İnflamatuvar Hastalıklarda Osteoporoza

Güncel Bakış

Dr Rana Terlemez

İÜC Cerrahpaşa Tıp Fakültesi
Fiziksel Tıp ve Rehabilitasyon AD

Kronik
Sistemik
İnflamasyon

Romatoid Artrit

KOAH

SLE

Hashimoto Tiroidit

Kronik Sistemik İnflamasyon

Spondiloartropatiler

İnflamatuvar Barsak Hastalığı

Psöriazis

Diabetes Mellitus


Osteopeni / Osteoporoz

İnflamatuvar hastalıklarda osteopeni/osteoporoz yeterince taranıyor mu?

Osteoporosis: An Underdiagnosed Problem in Patients with Ankylosing Spondylitis

Osteoporoz: Ankilozan Spondilitli Hastalarda Yeterince Tanı Konmamış Bir Problem

Yeşim Kirazlı, Ece Çınar

- Genç erkek hastalar nadiren taranmaktadır
- Klinisyenlerin primer amacı hastaların semptomlarını kontrol altına almak
- AS hastalarında OP taramasına yönelik bir kılavuz bulunmamaktadır

Turk J Osteoporos
2022;28:1-5

Arthritis Care & Research
Vol. 69, No. 5, May 2017, pp 642–648
DOI 10.1002/acr.22988

ORIGINAL ARTICLE

Variations in Radiographic Procedure Use for Medicare Patients With Rheumatoid Arthritis

GABRIELA SCHMAJUK,[1] CHRIS TONNER,[2] LAURA TRUPIN,[2] AND JINOOS YAZDANY[2]

- Retrospektif kohort çalışması
- 8051 RA'lı hasta, %81 kadın

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Table 1. Characteristics of Medicare beneficiaries with rheumatoid arthritis (n = 8,051)

	No.	(%)
Female sex	6,545	81.3
Age, years		
65–71	2,546	31.6
72–78	2,622	32.6
≥79	2,883	35.8
Nonwhite race	1,296	16.0

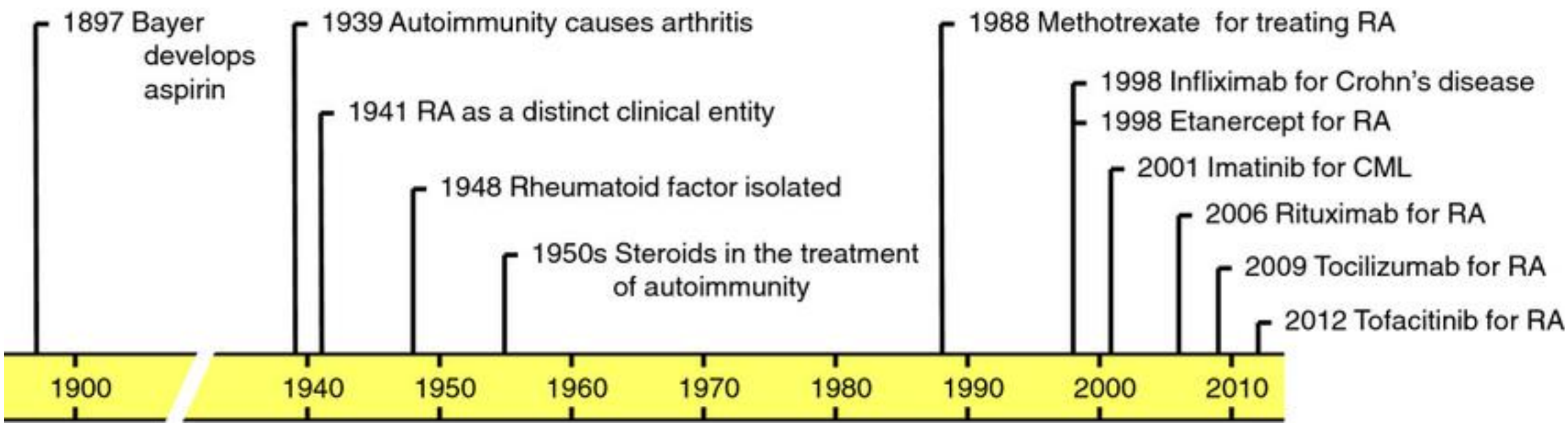
Table 2. Procedure utilization 2008–2009 among Medicare beneficiaries with rheumatoid arthritis*

	Peripheral joint MRI	Peripheral joint conventional radiograph	DXA scan
Total no.	8,051	8,051	6,545
Median no. procedures per beneficiary	0	1	1
Mean no. procedures per beneficiary	0.33	2.64	0.70
Range of procedures per beneficiary	0–50	0–33	0–11
No. (%) with ≥1 procedure	1,062 (13.1)	5,093 (59.9)	3,355 (51.3)
Median no. procedures among beneficiaries with ≥1 procedure	2	2	1
Mean no. procedures among beneficiaries with ≥1 procedure	2.50	4.14	1.36
No. (%) with ≥2 procedures	548 (6.8)	3,828 (47.5)	860 (13.4)
No. (%) with ≥2 procedures on same joint†	518 (6.4)	3,340 (41.4)	–

* MRI = magnetic resonance imaging; DXA = dual x-ray absorptiometry.
† Current Procedural Terminology codes do not specify laterality, so some procedures classified as being on the same joint may represent contralateral examinations.

65 y üstü RA’lı hastalarda rehberler net olmasına rağmen tarama ve tedavi oranları düşük

Variations in radiographic procedure use for medicare patients with rheumatoid arthritis. Arthritis Care Res 2017


1897 Bayer develops aspirin
1939 Autoimmunity causes arthritis
1941 RA as a distinct clinical entity
1948 Rheumatoid factor isolated
1950s Steroids in the treatment of autoimmunity
1988 Methotrexate for treating RA
1998 Infliximab for Crohn's disease
1998 Etanercept for RA
2001 Imatinib for CML
2006 Rituximab for RA
2009 Tocilizumab for RA
2012 Tofacitinib for RA
1900
1940
1950
1960
1970
1980
1990
2000
2010

JRD Journal of Rheumatic Diseases
J Rheum Dis 2023;30(1):3-17
https://doi.org/10.4078/jrd.22.0046
pISSN: 2093-940X, eISSN: 2233-4718
Review Article

Positive Effects of Biologics on Osteoporosis in Rheumatoid Arthritis

Yunkyung Kim, M.D., Ph.D., Geun-Tae Kim, M.D., Ph.D.

Division of Rheumatology, Department of Internal Medicine, Kosin University Gospel Hospital, Kosin University College of Medicine, Busan, Korea

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Biologics	Classification	Bone formation marker			Bone resorption marker				Others				
		PINP	OC	BAP	CTX	NTX	ICTP	DPD	OPG	RANKL	OPG/RANKL	MMP3	DKK1
Infliximab	TNF-α inhibitor	↑	↑	→	↓	↓	↓	↓	↑	↓	↓		
Adalimumab	TNF-α inhibitor	↑			↓						↑		
Golimumab	TNF-α inhibitor	↑			↓						↑		
Certolizumab	TNF-α inhibitor	↑			↓						↑		↓
Etanercept	TNF-α inhibitor	↑	↑	↑	↓	→			↑→	↓→	↓		↓
Abatacept	CTLA4-Ig								↑	↓			↓
Rituximab	Anti-CD20	↑		↑					↓	↓	↑		
Tocilizumab	IL-6 inhibitor	↑			↓				↑		↑	↓	↓

JOURNAL ARTICLE

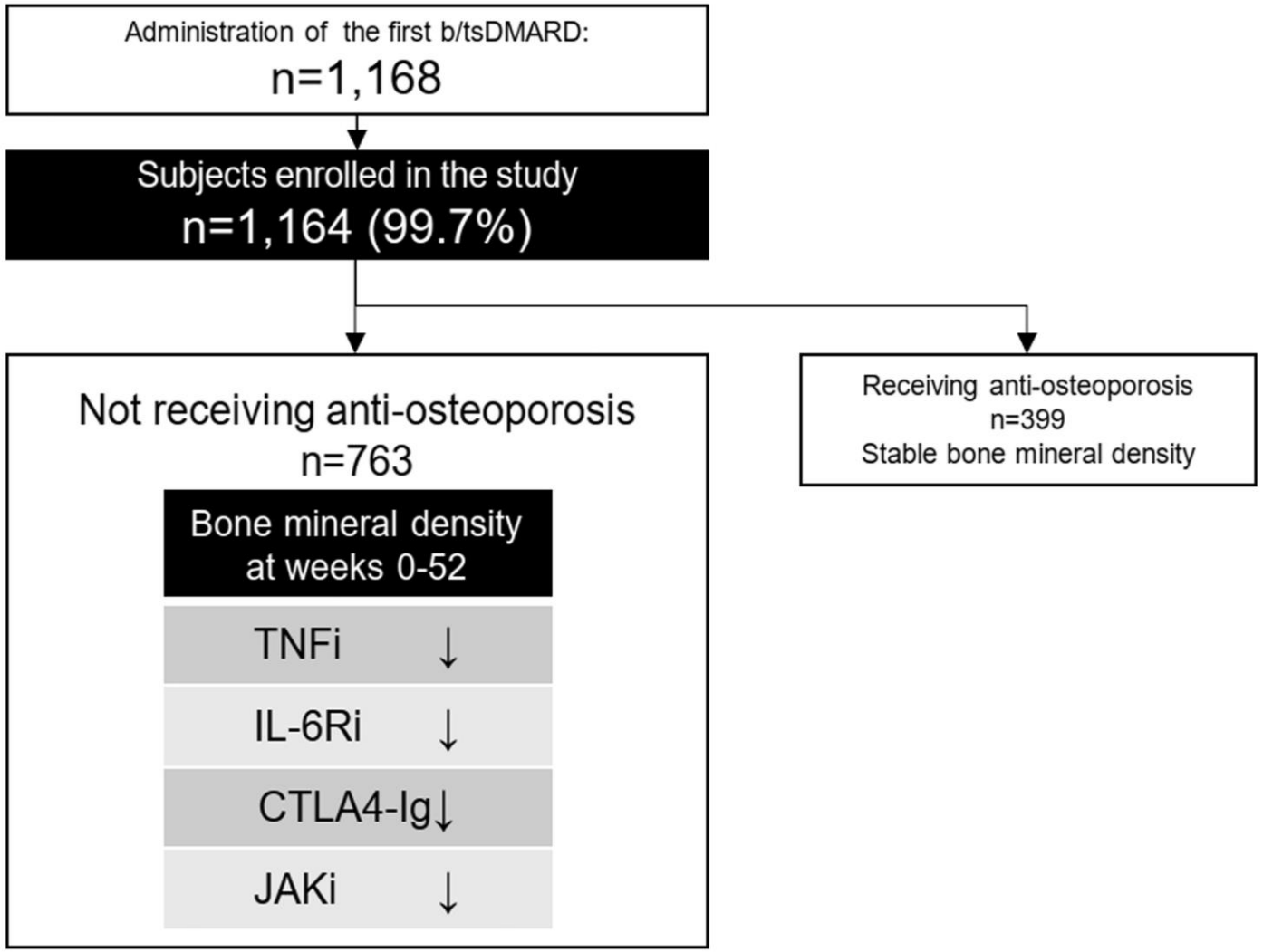
Biological/targeted synthetic DMARDs do not arrest bone loss in patients with rheumatoid arthritis: a multicenter prospective observational study

Get access ›

Koshiro Sonomoto, Shingo Nakayamada, Yoshihisa Fujino, Hiroko Miyata, Satoshi Kubo, Yuya Fujita, Yoshino Inoue, Satsuki Matsunaga, Shigeru Iwata,

- Çok merkezli, prospektif bir çalışma
- İlk kez bDMARD başlanan 1164 RA'lı hasta dahil edilmiş

Rheumatology, 2024



Rheumatology (Oxford), Volume 63, Issue 8, August 2024, Pages 2239–2248, https://doi.org/10.1093/rheumatology/kead579

Table 2. Changes in BMD, CDAI and bone turnover markers during treatment of the whole cohort

	Week 0	Week 2	Week 26	Week 52
BMD, Femoral neck (g/cm^3)				
Total	0.621 ± 0.132		0.615 ± 0.129*	0.615 ± 0.130*
Non-anti-OP	0.659 ± 0.126		0.649 ± 0.125*	0.646 ± 0.126*
anti-OP	0.551 ± 0.111		0.551 ± 0.111	0.555 ± 0.115
BMD, Radius (g/cm^3)				
Total	0.477 ± 0.116		0.476 ± 0.115*	0.470 ± 0.115*
Non-anti-OP	0.510 ± 0.110		0.507 ± 0.109*	0.500 ± 0.112*
anti-OP	0.415 ± 0.103		0.416 ± 0.104	0.415 ± 0.100

JOURNAL ARTICLE

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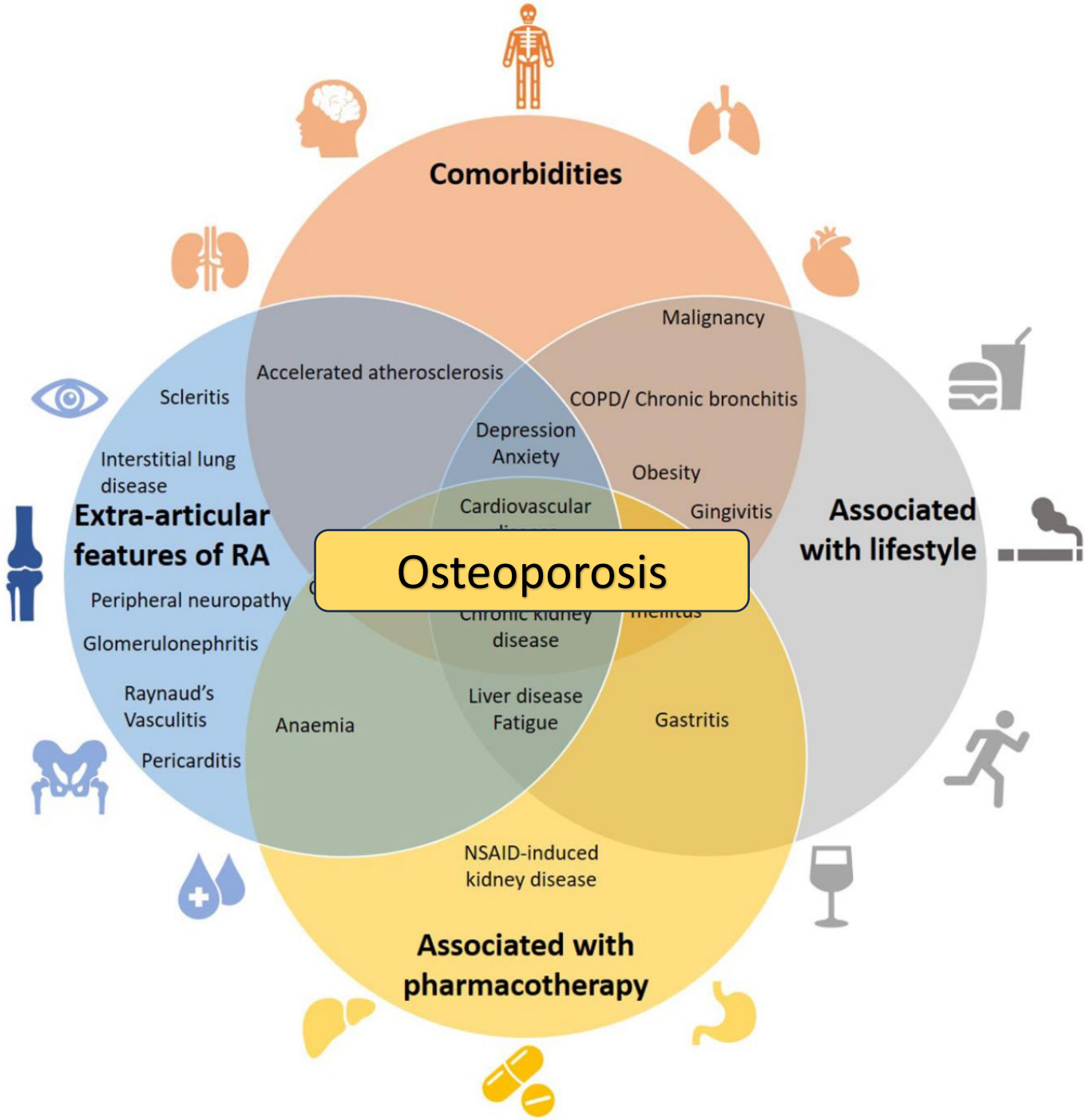
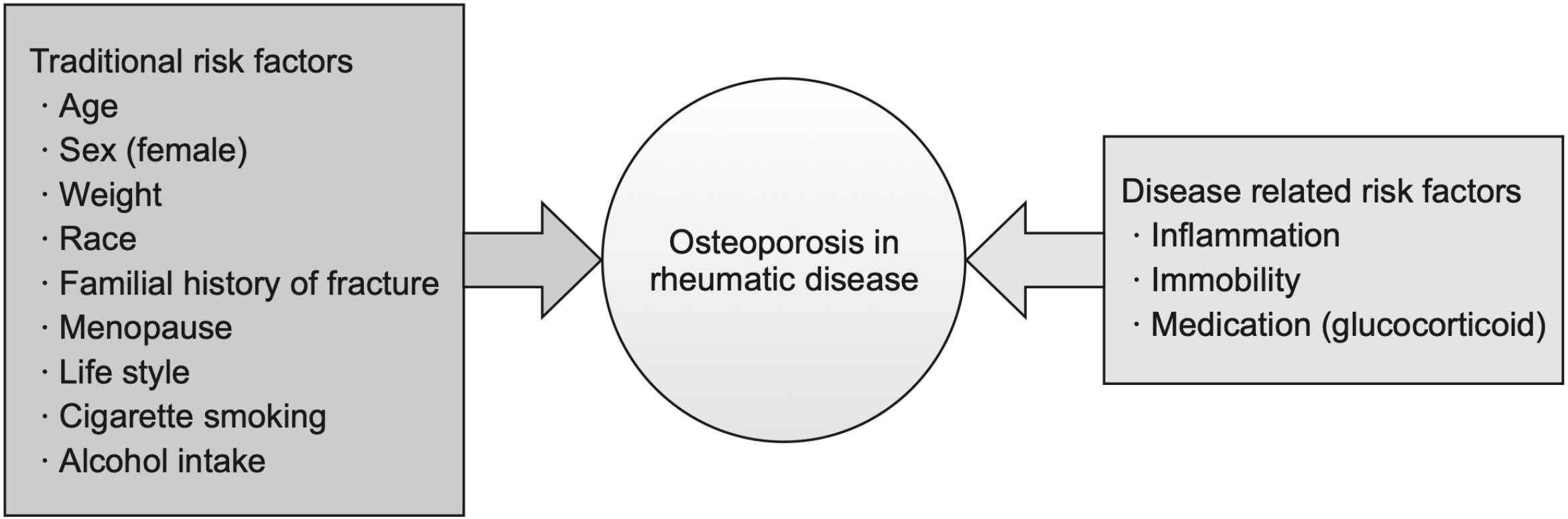
Conclusion

Our study suggested the progression of osteoporosis in RA patients during b/tsDMARDs treatment without anti-OP. BTMs may not reflect BMD change. Regular monitoring of BMD in RA should be considered for early management of osteoporosis.

Rheumatology, 2024



Rheumatology, 2023



J Rheum Dis 2023; 30(1): 3-17

Tüm sekonder osteoporoz olgularında olduğu gibi **altta yatan hastalığı (inflamasyonu) kontrol altına almak** ile birlikte **tüm risk faktörlerini değerlendirmek** temel tedavi yaklaşımını da oluşturuyor

Olgu 1

- 39 y, K
- RA tanılı (2017)
- Mtx 15 mg/hf ve etanercept 50 mg /hf ile remisyonda

Olgu 1- Hikaye

- Menarş yaşı: 13
- 2 gebelik (1 abortus, 1 canlı doğum)
- 1 yıl laktasyon

Olgu 1- Hikaye

- Düzenli egzersiz yapmakta
- Alkol, sigara kullanmıyor
- Kırık (kendisinde ya da ailesinde) hikayesi yok

Premenopozal RA’lı bir hastada kemik mineral yoğunluğu ne zaman istenmeli?

Evaluation and treatment of premenopausal osteoporosis

UpToDate®

Evaluation and treatment of premenopausal osteoporosis

✓ Premenopozal kadınlarda rutin KMY ölçümü önerilmiyor

Evaluation and treatment of premenopausal osteoporosis

- ✓ Premenopozal kadınlarda rutin KMY ölçümü önerilmiyor
- ✓ Ancak **frajilite kırığı hikayesi** ya da **sekonder osteoporoz** varlığı

Table 12
Causes of Secondary Osteoporosis in Adults[a]

Endocrine or metabolic causes	Nutritional/ GI conditions	Drugs	Disorders of collagen metabolism	Other
Acromegaly Diabetes mellitus Type 1 Type 2 Growth hormone deficiency Hypercortisolism Hyperparathyroidism Hyperthyroidism Hypogonadism Hypophosphatasia Porphyria Pregnancy	Alcoholism Anorexia nervosa Calcium deficiency Chronic liver disease Malabsorption syndromes/ malnutrition (including celiac disease, cystic fibrosis, Crohn disease, and gastric resection or bypass) Total parenteral nutrition Vitamin D deficiency	Anti-epileptic drugs[b] Aromatase inhibitors Chemotherapy/ immunosuppressants Medroxyprogesterone acetate Glucocorticoids Gonadotropin-releasing hormone agents Heparin Lithium Proton pump inhibitors Selective serotonin- reuptake inhibitors SGLT2-inhibitors Thiazolidinediones Thyroid hormone (in supraphysiologic doses)	Ehlers-Danlos syndrome Homocystinuria due to cystathionine deficiency Marfan syndrome Osteogenesis imperfecta	AIDS/HIV Ankylosing spondylitis Chronic obstructive pulmonary disease Gaucher disease Hemophilia Hypercalciuria Immobilization Major depression Myeloma and some cancers Organ transplantation Renal insufficiency/ failure Renal tubular acidosis Rheumatoid arthritis Systemic mastocytosis Thalassemia

AIDS = acquired immunodeficiency syndrome; GI = gastrointestinal; HIV = human immunodeficiency virus; SGLT2 = sodium-glucose cotransporter 2.
[a]Not meant to be a complete list.
[b]Phenobarbital, phenytoin, primidone, valproate, and carbamazepine have been associated with low bone mass.

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Olgu-1 DXA 2024

Results Summary:

Region	Area[cm^2]	BMC[(g)]	BMD[g/cm^2]	T-score	PR (Peak Reference)	Z-score	AM (Age Matched)
L1	12.86	12.13	0.943	-0.4	95	0.0	100
L2	13.13	13.40	1.021	-0.1	99	0.4	104
L3	14.08	14.02	0.995	-0.8	92	-0.3	96
L4	15.28	15.96	1.045	-0.1	98	0.3	104
Total	**55.35**	**55.51**	**1.003**	**-0.4**	**96**	**0.1**	**101**

Total BMD CV 1.0%, ACF = 1.034, BCF = 1.015, TH = 7.076

Olgu-1 DXA 2024

Results Summary:

Region	Area[cm²]	BMC[(g)]	BMD[g/cm²]	T-score	PR (Peak Reference)	Z-score	AM (Age Matched)
Neck	**5.22**	**4.38**	**0.838**	**-0.1**	**99**	**0.3**	**105**
Troch	8.18	5.90	0.722	0.2	103	0.4	106
Inter	18.15	21.04	1.159	0.4	105	0.5	108
Total	31.55	31.33	0.993	0.4	105	0.7	110
Ward's	1.31	0.96	0.737	0.0	100	0.9	117

Total BMD CV 1.0%, ACF = 1.034, BCF = 1.015, TH = 6.949

Evaluation and treatment of premenopausal osteoporosis

Non-Farmakolojik Yöntemler

- Yeterli kalsiyum ve vitamin D alımı
- Egzersiz
- Altta yatan hastalığın kontrolü

Evaluation and treatment of premenopausal osteoporosis

Farmakolojik Tedavi Kime Başlanmalı

- Kalça veya vertebra kırığı
- Multipl frajilite kırıkları
- KMY'de yılda ≥ % 3 düşüş

Olgu-1 DXA 2024

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Olgu-1 DXA 2022

Region	Area[cm^2]	BMC[(g)]	BMD[g/cm^2]
L1	13.34	13.10	0.982
L2	13.38	14.02	1.048
L3	14.40	15.01	1.043
L4	16.78	17.57	1.047
Total	**57.90**	**59.70**	**1.031**

Olgu-1 DXA 2024

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% 2.9

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Evaluation and treatment of premenopausal osteoporosis

Farmakolojik Tedavi Kime Başlanmalı

- Kalça veya vertebra kırığı
- Multipl frajilite kırıkları
- KMY'de yılda ≥ % 3 düşüş
- KMY'de kısa sürede hızlı düşüşe sebep olabilecek tedaviler

(GK, KT, hormonoterapiler, aromataz inhibitörleri)

< 40 y altı GK kullanan hastada farmakolojik tedavi

- Yakın tarihli frajilite kırığı
- Yılda 5 gram ve daha fazla GK kullanımı
- 6 ay süreyle 7.5 mg/g GK
- Z skoru < -3
- KMY'de 0.03 gr/cm^2 düşüş

2022 American College of Rheumatology Guideline for the Prevention and Treatment of Glucocorticoid-Induced Osteoporosis

Farmakolojik Tedavide Ne Başlanmalı?

- Antirezorptifler
- Anabolik ajanlar

Farmakolojik Tedavide Ne Başlanmalı?

- Antirezorptifler (Bifosfonatlar, denosumab)

- Anabolik ajanlar (Teriparatid)

Ne zaman Teriparatid?

- Kalça ya da vertebra kırığı varlığı
- Z skoru ≤-3.0 ve frajilite kırığı hikayesi
- Gebelik planlaması olan hasta

UpToDate®

Romosozumab versus denosumab in long-term users of glucocorticoids: A pilot randomized controlled trial

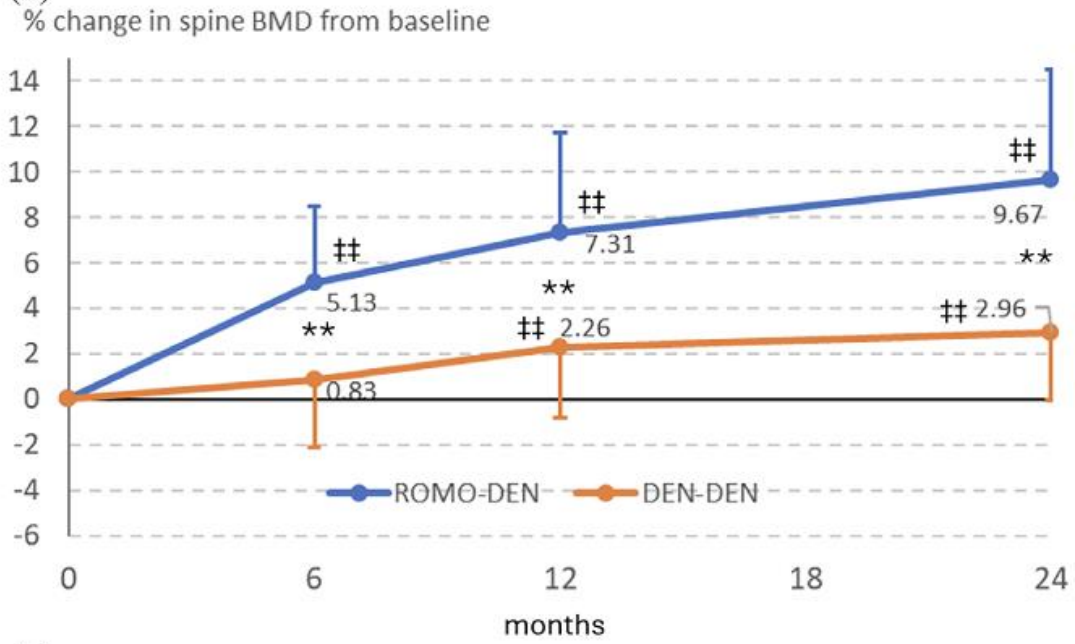
Chi Chiu Mok[1], Kar Li Chan[1], Sau Mei Tse[1], Sammy Pak Lam Chen[2], Kathryn Choon Beng Tan[3] & Wai Han Ma[4]

Table 1. *Clinical characteristics and osteoporosis risk factors of recruited patients.*

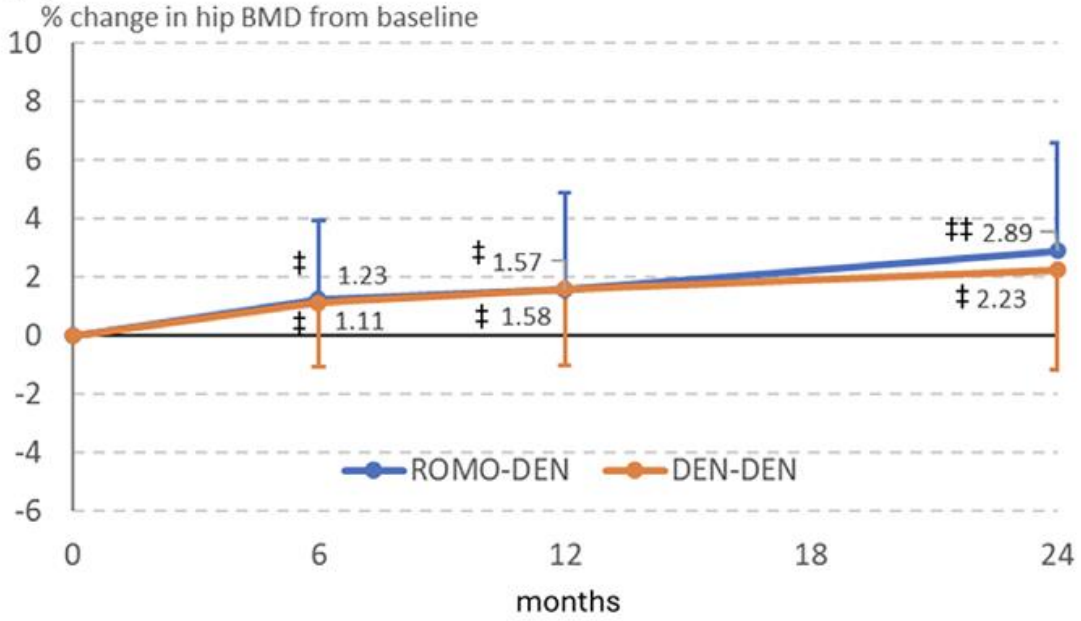
	Mean ± SD, number (%)		
	Romosozumab ($N = 35$)	Denosumab ($N = 35$)	p
Age, years	61.9 ± 8.3	63.3 ± 10.0	0.51
Women	34 (97.1)	33 (94.3)	1.00
Underlying disease			
SLE or lupus-like disease	17 (48.6)	19 (54.3)	0.63
Rheumatoid arthritis	14 (40.0)	6 (17.1)	
Primary Sjogren syndrome	2 (5.7)	1 (2.9)	
Inflammatory myopathy	2 (5.7)	4 (11.4)	
Systemic vasculitides	0 (0.0)	4 (11.4)	
Others	0 (0.0)	1 (2.9)	

J Intern Med. 2024 Dec;296(6):481

(a)



(b)



J Intern Med. 2024 Dec;296(6):481

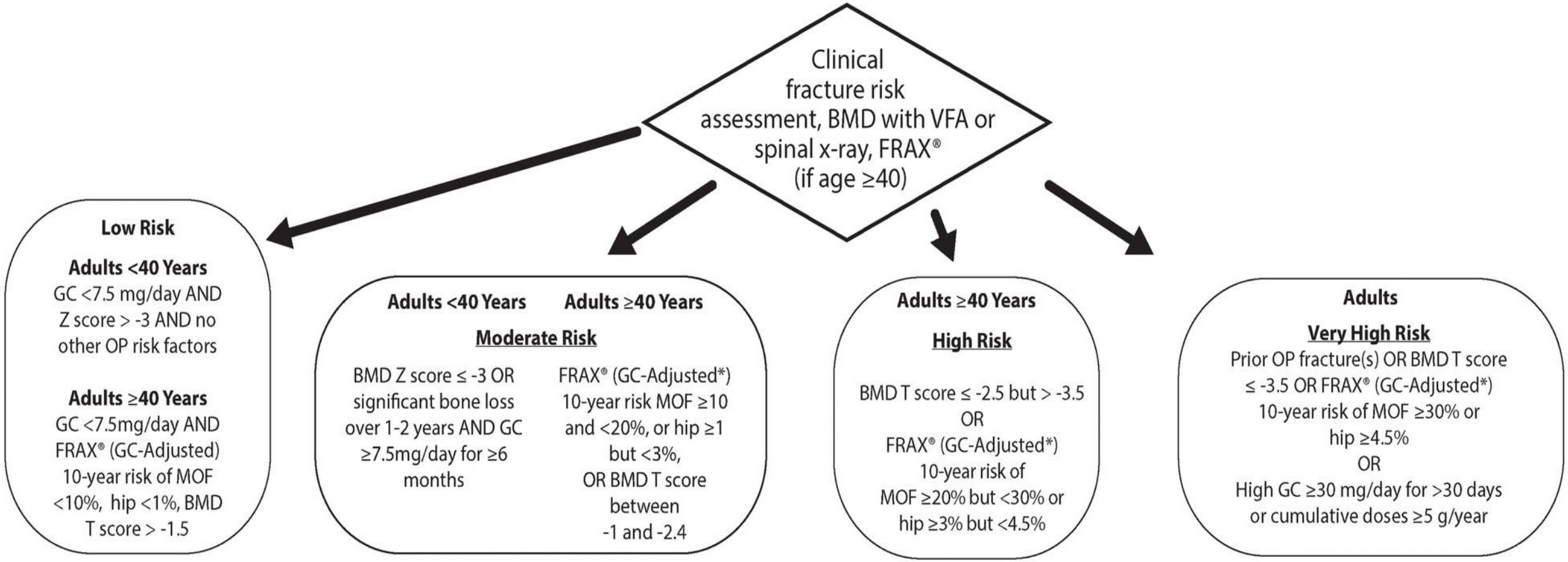
Olgu-2

- 63 y, seropozitif RA
- 28 yıldır RA tanılı
- Adalimumab, mtx, prednizolon 5mg /g
- Ek hastalık yok

Olgu-2

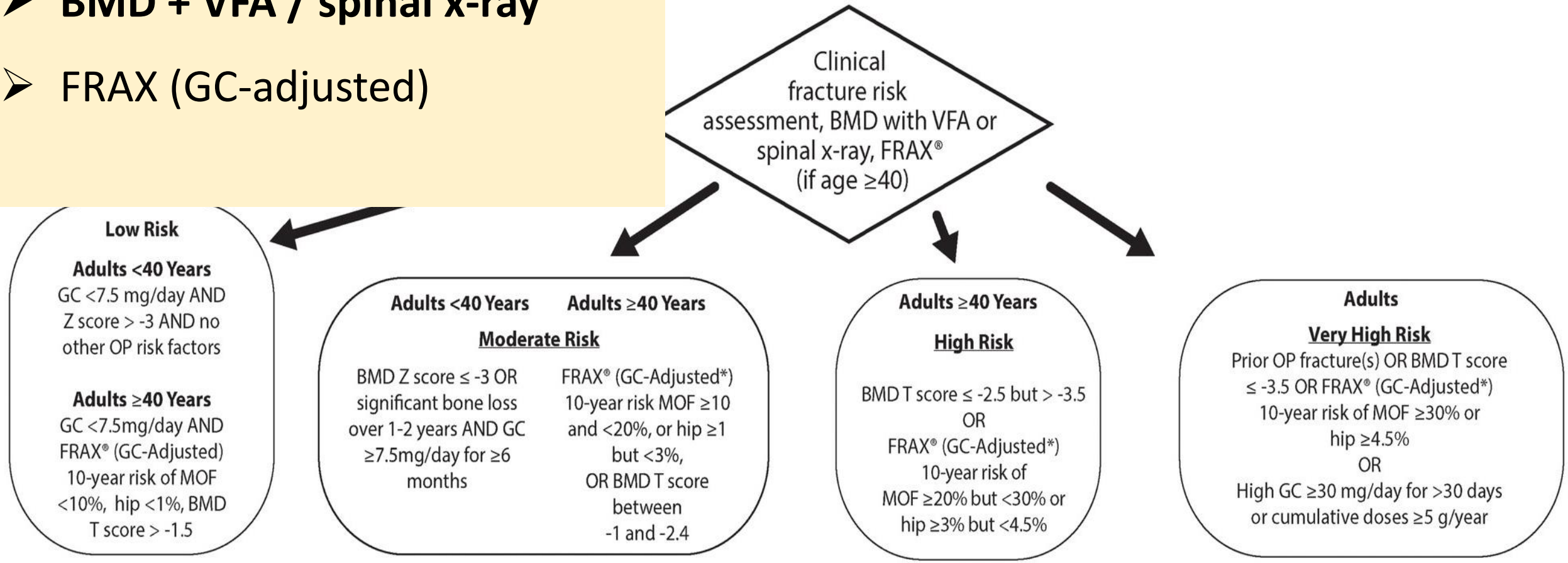
- Geçirilmiş kırık yok
- Menopoz yaşı 54, menarş yaşı?
- 2 canlı doğum, 2 yıl laktasyon
- Sigara, alkol kullanımı yok

2022 American College of Rheumatology Guideline for the Prevention and Treatment of Glucocorticoid-Induced Osteoporosis



2022 American College of Rheumatology Guideline for the Prevention and Treatment of Glucocorticoid-Induced Osteoporosis

- **BMD + VFA / spinal x-ray**
- FRAX (GC-adjusted)



Region	Area[cm^2]	BMC[(g)]	BMD[g/cm^2]	T-score	PR (Peak Reference)	Z-score	AM (Age Matched)
L1	13.02	9.36	0.719	-2.5	73	-1.0	87
L2	14.03	10.64	0.759	-2.4	74	-0.8	89
L3	14.86	13.43	0.904	-1.6	83	0.1	101
L4	16.15	14.56	0.902	-1.4	85	0.3	104
Total	**58.06**	**47.99**	**0.827**	**-2.0**	**79**	**-0.4**	**95**

Total BMD CV 1.0%, ACF = 1.034, BCF = 1.015, TH = 6.122

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Region	Area[cm^2]	BMC[(g)]	BMD[g/cm^2]	T-score	PR (Peak Reference)	Z-score	AM (Age Matched)
Neck	**5.07**	**3.09**	**0.609**	**-2.2**	**72**	**-0.7**	**88**
Troch	11.16	5.67	0.508	-1.9	72	-0.9	85
Inter	16.99	16.56	0.975	-0.8	89	0.1	101
Total	33.21	25.32	0.762	-1.5	81	-0.3	95
Ward's	1.11	0.41	0.372	-3.1	51	-0.9	77

Total BMD CV 1.0%, ACF = 1.034, BCF = 1.015, TH = 5.545

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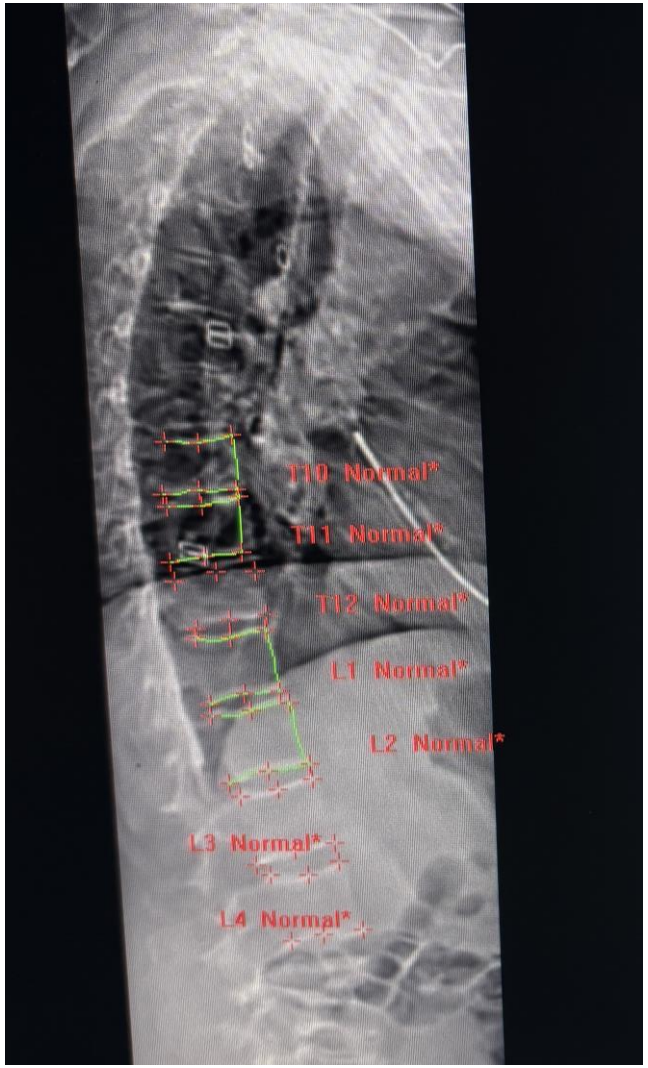
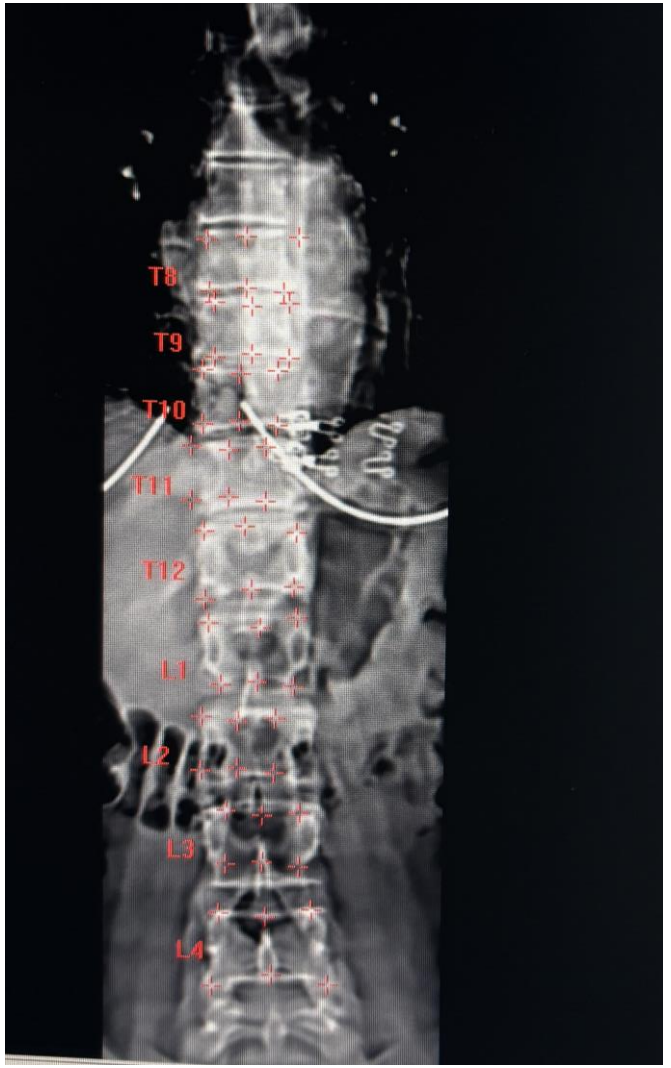
- Lomber total T skoru: -2.0
- Femur boyun T skoru: -2.2

Vertebral fracture assessment (VFA)



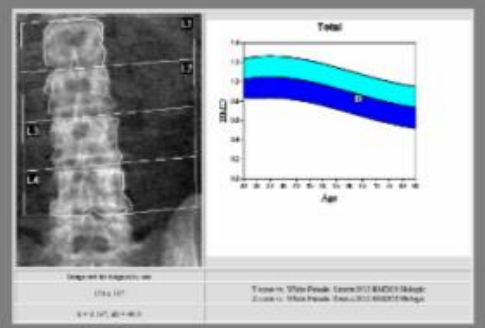


ISTANBUL UNIVERSITESI CERRAHPASA TIP FAKULTESI

NUKLEER TIP ANABILIMDALI

Patient Information:

Name:	AKSAKAL, AYSE
Patient ID:	34474682110
Identifier 2:	
Postal Code:	
Sex:	Female
Ethnicity:	White
Height:	158.0 cm
Weight:	52.0 kg
DOB:	16.05.1961
Age:	63
Menopause Age:	40
Referring Physician:	TERLEMEZ, RANA



Scan Information:

Scan Date:	01 October 2024 - A10012401
Scan Type:	(Lumbar Spine
Analysis Date:	01.10.2024 10:24
Analysis Protocol:	Spine
Report Date:	01.10.2024 10:25
Institution:	ISTANBUL UNIVERSITESI CERRAHPASA TIP FAKULTESI
Operator:	
Model:	Horizon W (S/N305833M)
Comment:	
Software version:	13.6.1.3

Results Summary:

Region	Area[cm²]	BMC[g]	BMD[g/cm²]	T-score	PR (Peak Reference)	Z-score	AM (Age Matched)
L1	13.02	9.36	0.719	-2.5	73	-1.6	87
L2	14.03	10.64	0.759	-2.4	74	-0.8	89
L3	14.86	13.43	0.904	-1.6	83	0.1	101
L4	16.15	14.36	0.902	-1.4	80	0.3	104
Total	**58.06**	**47.99**	**0.827**	**-2.0**	**79**	**-0.4**	**95**

Total BMD CV 1.0%, ACF = 1.034, BCF = 1.015, TH = 0.122

Fracture Risk: Increased; WHO Classification: Osteopenia

Comment:

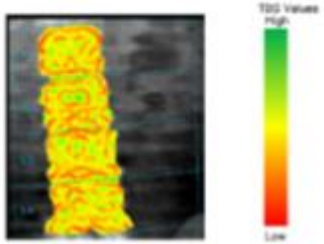
HOLOGIC®

Patient:	AKSAKAL, AYSE		
Patient ID:	34474682110	Date of birth - Age:	5/16/1961 - 63 years
Height - Weight - BMI:	158 cm - 52 kg - 20.8 kg/m²	Gender - Ethnicity:	Female - White
Referring physician:	TERLEMEZ, RANA	Acquisition date:	10/1/2024

BONE HEALTH REPORT

Inappropriate for clinical use due to one or more of the following:
DXA system not calibrated - BMI out of range - Age lower than 20

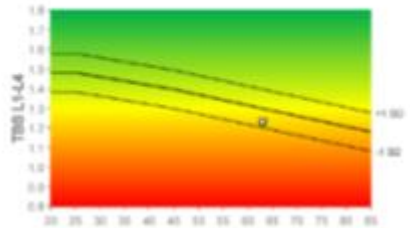
1 TBS Mapping



Non-diagnostic image

2 TBS Spine Results

TBS L1-L4 = 1.231 - Partially degraded microarchitecture

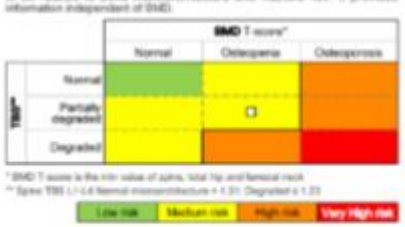


Reference population: USA (NHANES - Mediterranean) - White

3 Fracture Risk Assessment

Osteoporosis is a systemic skeletal disease characterized by low bone mass and microarchitectural deterioration of bone tissue, with a consequent increase in bone fragility and susceptibility to fractures.†

The TBS is derived from the texture of the DXA image and has been shown to be related to bone microarchitecture and fracture risk. It provides information independent of BMD.



* BMD T-score is the min value of spine, total hip and femoral neck

** Spine TBS L1-L4 Normal microarchitecture = 1.31; Degraded = 1.23

Color coded bone health categories based on Fracture Risk[2]

4 Therapeutic Decision Tools

The FRAX® 10-year probability of fracture:

Type of Fracture	Risk	Risk adjusted*
Major Osteoporotic	-	-
Hip	-	-

* Adjusted for TBS. † Validated only for Caucasian and Asian women and men. Refer to local guidelines before using these values.
FRAX has not been computed

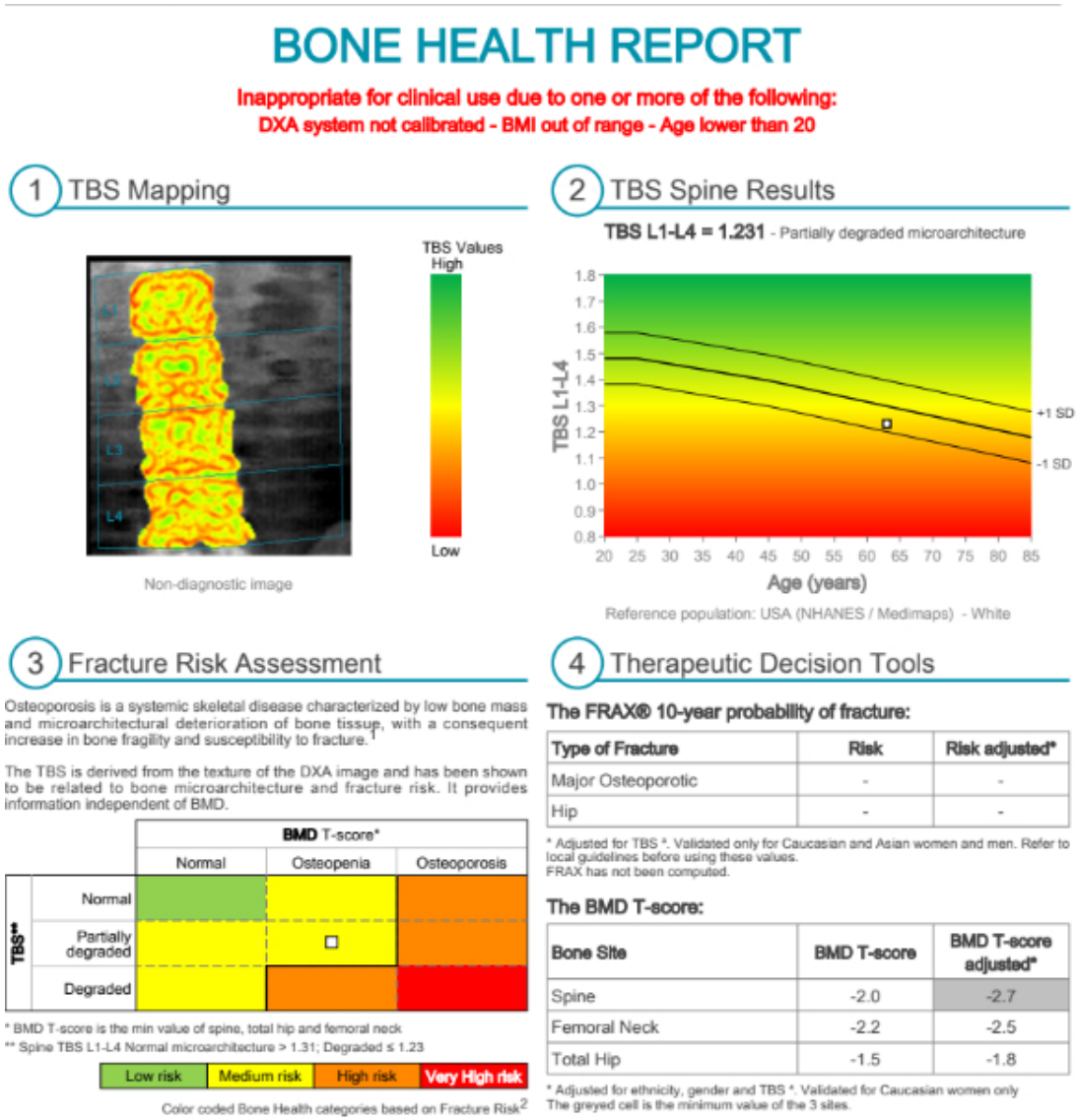
The BMD T-scores:

Bone Site	BMD T-score	BMD T-score adjusted*
Spine	-3.0	-2.7
Femoral Neck	-2.2	-2.5
Total Hip	-1.5	-1.8

* Adjusted for ethnicity, gender and TBS.† Validated for Caucasian women only
The grayed cell is the minimum value of the 3 sites

Trabecular Bone Score (TBS)

- Omurga DXA görüntülerinden üretilen doku tabanlı bir ölçümdür
- KMY'den bağımsız olarak kırık riskini tahmin etmeye yardımcı



BONE HEALTH REPORT

Inappropriate for clinical use due to one or more of the following:
DXA system not calibrated - BMI out of range - Age lower than 20

1 TBS Mapping

TBS Values
High
Low

Non-diagnostic image

2 TBS Spine Results

TBS L1-L4 = 1.231 - Partially degraded microarchitecture

TBS L1-L4
Age (years)
+1 SD
-1 SD

Reference population: USA (NHANES / Medimaps) - White

3 Fracture Risk Assessment

Osteoporosis is a systemic skeletal disease characterized by low bone mass and microarchitectural deterioration of bone tissue, with a consequent increase in bone fragility and susceptibility to fracture.[1]

The TBS is derived from the texture of the DXA image and has been shown to be related to bone microarchitecture and fracture risk. It provides information independent of BMD.

TBS** \ BMD T-score*	Normal	Osteopenia	Osteoporosis
Normal			
Partially degraded		□	
Degraded			

* BMD T-score is the min value of spine, total hip and femoral neck
** Spine TBS L1-L4 Normal microarchitecture > 1.31; Degraded ≤ 1.23

Low risk | Medium risk | High risk | Very High risk

Color coded Bone Health categories based on Fracture Risk[2]

4 Therapeutic Decision Tools

The FRAX® 10-year probability of fracture:

Type of Fracture	Risk	Risk adjusted*
Major Osteoporotic	-	-
Hip	-	-

* Adjusted for TBS *. Validated only for Caucasian and Asian women and men. Refer to local guidelines before using these values.
FRAX has not been computed.

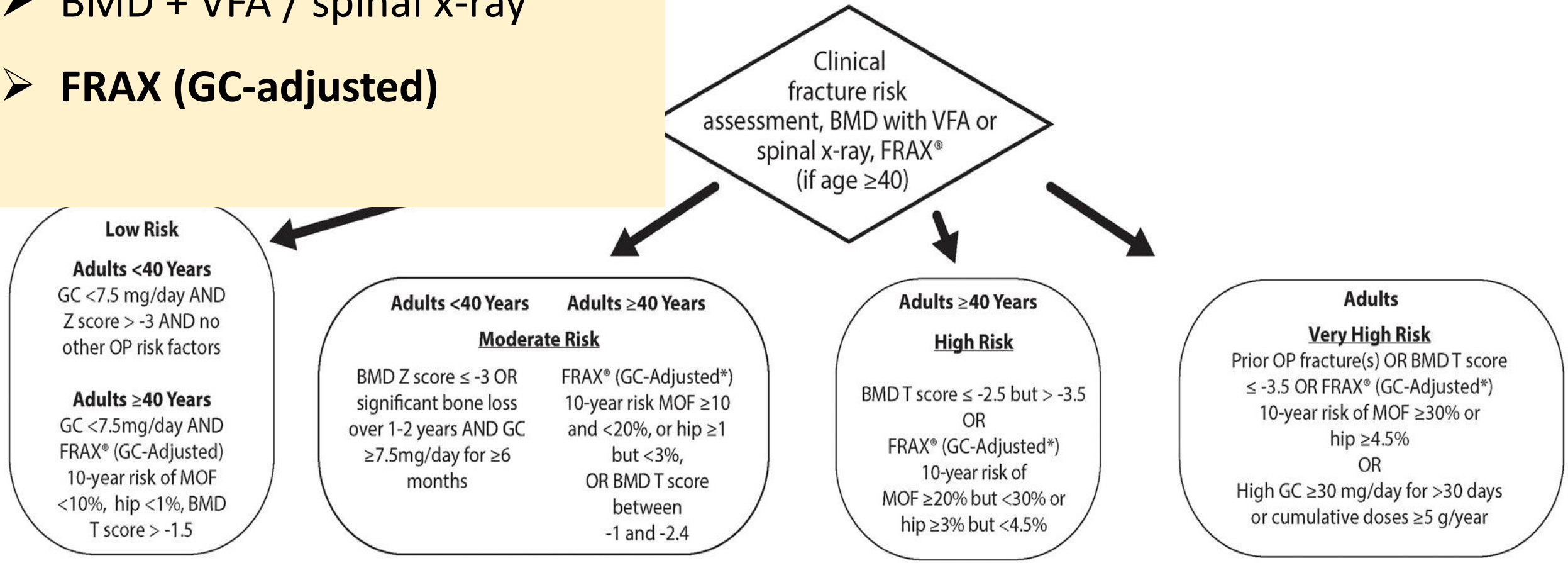
The BMD T-score:

Bone Site	BMD T-score	BMD T-score adjusted*
Spine	-2.0	-2.7
Femoral Neck	-2.2	-2.5
Total Hip	-1.5	-1.8

* Adjusted for ethnicity, gender and TBS *. Validated for Caucasian women only
The greyed cell is the minimum value of the 3 sites.

2022 American College of Rheumatology Guideline for the Prevention and Treatment of Glucocorticoid-Induced Osteoporosis

- BMD + VFA / spinal x-ray
- **FRAX (GC-adjusted)**



Hesaplama Aracı

Kemik mineral yoğunluğu (KMY) ile on yıllık kırık olasılığını hesaplamak için, lütfen aşağıdaki soruları cevaplayınız.

ülke: Türkiye Adı / ID: Risk faktörleri hakkında

Anket:

1. Yaş (40 ve 90 yaş arası) veya Doğum Tarihi
   Yaş: 63 Doğum tarihi: Y: 1961 A: 05 G: 19
2. Cinsiyet ○ Erkek ◉ Kadın
3. Vücut ağırlığı (kg) 56
4. Boy (cm) 160
5. Geçirilmiş kırık ◉ hayır ○ evet
6. Ebeveynde kalça kırığı ◉ hayır ○ evet
7. Mevcut sigara kullanımı ◉ hayır ○ evet
8. Glukokortikoidler ○ hayır ◉ evet
9. Romatoid artrit ○ hayır ◉ evet
10. Sekonder osteoporoz ◉ hayır ○ evet
11. Alkol tüketimi; günde 3 birim ve üstü ◉ hayır ○ evet
12. Femur boynu KMY (g/cm²)

Hologic 0.609

Temizle Hesapla

BMI: 21.9
The ten year probability of fracture (%)
with BMD

Major osteoporotic	12
Hip fracture	3.4

Eğer bir TBS değeri varsa, buraya tıklayınız: TBS ile ayarlayın

Vücut ağırlığı biriminin çevrilmesi

Pound → kg

Çevir

Boy ölçüm biriminin çevrilmesi

İnç → cm

Çevir

00431506
Individuals with fracture risk assessed since 1st June 2011

BMI: 21.9

The ten year probability of fracture (%)

with BMD

Major osteoporotic	12
Hip fracture	3.4

The 10 year probability of fracture (%)
Adjusted for TBS

Major Osteoporotic Fracture:	13
Hip Fracture:	3.7

Adjust your results with FRAX plus®

Adjust probability according to the dose of oral glucocorticoids

Adjusting conventional FRAX estimates of fracture probability according to corticosteroid dose (only available if glucocorticoids is yes).

- (●) High dose of corticosteroid ≥7.5 mg/day
- ( ) Dose above 2.5 and under 7.5 mg/day (no adjustment needed)
- ( ) Low dose of corticosteroid <2.5mg/day

Adjust probability

Hasta günlük 2.5-7.5 mg/gün arasında kullanıyorsa düzeltme yapmaya gerek yok

05.11.2024 11:09:24 Country : Turkey

FRAX® plus® Adjusted

Age	63	Currently Smoking	No	with BMD	
Sex	Female	Glucocorticoids	Yes	Machine	Hologic
Weight	56 kg	Rheumatoid arthritis	Yes	BMD value	0.61 g/cm²
Height	160 cm	Secondary osteoporosis	No	T-score	-2.1
Previous Fracture	No	Alcohol 3 or more units/day	No	BMI	21.88
Parent Fractured Hip	No				

THE TEN-YEAR PROBABILITY OF FRACTURE

Major osteoporotic	11.7%
Hip Fracture	3.35%

PROBABILITY ADJUSTED ACCORDING TO THE DOSE OF ORAL GLUCOCORTICOIDS

Glucocorticoids	High dose of corticosteroid ≥7.5 mg/day	
Adjusted Major osteoporotic		13.51%
Adjusted Hip Fracture		4.15%

05.11.2024 11:09:24 Country : Turkey

FRAX® plus® Adjusted

Age	63	Currently Smoking	No	with BMD	
Sex	Female	Glucocorticoids	Yes	Machine	Hologic
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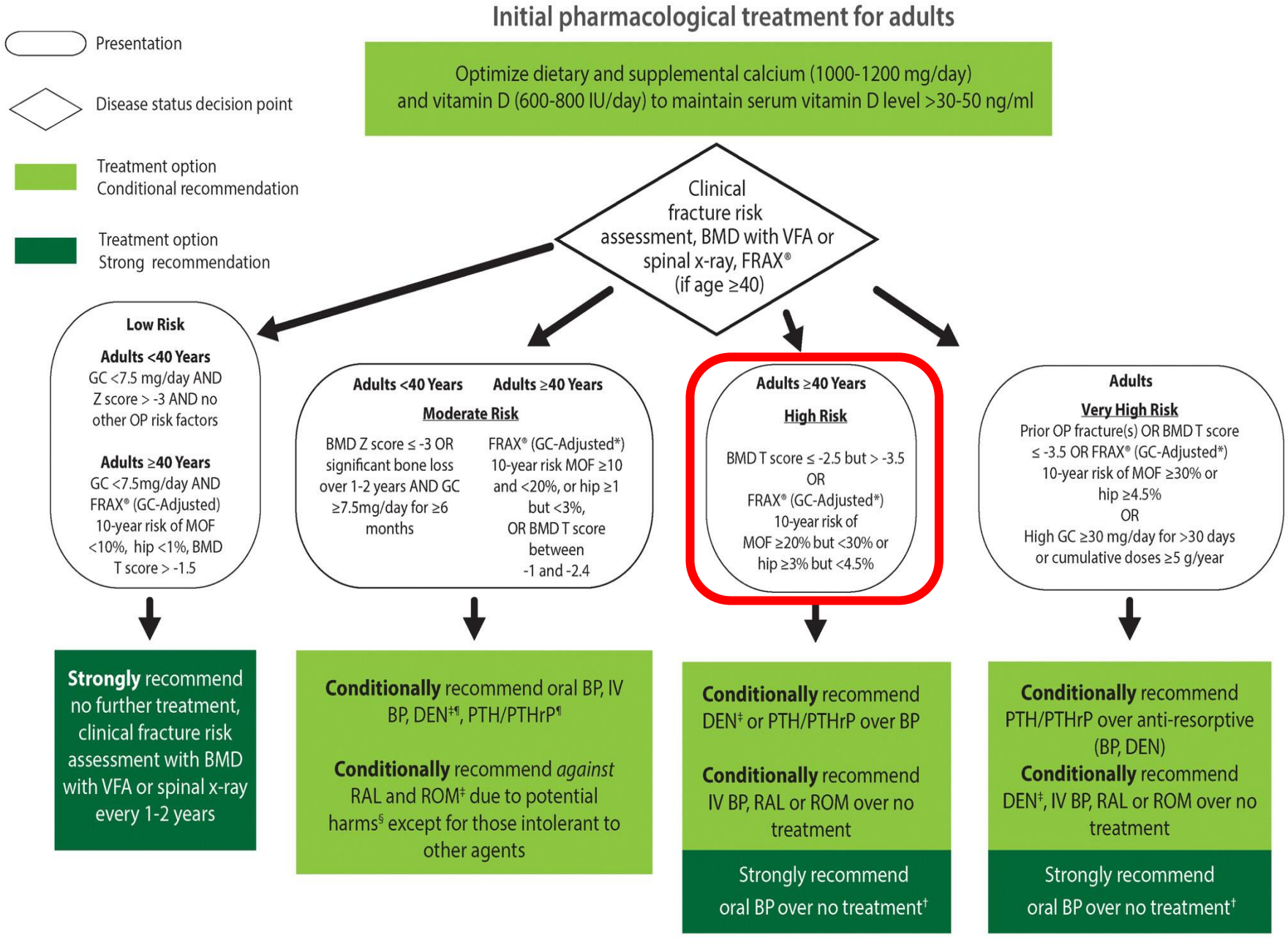


2022 American College of Rheumatology Guideline for the Prevention and Treatment of Glucocorticoid-Induced Osteoporosis

Denosumab ve bDMARD kombinasyonu enfeksiyon riskini arttırır mı?

J Rheumatol. 2018 Feb;45(2):170-176. doi: 10.3899/jrheum.161270. Epub 2017 Nov 15.

Occurrence of Serious Infection in Patients with Rheumatoid Arthritis Treated with Biologics and Denosumab Observed in a Clinical Setting

Table 3. Incidence of serious and opportunistic infections within the concurrent and biologic-alone groups.

Event Category Type	Concurrent, n = 102		Biologic Alone, n = 206	
	Event No./Total No. Patient-Yrs	Events per 100 Patient-Yrs (95% CI)	Event No./Total No. Patient-Yrs	Events per 100 Patient-Yrs (95% CI)
Serious infection requiring hospitalization				
Pneumonia	2/246	0.81 (0.10–2.94)	2/509	0.39 (0.05–1.42)
Bronchitis	—	—	1/509	0.20 (0.01–1.09)
Serious infection requiring ER visit and IV antibiotics				
Pneumonia	1/246	0.41 (0.01–2.26)	1/509	0.20 (0.01–1.09)
Opportunistic infection requiring hospitalization				
Pleural *Mycobacterium avium* complex	—	—	1/509	0.20 (0.01–1.09)
Serious infections	3/246	1.22 (0.25–3.56)	4/509	0.79 (0.21–2.01)
Serious or opportunistic infections (total)	3/246	1.22 (0.25–3.56)	5/509	0.98 (0.32–2.29)

Combination of denosumab and biologic DMARDs in inflammatory muscle-skeletal diseases and connective tissue diseases

Conclusion:

The combination of bDMARD and denosumab does not alter the efficacy and the safety profile of the bDMARD in patients with RMD/CTD. Future studies verifying the radiological disease inhibition could support denosumab use in RMD/CTD other than rheumatoid arthritis, when complicated by OP.

Eur J Rheumatol 2022 Jan 21;8(4):190

Denosumab ve RA

Anti-osteoporotik etkinliğinin yanında eklem hasarına karşı koruyucu ?

OPEN ACCESS

CLINICAL SCIENCE

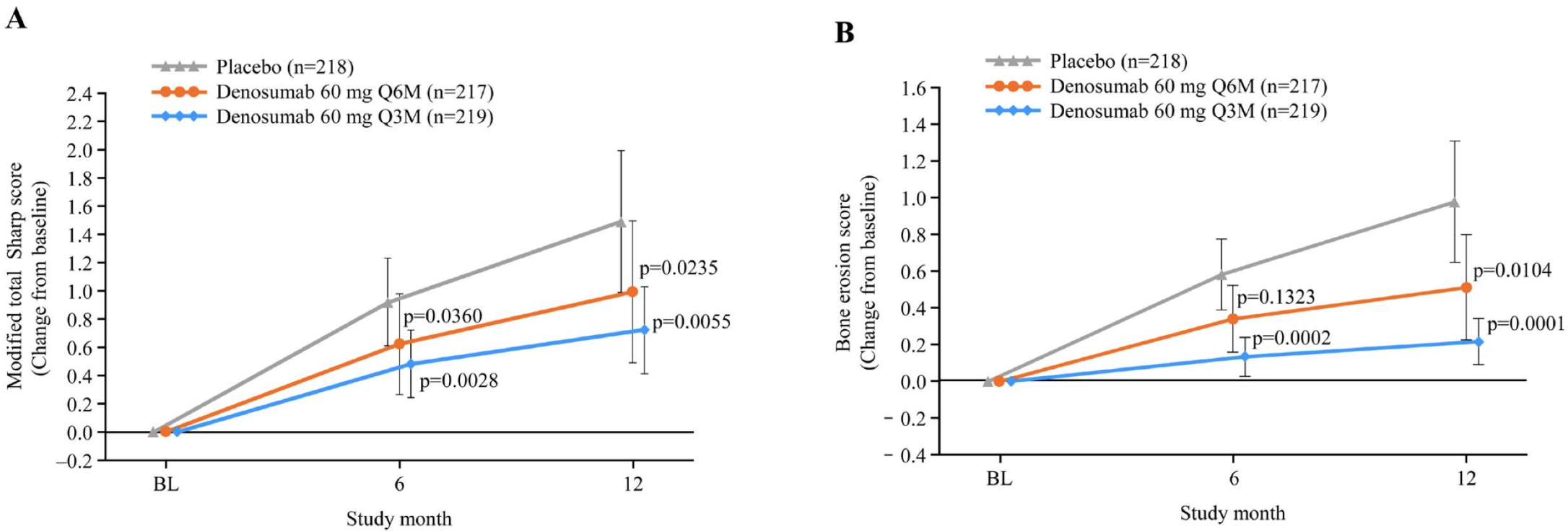
Effects of the anti-RANKL antibody denosumab on joint structural damage in patients with rheumatoid arthritis treated with conventional synthetic disease-modifying antirheumatic drugs (DESIRABLE study): a randomised, double-blind, placebo-controlled phase 3 trial

Tsutomu Takeuchi,[1] Yoshiya Tanaka,[2] Satoshi Soen,[3] Hisashi Yamanaka,[4] Toshiyuki Yoneda,[5] Sakae Tanaka,[6] Takaya Nitta,[7] Naoki Okubo,[8] Harry K Genant,[9] Désirée van der Heijde[10]

OPEN ACCESS

CLINICAL SCIENCE

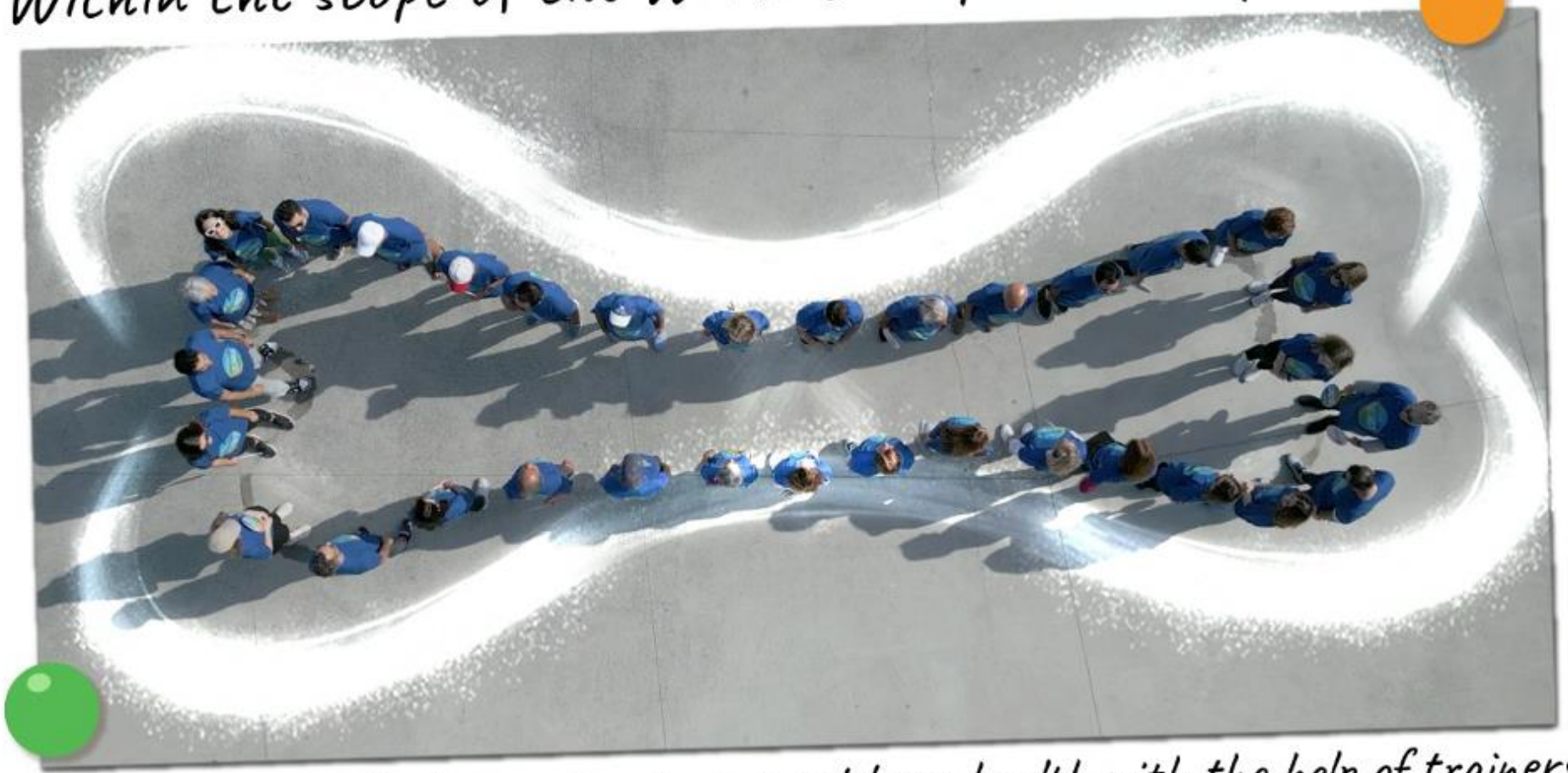
Effects of the anti-RANKL antibody denosumab on joint structural damage in patients with rheumatoid arthritis treated with conventional synthetic disease-modifying antirheumatic drugs (DESIRABLE study): a randomised, double-blind, placebo-controlled phase 3 trial



Osteoporosis: underrated, underdiagnosed and undertreated

Osteoporosis: underrated, underdiagnosed and undertreated

Within the scope of the World Osteoporosis Day,



we held an event to do exercises to support bone health with the help of trainers.


KEMİK SAĞLIĞIM
aklımda
osteoporoz
hasta olmayı

Teşekkürler

Osteoporosis International (2024) 35:25–40
https://doi.org/10.1007/s00198-023-06925-1

REVIEW

Bone density and fracture risk factors in ankylosing spondylitis: a meta-analysis

Fei Yan[1] · Linfeng Wu[1] · Juan Lang[1] · Zongju Huang[1]

Table 4 There are differences in fracture risk factors

Factors	Number	OR/WMD (95%CI)	I^2, $P_{heterogeneity}$	Test of OR = 1/ test of WMD = 0
Inflammatory bowel disease	3	0.46 (0.29, 0.74)	$I^2 = 0.0\%$, $P_{heterogeneity} = 0.753$	$z = 3.23$ $p = 0.001$
BASMI	3	−1.10 (−1.67, −0.53)	$I^2 = 0.0\%$, $P_{heterogeneity} = 0.360$	$z = 3.78$ $p = 0.000$
Finger-to-ground distance(cm)	3	−9.58 (−15.46, −3.70)	$I^2 = 56.7\%$, $P_{heterogeneity} = 0.100$	$z = 3.19$ $p = 0.001$
Total hip BMD (g/cm^2)	3	0.11 (0.08, 0.15)	$I^2 = 0.0\%$, $P_{heterogeneity} = 0.494$	$z = 6.72$ $p = 0.000$
total hip T-score	3	0.82 (0.52, 1.12)	$I^2 = 0.0\%$, $P_{heterogeneity} = 0.602$	$z = 5.30$ $p = 0.000$
BASRI	4	−2.04 (−2.53, −1.54)	$I^2 = 7.3\%$, $P_{heterogeneity} = 0.357$	$z = 8.02$ $p = 0.000$
Time of disease onset, year	4	−3.08 (−5.33, −0.84)	$I^2 = 25.5\%$, $P_{heterogeneity} = 0.261$	$z = 2.70$ $p = 0.007$
Pillow wall distance, cm	4	−4.35 (−8.27, −0.43)	$I^2 = 91.1\%$, $P_{heterogeneity} = 0.000$	$z = 2.17$ $p = 0.030$
Chest expansion, cm	4	0.73 (0.38, 1.07)	$I^2 = 44.6\%$, $P_{heterogeneity} = 0.144$	$z = 4.13$ $p = 0.000$
Femoral neck BMD (g/cm^2)	5	0.09 (0.06, 0.12)	$I^2 = 0.0\%$, $P_{heterogeneity} = 0.666$	$z = 6.09$ $p = 0.000$
BASFI	6	−0.78 (−1.24, −0.33)	$I^2 = 0.0\%$, $P_{heterogeneity} = 0.479$	$z = 3.36$ $p = 0.001$
CRP(mg/L)	6	0.73 (0.17, 1.29)	$I^2 = 46.6\%$, $P_{heterogeneity} = 0.095$	$z = 2.55$ $p = 0.011$
Lumbar vertebra BMD (g/cm^2)	7	0.04 (0.02, 0.06)	$I^2 = 25.8\%$, $P_{heterogeneity} = 0.232$	$z = 3.68$ $p = 0.000$
Course of disease,year	10	−2.75 (−4.69, −0.82)	$I^2 = 70.7\%$, $P_{heterogeneity} = 0.000$	$z = 2.79$ $p = 0.005$
Age, year	11	−4.42 (−5.78, −3.07)	$I^2 = 10.7\%$, $P_{heterogeneity} = 0.343$	$z = 6.40$ $p = 0.000$

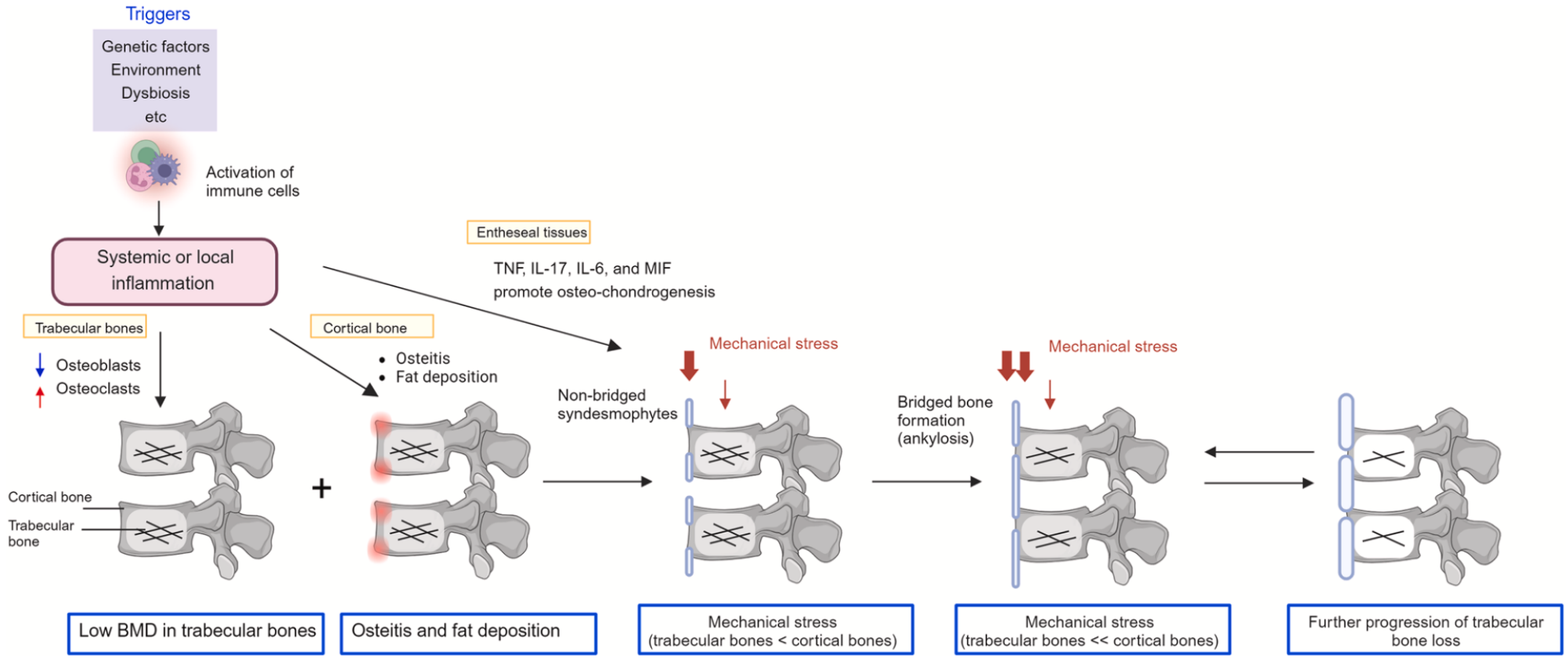


Fig. 1. Potential mechanisms of spinal trabecular bone loss and entheseal new bone formation in axSpA. Prior to the formation of bridged bone, a combination of systemic and local inflammation-induced low bone mineral density (BMD) in the trabecular bone, osteitis, and fat deposition at the edge of the cortical bone, along with type 3 immunity-driven enthesitis, contributes to the development of syndesmophytes. This phenomenon is attributed to increased mechanical loading on the cortical bone and entheseal lesions of the vertebrae. Upon completion of bone bridging, a reduction in mechanical loading on the trabecular bone occurs, leading to progressive bone loss. This perpetuates a vicious cycle, further promoting the bridging of the cortical bone. *BMD*, bone mineral density; *MIF*, macrophage migration inhibitory factor; TNF, tumor necrosis factor.

- While DXA is primarily used to measure BMD, TBS is a complementary
- technique that provides information about the quality of
- trabecular bone. TBS utilizes the same DXA scan data but analyzes the
- grayscale variations within the bone to evaluate the microarchitecture
- of the trabecular bone. It produces a numerical index that reflects the
- trabecular bone texture, with higher values indicating better trabecular
- bone microarchitecture and texture. Moreover, the addition of TBS to
- existing fracture prediction models, such as FRAX (Fracture Risk
- Assessment Tool), has the potential to enhance the predictive accuracy
- of such models [11

Clinical Rheumatology (2024) 43:1421–1433

- Rheumatoid arthritis (RA) and osteoporosis are two chronic disorders that are often seen together. RA is an autoimmune disorder that causes pain and inflammation in the joints, while osteoporosis is a disorder in which the bones become weak and fragile. Risk factors for bone loss in RA include disease activity, longer disease duration, erosive disease, autoantibody positivity, and joint damage leading to impaired physical activity. Recent research has shown that there is a complex interplay between immune cells, cytokines, and bone remodeling processes in both RA and osteoporosis. The bone remodeling process is regulated by cytokines and immune system signaling pathways, with osteoclasts activated through the RANK/RANKL/OPG pathway and the Wnt/DKK1/sclerostin pathway. Understanding these mechanisms can aid in developing targeted thera- pies for treatment of osteoporosis in RA patients. Current pharmacological approaches include anti-osteoporotic drugs such as bisphosphonates, denosumab, teriparatide, abaloparatide, raloxifene, and romosozumab. Conventional disease-modifying antirheumatic drugs such as methotrexate and biologicals including TNF inhibitors, IL-6 inhibitors, rituximab, and abatacept lower disease activity in RA and can improve bone metabolism by reducing inflammation but have limited impact on bone mineral density. This review will shed light on the relationship between osteoporosis and rheumatoid arthritis as well as the various factors that influence the onset of osteoporosis in RA patients. We also explore several treatment approaches to effectively managing osteoporosis in RA patients.

- Treatment for bone loss in axSpA
- The management of low BMD in axSpA patients has not received
- significant attention in routine practice, largely due to the underestimation
- of its prevalence and the absence of regular BMD assessments. In
- addition, despite the higher prevalence of bone loss in axSpA compared
- to the general population, current management guidelines recommended
- by ASAS-EULAR (Assessment of SpondyloArthritis International
- Society-European League Against Rheumatism) and ACR
- (American Colleague of Rheumatology) lack specific information about
- treatments for osteoporosis in axSpA patients [120,121]. Considering
- that axSpA patients face an elevated risk of both vertebral and
- non-vertebral fractures, coupled with low vitamin D levels [101], it is
- imperative to incorporate prophylactic and therapeutic interventions for
- bone loss into standard practice for axSpA patients.