarily reserved for patients with compromised immunity, persistent viremia, or B19-associated chronic erythropoietic disorders. Given that this patient had no explicit history of immunosuppression and presented primarily with immune-mediated hemolytic anemia and immune thrombocytopenia, the therapeutic focus was placed on corticosteroids and critical organ support. Following the escalation of the methylprednisolone dose, the patient's platelet count rapidly recovered, active bleeding ceased, and hemoglobin alongside hemolysis-related parameters progressively improved; this clinical course further corroborates the diagnosis of immune-mediated blood cell destruction. Nevertheless, because the patient concurrently received plasma exchange, red blood cell and platelet transfusions, CRRT, mechanical ventilation, and other supportive interventions, the recovery of hematological and organ functions cannot be solely attributed to corticosteroids. Likewise, these findings do not establish plasma exchange as a specific treatment for parvovirus B19-associated Evans syndrome.

Several limitations of this case report should be acknowledged. First, certain immunohematological tests, including the DAT, were performed after plasma exchange and blood product transfusions, meaning the results may have been influenced by these therapeutic interventions; additionally, haptoglobin was not measured, rendering the diagnostic chain of evidence for hemolysis incomplete. However, the positive DAT, presence of spherocytes, elevated indirect bilirubin and lactate dehydrogenase, and increased reticulocyte count collectively support peripheral immune-mediated hemolysis. Second, B19-specific IgM and IgG, along with quantitative PCR, were not evaluated; therefore, the exact infection stage and viral load dynamics cannot be accurately determined. Finally, because the

patient concurrently received corticosteroids, plasma exchange, blood product transfusions, CRRT, and other organ support measures, it is difficult to ascertain the independent contribution of any single therapeutic intervention to the observed improvements in hematological and organ functions.

This case suggests that human parvovirus B19-associated Evans syndrome can occur in adults with no explicit background of immunosuppression, primarily presenting with fulminant immune-mediated cytopenia, life-threatening gastrointestinal hemorrhage, and multiorgan dysfunction; its early clinical features can highly mimic TTP. For acute immune-mediated bicytopenia of unknown etiology, human parvovirus B19 should be included in the etiological differential diagnosis. Both pre- and post-treatment plasma mNGS detected only human parvovirus B19. Combined with evidence of profound immune thrombocytopenia and immune-mediated hemolysis, this provided a crucial basis for the diagnosis of B19-associated Evans syndrome[12]. For critically ill patients requiring emergency initiation of plasma exchange and blood product transfusion, samples for ADAMTS13, hemolysis, immunohematology, and etiology should be collected prior to treatment whenever possible, to enhance the reliability of subsequent differential diagnosis and etiological evaluation.

Abbreviations

AIHA
Autoimmune hemolytic anemia
APTT
Activated partial thromboplastin time
CRRT
Continuous renal replacement therapy
Cr
Creatinine
CT
Computed tomography
DAT
Direct antiglobulin test
DIC
Disseminated intravascular coagulation
ES
Evans syndrome
FDP
Fibrin degradation products
Hb
Hemoglobin
HUS
Hemolytic uremic syndrome
IBIL

Indirect bilirubin
ICU
Intensive care unit
IVIG
Intravenous immunoglobulin
LDH
Lactate dehydrogenase
mNGS
Metagenomic next-generation sequencing
PCR
Polymerase chain reaction
PLT
Platelet
PNH
Paroxysmal nocturnal hemoglobinuria
PT
Prothrombin time
Ret
Reticulocyte
TMA
Thrombotic microangiopathy
TTP
Thrombotic thrombocytopenic purpura

Declarations

Ethics approval and consent to participate

In accordance with the policies of the Medical Ethics Committee of Liaocheng People's Hospital, ethical approval is waived for anonymized case reports.

Consent for publication

Written informed consent for publication of clinical details or clinical images was obtained from the patient.

Data availability

The datasets generated and/or analysed during the current study are not publicly available due to strict patient privacy, confidentiality regulations, and the Human Genetic Resources Administration of China (HGRAC) regulations regarding clinical sequencing data. The metagenomic next-generation sequencing (mNGS) was performed exclusively for clinical diagnostic purposes rather than research, and the raw

sequencing data contains human genetic material. However, de-identified clinical data and the pathogen detection reports are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

ZPX wrote the manuscript and curated the data. XLZ and LNZ reviewed and edited the manuscript. All authors read and approved the final manuscript.

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Figures

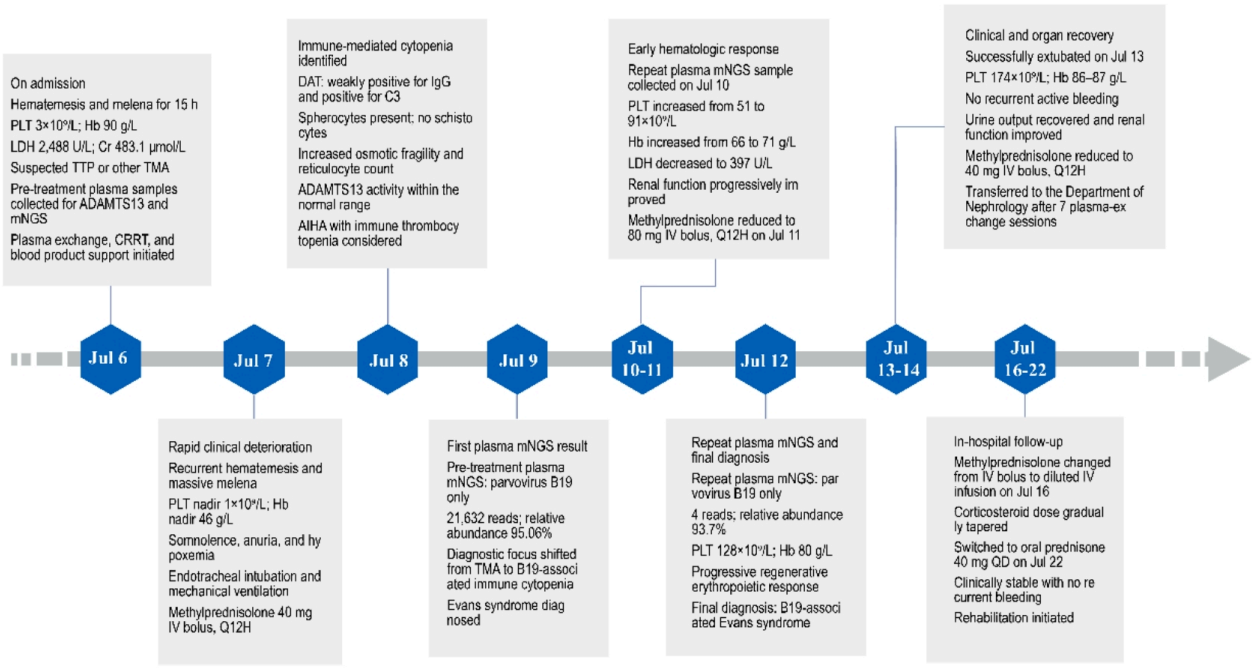


Figure 1

The treatment timeline of the case

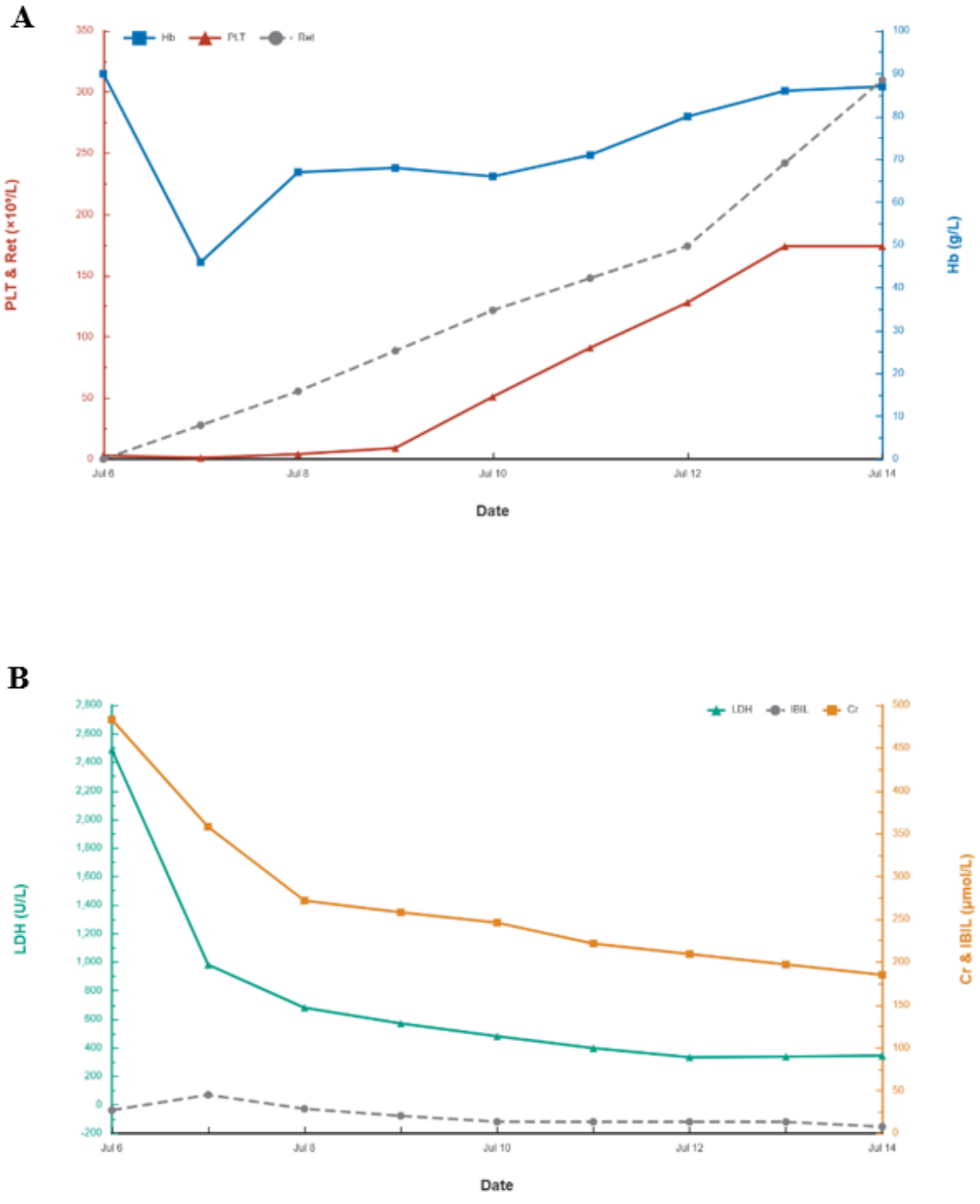


Figure 2

Dynamic changes in major laboratory parameters during the clinical course (A): Trends of PLT (left Y-axis), Hb, and Ret% (right Y-axis), highlighting the initial profound cytopenia and subsequent robust compensatory erythropoiesis. (B)**:** Trends of LDH (left Y-axis), Cr, and IBIL (right Y-axis), demonstrating the progressive resolution of hemolysis and renal dysfunction post-treatment.