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CASE REPORT

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When One Antibody Disappears, and Others Emerge: A Six-Year Evolution of Antiphospholipid Antibodies in a Pregnant Woman with Systemic Lupus Erythematosus

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Abstract

Antiphospholipid antibodies (aPL) are frequently detected in patients with systemic autoimmune diseases, particularly in systemic lupus erythematosus (SLE), and are associated with thrombotic events and adverse pregnancy outcomes. Although persistent aPL positivity is crucial for antiphospholipid syndrome (APS) classification, increasing evidence suggests that aPL profiles may evolve over time. Longitudinal observations documenting the transition between antiphospholipid antibody specificities remain limited.
We report the case of a 33-year-old woman with SLE diagnosed in 2019, who initially presented with persistent high-titer anti-β2GPI IgM (confirmed on repeat testing after 12 weeks) positivity. Lupus anticoagulant (LAC) and anti-β2GPI IgG were initially negative. During her first pregnancy in 2024, anti-β2GPI antibodies became negative, while LAC activity increased from 42.3 to 48.3 sec and anticardiolipin IgM antibodies became positive (60.6 U/mL). The pregnancy resulted in a preterm birth at 36 weeks of gestation. During the second pregnancy, in March 2026, LAC activity further increased to 55.1 sec, and both aCL IgM and IgG antibodies became positive. Throughout the six-year follow-up period, the patient remained free of arterial or venous thrombotic events and remained on stable treatment with hydroxychloroquine, methylprednisolone, and low-dose aspirin.
This case demonstrates a remarkable longitudinal evolution of the aPL profile in a woman with SLE, characterized by the disappearance of high-titer anti-β2GPI IgM antibodies and subsequent emergence of LAC and aCL antibodies. These findings emphasize the dynamic nature of antiphospholipid autoimmunity and support the importance of long-term monitoring of aPL profiles in patients with SLE, particularly during reproductive years. (**International Journal of Biomedicine. 2026;16(3):395-397.**)

Keywords: antiphospholipid antibodies • lupus anticoagulant • aCL antibodies • systemic lupus erythematosus

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Introduction

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterized by immune dysregulation and the production of a spectrum of autoantibodies. Among them, antiphospholipid antibodies (aPL), including lupus anticoagulant (LAC), anticardiolipin antibodies (aCL), and anti-β2GPI antibodies (anti-β2GPI), are of particular clinical importance due to their association with thrombotic events and pregnancy morbidity.[1-3] Antiphospholipid antibodies are detected in up to 50% of patients with SLE, while approximately 20% of patients with APS have underlying SLE.[3]

Clinical practice and current APS classification criteria primarily focus on the presence and persistence of aPL.[1] In contrast, relatively little attention has been given to the longitudinal evolution of individual antibody profiles. Although persistent aPL positivity remains central in APS classification, increasing evidence suggests that antiphospholipid antibody profiles may evolve over time, with fluctuations in antibody titer, disappearance of previously detected antibodies, and

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emergence of new antibody specificities.[4,5] Understanding these temporal changes may be relevant in women of reproductive age, in whom alterations in aPL profiles could influence risk stratification and pregnancy management and long- term clinical surveillance.[2]

This case describes a woman with SLE who demonstrated a longitudinal six-year evolution of her antiphospholipid antibody profile. Initial high-titer anti-β2GPI IgM positivity was confirmed on repeat testing at least 12 weeks later. Her antiphospholipid antibody profile evolved, with the disappearance of anti-β2GPI antibodies and the subsequent emergence of lupus anticoagulant and anticardiolipin antibodies during follow-up.

Case Presentation

A 33-year-old woman was diagnosed with SLE in 2019 based on clinical findings and immunological abnormalities, including positive antinuclear antibodies (ANA), positive anti-double-stranded DNA antibodies (anti-dsDNA), and decreased complement levels (C3 and C4). She fulfilled the 2019 ACR/EULAR classification criteria for SLE. Following the diagnosis, she was treated with hydroxychloroquine, methylprednisolone, and low-dose aspirin with regular rheumatologic follow-up.

Initial antiphospholipid antibody testing performed at the time of SLE diagnosis revealed isolated high-titer anti-β2GPI IgM positivity >200 RU/mL (reference value < 20 RU/mL), while anti-β2GPI IgG and lupus anticoagulant were negative. The patient had no history of arterial or venous thrombosis, previous pregnancy losses, or family history of APS.

In July 2024, during her first trimester of pregnancy, laboratory reassessment demonstrated persistently decreased C3 and C4 levels. ENA profile (SSA positive, anti-Sm positive), ANA and anti-dsDNA remained positive. Lupus anticoagulant activity was measured at 42.3 sec, while both anti-β2GPI IgM and IgG were negative on repeat testing, indicating the disappearance of the previously detected high-titer anti-β2GPI IgM antibodies. Repeat testing in November 2024 showed further changes in antiphospholipid antibody profile. Lupus anticoagulant activity increased to 48.3 sec and anticardiolipin IgM antibodies became positive at 60.6 U/mL (reference value <12 U/mL). No thrombotic manifestations were observed. The patient's first pregnancy subsequently resulted in a preterm birth at 36 weeks of gestation.

In March 2026, during her second pregnancy, repeat antiphospholipid antibody testing revealed continued evolution of the antiphospholipid antibody profile. Lupus anticoagulant activity increased to 55.1 sec. Anticardiolipin antibodies were positive for both IgM and IgG, with values of 38.4 U/mL and 20.7 U/mL (reference value <12 U/mL), respectively.

Throughout the six-year follow-up period, the patient remained free of documented arterial or venous thrombotic events, and no major therapeutic modifications were introduced. Serial laboratory assessments demonstrated the longitudinal evolution of the antiphospholipid antibody profile, characterized by the disappearance of high-titer anti-β2GPI IgM antibodies and the subsequent emergence of lupus anticoagulant and anticardiolipin antibodies.

Disussion

This case report illustrates a remarkable longitudinal evolution of the antiphospholipid antibody profile in a woman with SLE over a six-year follow-up. The most notable finding was the transition from isolated high-titer anti-β2GPI IgM positivity at the time of diagnosis to the subsequent disappearance of anti-β2GPI antibodies and emergence of LAC and anticardiolipin antibodies during this period. These changes occurred despite stable treatment with hydroxychloroquine, methylprednisolone, and low-dose aspirin, with no documented thrombotic manifestations.

Antiphospholipid antibodies are frequently detected in SLE patients and are associated with an increased risk for thrombotic and obstetric complications.[2,3] Current classification criteria focus on the persistence of aPL positivity and require laboratory confirmation after at least 12 weeks.[1] Accumulating evidence suggests that the aPL profile is dynamic and may change throughout the disease's course. This case provides a clear illustration of this phenomenon.

Table 1.

Evolution of antiphospholipid antibody profile during follow-up.

Time point	Clinical status	LAC (sec)	aCL IgM (U/mL)	aCL IgG (U/mL)	Anti-β2GPI IgM (RU/mL)	Anti-β2GPI IgG (RU/mL)	Other findings
2019	SLE diagnosis	Neg	-	-	>200	Neg	ANA + anti-dsDNA + low C3/C4
07.2024	First pregnancy (1st trimester)	42.3	Neg	Neg	Neg	Neg	ANA + anti-dsDNA + low C3/C4, ENA +
11.2024	Follow-up during pregnancy	48.3	60.6	Neg	Neg	Neg	No thrombotic events
2025	Pregnancy outcome						Preterm birth at 36 weeks
03.2026	Second pregnancy (1st trimester)	55.1	38.4	20.7	Neg	Neg	Low C3/C4

LAC-lupus anticoagulant, aCL-anticardiolipin antibodies, anti-β2GPI-anti-beta-2 glycoprotein I, C3- complement component 3, C4-complement component 4, ENA-extractable nuclear antigen.

In longitudinal studies of SLE patients, similar fluctuations and serological transitions have been reported.[4] aPL may exhibit variable patterns over time, including seroconversion from positive to negative status and the appearance of new antibody specificities. These observations support the concept that antiphospholipid autoimmunity represents a dynamic process.[5]

The mechanism underlying these changes remains incompletely understood. Persistent immune dysregulation and ongoing B-cell activation in SLE may contribute to shifts in autoantibody profiles over time, although the factors driving longitudinal evolution remain unclear.[6] The transition observed in this patient, characterized by the disappearance of anti-β2GPI antibodies and subsequent emergence of LAC and aCL antibodies, may reflect the natural evolution of antiphospholipid autoimmunity rather than a stable antibody pattern.

The absence of arterial or venous thrombosis despite the longitudinal evolution of aPL profile is another noteworthy aspect. LAC is considered the strongest laboratory predictor of thrombotic and obstetric manifestations among antiphospholipid antibodies. The progressive increase in LAC activity observed in this patient may be clinically relevant despite the absence of thrombotic manifestations. However, not all individuals with positive aPL develop clinical APS. The occurrence of thrombosis is thought to depend on additional genetic, inflammatory, or environmental factors known as a "second hit" in susceptible individuals.[7] The present findings also highlight the importance of repeated testing, as a single negative result may not accurately reflect future risk and may fail to capture the dynamic evolution of aPL profile.

The longitudinal evolution of aPL antibody profiles remains incompletely understood. While fluctuations in aPL status have been reported, no consistent pattern of antibody evolution has been established. Factors such as previous births, the interpregnancy interval, hormonal changes, and environmental exposures have been suggested as potential modulators of aPL dynamics; however, robust evidence supporting these associations is still lacking.

This woman experienced two pregnancies during the follow-up period, with a preterm birth at 36 weeks of gestation during the first pregnancy. Pregnant women with SLE and aPL positivity have an increased risk for adverse obstetric outcomes.[2]

Although a direct relationship cannot be established from a single case, the emergence and progressive increase of LAC during pregnancy may be clinically relevant, given the well-recognized association between LAC positivity and adverse pregnancy outcomes. This observation further emphasizes the importance of close laboratory surveillance in pregnant women with SLE.

This report has some limitations. As a single case report, it cannot establish causal relationships between changes in antiphospholipid antibody profiles and pregnancy, hormonal influences, or other clinical factors. Furthermore, these findings cannot be generalized to all patients with SLE or APS. Nevertheless, the prolonged six-year follow-up with repeated laboratory assessments provides a unique opportunity to document the longitudinal evolution of antiphospholipid antibody profiles.

Conclusion

This case demonstrates a six-year longitudinal evolution of the aPL profile in a woman with SLE, characterized by the disappearance of high-titer anti-β2GPI IgM antibodies and the subsequent emergence of LAC and aCL antibodies. These changes occurred despite stable treatment and in the absence of thrombotic events. The findings support the concept that antiphospholipid autoimmunity may evolve over time and emphasize the value of long-term monitoring of aPL profiles in SLE patients, particularly during the reproductive years. Further research is warranted to identify the factors that drive the longitudinal evolution of antiphospholipid antibody profiles and to clarify their clinical significance.

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Conflict of Interest

The authors declare that they have no competing interests.

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