Η σημασία των προγνωστικών παραγόντων στην εμφάνιση και εξέλιξη της νόσου

Νέστορας Αυγουστίδης
Ρευματολόγος, Επιμελητής Α'
Ρευματολογική Κλινική ΠΑΓΝΗ

ΠΑΝΕΠΙΣΤΗΜΙΑΚΟ ΝΟΣΟΚΟΜΕΙΟ ΗΡΑΚΛΕΙΟΥ

- No conflict of interest

Περίγραμμα ομιλίας

- Παράγοντες κινδύνου (risk factors) για την εμφάνιση της νόσου

- Κλασσικοί παράγοντες κακής έκβασης της νόσου (poor prognostic factors) όπως τους γνωρίζουμε μέχρι σήμερα από τυχαιοποιημένες μελέτες, κατευθυντήριες οδηγίες και κοορτές ασθενών.

- Νέα εργαλεία κατηγοριοποίησης νόσου-ασθενών και εκτίμησης κινδύνου κακής έκβασης , υποτροπών και βέλτιστων θεραπευτικών επιλογών

1. Disease multi-trajectories
2. Molecular portraits and genomics/transcriptomics/RNA-sequencing
3. Artificial intelligence and deep machine learning

Παράγοντες κινδύνου (risk factors) για την εμφάνιση της νόσου

Sex

- Approximately two-thirds of individuals who develop RA are women . The cumulative risk of developing RA in adult population has been estimated at 3.6% for women and 1.7% for men

Family History of RA

- There is an increased prevalence of RA in those families with an 40–50% risk for seropositive RA, being strongest in first-degree relatives (FDRs)

Auto-Antibodies

- The presence of different auto-Abs in serum can be detected years before RA disease onset . Several longitudinal studies have linked the presence of ACPA to the onset of clinical arthritis

Novella-Navarro et al. Risk Factors for RA Development. Frontiers in Medicine 2021

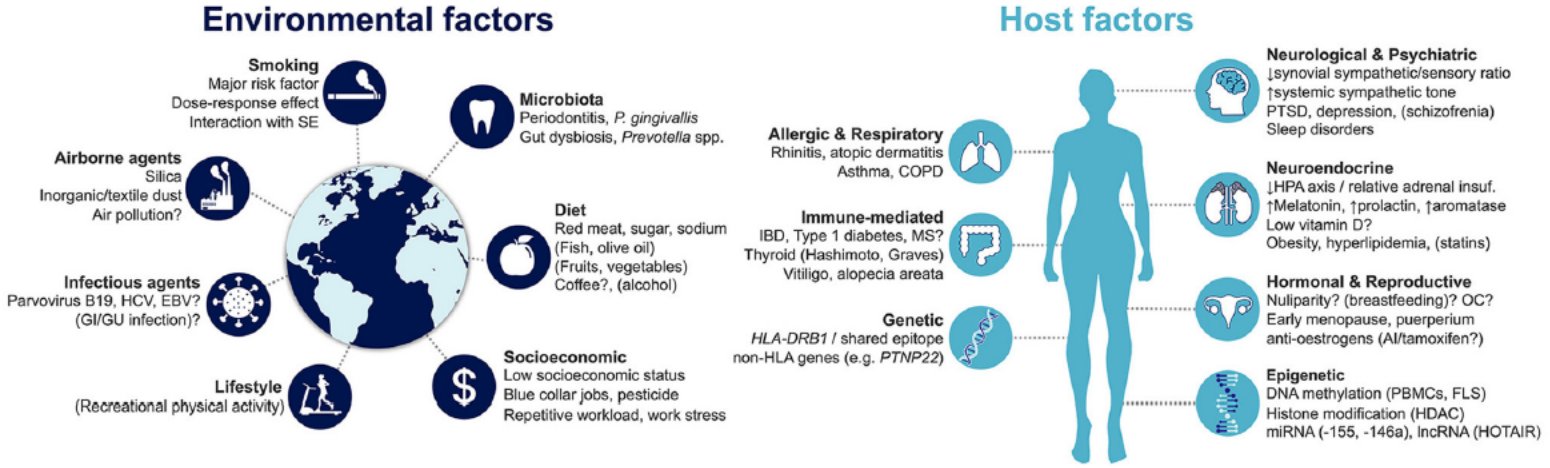


FIGURE 1 | Summary of risk factors for the development of rheumatoid arthritis. Factors that are associated with decreased risk are represented in brackets. Factors for which evidence is conflicting and uncertainty remains are followed by a question mark. AI, aromatase inhibitors; COPD, chronic obstructive pulmonary disease; EBV, Epstein-Barr virus; FLS, fibroblast-like synoviocyte: GI, gastrointestinal; GU, genitourinary; HCV, hepatitis C virus; HDAC, histone deacetylases; HLA, human leukocyte antigen; HPA, hypothalamic-pituitary-adrenal; IBD, inflammatory bowel disease; lncRNA, long non-coding RNA; miRNA, micro RNA; MS, multiple sclerosis; OC, oral contraceptives; PBMCs, peripheral blood mononuclear cells; PTSD, post-traumatic stress disorder; SE, shared epitope.

Novella-Navarro et al. Risk Factors for RA Development. Frontiers in Medicine 2021

Παράγοντες κινδύνου (risk factors) για την εμφάνιση της νόσου

Smoking

- Smoking is the most important environmental risk factor for RA development. The association between smoking and RA presents an OR > 2, and smoking is estimated to account for approximately 20–30% of the environmental risk for RA

Periodontal Disease

- Periodontitis (PD), is a disease that is more frequent in subjects with RA than in healthy populations and P. gingivalis is the main causative agent of PD. It is the only bacterium known expressing peptidyl arginine deiminase (PAD) enzyme necessary for protein citrullination

Silica/Dust Inhalation

- Multiple studies have demonstrated an association between exposure to occupational silica/dust and RA, mainly in ACPA positive RA .

1.Novella-Navarro et al. Risk Factors for RA Development. Frontiers in Medicine 2021
2.JM Kroese et al. Oral microbiome in early RA . Arthritis and Rheumatology 2021

Clinical Application

Modifiable

Cigarette smoking	Strong evidence supports an association of smoking with RA development . After twenty years of smoking cessation, RA risk returns to the risk of a non-smoker, suggesting modifiability
Overweight/obesity	Increased body mass index, inflammation, and RA are linked by a variety of mechanisms, including adipokines and chemokines .Several studies associate overweight and obesity with RA development
Low fish intake	Oily fish intake has a protective effect for RA, perhaps due to omega-3 polyunsaturated fatty acids
Periodontitis	Periodontal disease has been consistently associated with RA . *Porphyromonas gingivalis*, a bacterium that causes periodontitis, may mediate citrullination of peptides that leads to the development of RA-related autoimmunity

Modification of RA risk-related behaviours

Sparks JA, et al. Arthritis Care Res (Hoboken). 2018
Sparks JA, *et al. Personalized risk* estimator for rheumatoid arthritis (PRE-RA) family study .Contemp Clin Trials. 2014

Clinical Application

Table 6 Core risk factors for development of arthritis in individuals at risk of RA according to population

At-risk population	Subpopulations	Core risk factors for arthritis
Asymptomatic at-risk individuals	Relatives of RA probands	Serum ACPA level±RF
	Indigenous at-risk populations	
	ACPA + individuals identified by population screening	
MSK symptoms without arthritis	ACPA + with MSK symptoms	Serum ACPA level±RF MSK symptoms Subclinical joint inflammation on US Subclinical joint and tendon inflammation on MRI
	ACPA+/RF + with arthralgia	Serum ACPA level±RF MSK symptoms Subclinical joint inflammation on US
	Clinically suspect arthralgia	Serum ACPA level±RF MSK symptoms Subclinical joint and tendon inflammation on MRI
Early clinical arthritis	Palindromic rheumatism	Serum ACPA level±RF MSK symptoms
	Undifferentiated arthritis	Serum ACPA level±RF MSK symptoms Subclinical joint inflammation on US Subclinical joint and tendon inflammation on MRI

ACPA, anticitrullinated protein antibodies; MSK, musculoskeletal; RA, rheumatoid arthritis; RF, rheumatoid factor; US, ultrasound.

Close follow-up and serology profile of patients with suspected inflammatory arthralgia

Use of more advanced imaging techniques (MRI+U/S) for selected patients

Mankia K, *et al. RMD Open* 2021

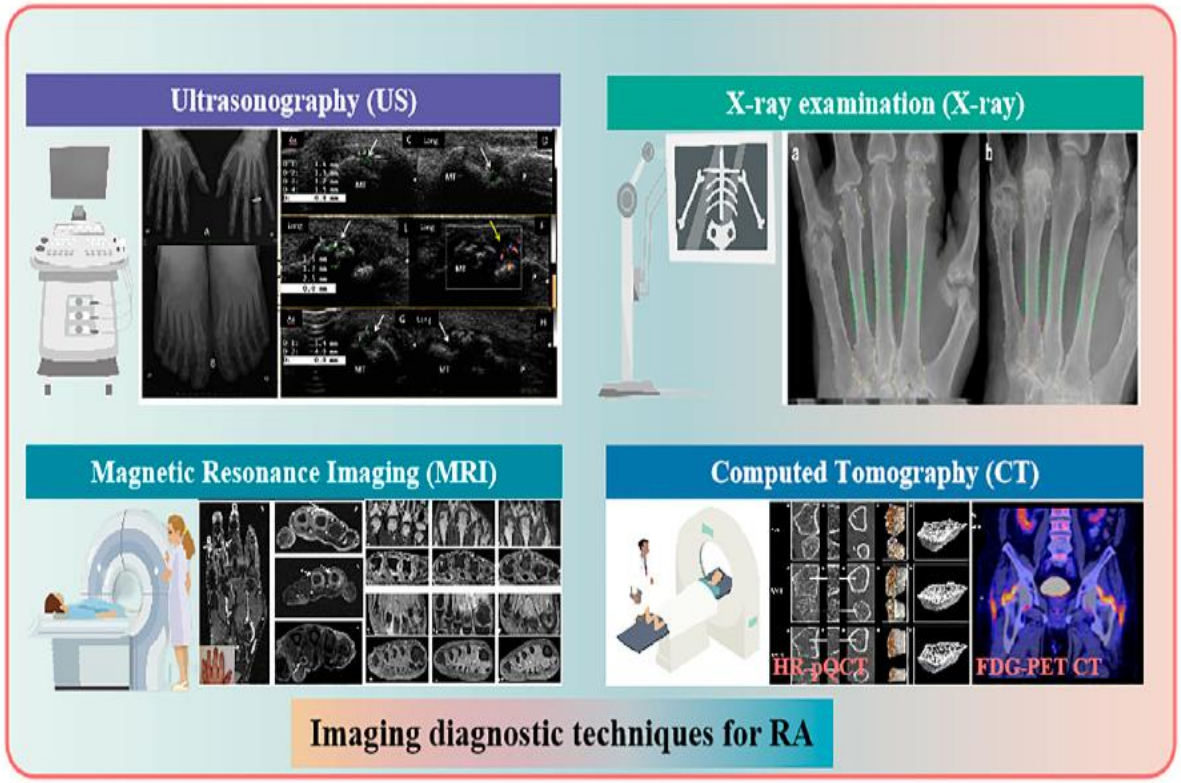


FIGURE 4 | Different imaging diagnostic techniques for RA.

Ultrasound

	First author	Sample size	Duration of follow-up	Subjects recruited	Main results
US	Nam JL[18]	136	Median 18.3 months	Musculoskeletal symptoms, ACPA positive, no clinical synovitis	Doppler signal and erosion over hand and foot joints associated with development of inflammatory arthritis
	Van Beers-Tas MH[19]	163	Median 12 months	Arthralgia, RF or ACPA positive, no clinical arthritis	Synovial thickening of hand joints associated with development of clinical arthritis
	Filer A[20]	58	18 months	Clinical synovitis at least one joint, symptom duration </= 3 months	Synovial thickening of wrists and MCPJ, and power Doppler signal of MTPJ predictive of RA
	Sahbudin I[21]	107	18 months	Clinical synovitis at least one joint, symptom duration </= 3 months	Tenosynovitis of digit flexor predictive of RA
	Zufferey P[22]	80	Mean 18 months	Polyarthralgia, no RF or ACPA, no clinical synovitis	Synovial thickening of hands, elbows and knees predictive of RA

- US-defined synovial thickening
- power Doppler signal
- tenosynovitis
- bone erosion

} predictive value for inflammatory arthritis

Important considerations

1. The subclinical inflammation detectable by US might be a late feature in the development of inflammatory arthritis.
2. The narrow window between the detection of US abnormalities and clinical arthritis might not allow any meaningful intervention
3. Lastly, it is also not clear which and how many joints need to be imaged for optimum predictive accuracy.

Ho So et al. Rheumatology and Immunology Research 2021

MRI

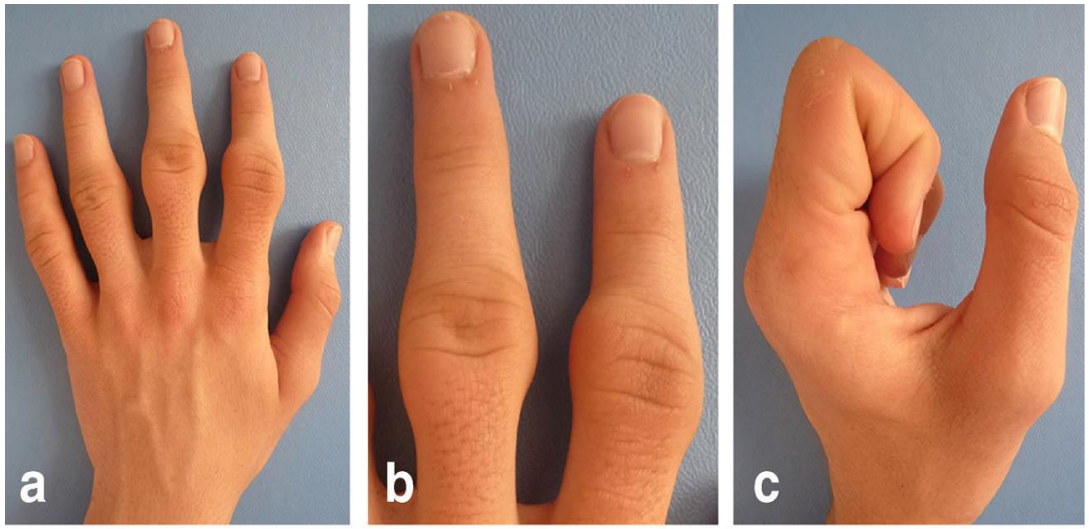
	Di Matteo A[23]	419	Median 41.4 months	Musculoskeletal symptoms, ACPA positive, no clinical synovitis	Bone erosion in > 1 hand or foot joints, and bone erosion with synovitis in foot joints predictive of inflammatory arthritis
MRI	Tamai M[28]	129	12 months	Undifferentiated arthritis	Synovitis and bone marrow edema or erosion over hand joints in conjunction of autoantibodies predictive of RA
	Ji L[29]	31	Median 15 months	Undifferentiated arthritis	Synovitis and bone erosion in writs associated with the development of RA
	Van Steenbergen HW[30]	150	Median 6.3 months	Arthralgia of small joints <1 year, no clinical arthritis, suspected to progress to RA by rheumatologists	MRI inflammation score (sum of synovitis, bone marrow edema and tenosynovitis) over hands and feet predictive of inflammatory arthritis
	Wouters F[32]	490	Progressors: median 1.2 months, Non-progressors: median 8.6 months	Arthralgia of small joints <1 year, no clinical arthritis, suspected to progress to RA by rheumatologists	Bone erosion in hands and feet associated with development of inflammatory arthritis, but not after adjustments for age and MRI inflammation

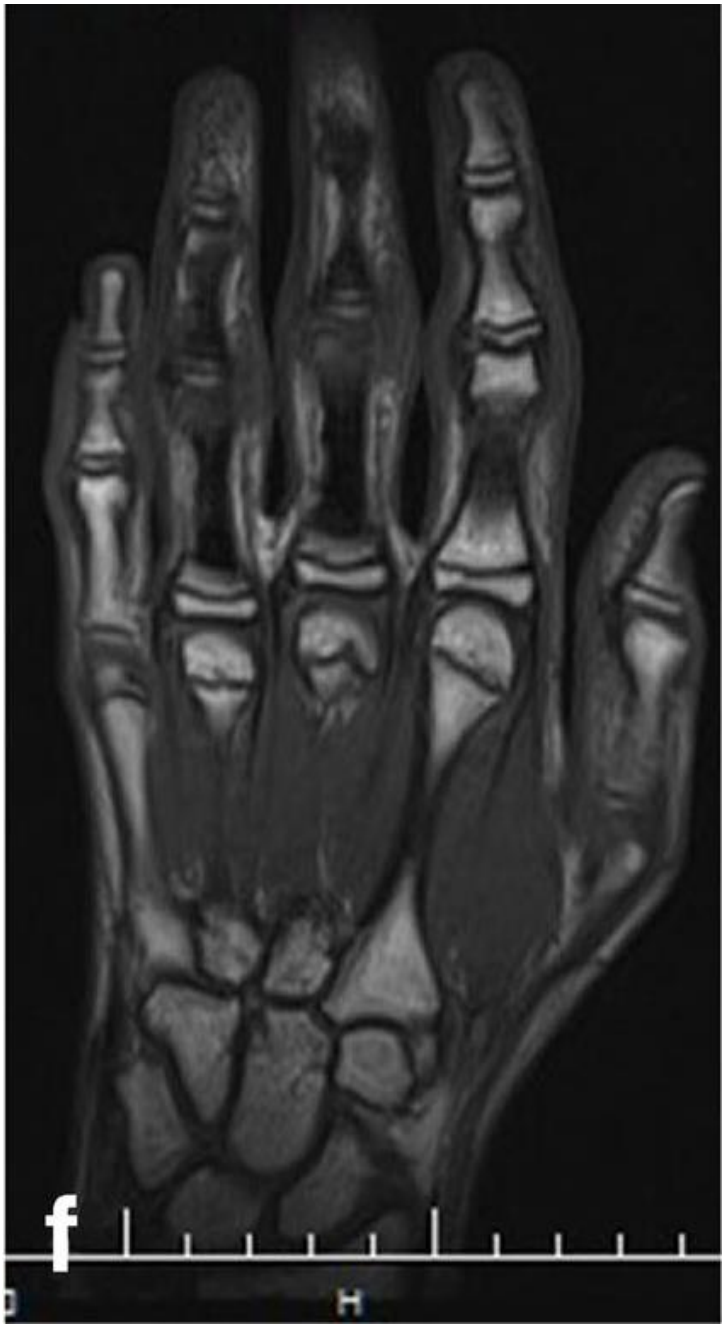
- Synovitis
- Tenosynovitis
- Bone marrow oedema

} could predict the development of inflammatory arthritis.

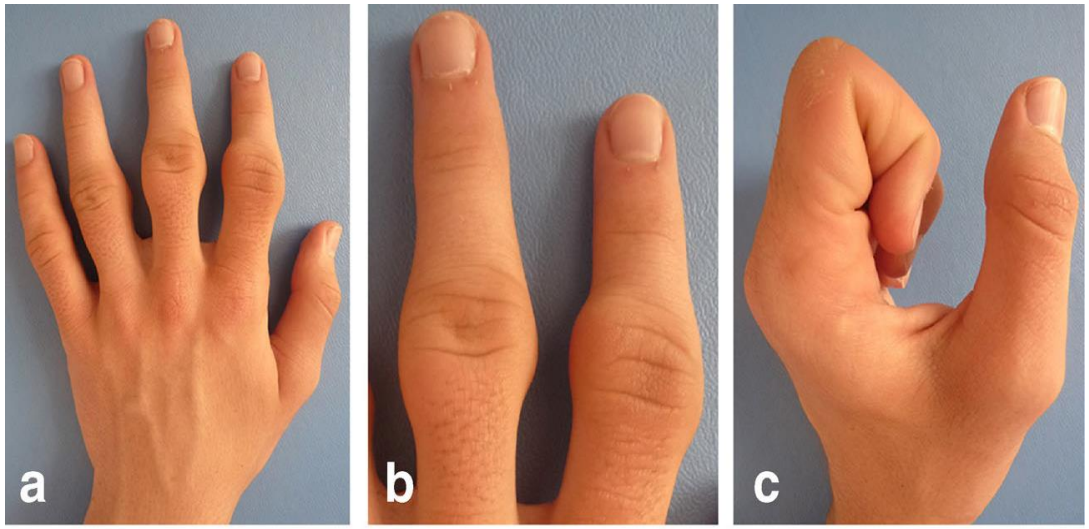
- Due to the relatively long scanning time and limited access the use of MRI is generally recommended only in difficult patient cases at least for the management of early arthritis.

Ho So et al. Rheumatology and Immunology Research 2021

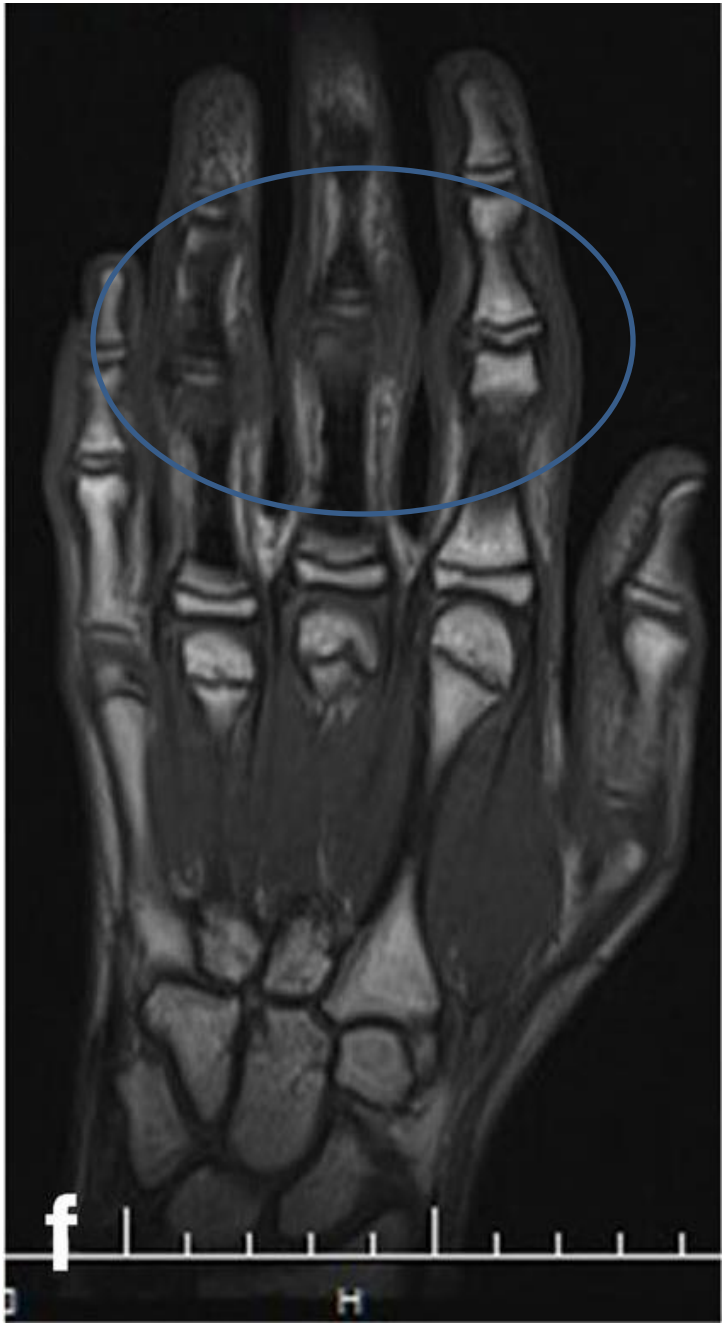




Curr Rheumatol Rep (2014) 16:442



Pachydermodactyly



Curr Rheumatol Rep (2014) 16:442

RA complexity and heterogeneity

- Pre RA
- Seronerative versus Seropositive
- Early versus established
- Erosive versus non erosive
- With or without extraarticular manifestations
- Difficult to treat RA
- NIRRA versus PIRRA

Prognostic factors

- Prognostic factors are used for RA diagnosis, treatment decisions, and prognosis of disease severity. So far, there is no uniform definition of poor prognostic factors.

- Prognostic factors are used as inclusion criteria in randomized controlled trials. They are defined heterogeneously and the relevance of a single or combined presence of poor prognostic factors remains unclear.

- Factors that are used predominantly for treatment decisions are:

1. high disease activity
2. early presence of erosions
3. autoantibody positivity
4. extraarticular disease
5. functional decline (HAQ score)

Albrecht and Zink Arthritis Research & Therapy (2017)

Table 1 Identification of poor prognostic factors in randomized trials and observational cohort studies

Poor prognostic factor	Outcome	Study type	References
Increased DAS28 or single components	Radiographic progression Absence of remission	RCT, cohort Cohort	[7, 9, 11, 15] [12, 42]
Increased MBDA	Radiographic progression	RCT, cohort	[21, 23]
Presence or high titers of RF and/or ACPA	Radiographic progression Absence of remission	RCT, cohort Cohort	[7, 9, 32] [42]
Presence of erosions	Radiographic progression	RCT, cohort	[9, 11, 14, 32]
Increased HAQ	Absence of remission Functional limitation	Cohort RCT	[12, 44] [13]
Smoking	Radiographic progression	RCT	[15]
Delayed diagnosis/treatment initiation	Absence of remission	Cohort	[16]
Ultrasound Doppler activity	Radiographic progression Absence of remission	Cohort	[24, 25]
MRI bone edema	Radiographic progression	RCT, cohort	[26–28]
Genetic predisposition (relatedness)	Radiographic damage	Cohort	[29]

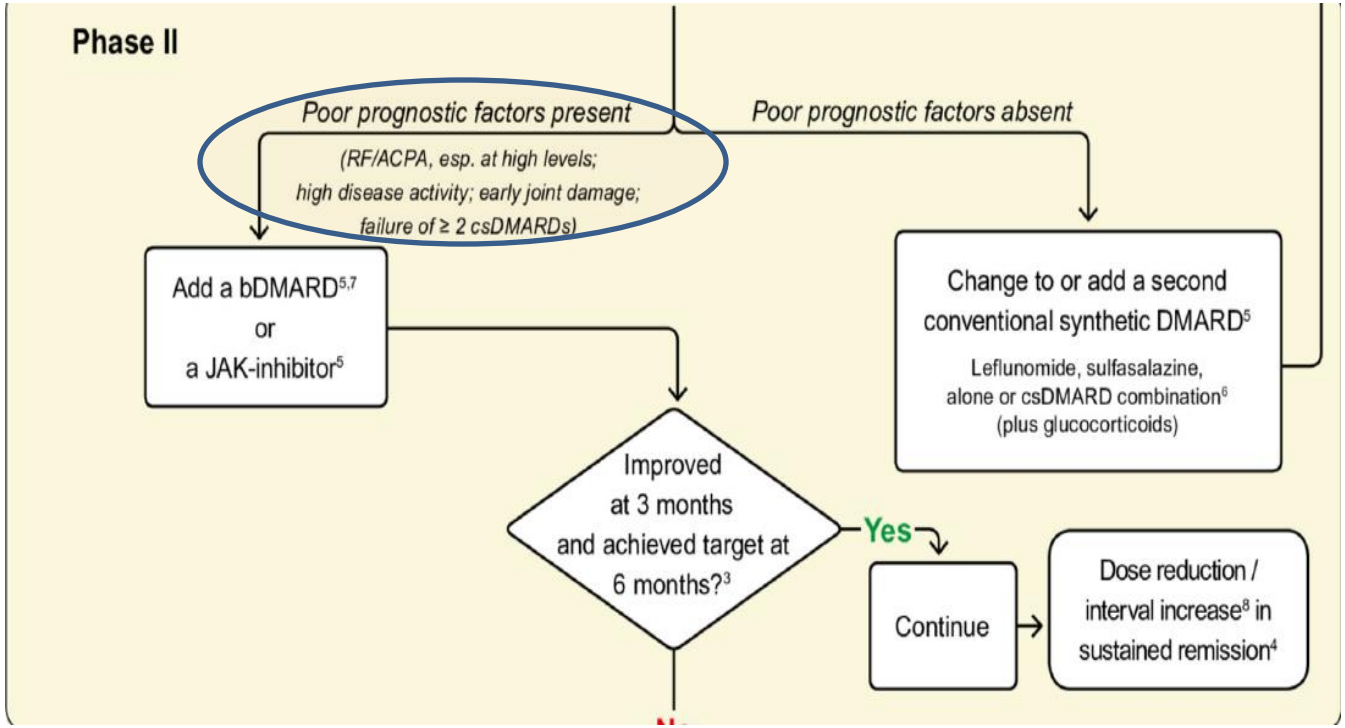
ACPA anti-citrullinated protein-peptide antibodies, *HAQ* Health Assessment Questionnaire, *MBDA* multibiomarker disease activity score, *MRI* magnetic resonance imaging, *DAS28* disease activity score of 28 joints, *RCT* randomized controlled trial, *RF* rheumatoid factor

Albrecht and Zink *Arthritis Research & Therapy* (2017)

Table 3 Treatment recommendations with poor prognostic factors as decision-criteria

	RA state	Poor prognostic factors	Presence allows for	Treatment target
EULAR [3]	RA, first DMARD failure	High disease activity, RF/ACPA positivity, early presence of joint damage	bDMARDs	Low disease activity or remission
ACR [4]	Early RA <6 months	Moderate disease activity + ≥1 of functional limitation, extraarticular disease, RF/ACPA positivity, erosions	csDMARD combination	
		High disease activity + one or more of functional limitation, extra-articular disease, RF/ACPA positivity, erosions	bDMARD or csDMARD combination	
	Established RA (≥6 months or 1987 ACR criteria)	LDA + one or more of functional limitation, extraarticular disease, RF/ACPA positivity, erosions or at least moderate disease activity	csDMARD combination, bDMARD at 3 months	
Italy [5]	RA, DMARD failure	1. High disease activity (DAS28 > 5.1 for ≥1 months 2. Moderate disease activity (DAS >3.2) + ACPA/RF positive and elevated CRP or ESR, persistence of one or more swollen joint, bone erosions on X-rays, active synovitis with power Doppler signal 3. New erosions	bDMARD	
France [35]	RA, DMARD failure	Existence or progression of structural damage, high clinical and/or laboratory activity, high RF/ACPA titers	bDMARD	
Germany [34]	RA, 1st DMARD failure	High disease activity, RF/ACPA positivity, early presence of joint damage	bDMARD	
Canada [36]	RA	Not further specified	Initial csDMARD combination	

ACPA anti-citrullinated protein-peptide antibodies, *ACR* American College of Rheumatology, *CRP* C-reactive protein, *ESR* erythrocyte sedimentation rate, *EULAR* European League Against Rheumatism, *DAS* disease activity score, *bDMARD* biologic disease-modifying antirheumatic drug, *csDMARD* conventional synthetic disease-modifying antirheumatic drug, *RA* rheumatoid arthritis, *RF* rheumatoid factor



A minimal initial treatment goal of low disease activity is **conditionally** recommended over a goal of remission.

Addition of a bDMARD or tsDMARD is **conditionally** recommended over triple therapy for patients taking maximally tolerated doses of methotrexate who are not at target.

Smolen JS, *et al*. *Ann Rheum Dis* 2020

ACR GUIDELINE FOR TREATMENT OF RA

Arthritis Care & Research
Vol. 73, No. 7, July 2021, pp 924–939

Predicting disease progression and poor outcomes in patients with moderately active rheumatoid arthritis: a systematic review

14 studies were prioritized, because they specifically analysed patients with moderate RA.

9 studies reported radiographic progression outcomes for 3241 patients

3 studies reported disease activity progression for 1516 patients, and two studies reported other relevant outcomes for 2094 patients.

TABLE 4 Identified prognostic factors and reported thresholds for patients with moderate RA

Factor	Threshold for progression	Sources
DAS28	>4.2 at baseline	Kiely *et al.*, 2011 [20] Nikiphorou *et al.*, 2015 [28]
Presence of anti-CCP antibodies	(Presence at baseline)	Alemao *et al.*, 2014 [8] Alemao *et al.*, 2016 [7] Kroot *et al.*, 2000 [21] Barra *et al.*, 2013 [10]
PDUS	PDUS score ≥1 at baseline	De Miguel *et al.*, 2015 [14]

Abbreviations: DAS28: 28-joint DAS; PDUS: power Doppler ultrasound.

Key messages

- Three prognostic factors were identified in moderately active RA patients at greater risk of disease progression.
- DAS28 ≥4.2, anti-CCP antibody presence and power Doppler ultrasound score ≥1 were identified as indicators of potential progression.
- Higher-risk RA patients with moderate disease activity may benefit from more intensive treatment strategies.

Christopher J. Edwards *et al.* Rheumatology Advances in Practice 2019


OLD WAY
NEW WAY

Disease multi-trajectories

- Response criteria based on current versus previous scores irrespective of which composite score or response criteria are used they only give a snapshot of patients' health at a specific time-point as they lack the time component.

- Longitudinal approach that identifies subgroups of patients while capturing their evolution across several clinical outcomes simultaneously (multi-trajectories).

- Identifying subgroups of patients with similar progression is essential for understanding what hinders improvement.

- It is essential to identify such subgroups in order to find prediction factors of disease evolution or for treatment response

Disease activity trajectories

Method: early-RA cohort BARFOT (n = 2829) was used to identify 24 month post-diagnosis simultaneous trajectories of 28-joint Disease Activity Score and its components.

For validation, the early-RA-TIRA-2 cohort (newer), (n = 504) was used.

Results: Three multi-trajectories were identified

- ✓39.6% of the patients in the lowest
- ✓18.9% in the highest (worst) trajectory.

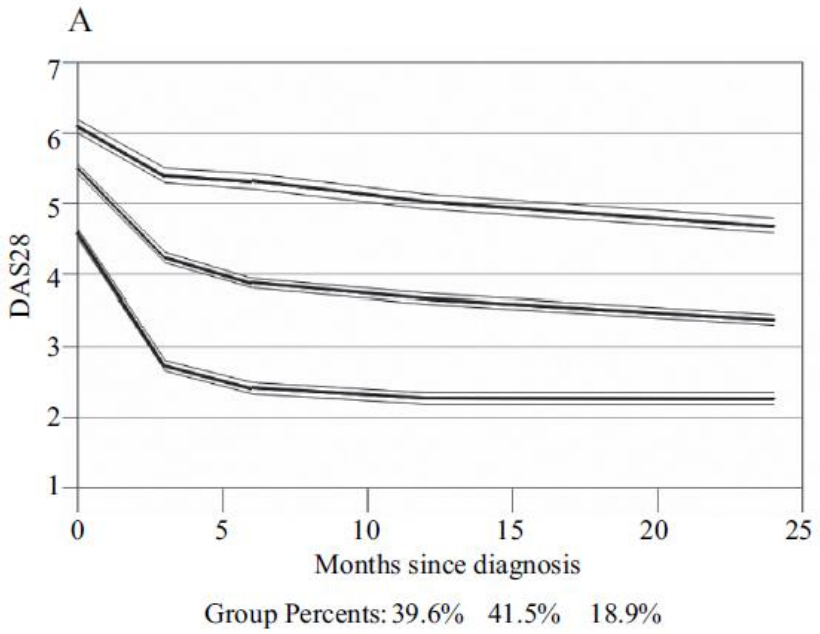
Patients in the worst trajectory had on average eight tender and six swollen joints after 24 months.

Radiographic changes at 24 and 60 months were significantly increased from the lowest to the highest trajectory.

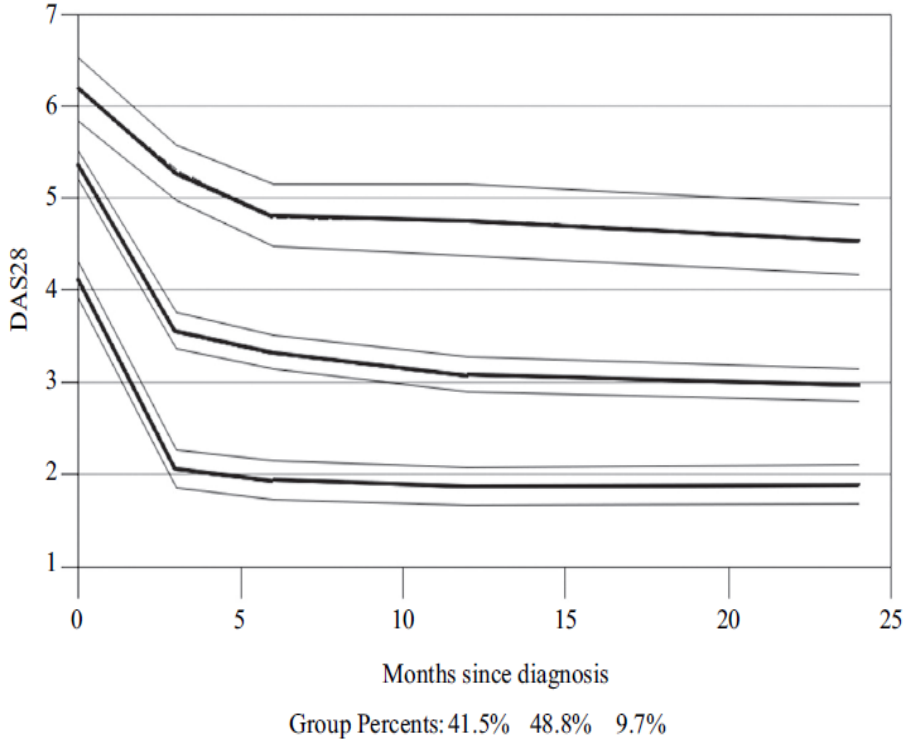
M Leu Agelii et al. Scand J Rheumatol 2021

Disease activity trajectories

BARFOT cohort (n = 2838)



TIRA-2 cohort (n = 504)



Estimated trajectories are shown as thick lines with surrounding 95% confidence bands (thin lines). Mean statistics are shown as dashed lines.

Conclusion: Multi-trajectories constitute a powerful tool for identifying subgroups of RA patients and could be used in future studies searching for predictive biomarkers for disease progression.

M Leu Agelii et al. Scand J Rheumatol 2021

RNA Identification of PRIME Cells Predicting Rheumatoid Arthritis Flares

- a clinical and technical protocol for repeated home collection of blood (fingerstick collection of three drops) of blood at home in patients with rheumatoid arthritis to allow for longitudinal RNA sequencing (RNA-seq).

- Specimens were obtained during eight flares over a period of 4 years in index patient, as well as from 235 time points during flares in three additional patients.

- Identification of transcripts that were differentially expressed before flares and compared these with data from synovial single cell RNA-seq.

Dana E, et al. NEJM July 16, 2020

RNA Identification of PRIME Cells Predicting Rheumatoid Arthritis Flares

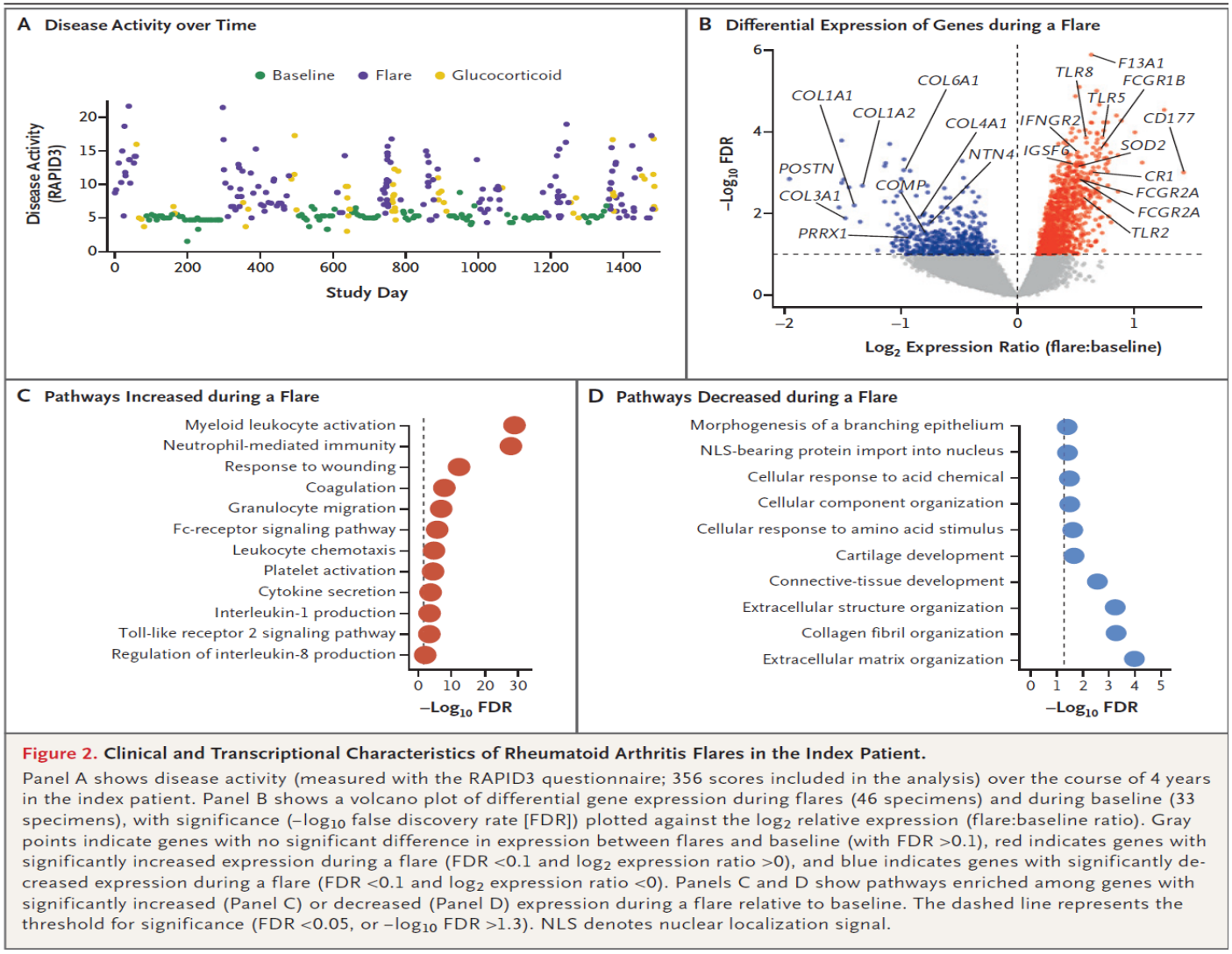


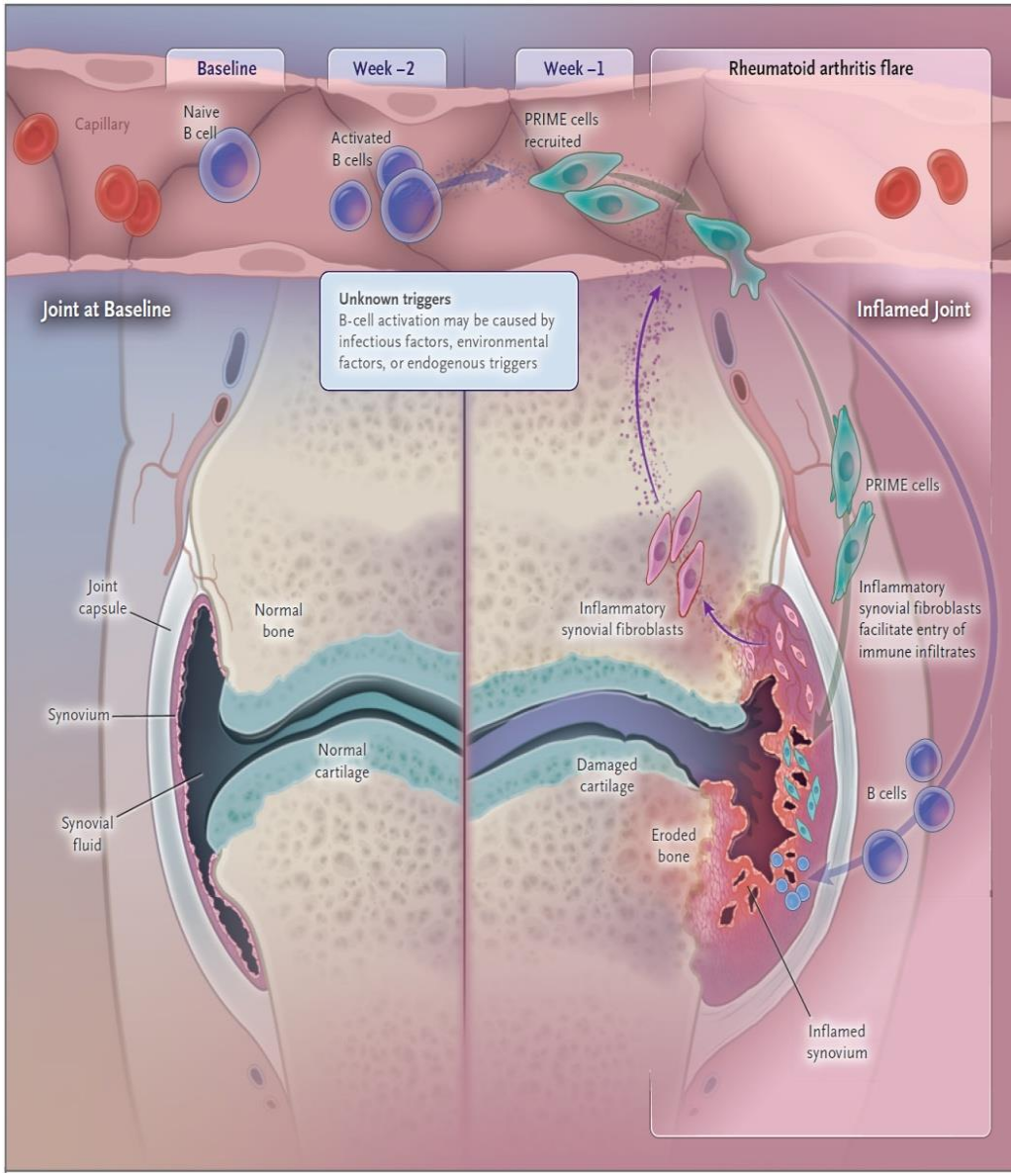
Figure 2. Clinical and Transcriptional Characteristics of Rheumatoid Arthritis Flares in the Index Patient.

Panel A shows disease activity (measured with the RAPID3 questionnaire; 356 scores included in the analysis) over the course of 4 years in the index patient. Panel B shows a volcano plot of differential gene expression during flares (46 specimens) and during baseline (33 specimens), with significance ($-\log_{10}$ false discovery rate [FDR]) plotted against the $\log_2$ relative expression (flare:baseline ratio). Gray points indicate genes with no significant difference in expression between flares and baseline (with FDR >0.1), red indicates genes with significantly increased expression during a flare (FDR <0.1 and $\log_2$ expression ratio >0), and blue indicates genes with significantly decreased expression during a flare (FDR <0.1 and $\log_2$ expression ratio <0). Panels C and D show pathways enriched among genes with significantly increased (Panel C) or decreased (Panel D) expression during a flare relative to baseline. The dashed line represents the threshold for significance (FDR <0.05, or $-\log_{10}$ FDR >1.3). NLS denotes nuclear localization signal.

Dana E, et al. NEJM July 16, 2020

RNA Identification of PRIME Cells Predicting Rheumatoid Arthritis Flares

Longitudinal genomic analysis of rheumatoid arthritis flares revealed PRIME(pre-inflammatory mesenchymal cells) in the blood during the period before a flare and suggested a model in which these cells become activated by B cells in the weeks before a flare and subsequently migrate out of the blood into the synovium.



Dana E, et al. NEJM July 16, 2020

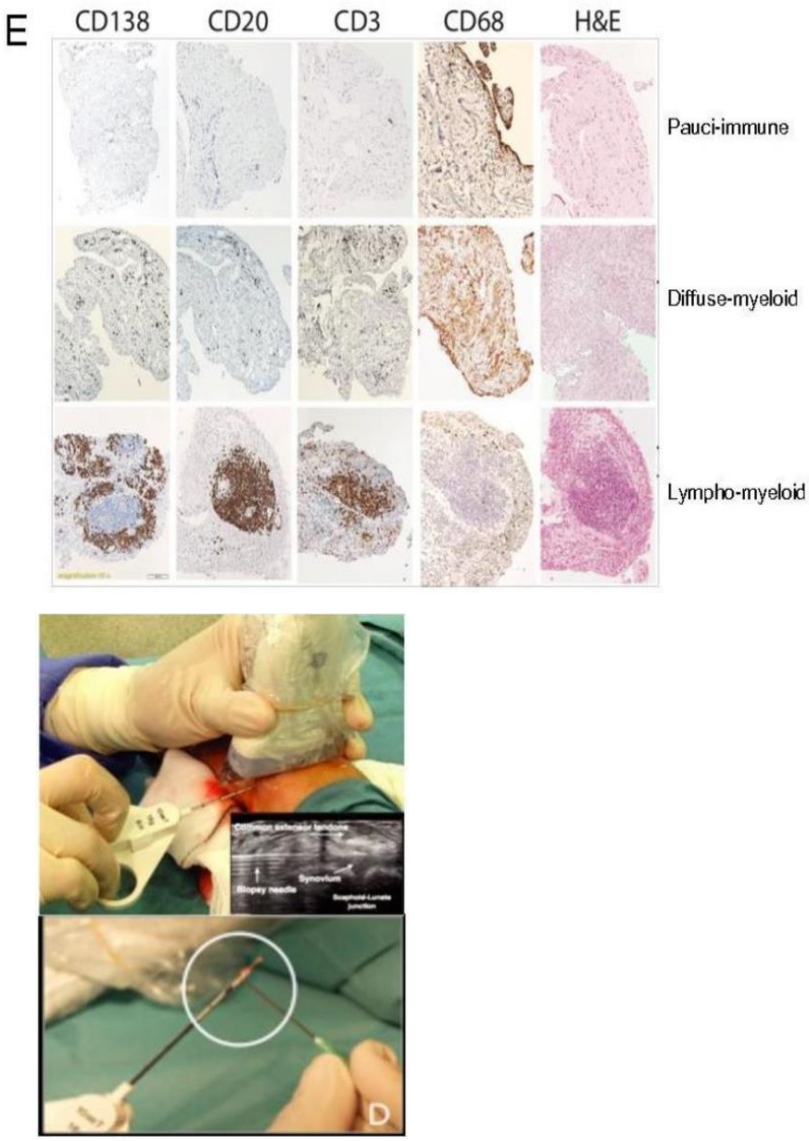
Clinical application

- it may become possible to intervene to prevent clinical flares when the changes associated with flare immunopathogenesis are detected and before clinical symptoms emerge.

But

- since a small number of patients were studied and medications were not considered, and it remains likely that distinct cellular and molecular mechanisms are at play in different subpopulations of patients with rheumatoid arthritis

Synovial cellular and molecular signatures stratify clinical response to csDMARD therapy and predict radiographic progression in early rheumatoid arthritis patients



Baseline clinical and histological parameters stratified according to three pathological subtypes, adjusted for joint type (n=129),

N=129		Pauciimmune-fibroid n=34 (26.4) [NA]	Diffuse-Myeloid n=44 (34.1) [NA]	Lympho-myeloid n=51 (39.5) [NA]	P value
Age (years) mean(SD)		50.8 (14.7) [0]	52 (18) [0]	53.9 (15.6) [0]	0.91
Disease duration (months) mean (SD)		6 (3.4) [1]	4.6 (2.7) [1]	5.7 (3.3) [2]	0.24
ESR (mm/hr) mean (SD)		22.9 (21.5) [1]	29.8 (25.3) [0]	53 (29.8) [0]	$4.5x10^{-5}$ ***
CRP (mg/l) mean (SD)		7.7 (27.7) [0]	13.3 (19.3) [1]	28.4 (33.1) [1]	0.032*
RF positivity n (%)		17 (50) [0]	29 (65.9) [0]	39 (76.5) [0]	0.042*
ACPA positivity n (%)		18 (52.9) [0]	29 (65.9) [0]	40 (78.4) [0]	0.047*
RF titre mean (SEM)		41 (21.8) [0]	125 (26.1) [0]	147 (27.2) [0]	0.044*
ACPA titre mean (SEM)		169 (50.9) [0]	179 (32.1) [0]	250 (30.3) [0]	0.11
SJ mean (SD)		5.3 (5.1) [0]	7.2 (5.2) [0]	8.8 (5.9) [0]	0.045*
TJ mean (SD)		10.3 (8) [0]	10.6 (6.3) [0]	13.2 (7.2) [0]	0.16
VAS mean (SD)		58.1 (25.8) [0]	62.9 (26.2) [0]	67.6 (24) [0]	0.19
DAS28 mean (SD)		4.9 (1.4) [0]	5.3 (1.5) [0]	6.2 (1.3) [0]	$2.9x10^{-1}$ ***
HAQ mean (SD)		1.4 (0.7) [0]	1.4 (0.7) [0]	1.6 (0.8) [0]	0.58
SHSS mean (SD)	Erosions	0.4 (1.4) [5]	0.4 (0.9) [7]	1 (2.6) [8]	0.0039**
	JSN	1.1 (2.5) [5]	1.1 (2.2) [7]	3.5 (7.4) [8]	$1.3x10^{-6}$ ***
	Total	1.5 (3.5) [5]	1.5 (2.5) [7]	4.5 (9.7) [8]	$1.8x10^{-8}$ ***
US score of biopsied joint (0-3) mean (SD)	ST	1.8 (0.7) [3]	2 (0.8) [3]	2.7 (0.5) [8]	0.030*
	PD	0.8 (0.9) [3]	1.5 (1) [3]	2.3 (0.7) [8]	$8.2x10^{-6}$ ***
US 12 max score (0-36) mean (SD)	ST	12.7 (7.5) [8]	15.9 (8.6) [10]	17.5 (9.6) [14]	$5.5x10^{-6}$ ***
	PD	2.7 (3.6) [8]	6.6 (5.7) [10]	8.1 (6.6) [14]	$1.0x10^{-13}$ ***

Humby F, *et al*. *Ann Rheum Dis* 2019;

A

N=106		Pauci-immune n=30(28.3)[NA]	Diffuse-myeloid n=33(31.1)[NA]	Lympho-myeloid n=43(40.6)[NA]	P Value
6 month DAS28 Mean (SD)		-1.6(1.5)[4]	-2(2)[11]	-2.4(1.6)[8]	0.254
Change in DAS Mean(SD)		3.2(1.9)[4]	3.7(1.9)[11]	3.8(2)[8]	0.293
EULAR responder N(%)		25(83.3)	24(72.7)	33(76.7)	0.599
DAS <3.2/>3.2 N(%)		19(63.3)	13(39.4)	17(39.5)	0.085
N=115		Pauci-immune n=31(27)[NA]	Diffuse-myeloid n=38(33)[NA]	Lympho-myeloid n=46(40)[NA]	P Value
DMARD therapy to 6 months (N=115) n(%)	Non MTX	6(19.4)	0(0)	6(13)	0.014[1]
	MTX only	4(12.9)	2(5.3)	2(4.3)	
	MTX combination	21(67.7)	36(94.7)	38(82.6)	

12 months (n=89)		Pauciimmune-fibroid/Diffuse-Myeloid n=55 (61.8%)	Lympho-myeloid n=34 (38.2%)	P value
SHSS	Erosions	0.49 (1.23)	0.71 (1.68)	0.759
	JSN	1.71 (3.66)	3.62 (4.96)	0.044*
	Total	2.2 (4.05)	4.32 (6.04)	0.068
ΔSHSS		0.44 (2.92)	0.85 (2.22)	0.042*
Progressors/non-progressors (ΔSHSS ≥1)		5/50	9/25	0.029*

Humby F, *et al*. *Ann Rheum Dis* 2019;

Key messages

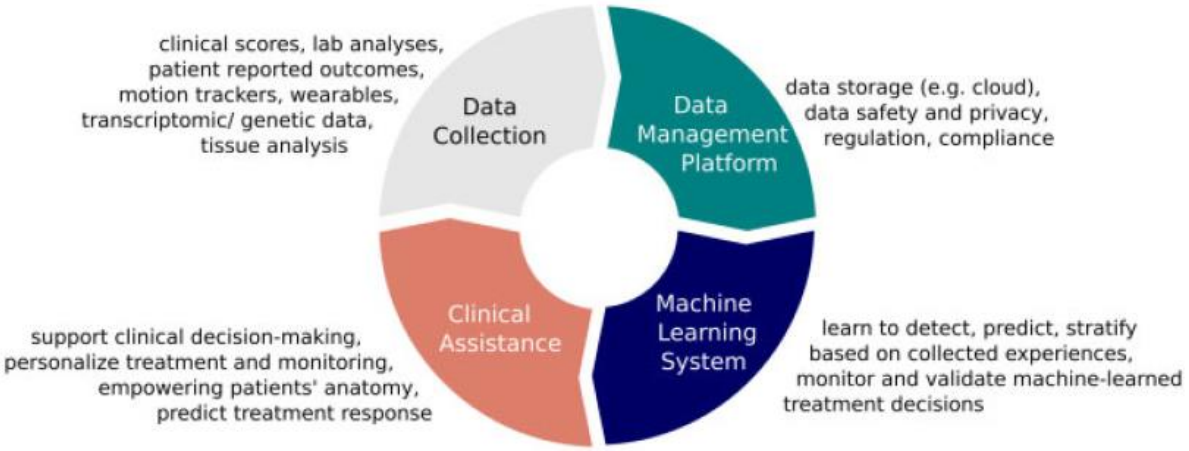
How might this impact on clinical practice?

- This study demonstrates that the cellular and molecular signatures define pathobiological endotypes early in the disease process, prior to treatment modification, that have a significant impact on disease prognosis and treatment outcome.
- This offers the potential for a more accurate patient stratification of this severe disabling disease where early disease modification is crucial to life course outcome and quality of life.

Humby F, *et al*. *Ann Rheum Dis* 2019;

Artificial Intelligence and DML

FIG. 6 Cycle of artificial intelligence-supported data management and clinical decision-making in rheumatology



Key messages

- Deep learning can assist rheumatologists by disease classification and prediction of individual disease activity.
- Artificial intelligence has the potential to empower autonomy of patients, e.g. by providing individual treatment propositions.
- Automated image recognition and natural language processing will be likely to pioneer implementation of artificial intelligence in rheumatology.

Maria Hugle et al. Rheumatology Advances in Practice 2020

Συμπεράσματα

- Δυστυχώς πάρα την σημαντική πρόοδο στην κατανόηση και στην θεραπεία της Ρευματοειδούς Αρθρίτιδας δεν έχουμε αξιόπιστους προγνωστικούς δείκτες έκβασης και ανταπόκρισης στην θεραπεία.

- Παρά τα εντυπωσιακά ευρήματα από τις γονιδιακές μελέτες, τις βιοψίες αρθρικού υμένα αλλά και την χρήση τεχνητής νοημοσύνης λείπουν εκείνοι οι βιοδείκτες που θα είναι εύκολα διαθέσιμοι στον κλινικό ιατρό προκειμένου να γνωρίζει την έκβαση των ασθενών αλλά και την βέλτιστη θεραπευτική επιλογή