

Platelet glutamate dehydrogenase as a biomarker for prediction of antipsychotic therapy efficacy in patients with endogenous psychosis

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Introduction: Individual prognosis of antipsychotic therapy efficacy in patients with endogenous psychosis remains an unsolved problem, and no reliable biological or clinical markers are proposed still for development of approach to elevation of individual antipsychotic therapy efficacy.

Methods: Glutamate dehydrogenase (GDH) enzymatic activity was determined in platelets of patients with endogenous psychosis (diagnosed as Schizophrenia (n=48) or Schizoaffective disorder (n=21) in comparison with control group (n=34), and links were searched between their platelet GDH activity and clinical psychopathological condition.

Results: Generally, baseline GDH activity in patients was significantly lower, than in the control group (p<0,001, Mann-Whitney U-test). Significant differences were revealed in baseline GDH activity between subgroups of patents with first episode psychosis (FEP, n=34), chronic patients (n=35) and control group (p<0,002, Kruskal-Wallis test), wherein baseline GDH activity correlated with PANSS assessed before the treatment course in FEP patients: PANSStot (Spearman R= - 0,44, p<0,008), PANSSneg (R= - 0,48, p<0,003) and PANSSpsy (R= -0,37, p<0,03). No links were found in patients with chronic schizophrenia. Besides, significant links between baseline GDH activity

and PANSS scores after the treatment were found in FEP patients: the higher were levels of GDH activity, the lower were their PANSS scores after the treatment: PANSStot (R=-0.39, p<0.04), PANSS neg (R=-0.39, p<0.04) and PANSSpsy (R=-0.37, p<0.04).

Conclusion: Data on initial (baseline) levels of platelet GDH activity can help to develop individual prognosis of antipsychotic pharmacotherapy efficacy in patients with FEP.

