

ACE, KALLIKREIN AND ASPARTIC PEPTIDASE ACTIVITIES IN PLASMA AND URINE IN PRE-ECLAMPSIA

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Pre-eclampsia is a human pregnancy specific disorder characterized by increased blood pressure, oedema and proteinuria after twenty weeks of gestation in previously normotensive women. Its exact etiology is unknown, but several studies on endothelial cell, placenta ischemia, vasospasm and changes of pressor substances have been done aimed to clarify the physiopathological mechanism of the disease. Enzymatic systems involved in vasoactive peptides releasing as renin-angiotensin system (RAS) and kallikrein-kinin system (KKS) have been evaluated. An aspartic peptidase expressed in human decidua, similar to cathepsin D, has been related to the uterus-placental development during normal placentation. As these systems have common components, a reciprocal regulation between RAS and kininogenase-kinin systems should be possible. This study evaluated the activities of angiotensin converting enzyme (ACE), kallikrein (KK) and aspartic peptidase (AP) in plasma and urine (24 hours samples) of normal pregnant (control group, 14) and pre-eclamptic (27) women after 20 weeks gestation. Enzymatic activities were determined by a continuous fluorescent assay using peptide sequences susceptible to hydrolysis by ACE (Abz-YRK(Dnp)-P-OH), kallikrein (Abz-MISLMKRPQ-Eddnp, Abz-RPPGFSPFRSSRQ-Eddnp) and aspartic peptidase (Abz-KPIEFFRLQ-Eddnp) in adequate pH. Statistical analyses showed no significant difference in plasma enzymatic activities between the control and pre-eclamptic groups. Meanwhile, urinary KK and ACE activities were significantly lower in pre-eclamptic patients than control group ($p < 0.05$). AP urinary activity showed no significant difference. There was no correlation between urinary and plasma enzymatic activities as in normal pregnant as in pre-eclamptic women. These results suggest that plasma and renal regulation of kinin releasing and hydrolysis are independent. Low urinary KK activity in pre-eclamptic condition could contribute to increase blood pressure. If the decrease of urinary ACE activity is a consequence of higher blood pressure levels in pre-eclamptic group remains to be determined. Financial support: CAPES, CNPq, UFTM, UNIFESP.