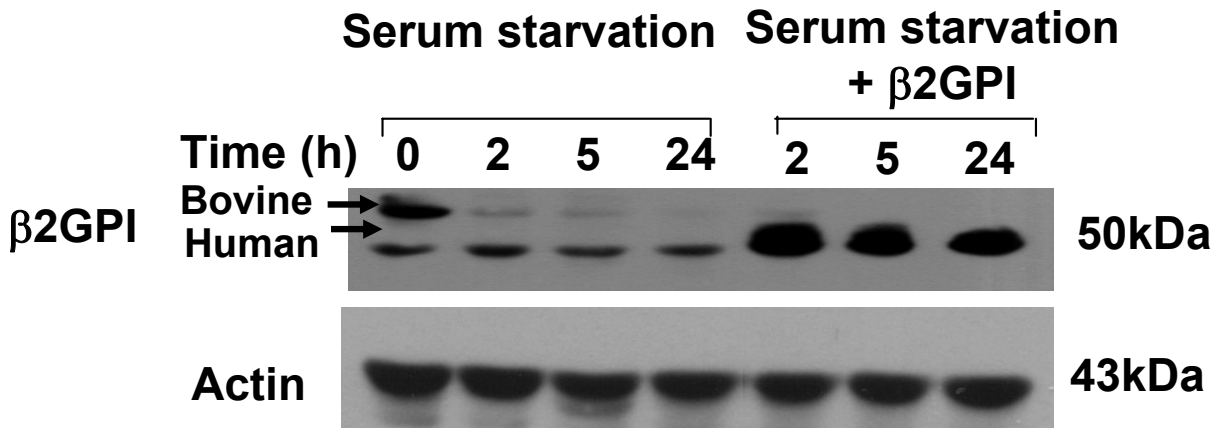
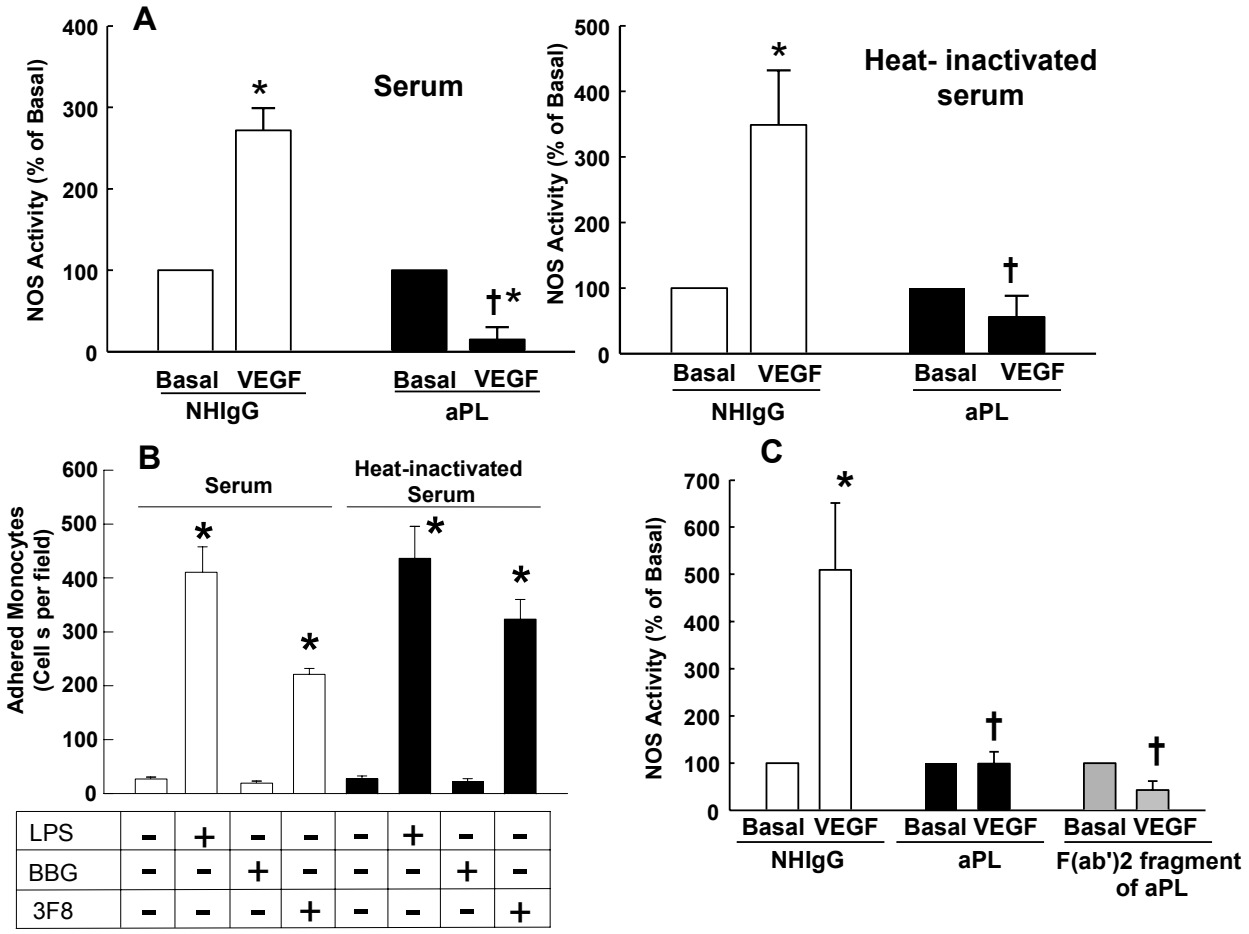
Supplemental Table. Clinical and laboratory features of the patients used as a source of human polyclonal aPL

Patient	Age	Sex	aCL(>80 LA PGA)	anti-β2GPI	Clinical Features
1	43	M	+	+	Arterial thrombosis, digital infarct, leg ulcer
2	50	F	+	+	Arterial thrombosis, pregnancy losses, catastrophic APS, myocardial infarction
3	57	M	+	+	Arterial thrombosis, recurrent pulmonary hemorrhage, catastrophic APS
4	36	F	+	+	Deep venous thrombosis, renal microthrombotic angiopathy, stroke

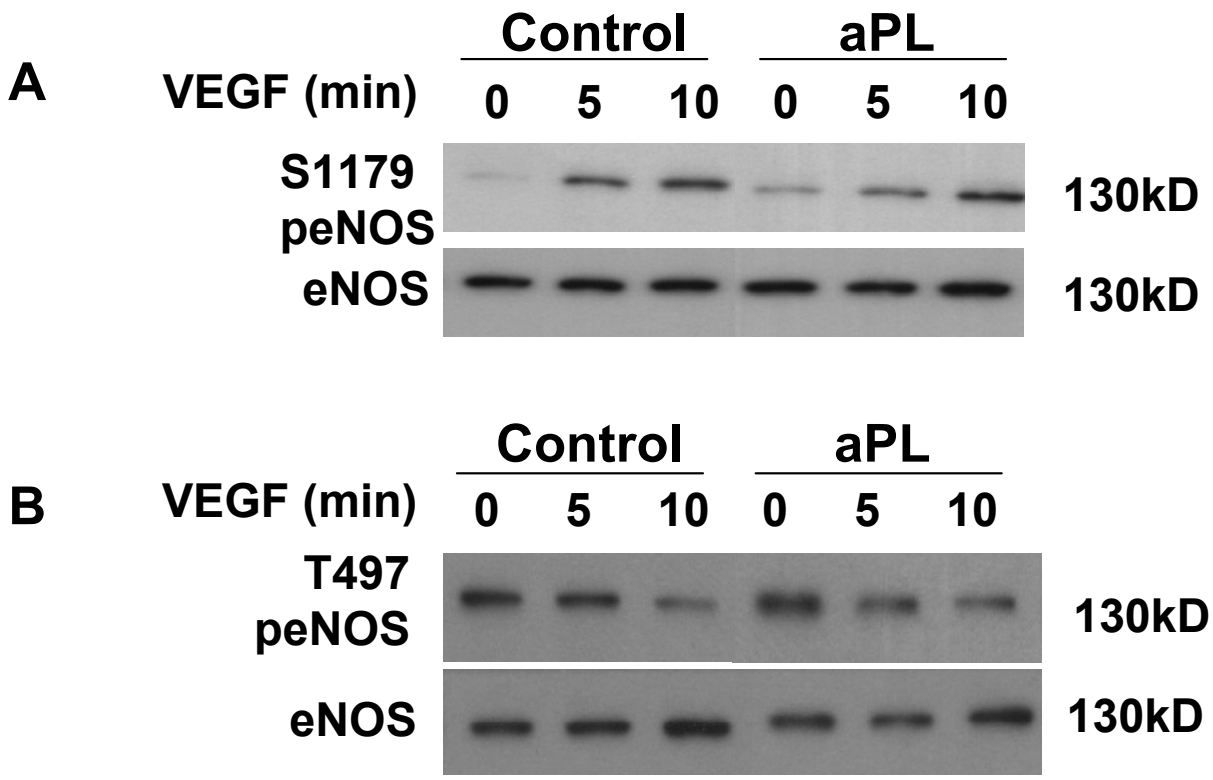


Supplemental Figure 1: Loss of β2GPI with serum starvation, and β2GPI reconstitution. Bovine aortic endothelial cells (BAEC) were serum starved for 0-24h and harvested, or treated with human β2GPI (5 μg/ml) for 1h after serum starvation for 2-24h. Whole cell lysates were immunoblotted for β2GPI. Findings shown are representative of 3 independent experiments.



Supplemental Figure 2: aPL inhibition of eNOS and resulting increase in adhesion occur independent of complement via Fab'.

(**A**) BAEC cultured in media containing untreated serum (left panel) or heat-inactivated, complement-free serum (right panel) were pretreated for 15 min with NHIgG or aPL (100 µg/ml), and eNOS activity was measured under basal conditions or in the presence of VEGF (100ng/ml) for 15 min. Values are mean±SEM, n=4, *p<0.05 vs basal, †p< 0.05 vs. NH IgG. (**B**) BAEC were cultured in untreated serum or heat-inactivated serum in the presence of vehicle, LPS (100 ng/ml), control IgG (BBG, 10 µg/ml) or anti-β2GPI antibody (3F8, 10 µg/ml) for 18h, and monocyte adhesion to BAEC was evaluated. Values are mean±SEM, n=4, *p< 0.05 vs no treatment. (**C**) BAEC were pretreated for 15 min with NHIgG, intact aPL, or Fab' fragments of aPL (100 µg/ml), and eNOS activity was measured under basal conditions or in the presence of VEGF for 15 min. Values are mean±SEM, n=4, *p< 0.05 vs basal, †p< 0.05 vs NHIgG.



Supplemental Figure 3: aPL does not alter eNOS S1179 or T497 phosphorylation in the absence of β2GPI. BAEC were serum-starved for 24h to remove β2GPI, pretreated for 15 min with NHIgG or aPL (100 μg/ml), and incubated with VEGF (100ng/ml for 0-10 min). Whole cell lysates were prepared and immunoblotted for phospho-eNOS and total eNOS using polyclonal phospho-S1179 eNOS antibody and monoclonal eNOS antibody, respectively (**A**). Samples were independently immunoblotted for phospho-T497 eNOS and total eNOS (**B**). Findings shown are representative of 3 independent experiments.

Legends for Leukocyte Rolling Videos

Leukocyte adhesion was determined as described in Materials and Methods. Video 1-4: $eNOS^{+/+}$ (**Video 1 and 2**) or $eNOS^{-/-}$ (**Video 3 and 4**) mice were injected intraperitoneally with NHIgG (**Video 1 and 3**, 100 µg) or aPL (**Video 2 and 4**, 100 µg), and 24 h later prepared for intravital microscopy. Video 5-8: $apoER2^{+/+}$ (**Video 5 and 6**) or $apoER2^{-/-}$ (**Video 7 and 8**) mice were injected intraperitoneally with NHIgG (**Video 5 and 7**, 100 µg) or aPL (**Video 6 and 8**, 100 µg), and 24 h later prepared for intravital microscopy. All leukocytes were fluorescence-labeled and the mesentery was exposed for the observation and recording of images of leukocyte adhesion and rolling.