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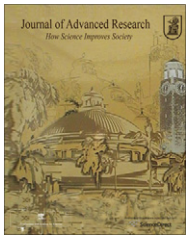

ORIGINAL ARTICLE

Jaccoud's arthropathy in patients with systemic lupus erythematosus: One centre study

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Abstract Jaccoud's arthropathy (JA) is a chronic, deforming, non-erosive arthropathy occurring in a subset of patients with systemic lupus erythematosus (SLE). In this research we aimed to evaluate clinical and immunological features in patients with SLE complicated by JA. Eighty seven consecutive SLE patients with a history of arthritis were included in the present study. These patients were subdivided according to "Jaccoud's arthropathy index" as follows: non-deforming arthropathy, mild deforming and definite Jaccoud. Demographic data, disease activity and disability were recorded. Rheumatoid factor (RF), anti-cardiolipin antibodies (ACL), antiSSA/Ro, and anti SSB/La antibodies, were assessed in all patients. We found clinical deforming arthropathy in 12 patients, among whom seven had definite JA. Both the mean duration of the disease and of arthritis were longer in the JA group compared to the non-deforming arthropathy group. JA patients presented a trend toward a lower quality of life. The prevalence of Sicca syndrome (SS) and antiphospholipid syndrome were significantly higher in the JA group than in the patients with non-deforming arthropathy ($p = 0.011$ and 0.012, respectively). ACL and RF were more frequent

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among patients with JA ($p = 0.013$ and 0.036; respectively). These data suggest that JA is not rare and represents a subset of SLE with specific clinical and serological features. Future studies are needed to reveal the pathogenesis, the genetic association, the prevention, the stabilization and the appropriate cure for these patients.

Introduction

Joint involvement in systemic lupus erythematosus (SLE) is one of the earliest and most common manifestations of this multi-systemic disease [1]. Lupus arthropathy is usually transient, migratory, non-erosive and reversible [2]. Occasionally, it may take a more chronic course, leading to non-erosive joint deformities, although erosive features indistinguishable from rheumatoid arthritis occur rarely [3].

Non-erosive arthropathy with marked articular dislocation or subluxation has been first described by Jaccoud in patients with rheumatic fever. Later investigators have reported complications of Jaccoud's arthropathy (JA) in other rheumatic diseases such as SLE [1–3]. JA has not been adequately evaluated because it is more easily manageable compared to life-threatening involvements such as renal disorders [1].

Given the wide variety of clinical features associated with SLE, there have been many attempts to identify subsets of patients for whom a given antibody specificity can be identified with JA. Several associations, such as the presence of antibodies against U1 RNP, RA 33, SS-A/Ro and SS-B/La, anti cardiolipin (aCL), lupus anticoagulants (LAC), and anti-mutated citrullinated vimentin, have been reported previously [1,4,5]. To our knowledge, no previous studies had dealt with the description of JA and its relation to clinical and immunological profiles among Egyptian SLE patients with arthritis. In that field, well-designed replication studies in populations with different ethnic backgrounds are necessary.

In this research, we aimed to evaluate clinical and immunological features in patients with SLE complicated by JA.

Patients and methods

A group of 87 consecutive patients affected by SLE with a documented history of arthritis were prospectively assessed. They all attended the Department of Rheumatology and Rehabilitation Kasr El-Eini Hospital, Cairo University, and fulfilled the American College of Rheumatology (ACR) criteria for the diagnosis of SLE [6]. There were 83 women and four men with a mean age of 25.07 ± 6.77 years (14–47 years); the mean duration of SLE was 7.20 ± 4.06 years (5–17 years). Disease activity was assessed for all the patients using the SLE disease activity index (SLEDAI) [7]. The definition of JA had been based on clinical criteria (reversible articular deformities) together with the absence of bone erosions on radiographs. These patients were clinically evaluated, underwent a detailed physical examination and had their medical records revised.

Articular evaluation

Physical examination included a detailed standardized examination of the hands and feet. The following items were evaluated in each case: signs of arthritis of wrists and small joints of the hand, ulnar deviation of fingers, MCP subluxation, swan neck deformities of the fingers, Z deformity of the thumb, boutonnière deformities and deformities of the feet. Previous history with special attention to the presenting manifestation of SLE, cumulative ACR criteria, and time between arthritis and the development of deformity, were obtained from medical records. Deforming arthropathy was considered positive if there is deviation from any of the metacarpus finger axes (assessed with an angle goniometer) [8]. Those patients were then assessed for the presence of definite Jaccoud's arthropathy, using a JA index [9], which is dependent upon the different clinical symptoms and the severity of the deformities (details in Table 2). Patients who had scores exceeding five points were considered to have JA. The remaining patients were classified as having mild deforming arthropathy. Assessment of disability was done using the Health Assessment Questionnaire Disability Index (HAQ-DI) [10]. All the patients had recent X-ray film of the hands (postero-anterior view).

Organ system assessment

Clinical features were defined according to the ACR 1982 revised classification criteria for SLE [6]. Neuropsychiatric manifestations were defined according to the ACR nomenclature and case definitions for neuropsychiatric lupus [11]. Renal involvement was defined as glomerulonephritis on biopsy or with diastolic blood pressure >90 mm Hg, edema requiring diuretic therapy, proteinuria >0.5 g/24 h, abnormal urinary sediment manifested by RBC and leukocytes, creatinine clearance <60 ml/min or raised serum creatinine level >124 umol/l. Renal biopsy was available for 22 patients and evaluated according to the World Health Organization (WHO) classification of histological types of lupus nephritis [12]. Antiphospholipid syndrome was considered to be present if at least one of deep venous thrombosis (DVT), arterial thrombosis confirmed by Doppler imaging, or pregnancy morbidity was present, in addition to the presence of LAC and/or aCL [13]. Other organ system affections were defined as previously described [14].

Laboratory and immunological investigations

Routine laboratory examinations were collected from the patients' records. Detection of IgM rheumatoid factor (RF) was done by latex agglutination (Rose-Waaler test), antinuclear antibody (ANA) by indirect immunofluorescence on Hep-2 cells, and anti double stranded DNA (anti-dsDNA) antibody using a modified Farr assay. Anti-Ro/SSA and anti-La/SSB were searched by immunodiffusion (ImmunoConcepts, Sacramento, CA, USA). Anti-cardiolipin antibodies (aCL) were detected by ELISA using commercial kit (ImmunoConcepts, Sacramento, CA, USA). Lupus anticoagulants (LAC) were assessed by the dilute Russell's viper venom time and confirmatory tests [15].

The study was approved by our institutional ethics committee and informed consent was obtained from all the patients.

Statistical analysis

The statistical package for social sciences (SPSS) version 10 (LEAD Technology Inc., Charlotte, NC, USA) was used to analyze the data. Data were statistically described in terms of range, mean ± standard deviation (±SD), frequencies (number of cases) and relative frequencies (percentages) when appropriate. Statistical analysis was performed using the chi square method with Yates' correction, Fisher's exact test. The Student *t*-test was used to compare the differences of the mean of two groups in ordinal variables. A difference was considered to be statistically significant when the probability (p) value was <0.05.

Results

We identified 12 patients with clinical deforming arthropathy of the hands. Among them, seven patients had a JA index greater than five points (>5) and were considered to have definite JA.

The mean age of JA patients was insignificantly different compared to patients with mild deformity and those with non-deforming arthropathy. Both the disease duration and the duration of arthritis were longer in JA patients than in those with non-deforming arthropathy ($p = 0.000$ and 0.04, respectively).

Lupus activity using a SLEDAI score was comparable between the three groups. The mean score of the disability was higher in the JA group compared to the other groups; however, differences were not significant ($p > 0.05$). Demographic data, activity and disability of the three types of arthritis are presented in Table 1.

Arthritis was presented as the initial disease manifestation in six out of seven JA patients. Their early symptoms consisted of morning stiffness and/or minimal to mild arthralgias and the duration from the initial joint symptoms to the typical deformity was 12.5 ± 5.3 years.

The JA index and articular indices of the two types of deforming arthropathy are presented in Table 2. Foot involvement was observed in three patients of the JA group, two had hullux valgus and one had metatarsophalangeal joint sublaxation. Laxity of ligaments, other than those of hands and feet, were not observed in any other joints of the body.

The radiological findings of the wrists in the JA group included joint space narrowing in one patient, cystic changes in two patients, bone irregularity in two patients, and ulnar laxation in one patient. Bone irregularities, cystic changes, and MCP hooks, were found in the fingers of three patients.

In order to evaluate whether JA patients constitute, apart from the presence of deformities, a distinct subgroup within the range of SLE, we compared the clinical characteristics and serological findings of seven patients with definite JA and with those of the group of our SLE population with non-deforming arthropathy (Table 3). Renal affection was present in 52/87 patients of our patient cohort; none of our patients suffered from chronic renal failure. Renal affection was less frequent among the JA group than the non-deforming group; however, the difference was insignificant ($p = 0.08$). The prevalence of Sicca syndrome (SS) and antiphospholipid syndrome were significantly higher in the JA group compared with the patients with non-deforming arthropathy ($p = .0.011$ and 0.012, respectively). ACL and IgM RF were more frequent among patients with JA ($p = 0.013$ and 0.036; respectively). No significant differences were found with respect to other clinical and serological characteristics between the two groups. The group with mild deforming arthropathy did not differ statistically in any aspect (from the SLE patients without deforming arthropathy (data not shown).

Discussion

Joint involvement occurs in the majority of patients with SLE and is one of the initial manifestations, ranging from minor arthralgia to severe deforming arthritis, or so-called JA [1]. JA of the hands was identified in seven (8%) of our series of lupus patients with arthritis. JA is noteworthy to be recognized since it is difficult to manage and may trouble patients' quality of life [16]. The estimated prevalence of JA in the published literature ranged from 3% to 10% according to the population studied and their criteria of patient selection [5,9,16,17]. JA was first described in 1867 in patients with rheumatic fever [18]; later it was observed in patients with SLE [5,9,16,17], in other connective tissue diseases [19–21], and in other different clinical conditions [22–24]. Regardless of the associated disease condition, the etiopathogenesis of JA is still unknown. In the present series of SLE patients, it was observed that the group with JA had a significantly longer duration both of the disease and of arthritis compared to the non-deforming group. Spronk et al. have observed that high disease activity for prolonged periods of time may lead to the development of hand deformities [9]. Furthermore, previous studies have confirmed that typical JA appears to be a late manifestation of longstanding

Table 1 Demographic data, activity and disability of the three types of arthropathy.

Patient characteristic	Mild deforming ($n = 5$)	Jaccoud's ($n = 7$)	Non-deforming ($n = 75$)
Age (years)	25.0 ± 4.52	27.57 ± 7.76	25.12 ± 6.91
Disease duration (years)	6.20 ± 2.40	13.28 ± 2.65[a,b]	6.65 ± 3.68
Arthritis duration	5.97 ± 3.61	9.62 ± 4.97[b]	5.61 ± 4.93
SLEDAI	12.80 ± 8.50	13.57 ± 9.08	17.45 ± 8.36
HAQ-DI	1.2 ± 0.7	1.5 ± 1.2	0.9 ± 0.9

Data are mean ± SD.
SLEDAI = systemic lupus erythematosis disease activity index, HAQ-DI = Health Assessment Questionnaire Disability Index.
[a] Significant compared to mild deforming group.
[b] Significant compared to non-deforming group.

Table 2 Jaccoud arthropathy index of the hand of two types of deforming arthropathy.

Jaccoud arthropathy index		Mild deforming patients no ($n = 5$)	Jaccoud's patients no ($n = 7$)
Swan neck deformity	1–4 fingers (2 points)	1/5	3/7
	5–8 fingers (3 points)	0/5	3/7
Ulnar deviation	1–4 fingers (2 points)	2/5	4/7
	5–8 fingers (3 points)	0/5	2/7
Boutonnière deformity	1–4 fingers (2 points)	1/5	3/7
	5–8 fingers (3 points)	1/5	1/7
Limited MCP extension	1–4 fingers (2 points)	2/5	2/7
	5–8 fingers (3 points)	0	2/7
Z deformity	One thumb (1 point)	1/5	1/7
	Both thumbs (2 points)	1/5	1/7

MCP = metacarpophalangeal joints.

Table 3 Clinical and immunological characteristics of the three groups of patients with lupus arthropathy.

	Mild-deforming ($n = 5$)	Jaccoud ($n = 7$)	Non-deforming ($n = 75$)
ACR criteria of SLE			
Malar rash	2 (40)	4 (57.1)	45 (60)
Discoid rash	1 (20)	0	8 (10.6)
Photosensitivity	2 (40)	4 (57.1)	47 (62.6)
Oral Ulcers	2 (40)	2 (28)	27 (36)
Arthritis	5 (100)	7 (100)	75 (100)
Serositis	2 (40)	5 (71.4)	51 (68)
Renal disorders	2 (40)	2 (28)	48 (64)
Neuropsychiatric	1 (20)	1 (14.2)	13 (17.3)
Haematological disease	1 (20)	3 (42.8)	16 (21.3)
Anti-nuclear ab	5 (100)	7 (100)	75 (100)
Anti-dsDNA	2 (40)	3 (42.8)	51 (68)
Other clinical manifestations			
Anti-phospholipid syndrome	1 (20)	4 (57.1)*	9 (12)
Sicca syndrome	1 (20)	3 (42.8)*	4 (5)
Cardiopulmonary	1 (20)	2 (28)	26 (34.6)
Cutaneous vasculitis	1 (20)	3 (42.8)	14 (18.6)
Other serological findings			
RF (IgM)	1 (20)	3 (42.8)*	7 (9)
aCL (IgG and/or M)	1 (20)	5 (71.4)*	17 (22.6)
LAC	1 (20)	3 (42.8)	9 (12)
Anti-Ro/anti-La	1 (20)	3 (42.8)	13 (17.3)

Data are number of patients (%); anti-dsDNA, anti-double stranded DNA; RF, rheumatoid factor; aCL, anticardiolipin; LAC, lupus anticoagulant.
* Significantly different compared to the non-deforming group.

arthritis [1,25,26]. Therefore, we believed that lupus activity occurring over a long duration together with persistent tenosynovitis, might cause capsule retraction and ligamentous laxity leading to muscle imbalance, with subsequent development of JA [1,25]. Based on histological findings, microvascular changes and mild but typical fibrous synovitis with little or no round cell infiltration have been found in JA, which have been mainly located in the articular capsule and tendons, leading to later fibrosis and deformity [17]. These findings have been confirmed by ultrasonographic and magnetic resonance imaging (MRI) of the hands in JA where capsular swelling, and edematous and proliferative tenosynovitis were the most prominent findings [26,27]. Recently, Sá Ribeiro et al. have performed a detailed MRI analysis of the hands of 20 patients with JA secondary to SLE. They found some degree of synovitis, bone cysts, subchondral bone edema, and tiny areas of erosion, which were not detected on conventional radiographs of the hands [28].

Previous published studies have shown conflicting results with respect to the association of JA with auto antibodies in lupus patients. Rheumatoid factor and ACL were found associated with JA in our patient cohort. A possible role for RF has been suggested in the pathogenesis of JA by participating in the formation of immune complexes and, as such, acting as a local inducer of an inflammatory reaction [1]. Numerous authors have observed that the patients with JA frequently display elevated C-reactive protein [9], RF [1], anti-cardiolipins [1,17,29], anti-SSA/Ro and anti-SSB/La antibodies [8,30]. Another study has demonstrated an association between JA in the patients with SLE and anti-thyroglobulin antibodies [31]. A recent study conducted in Brazil comparing the frequency of various autoantibodies such as anti-dsDNA, anti-SSA/Ro, anti-SSB/La, anti-cyclic citrullinated peptides and anti-mutated citrullinated vimentin in SLE patients, with or without JA, found no statistically significant differences between the groups except for anti-dsDNA and anti-mutated citrullinated vimentin antibodies [5]. The variable results presented in the literature might be related to ethnical or methodological differences. Whether these antibodies have an etiopathogenic role in JA is still entirely unknown.

We described an association of anti-phospholipid syndrome and JA. Van Vugt et al. [17] have previously described such an association and have suggested that small vessel vasculopathy may play a part in the genesis of the periarticular fibrosis. A growing body of evidence for this hypothesis can be found in "fibrin like material obliterating small vessel lumens" described in synovial biopsy specimens [32]. Bywaters [18] has already reported a correlation with mitral stenosis and Libman-Sacks endocarditis; both are known to be associated with antiphospholipid syndrome. One recent study has demonstrated an association between the presence of valvular heart disease and JA in the patients with SLE [33].

The high prevalence of SS in our patients with JA was consistent with previous observations [4,8,34]. Villiaumey et al.

[35] have reported the association of SS and JA in collagen diseases and raised the possibility that lacrimal and salivary gland involvement in SS can reflect a systemic disorder, including synovial inflammation, which may lead to a capsulo-ligamentous dislocation or JA.

The association between lupus nephritis and JA is, however, unclear: Van Vugt et al. have found renal affection to be significantly less in their JA group with SLE than the non-deforming group [17]. On the other hand, Zeier has described a case with glomerulonephritis and JA and coined the term Jaccoud's nephropathy [36]. A close association has been described between the presence of chronic renal failure complicated by secondary hyperparathyroidism in SLE patients, and responsibility for tendinous elongation and/or Jaccoud's syndrome [37]. The rarity of renal involvement and chronic renal failure in our JA patients might be related to the elevated RF, which has been considered as a protecting factor against renal affection [29].

Conclusion

To sum up, in our patients with lupus arthropathy JA is not rare and seems to be more frequent among patients with long disease duration. JA patients presented a trend toward a lower quality of life (using HAQ-DI) compared with the patients with SLE without deforming arthropathy. JA is notable for positive association with IgM RF, SS, anti-phospholipid syndrome, with unique radiological features, and foot involvement. On the other hand, those with a ''mild deforming arthropathy'' do not seem to differ in any respect from SLE patients without deforming arthropathy. Future multicentre studies, on a larger cohort, are needed to reveal the pathogenesis, the genetic association, the prevention, the stabilization and the appropriate cure for these patients.

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