POST-ACUTE & LONG-TERM CARE — CME PRESENTATION

A Review in the Management of Rheumatoid Arthritis in the Long-Term Care Setting

Diagnosis, Management, and Complications

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Disclosures: None

SECTION

01

Introduction

Slides 1–3

Objectives

1. Pathophysiology & the preclinical continuum
2. Clinical, extra-articular & radiographic features
3. 2010 ACR/EULAR classification & work-up
4. csDMARD vs. bDMARD vs. tsDMARD
5. Treat-to-target, monitoring & deprescribing
6. Long-term prognosis in the frail elderly

6

objectives for today's session

Why RA Matters in PALTC

1. Systemic autoimmune disease — excess mortality from CV disease, infection, ILD, and disability

2. Late-onset RA (≥65 yr) increasingly common — more functional decline and adverse events than younger-onset disease

3. Central theme: tight inflammatory control protects against lethal complications, but the drugs demand age-attentive safety management

≥65

yr: late-onset RA is rising in prevalence

SECTION

02

Pathophysiology

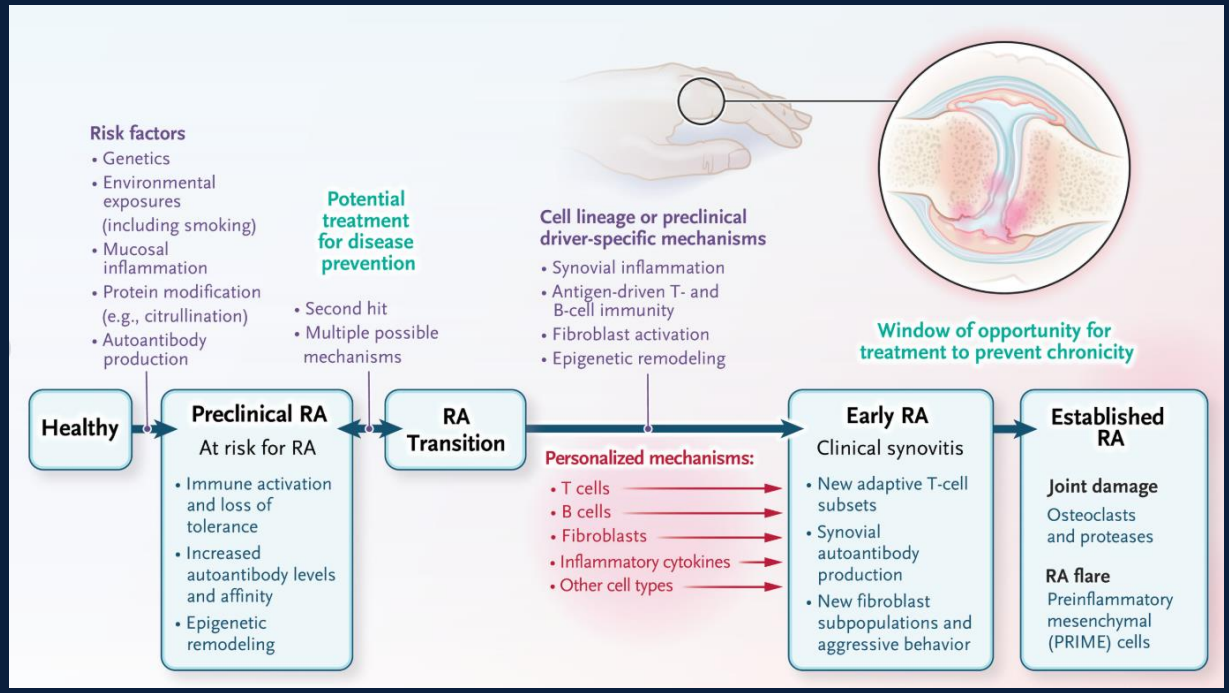
Slides 4–6

The Disease Continuum

1. Genetic risk → environmental exposure → systemic autoimmunity → clinically suspect arthralgia → classifiable RA

2. Autoimmunity precedes clinical disease by years — ACPAs detectable a median ~4.5 yr (IgG ACPA up to ~17 yr) before symptoms

3. Opens a window for prevention/interception



Initiation and progression of RA: health → preclinical → transition → early synovitis → established destructive disease.

Gravallese & Firestein, N Engl J Med. 2023.

Genetics and Environment

1. Heritability ~40–65% for seropositive disease
2. HLA-DRB1 “shared epitope” → 2–3× risk for ACPA+ RA; key non-HLA gene PTPN22
3. Triggers: smoking, silica, periodontitis (P. gingivalis), gut dysbiosis (Prevotella copri)
4. ACPA+ vs. ACPA− RA are genetically distinct entities

20×

risk in ACPA+ smokers with 2 shared-epitope copies

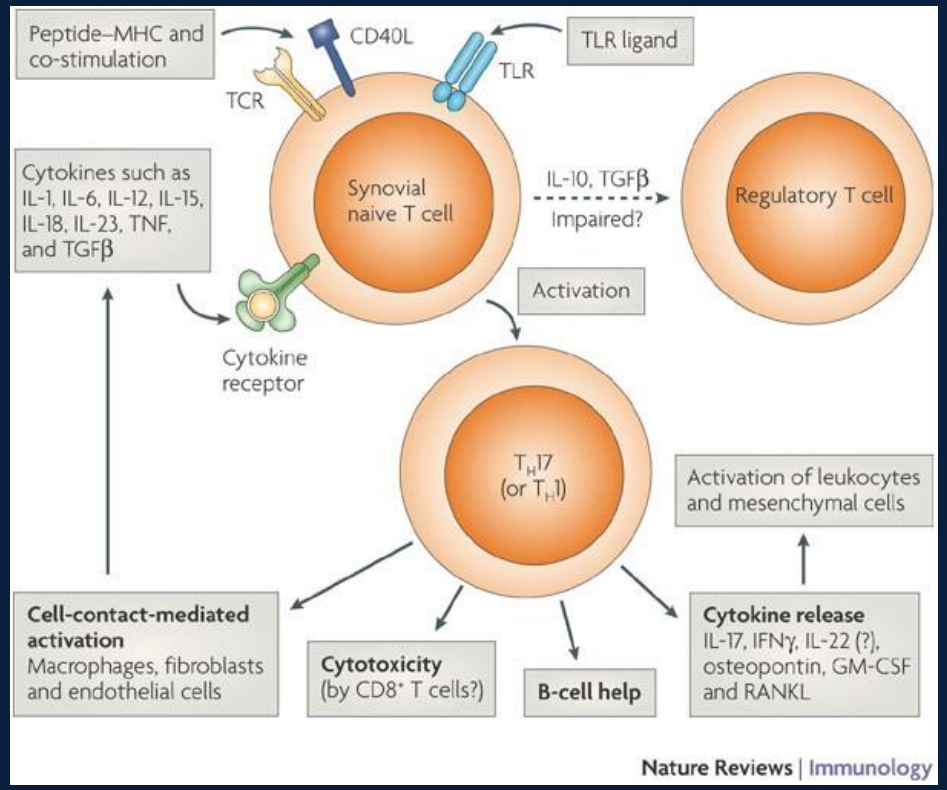
Effector Mechanisms & Cytokine Hubs

1. Citrullination (PAD enzymes) → neoantigens → ACPA immune complexes activate macrophages, osteoclasts, and NETs

2. RA is the prototypic disease with combined TNF-α and IL-6 dependency

3. Fibroblast-like synoviocytes: “tumor-like” invasive pannus; MMPs and RANKL drive erosion

4. JAK–STAT pathway transmits IL-6/interferon signaling — target of JAK inhibitors



TNF-α + IL-6: the twin drivers of RA inflammation

SECTION

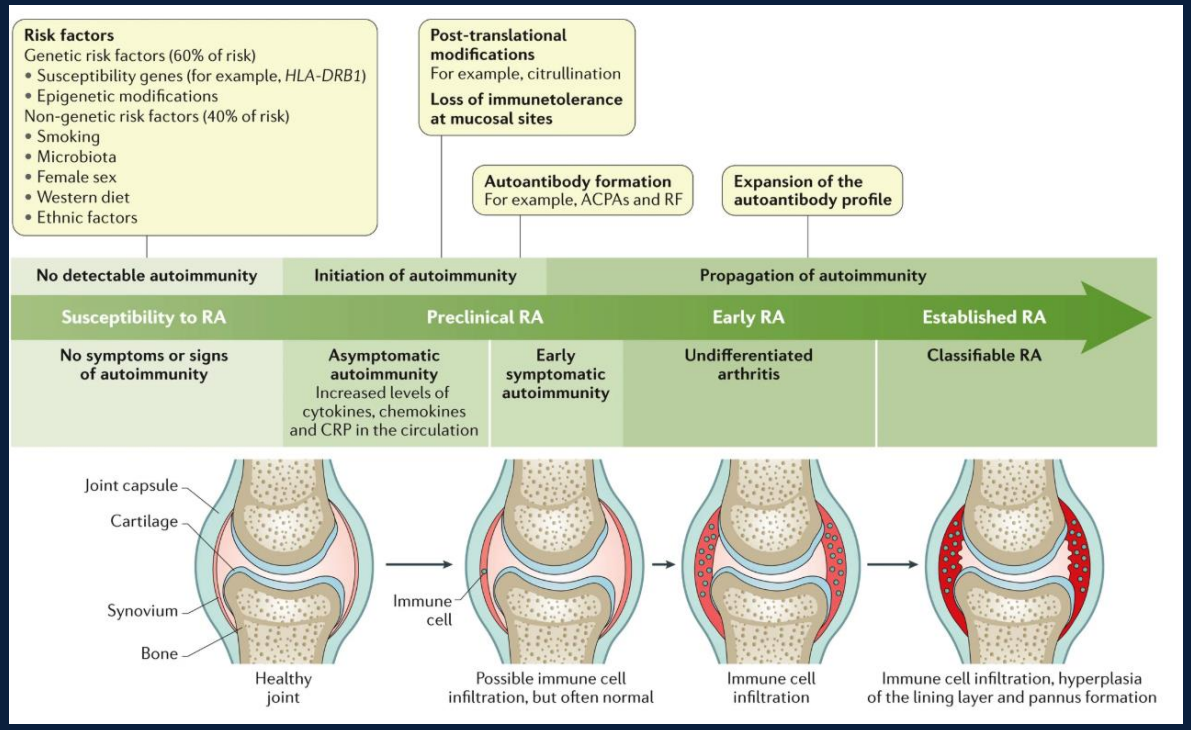
03

Clinical & Radiographic Features

Slides 7–10

Articular Manifestations

1. Insidious, symmetric polyarthritis; morning stiffness ≥30–60 min
2. Wrists, MCP, PIP, MTP — spares DIP and axial spine except C1–C2
3. “Soft” boggy synovial swelling (vs. hard bony OA swelling)
4. Late deformities: ulnar deviation, swan-neck, boutonnière, Z-thumb



Clinical manifestations spectrum: early MCP/PIP swelling → deformities → mutilating foot disease → hand radiographs → C1–C2 pannus MRI → nodules.

Smolen et al., Nat Rev Dis Primers. 2018.

Cervical Spine — PALTC Safety Priority

1. C1–C2 is the only axial joint typically involved; cervical involvement in 25–86%
2. Patterns: atlantoaxial subluxation, vertical subluxation/cranial settling, subaxial subluxation
3. Often asymptomatic — assess cervical spine (flexion-extension films ± MRI) before any general anesthesia/intubation (cord-compression risk)

25–86%

of RA patients have cervical spine involvement

Extra-Articular Manifestations (ExRA)

1. Occur in 30–40% — long-standing, seropositive, smoking, undertreated patients

2. Nodules (most common), ILD, pleuropericardial disease, scleritis/episcleritis, sicca, vasculitis, neuropathy, Felty syndrome

3. Most ExRA declining with modern therapy — but RA-ILD incidence has NOT declined (major mortality driver)

30–40%

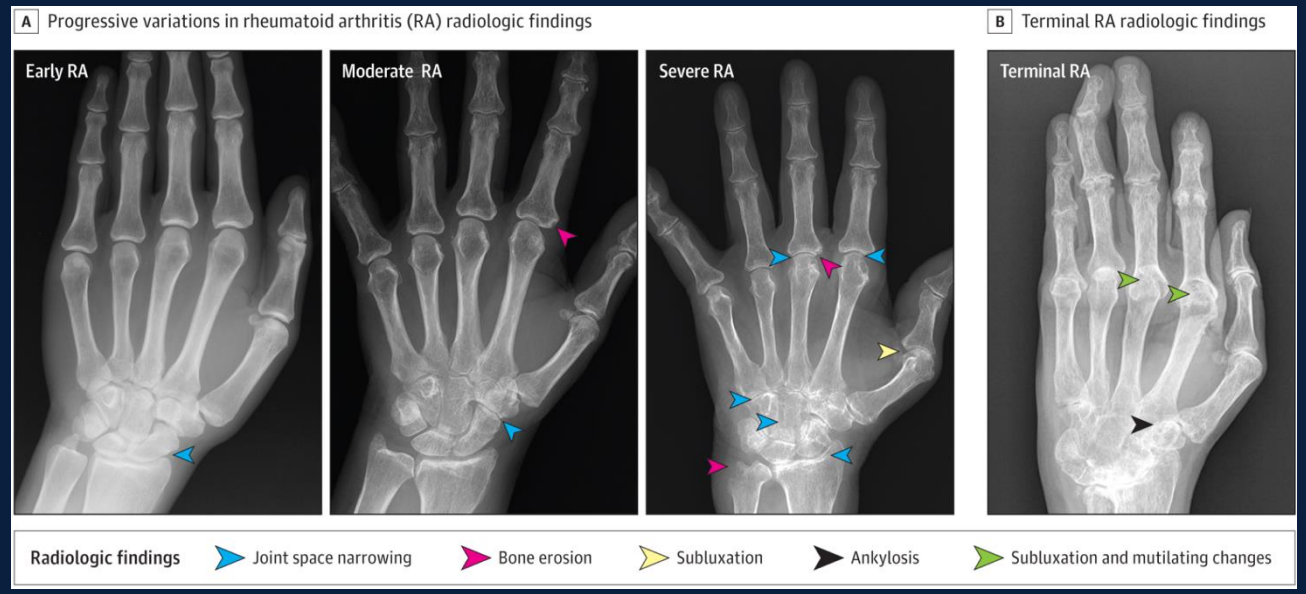
of patients develop extra-articular disease

Radiographic Features

1. Plain radiography = EULAR first-line structural test

2. Order: soft-tissue swelling → osteopenia → marginal erosions → uniform JSN → subluxation; bilateral/symmetric; no osteophytes

3. MRI bone marrow edema/osteitis = strongest predictor of future erosion



Structural phenotypes of RA across disease stages (radiographic progression).

Aletaha & Smolen, JAMA. 2018.

SECTION

04

Differential Diagnosis

Slide 11

Differential Diagnosis of Inflammatory Polyarthritis

PALTC pearls: elderly-onset RA can present PMR-like; polyarticular CPPD frequently masquerades as RA.

Category	Key entities	Distinguishing features
Degenerative	Osteoarthritis	DIP/first CMC, bony swelling, no systemic inflammation
Spondyloarthritis	PsA, AS, IBD-associated, reactive	DIP disease, dactylitis, enthesitis, axial, asymmetry
Crystal	Gout, CPPD/pseudogout	Crystal analysis, chondrocalcinosis; CPPD mimics RA in elderly
Connective tissue	SLE, Sjögren, scleroderma	ANA, sicca, Raynaud, organ features
Infectious	Septic, viral (parvovirus B19, HBV/HCV, HIV), Lyme	Acute/self-limited (<6 wk viral); synovial fluid/serology
Older-adult mimics	PMR, RS3PE	Proximal girdle stiffness >50 yr; pitting edema, steroid-responsive

SECTION

05

Classification Criteria

Slides 12–13

2010 ACR/EULAR Classification Criteria

Entry: ≥1 joint with clinical synovitis not better explained otherwise; typical erosions → classify as RA regardless of score . Score ≥6/10 = definite RA.

Domain	Category	Points
Joints	1 large	0
	2–10 large	1
	1–3 small (± large)	2
	4–10 small (± large)	3
	>10 (≥1 small)	5
Serology	Negative RF & ACPA	0
	Low-positive (≤3× ULN)	2
	High-positive (>3× ULN)	3
Acute-phase	Normal CRP & ESR	0
	Abnormal CRP or ESR	1
Duration	<6 weeks	0
	≥6 weeks	1

Criteria: Performance and Limitations

1. Pooled sensitivity ~0.82, specificity ~0.61
2. Under-identifies seronegative RA (only 25–51% early-classified; need >10 joints to reach threshold)
3. These are classification (research), not diagnostic, criteria — a patient may have RA without meeting them
4. 2025 EULAR/ACR risk-stratification criteria now address the at-risk arthralgia stage (AUC 0.80; 0.87 with MRI)

0.82 / 0.61

pooled sensitivity / specificity

SECTION

06

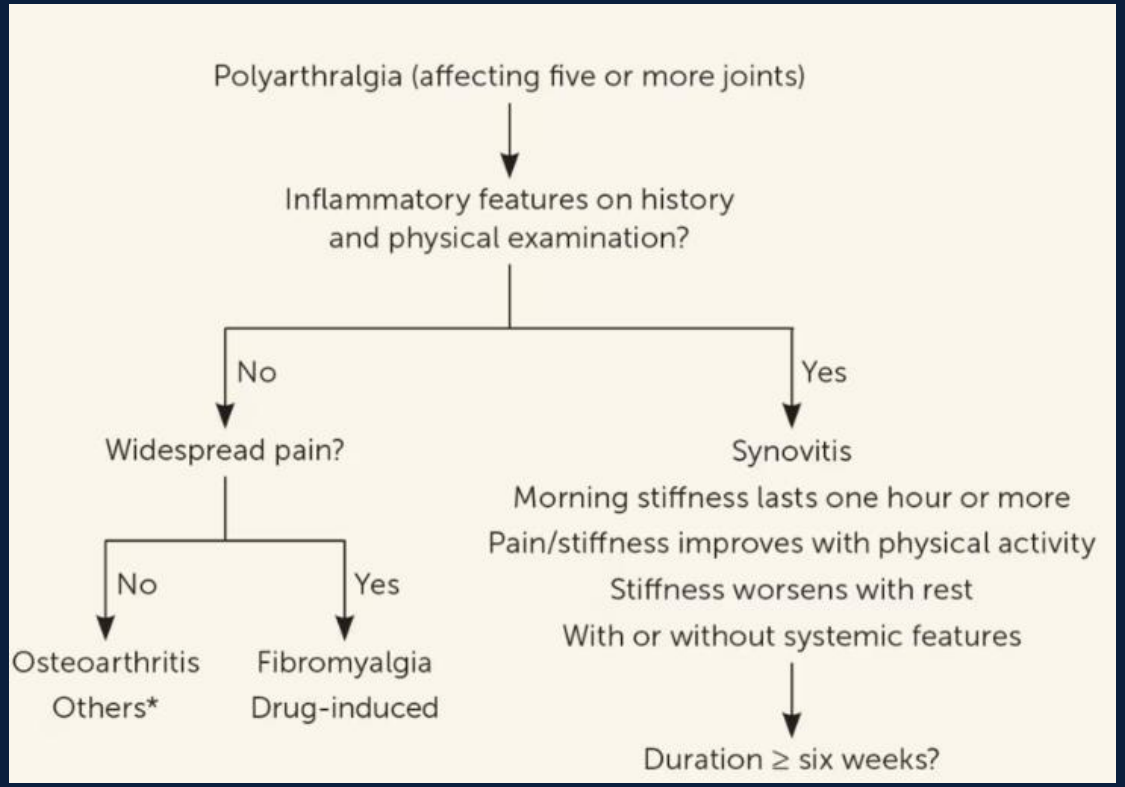
Work-Up Framework

Slides 14–19

Framework: Two Branch Points

1. Branch 1 — inflammatory vs. non-inflammatory: morning stiffness ≥30 min, improves with activity/worsens with rest, nocturnal pain, objective swelling

2. Branch 2 — symptom duration: 6-week threshold separates likely self-limited (viral/reactive) from chronic inflammatory arthritis

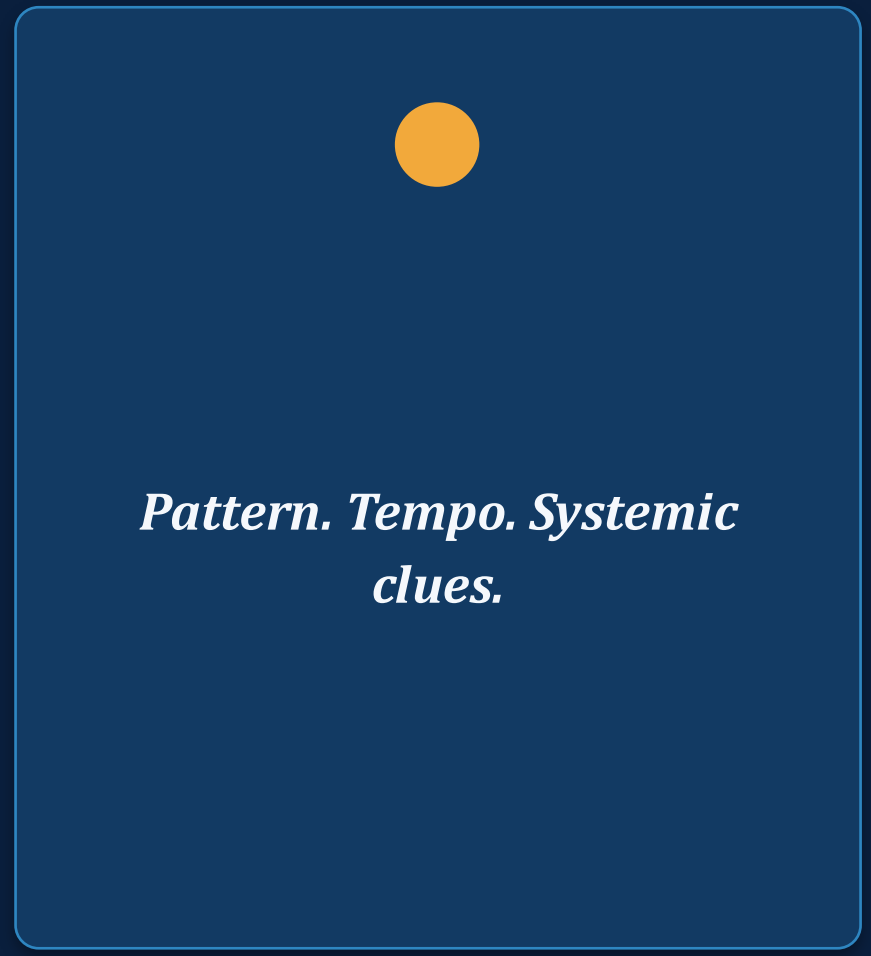


General approach to polyarticular pain: inflammatory vs. non-inflammatory, then 6-week duration.

Foster et al., Am Fam Physician. 2023.

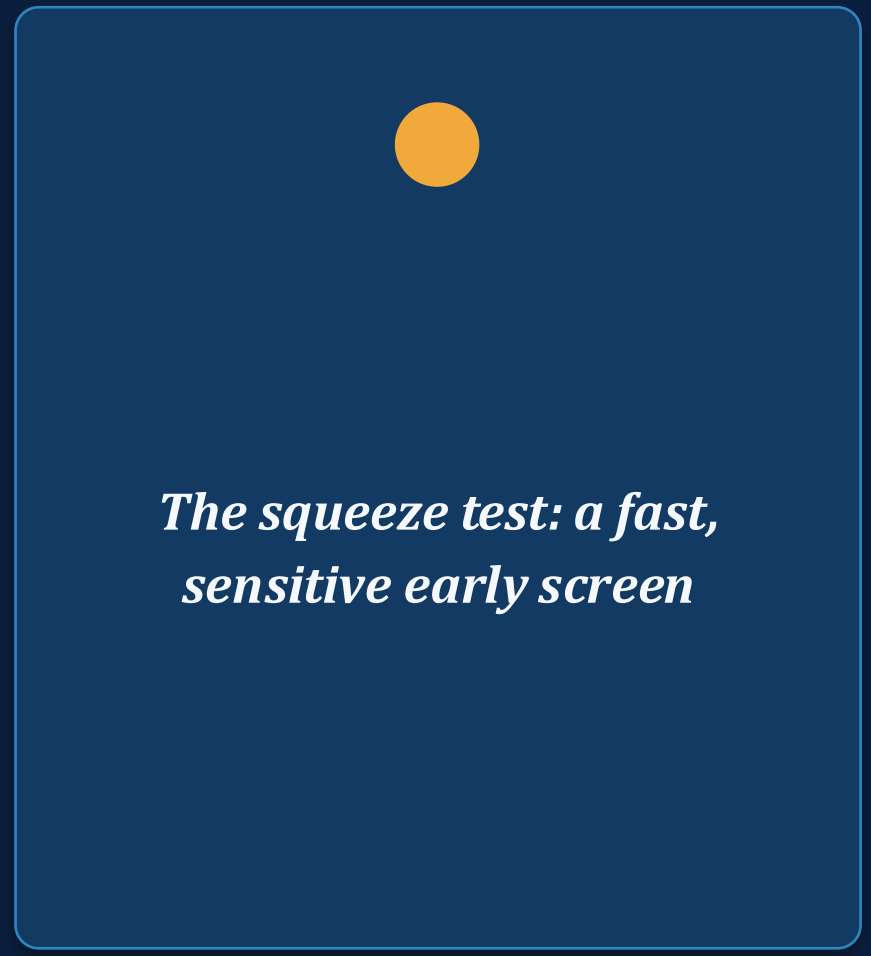
History

1. Pattern: mono/oligo/polyarticular, symmetric vs. asymmetric — MCP/PIP/wrist/MTP favor RA; DIP/dactylitis/enthesitis/axial → SpA/PsA
2. Tempo: additive/persistent (RA) vs. migratory (gonococcal, rheumatic fever, viral)
3. Systemic/ExRA clues & exposures: psoriasis, sicca, Raynaud, uveitis, ticks, sexual/blood exposure, smoking, family history
4. Older adults: watch for PMR-like presentation and polyarticular CPPD



Physical Examination

1. Confirm synovitis (soft/boggy swelling, warmth, tenderness, effusion); count and map involved joints
2. Distinguish boggy synovitis from hard OA nodes (Heberden/Bouchard)
3. MTP/MCP squeeze (compression) test — sensitive early screen; forefoot tenderness often earliest sign
4. Look for tenosynovitis, dactylitis, nodules, tophi, nail pitting, scleritis, enthesitis, lymphadenopathy



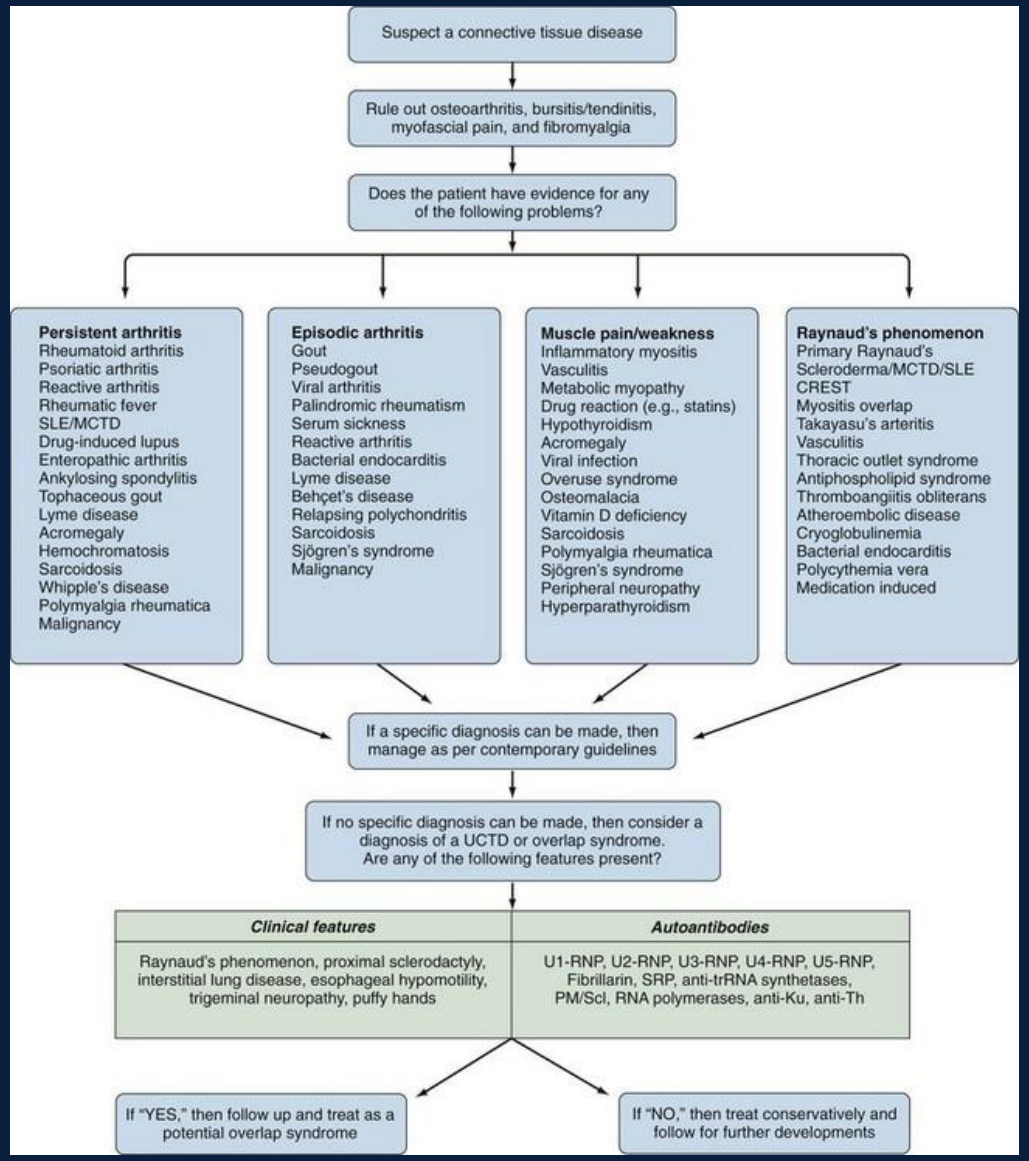
Initial Laboratory Panel

Test	Purpose	Key point
Anti-CCP (ACPA)	Most specific serology	Sens ~57–67%, spec ~95–96%; +LR ~12.5
Rheumatoid factor	Criterion serology	Sens ~67–69%, spec ~85%; also Sjögren, HCV, elderly
ESR & CRP	Acute-phase reactants	Nonspecific; ESR age-elevated
CBC with diff	Cytopenias, ACD, thrombocytosis	Leukopenia/lymphopenia → SLE
CMP (LFTs, Cr)	Organ function; baseline pre-DMARD	eGFR guides MTX dosing
Uric acid	Gout evaluation	Normal level does not exclude gout
ANA	Only if CTD features	Low-titer common in healthy people
Hep B & C serologies	Viral mimic; pre-immunosuppression	Reserve for risk factors/pre-biologic

Lab Stewardship Pearls

1. ANA: reserve for patients with CTD features (a priori threshold missed no SLE/CTD over 1 yr) — reflexive ordering is low-yield

2. Viral serologies: routine screening added little in recent-onset polyarthritis — reserve for risk factors or before starting MTX/biologics



Test smarter, not more

Imaging, Aspiration, and Referral

1. Baseline hand/foot radiographs (EULAR first-line) + chest radiograph (ILD/sarcoid/infection)
2. Ultrasound/MRI when diagnosis uncertain (MRI osteitis strongest erosion predictor)
3. Joint aspiration mandatory whenever effusion could be septic/crystal-induced (synovial WBC >50,000/mm³ → suspect infection)
4. Refer early: rheumatology within 6 weeks; 3-month “window of opportunity” for early DMARDs

6 wks

target window to refer to rheumatology

SECTION

07

DMARD Therapy

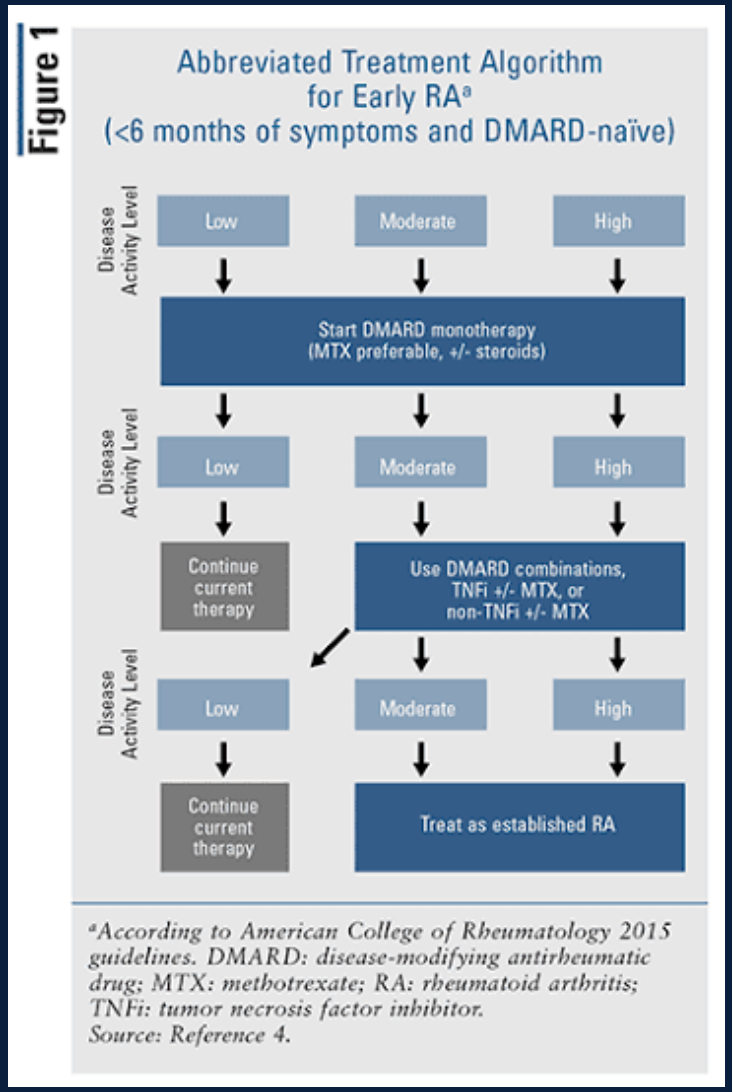
Slides 20–24

DMARD Classes: Overview

1. Three classes: conventional synthetic (csDMARD), biologic (bDMARD), targeted synthetic (tsDMARD/JAKi)

2. csDMARDs anchor first-line therapy

3. b/tsDMARDs added after inadequate response



Conventional Synthetic DMARDs (csDMARDs)

Monitoring: MTX/LEF/SSZ — CBC, transaminases, Cr q2–4 wk ✕ 3 mo → q8–12 wk → q12 wk; HCQ — baseline + annual retinal exam after 5 yr.

Drug	Typical dosing	Common side effects	Serious adverse reactions	Key contraindications
Methotrexate	15 mg/wk PO/SC → 25–30 mg/wk; folic acid mandatory; reduce if eGFR <60, avoid <30	Transaminitis, nausea, stomatitis, cytopenias, alopecia	Myelosuppression, hepatic fibrosis, pneumonitis, serious infection	Pregnancy, eGFR <30, liver disease, active infection, cytopenias
Leflunomide	20 mg/day (10 if not tolerated); skip loading in elderly	Diarrhea, alopecia, transaminitis, HTN, rash	Fatal hepatotoxicity (boxed), pancytopenia, SJS/TEN, ILD, neuropathy	Pregnancy (boxed), severe hepatic impairment; long half-life → cholestyramine washout
Sulfasalazine	500 mg → 2 g/day (1 g BID); enteric-coated	Nausea/dyspepsia, rash, headache, reversible oligospermia, orange discoloration	Agranulocytosis, aplastic anemia, hepatitis, SJS/TEN, DRESS, hemolysis (G6PD)	Sulfa/salicylate allergy, GI/urinary obstruction, porphyria
Hydroxychloroquine	200–400 mg/day, ≤5 mg/kg/day	Nausea, diarrhea, rash, pigment change	Irreversible retinopathy, QT/cardiomyopathy, myopathy, cytopenias	4-aminoquinoline hypersensitivity; caution psoriasis, porphyria, G6PD

Biologic DMARDs (bDMARDs)

Combine with MTX (superior) — except IL-6R inhibitors, which retain monotherapy efficacy (ADACTA).

Class / example	Dosing	Common side effects	Serious adverse reactions	Precautions
TNF inhibitors (adalimumab, etanercept, infliximab)	SC or IV; loading for IV agents	Injection/infusion reactions, URTI, headache	Serious infection, TB & HBV reactivation, demyelination, HF worsening, lupus-like	Active infection, untreated latent TB, active HBV, NYHA III–IV HF, demyelinating disease
IL-6R inhibitors (tocilizumab, sarilumab)	SC or IV; full monotherapy efficacy	Neutropenia, ↑LFTs, hyperlipidemia	Serious infection, GI perforation, hepatotoxicity	Avoid if ANC <2,000, plt <100K, ALT >1.5 × ULN; LFT/CBC q3mo, lipids q6mo
T-cell costimulation blocker (abatacept)	SC weekly or weight-based IV	Headache, URTI, nausea	Infection (lower than TNFi), COPD exacerbation	Preferred with recurrent infection; caution COPD
B-cell depletion (rituximab)	IV; premedicate with steroid	Infusion reactions, HTN, hypophosphatemia	HBV reactivation (boxed), PML (boxed), hypogammaglobulinemia	Screen HBsAg/anti-HBc; reserve after ≥1 TNFi

Targeted Synthetic DMARDs (JAK Inhibitors)

1. Oral agents: tofacitinib 5 mg BID (or 11 mg XR daily); baricitinib 2 mg daily (US); upadacitinib 15 mg daily
2. Efficacy superior to MTX; comparable to adalimumab (SELECT-COMPARE)
3. Common: URTI, headache, hyperlipidemia, transaminitis, cytopenias, herpes zoster
4. Monitoring: baseline TB/HBV/HCV; LFTs/CBC at 4–8 wk then q3mo; lipids at 4–8 wk then q6mo

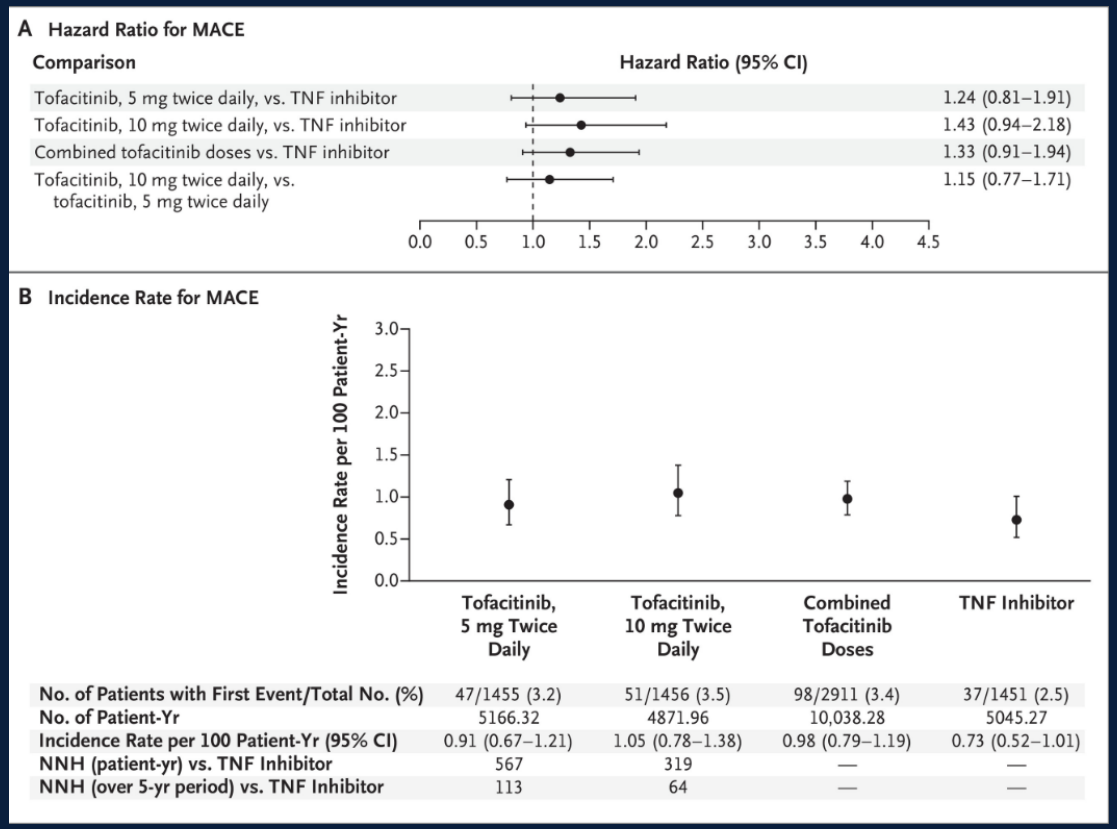
12.4%

herpes zoster with JAKi vs. 4.0% with TNFi

JAK Inhibitors: The ORAL Surveillance Signal

PALTC-critical

1. Trial: age ≥50 + ≥1 CV risk factor, tofacitinib vs. TNFi, median ~4 yr
2. MACE HR 1.33; malignancy (excl. NMSC) HR 1.48; dose-dependent VTE; more zoster
3. Excess risk concentrated in ≥65 yr, prior ASCVD, ever-smokers
4. → FDA boxed warnings on all JAK inhibitors; restricted to inadequate response/intolerance to ≥1 TNFi



Hazard ratios and incidence rates for adjudicated cancers (excl. NMSC), tofacitinib vs. TNFi.

Ytterberg et al. (ORAL Surveillance), N Engl J Med. 2022.

SECTION

08

Treatment Strategy

Slides 25–29

Treat-to-Target

1. Target: remission (or low disease activity if remission not feasible)
2. Reassess q1–3 mo; require ≥50% improvement by 3 mo; target attainment by 6 mo, then adjust
3. Correct sequential strategy → low disease activity/remission in >75–80%

>75–80%

reach LDA/remission with correct sequential strategy

Grading RA Severity: Composite Disease-Activity Measures

Composite indices — not any single lab test — quantify disease activity; the 2019 ACR update endorses CDAI, DAS28-ESR/CRP, SDAI, RAPID3, and PAS-II for routine use.

Feature	RAPID3	DAS28-ESR
Component 1	Physical function (MDHAQ: 10 activities, 0–3 each, converted to 0–10)	Tender joint count (28 joints)
Component 2	Pain (VAS 0–10)	Swollen joint count (28 joints)
Component 3	Patient global assessment (VAS 0–10)	ESR (mm/hr)
Component 4	— (none)	Patient global health assessment (VAS)
Provider joint count required?	No	Yes (28 tender + 28 swollen)
Lab required?	No	Yes (ESR)
Total score range	0–30 (sum of three 0–10 items)	0–9.4
Time to score	~5–10 seconds	~114 seconds

Disease-Activity Cut Points: Remission, Low, Moderate, High

Pick one instrument and use it consistently — the trend within a patient matters more than cross-instrument comparison.

Measure (range)	Remission	Low	Moderate	High
DAS28-ESR (0–9.4)	<2.6	2.6–<3.2	3.2–5.1	>5.1
DAS28-CRP (0–~9)	<2.6*	2.6–3.2	>3.2–5.1	>5.1
CDAI (0–76)	≤2.8	>2.8–10	>10–22	>22
SDAI (0–86)	≤3.3	>3.3–11	>11–26	>26
RAPID3 (0–30)	≤3	3.1–6	6.1–12	>12
PAS / PAS-II (0–10)	0–0.25	>0.25–3.7	>3.7–<8.0	≥8.0

What Counts as DMARD Failure?

Treat-to-Target Definitions

1. <50% improvement by 3 mo, or missing the target by 6 mo → change therapy
2. Reassess active disease every 1–3 months — escalate promptly
3. ≥2 csDMARD failures → advance to a bDMARD or JAKi (MTX alone fails in up to ~50%)
4. Primary non-response (no benefit by 3–6 mo) vs. secondary non-response (loss of benefit, often anti-drug antibodies)
5. 50–60% miss target on the first DMARD course; response rates fall with each successive failure

50–60%

do not meet target on the first DMARD course

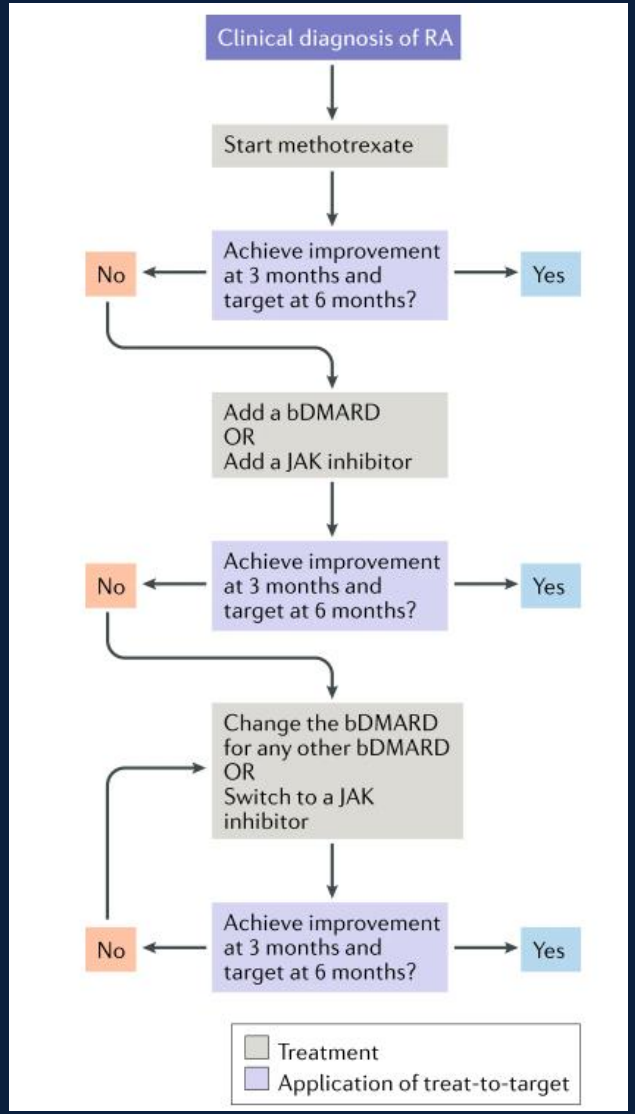
Escalation After Failure & Difficult-to-Treat RA

1. csDMARD failure: stratify by poor-prognostic factors → add a bDMARD (preferred) or JAKi; 2021 ACR favors b/tsDMARD over triple therapy for faster onset

2. After a first TNFi failure: switch mechanism (IL-6R, abatacept, rituximab, JAK) or cycle to another TNFi — a second TNFi can be as effective as changing class

3. Registries: switching first-line targeted therapy when LDA isn't reached by 6 mo beats a conservative “stay-the-course” approach

4. D2T RA (EULAR 2021, all 3): ≥2 b/tsDMARD failures of different mechanisms + active/progressive disease + management perceived as problematic



Treat-to-target algorithm: MTX → add bDMARD/JAKi → switch on failure at the defined 3- and 6-month decision points.

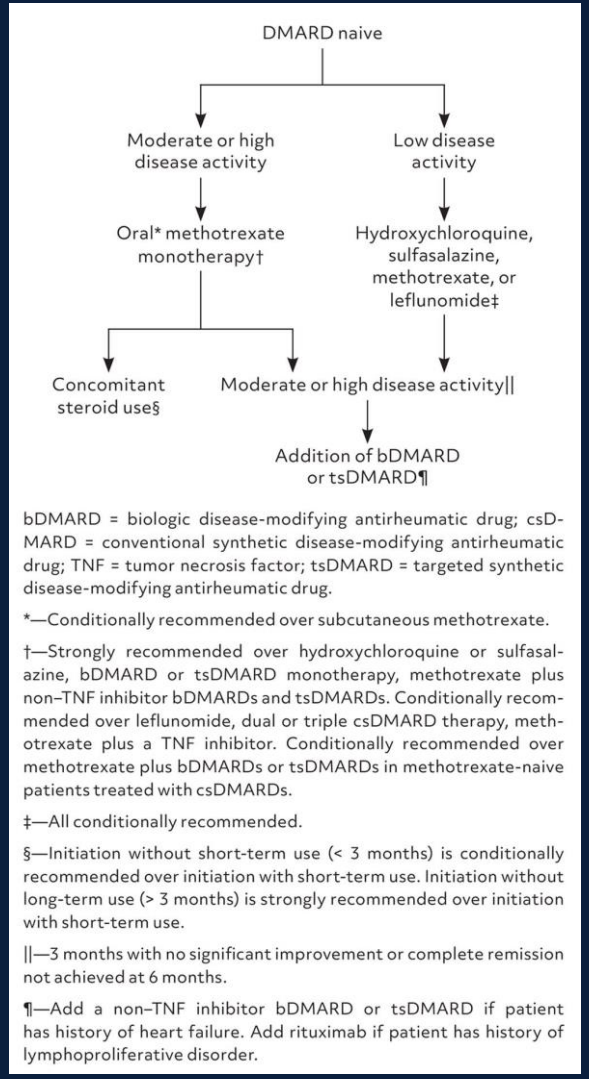
van Vollenhoven R, Nat Rev Rheumatol. 2019.

Step-Up Therapy After Treatment Failure

1. Phase 1 (naïve): start MTX; EULAR = short-term bridging steroids tapered off by ~3 mo; minimize steroids in PALTC

2. Phase 2 (csDMARD failure): stratify by poor-prognosis factors → add bDMARD (preferred) or JAKi after risk assessment

3. Phase 3 (first b/tsDMARD failure): EULAR permits same-class cycling; ACR favors switching mechanism after TNFi failure



ACR treatment algorithm for RA in adults.
Peterson et al., Am Fam Physician. 2024.

Sustained Remission and Tapering

1. After sustained remission: taper, do not stop — hierarchy: continue > dose-reduce > gradually discontinue > abruptly discontinue

2. Complete withdrawal → flare in majority; ~40–60% maintain control after dose reduction; ~90% regain control on restarting

3. ACR tapers MTX first; EULAR tapers b/tsDMARD first

~90%

regain control after restarting therapy

Drug Monitoring: Before and During Therapy

1. Before MTX: CBC, ALT/AST, Cr, chest radiograph, HBV serologies, FIB-4
2. Before any b/tsDMARD: latent TB (IGRA), HBV (HBsAg + anti-HBc), HCV
3. Highest reactivation risks: rituximab → HBV; TNFi → TB
4. csDMARD/JAKi/IL-6i lab schedules per prior slides

Screen before you suppress

Vaccination (2022 ACR Guideline)

1. Pneumococcal and recombinant zoster (Shingrix, non-live) strongly recommended on immunosuppression — especially with JAK/IL-6 inhibitors

2. Give non-influenza vaccines ≥6 mo after rituximab; avoid live vaccines on immunosuppression

Vaccinate before you immunosuppress

SECTION

09

Special Populations

PALTC Focus — Slides 30–34

Difficult-to-Treat (D2T) RA

1. 2021 EULAR definition (all 3): failure of ≥2 b/tsDMARDs of different mechanisms; active/progressive disease; management perceived as problematic
2. Prevalence ~11.7%
3. PIRRA (persistent inflammatory refractory) vs. NIRRA (non-inflammatory refractory — cycling futile)
4. First verify diagnosis and confirm objective inflammation; IL-6R inhibitors and JAKi show superior retention in D2T-RA

~11.7%

prevalence of difficult-to-treat RA

DMARD Dosing in Renal Impairment

CKD + MTX → ~2 ✕ hospitalization for myelosuppression/sepsis/hepatotoxicity; watch fatal daily-instead-of-weekly dosing errors in cognitively impaired elders.

Agent	Renal dosing guidance
Methotrexate	Reduce if eGFR <60; avoid <30
Leflunomide	Caution; avoid loading dose in elderly
Hydroxychloroquine	≤5 mg/kg/day; caution severe CKD
Baricitinib	eGFR 30–60: 1 mg daily; <30: not recommended
Tofacitinib	eGFR <50: reduce dose 50%
Upadacitinib	No adjustment for any renal impairment
bDMARDs (TNFi, abatacept, tocilizumab, rituximab)	No adjustment — not renally cleared

Deprescribing and Tapering in the Elderly

1. Taper after remission/LDA sustained ≥6 mo
2. Dose reduction: ↑risk of losing remission (RR 1.23) but not LDA; no meaningful radiographic progression
3. Complete discontinuation >2× flare risk (RR 2.13); ~87.5% recapture control on re-treatment; tapering does not increase serious infection
4. Predictors of success: ACPA-negative, deep sustained remission, concomitant csDMARD

RR 2.13

flare risk with complete discontinuation

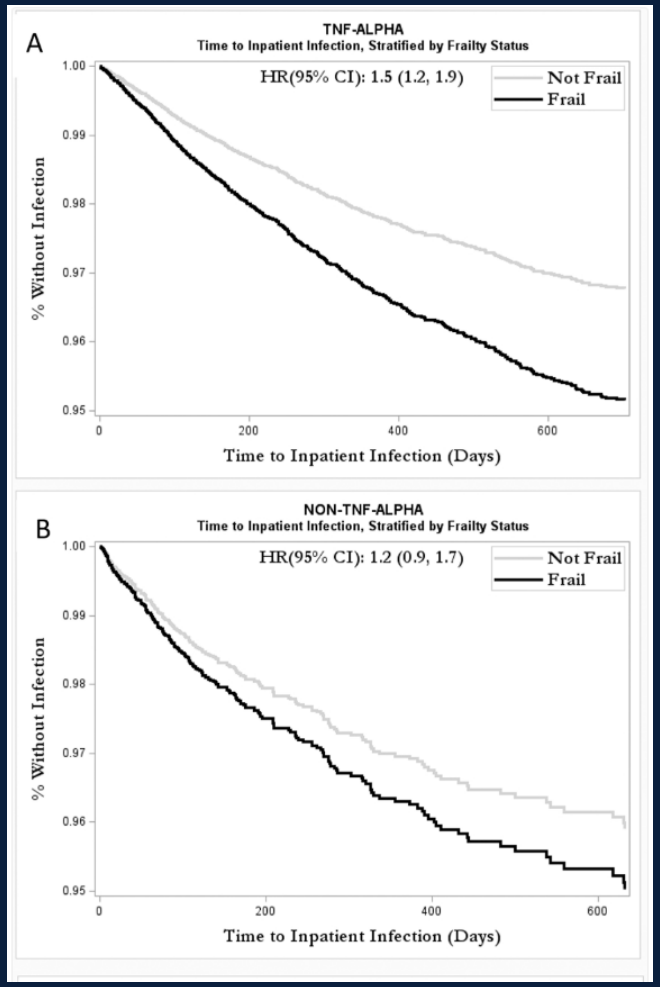
Frailty, Polypharmacy, and Agent Selection

1. Frailty independently predicts MTX discontinuation for AEs (HR 2.34) and ↑serious infection on b/tsDMARDs

2. Abatacept: lowest AE discontinuation, lower hospitalized-infection risk than TNFi → favored in infection-prone elders

3. Polypharmacy: each added drug ↓odds of biologic response; watch MTX + trimethoprim/NSAIDs/PPIs

4. Glucocorticoid restraint even <5 mg/day (infection, osteoporosis, diabetes, delirium); structured review: Beers, STOPP/START



Risk of serious infections, frail vs. non-frail patients by drug class (TNFi highest).

Singh et al., Arthritis Care Res. 2024.

Treat-to-Target Modifications by Age

1. <75 yr: standard remission-targeted algorithm

2. ≥75 yr: initial target = low disease activity (escalate only if safety permits)

3. Use CDAI or SDAI rather than DAS28-ESR (ESR age-elevated); monitor more frequently for AEs and frailty

Same target, gentler pace after 75

SECTION

10

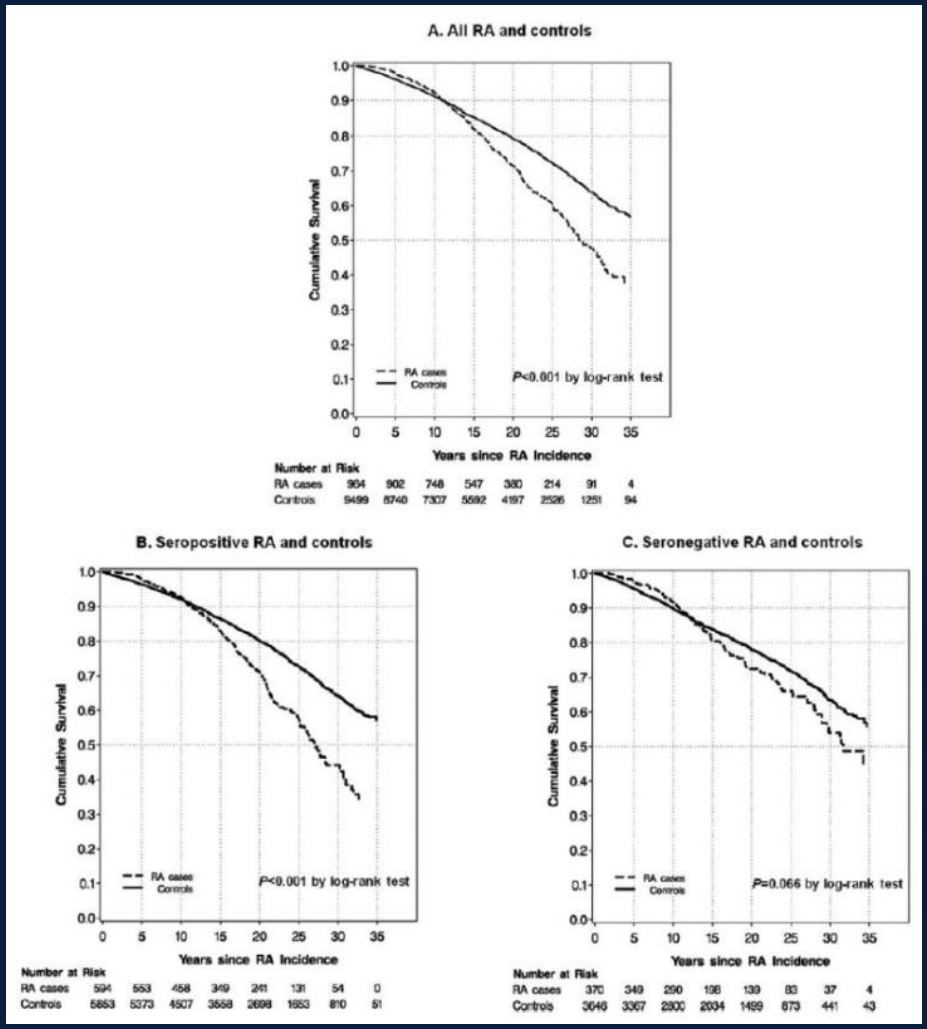
Prognosis & Complications

Slides 35–41

Mortality and Prognosis

1. Excess mortality ~1.4–1.6×; life expectancy shortened ~6–7 yr; driven by seropositive disease

2. Persistently high activity (DAS28 >5.1) ~doubles mortality (HR 2.43) → tight control is protective



Kaplan–Meier survival curves — mortality excess confined largely to seropositive RA.

Sparks et al. (Nurses' Health Study), Arthritis Care Res. 2016.

Cardiovascular Disease

1. Leading cause of death (30–40%); ~1.5–2× coronary risk, 68% ↑MI, 41% ↑stroke, ~2× heart failure

2. EULAR: apply 1.5× multiplier on CV risk scores; “lipid paradox” (active RA lowers cholesterol, rises with treatment)

3. Recently diagnosed patients approach non-RA CV outcomes with tighter control

68%

increase in myocardial infarction risk

Infection

1. ~2× serious-infection risk; amplified by glucocorticoids (dose-dependent, up to ~9×), TNFi (TB), rituximab (HBV), JAKi (zoster)

2. Elderly nonserious infections: 47.5 per 100 patient-years — central concern in congregate PALTC settings

~2x

serious infection risk vs. general population

Malignancy

1. Modest ~20% overall increase

2. Lymphoma 2–2.5× (DLBCL predominant), driven by cumulative inflammatory activity more than treatment; lung cancer ~1.4–1.7×

2–2.5×

lymphoma risk (DLBCL predominant)

Osteoporosis and Fracture

1. Prevalence ~30–50% (2× general population)
2. RA independently ↑fracture risk (HR 1.49 any; 2.23 hip); embedded in FRAX®; glucocorticoids compound
3. High-yield PALTC intervention target

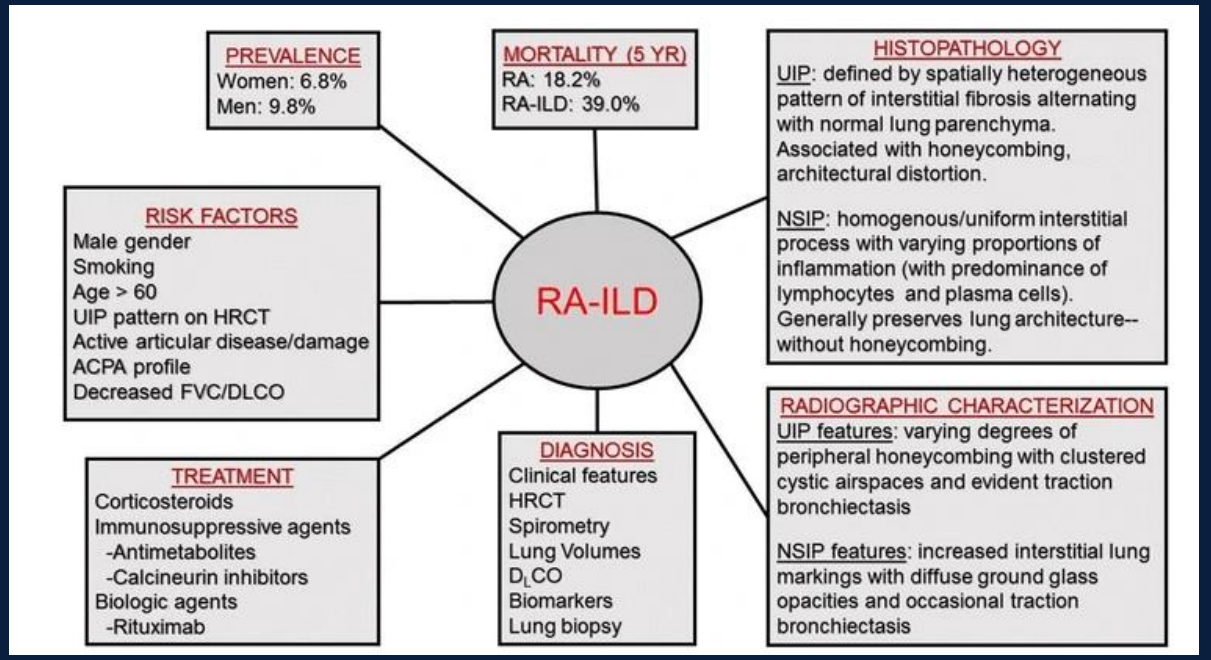
30–50%

osteoporosis prevalence in RA

RA-Associated ILD

1. Clinically apparent ~10% (subclinical 20–60%); poor prognosis (median survival ~2.6–8 yr; ~3× mortality vs. RA without ILD; UIP worst)

2. Respiratory disease may now rival CVD as leading contributor to excess mortality



Overview of RA-ILD: epidemiology, diagnosis, management.

Brito et al., Curr Rheumatol Rep. 2017.

Function in Older Adults

1. Late-onset RA (≥65): similar disease-activity improvement but greater functional decline and more AEs (adj HR 4.70 death; 2.58 malignancy)

2. Functional deterioration accelerates 1.71× in 60s–70s and 3.48× in ≥80 (cumulative damage, comorbidity, sarcopenia)

3.48×

faster functional decline at age ≥80

SECTION

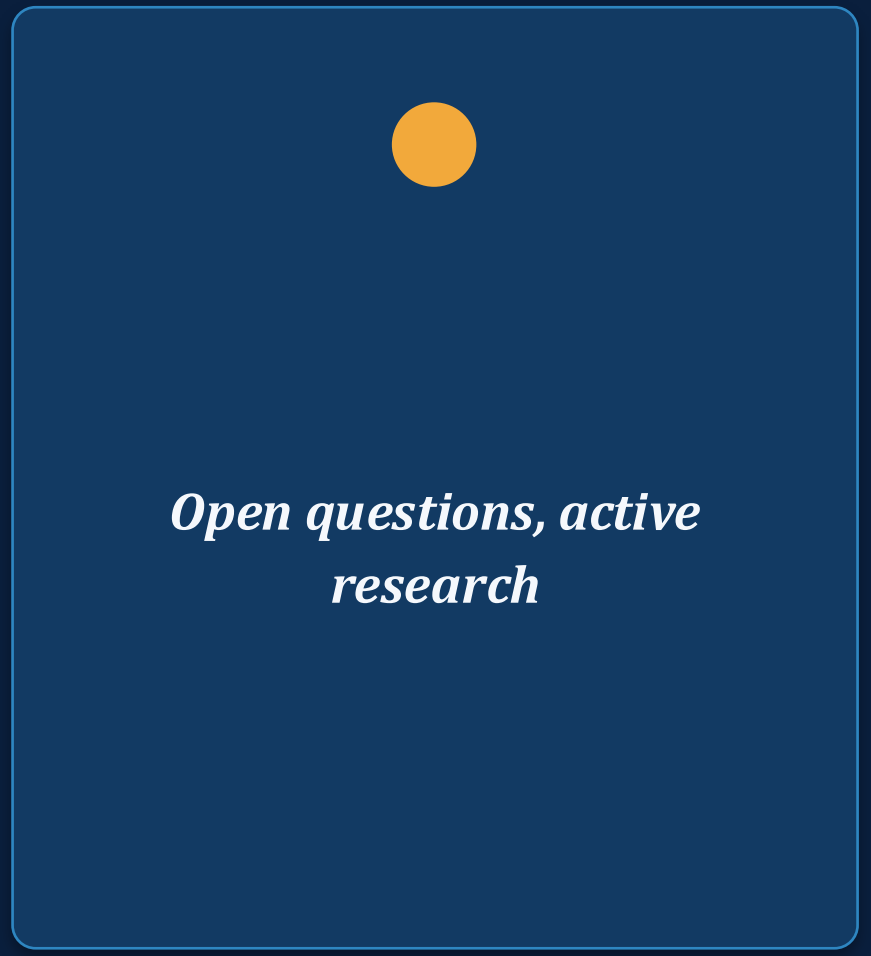
11

Gaps & Summary

Slides 42–44

Evidence Gaps and Emerging Data

1. Is the ORAL Surveillance signal a class effect or tofacitinib-specific? (registries reassuring)
2. Lack of head-to-head JAKi trials and robust sequencing data after multiple bDMARD failures
3. No validated biomarkers to predict individual DMARD response; no RA-specific osteoporosis guidance; no routine ILD screening standard
4. Prevention/interception trials (abatacept promising; TREAT EARLIER MTX modified symptoms but did not prevent progression)



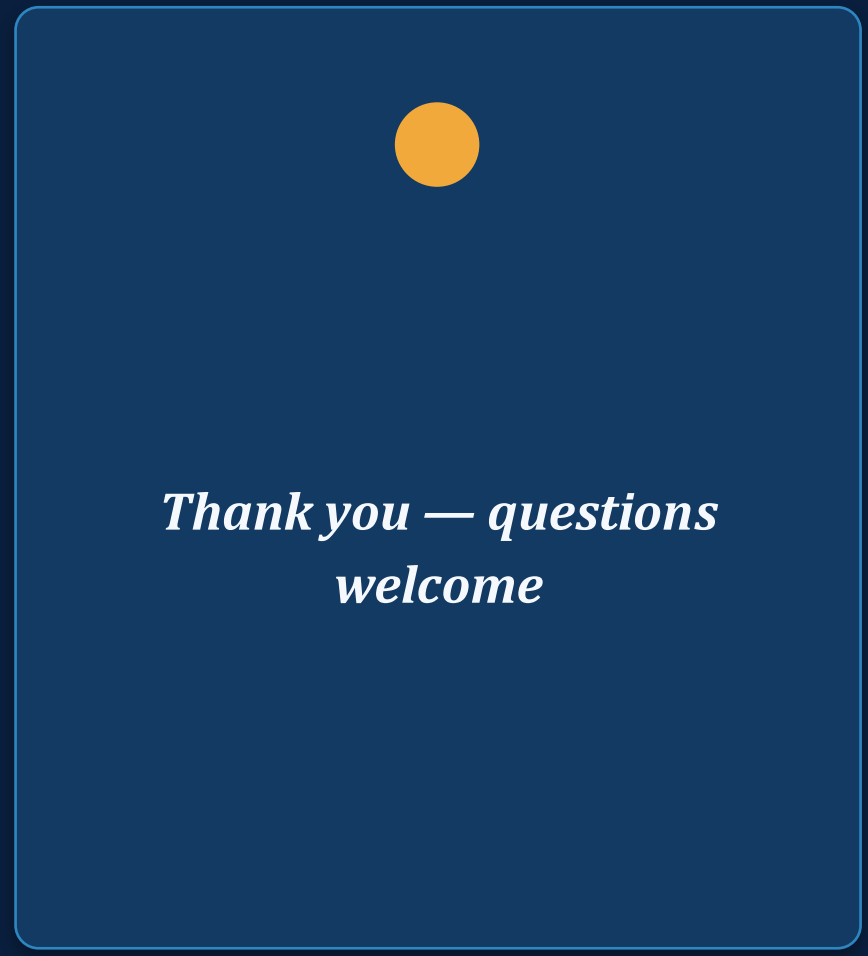
Summary / Take-Home Points

1. RA = systemic, ACPA-driven, TNF/IL-6–dependent disease; remission/LDA now attainable in most patients

2. PALTC throughline: tight inflammatory control prevents the lethal complications (CVD, ILD, lymphoma, disability) — the drugs demand age-attentive safety

3. Minimize steroids, screen/vaccinate before biologics, weigh JAKi CV/malignancy risk in ≥65 or smokers

Control the fire. Protect the patient.

Questions / Discussion

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