parathyroidectomy , when it is necessary

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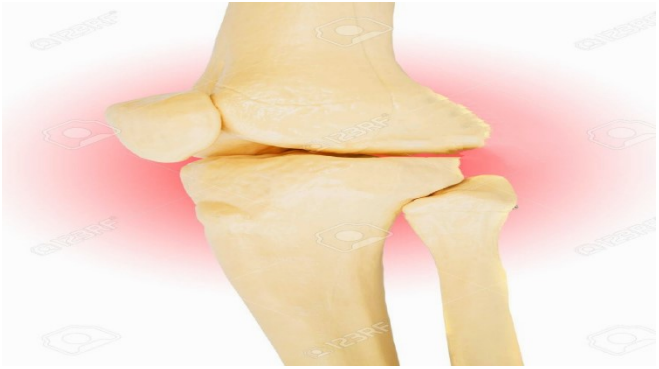
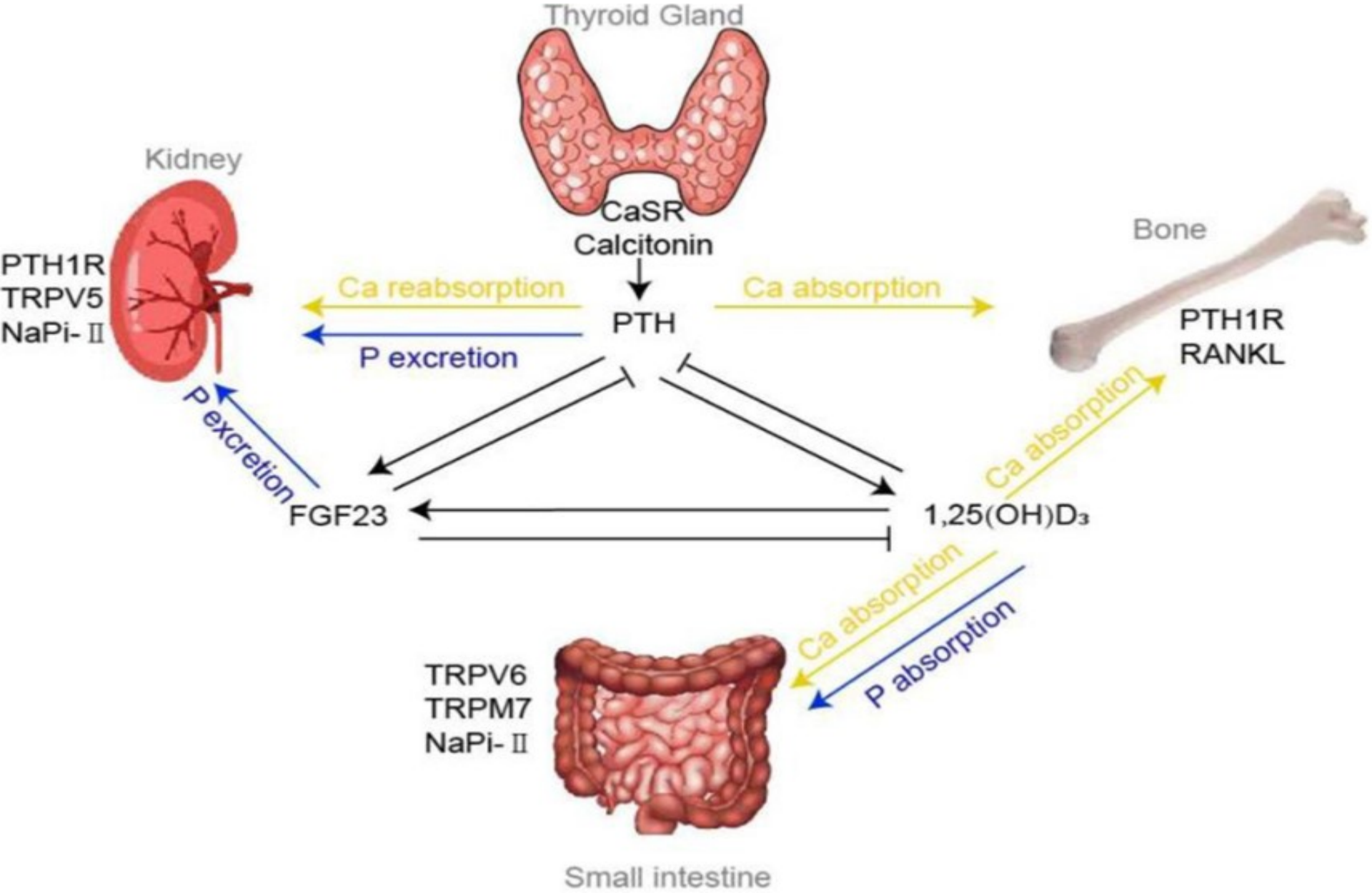


INTRODUCTION

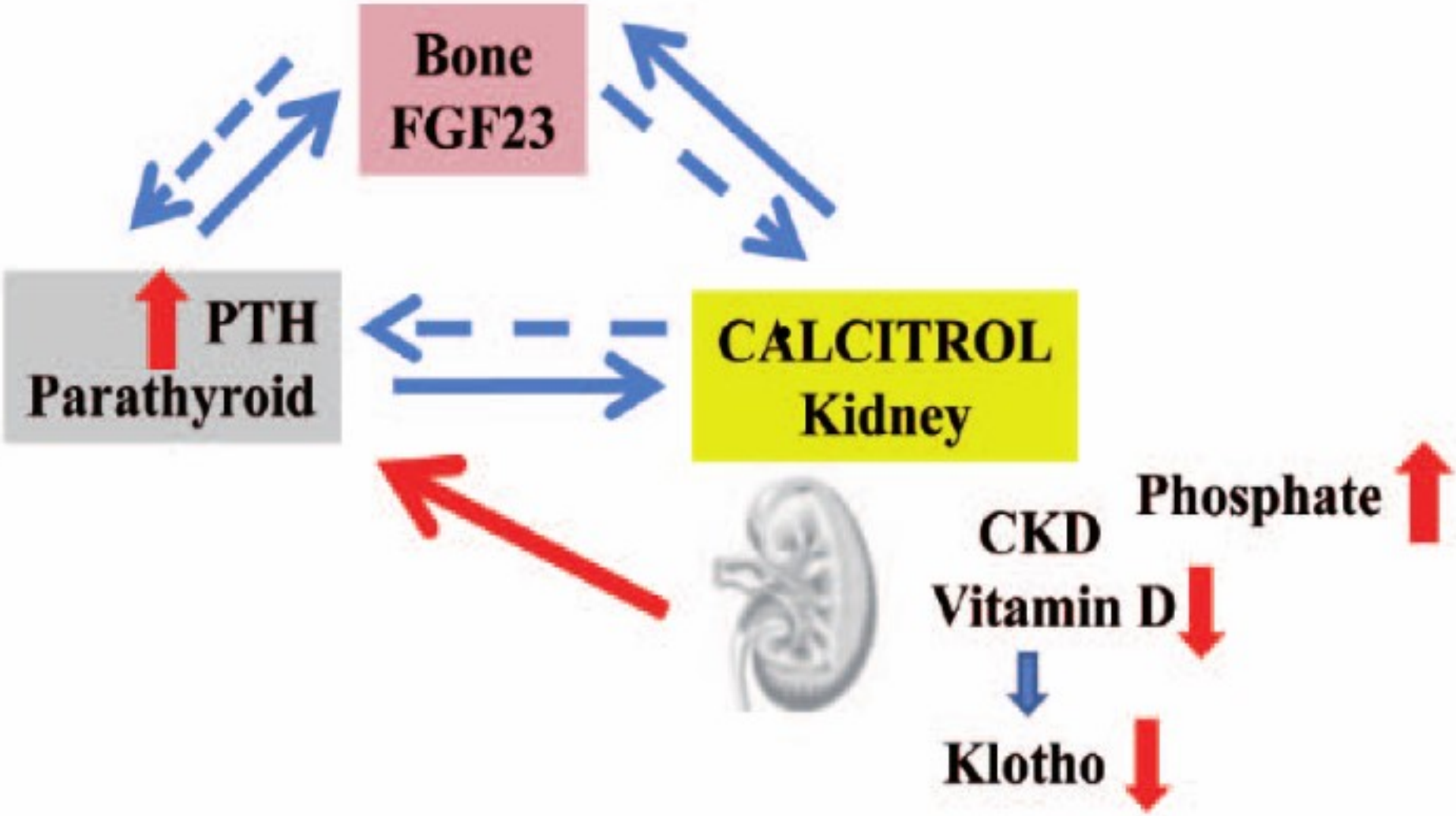
- Disturbances in mineral and bone metabolism are almost a constant feature of chronic kidney disease, and constitute a major cause of morbidity and mortality through their metabolic, bone and, above all, cardiovascular consequences.

- This justifies prevention and treatment based on physiopathological knowledge and international recommendations.

PHOSPHOCALCIC HOMEOSTASIS



During chronic kidney disease



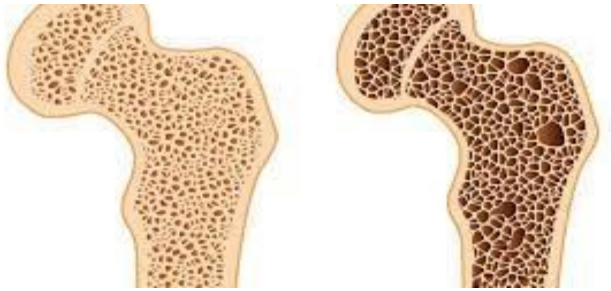
CLASSIFICATION

Bone metabolism disorders (BMD) with high bone remodeling:

Fibrosis osteitis : high PTH levels:

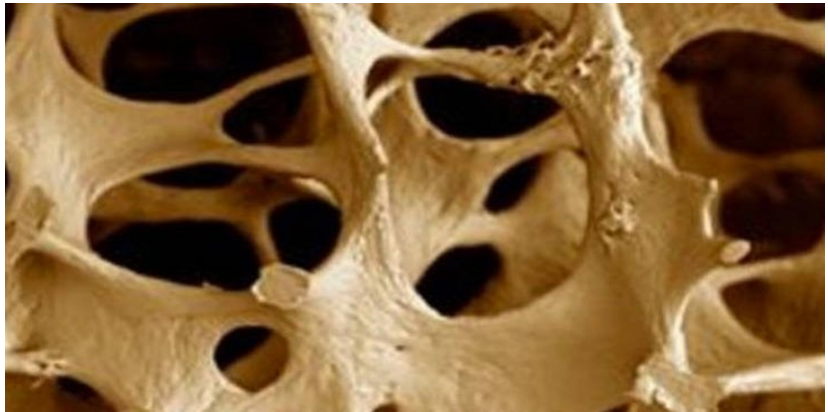
TMO with low bone remodelling:

Osteomalacia:



- aluminic osteomalacia
- no-aluminic osteomalacia

Adynamiquic bone



Treatment

Parameter targets according to KDIGO 2017

It is advisable to maintain a PTH level within a range of around two to nine times the upper limit of normal, (150-300 pg/l)

It is advisable to maintain :

Blood phosphate levels within laboratory norms (0.8 to 1.45mmol/l) = 25-55 mg/l

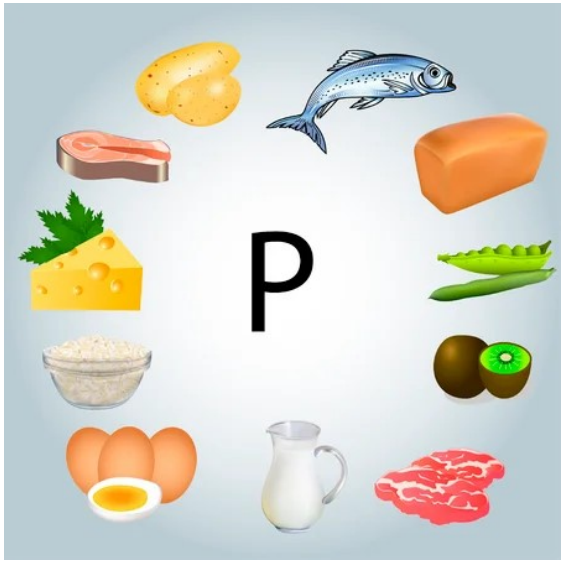
Normal blood calcium 2.1-2.37mmol/l= 80-100 mg/l

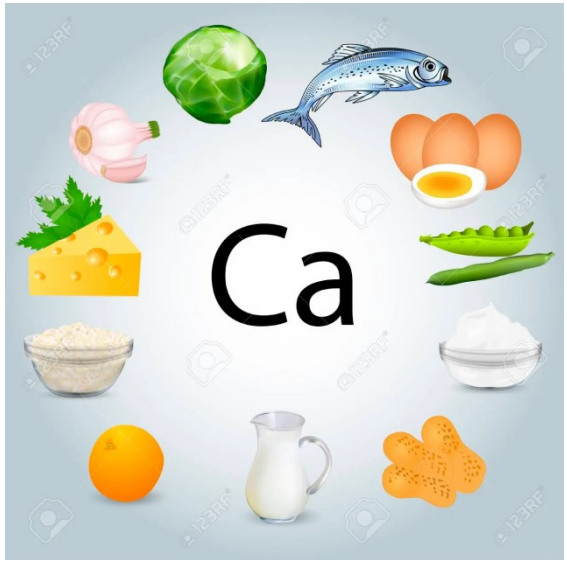
$P*Ca < 4.5\ (mmol/l)^2$

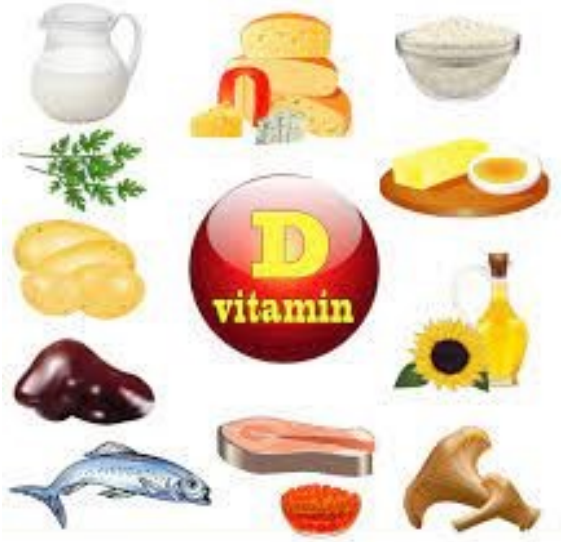
Native vit D reserves around 40 ng/l

TREATEMENT

- GOOD DIALYSIS ++++++
- The right diet ....! Difficult



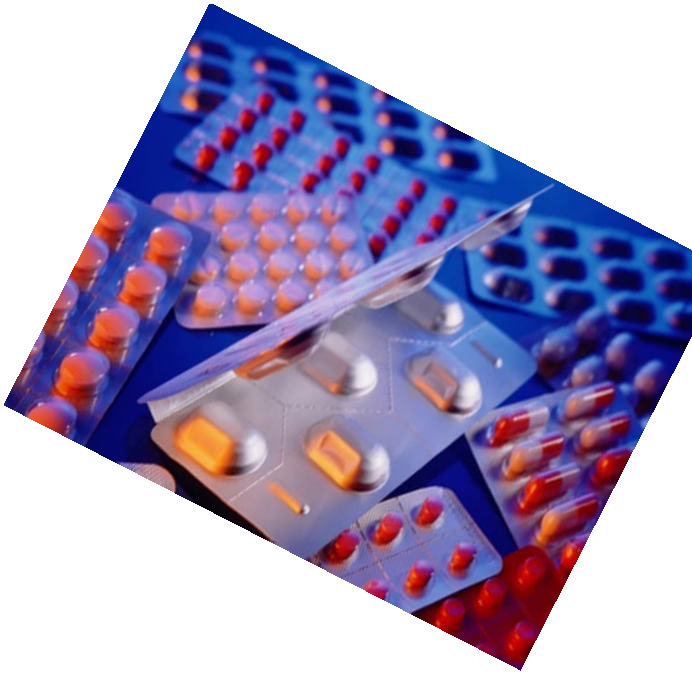




1-Treatment of hyperphosphatemia:

*Calcium phosphate binders

*No-calcium phosphate binders



2- Treatment of hypocalcaemia:

*Calcium substitution outside of meals.

*Monitoring ++++++

3-Vit D replacement:

*Native vit D: if low reserves of (25 OH vit D).

*1- alfa: (1, OH vit D) substitution obligatory from stage IIIb to prevent CKD-MBD.

4-Calcimimetic (Cinacalcet):

*after correction of serum calcium

*for a PTH>300

5-Parathyroidectomy(PTX)

Indications:

- Intact PTH> 800 pg/ml(about 7 times normal).
- hypercalcaemia and severe hyperphosphataemia or PxCa > 4.5
- nodular or diffuse hyperplasia.
- One or more parathyroid glands with a volume>0.5Cm3 or diameter>1cm or parathyroid carcinoma.
- radiological signs of hyperparathyroidism (cortical thinning, subperiosteal resorption)
- skin necrosis associated with calcifying arteriopathy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Case N°1

T.A is 24 years old man and has been on haemodialysis since the age of 21 years old.

*His history reports :

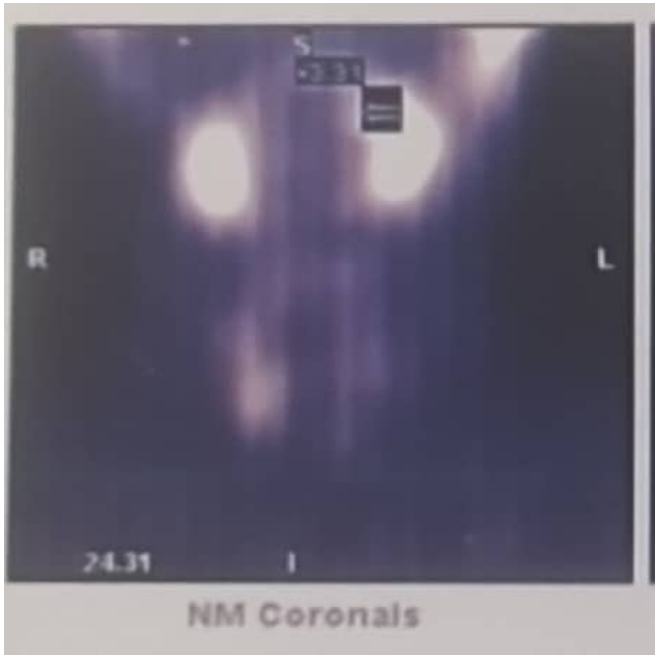
-He had been treated for hypercalcaemia since childhood complicated by recurrent renal lithiasis and bilateral nephrocalcinosis.

-Then chronic renal failure (CRF) at the age of 14.

*Analysis of the case history revealed hypercalcaemia with normo-hypophosphataemia during childhood, in favour of PTH I.

*After the onset and evolution of CKD and its complications, the pho/Ca balance reversed (ph:80-129mg/l, Ca:70-80mg/l) with a drop of Vit D, PAL up to 1800ui, and PTH already at 2200pg/l at the start of dialysis.

* Parathyroid scintigraphy revealed hyperplasia (Fig1).



Parathyroidectomy was suggested at the very begining but not performed!

Treatment was continued with calcium, pho chelation, VitD, calcimimetics.

the PTH did not stop increasing up to (5400pg/l), leading to the installation of all the complications of HPT such as:

-Bone deformity, pain and demineralisation

-EPO resistance (Hb:7-8 g/dl, Ferretinemia: 700 ng/l)

-Significant weight loss, loss of appetite, chronic asthenia and tendency to depression

-Vascular calcifications and impaired blood supply to the extremities and skin necrosis(Fig2).

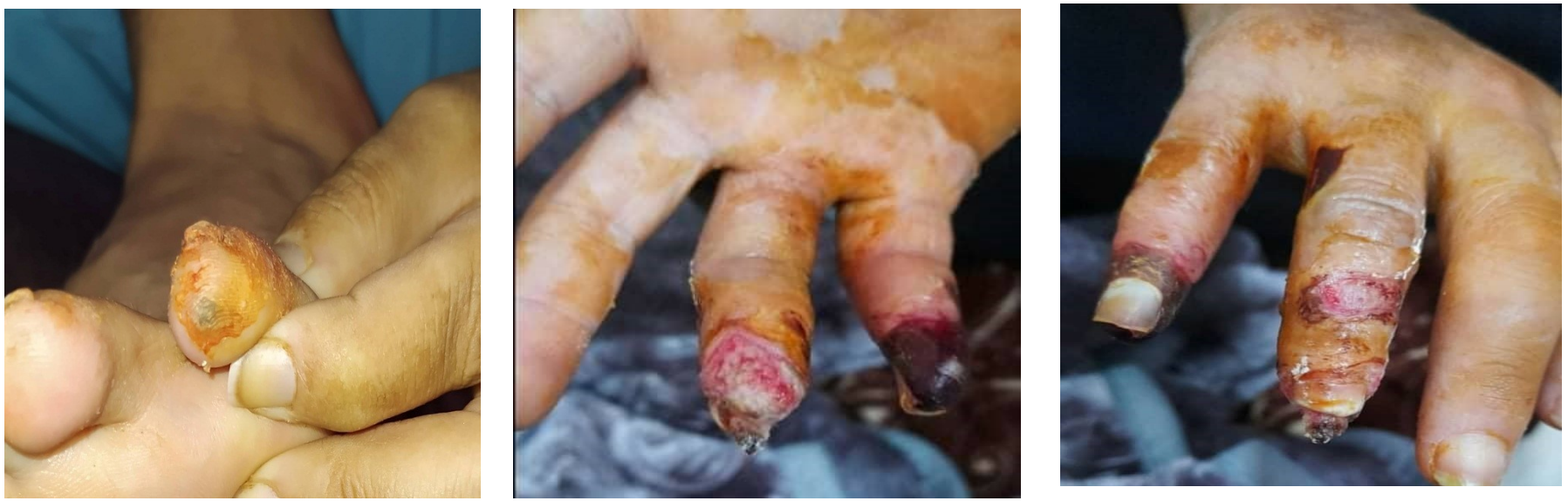


Fig 2 skin necrosis affecting the extremities

After agreement of the patient (3 years of evolution) a parathyroidectomy 7/8th parathyroidectomy was performed.

- Histological examination was in favour of parathyroid hyperplasia with no signs of malignancy.
- The clinical course after three months was marked by : resumption of appetite, weight gain, resolution of bone pain and and improvement in his walking,
- Biologically: Ca+:82 mg/l, Pho:40mg ,Hb:12.8g/dl, Férritinémie:229ng/l, PAL:224ui/l.

Case N°2

- B.S, aged 32, with a history of CKD for 7 years due to undetermined nephropathy.

- After 6 years of dialysis, he began to developpe bone and muscle pain.

- Biological findings:

Hypo/normocalcaemia: 70-88mg/l,

Normo/hyperphosphatemia: 45-65mg/l

PAL: 700ui/l

EPO resistance (hb:8-9g/dl, Ferretinemia:1100ng/l),

Native Vit D: tendency to hypo

PTH: 800pg/ml .....1200pg/ml(Calcimimetic started but unable to increase dose to over 90mg/d due to digestive intolerance)

Ultrasound : parathyroid nodule.

7/8th parathyroidectomy performed; complicated by severe hypocalcaemia despite supplementation before and after surgery, which took 3 weeks to achieve normal Ca++ levels.

Progression after 3 months:

Disappearance of asthenia, bone and muscle pain

Weight gain of 1.5 kg

Calcaemia: 87mg/l

Blood phosphate: 45mgl

Native vit D: 48ug/l

PTH: 59pg/ml

Hb: 11.3g/dl with serum ferritin: 870ng/l

Case N°3

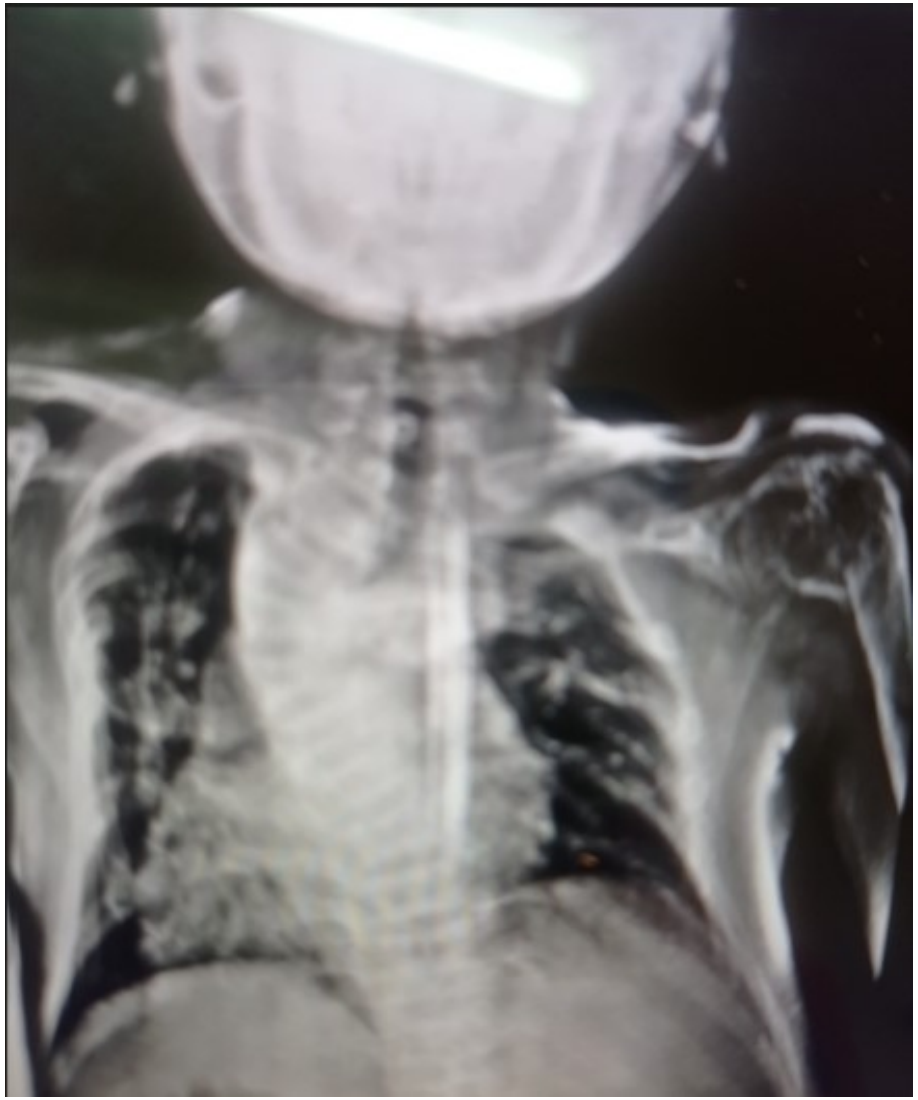
F,S aged 25 years with CKD for 10 years due to undetermined nephropathy (probably glomerular disease according to the history of CKD).

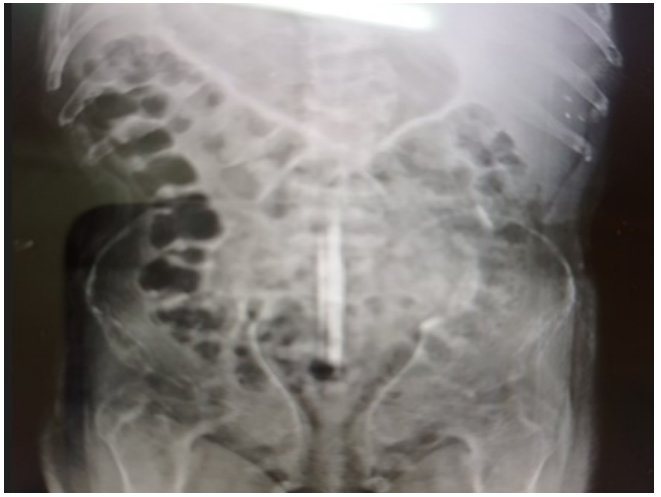
Clinical history: Bed-ridden for about 2 years after a fall from a height that caused several fractures.

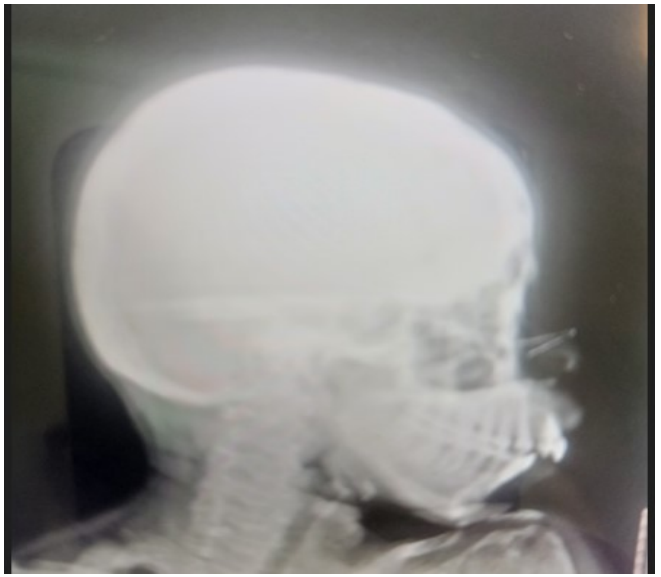
Significant skeletal deformities (thorax, skull, limbs, spine)

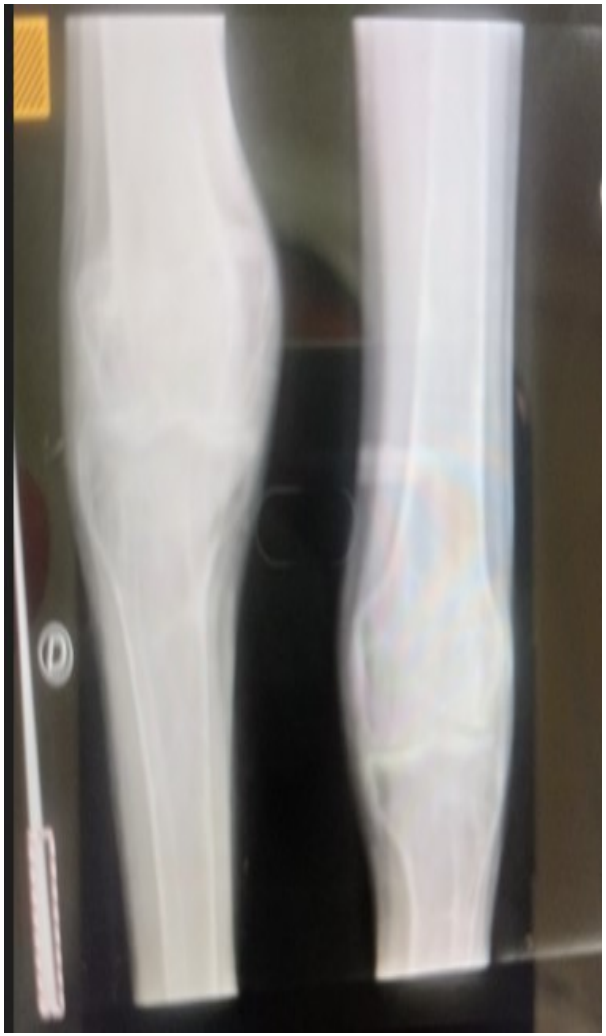
Biology: PTH > 1500Phosphatemia between 60 and 80 mg/l Calcaemia between 75 and 90 mg/l Native Vit D within normal range

Radiology: diffuse demineralisation with significant bone deformations.





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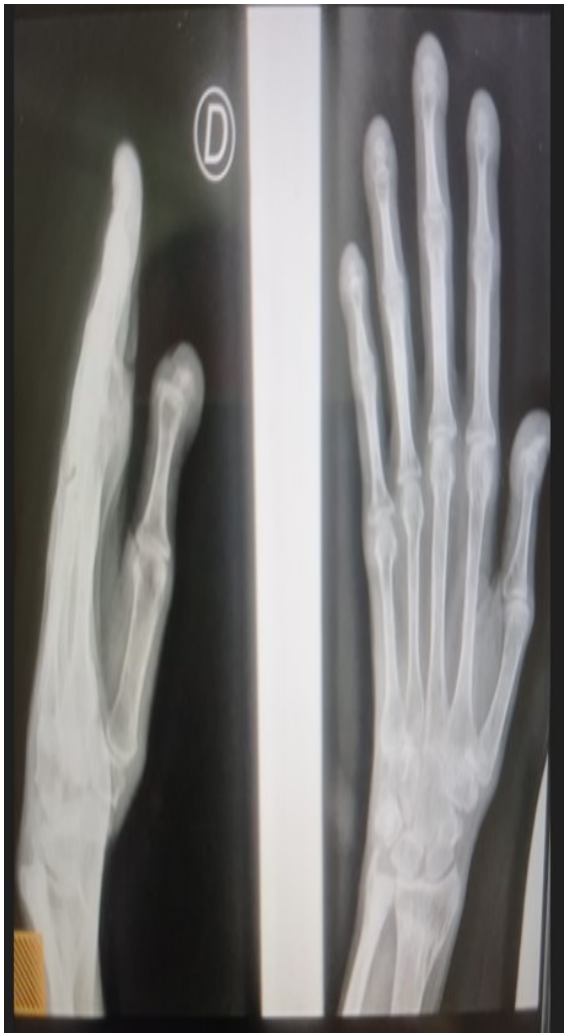
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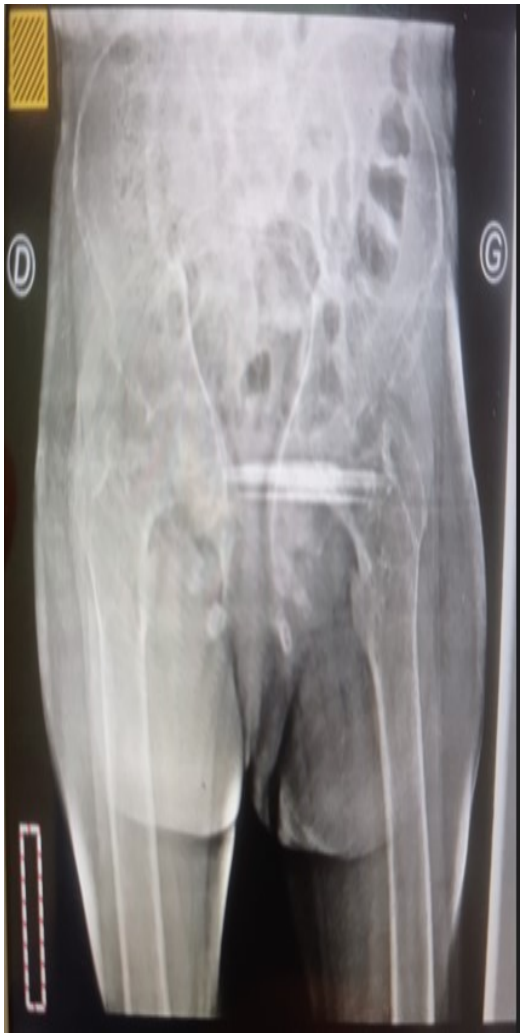
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Evolution:

The patient is in stable condition Still on medical treatment PTX cannot be considered as intubation is impossible.

Poor compliance with treatment since its inception, mainly due to digestive intolerance.

CONCLUSION

Up to the present day in 2023, despite the availability of several medical treatments to prevent the complications of HPT II in haemodialysis patients, this treatment may not be effective and surgical treatment is required, with better results, which must also be indicated in good time.

TNANK YOU

FOR YOUR ATTENTION