CKD-MBD και Οστεοπόρωση

Βίοι παράλληλοι αλλά με αντίθετη κατεύθυνση;

Χρυσόστομος Δημητριάδης

Νεφρολόγος, Διευθυντής ΕΣΥ

Α' Νεφρολογική Κλινική ΑΠΘ

Ιπποκράτειο ΓΠΝ Θεσσαλονίκης

ΟΡΙΣΜΟΣ ΔΙΑΤΑΡΑΧΗΣ ΜΕΤΑΛΛΩΝ ΚΑΙ ΟΣΤΩΝ (CKD-Mineral Bone Disorder)

σε ασθενείς με χρόνια νεφρική νόσο

Definition, evaluation, and classification of renal osteodystrophy: A position statement from Kidney Disease: Improving Global Outcomes (KDIGO)

ΟΡΜΟΝΕΣ/ ΜΕΤΑΛΛΑ

Table 3 | Definition of CKD-MBD

A systemic disorder of mineral and bone metabolism due to CKD manifested by either one or a combination of the following:

Abnormalities of calcium, phosphorus, PTH, or vitamin D metabolism

Abnormalities in bone turnover, mineralization, volume, linear growth, or strength

Vascular or other soft tissue calcification

ROD -
Renal OsteoDystrophy

Kidney International (2006) 69, 1945–1953

ΟΡΙΣΜΟΣ ΔΙΑΤΑΡΑΧΗΣ ΜΕΤΑΛΛΩΝ ΚΑΙ ΟΣΤΩΝ (CKD-Mineral Bone Disorder)

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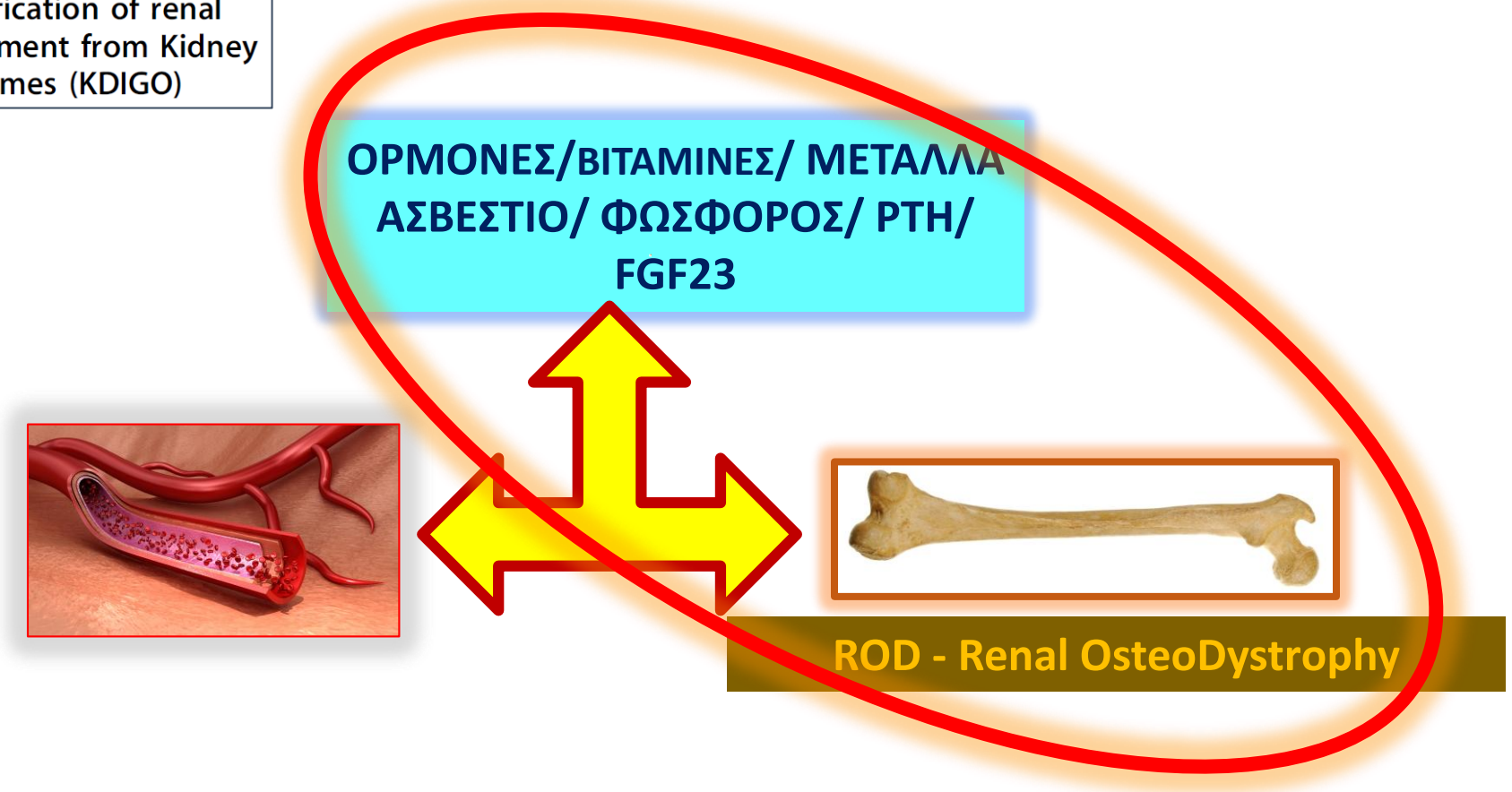
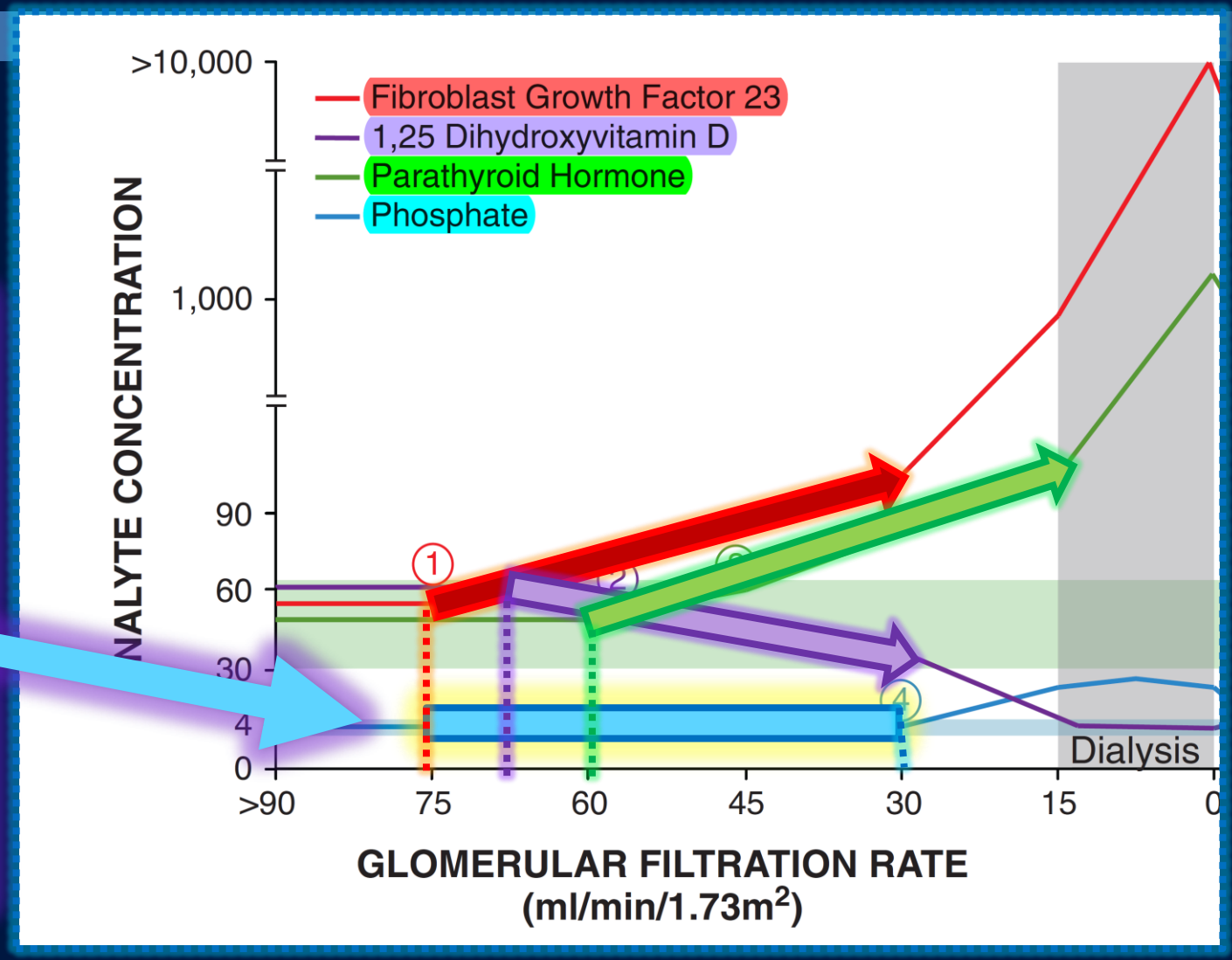
Definition, evaluation, and classification of renal osteodystrophy: A position statement from Kidney Disease: Improving Global Outcomes (KDIGO)



Kidney International (2006) 69, 1945–1953

Φώσφορος, FGF23, 1,25-D_3 και PTH σε ασθενείς με ΧΝΝ σταδίου 1-5

Η συνολική προσπάθεια να διαχείρισης του *φωσφόρου* είναι η αιτία των διαταραχών στην CKD-MBD



Wolf M.

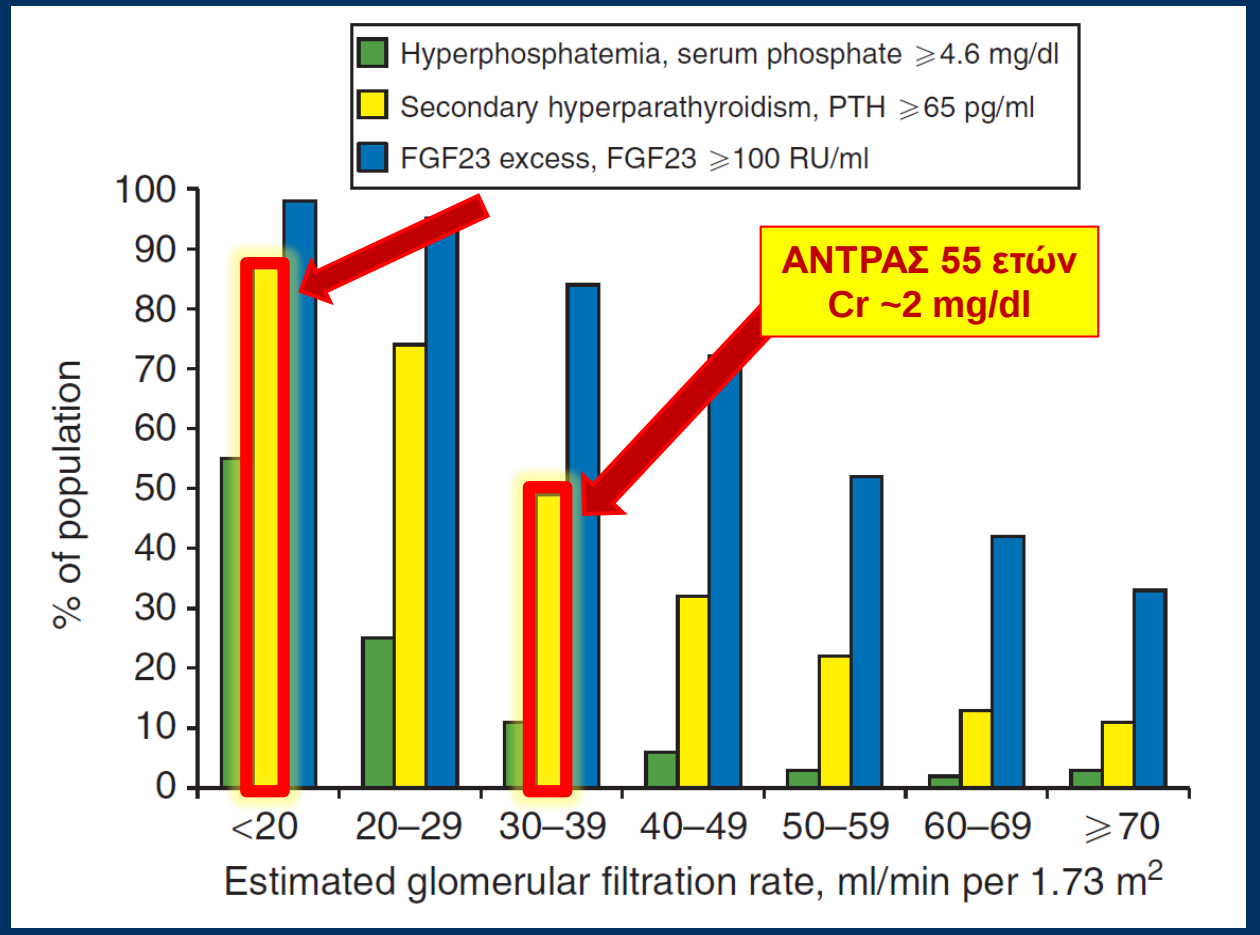
J Am Soc Nephrol 21:1427, 2010

ΝΕΦΡΙΚΗ ΟΣΤΕΟΔΥΣΤΡΟΦΙΑ ΚΑΙ ΠΑΡΑΘΟΡΜΟΝΗ

~50 % σε GFR 30-40 ml/min

>80 % σε GFR <20 ml/min

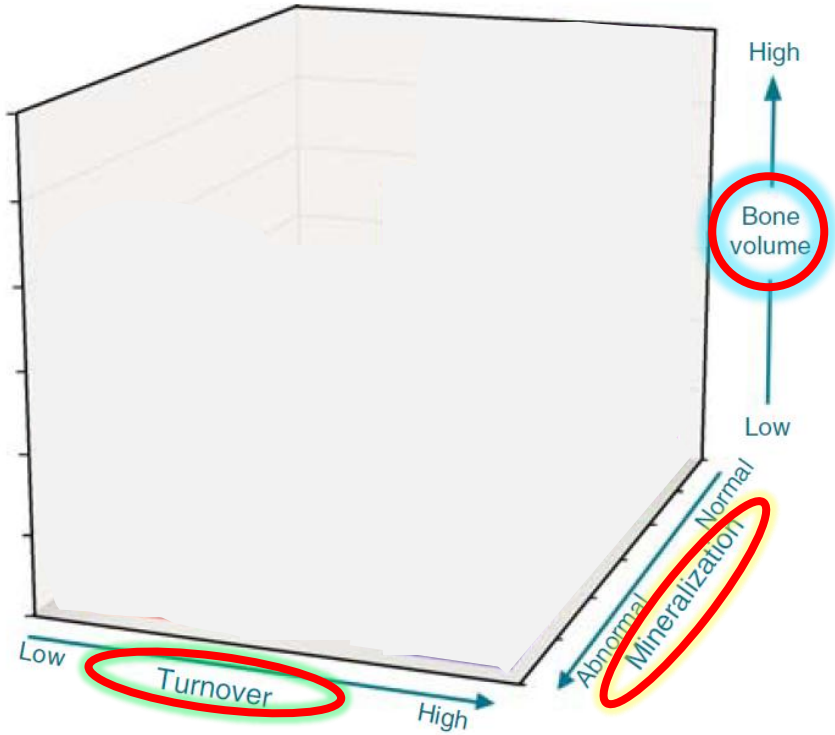
έχει αυξημένες τιμές PTH !!



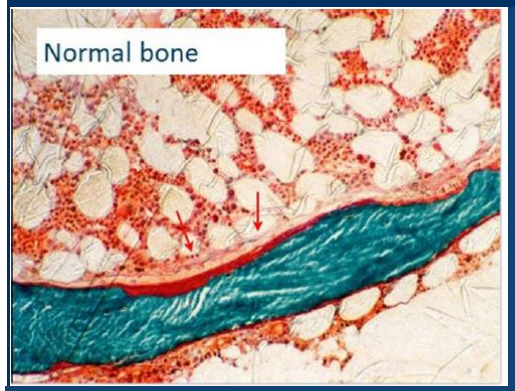
ΝΕΦΡΙΚΗ ΟΣΤΕΟΔΥΣΤΡΟΦΙΑ ΣΤΗ ΧΝΝ

ΔΕΝ ΕΙΝΑΙ ΕΝΑ ΕΙΔΟΣ ΟΣΤΙΚΗΣ ΝΟΣΟΥ !!

(OM, osteomalacia)
(AD, adynamic bone disease)
(mild HPT, mild hyperparathyroid related bone disease)
(OF, osteitis fibrosa or advanced hyperparathyroid-related bone disease)
(MUO, mixed uremic osteodystrophy)



ΑΔΥΝΑΜΙΚΗ ?!
Οστική Νόσος



Aluminum ??
Έλλειψη Vit D

PTH ↑↑↑ !!
«Κλασσικός» 2παθής Υπερπαραθυρεοειδισμός
Ινώδης κυστική Οστεϊτις

Kidney International (2006) 69, 1945–1953

ΝΕΦΡΙΚΗ ΟΣΤΕΟΔΥΣΤΡΟΦΙΑ

ΔΕΝ ΕΙΝΑΙ ΕΝΑ ΕΙΔΟΣ ΟΣΤΙΚΗΣ ΝΟΣΟΥ !!

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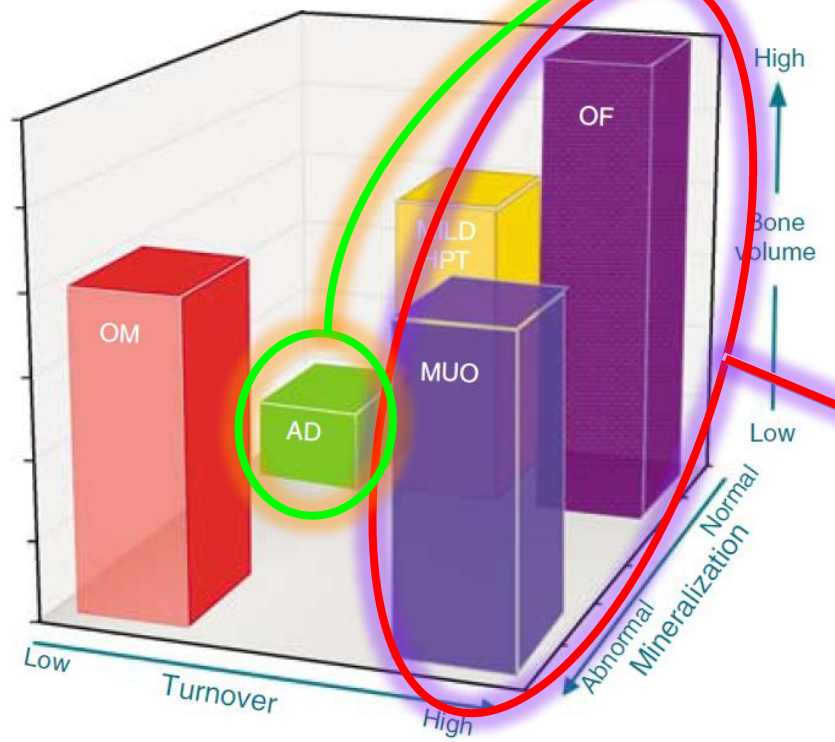


Kidney International (2006) 69, 1945–1953

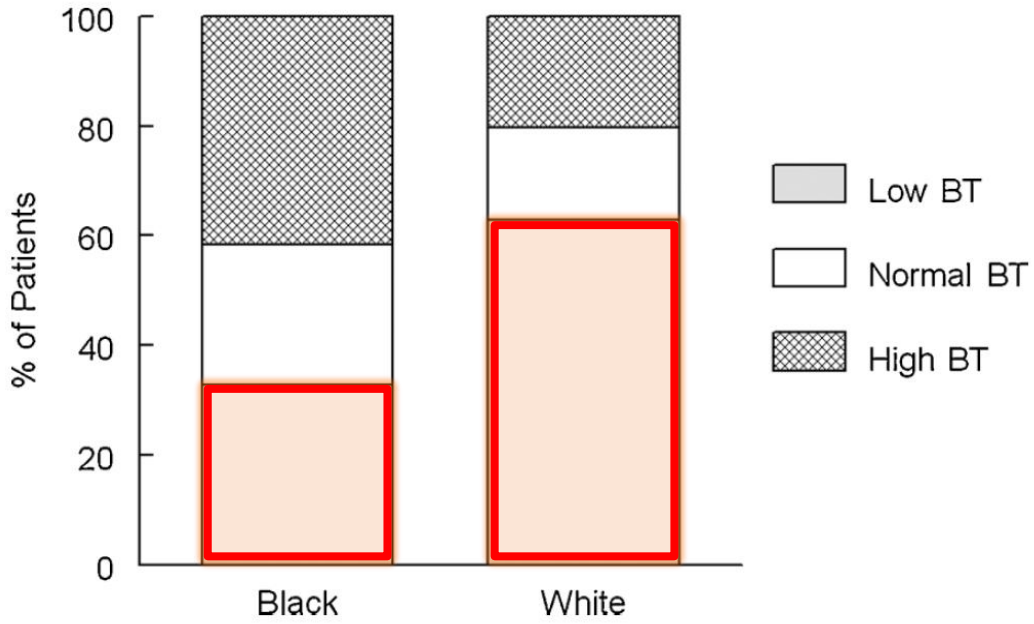
ΑΔΥΝΑΜΙΚΗ ?!
Οστική Νόσος

PTH ↑↑↑ !!
«Κλασσικός» 2παθής Υπερπαραθυρεοειδισμός
Ινώδης κυστική Οστεϊτις

Συχνότητα Αδυναμικής Οστικής νόσου σε ασθενείς υπό αιμοκάθαρση

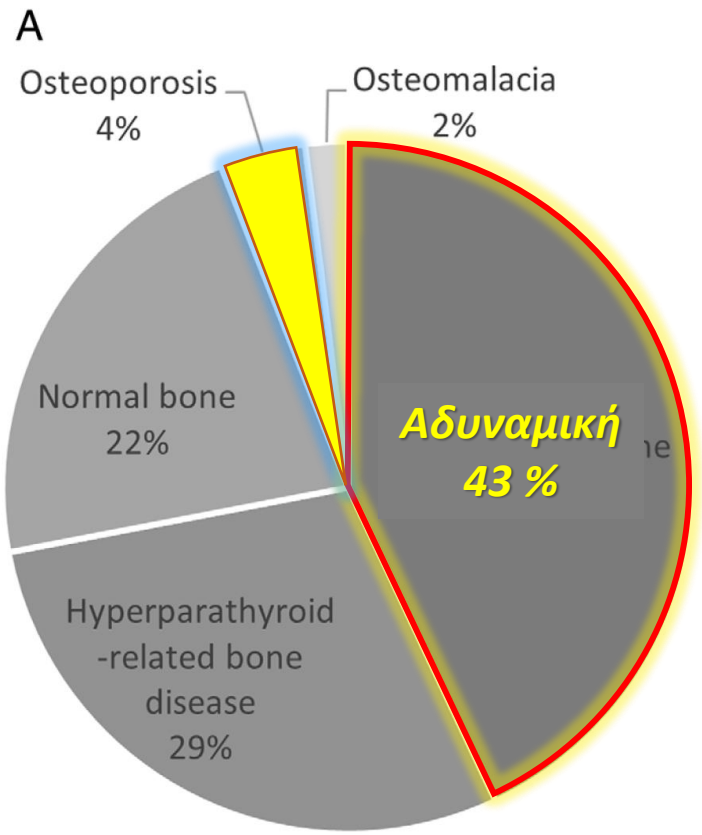
Renal Osteodystrophy in the First Decade of the New Millennium: Analysis of 630 Bone Biopsies in Black and White Patients

Hartmut H Malluche, Hanna W Mawad, and Marie-Claude Monier-Faugere
Division of Nephrology, Bone and Mineral Metabolism, University of Kentucky, Lexington, KY, USA



J Bone Miner Res. 2011 Jun; 26(6): 1368–1376

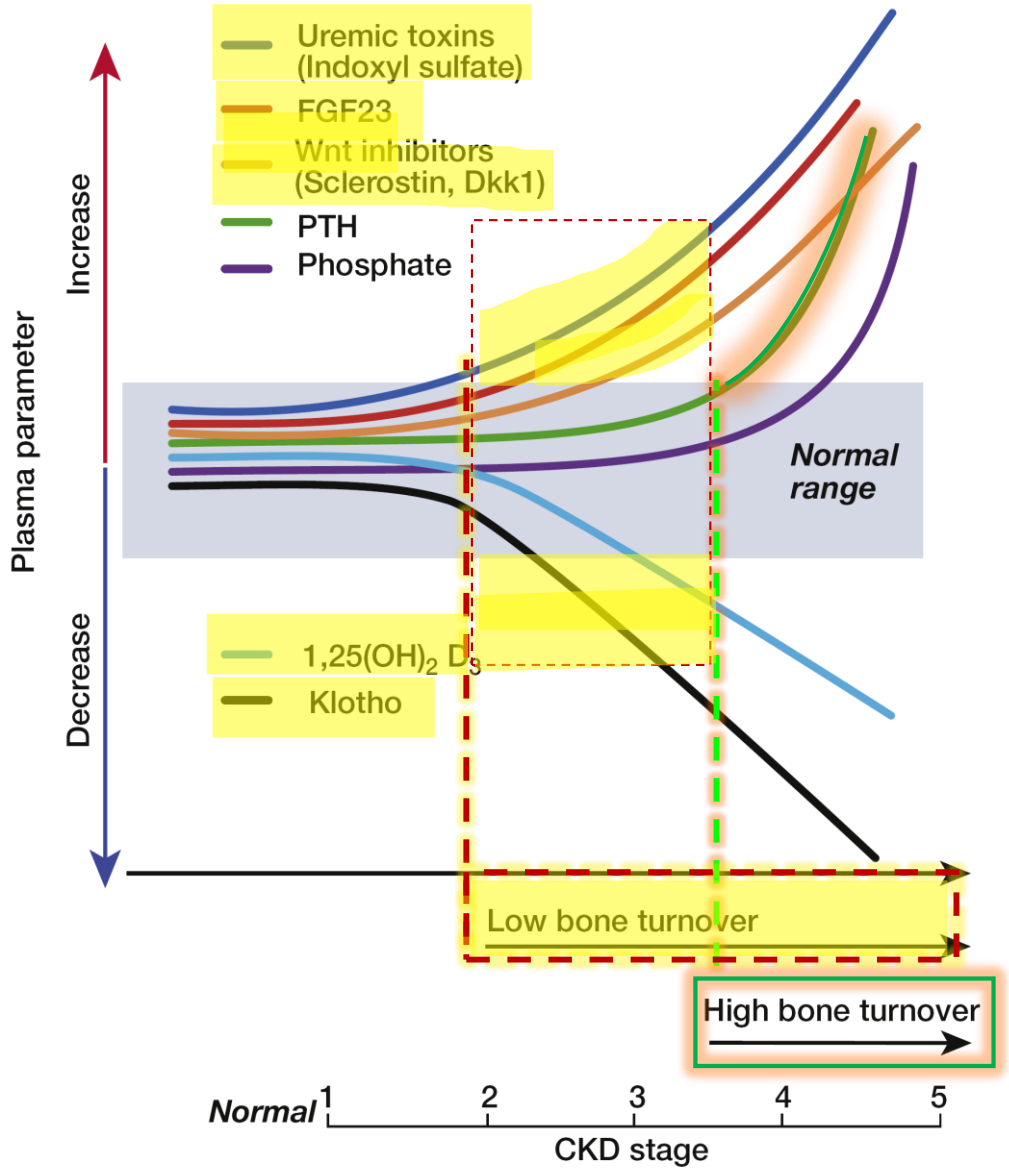
Οστική Βιοψία /Οστεοδυστροφία σε ασθενείς υπό περιτοναϊκή κάθαρση



Pereira L et al

J Bone Miner Res. 2022 Sep;37(9):1689-1699

Evidence of low-turnover bone disease in early, as compared to late, chronic kidney disease

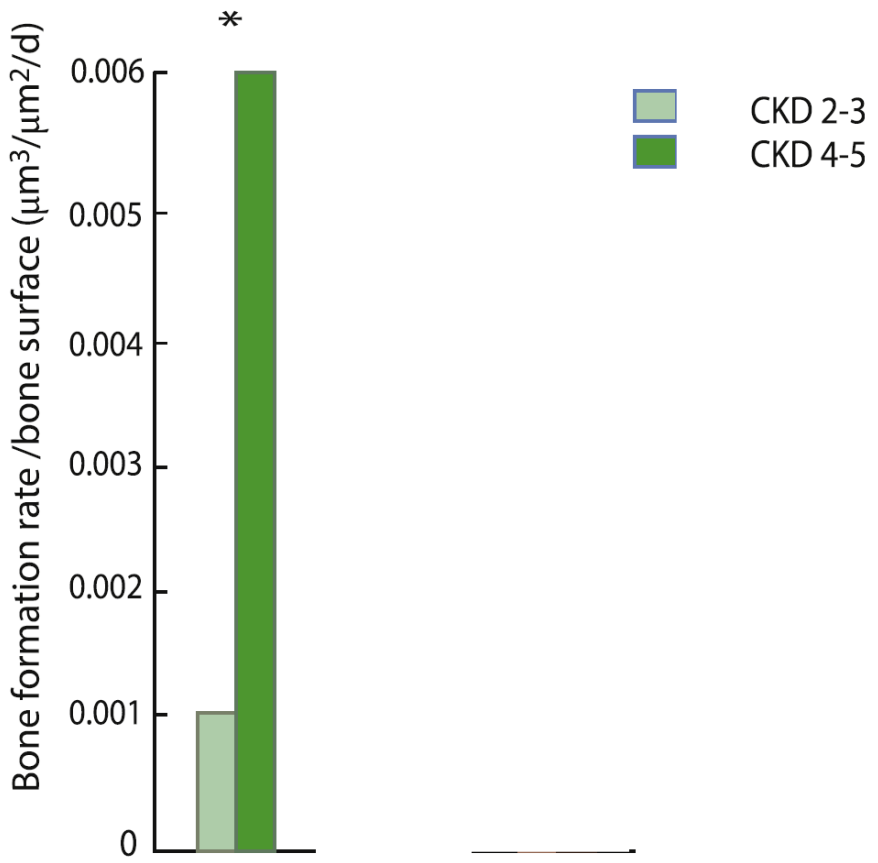


Drüeke TB, Massy ZA

Kidney Int. 2016 Feb;89(2):289-302

Evidence of low-turnover bone disease in early, as compared to late, chronic kidney disease

ΜΕΤΑΒΟΛΙΚΟΣ ΡΥΘΜΟΣ ΣΕ ΒΙΟΨΙΑ ΟΣΤΟΥ



Drüeke TB, Massy ZA

Kidney Int. 2016 Feb;89(2):289-302

Η i-PTH ***δεν είναι*** ευαίσθητος και ειδικός δείκτης του τύπου της οστικής νόσου

Diagnostic Accuracy of Bone Turnover Markers and Bone Histology in Patients With CKD Treated by Dialysis

Stuart M. Sprague, DO,[1] Ezequiel Bellorin-Font, MD,[2] Vanda Jorgetti, MD, PhD,[3] Aluizio B. Carvalho, MD, PhD,[4] Hartmut H. Malluche, MD,[5] Aníbal Ferreira, MD, PhD,[6] Patrick C. D'Haese, PhD,[7] Tilman B. Drüeke, MD,[8] Hongyan Du, MB, MS,[1] Thomas Manley, RN, CRNA,[9] Eudocia Rojas, MD,[2] and Sharon M. Moe, MD[10]

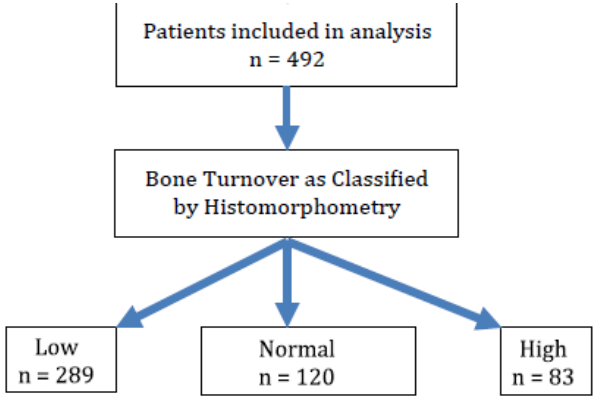


Table 2. Discrimination of Bone Turnover by Serum Biochemistry

	Low (n = 289)	Normal (n = 120)	High (n = 83)	Reference Range
iPTH, pg/mL	68.2 [23.2-186.3]	180.7 [50.0-717.9]	382.6 [139.5-865.5]	15.0-65.0

Sprague et al

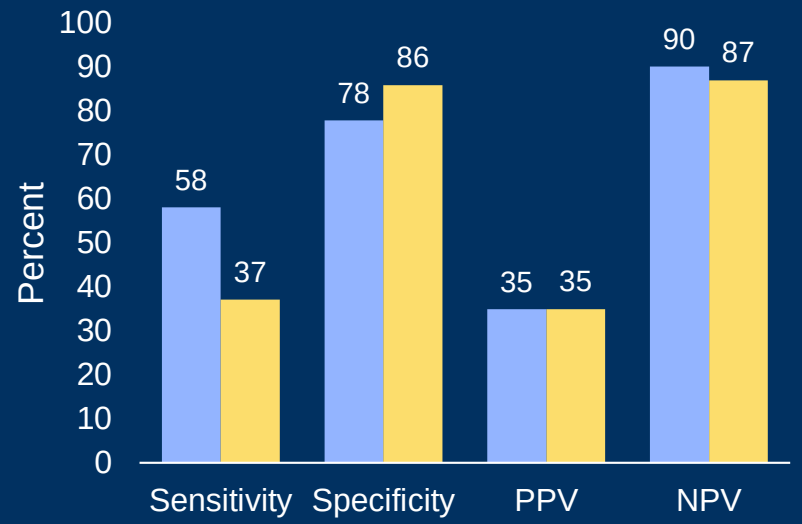
Am J Kidney Dis. 2016;67(4):559-566

Οι τιμή της i-PTH δεν είναι ευαίσθητος και ειδικός δείκτης του τύπου της οστικής νόσου σε ασθενείς με ΧΝΝ τελικού σταδίου...

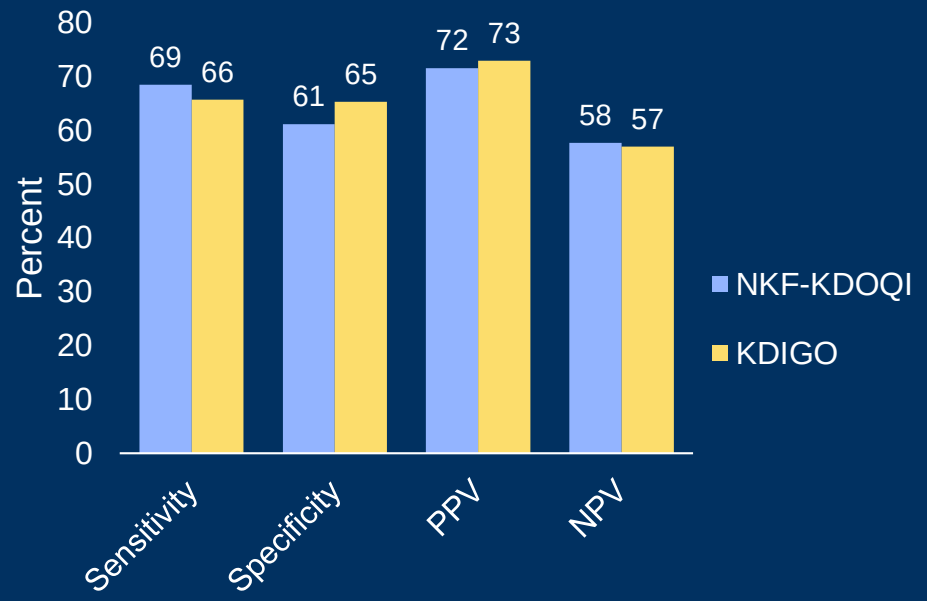
ούτε για τη θεραπεία της με βάση τα προτεινόμενα όρια !

ΣΕ ΕΞΩΝΕΦΡΙΚΗ ΚΑΘΑΡΣΗ

Differentiating high from non-high turnover bone disease, or "When do I *START therapy?*"



Differentiating low from non-low turnover bone disease, or "When do I *STOP therapy?*"



Sprague et al

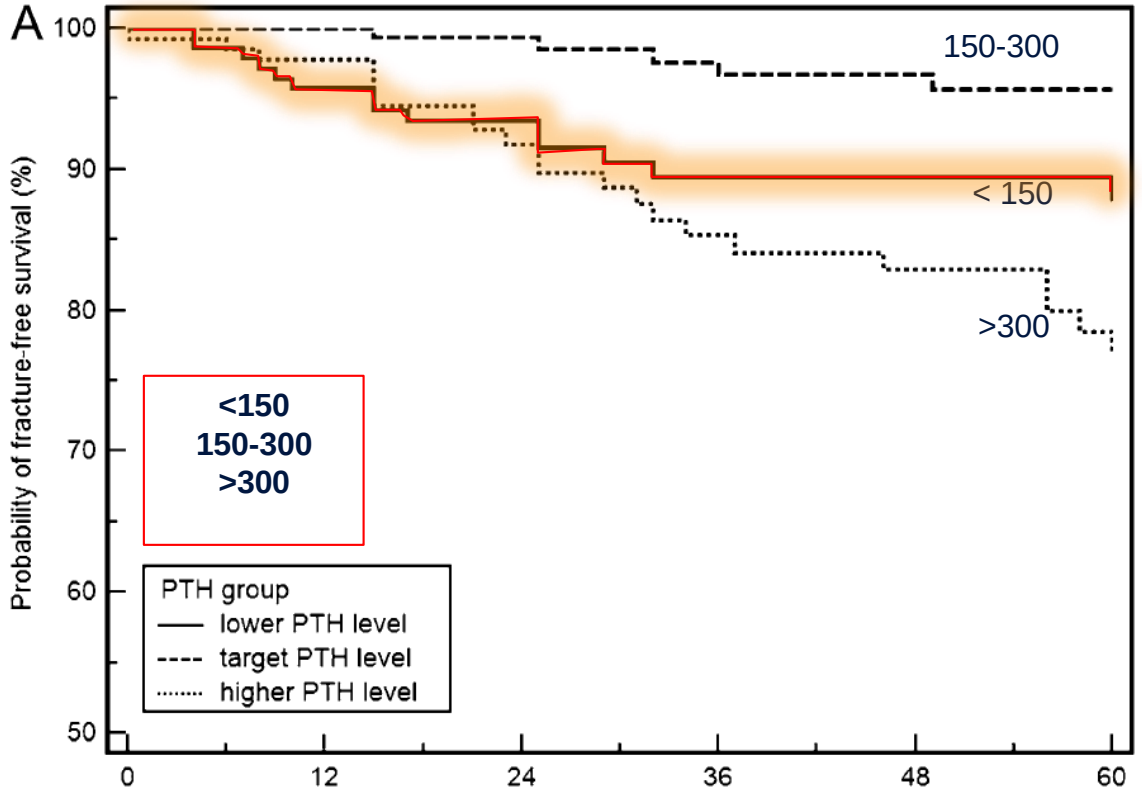
Am J Kidney Dis. 2016;67(4):559-566

Οι αυξημένες τιμές παραθορμόνης συσχετίζονται με *κίνδυνο καταγμάτων* ...

... *το ίδιο όμως και οι χαμηλές*

Diagnostic usefulness of bone mineral density and biochemical markers of bone turnover in predicting fracture in CKD stage 5D patients—a single-center cohort study

ΣΕ ΕΞΩΝΕΦΡΙΚΗ ΚΑΘΑΡΣΗ



Iimori S et al..

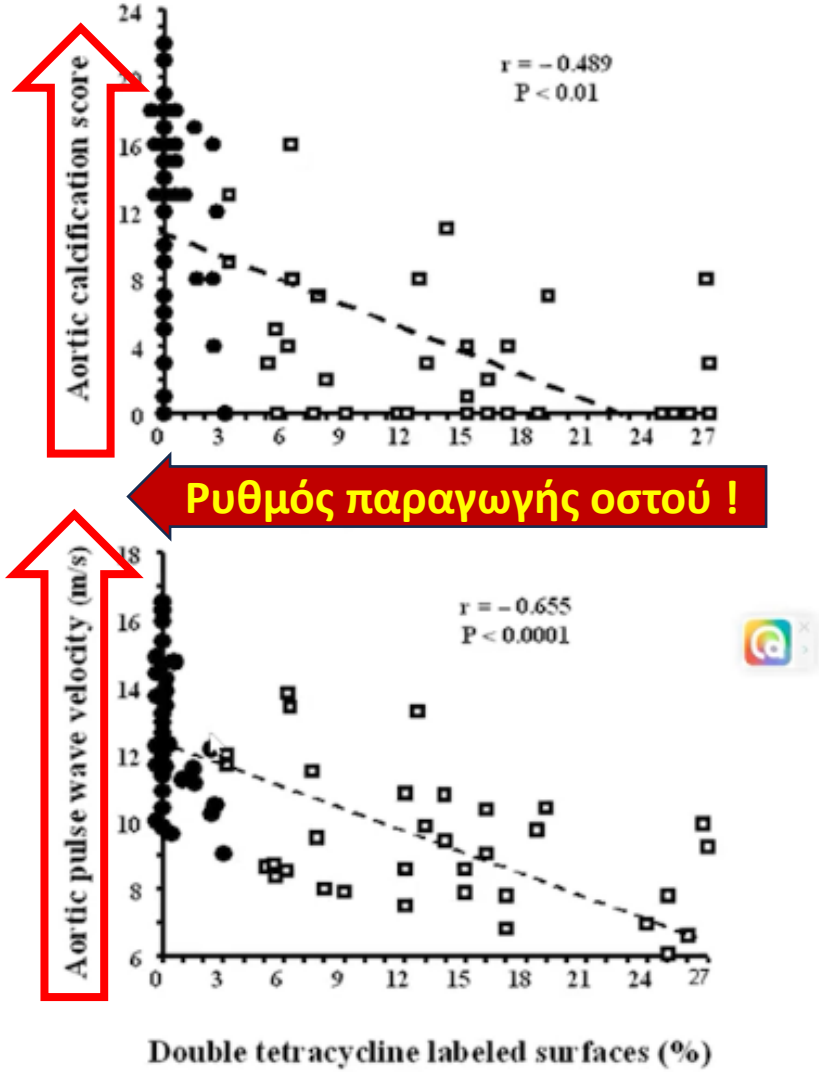
Nephrol Dial Transplant. 2012;27:345–351

Οι χαμηλές τιμές παραθορμόνης και ο *χαμηλός οστικός μεταβολισμός* συσχετίζονται με ...

Αγγειακές επασβεστώσεις !!

Bone turnover disorders are associated with vascular calcification in hemodialysis patients

London M et al. J Am Soc Nephrol. 2008 Sep;19(9):1827–1835.

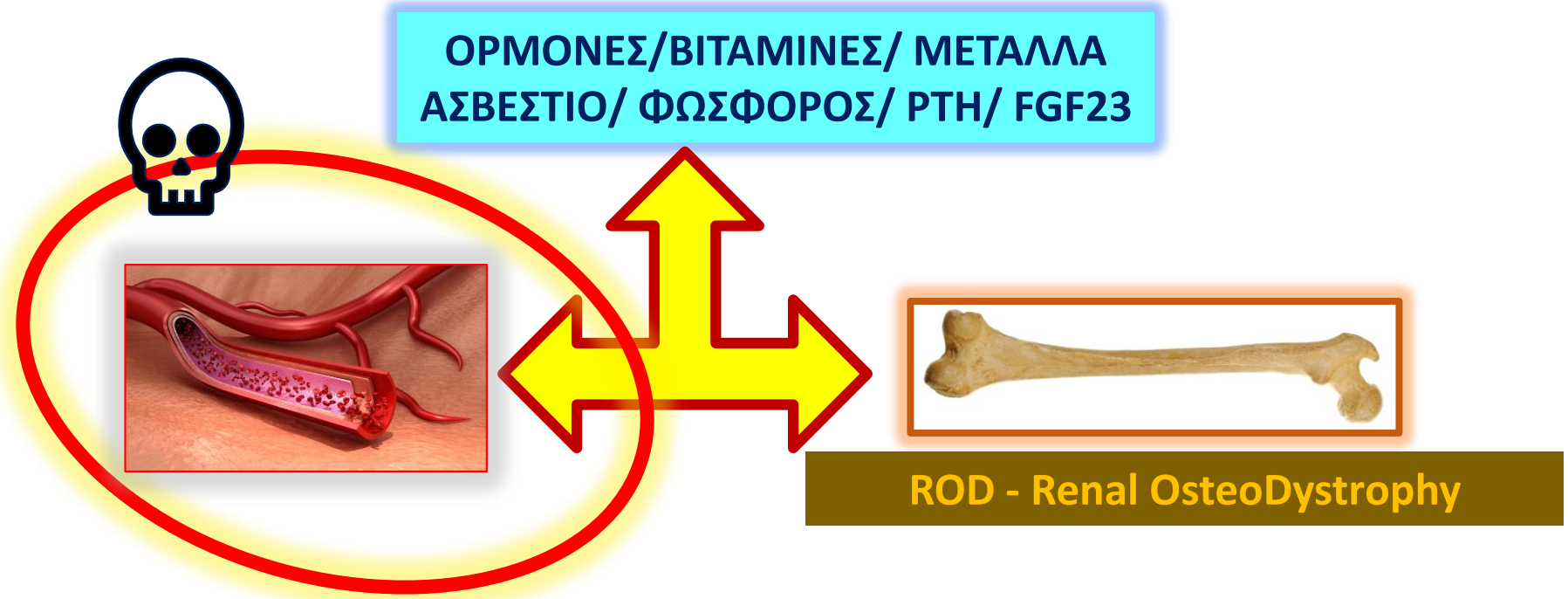


ΔΙΑΤΑΡΑΧΗ ΜΕΤΑΛΛΩΝ ΚΑΙ ΟΣΤΩΝ ΣΕ ΧΝΝ (CKD-Mineral Bone Disorder)

ΑΠΟ ΤΑ ΟΣΤΑ ΣΤΑ ΑΓΓΕΙΑ ...

θεραπευτικές αποφάσεις για την οστεοδυστροφία ...

σχετίζονται με αγγειακή νόσο και καρδιαγγειακή θνητότητα !!



Kidney International (2006) 69, 1945–1953

ΔΙΑΤΑΡΑΧΕΣ ΤΟΥ ΜΕΤΑΒΟΛΙΣΜΟΥ ΤΩΝ ΜΕΤΑΛΛΩΝ ΣΤΗ ΧΝΝ

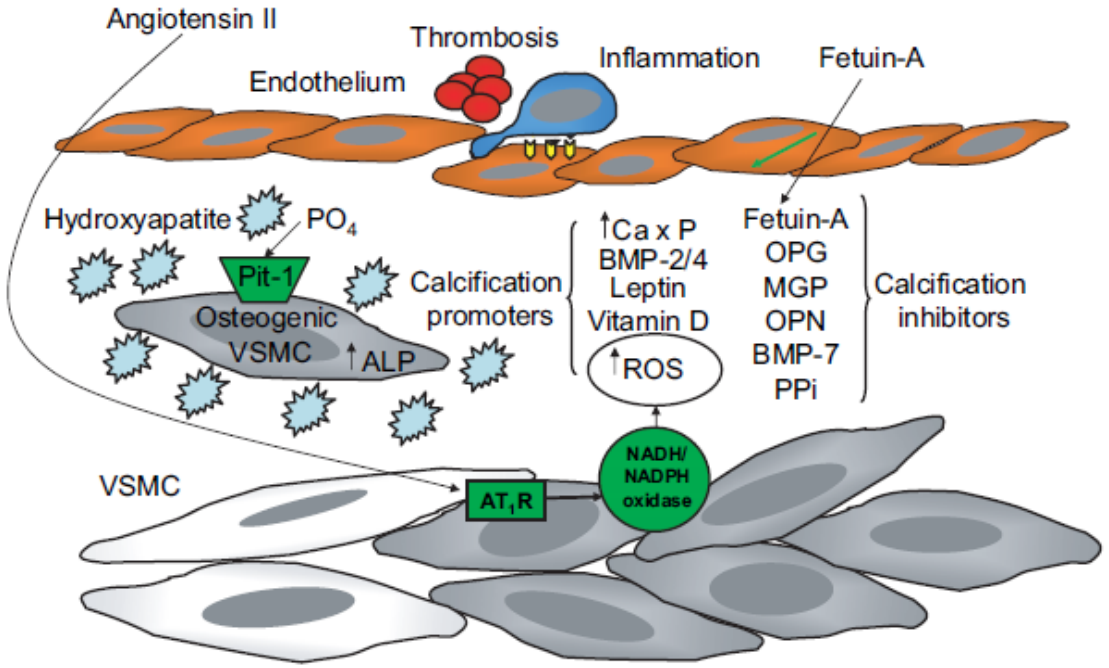
ΚΑΙ ΚΑΡΔΙΑΓΓΕΙΑΚΕΣ ΕΠΑΣΒΕΣΤΩΣΕΙΣ

Προάγουν την επασβέστωση:

Φωσφόρος, Ασβέστιο, βιτ-D, Υπερπαραθυρεοειδισμός, ουραιμία, φλεγμονή , Vit K ανταγωνιστές,

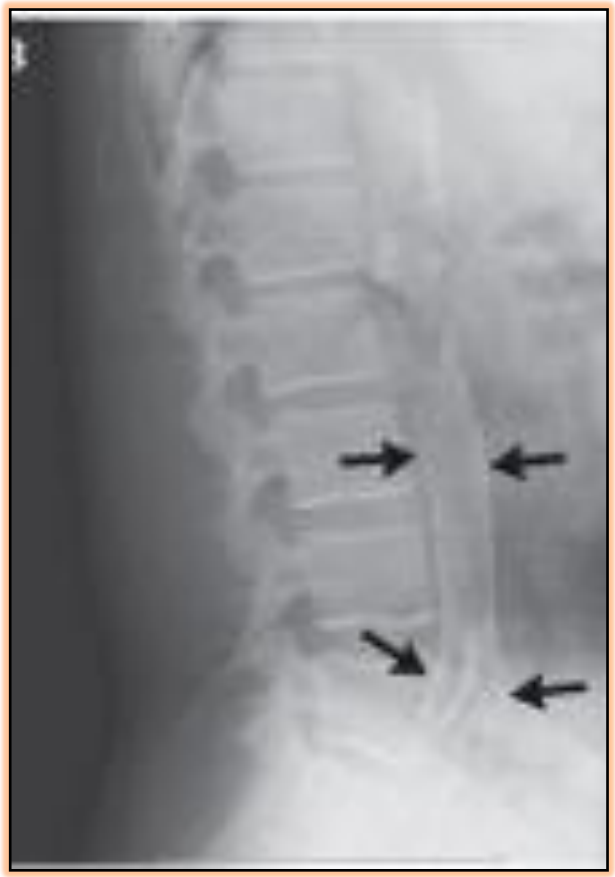
Αναστολείς των επασβεστώσεων:

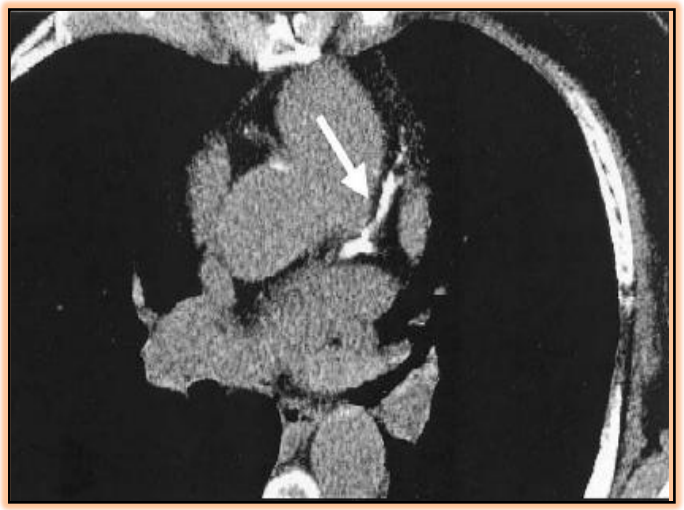
Φετουϊνη-Α, BMP-7, OPG, Μαγνήσιο, mGP, Vit K, PPi

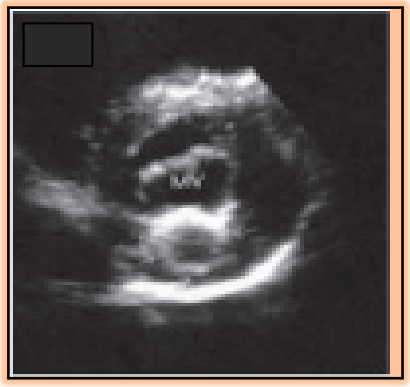


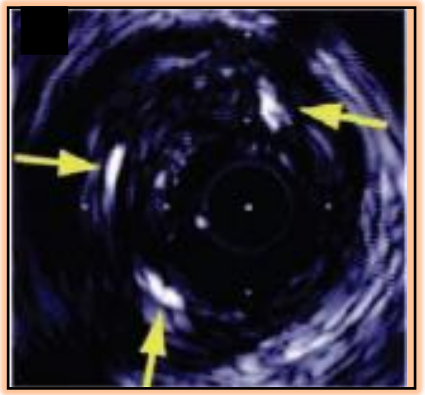
Circulation 2007;116:85-97

Καρδιαγγειακές επασβεστώσεις σε ΧΝΝ

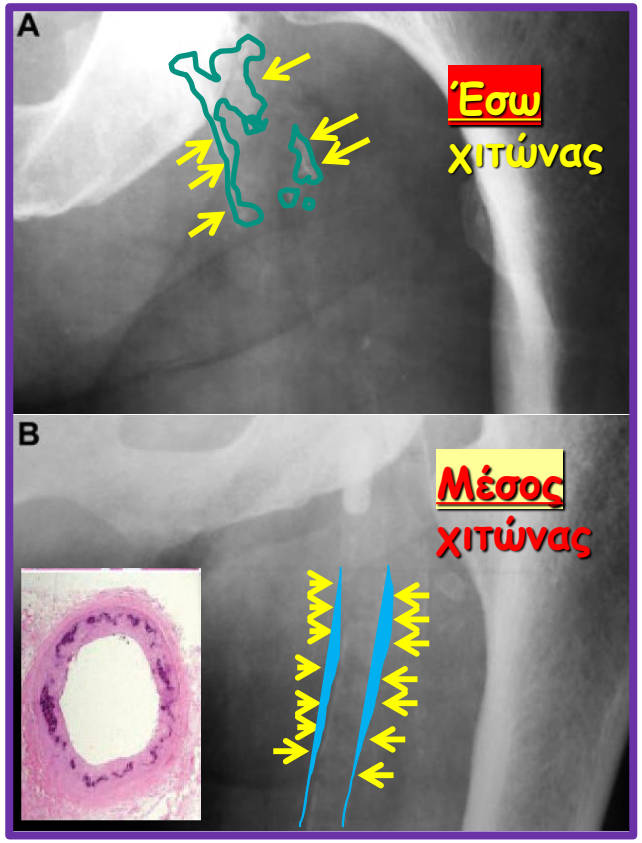








ΑΘΗΡΟΣΚΛΗΡΩΤΙΚΕΣ ΚΑΙ ΑΡΤΗΡΙΟΣΚΛΗΡΥΝΤΙΚΕΣ ΑΓΓΕΙΑΚΕΣ ΕΠΑΣΒΕΣΤΩΣΕΙΣ



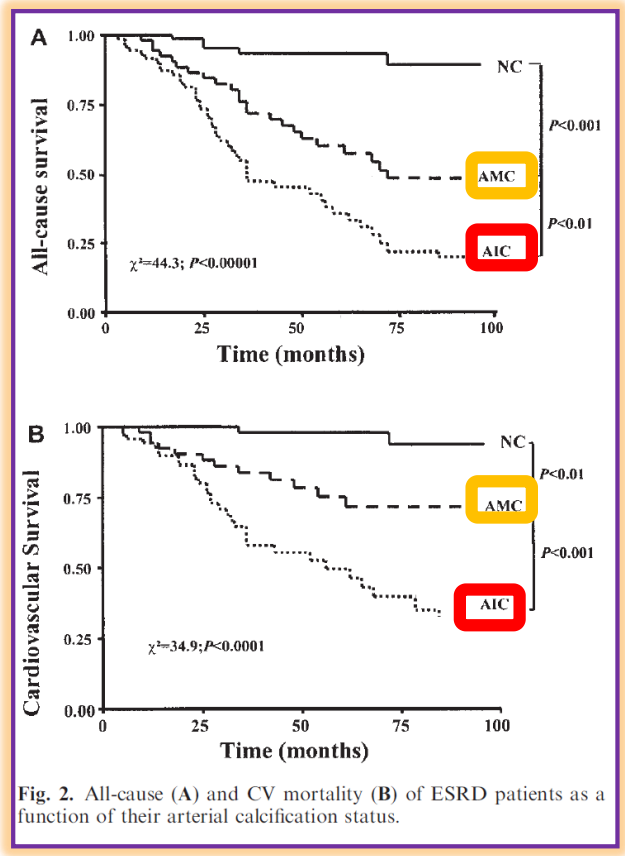
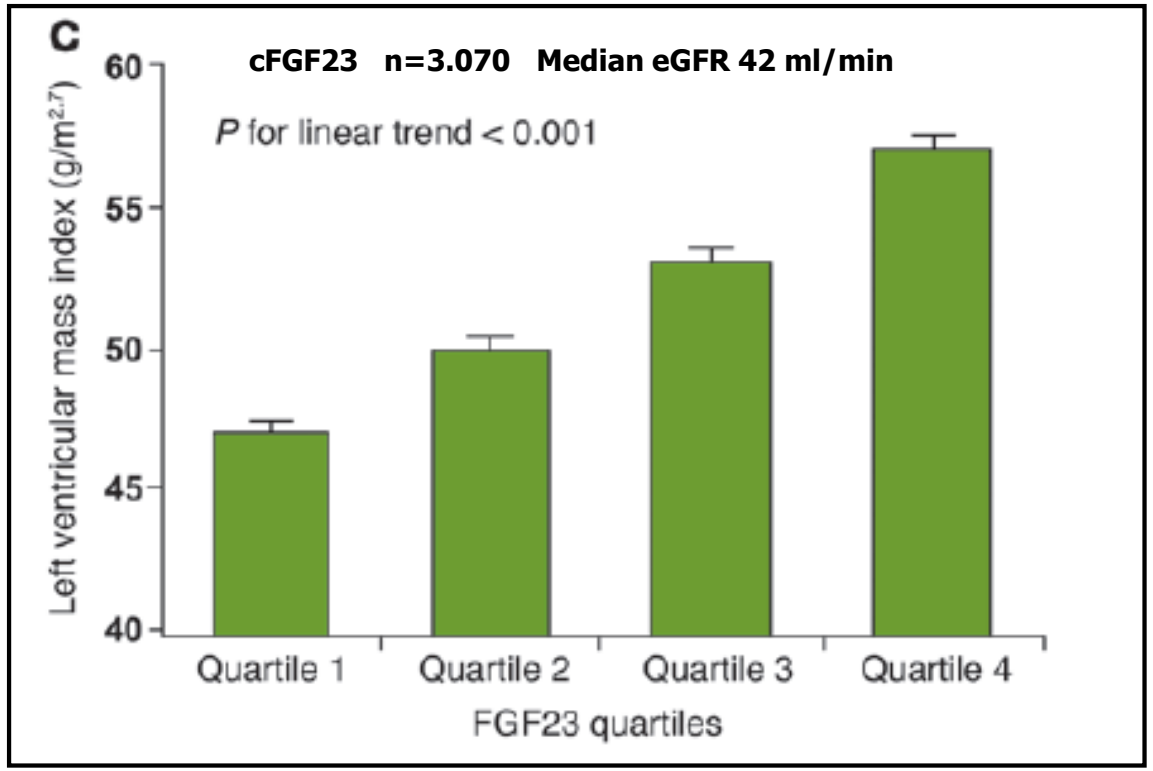


Fig. 2. All-cause (A) and CV mortality (B) of ESRD patients as a function of their arterial calcification status.

London G

Nephrol Dial Transplant (2003) 18: 1731–1740

FGF23 και νεοεμφανιζόμενη LVH σε νορμοτασικούς ασθενείς με ΧΝΝ



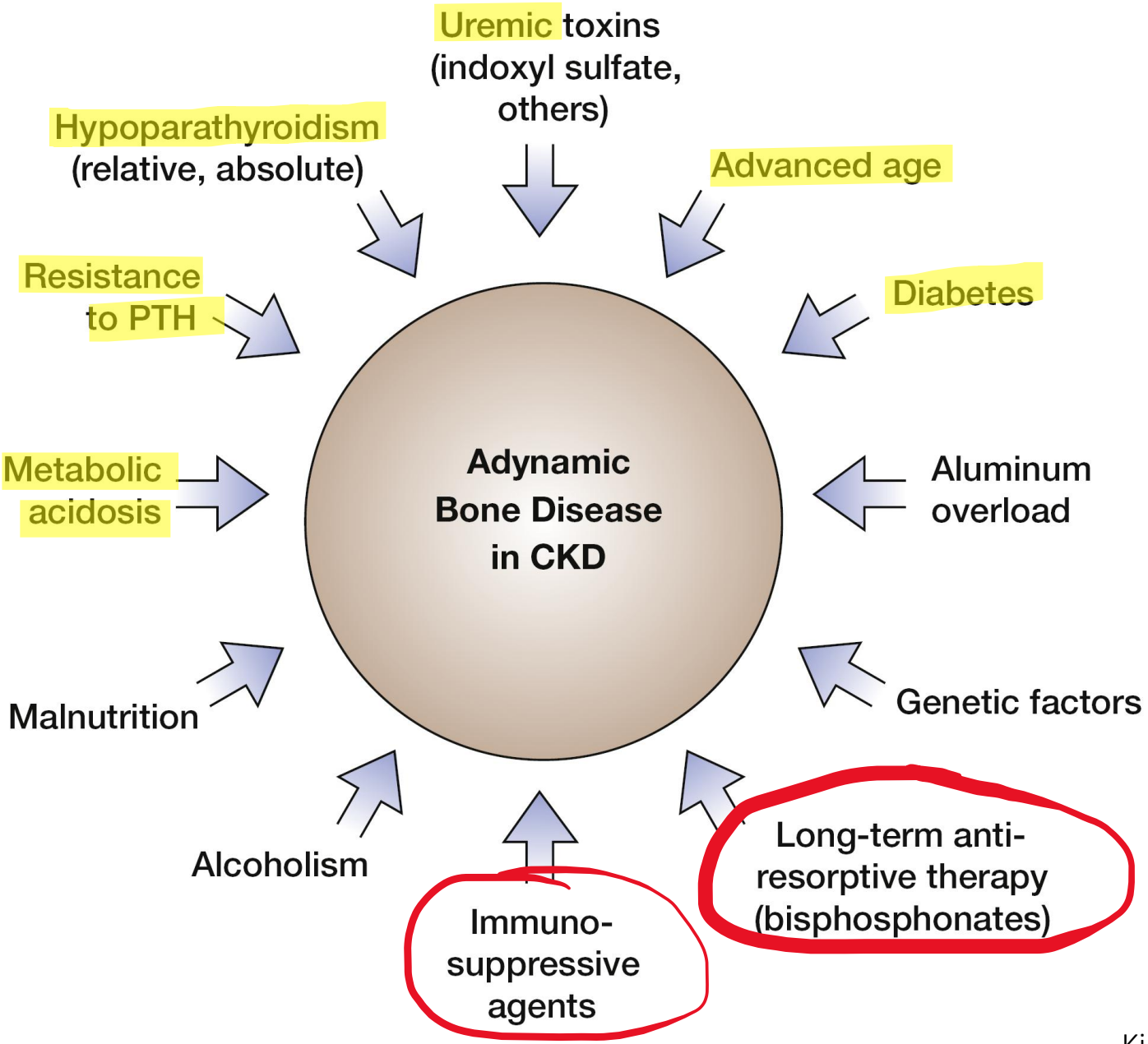
Elevated FGF23 is associated with increased risk of <u>new-onset LVH</u>

411 pts normal LV geometry at baseline,
84 (20%) new onset LVH (2.9±0.5 years later)

Elevated FGF23 levels at baseline were associated with increased future risk of new onset LVH both in normotensive subjects

Faul C et al.

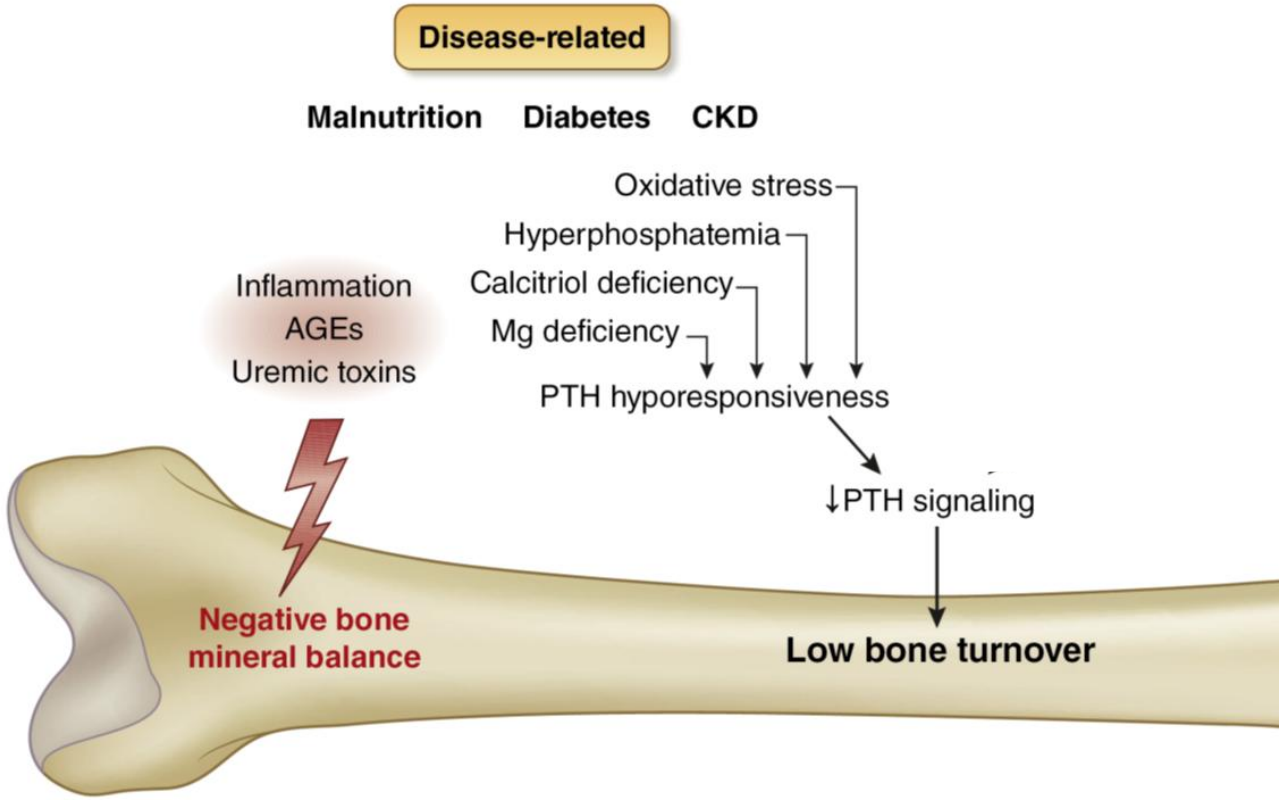
J Clin Invest 121: 4393, 2011



Drüeke TB, Massy ZA

Kidney Int. 2016 Feb;89(2):289-302

Αδυναμική οστική Νόσος στη ΧΝΝ και αντι-οστεοπορωτική θεραπεία ?



Haarhaus et al

Kidney Int. 2021 Sep;100(3):546-558

Η DXA-BMD ΔΕΝ ΜΠΟΡΕΙ ΝΑ ΔΙΑΚΡΙΝΕΙ ΤΟ ΕΙΔΟΣ ΤΗΣ ΟΣΤΙΚΗΣ ΝΟΣΟΥ

Diagnostic Accuracy of Biomarkers and Imaging for Bone Turnover in Renal Osteodystrophy

69 patients with CKD stages 4–5
BONE BIOPSY

	Low, *n*=11	Normal, *n*=15	High, *n*=17	*P* Value
DXA BMD Z score				
Forearm	−0.24 (0.88)	−0.31 (0.99)	−0.94 (1.33)	0.18
Total hip	−0.07 (0.76)	−0.35 (0.96)	−0.38 (1.05)	0.68
Lumbar spine	−0.10 (−0.5 to 0.6)	0.2 (−0.8 to 1.0)	0 (−0.8 to 1.3)	0.86

Salam S

J Am Soc Nephrol. 2018 May;29(5):1557-1565

ΒΙΟΔΕΙΚΤΕΣ ΟΣΤΙΚΟΥ ΜΕΤΑΒΟΛΙΣΜΟΥ ΣΤΗ ΧΝΝ

Bone formation markers

- PINP (recommended as the reference bone formation marker by IOF and IFCC)
- Osteocalcin
- Bone alkaline phosphatase
- C-terminal propeptide of type I procollagen

Bone resorption markers

- CTX-I (recommended as the reference bone resorption marker by IOF and IFCC)
- NTX-I
- CTX-I generated by MMPs (CTX-MMP)
- Helical peptide 620–633 of the α1 chain of type I collagen
- Deoxypyridinoline
- TRAP5b (also known as ACP5)

Δεν απεκκρίνονται ή αποδομούνται από τους νεφρούς και αποτελούν αξιόπιστους δείκτες του οστικού μεταβολισμού στη ΧΝΝ:

- *bone Alkaline Phosphatase (b-ALP)*
- *intact PINP*
  (Procollagen type I N-terminal propeptide)
- *TRAP5b*
  (Tartrate-resistant acid phosphatase 5b)

Eastell R, Szulc P

The Lancet Diabetes & Endocrinology, 2017; 5, 908-923

ΒΙΟΔΕΙΚΤΕΣ ΣΤΗ ΔΙΑΦΟΡΙΚΗ ΔΙΑΓΝΩΣΗ ΝΟΣΟΥ ΧΑΜΗΛΟΥ ΟΣΤΙΚΟΥ ΜΕΤΑΒΟΛΙΣΜΟΥ

Diagnostic Accuracy of Biomarkers and Imaging for Bone Turnover in Renal Osteodystrophy

69 patients with CKD stages 4–5
BONE BIOPSY

Table 3. Diagnostic accuracy of biomarkers and radius high-resolution peripheral quantitative computed tomography for identifying patients with low bone turnover

Variables	AUC (95% CI)	Criterion	Sensitivity, %	Specificity, %	PPV, %	NPV, %
Biomarkers						
iPTH	0.563 (0.40 to 0.72)	≤183 pg/ml	70	53	32	85
Intact PINP	0.794 (0.64 to 0.90)	≤57 ng/ml	80	75	50	92
bALP	0.824 (0.67 to 0.93)	≤21 μg/L	89	77	53	96
tALP	0.753 (0.60 to 0.87)	≤88 IU/L	91	63	46	95
TRAP5b	0.799 (0.64 to 0.91)	≤4.6 U/L	89	71	47	96

Salam S

J Am Soc Nephrol. 2018 May;29(5):1557-1565

ΒΙΟΔΕΙΚΤΕΣ ΣΤΗΝ ΠΡΟΒΛΕΨΗ ΤΟΥ ΟΣΤΙΚΟΥ ΜΕΤΑΒΟΛΙΣΜΟΥ ΣΕ ΧΝΝ G4-5D

Table 4. Diagnostic Performance of Biochemical Markers for a Diagnosis of High or Low Bone Turnover

	Exploration Cohort (n = 100)		Validation Cohort (n = 99)				
	AUC	Cutoff	Sensitivity	Specificity	PPV	NPV	Accuracy
High Turnover							
Biointact PTH, pg/mL	0.78 (0.67, 0.86)	>143.5	70%	74%	57%	84%	73%
Total AP, U/L	0.77 (0.65, 0.86)	>97	76%	77%	61%	87%	77%
BsAP, μg/L	0.83 (0.73, 0.91)	>33.7	73%	86%	71%	88%	82%
Intact PINP, ng/mL	0.85 (0.74, 0.93)	>120.7	73%	94%	85%	89%	88%
TRAP5b, U/L	0.78 (0.66, 0.86)	>5.05	77%	76%	59%	88%	76%
BsAP + intact PINP	0.84 (0.74. 0.94)	As above	63%	97%	90%	85%	86%
BsAP + TRAP5b	0.79 (0.70, 0.88)	As above	63%	91%	76%	85%	82%
PINP + TRAP5b	0.84 (0.74. 0.94)	As above	82%	94%	88%	91%	90%

Jørgensen HS

Am J Kidney Dis. 2022 May;79(5):667-676.e1

Bone turnover markers in the management of CKD-associated osteoporosis – a European consensus

Focus of study was to improve management of patients with chronic kidney disease (CKD)-associated osteoporosis by addressing methodological aspects and clinical usefulness of bone turnover markers (BTM) in adults and children with CKD.

Methods

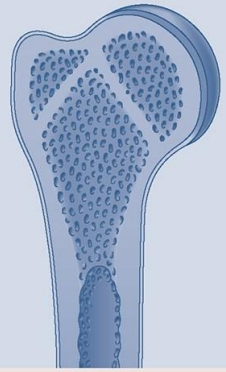
Literature review by expert panel; consensus conference

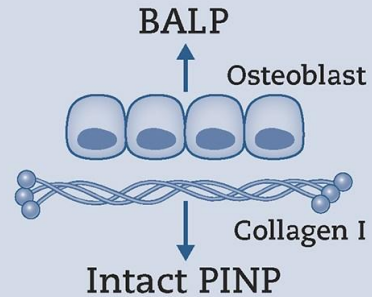
Delphi survey, including external experts

Final consensus based on survey replies

Results

Recommended BTM in CKD





Utility in CKD

Diagnosis

Risk assessment

Treatment decisions

Treatment monitoring

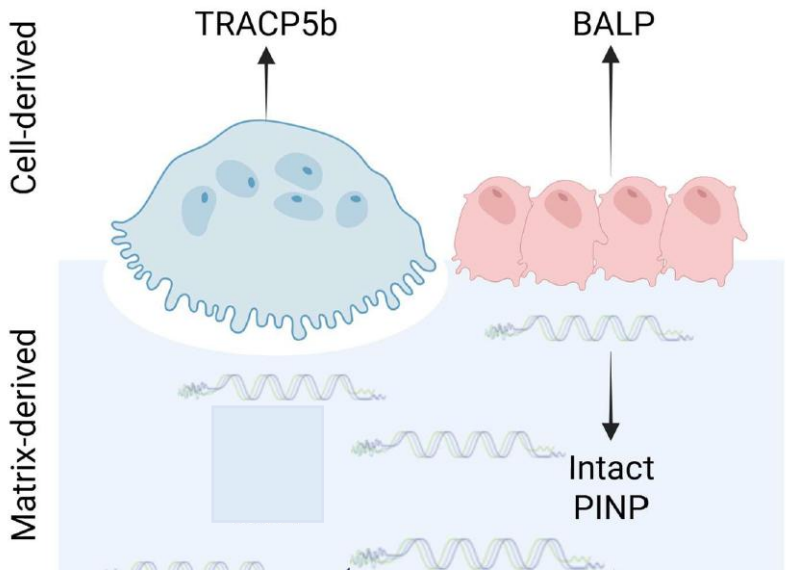
Key recommendations

- BALP and TRAcP5b are reference BTM in CKD, intact PINP is also useful
- Current skeletal remodeling rate can be assessed using BTM
- High and increasing levels of BTM indicate risk of bone loss and fractures
- BTM may guide treatment decisions in CKD-associated osteoporosis

Haarhaus, M. *et al.*
NDT (2026)
@NDTSocial

This consensus provides key evidence and clinical practice points on the utility of bone turnover markers in diagnosis, risk prediction, therapeutic decisions, and treatment evaluation in adults and children with CKD-associated osteoporosis.

ΒΙΟΔΕΙΚΤΕΣ ΣΤΗΝ ΠΡΟΒΛΕΨΗ ΤΟΥ ΟΣΤΙΚΟΥ ΜΕΤΑΒΟΛΙΣΜΟΥ ΣΕ ΧΝΝ



Clinical practice points

- A low bone turnover state may be suspected if BALP <20 µg/L (IDS iSYS) or 30 U/L (Quidel) or iPINP <50 µg/L (IDS iSYS) or TRACP5b <4.0 U/L..

- A high bone turnover state may be suspected if BALP >35 µg/L (IDS iSYS) or 40 U/L (Quidel), iPINP >120 µg/L (IDS iSYS) or TRACP5b >5.0 U/L (IDS iSYS).
- Consider the possibility of a mineralization defect if BALP levels are very high,

- ***intact PINP*** *(Procollagen type I N-terminal propeptide)*
- ***bone Alkaline Phosphatase*** *(b-ALP)*
- ***TRAP5b*** *(Tartrate-resistant acid phosphatase 5b)*

Haarhaus M

Nephrol Dial Transplant. 2026 Feb 17:gfag033

Η «οστεοπόρωση» σε προχωρημένη ΧΝΝ <u>υποδιαγιγνώσκεται *και υποθεραπεύεται*</u>

εξαιτίας…

- της πολυπλοκότητας της υποκείμενης οστικής νόσου
- της έλλειψης στοιχείων από μεγάλες κλινικές δοκιμές
- του φόβου αρνητικής αλληλεπίδρασης με την υποκείμενη CKD-MBD (ειδικά ενδεχόμενη αδυναμική οστική νόσο)
- πιθανής αρνητικής επίδρασης στην υπολειπόμενη νεφρική λειτουργία

<u>Κίνδυνοι</u> παράλληλοι αλλά με αντίθετη κατεύθυνση;

ΚΑΡΔΙΑΓΓΕΙΑΚΟΣ ΚΙΝΔΥΝΟΣ

- Συννοσηρότητα/θνητότητα
- Θεραπείας της CKD-MBD
- *Ποια CKD-MBD?*
- *Ποια θεραπεία ?*

ΚΙΝΔΥΝΟΣ ΚΑΤΑΓΜΑΤΩΝ

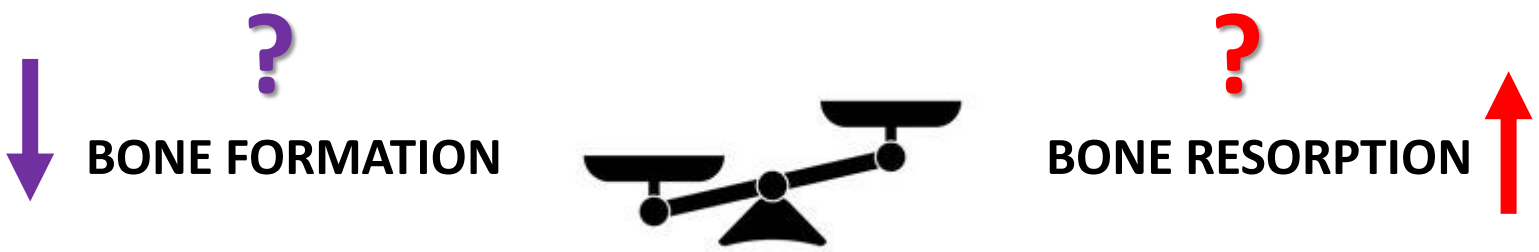
Συννοσηρότητα/θνητότητα

Θεραπεία της οστεοπόρωσης

Ποια οστεοπόρωση?

Ποια θεραπεία ?

ΟΥΤΕ Η ΟΣΤΕΟΠΟΡΩΣΗ ΕΧΕΙ ΠΑΝΤΑ ΤΟΥΣ ΙΔΙΟΥΣ ΜΗΧΑΝΙΣΜΟΥΣ



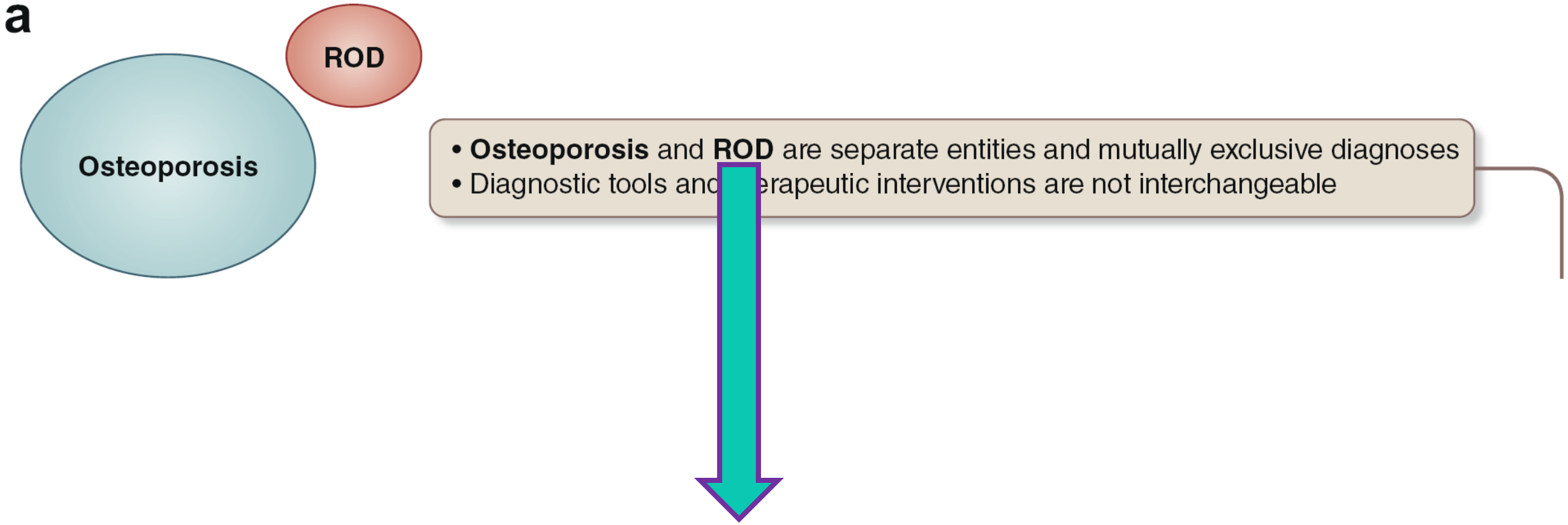
Postmenopausal estrogen deficiency		predominance of bone resorption
Aging		bone formation decreases
Glucocorticoids	*AND*	reduce function of osteoblasts increase bone resorption
Osteomalacia /rickets		defect in the mineralization

Pazianas M, Miller PD

Am J Kidney Dis. 2021 Oct;78(4):582-589

ΝΕΦΡΙΚΗ ΟΣΤΕΟΔΥΣΤΡΟΦΙΑ ΚΑΙ ΟΣΤΕΟΠΟΡΩΣΗ:

ΔΥΟ ΞΕΧΩΡΙΣΤΕΣ ΟΝΤΟΤΗΤΕΣ ΜΕ ΚΟΙΝΟ ΤΟΠΟ ΤΟΝ ΚΙΝΔΥΝΟ ΚΑΤΑΓΜΑΤΩΝ

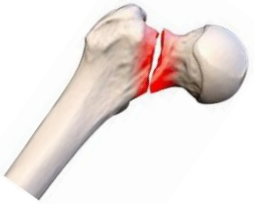


Ketteler M, Evenepoel P et al

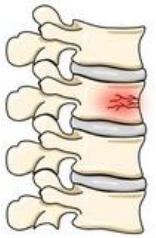
Kidney Int. 2025 Mar;107(3):405-423

Διαφορική διάγνωση: οστεοπόρωση έναντι νεφρικής οστεοδυστροφίας

- Στη νεφρική οστεοδυστροφία, είναι το φλοιώδες οστό που επηρεάζεται κυρίως



- Στην οστεοπόρωση, αντίθετα, το σπογγώδες οστό επηρεάζεται περισσότερο



- CKD MBD μπορεί να αναπτυχθεί σε έδαφος φυσιολογικού οστού ή οστεοπορωτικού
- η ΧΝΝ συχνά συνοδεύεται από άλλες καταστάσεις (υπογοναδισμός και γήρανση)

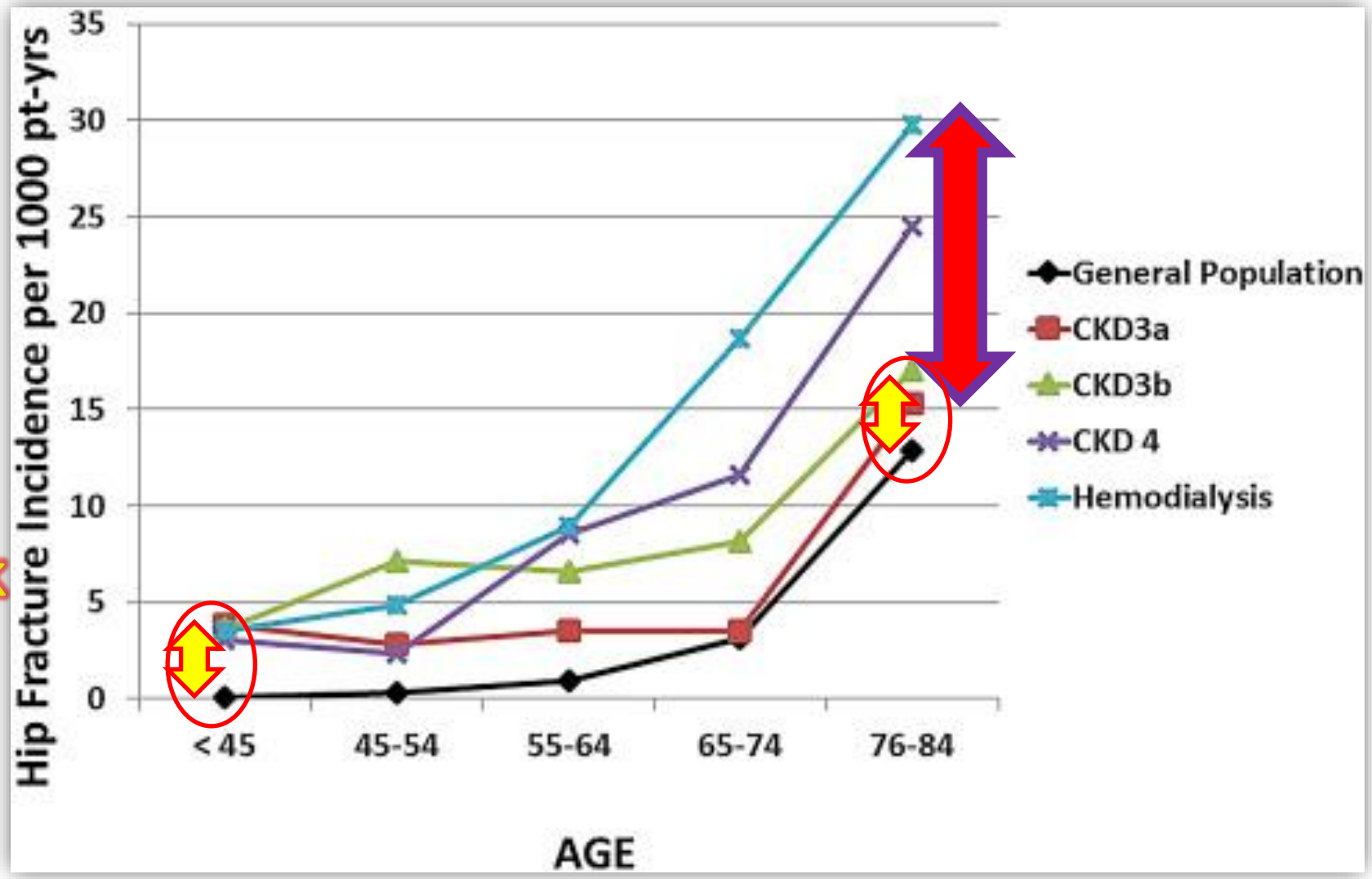
Pazianas M, Miller PD

Am J Kidney Dis. 2021 Oct;78(4):582-589

Επίπτωση καταγμάτων σε ασθενείς με ΧΝΝ, ανάλογα με το GFR

×4

fracture risk



Clin J Am Soc Nephrol 11: 1929–1931, 2016

Εντόπιση των καταγμάτων σε ασθενείς με ΧΝΝ, ανάλογα με το GFR

Table 3 | Location of the first fracture in follow-up according to kidney function

	eGFR (ml/min per 1.73 m^2)				
Fracture location	⩾60	45–59	30–44	15–29	<15 or chronic dialysis
Hip (%)	27.2	46.3	47.9	54.3	54.2
Forearm (%)	47.6	28.4	22.7	20.6	19.2
Proximal humerus (%)	15.0	13.5	13.8	11.4	8.4
Pelvis (%)	10.2	11.8	15.6	13.7	18.2

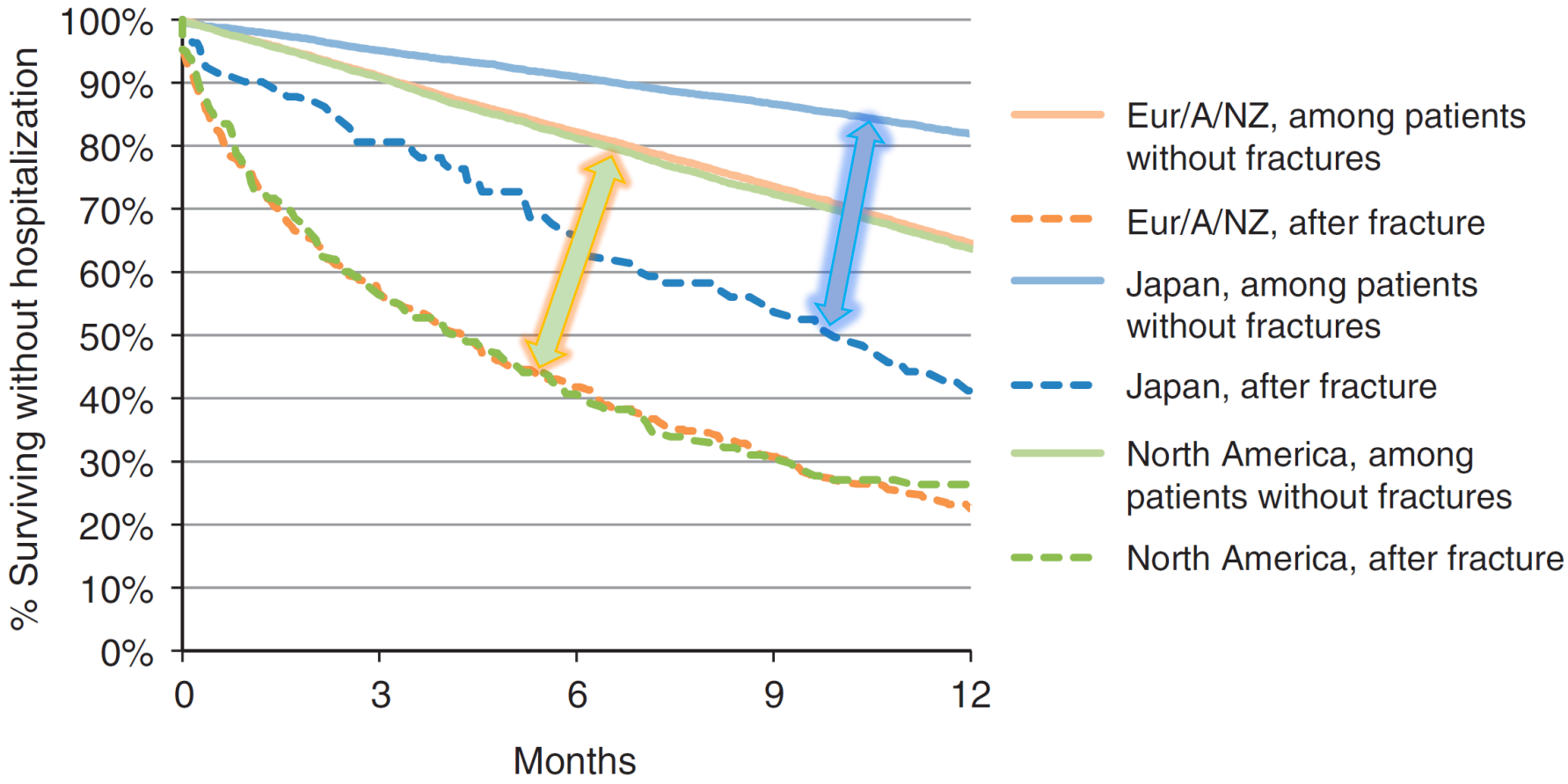
Abbreviation: eGFR, estimated glomerular filtration rate.

Naylor KL et al

Kidney Int. 2014 Oct;86(4):810-8

ΚΑΤΑΓΜΑΤΑ ΚΑΙ ΝΟΣΗΡΟΤΗΤΑ / ΘΝΗΤΟΤΗΤΑ ΣΕ ΑΣΘΕΝΕΙΣ ΜΕ ΧΡΟΝΙΑ ΝΕΦΡΙΚΗ ΝΟΣΟ

High rates of death and hospitalization follow bone fracture among hemodialysis patients



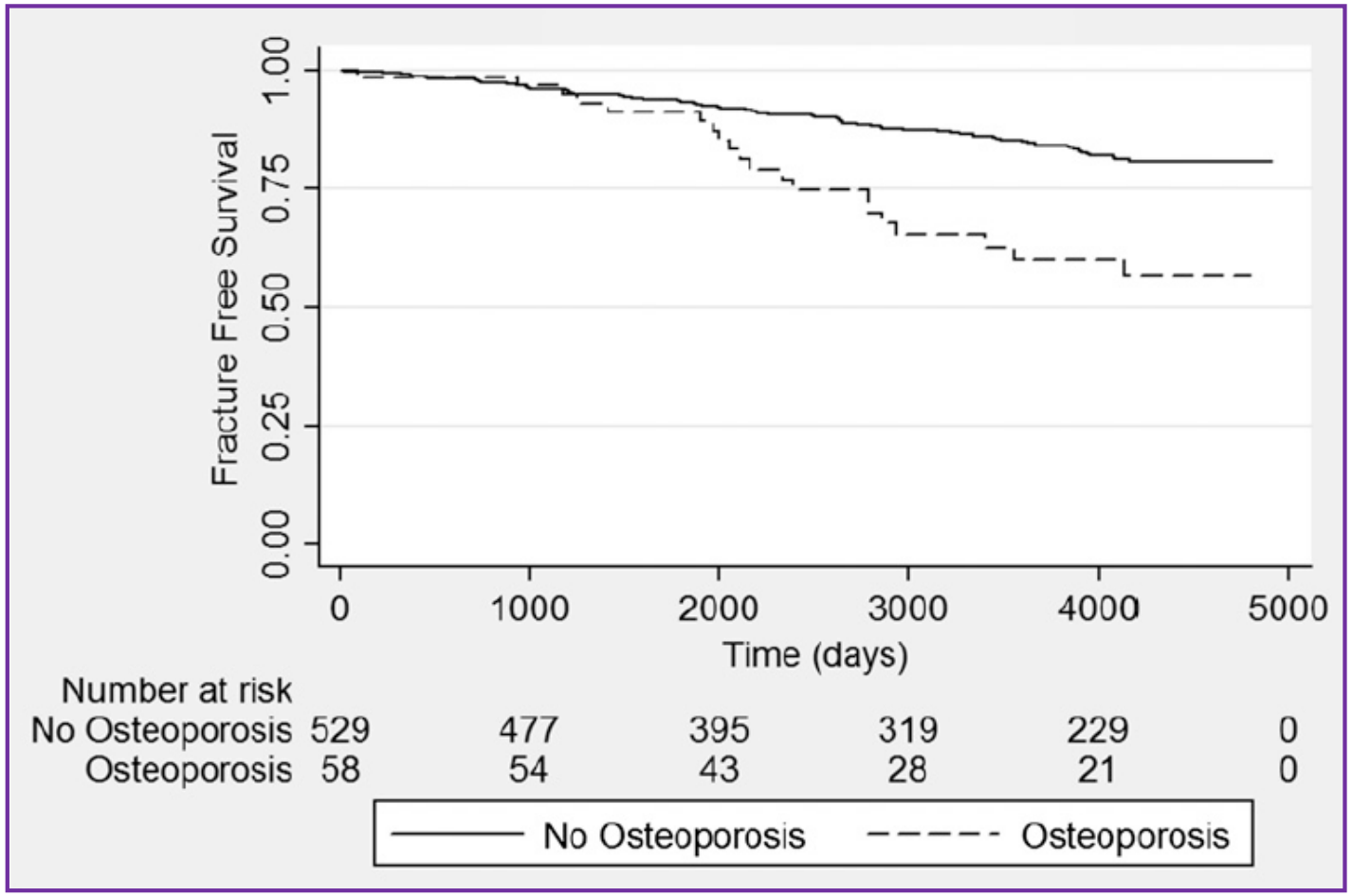
Kidney Int (2013) 85, 166–173

Ποιές εξετάσεις προβλέπουν τον κίνδυνο καταγμάτων στη ΧΝΝ ?

- *Η οστική πυκνότητα με DXA ?*
- *FRAX score?*
- *TBS (Trabecular bone score) ?*

Η οστική πυκνότητα προβλέπει τον κίνδυνο καταγμάτων σε ασθενείς με ΧΝΝ 3-5

Bone Mineral Density and Fracture Risk in Older Individuals with CKD



Health, Aging, and Body Composition Study
2754 συμμετέχοντες (μέση ηλικία ~73,6 έτη)

Εμφάνιση κατάγματος στους ασθενείς με ΧΝΝ, ανάλογα με την παρουσία οστεοπόρωσης σε DEXA

Yenchek R et al.

Clin J Am Soc Nephrol. 2012;7:1130–1136

ΟΣΤΙΚΗ ΠΥΚΝΟΤΗΤΑ ΜΕ DXA και QCT στην ΧΝΝ 5D

Diagnosis of low bone mass in CKD-5D patients

Gustav A. Blomquist[1], Daniel L. Davenport[2], Hanna W. Mawad[3], Marie-Claude Monier-Faugere[3], and Hartmut H. Malluche[3]

[1]Department of Radiology, [2]Department of Surgery, and [3]Division of Nephrology, Bone and Mineral Metabolism, University of Kentucky, Lexington, KY, USA

~20-22 % Οστεοπορωτική τιμή σε ΧΝΝ τελικού σταδίου <u>υπό αιμοκάθαρση</u>

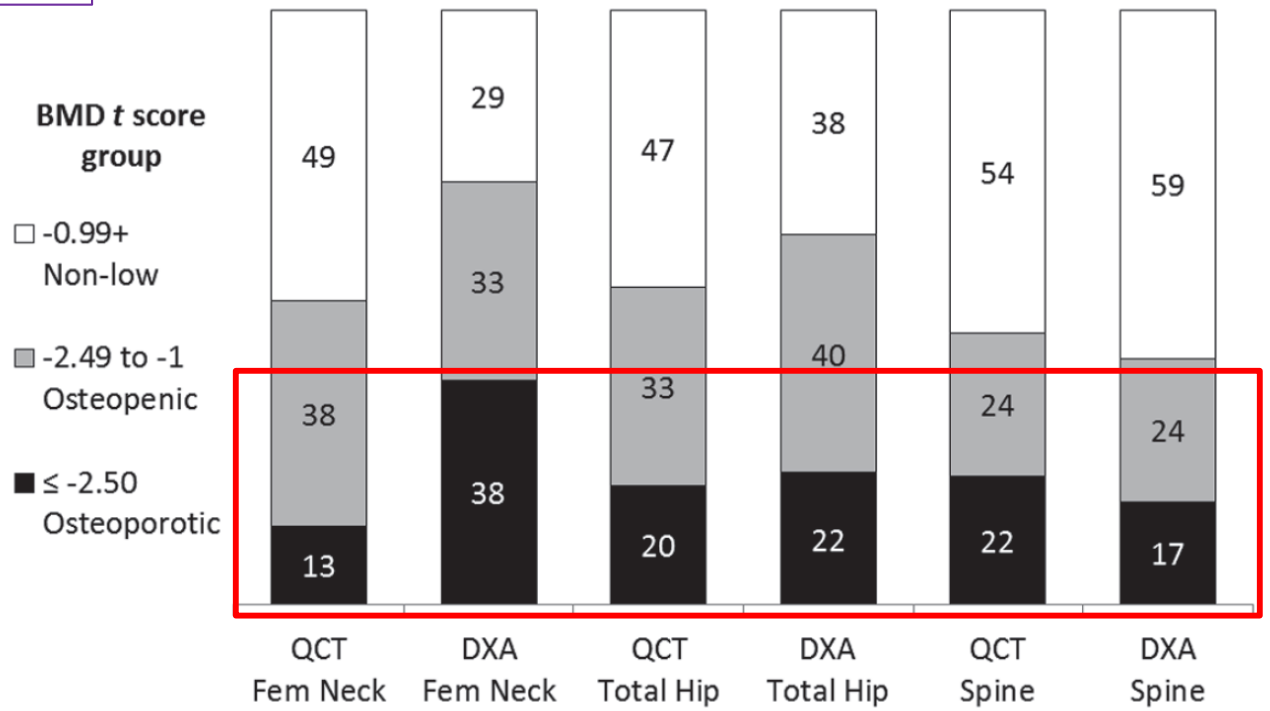


Figure 3. Percent of CKD-5D patients with osteopenia or osteoporosis by DXA or QCT at different sites.

Clinical Nephrology, Vol. 85 – No. 2/2016 (77-83)

Η οστική πυκνότητα προβλέπει τον κίνδυνο καταγμάτων σε ασθενείς με ΧΝΝ 5-5D

Bone turnover markers are associated with bone density, but not with fracture in end stage kidney disease: a cross-sectional study

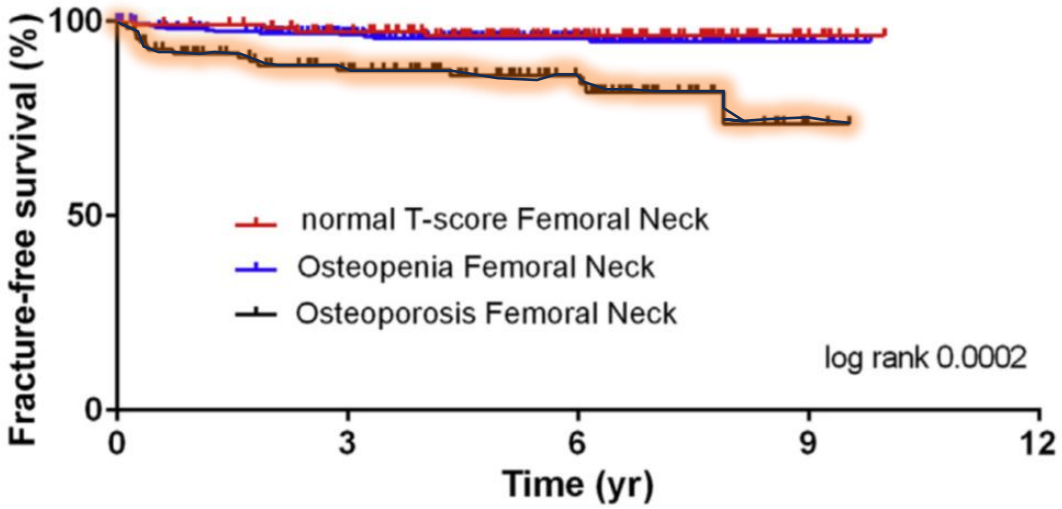
Table 4 Bone density measurements in adult kidney transplantation candidates

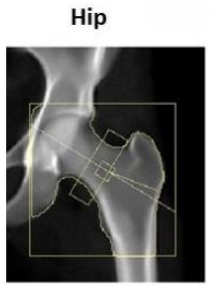
	No fracture (n = 129)	Any fracture (n = 28)	
Volumetric BMD, mg/cm^3			
Lumbar spine	126 ± 38	103 ± 37	†
Total hip	237 ± 43	196 ± 35	‡
Femoral neck	242 ± 50	195 ± 37	‡
Areal BMD, mg/cm^2			
Total hip	0.71 ± 0.12	0.61 ± 0.10	‡
Femoral neck	0.60 ± 0.10	0.51 ± 0.09	‡
Z-score			
Lumbar spine	−0.30 ± 1.37	−1.11 ± 1.21	†
Total hip	−1.08 ± 1.05	−0.94 ± 1.04	‡
Femoral neck	−0.91 ± 0.94	−1.77 ± 0.86	‡
T-score			
Lumbar spine	−1.77 ± 1.43	−2.60 ± 1.44	†
Total hip	−1.79 ± 1.05	−2.73 ± 0.89	‡
Femoral neck	−1.74 ± 0.93	−2.60 ± 0.79	‡

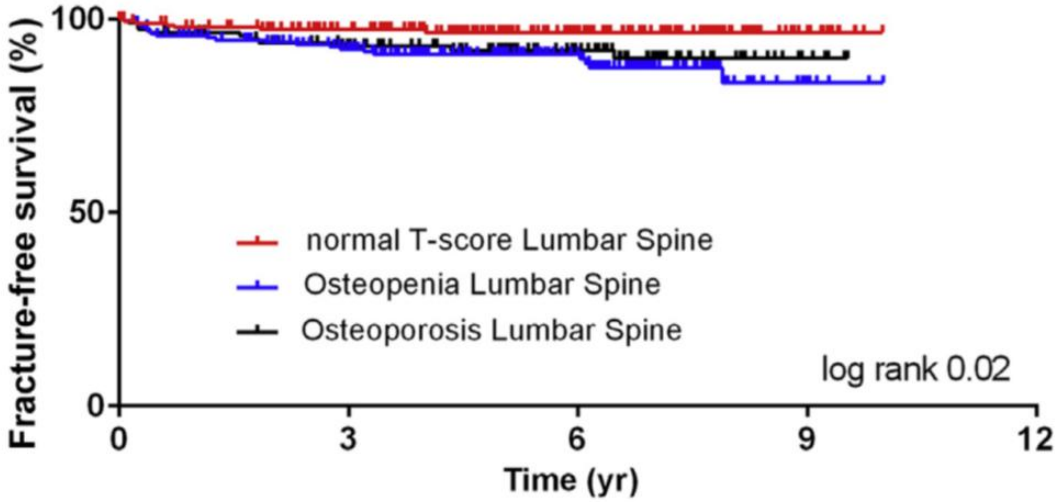
Jørgensen et al.

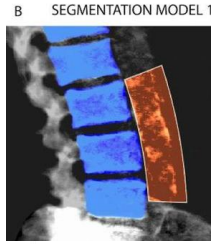
BMC Nephrology (2017) 18:284

Η BMD στο Ισχίο προβλέπει τον κίνδυνο κατάγματος σε μεταμοσχευμένους ασθενείς









Evenepoel P

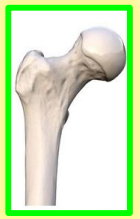
Kidney Int. 2019 Jun;95(6):1461-1470

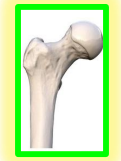
Η ΕΛΑΤΤΩΣΗ BMD ΤΑ ΠΡΩΤΑ 2 ΧΡΟΝΙΑ ΣΕ ΑΙΜΟΚΑΘΑΡΣΗ

ΑΦΟΡΑ ΚΥΡΙΩΣ ΤΟ ΦΛΟΙΩΔΕΣ ΟΣΤΟ! - ΙΣΧΙΟ !!

Table 2 Changes in BMD (±SD) after 1 and 2 years

	At 1 year		At 2 years	
No. of paired measurements	137		89	
BMD method and location	Mean absolute change ± SD	Percent change ± SD	Mean absolute change ± SD	Percent change ± SD
QCT total hip BMD (mg/cm^3)	− 11.5 ± 23.6***	− 4.0% ± 7.8***	− 17.3 ± 22.1***	− 5.9% ± 8.5***
Total hip cortex				
Total hip cortical mass (g)	− 0.8 ± 2.2***	− 4.0% ± 15.0**	− 1.5 ± 2.9***	− 7.3% ± 17.1***
Total hip cortical volume (cm^3)	− 1.1 ± 2.8***	− 6.4% ± 18.8***	− 2.2 ± 3.5***	− 10.0% ± 20.0***
Total hip trabeculum				
DXA total hip BMD (g/cm^2)	− 0.011 ± 0.047**	− 1.2% ± 5.2**	− 0.031 ± 0.057***	3.1% ± 5.9***





Malluche HH

Osteoporos Int. 2018 Jan;29(1):125-134

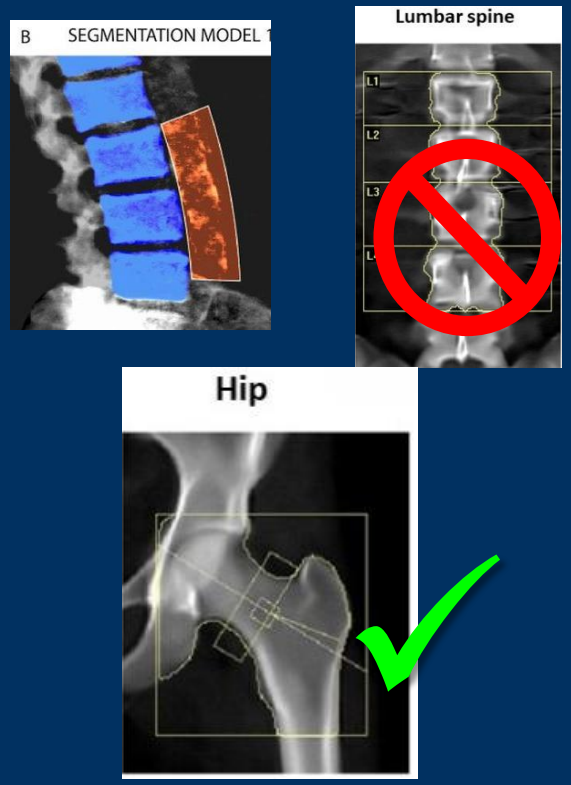
Ποιά Οστική Πυκνότητα ?

Chronic kidney disease–mineral and bone disorder: conclusions from a Kidney Disease: Improving Global Outcomes (KDIGO) Controversies Conference **2025**

*Σε **ΧΝΝ και υπερπαραθυρεοειδισμό, το φλοιώδες οστό** είναι το πιο προσβεβλημένο*

Η DEX της ΟΜΣΣ έχει δύο προβλήματα:

1. Η ΟΜΣΣ αποτελείται **>90% από δοκιδώδες οστό**
2. η προσθιοπίσθια **DXA περιλαμβάνει και την δυνητικά ασβεστοποιημένη αορτή**

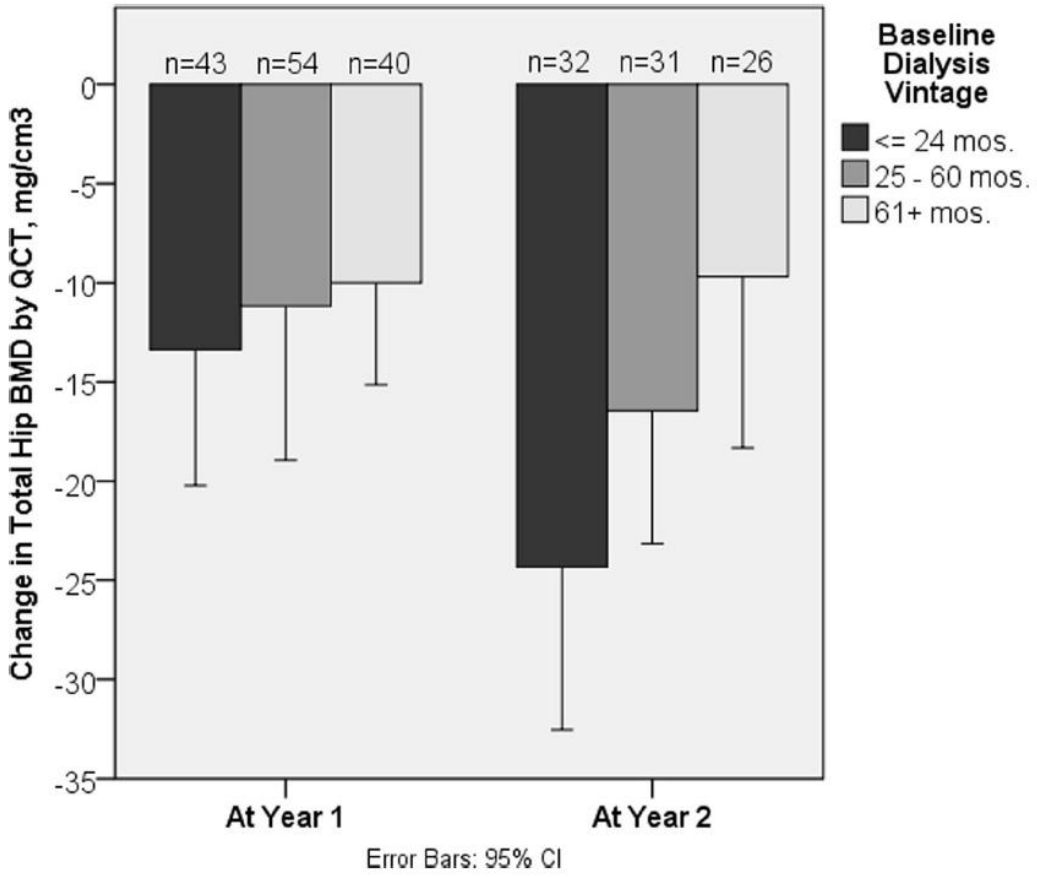


The total hip is the preferred site to measure BMD !
(*mixture of cortical and trabecular bone*)

Ketteler M, Evenepoel P et al

Kidney Int. 2025 Mar;107(3):405-423

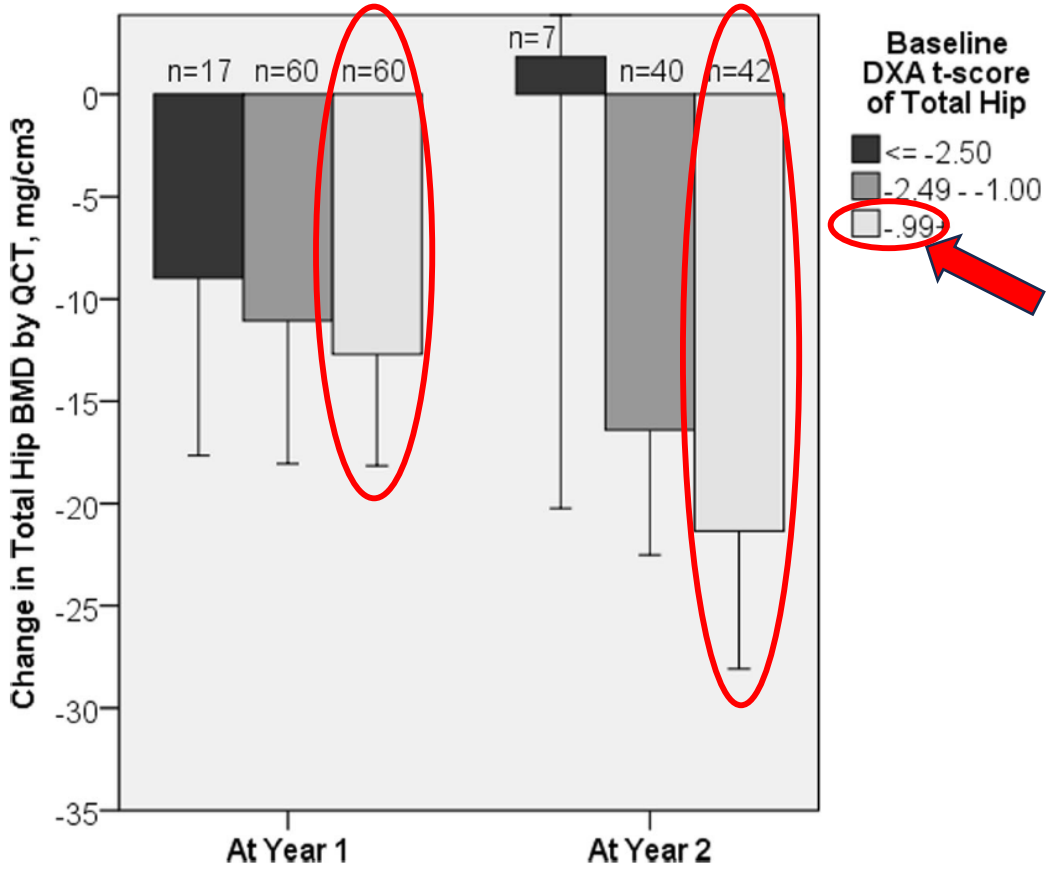
Η ΕΛΑΤΤΩΣΗ BMD ΕΙΝΑΙ ΜΕΓΑΛΥΤΕΡΗ ΤΑ ΠΡΩΤΑ 2 ΧΡΟΝΙΑ ΣΕ ΑΙΜΟΚΑΘΑΡΣΗ



Malluche HH

Osteoporos Int. 2018 Jan;29(1):125-134

Η ΕΛΑΤΤΩΣΗ BMD ΕΙΝΑΙ ΜΕΓΑΛΥΤΕΡΗ ΤΑ ΠΡΩΤΑ 2 ΧΡΟΝΙΑ ΣΕ ΑΙΜΟΚΑΘΑΡΣΗ

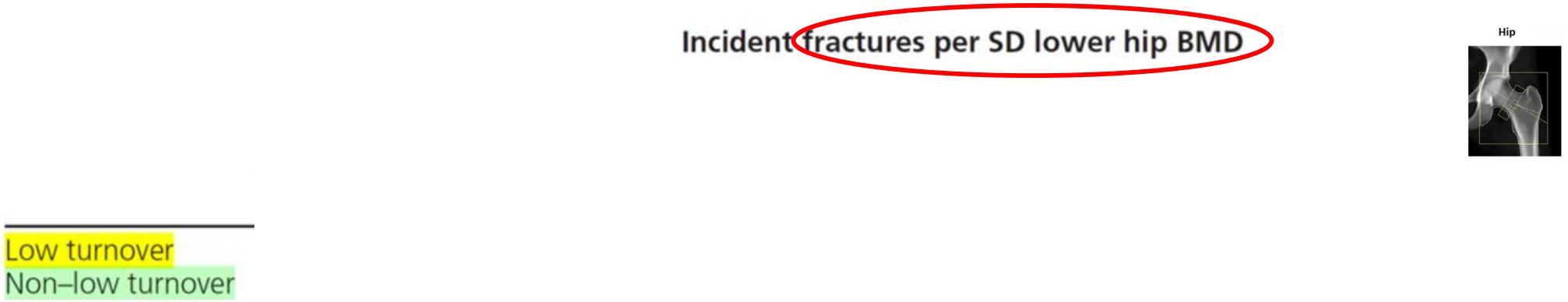


ΚΑΙ ΜΕΓΑΛΥΤΕΡΗ ΓΙΑ ΑΥΤΟΥΣ ΠΟΥ ΕΙΧΑΝ ΑΡΧΙΚΑ ΚΑΛΥΤΕΡΗ BMD !

Malluche HH

Osteoporos Int. 2018 Jan;29(1):125-134

Η BMD ΣΧΕΤΙΖΕΤΑΙ ΜΕ ΜΕΓΑΛΥΤΕΡΟ ΚΙΝΔΥΝΟ ΚΑΤΑΓΜΑΤΟΣ ΣΕ ΑΣΘΕΝΕΙΣ ΜΕ ΧΝΝ ΚΑΙ ΔΕΙΚΤΕΣ ΧΑΜΗΛΟΥ ΟΣΤΙΚΟΥ ΜΕΤΑΒΟΛΙΣΜΟΥ



<u>Για την ίδια ελάττωση της οστικής πυκνότητας (T-score)</u>

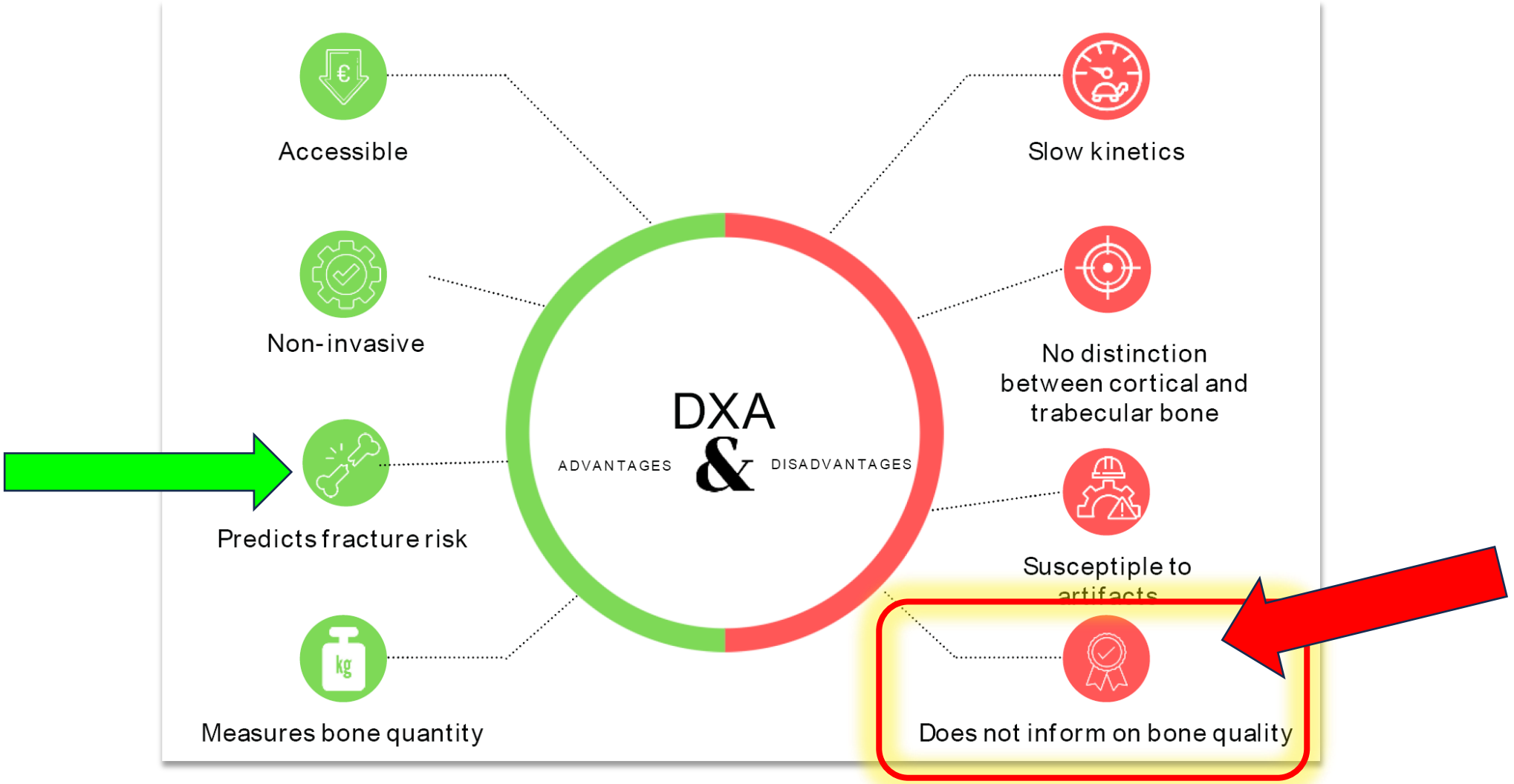
<u>οι ασθενείς με χαμηλό οστικό μεταβολισμό έχουν σχεδον 4πλάσιο κίνδυνο</u>

κατάγματος σε σύγκριση με αυτούς με αυξημένο οστικό μεταβολισμό

Hughes-Austin JM

J Clin Endocrinol Metab. 2020 Aug 1;105(8):e2903–11

ΠΛΕΟΝΕΚΤΗΜΑΤΑ ΚΑΙ ΜΕΙΟΝΕΚΤΗΜΑΤΑ ΤΗΣ DEXA BMD

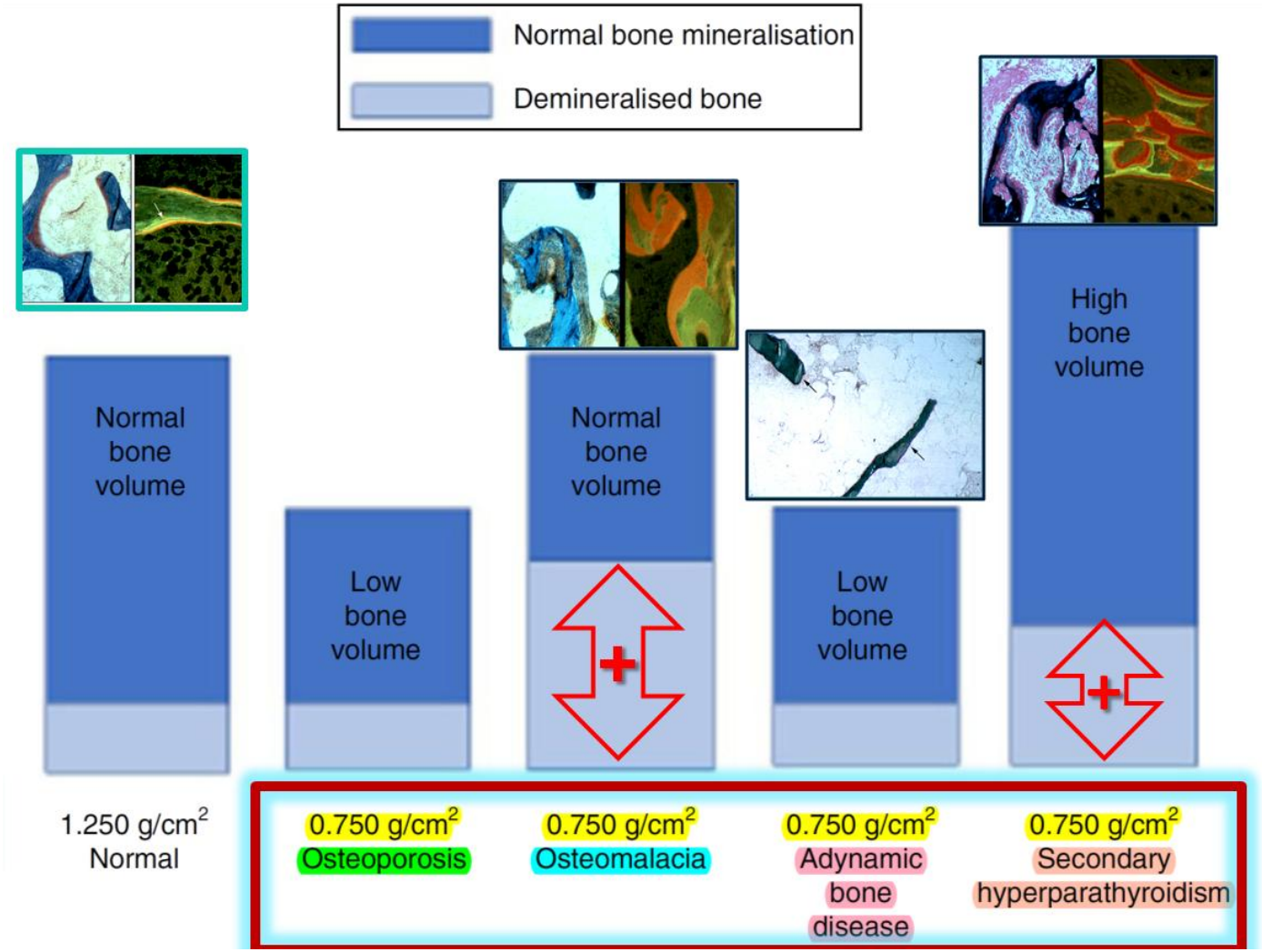


Lloret MJ et al

J Clin Med 2024;13:1010

ΝΕΦΡΙΚΗ ΟΣΤΕΟΔΥΣΤΡΟΦΙΑ και ΟΣΤΕΟΠΟΡΩΣΗ ΣΤΗ ΧΝΝ

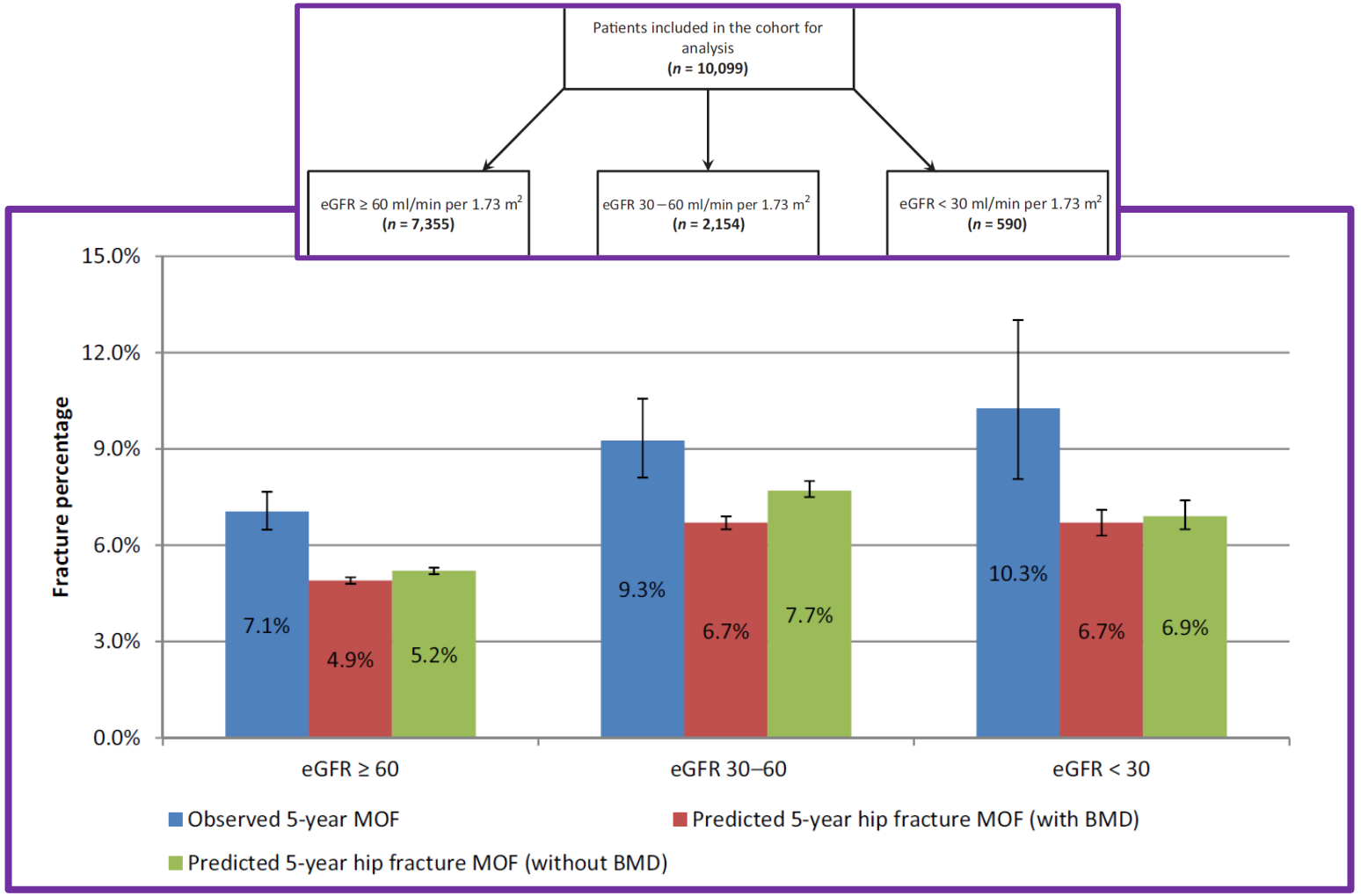
ΔΕΝ ΕΙΝΑΙ ΕΝΑ ΕΙΔΟΣ ΟΣΤΙΚΗΣ ΝΟΣΟΥ ... ΑΛΛΑ Η DEXA ΜΠΟΡΕΙ ΝΑ ΕΙΝΑΙ Η ΙΔΙΑ !!!



Bover J

Nefrologia (Engl Ed). 2018 Sep-Oct;38(5):476-490

FRAX και πρόγνωση καταγμάτων σε ΧΝΝ σε όλο το εύρος GFR



Whitlock R et al

Kidney Int. 2019 Feb;95(2):447-454

FRAX και πρόγνωση καταγμάτων σε ΧΝΝ σε υπό Αιμοκάθαρση

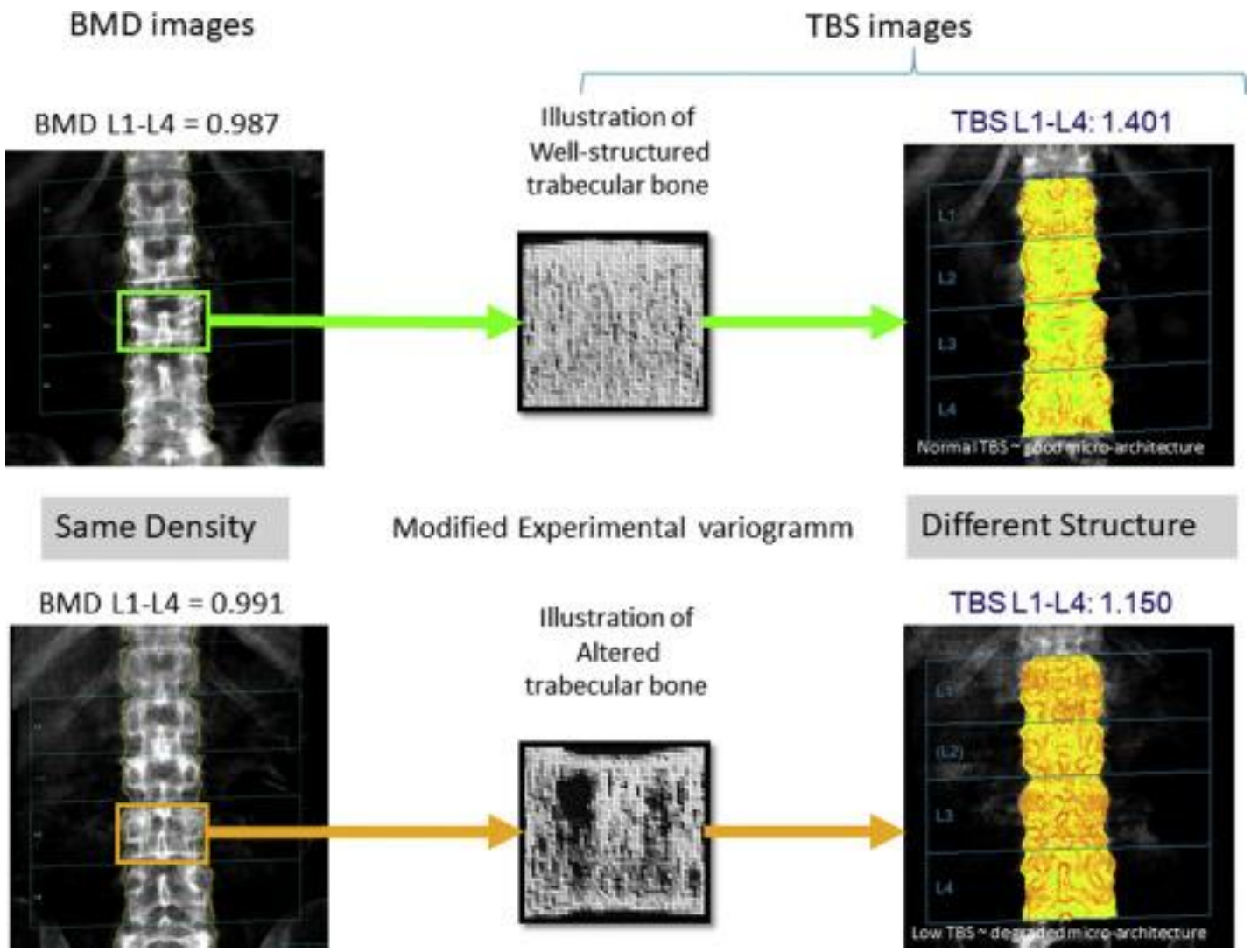
The utility of FRAX® in predicting bone fractures in patients with chronic kidney disease on hemodialysis: a two-year prospective multicenter cohort study

Risk factors	Major fracture		
	No. of analysis	AUC	95% CI
FRAX® (%)	718	0.76	0.69–0.84
Age (years)	718	0.66	0.55–0.76
Sex (women/men)	718	0.54	0.47–0.64
BMI (kg/m^2)	718	0.56	0.47–0.64
Previous fracture	718	0.59	0.50–0.67
Parent fractured hip	718	0.55	0.49–0.62
Current smoking	718	0.47	0.41–0.54
Glucocorticoids current/past	718	0.61	0.52–0.70
Rheumatoid arthritis	718	0.50	0.47–0.54
Alcohol	718	0.51	0.47–0.56
Diabetes (all types)	718	0.47	0.39–0.55
Diabetes type 1	718	0.49	0.49–0.50

Przedlacki J et al

Osteoporos Int. 2018 May;29(5):1105-1115

Trabecular Bone Score / εκτίμηση της δοκίδωσης του σπογγώδους οστού



Προγνωστική αξία Trabecular Bone Score σε ασθενείς με ΧΝΝ

Table 2 Hazard ratios for lumbar spine trabecular bone score to predict MOF stratified by kidney function

HR per SD decrease (95% CI)	eGFR ≥ 60 (n = 6224)	eGFR < 60 (n = 2065)	eGFR 30–60 (n = 1624)	eGFR < 30 (n = 441)	Interaction (TBS*CKD)
TBS adjusted for FRAX MOF with BMD	*1.12 (1.04–1.20)*	1.10 (0.98–1.23)	1.10 (0.97–1.25)	1.07 (0.85–1.35)	0.77
Total MOF	416	177	137	40	

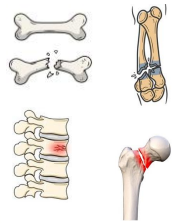
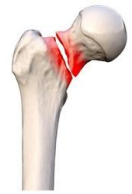


Table 3 Hazard ratios for lumbar spine trabecular bone score to predict hip fractures stratified by kidney function

TBS adjusted for FRAX hip with BMD	1.08 (0.93–1.26)	1.11 (0.93–1.32)	1.11 (0.91–1.36)	1.08 (0.72–1.61)	0.91
Total hip fractures	95	68	55	13	



Rampersad C.

Osteoporos Int. 2020 Oct;31(10):1905-1912

ΠΛΑΓΙΕΣ ακτινογραφίες ΘΜΣΣ και ΟΜΣΣ

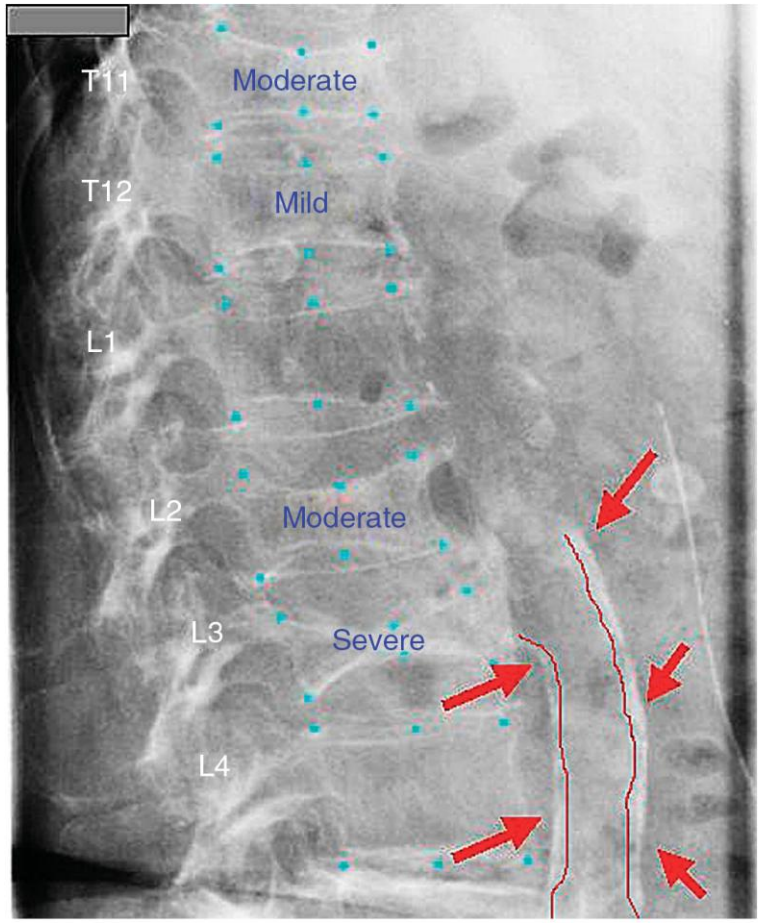


Figure 1 | Example of assessment of vertebral fractures with the aid of quantitative vertebral morphometry (QVM). Turquoise dots allow the delimitation of vertebral heights

ΘΕΡΑΠΕΙΑ

Οστεοπόρωσης(±CKD-MBD)

σε ασθενείς με Χρόνια Νεφρική Νόσο

Αντιοστεοπορωτικά φαρμάκα και ΧΝΝ

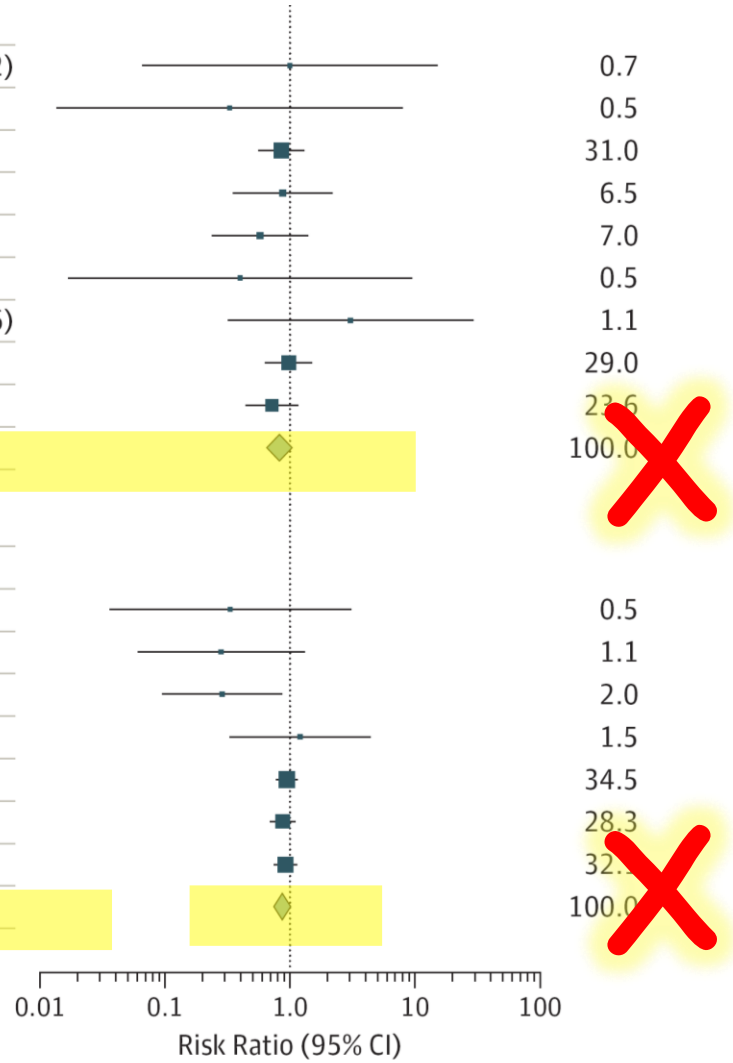
	Advantages	Disadvantages
Calcium	Can reduce risk of sHPT and skeletal mineralization defects Lowers phosphorus load May improve BMD in combination with vitamin D	Excessive use may increase cardiovascular risk and risk for kidney stones
Vitamin D	Can reduce risk of sHPT and skeletal mineralization defects May improve BMD in combination with calcium Active vitamin D preferrable in CKD 5D Nutritional vitamin D preferable in CKD 4–5	Stimulates FGF23 Excessive use may increase cardiovascular risk Active vitamin D should be restricted to patients with SHPT and can in that case also be used as adjuvant therapy with bone-specific agents
Bisphosphonates	Can improve BMD in all stages of CKD Persistent effect after cessation No evidence for increased cardiovascular risk	Increased systemic retention May be associated with CKD progression in CKD 4–5 and reduced residual renal function in CKD 5D Occasional reports of AKI with intravenous use
Denosumab	Can improve BMD in all stages of CKD No evidence of increased cardiovascular or renal risk No dose adaptation needed in any stages of CKD, including CKD 5D	Risk for hypocalcemia (especially in severe HPT) Rapid BMD deterioration and increased fracture risk after cessation
PTH analogues	Can improve BMD in all stages of CKD May improve suppressed BFR	Safety uncertain Optimal dosing uncertain May aggravate existing hyperparathyroidism
Romosozumab	May improve BMD in all stages of CKD Anabolic and antiresorptive effect	May induce hypocalcaemia Cardiovascular safety uncertain Optimal dosing uncertain
HRT	Can improve BMD in all stages of CKD	Safety uncertain Limited to early menopause

Συμπληρώματα ασβεστίου και βιταμίνης D

στη θεραπεία της οστεοπόρωσης και στη ΧΝΝ

Ασβέστιο και κίνδυνος κατάγματος ...

Vertebral fracture						
Hansson and Roos,[41] 1987	1	25	1	25	1.00 (0.07-15.12)	0.7
Reid et al,[42] 1993	0	68	1	67	0.33 (0.01-7.92)	0.5
Recker et al,[44] 1996	27	95	34	102	0.85 (0.56-1.30)	31.0
Riggs et al,[46] 1998	8	119	9	117	0.87 (0.35-2.19)	6.5
Peacock et al,[49] 2000	7	126	13	135	0.58 (0.24-1.40)	7.0
Avenell et al,[51] 2004	0	29	1	35	0.40 (0.02-9.46)	0.5
RECORD,[54] 2005	3	1311	1	1332	3.05 (0.32-29.26)	1.1
Prince et al,[56] 2006	38	730	39	730	0.97 (0.63-1.51)	29.0
Reid et al,[57] 2006	27	732	38	739	0.72 (0.44-1.16)	23.6
Total	111	3235	137	3282	0.83 (0.66-1.05)	100.0
Heterogeneity: $\tau^2 = 0.00$; $\chi^2_8 = 3.37$ ($P = .91$); $I^2 = 0\%$						
Test for overall effect: $z = 1.52$ ($P = .13$)						
Total fracture						
Inkovaara et al,[40] 1983	1	42	3	42	0.33 (0.04-3.08)	0.5
Reid et al,[42] 1993	2	68	7	67	0.28 (0.06-1.31)	1.1
Baron et al,[47] 1999	4	464	14	466	0.29 (0.10-0.87)	2.0
Avenell et al,[51] 2004	4	29	4	35	1.21 (0.33-4.41)	1.5
RECORD,[54] 2005	166	1311	179	1332	0.94 (0.77-1.15)	34.5
Prince et al,[56] 2006	110	730	126	730	0.87 (0.69-1.10)	28.3
Reid et al,[57] 2006	134	732	147	739	0.92 (0.75-1.14)	32.1
Total	421	3376	480	3411	0.88 (0.75-1.03)	100.0
Heterogeneity: $\tau^2 = 0.01$; $\chi^2_6 = 7.63$ ($P = .27$); $I^2 = 21\%$						
Test for overall effect: $z = 1.56$ ($P = .12$)						



Zhao JG

JAMA. 2017 Dec 26;318(24):2466-2482

Βιταμίνη D και κίνδυνος κατάγματος …

JAMA | Original Investigation

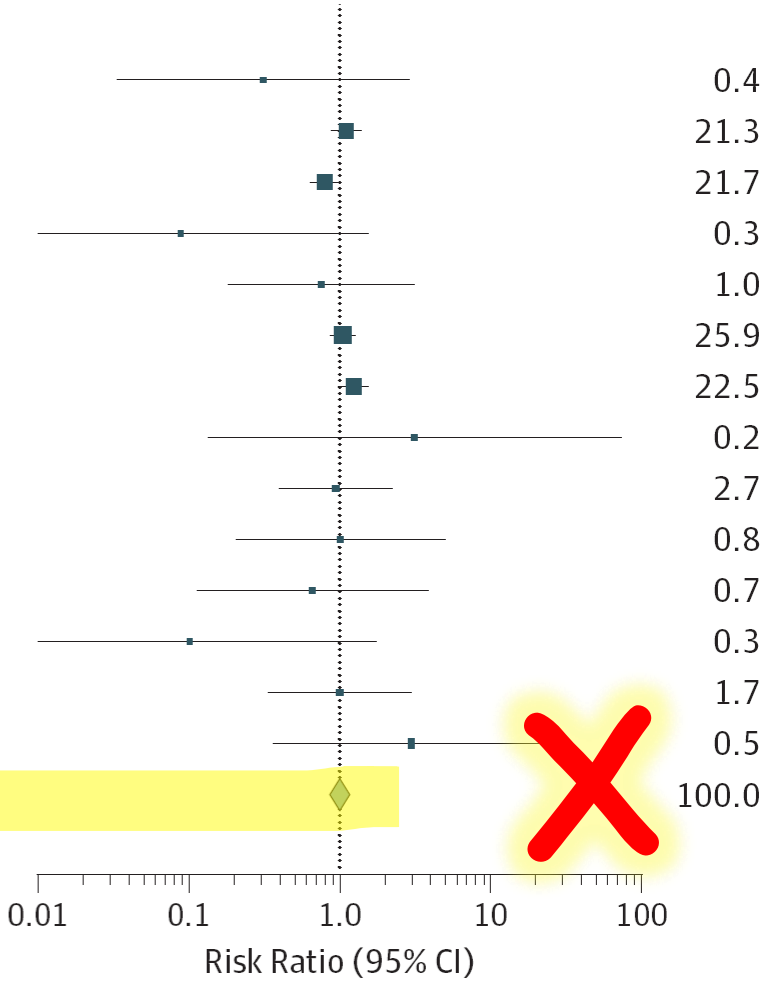
Association Between Calcium or Vitamin D Supplementation and Fracture Incidence in Community-Dwelling Older Adults

A Systematic Review and Meta-analysis

Total fracture						
Inkovaara et al,[40] 1983	1	45	3	42	0.31 (0.03-2.88)	0.4
Lips et al,[43] 1996	135	1291	122	1287	1.10 (0.87-1.39)	21.3
Trivedi et al,[50] 2003	119	1345	149	1341	0.80 (0.63-1.00)	21.7
NoNOF,[52] 2004	0	38	5	37	0.09 (0.01-1.55)	0.3
Avenell et al,[51] 2004	3	35	4	35	0.75 (0.18-3.11)	1.0
RECORD,[54] 2005	188	1343	179	1332	1.04 (0.86-1.26)	25.9
Vital D,[61] 2010	155	1131	125	1127	1.24 (0.99-1.54)	22.5
Mitri et al,[62] 2001	1	23	0	24	3.13 (0.13-73.01)	0.2
Glendenning et al,[63] 2012	10	353	10	333	0.94 (0.40-2.24)	2.7
TIDE,[32] 2012	3	607	3	614	1.01 (0.20-4.99)	0.8
VitDISH,[64] 2013	2	80	3	79	0.66 (0.11-3.83)	0.7
VitaDial,[34] 2014	0	26	5	29	0.10 (0.01-1.74)	0.3
DEX,[36] 2015	6	102	6	102	1.00 (0.33-3.00)	1.7
BEST-D,[37] 2017	6	204	1	101	2.97 (0.36-24.34)	0.5
Total	629	6623	615	6483	1.01 (0.87-1.17)	100.0

Heterogeneity: $\tau^2 = 0.01$; $\chi^2_{13} = 16.27$ ($P = .23$); $I^2 = 20\%$

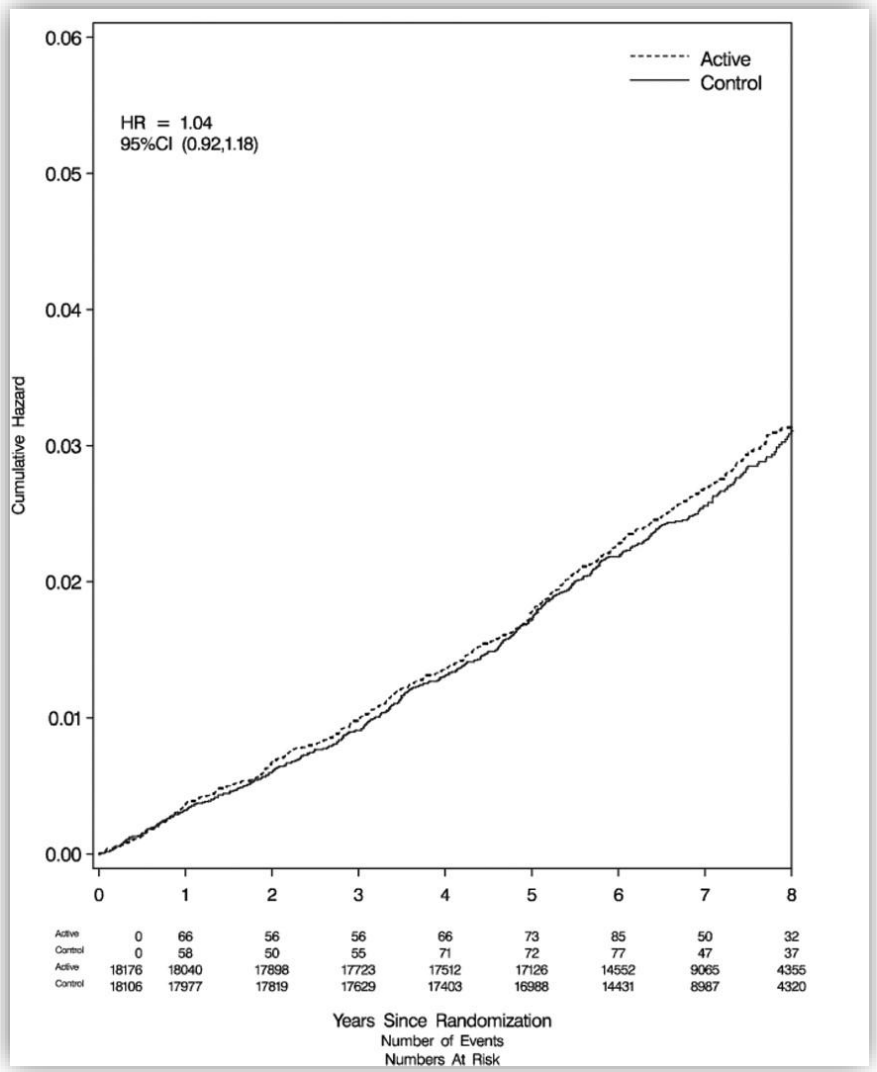
Test for overall effect: $z = 0.17$ ($P = .87$)



Zhao JG

JAMA. 2017 Dec 26;318(24):2466-2482

Ασβέστιο και βιταμίνη D και καρδιαγγειακός κίνδυνος ?...



- Τυχαιοποιημένη, πολυκεντρική μελέτη
- 36.282 μετεμμηνοπαυσιακές γυναίκες 50-79 ετών, 7 έτη παρακολούθησης
- Λήψη ανθρακικού ασβεστίου 500 mg με vitamin D 200 IU δυο φορές/ημερα vs placebo

Τα συμπληρώματα ασβεστίου/βιταμίνης D δεν αύξησαν, αλλά ούτε μείωσαν τα καρδιαγγειακά συμβάματα (ΟΕΜ/ΑΕΕ)

Circulation. 2007;115:846-854

ΧΝΝ και συμπληρώματα ασβεστίου

Για ασθενείς με **eGFR<30** προτείνεται συνολική ημερήσια πρόσληψη ασβεστίου 800-1000 mg και βιταμίνης D 800 IU

- *Η ημερήσια πρόσληψη ασβεστίου να μην υπερβαίνει τα 1500mg, λόγω του κινδύνου αγγειακής ασβεστοποίησης*
- ***Συμπληρώματα ασβεστίου μέχρι 500 mg ημερησίως και τα υπόλοιπα από διατροφή***

European Consensus Statement on the diagnosis and management of osteoporosis in chronic kidney disease stages G4-G5D.
Nephrol Dial Transplant. 2021;36(1):42-59

Recommended calcium intake in adults and children with chronic kidney disease – a European consensus statement

Focus of study was to establish optimal calcium intake in chronic kidney disease (in adults and children) which is not addressed in current clinical practice guidelines

Methods

Literature review by expert panel

Delphi survey

Revision based on survey response

Results

Key recommendations:

Adults

Total calcium intake (diet and medications): 800–1000 mg/day

Children

Total calcium intake: age-appropriate normal range

Evenepoel, P. *et al.*
NDT (2023)
@NDTSocial

This consensus statement provides key evidence and clinical practice points on calcium management that may assist in clinical decision-making in children and adults with CKD.

Δεδομένα και προτεινόμενη λήψη Vit D στη ΧΝΝ *για πρόληψη καταγμάτων*

The role of nutritional vitamin D in chronic kidney disease–mineral and bone disorder in children and adults with chronic kidney disease, on dialysis, and after kidney transplantation—a European consensus statement

3.2 Vitamin D supplementation and bone outcomes in CKD

Key evidence points

- There is no evidence for a benefit of vitamin D supplementation alone on the risk of bone loss and fractures in adults or children with CKD, or for improved growth in children with CKD.

- Vitamin D supplementation improves the control of hyperparathyroidism after kidney transplantation.
- Vitamin D supplementation may reduce the risk of bone loss and fractures after kidney transplantation.

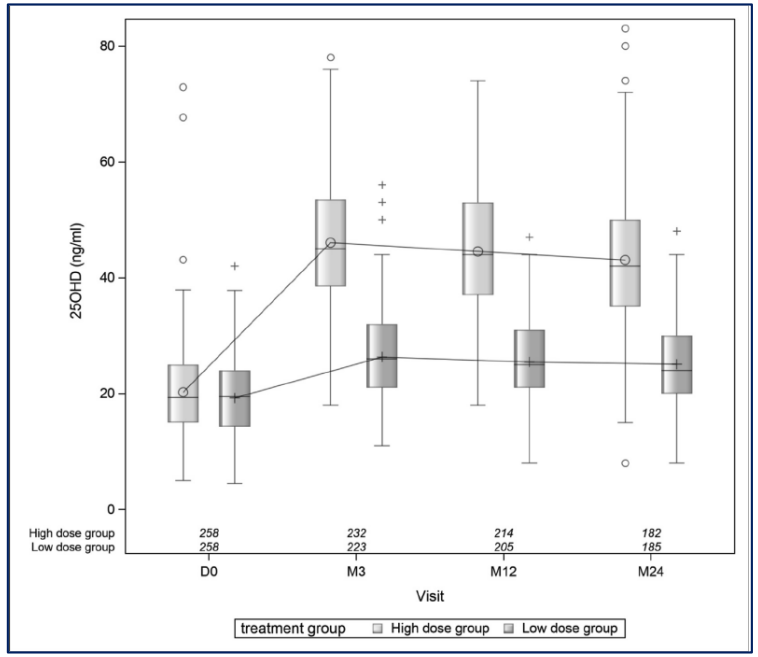
Jørgensen HS et al

Nephrol Dial Transplant. 2025 Apr 1;40(4):797-822

ΟΦΕΛΟΣ (ΚΑΤΑΓΜΑΤΑ) ΑΠΟ ΤΗ ΧΟΡΗΓΗΣΗ Vit D ΜΕΤΑ ΤΗ ΜΕΤΑΜΟΣΧΕΥΣΗ

Original Article

Nonskeletal and skeletal effects of high doses versus low doses of vitamin D_3 in renal transplant recipients: Results of the VITALE (VITamin D supplementation in renAL transplant recipients) study, a randomized clinical trial



Variable	Treatment group	HR (95% CI)	*P* value
Composite endpoint[a]	Low-dose group: 42 (16)	1.0	.78
	High-dose group: 40 (15)	0.94 (0.60-1.48)	
Major cardiovascular events	Low-dose group: 14 (5)	1.0	.47
	High-dose group: 11 (4)	0.75 (0.34-1.65)	
De novo diabetes mellitus	Low-dose group: 11 (4)	1.0	.66
	High-dose group: 9 (3)	0.82 (0.34-1.98)	
De novo cancer	Low-dose group: 16 (6)	1.0	.31
	High-dose	1.34	
Symptomatic fracture[b]	Low-dose group: 12 (4)	1.0	.03
	High-dose group: 3 (1)	0.24 (0.07-0.86)	
Infection (all infections)	Low-dose group: 126 (47)	1.0	.49
	High-dose group: 135 (51)	1.13 (0.80-1.58)	
Infection (CMV)	Low-dose group: 7 (3)	1.0	.47
	High-dose group: 10 (4)	1.43 (0.54-3.83)	
Infection (other than CMV)	Low-dose group: 126 (47)	1.0	.60
	High-dose group: 133 (49)	1.09 (0.78-1.54)	
Acute rejection	Low-dose group: 6 (2)	1.0	.60
	High-dose group: 8 (3)	1.33 (0.46-3.90)	
Dialysis or loss of transplant	Low-dose group: 1 (0.3)	NA	NA
	High-dose group: 1 (0.3)		
Symptomatic fracture[b]	Low-dose group: 12 (4)	1.0	.03
	High-dose group: 3 (1)	0.24 (0.07-0.86)	

Nephrol Dial Transplant, 2025, **40**, 797–822
https://doi.org/10.1093/ndt/gfae293
Advance access publication date: 28 January 2025

The role of nutritional vitamin D in chronic kidney disease–mineral and bone disorder in children and adults with chronic kidney disease, on dialysis, and after kidney transplantation—a European consensus statement

Hanne Skou Jørgensen[1,2], Marc Vervloet[3], Etienne Cavalier[4], Justine Bacchetta[5], Martin H. de Borst[6], Jordi Bover[7], Mario Cozzolino[8], Ana Carina Ferreira[9], Ditte Hansen[10,11], Markus Herrmann[12], Renate de Jongh[13], Sandro Mazzaferro[14], Mandy Wan[15], Rukshana Shroff[16] and Pieter Evenepoel[17]; on behalf of the European Renal Osteodystrophy (EUROD) initiative under the Chronic Kidney Disease–Mineral and Bone Disorder working group of the European Renal Association (ERA) and the Dialysis and Chronic Kidney Disease–Mineral and Bone disorder working groups of the European Society of Paediatric Nephrology (ESPN)

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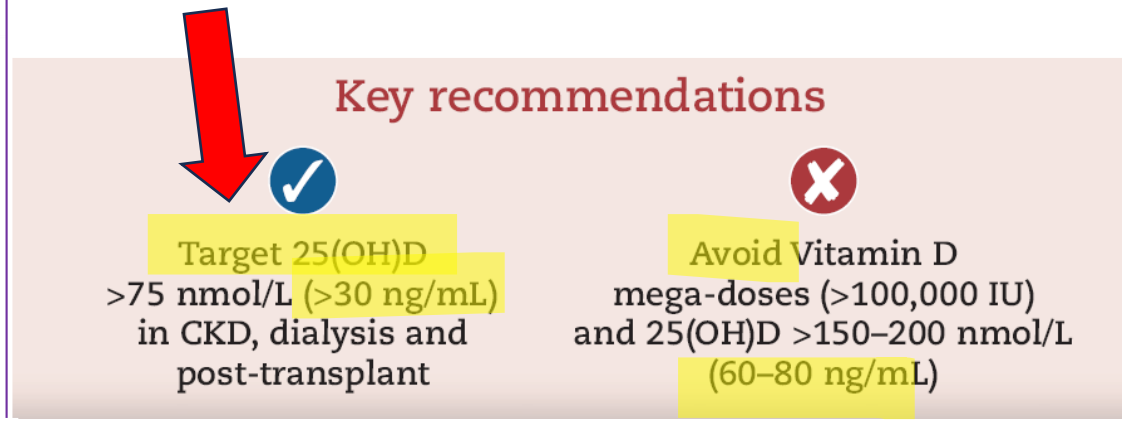
Δεδομένα και προτεινόμενη λήψη Vit D στη ΧΝΝ *για έλεγχο του ΔΥΠΘ*

3.1 Vitamin D supplementation and PTH control in CKD

Clinical practice points

- We recommend supplementing vitamin D to >75 nmol/l (>30 ng/ml) in adults and children with CKD G2–5D to delay the onset, or improve the control, of secondary hyperparathyroidism, recognizing that the effect in CKD G5–5D is uncertain.



Jørgensen HS et al

Nephrol Dial Transplant. 2025 Apr 1;40(4):797-822

Αποφυγή συστηματικής χορήγησης ενεργού βιταμίνης D για έλεγχο ΔΥΠΘ πριν την ένταξη σε εξωνεφρική κάθαρση

Chapter 4.2: Treatment of abnormal PTH levels in CKD-MBD

4.2.1: In patients with CKD G3a–G5 not on dialysis, the optimal PTH level is not known. However, we suggest that patients with levels of intact PTH progressively rising or persistently above the upper normal limit for the assay be evaluated for modifiable factors, including hyperphosphatemia, hypocalcemia, high phosphate intake, and vitamin D deficiency (*2C*).

4.2.2 In adult patients with CKD G3a–G5 not on dialysis, we suggest that calcitriol and vitamin D analogs not be routinely used (*2C*). It is reasonable to reserve the use of calcitriol and vitamin D analogs for patients with CKD G4–G5 with severe and progressive hyperparathyroidism (*Not Graded*).

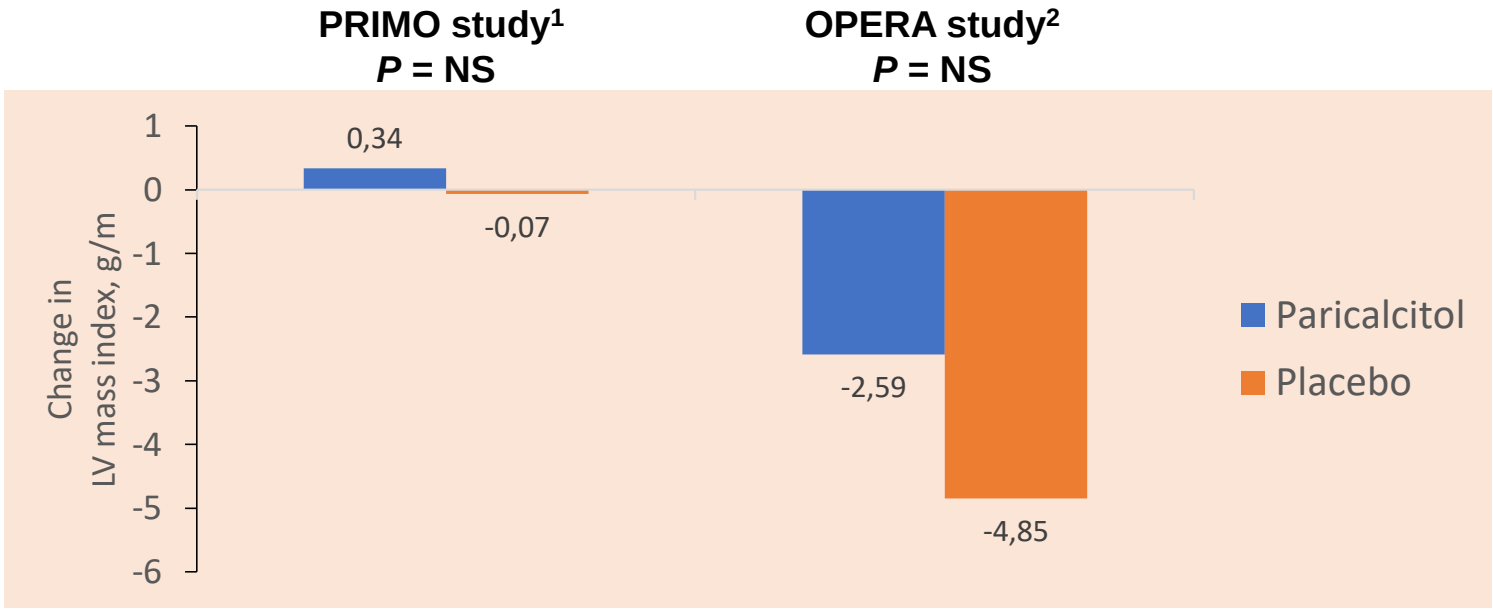
In children, calcitriol and vitamin D analogs may be considered to maintain serum calcium levels in the age-appropriate normal range (*Not Graded*).

KDIGO 2017 Clinical Practice Guideline Update for the Diagnosis, Evaluation, Prevention, and Treatment of Chronic Kidney Disease–Mineral and Bone Disorder (CKD-MBD)

Kidney Int Suppl. 2017;7:1-59

Αποφυγή συστηματικής χορήγησης ενεργού βιταμίνης D για έλεγχο ΔΥΠΘ πριν την ένταξη σε εξωνεφρική κάθαρση

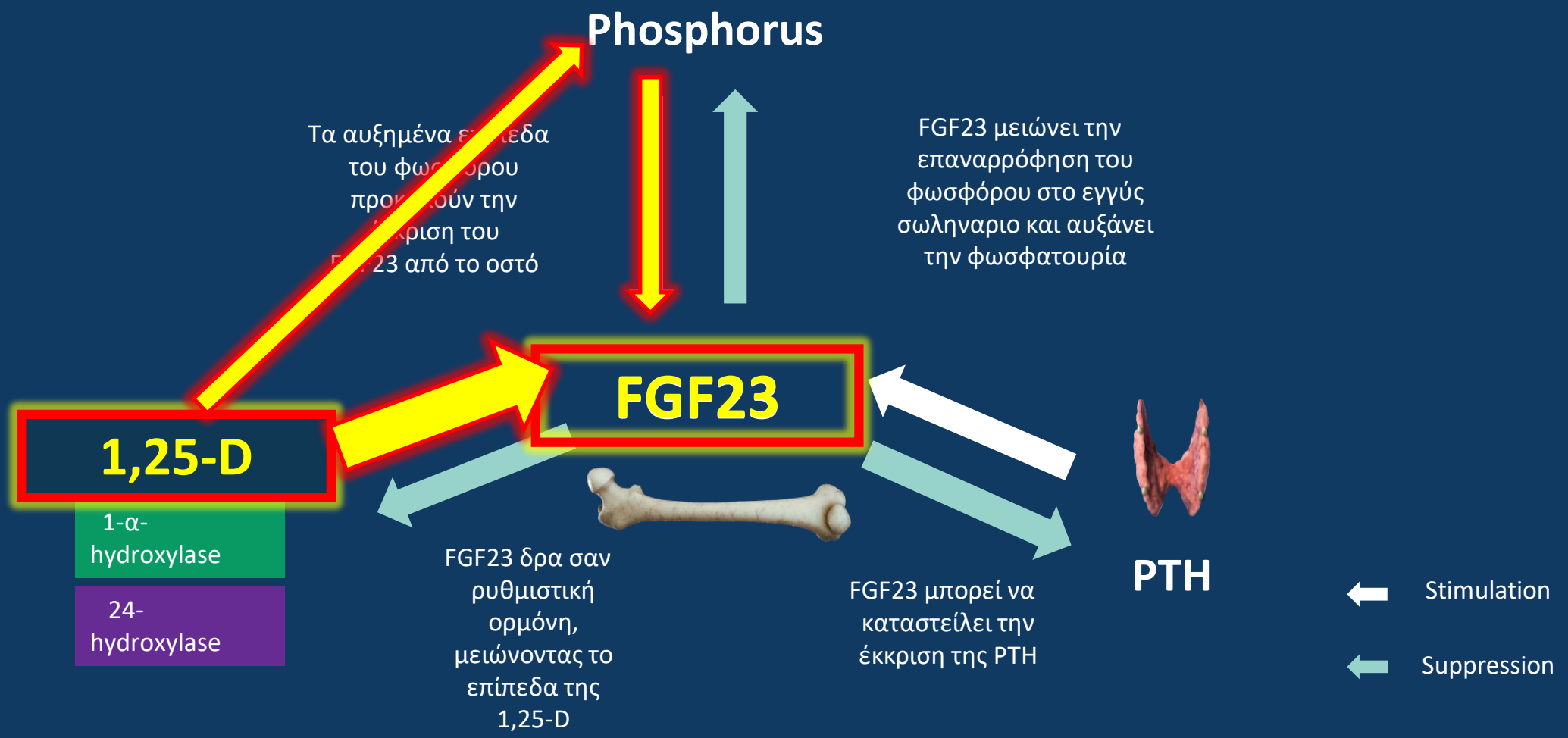
Left Ventricular Mass Index Change From Baseline to End of Study



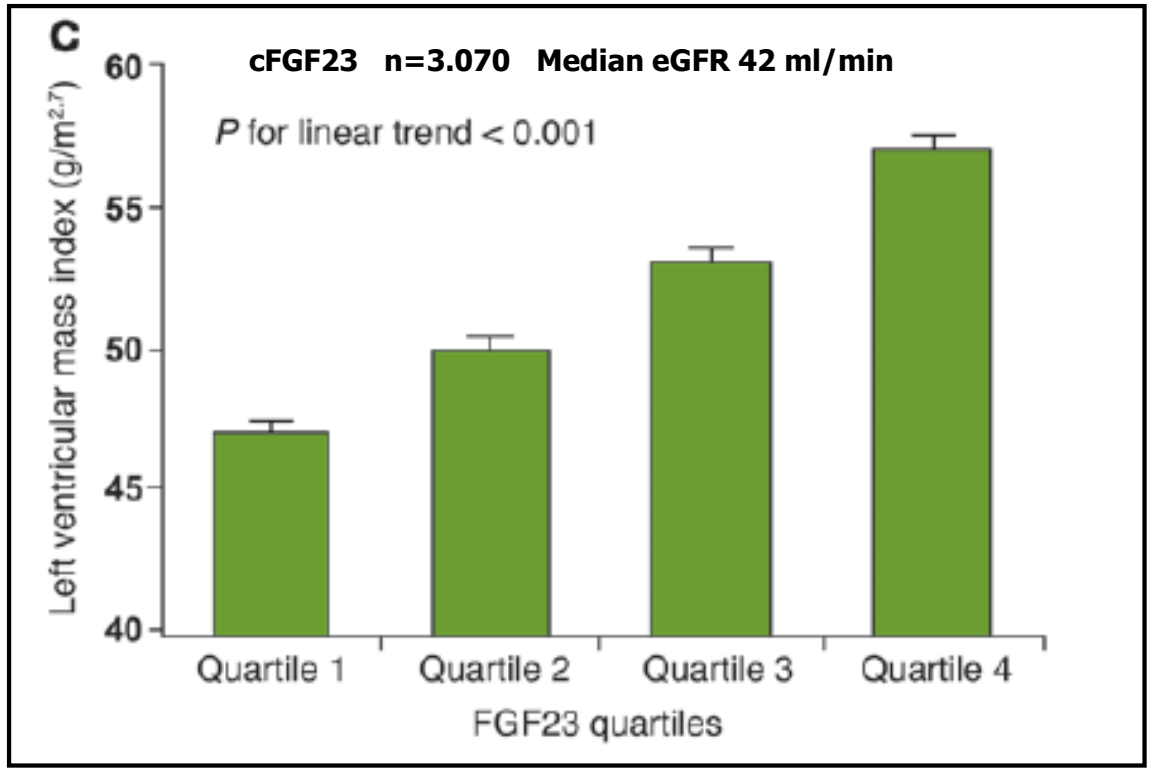
Thadhani R, et al.
Wang AY-M, et al.

JAMA. 2012;307:674-684.
J Am Soc Nephrol. 25:175-186

Η αλληλεπίδραση των FGF23, φωσφόρου, PTH και βιταμίνης D (1,25-D) στην ΧΝΝ



FGF23 και νεοεμφανιζόμενη LVH σε νορμοτασικούς ασθενείς με ΧΝΝ



Elevated FGF23 is associated with increased risk of <u>new-onset LVH</u>

411 pts normal LV geometry at baseline,
84 (20%) new onset LVH (2.9±0.5 years later)

Elevated FGF23 levels at baseline were associated with increased future risk of new onset LVH both in normotensive subjects

Faul C et al.

J Clin Invest 121: 4393, 2011

Αντιοστεοπορωτική θεραπεία στην οστεοπόρωση της ΧΝΝ:

Ξεκινήστε από τις διαταραχές της CKD-MBD

A consensus statement on the management of vertebral fractures in CKD stages G4–G5D

→ Before initiating antiresorptive or anabolic therapy for CKD-associated osteoporosis, it is good clinical practice to first

→ correct CKD-associated uraemic and mineral metabolism

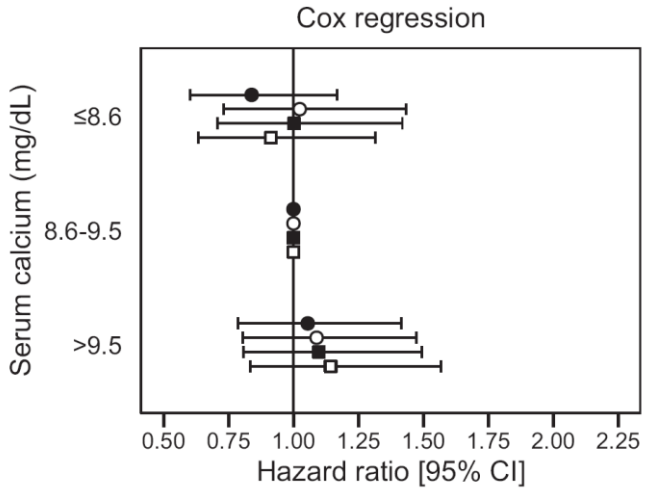
Consensus statement on the management of vertebral fractures in CKD stages G4-G5D

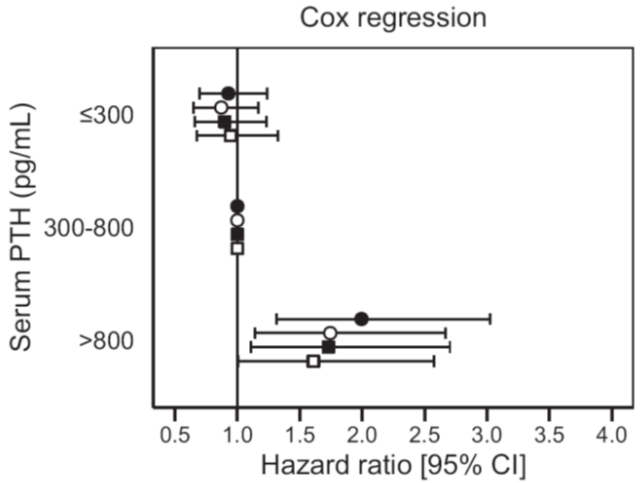
Nephrol Dial Transplant. 2026 Feb 27;41(3):584-598

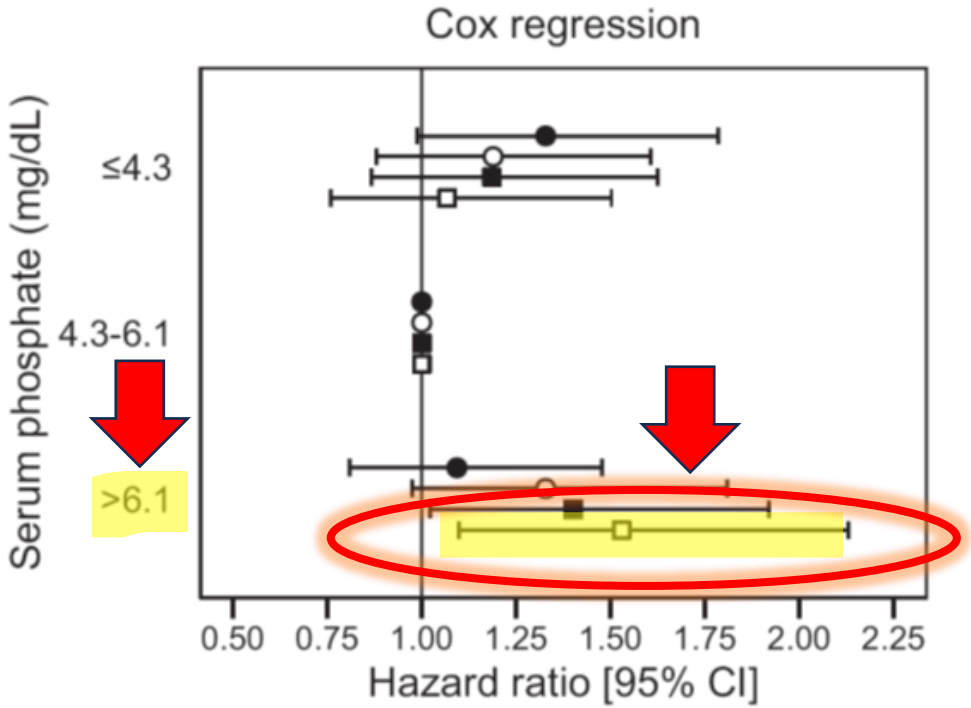
Αντιοστεοπορωτική θεραπεία στην οστεοπόρωση της ΧΝΝ:

Ξεκινήστε από τις διαταραχές της CKD-MBD

Serum phosphate is associated with increased risk of bone fragility fractures in haemodialysis patients







Multivariate adjustments; Model 1: age, sex, body mass index, aetiology of CKD, time on haemodialysis, smoking habit, diabetes, cardiovascular disease history, bone fracture history in the previous 12 months, vascular or valvular calcification, and parathyroidectomy. Model 2: Model 1 plus dialysis type, calcium concentration in the dialysate, hours of haemodialysis per week, treatment with erythropoietin-stimulating agents (ESAs), prescription of vitamin D metabolites/analogues, native vitamin D or calcidiol, phosphate-binding agents. ***Model 3****: Model 2* ***plus*** *haemoglobin, albumin,* ***PTH and calcium***

Barrera-Baena P et al

Nephrol Dial Transplant. 2024 Mar 27;39(4):618-626

Αντιοστεοπορωτική θεραπεία στην οστεοπόρωση της ΧΝΝ:

Calcimimetics και βελτίωση της οστικής πυκνότητας σε ΔΥΠΘ

Changes in Bone Quality after Treatment with Etelcalcetide

Biochemicals	Baseline Visit	36-wk Visit	Change (%)	*P* Value
Whole PTH 1–84, pg/ml,[a]	692 (561–732)	156 (82–239)	−67±9	<0.001
Calcium, mg/dl	9.3 (9.0–9.6)	9.3 (8.4–10.1)	−2±3	0.5
Phosphate, mg/dl	6.2 (5.2–6.8)	5.9 (4.8–6.8)	−1±11	0.7
Bone-specific alkaline phosphatase, U/L,[b]	59.9 (40.0–71.3)	26.0 (20.5–36.7)	−45±4	<0.001
C-telopeptide, ng/ml,[b]	6.7 (3.8–10.6)	2.2 (0.8–5.4)	−63±6	<0.001
FGF23 C-terminal, RU/mL,[c]	9148 (6678–13,176)	8856 (1694–19,465)	−15±13	0.2
FGF23 intact, pg/ml,[c]	10,868 (5544–13,834)	8569 (1755–22,530)	−18±13	0.2
Sclerostin, ng/ml,[b]	1.98 (1.11–2.46)	2.18 (1.38–3.30)	34±11	0.01
Areal bone mineral density (mg HA/cm^2)			**mean±SEM**	
Lumbar spine	0.99±0.17	1.01±0.17	3±1	0.04
Femoral neck	0.72±0.13	0.77±0.15	7±2	0.002
Total hip	0.83±0.14	0.85±0.15	3±1	0.04

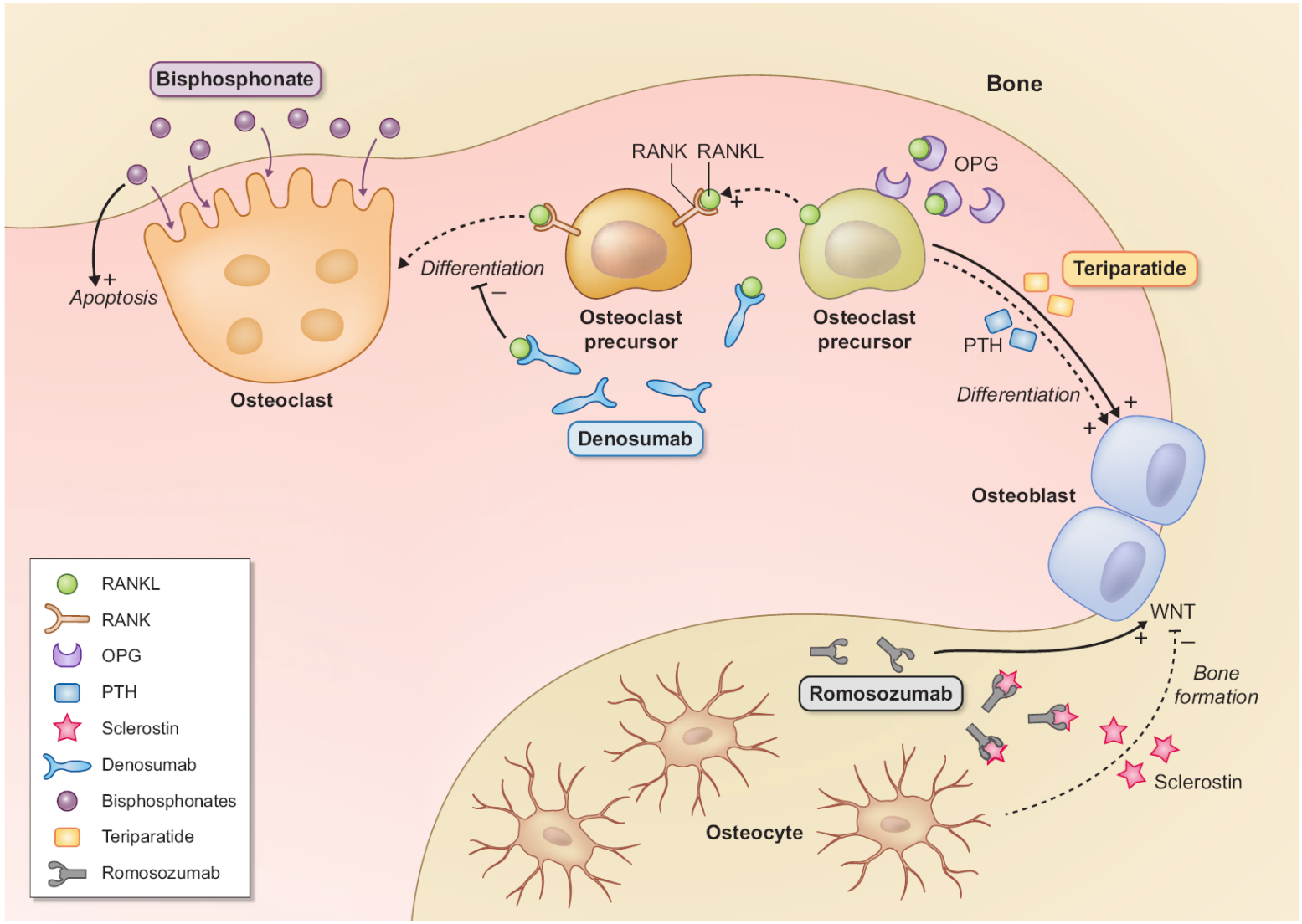
Khairallah P

Clin J Am Soc Nephrol. 2023 Nov 1;18(11):1456-1465

Αντιοστεοπορωτικά φαρμάκα και ΧΝΝ

	Advantages	Disadvantages
Calcium	Can reduce risk of sHPT and skeletal mineralization defects Lowers phosphorus load May improve BMD in combination with vitamin D	Excessive use may increase cardiovascular risk and risk for kidney stones
Vitamin D	Can reduce risk of sHPT and skeletal mineralization defects May improve BMD in combination with calcium Active vitamin D preferrable in CKD 5D Nutritional vitamin D preferable in CKD 4–5	Stimulates FGF23 Excessive use may increase cardiovascular risk Active vitamin D should be restricted to patients with SHPT and can in that case also be used as adjuvant therapy with bone-specific agents
Bisphosphonates	Can improve BMD in all stages of CKD Persistent effect after cessation No evidence for increased cardiovascular risk	Increased systemic retention May be associated with CKD progression in CKD 4–5 and reduced residual renal function in CKD 5D Occasional reports of AKI with intravenous use
Denosumab	Can improve BMD in all stages of CKD No evidence of increased cardiovascular or renal risk No dose adaptation needed in any stages of CKD, including CKD 5D	Risk for hypocalcemia (especially in severe HPT) Rapid BMD deterioration and increased fracture risk after cessation
PTH analogues	Can improve BMD in all stages of CKD May improve suppressed BFR	Safety uncertain Optimal dosing uncertain May aggravate existing hyperparathyroidism
Romosozumab	May improve BMD in all stages of CKD Anabolic and antiresorptive effect	May induce hypocalcaemia Cardiovascular safety uncertain Optimal dosing uncertain
HRT	Can improve BMD in all stages of CKD	Safety uncertain Limited to early menopause

ΦΑΡΜΑΚΑ ΚΑΙ ΠΑΘΟΦΥΣΙΟΛΟΓΙΑ ΤΗΣ ΟΣΤΕΟΠΟΡΩΣΗΣ



Henry N, Hildebrand S, Cunningham J.

Clin Kidney J. 2025 Jul 16;18(10):sfaf228

Αντιοστεοπορωτικά φαρμάκα και ΧΝΝ σταδίων 1-3

A consensus statement on the management of vertebral fractures in CKD stages G4–G5D

Antiresorptive (bisphosphonates and denosumab), anabolic (teriparatide and abaloparatide) and mixed agents (romosozumab) can be utilized for osteoporosis therapy in CKD G1–3, as in the general population.

ΣΤΑΔΙΟ 1-3 : ΟΠΩΣ ΣΤΟ ΓΕΝΙΚΟ ΠΛΗΘΥΣΜΟ !!

Consensus statement on the management of vertebral fractures in CKD stages G4-G5D

Nephrol Dial Transplant. 2026 Feb 27;41(3):584-598

Αντιοστεοπορωτικά φαρμάκα και ΧΝΝ σταδίων 4-5D

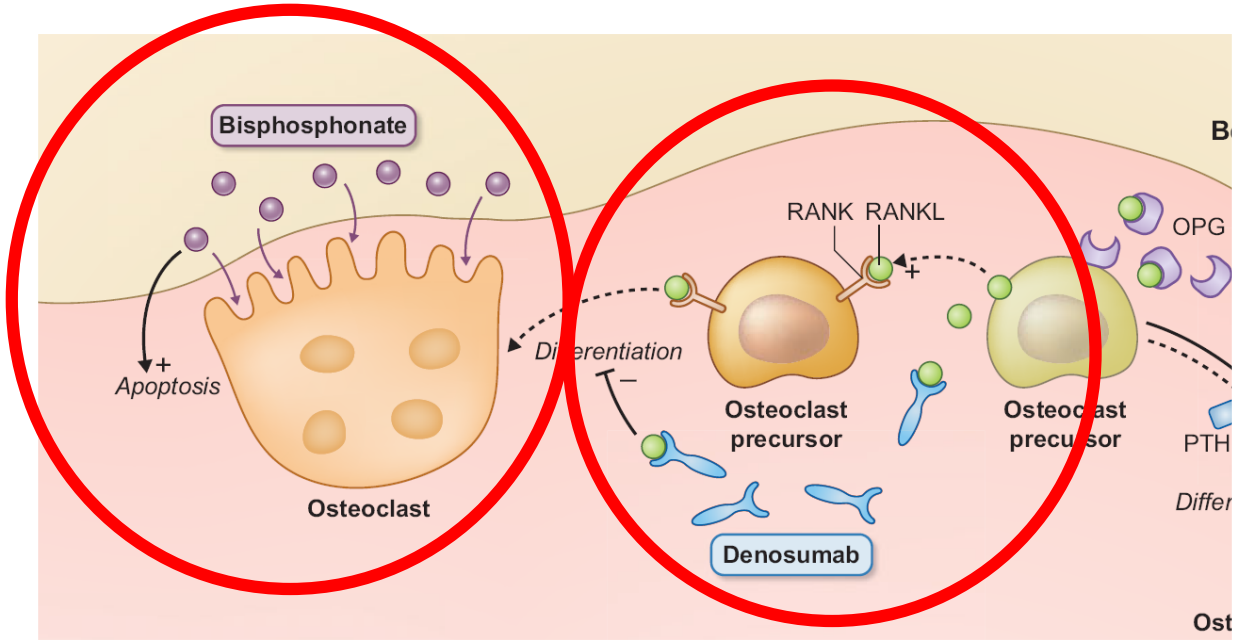
A consensus statement on the management of vertebral fractures in CKD stages G4–G5D

There is currently no available data on the effects of standard osteoporosis therapies on VFs in advanced CKD G4–5D.

Consensus statement on the management of vertebral fractures in CKD stages G4-G5D

Nephrol Dial Transplant. 2026 Feb 27;41(3):584-598

ΦΑΡΜΑΚΑ ΠΟΥ ΑΝΑΣΤΕΛΛΟΥΝ ΤΗΝ ΟΣΤΙΚΗ ΑΠΟΡΡΟΦΗΣΗ (ΚΥΡΙΩΣ ΤΟΥΣ ΟΣΤΕΟΚΛΑΣΤΕΣ)



Henry N, Hildebrand S, Cunningham J.

Clin Kidney J. 2025 Jul 16;18(10):sfaf228

Αναστολείς της οστικής απορρόφησης στη ΧΝΝ

Table 2. Osteoporosis Drugs in Advanced CKD

Drug	Trials in eGFR < 45 (No. of Patients)	Trials in Dialysis Patients (No. of Patients)	Suggested Dosing Options	Side Effects	Efficacy
Antiresorptive					
Bisphosphonates	Yes (59-581)	Yes (ongoing)	(1) Oral alendronate 35 mg, weekly (2) IV pamidronate 60 mg, every other month	Hypocalcemia, AKI, eGFR decline, osteonecrosis	Benefits seen in CKD3; data lacking in CKD4 and dialysis
RANK ligand inhibitor	Yes (55)	Yes (8-12)	SC denosumab, 60 mg, ×1	Hypocalcemia (severe), rebound osteoclast activity	Efficacy seen in dialysis in observational studies

Ginsberg C, Ix JH

Am J Kidney Dis. 2022 Mar;79(3):427-43

Δεδομένα για τη χορήγηση Διφωσφονικών σε αιμοκάθαρση

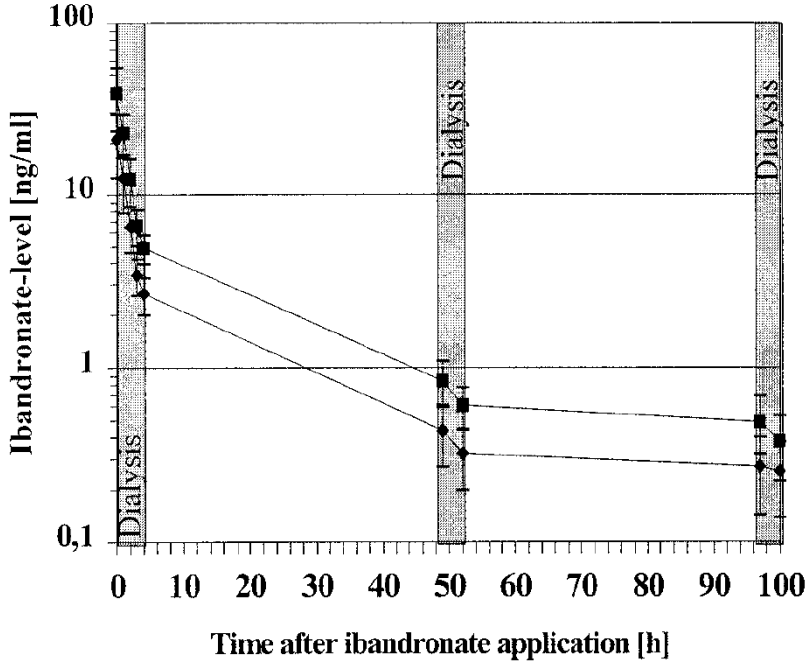
Nephrol Dial Transplant (2002) 17: 1281–1285

Nephrology Dialysis Transplantation

Original Article

Elimination of intravenously administered ibandronate in patients on haemodialysis: a monocentre open study

Raoul Bergner[1], Katja Dill[1], Dietmar Boerner[2] and Michael Uppenkamp[1]



ibandronate application every 1–3 months is sufficient for therapy in osteoporosis and metastatic bone disease. Normal doses of ibandronate administered every 1–3 months will be removed effectively by haemodialysis, therefore no accumulation will occur in patients who undergo haemodialysis three times weekly. Further studies are needed to demonstrate the effects on renal bone disease.

Αναστολείς της οστικής απορρόφησης στη ΧΝΝ

Table 2 | Bisphosphonates vs. denosumab in advanced CKD (pros and cons)

	Bisphosphonates	Denosumab
Pros	Improves BMD in all CKD stages	Improves BMD in all CKD stages
	Oral or i.v. dose (can be administered during dialysis)	Subcutaneous dosing every 6 mo
	Low risk of severe hypocalcemia	Continued effectiveness for at least 10 yr (in patients without CKD)
	Can be stopped after limited treatment time	
Cons	Risk of kidney damage in CKD 4–5	Risk of severe hypocalcemia
	Wear out after several years	Risk of fractures if stopped
	Osteonecrosis of the jaw	Osteonecrosis of the jaw
	Atypical femoral fractures	Atypical femoral fractures
	Acute phase reaction (i.v. bisphosphonates only)	Risk of infections
	Esophagitis	
	Uveitis	
	Atrial fibrillation	

Haarhaus et al

Kidney Int. 2021 Sep;100(3):546-558

ΑΣΦΑΛΕΙΑ ΔΙΦΩΣΦΟΝΙΚΩΝ ΣΕ ΧΝΝ 3-4/5

Safety of Oral Bisphosphonates in Moderate-to-Severe Chronic Kidney Disease: A Binational Cohort Analysis

GFR <45 ml/min που έλαβαν διφωσφονικά VS Propensity Score matched OXI

Table 2. Numbers of Events, Incidence Rates, and Hazard Ratios per 1000 person-years for All Analyses

		CPRD		SIDIAP		
		BP	Non-BP	BP	Non-BP	Combined
Chronic kidney disease progression	Unmatched no. events	614	15,411	471	13,462	
	Unmatched incidence rates	90.8 (83.9, 98.3)	73.3 (72.1, 74.4)	119.0 (108.5,130.2)	104.7 (102.9,106.5)	
	Unadjusted HR	**1.25 (1.15, 1.36)**		**1.13 (1.03, 1.23)**		**1.19 (1.12, 1.27)**
	Fully adjusted HR	**1.18 (1.08, 1.29)**		**1.19 (1.08, 1.31)**		**1.18 (1.11, 1.26)**
	PS-matched no. events	576	1996	467	2015	
	PS-matched incidence rates	89.1 (82.1, 96.7)	85.6 (82.0, 89.5)	118.4 (107.9,129.6)	100.0 (95.7,104.5)	
	PS-matched sub-HR	**1.14 (1.04, 1.26)**		**1.15 (1.04, 1.27)**		**1.14 (1.07, 1.23)**

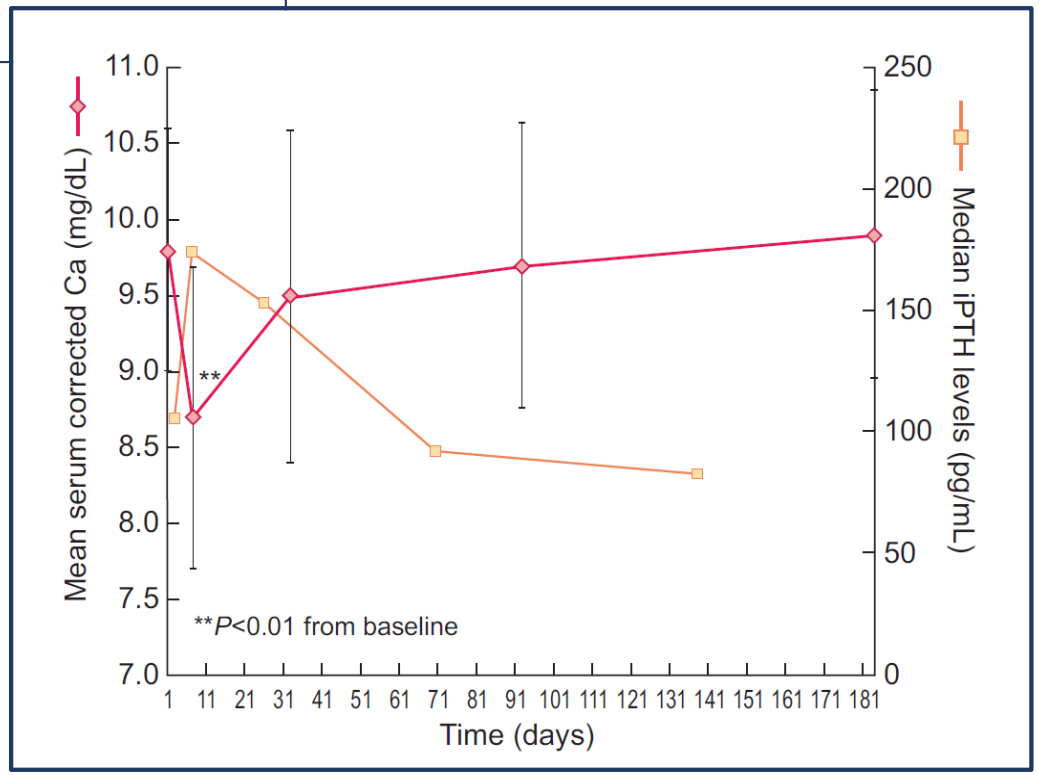
~15 % κίνδυνο για επιδείνωση ΧΝΝ/ένταξη σε κάθαρση

Robinson DE et al

J Bone Miner Res. 2021 May;36(5):820-832

Υπασβεστιαιμία και Ασφάλεια DENOSUMAB σε ΧΝΝ 5D/HD

Hypocalcemia and bone mineral changes in hemodialysis patients with low bone mass treated with denosumab: a 2-year observational study



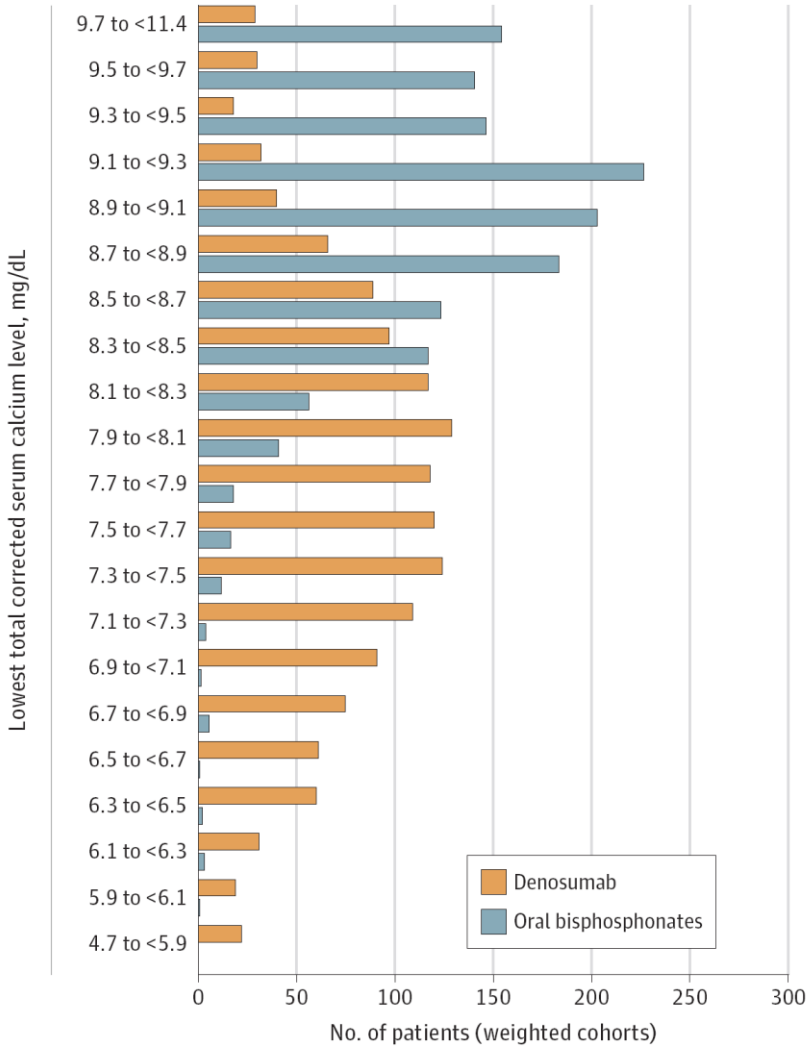
- **Η συχνότητα άμεσης σημαντικής υπασβεσταιμίας ~25% των ασθενών**
- **Ναδίρ ~ 7 ημέρες μετά τη χορήγηση**

Broadwell A

J Clin Endocrinol Metab. 2021 Jan 23;106(2):397-409

Denosumab vs διφωσφονικά: υπασβεστιαιμία σε ηλικιωμένες γυναίκες υπό αιμοκάθαρση

Figure 3. Lowest Total Albumin-Corrected Serum Calcium Level



Medicare >3000 patients
aged 65 years or older

- Severe hypocalcemia <7.5 mg/dL
- Very Severe <6.5 mg/dL

Table 2. Outcomes for Severe and Very Severe Hypocalcemia in the Weighted Cohorts[a]

Outcomes	No. of patients	Incidence of study outcomes, No.	Weighted cumulative incidence, % (95% CI)
Primary: severe hypocalcemia			
Denosumab	1523	607	41.1 (38.5-43.6)
Oral bisphosphonates	1501	29	2.0 (1.0-3.0)
Secondary: very severe hypocalcemia			
Denosumab	1523	161	10.9 (9.3-12.4)
Oral bisphosphonates	1501	6	0.4 (0.0-0.9)

Bird ST et al

JAMA. 2024 Feb 13;331(6):491-499

Denosumab vs διφωσφονικά: RCT σε ασθενείς υπό αιμοκάθαρση

Effects of Denosumab and Alendronate on Bone Health and Vascular Function in Hemodialysis Patients: A Randomized, Controlled Trial

- prior fracture
- BMD 70% or T score < –2.5

- HD > 6 months
- 60 pg/mL < i-PTH < 240 pg/mL

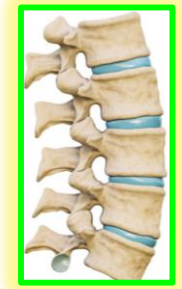
	Total ($n = 46$)	Denosumab ($n = 22$)	Alendronate ($n = 24$)	p Value
Age (years)	71.4 ± 9.8	71.3 ± 10.5	71.5 ± 9.3	0.9505
Body mass index (kg/m^2)	20.5 (18.8–23.4)	19.9 (19.1–22.5)	20.7 (17.9–24.6)	0.7498
Female/male	18/28	9/13	9/15	1.000
Duration of hemodialysis (years)	7.1 (2.9–11.7)	9.0 (2.4–13.2)	6.1 (3.7–11.2)	0.9474
Previous fracture, *n* (%)	17 (37.0%)	10 (45.5%)	7 (29.2%)	0.3608
History of diabetes, *n* (%)	20 (43.5%)	9 (40.9%)	11 (45.8%)	0.7666
Drug therapy, *n* (%)				
Active vitamin D preparations use	39 (84.7%)	20 (90.9%)	19 (79.2%)	0.4177
Alfacalcidol (po)	28 (60.9%)	13 (59.1%)	15 (62.5%)	
Calcitriol (iv)	6 (13.0%)	5 (22.7%)	1 (4.2%)	
Maxacalcitriol (iv)	5 (10.9%)	2 (9.1%)	3 (12.5%)	
Cinacalcet	23 (50.0%)	9 (40.9%)	14 (58.3%)	0.3762

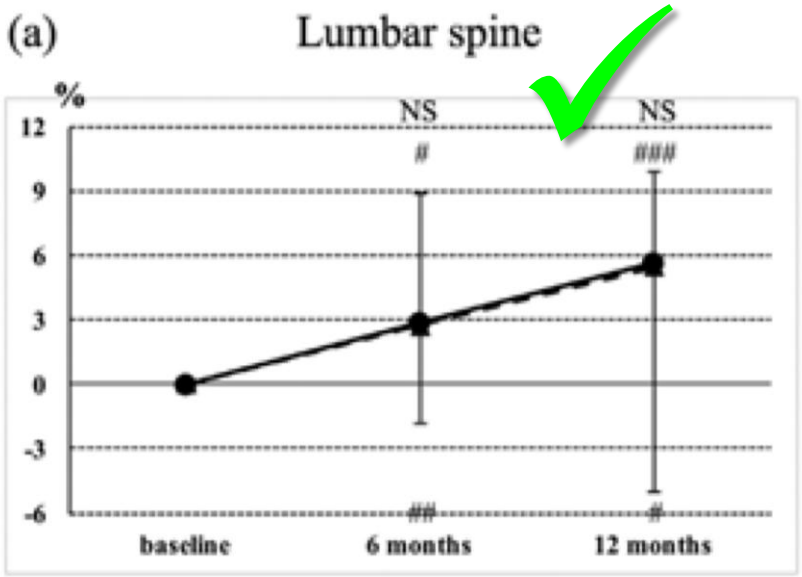
Iseri K

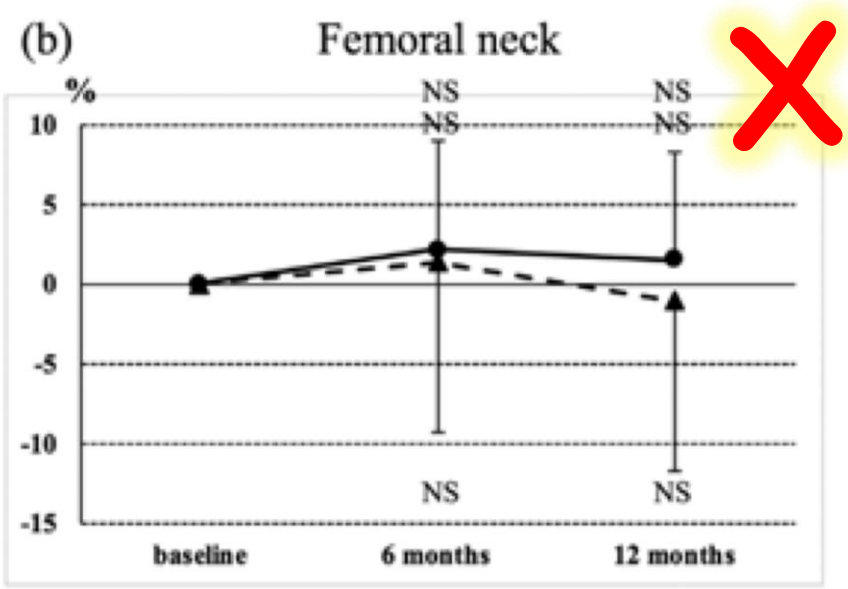
J Bone Miner Res. 2019 Jun;34(6):1014-1024

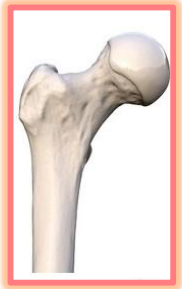
Denosumab vs διφωσφονικά RCT σε ασθενείς υπό αιμοκάθαρση

Βελτίωση BMD μόνο στην ΟΜΣΣ







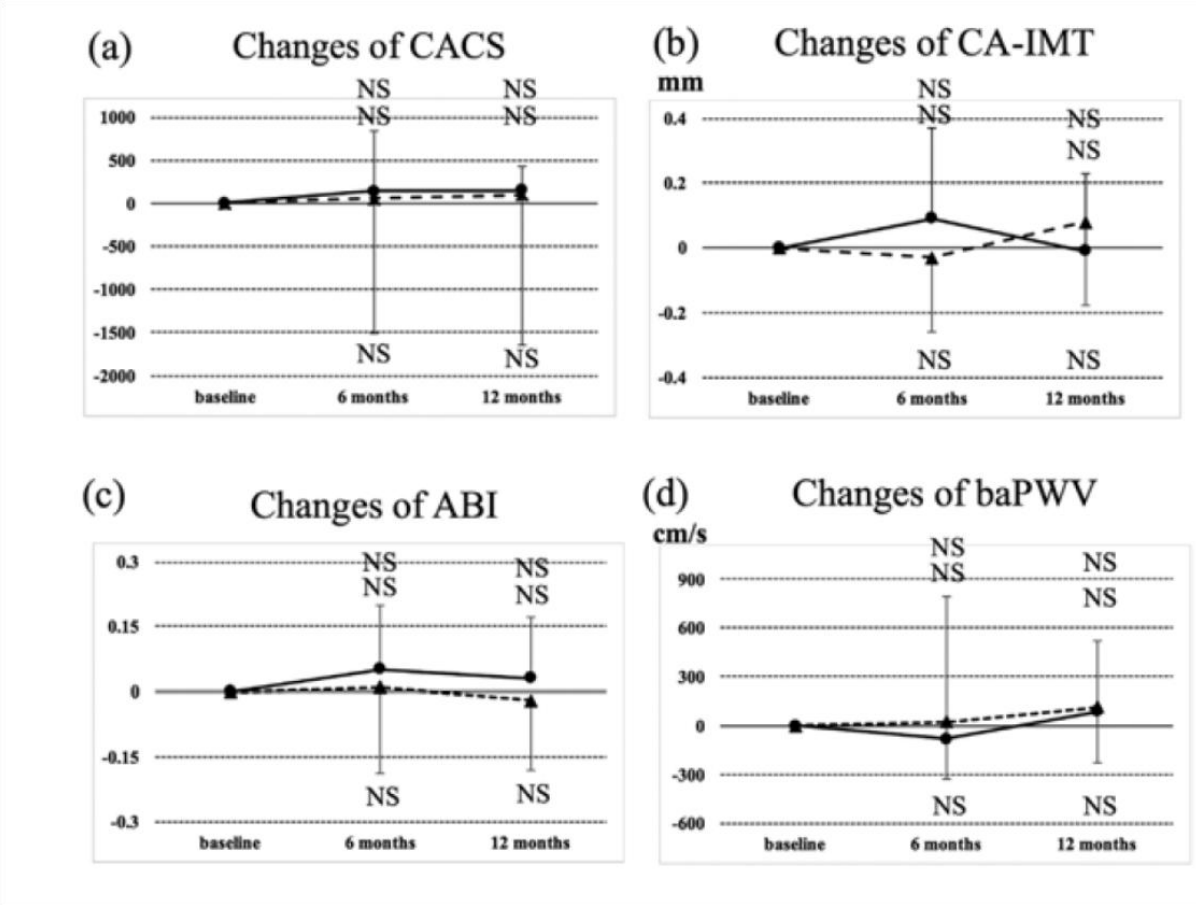


Iseri K

J Bone Miner Res. 2019 Jun;34(6):1014-1024

Denosumab vs διφωσφονικά RCT σε ασθενείς υπό αιμοκάθαρση

Ασφαλή και χωρίς διαφορά στις αγγειακές επασβεστώσεις



Iseri K

J Bone Miner Res. 2019 Jun;34(6):1014-1024

Denosumab *και Κατάγματα* σε ασθενείς υπό αιμοκάθαρση με οστεοπόρωση

512 patients / 52 denosumab
retrospective cohort study

Table 2. Risk of fracture with competing risk in denosumab users versus non-users.

	Events [n (%)]		Hazard Ratio (95%CI)	P Value
Non-denosumab	60 (37.0)		reference	
Denosumab	21 (40.4)	Unadjusted	0.53 (0.24–1.14)	0.104
		Adjusted	0.64 (0.27–1.51)	0.310

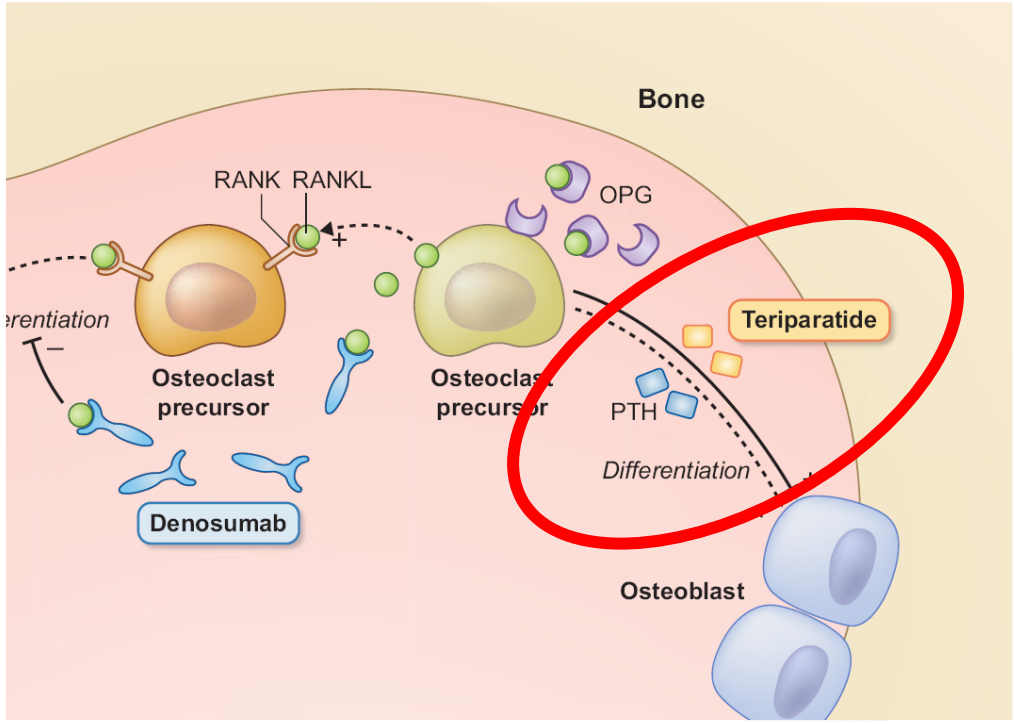
adjusted: Age, sex, history of bone fractures, bone mineral density, intact parathyroid hormone added to the unadjusted model

Kobayashi A et al

PLoS One. 2024 Aug 29;19(8):e0309657

Να αυξήσουμε την παραγωγή νέου οστού ??

Παραθορμόνη ?? !!



Θεραπεία με ανάλογα της PTH σε ασθενείς με ΧΝΝ G4–G5D

Table 2. Osteoporosis Drugs in Advanced CKD

Drug	Trials in eGFR < 45 (No. of Patients)	Trials in Dialysis Patients (No. of Patients)	Suggested Dosing Options	Side Effects	Efficacy
Anabolic					
PTH analogues	Yes (168-736)	Yes (7)	(1) SC teriparatide, 20 μg, daily(2) SC abaloparatide, 80 μg, daily	Hypercalcemia, nausea, URI	Efficacy in CKD, unclear in dialysis

PTH analogues are generally not recommended in

- patients with open epiphyses (i.e. children)
- cancer patients at risk of bone metastases
- < 2 χρόνια αγωγής ?!

Ginsberg C, Ix JH

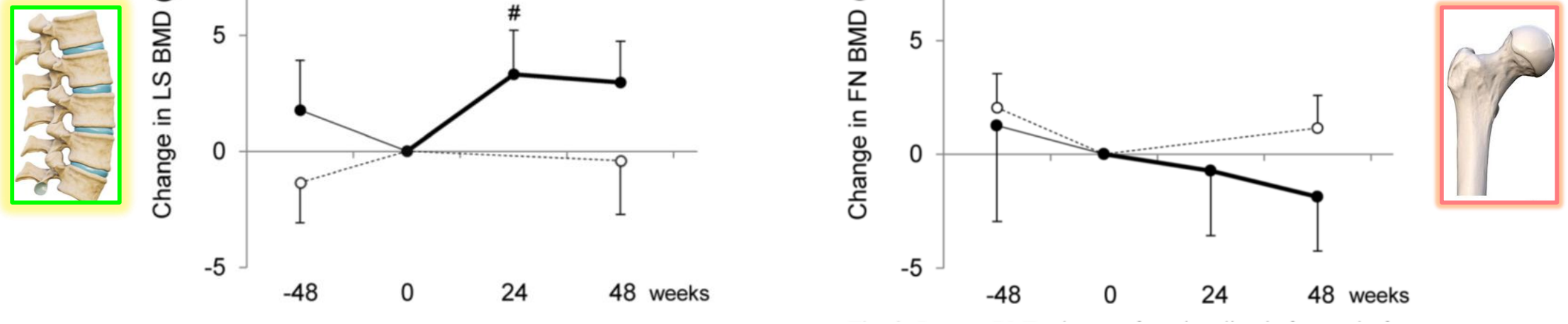
Am J Kidney Dis. 2022 Mar;79(3):427-43

PTH Analog Therapy in CKD G4–G5D

Author, Year	Drug	Type CKD	Number of Patients	Control Group	Dose of PTH Analog	Duration of PTH Analog	Lumbar BMD	Femur BMD	Fracture Incidence	Safety
Nishikawa et al., 2016 [69]	Teriparatide	CKD 4	30	–	20 µg/day	24 months	↑ BMD overall	↑ BMD (trend)	1 new fracture (CKD 5)	No serious AEs (4 mild in 33 patients)
		CKD 5	3	–	20 µg/day	24 months	↑ BMD (trend)	–	–	–
Cejka et al., 2010 [70]	Teriparatide	CKD 5	7	–	20 µg/day	6 months	↑ BMD lumbar (significant)	↑ BMD (not significant)	Not reported	No severe AEs reported
Mitsopoulos et al., 2012 [71]	Teriparatide	CKD 5	9	Yes	20 µg/day	13–16 months	↑ BMD lumbar (+4.9%)	↑ BMD femoral neck (+2.7%)	Not reported	No significant AEs
Sumida et al., 2016 [72]	Teriparatide	CKD 5	22	Yes (n = 8)	56.5 µg/ week	48 weeks	↑ BMD lumbar (+3%)	No change	Not reported	10/22 discontinued due to AEs (transient hypotension)
Yamamoto et al., 2020 [60]	Teriparatide	CKD 5	10	Yes (n = 5)	56.5 µg/ week	12 months	↑ BMD lumbar (+2.5% at 12 M)	No change	Not reported	40% dropout due to AEs (mainly hypotension)

PTH Analog Therapy in CKD G4–G5D:

Trabecular (LS), but not Cortical (Femoral Neck) bone benefit

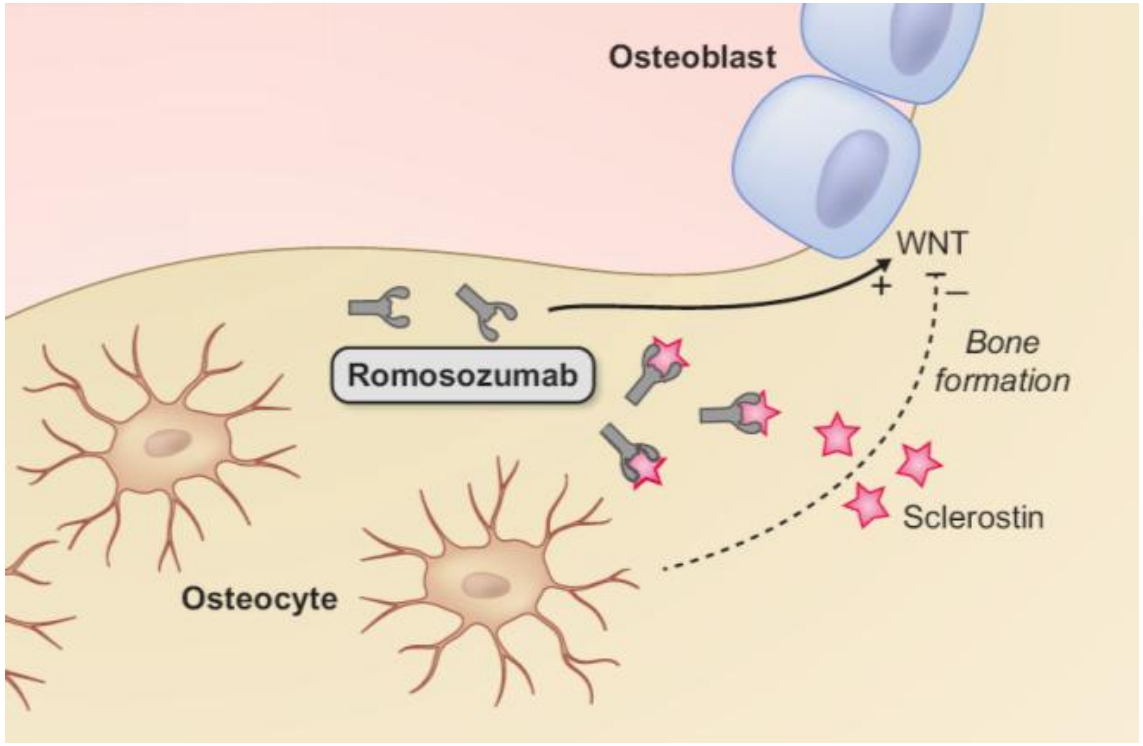


Cejka et al

Osteoporos Int (2016) 27:1441–1450

Να αυξήσουμε την παραγωγή νέου οστού ??

- Να μπλοκάρουμε την σκληροστίνη !!

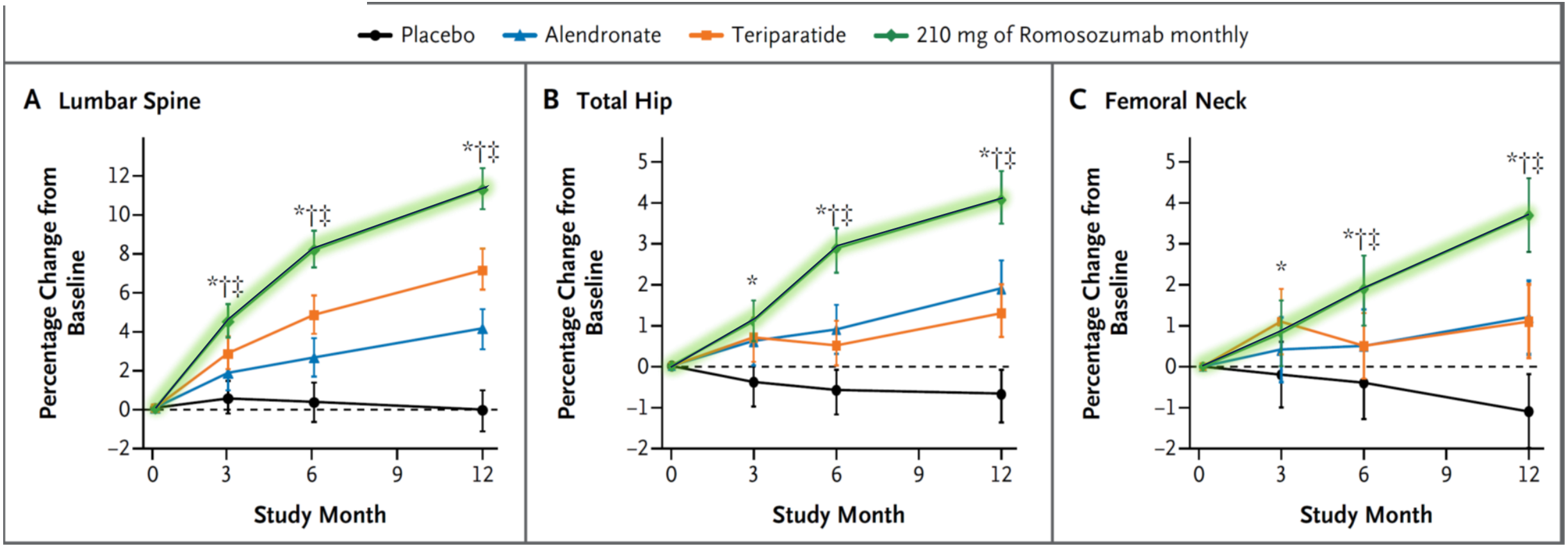


Romosozumab: μπλοκάροντας τη σκληροστίνη

με σημαντική αύξηση της BMD

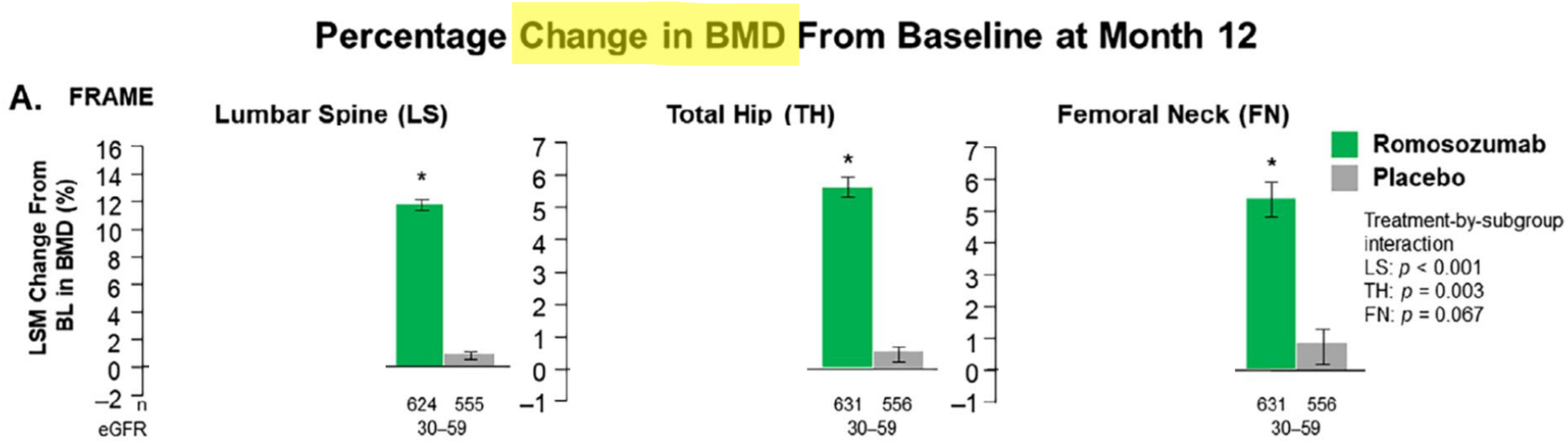
ORIGINAL ARTICLE

Romosozumab in Postmenopausal Women with Low Bone Mineral Density

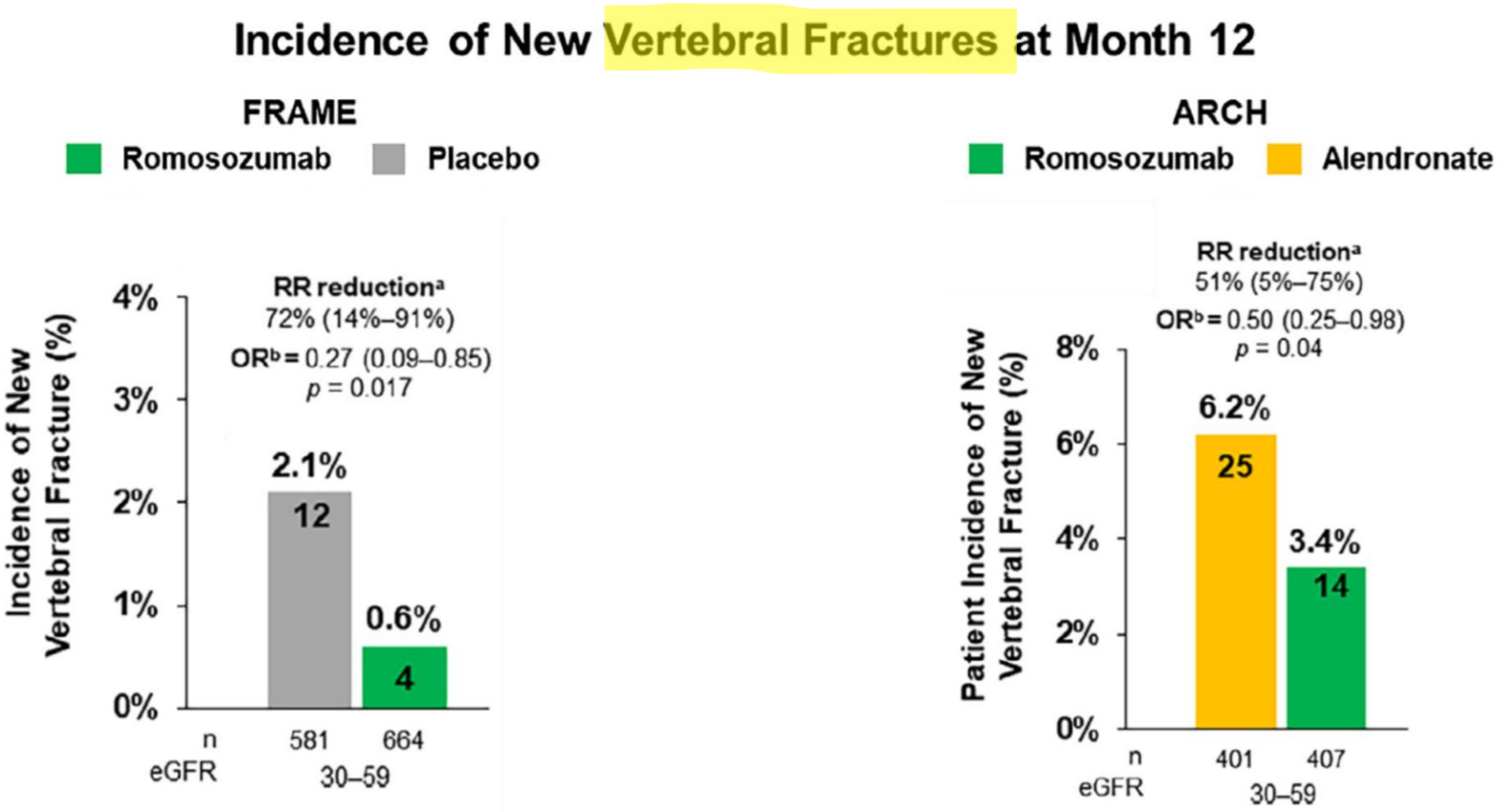


N Engl J Med 2014;370:412-20

ROMOSOZUMAB vs PLACEBO vs ALENDRONATE / CKD G3



ROMOSOZUMAB vs PLACEBO vs ALENDRONATE / CKD G3



ROMOSOZUMAB *vs* ALENDRONATE

Table 2. Adverse Events.

Event	Month 12: Double-Blind Period	
	Alendronate (N = 2014)	Romosozumab (N = 2040)
	number of pat	
Adverse event during treatment	1584 (78.6)	1544 (75.7)
Back pain†	228 (11.3)	186 (9.1)
Nasopharyngitis†	218 (10.8)	213 (10.4)
Serious adverse event	278 (13.8)	262 (12.8)
Adjudicated serious cardiovascular event‡	38 (1.9)	50 (2.5)
Cardiac ischemic event	6 (0.3)	16 (0.8)
Cerebrovascular event	7 (0.3)	16 (0.8)
Heart failure	8 (0.4)	4 (0.2)
Death	12 (0.6)	17 (0.8)

Saag K et al

N Engl J Med 2017;377:1417-27

Θεραπεία με Romosozumab σε ασθενείς με ΧΝΝ G4–G5D

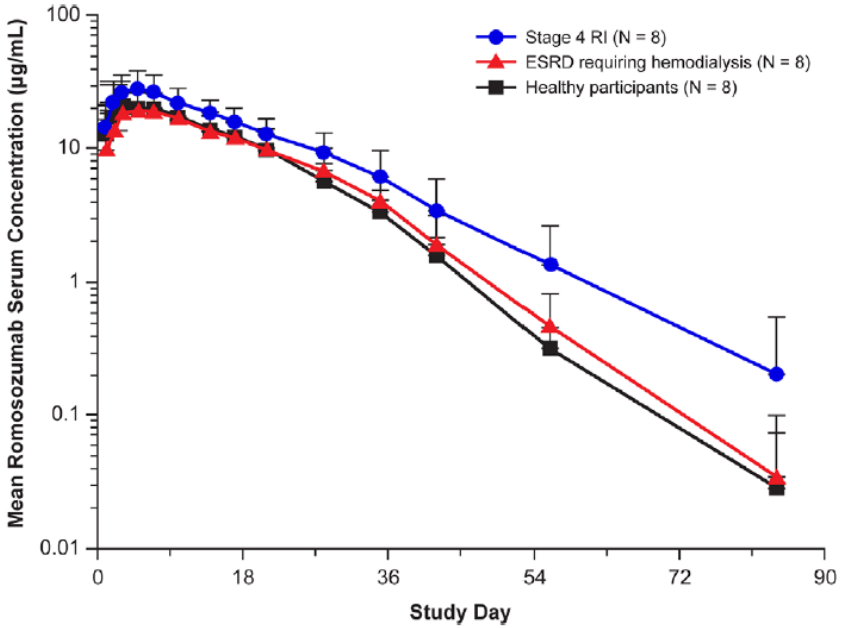
Table 2. Osteoporosis Drugs in Advanced CKD

Drug	Trials in eGFR < 45 (No. of Patients)	Trials in Dialysis Patients (No. of Patients)	Suggested Dosing Options	Side Effects	Efficacy
Mixed					
Antisclerostin antibody	Yes (430)	Yes (12)	Romosozumab, 210 mg, monthly	CVD, hypocalcemia, arthralgias	Efficacy in CKD, unclear in dialysis

Ginsberg C, Ix JH

Am J Kidney Dis. 2022 Mar;79(3):427-43

ROMOSOZUMAB: ΦΑΡΜΑΚΟΚΙΝΗΤΙΚΗ ΣΕ ΧΝΝ G4-5D



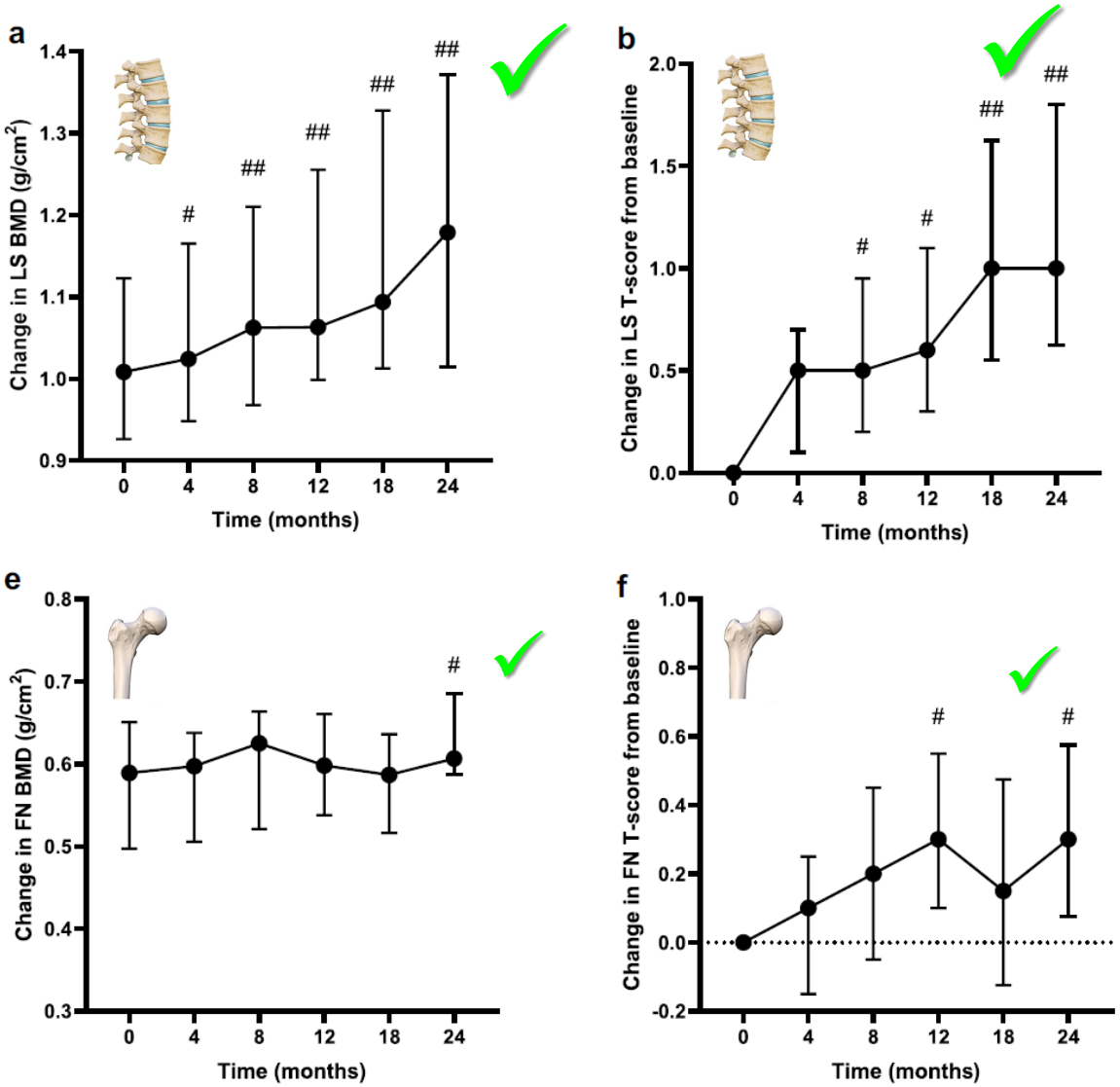
Influence of Renal Function on Pharmacokinetics, Pharmacodynamics, and Safety of a Single Dose of Romosozumab

	Patients With Stage 4 RI (N = 8)	Patients With ESRD-RH (N = 8)	Healthy Participants (N = 8)
All treatment-emergent adverse events	7 (87.5)	6 (75.0)	0 (0.0)
Serious adverse events	1 (12.5)	1 (12.5)	0 (0.0)
Leading to discontinuation of romosozumab	0 (0.0)	0 (0.0)	0 (0.0)
Fatal adverse events	0 (0.0)	0 (0.0)	0 (0.0)
Most common treatment-emergent adverse events (>5%)			
Hypocalcemia	1 (12.5)	4 (50.0)	0 (0.0)
Hyperparathyroidism secondary	4 (50.0)	0 (0.0)	0 (0.0)
Arthralgia	1 (12.5)	1 (12.5)	0 (0.0)
Constipation	0 (0.0)	2 (25.0)	0 (0.0)
Vomiting	1 (12.5)	1 (12.5)	0 (0.0)

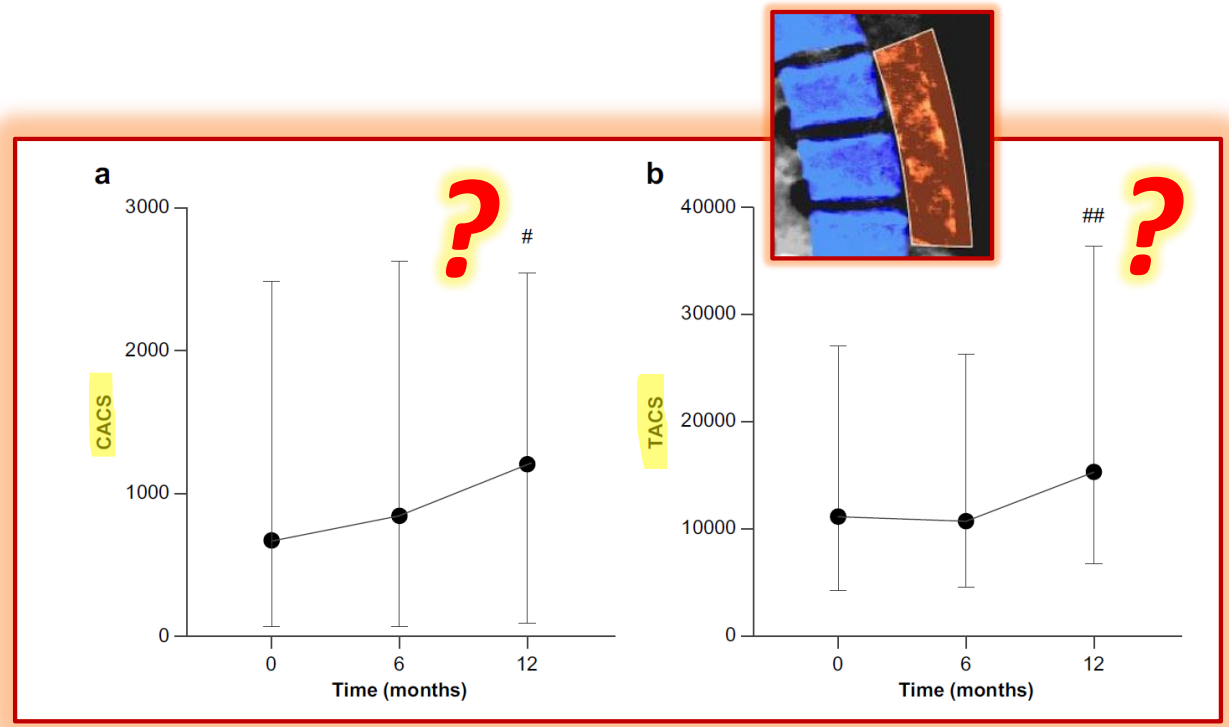
Hsu CP

J Clin Pharmacol. 2022 Sep;62(9):1132-1141

ROMOSOZUMAB + DENOSUMAB / DIALYSIS



One-Year Romosozumab Treatment Followed by One-Year Denosumab Treatment for Osteoporosis in Patients on Hemodialysis: An Observational Study



Saito T.

Calcif Tissue Int. 2023 Jan;112(1):34-44

Sclerostin: Χαμηλά επίπεδα *και χειρότερη επιβίωση !!* στην αιμοκάθαρση

High levels of circulating sclerostin are associated with better cardiovascular survival in incident dialysis patients: results from the NECOSAD study

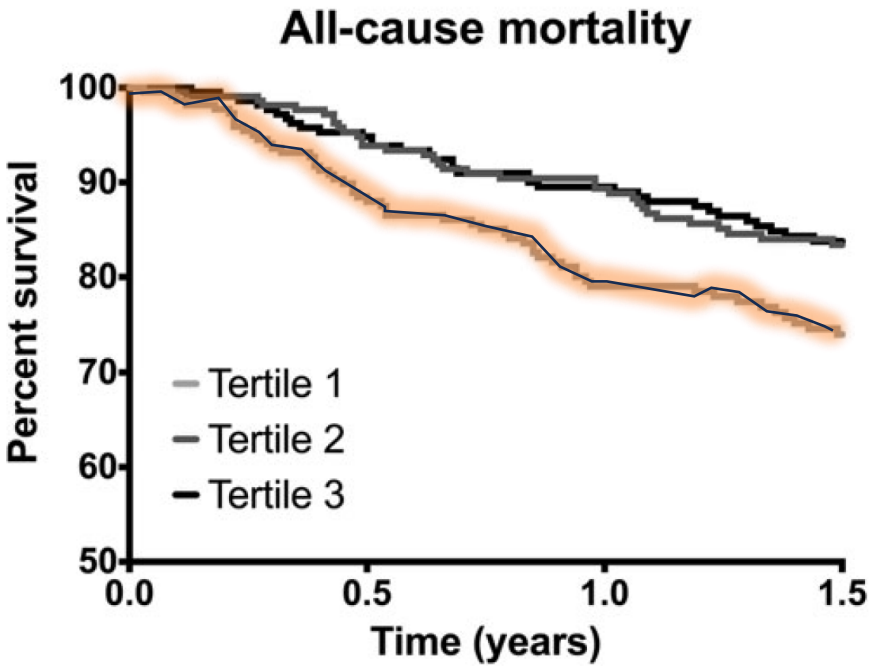


FIGURE 1: Kaplan–Meier analyses showing all-cause mortality according to baseline tertiles of sclerostin concentration.

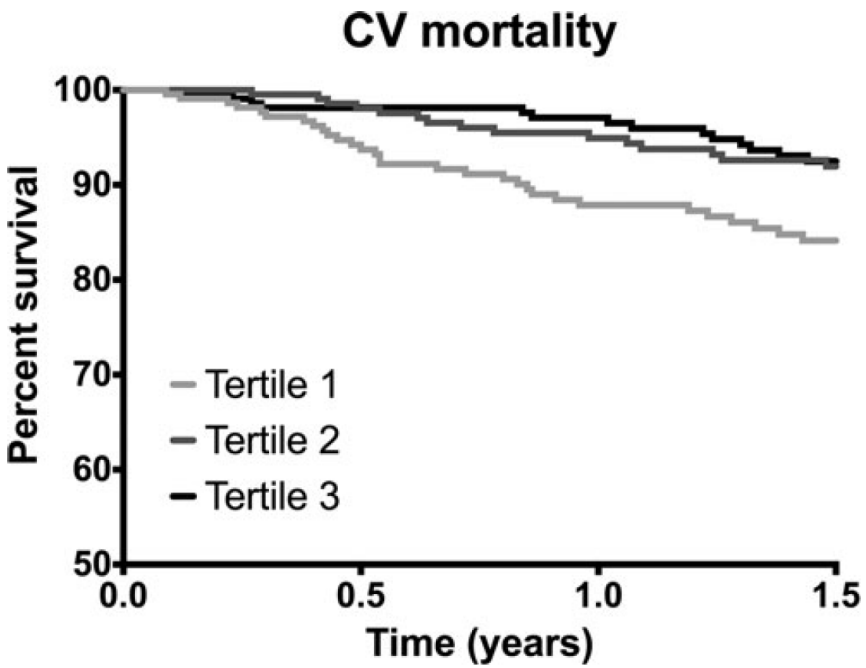
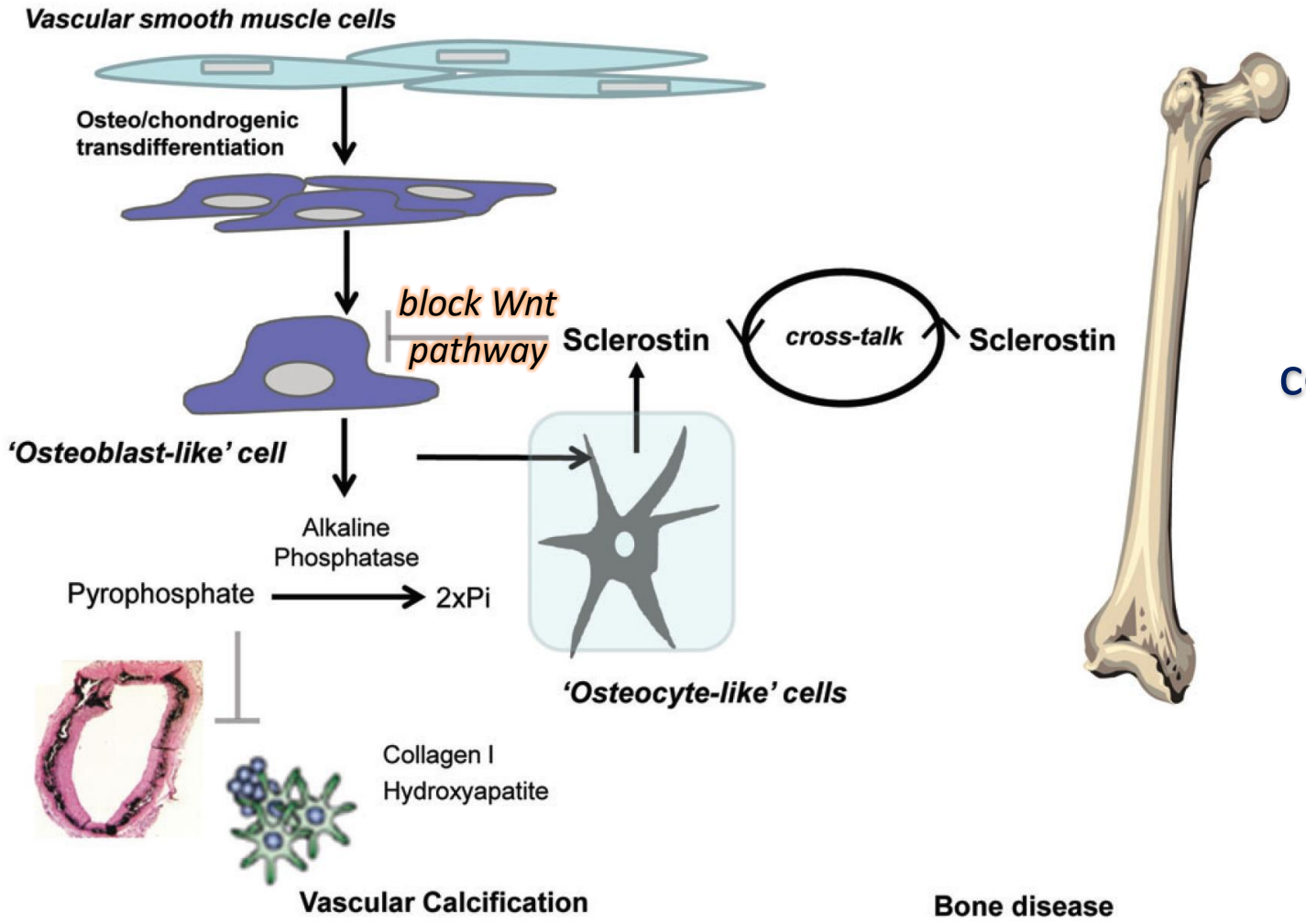


FIGURE 2: Kaplan–Meier analyses showing cardiovascular mortality according to baseline tertiles of sclerostin concentration.

Drechsler C et al

Nephrol Dial Transplant. 2015 Feb;30(2):288-93

Sclerostin: a vascular defensive feedback loop against calcification ?

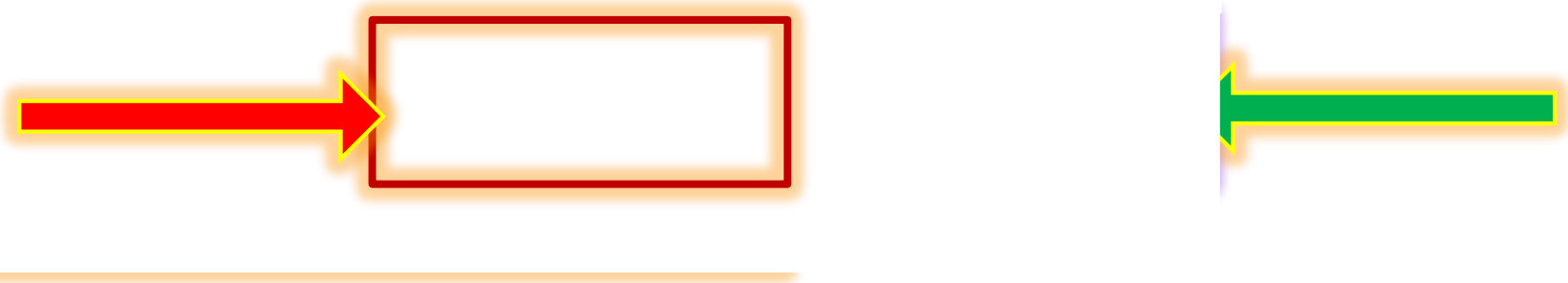


Bone health : collateral damage ? in a defensive mechanism ?

FIGURE 2: Vascular smooth muscles cells undergo osteo/chondrogenic transdifferentiation in a pro-calcifying environment. The resulting osteoblast-like cells induce alkaline phosphatases. These alkaline phosphatases catalyze the hydrolysis of PPi. In the late phase of VC, sclerostin is expressed. This can be interpreted as a defensive response that aims to block the Wnt pathway in order to reduce the mineralization in the vascular tissue. Sclerostin may spill over to the circulation and may reciprocally inhibit bone metabolism.

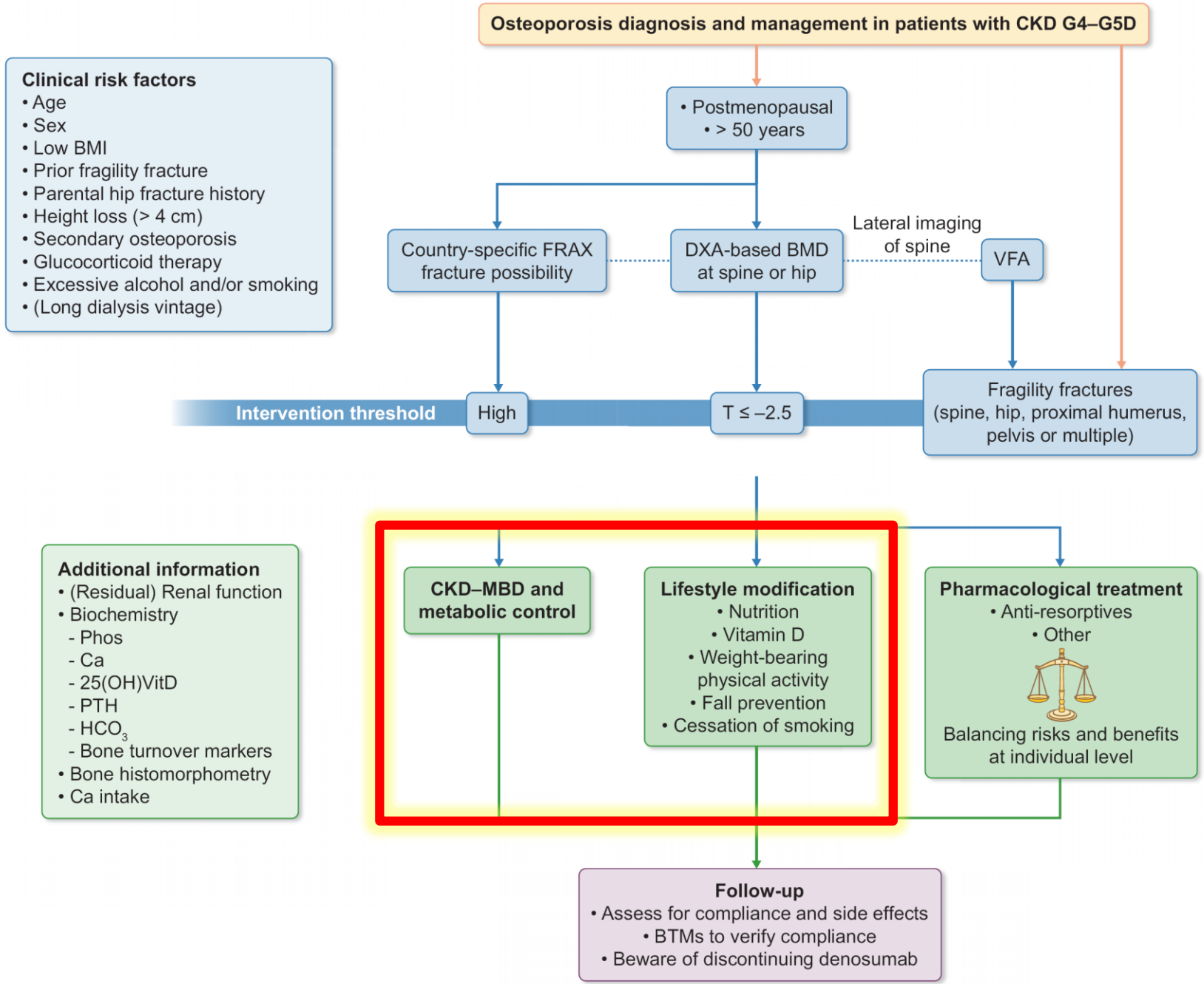
Differences between CKD-MBD and postmenopausal osteoporosis

Clinical factor	CKD-MBD	Postmenopausal osteoporosis
PTH levels	Increased	Usually normal*
Alkaline phosphatase	Increased	Usually normal*
Bone mineral density	Weakly related to fracture risk	Predicts risk of fracture



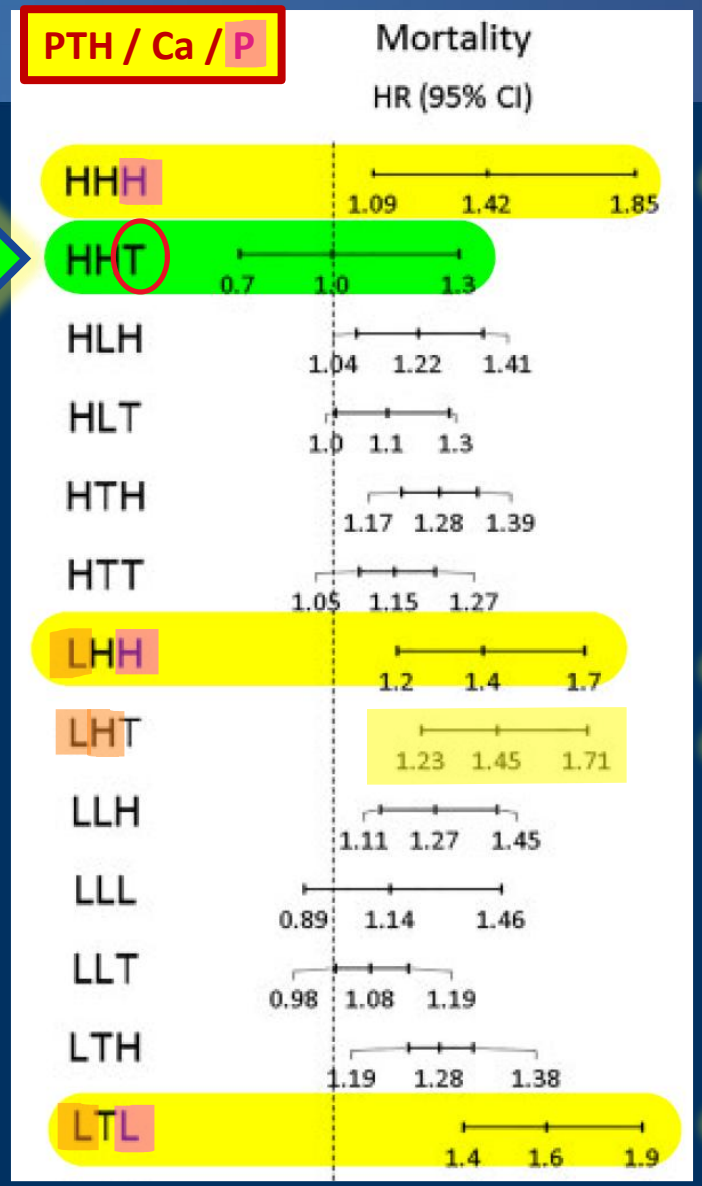
Ott SM

Nat Rev Nephrol 2013; 9:681


Osteoporosis diagnosis and management in patients with CKD G4–G5D
Clinical risk factors
• Age
• Sex
• Low BMI
• Prior fragility fracture
• Parental hip fracture history
• Height loss (> 4 cm)
• Secondary osteoporosis
• Glucocorticoid therapy
• Excessive alcohol and/or smoking
• (Long dialysis vintage)
• Postmenopausal
• > 50 years
Country-specific FRAX fracture possibility
DXA-based BMD at spine or hip
Lateral imaging of spine
VFA
Intervention threshold
High
T ≤ –2.5
Fragility fractures (spine, hip, proximal humerus, pelvis or multiple)
Additional information
• (Residual) Renal function
• Biochemistry
- Phos
- Ca
- 25(OH)VitD
- PTH
- HCO3
- Bone turnover markers
• Bone histomorphometry
• Ca intake
CKD–MBD and metabolic control
Lifestyle modification
• Nutrition
• Vitamin D
• Weight-bearing physical activity
• Fall prevention
• Cessation of smoking
Pharmacological treatment
• Anti-resorptives
• Other
Balancing risks and benefits at individual level
Follow-up
• Assess for compliance and side effects
• BTMs to verify compliance
• Beware of discontinuing denosumab

Η *Αδυναμική Οστική νόσος και ο φωσφόρος* συσχετίζονται με *θνητότητα*

Detecting high-risk chronic kidney disease–mineral bone disorder phenotypes among patients on dialysis: a historical cohort study



↑PTH / OK Phos

↑PTH + Phos

ΑΔΥΝΑΜΙΚΗ

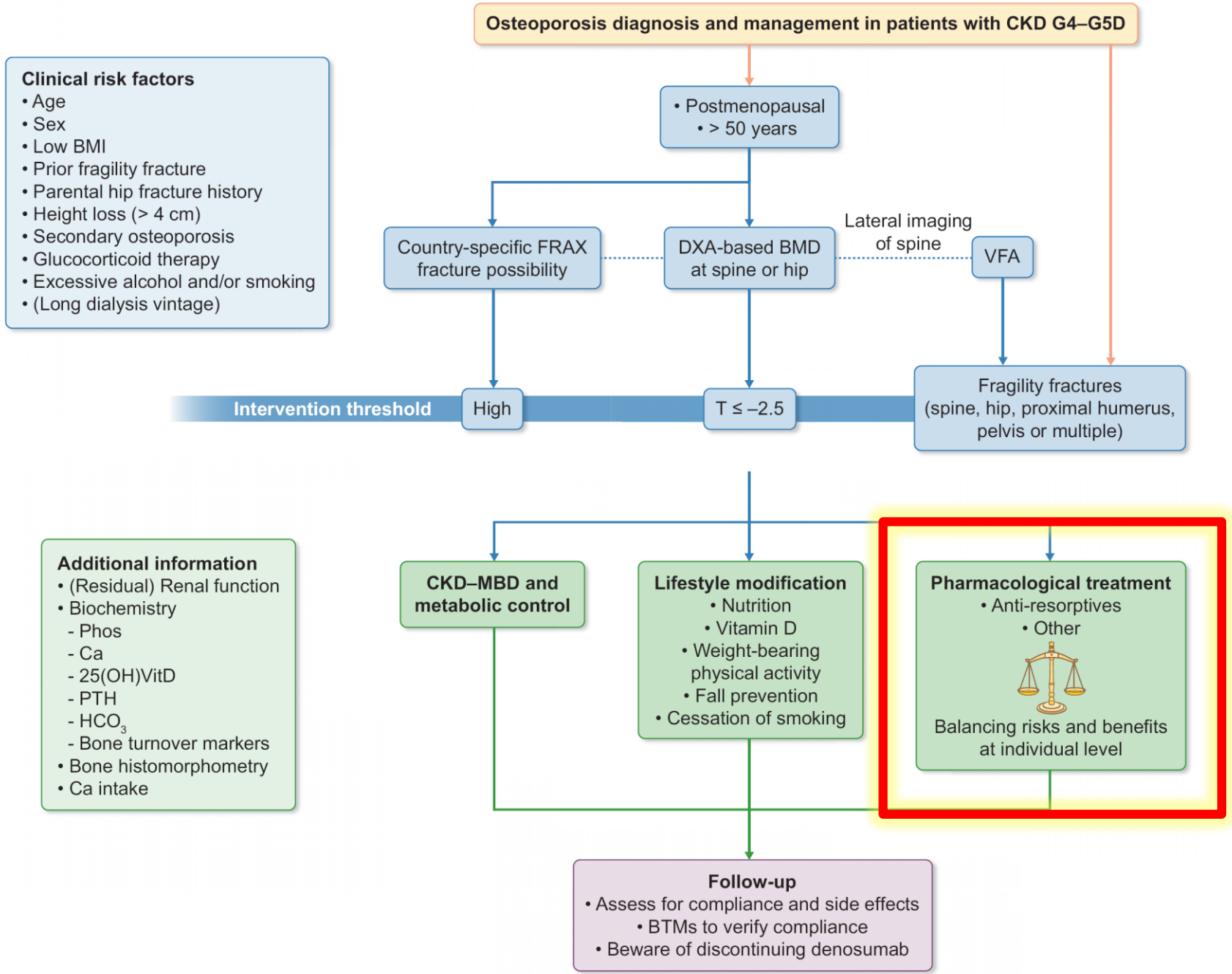
ΑΔΥΝΑΜΙΚΗ

ΑΔΥΝΑΜΙΚΗ

35.721 αιμοκαθαιρόμενοι

Neri et al

Nephrol Dial Transplant (2019) 34: 682–691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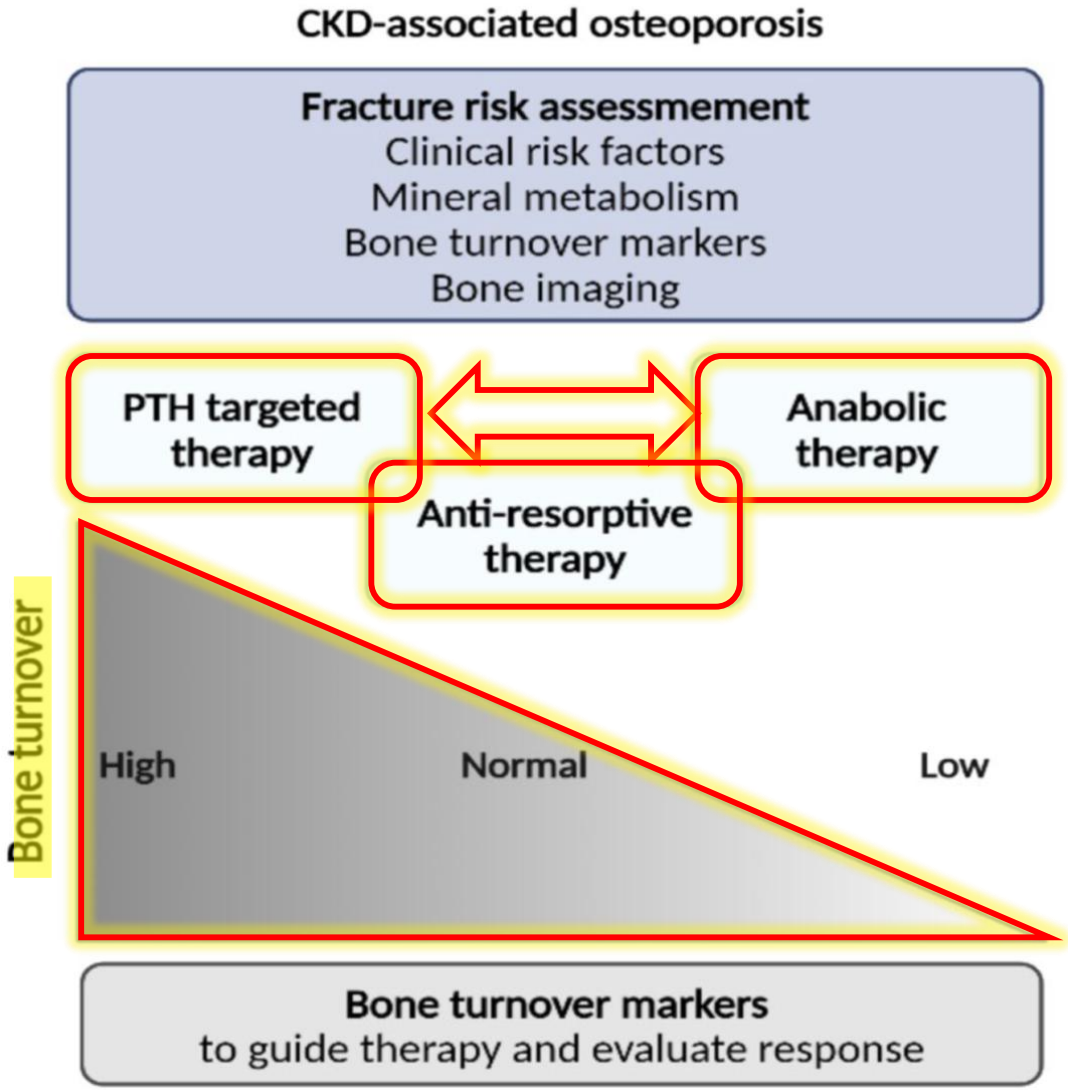
Επιλογή θεραπείας με γνώμονα την υποκείμενη διαταραχή του οστικού μεταβολισμού

POSITION PAPER

Update on the role of bone turnover markers in the diagnosis and management of osteoporosis: a consensus paper from The European Society for Clinical and Economic Aspects of Osteoporosis, Osteoarthritis and Musculoskeletal Diseases (ESCEO), International Osteoporosis Foundation (IOF), and International Federation of Clinical Chemistry and Laboratory Medicine (IFCC)

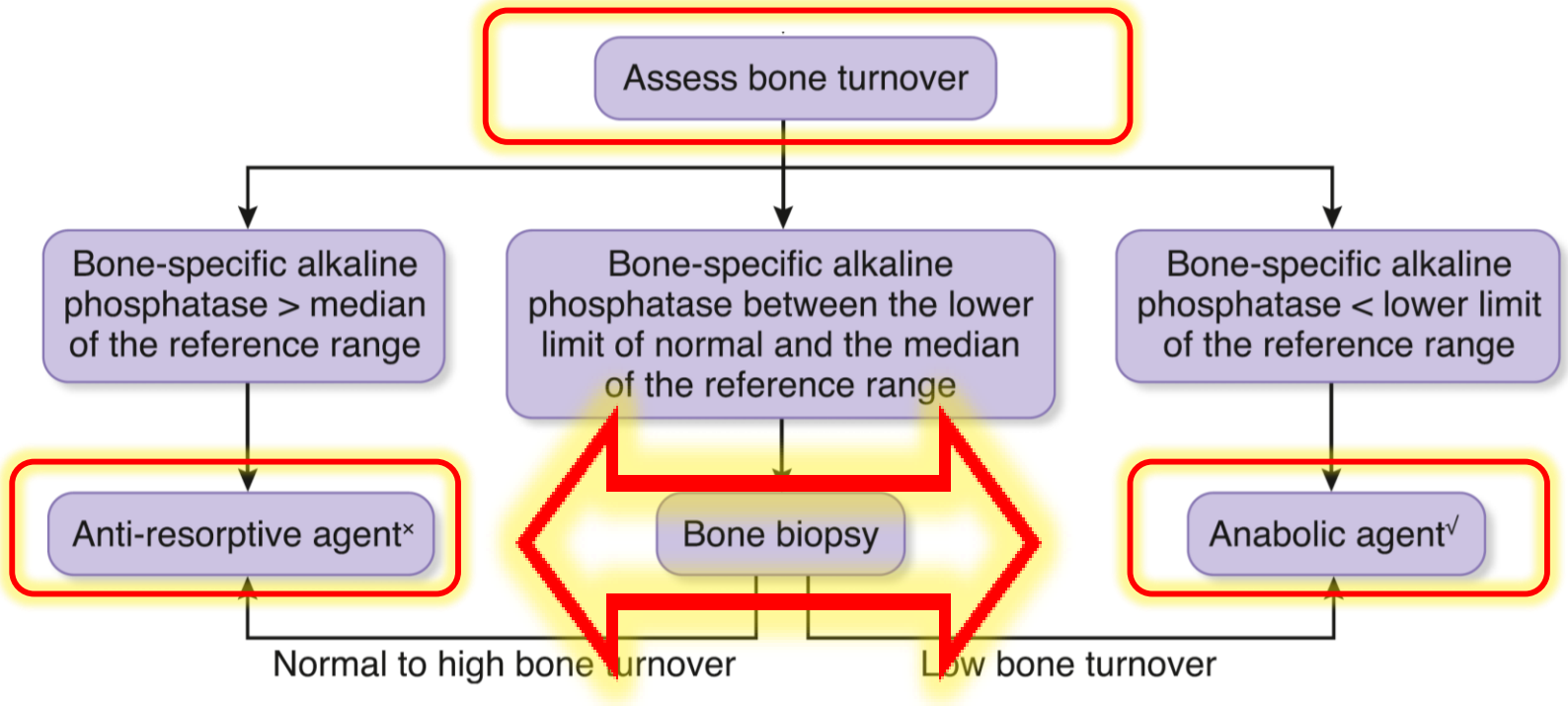
Optimal nutrition
Increased physical activity
Sufficient minerals and vitamin D
Acidosis control
Inflammation control
Glycemic control
Uremia control



Bhattoa HP

Osteoporos Int. 2025 Apr;36(4):579-608

Επιλογή θεραπείας με γνώμονα την υποκείμενη διαταραχή του οστικού μεταβολισμού

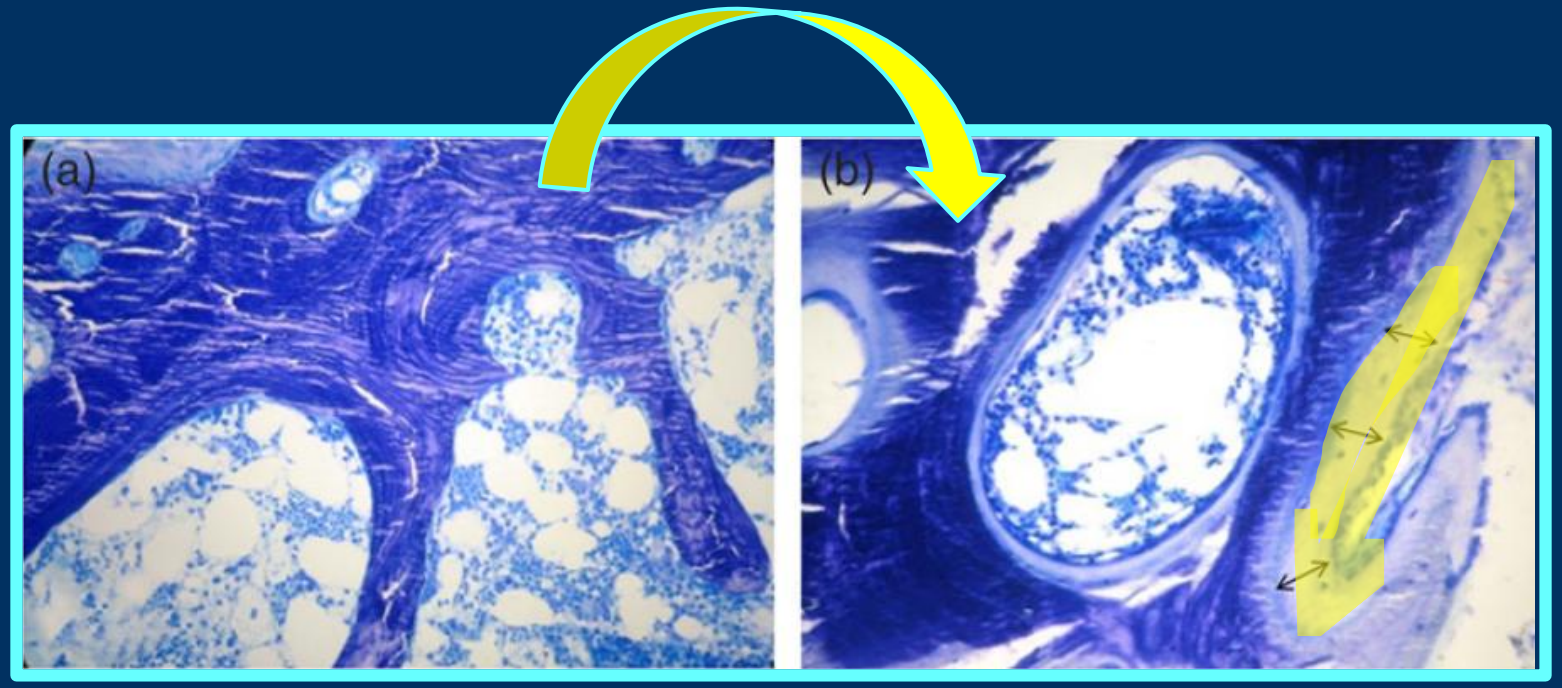


Khairallah P, Nickolas TL
Clin J Am Soc Nephrol. 2018;13(6):962-969

Treatment of adynamic bone disease in a haemodialysis patient with teriparatide

Panagiotis Giamalis, Dominiki Economidou, Chrysostomos Dimitriadis, Dimitrios Memmos, Aikaterini Papagianni and Georgios Efstratiadis

Επειδή , όταν η ιατρική γίνεται προσωπική υπόθεση και πάθος, δεν ισχύει το «Ουδείς αναντικατάστος» ....



Clin Kidney J (2015) 8: 188–190

- Η οστεοπόρωση/CKD-MBD είναι μία σύνθετη νόσος ειδικά στην ΧΝΝ σταδίων 4-5D

- Πρέπει να αντιμετωπίζεται γιατί συνδέεται με υψηλό κίνδυνο καταγμάτων και νοσηρότητας / θνητότητας

- Ωστόσο, το φάσμα της υποκείμενης οστικής νόσου είναι μεγάλο και η οστική πυκνότητα δεν απεικονίζει το είδος της υποκείμενης διαταραχής

- Ο συνδυασμός των δεικτών οστικού μεταβολισμού P1NP, bALP και TRAP5B23 , που δεν εξαρτώνται από τη νεφρική λειτουργία, παρέχει κρίσιμες πληροφορίες πριν την επιλογή θεραπείας

- ΔΕΝ ΕΙΝΑΙ ΩΣΤΟΣΟ ΑΠΟΚΛΕΙΣΤΙΚΑ ΟΣΤΙΚΗ ΝΟΣΟΣ: οι αγγειακές βλάβες καθορίζουν την επιβίωση των ασθενών !

- Η αδυναμική οστική νόσος είναι συχνή και συνδέεται τόσο με αυξημένη καρδιαγγειακή θνητότητα όσο και με ΜΕΓΑΛΥΤΕΡΟ κίνδυνο καταγμάτων !

- Αντιμετωπίστε πρώτα τις διαταραχές που σχετίζονται με τη ΧΝΝ (ΔΥΠΘ, Υπερφωσφαταιμία)

- Επιλέξτε περαιτέρω θεραπεία αφού προσπαθήσετε να αναγνωρίσετε το ρυθμό του οστικού μεταβολισμού !

Hansen D

Nephrol Dial Transplant. 2024 Dec 20;40(1):48-59

ΕΥΧΑΡΙΣΤΩ ΠΟΛΥ ΓΙΑ

ΤΗΝ ΠΡΟΣΟΧΗ ΣΑΣ !!!