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LATENT PREDICTORS OF SEVERE PREECLAMPSIA AS A FORM OF SECONDARY THROMBOTIC MICROANGIOPATHY IN PREGNANT WOMEN.

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Abstract:

Objective: To analyze the clinical-demographic characteristics, latent risk factors, and functional (laboratory and instrumental) predictors of severe preeclampsia (sPE) as a form of secondary thrombotic microangiopathy (TMA).

Methods: The study was conducted on a cohort of 327 patients with sPE, representing 87.4% of all TMA cases in the study population, and a control group of 50 women with physiologically normal pregnancies. The research involved a detailed analysis of medical history, clinical and laboratory parameters, and obstetric outcomes. Laboratory assessment included complete blood count, biochemical profile, and hemostasis parameters. Instrumental evaluation was performed using Doppler ultrasound of the uteroplacental and fetal circulation.

Results: It was established that sPE is characterized by a significantly earlier gestational age at delivery, lower birth weight and length of newborns, elevated liver enzymes, hemolytic anemia, and thrombocytopenia. Anamnestic analysis revealed a high prevalence of pre-existing risk factors, including chronic hypertension and a history of adverse obstetric events. Laboratory and Doppler parameters demonstrated significant deviations, indicating systemic endothelial damage, placental hypoperfusion, and fetal distress.

Conclusion: The identified clinical, anamnestic, and functional associations underscore the necessity for intensive dynamic monitoring in high-risk groups to prevent life-threatening complications. The combination of these latent predictors and indicators allows for a more comprehensive assessment of the risk of adverse outcomes.

Keywords: Preeclampsia, thrombotic microangiopathy, pregnancy, obstetric complications, placental insufficiency, endothelial dysfunction, Doppler ultrasound, latent predictors

INTRODUCTION. Preeclampsia (PE) is a major cause of maternal and perinatal mortality globally, representing a multisystemic disorder that develops after 20 weeks of gestation. It complicates up to 5–8% of all pregnancies and remains a leading cause of preterm birth, fetal growth restriction, and multi-organ dysfunction[] . In recent years, there has been a growing interest in studying PE not only as an isolated gestational complication but also as a manifestation of systemic thrombotic microangiopathies (TMAs). Severe preeclampsia is considered a key trigger for secondary TMAs (sTMAs) in pregnant women due to its close pathogenetic link with endothelial dysfunction, inflammation, and microthrombosis[2,3].

The core clinical challenge lies in the fact that a similar clinical presentation of severe PE can conceal fundamentally different pathogenetic processes that determine the subsequent disease trajectory. While one patient's condition may stabilize, another's can rapidly transform into a fulminant form of TMA such as HELLP syndrome. Therefore, the primary challenge for the clinician is not only the initial diagnosis of the manifest

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pathology but also the assessment of the latent (hidden) risk of its malignant transformation and the determination of the individual vector of progression[1,4,6].

In the context of this study, "latent predictors" are understood as hidden, clinically non-obvious factors (anamnestic, as well as specific patterns of laboratory and instrumental changes) that, while not being direct diagnostic criteria, determine the hidden potential for the disease to worsen. The complexity of diagnosis lies in the fact that the manifestations of these latent processes are often masked by the symptoms of the primary trigger disease, significantly hindering their timely verification. This delay in recognizing the latent potential can lead to the rapid, sometimes catastrophic, development of multi-organ dysfunction, critically worsening the prognosis for both mother and child[5,7]. This study aims to fill this scientific lacuna through an in-depth analysis of the profile of patients with severe PE and the identification of its specific latent predictors and indicators.

MATERIALS AND METHODS. This was a prospective comparative study that included 327 pregnant women with severe PE and a control group of 50 women with uncomplicated pregnancies. Inclusion criteria for the main group were based on international standards for the diagnosis of severe PE. All patients provided informed consent. The study protocol was approved by the Local Ethics Committee. A comprehensive analysis was performed, including the collection of clinical-anamnestic data, general and biochemical laboratory tests, hemostasis parameters, and Doppler ultrasound of uteroplacental and fetal blood flow. Statistical data processing was performed using the Mann–Whitney U-test for quantitative variables and the χ^2 test (with Fisher's exact test where appropriate) for qualitative variables. Differences were considered statistically significant at $p<0.05$.

RESULTS. Clinical-Demographic Characteristics. The demographic analysis confirmed that the study groups were comparable in age (median 27.0 years in both groups; $p=0.224$), which excludes age as a potential confounding factor. However, statistically significant differences were observed for all major obstetric outcomes. The median gestational age at delivery in the sPE group was 36.0 [35.00-38.00] weeks, which was significantly lower than in the control group (median 39.0 [38.25-40.00] weeks; $p<0.001$). This was accompanied by a significant reduction in neonatal anthropometric parameters: the median birth weight in the sPE group was 2550.0 g (control: 3410.0 g; $p<0.001$), and the median length was 48.0 cm (control: 53.0 cm; $p<0.001$).

Anamnestic Profile and Latent Predictors. An in-depth analysis of anamnestic data revealed a substantial background burden in patients with sPE, which plays the role of latent predictors. A history of chronic arterial hypertension was found in 88.4% of sPE patients versus 28.0% in the control group ($p<0.001$). A family history of thrombosis was identified in 38.5% of sPE patients, while being absent in the control group ($p<0.001$). Similarly, recurrent pregnancy loss (61.8% vs. 0%; $p<0.001$) and preeclampsia in previous pregnancies (50.8% vs. 0%; $p<0.001$) were powerful latent predictors. A history of anemia (62.1% vs. 16.0%; $p<0.001$), urinary tract infections (48.3% vs. 24.0%; $p=0.001$), and infertility (31.5% vs. 8.0%; $p<0.001$) further confirmed the profile of high latent vulnerability. These data convincingly demonstrate that severe PE develops in patients with a pre-existing compromised somatic and obstetric status.

Laboratory and Instrumental Profile: Latent Indicators of Systemic Damage. The clinical manifestation of sPE was accompanied by characteristic changes in laboratory and instrumental parameters, which act as latent indicators of disease progression. In the complete blood count, the platelet count was significantly lower in the sPE group (median 181.0 x10^9/L) compared to the control group (median 233.5 x10^9/L; $p<0.001$), indicating increased platelet consumption. In the biochemical analysis, the level of Lactate Dehydrogenase (LDH), an integral marker of hemolysis and cytolysis, was significantly elevated in the sPE group (median 280.0 U/L) compared to the control group (median 212.0 U/L; $p<0.001$). Homocysteine, a key marker of endothelial dysfunction, was also significantly higher (median 16.0 µmol/L vs. 9.49 µmol/L; $p<0.001$). A significant increase in creatinine ($p<0.001$) and protein in a random urine sample ($p<0.001$) indicated renal involvement. In the coagulation profile, the D-dimer level was significantly different between the groups ($p<0.001$).

Instrumental Doppler ultrasound revealed severe uteroplacental blood flow disturbances, which are a major latent functional substrate of placental dysfunction. The Pulsatility Index (PI) in the uterine arteries was significantly higher in the sPE group (median 1.43) compared to the control group (median 0.68; $p<0.001$), indicating impaired trophoblast invasion and increased resistance. Concurrently, the PI in the fetal middle cerebral artery was significantly lower in the sPE group (median 0.98) compared to the

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control group (median 1.63; p<0.001), which is a sign of compensatory fetal circulatory centralization in response to chronic hypoxia (the "brain-sparing" phenomenon).

DISCUSSION AND CONCLUSION. The data obtained in this study confirm the multifactorial nature of severe preeclampsia as a significant manifestation of secondary TMA in pregnant women. The combination of clinical-demographic, anamnestic, and functional parameters identified in the study group indicates that the development of sPE is conditioned by the interaction of pre-existing vascular pathology, immune disorders, and obstetric history. The anamnestic factors identified, such as chronic hypertension and recurrent pregnancy loss, serve as powerful latent predictors, highlighting a population of women with high baseline vulnerability.

The laboratory and instrumental profiles revealed a pattern of systemic endothelial dysfunction, microvascular damage, and impaired placental perfusion. The observed changes in platelets, LDH, homocysteine, and Doppler indices act as latent indicators, allowing for a quantitative assessment of the severity of the pathological process, even before the development of life-threatening complications. As demonstrated by multiple meta-analyses, including reports from the American College of Obstetricians and Gynecologists (ACOG, 2023), early identification of these risk factors and indicators is crucial for risk stratification and timely intervention.

In conclusion, a comprehensive assessment, integrating a detailed analysis of the patient's history with dynamic monitoring of laboratory and instrumental markers, is essential for the early identification of patients at high risk of developing a complicated course of sPE. This approach allows for a shift from a reactive to a proactive and personalized management strategy, which is critical for preventing severe maternal and perinatal morbidity and mortality.

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