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KIDNEY PROBLEMS IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Kidney involvement in RA is clinically meaningful because it worsens the course of primary disease and increases mortality. The pathogenesis of renal damage in rheumatoid arthritis (RA) is a complex combination of congenital and acquired defects in immunoregulatory mechanisms . Subjects hospitalized for RA are significantly more likely to have a recorded cause of death due to renal failure. Proteinuria may be the first clinical sign in many renal disorders, for example, in amyloidosis patients.

The aim: to study the changes of cytokines at different stages of chronic kidney disease (CKD) progression in patients with RA.

The study involved 120 patients with RA II-III. The presence of CKD was established according the classification adopted by the 2nd Congress of Nephrology Ukraine. The formula CKD-EPI was used for determination of glomerular filtration rate (GFR). Patients were divided into four groups according presence of CKD: I- RA patients without CKD (n=22), II- RA with CKD stage I (n=34), III- RA with CKD stage II (n=33), IV- RA with CKD stage III (n=31)). Comparison group was 20 healthy individuals. In addition to conventional laboratorial tests, serum cytokins (transforming growth factor-β1 (TGF-β1), tumour necrosis factor-α (TNF-α), interleukin-1β (IL-1β), IL-10) were studied by ELISA.

In patients with RA in the evolution of CKD I-III the disbalance of pro- and anti-inflammatory cytokines was found: increase in the level of proinflammatory IL-1β ($p<0,05$) and TGF-β1 ($p<0,05$) in all patients with rheumatoid arthritis, decrease in the level of anti-inflammatory IL-10 ($p<0,05$), which was more pronounced in the CKD stage III.

Correlation relationships were established between TGF-β1 and the age of patients ($r=0,62$) ($p<0,05$) and daily proteinuria ($r=0,68$) ($p<0,05$), between TNF-α and proteinuria ($r=0,63$) ($p<0,05$) inverse correlation between TGF-β1and GFR ($r=-0,55$) ($p<0,05$).

Thus, there was established the active participation of pro- and anti-inflammatory cytokines in the development of immune inflammation in patients with RA, a large prognostic value for the growth of TGF-β1 in the progression of CKD in RA. These studies will allow in further to adjust the treatment of patients with rheumatoid arthritis and to prevent the progression of complications from kidney disease in time.

Kvasnytska O. B.

PATHOGENETIC MECHANISMS OF FUNCTIONAL RENAL IMPAIRMENT IN PATIENTS WITH CHRONIC HEPATITIS

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In patients with chronic hepatitis (CH) in the period of exacerbation the development of edema syndrome is possible. It should be kept in mind that in the emergence of water-electrolyte imbalance not only hypoproteinemia, but also changes in the function of the kidneys play a role. Today there is no doubt that a significant role in the pathogenesis of the progression of liver diseases of different etiologies belongs to the free radical processes (FRP) and the syndrome of endogenous intoxication (SEI).

Objective of the work is to establish changes in renal function in patients with CH, as well as clarify the role of FRP and the SEI on the development of renal dysfunction.

22 patients with low-active CH of non-viral etiology with disease duration from 3 to 6 years were examined. Clinically peripheral edema was diagnosed in 21% of patients. The functional state of the kidneys was studied under conditions of 12-hour spontaneous diuresis and when conducting a water load in the amount of 0.5% of body weight. FRP were assessed by the level of malonic aldehyde in the blood (MA), the degree of endotoxicosis by the level of medium molecular peptides (MMP). The control group consisted of 20 healthy persons of the corresponding age.

The results of the study showed that under conditions of spontaneous diuresis, significant changes in the function of the kidneys in patients were absent. At the same time, during the water loading, the diuresis was reduced, both absolute and standardized; glomerular filtration (GF) was reduced by 3 times ($p<0.05$) in relation to a group of healthy individuals. There was also a significant impairment of the ion-regulating function of the kidneys: if in healthy individuals, sodium excretion increased by 50% in relation to spontaneous diuresis, then it was to decrease in patients with CH ($p<0.05$). Similar changes were observed regarding the excretion of ammonia and titrated acids ($p<0.05$)

Correlation analysis showed the relationship between the MA indicator in the blood and the specific gravity of urine ($r= 0.81$, $p<0.05$), as well as GF ($r=-0.56$, $p<0.05$); and the level of MMP 280 correlated with the level of sodium in the blood and its excretion ($r=-0.54-0.58$, $p<0.05$)

Thus, in patients with chronic hepatitis in the early stages of the disease during the water load, there is a disturbance of the adaptive properties of the kidneys by reducing GF. At the same time, both FRP and the SEI may have a certain role in the development of these disorders, which requires further study.

Liakhovych O.D.

FEATURES OF THE CLINICAL COURSE OF NON-ALCOHOLIC STEATOHEPATITIS DEPENDING ON THE PRESENCE OF COMORBID DISEASES: OBESITY AND OSTEOARTHRITIS

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Today non-alcoholic fatty liver disease (NAFLD) is one of the most common diseases in hepatology, which leads to poor quality of life, reducing its duration. With regard to the etiology of NAFLD, it is quite diverse, although its close relationship with insulin resistance (IR) is noted. The liver is a major target of lesions in conditions characterized by IP, which is a factor in the risk of progression of liver steatosis in NASH, with an inherent risk of progression to cirrhosis.

Objective – to find out the features of the clinical course of non-alcoholic steatohepatitis (NASH) depending on the presence of comorbid diseases: obesity (Ob) and osteoarthritis (OA), the degree of obesity. 140 patients with NASH, OA, obesity or with their combination, were examined including 30 patients with OA and normal weight (BMI = 21-25 kg /m^2), 80 patients with OA, NASH and obesity (BMI higher than 30 kg/m^2), 30 patients with NASH and obesity without OA (BMI>30 kg/m^2). The average age (63.1±5.3) years. The duration of NASH disease ranged from 2 to 11 years. The control group consisted of 30 healthy individuals with normal body weight, including 12 men and 18 women.

Clinical manifestation of non-alcoholic steatohepatitis during comorbid obesity and osteoarthritis (compared with the course without OA) includes prevailing astheno-vegetative syndrome and hepatomegaly (96,3% and 100,0% of cases versus 50,0% and 93,3% in patients without osteoarthrosis), dyspeptic (OR=2,61, 95% CI [1,13-6,03]) and cholestatic (OR=3,40, 95% CI [1,21-9,58]) syndromes (with frequency of 78,8% and 56,3% of cases vs. 30,0% and 16,7% for NASH without OA), splenomegaly (OR=4,75, 95% CI [1,04-21,75]) (31,7% vs. 6,7%) frequency and intensity of biochemical syndromes: cholestasis (65,0% vs. 23,3%), mesenchymal inflammation (100,0% vs. 40,0%), hepatic cell failure (41,3% vs. 10,0%), which significantly exceeded their intensity in NASH patients without OA ($p<0,05$).

With increase of obesity from I to II degree in patients suffering from NASH with OA the frequency of asthenia, cholestasis, splenomegaly increased among biochemical clinical signs: degree of liver steatosis (Steato-test), mesenchymal inflammation and cholestasis ($p<0,05$). With increase of obesity from the II to III degree the frequency of dyspepsia, cholestasis, abdominal discomfort, splenomegaly increased among clinical signs. The frequency of cholestasis and hepatic cell failure (HCF) increased among biochemical syndromes ($p<0,05$). The chance of occurrence of biochemical syndromes: mesenchymal inflammation (OR = 2,50, 95% CI [1,17-5,34]), cholestasis (OR=2,79, 95% CI [1,11-6,96]), HCF (OR = 4,50, 95% CI [1,27-16,04]) and the formation of