Editorial

International Journal of Clinical Rheumatology

Antiphospholipid Syndrome (APS): Pathogenesis, Clinical Features, and Management

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Received: 02-Feb-2025, Manuscript No. fmijcr-26-185403; **Editor assigned:** 04- Feb-2025, Pre-QC fmijcr-26-185403 (PQ); **Reviewed:** 17-Feb-2025, QC No. fmijcr-26-185403; **Revised:** 22-Feb-2025, Manuscript No. fmijcr-26-185403 (R); **Published:** 27-Feb-2025, **DOI:** 10.37532/1758-4272.2025.20(2).414-414

Introduction

Antiphospholipid syndrome (APS) is a systemic autoimmune disorder characterized by recurrent arterial or venous thrombosis and pregnancy-related complications in the presence of persistent antiphospholipid antibodies (aPL). First recognized in the 1980s, APS has become an important cause of acquired thrombophilia. It may occur as a primary condition or in association with other autoimmune diseases, particularly systemic lupus erythematosus. Early recognition is essential to prevent life-threatening complications and long-term morbidity.

Pathogenesis

APS is mediated by autoantibodies directed against phospholipid-binding proteins, most notably beta-2 glycoprotein I. The principal laboratory markers include lupus anticoagulant, anticardiolipin antibodies, and anti–beta-2 glycoprotein I antibodies. Rather than causing bleeding, these antibodies promote a hypercoagulable state.

Clinical Manifestations

The hallmark feature of APS is thrombosis. Venous thrombosis, especially deep vein thrombosis of the lower limbs, is most common, but arterial events such as stroke and myocardial infarction may occur. Recurrent pregnancy loss, preeclampsia, intrauterine growth restriction, and premature birth are characteristic obstetric complications.

Other manifestations include thrombocytopenia, livedo reticularis, heart valve abnormalities, and neurological symptoms. A rare but severe form, catastrophic APS, involves rapid multiorgan thrombosis and carries high mortality if not promptly treated.

Management and Treatment

Long-term anticoagulation is the cornerstone of APS management. Vitamin K antagonists such as warfarin are commonly used to prevent recurrent thrombosis. In selected patients, direct oral anticoagulants may be considered, though their use remains controversial in high-risk APS.

For obstetric APS, combination therapy with low-dose aspirin and low-molecular-weight heparin significantly improves pregnancy outcomes. In catastrophic APS, aggressive treatment including anticoagulation, corticosteroids, plasma exchange, and intravenous immunoglobulin is required.

Conclusion

Antiphospholipid syndrome is a complex autoimmune disorder marked by recurrent thrombosis and pregnancy morbidity driven by pathogenic antiphospholipid antibodies. Through mechanisms involving endothelial activation and complement-mediated injury, APS creates a persistent prothrombotic state. Early diagnosis and appropriate long-term anticoagulation substantially reduce morbidity and mortality. Continued research into immunological pathways and targeted therapies holds promise for improving outcomes and refining management strategies in this potentially life-threatening condition.