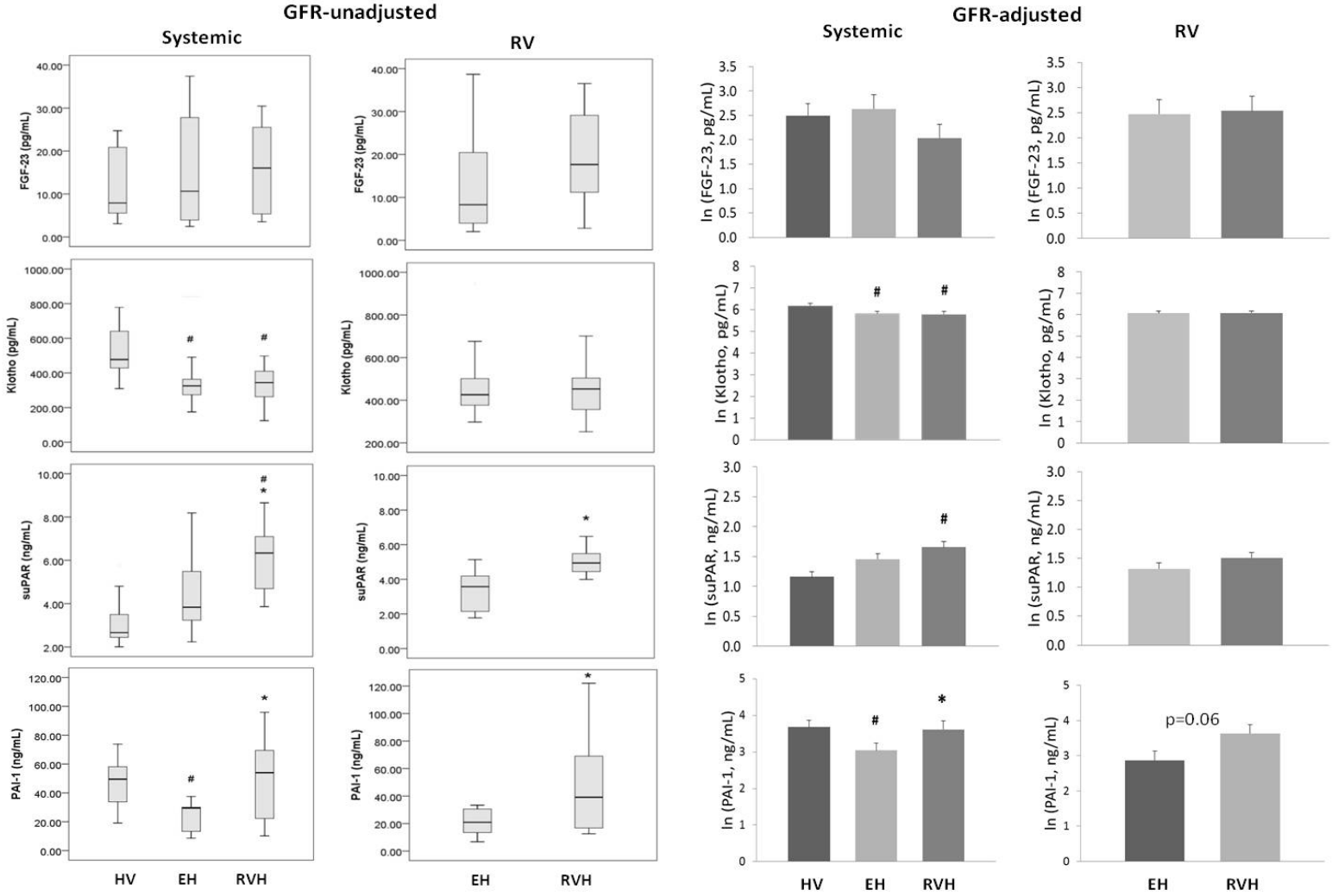
Supplementary Figure 1: Systemic and RV levels of FGF-23, Klotho, suPAR, and PAI-1 in patients with EH or RVH, compared with systemic levels in HVs, unadjusted or adjusted for GFR. Unadjusted systemic and RV levels of FGF-23 were not different among the groups, whereas systemic level of Klotho was significantly lower in the RVH and EH groups compared with HVs. Levels of suPAR in the RVH group were higher in the systemic circulation compared with both HVs and patients with EH, and PAI-1 in patients with RVH versus EH. The adjusted systemic level of Klotho remained significantly reduced in the RVH and EH groups compared with HVs, and the adjusted level of suPAR remained elevated in patients16 with RVH compared with HVs. PAI-1 RV levels tended to be elevated in RVH versus EH. *P≤0.05 versus EH; #P≤0.05 versus HV. EH, essential hypertension; FGF, fibroblast growth factor; HV, healthy volunteer; PAI, plasminogen activator inhibitor; RV, renal vein; RVH, renovascular hypertension; suPAR, soluble urokinase plasminogen activator receptor.



Supplementary Figure 2: Gradient and net release of FGF-23, Klotho, suPAR, and PAI-1 in patients with EH, and both the stenotic kidneys (RVH) and CLKs in patients with RVH, unadjusted or adjusted for GFR. Only unadjusted FGF-23 gradient was greater in the stenotic RVH kidney versus EH, and Klotho net release tended to be as well. Gradient and net release of GFR-adjusted markers in EH and RVH were not significantly different among the groups. A negative suPAR gradient was greater in the CLK compared with the stenotic kidney, and its net release also decreased in the CLK. Net release of PAI-1 was lower in the CLK compared with the stenotic kidney. *P≤0.05 versus EH; **P≤0.05 versus RVH. CLK, contralateral kidney.