Other

INTERLEUKINS SERUM LEVELS AND MITOCHONDRIAL DYSFUNCTION IN THE POST-INJURY PERIOD FOLLOWING TRAUMATIC BRAIN INJURY

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Mitochondrial dysfunction and subsequent neuron energy failure play a key role in pathogenesis of the post-injury period following traumatic brain injury (post-TBI).

Objective: To identify lactate, lactate dehydrogenase, pyruvate plasma levels in the post-TBI in relation to proinflammatory interleukins (IL-6, IL-8) involved into microglial-neuronal interactions and latent neuroinflammation.

Patients and Methods: Eighty-four patients at post-TBI (men age±SD 36.3±11.08 years) were investigated; plasma lactate and pyruvate levels were measured by spectrophotometric method according to manufacturer protocols; ILs were measured in sera by standard immunoenzyme assays using Vector Best reagents.

Results: The highest plasma lactate levels in the patients with post-TBI was 0.92±0.04 and 0.7±0.02 mmol/L in controls (p0.05). Lactate data did not distinguish with the respect to gender and age. These changes reflecting oxidative dysmetabolism were depended on the disease duration more than 15 years (p0.05). Post-TBI has revealed higher serum proinflamatory IL-6 levels suggesting that increased IL-6 (Mean 21.9±4.6, range: 11-26.5 vs. HC 3.52±1.38 pg/ml) might be generated from activation of cytokine-astrocytes network (p0.01; t=3.1); high IL-6 data were associated with long-course and microglia-astrocyte activation could be pronounced as a potential cause of higher interleukin data. The results showed no significant differences in pyruvate, lactate dehydrogenase and IL-8 levels.

Conclusions: This study shows data, revealing higher lactate levels in the post-TBI reflecting post-injury oxidative dysmetabolism. Post-TBI revealed higher baseline proinflammatory IL-6 cytokine levels which may play role in chronic course. IL-6–mediated neuroinflammatory processes are important of clinical understanding of the post-TBI there maybe potential role for anti-inflammatory therapy targeting cytokines.