



Alexa, 32, has lived with systemic lupus erythematosus (SLE) since her early twenties.

From the start, her disease carried a clear serologic signature: she remained persistently anti-dsDNA positive, with clinical flares often mirroring changes in inflammatory activity.

Her course worsened after she developed secondary antiphospholipid syndrome (APS). Antiphospholipid testing showed anticardiolipin IgA and anti-β2-glycoprotein I (β2GPI) IgA positivity, with a lupus anticoagulant profile and persistent positivity over time. Clinically, she suffered recurrent thromboses and a transient ischemic attack—requiring lifelong anticoagulation.

Despite immunosuppressive therapy, she progressed to end-stage renal disease within 18 months and started dialysis while being listed for a kidney transplant. APS made every step precarious: clotting risk on one side, bleeding risk on the other—especially around procedures and transfusions.

A sudden turn in the emergency department

One winter morning, Alexa arrived at the emergency department exhausted, tachycardic, and short of breath. Her partner had noticed a yellowish skin tone, and Alexa reported:

- Dark urine for several days
- Fatigue far worse than usual
- New dyspnoea and mild chest tightness

On examination she was pale and mildly jaundiced, heart rate 118 bpm, respiratory rate 24/min with shallow effort, and reduced breath sounds at the bases.

Because her symptoms suggested impaired oxygen delivery, the team rapidly reassessed oxygenation and perfusion (including serial blood gas checks) while urgent labs revealed severe anaemia with clear evidence of haemolysis:

- Haemoglobin 6.6 g/dL (10.8 g/dL two weeks earlier)
- Reticulocytosis
- LDH elevated, indirect bilirubin elevated
- Haptoglobin undetectable
- Positive direct antiglobulin test (DAT): IgG warm autoantibody

The diagnosis was warm autoimmune haemolytic anaemia (AIHA)—a known but serious SLE complication—now threatening oxygen delivery in a dialysis patient already at high thrombotic risk due to APS.

Transfusion under constraints

Transfusion was necessary, but not straightforward. With a positive DAT and warm autoantibody interference, compatibility testing and unit selection required an extended, cautious approach. APS primarily influenced the anticoagulation balance and monitoring strategy around transfusion.

The transfusion plan prioritized safety and predictability:

- Extended immunohematology work-up with Rh phenotype and Kell (K) typing
- Careful transfusion of compatible RBC concentrates under close monitoring
- Close coordination between intensive care, nephrology, and the transfusion laboratory to reduce delays and avoid over-transfusion in a dialysis patient

Stabilization through multidisciplinary care

Alexa was admitted to intensive care. Management focused on controlling haemolysis, protecting organs, and balancing anticoagulation:

- High-dose IV steroids for AIHA (and to cover possible lupus pulmonary involvement)
- Oxygen support with frequent reassessment, including serial blood gases
- Careful optimization of anticoagulation for APS (reassessed daily against bleeding risk)
- Dialysis adjustments to avoid volume overload

Over the next 72 hours, haemoglobin improved, bilirubin and LDH began to fall, and respiratory status stabilized. She stepped down from ICU care after gradual weaning to low-flow oxygen.

The transplant moment: antibody risk defined, compatibility confirmed

Weeks later, Alexa received a kidney offer. Given her autoimmune background and recent transfusion exposure (a potential sensitizing event), the transplant team approached her as elevated immunologic-risk and performed a structured pre-transplant antibody and compatibility assessment:

- High-resolution HLA typing of donor and recipient to define the match and anticipate immunologic risk
- Anti-HLA antibody screening to assess sensitization status
- Antibody identification with single antigen bead (SAB) testing to map specificity and detect any donor-specific antibodies (DSA)
- A virtual crossmatch derived from the antibody specificity profile, aligned with the donor's HLA type
- When results were borderline, complement-binding DSA assessment (e.g., C1q/C3d) helped refine the risk of antibody-mediated rejection and guide peri-transplant immunosuppression intensity

With immunologic compatibility confirmed and a tailored immunosuppression plan in place, surgery proceeded.

Intraoperative bleeding: viscoelastic testing–guided hemostasis

During transplantation, unexpected diffuse bleeding developed despite meticulous surgical technique. With no clear surgical source—and given the potential for dilutional and uraemia-related coagulopathy—the team performed intraoperative viscoelastic testing:

- EXTEM CT: prolonged
- INTEM CT: prolonged

Interpretation: delayed clot initiation consistent with coagulation factor deficiency (with dilutional and uraemia-related contributions).

- FIBTEM A10: markedly reduced
- FIBTEM MCF: markedly reduced

Interpretation: low functional fibrinogen as the primary driver of weak clot firmness.

- LI30 (EXTEM): within normal range

Interpretation: no evidence of hyperfibrinolysis.

Instead of empiric escalation of blood products, the team applied a targeted, test-guided strategy (carefully weighed

against APS-related thrombotic risk):

- Fibrinogen concentrate to correct the low FIBTEM A10/MCF and strengthen clot formation
- Prothrombin complex concentrate (PCC) to address prolonged EXTEM/INTEM clotting times and support thrombin generation Bleeding rapidly decreased, surgical conditions improved, and the transplant proceeded with stable hemostasis and good graft perfusion.

Forward look: complex, but possible

Alexa's story did not "end" with surgery. It became a long-term balance:

- preventing APS-related thrombosis with tailored anticoagulation
- vigilance for AIHA relapse
- individualized immunosuppression guided by ongoing immune monitoring, including post-transplant antibody surveillance when indicated She left the hospital still fragile—but stable. Not a miracle: a chain of decisions, multidisciplinary teamwork, and data-guided medicine—making the next step possible.