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CLINICAL FEATURES OF THE COURSE OF PREGNANCY IN WOMEN WITH ANTIPHOSPHOLIPID SYNDROME (APS)

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Abstract

Antiphospholipid syndrome (APS) is one of the most significant forms of acquired thrombophilia and is associated with severe obstetric complications. It is estimated that APS accounts for approximately 10–20% of recurrent pregnancy losses. This study aims to analyze the clinical course of pregnancy in women with APS, with particular emphasis on pathophysiological mechanisms, diagnostic criteria, and evidence-based management strategies. APS-related pregnancy is characterized by impaired placentation, microthrombosis, and inflammatory activation, leading to adverse maternal and fetal outcomes. Early diagnosis and combined anticoagulant therapy significantly improve pregnancy outcomes, with live birth rates reaching up to 80–85%.

Keywords

Antiphospholipid syndrome, pregnancy, thrombosis, placental insufficiency, preeclampsia, enoxaparin, aspirin

1. Introduction

Antiphospholipid syndrome (APS) is an autoimmune disorder characterized by the persistent presence of antiphospholipid antibodies directed against phospholipids and phospholipid-binding proteins, particularly β2-glycoprotein I.

Epidemiological data indicate that APS occurs in approximately 1–5% of the general population, while its prevalence increases to 15–20% among women with recurrent pregnancy loss and 10–15% in cases of severe preeclampsia.

Obstetric APS is strongly associated with the following clinical manifestations:

Three or more consecutive early miscarriages

One or more fetal losses after 10 weeks of gestation

Severe preeclampsia or placental insufficiency before 34 weeks

2. Materials and Methods

This study is based on a comprehensive review and analysis of current scientific literature on APS and pregnancy outcomes. Clinical data were interpreted in accordance with established international diagnostic criteria (Sydney criteria, 2006) and modern evidence-based treatment recommendations.

3. Results and Discussion

3.1. Pathophysiological Mechanisms

The adverse outcomes of pregnancy in APS are mediated by multiple interrelated mechanisms:

Thrombotic mechanism:

- Microthrombi formation in placental vessels leads to a reduction in uteroplacental blood flow by up to 30–40%, resulting in fetal hypoxia.

Impaired trophoblast invasion:

Antiphospholipid antibodies inhibit trophoblast proliferation and disrupt spiral artery remodeling, leading to defective placentation.

Inflammatory and complement activation:

Activation of the complement system (C3, C5) induces placental inflammation, necrosis, and tissue damage. Studies suggest that complement inhibition may reduce adverse outcomes.

3.2. Clinical Features

Early pregnancy complications (≤10 weeks):

- Recurrent miscarriage (65–80%)
- Anembryonic pregnancy
- Biochemical pregnancy

Late pregnancy complications (>10 weeks):

- Preeclampsia (30–50%), often early-onset and severe
- Fetal growth restriction (20–30%)
- Placental abruption (5–10%)
- Antenatal fetal death (10–15%), чаще in the third trimester

3.3. Diagnostic Criteria

Diagnosis of APS is based on the Sydney criteria (2006), requiring at least one clinical and one laboratory criterion.

Laboratory criteria:

- Lupus anticoagulant (LA)
- Anticardiolipin antibodies (aCL) IgG/IgM (>40 GPL/MPL or >99th percentile)
- Anti-β2-glycoprotein I antibodies

These tests must be positive on two occasions at least 12 weeks apart.

Clinical criteria:

- Arterial or venous thrombosis
- Obstetric complications
- The presence of "triple positivity" (LA + aCL + anti-β2GP1) is associated with the highest risk of adverse outcomes.

3.4. Management and Treatment

The standard of care for pregnant women with APS includes combined therapy:

- Low molecular weight heparin (LMWH) (e.g., enoxaparin)
- Low-dose aspirin (75–150 mg/day)

This combination significantly improves live birth rates (up to 80–85%).

In refractory cases, additional therapies may include:

- Hydroxychloroquine
- Glucocorticoids
- Intravenous immunoglobulin (IVIG)

4. Conclusion

Pregnancy in women with APS is considered high-risk and requires careful monitoring. The key pathogenic mechanisms include thrombosis, inflammation, and impaired placentation. Early diagnosis and individualized management, particularly combined anticoagulant therapy, significantly improve maternal and fetal outcomes.

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