Conclusion. Mutation of LGI1 gene, disruption of interaction between LGI proteins and ADAM proteins, ADAM proteins defects, lead to TLE phenotype, manifested by seizure, halucination, auditive disorders, memory disorders. At the same time the presence of antibodies anti-LGI or anti-NMDA leadt to LE, manifesting by lose of memory, iritability, headache, seizures and psychosis.

Key words: LGI, epilepsy, mutation.

274. BIOCHEMICAL DATA IN ACUTE MYOCARDIAL INFARCTION.

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Introduction: Acute myocardial infarction (AMI) is one of the most usual diagnosis in hospitalized patients. Hyperglycemia, hypertension, and hypercholesterolemia evaluated on admission in patients with AMI are considered negative predictors of short- and long-term clinical outcomes.

Aim: We performed statistical analyses to identify correlations between biochemical parameters in patients with AMI Associated with hypertension stage II/III.

Materials and methods: Our study was performed on 33 patients with AMI admitted to the Intensive Care Unit of the Public Institution Institute of Cardiology. Patients were divided into three groups: L1- AMI Associated with hypertension stage II (n=13); L2- AMI Associated with hypertension stage III (n=8); L3- sham AMI (n=12). On admission in all the patients were evaluated plasma levels of cholesterol, LDL and HDL cholesterol, triglycerides (TAG), and glucose. The obtained data were represented by median and percentiles. For comparison the Mann Whitney and Kruskal-Wallis nonparametric tests were performed using SPSS statistical program.

Discussion results: Statistically significant differences were found in parameters of age (χ26.901 df=2 p=0.032) and TAG (χ26.559 df=2 p=0.038). The age of patients in L1 was lower (median 60.0) compared to L2 (median 65.0, Mann-Whitney U=32.0, p=0.161), but higher than in L3 (median 55.0, Mann-Whitney U=16.5, p=0.012). TAG value was higher in L1 (median 2.24) compared to L2 (median 1.35, Mann-Whitney U=22.5, p=0.03) and L3 (median 1.37, Mann-Whitney U=46.5, p=0.91). We noticed a slight difference in value of glucose (χ24.828 df=2 p=0.038): it was lower in L1 (median 6.8) compared to L2 (median 11.2, Mann-Whitney U=27.0, p=0.076) and L3 (median 7.1, Mann-Whitney U=21.0, p=0.039). The investigated groups showed no statistically significant differences in cholesterol value (L1 median 5.5; L2 median 5.35; L3 median 5.3; Kruskal-Wallis test χ20.688 df=2 p=0.709), LDL-cholesterol (L1 median 3.02; L2 median 3.4; L3 median 3.0, Kruskal-Wallis test χ21.373 df=2 p=0.503), HDL-cholesterol (L1 median 1.3; L2 median 1.28; L3 median 1.27, Kruskal-Wallis test χ21.462 df=2 p=0.481).

Conclusion: Atherosclerosis is main cause of AMI. Hypertension and hyperglycemia after acute coronary syndrome are Associated with an increased risk of in-hospital mortality and severe complications. The major plasma lipid traits, low-density lipoprotein cholesterol (LDL-C), triglycerides,

and high-density lipoprotein cholesterol (HDL-C) are all predictive of cardiovascular risk and are considered targets for therapeutic intervention.

Key Words: AMI, hypertension, dislipidemia, hyperglycemia.

275. LEUKEMIA CAUSED BY CHIMERIC ONCOGENES

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Introduction: The involving of chimeric oncogenes in the molecular-genetic mechanism of leukemia's occurrence is currently discussed a lot. Their activity is explained by the transformation of the hematopoietic cells into leukemic cells using different kind of genetic disorders. It involves the disruption of a normal survival, proliferation and differentiation of the hematopoietic's progenitors. As an example of these chimeric oncogenes is the BCR-ABL gene, which is responsable for the creation of an abnormal protein kinase and is has been proved that almost 95% of Chronic Myeloid Leukemia patients have this gene in their leukemic cells. Therefore, it is important to realise that medical examination of patients with hematologic malignancies should involve cytogenetic technics (RT-PCR qualitative or quantitative, FISH) as an essential method of diagnostic as they play a major role in establishing a more targeted treatement.

Materials and methods: The current study includes 704 pacients followed at the CHU Amiens, France with a suspicion for Chronic Myeloid Leukemia during 2012-2015. Their diagnostic was put based on their blood test, myelogram analyze and finally by the RT-PCR qualitative method, which played the most precise role in establishing the disease. The statistical used method is the descriptive one, since we made our study based on their medical records and their results.

Discussion results: After making this study, we have obtained the following data: it included 374 men (53%) and 330 women (47%). The average age when this investigation method was applied is 62.67 years, including 63.14 years for males and 62.15 years for females. 109 patients (15.42%) have presented a positive diagnostic: 96 patients (13.63%) had a M-BCR-ABL transcript and 13 patients (1.84%) had a m-BCR-ABL transcript. In 591 cases (83.94%) the BCR-ABL trasncript was absent, but in 4 cases (0.56%) the transcript could not be identified because of the extracted ARN's bad quality.

Conclusion: In the end, after analysing this study's results, we can conclude that most part of leukemias can be certainly confirmed by using RT-PCR. For the establishment of a leukemia diagnostic we use RT-PCR qualitative, but for the disease's evolution we practice RT-PCR quantitative. Also we have observed the role of BCR-ABL oncogene in Chronic Myeloid Leukemia's etiology and the variety of regions where chromosomal translocation may occur. Therefore using molecular-genetic techniques in the diagnosis of leukemia has a fundamental significance for the development of a targeted treatment.

Key words: Chimeric oncogenes, BCR-ABL, RT-PCR.