

FGF23, PHOSPHATURIA, AND VASCULAR CALCIFICATION IN A PERITONEAL DIALYSIS POPULATION WITH RESIDUAL RENAL FUNCTION



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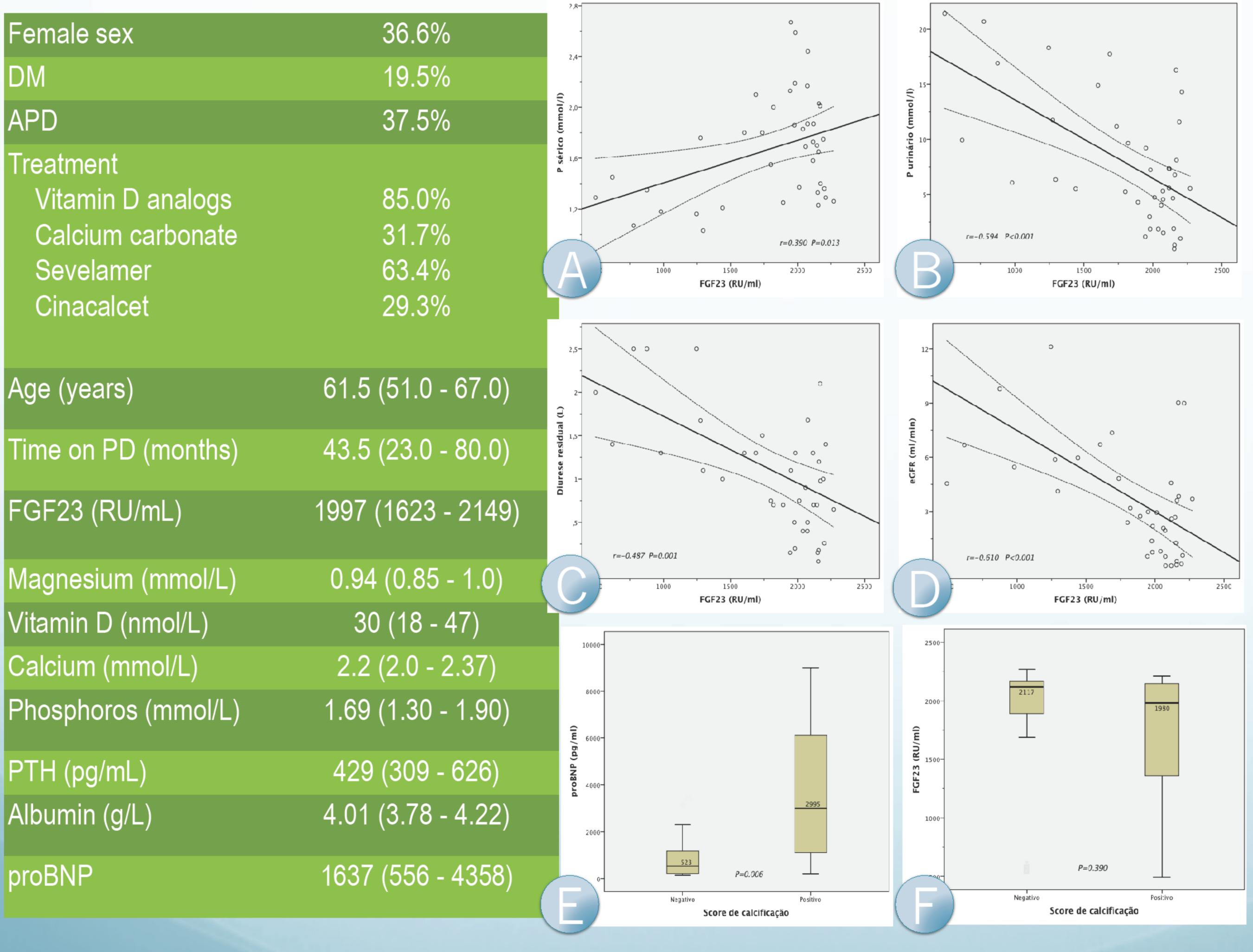
INTRODUCTION AND AIMS:

Fibroblast growth factor-23 (FGF23) induces phosphaturia. In dialysis patients serum levels are 100-1000 times higher than that in the general population. Its clinical impact is beyond mineral bone disease in CKD, being coupled with vascular calcification and mortality. Residual renal function (RRF) is associated with significant capacity to excrete phosphate in Peritoneal Dialysis (PD) patients. Therefore, investigation of the relationship of FGF23 with GFR and phosphate excretion is opportune, to increase the knowledge on its clinical relevance in this disease stage.

METHODS:

FGF23 (C-terminal assay, Immunotopics) was measured in 40 prevalent stable chronic PD patients with >3 months on PD and with RRF. Clinically relevant variables including CKD-bone laboratory parameters, serum vitamin D magnesium (Mg) levels, dialysis adequacy, glomerular filtration rate (GFR), urinary phosphate, fractional excretion of phosphorus (FePi), and vascular calcification score (Adragão et al, NDT, vol. 19, No 6, 2004) were explored cross-sectionally. Non parametric tests were applied (Spearman Correlations and Mann-Whitney U Test).

RESULTS:



FGF23 correlated positively with:

(A) serum phosphate (r=0.39, p= 0.013)

FGF23 correlated negatively with:

- (B) urine volume (r=-0.48, p= 0.001)
- (C) phosphaturia (r=-0.594, p<0.0001)
- (D) GFR (r=-0.61, p<0.0001)

However FGF23 was not significantly correlated with age, time on dialysis, FePi, Mg, nor vitamin D.

Lower levels of albumin, lower levels of magnesium and higher levels of proBNP (image E) significantly differed in calcified versus non calcified patients (all with p<0.05).

Neither FGF23 (F) nor low FePi/FGF23 ratio was significantly associated with vascular calcification score.

CONCLUSIONS:

A sustained increase of FGF23 in PD patients signalizes an active endocrine phosphaturic process compensating residual renal function loss. It alerts for dietetic and therapy optimization. However, in our population, it was not associated with vascular calcification.









