The red blood cell distribution width is a standard component of a routine complete blood count test. RDW quantifies the variation of individual red blood cell volumes, which vary from one cell to the next and for the same cell as it circulates during its approximately 115-day lifespan. Elevated RDW is associated with an increased risk for all-cause mortality. In our opinion, increasing of the RDW-CV is also an evidence of the important pathogenetic role of disintegration processes that take place in the pancreas, and of the development of active inflammatory process in the latter.

The higher levels of RDW-CV, which exceeded 14.5%, were observed in 10.5% of the patients with chronic pancreatitis exacerbation. On the contrary to these data, in patients with acute pancreatitis with development purulent-necrotic complications in the future the exceeded 14.5% level was found in 37.3%. The severe course of acute pancreatitis with a high level of RDW-CV was confirmed clinically (the occurrence of purulent-necrotic complications), and by the laboratory examinations (increase in the level of peripheral blood leukocytes, leukocyte intoxication index, C-reactive protein). The obtained clinical observation data confirm that a higher RDW-CV may be a predictor of complicated acute pancreatitis.

Thus, the proposed method of predicting the course of acute pancreatitis by RDW-CV showed high clinical efficiency of prediction and availability, and has no contraindications. Its use will provide early prediction and stratification of a more severe course of acute pancreatitis with the development of purulent-necrotic complications.

Kolodnitska T.L.

CURRENT VIEWS ON THE PM2.5 EXPOSURE EFFECT ON COAGULATION, INFLAMMATION AND ENDOTHELIAL FUNCTION

Department of Internal Medicine, Physical Rehabilitation and Sports Medicine
Bukovinian State Medical University

Many epidemiological and clinical studies demonstrate that particulate matter (PM) increases the risk of cardiopulmonary disorders, such as asthma, bronchitis, arrhythmias, atherosclerosis, and so on, while PM-induced oxidative stress and inflammation are possibly responsible for these different diseases. There is also evidence that air pollution is related to thrombosis and endothelial dysfunction. A lot of researchers nowadays try to find the most susceptible to PM exposure people. However, mechanisms and sources of susceptibility are still unclear. This may be due to comorbidity and epigenetic states.

The aim is to analyze changes in markers of coagulation, inflammation and endothelial function associated with PM2.5 exposures. Research methods are informational-analytical, content-analysis.

Exposure to PM leads to kinds of cardiopulmonary diseases, such as asthma, COPD, arrhythmias, lung cancer, etc., which are related to PM-induced inflammation. It was found that PM2.5 (aerodynamics diameter <2.5 mm) exposure induces inflammatory response both in vivo and in vitro. Since the toxicity of PM is tightly associated with its size and components, PM1 (aerodynamics diameter <1.0 mm) is supposed to be more toxic than PM2.5. However, the mechanism of PM1-induced inflammation is not clear.

Particulate air pollution has been associated with triggering of myocardial infarctions and increased cardiovascular mortality. Potential pathways for these effects include increased systemic cytokine-mediated inflammation, endothelial dysfunction, increased thrombosis, decreased plaque stability, and increased arrhythmias. Previous studies have found that air pollution influences markers of coagulation (such as fibrinogen), inflammation (such as C-reactive protein), and endothelial function (such as ICAM-1 and VCAM-1). Elevations in these blood markers, in turn, have been associated with an increased risk of adverse cardiovascular events.

Air pollution effects on cardiovascular disease (CVD) are stronger among subjects with fibrinogen and IL-6 gene variants. However, epigenetic modifications may be as important as genetic polymorphisms in CVD pathogenesis.

Air pollution may affect some plausible biological mechanisms that could explain some of the exacerbation of CVD morbidity and mortality. Air pollution exposure may increase systemic cytokine-mediated inflammation and prothrombotic activity. In susceptible people, ultrafine particles were able to provoke alveolar inflammation, with the release of mediators capable of increasing blood coagulability. Increased plasma viscosity is a potential mechanism explaining why high fibrinogen levels are related to increased CVD risk. Similarly, elevated C-reactive protein, ICAM-1, and VCAM-1 levels have been associated with inflammation and cardiovascular risk. An increase in C-reactive protein may reflect arterial damage from white blood cell invasion and inflammation within the wall due to air pollution exposure, thus inducing cardiovascular events.

PM exposure effect on markers of coagulation, inflammation and endothelial function. This association should be modified by race, sex, and age. The question about the most susceptible people is still not answered.

Lukashevych I.V.

DYNAMICS OF INDICATORS ANTIOXIDANT PROTECTION IN PATIENTS WITH CHRONIC HEPATITIS DURING THE COMPREHENSIVE TREATMENT WITH INCLUSION “HEPTRAL” BELONGS TO DISEASE WITH CHRONIC HEPATITIS

Department of Propedeutics of Internal Diseases
Bukovinian State Medical University

The aim of our study was to study the effect of “Heptral” on the results of treatment of patients with chronic hepatitis non-viral origin. 41 patients with chronic hepatitis aged from 22 to 75 (51,3 ± 14,5) have been explored. According to the treatment, patients are divided into two groups. The basic group consisted of 21 patients, whom together with standard treatment prescribed pills “Heptral” 1 tablet three times a day 30 minute before meal for 15-18 days.The group for compare were 20 patients with chronic hepatitis non-viral origin,who received the standard treatment. The group for check up were 20 practically healthy volunteers. We researched the concentration in the blood of the reaction products thiobarbituric acid content of glutathione in the blood, activity of catalase, glutathione peroxidase.

As a result of research discovered a significant increase in the concentration of reduced glutathione during treatment in patients who additionally received “Heptral”.They had contents of reduced glutathione after treatment higher by 26,1% ($p<0,05$) in compare with contents before treatment. The trend to reduced activity of glutathione peroxidase observed during treatment in both groups of patients, but it was not credible. Blood catalase activity significantly increased after treatment in patients who took “Heptral” on average by 20,4% ($p <0,05$) in compare with that before treatment, in patients of the group of compare – by 13,8% ($p <0,05$). After treatment we could see decrease of concentration of reaction products of thiobarbituric acid in patients of both group, more reduction of their content noted in patients , whom to complex treatment was included “Heptral”.

During two weeks of treatment better antioxidant status was adjusted in patients with chronic hepatitis, whom in addition to standard treatment took “Heptral”. For full correction of the clinical manifestations of the disease and antioxidant status should follow the chosen schemes of treatment as the maintenance dose to begin of stable remission in outpatient stage.

Nesterovska R.A.

COLCHICINE EFFICACY AND SAFETY FOR THE TREATMENT WITH ISCHEMIC HEART DISEASE: A SYSTEMATIC REVIEW AND META-ANALYSIS OF RANDOMIZED TRIALS

Department of Internal Medicine, Physical Rehabilitation and Sports Medicine
Bukovinian State Medical University

Colchicine, an anti-inflammatory drug that has been used in rheumatology for a long time to treat gout and prevent seizures; it was firstly presented in cardiology to reduce the recurrence rate of