

Methods: Whole blood of apparently healthy donors (n=24) and persons with clinical manifestations of atherosclerosis (n=27) was used. Determination of cholesterol and LDL concentrations was carried out by methods generally accepted in the clinic. The assessment of the degree of hemolysis was determined by the change in the light transmission of a suspension of erythrocytes suspended in 0.9% NaCl in the presence of HCl (hemolytic) at 500 nm. It is not hemolysis that is recorded, but damage to the erythrocyte membrane, since light scattering changes due to the collapse of the membrane and the erythrocyte cytoskeleton.

Results: In the group of apparently healthy donors, the cholesterol level was 4.1 ± 0.5 mmol/l, the concentration of LDL was 2.1 ± 0.3 mmol/l, the degree of HCl-induced erythrocyte hemolysis was $31.5 \pm 4.6\%$. In the group of patients with atherosclerosis, the cholesterol level was 6.9 ± 1.3 mmol/l, the LDL concentration was 4.7 ± 0.8 mmol/l, the degree of erythrocyte hemolysis induced by HCl was $48.4 \pm 8.2\%$.

Conclusions: The high levels of cholesterol and LDL are associated with the degree of HCl-induced erythrocyte hemolysis ($p < 0.01$).

HCl-induced hemolysis of erythrocytes reflects the degree of metabolic damage to cells. An increase in the degree of induced hemolysis of erythrocytes can be a marker for assessing damage to endothelial cells.

P454 / #713, TOPIC: AS03 DYSLIPIDEMIA AND RISK FACTORS / AS03.17 OTHER.

ASSOCIATION BETWEEN HYPERCHOLESTEROLEMIA AND OXALATE HOMEOSTASIS IN A RATS

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Background and Aims: Hypercholesterolemia is closely related to impaired kidney function and, consequently, increased plasma oxalic acid (POx) concentrations. However, whether hypercholesterolemia per se is associated with impaired oxalate homeostasis remains unknown. In this experimental study, we compared the association between hypercholesterolemia and POx concentrations and urine oxalate (UOx) excretion in rats with and without acute kidney injury (AKI).

Methods: Male Wistar rats (200–300 g, n=20) were randomly divided into 2 groups. After 24 hours of water deprivation, rats in group 1 (n=10) received an intramuscular injection of 50% glycerol (10 ml/kg body weight), while group 2 (n=10) served as the control group. During the 10-week experimental period, POx concentration, UOx excretion, serum creatinine, and total cholesterol levels were measured in each group of rats. Data analysis and all graphs were generated using MedCalc software.

Results: At 10 weeks following AKI initiation, significantly lower UOx and higher concentrations of serum creatinine, total cholesterol, and POx were observed in the experimental group compared with the control group (Fig. 1).

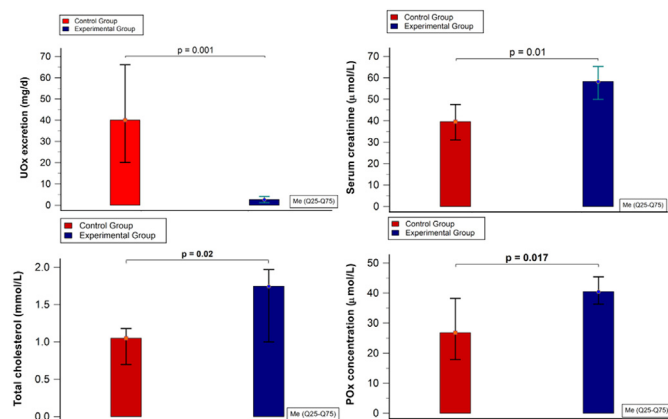


Fig. 1. Daily UOx, serum creatinine, total cholesterol and POx concentrations in the experimental and control rats

Obviously, serum creatinine level was directly associated with total cholesterol ($r=0.57$, $p=0.03$) and POx ($r=0.55$, $p=0.002$) concentrations, and showed an inverse correlation with UOx excretion ($r=0.37$, $p=0.04$) in the experimental group. However, a direct strong correlation between cholesterol and POx levels was also observed in the control group (Fig. 2).

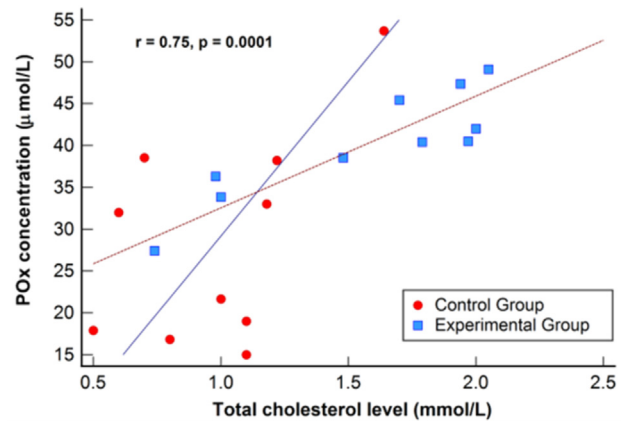


Fig. 2. The association between total cholesterol and POx concentrations in rats

In the partial correlation analysis, total cholesterol level was significantly associated with POx concentration independently of creatinine level ($r=0.53$, $p=0.03$).

Conclusions: Hypercholesterolemia is associated with increased POx concentration in rats independent of kidney function.

P455 / #1490, TOPIC: AS03 DYSLIPIDEMIA AND RISK FACTORS / AS03.17 OTHER.

THE IMPORTANCE OF CASCADE SCREENING OF FAMILIAL HYPERCHOLESTEROLEMIA IN ROUTINE CLINICAL PRACTICE

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Background and Aims: The problem of late diagnosis of familial hypercholesterolemia (FH) is common in routine clinical practice. Cascade screening within the family is necessary to ensure early diagnosis of FH for timely initiation of treatment. We report a three-generation pedigree where 7 cases of FH were identified.

Methods: FH was documented by DLCN criteria. Mutation was identified by targeted NGS sequencing for the proband and subsequently by Sanger sequencing in family members.

Results: The proband was a female with FH diagnosed at the age of 33. She has a tendon xanthomas and atherosclerotic lesions of carotids. Maximal level of total cholesterol (TC) was 16 mmol/l. In her twin sons high level of TC (10,5 and 12,0 mmol/l) was detected at the age of 3. The proband's mother and elder sister of 37 years have TC level 9,0 and 13,0 mmol/l respectively. Two of the three children of the sib (the girl of 12 years and boy of 3 years) also have elevated level of TC: 8,77 and 6,89 mmol/l. The mother of the proband has a history of acute ischemic insult at the age of 53. Pathogenic variant of LDLR: rs121908038 c.1202T>A p.(Leu401His), was detected in the proband, her sib and their affected siblings. Combined lipid lowering therapy was initiated in the proband and her sib. The siblings with FH are supervised by pediatric and lipidologist.

Conclusions: The cascade screening of FH provides the necessary ensures for early diagnosis and timely initiation of treatment to prevent the cardiovascular events.