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Kallikrein and plasmin in ascitic fluid require exogenous inhibitors in ovarian cancer and tuberculosis of uterine appendages

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Objective.

Ascites is the dominant syndrome in stage III-IV ovarian cancer (OC, cystadenocarcinoma), and the microvasculature is directly exposed to bradykinin. The kallikrein-kinin system (KKS), plasmin (P), and α2macroglobulin (α2M) are involved in the vascular status control. Our aim was to study parameters of KKS in the ascitic fluid (AF) in OC and tuberculosis of uterine appendages (TUA).

Material and methods.

AF was studied in OC patients (n=26, $T_{3\text{-}4}N_0M_0$), comparison group: AF of TUA patients (n=13); age 57.5±2.6, menopause. AF was considered an exudate due to its high protein content in OC and TUA. Levels of kallikrein (K), prekallikrein (PK), carboxypeptidase N (CPN), P, plasminogen (PG) and α2M were determined by spectrophotometry and ELISA. An exudate comes from vessels damaged by inflammation, so AF was compared with plasma of 32 healthy donors (N).

Results.

PK/K balance in N was 7.4±0.5; in AF: in TUA 1.6±0.1, in OC 1.1±0.1. The ratio of kinin formation and degradation rates (K/CPN) in TUA was 195±10.3; in OC 129±6.8, vs. 60.4±3.8 in N. In low PK/K, KKS in AF cannot perform its regulatory and adaptive protective functions. PG/P ratio was in TUA 2.2±0.1; in OC 0.6±0.1, vs. 2.3±0.2 in N. High P levels are dangerous for all structures, especially since the activity of α2M in OC was similar to N, and in TUA it decreased by 1.4 times, which was not enough in activated proteolysis.

K/ α2M and P/α2M ratios in TUA were 52.1±2.9 and 1.3±0.1, vs. 11.3±0.8 and 0.2±0.01 in N.

Conclusions.

Period after the AF removal requires treatment with exogenous protease inhibitors, since the pathological functions of KKS and P in the continuing exudate will ensure the spread of the pathological process.

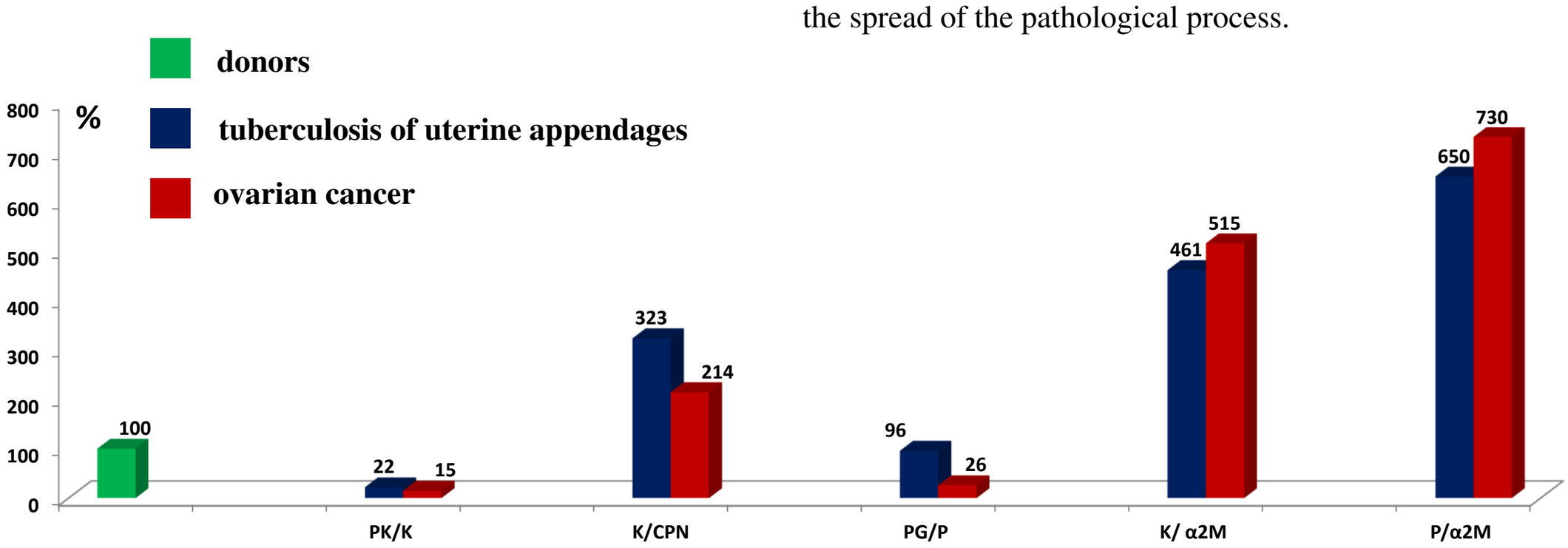

Figure 1. - Parameters of kallikrein-kinin system in the ascitic fluid of patients with ovarian cancer and tuberculosis of uterine appendages