Oncology of the chest

The effect of mitochondrial respiratory system in peripheral blood mononuclear cells on cognition during the course of lung cancer

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Background. Peripheral blood mononuclear cells (PBMC) represent easy available population of cells for the studies on remote effects of lung cancer. NADH dehydrogenase (ubiquinone)Fe-S protein-1 (Ndufs1) transfers electrons from NADH to respiratory system. Mitochondrially encoded cytochrome c oxidase I (MT-CO1) couples oxygen reduction and proton pumping. Mitochondrial dysfunction may be crucial pathomechanism leading to neurological deficits, including cognitive impairment.

Material and methods. The study involved 80 consecutive lung cancer patients hospitalized in Wielkopolska Center of Pulmonology and Thoracosurgery and Chair and Clinic of Oncology. Ndufs1 and MTCO1 expression in PBMC was evaluated by means of ELISA. Mini-Mental State Examination (MMSE), Trail Making Test (TMT-A and B) were performed at baseline and after 6 months. Sera were tested for onconeural antibodies with indirect immunofluorescence and Line blot (EUROIMMUN, Germany).

Results. Stimulation of Ndufs1 in PBMC relates to impaired performance of TMT-A (13.61±3.13s) and TMT-B (162.48±46.40s) compared to lowered Ndufs-1 cases (8.60±4.51s; P=0.003 and 124.78±51.77s;P<0.05, respectively). The correlations between Ndufs1 expression and MMSE after 6 months follow-up (tau=-0.310;P=0.0236), TMT-A (tau=0.301;P=0.0001) and TMT-B (tau=0.199;P=0.0120) at baseline were found. Higher MTCO1 expression accompany worse TMT-A results (11.05±5.81s) than in inhibited MTCO1 (8.52±4.14s;P=0.048). MTCO1 expression correlated with TMT-A results (tau=0.167;P=0.0344) at baseline. No association between Ndufs1 and MTCO1 expression and onconeral antibodies production was found.

Conclusion. Stimulation of PBMC mitochondrial function in lung cancer patients is associated with cognitive impairment. Mitochondrial dysfunction in PBMC may reflect cytotoxic responsible for neurological deficits.

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