RELATIONSHIP OF DIETARY PHOSPHATE INTAKE WITH SUBCLINICAL VASCULAR DAMAGE IN STAGE 3-5 NON-DIALYSIS CHRONIC KIDNEY DISEASE

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Introduction

- Higher serum phosphate (P) is associated with risk of death, CVD and progression to ESRD in earlier stages of CKD, even among persons with normal kidney function.
- Dietary P intake is a key determinant of serum P in patients on dialysis.
- KDIGO guidelines recommend the use of intestinal P binders and limiting dietary P intake to maintain serum P within the normal range in stage 3-5 non-dialysis CKD.
- However, the benefit of this assumption is controversial:
  - Serum P is only weakly related with dietary phosphate intake in CKD-ND
  - P binders in CKD 3-5ND are able to reduce urinary phosphate excretion but **the effects on serum phosphate arte minimal or absent**
  - The association of dietary phosphate intake with mortality in CKD-ND are unknown

Aim

- To analyze the association of dietary P intake with subclinical organ damage in stage 3-5 non-dialysis CKD patients.

Material and methods

- Design: transversal, observational study
- inclusion: 60 consecutives patients evaluated:
  - Age: 40 - 65 years
  - CKD 3-5ND
  - asymptomatic, without previous CV disease
  - P < 5mg/dl without phosphorous binders
- Estimation of P intake was based on:

  1- a systematic three consecutive days dietary interview held by a dietician
  - a digital scale was provided for weighing food.
  - information was completed with **photo album SUVIMAX** and with precooked food labels
  - to quantify the composition we used the **CESNID nutritional calculator program**

  2- measurement of 24-h urinary P excretion

Material and methods

Vascular function was assessed:

1- with the SphygmoCor system, to obtain carotid-femoral pulse wave velocity, estimates of central aortic pressures and augmentation index

2- vascular structural changes were analysed with measurement of intima-media thickness and evaluation of atheroma plaques using carotid ultrasound (ANTARES-Siemens), image and measurements were carried out with the Syngo Art Health-US Workplace 3.0 software

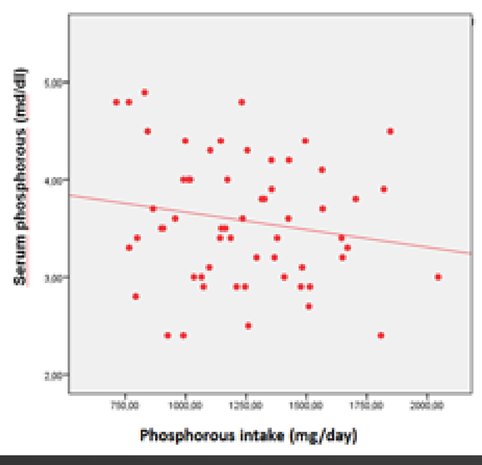
3- left ventricular hypertrophy and valve calcification were assessed by echocardiography study

4- vascular calcification scores were obtained with Kauppila and Adragao indexes

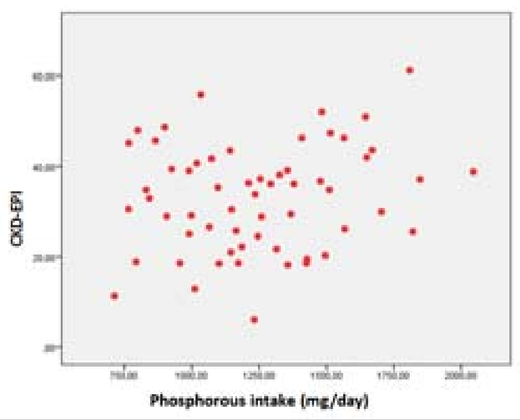
Results

- Mean age: 54±8; 28% women
- Aetiology of CKD:
  - nephrosclerosis (biopsy proven): 28 (9)
  - diabetic nephropathy: 3
  - chronic glomerulonephritis : 8
  - polycystic kidney disease: 7
  - others: 12
  - unknown: 2
- Cr: 2.35±0.98 mg/dl
- eGFR(CKD-EPI): 32.8±11 mL/min/1.73m²
  - 3a: 11
  - 3b: 23
  - 4: 23
  - 5: 3
- Serum P: 3.59±0.66 mg/dl; (2,2-4,9)
- Serum Ca: 9.41±0.54 mg/dl; (8,09-11,5)
- P intake: 1242±310 mg/d; (712-2044 mg)
- 24 –h urinary P excretion: 847±308 mg/d; (270-2190 mg)

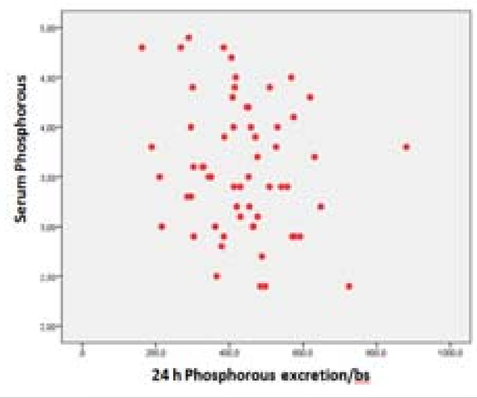
Serum P and P intake correlation: R= -0.17 (p NS)



P intake and CKD-EPI correlation: R=0,189 (p 0,15)

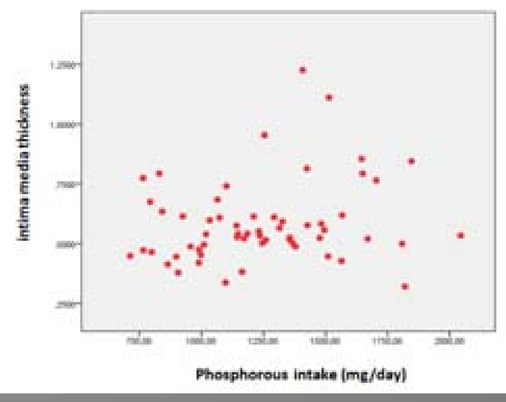


24h P excretion/bs and serum P correlation: R -0,19; (p NS)



P intake and 24 h urinary P excretion correlation: R=0,435 (p=0,001)

P intake and intima media thickness correlation: 0,2 (p 0,13)



Baseline characteristics and vascular results by terciles of P intake

	T1	T2	T3	p
P intake	712 – 1073	1096 – 1367	1378 - 2044	
age	54.7 ± 8.9	53.7 ± 8.9	53.05 ± 8.3	ns
CKD-EPI	33.6 ± 12.4	28.2 ± 9.3	37.5 ± 11.9	0.043
P	3.6 ± 0.7	3.6 ± 0.5	3.4 ± 0.5	ns
Ca	9.4 ± 0.6	9.4 ± 0.5	9.3 ± 0.3	ns
Pulse wave velocity	**10.2 ± 3.4**	**9.8 ± 2.5**	**9.2 ± 1.8**	**ns**
Systolic Aortic Pressure	**128 ± 20**	**134 ± 16**	**134 ± 16**	**ns**
Augmentation Index	**30 ± 8.8**	**24 ± 8.6**	**26 ± 7.4**	**0.05**
Intima media thickness	**0.54 ± 0.12**	**0.55 ± 0.12**	**0.65 ± 0.23**	**ns**
Ventricular mass/sc	**82 ± 27**	**96 ± 41**	**89 ± 30**	**ns**
Kauppila score	**4.7 ± 1.4 SE**	**2.6 ± 1.2 SE**	**1.8 ± 0.9 SE**	**ns**
Adragao score	**0.4 ± 0.16 SE**	**0.6 ± 0.35 SE**	**0.6 ± 0.28 SE**	**ns**

Baseline characteristics and vascular results by terciles of 24h urinary P excretion/bs

	T1	T2	T3	p
Urinary P excretion/bs	162 - 384	384 - 475	476 - 880	
P intake	1062 ± 248	1407 ± 277	1263 ± 312	0.001
age	57 ± 8.8	51 ± 8.9	53 ± 7.3	0.07
CKD-EPI	28 ± 13	31 ± 10	38 ± 10	0.017
P	3.6 ± 0.7	3.7 ± 0.5	3.4 ± 0.6	ns
Ca	9.5 ± 0.6	9.4 ± 0.5	9.3 ± 0.4	ns
Pulse wave velocity	**10.5 ± 3.3**	**9.9 ± 2.6**	**9 ± 20**	**ns**
Systolic Aortic Pressure	**130 ± 19**	**132 ± 18**	**136 ± 16**	**ns**
Augmentation Index	**27 ± 10**	**25 ± 7**	**29 ± 8**	**ns**
Intima media thickness	**0.56 ± 0.12**	**0.60 ± 0.19**	**0.59 ± 0.18**	**ns**
Ventricular mass/sc	**85 ± 30**	**89 ± 40**	**92 ± 30**	**ns**
Kauppila score	**5.25 ± 1.5 SE**	**2.25 ± 1.1 SE**	**1.60 ± 0.79 SE**	**ns**
Adragao score	**0.70 ± 0.28 SE**	**1 ± 0.51 SE**	**0.3 ± 0.14 SE**	**ns**

Discussion

- KDIGO recommendation limiting P intake and/or using P binders in CKD stages 3-5ND is debatable: these strategies only minimally or do not affect serum P levels
- Still could be beneficial by reducing FGF-23 or Intact PTH levels?
- In any case, it does not seem that a higher intake of P have worst consequences
- Conversely, advise to lower P intake will result in lowering protein intake, increasing the risk of protein malnutrition, something unwise
- Other factors not directly linked to the intestinal P absorption should be determinants of serum P levels (altered renal tubule phosphate transport, bone buffering, intra-extra cellular phosphate shifts....)

Existence of "plaque" or "calcified plaque" on ultrasound

plaque	Yes (n=22)	No (n=38)	P
P intake	1253±327	1220±282	NS
24h urinary P excretion	856±328	830±278	NS

Calcified plaque	Yes (n=16)	No (n=44)	P
P intake	1145±338	1275±296	NS
24h urinary P excretion	690±231	899±316	0,01

Conclusions

- Phosphate intake in CKD stages 3-5ND is not closely correlated with serum phosphate concentration
- Phosphate intake is not associated with subclinical vascular damage
- Other not well defined factors may be key determinants of serum phosphate concentration

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