

IMBALANCE OF PLATELET PRODUCTION AND DESTRUCTION IN PREECLAMPSIA

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Background and aims. Preeclampsia (PE) is characterized by endothelial dysfunction and platelet involvement, with increasing evidence pointing to altered platelet turnover. Enhanced platelet consumption may trigger compensatory production, leading to measurable changes in circulating biomarkers. Thrombopoietin (TPO) reflects platelet production, while glycocalicin (GC) indicates platelet destruction. Thiazole orange (TO) identifies immature, reticulated platelets and provides insight into bone marrow response and platelet activity. This study aimed to investigate longitudinal changes in TPO, GC, and TO in high-risk pregnancies treated with low-dose aspirin (ASA), to better characterize platelet turnover and its relationship with PE development.

Methods. This observational study included 25 high-risk pregnant women on ASA and 12 controls with uncomplicated pregnancies. Blood samples were collected at baseline (T0), 21 gestational weeks (T4), and 27 gestational weeks (T5). Platelet-related biomarkers TPO and GC, indicating platelet production and destruction, respectively; and TO were measured longitudinally.

Results. Late onset PE occurred in 6/25 (24%) high-risk women and in none of the controls, while there was no case of early onset disease. Within the high-risk group, TPO and GC levels remained relatively stable in women without events, whereas in women who developed PE, TPO levels increased and were linked to disease onset (overall p=0.036), with more notable differences at T4–T5 (weeks 21 and 27); conversely, GC levels were higher at baseline (p=0.048) and decreased over time in women who developed PE. Using the latest PE risk algorithm, clinical stratification showed high sensitivity and NPV (both 1.00) but low specificity (0.39) and PPV (0.24). Adding baseline glycocalicin improved prediction, with higher thresholds (≥0.80) markedly increasing specificity (0.95) and PPV (0.80), supporting a two-step algorithm–biomarker approach.

Conclusions. Preeclampsia is characterized by a dynamic imbalance in platelet turnover, with early alterations in glycocalicin and subsequent increases in TPO, suggesting temporally distinct changes in platelet destruction and production. While the current PE risk algorithm shows excellent rule-out performance, its limited specificity may be improved by incorporating platelet biomarkers. In particular, baseline glycocalicin enhances risk stratification at higher thresholds, supporting a combined algorithm–biomarker approach for identifying high-risk women despite aspirin prophylaxis.

