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**“DIGITALIZATION” IN CARDIOLOGY:  
CHANGES IN ECG MARKERS DURING COVID-19**

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COVID-19 pandemic adjusts care for patients with cardiovascular disease. According to the European Association of Cardiologists Recommendations, no specific electrocardiogram (ECG) differences were found in patients with and without confirmed COVID-19. The minimal findings were rather signs of myocarditis (apparently caused by the virus itself) and a small number of arrhythmias in such patients. It is clear that the final diagnosis of COVID-19 can be made only in the presence of a positive PCR or ELISA test, and the presence of other “specific” signs of SARS-CoV-2 infection. However, this little informativeness applies to the routine ECG performed on all patients admitted to the hospital.

The aim of the study is to establish possible differences in the ECG of patients with / without a confirmed diagnosis of COVID-19 and various cardiac pathology in digital processing of routine ECG using the software-diagnostic complex “Smart ECG” and the ability to assess the course of treatment of these patients.

The routine ECG was digitally processed to determine the angle  $\alpha$  of the ST segment slope and the extension height H of the ST segment slope (ST, mV). The first derivative of the T wave with the calculation of the maximum velocities ratio (MVR) and the adjacent extreme values ratio (AEVR) were obtained. These parameters were evaluated in a patient diagnosed with COVID-19 and probable myocarditis of viral etiology.

In the analysis of the studied parameters obtained by “digitalization” of routine ECG made on the 1st, 5th and 10th days of treatment in a patient with diagnosed COVID-19 and probable myocarditis of viral etiology. The dynamics of changes in the background of therapy shows the normalization of MVR (0.393; 0.417 and 0.833), which indicates a positive effect of anti-ischemic therapy, as well as a decrease in microvascular myocardial damage COVID-19, with the development of pericyte damage, which can also lead to ischemia. At the same time, the growth of AEVR with its subsequent decline (1,167; 1,375 and 1,0), as well as changes in the angle  $\alpha$  (8,53; 6,84 and 4,54) and its continuation height H (0,37; 0,30 and 0.40), may reflect the dynamics of COVID-19 in this patient and treatment efficacy.

The use of “digitalization” of the ECG in patients with cardiovascular disease and the presence / absence of COVID-19, can significantly improve the informativeness and specificity of the classical ECG and improve its diagnostic and prognostic value in this group of patients. The dynamics of changes in the indicators obtained during “digitalization” on the background of therapy demonstrates their normalization, which indicates a positive effect of therapy, and may reflect the course of COVID-19 and concomitant cardiac pathology.

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**ASSOCIATION OF RED BLOOD CELL DISTRIBUTION WIDTH WITH THE ACUTE  
PANCREATITIS AND CHRONIC PANCREATITIS EXACERBATION FROM THE  
POSITION OF THE PROGNOSIS**

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The aim of the research was to investigate the association of red blood cell distribution width (RDW) with the acute pancreatitis and chronic pancreatitis exacerbation as a possible prediction factor. Moreover, biomarkers are urgently needed for patient risk stratification. This study included adults diagnosed with acute pancreatitis and chronic pancreatitis exacerbation admitted to Emergency Hospital in Chernivtsi, between January, 2017, and January, 2020. A total of 123 patients were included in the study. The clinical data were retrospectively analysed for all patients. The measures included RDW at admission or during the first 24 hours, with an elevated RDW-coefficient of variation (RDW-CV) defined as more than 14.5%.

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 PM<sub>2.5</sub> EXPOSURE EFFECT ON COAGULATION,  
INFLAMMATION AND ENDOTHELIAL FUNCTION**

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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 PM<sub>2.5</sub> 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 PM<sub>2.5</sub> (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, PM<sub>1</sub> (aerodynamics diameter <1.0 mm) is supposed to be more toxic than PM<sub>2.5</sub>. However, the mechanism of PM<sub>1</sub>-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.