pulmonary inflammation, the induction of increased blood coagulability, and endothelial dysfunction. It also might activate several signaling mechanisms such as mitogen-activated protein kinases, the antioxidant responsive element, and nuclear factor κβ cascade. Initiating of oxidative stress through translocation from the lungs into the vascular tree induces the release of pro-inflammatory mediators and the activation of phosphatidylinositol 3-kinase pathways. Exposure to air pollution increases levels of malondialdehyde and protein carbonyls in the plasma, promotes lipid peroxidation, decreases in catalase and superoxide dismutase activity and glutathione levels, increases hs-CRP, IgM, IgG, and IgE, interleukin 1β, IL-6, tumor necrosis factor α, and interferon γ and decreases CD8 and IgA levels. Intercellular adhesion molecule-1 is expressed mainly on endothelial cells under the stimulation of pro-inflammatory cytokines and is a good predictor of cardiovascular risk. Considering the potential role of these biomarkers in assessing atherosclerotic disease, these results provide an association between PM2.5 exposures and the development and progression of cardiovascular disease. Air pollution exposure and the inflammatory process could be responsible for vasoconstriction and endothelial dysfunction, leading to the autonomic imbalance of the nervous system too.

The novel approaches in molecular biology and the greater sensitivity of the analytical methods are important advances to detect early changes in biomarkers and thus prevent the development of diseases associated with PM2.5 exposures. The screening of several biomarkers could be suggested as a preventive measure to monitor health of individuals who remain chronically exposed for several hours daily. There is a clear need for more epidemiologic studies of subjects occupationally exposed to PM2.5, especially using the new toxicological approaches to identify early effects and genetic susceptibility.

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RANOLAZINE "HAS A FINGER IN EVERY PIE" – FICTION OR REALITY

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Ranolazine, an active piperazine derivative, is a well-tolerated medication that selectively inhibits the late sodium current. Analysis of clinical studies (CARISA, MARISA, ERICA, TERISA, MERLIN-TIMI 36, RIVER-PCI, RIMINI-TRIAL) proved the effectiveness of ranolazine as an antianginal and anti-ischaemic drug.

In the European Society of Cardiology (ESC) guidelines of the management of stable angina, ranolazine is given a class IIa (level of evidence B) recommendation as a second-line treatment to reduce angina frequency and improve exercise tolerance in patients who cannot tolerate, have contraindications to, or whose symptoms are not adequately controlled by β-adrenergic blockers, calcium channel blockers, and long-acting nitrates. In patients with baseline low heart rate and low blood pressure, ranolazine may be considered as a first-line drug to reduce angina frequency and improve exercise tolerance - class IIa (level of evidence B) recommendation. ESC`2020 guidelines for the acute coronary syndrome (ACS) without ST segment elevation - in the section "Other antianginal drugs" shows that ranolazine with antianginal action inhibits the late sodium current with the unproven effect of reducing the main cardiovascular events to reduce ischemia in patients diagnosed with ACS without ST segment elevation (MERLIN-TIMI 36), but reduces the frequency of recurrent ischemia.

We conducted an open controlled single-centre study "Smart ECG- Ranolazine". The aim of our work is to implement ECG digitalization and our program "Smart ECG" to estimate the effectiveness of ranolazine and to upgrade the treatment of ST elevation syndrome. We evaluated 40 patients with Q wave myocardial infarction (STEMI), were instituted basic therapy according to the modern recommendations with the addition of ranolazine (group I, 30 patients diagnosed STEMI). Control group II consisted of 10 patients with STEMI, who were instituted basic therapy without the addition of ranolazine.

Analog scale EQ–VAS indicates a positive effect of ranolazine on the quality of life of patients diagnosed with STEMI (EQ-VAS, showed a better quality of life on the 1st day in the

group of ranolazine (38%, p<0.001), the dynamics was maintained on the 10th day (5,4%, p>0.5). Ranolazine (STEMI diagnosed patients) had a positive impact on the markers of electrical myocardial instability and its ischemia - as evidenced: decreasing of the heart rate, probability of cases of SDNN (standard deviation of NN intervals) decrease (decreasing of SDNN is a strong, independent and consistent risk factor for overall and cardiac mortality), QT dispersion (increased QT variability is a risk factor for sudden cardiac death) and maybe increase of the ratio of maximum velocity for differentiated deflection T.

This paper presents the possibility and expediency of using information systems in diagnostics – ECG digitalization and "Smart-ECG" which showed ranolazine efficiency for optimization of treatment of ST segment elevation syndrome, therefore, this study is relevant and needs further implementation.

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PREVENTION OF POSTPNEUMONIC PULMONARY FIBROSIS IN PATIENTS WITH BILATERAL VIRAL-BACTERIAL PNEUMONIA

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Improving the effectiveness of viral-bacterial pneumonia treatment related to the COVID-19 pandemic has become particularly important, as it is this complication of viral infection that is associated with mortality or reducing quality of life. A common pathomorphological feature of viral pneumonia is the tendency to form thrombotic masses in the capillaries of the pulmonary circulation, in contrast to bacterial pneumonia, when pathological process dominates by intra-alveolar fibrinous exudation.

In both cases, with fibrinolysis insufficiency and low resorptive potential, there is a risk of developing postpneumonic pulmonary fibrosis (PPF) or in the form of lung carnification (intraalveolar fibrosis) or a process similar to fibrosing alveolitis. The development of postpneumonic pulmonary fibrosis is associated with the development of a restrictive type of chronic pulmonary insufficiency of varying severity depending on the area of the affected lung tissue.

In the infectious department of MNE "City Clinical Hospital № 3" we tested the method of a combined use of two drugs to prevent the development of PPF with different mechanisms of antifibrotic activity: peroral administration of seratthiopeptidase tablets of 20 mg twice a day and inhalation of budesonide 500 mcg twice a day.

Serratiopeptidase was chosen by us due to its inherent fibrinolytic, anti-inflammatory and anti-edematous activity. Serratiopeptidase, a serine protease with a molecular weight 60 kDa, has been significantly reported for its potent anti-inflammatory activity. The clinical use of enzyme was reported for many diseases like arthritis, sinusitis, inflammatory bowel disease and bronchitis etc. The current challenge toward developing serratiopeptidase into an effective broadspectrum anti-inflammatory drug is due to lack of precise molecular mechanism. This proteolytic enzyme was reported effective in many diseases precisely during surgical events for a long time, but there is a lack of research evidence and available literature (Tiwari M., 2017).

The drug also attracted our attention due to its hydrolytic inactivation of bradykinin, histamine and serotonin, it is able to directly reduce the dilation of capillaries and control their permeability. Serrathiopeptidase blocks plasmin inhibitors by stimulating its fibrinolytic activity. Due to a possible potentiation of anticoagulants, which are widely used in pneumonia caused by coronavirus, the use of the drug is not recommended in the presence of hemoptysis.

The local activity of the budesonide molecule is known to interact with specific receptors in the cytoplasm of the cell and form a complex that binds to DNA and stimulates the formation of mRNA after penetrating the cell. It leads to changes in the ribosomes of proteins corresponding to the properties of cells. The drug stimulates the synthesis of lipocortin, which inhibits the enzyme phospholipase A2, resulting in inhibition of the synthesis of prostaglandins and leukotrienes involved in the development of inflammatory reactions.