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Preeclampsia: How Collaborative Work between Laboratories and Obstetrician-Gynecologists Helps Predict, Diagnose, and Treat

N.N. Ghukasyan

"Erebouni" Medical Center,
Titogradyan St. 14, 0087, Yerevan

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Introduction

Preeclampsia remains a leading cause of maternal and perinatal morbidity and mortality worldwide. This hypertensive disorder, often presented after 20 weeks of gestation, is characterized by proteinuria and systemic organ involvement, including liver, kidney, and hematological complications. While the exact etiology of preeclampsia remains elusive, its pathophysiology is thought to involve abnormal placentation, immune dysfunction, and endothelial damage [3, 7].

The coexistence of autoimmune conditions like systemic lupus erythematosus (SLE), thrombophilia disorders, and hereditary autoinflammatory syndromes such as familial Mediterranean fever (FMF) presents unique challenges in pregnancy [1, 5]. These conditions independently predispose individuals to vascular complications, and their intersection with pregnancy increases the likelihood of adverse outcomes. This article explores the complex interplay of these disorders through case studies, emphasizing the need for collaborative work between laboratories and clinicians to predict, diagnose, and treat preeclampsia effectively [8].

Case 1: Complex Obstetric History with Isolated Low Protein S Levels

A 28-year-old woman, currently at 25 weeks of gestation (G3P1), presented for antenatal care. Her obstetric history revealed significant complications.

Obstetric History: 2016 (First pregnancy): Miscarriage at 28 weeks due to preeclampsia; 2017 (Second pregnancy): Premature birth at 26 weeks with elevated liver enzymes, hypertension, and proteinuria, consistent with severe preeclampsia.

Current Pregnancy:

- Comorbidities: Diagnosed with hypothyroidism during her first pregnancy, currently on levothyroxine.
- Past Medical History: Cholecystectomy without complications.

- Allergies: Reports an allergy to antibiotics, though specific agents are unclear.

Laboratory Findings:

- Coagulation Panel: Normal factor V and II mutation studies, no abnormalities in antithrombin III or protein C levels. However, isolated protein S deficiency (20%) was detected.
- Management: Initiated low-molecular-weight heparin (40 mg/day) after aspirin cardio (100 mg) was discontinued due to concerns over thrombosis risk.

Analysis: Protein S deficiency is a known risk factor for thromboembolic events and preeclampsia. Its isolated nature here suggests a potential hereditary component. Regular monitoring of maternal coagulation status, fetal growth, and placental function is crucial. The patient's management plan includes biweekly Doppler ultrasound assessments to monitor uteroplacental perfusion [6, 9].

Case 2: SLE-Associated Pregnancy Complications

A 31-year-old woman with a 17-year history of systemic lupus erythematosus (SLE) presented for antenatal care at 18 weeks of gestation.

Medical History: Diagnosed with SLE at the age of 14; treated with pulse therapy (methylprednisolone) until the age of 18. Treatment was discontinued after marriage, which exacerbated her disease activity. Since 2023, she has been on continuous Plaquenil (hydroxychloroquine) therapy.

Obstetric History: 19 years old (First pregnancy): Stillbirth at 25 weeks due to preeclampsia. 21 years old (Second pregnancy): Preterm delivery at 26-27 weeks following severe preeclampsia. Complications included pyelonephritis requiring prolonged hospitalization and antibiotics. Delivered a premature infant via cesarean section at 34 weeks. 30 years old (Third pregnancy): Non-developing fetus detected at 10 weeks.

Current Pregnancy:

- Laboratory findings showed normal complement levels (C3, C4), stable anti-dsDNA titers, and normal platelet counts.
- Renal function is stable with no evidence of proteinuria.
- Currently, there are no clinical or laboratory signs of active lupus.

Analysis: SLE significantly increases the risk of adverse pregnancy outcomes, including preeclampsia, intrauterine growth restriction (IUGR), and fetal loss. The continuation of Plaquenil has likely contributed to disease stabilization. Low-dose aspirin has been initiated to mitigate the risk of preeclampsia, and close surveillance includes fetal biometry and maternal blood pressure monitoring [3, 5].

Case 3: Familial Mediterranean Fever and HELLP Syndrome

A 20-year-old woman was admitted at 27 weeks of gestation with rapidly progressing hypertension, proteinuria, and transaminase elevation, consistent with HELLP syndrome.

Medical History: No prior pregnancies. Diagnosis: Previously undiagnosed familial Mediterranean fever (FMF) confirmed post-admission through genetic testing (MEFV mutation). Family History: Positive for FMF, with relatives experiencing recurrent febrile episodes and abdominal pain.

Clinical Course:

- The patient developed severe thrombocytopenia (platelet count: 45,000/μL), ALT/AST levels >500 U/L, and significant hemolysis.
- Emergency cesarean section was performed due to non-reassuring fetal status, resulting in the delivery of a 900 g infant requiring neonatal intensive care.

Management:

- Initiation of colchicine postoperatively to manage FMF symptoms and reduce systemic inflammation.
- Postpartum recovery was complicated by persistent hypertension requiring antihypertensive therapy and close monitoring for end-organ damage.

Analysis: FMF and associated chronic inflammation may predispose to endothelial dysfunction, a key contributor to preeclampsia and HELLP syndrome. Early diagnosis of FMF and preemptive management with colchicine could have potentially mitigated risks. This case highlights the need for genetic counseling and family history evaluation during antenatal care [1, 2].

Discussion. These cases collectively underscore the intricate interplay between autoimmune, thrombotic, and genetic conditions in predisposing to preeclampsia. Analysis of each case reveals:

1. Case 1: The significance of isolated protein S deficiency as an independent risk factor for preeclampsia, necessitating individualized thromboprophylaxis.
2. Case 2: The importance of disease control in SLE through maintenance therapy to improve pregnancy outcomes.
3. Case 3: The critical role of genetic testing in identifying FMF, which may otherwise remain undiagnosed and complicate pregnancy outcomes.

Emerging biomarkers and personalized therapeutic strategies, such as the use of low-dose aspirin, hydroxychloroquine, and colchicine, demonstrate promise in mitigating risks in high-risk pregnancies. Further research is essential to elucidate the precise mechanisms linking these conditions to pre-eclampsia [2, 7].

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Преэклампсия: как совместная работа лабораторий и акушеров-гинекологов помогает предсказывать, диагностировать и лечить

Н.Н. Гукасян

Преэклампсия, многосистемное нарушение во время беременности, представляет значительный риск для здоровья матери и плода. В данной статье представлены три клинических случая, подчеркивающие взаимосвязь системной красной волчанки, тромбофилии и семейной средиземноморской лихорадки с развитием преэклампсии. Эти случаи акцентируют важность междисциплинарного сотрудничества и персонализированных стратегий ухода при ведении беременностей высокого риска.

Պրեէկլամպսիա. ինչպես է լաբորատորիաների և մանկաբարձ-գինեկոլոգների համագործակցությունը օգնում կանխատեսել, ախտորոշել և բուժել

Ն.Ն. Ղուկասյան

Պրեէկլամպսիան հղիության բազմահամակարգ խանգարում է և էական վտանգ է ներկայացնում մոր և պտղի առողջության համար: Ներկայացվում են երեք կլինիկական դեպքեր, որոնք ընդգծում են համակարգային կարմիր գայլախտի, թրոմբոֆիլիայի և ընտանեկան միջերկրածովյան տենդի կապը պրեէկլամպսիայի զարգացման հետ: Այս դեպքերով կարևորվում է միջգիտակարգային համագործակցության և անհատականացված խնամքի ռազմավարությունների անհրաժեշտությունը բարձր ռիսկի հղիությունների կառավարման մեջ:

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