Effect of CKD on behavioral and recognition in the hippocampus

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Objectives: CKD is either occurring naturally in humans or induced surgically in rat causes alterations in behavior and motor functions. Clinical studies have demonstrated a high risk for dementia and cognitive impairment in patients with CKD. Moreover, the evidence in animal studies indicates that uremia impairs synaptic transmission in the rat hippocampus. However, the effect of association to behavior, neuropathological and perturb synaptic function in the CKD is imprecision. Therefore in this study, we are identifying the mechanisms that psychomotor functional disorders by renal and synaptic malfunction at the CKD.

Methods: SD rats undergone 5/6 nephrectomy. At 4, 10 week after 5/6 nephrectomy, cognitvie function were investigated.

Results: we were confirmed body weight and creatinine concentration in CKD models. Furthermore to characterize of behavioral disturbance by cognitive dysfunction, we were experimented behavior test. Compared to control, the CKD group was markedly suppressed locomotor activity and enhanced anxiety. And then working memory was similarly to control but, special working memory of hippocampus dependent was decreased then control and cross-check results of behavior test through llocal field potential (LFP). In addition, we were investigated field excitatory postsynaptic potential (fEPSP) in the hippocampus for identifying impairs synaptic transmission, observed that resultant slope of fEPSP was markedly reduced more than control level. Morphological investigations in the hippocampus showed sclerotic features of glomeruli by PAS stain and reducing of interneurons by cresyl violet staining at CKD group. Additionally, immunoreactivity of glial fibrillary acidic protein (GFAP) was enhanced that soma and proximal process compared with control.

Conclusions: Our study suggest that elevations in uremic toxin levels functionally perturb synaptic function of the hippocampus and leads to neuronal cell death and subsequent cognitive dysfunction.