## **OP 12**

## Upregulation of the olfactomedin 4 gene enables the disease progression in chronic kidney disease patients

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**Background**: Olfactomedin 4 is a glycoprotein encoded by the gene, OLFM4 and found in various tissues including pancreatic  $\beta$ -cells. It involves various pathways of innate immunity, inflammation, and malignancies. However, the clinical significance of its expression in Chronic Kidney Disease (CKD) was not studied so far.

**Objective**: To identify the *OLFM4* gene expression in CKD patients with comorbidities of diabetes (DM), hypertension (HT), and other causes (O) and its association with disease progression.

**Methods & Materials**: Total RNA was extracted from the urine samples of the study population (n=43): CKD + DM (n=8); CKD + HT (n=16); CKD + DM and HT (n=7); CKD + O (n=6), and healthy volunteers (n=6). Reverse transcription-quantitative polymerase chain reaction was carried out using gene-specific primers. Normalization was carried out using reference gene, β-2 microglobulin, and Fold Changes (FC) were calculated using the relative quantification method. *OLFM4* expression in early (n=15) and late-stage of CKD (n=23) was also analyzed irrespective of the etiology. Log2normalized fold changes were compared with existing biomarkers: serum creatinine and estimated Glomerular Filtration Rate (eGFR) of CKD.

**Results**: OLFM4 gene was upregulated at  $10.47\pm2.62$ -fold in CKD patients while the highest upregulation was found in CKD patients with both DM and HT (FC: 12.26) compared to other study categories. However, the upregulation of OLFM4 was significantly high in the late-stage of CKD than the early-stage (p<0.05). OLFM4 expression in CKD subjects poorly correlated with serum creatinine (r=0.22; p>0.05) and eGFR (r=0.015; p>0.05).

**Conclusion:** Both diabetes and hypertension enhance the expression of the *OLFM4* gene and it could be used as a candidate biomarker for CKD progression. However further validation is required with more samples before clinical application.