

IMPAIRED ENDOTHELIAL FUNCTION IN ISOLATED HUMAN UREMIC RESISTANCE ARTERIES

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Background: Patients with chronic renal failure (CRF) face a markedly increased risk of cardiovascular death. CRF is frequently complicated by hypertension and changes in both the heart (left ventricular hypertrophy) and the vasculature (endothelial dysfunction and accelerated atherosclerosis). The mechanisms underlying changes in vascular function and specifically endothelial dysfunction are unclear. This present study therefore examined subcutaneous resistance artery function in vitro, comparing adult uremic patients and controls using wire myography.

Materials and methods: Subcutaneous fat biopsies were obtained from 12 patients with CRF (median serum creatinine 735 micromol/L) at the time of renal transplantation or peritoneal dialysis catheter insertion, and from eight controls without renal disease at the time of abdominal surgery. Resistance arteries were mounted on a wire myograph. Their contractile ability was tested with high potassium depolarization, and endothelial integrity was tested by relaxation to acetylcholine. Cumulative concentration-response curves were then constructed for norepinephrine, endothelin-1, acetylcholine, and sodium nitroprusside (SNP).

Results: Following precontraction with norepinephrine,

vessels from uremic patients vasodilated less well to acetylcholine compared with vessels from controls [maximum % relaxation 77% (range 41, 97) vs. 98% (78, 100), $P < 0.001$]. The vasodilation to SNP was similar [95% (63, 100) vs. 94% (71, 100), $P = 0.751$]. There was a trend toward increased maximum pressure achieved with both norepinephrine and endothelin-1 in vessels from uremic patients, and the contractions to both of these agents were more prolonged in the uremic vessels.

Conclusions: The pattern of normal vasodilation to SNP but reduced vasodilation to acetylcholine is consistent with endothelial dysfunction due to impaired nitric oxide (NO) production in uremic vessels. Similar results have been demonstrated in vivo in uremia, one suggested mechanism being accumulation of endogenous inhibitors of NO synthase such as asymmetric dimethylarginine. This in vitro study suggests that a short-lived circulating factor is not entirely responsible and that there may be an inherent abnormality in endothelial function in uremia, although the exact pathophysiology remains unclear. Endothelial dysfunction may predispose the patient to accelerated atherosclerosis and may be involved in the pathogenesis of hypertension in end-stage renal failure.

THE ABSENCE OF INCREASED ATHEROGENICITY IN PATIENTS WITH CENTRAL FORM OF BECHTEREW'S DISEASE WITH A MINIMUM DEGREE OF DISEASE ACTIVITY

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Actuality: Cardiovascular events are the most unfavorable prognostic complications in patients with rheumatic diseases. The course of these diseases is accompanied by an increased risk of cardiovascular death. For example, the risk of myocardial infarction in patients with rheumatoid arthritis is 60% higher than in the general population. Patients with systemic lupus erythematosus have a 50 times higher risk myocardial infarction than the healthy population. Bechterew's disease (BD) belongs to a group of rheumatic diseases. In accordance from the views of some researchers the level of cardiovascular mortality in patients with BD increased 1.5-2 times compared with the general population rate. However, other researchers have noted no increase in cardiovascular risk in patients with BD. One of cardiovascular risk assessment methods is the study of the levels of specific biochemical markers. Elevated serum levels of uric acid, total cholesterol, low density lipoprotein cholesterol (LDL- cholesterol) and decreased serum level of high density lipoprotein cholesterol (HDL- cholesterol) are proven biochemical markers of cardiovascular risk. Changing one of these markers is an independent risk factor for cardiovascular events, but the combined involvement of multiple markers increases this risk many times.

Objective: To determine the content of biochemical markers of cardiovascular risk such as: uric acid, total cholesterol, LDL- cholesterol and HDL- cholesterol in the blood serum of patients with BD with minimal activity.

Materials and methods: 20 patients (all of them were male), aged 41-69 years, with a central form of BD with 1 degree activity formed a main group. 20 healthy men of similar age formed the control group. Uric acid, total cholesterol, LDL-cholesterol and HDL-cholesterol was detected in the serum of blood in all examined persons. Persons with clinical manifestations of atherosclerosis (coronary heart disease, cerebral atherosclerosis, atherosclerosis of peripheral vessels of the lower extremities), gout, liver diseases and renal insufficiency were excluded from the study. The obtained results were processed using parametric statistical methods. The critical level of significance at check of statistical hypotheses was 0.05.

Results: The level of total cholesterol in serum of patients of the main group was $4,2 \pm 0,14$ mmol / l, LDL- cholesterol - $2,6 \pm 0,11$ mmol / l, HDL- cholesterol - $0,8 \pm 0,06$ mmol / l, uric acid - $402,5 \pm 12,23$ μ mol / l. Similar rates of the control group were respectively $4,5 \pm 0,14$ mmol / l, $2,8 \pm 0,13$ mmol / l, $1,0 \pm 0,07$ mmol / l and $378,2 \pm 13,17$ μ mol / l.

Statistical analysis has not found significant differences ($p > 0,05$) in the serum concentrations of total cholesterol, LDL-cholesterol and uric acid between groups of persons surveyed. At the same time, decrease in the level of HDL-cholesterol in patients of the main group compared to the control was more pronounced than the variation of other studied parameters. This decrease was also unreliable, but it was as though trend ($t = 1,881$, $p = 0,073$).

Conclusions: Reliable changes in the content of

biochemical markers of cardiovascular risk (uric acid, total cholesterol, HDL-cholesterol and LDL-cholesterol) in blood serum of patients with central form of BD with 1 degree of activity were not detected. This result is evidence in favor of inflammatory genesis of increase the cardiovascular risk in patients with BD. However, in the examined patients the activity of inflammation was very low. Another explanation for this phenomenon may be the absence of increase cardiovascular risk in patients with BD in general.

SYSTEMIC MARKERS OF CARDIOVASCULAR RISK IN PATIENTS WITH COMORBIDITY OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND CHRONIC PANCREATITIS

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The aim of the current study is to determine C-reactive protein and homocysteine levels, as systemic markers of cardiovascular risk in patients with comorbidity of chronic obstructive pulmonary disease (COPD) and chronic pancreatitis.

Materials and methods: 148 COPD patients have been examined: 76 COPD patients in combination with chronic pancreatitis have been regarded as a main group, 72 patients with an isolated course of COPD made up a compared group. Standard values were obtained while examining 20 almost healthy patients of the same age and gender. The latter made up a control group. The homocysteine and C-reactive protein has been performed by ELISA. Statistical data has been performed on workstation by means of software "Microsoft Excel" and "STATISTICA 6.0".

Results: The study showed that COPD exacerbation was accompanied with an increase of homocysteine blood level both in groups with isolated COPD and in groups with comorbidity in comparison with almost healthy patients. It has been found out that patients with comorbid pathology are characterized by the significant increase of homocysteine blood level up to $17,1 \pm 1,5 \mu\text{mol/L}$, in comparison with control

group – $9,6 \pm 0,5 \mu\text{mol/L}$ ($p < 0,05$). At the same time, patients with isolated COPD homocysteine level increase to $13,8 \pm 1,2 \mu\text{mol/L}$ ($p < 0,05$). Simultaneously it has been found out that significantly increase of C-reactive protein in both groups – $6,2 \pm 0,25 \text{ mg/L}$ and $4,1 \pm 0,22 \text{ mg/L}$ respectively, in comparison with almost healthy patients – $1,43 \pm 0,06 \text{ mg/L}$ ($p < 0,05$). The comparative analysis of the examined groups has proved the significant difference ($p < 0,05$) in levels of homocysteine and C-reactive protein.

Conclusions: Thus, as a result of studies, it has been found out that there is an exacerbation of COPD, in the isolated course of disease as well as in disease combined with chronic pancreatitis, there is an observed increased activity of C-reactive protein and homocysteine, which may cause the development of cardiovascular complications in such patients. At the same time, changes in patients with comorbidities of COPD and chronic pancreatitis were significantly deeper and had significant differences from those in patients with isolated COPD, reflecting significant increase of cardiovascular risk in patients with comorbidity.