

between 20 and 40 years respectively. Treatment outcomes appeared as acute toxicity, graded as 1, 2, 3 and 4 grades. Mainly was 1 and 2 grade, included signs as: edema, acute hematologic toxicity, nausea and vomiting. All data included positive 5-year and 10-year survival prognosis at early stage of disease.

Conclusion: treatment of testicular cancer must begin as early as possible. Due to positive results of early diagnosis will be positive goals of treatment. Also prophylaxis of testicular cancer must be provided everywhere, in all levels and groups of people.

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PREDICTIVE FACTORS OF ATTAINMENT THERAPEUTIC GOALS OF LIPIDS IN HIGH CARDIOVASCULAR RISK PATIENTS

DORONINA A., VOROBYEVA M.

PEOPLES' FRIENDSHIP UNIVERSITY OF RUSSIA, MOSCOW, RUSSIAN FEDERATION

FACULTY OF GENERAL MEDICINE, DEPARTMENT OF PROPEDEUTICS OF INTERNAL DISEASES

SCIENTIFIC ADVISER(S): VILLEVALE V., SVETLANA, MD, PhD, DSc

KOBALAVA D. JANNA, MD, PhD, DSc

Objective: Dyslipidemia is recognized as a prominent risk factor for cardiovascular disease. The recent cholesterol guidelines recommend aggressive statin therapy in patients with high cardiovascular risk. However, high-intensity statin therapy may be insufficient to achieve LDL-C targets. The aim of the study was to investigate the efficacy and predictors of attainment of target LDL-C during short-term high-intensity statin therapy in patients with high cardiovascular risk.

Methods: In 72 patients with history of clinically evident cardiovascular disease and fasting low-density lipoprotein cholesterol (LDL-C) >1.8 mmol/l or non-high-density lipoprotein cholesterol (non-HDL-C) >2.6 mmol/l (63.9% male, 60.8±8.6 (M±SD) years, current smoking 40.3%, abdominal obesity 63.9%, arterial hypertension 86.1%, myocardial infarction 73.6%, percutaneous coronary intervention 58.3%, coronary artery bypass surgery 12.5%, non-hemorrhagic stroke 29.1%, diabetes mellitus 22.2%, symptomatic peripheral arterial disease 6.9%, chronic heart failure NYHA II 54%, chronic kidney disease 11.1%, total cholesterol (TC) 5.6±1.6 mmol/l, HDL-C 1.1±0.3 mmol/l, LDL-C 3.5±1.3 mmol/l, triglycerides (TG) 2.2±1.4 mmol/l, very LDL-C (VLDL-C) 0.9±0.4 mmol/l, non-HDL-C 4.5±1.5 mmol/l, previous statin therapy 73.6%) efficacy and safety of 1 month of high-intensity statin therapy (atorvastatin 80 mg/day) was assessed. Wilcoxon test and multivariate logistic were performed. $P < 0.05$ was considered significant.

Results: 29 patients (40.3%) achieved target LDL-C level <1.8 mmol/l. Patients who attained (G1) vs did not attain (G2) target LDL-C had lower baseline levels of TC (4.8±1.1 vs 5.6±1.5 mmol/l), LDL-C (2.8±1.1 vs 3.6±1.3 mmol/l), non-HDL-C (3.8±0.9 vs 4.5±1.5 mmol/l), $p < 0.0001$. LDL-C significantly decreased in both groups: from 2.8±1.1 to 1.6±0.2 mmol/l ($p = 0.01$) and from 3.6±1.3 to 2.4±0.5 mmol/l ($p < 0.0001$) in G1 and G2 respectively. Multivariate logistic regression analysis revealed the following independent predictors of LDL-C goal attainment: baseline LDL-C (OR=0.042, 95% CI 0.005-0.363), baseline TC (OR=0.053, 95% CI 0.01-0.294) and baseline non-HDL-C (OR=0.0853, 95% CI 0.015-0.434).

Conclusion: Short term high-intensity statin therapy is effective only in 40.3% of high cardiovascular risk patients. Baseline LDL-C may be a predictor of early response in this patient population. Lower baseline LDL-C is associated with attainment of target LDL-C.

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ФЕНОМЕН АНТИТЕЛО-ЗАВИСИМОГО УСИЛЕНИЯ ИНФЕКЦИИ В РАЗВИТИИ ГЕМОРАГИЧЕСКОЙ ЛИХОРАДКИ ДЕНГЕ

МИСЭЛЕ Р., ГРИГОРЯН С.

РОССИЙСКИЙ УНИВЕРСИТЕТ ДРУЖБЫ НАРОДОВ

КАФЕДРА ИНФЕКЦИОННЫХ БОЛЕЗНЕЙ И ЭПИДЕМИОЛОГИИ

НАУЧНЫЙ(ЫЕ) РУКОВОДИТЕЛЬ(И): Д. М. К. КОЗЕВНИКОВА Г. М.,

ANTIBODY-DEPENDENT ENHANCEMENT AS A THEORY FOR THE DEVELOPMENT OF DENGUE

MICHELLE R., GRIGORYAN S.

PEOPLES' FRIENDSHIP UNIVERSITY OF RUSSIA, RUSSIA, MOSCOW

DEPARTMENT OF INFECTIOUS DISEASES AND EPIDEMIOLOGY

SCIENTIFIC ADVISER(S): PROFESSOR GAUINA M. KOZHEVNIKOVA

Dengue fever (DF) may clinically develop into DF, Dengue Hemorrhagic Fever (DHF) or Dengue Shock Syndrome (DSS). The possible cause for the development of the more severe forms of the infection is the immune response caused by the host with secondary infection, also known as antibody-dependent enhancement.

Dengue fever (DF) is the most rapid spreading mosquito-borne viral hemorrhagic fever, without any available specific treatment or prophylactics. It may clinically develop into DF, Dengue Hemorrhagic Fever (DHF) or Dengue Shock Syndrome (DSS). In 2014 in 8 months 77 cases of imported DF were registered in Russia. Among returned travelers the most common pathologies, overall, are dermatologic conditions, diarrheal syndromes, febrile systemic illness. DF and malaria are the most frequent causes of fever after travel. Aim: This publication is intended to contribute to prevention and control of dengue and to serve as a reference source for health workers and researchers. Methods: Retrospective analysis of patient history (2014) in the Clinical Hospital for Infectious Diseases №2 in Moscow, Russia. Results: We observed adult patients, admitted after returning from Southeast Asia. All 8 of our patients initially presented with a fever (38°C, 5-7 days), weakness, myalgia, nausea, diarrhea, severe thrombocytopenia, leukocytopenia and monocytosis – a feature of the host-virus interaction. 4 patients developed hepatomegaly and had elevated ALAT. 7 patients developed a petechial rash around ankles and torso. They were diagnosed during the 3-5 day of hospital stay – in the case of development of the dengue hemorrhagic fever (DHF) this would have been fatal. Diagnosis criteria were positive PCR, IgM and EUSA tests, clinical manifestations. Treatment: supportive, non-specific. Conclusions: 1) The severe form DHF usually develops due to a secondary infection with another type of dengue virus. The neutralization of the virus by the host's antibodies does not occur, which were formed during the primary infection, on the other hand the virus replicates more intensively in the host's monocytes in the peripheral blood. All patients had primary infection. Infections with DEN-2 have been linked with the development of the more severe form of the disease. 2) The diarrheal syndrome in our patients was secondary to the intoxication due to the DF. 3) Dengue may mimic a several number of other conditions, such as malaria, diarrheal syndromes, influenza, typhoid fever – our patients were not diagnosed with any of these infections. 4) The temperature curve in DF is a typical "saddle back" temperature curve, which helps to narrow down the diagnosis. Patients with DF usually progress into DHF and DSS after the temperature falls, therefore the correct interpretation of the temperature curve is imperative, so patients must always be reevaluated even after the phase of defervescence. 5) The main factor involved in the development of dengue hemorrhagic fever is secondary dengue infection with a different serotype, but factors like viral virulence and host characteristics are also important. Severe disease is the result of a complex interaction between the virus and the immune response caused by the host with secondary infection.

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THE ROLE OF CARDIAC MAGNETIC RESONANCE IMAGING IN THE DIFFERENTIATION OF PHYSIOLOGIC SPORT ADAPTION FROM HYPERTROPHIC CARDIOMYOPATHY

KINGA KECSKES, CSILLA CZIMBALMOS, IBOLYA CSÉCS

SEMMELWEIS UNIVERSITY, BUDAPEST, HUNGARY

SCIENTIFIC ADVISER(S): HANALKA VÁGÓ

Introduction: Hypertrophic cardiomyopathy (HCM) is the most common cause of sudden death in young competitive athletes, distinction from physiologic sport adaption is critical, especially in male athletes with expressed ventricular remodeling and frequent sudden death.

Study aims: Our aim was to compare volumes, mass and hypertrophic patterns of both ventricles of professional athletes and HCM patients using magnetic resonance imaging (CMR).

Methods: At our Clinic 75 professional male athletes free of complaint (28±6 years), 92 HCM male patients (43±8 years) and 41 male healthy volunteers were examined by CMR. On the basis of motion images left and right ventricular end-diastolic, end-systolic volume indices, stroke volume index, ejection fraction (LVEF, RVEF), mass index (LVMI) and maximal diastolic wall thickness (MDWT) were determined. Derived parameters were calculated to evaluate the hypertrophic pattern [1.diastolic wall thickness ratio: maximum-minimum wall thickness ratio (DWTR), 2. left

ventricular maximum diastolic wall thickness-end-diastolic volume index ratio (DWT_V). 3. left ventricular mass index-end-diastolic volume index ratio (M_V).

Results: In athletes left ventricular end-diastolic (122±15 vs 90.7±16.5 ml/m²), end-systolic volume indices (52±10 vs 35±10 ml/m²), stroke volume index (70±8 vs 58±11 ml/m²) and right ventricular end-diastolic (128±17 vs 83±17 ml/m²), end-systolic volume indices (57±12 vs 31±8 ml/m²), stroke volume index (139±31 vs 54±45 ml/m²) were significantly higher, left and right ventricular EF (LVEF: 58±4 vs 63±6 RVEF: 56±4 vs 63±6%) were lower in athletes compared to HCM patients. The LVMI did not differ significantly in athletes and HCM patients. The MDWT (12.9±1.7 vs 21.8±5.2 mm), DWTR (2.0±0.4 vs 3.8±1.3), DWT_V (0.11±0.02 vs 0.26±0.09 mm/m²) and M_V (0.77±0.08 vs 1.16±0.36 g/ml) were significantly higher in the HCM group compared to athletes. These differences were also observed in young HCM patients with the same age of professional athletes.

Conclusion: CMR may have important role in the distinction of athlete's heart from pathological forms providing exact functional and morphological informations.

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РАСПРОСТРАНЕННОСТЬ И ПРОГНОСТИЧЕСКОЕ ЗНАЧЕНИЕ Контрастиндуцированного острого повреждения почек у пациентов с первичным чрескожным коронарным вмешательством

Юсупов Т., Гаскина А.А.

РОССИЙСКИЙ УНИВЕРСИТЕТ ДРУЖБЫ НАРОДОВ, РОССИЯ, МОСКВА
МЕДИЦИНСКИЙ ФАКУЛЬТЕТ, КАФЕДРА ПРОПЕДИТИКИ ВНУТРЕННИХ БОЛЕЗНЕЙ
НАУЧНЫЙ(ЫЕ) РУКОВОДИТЕЛЬ(И): Д.М.Н., ПРОФЕССОР КОБАЛАВА Ж.Д.

PREVALENCE AND PROGNOSTIC VALUE OF CONTRAST-INDUCED ACUTE KIDNEY INJURY IN PATIENTS WITH PRIMARY PERCUTANEOUS CORONARY INTERVENTION

YUSUPOV T., GASKINA A.A.

PEOPLES' FRIENDSHIP UNIVERSITY OF RUSSIA, RUSSIA, MOSCOW
THE FACULTY OF MEDICINE, DEPARTMENT OF PROPEDIATICS OF INTERNAL DISEASES
SCIENTIFIC ADVISER(S): MD, PH.D., DSc KOBALAVA Z.D.

Objective: The incidence of contrast-induced acute kidney injury (CI-AKI) is rising due to increased use of coronary angiography and percutaneous coronary intervention (PCI). Patients undergoing primary PCI are at high risk of CI-AKI, a complication that negatively affects outcomes. The aim of the study was to evaluate the incidence, predictors and outcomes of CI-AKI in patients with ST-segment elevation myocardial infarction (STEMI) and primary PCI.

Methods: 216 patients with STEMI and primary PCI (143 male, 64±13 years, weight 80±14 kg (M±SD), smoking 49%, arterial hypertension 90%, previous myocardial infarction 27%, diabetes mellitus 21%, known chronic kidney disease 7%, anemia 14%, COPD 39%, history of cancer 1.4%, history of stroke 14%, hepatic disease 4%, heart failure 62%, left ventricular ejection fraction (LV EF) 44±15%) were examined. According to the PCI patients was no damaged to the coronary arteries 1.7%, damaged to the coronary arteries <50% - 13%, damaged to the 1 or 2 coronary arteries - 29%, 3 or more - 64%, main left coronary artery disease - 10%. The median volume of contrast used was 244 ml (interquartile range: 100 to 500 ml), and the median duration of the PCI was 59 minutes (interquartile range: 10 to 120 minutes). CI-AKI was defined using 2012 KDIGO Guidelines: as an increase in serum creatinine ≥ 0.5 mg/dl above baseline within 3 days after contrast administration. Mann-Whitney test and multivariate logistic regression analysis were performed. P < 0.05 was considered statistically significant.

Results: 20% of patients developed CI-AKI. Stages 1 and 2 of CI-AKI were found in 81 and 19% of cases accordingly. CI-AKI developed in 75% of cases in first 48 hours after PCI. Patients with versus without CI-AKI were older (69±13 vs 63±12 years, p<0.05), had higher baseline serum creatinine (104±31 vs 87±22 μmol/l, p<0.001), lower LV EF (37±10 vs 41±14%, p<0.05), higher rate of CKD (21 vs 3.5%, p<0.001) and higher rate of therapy with nephrotoxic antibiotics (19 vs 3.5%, p<0.05), loop diuretics (72 vs 39%, p<0.05), mineralocorticoid receptor antagonists (56 vs 37%, p<0.05), higher volume of contrast agent (282±94 vs 236±85 ml, p<0.05), contrast media volume/estimated glomerular filtration rate ratio (CV/eGFR) (4.02±2.15 vs 2.32±1.08, p<0.05), higher rate of multivessel coronary damage (84 vs 59%, p<0.05). Main predictors of CI-AKI were CKD (OR 7.37;

95% CI 2.46-22.06; p<0.001), therapy with nephrotoxic antibiotics (OR 6.36; 95% CI 2.08-19.49; p<0.05), loop diuretics (OR 3.98; 95% CI 1.92-8.30; p<0.01), multivessel coronary damage (OR 3.58; 95% CI 1.51-8.49; p<0.05), therapy with mineralocorticoid receptor antagonists (OR 2.15; 95% CI 1.09-4.23; p<0.05).

Patients with CI-AKI had higher risk of 30-days mortality (10 vs 3%, p<0.05) and similar rate of 6 months rehospitalizations (66 vs 40%, p>0.05).

Conclusions: CI-AKI in patients with STEMI and primary PCI developed in 20% of cases, predominantly in first 48 hours after PCI. CI-AKI was associated with higher rate of CKD, therapy with nephrotoxic drugs, multivessel coronary damage, higher baseline serum creatinine and volume of contrast agent. CI-AKI had negative impact on 30-days mortality.

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THE LATE ACTIVATION TIME VARIABILITY IN ECG PREDICTS MORTALITY AND RESPONSE TO CARDIAC RESYNCHRONIZATION THERAPY

SIMON SCHLÖGL

SEMMELWEIS UNIVERSITY, BUDAPEST, HUNGARY

HEART AND VASCULAR CENTER

SCIENTIFIC ADVISER(S): GABOR SZÉPLAKI MD, PhD

ANDRÁS MIHÁLY BORDOS MD

Aims: Ventricular dyssynchrony is of key importance in the success of the resynchronization therapy of heart failure. Dyssynchrony correlates with the QRS width in ECG, which is composed of the intrinsicoid deflection (ID) time and late activation time (LAT).

We hypothesized that the variability of QRS, ID time and LAT between leads V1 and V2 would predict not only the 6-month response (at least 15% improvement in the left ventricular ejection fraction), but also the 2-year mortality of the patients.

Methods: The surface 12-lead ECGs of 127 heart failure patients were analyzed in a prospective, single-center observational study before and 6 months after CRT implantation.

Results: Up to a maximum follow-up period of 1213 days (median 787 days), 29 patients died. The responders (n=85) tended to exhibit a wider QRS and ID time before implantation [odds ratio (OR)=1.57 (1.00-2.48), p=0.05, and OR=1.47 (0.95-2.25), p=0.07]. A smaller variability of QRS and LAT proved to predict the response [OR=0.62 (0.39-0.98), p=0.04 and OR=0.64 (0.41-0.98), p=0.04]. Adjusted multivariable Cox regression analysis revealed that only the LAT variability was of predictive power as concerns the mortality [hazard ratio (HR) = 1.49 (1.09-2.04), p=0.001, per 0.5 msec increase].

The optimum cut-off value for LAT variability in the prediction of mortality and response was 4.3 msec [mortality: OR=3.82 (1.07-13.6), sensitivity=89%, specificity = 30%, p=0.03, and response: OR=7.95 (1.76-35.9), sensitivity = 92%, specificity = 37%, p=0.001].

Conclusion: The results demonstrated the usefulness of the LAT in the prediction of 2-year mortality of the patients and the 6-month response to CRT.

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КЛИНИЧЕСКИЕ МАРКЕРЫ ДЛЯ РАЗВИТИЯ ТЯЖЕЛОЙ ИЛИ ОСЛОЖНЕННОЙ МАЛЯРИИ

Мислере Р., Фризони Т., Эстефес М.

РОССИЙСКИЙ УНИВЕРСИТЕТ ДРУЖБЫ НАРОДОВ, РОССИЯ, МОСКВА

МЕДИЦИНСКИЙ ФАКУЛЬТЕТ

КАФЕДРА МИКРОБИОЛОГИИ И ВИРОЛОГИИ

НАУЧНЫЙ(ЫЕ) РУКОВОДИТЕЛЬ(И): К.Б.Н. САЧИНКИНА НАДЕЖДА ПАВЛОВНА

CLINICAL MARKERS FOR SEVERE MALARIA DEVELOPMENT

MICHELLE R., FRIZONI T., ESTEVES M.

PEOPLES' FRIENDSHIP UNIVERSITY OF RUSSIA, RUSSIA, MOSCOW

THE FACULTY OF MEDICINE

DEPARTMENT OF MICROBIOLOGY AND VIROLOGY

SCIENTIFIC ADVISER(S): CANDIDATE OF BIOLOGICAL SCIENCES SACHINKHINA NADEZHDA PAVLOVNA

Introduction: Malaria is a protozoal systematic infection caused by Plasmodium falciparum, P. vivax, P. malariae, P. ovale and P. knowlesi. Markers of severe malaria were: fever over 40° C, > 8 days or more, early hepato- and splenomegaly (from the 3rd day of clinical onset), prevalence of febrile atypia, ECG changes such as deformation of the last