Original Research Article

Comparison of comorbidities between rheumatoid and psoriatic arthritis in a tertiary care rheumatic center

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International Archives of Integrated Medicine, Vol. 5, Issue 12, December, 2018.

Available online at http://iaimjournal.com/
ISSN: 2394-0026 (P) **ISSN: 2394-0034 (O)**
Received on: 22-11-2018 **Accepted on:** 26-11-2018
Source of support: Nil **Conflict of interest:** None declared.
How to cite this article: T.N. Tamil Selvam, V.A. Sowndhariya, N.K. Senthil Nathan. Comparison of comorbidities between rheumatoid and psoriatic arthritis in a tertiary care rheumatic center. IAIM, 2018; 5(12): 29-34.

Abstract

Introduction: Rheumatoid arthritis (RA) and psoriatic arthritis (PsA) have key differences in clinical presentation, radiographic findings, comorbidities and pathogenesis to distinguish between these common forms of chronic inflammatory arthritis. Joint involvement is typically, but not always, asymmetric in PsA, while it is predominantly symmetric in RA. Bone erosions, without new bone growth, and cervical spine involvement are distinctive of RA, while axial spine involvement, psoriasis, and nail dystrophy are distinctive of PsA.
Aim of the study: To Compares the comorbidities between Rheumatoid Arthritis (RA) and Psoriatic Arthritis (PSA).
Materials and methods: This was a prospective observational study conducted for a period of 6 months at Institute of Rheumatology, Government K.A.P.V. Medical College and MGM Government Hospital. Totally 241 patients who were fulfilling inclusion criteria has been included. Patients diagnosed with and treated for PsA on the basis of clinical, radiological and laboratory findings and expert opinion was monitored using standardized examination methods and jointly prepared forms. Clinical status, accompanying systemic diseases and surgical history was recorded. Routine clinical examination and basic comorbid findings have been assessed by standardized methods.

Results: Two hundred and forty-one patients were included in the study. 179 patients were diagnosed with RA (male -15, female -164) and 62 were diagnosed with PsA (male - 44, female -18). Comorbid systems such as diabetes, hypertension, metabolic syndrome, cardiovascular disease, transaminitis, malignancy, hyperuricemia were more in RA group when compares to PsA. Which shows statistically significant of p-value <0.005.

Conclusion: Though both Rheumatoid arthritis and Psoriatic arthritis are associated with enhanced cardiovascular morbidity, markers of morbidity are more in the RA group when compared to PsA except that patients with psoriatic arthritis have more hyperuricemia and transaminitis.

Key words

Rheumatoid arthritis (RA), Psoriatic arthritis (PsA), Cardiovascular manifestations, Comorbid symptoms.

Introduction

Rheumatoid arthritis (RA) and psoriatic arthritis (PsA) are common chronic inflammatory diseases; both are characterized by pain and swelling in the joints and have significant systemic manifestations [1]. If not diagnosed and treated early, both can lead to joint destruction with loss of function. For this reason, early diagnosis is important to determine therapeutic strategies that will optimize clinical and radiographic outcomes. Differentiating between RA and PsA can be clinically challenging because there are many similarities in their clinical presentation and manifestations [2]. Both also have similarities with other inflammatory diseases5 and association with prevalent forms of arthritis, such as gout and secondary osteoarthritis. RA is an autoimmune systemic inflammatory disease characterized by synovitis, bony erosions and cartilage damage [3]. PsA is a heterogeneous autoimmune systemic disease with diverse clinical and radiographic manifestations. The presence of psoriasis precedes the development of PsA in 85% of patients, and PsA typically develops about 10 years after the onset of psoriasis [4]. Other common clinical features of PsA include synovitis with subsequent osteolysis and/or joint fusion of peripheral joints, axial involvement, sacroiliitis, and extra-articular manifestations, including nail dystrophy, enthesitis, and dactylitis; not all are present in every patient [5]. A combination of genetic factors and environmental triggers is thought to elicit autoimmune inflammatory responses in both RA and PsA. The pathogenesis of RA and PsA is not completely understood. In addition to the known association with human leucocyte antigen (HLA)-DR4 in RA, one theory is the development of lung inflammation, typically prior to joint symptoms, with the production of antibodies to citrullinated protein antigens that mediate pathogenesis. Gut dysbiosis has been linked with the pathogenesis of PsA [6]. While there is some overlap in the development of inflammation in PsA and RA, some important differences are evident [7]. In both PsA and RA, HLA alleles have been shown to affect disease susceptibility and severity; however, the primary genotypes associated with each disease are different. In PsA, HLA-B27 is associated with the development of enthesitis and symmetric sacroiliitis, and HLA-B08 is associated with joint fusion, asymmetric sacroiliitis and dactylitis [8]. In RA, HLA-DRB1 alleles are associated with disease susceptibility and severity in patients who have positive findings for rheumatoid factor (RF) and cyclic citrullinated peptide (CCP) antibodies [9]. In both PsA and RA, inflammatory responses are characterized by the increased production of proinflammatory molecules that act in synergy to propagate chronic inflammation. In PsA, activated T cells and macrophages induce production of inflammatory chemokines and cytokines, including interleukin (IL)-17, IL-23, IL-22, IL-1β, IL-6, interferon-γ, and tumor necrosis factor-α (TNF-α). Specifically, elevated levels of IL-17

+ CD8+ T cells have been observed in the joints of patients with PsA but not in those with RA [10]. In addition, recent studies have implicated increased expression of IL-9 and its receptor (IL-9R) in the promotion of pathological T-cell growth and subclinical gut inflammation [11].

Materials and methods

This was a prospective observational study conducted for a period of 6 months at Institute of Rheumatology, Government K.A.P.V Medical College and MGM Government Hospital. Totally 241 Patients who were fulfilling inclusion criteria has been included. Patients diagnosed with and treated for PsA on the basis of clinical, radiological and laboratory findings and expert opinion was monitored using standardized examination methods and jointly prepared forms. Clinical status, accompanying systemic diseases and surgical history was recorded. Inclusion Criteria: Consecutive patients with Psoriatic arthritis (CASPAR CRITERIA) and Rheumatoid arthritis (2010 ACR-EULAR criteria). Demographic characteristics and comorbidities at baseline and done again annually were recorded.

Exclusion criteria

- Patients who were not willing for screening/blood investigations.
- Patients who were lost to follow up.
- Patients who were started on drugs for dyslipidemia /antidiabetic drugs before screening for any other reason.

Demographic information and prevalence of comorbidities were summarised using descriptive statistics. Prevalence of comorbidities in PsA and RA were compared using logistic regression, adjusting for age, gender, disease duration, education, employment and prednisone use. Routine clinical examination and basic comorid findings have been assessed by standardized methods.

Statistical analysis

Statistical analysis was performed using the SPSS software Differences between parameters were analyzed using Student's t-test. Categorical parameters analyzed using Chi-square test. Comparisons between groups using odds ratio and 95% confidence intervals were done. p-value of <0.05 was considered significant.

Table – 1: Comparison of gender between rheumatoid arthritis and psoriatic arthritis groups.

Gender	Rheumatoid arthritis	%	Psoriatic arthritis	%
Male	15	8.4%	18	29.0%
Female	164	91.6%	44	71.0%
Total	179	100%	62	100%

**P<0.001

Table – 2: Comorbidities between rheumatoid arthritis and psoriatic arthritis groups.

Comorbidities	Rheumatoid arthritis N=179	Psoriatic arthritis N=62	P value
Diabetes mellitus	32	13	0.818
Systemic hypertension	38	7	0.112
Metabolic syndrome	34	16	0.072
Cardiovascular diseases	18	1	0.998
Transaminitis	3	13	<0.0001
Malignancy	1	1	0.434
Hyperuricemia	12	11	0.01

Results

Two hundred and forty-one patients were included in the study. 179 patients were diagnosed with RA (male - 15, female – 164) and 62 were diagnosed with PsA (male - 44, female – 18). Comorbid systems such as diabetes, hypertension, metabolic syndrome,

cardiovascular disease, transaminitis, malignancy, hyperuricemia were more in RA group when compares to PsA which showed statistically significant of p-value <0.005 (**Table – 1, 2**).

Discussion

For RA, the American College of Rheumatology (ACR)/ European League Against Rheumatism classification criteria were designed for patient characterization and use in clinical trials. The key clinical characteristic is the confirmation of definite, persistent, clinical synovitis in at least one joint [12]. The criteria include the number of joints involved, duration of symptoms, and the demonstration of serological markers and an elevated acute-phase reactant. For PsA, the Classification Criteria for Psoriatic Arthritis help categorize patients with inflammatory articular disease for clinical trials [12]. Key clinical characteristics include a personal or family history of psoriasis, psoriatic nail dystrophy and dactylitis. Neither classification criteria should be confused as diagnostic criteria. Joint involvement is predominantly symmetric in RA and often, but not always, asymmetric in PsA [13]. In both RA and PsA, most patients have polyarthritis (≥5 involved joints), although joint involvement can be oligoarticular or polyarticular. The monoarticular disease is less common in PsA; however, 5%–10% of patients may present with isolated distal joint involvement [14]. In PsA, prognosis worsens and symmetry of joint involvement tends to increase as the number of affected joints increases [15]. Typically, RA affects the shoulder, elbow, wrist, metacarpophalangeal, proximal interphalangeal, hip, knee, ankle, and metatarsophalangeal joints. In PsA, the distal interphalangeal joints of the hands and feet, large joints of the lower extremities, the axial spine and sacroiliac joints are commonly affected; the metacarpophalangeal and metatarsophalangeal joints and wrist can be involved as well. PsA, rather than RA, is included in the spectrum of spondyloarthritis as PsA can affect the axial skeleton (eg, sacroiliac joints and spine) [16]. It is estimated that up to 50% of patients with PsA experience inflammation in the axial skeleton. Axial involvement can be a differentiating feature of PsA because it is not present in RA other than cervical spine involvement, which has been reported in up to 80% of patients with RA. Although TNF-α induces angiogenesis in both RA and PsA, differences in synovial vascularity can help differentiate the diseases. Both RA and PsA exhibit proliferation of endothelial cells, but topological differences in endothelial cells suggest differing pathological features [17]. Among patients with knee synovitis, straight, branching vessels are observed in RA, and predominantly tortuous, bushy vessels are observed in PsA. These physiological differences may be caused by varied patterns of synovial cytokine expression as significantly higher levels of IL-1β, IL-2, IL-10, and IFN-γ are found in PsA synovial explants compared with RA synovial explants [18]. Overall, comorbidity burden may be higher in RA than in PsA, but both diseases are similarly associated with increased risk for comorbidities linked to systemic inflammation. Han and colleagues found that patients with RA and PsA had similarly increased prevalence ratios of ischaemic heart disease, atherosclerosis, peripheral vascular disease, congestive heart failure, cerebrovascular disease, hyperlipidemia, and hypertension compared with healthy controls [19]. However, registry data suggest that the rates of obesity, diabetes mellitus, and metabolic syndrome are significantly higher in patients with PsA compared with those with RA. Notably, most patients with PsA are overweight or obese. Cardiometabolic comorbidities of PsA are associated with higher levels of systemic inflammation and increased disease severity [20].

Conclusion

Though both Rheumatoid arthritis and Psoriatic arthritis are associated with enhanced cardiovascular morbidity, Markers of morbidity including the incidence of Hypertension, Type II DM and Metabolic syndrome are more in the RA Group when compared to PsA Group.

Hyperuricemia, transaminitis are commonly seen in the PsA Group.

Acknowledgments

The authors would like to thank the whole team at the Medicine and Rheumatology Department's Centre of Government K.A.P.V Medical College And MGM Government Hospital, and research center, Trichy for helping with data collection and laboratory analyses.

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