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Background: POCD is actual problem nowadays.

Objective: The study aim was assessment of association of patient cognitive dysfunction and systemic inflammatory markers and matrix metalloproteinase (MMP-2, MMP-9) serum levels after coronary artery bypass grafting (CABG).

Patients and Methods/Material and Methods: Seventy patients (age 61,6 ± 6,7) with ischemic heart disease scheduled for CABG were included. They were divided by gender and age into 2 groups based on surgery type (42 on-pump, 28 off-pump groups). Cognitive level decline was evaluated by neuropsychological tests.

Results: Concentration of cytokines (IL-5, IL-8, IL-9, IL-13, IL-15, Eotaxin, FGF-2, G-CSF, PDGF-bb, VEGF), MMP-2 and MMP-9 was evaluated a day before (T0) and within 2 hours after surgery (T1). The most considerable and statistically significant changes were in T1 point in on-pump and off-pump groups, where IL-8, VEGF, Eotaxin and MMP9 levels increased. PDGF-bb, G-CSF and IL-13 levels statistically significant increased only in on-pump group. Comparing on-pump and off- pump IL-8, MMP9, Eotaxin growth level was statistically more significant in on- pump group. Dependence between cognitive decline on the 7th day and plasma biomarkers levels in 2 hours after surgery was revealed for IL-5 ($r = 0{,}348$, $p = 0{,}05$), IL-8 ($r = 0{,}278$, $p = 0{,}05$) and MMP9 ($r = 0{,}549$, $p = 0{,}01$). Dependence between cognitive decline and levels of VEGF ($r = 0{,}398$, $p = 0{,}05$) and G-CSF ($r = 0{,}337$, $p = 0{,}05$) was discovered only for on-pump group. We found out dependence of MMP-2 serum level (before and after surgery) and cognitive decline on the 7th day after surgery ($r = 0{,}598$, $p = 0{,}01$).

Conclusion: The systemic inflammatory reaction occurs after CABG accompanied by cytokines, MMP-2 and MMP-9 levels elevation, that correlates with cognitive dysfunction.

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SHIFT 6 - DEMENTIA

The renal cognitive impairment: The association of CKD and cognitive impairment

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Background: Higher risk of cognitive impairment (CI) in CKD has been generally explained by influence of SVD.

Objective: We determine whether CKD is associated with CI independent of SVD burden in non-stroke patients.

Patients and Methods/Material and Methods: We enrolled consecutive patients without dementia or stroke who underwent detailed neuropsychological tests and brain MRI. Renal dysfunction was classified as no CKD (GFR≥60), mild CKD (45≤GFR<60) and moderate to severe CKD (15≤GFR<45). And we used 7, 4, 9 and 6-point visual rating scale of brain MRI to evaluate severity of SVD, MTA, cortical and subcortical atrophy respectively. We performed a linear regression analysis to assess correlation of CKD and CI adjusting for age, sex, educational level, vascular risk factor and MRI findings.

Results: A total of 446 patients were included (mean age 75 ± 5.4 years, 33% male, 7.5 ± 5.2 years of education) and CKD was present in 87 (55 mild, 32 moderate to severe). Cognitive decline was correlated with severity of SVD and brain atrophy. In group analysis, moderate to severe CKD was associated with decreased language domain function in models adjusted for age, sex, and level of education. The associations of moderate CKD with language dysfunction remained significant even after adjusting for SVD and brain atrophy in comparison with no CKD (95% CI -1.52, -0.14) or mild CDK group (95% CI -1.92, -0.10)

Conclusion: CI in CKD may be independent of underlying SVD or brain atrophy. These results may suggest the hypothetical concept of "renal cognitive impairment", which need to be validated by further larger prospective study.

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SHIFT 6 - DEMENTIA

Genetic heterogeneity in Alzheimer's disease derived from five brain regions suggest new counteractions

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Background: The implementation of personalized medicine in the prevention of Alzheimer's disease (AD) depends on understanding subtypes of AD. Recent reports suggest that diagnosis the clinical onset of AD may be brain region specific.

Objective: Here, in order to clarify possible differences in regulatory patterns in brain regions in AD, we studied public gene expression microarray data from five brain regions–entorhinal cortex (EC), hippocampus (HC), medial temporal gyrus (MTG), posterior cingulate (PC), and superior frontal gyrus (SFG)–of AD patients and controls.

Patients and Methods/Material and Methods: We used the method of differential expression genes (DEG), gene set enrichment analysis (GSEA) and miRNA analysis in this study. Five region-specific gene-gene interaction networks (GGNs) were also constructed from gene pairs that were differentially co-expressed and had known protein-protein interactions.

Results: DEG analysis indicated that up-regulated islets/type 2 diabetes (T2D) and down-regulated HC/AD DEGs have significant overlaps (Fisher's exact test, $p < 2 \times 10^{-256}$), and this was corroborated in GSEA analysis. The 1,660 DEGs common to HC/AD and T2D are significantly enriched in oxidative phosphorylation and metabolic process related pathways, and have significant connections to four miRNAs-targets: hsa-mir-29a (CFH), hsa-mir-144 (IRS1), hsa-mir-29a (NAV3) and hsa-mir-195 (BACE1). GGNs results showed that enriched pathways depended on region. For instance, cancer pathways were seen to be highly enriched PC/AD, but were absent in HC/AD.

Conclusion: Our results highlighted the heterogeneity of AD in the five brain regions, and indicated a possible AD-T2D connection. These may be useful in devising strategies for the early detection of AD as well as T2D.

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