Supplementary material

Methods

Clinical assessment

All patients were followed at the outpatient rheumatology clinic of the Department of Pathophysiology, National and Kapodistrian University of Athens, Greece. Moreover, all patients satisfied the classification criteria for APS (2023 ACR/EULAR), but not for any other autoimmune disease, including the 2012 SLICC and the 2019 EULAR/ACR classification criteria for systemic lupus erythematosus. None of the patients had a high-risk venous thromboembolism (VTE) profile. The diagnosis of primary APS was established following the exclusion of alternative causes of VTE, including inherited thrombophilias, malignancy, infections, and systemic autoimmune rheumatic diseases.

Supplementary Table S1. Serum autoantibody profile of the three APS patients during follow-up.

Immunological profile	At APS diagnosis	Prior to BLM administration	After BLM administration
Patient 1			
APS-related			
anti-CL-IgM	low titre	-	-
anti-CL-IgG	-	-	-
anti-β2GPI-IgM	-	-	-
anti-β2GPI-IgG	-	-	-
LAC	positive	positive	-
ANA	-	-	1/160
Anti-dsDNA	-	-	-
Anti-ENA	-	-	-
Patient 2			
APS-related			
anti-CL-IgM	-	-	-
anti-CL-IgG	moderate titre	low titre	low titre
anti-β2GPI-IgM		-	-
anti-β2GPI-IgG	-	-	-
LAC	-	positive	-
ANA	positive	1/640	1/1280
Anti-dsDNA	1/160	-	-
Anti-ENA	-	-	anti-Ro/SSA
Patient 3			
APS-related			
anti-CL-IgM	-	-	-
anti-CL-IgG	high titre	high titre	high titre
anti-β2GPI-IgM	-	-	-
anti-β2GPI-IgG	high titre	high titre	high titre
LAC	-	-	-
ANA	1/1280	1/160	1/640
Anti-dsDNA	-	-	-
Anti-ENA	-	-	-

At the time of APS diagnosis, Patient 1 had low-titre IgM anti-CL antibodies (27 IU), which became negative following mycophenolate/rituximab therapy, while LAC remained persistently positive. Twelve months after belimumab administration, the patient manifested negative LAC, as well as positive ANA at a titre of 1/160. Prior to belimumab administration, Patient 2 presented positive LAC, moderate IgG anti-CL (49 IU) and low IgG anti-CL (33 IU) levels, as well as high ANA titre (1/640). In this patient, belimumab treatment was followed by conversion to negative LAC, along with an increase in ANA titre (1:1280) and the emergence of positive anti-Ro/SSA antibody levels. Patient 3 exhibited high-titre anti-CL IgG (97 IU) and anti-β2GPI IgG (174 IU), with an ANA titre of 1:160 prior to BLM initiation. After initiation of treatment, IgG anti-CL and IgG anti-β2GPI persisted at high levels (108 IU and >300 IU, respectively), with a concurrent rise in ANA titre to 1:640. In the table, negative values are indicated by a dash.
BLM: belimumab; anti-CL: anticardiolipin antibodies; anti-β2GPI: anti-β2 glycoprotein-I antibodies; LAC: lupus anticoagulant; ANA: anti-nuclear antibodies; Anti-dsDNA: anti-double strand DNA; Anti-ENA: autoantibodies to extracted nuclear antigens (Ro/SSA, La/SSB, Sm, U1nRNP, Jo-1, Scl70). Normal values for anti-CL and anti-β2GPI were 0–18 IU.

Supplementary Fig. S1. Time course of platelet count recovery following belimumab therapy. The initiation of belimumab (timepoint zero) was found to gradually restore all patients' platelet counts to normal levels.
Moreover, Patient 1 and Patient 3 successfully discontinued steroids without relapse of thrombocytopenia at 10 and 6 months, respectively (arrows).