Erken Romatoid Artrit

5N1K

Ahmet Kıvanç Cengiz

19 Mayıs Üniversitesi

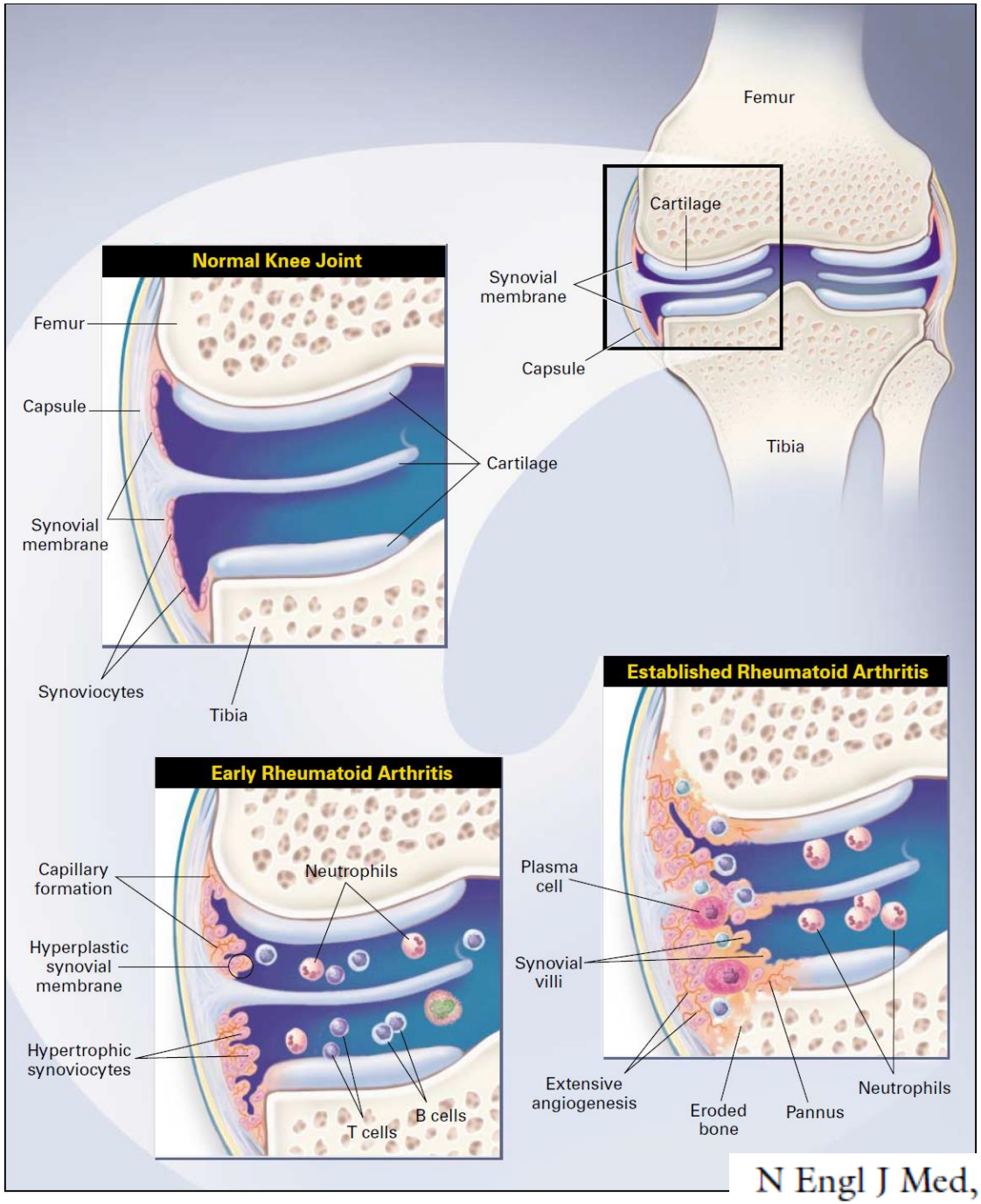
Fiziksel Tıp ve Rehabilitasyon AD

Romatoloji BD


Cüneyt Özdemir
5N1K


TAFF PICTURES SUNAR
UĞUR YÜCEL
BİNNUR KAYA
CENGİZ BOZKURT
FEYYAZ YİĞİT
MERT DENİZMEN
MEHMET ÖZGÜR
'ÖLÜMLÜ DÜNYA'NIN YARATICI EKİBİNDEN
CİNAYET SÜSÜ
YÖNETMEN ALİ ATAY
25 EKİM'DE SİNEMALARDA
CJ ENTERTAINMENT
POWER TÜRK
TAFF PICTURES


KAÇTA?
HANGİ?
NE İLE?
NİÇİN?
N'OLMUŞ?
KİMİ?
NERDE?
NASIL?
NE ZAMAN?
CİNAYET SÜSÜ
KİMDEN?
NEYİ?
NE BELLİ?
NEYE?
KİM?



N Engl J Med, Vol. 344, No. 12 · March 22, 2001

Commentary

Pathogenesis of rheumatoid arthritis: how early is early?

Gary S Firestein

Erken RA: ilk 2 yıl? 6 hafta?

Tetikleyici?➔doğal imm. Sis. ➔adaptif imm. Sis.

Th1 ağırlıklı ama IL4, IL13 gibi Th2 sitokinleri de çok

Özellikle ilk 3 ay T hücre sitokinleri ağırlıklı

Arthritis Research & Therapy August 2005 Vol 7 No 4

REDUCTION OF SYNOVIAL INFLAMMATION AFTER ANTI-CD4 MONOCLONAL ANTIBODY TREATMENT IN EARLY RHEUMATOID ARTHRITIS

PAUL P. TAK, PETER A. VAN DER LUBBE, ALBERTO CAULI, MOHAMED R. DAHA, TOM J. M. SMEETS, PHILIP M. KLUIN, A. EDO MEINDERS, GHADA YANNI, GABRIEL S. PANAYI, and FERDINAND C. BREEDVELD

8 erken RA hastası (18 aydan az)
Anti-CD4
Sinoviyal biyopsi
T hücre sayısı belirgin azalmış
TNF, IL-1 yüksek
Klinik rahatlama yok

ARTHRITIS & RHEUMATISM
Vol. 38, No. 10, October 1995, pp 1457–1465

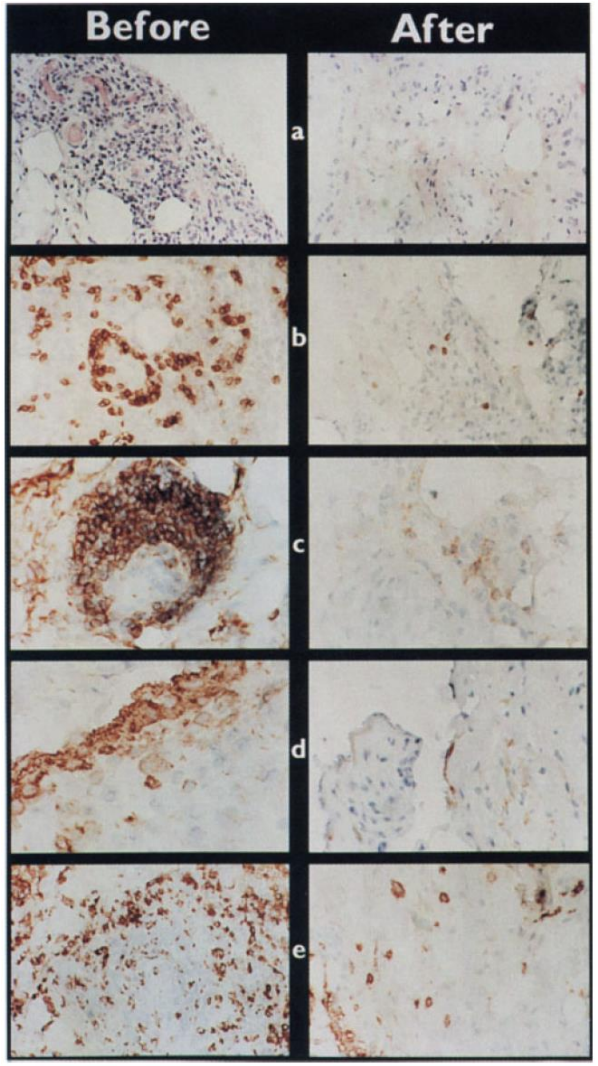
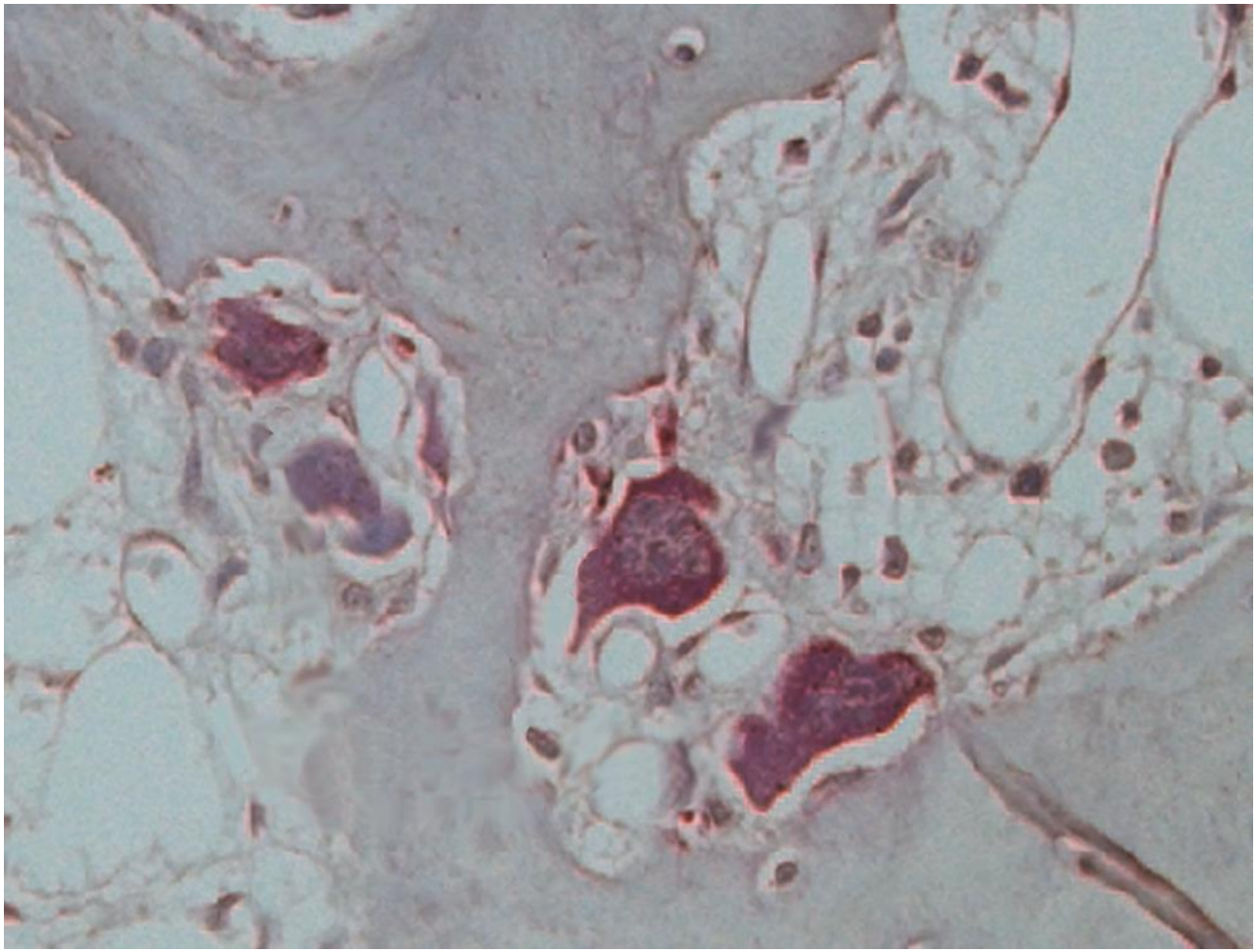
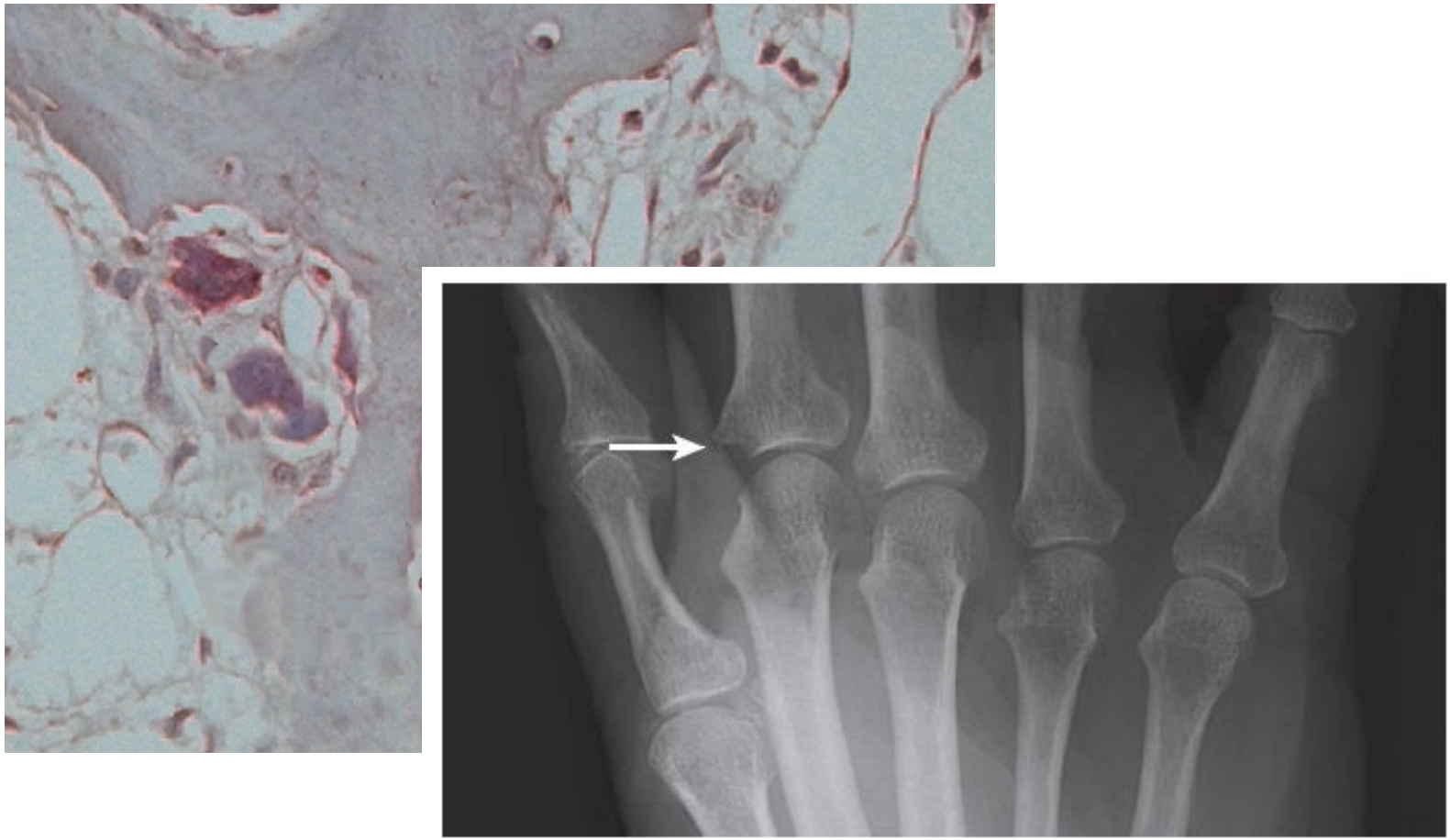
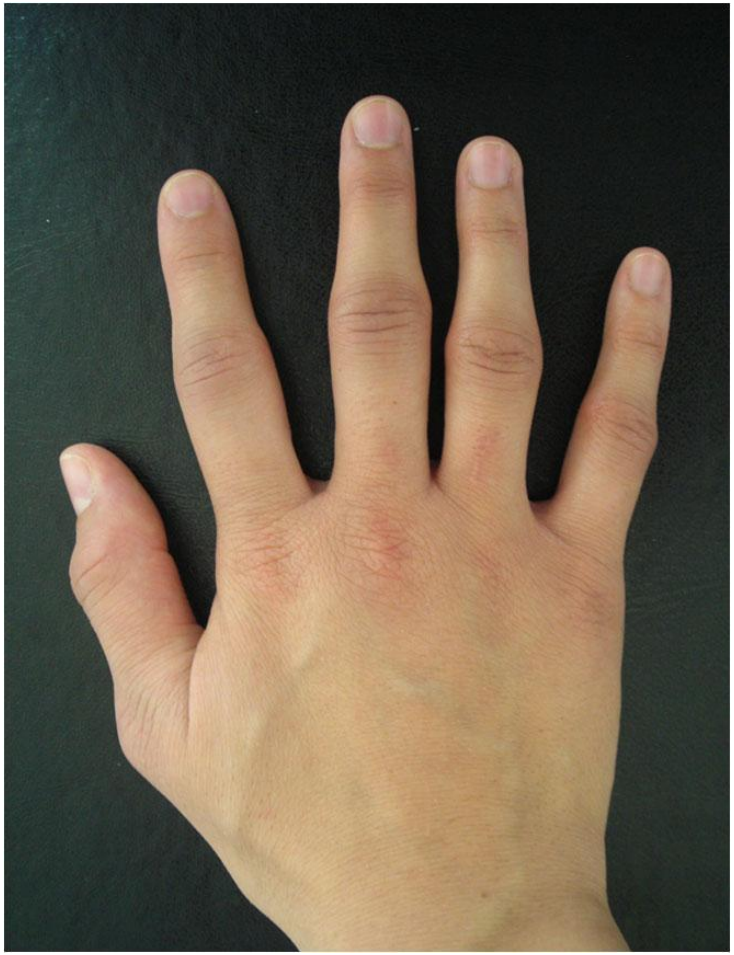


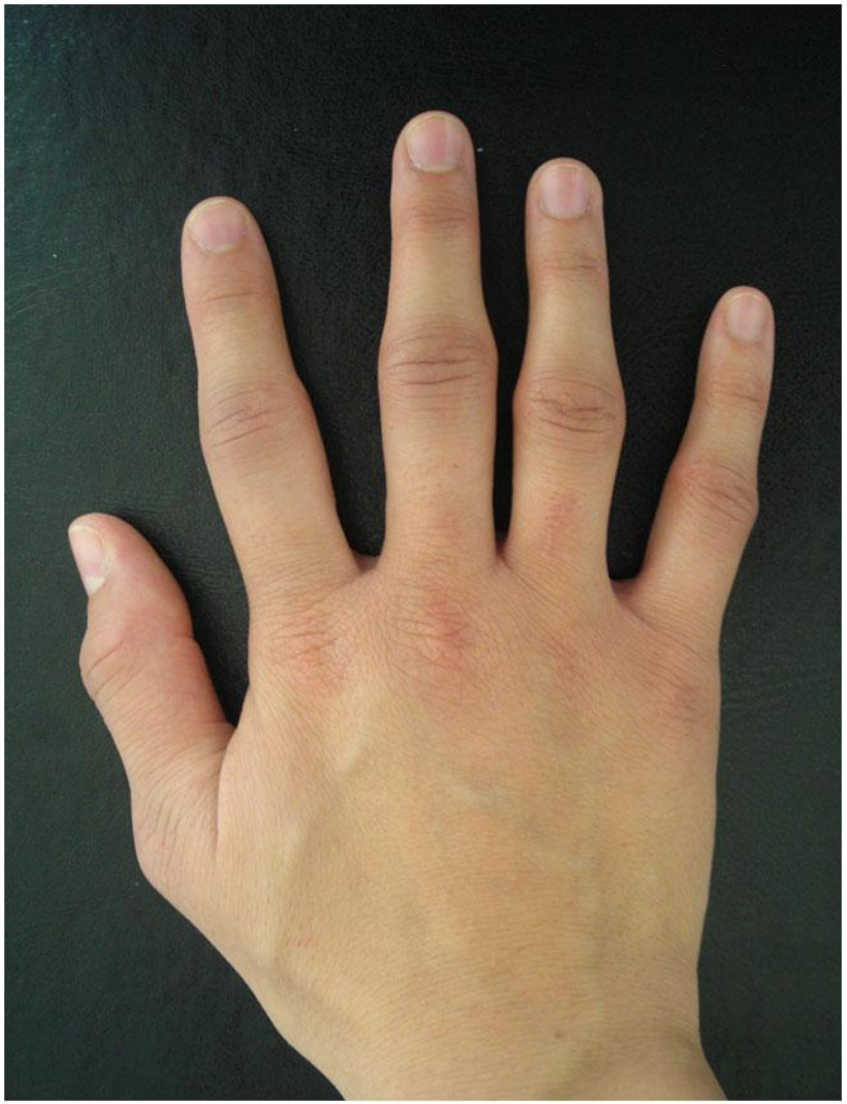
Figure 1. Rheumatoid synovial tissue obtained from patient 6 before and 4 weeks after anti-CD4 therapy, showing a decrease in inflammation (**a**) (hematoxylin and eosin stained) and in infiltration with CD3+ T cells (**b**), CD4+ cells (**c**), Mab67+ type B synoviocytes (**d**), and CD68+ macrophages (**e**) after therapy. (Original magnification × 250 in **a**, **b**, and **e**; × 400 in **c** and **d**.)

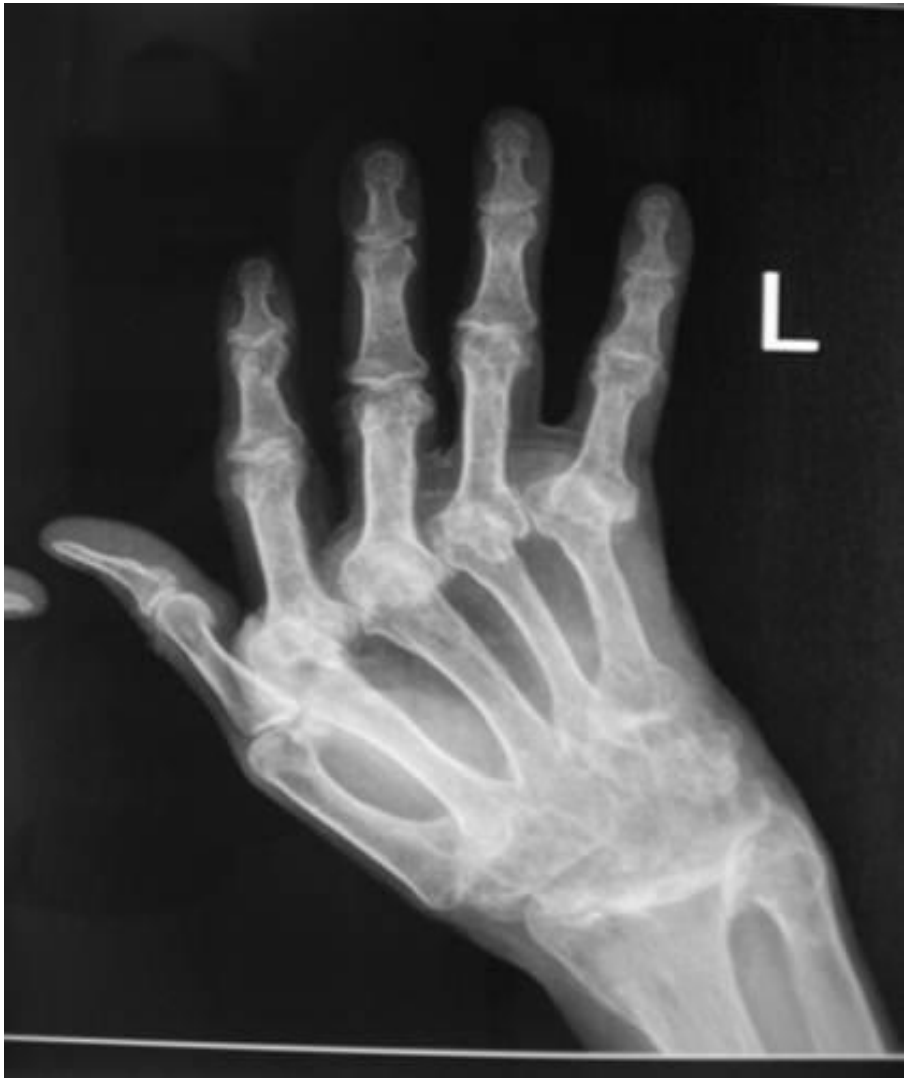




• **Fig. 76.2** Typical rheumatoid arthritis erosion *(arrow)* along the periarticular area where the synovium wraps back on itself, the so-called *bare area*. (Courtesy Dr. Gerald Moore.)



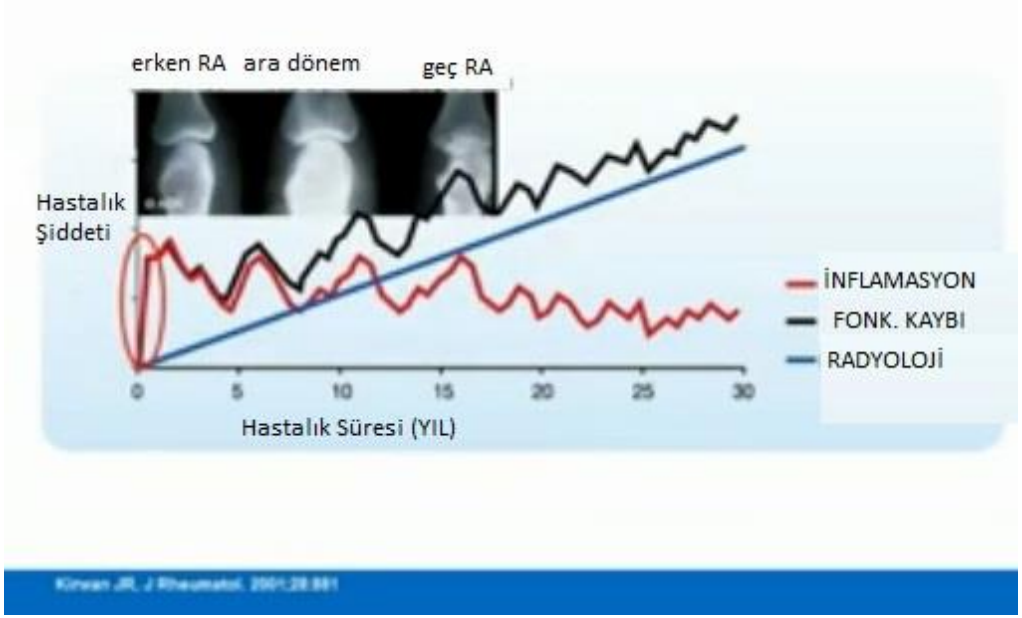



L

Links Between Radiological Change, Disability, and Pathology in Rheumatoid Arthritis

JOHN R. KIRWAN

Romatoid artritte hastalık aktivitesi,radyolojik hasar ve fonksiyon kaybı arasındaki ilişki iyi bilinmektedir



•Hastalık aktivitesinin erken dönemde baskılanması radyolojik hasarı ve dolayısıyla fonksiyon kaybının önler

•Bu nedenle hastalara erken tanı konması ve tedavinin erken dönemde başlanması önemlidir

Early Rheumatoid Arthritis — Is There a Window of Opportunity?

JOHN J. CUSH

The Journal of Rheumatology 2007; 34 Suppl 80

Table 1. Subanalyses comparing response rates with TNF inhibitors used in patients with early versus established RA.

		Early RA Patients			Overall RA Population (Established + Early Patients)		
Study	Agent	Disease Duration, yrs	N	ACR 20 Responses, %	Disease Duration, yrs	N	ACR 20 Responses, %
DE019[19]	Adalimumab	< 2	55	70	≥ 2	363	62
TEMPO[20,21]	Etanercept	≤ 3	77	77.9		503	75
ATTRACT[22]	Infliximab*	< 3	82	37	> 3	428	42

* 3mg/kg q 8 wks.

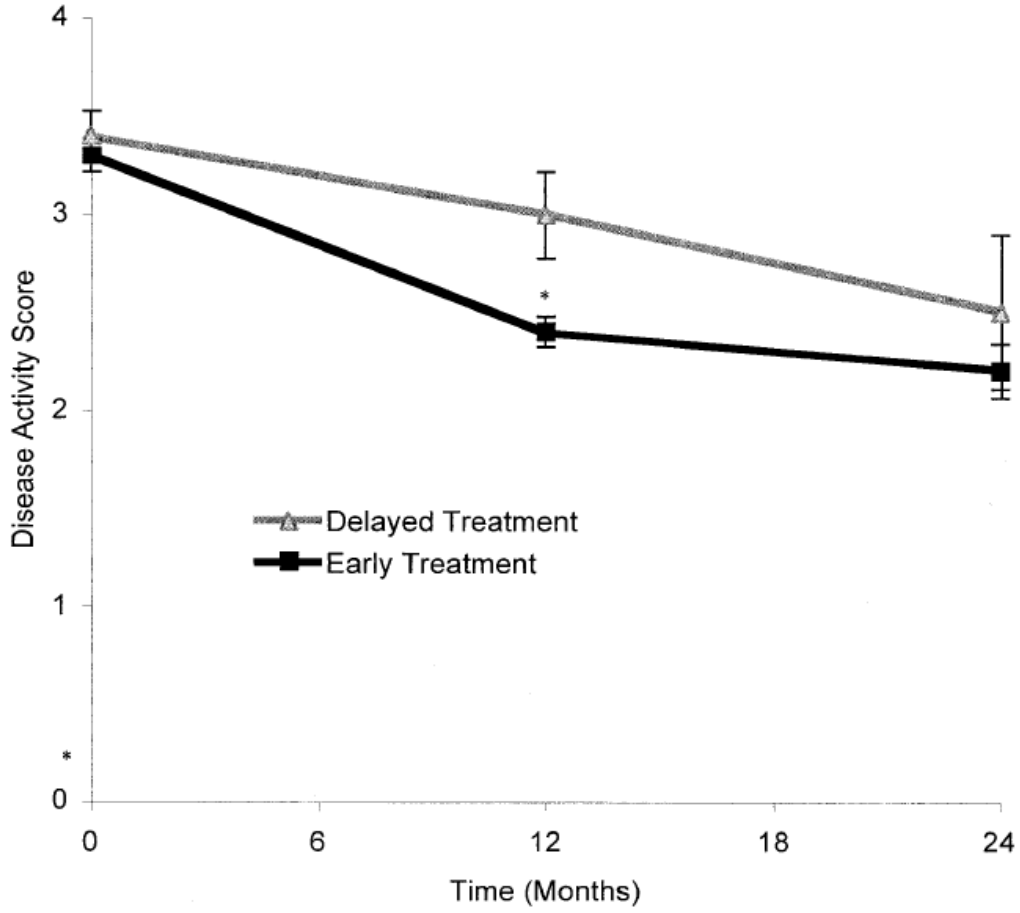
Early versus Delayed Treatment in Patients with Recent-onset Rheumatoid Arthritis: Comparison of Two Cohorts Who Received Different Treatment Strategies

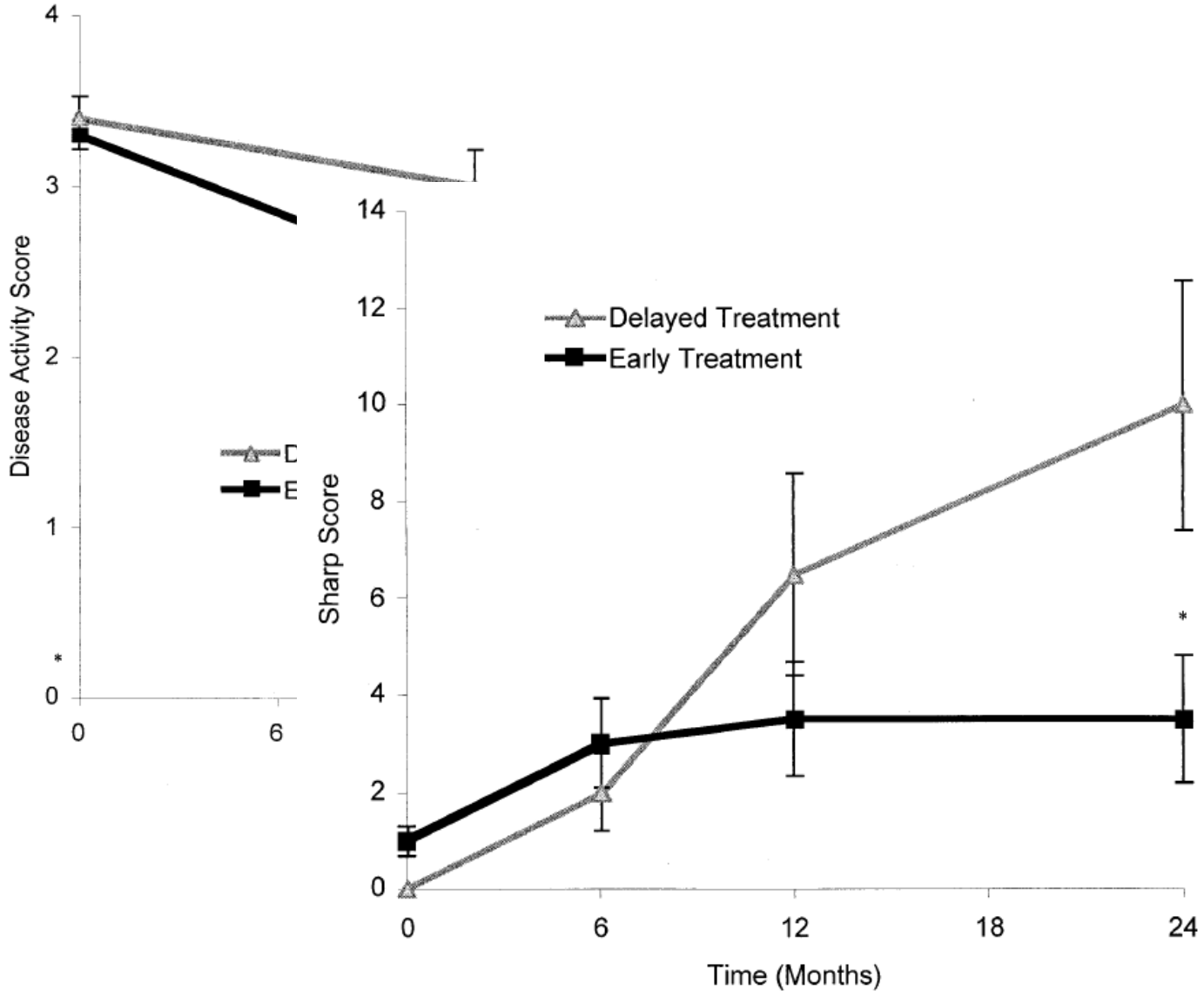
Leroy R. Lard, MD, Henk Visser, MD, Irene Speyer, MD, Irene E. vander Horst-Bruinsma, MD, Aeilko H. Zwinderman, PhD, Ferdinand C. Breedveld, MD, Johanna M.W. Hazes, MD, PhD

Table 2. Characteristics of the Initial Disease-modifying Drug Treatment

Treatment	Delayed Group (n = 109)	Early Group (n = 97)
	Number (%)	
Chloroquine	35 (32)	46 (47)
Salazopyrine	21 (19)	51 (53)
No disease-modifying drug*	42 (39)	0 (0)
Other†	11 (10)	0 (0)

1993-1995
Tanı sonrası ilk 15 günde DMARD başlananlar
Ortalama 123 gün gecikme ile DMARD başlananlar

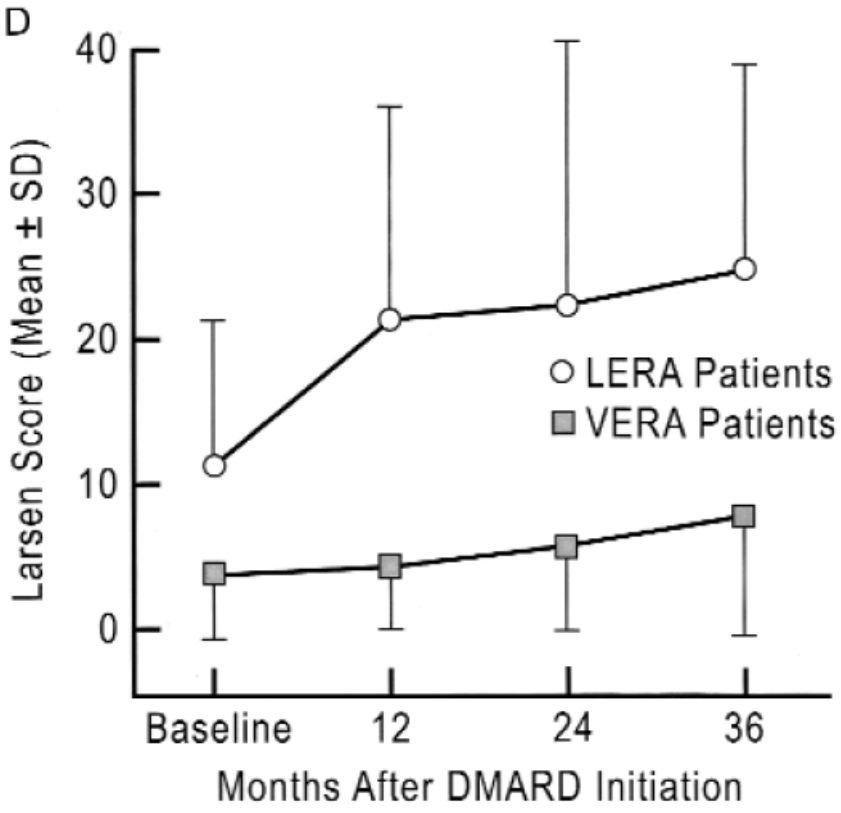

4
3
2
1
0
*
*
Disease Activity Score
Delayed Treatment
Early Treatment
0
6
12
18
24
Time (Months)


Disease Activity Score
4
3
2
1
0
*
0
6
D
E
Sharp Score
14
12
10
8
6
4
2
0
Delayed Treatment
Early Treatment
*
0
6
12
18
24
Time (Months)

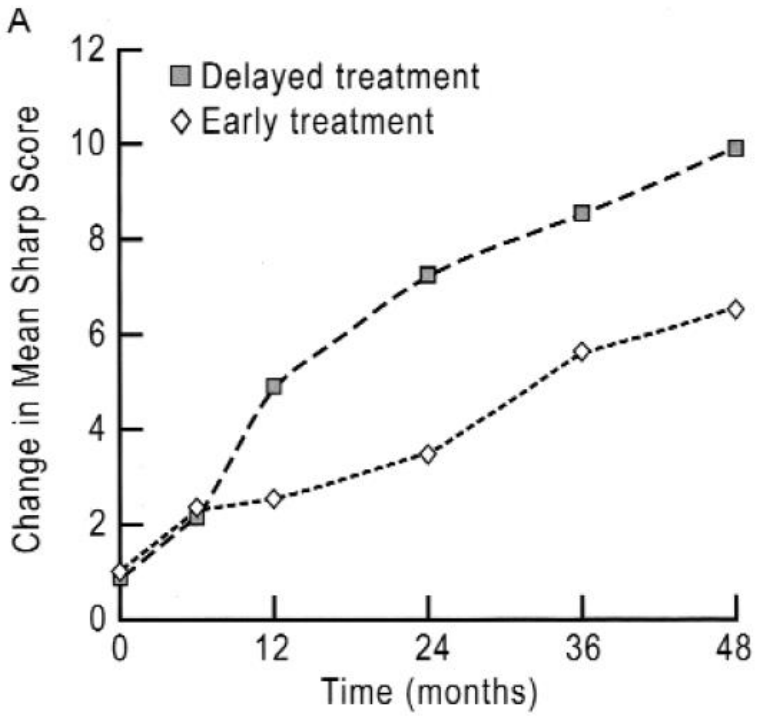
Early Rheumatoid Arthritis: A Medical Emergency?

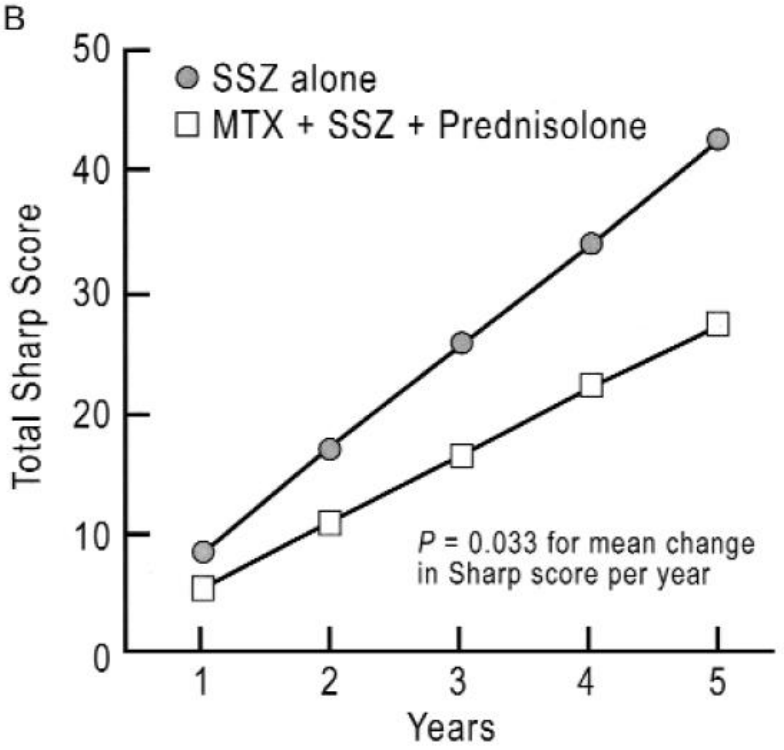
Larry W. Moreland, MD, S. Louis Bridges, Jr, MD, PhD

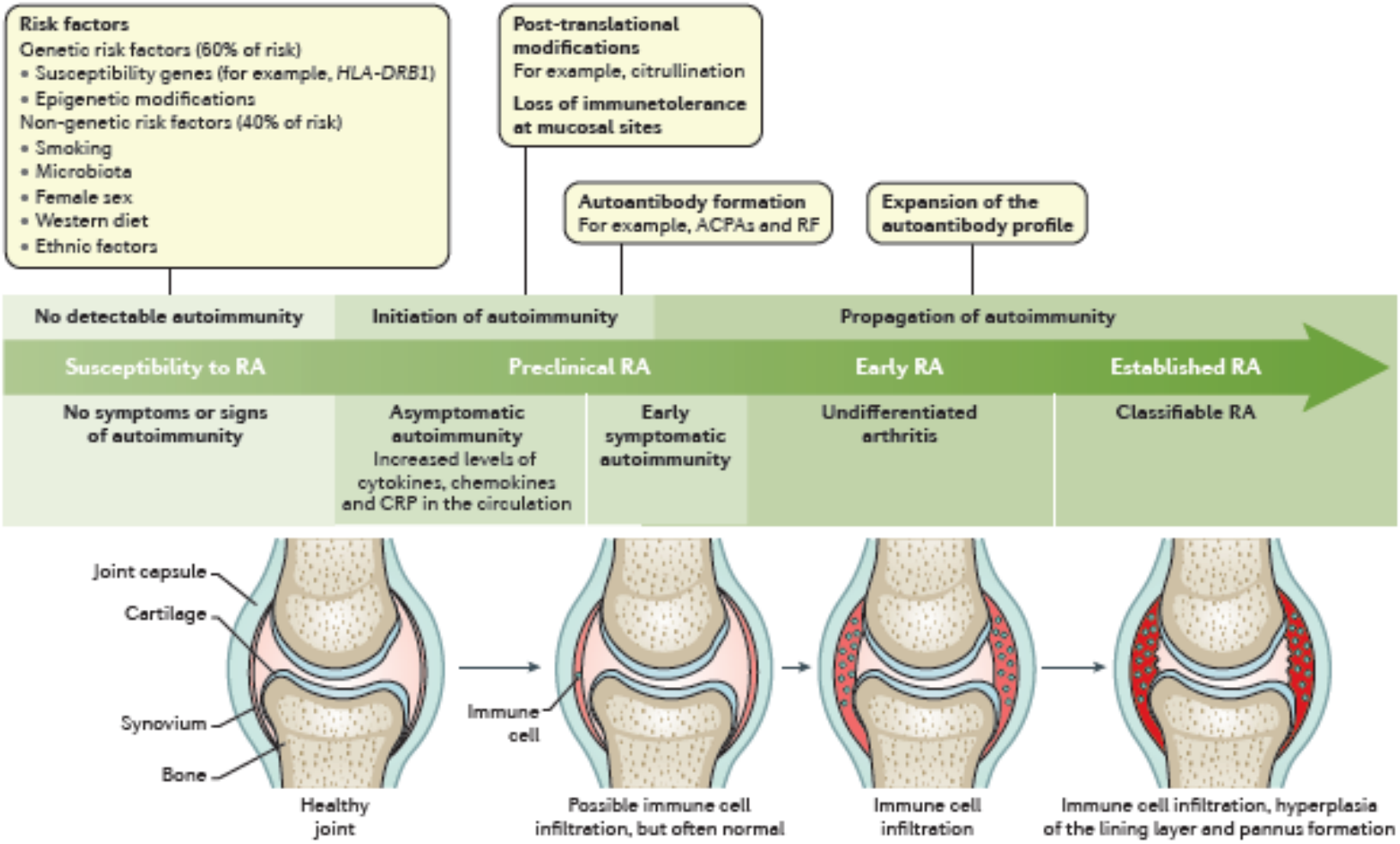
In this issue of *The American Journal of Medicine*, Lard et al. (1) report that early treatment (within 2 weeks of diagnosis) of patients with rheumatoid arthritis provides better outcomes at 2 years of disease than does delayed treatment, and they conclude that both patients and physicians should consider early-onset rheumatoid arthritis as a medical emergency. Their study corroborates previous reports on the benefit of early management of the disease (2–4).



VERA: ilk 3 ay
LERA: 9ay-3,5 yıl

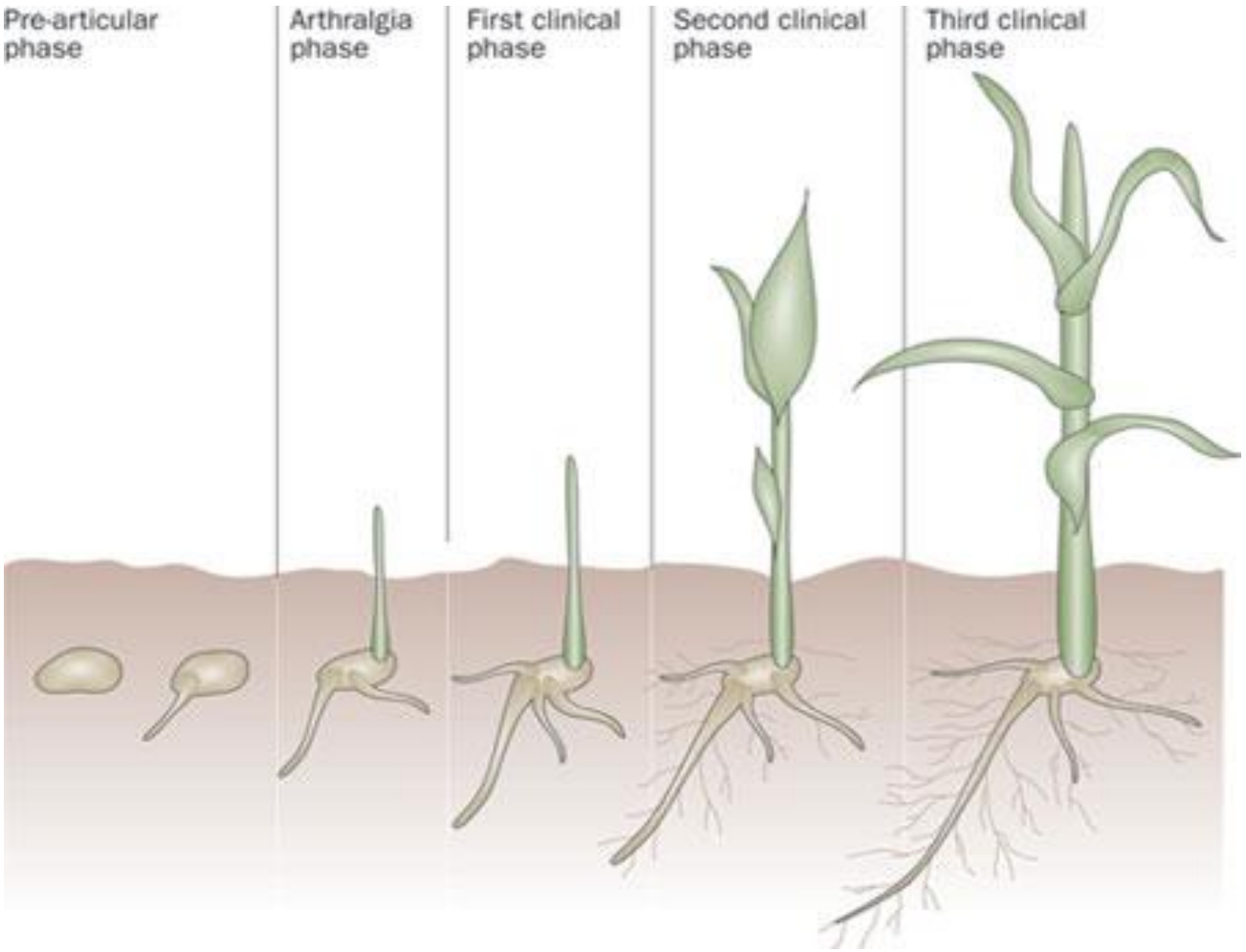

A
Change in Mean Sharp Score
Delayed treatment
Early treatment
0
2
4
6
8
10
12
0
12
24
36
48
Time (months)


B
Total Sharp Score
SSZ alone
MTX + SSZ + Prednisolone
P = 0.033 for mean change in Sharp score per year
0
10
20
30
40
50
1
2
3
4
5
Years


Risk factors
Genetic risk factors (60% of risk)
• Susceptibility genes (for example, HLA-DRB1)
• Epigenetic modifications
Non-genetic risk factors (40% of risk)
• Smoking
• Microbiota
• Female sex
• Western diet
• Ethnic factors
Post-translational modifications
For example, citrullination
Loss of immunetolerance at mucosal sites
Autoantibody formation
For example, ACPAs and RF
Expansion of the autoantibody profile
No detectable autoimmunity
Initiation of autoimmunity
Propagation of autoimmunity
Susceptibility to RA
Preclinical RA
Early RA
Established RA
No symptoms or signs of autoimmunity
Asymptomatic autoimmunity
Increased levels of cytokines, chemokines and CRP in the circulation
Early symptomatic autoimmunity
Undifferentiated arthritis
Classifiable RA
Joint capsule
Cartilage
Synovium
Bone
Immune cell
Healthy joint
Possible immune cell infiltration, but often normal
Immune cell infiltration
Immune cell infiltration, hyperplasia of the lining layer and pannus formation

The epidemiology of early inflammatory arthritis.

Hazes JM[1], Luime JJ.



Nature Reviews Rheumatology **7**, 381-390 (July 2011) | doi:10.1038/nrrheum.2011.78

Hastalık nerede başlıyor?

- Uzun preklinik evre boyunca hiç eklem bulgusu yok
- Görüntüleme çalışmaları, sinovial biyopsilerde saptanan sinovit yok
- Ekstra-artiküler başlangıç
- IgA tipi otoantikorlar, akciğer bulguları ile otoantikorların ilişkisi, periodontal hastalıklarla otoantikorların ilişkisi…

Published in final edited form as:
Arthritis Rheum. 2012 June ; 64(6): 1756–1761. doi:10.1002/art.34344.

Airways abnormalities and rheumatoid arthritis-related autoantibodies in subjects without arthritis: early injury or initiating site of autoimmunity?

M. Kristen Demoruelle[1], Michael H. Weisman[2], Philip L. Simonian[3], David A. Lynch[4], Peter B. Sachs[5], Isabel F. Pedraza[6], Annie R. Harrington[6], Jason R. Kolfenbach[1], Christopher C. Striebich[1], Quyen N. Pham[7], Colin D. Strickland[5], Brian D. Petersen[5], Mark C. Parish[1], Lezlie A. Derber[1], Jill M. Norris[8], V. Michael Holers[1], and Kevin D. Deane[1]

İnflamatuvar artriti olmayan fakat RF, Anti-CCP pozitifliği saptanan preklinik evredeki 42 birey ve 12 sağlıklı seronegatif birey YRBT
Bronşial kalınlaşmalar, Sentrilobüler opasiteler sık
'Akciğerler otoimmünite ilişkili hasarın erken hedeflerinden olabilir'

- Sigara, Peptidylarginine Deiminase (PAD) uyarımı ile sitrilünizasyonu arttırıyor
- Periodontal hastalıklar, Porphyromonas Gingivalis enfeksiyonları ile RA ilişkisi
- Başlangıç yeri mukozal yüzeyler olabilir: Akciğerler, oral mukoza, GIS, GÜS ?

Autoimmunity Reviews 20 (2021) 102797

Contents lists available at ScienceDirect

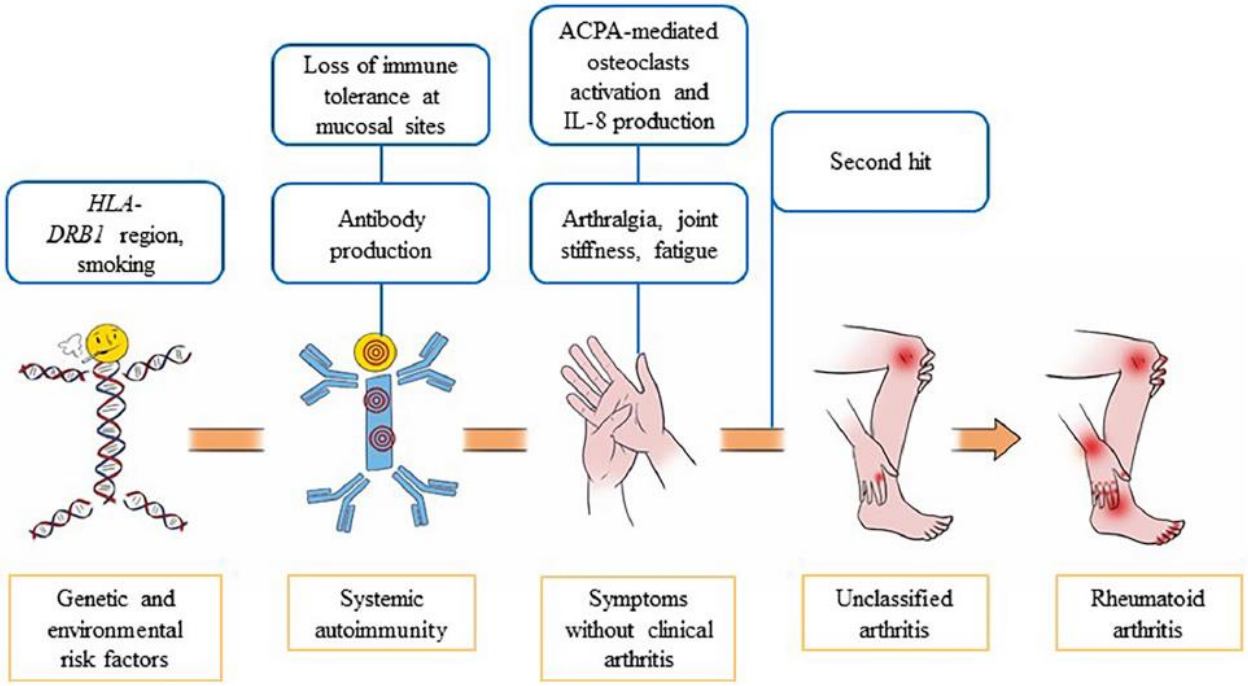
Autoimmunity Reviews

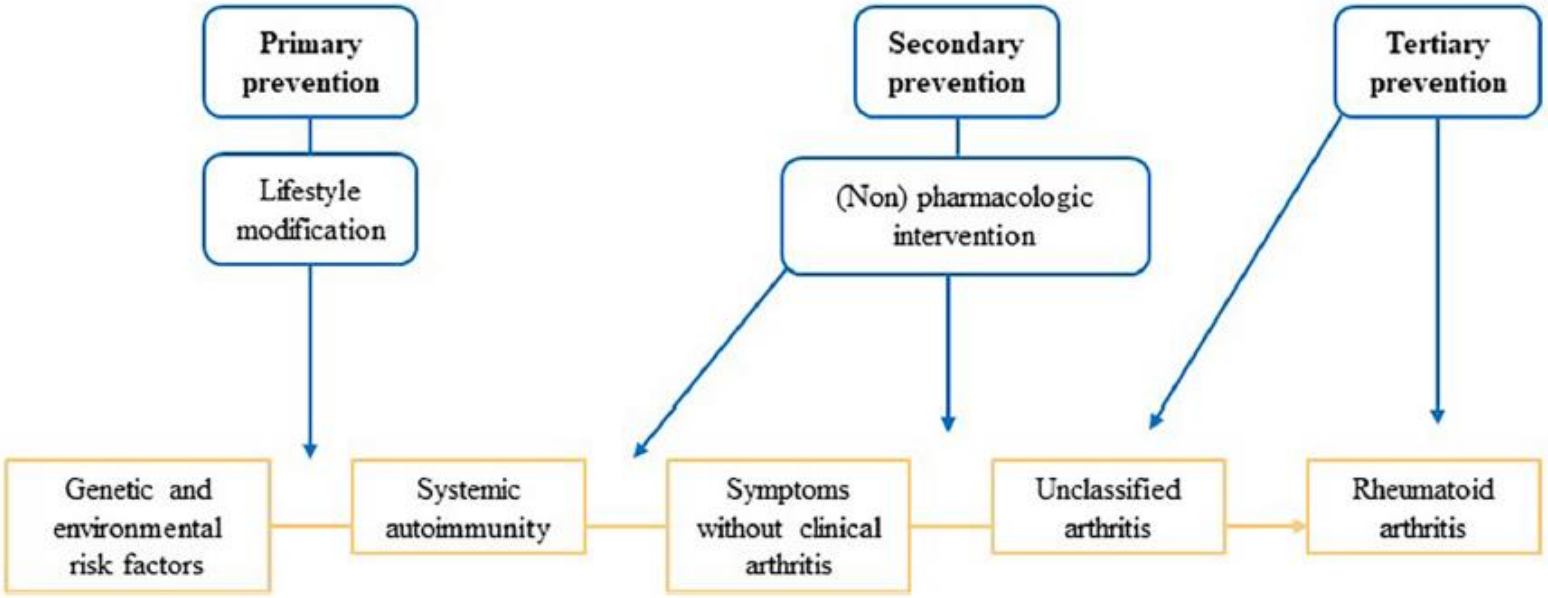
journal homepage: www.elsevier.com/locate/autrev

Review

The pre-clinical phase of rheumatoid arthritis: From risk factors to prevention of arthritis

Nora Petrovská *, Klára Prajzlerová, Jiří Vencovský, Ladislav Šenolt, Mária Filková




Primary prevention
Lifestyle modification
Secondary prevention
(Non) pharmacologic intervention
Tertiary prevention
Genetic and environmental risk factors
Systemic autoimmunity
Symptoms without clinical arthritis
Unclassified arthritis
Rheumatoid arthritis

Yağ Dokusu

- VKİ 25’in üstü: RA riski %15 artıyor
- VKİ 30’un üstü: risk artışı %21-31
- Obesite+ sigara: risk artışı %60’larda

Table 1
Food associated with risk of rheumatoid arthritis.

Food		
General	**protective effect**	**increasing the risk of RA**
	moderate alcohol intake	red meat
	fruit and vitamin C	sugar-sweetened soda
	olive oil	
	cooked vegetable	
	mushrooms	
	beans	
	poultry	
	dairy products	

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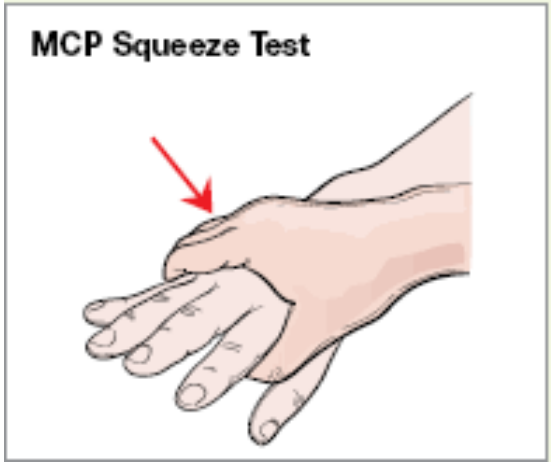
Review

The pre-clinical phase of rheumatoid arthritis: From risk factors to prevention of arthritis

Nora Petrovská *, Klára Prajzlerová, Jiří Vencovský, Ladislav Šenolt, Mária Filková

Kimlere dikkat etmeli:

- Son 1 yılda başlayan artralji
- MKF eklemler
- Sabah tutukluğu
- Sabahları yumruk yapmakta zorlanmak
- MKF sıkma testi+
- Aile öyküsü+



İlginiz için teşekkürler…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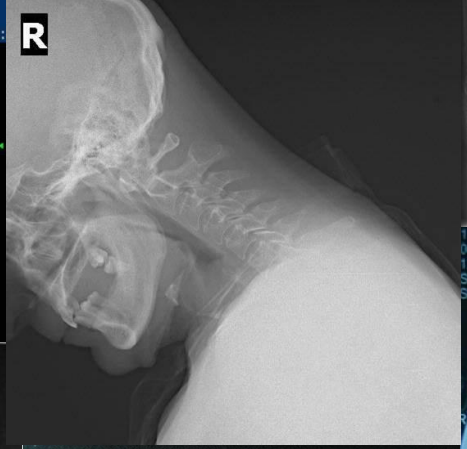
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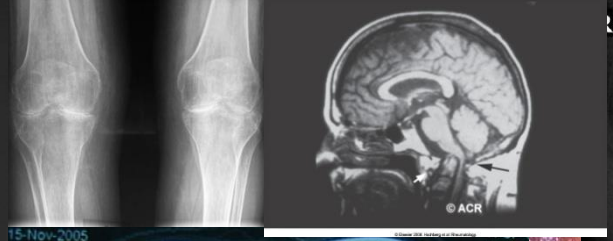
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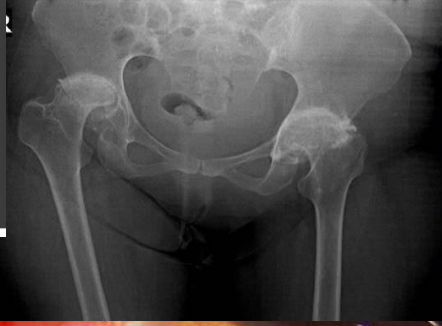

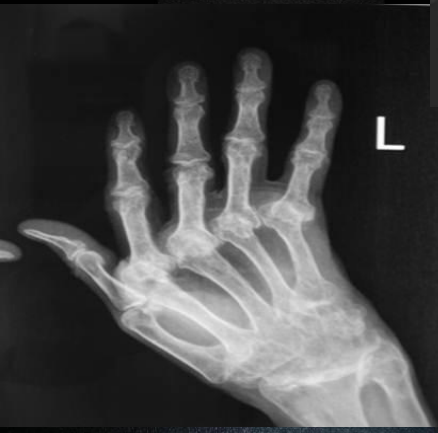
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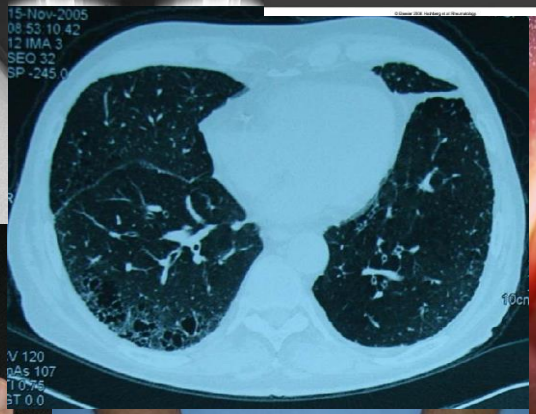
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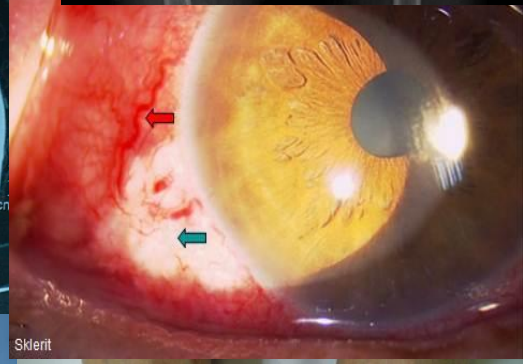
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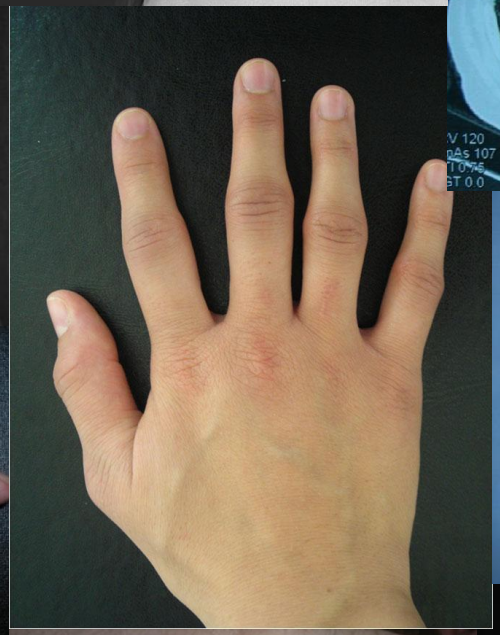

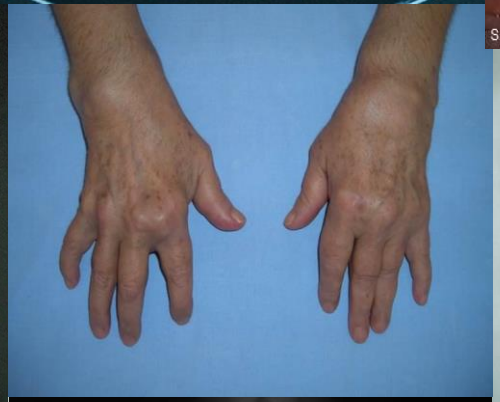

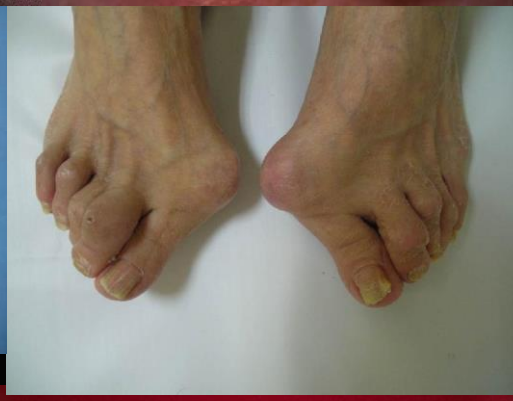

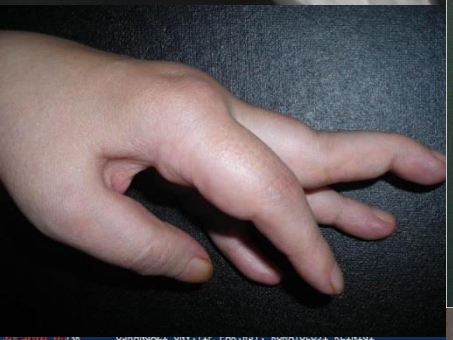

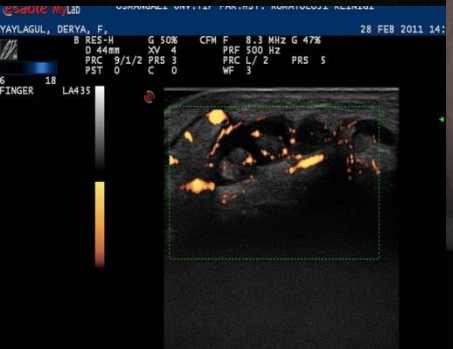
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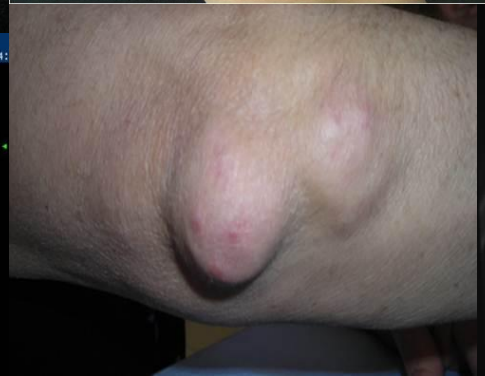

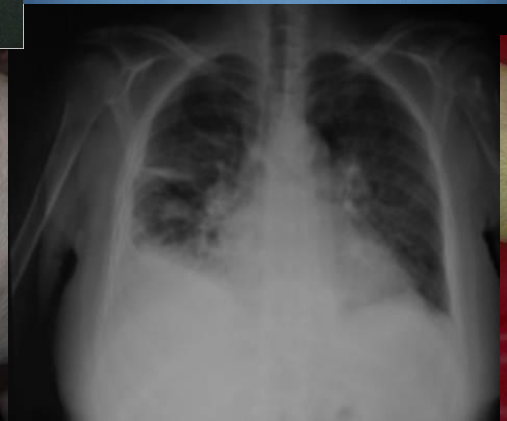

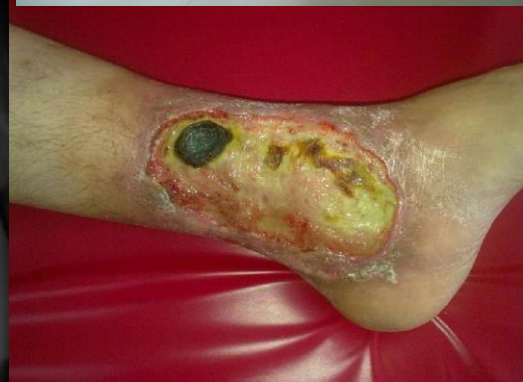


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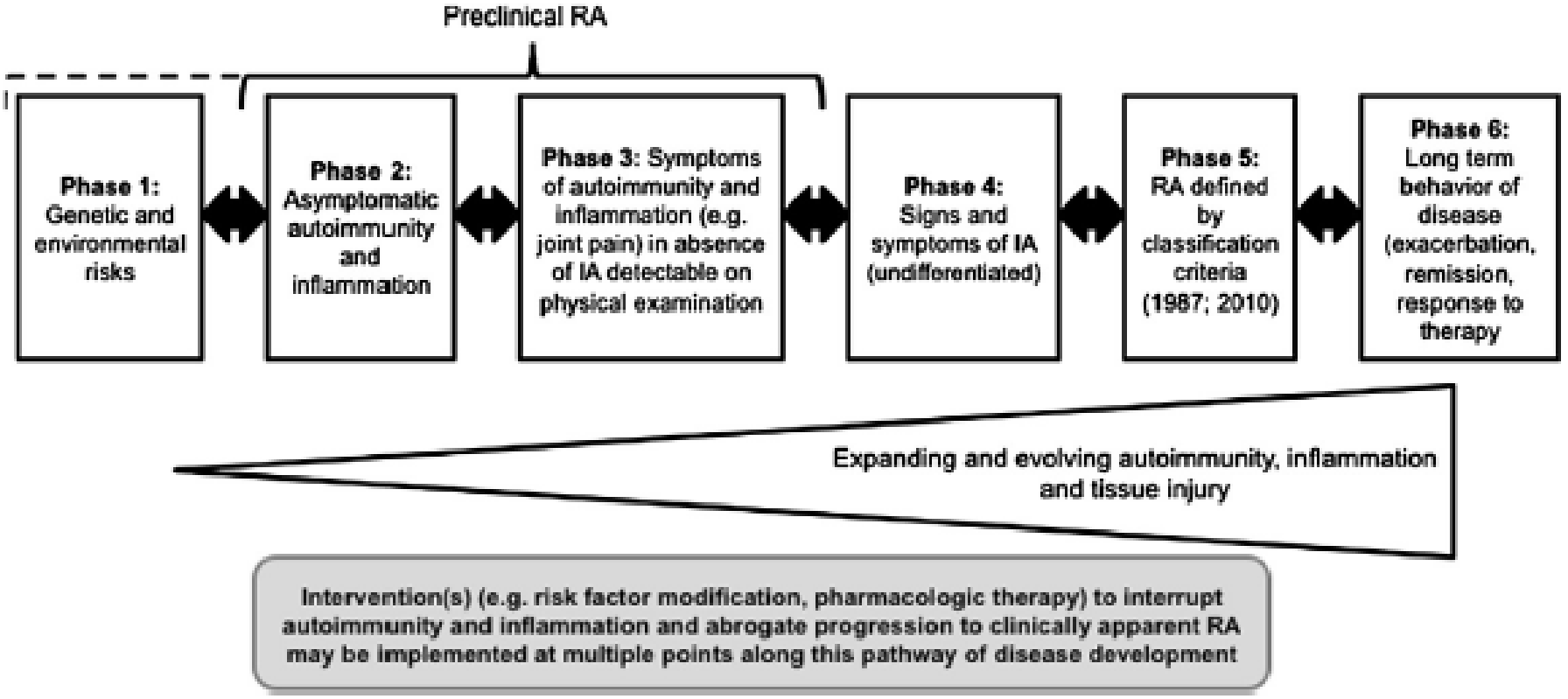

K.D. Deane / Best Practice & Research Clinical Rheumatology 27 (2013) 467–485

REVIEW ARTICLE

WILEY Journal of Cellular Physiology

Strategies toward rheumatoid arthritis therapy; the old and the new

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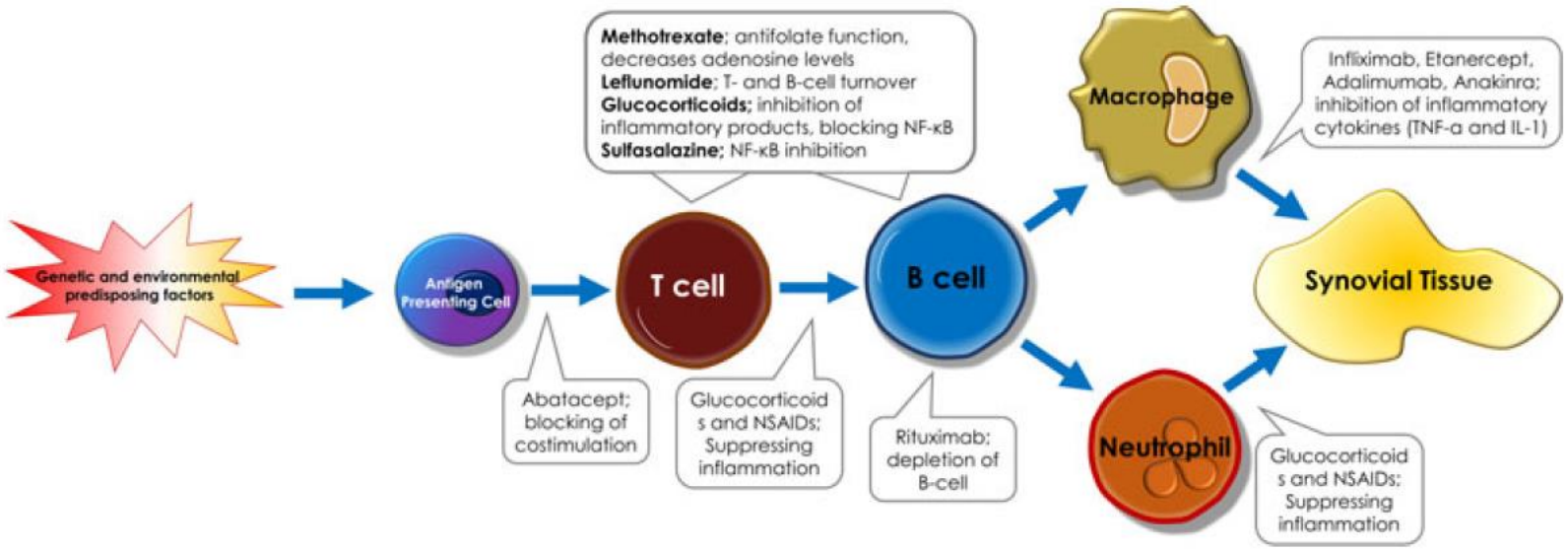

TABLE 1 Categorization of treatment options for RA therapy

Drug names	Functions	Outcome
NSAIDs	COX inhibitors	Resolution of inflammation and pain
Glucocorticoids	Inhibition of inflammatory genes	Resolution of inflammation and pain
DMARDs	Suppression of immune cell proliferation Reducing the accumulation of toxic elements in joints Decrease intracellular levels of glutathione, resulting in diminished tissue damage due to toxic oxygen metabolites Promoting the extracellular levels of adenosine	Resolution of inflammation and pain
Adalimumab Infliximab Etanercept	Targeting TNF Targeting TNF Targeting TNF	Anti-inflammatory properties
Abatacept	Targeting CTLA4	Reducing effector T cells
Tofacitinib	Inhibition of JAK	Reducing inflammatory cytokine production
Rituximab	Targeting CD20 molecule on B cells	Reducing B-cell count and function
Trichostatin A	Inhibition of HDACs	Reducing IL-6 level and inflammation
Givinostat	Inhibition of HDACs	Reducing IL-6 level and inflammation
Vorinostat	Inhibition of HDACs	Suppression of fibrosis
Entinostat	Inhibition of HDACs	Suppression of NF-κB
Valproic acid	Inhibition of HDACs	Stimulation of Tregs
Mesenchymal stem cell	Cell–cell contact Soluble mediator production	Decreasing inflammation

Note. CD: cluster of differentiation; COX: cyclooxygenase; CTLA: cytotoxic T lymphocyte antigen; DMARD: disease-modifying rheumatic disease; GC: glucocorticoid; HAT: histone acetyltransferase; HDAC: histone deacetylase; IL: interleukin; JAK: Janus-activated kinase; MSC: mesenchymal stem cell; NF-κB: nuclear factor-κB; NSAID: nonsteroidal anti-inflammatory drugs; TNF, tumor necrosis factor; Treg, regulatory T cell.