FGF23 MAINLY REGULATES PHOSPHATE HOMEOSTASIS IN STAGE 3 CHRONIC KIDNEY DISEASE

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OBJECTIVES

It has been reported that the phosphorus overload per residual nephron may induce kidney damage contributing to the *interstitial* fibrosis and inflammation. So phosphorus is presumed one of chronic kidney disease (CKD) progression factors. Fibroblast growth factor 23 (FGF23) and parathyroid hormone (PTH) as phosphaturic hormone are known for an appropriate biomarker of phosphorus overload per residual nephron. Fractional excretion of phosphorus (FEP) is the ratio of clearance of phosphorus and creatinine, which is in theory indicating the phosphorus load per residual kidney function (nephron). We evaluate the relationship between FEP FGF23, and PTH in patients with kidney donors before and after nephrectomy (Nx).

METHODS

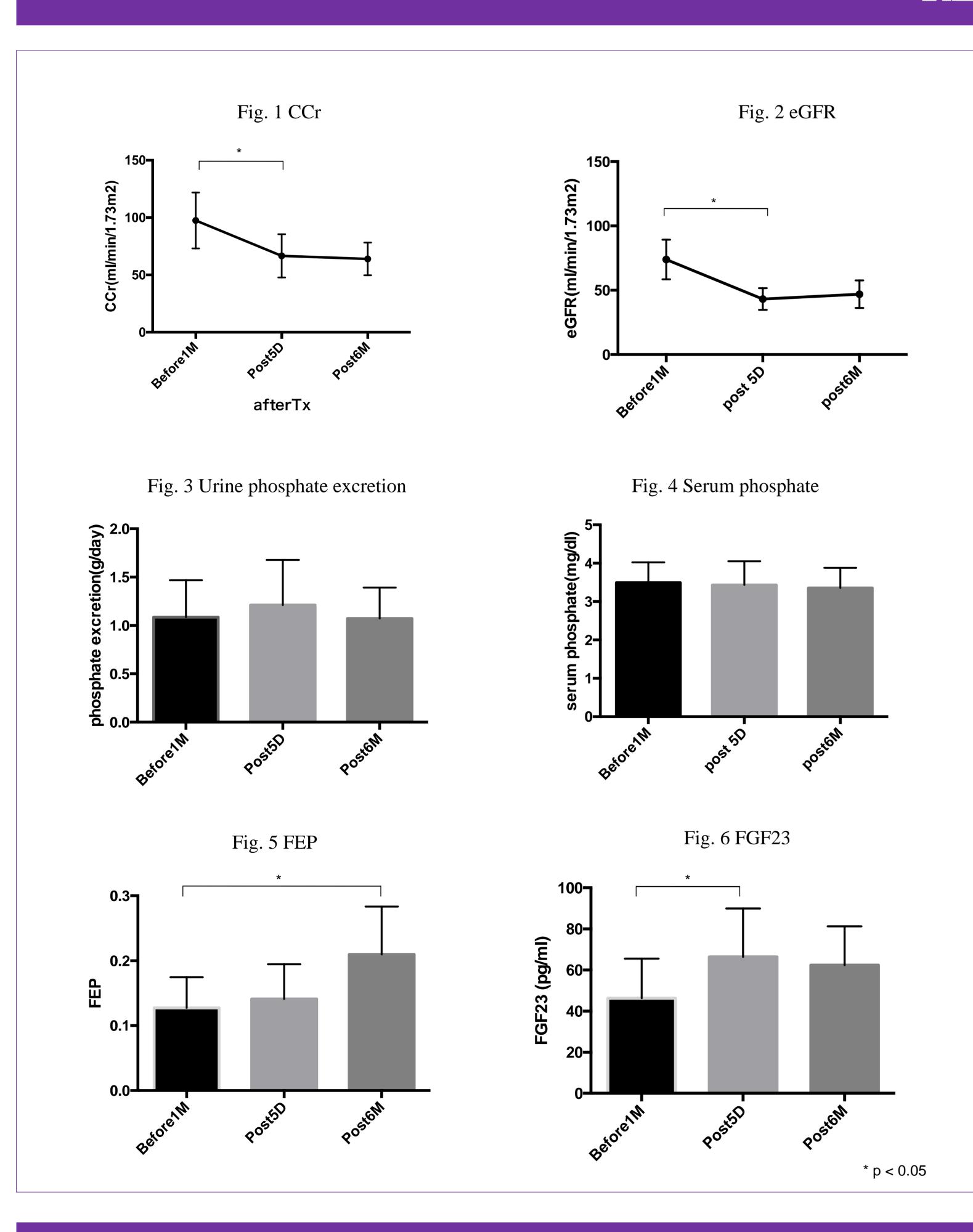
- Twenty-one kidney donors were inrolled.
- Average age was 53.2 ± 5.7 years old.
- Ten of them were male donors.

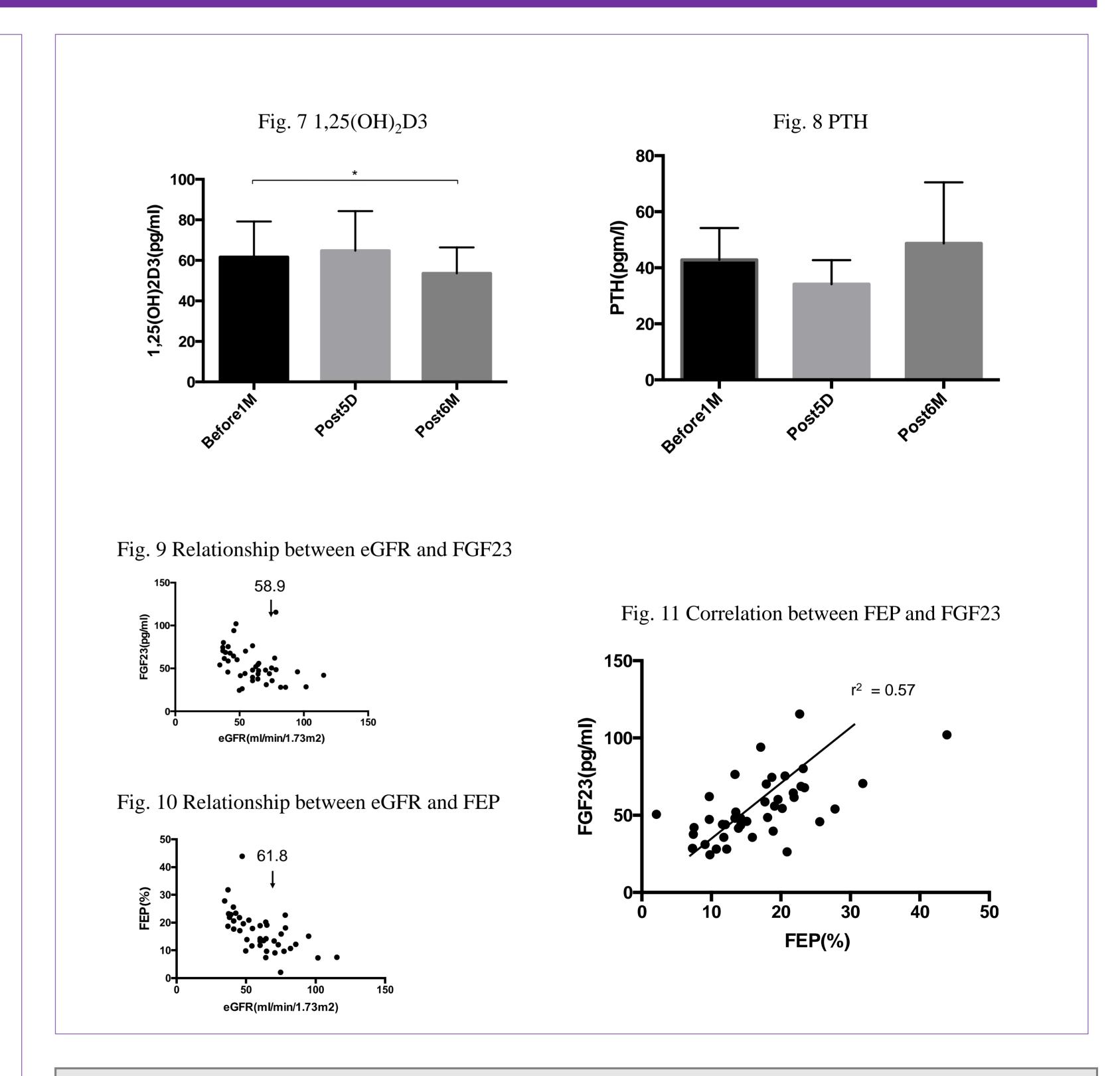


One month before, 5 days after, and 3 months after nephrectomy (Nx), following parameters were measured.

- Creatinine Clearance (CCr)
- Estimated glomerular filtration rate (eGFR)
- Urine phosphate excretion
- Serum phosphate
- Fractional excretion of phosphate (FEP)
- Fibroblast growth factor 23 (FGF23)
- Vitamin D (1,25(OH)₂VitD3)
- Parathyroid hormone (PTH)

RESULTS





- CCr and eGFR decreased immediately after Nx.
- FGF23 and FEP increased after Nx, but not PTH.
- Both FGF23 and FEP began to increase at eGFR 60 ml/min/1.73m² (CKD stage 3).
- FGF23 was positively correlated with FEP.

CONCLUSIONS

The compensatory mechanism for the phosphate homeostasis is induced at CKD stage 3 and the major contributing factor is FGF23 but not PTH









