Міністерство освіти і науки України
Харківський національний університет імені В. Н. Каразіна
Медичний факультет

XVI МІЖНАРОДНА НАУКОВА КОНФЕРЕНЦІЯ
СТУДЕНТІВ, МОЛОДИХ ВЧЕНИХ ТА ФАХІВЦІВ

АКТУАЛЬНІ ПИТАННЯ СУЧАСНОЇ МЕДИЦИНИ

Тези доповідей

28-29 березня
2019 року

do 25-річчя від дня відродження кафедр внутрішньої медицини, хірургічних хвороб, загальної та клінічної імунології та алергології медичного факультету Харківського національного університету імені В.Н. Каразіна
EARLY KIDNEY DAMAGE IN ADOLESCENTS WITH PRIMARY ARTERIAL HYPERTENSION
Simpson Tarek S., Waugh Owen O. .................................................................373
CORONARY ATHEROSCLEROSIS MORPHOLOGY ASSOCIATED WITH CLINICAL PRESENTATION
Sood Purva..........................................................374
COMPARISON OF EARLY POSTOPERATIVE RESULTS AFTER UNICONDYLAR AND TOTAL KNEE ARTHROPLASTY. PILOT STUDY
Utkus Simonas, Petrauskas Vidas, Versocki Artūr ........................................375
SURGICAL CONCEPT OF SOLVING TROPHOLOGICAL PROBLEMS IN PALLIATIVE PATIENTS
Vivcharuk V. P., Pashchenko K.Yu , Pidkova A.V. ........................................376
MIR-138-1 EXPRESSION IN TUMOR AND PATIENT BLOOD PLASMA AS DIAGNOSTIC BIOMARKER OF CLEAR CELL RENAL CELL CARCINOMA
VIRAL HEPATITIS C AS A FACTOR OF ATHEROSCLEROSIS AND ISCHEMIC HEART DISEASE
Volobuiev D. A..............................................378
PECULIARITIES OF FOETAL CIRCULATION
Waugh Owen O., Simpson Tarek S., Komaromi N. A .................................379
UNTYPICAL CASE OF SYNCOPE AS A CHEST PAIN EQUIVALENT IN PATIENT WITH ACUTE MYOCARDIAL INFARCTION
Waugh Owen, Simpson Tarek .................................................................380
DIABETES MELLITUS AND CARDIOGENIC SHOCK COMPLICATING ACUTE MYOCARDIAL INFARCTION
Yasser S ...........................................................................381
MORPHOLOGICAL AND PHYSIOLOGICAL CHANGES OF LUNGS WITH THE COMPLEX EFFECT OF IMMOBILIZATION AND HYPOTHERMIA
Zhiyengaliyeva A. K., Ostanin A. A., Khamchiyev K. M., Tuleubayeva A. A .......382
present in anamnesis vitae or anamnesis morbi. No history of epilepsy or head trauma. ECG revealed sinus rhythm, heart rate 60 in min, ventricular premature contraction, repolarization abnormalities in V4-V6. During an heart ultrasound was found hypokinesia of left ventricle’s posterior segment, global contractility decline with ejection fraction (EF) – 44%, dilation of left atrium, no pulmonary hypertension, atherosclerotic changes of aorta present, no ventricular septal defect or mitral valve significant changes were noted. Because of appearance of non-numerous ventricular premature contractions on ECG was infrequent performed 24h-ECG monitoring demonstrated a tendency to bradyarrhythmia at night (till 42/min), supraventricular extrasystoles – 31 per hour, short term bigemines, once - triplet (during activity); ventricular extrasystoles – 31 per hour, short term allorhythmia of bigeminy type, unifocal and infrequent, grade 1 by Laun. On background of lipid lowering therapy with atorvastatin 20mg/day total cholesterol – 3,3 mmol/l (N < 6,2), electrolytes within normal limits (potassium – 4,2 mmol/l (N – 3,8-6,2), sodium – 136 mmol/l (N – 130-160), Ca – 2,13 mmol/l (N – 2,15-2,5)), blood glucose – 3,8 mmol/l (N 4,1-5,9), GFR – 75 ml/min/1,73m2, C-reactive protein – negative.

**Conclusion.** Cardiac origin of syncope as usual is represented by arrhythmias with ventricular tachycardia significant prevalence. Also, syncope could be associated with bradycardia and the sick sinus syndrome. In our patient’s case the cause of syncope probably is a coronary arteries disease origin global myocardium hypoperfusion with transient sinus node and heart conduction system dysfunction causing appearance of life-threatening bradyarrhythmias as primary manifestation of MI.

**UDC 616.127-005.8-061**

**DIABETES MELLITUS AND CARDIOGENIC SHOCK COMPLICATING ACUTE MYOCARDIAL INFARCTION**

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**Introduction.** In a big survey with 72,765 cardiogenic shock patients (J. B. Echouffo-Tcheugui, 2018) it was revealed that pre-existing diabetes (DM) was associated with an increased risk of cardiogenic shock and its worsen outcomes. Could percutaneous coronary intervention (PCI) decrease death rate and improve prognosis for patients as such?

**The aim of the study.** To study the possible prognostic risks of lethal outcome in patients with DM after PCI.

**Clinical case:** A 72-year-old woman presented with typical symptoms of myocardial infarction, general weakness, dyspnoea increasing in horizontal position and in minimal exertion. andintensive chest pain bothered patient from 9pm. Patient took one by one 6 tablets of nitroglycerin without relief. Felt ill in 2010, when first time
appeared retrosternal chest pain. She has been hypertensive since 1999 and DM type2 since 1995 treated with combined therapy (insulin 40U/day and glybenclimide 5 mg/day). In 2011 PCI was performed due to presence of both coronary arteries stenosis more than 60%, TIMI1 circulation type, after PCI with double stenting – no chest pain till the day of admission. During admission on ECG was found complete Left bundle branch block previously absent. Echo was performed: dilation of left heart chambers with diffuse contractility decline, EF -35%. Troponin I was 0.84 ng/ml (N – less 0.5). Since admission time (0:00 am) clinical picture of MI was presented with signs of acute heart failure and pulmonary edema without changes of ECG picture (in objective exam were remarkable: over all lung surface – wheezing, below scapular angles in both sides - rales during lung auscultation; BP 90/60 mm Hg on dopamine infusion background, pitting edema and liver size enlargement +2cm). At 2pm cardiogenic shock developed despite treatment prescribed. At 06:35 pm was registered clinical death and 6:55 – patient died.

**Conclusion.** DM is not only a risk factor for coronary artery disease but furthermore it might be an independent predictor of mortality in patients with left ventricular dysfunction after acute myocardial infarction. As a probable causes: re-stenosis after PCI, progression of a separate untreated plaques, or the development of new ones with acceleration of negative remodeling owing to neointimal proliferation after PCI and increased platelet aggregation. Also DM patients tend to have more severe and diffuse coronary disease with smaller distal vessels microangiopathy and reduced collateral blood flow as a factor of non-reversible myocardial damage area augmentation.

**UDC 612.014.2-215.8:616-089.22-001.186-092.9**

**MORPHOLOGICAL AND PHYSIOLOGICAL CHANGES OF LUNGS WITH THE COMPLEX EFFECT OF IMMOBILIZATION AND HYPOTHERMIA**

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**Introduction.** Environmental factors are often extreme for an organism, and as a rule affect it in a complex. Some literary data testify that the complex of jointly functioning irritants often leads to the complication of the shifts arising in an organism, in comparison with their isolated influence. In other works, on the contrary it is found a protective effect of one of the stressors during the complex influence of several ones.

**The aim of the study.** The aim of the research was to study lungs morphofunctional changes under the influence of the complex effect of hypothermia and immobilization.

**Materials and methods.** The complex effect of hypokinesia and hypothermia was simulated by the placing of experimental rats into the 80 cm³ camera designed by us, which has connection with the ambient environment within 6 hours for 10 days at the temperature of +3+4°C. Registration of pulmonary hemodynamics was carried out with the help of rheogram record (RG) on the RPG2 – 02 unit by the