All patients were diagnosed with ventricular extrasystoles (VE) and supraventricular extrasystoles (SVE) according to HM results. According to the results of clinical examinations, patients were divided into groups SA (10 patients), PIMF (12 patients), NCD (13 patients) and AH (11 patients), in constellation with SA or PIMF. All patients underwent HM ECG with analysis of electrocardiographic changes and assessment of "turbulence onset" by the formula:

HRTO = (RR_1 + RR_2) - (RR_{-2} + RR_{-1}) / (RR_{-2} + RR_{-1}) × 100 [%], where RR_{-2} і RR_{-1} – intervals before VE / SVE, RR_1 і RR_2 – two RR intervals immediately following the compensatory pause. According to the accepted criteria, the indicator HPTO <0% was considered as norm.

All patients underwent analysis of HRT parameters. The magnitude of the increasing of the sinus rhythm frequency after VE is recognized as normative at TO <0% and pathological at TO> 0%, which led to the formation of two groups (normative and pathological) with a significant difference of TO (-0,040±0,0113 against 0,062±0,0159 %, p<0,001). Patients with NCD against groups with structural myocardial diseases were characterized by a reliable normative distribution of TO (negative -0,022±0,0198 against positive 0,030±0,0163%, p<0,05). At the same time, differences in the groups of present / absent SA (0,027±0,0208 against 0,015±0,0183%, p>0,5) and/or PIMF (0,031±0,0238 against 0,008±0,0157%, p>0,2), in particular depending on the impact of AH (0,013±0,0186 against 0,022±0,0198%, p>0,5) was not detected, all indicators were positive and showed a level of TO>0%, unfavorable prognostic distribution of HRT was detected in the presence of structural heart diseases - SA, PIMF and AH, which can predict the risk of SCD.

Patients with functional pathology (NCD) are characterized by a normative distribution of the "turbulence onset" (TO <0%, p <0.05) against groups with structural myocardial damage (SA, PIMF) with present VE, differences in groups of present / absent SA or PIMF, in particular, depending on the influence of AH was not detected, all indicators showed an unfavorable prognostic distribution of the onset of turbulence (TO> 0%).

Kolodnitska T.L.

BIOMARKERS OF THE PM2.5 EXPOSURE EFFECT ON THE CARDIOVASCULAR SYSTEM

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Numerous epidemiological studies have shown that air pollution exposure is associated with negative health impacts, such as increased mortality and morbidity due to respiratory and cardiovascular diseases and cancer. Urban areas are usually characterized by an elevated rate of air pollution through PM emitted by cars burning fossil fuel or during abrasion processes of vehicle tires and brakes. Toxicological studies indicate that urban traffic-related air pollution contains several compounds, e.g. metals and polycyclic aromatic hydrocarbons, which are absorbed into the PM2.5 surfaces. These particles can go through the nose and enter the lower respiratory tract, causing serious health effects when inhaled.

Aim: To analyze changes in biomarkers associated with PM2.5 exposures. Research methods: informational-analytical, content-analysis.

Air pollution causes harmful effects on people, leading to both acute and chronic diseases. The general population is exposed to environmental pollutants. However, individuals who remain chronically exposed for several hours daily are more vulnerable to developing exposure-related toxicological effects.

Biomarkers used in human health monitoring are divided into three classes: biomarkers of exposure, effect, and susceptibility. Biomarkers of exposure are considered an important tool, as they estimate the levels at which chemical substances are absorbed by quantifying the toxic substances or metabolites in biological samples. Biomarkers of effect are biological parameters that reflect the interaction between xenobiotics and cellular, biochemical, or molecular targets. They relate to oxidative stress, the inflammation process, and genotoxicity.

Fine airborne particles lead to reactive oxygen species production and consequently macromolecule damage, as well as activation of inflammatory mediators capable of exacerbating

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