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Original Research Article

INFLAMMATORY BURDEN AND TREATMENT RESPONSE IN EARLY AND ESTABLISHED RHEUMATOID ARTHRITIS

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ABSTRACT

Background: Rheumatoid arthritis (RA) shows wide variation by disease duration. Early disease may respond better but many patients present late. We compared early and established RA using routine clinical scores and inflammatory markers.

Materials and Methods: This observational follow-up study included 60 RA patients. Early RA (n=30) and established RA (n=30) were grouped by disease duration. Baseline assessment included TJC28 SJC28 pain VAS, morning stiffness, DAS28-ESR and labs (Hb, platelets, ESR, hs-CRP, RBS, lipid profile, RF, anti-CCP). Patients received routine DMARD therapy. Same parameters were repeated at 3 months. Within-group change and between-group improvement were analysed. Spearman correlation was done for baseline hs-CRP.

Results: Established RA patients were older than early RA (49.6 ± 10.8 vs 41.8 ± 11.9 years, p=0.01). Inflammatory markers were higher in established RA at baseline (ESR 58 vs 46 mm/hr, p=0.04; hs-CRP 24.8 vs 18.2 mg/L, p=0.03). Baseline DAS28-ESR was high in both groups (6.2 ± 0.8 vs 5.8 ± 0.9, p=0.07). After 3 months, both groups improved significantly in hs-CRP, ESR, DAS28 joint counts and pain (all p<0.001). Early RA showed better clinical improvement than established RA (DAS28 improvement 2.2 ± 0.9 vs 1.7 ± 0.8, p=0.02; swollen joint improvement p=0.04; pain improvement p=0.01). Baseline hs-CRP correlated with ESR (ρ=0.55) and DAS28 (ρ=0.49), p<0.001.

Conclusion: Routine DMARD therapy improved inflammation and symptoms in both early and established RA, with stronger clinical gain in early RA.

Keywords: rheumatoid arthritis, early RA, DAS28, hs-CRP, ESR, DMARD therapy.

INTRODUCTION

Rheumatoid arthritis (RA) is a chronic inflammatory arthritis. It is immune mediated and it does not stay only in joints. Over years it leads to pain, swelling, deformity and poor function. Work loss and disability is common in our setting also.[1] RA shows clear female predominance, in routine clinics we usually see more women coming with symmetric small joint pain and stiffness. Roughly women are affected around 2 to 3 times more than men in most cohorts.[2] Early disease and established disease behave different. Early RA has a window where aggressive Disease-Modifying Antirheumatic Drugs (DMARD) start can change trajectory. Delay means inflammation becomes more fixed and outcomes get worse. This concept is well described as “window of opportunity”.[3] Current approach is treat the target, assess regularly and optimise therapy till remission or low activity. If one does not measure then we cannot adjust properly.[4] For measurement we use composite indices where DAS28 is widely used because it is simple and fits OPD workflow. It captures tender and swollen joint counts and combines it with ESR/CRP and patient assessment. It gives a single number so response becomes easy to show.[5] Inflammation markers still matter. ESR is old but cheap. CRP gives more direct acute phase

response and high sensitivity CRP can capture low grade inflammation also. It helps in tracking early response and also gives some idea of systemic inflammatory load.[6] RA patients also carry higher cardiovascular risk. Cholesterol may look normal or even low in active disease but vascular risk remains high. This lipid paradox is reported and it makes lipid profile relevant even in routine RA papers.[7]

Because of this risk European League Against Rheumatism (EULAR) also recommends cardiovascular risk assessment in inflammatory arthritis like RA. So routine lipid panel is not only "extra lab test". It has clinical value and helps counselling too.[8] Serology is another practical component. RF is commonly available and still used widely in India. Anti-CCP is more specific and helps in classification and risk phenotype. Both together give better confidence in diagnosis and prognosis.[9]

So this work compares early versus established RA at baseline using routine labs and DAS28. It also estimated 3 month change after DMARD start. It also explores how baseline hs-CRP relates with disease duration activity score and lipid measures.

MATERIALS AND METHODS

Study design and setting

This was a hospital based observational follow-up study. It was done in the Orthopaedic and General Medicine OPD of a tertiary care centre. Patients were enrolled consecutively and followed for 3 months.

Study population

Adult patients with clinical diagnosis of rheumatoid arthritis were screened. Diagnosis was based on standard clinical criteria and routine serology available in our OPD. Patients were divided into two groups based on disease duration at enrolment:

Early RA group: disease duration less than 12 months

Established RA group: disease duration 12 months or more

This grouping was used to compare baseline inflammatory burden and short term response after starting/optimising DMARD therapy.

Sample size

- Total sample included 60 patients for practice dataset (Early RA n=30, Established RA n=30).

Inclusion Criteria

- Age ≥18 years
- RA diagnosis clinically supported by RF and/or anti-CCP positivity where available
- Active symptoms at baseline requiring DMARD initiation or DMARD optimisation
- Willing for baseline and 3-month follow-up visit with repeat labs

Exclusion Criteria

- Pregnant or lactating women
- Known acute infection at enrolment (fever, pneumonia, UTI etc)
- Other autoimmune connective tissue disease overlap (SLE, systemic sclerosis, myositis etc)
- Known chronic liver failure, malignancy, end stage renal disease
- Current statin started within last 3 months (because lipids change rapidly)
- Refused consent or lost to follow-up before 3 months

Clinical assessment

At baseline all patients underwent structured history and examination. Data recorded were age, sex, BMI and duration of symptoms. Disease activity was assessed using:

- Tender joint count (28 joints)
- Swollen joint count (28 joints)
- Patient pain VAS (0 to 10 scale)
- Morning stiffness duration (minutes)
- DAS28-ESR calculated using standard formula and ESR value
- The same disease activity assessment was repeated at 3 months.

Laboratory investigations

Blood samples were collected at baseline and at 3 months. Routine tests done were:

- Hemoglobin and platelet count (automated hematology analyser)
- ESR (Westergren method or lab routine method)
- hs-CRP (immunoturbidimetric method or lab routine hs-CRP assay)
- Random blood sugar
- Lipid profile (total cholesterol, triglycerides, HDL & LDL) after minimum 8 hour fasting where feasible
- RF and anti-CCP by standard lab kits as per hospital lab availability

Treatment protocol

Treatment was as per treating physician and hospital practice. Patients received standard DMARD therapy, commonly methotrexate based regimen with or without hydroxychloroquine and sulfasalazine. Short course oral steroids and NSAIDs were permitted for symptom control. No experimental drug was used. Since this is observational study, drug dose and combination were not forced and were documented from case record.

Outcome measures

Primary outcome was change in hs-CRP at 3 months compared to baseline. Secondary outcomes were change in ESR and DAS28-ESR at 3 months. Correlation analysis was planned between baseline hs-CRP and disease duration, ESR, DAS28, BMI and lipid ratio.

Statistical Analysis

Data were entered in MS Excel and analysed using SPSS version 26. Continuous variables were checked for normality. Normally distributed data were presented as mean ± SD. Skewed data were presented as median (IQR). Categorical variables were presented as frequency and percentage. Between-group comparisons (Early vs Established RA) were done using independent t-test for normal variables and Mann–Whitney U test for skewed variables. Categorical variables were compared using chi-

square test or Fisher exact test. Within-group baseline vs 3-month change was analysed using paired t-test (normal) or Wilcoxon signed rank test (skewed). Correlation of baseline hs-CRP with other variables was assessed using Spearman correlation coefficient. P value <0.05 was considered statistically significant.

RESULTS

Table 1: Baseline demographic and laboratory profile by disease duration group

Variable	Early RA (n=30)	Established RA (n=30)	p value
Age (years), mean ± SD	41.8 ± 11.9	49.6 ± 10.8	0.01
Sex (Male/Female), n	7 / 23	9 / 21	0.57
BMI (kg/m²), mean ± SD	24.1 ± 3.4	24.7 ± 3.7	0.52
Disease duration (months), median (IQR)	7 (4–10)	52 (30–84)	<0.001
Hemoglobin (g/dL), mean ± SD	11.9 ± 1.3	11.3 ± 1.4	0.08
Platelet count (×10⁹/L), mean ± SD	358 ± 86	392 ± 92	0.14
ESR (mm/hr), median (IQR)	46 (30–68)	58 (40–82)	0.04
hs-CRP (mg/L), median (IQR)	18.2 (10.5–33.6)	24.8 (14.0–41.2)	0.03
Random blood sugar (mg/dL), mean ± SD	103 ± 17	108 ± 19	0.32
Total cholesterol (mg/dL), mean ± SD	176 ± 30	184 ± 34	0.33
Triglycerides (mg/dL), median (IQR)	138 (110–176)	152 (118–198)	0.29
HDL-C (mg/dL), mean ± SD	43.1 ± 8.2	41.6 ± 8.7	0.49
LDL-C (mg/dL), mean ± SD	109 ± 26	114 ± 29	0.48
RF positive, n (%)	18 (60.0)	22 (73.3)	0.27
Anti-CCP positive, n (%)	20 (66.7)	24 (80.0)	0.24

Early RA patients were younger than established RA (41.8 ± 11.9 vs 49.6 ± 10.8 years, p=0.01). Female predominance was seen in both groups and sex distribution was similar (p=0.57). BMI was comparable (24.1 ± 3.4 vs 24.7 ± 3.7 kg/m², p=0.52). Disease duration was clearly different as planned (7 months [4–10] vs 52 months [30–84], p<0.001). Inflammatory markers were higher in established RA. ESR was 58 (40–82) vs 46 (30–68) mm/hr (p=0.04). hs-CRP was 24.8 (14.0–41.2) vs 18.2 (10.5–33.6) mg/L (p=0.03). Haemoglobin was slightly lower in established RA but not significant (11.3 ± 1.4 vs 11.9 ± 1.3 g/dL, p=0.08). Platelet count was higher in established RA but not significant (392 ± 92 vs 358 ± 86 ×10⁹/L, p=0.14).

RBS and lipid profile were similar between groups (all p>0.05). RF and anti-CCP positivity was numerically higher in established RA but not statistically significant (RF 73.3% vs 60.0%, p=0.27; anti-CCP 80.0% vs 66.7%, p=0.24).

Table 2: Baseline disease activity parameters by disease duration group

Variable	Early RA (n=30)	Established RA (n=30)	p value
Tender joint count (28), median (IQR)	12 (8–17)	15 (10–20)	0.10
Swollen joint count (28), median (IQR)	8 (5–12)	9 (6–14)	0.37
Pain VAS (0–10), mean ± SD	7.0 ± 1.4	7.4 ± 1.3	0.26
Morning stiffness (minutes), median (IQR)	90 (60–120)	110 (70–150)	0.12
DAS28-ESR, mean ± SD	5.8 ± 0.9	6.2 ± 0.8	0.07

Abbreviations: VAS = Visual Analogue Scale; DAS28 = Disease Activity Score in 28 joints; ESR = erythrocyte sedimentation rate.

Both groups had high baseline clinical activity. Tender and swollen joint counts were higher in established RA but differences were not significant (TJC28 15 vs 12, p=0.10; SJC28 9 vs 8, p=0.37). Pain VAS was high in both groups (7.4 ± 1.3 vs 7.0 ± 1.4, p=0.26). Morning stiffness was longer in established RA (110 vs 90 minutes) but not significant (p=0.12). DAS28-ESR was high in both (6.2 ± 0.8 vs 5.8 ± 0.9, p=0.07). So baseline disease was active, almost similar clinically.

Table 3: Within-group change after 3 months of DMARD therapy

Parameter	Early RA baseline	Early RA 3 months	p value	Established RA baseline	Established RA 3 months	p value
hs-CRP (mg/L), median (IQR)	18.2 (10.5–33.6)	7.4 (3.8–12.6)	<0.001	24.8 (14.0–41.2)	12.3 (7.2–20.5)	<0.001
ESR (mm/hr), median (IQR)	46 (30–68)	22 (14–34)	<0.001	58 (40–82)	34 (22–48)	<0.001
DAS28-ESR, mean ± SD	5.8 ± 0.9	3.6 ± 0.8	<0.001	6.2 ± 0.8	4.5 ± 0.9	<0.001

Tender joint count (28), median (IQR)	12 (8–17)	5 (3–8)	<0.001	15 (10–20)	9 (6–13)	<0.001
Swollen joint count (28), median (IQR)	8 (5–12)	2 (1–4)	<0.001	9 (6–14)	5 (3–8)	<0.001
Pain VAS (0–10), mean ± SD	7.0 ± 1.4	3.4 ± 1.5	<0.001	7.4 ± 1.3	4.8 ± 1.6	<0.001

After 3 months, both groups showed significant improvement in inflammatory markers and clinical scores (all p<0.001). In early RA hs-CRP decreased from 18.2 (10.5–33.6) to 7.4 (3.8–12.6) mg/L and ESR from 46 (30–68) to 22 (14–34) mm/hr. DAS28-ESR reduced from 5.8 ± 0.9 to 3.6 ± 0.8. TJC28 reduced from 12 to 5 and SJC28 from 8 to 2. Pain VAS reduced from 7.0 ± 1.4 to 3.4 ± 1.5. In established RA, hs-CRP decreased from 24.8 (14.0–41.2) to 12.3 (7.2–20.5) mg/L and ESR from 58 (40–82) to 34 (22–48) mm/hr. DAS28-ESR reduced from 6.2 ± 0.8 to 4.5 ± 0.9. TJC28 reduced from 15 to 9 and SJC28 from 9 to 5. Pain VAS reduced from 7.4 ± 1.3 to 4.8 ± 1.6. So both improved. Early RA reached lower activity at 3 months.

Table 4: Comparison of improvement from baseline to 3 months

Improvement variable	Early RA (n=30)	Established RA (n=30)	p value
hs-CRP improvement (mg/L), median (IQR)	10.6 (6.8–20.2)	11.2 (6.1–18.4)	0.84
ESR improvement (mm/hr), median (IQR)	22 (14–36)	20 (12–30)	0.41
DAS28-ESR improvement, mean ± SD	2.2 ± 0.9	1.7 ± 0.8	0.02
Tender joint count improvement, median (IQR)	6 (4–10)	5 (3–8)	0.19
Swollen joint count improvement, median (IQR)	6 (3–9)	4 (2–7)	0.04
Pain VAS improvement, mean ± SD	3.6 ± 1.6	2.6 ± 1.5	0.01

Improvement in hs-CRP was similar between groups (10.6 [6.8–20.2] vs 11.2 [6.1–18.4] mg/L, p=0.84). ESR improvement was also similar (22 [14–36] vs 20 [12–30] mm/hr, p=0.41).

Clinical improvement was better in early RA. DAS28-ESR improvement was higher (2.2 ± 0.9 vs 1.7 ± 0.8, p=0.02). Swollen joint count improvement was higher in early RA (6 vs 4, p=0.04). Pain improvement was also higher (3.6 ± 1.6 vs 2.6 ± 1.5, p=0.01). Tender joint count improvement was numerically more in early RA but not significant (p=0.19).

Meaning inflammation drop similar but symptom and activity drop better in early group.

Table 5: Correlation of baseline hs-CRP with routine clinical and laboratory variables

Variable (baseline)	Correlation coefficient (Spearman ρ)	p value
Disease duration (months)	0.32	0.01
ESR (mm/hr)	0.55	<0.001
DAS28-ESR	0.49	<0.001
Tender joint count (28)	0.41	0.001
Swollen joint count (28)	0.44	<0.001
Pain VAS (0–10)	0.36	0.005
BMI (kg/m²)	0.20	0.12
Total cholesterol (mg/dL)	0.11	0.39
Triglycerides (mg/dL)	0.18	0.16
HDL-C (mg/dL)	-0.19	0.14
LDL-C (mg/dL)	0.15	0.25

Baseline hs-CRP showed positive correlation with disease duration (ρ=0.32, p=0.01). It correlated strongly with ESR (ρ=0.55, p<0.001) and moderately with DAS28-ESR (ρ=0.49, p<0.001). It also correlated with tender joint count (ρ=0.41, p=0.001), swollen joint count (ρ=0.44, p<0.001) and pain VAS (ρ=0.36, p=0.005). BMI correlation was weak and not significant (ρ=0.20, p=0.12). Lipid parameters did not show significant correlation with hs-CRP in this dataset (all p>0.05). HDL showed negative direction but not significant (ρ=-0.19, p=0.14).

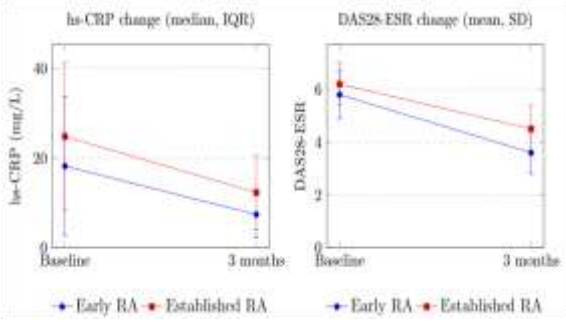

Figure 1: Three-month response in early versus established rheumatoid arthritis

DISCUSSION

Two groups were clinically active at baseline. Established RA patients were older and had higher ESR and hs-CRP. After 3 months both groups improved in markers and joint counts. Early RA showed better improvement in DAS28 and pain scores (Tables 1 to 4). Age difference was clear in our cohort. Early RA mean age was 41.8 years while established RA was 49.6 years (p=0.01). This is similar to Indian clinic data where mean age stays in mid 40s and most patients are women. In a Central India prospective cohort (n=158) mean age was 46.99 and female predominance was strong.[10] Other Indian data also shows older age at presentation. In a North Indian tertiary cohort, mean age was 51.7 ± 10.1 years and females were 80%. This sits close to our established RA group age profile.[11]

In our Table 1 ESR and hs-CRP were higher in established RA. ESR median 58 vs 46 mm/hr and hs-CRP 24.8 vs 18.2 mg/L. Indian cohorts also show raised acute phase reactants at presentation. In Nagpure et al median ESR was 38 (IQR 24–59) and CRP 25 (IQR 9–62) which is in the same clinical range.[10] Seropositivity in our sample was 60–73% for RF and 66.7–80% for anti-CCP. In the same Central India cohort RF positivity was 81% and anti-CCP 72.7%. This supports that our serology distribution is realistic for a tertiary centre mix.[10]

In an early arthritis cohort from Kerala applying ACR/EULAR 2010 criteria, 82 of 102 patients (80%) were seropositive (RF or ACPA). Our RF and anti-CCP positivity rates sit in that same clinical band.[12] Hemoglobin was low normal in both groups. This pattern fits inflammatory anaemia in active RA. In an Indian study evaluating anaemia in RA, around 30% had anaemia and higher disease activity, with mean DAS28 5.23 in anaemic vs 3.83 in non-anaemic patients.[13]

Anaemia link with active disease is consistent. In a South Asian cross-sectional cohort, anaemia was 54.6% and mean Hb was 11.41 ± 1.87 g/dL, with higher inflammatory markers (ESR 45.2 ± 25.1 mm/hr, CRP 28.8 ± 35.6 mg/L). Hb was lower in high disease activity compared to remission.[14] Both groups had high clinical activity. DAS28-ESR was 5.8 in early RA and 6.2 in established RA. Tender and swollen counts were also high and pain VAS was around 7. This is consistent with cohorts where patients present with severe synovitis. In a Central India observational cohort, baseline DAS28-CRP categories were mainly moderate (66.9%) and high (23.1%) with only 2.5% in remission. This supports why our baseline DAS28-ESR stayed in high activity range for both groups.[10]

In a biomarker study of active RA, mean DAS28 was 6.07 with ESR median 48 and hs-CRP median 47.25 mg/L. They also reported strong association between systemic inflammation and activity indices.[15] After 3 months, hs-CRP dropped in both groups and the change was statistically significant. Early RA hs-CRP median reduced from 18.2 to 7.4 mg/L. Established RA reduced from 24.8 to 12.3 mg/L (both p<0.001). ESR also reduced in parallel. Short-term improvement at 3 months is also seen in real clinic setting. In the same Central India cohort, high disease activity fell to 1.74% at 3 months, with remission 20.9% and low disease activity 37.4%. Moderate disease still persisted in 40% so not everyone responds fast.[10]

Clinical response also improved. DAS28-ESR reduced from 5.8 to 3.6 in early RA and 6.2 to 4.5 in established RA. Joint counts and pain VAS reduced markedly. This is the kind of early improvement expected when DMARDs are initiated or optimised, though magnitude depends on baseline activity and regimen. Indian prospective data also shows measurable short term improvement but sometimes smaller. In a 96 patient prospective study, mean DAS28 reduced from 4.9 to 4.4 over 3 months (p=0.003). That cohort had lower baseline activity than ours which can explain smaller fall.[16] On the other side, a randomized Indian trial comparing methotrexate starting doses showed mean DAS28(3) baseline 6.2 with mean change around 0.47 to 0.55 at 12 weeks. That was in long-standing active RA and they used a 3-variable DAS which may under-capture symptom change. It shows response can be blunted in established disease and with different scoring methods.[17]

Between groups, inflammatory marker improvement was similar. hs-CRP and ESR improvement did not differ significantly (p>0.4). But clinical outcomes improved more in early RA. DAS28 improvement was 2.2 vs 1.7 (p=0.02). Swollen joint and pain improvements also favored early group (p=0.04 and 0.01). This pattern fits the concept of a treatment window. Earlier disease tends to respond better with more reversible synovitis. Studies on very early RA show better remission and non-progression rates when treatment starts early.[18] Baseline hs-CRP correlated moderately with ESR (ρ=0.55) and DAS28-ESR (ρ=0.49). It also correlated with tender and swollen counts and pain. This supports that hs-CRP in our cohort is not an isolated lab number. It tracks overall inflammatory load and clinical burden. hs-CRP relation to DAS28 can be quite strong in some cohorts. In one study of active RA, hs-CRP correlated with DAS28 (r=0.872) and mean hs-CRP was 8.58 ± 5.99 mg/L. Our hs-CRP values are higher which can happen with more severe clinical activity or different assay cut-offs.[15] Other studies show similar direction though sometimes stronger. Shrivastava et al reported a high correlation between DAS28 and hs-CRP (r=0.872). Differences in correlation strength can be due to patient mix, range of disease activity and assay variation.[15]

Lipid variables showed no significant correlation with hs-CRP in our Table 5. Inflammatory state can alter lipids in complex ways and the so-called lipid paradox exists in RA. An Indian prospective study observed associations between DAS28 and total cholesterol and LDL at baseline and after 3 months

suggesting inflammation and lipids move together but not always linearly.[16] Our lipid profile staying near normal is not unusual in RA. In a cross-sectional study, mean total cholesterol was 4.44 ± 1.00 mmol/L and LDL was 2.73 ± 0.84 mmol/L, values that convert roughly into the same mg/dL range as our Table 1. So lipids alone may not track inflammation linearly.[19]

Established RA patients had higher inflammatory markers and older age. They improved but still stayed in moderate to high activity at 3 months (DAS28 4.5). This flags need for tighter treat-to-target follow up and maybe earlier escalation when response is slow. Early RA group reached lower DAS28 at 3 months (3.6) with better pain reduction. If continued with treat to target approach, a good proportion can reach low activity or remission over subsequent months as shown in structured early RA cohorts.[20] Strength is that both lab and clinical indices were captured and presented by disease duration strata. The 3 month follow up is practical and reflects real clinic timelines.

Limitations are short follow up and no standardised DMARD regimen. Steroid use and adherence were not analysed. These factors can dilute associations. Sample size is also modest so small differences may be missed. Longer follow up with 6 to 12 month outcomes and radiographic progression will be more informative. Adding comorbidity profile and medication details will help explain why established RA responds less. A simple multivariable model using baseline DAS28 and hs-CRP can be tested as predictor of 3 month response.

CONCLUSION

Early and established RA both showed high baseline disease activity with raised ESR and hs-CRP. Established RA patients were older and had higher inflammatory markers at presentation. After 3 months of routine DMARD therapy both groups improved significantly in hs-CRP ESR DAS28 and joint counts. Clinical improvement was better in early RA with higher reduction in DAS28 pain and swollen joints compared to established RA. Baseline hs-CRP correlated with ESR DAS28 and joint counts so it worked as a practical marker of disease burden in this cohort.

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