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MECHANISMS OF THROMBOSIS AND BLEEDING IN CANCER

DYSFUNCTION OF THE ADAMTS-13 / VON WILLEBRAND FACTOR AXIS, MEDIATED BY NETOSIS, AS A DRIVER OF PROTHROMBOTIC STATE AND UNFAVORABLE PROGNOSIS IN ONCOGYNECOLOGICAL PATIENTS

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Introduction. An imbalance between von Willebrand factor (VWF) and its primary protease, ADAMTS-13, plays a crucial role in the pathogenesis of microthrombosis and endotheliopathy. The aim of this work was to study the functioning of the ADAMTS-13/VWF axis and its interaction with thromboinflammatory reactions (NETosis) in oncogynecological patients undergoing antitumor therapy.

Methods. The study included 262 patients (cancer of the endometrium, ovaries, cervix, and breast). The concentration of VWF, the antigen concentration and activity of ADAMTS-13, its inhibitor levels, NETosis markers (CitH3, MPO), and hemostasis activation markers were determined dynamically (before and after surgical treatment/chemotherapy).

Results. Oncogynecological patients exhibited a significant increase in VWF concentration and a decrease in ADAMTS-13 concentration/activity ($p<0.0001$). The VWF/ADAMTS-13 ratio was significantly elevated in all groups, with a maximum after chemotherapy (6.42 vs. 0.94 in the control). A strong negative correlation was found between NETosis markers (CitH3, MPO) and ADAMTS-13 parameters in patients with ovarian cancer (ρ up to -0.69, $p<0.01$). The increase in VWF and the VWF/ADAMTS-13 ratio after chemotherapy strongly correlated with coagulation activation (TAT complexes, $p<0.05$).

Conclusions. An acquired deficiency of ADAMTS-13, induced in part by active NETosis, combined with increased VWF release leads to a critical disruption of microthrombosis regulation. The assessment of the ADAMTS-13/VWF axis, together with NETosis markers, is a promising tool for stratifying the risk of thrombosis and disease prognosis, as well as a target for pathogenetically based therapy.