CONCLUSIONS: In conclusion, this study suggests that not only patients with low eGFR decline, but also those with preserved renal function (Group B) may come early in the course of ADPKD and could be a cause rather than a result of disease progression and dysfunction. These results could lead to the hypothesis that local microcirculatory changes indicative for endothelial dysfunction, angiogenesis and hypoxia in comparison to ADPKD patients, we noted strong correlations of all studied markers with ADMA (nM), angiopoietin-2 (ng/ml), and HIF-1α (endothelial cell-specific molecule-1) angiopoietin-2, and hypoxia-induced-factor-1α (HIF-1α) were determined by ELISA techniques.

RESULTS: Patients in Group A had significantly higher levels of endocan (7.17 ng/ml), angiopoietin-2 (5.595.43 ng/ml), and HIF-1α (163.68 ng/ml, p<0.001, angiopoietin2 r²=0.69, Angiopoietin-1 r²=0.59 ng/ml, p<0.018, HIF-1α r²=0.69, respectively) or controls (4.83 ng/ml, p<0.001, angiopoietin2 r²=0.70, Angiopoietin-1 r²=0.69, HIF-1α r²=0.69, respectively). There was a significant positive correlation between MBPS and LVMI (r²=0.375 respectively). In an ordered logistic regression model adjusted for age, gender, and BMI, ADPKD patients had significantly higher odds of high LVMI (OR 6.4, p<0.001). The LVMI and CIMT were significantly higher in patients with ADPKD (108±2.5 vs 94±2.0, p=0.003 and 6.8±2.3 vs 3±1.1, p=0.001, respectively). There was a significant positive correlation between MBPS and LVMI in ADPKD group but not in controls (figure). FMD% was significantly diminished in ADPKD (16.6±6.4 vs 16.6±6.4, p=0.001). In ADPKD group, the LVMI, CIMT, hsCRP, and microalbuminuria were significantly higher in MBPS (+) cases than MBPS (-) cases. However, there were no significant differences in
these parameters between MBPS (+) and MBPS (-) cases in control group. In the linear regression model, hsCRP, BMI and MBPS with uric acid were main determinants of LVH in ADPKD patients ($R^2=0.641$, $\beta=0.621$ and $p<0.001$).

CONCLUSIONS: In conclusion, MBPS is one of the most important determinants of LVM, ED and early atherosclerosis in early ADPKD patients. Reduction of the MBPS may be a new therapeutic target for subsequent cardiovascular events in ADPKD patients.