Cas Rapportés
et réflexions néphrologiques
en médecine générérale

Néphrologie

DODECA LAEKEN 2012
Quizz médical à visée éducative

Patient n° 1

- Femme de 75 ans,
- Diabète de type II depuis 4 ans, ADO, Hba1c 6,8%, FO normal,
- LDL 94 mg/dL
- GFR 74 ml/min/1,73 m2
- HTA sous contrôle (Anti Calcique-BB).
- Tigelle Protéinurie +

Attitude ?

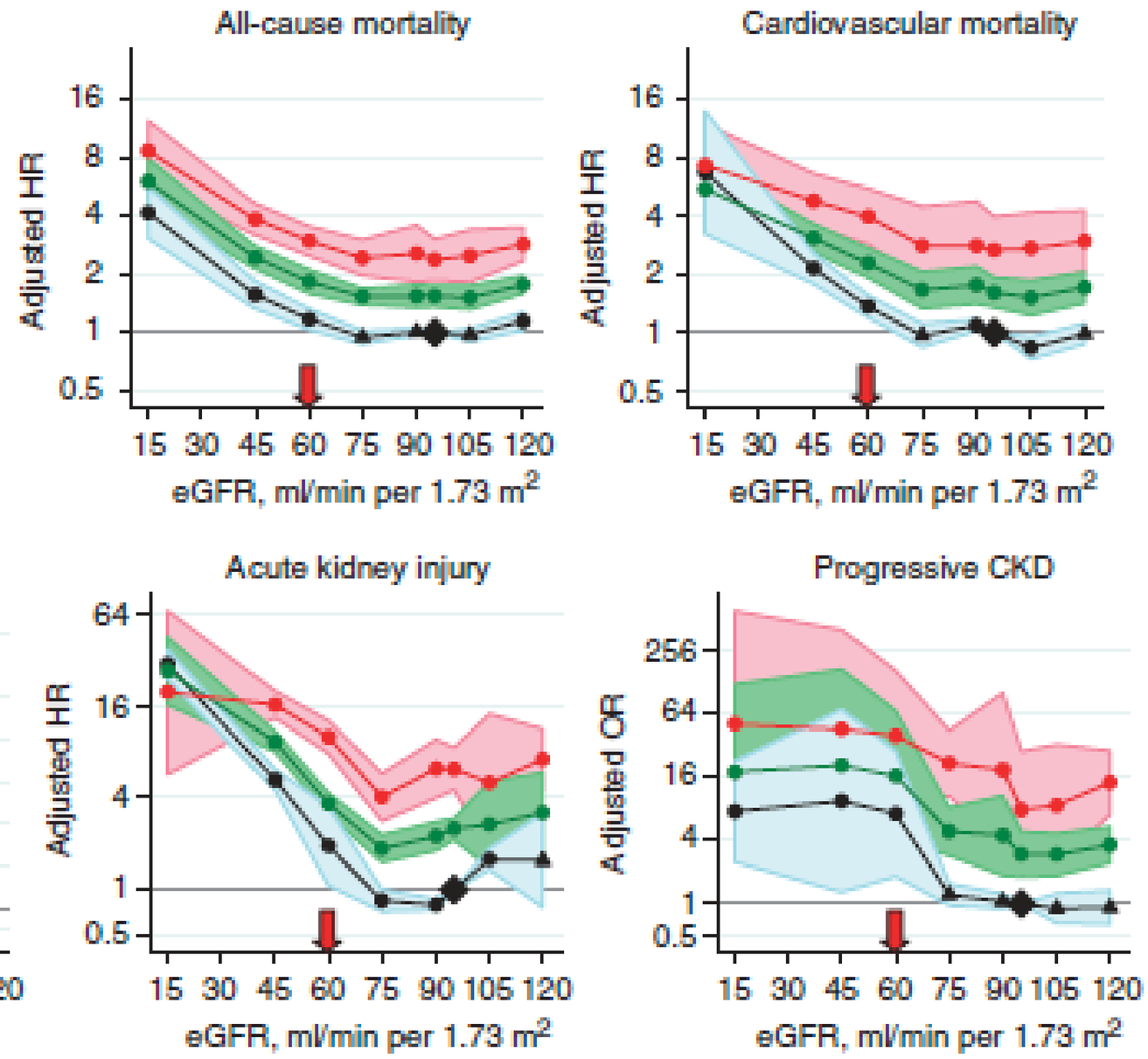
- Dans le doute référer au néphrologue
- GFR > 60 non prédictive et fausse: Ne prendre aucune initiative.
- Faire réaliser une clearance isotopique
- Tigelle Protéinurique +: start IEC
- Urine de 24h dosage protéinurie/albuminurie
- Tigelle en dessous de < ++ non prédictive: prochain contrôle dans six mois.
- Autres ?

a. de la GFR... et la classification

- DFG / GFR: taux de filtration glomérulaire
- MDRD > 60 ml/min/1,73 m2 n’est pas prédictible.
- Gold standard U 24h: Est ce que cela va changer le prise en charge ?

The definition, classification, and prognosis of chronic kidney disease: a KDIGO Controversies Conference report

Summary of relative risks from continuous meta-analysis



Kidney International (2011) **80**, 17–28

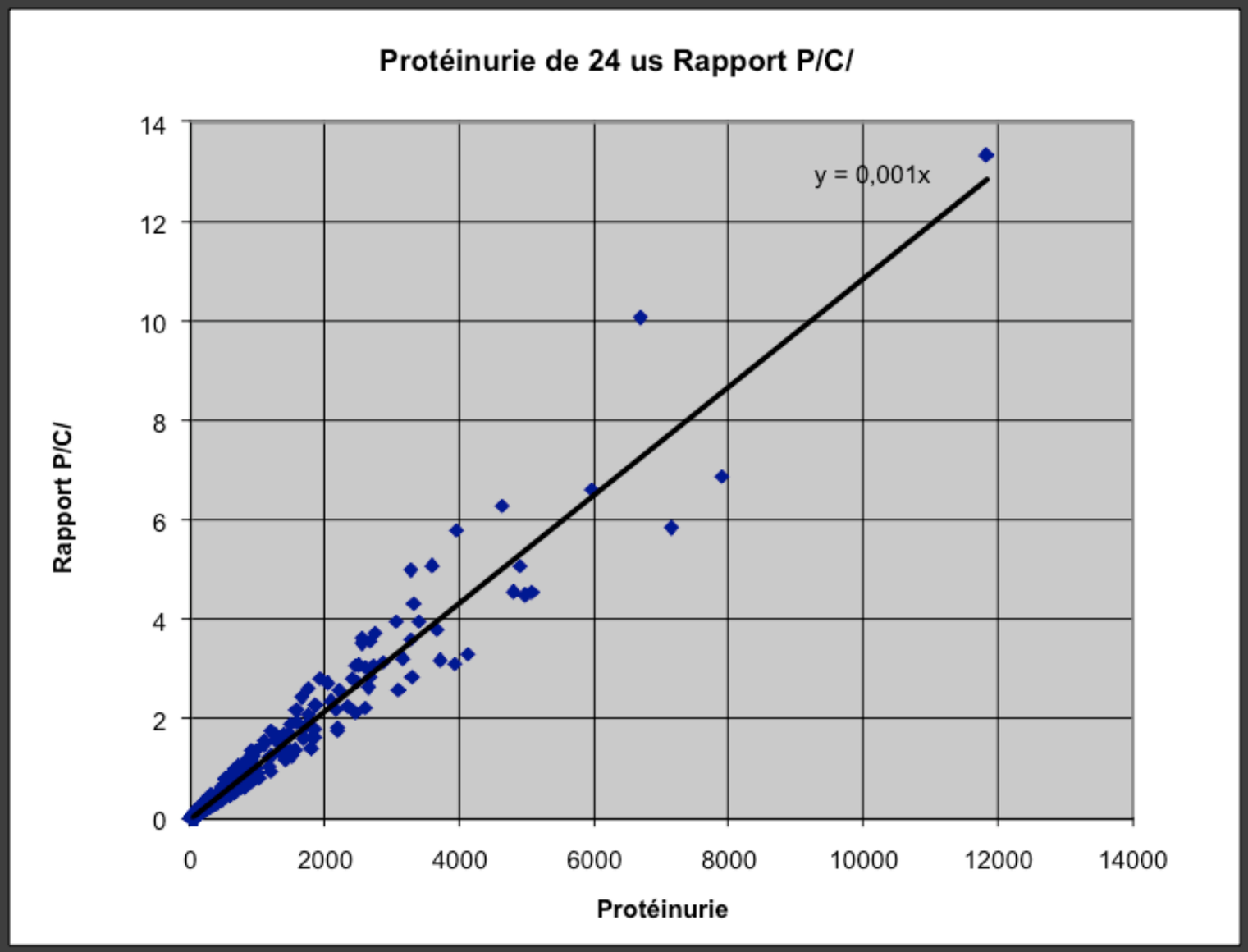
b. tigelle

- moyen de dépistage avec une précision limitée.(défaut de dilution-concentration)
- employer la protéinurie et mieux le ratio soit protéinurie / créatinurie soit le ratio albuminurie / créatinurie.
- Facteur prédictif du risque rénal et cardiovasculaire.

Patient n° 1

RATIO Prot/C ALB/C

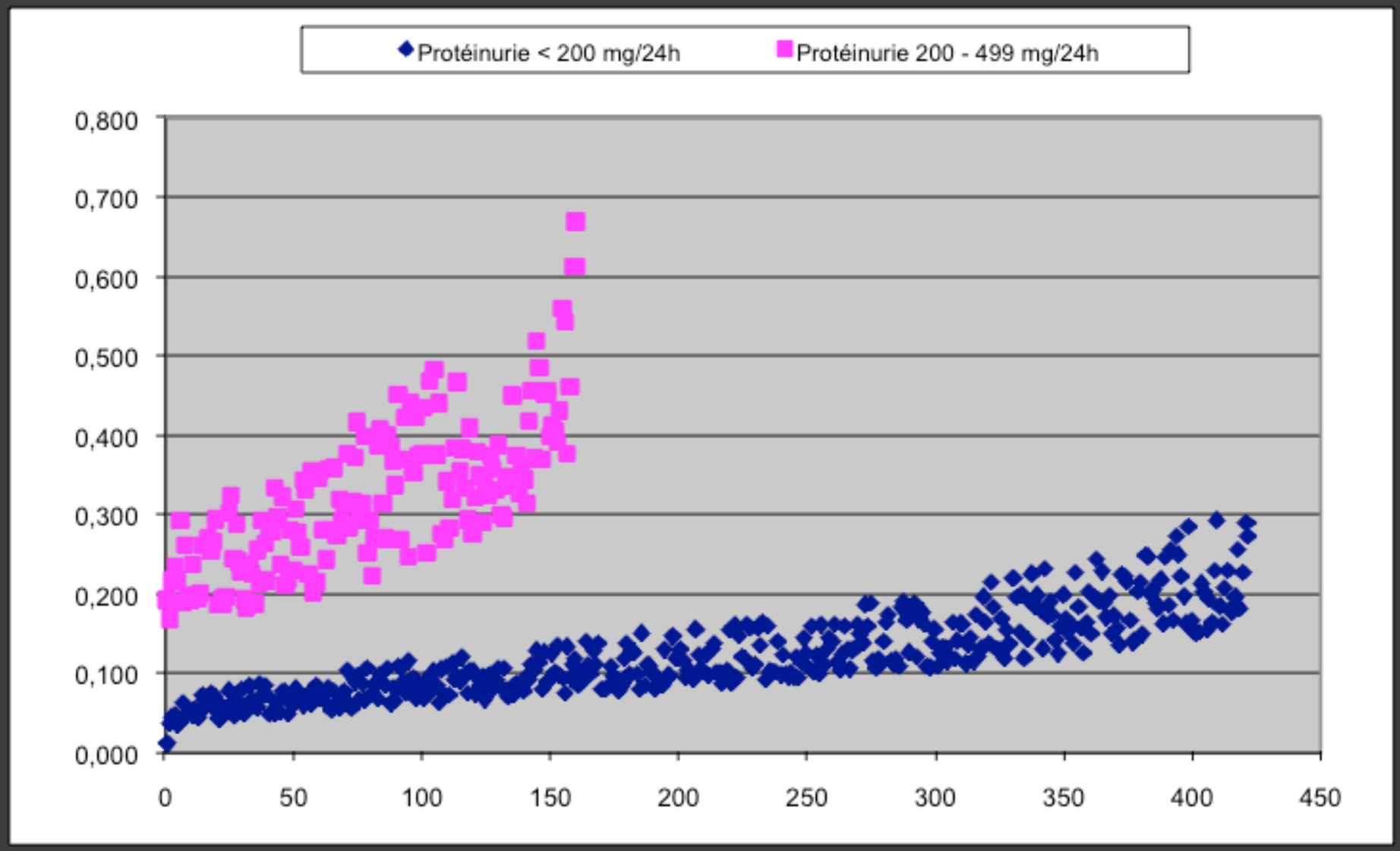
- Analyse 2065 urine de 24h (C H H F)



Patient n° 1

Rapport P/C A/C

Analyse	Physio	+-	+	++	+++
P/C	< 0,3	0,2 - 0,4	0,5-1	1-3	>3
Alb (mg/g)	<10	10-29	30-300	>300	



Composite ranking for relative risks by GFR and albuminuria (KDIGO 2009)

GFR stages, description and range (ml/min per 1.73 m²)			Albuminuria stages, description and range (mg/g)				
			A1		A2	A3	
			Optimal and high-normal		High	Very high and nephrotic	
			<10	10–29	30–299	300–1999	⩾2000
	G1	High and optimal	>105				
			90–104				
	G2	Mild	75–89				
			60–74				
	G3a	Mild-moderate	45–59				
	G3b	Moderate-severe	30–44				
	G4	Severe	15–29				
	G5	Kidney failure	<15				

Age

Comorbidité

All-cause mortality

	ACR <10	ACR 10–29	ACR 30–299	ACR ⩾300
eGFR >105	1.1	1.5	2.2	5.0
eGFR 90–105	Ref	1.4	1.5	3.1
eGFR 75–90	1.0	1.3	1.7	2.3
eGFR 60–75	1.0	1.4	1.8	2.7
eGFR 45–60	1.3	1.7	2.2	3.6
eGFR 30–45	1.9	2.3	3.3	4.9
eGFR 15–30	5.3	3.6	4.7	6.6

Cardiovascular mortality

	ACR <10	ACR 10–29	ACR 30–299	ACR ⩾300
eGFR >105	0.9	1.3	2.3	2.1
eGFR 90–105	Ref	1.5	1.7	3.7
eGFR 75–90	1.0	1.3	1.6	3.7
eGFR 60–75	1.1	1.4	2.0	4.1
eGFR 45–60	1.5	2.2	2.8	4.3
eGFR 30–45	2.2	2.7	3.4	5.2
eGFR 15–30	14	7.9	4.8	8.1

Kidney failure (ESRD)

	ACR <10	ACR 10–29	ACR 30–299	ACR ⩾300
eGFR >105	Ref	Ref	7.8	18
eGFR 90–105	Ref	Ref	11	20
eGFR 75–90	Ref	Ref	3.8	48
eGFR 60–75	Ref	Ref	7.4	67
eGFR 45–60	5.2	22	40	147
eGFR 30–45	56	74	294	763
eGFR 15–30	433	1044	1056	2286

Acute kidney injury (AKI)

	ACR <10	ACR 10–29	ACR 30–299	ACR ⩾300
eGFR >105	Ref	Ref	2.7	8.4
eGFR 90–105	Ref	Ref	2.4	5.8
eGFR 75–90	Ref	Ref	2.5	4.1
eGFR 60–75	Ref	Ref	3.3	6.4
eGFR 45–60	2.2	4.9	6.4	5.9
eGFR 30–45	7.3	10	12	20
eGFR 15–30	17	17	21	29

Progressive CKD

	ACR <10	ACR 10–29	ACR 30–299	ACR ⩾300
eGFR >105	Ref	Ref	0.4	3.0
eGFR 90–105	Ref	Ref	0.9	3.3
eGFR 75–90	Ref	Ref	1.9	5.0
eGFR 60–75	Ref	Ref	3.2	8.1
eGFR 45–60	3.1	4.0	9.4	57
eGFR 30–45	3.0	19	15	22
eGFR 15–30	4.0	12	21	7.7

Patient n° 1: en pratique

- Confirmation de la protéinurie et de la GFR
- Si confirmée:
- <u>FAIRE UN DIAGNOSTIC NEPHROLOGIQUE</u>
  - Imagerie rénale
  - Bilan protéinurique biologique extensif
  - selon bilan nephro-diagnostic PBR.

Patient 2

- Développer Effet protéinurie sur le GFR
- Développer Inhibition axe RAA.

Patient 2

- Homme de 58 ans
- Nephrectomie 2010 pour hypernephrome
- HTA depuis 2005 actuellement 140/90: Nobiten 5, Moxonidine 0,4, Vasexten 10 mg. Clinique pas de signe de surcharge vasculaire. Régime diabetique
- Bilan: créatinine 1,1 mg/dL, protéinurie de 24h : 2900 mg. gly 105 Hba1c 50.
- Imagerie: pas de récidive néoplasique
- Auto Immunité négative, pas de Sd nephrotique, pas de myélome.
- PBR Haut risque: rein unique

Patient 2: Propositions

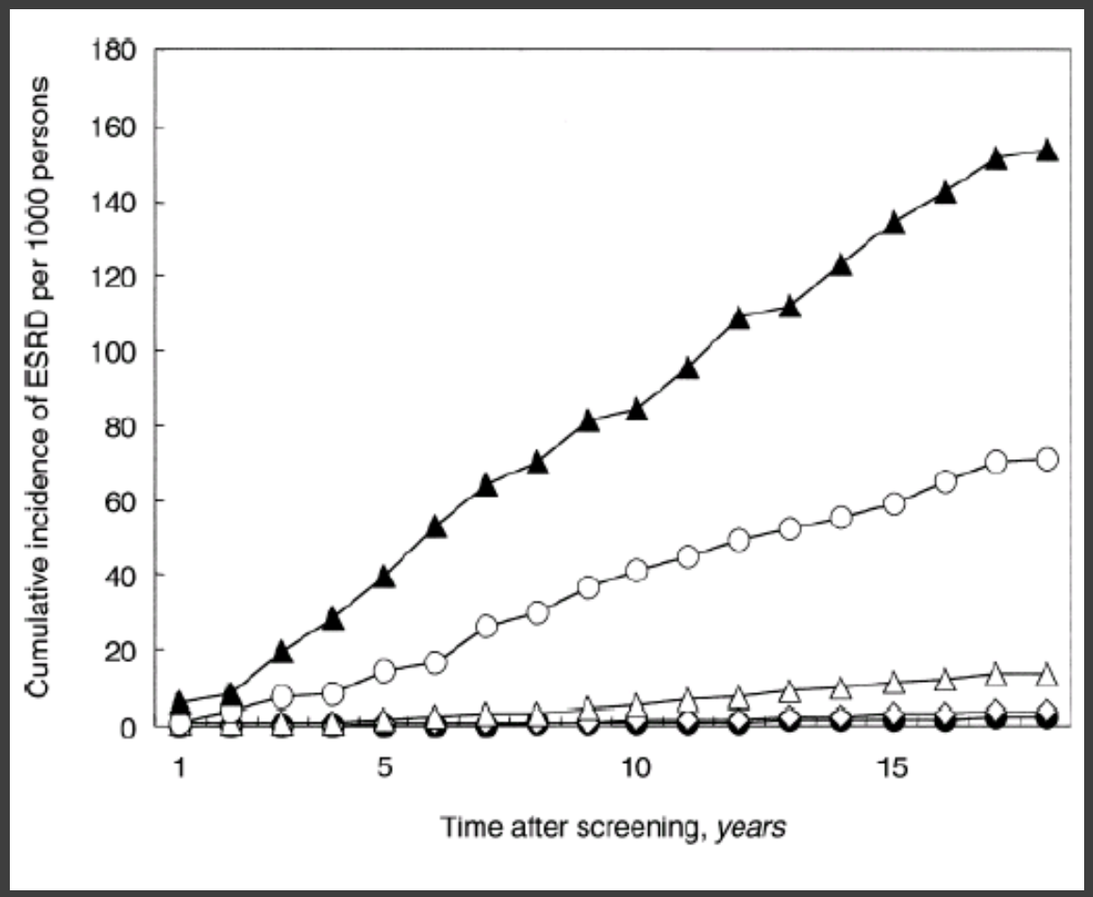
- Biopsie rénale
- Contre Indication à une inhibition de l’axe rénine angiotensine: rein unique
- Indication d’un inhibition de l’axe rénine angiotensine
- Start IEC d’abord
- Start ARBs d’abord
- Start IDR (aliskiren) d’abord
- Idem 2 mais double inhibition nécessaire et souhaitable
- Effet possible de l’anticalcique: arrêt de celui-ci
- Intensification du traitement diabetique
- Autres…

Axe RAAS et Médication

Proteinuria and the risk of developing end-stage renal disease

KUNITOSHI ISEKI, YOSHIHARU IKEMIYA, CHIHO ISEKI, and SHUICHI TAKISHITA

Dialysis Unit and Third Department of Internal Medicine, University of The Ryukyus and Okinawa General Health Maintenance Association, Okinawa, Japan



Composite ranking for relative risks by GFR and albuminuria (KDIGO 2009)				Albuminuria stages, description and range (mg/g)				
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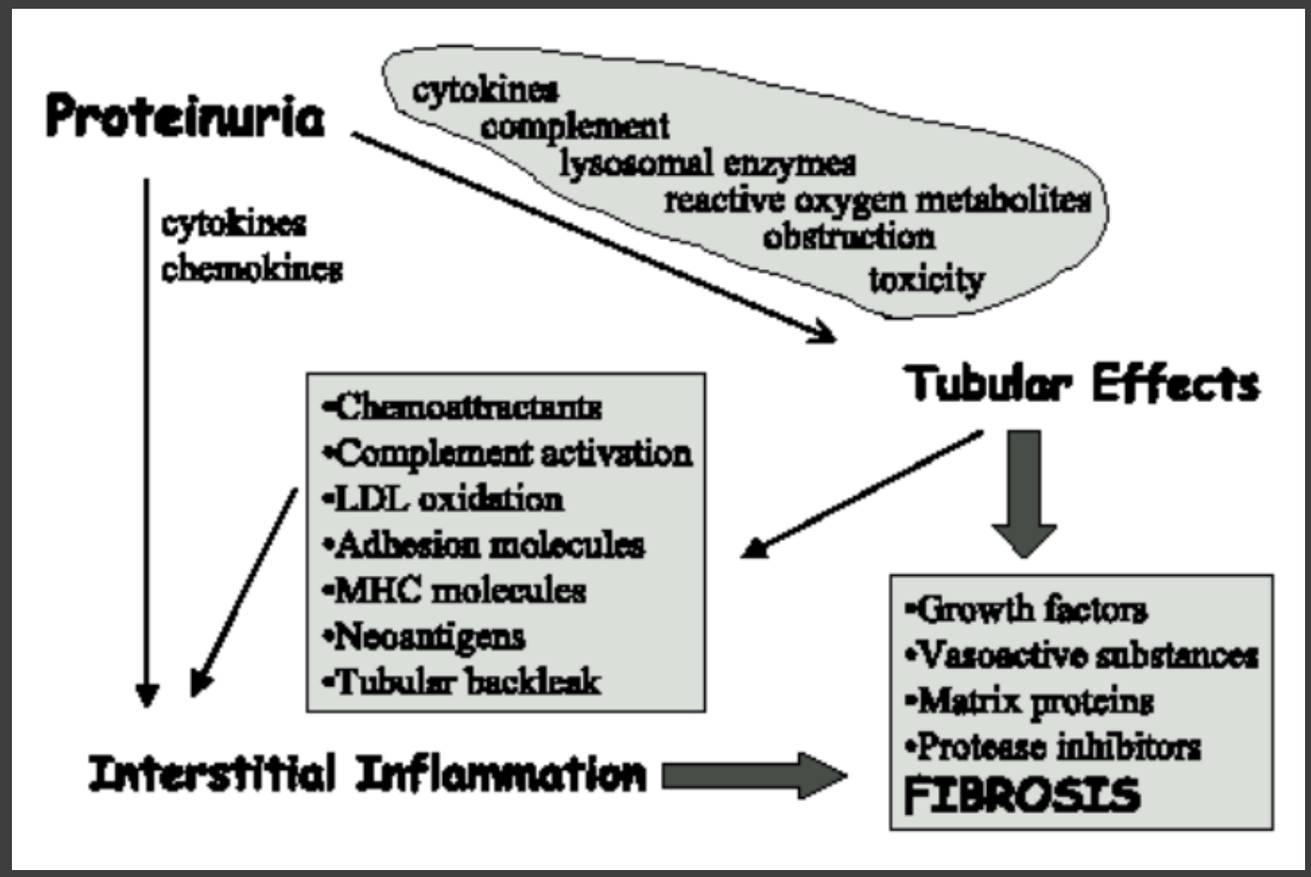
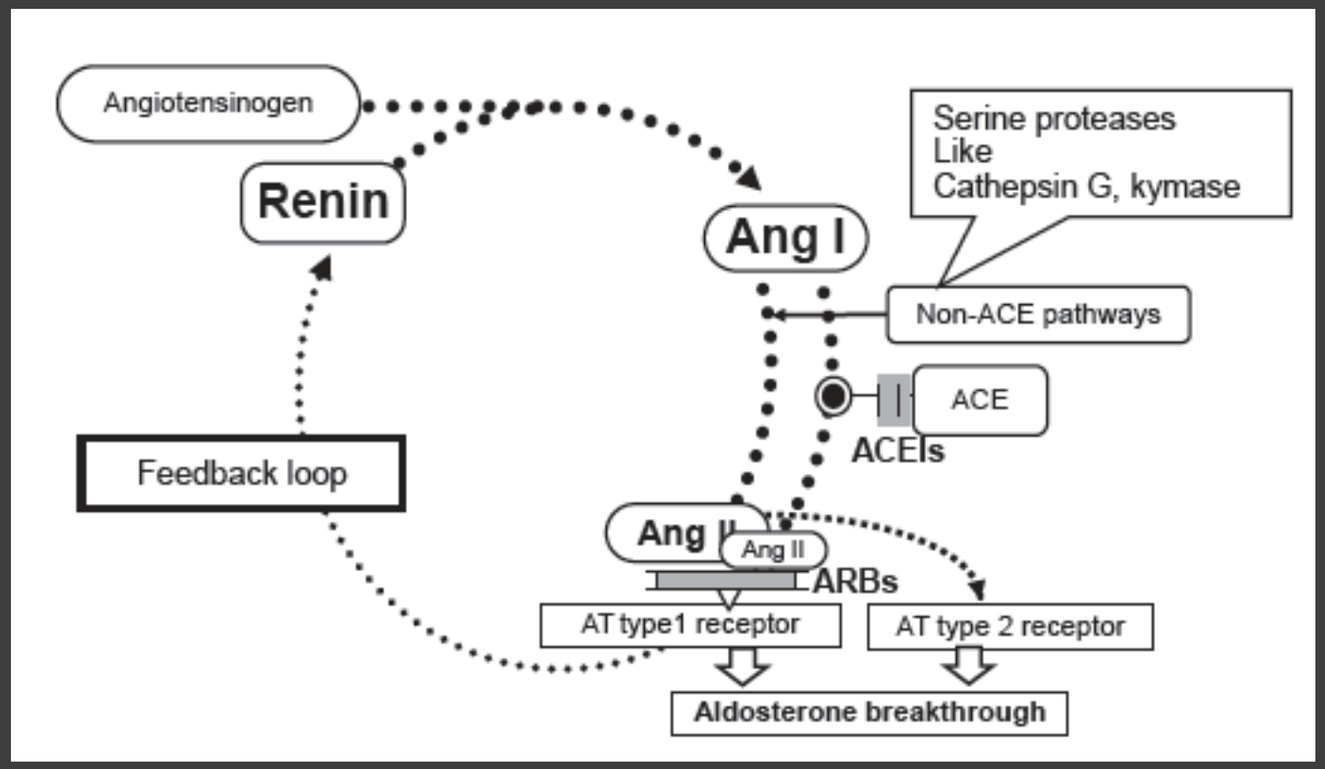
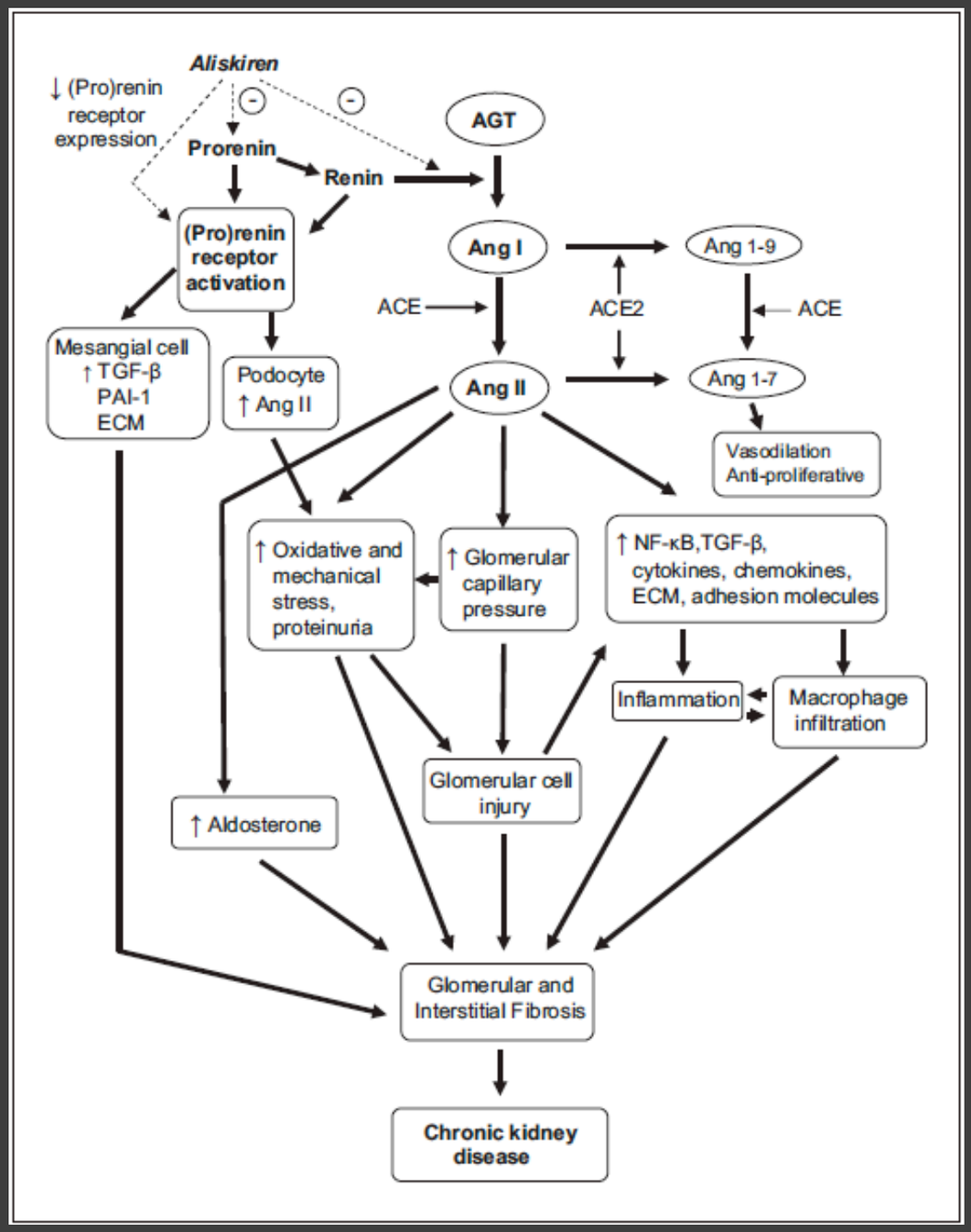


Axe RAAS et Médication





Axe RAAS et Médication

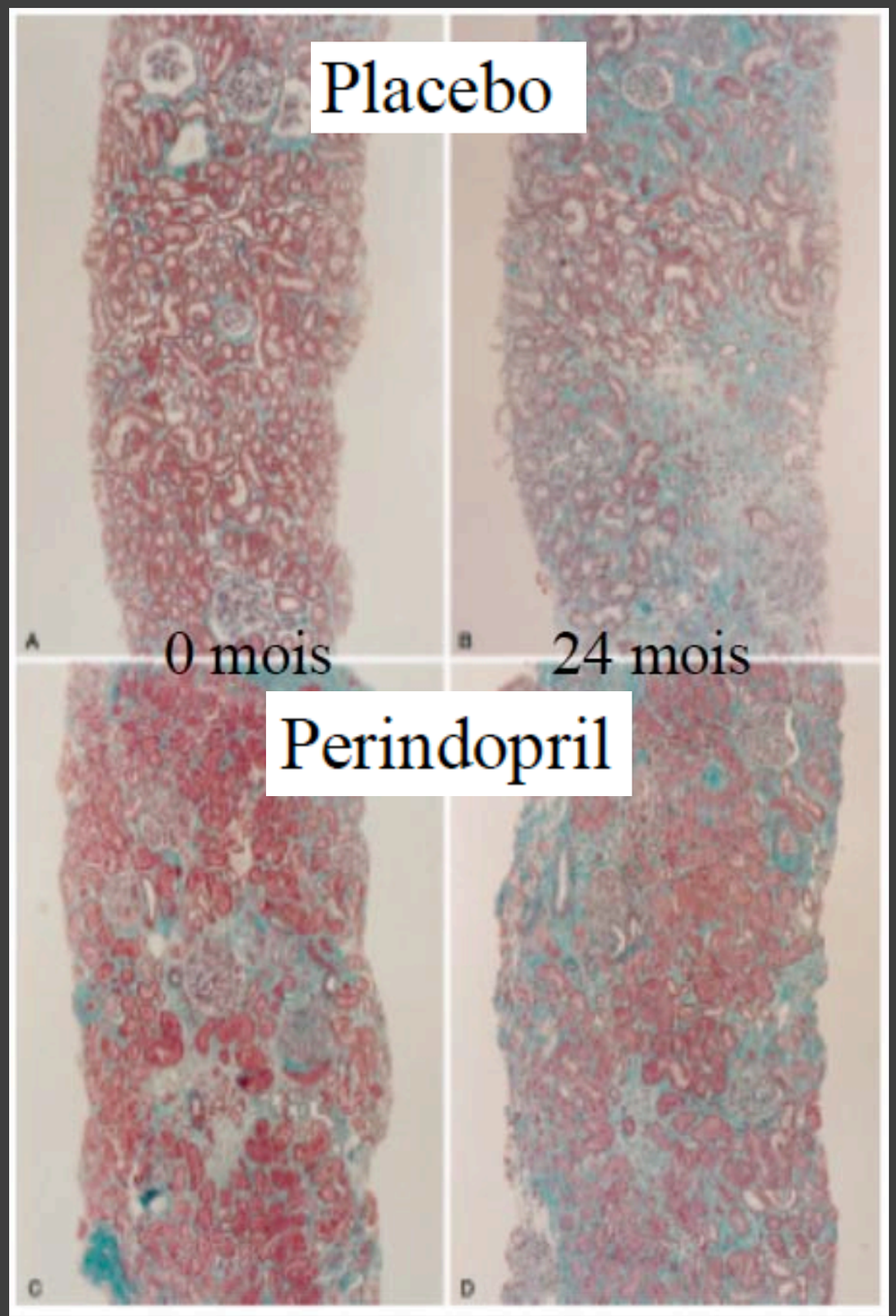
Expansion of Cortical Interstitium Is Limited by Converting Enzyme Inhibition in Type 2 Diabetic patients with Glomerulosclerosis

DANIEL J. CORDONNIER,* NICOLE PINEL,§ CLAIRE BARRO,CLAIRE MAYNARD,* PHILIPPE ZAOUI,* SERGE HALIMI,*BRUNO HURAULT DE LIGNY,† YVES REZNIC,† DOMINIQUE SIMON,‡RUDOLF W. BILOUS,FOR THE DIABIOPSIES GROUPa

J Am Soc Nephrol 10: 1253–1263, 1999

Table 4. Longitudinal renal function[a]

Group	Mean BP (mmHg)		Creatinine Clearance (ml/min)		Proteinuria[b] (mg/24 h)		Urine Na (mmol/24 h)	
	M0	M24	M0	M24	M0	M24	M0	M24
Placebo (n = 10)	109 ± 12	108 ± 10	119 ± 54	102 ± 53	547 (191,1567)	881 (239,3242)	145 ± 75	120 ± 68
Perindopril (n = 9)	101 ± 12	96 ± 11	124 ± 23	109 ± 31	668 (226,1369)	436 (187,1016)	202 ± 136	171 ± 88



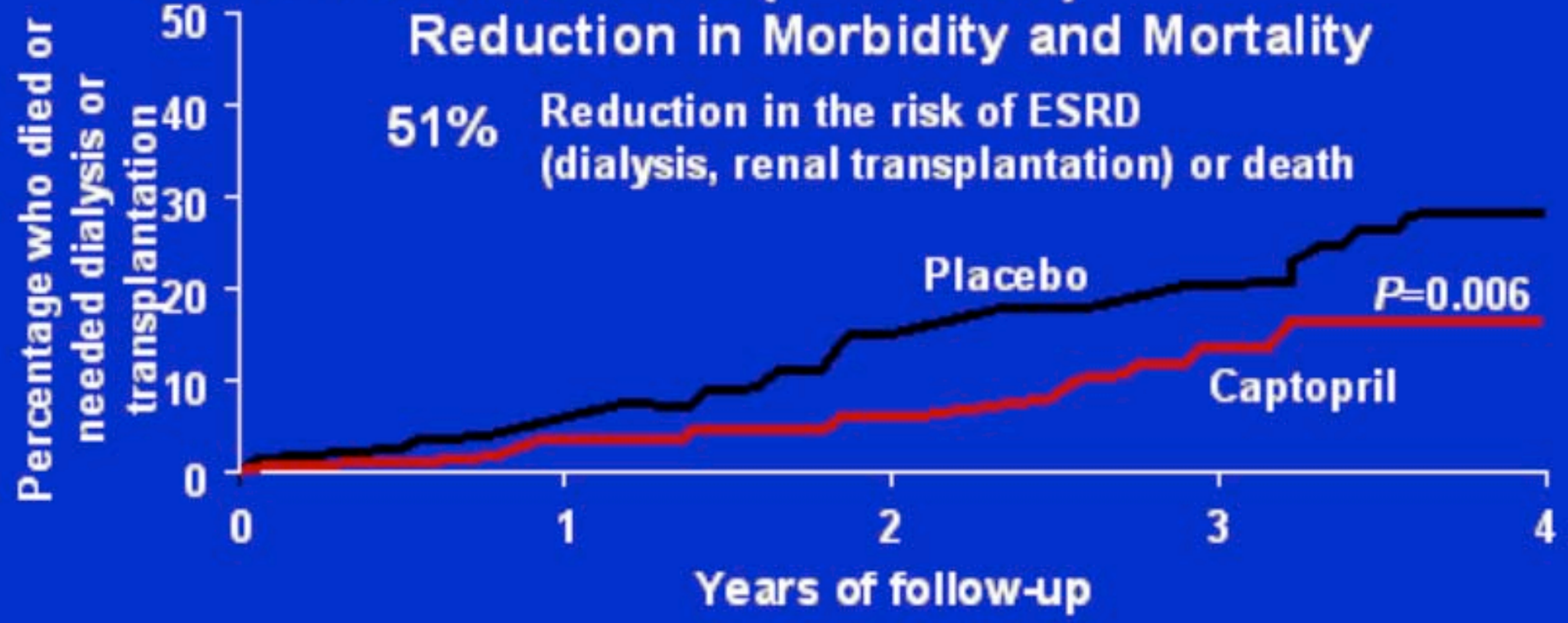
EBM IEC

Effect of ACE Inhibition on Diabetic Kidney Disease

- 48% reduction in the risk of doubling of serum creatinine (*P*=0.007)

Reduction in Morbidity and Mortality

51% Reduction in the risk of ESRD (dialysis, renal transplantation) or death



Lewis et al. N Engl J Med. 1993;329:1456.

Patient n° 2

EBM ARBs

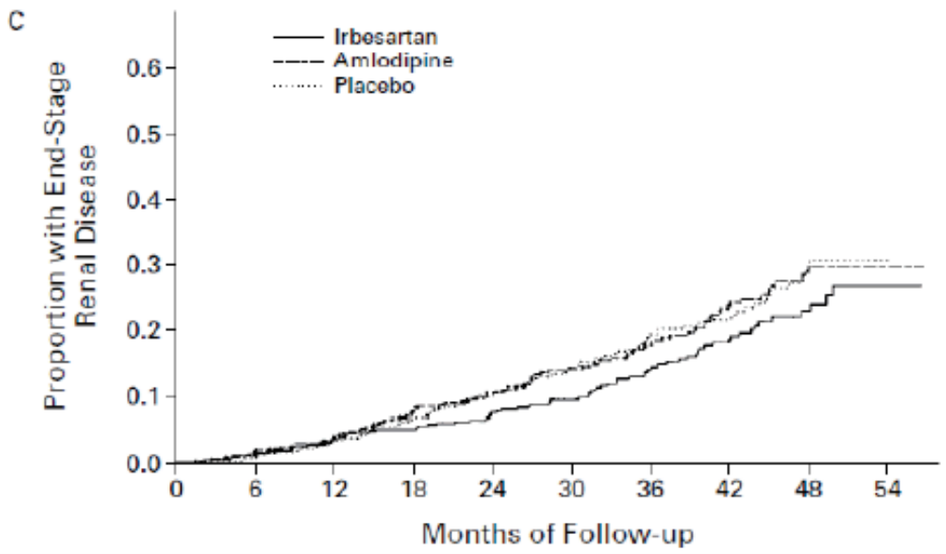
VOLUME 345 SEPTEMBER 20, 2001 NUMBER 12

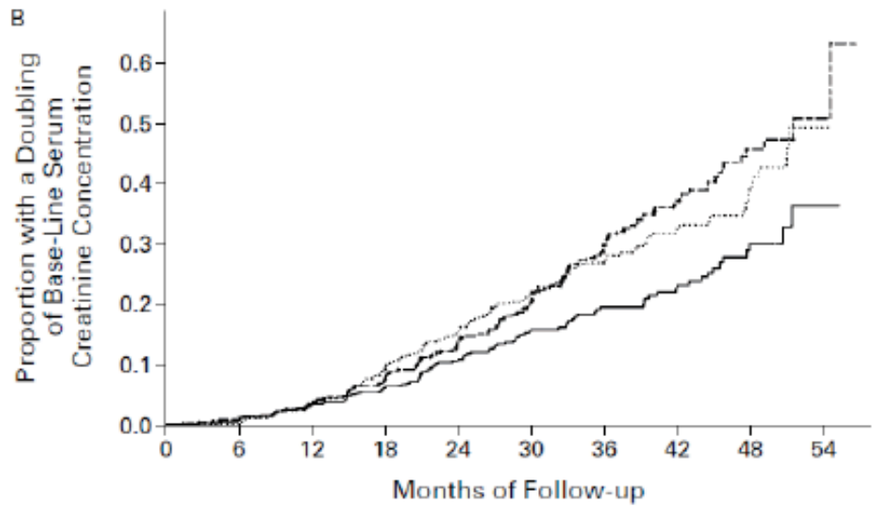
RENOPROTECTIVE EFFECT OF THE ANGIOTENSIN-RECEPTOR ANTAGONIST IRBESARTAN IN PATIENTS WITH NEPHROPATHY DUE TO TYPE 2 DIABETES

EDMUND J. LEWIS, M.D., LAWRENCE G. HUNSICKER, M.D., WILLIAM R. CLARKE, PH.D., TOMAS BERL, M.D., MARC A. POHL, M.D., JULIA B. LEWIS, M.D., EBERHARD RITZ, M.D., ROBERT C. ATKINS, M.D., RICHARD ROHDE, B.S., AND ITAMAR RAZ, M.D., FOR THE COLLABORATIVE STUDY GROUP*

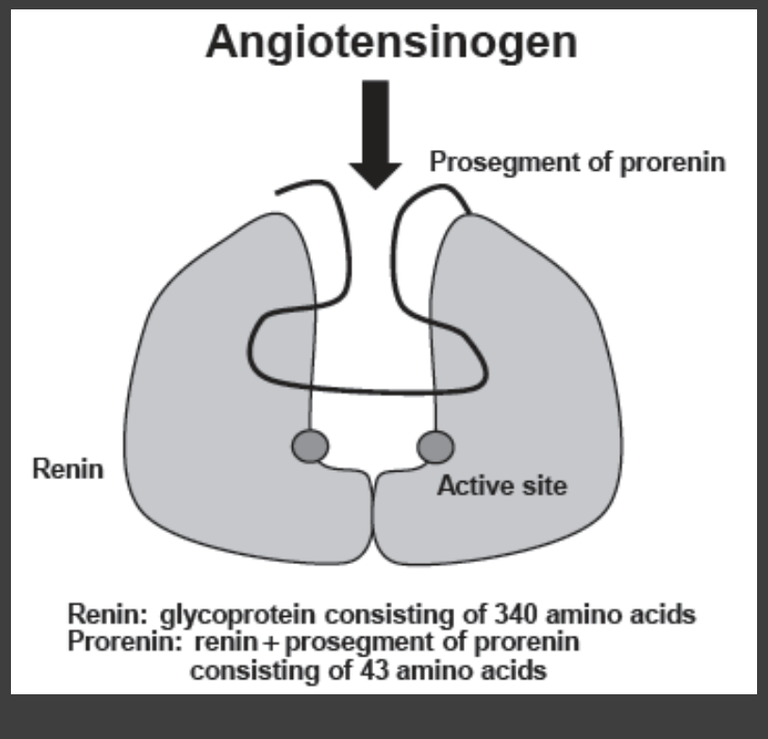
TABLE 1. BASE-LINE CHARACTERISTICS OF THE PATIENTS.*

CHARACTERISTIC	IRBESARTAN GROUP (N=579)	AMLODIPINE GROUP (N=567)	PLACEBO GROUP (N=569)	P VALUE





EBM IDR



Study design	Population	Treatment	Outcome	Reference
Open-label, single-center (n = 15)	mean age 63.5 years, 87% male, 100% white, UACR 173 mg/g, 24 h maBP 143/75 mm Hg	300 mg aliskiren for 28 days	UACR decreased by 44% vs. baseline ($p < 0.001$). maBP decreased ($p = 0.035$)	Persson et al. [50]
Randomized, double-blind, multicenter (n = 599)	mean age 59.8–61.8 years, 68.4–74.2% male, 86.0–87.6% white, UACR 513–553 mg/g	losartan 100 mg vs. losartan 100 mg and aliskiren 150 mg for 3 months titrated to 300 mg for 3 months	UACR decreased by 20% with aliskiren/losartan vs. losartan ($p < 0.0001$) with no significant differences in BP	AVOID Trial Parving et al. [59]
Randomized, double-blind, crossover, single-center (n = 26)	mean age 59.8 years, 77% male, 100% white, UAER 275 mg/day	placebo, 300 mg aliskiren, 300 mg irbesartan or 300 mg aliskiren/ 300 mg irbesartan for 2 months	change in UAER (%) vs. placebo aliskiren –48%, irbesartan –58% aliskiren/irbesartan –71%	Persson et al. [51]

BP = Blood pressure; maBP = mean ambulatory blood pressure; UACR = urinary albumin creatinine ratio; UAER = urinary albumin excretion rate.

EBM IRMC

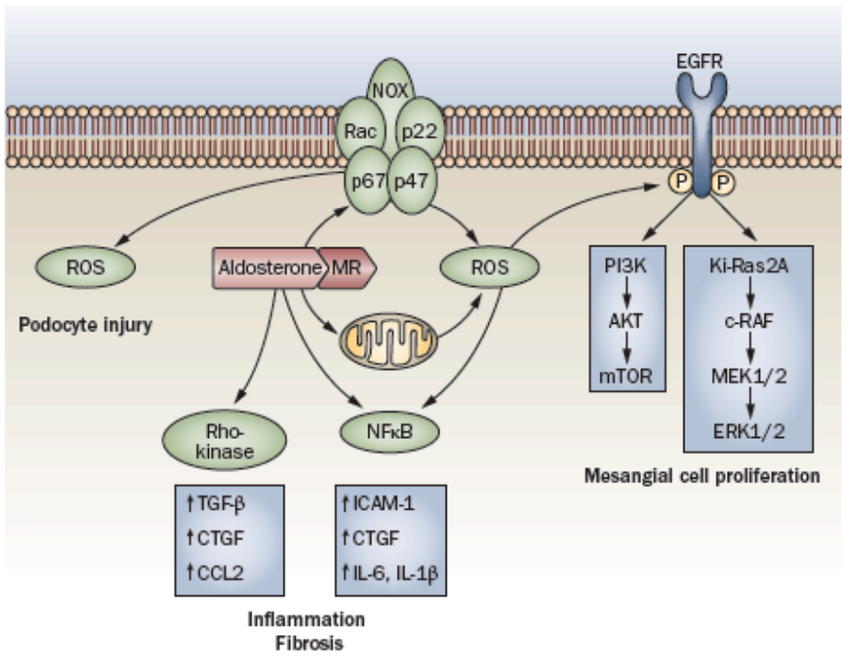


Figure 2 | Mechanisms of aldosterone-induced kidney injury. Aldosterone acts via the mineralocorticoid receptor to induce the production of ROS either by the stimulation of NADPH oxidase or via mitochondria. ROS induce podocyte damage, inflammation and fibrosis. Aldosterone activates PI3K–AKT and MAPK via the mineralocorticoid receptor and transactivation of EGFR, which leads to mesangial cell proliferation. Abbreviations: MR, mineralocorticoid receptor; P, phosphorylation; ROS, reactive oxygen species.

Table 1 | Interventional studies with mineralocorticoid receptor antagonism in chronic kidney disease

Authors	Design	*n*	Follow-up (weeks)	Type of patient	Interventions	Outcomes	Main result
Schjoedt *et al.* (2005)[66]	Crossover, randomized, double-blind	20	8	Type I diabetes Albuminuria >300 mg daily GFR >30 ml/min/1.73 m²	SPIRO 25 mg daily + ARB vs placebo + ARB	Albuminuria GFR BP Kalemia	–30% albuminuria with SPIRO vs placebo, independently of BP, no change in GFR
Rossing *et al.* (2005)[67]	Crossover, randomized, double-blind	22	8	Type II diabetes Albuminuria >300 mg daily GFR >30 ml/min/1.73 m²	SPIRO 25 mg daily + ARB vs placebo + ARB	Albuminuria GFR BP Serum K Kalemia	–33% albuminuria with SPIRO vs placebo, independently of BP, no change in GFR
Chrysostomou *et al.* (2006)[68]	Randomized, double-blind, placebo-controlled	41	12	CKD Albuminuria >1.5 g daily Creatinine <200 µmol/l ACEi >6 months	Ramipril vs ramipril + irbesartan vs ramipril + SPIRO 25 mg daily vs ramipril + irbesartan + SPIRO 25 mg daily	Albuminuria GFR BP Kalemia	–1.4% albuminuria, group 1 –16% albuminuria, group 2 –42% albuminuria, group 3 –48% albuminuria, group 4 No change in eGFR, BP
Bianchi *et al.* (2006)[64]	Randomized, open-labeled	165	52	CKD Albuminuria >1 g daily eGFR >30 ml/min/1.73 m² Without systemic disease ACEi or ARB >1 year	SPIRO 25 mg daily + ARB or ACEi vs ARB or ACEi	Albuminuria GFR BP Kalemia	–54% albuminuria with SPIRO vs conventional therapy, independently of BP, eGFR slope lower in SPIRO group
Meiracker *et al.* (2006)[65]	Randomized, double-blind, placebo-controlled	59	52	Type II diabetes Albuminuria >300 mg daily Creatinine <156 µmol/l	Usual patient treatment + SPIRO 50 mg daily vs usual patient treatment + placebo	Albuminuria GFR BP Kalemia	–44% albuminuria with SPIRO, no change with placebo, independently of BP, GFR slope higher in SPIRO group
Schjoedt *et al.* (2006)[69]	Crossover, randomized, double-blind	20	8	Type I or II diabetes Albuminuria >2.5 g daily eGFR >30 ml/min/1.73 m²	Usual patient treatment + SPIRO 25 mg daily vs usual patient treatment + placebo	Albuminuria GFR BP Kalemia	–32% albuminuria with SPIRO vs placebo, independently of BP, no change in eGFR
Epstein *et al.* (2006)[70]	Randomized, double-blind, placebo-controlled	268	12	Type II diabetes UACR >50 mg/g eGFR >70 ml/min/1.73 m²	Enalapril vs enalapril + eplerenone 50 mg daily vs enalapril + eplerenone 100 mg daily	Albuminuria BP Kalemia	–7.4% UACR in placebo group –41% in EPL 50 group –48.4% in EPL 100 group Independently of BP
Furumatsu *et al.* (2008)[71]	Randomized open-labeled, controlled	32	52	CKD eGFR >30 ml/min/1.73 m² Losartan 50 mg and enalapril 5 mg >12 weeks Albuminuria >0.5 g daily	Enalapril + losartan + SPIRO 25 mg vs enalapril + losartan + diuretic	Albuminuria GFR BP Kalemia	–58% albuminuria in SPIRO group, no change in control group, no change in eGFR, no change in kalemia
Tylicki *et al.* (2008)[72]	Crossover randomized open-labeled	18	24	Nondiabetic CKD eGFR >45 ml/min/1.73 m² Albuminuria >0.3 g daily	Telmisartan + cilazapril + SPIRO 25 mg vs Telmisartan + cilazapril	Albuminuria GFR BP	–55% albuminuria in SPIRO vs control group, no change in BP

Abbreviations: ACEi, angiotensin-converting-enzyme inhibitor; ARB: angiotensin-receptor blocker; BP, blood pressure; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; EPL, eplerenone; SPIRO, spironolactone; UACR, urinary albumin:creatinine ratio.

EBM : IEC +- ARBs +- IDR +- IRMC

- “PETITES ETUDES TRES CIBLEES” encourageante.
- “Grandes ETUDES avec Large population:
  - HTA: échec
  - Post Infar: > 40 % FE echec
  - Kidney Disease: echec

- Problème majeur : E.S.:
  - Hypotension
  - Hyperkaliémie
  - Ins Rénale Aigue

Combination inhibition of the renin–angiotensin system: is more better?

Table 4 | Summary of recommendations based on clinical evidence for the use of dual blockade of the renin–angiotensin system with ace inhibitors and angiotensin receptor blockers for cardiovascular and chronic kidney disease

Cardiovascular disease
- Hypertension
  - Level of evidence D: No clinical evidence that supports recommendation for combination therapy in hypertension
- Congestive heart failure
  - Preserved ejection fraction
    - Level of evidence D: No clinical evidence that supports recommendation for combination therapy
  - Reduced ejection fraction
    - Level of evidence B: No clinical evidence that supports recommendation for combination therapy for all-cause mortality, consideration for combination therapy to reduce hospitalization for congestive heart failure or reduce cardiovascular death
- Ischemic heart disease
  - Preserved ejection fraction
    - Level of evidence D: No clinical evidence that supports recommendation for combination therapy
  - Reduced ejection fraction
    - Level of evidence D: No clinical evidence that supports recommendation for combination therapy

Chronic kidney disease
- Diabetic kidney disease
  - Microalbuminuria
    - Level of evidence I: Limited clinical evidence that supports recommendation for combination therapy
  - Macroalbuminuria
    - Level of evidence I: Limited clinical evidence that supports recommendation for combination therapy and awaiting further clinical trial evidence for guidance
- Non-diabetic kidney disease
  - Proteinuria < 500 mg/day
    - Level of evidence I: Limited clinical evidence that supports recommendation for combination therapy and awaiting further clinical trial evidence
  - Proteinuria $\geqslant 500$ mg/day
    - Level of evidence C: Limited clinical evidence that supports recommendation for combination therapy but favors use while awaiting further clinical trial evidence

Level of evidence based on the US Preventive Services Task Force. Level A: good scientific evidence suggests that the benefits of the clinical service substantially outweigh the potential risks. Clinicians should discuss the service with eligible patients. Level B: at least fair scientific evidence suggests that the benefits of the clinical service outweigh the potential risks. Clinicians should discuss the service with eligible patients. Level C: at least fair scientific evidence suggests that there are benefits provided by the clinical service, but the balance between benefits and risks are too close for making general recommendations. Clinicians need not offer it unless there are individual considerations. Level D: at least fair scientific evidence suggests that the risks of the clinical service outweigh potential benefits. Clinicians should not routinely offer the service to asymptomatic patients. Level I: Scientific evidence is lacking, of poor quality, or conflicting, such that the risk versus benefit balance cannot be assessed. Clinicians should help patients understand the uncertainty surrounding the clinical service.

Kidney International (2011) **80**, 245–255

Patient n° 2

Patient 2 en pratique

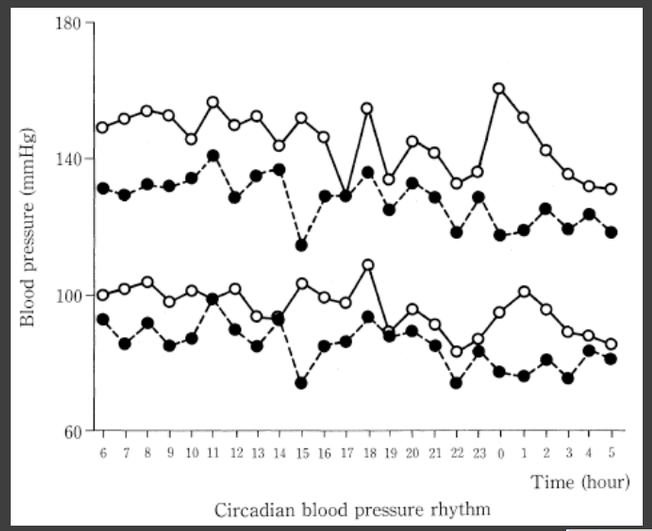
- Si bilan biopsie rénale négative
- 1. START IEC --> ARBS --> IDR: Objectif 125/75 mmHg
- 2. Diminution Apport sodé et si HTA discussion diurétique
- 3. Si échec discussion double inhibition mais risque Hyperkaliémie et nécessité de haute compliance
- 4. Contrôle vitaminique D
- 5. relire l’anapath du rein nephrectomisé

Vitamine D et RAAS

Internal Medicine Vol. 38, No. 1 (January 1999)

Table 2. Calcium, Hormones, and Renal Function before and during Supplementation

	before supplementation	during supplementation
Supine position		
Albumin corrected calcium (mg/dl)	7.0	8.5
Pi (mg/dl)	3.6	4.1
Intact-PTH (pg/ml)	290	23
Plasma renin activity (ng/ml/h)	2.6	0.9
Glomerular filtration rate (ml/min)	130	125
Renal plasma flow (ml/min)	470	429
Fractional excretion of sodium (%)	1.0	1.2
Standing position		
Plasma renin activity (ng/ml/h)	5.5	2.3



Circadian blood pressure rhythm

The NEW ENGLAND JOURNAL of MEDICINE

MEDICAL PROGRESS

Vitamin D Deficiency

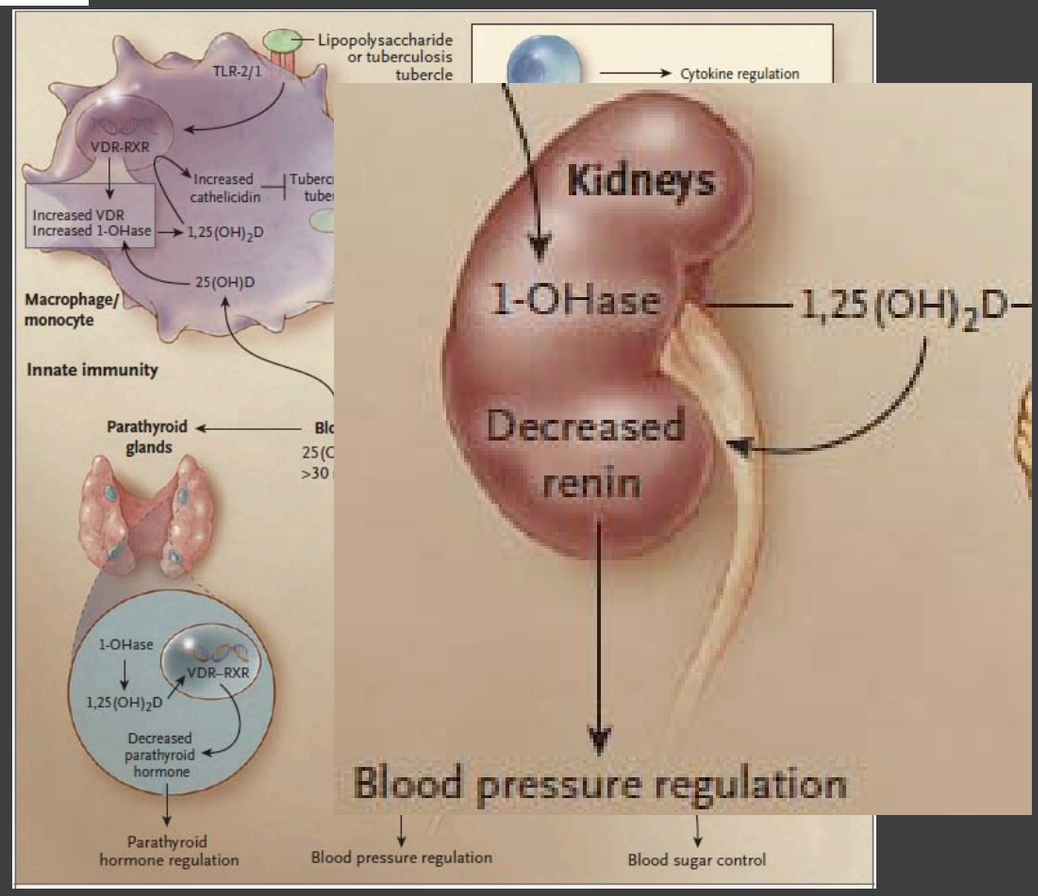
Michael F. Holick, M.D., Ph.D.

N ENGL J MED 357;3 WWW.NEJM.ORG JULY 19, 2007

1,25-Dihydroxyvitamin D_3 is a negative endocrine regulator of the renin-angiotensin system

[1]Department of Medicine, University of Chicago, Chicago, Illinois, USA
[2]Department of Anesthesiology, Washington University School of Medicine, St. Louis, Missouri, USA
[3]Department of Biomedical Engineering, Northwestern University, Evanston, Illinois, USA

J. Clin. Invest. **110**:229–238 (2002). doi:10.1172/JCI200215219.



Patient n° 2

patient 3

- Homme 72 ans, BPCO post Tabac (24 UAP), BMI 31, CKD IIIb (GFR 49 créat 1,7mg/dL), Diab type II - ADO. compliant
- Amlodipine 10- Valsartan 100 - Atorvastatine 40 - MTFmx 500*3 - AAS 100
- Visite de contrôle en MG trimestrielle
- Plainte: Dyspnée modérée à l'effort.

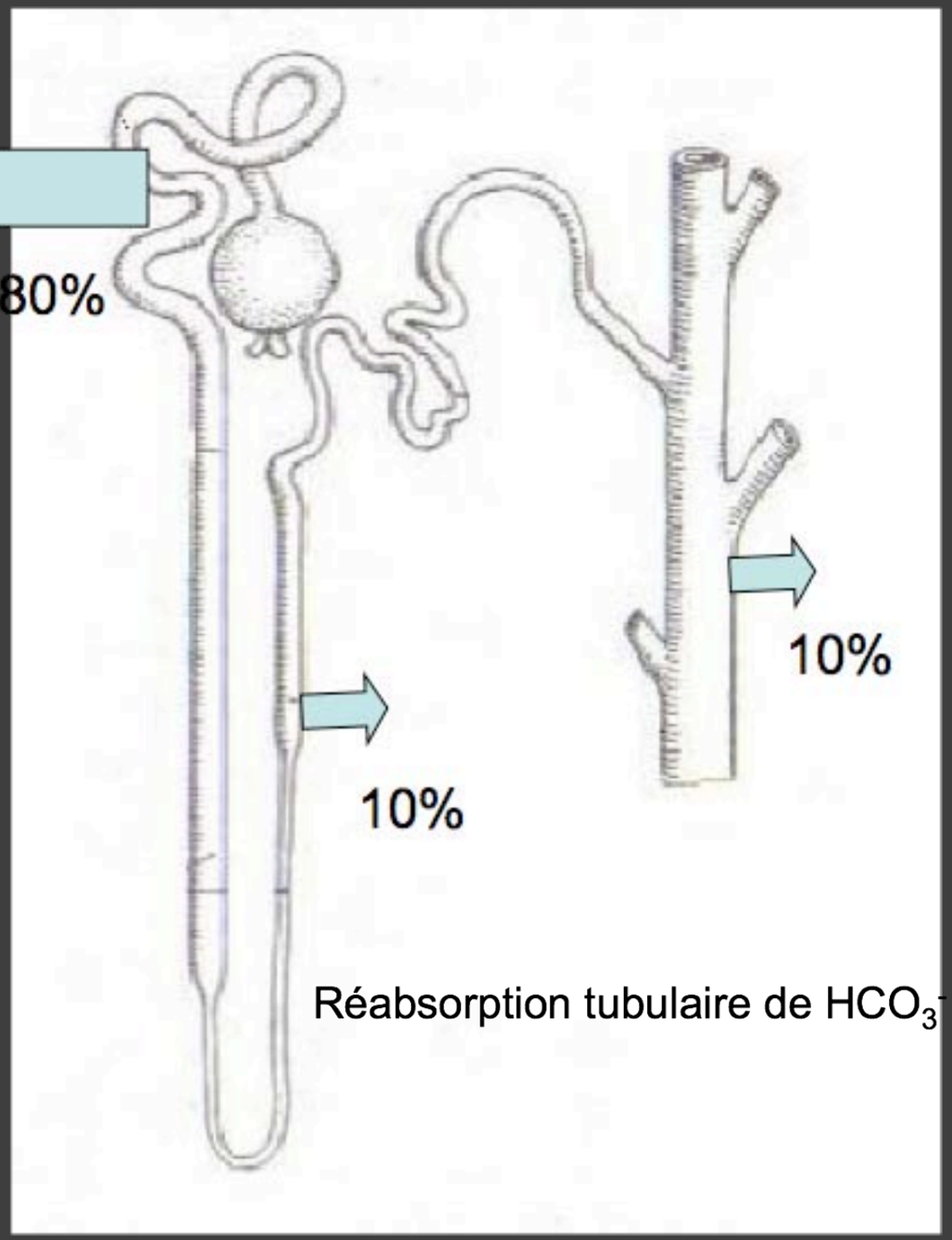
BILAN

Anamnèse	Clinique	Biologie
- Dyspnée IIb - Pas de toux - Pas de symptôme d'angor - Pas d'OMI	TA 145 / 88 Jugulaires - OMI - Coeur: b1b2 85/min Poumon: FRr 20, BRN, VV +, Perc normale, ampliation normale Pâleur: non	créat: 1,7 --> 1,9 Na 138, K 5,3, Ca 9,4, P 4,7 Hb: 12,9 (stable) Hba1c 7,3 % (us 6,7 %) Protéinurie negative

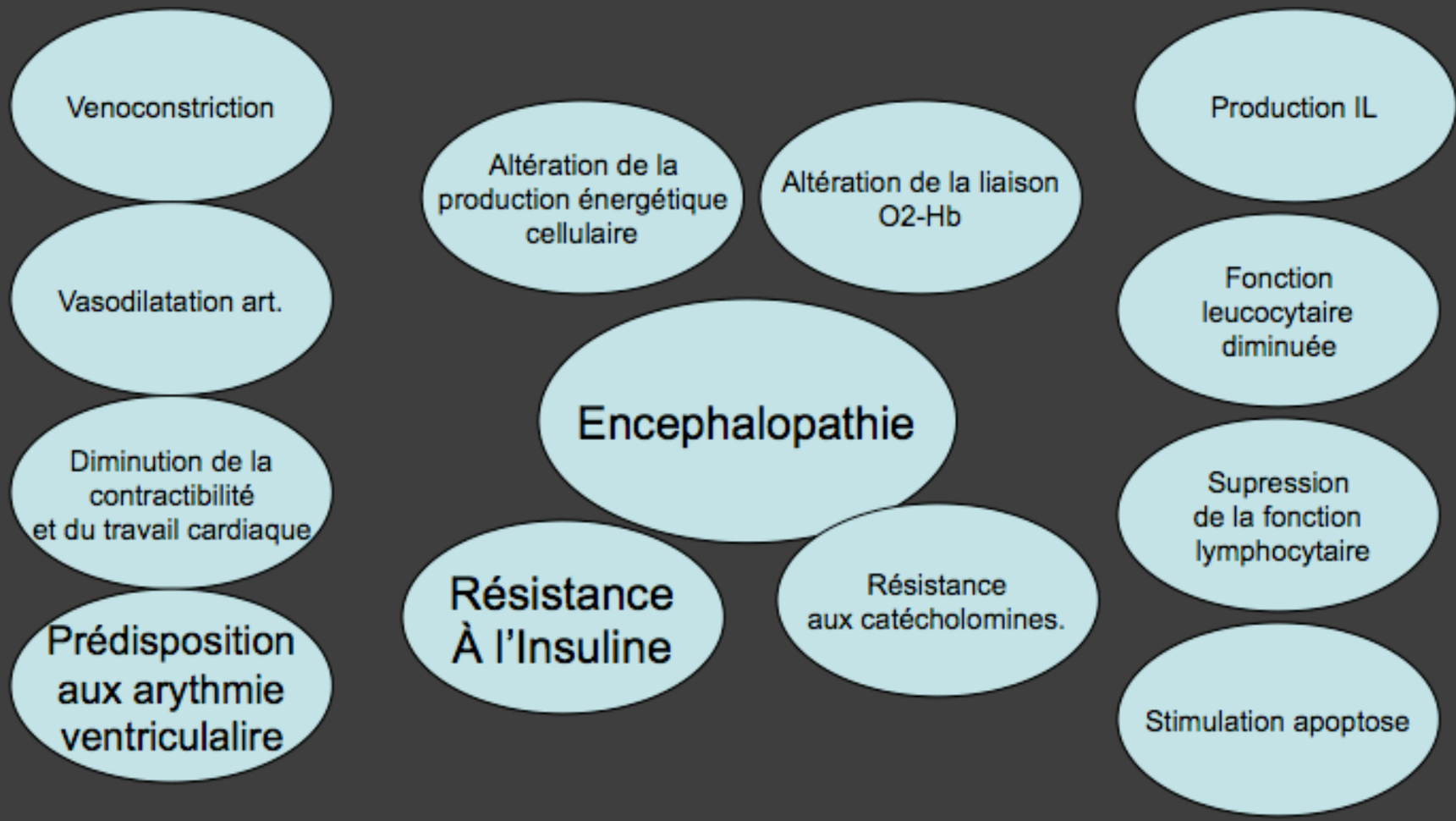
PLAN ?

- ECG ?
- Echo Coeur ?
- Rx Thorax ?
- EFR ?
- Arrêter la MTFmx ?
- Autres ?
- R/ alprazolam...
- Dosage Hb.
- Angio CT poumon

ACIDOSE - Physiopathologie

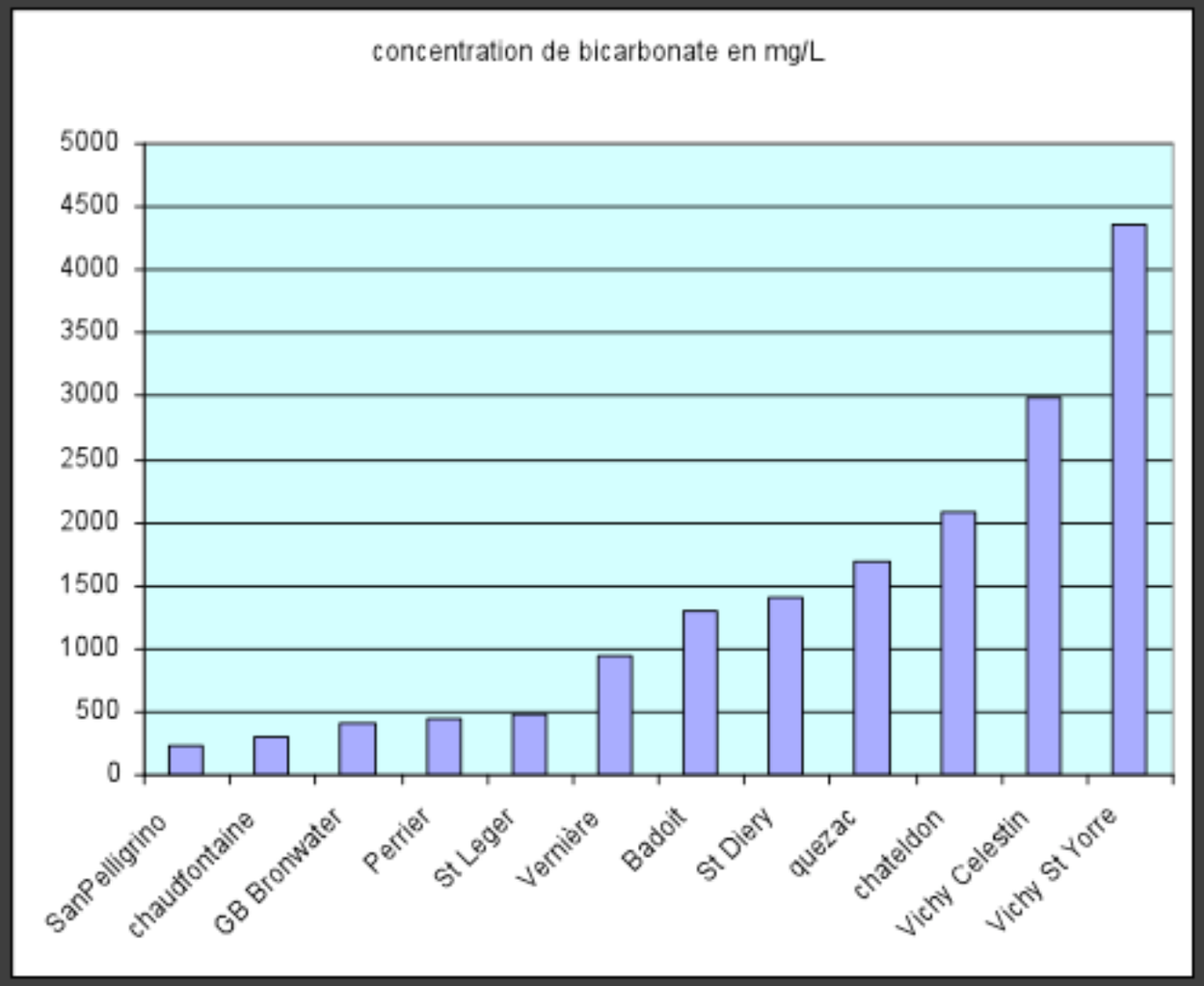


ACIDOSE: EFFET



Acidose - Effet

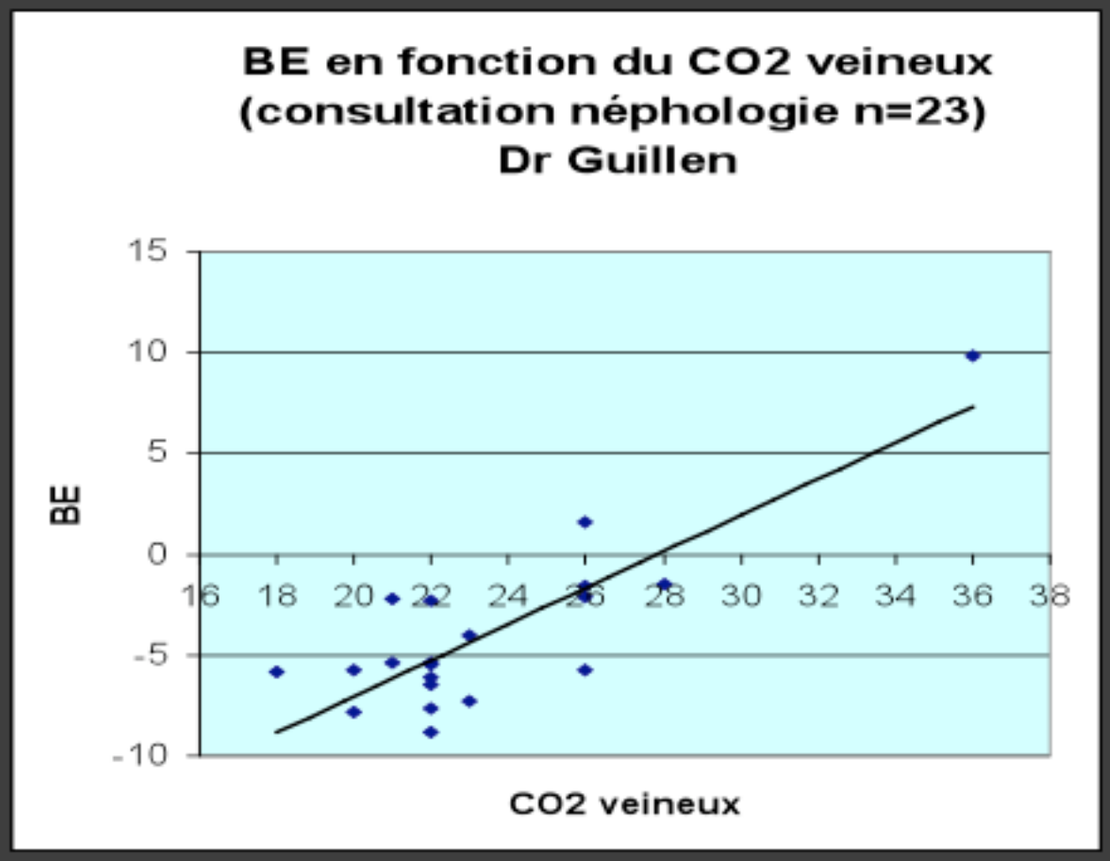
Acidose - Traitement



http://www.mineralwaters.org/

Bilan de Laboratoire

- Veineux:
  - CO2 / R.Alc / Bicar
  - Trou anionique
- Gaz art:
  - BE < -2; pCO diminiunée



Acidose - EBM

Etude	Espèce	CKD	GFR	Nutrition	Autres
Mahajan et al. (KI 2010)	Humain	Stade II	Diminution de la pente	NR	Amélioration de l'excrétion potassique et diminution de l'endotheline urinaire.
Phisitkul et al (KI 2010)	Humain	Stade II, III, IV (>20 ml)	Stabilisation dans le groupe alcalinisé	NR	Diminution de la proté-inurie et des paramètres inflammatoires urinaires
DeBrito et al (JASN2009)	Humain	Stade IV / V	Stabilisation (diminution de la perte néphronique)	Amélioration MAMC / Albumine	Survie sans dialyse 90% us 70%.
Bommer et al (AJKD 2004)	Humain	Stade Vd			Diminution des hospitalisations.
D E Wesson (KI 2010)	Rat Witstar	2/3 nephrectomie	stabilisation	NR	Diminution des paramètres inflammatoires urinaires.

Patient 4 .

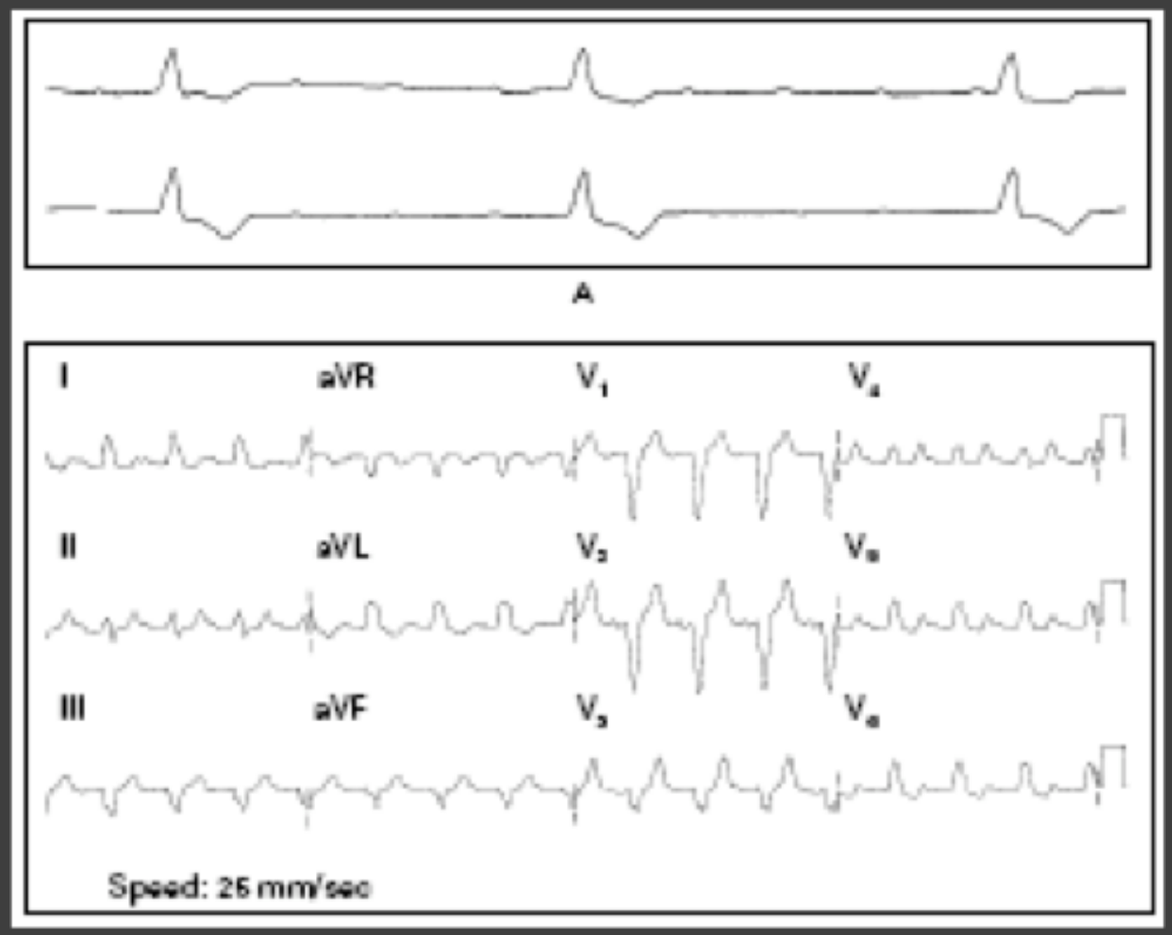
- Homme de 55 ans
- ATCDT: Infarctus myocarde - HTA - CKD II non proétinurique.
- R/ AAS - Statine - BB - ARBs- I MR (aldactone 50 mg).
- Contrôle biologique au Laboratoire:
  - Créatinine 1,6 mg/dL (MDRD 45) , Na 138, Cl 97, K 5.8

HyperKaliémie

- Contrôle du potassium
- Stop aldactone ?
- Stop ARBs ?
- Start Régime pauvre en K ?
- Chélateur Potassium ?
- Ajout Diurétique de l’Anse. ?
- Autres ?
- Nephrologue ?

signe d’alerte

- ECG: onde T pointue
- Faiblesse musculaire importante
- . . . . .
- Décès



Potassium Elevé

Anamnèse Aliment riche en K

Pseudo Hyperk
ponction difficile

Shift sur Acidose

Vrai Hyper K
Cause 1: traitement
IEC - ARBS - IDR - IMR

Correction Régime

Diminution Traitement

Ajout Diurétique de l’anse

Résine

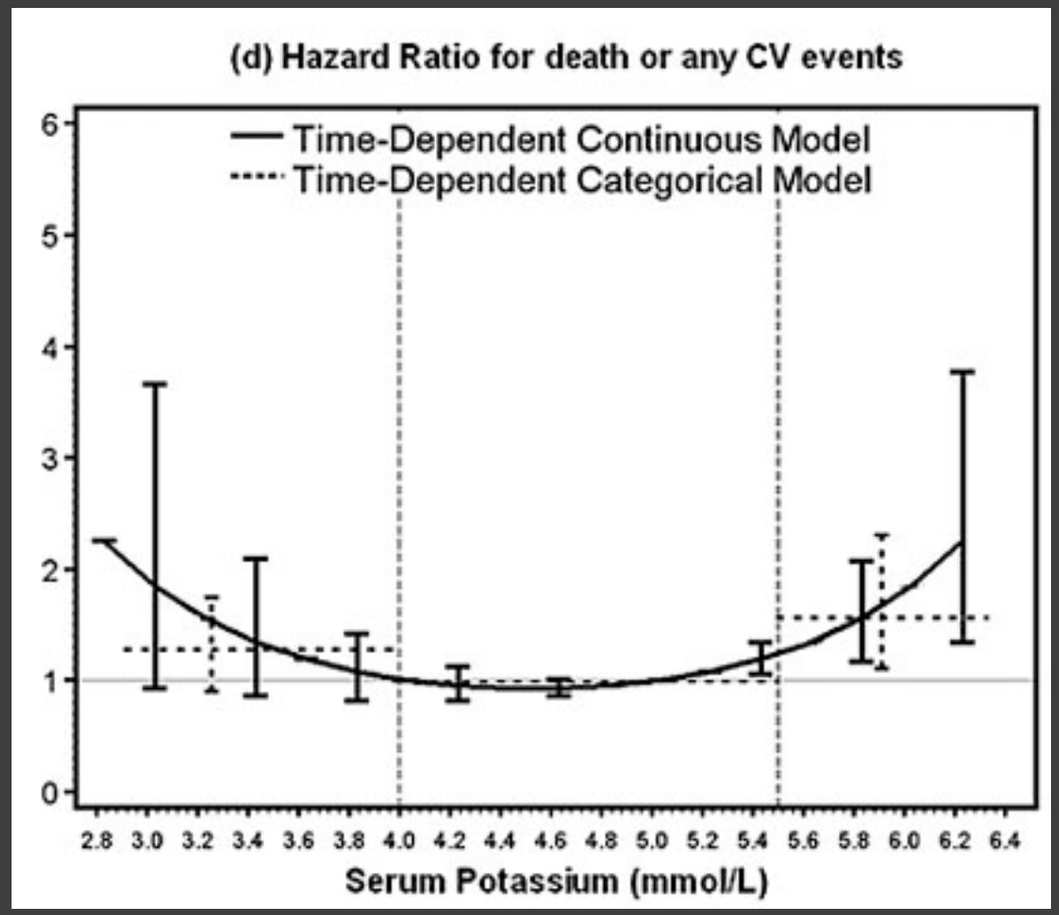
Patient n° 4

Résine:

- Kayexalate calcique :
  - Acidose
  - Phosphore
- Kayexalate Sodique :
  - Apport Sodique
  - Phosphore
- Sorbisterit :

Tolérance DIGESTIVE

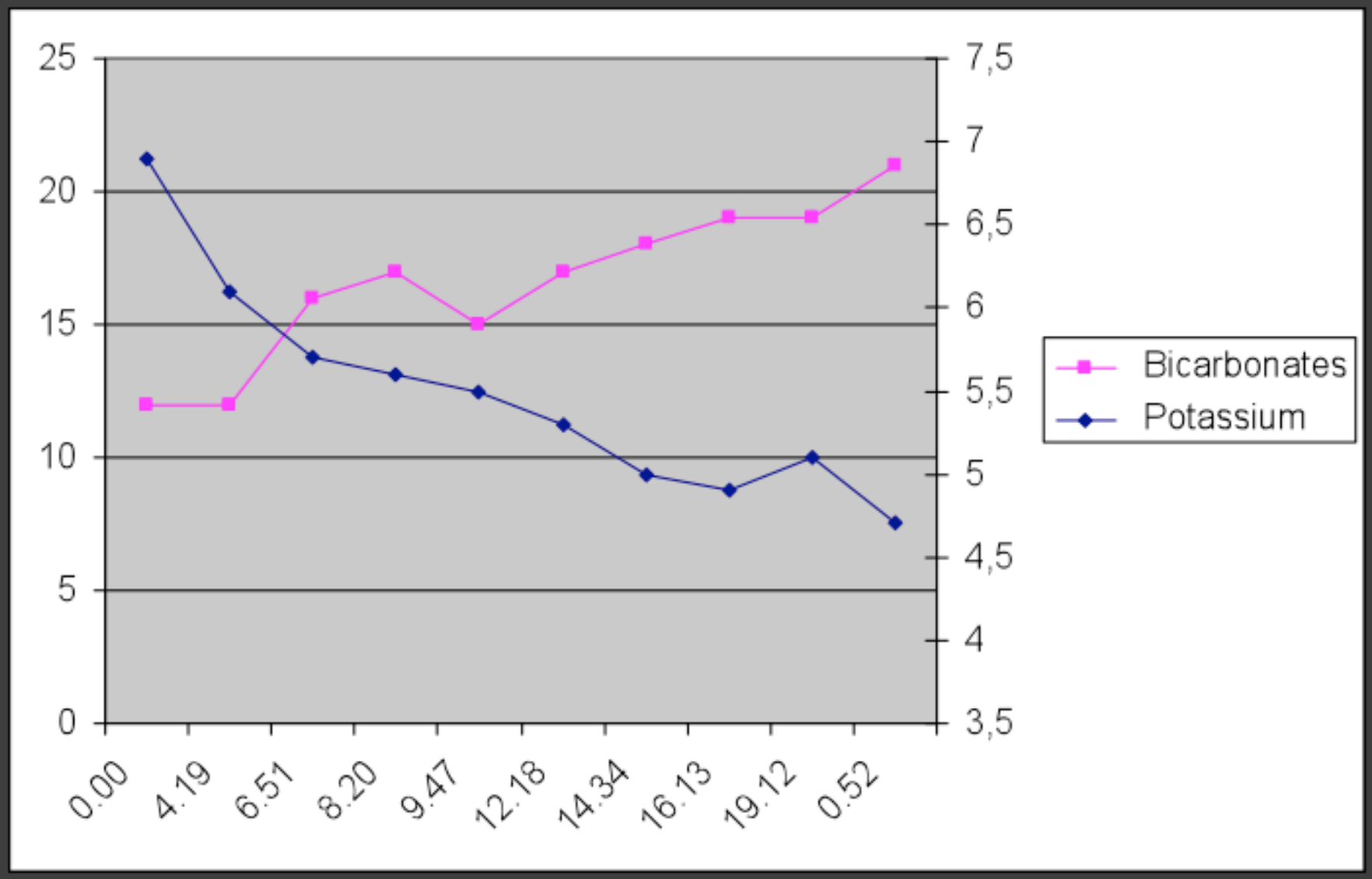
Serum Potassium and Outcomes in CKD: Insights from the RRI-CKD Cohort Study



Clin J Am Soc Nephrol 5: 762–769, 2010. doi: 10.2215/CJN.05850809

Patient n° 4

1 meq K = 4 meq CO2



ANURIE en UROLOGIE

Patient n° 4

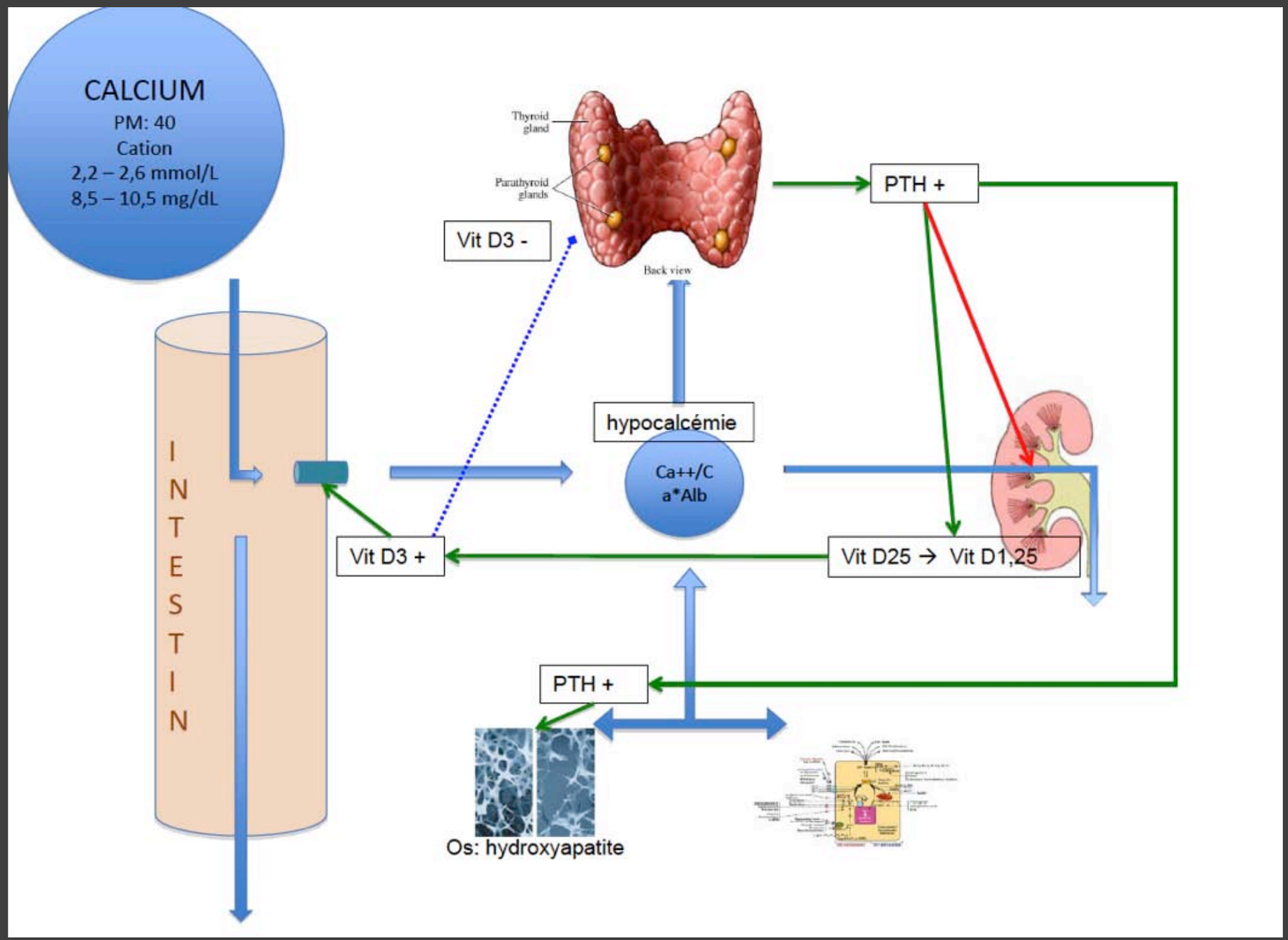
Patient n° 5

- Homme de 68 ans
- ATCDT: Infarctus myocarde - HTA - CKD III non proétinurique.-BPCO - SAHOS
- R/ AAS - Statine - BB - ARBs- I MR (aldactone 50 mg). - Seretide puff - Kayexalate calcique
- Contrôle biologique au Laboratoire:
  - Créatinine 2,6 mg/dL , Na 138, Cl 97, K 5.8, CO 19
  - Calcium 8,7 mg/dL, Phosphore 4,9 mg/dL, Vit D 25.6 UI, Parathormone: 107 ng/ml

Cas 5 - Hyperparthyroïdie

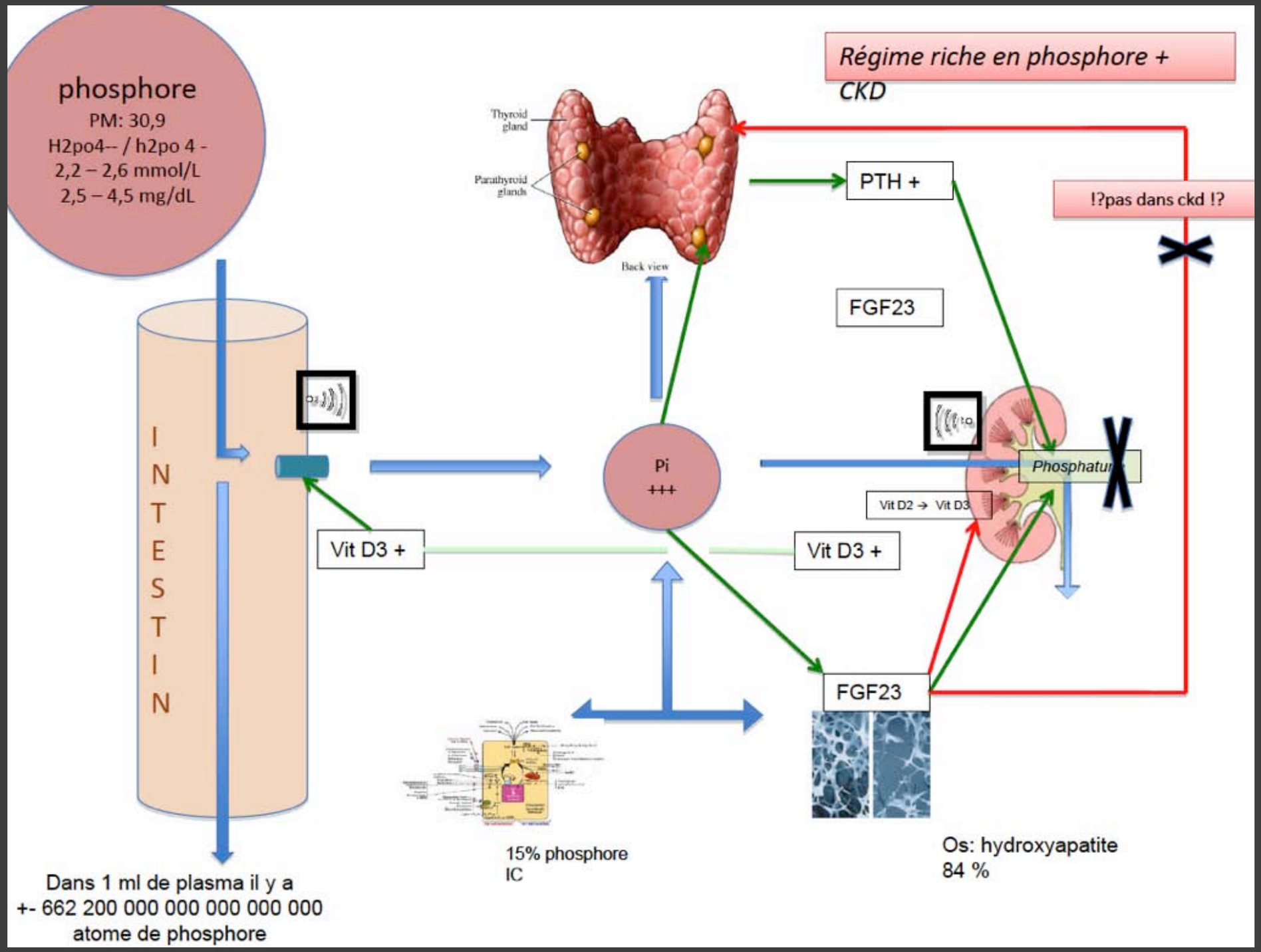
- Hyperparathyroïdie Primaire : calcium bas car IRC --> Bilan Endrocrino
- Hyperparthyroïdie Secondaire mais taux de PTH normal pour le niveau d’IRC.
- Hyperparathyroïdie Secondaire mais traitement par Calcium - Vitamine D suffisant
- Hyperparathyroïdie Tertiaire vu le taux de phosphore --> Scintigraphie Osseuse et Parathyroïdienne

Maladie Osseuse Rénale
en résumé



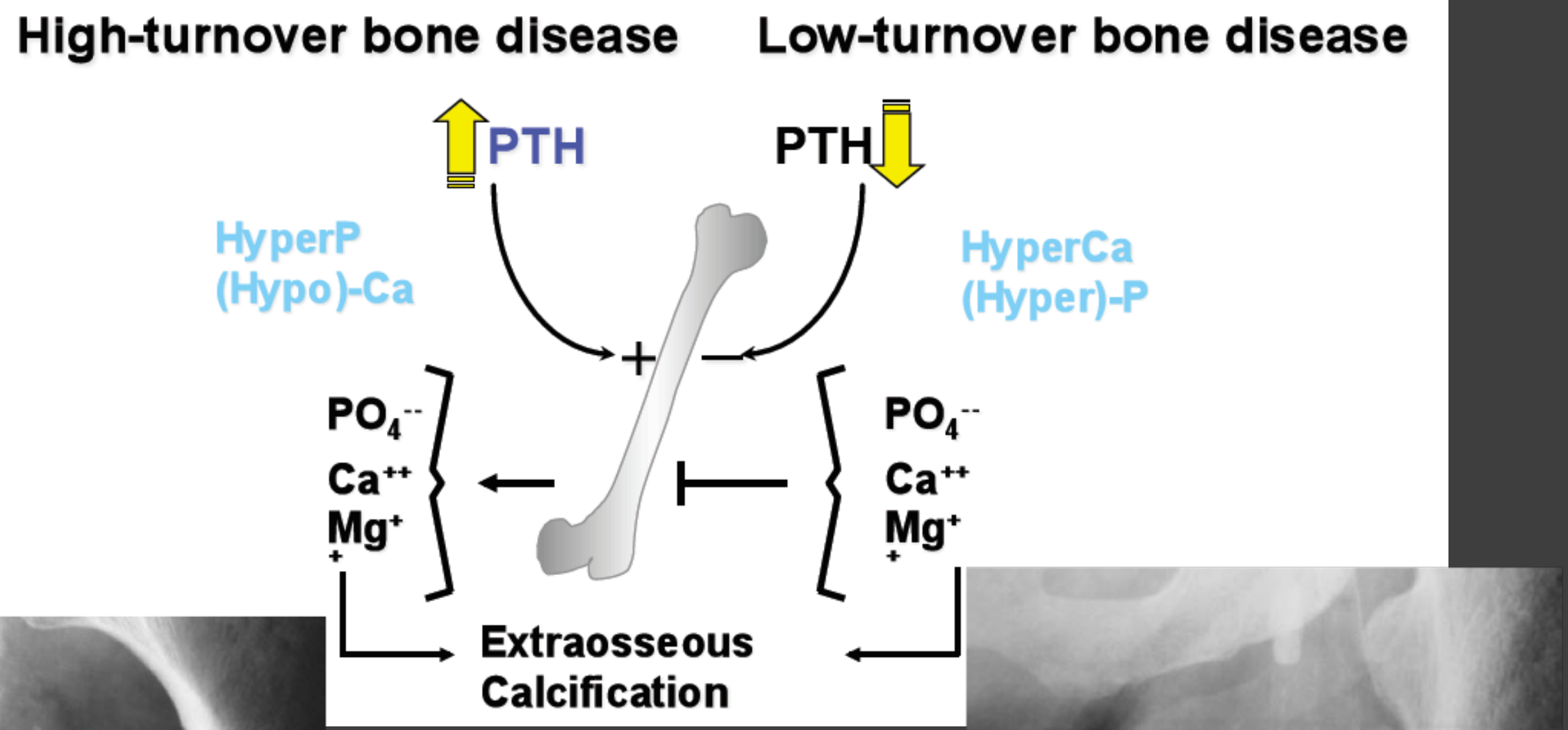
Patient n° 5

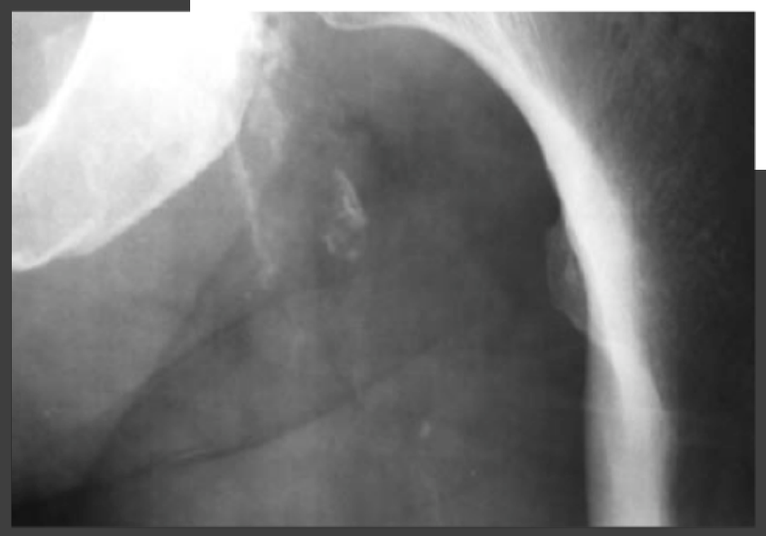
Maladie Osseuse Rénale
en résumé

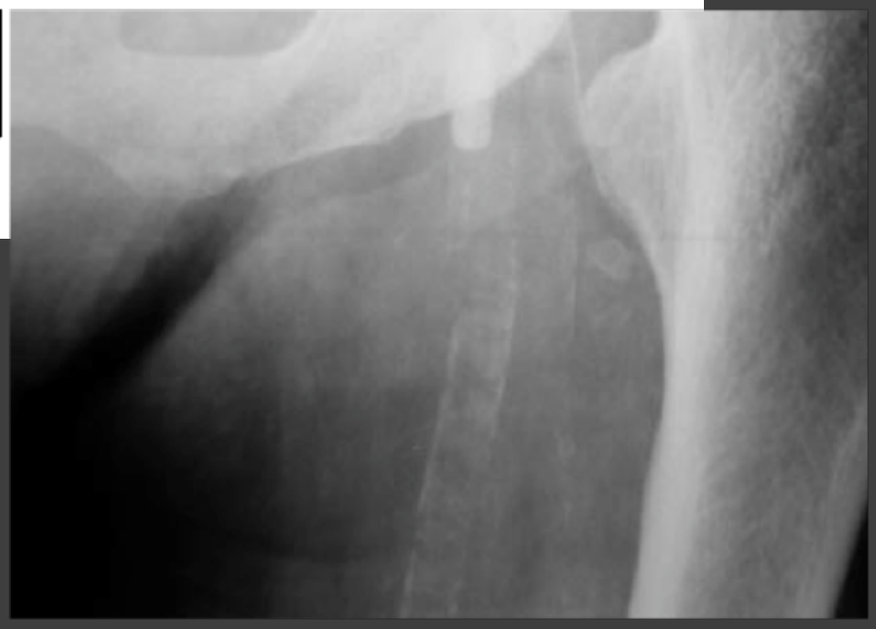


Patient n° 5

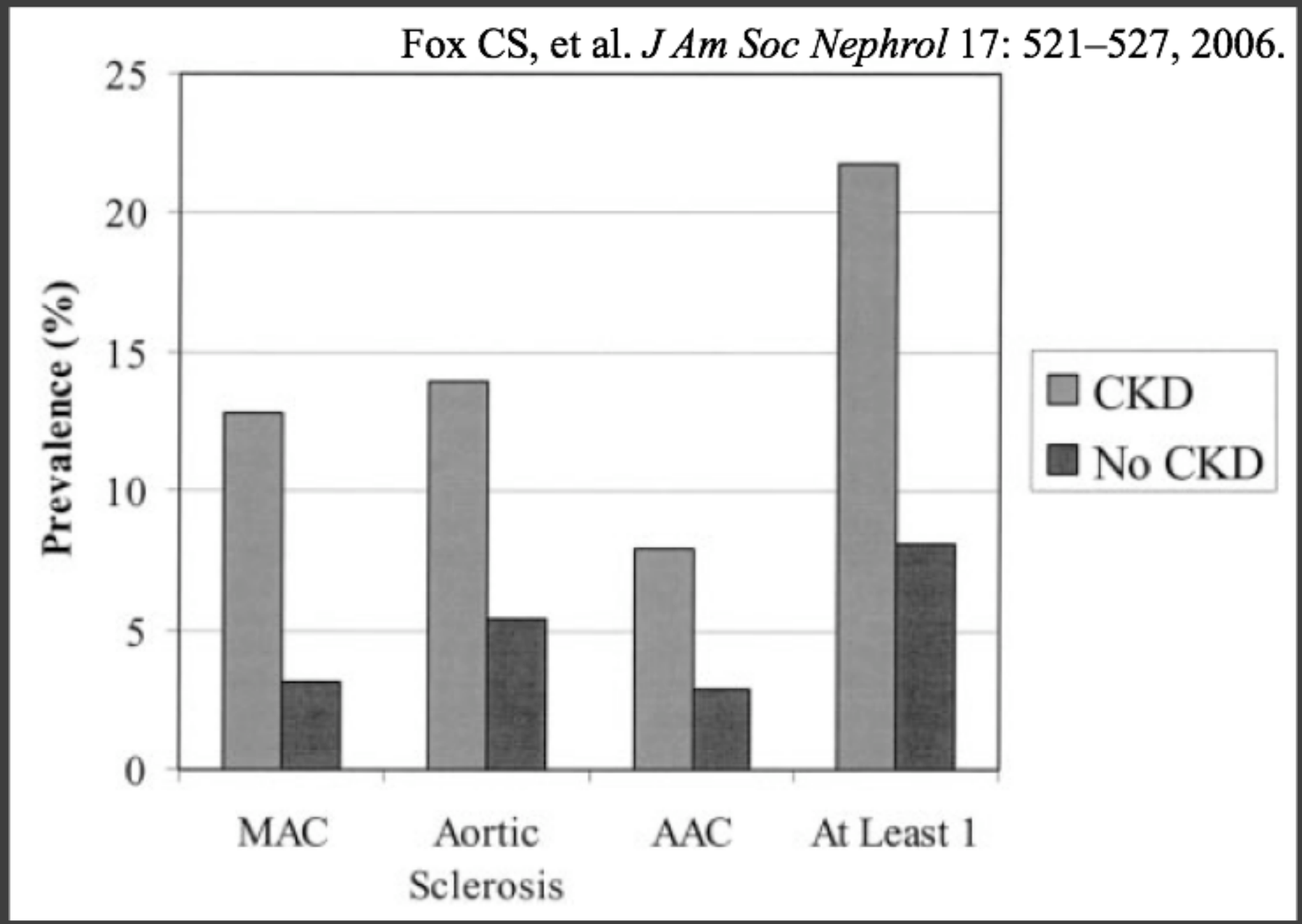
Relation between Type of Bone Disease and Metastatic Calcification







Patient n° 5


Fox CS, et al. *J Am Soc Nephrol* 17: 521–527, 2006.
25
20
15
10
5
0
Prevalence (%)
CKD
No CKD
MAC
Aortic Sclerosis
AAC
At Least 1

Patient n° 5

Maladie Osseuse Rénale
traitement en Prédialyse

- Monitoring: Selon Niveau de comorbidité du patient

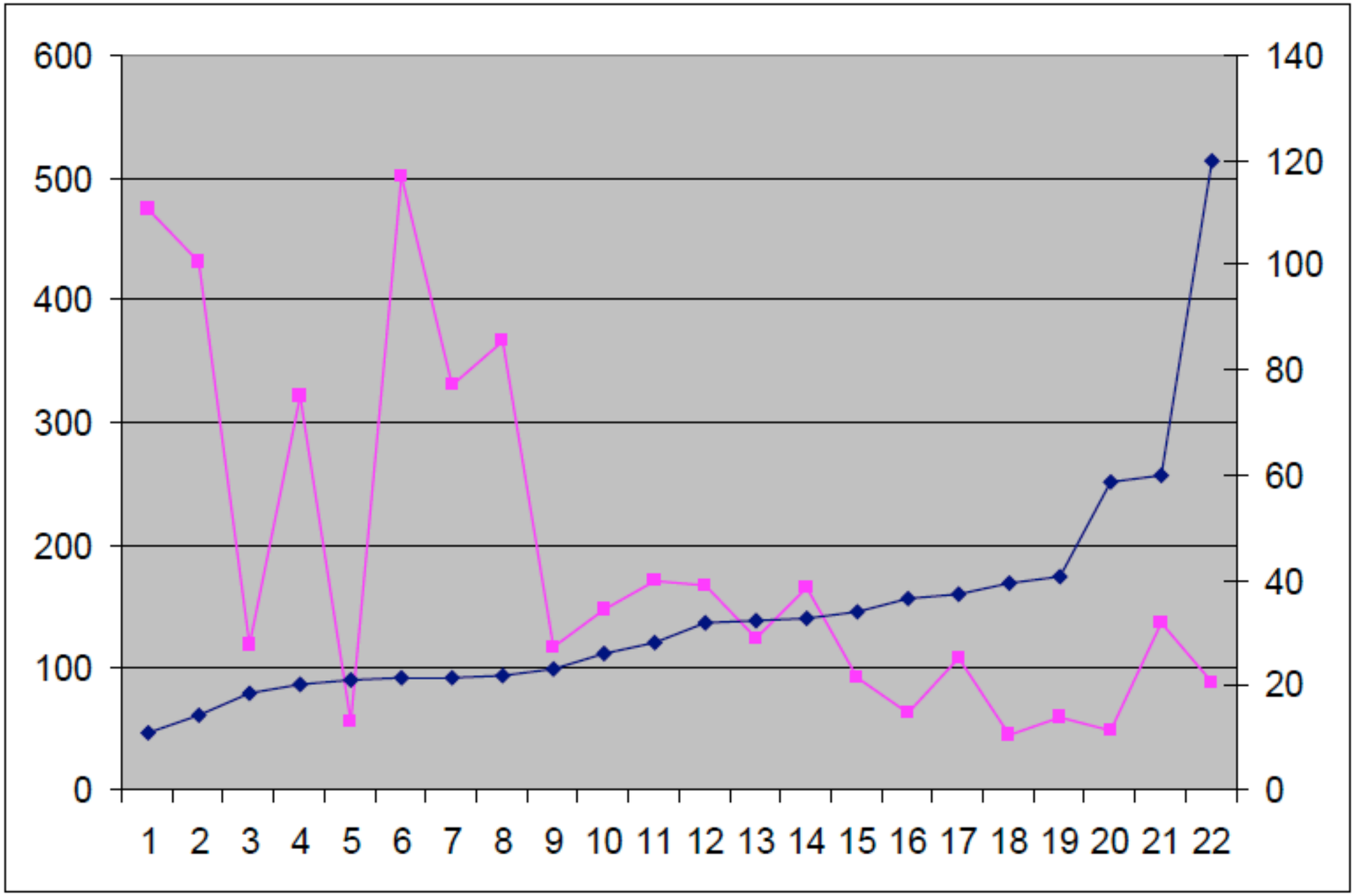
- Apport Vitaminique D

- Apport Calcium ? Oui comme chélateur du phosphore.

- Diminution Apport de Phosphore.

- Méfiance des diurétique de l’anse

iPTH (rose) // Créat eGFR (bleue)



Patient n° 5

Physiologic Regulation of the Serum Concentration of 1,25-Dihydroxyvitamin D by Phosphorus in Normal Men

Anthony A. Portale, Bernard P. Halloran, and R. Curtis Morris, Jr.
General Clinical Research Center, Departments of Pediatrics and Medicine, University of California, San Francisco, California 94143; and Veterans Administration Medical Center, San Francisco, California 94143

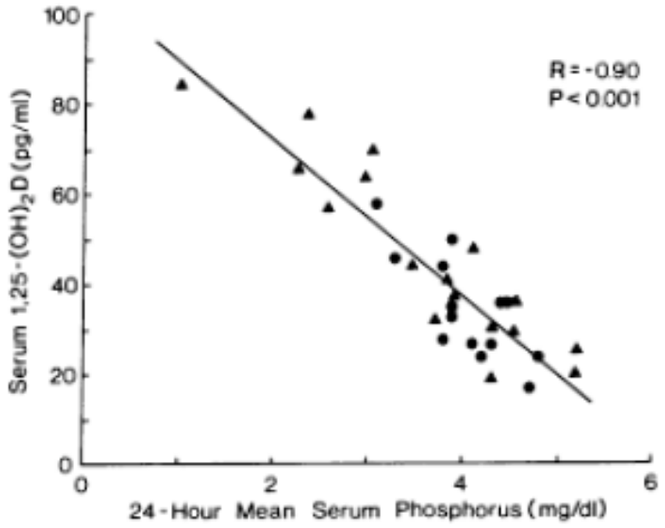


Figure 4. The relationship between serum levels of $1,25(OH)_2D$ and 24-h mean serum levels of phosphorus in 12 normal men. Each point depicts data obtained during five separate study periods of 5–10 d duration, in which dietary phosphorus was maintained at either < 50, 1,500, or > 3,000 mg/d (▲) (data from reference 27); and 625 or 2,300 g/d (●) (this study). For each point depicted, the values of serum $1,25(OH)_2D$ and phosphorus were calculated as in Fig. 3.

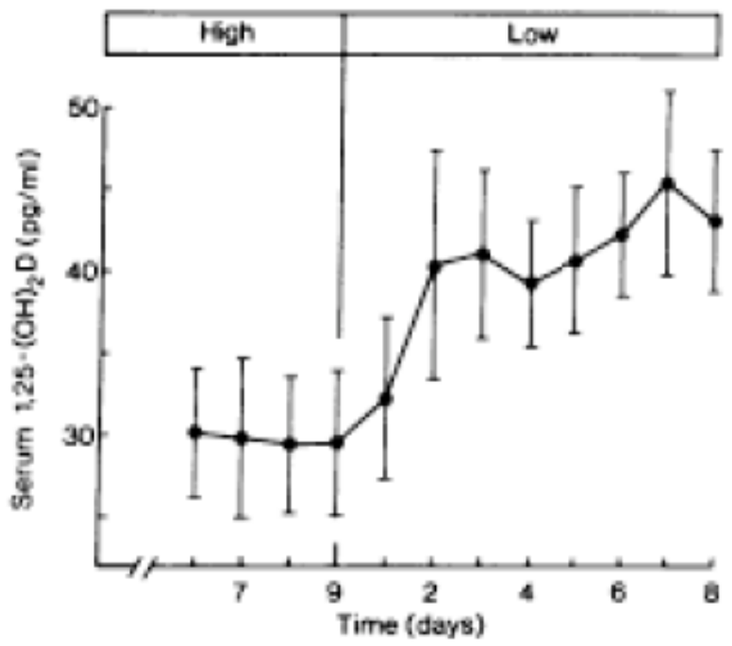


Figure 1. Effect of decreasing dietary phosphorus within its normal range on the daily serum concentration of $1,25(OH)_2D$ in seven normal men. Dietary phosphorus was first maintained at 2,300 mg/d for 9 d, then decreased to 625 mg/d for 8 d. Depicted are mean values±SEM.

Patient n° 5

Cas 5 - Hyperparthyroïdie

- Calcium 8,7 mg/dL, Phosphore 4,9 mg/dL, Vit D 25.6 UI, Parathormone: 107 ng/ml

- Hyperparathyroïdie Secondaire mais traitement par Calcium pour chélation phosphore mais diète hypophosphorée si possible
- Vitamine D pour améliorer taux calcique et PTH, vit D hydroxylée au besoin
- Si réfractaire

Patient 6

- Homme de 73 ans
- ATCDT: Infarctus myocarde (FE 40%)- HTA - CKD IV non protéinurique (Pente -4 ml/ans).-BPCO - SAHOS - PTA iliaque - Hernie inguinale droite et ombilicale non opérée.
- R/ AAS - Statine - BB - ARBs- I MR (aldactone 50 mg). - Seretide puff - Kayexalate calcique - EPO
- Contrôle biologique au Laboratoire:
  - Créatinine 3,9 mg/dL (MDRD 15), Na 138, Cl 97, K 5.8, CO 19, Hémoglobine 11,2 g/dL
  - Calcium 9,2 mg/dL, Phosphore 4,2 mg/dL, Vit D 25.6 UI, Parathormone: 157 ng/ml

Patient N° 6 - suite

- Docteur, On m’a parlé de m’informer pour la dialyse. Et vous qu’en pensez vous ?

... ? ...
Info Patient - Dialyse
Info Médecin générale.

- Toujours plusieurs mois avant la dialyse.
- Implication:
  - Hémodialyse = Fistule AV !
  - Hémodialyse en centre ?
  - Hémodialyse à la Maison ?
- Dialyse Péritonéale = Protection abdominale !
  - Dialyse Péritonéale autonome.
  - Dialyse Péritonéale aidé par nursing .

LE MG reste acteur de son patient

MG et Epuration rénale

	Hémodialyse	Dialyse Péritonéale
Fièvre	!!! Acces Vasculaire !!!	!!! Péritonite à bas bruit !!!
Dyspnée	OAP débutant	Ep Pleural de DP
Examen à Prescrire	apprécier Effet dialyse	
Particularité	Héparine	patient plus libre

RESUME

- Cas1:
  - Développer concept MDRD classification
  - Développer mesure de la protéinurie - FR rénal.
- Cas 2:
  - Développer Effet protéinurie sur le GFR
  - Développer Inhibition axe RAA.
- Cas 3:
  - Développer acidose
- Cas 4:
  - Développer Kaliémie
- Cas 5:
  - Développer Calcium Phosphore PTH
- Cas 6:
  - “On m’a parlé de dialyse”

Message

- 1) Diagnostic. TOUJOURS UN DIAGNOSTIC
- 2) Evaluation du risque (stadification KDIGO)
- 3) Correction des paramètres usuels:
  - HTA - Protéinurie - Acidose - Kaliémie - Anémie- Osteodystrophie rénale - Anémie
- 4) Au pire cela se prépare aussi.

Un dernier pour la route

- Docteur, mon père (CKD IV) que vous soignez ne va pas bien au Brésil.
- Les docteurs disent qu’il a une méningite.
- Cela a commencé par du hocquet avant qu’il soit confus me dit ma mère.
- En plus ils vont le dialyser