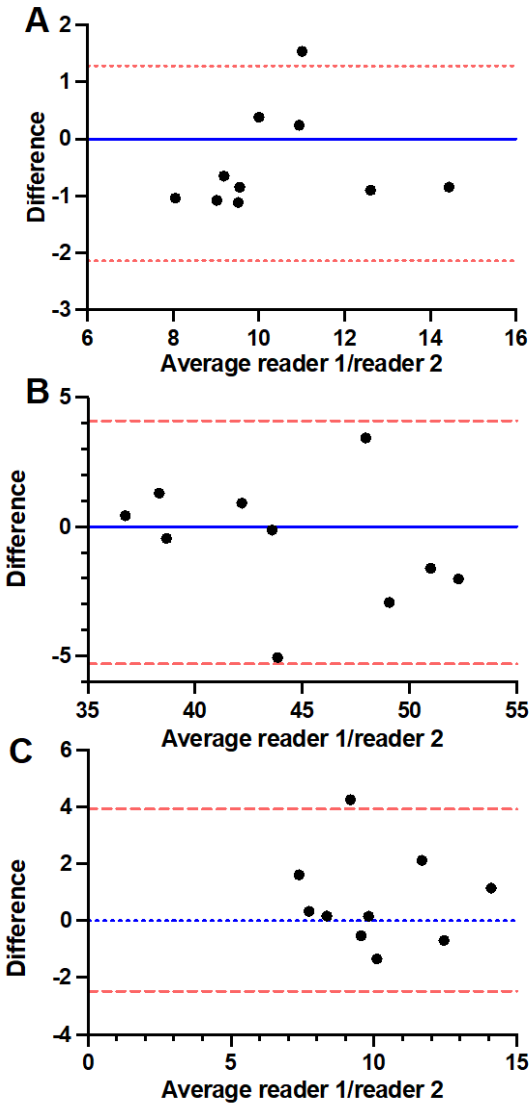
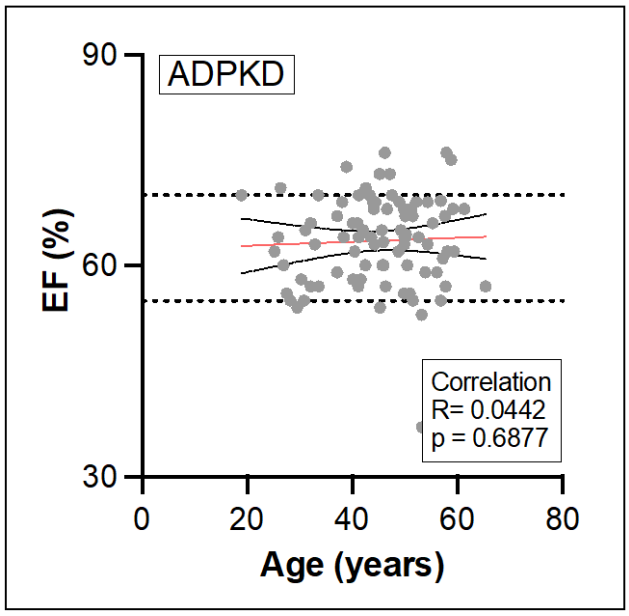
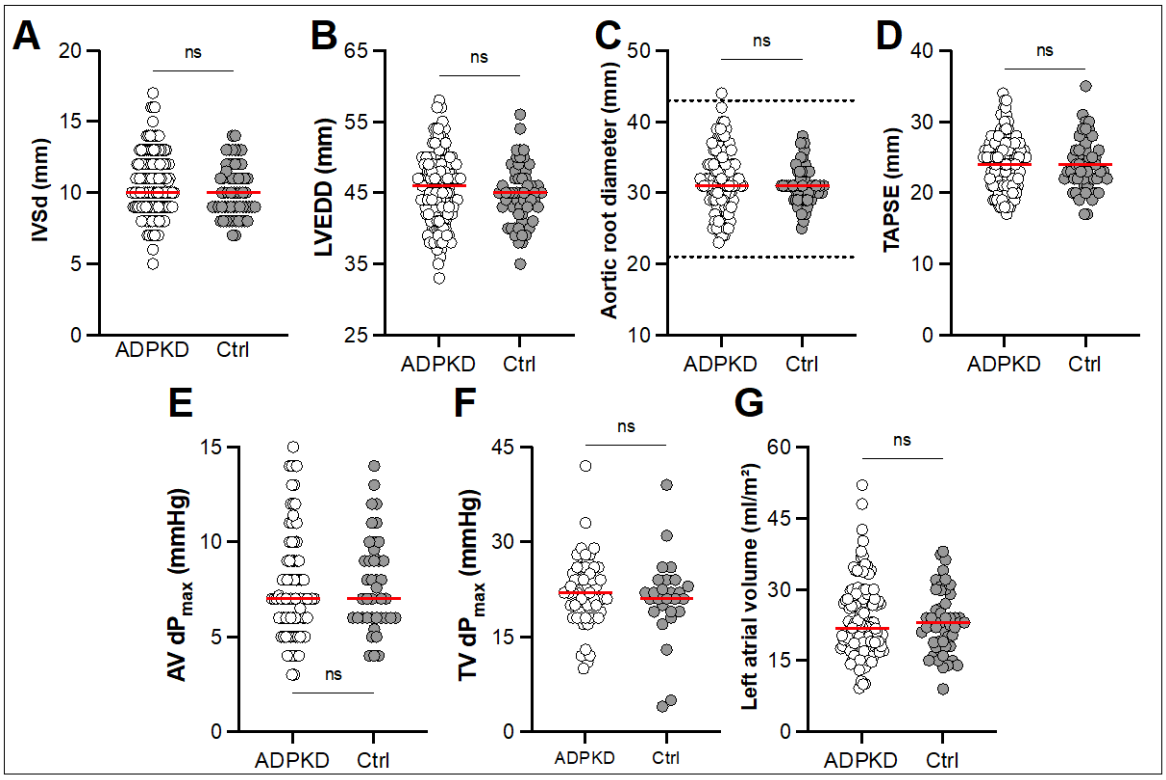
Supplemental Section



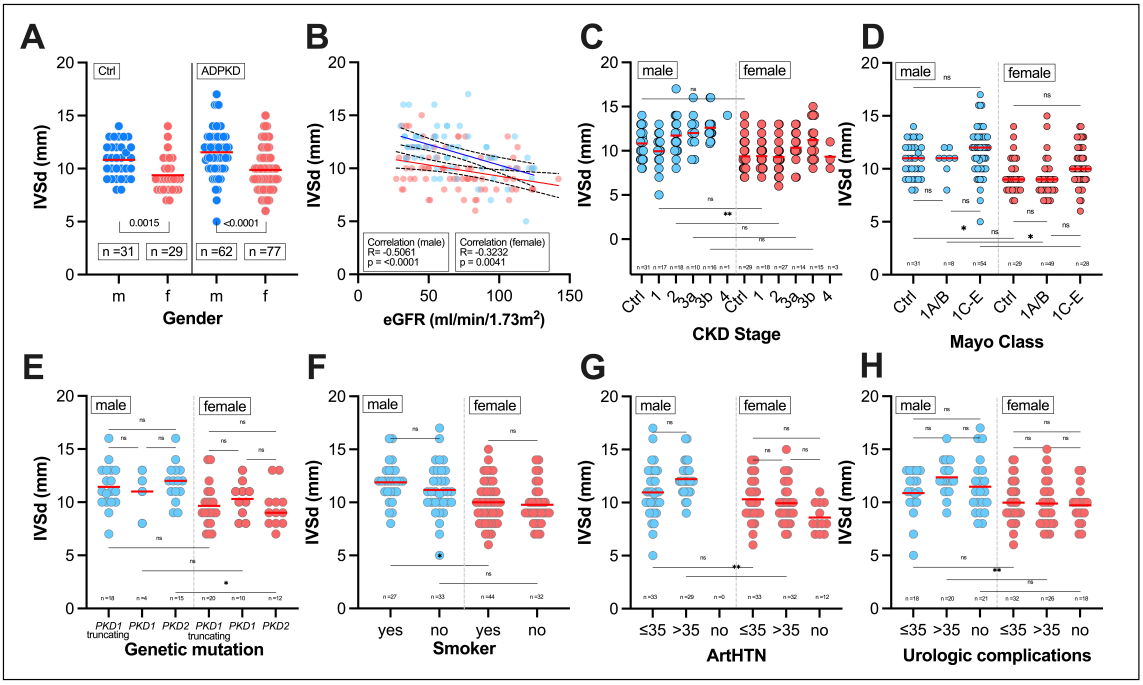
Supplemental Figure 1: Bland-Altman plot for interpretation of inter-observer reproducibility.
A) IVSd (Control) B) LVEDD C) IVSd (ADPKD)



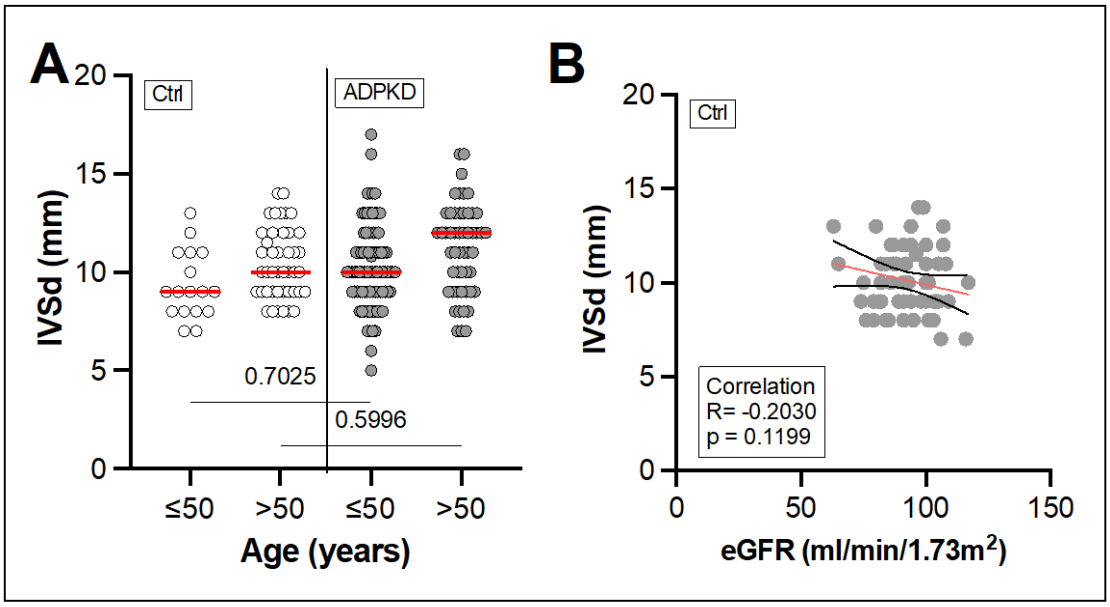
Supplemental Figure 2 Correlation of ejection fraction and age of ADPKD patients. *Linear regression is denoted by a red line, while the 95% confidence interval is denoted by dotted lines.*



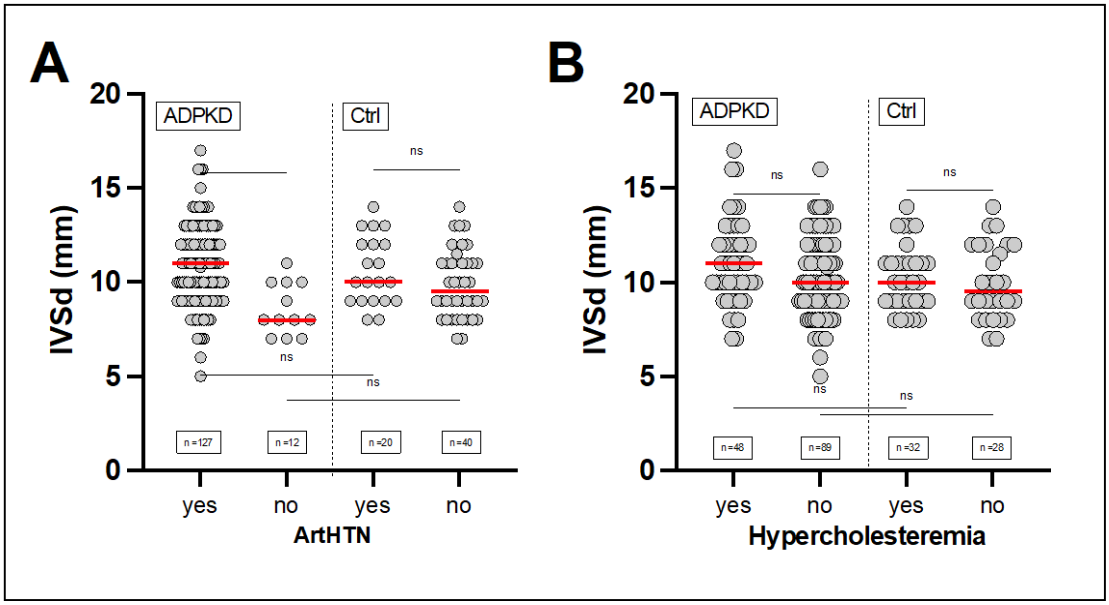
Supplemental Figure 3 Comparison of echocardiographic parameters of ADPKD patients versus controls. *Mean values are indicated as red line. IvSD = end-diastolic left ventricular septal wall thickness, LVEDD = left ventricular end-diastolic diameter, TAPSE = tricuspid annular plane systolic excursion, AV = aortic valve, TV = tricuspid valve*



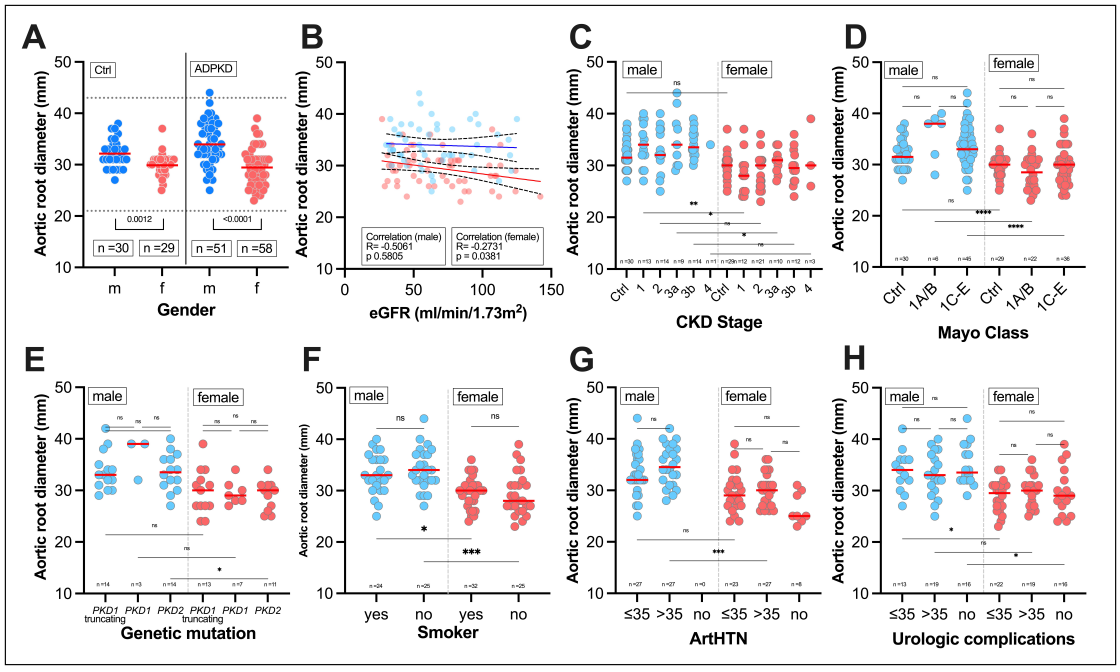
Supplemental Figure 4 Association of interventricular septal wall thickness (IVSd) with clinical characteristics among male and female ADPKD patients and controls *A) Gender distribution of IVSd in ADPKD patients (Mean±SD: male 11.55±2.18 mm and female 9.88±1.97 mm) and controls (Mean±SD: male 10.82±1.65 mm and female 9.38±1.70 mm) B) Spearman correlation of IVSd and eGFR in ADPKD patients. C) IVSd according to CKD stages 1-4 in ADPKD patients versus controls D) IVSd according to Mayo classification versus 1C/D/E in ADPKD patients versus controls. E) IVSd in ADPKD patients among respective gene products (PKD1 and PKD2). Truncating mutations included non-sense, frameshift, splicing mutations, and large rearrangements, while non-truncating mutations included missense mutations and in-frame short deletions and insertions. F-H) IVSd according to smokers and relevant comorbidities. Horizontal red lines indicate mean values. ArtHTN indicates arterial hypertension, ns indicates not significant.*



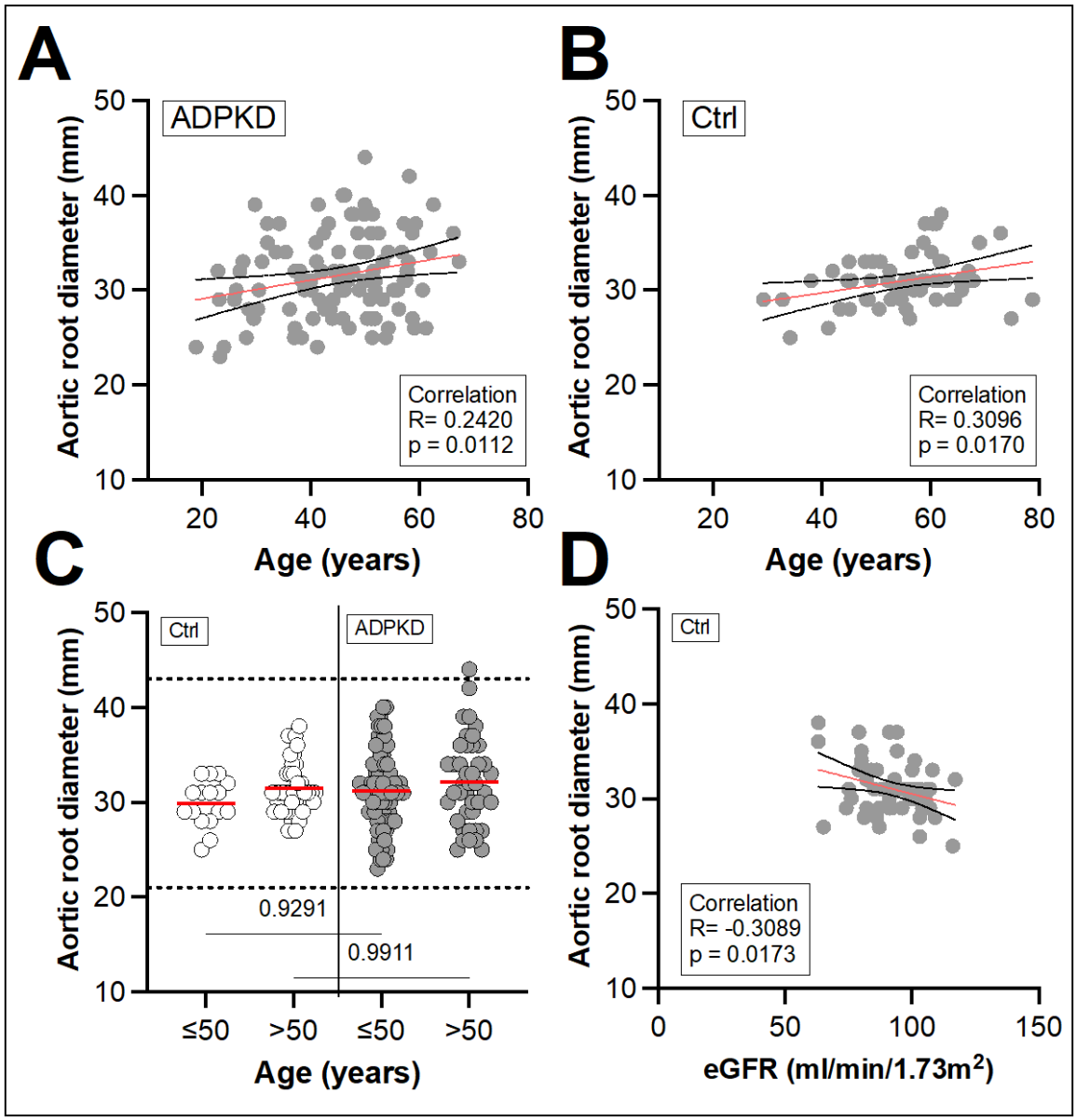
Supplemental Figure 5 Association of left ventricular septum thickness (IVSd) with age (A) and eGFR (B) in ADPKD patients (A) and controls (A,B). *The red lines indicate mean value±SD for control (≤50 9.35±1.73 mm, >50 10.43±1.77 mm) and ADPKD patients (≤50 10.27±2.13 mm, >50 11.21±2.25 mm).*



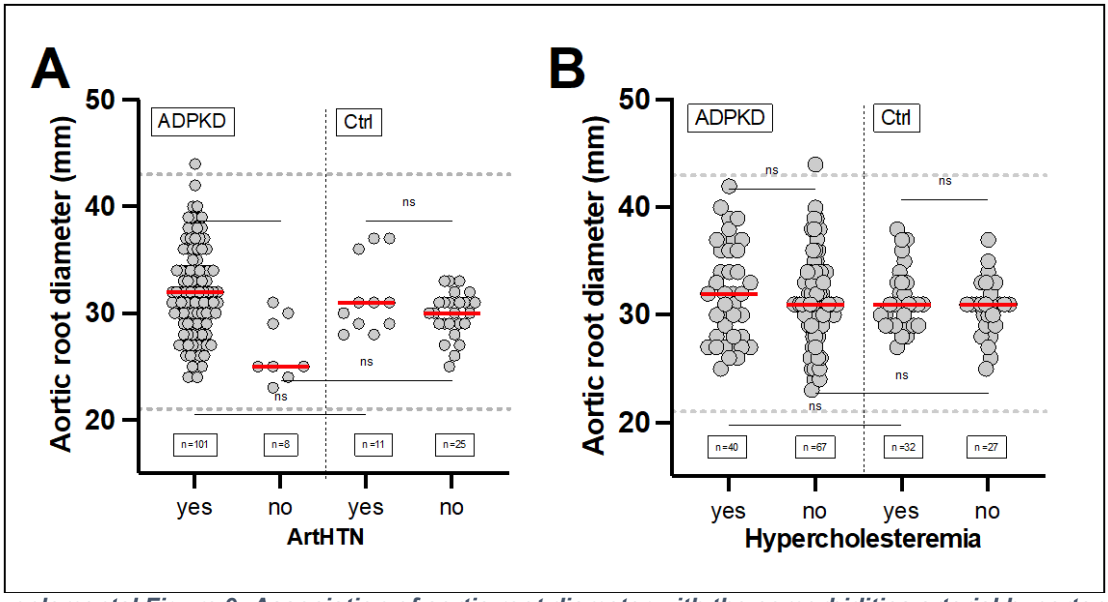
Supplemental Figure 6: Association of IVSd with the comorbidities arterial hypertension (A) and hypercholesteremia (B) for ADPKD patients and controls.



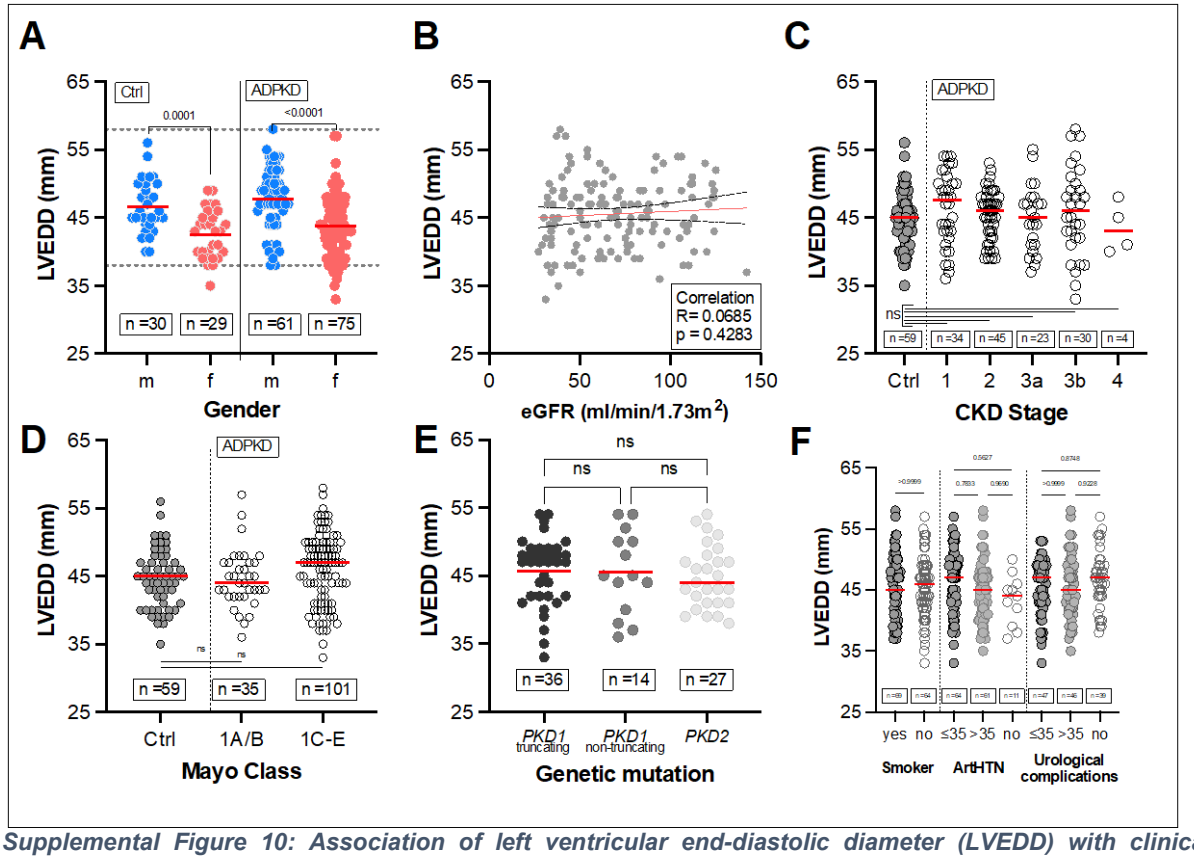
Supplemental Figure 7 Association *of aortic root diameter with clinical characteristics among male and female ADPKD patients and controls* A) Gender distribution of aortic root diameter in ADPKD patients (*Mean±SD: male 33.96±4.06 mm and female 29.45±3.52 mm*) and controls (*Mean±SD: male 32.13±2.75 mm and female 29.90±2.42 mm*) B) Spearman correlation of aortic root diameter and eGFR in ADPKD patients. C) Aortic root diameter according to CKD stages 1-4 in ADPKD patients versus controls D) Aortic root diameter according to Mayo classification versus 1C/D/E in ADPKD patients versus controls. E) Aortic root diameter in ADPKD patients among respective gene products (*PKD1* and *PKD2*). Truncating mutations included non-sense, frameshift, splicing mutations, and large rearrangements, while non-truncating mutations included missense mutations and in-frame short deletions and insertions. F-H) Aortic root diameter according to smokers and relevant comorbidities. Horizontal red lines indicate mean values. ArtHTN indicates arterial hypertension, ns indicates not significant.



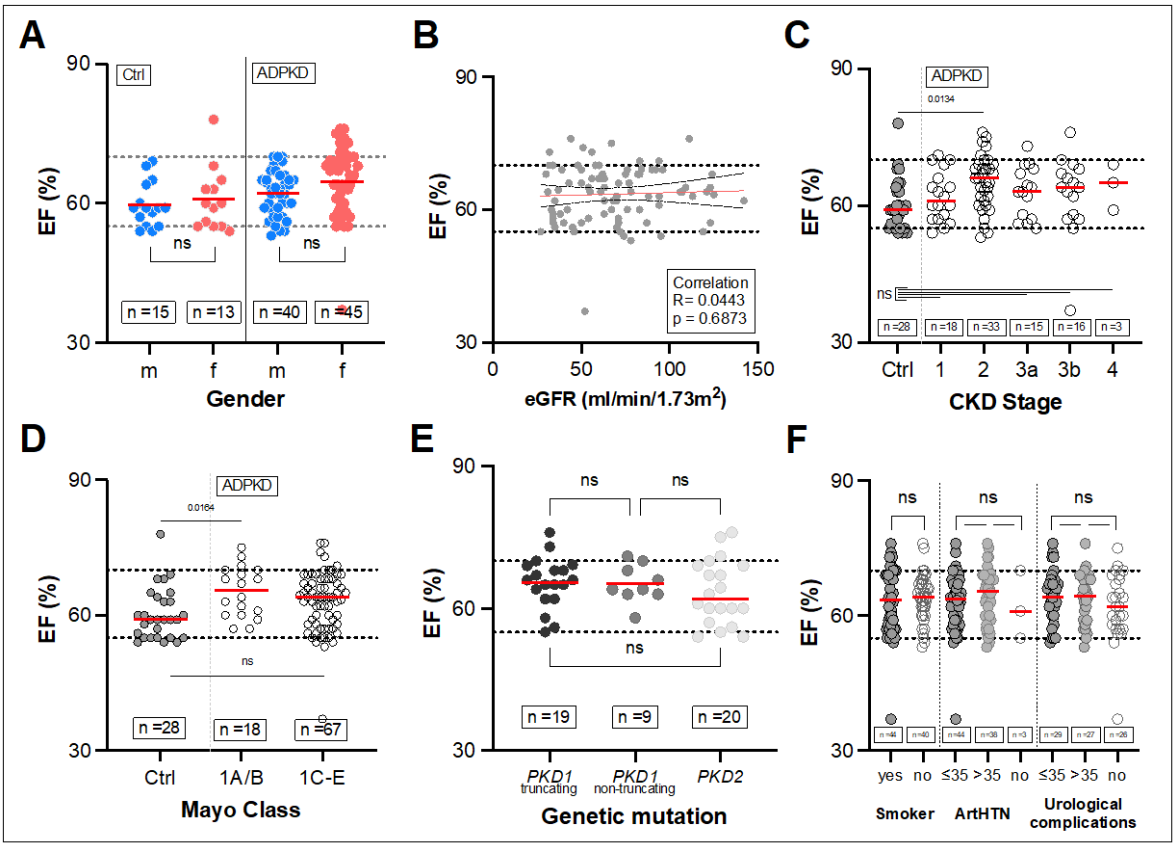
Supplemental Figure 8 Association of aortic root diameter and age (A-C) and eGFR (D) in ADPKD patients and controls. *Correlation of aortic root diameter with age in ADPKD patients (A) and controls (B). Linear regression is denoted by a red line, while the 95 percent confidence interval is denoted by dotted lines. C) Aortic root diameter among different age groups in ADPKD patients and controls. The red lines indicate mean value for control (≤50 29.88±2.42 mm, >50 31.47±2.75 mm) and ADPKD patients (≤50 31.18±4.24, >50 32.17±4.62 mm). D) Correlation of aortic root diameter with eGFR. Linear regression is denoted by a red line, while the 95 percent confidence interval is denoted by dotted lines.*



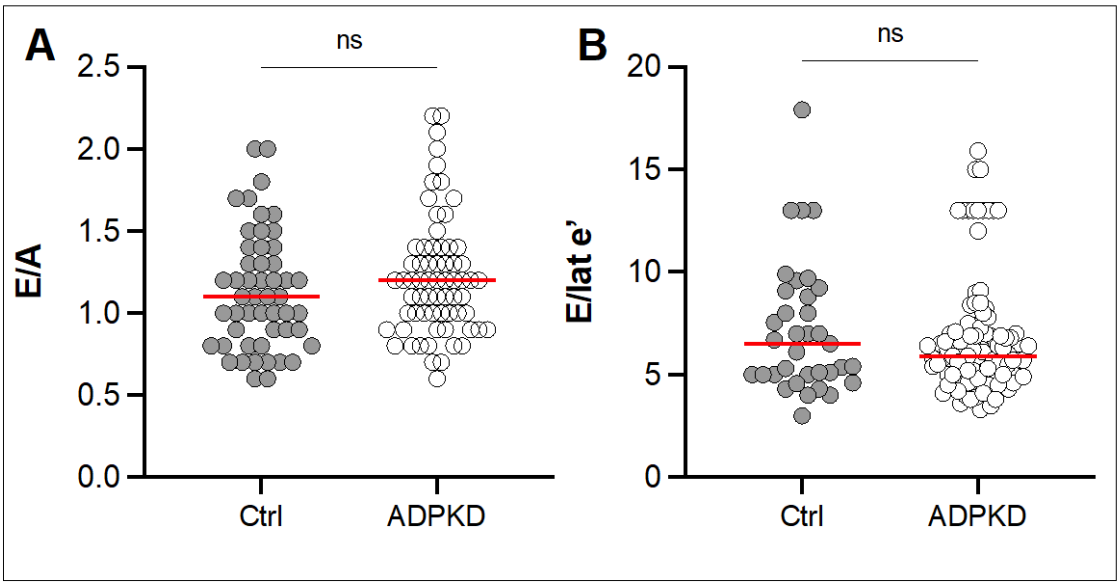
Supplemental Figure 9: Association of aortic root diameter with the comorbidities arterial hypertension (A) and hypercholesteremia (B) for ADPKD patients and controls.



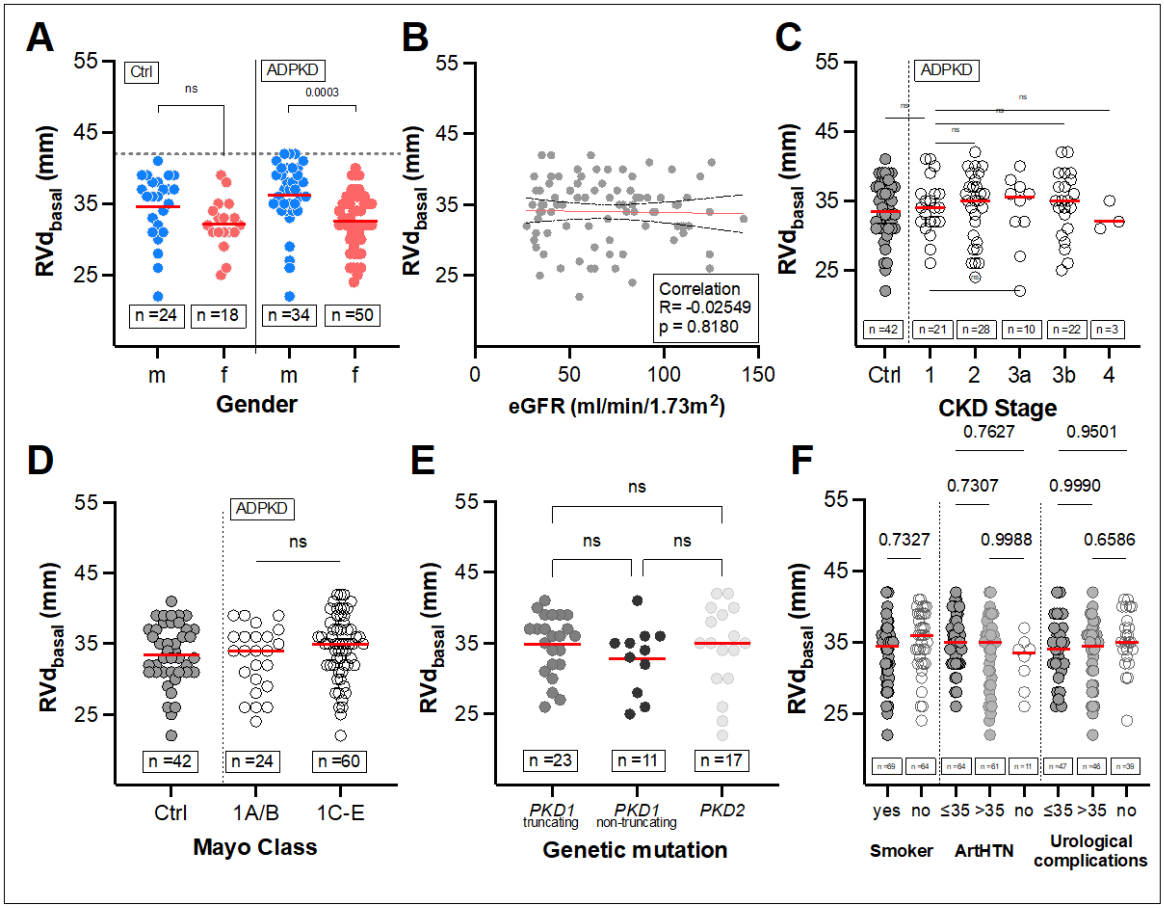
Supplemental Figure 10: Association of left ventricular end-diastolic diameter (LVEDD) with clinical characteristics *A) Gender distribution of left ventricular end-diastolic diameter in ADPKD patients* (Mean±SD: male 47.82±4.55 mm and female 43.76±4.66 mm) and controls (Mean±SD: male 46.60±3.87 mm and female 42.59±3.62 mm) *by gender B) Correlation of left ventricular end-diastolic diameter and eGFR in ADPKD patients. C) Left ventricular end-diastolic diameter according to CKD stages 1-4 in ADPKD patients versus controls. D) Left ventricular end-diastolic diameter according to Mayo classification (1A/B versus 1C/D/E) in ADPKD patients versus controls. E) Left ventricular end-diastolic diameter in ADPKD patients among respective gene products (PKD1 and PKD2). Truncating mutations included non-sense, frameshift, splicing mutations, and large rearrangements, while non-truncating mutations included missense mutations and in-frame short deletions and insertions. F) Left ventricular end-diastolic diameter according to relevant comorbidities.* Horizontal red lines indicate mean values. ArtHTN indicates arterial hypertension, ns indicates not significant



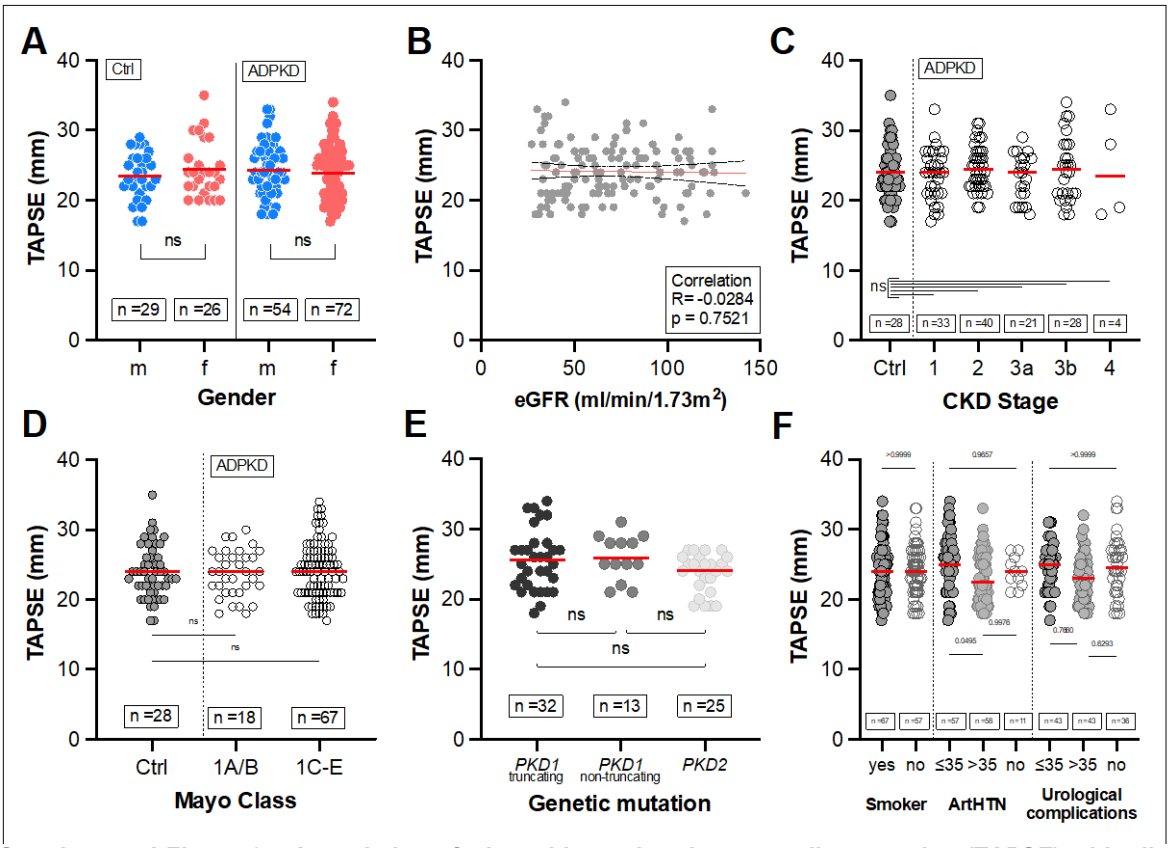
Supplemental Figure 11: Association of ejection fraction (EF) with clinical characteristics *A) Gender distribution of ejection fraction in ADPKD patients and controls by gender B) Correlation of ejection fraction and eGFR in ADPKD patients. C) Ejection fraction according to CKD stages 1-4 in ADPKD patients (Mean±SD: CKD1 62.00±5.48 mm, CKD2 65.19±5.91 mm, CKD3a 62.97±5.62 mm, CKD3b 62.38±8.72mm, CKD4 64.33±5.03 mm) versus controls (Mean±SD 60.21±5.72 mm) D) Ejection fraction according to Mayo classification (1A/B (Mean±SD: 65.22±5.59mm) versus 1C/D/E (Mean±SD: 63.07±6.54mm)) in ADPKD patients versus controls (Mean±SD: 60.21±5.72mm). E) Ejection fraction in ADPKD patients among respective gene products (PKD1 and PKD2). Truncating mutations included non-sense, frameshift, splicing mutations, and large rearrangements, while non-truncating mutations included missense mutations and in-frame short deletions and insertions. F) Ejection fraction according to relevant comorbidities. Horizontal lines indicate median values.*



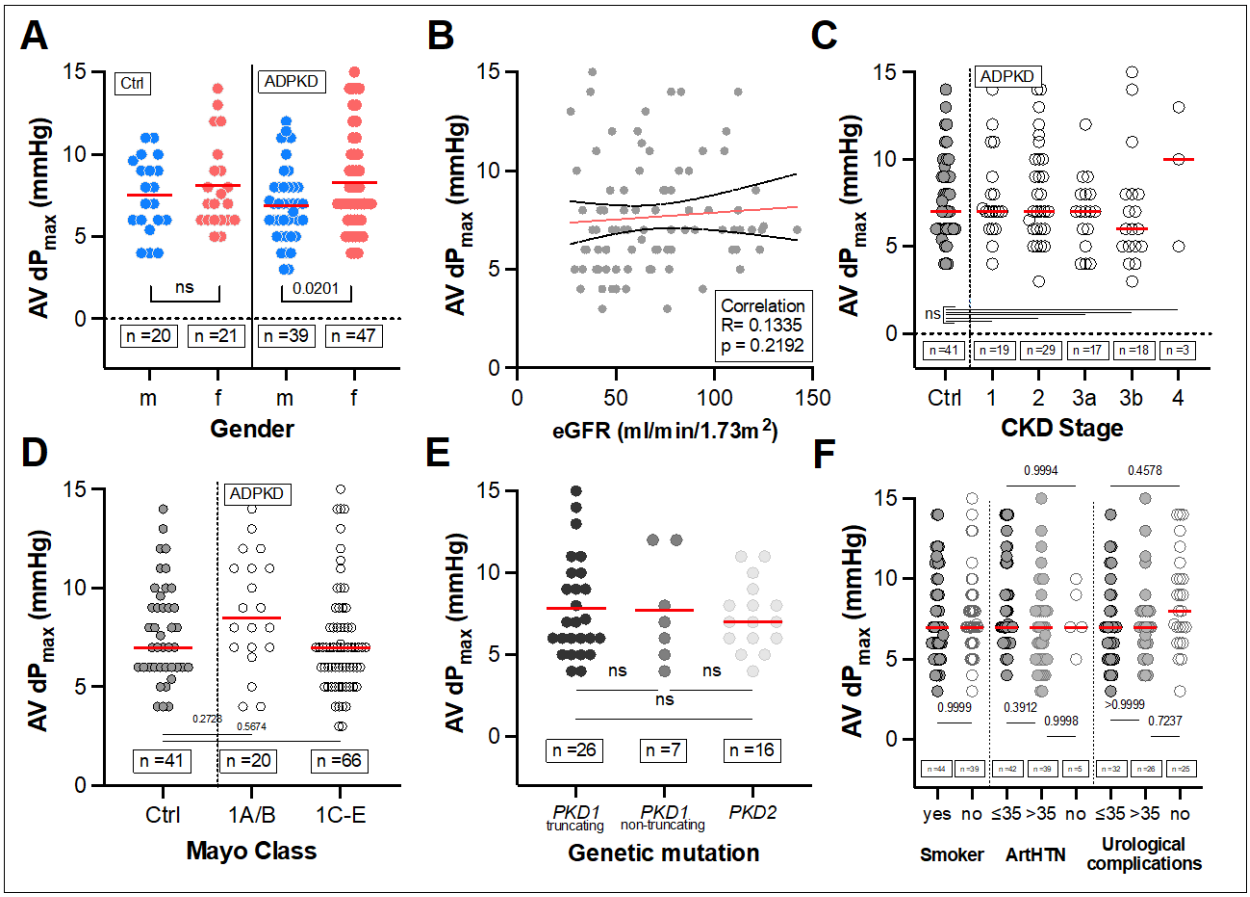
Supplemental Figure 12: Evaluation of left ventricular function using E/A (A, Mean±SD: controls 1.13 ± 0.36 vs. ADPKD patients 1.22 ± 0.36) and E/lat e' (B, Mean±SD: controls 7.20 ± 3.23 vs. ADPKD patients 6.77 ± 2.86) among control individuals and ADPKD patients. *ns indicates not significant*



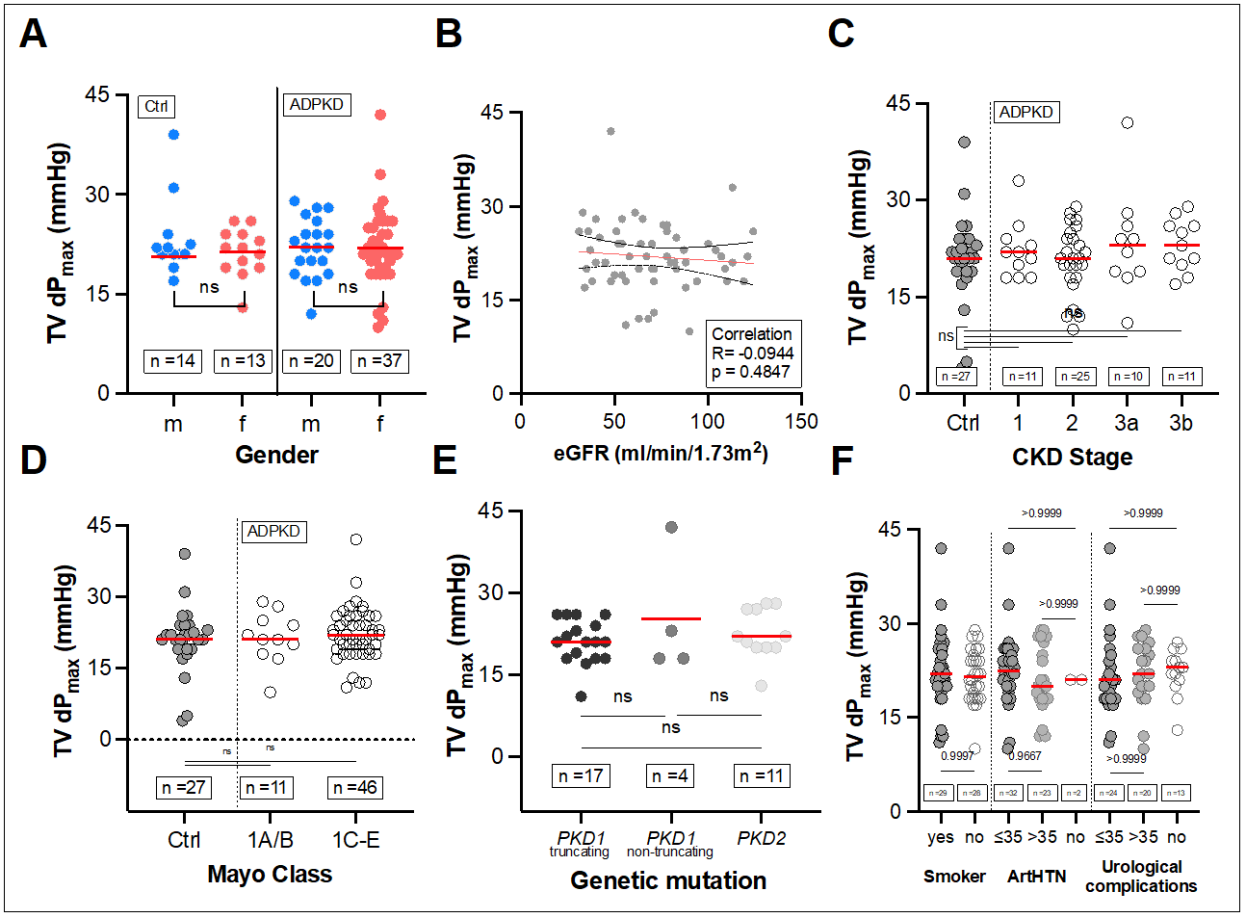
Supplemental Figure 13: Association of Right ventricular basal diameter (RVD) with clinical characteristics
A) Gender distribution of RVD in ADPKD patients (Mean±SD: male 36.21±4.66 mm and female 32.58±3.99 mm) and controls (Mean±SD: male 34.63±4.68 mm and female 32.17±3.50 mm) by gender B) Pearson correlation of RVD and eGFR in ADPKD patients. C) RVD according to CKD stages 1-4 in ADPKD patients (Mean±SD: CKD1 34.14±3.96 mm, CKD2 34.00±5.05 mm, CKD3a 33.50±5.46 mm, CKD3b 34.45±4.74 mm, CKD4 32.67±2.08 mm) versus controls (Mean±SD 33.57±4.35 mm) D) RVD according to Mayo classification (1A/B (Mean±SD: 32.96±4.55mm) versus 1C/D/E (Mean±SD: 34.48±4.60mm)) in ADPKD patients versus controls (Mean±SD 33.57±4.35mm). E) RVD in ADPKD patients among respective gene products (PKD1 and PKD2). Truncating mutations included non-sense, frameshift, splicing mutations, and large rearrangements, while non-truncating mutations included missense mutations and in-frame short deletions and insertions. F) RVD according to (ever) smokers and relevant comorbidities (ArtHTN). Horizontal red lines indicate mean values. ArtHTN indicates arterial hypertension, ns indicates not significant.



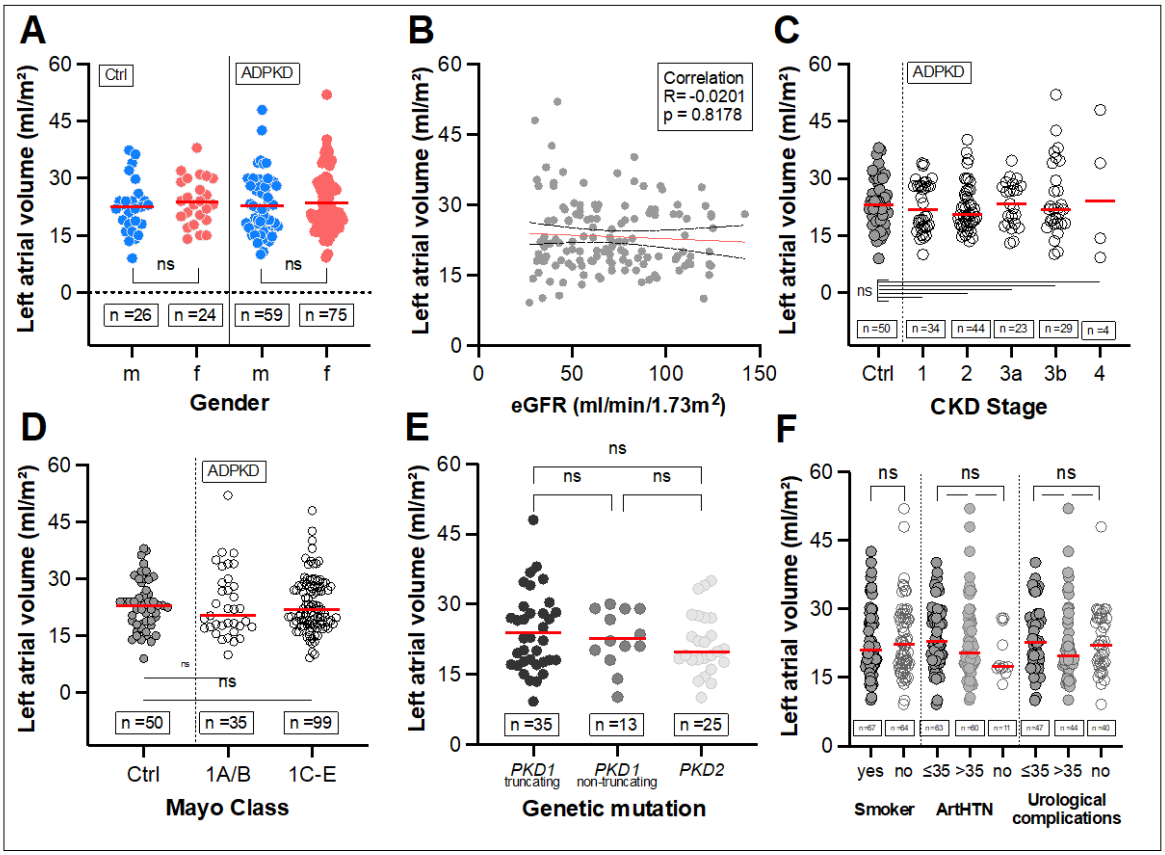
Supplemental Figure 14: Association of tricuspid annular plane systolic excursion (TAPSE) with clinical characteristics *A) Gender distribution of TAPSE in ADPKD patients and controls by gender B) Correlation of TAPSE and eGFR in ADPKD patients. C) TAPSE according to CKD stages 1-4 in ADPKD patients versus controls with maintained kidney function (eGFR ≥ 90ml/min/1.73m²). D) TAPSE according to Mayo classification (1A/B versus 1C/D/E) in ADPKD patients versus controls. E) TAPSE in ADPKD patients among respective gene products (PKD1 and PKD2). Truncating mutations included non-sense, frameshift, splicing mutations, and large rearrangements, while non-truncating mutations included missense mutations and in-frame short deletions and insertions. F) TAPSE according to relevant comorbidities. Horizontal lines indicate median values. Significance levels are only indicated by an asterisk if detected differences were significant.*



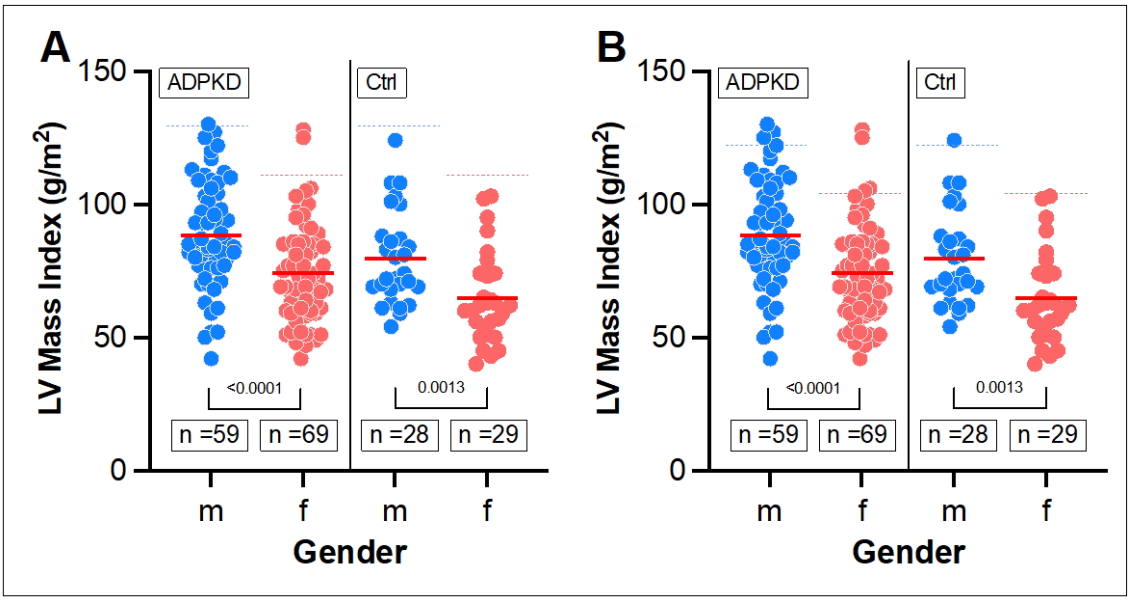
Supplemental Figure 15: Association of maximal pressure gradient across the aortic valve (AV dP_{max}) with clinical characteristics *A) Gender distribution of AV dP_{max} in ADPKD patients and controls by gender B) Correlation of AV dP_{max} and eGFR in ADPKD patients. C) AV dP_{max} according to CKD stages 1-4 in ADPKD patients versus controls with maintained kidney function (eGFR ≥ 90ml/min/1.73m^2). D) AV dP_{max} according to Mayo classification (1A/B versus 1C/D/E) in ADPKD patients versus controls. E) AV dP_{max} in ADPKD patients among respective gene products (PKD1 and PKD2). Truncating mutations included non-sense, frameshift, splicing mutations, and large rearrangements, while non-truncating mutations included missense mutations and in-frame short deletions and insertions. F) AV dP_{max} according to relevant comorbidities. Horizontal lines indicate median values.*



Supplemental Figure 16: Association of peak pressure gradient across the tricuspid valve (TV dP_{max}) with clinical characteristics *A) Gender distribution of TV dP_{max} in ADPKD patients and controls by gender B) Correlation of TV dPmax and eGFR in ADPKD patients. C) TV dPmax according to CKD stages 1-4 in ADPKD patients versus controls with maintained kidney function (eGFR ≥ 90ml/min/1.73m^2). D) TV dP_{max} according to Mayo classification (1A/B versus 1C/D/E) in ADPKD patients versus controls. E) TV dP_{max} in ADPKD patients among respective gene products (PKD1 and PKD2). Truncating mutations included non-sense, frameshift, splicing mutations, and large rearrangements, while non-truncating mutations included missense mutations and in-frame short deletions and insertions. F) TV dP_{max} according to relevant comorbidities. Horizontal lines indicate median values. Significance levels are only indicated by an asterisk if detected differences were significant.*



Supplemental Figure 17: Association of left atrial volume with clinical characteristics *A) Gender distribution of left atrial volume in ADPKD patients and controls by gender B) Correlation of left atrial volume and eGFR in ADPKD patients. C) Left atrial volume according to CKD stages 1-4 in ADPKD patients versus controls with maintained kidney function (eGFR ≥ 90ml/min/1.73m²). D) Left atrial volume according to Mayo classification (1A/B versus 1C/D/E) in ADPKD patients versus controls. E) Left atrial volume in ADPKD patients among respective gene products (PKD1 and PKD2). Truncating mutations included non-sense, frameshift, splicing mutations, and large rearrangements, while non-truncating mutations included missense mutations and in-frame short deletions and insertions. F) left atrial volume according to relevant comorbidities. Horizontal lines indicate mean values.*



Supplemental Figure 18: LV mass index among ADPKD patients (Mean±SD: male 88.32±20.45 mm and female 74.32±18.43 mm) and kidney donor candidates (Mean±SD: male 79.57±17.56 mm and female 64.66±17.02 mm). Thresholds were defined according to Chapman *et al.*[11] (A) or Perrone *et al.*[13] (B) and indicated by the dashed line.