observation period, in particular, 94 (25,54%) patients – during 28-day staying in hospital, and 29 (7,88%) – during a year of observation.

With a purpose to create prognostic models of AMI complicated by acute left-ventricular failure (ALVF), all the patients were divided into 2 groups: group 1 – with favorable AMI outcome, and group 2 – with fatal outcome.

Patients who died were on an average 9 years older comparing those with favorable outcome. Males were prevalent amongst ($p<0,001$). Besides, repeated AMI was registered much more frequently in 2 group patients (79,6% vs 39,19% in group 1, $p<0,001$). Class 2-4 ALVF signs by T.Killip were significantly more frequent in group 2 patients ($p<0,001$). Frequency of arterial hypertension (AH) and diabetes mellitus (DM) presence in anamnesis was significantly higher in group 2 patients as well ($p<0,01$). Risk factors prevalence analysis among patients of both groups revealed significant prevalence of active smoking ($p<0,01$) and obesity ($p<0,001$) in group 2 patients as well.

Single-factor regression analysis results indicated the fact that risk of lethal event occurrence increased with age: increase of risk by a factor of 1.5 follows each additional 5 years over 50. Risk of lethal event appearance raised twice with every ALVF class by Killip increase, 1.02 times more with income HR increase on 10 b.p.m. after 60 b.p.m., 1.3 times more in patients with DM, 1.15 times more in case of obesity presence, three times more in patients with chronic heart failure (CHF), 1.2 times more in case of ejection fraction (EF) below 40% detected during 1-2 days after patient's admission, and 4.5 times more in case of anterior AMI localization.

IL-1 α content analysis revealed its significant predominance in group 2 patients (48,94+7,05 vs 22,43+3,41 pg/ml (group 1), $p<0,01$). IL-6 level was markedly higher in group 2 patients as well (51,63+7,86 vs 16,84+3,94 pg/ml, $p<0,01$), and level of anti-inflammatory cytokine IL-10 was lower in group 2 patients comparing group 1 (2,45+0,51 vs 4,03+0,73 pg/ml, $p>0,05$).

Tumor-necrotizing factor (TNF) and neopterine (Np) levels analysis in the groups indicates significant predominance of these both values in group 2 patients comparing group 1: $63,41\pm3,78$ vs $43,1\pm2,62$ pg/ml for TNF ($p<0,01$) and $24,28\pm4,32$ vs $15,08\pm1,76$ nmol/l for Np ($p<0,05$).

Elder patients age, higher class of ALVF, presence of DM and CHF, anterior localization of AMI, smoking and obesity, EF low then 40% are independent predictors of lethal event development in patients with AMI and ALVF. Besides, increase in pro-inflammatory cytokines level (IL-1α, IL-6, TNF and Np) parallel with worsening of EchoKG EF results promote increase of lethal event onset probability in the mentioned category of patients.

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CHANGES OF HEART RATE TURBULENCE AS A PREDICTOR OF RISK IN PATIENTS WITH CORONARY AND NON-CORONARY HEART DISEASES AND VENTRICULTURAL EXTRASYSTOLS

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Circulatory diseases remain the leading cause of disability and mortality, despite a wide range of diagnostic methods to objectify the cardiovascular system. Electrocardiography (ECG) and Holter ECG monitoring (HM) remain the key methods for assessing heart rate and electrical processes in the myocardium. HM provides an opportunity to analyze the heart rate turbulence (HRT) with assessment of "turbulence onset" (TO), especially in high-risk groups, and helping assess prognostic value in the development of sudden cardiac death (SCD).

The aim of the study was to evaluate changes in HRT and features of TO in patients with stable angina (SA), postinflammatory myocardial fibrosis (PIMF), neurocirculatory dystonia (NCD) and the effects of arterial hypertension (AH).

An analysis of the results of examinations of 35 patients consulted at the Department of Internal Medicine, Physical Rehabilitation and Sports Medicine of HSEE "Bukovinian State Medical University" with complaints of cardiac pain of unknown origin.

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%).

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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