DISTAL INTERPHALANGEAL JOINT INVOLVEMENT IN EGYPTIAN PATIENTS, DEMOGRAPHIC, CLINICAL AND RADIOLOGICAL CHARACTERISTICS: MULTI-CENTER STUDY

Hassan A.M Elsonbaty[1], Eman M.S Ahmed[2], Farag khalil[3], Hendawy Zidan[4], Mahmoud A. Ghalab[5], Mohammed Abdel- Hassib[6], Mohamed Hassan Attia[7], Tarek M. Nasrallah[8], Amr M. Hawwash[9]

Article History: Received: 10.05.2023 **Revised:** 15.06.2023 **Accepted:** 20.06.2023

Abstract

Introduction: Distal interphalangeal (DIP) joint involvement is a feature of hand osteoarthritis (OA), psoriatic arthritis (PsA) and Gout.

Objectives: to explore the demographic, clinical and radiological features of DIP- joint involvement in Egyptian Patients.

Patients and methods: 320 patients with DIP-joint arthropathy were involved from the Rheumatology and Immunology units of three Egyptian University Hospitals. Data on demographics, clinical conditions, and treatments were gathered. Patients were examined for evidence of osteoarthritis, Psoriasis, gout and Rheumatoid Arthritis (RA) and examined using standard hand radiography. Erythrocyte Sedimentation rate (ESR) and C-reactive protein (CRP) were done. Prevalence's were determined on all included fingers and differences in prevalence's were tested using Chi-square statistics.

Results: DIPJ-OA was diagnosed in 275 cases (85.9%), with erosions in 6 cases (2.2%). The remaining 45 (14.1%) were diagnosed as non-OA-DIPJ (31 psoriasis, 9 gout, and 5 RA), with erosions in 5 cases (11.1%), all of them had a diagnosis of PsA. Compared to non-OA-DIPJ, DIPJ-OA showed statistically significantly higher morning stiffness (MS) 5-15 minutes, duration of symptoms up to 10 years, symptomatic knee OA, and statistically significantly lower erosions, family history of DIPJ involvement, diabetes, osteophytes, ESR, and CRP. Statistically significantly higher erosions, MS, symptom duration > 10 years, positive family history of DIPJ involvement, psoriasis in a 1st degree relative, dactylitis, and joint space narrowing were found in PsA vs. non-PsA.

Conclusion: The demographic and Clinical features of Egyptian patients with DIP-Joint involvement are comparable to those from other countries.

Keywords: Distal interphalangeal joints; osteoarthritis; Psoriatic arthritis; Gout; Egypt.

1. Lecturer of Rheumatology and Rehabilitation, Faculty of Medicine, Helwan University, Cairo, Egypt
2. Assistant professor of Dermatology, Venereology and Andrology, Faculty of Medicine, Helwan University, Cairo, Egypt
3. Assistant professor of Internal Medicine and immunology, Faculty of Medicine, Al-Azhar University, Cairo, Egypt
4. Assistant professor of Internal Medicine and immunology, Faculty of Medicine, Al-Azhar University, Cairo, Egypt
5. Lecturer of radiodiagnosis and intervention, Department of Radiology,Faculty of Medicine, Kafrelsheikh University, Egypt
6. Department of Internal Medicine, Faculty of Medicine, Al-Azhar University, Cairo, Egypt
7. Lecturer of internal medicine, Faculty of Medicine, Al-Azhar University, Cairo, Egypt
8. Assistant professor of Rheumatology and Rehabilitation, Faculty of Medicine, Al-Azhar University, Dimuta, Egypt
9. Lecturer of internal medicine and Rheumatology, Faculty of Medicine, Ain Shams University, Cairo, Egypt

***Corresponding author:** Mahmoud A. Ghalab**; Email:** drmahmoudghalab@gmail.com; **Mobile:** +201003999893
Lecturer of radiodiagnosis and intervention, Department of Radiology, Faculty of Medicine, Kafrelsheikh University, Kafrelsheikh, Egypt.

INTRODUCTION

One of the fundamental concepts in diagnosis of DIP-Joint arthropathy is to differentiate osteoarthritis (OA) From psoriatic arthritis (PsA) and also from gout since the management is different.[1]

OA is a prevalent joint disease worldwide. Furthermore, it is the primary contributor to senior people's locomotor impairment. Having correlations with OA at other locations, [2,3] and atherosclerosis, it is also a potential indicator of the systemic nature of osteoarthritis. [4]

According to Peter et al.[5], erosive DIPJ-OA is a rare form of OA that mostly affects females and is marked by degenerative alterations and inflammatory assaults of DIPJ arthropathy. Subchondral erosion and bony ankylosis are radiographical features of EHOA that meet the criteria stipulated by the European League Against Rheumatism (EULAR).[6]

Contrarily, PsA encompasses a variety of clinical manifestations such as a polyarthritis that can occasionally be difficult to distinguish from rheumatoid arthritis (RA), enthesitis, oligoarthritis, spondylitis, dactylitis, asymmetric DIP joint

involvement, and a variety of osseous diseases such as mutilating arthritis, bon.[7,8]

According to CASPAR criteria, approximately 30% of psoriatic patients have PsA.[9] Similar to EHOA, PsA may progress to destructive disabling form of DIP-Joints.[10] Indeed, erosions are found in up to 47% of PsA.[11]

Both PsA and EHOA have erosion typically affects DIP- joints, with different distribution, symmetric in EHOA while asymmetrical in PsA.[12]

To differentiate between EHOA and PsA, Heberden's nodes (DIP) could be a helpful indicator. Similar to HEOA, PsA could develop limitation of Hand Functions.[13]

Loss of hand function decreases quality of life of patients with DIP-Join Arthropathy and also increases the risk of depression.[14]

The management gets more difficult when PsA occurs first and skin ailment follows. Diseases that frequently affect the hand include hand OA and (PsA) with DIP-Joint arthropathy. Both of them result in stenosing tendonitis and a gradual restriction in the range of motion of the affected joints. There are currently few effective pharmaceutical treatments for the management of OA, and those that are available are primarily symptomatic. Furthermore, there are little statistics about PsA with DIP-Joint Arthropathy's drug effectiveness.[15]

The presence of OA with a clinical diagnosis was strongly correlated with the joint sites affected by acute gout bouts. Although some case reports and small case series16–18 demonstrate that DIP-JOA predisposes to the production of urate crystals at the DIP-joint, RA seldom affects the DIP -joint.[19]

X-ray is usually helps when a patient is initially diagnosed with inflammatory arthritis. It is crucial in the initial differentiation of arthritis.[20] Anti-inflammatory dosages of Non-steroidal Anti Inflammatory drugs (NSAIDs),[21] Glucocorticoids,[22] DMARDs[23,24] and Biological Therapy provide good symptomatic effects..[25-28]

No countrywide study has been conducted in Egypt to assess the clinical characteristics and epidemiological distribution of DIP Joint Arthropathy. The present research aimed to explore the demographic, clinical and radiological characteristics of DIP joint involvement in patients attending the Rheumatology clinics and units of Helwan, Ain shams, and Al-Azhar University Hospitals in Egypt.

2. PATIENTS AND METHODS

In the present cross-sectional observational research, 355 patients were invited to participate, 35 were excluded (27 due to traumatic cause, and 8 due to refusal to participate). Finally, 320 consecutive patients with DIP Joint Arthropathy were recruited from the Rheumatology and Immunology Units of Helwan, Ain shams and Al-Azhar University Hospitals. All patients were informed about the study's objectives and given the opportunity to provide written informed consent. The Helwan University ethics committee gave the study the thumbs up. The diagnosis of DIPJ OA was confirmed according to the ACR standards for hand OA,[29] as well as CASPAR criteria for PsA,[30] 2015ACR/EULAR classification criteria for Gout,[31] And 2010 standards for RA from the European League Against Rheumatism (EULAR) and the American College of Rheumatology (ACR).[32]

Age, sex, and socioeconomic level were all collected as demographic information. The length of the disease was noted, and clinical information including the existence of Low back pain, BMI, and symptomatic OA in the knee was assessed. The distribution of Heberden's nodes and any symptoms of dactylitis were carefully examined on the fingers. The skin was thoroughly checked to look for any nodes or psoriatic skin lesions. Additionally, thorough information was gathered on family history of gout, psoriasis, diabetes mellitus, hypertension, and DIPJ arthropathy. CRP and ESR measurements were done. NSAIDs, steroids, disease-modifying antirheumatic medications (DMARDs), and biologic therapy were all mentioned in the patient's descriptive therapeutic history. Plain postero-anterior radiographs of Hands was evaluated for entheseal bone-change, erosions, Osteophyte and Joint space narrowing, using the OARSI atlas.[33,34]

STATISTICAL ANALYSIS

Utilizing Statistical Package for the Social Sciences (SPSS) software, version 25, data of the present study were expressed as n (%) or median and interquartile range (IQR). For comparisons, Chi-Square, or Fisher's exact test for categorical data and Mann-Whitney U test for quantitative data were considered. Results were regarded as significant if p-Value < 0.05.

RESULTS

In this study, 355 patients were invited to participate, 35 were excluded (27 due to traumatic cause, and 8 due to refusal to participate). Finally, 320 patients were enrolled. DIPJ-OA was diagnosed in 275 cases (85.9%), with erosions in only 6 cases (2.2%). The remaining 45 (14.1%) were diagnosed as non-DIPJ-OA (31 psoriasis, 9 gout, and 5 RA), with erosions in 5 cases (11.1%), all of them have a diagnosis of psoriasis. This is illustrated in figure (1). Their median age of the cases was 51 years, ranging from 30 to 70 years. They were 154 male and 166 female patients. Table (1) shows descriptive statistics of the whole study cases.

Characteristics of individual diagnoses:

1. O.A.:

The main diagnosis was OA (275 cases). Table (2) shows a statistical comparison between DIPJ-OA and non-OA-DIPJ.

Notes: For categorical traits, the Chi-square test or Fisher's exact test (FET) is the test of significance; for quantitative features, the Mann-Whitney U-test.

Table (2) shows a statistically significantly higher morning stiffness 5-15 minutes, duration of symptoms up to 10 years, symptomatic knee OA, and osteophytes grades 0-2, and a statistically significantly lower erosions, family history of DIPJ involvement, morning stiffness around 20 minutes, duration of symptoms > 10 years, diabetes, osteophytes grade 3, ESR, and CRP in those with DIPJ-OA vs. those without DIPJ-OA.

Binary logistic regression was utilized to confirm the impact of 9 variables on the likelihood that patients presenting with DIPJ involvement will exhibit DIPJ osteoarthritis. This can be shown in table (3).

On univariable analysis, all these parameters were statistically substantial predictors of DIPJ osteoarthritis. Accordingly, all these 9 variables were included in multivariable analysis model. The model was statistically substantial ($\chi^2[9] = 222.491$, $P<0.001$). The model correctly classified 97.5% of cases with a sensitivity and specificity of 98.9% and 88.9%, respectively. The model explains 90.1% of the variance in the diagnosis of DIPJ OA vs. DIPJ non-OA. Of the 9 variables, only 4 were statistically substantial independent predictors of DIPJ OA. These variables are ESR ≤58, CRP ≤9, osteophyte radiological grading of 0-2, and absence of diabetes. Patients with ESR ≤58, CRP ≤9, osteophyte radiological grading of 0-2, and absence of diabetes have 277.1-, 19.7-, 80.1-, and 77.2-times higher odds to exhibit DIPJ OA.

2. Psoriasis:

This was the second diagnosis in out cohort (31 cases). Table (4) shows a statistical comparison between PsA and non-PsA cases.

Table (4) shows a statistically significantly higher erosions, morning stiffness, symptom duration > 10 years, osteophyte radiological grades 3-4, positive family history of DIPJ involvement, inflammatory LBP, psoriasis in a 1st degree relative, dactylitis, and joint space narrowing, and a statistically significantly lower symmetrical distribution in PsA vs. non- PsA.

Table (1): Descriptive statistics of the studied cases (n=320):

Characteristic	Statistic
Categorical	N (%)
Female sex	166 (51.9%)
Socio-economic status	
Very low / Low	218 (68.1%)
Middle / High	102 (31.9%)
Occupation	
Manual	146 (45.6%)
Office	100 (31.3%)
Housewife	74 (23.1%)
Diabetes	34 (10.6%)
Psoriasis in a first degree relative	9 (2.8%)
Family history of DIPJ involvement	70 (21.9%)
History of gout	9 (2.8%)
Duration of symptoms (years)	
<1	52 (16.3%)
1 – <5	91 (28.4%)
5 – 10	80 (25%)
> 10	97 (30.3%)
Morning stiffness in DIPJ (minutes)	
Around 5 minutes	149 (46.6%)
Around 10 minutes	49 (15.3%)
Around 15 minutes	80 (25%)
Around 20 minutes	42 (13.1%)
Inflammatory LBP	25 (7.8%)
BMI categories	
Ideal	40 (12.5%)
Overweight	103 (32.2%)
Grade I obesity	177 (55.3%)

Clinical features	
Heberden's nodes	237 (74.1%)
Symmetrical distribution	206 (86.9%)
Grade 1 / Grade 2	173 (73%) / 64 (27%)
Psoriasis	31 (9.7%)
Dactylitis	23 (74.2%)
Rheumatoid arthritis	5 (1.5%)
Symptomatic Knee OA	283 (88.4%)
DIPJ OA	275 (85.9%)
Radiological features	
Erosion	11 (3.4%)
Gull wing appearance	6 (1.9%)
Joint space narrowing	235 (73.4%)
Osteophyte radiological grading	
Grade 0	7 (2.2%)
Grade 1	96 (30%)
Grade 2	190 (59.4%)
Grade 3	27 (8.4%)
Treatments received	
NSAIDs	320 (100%)
Methotrexate	58 (18.1%)
Hydroxychloroquine	140 (43.8%)
Systemic steroids	67 (20.9%)
Biologic therapy	8 (2.5%)
Quantitative	Median (Q1 – Q3)
Age (years)	51 (43 – 59)
BMI (kg/m^2)	30 (26 – 33)
Number of joints affected by Heberden's nodes	2 (2 – 6)
CRP (mg/dl)	7 (5 – 9)
ESR (mm/first hour)	44 (37 – 54)

Table (2): Comparisons between those with and without DIPJ OA

Characteristic	**DIPJ-OA (N=275)**	**Non-DIPJ-OA (N=45)**	**Test of sig**	
Categorical	N (%)	N (%)	χ^2	p-value
Female sex	146 (53.1%)	20 (44.4%)	1.158	0.282
BMI category				
Ideal	35 (12.7%)	5 (11.1%)		
Overweight	83 (30.2%)	20 (44.4%)		
Grade 1 obesity	157 (57.1%)	20 (44.4%)	3.643	0.162
Erosions	6 (2.2%)	5 (11.1%)	FET	**0.011**
Occupation				
Manual	122 (44.4%)	24 (53.3%)		
Office	88 (32%)	12 (26.7%)		
Housewife	65 (23.6%)	9 (20%)	1.255	0.534
Socio-economic status				
Very low / Low	183 (66.5%)	35 (77.8%)		
Middle / High	92 (33.5%)	10 (22.2%)	2.247	0.134
Family history of DIPJ involvement	37 (13.5%)	33 (73.3%)	81.134	**<0.001**
Morning stiffness in DIPJ (minutes)				
Around 5 minutes	143 (52%) a	6 (13.3%) b		
Around 10 minutes	46 (16.7%) a	3 (6.7%) a		
Around 15 minutes	76 (27.6%) a	4 (8.9%) b		
Around 20 minutes	10 (3.6%) a	32 (71.1%) b	154.557	**<0.001**
Duration of symptoms (years)				
<1	50 (18.2%) a	2 (4.4%) b		
1 – <5	87 (31.6%) a	4 (8.9%) b		
5 – 10	77 (28%) a	3 (6.7%) b		
> 10	61 (22.2%) a	36 (80%) b	61.216	**<0.001**
Symptomatic knee OA	270 (98.2%)	13 (28.9%)	181.587	**<0.001**

Diabetes	15 (5.5%)	19 (42.2%)	FET	**<0.001**
Joint space narrowing	204 (74.2%)	31 (68.9%)	0.555	0.456
Osteophyte radiological grading Grades 0 – 2 Grades 3 –4	 260 (94.5%) 15 (5.5%)	 22 (48.9%) 23 (51.1%)	 77.032	 **<0.001**
Quantitative	Median (Q1-Q3)	Median (Q1-Q3)	Z	**p-value**
Age (years)	51 (43 – 59)	52 (44 – 61.5)	-0.808	0.419
BMI (kg/m^2)	30 (26 – 33)	29 (26 – 32)	-1.176	0.240
ESR (mm/first hour)	43 (34 – 50)	69 (66 – 70)	-10.282	**<0.001**
CRP (mg/dl)	6 (4 – 8)	15 (12 – 17)	-10.244	**<0.001**

Table (3): Predictors of DIPJ osteoarthritis

Predictor	Univariate			Multivariate		
	p-values	COR	95% CI	p-values	AOR	95% CI
ESR >58 ≤58	**<0.001**	r(1) 342.1	r(1) 98.6-1187.4	**<0.001**	r(1) 277.1	r(1) 15.4-4970
CRP >9 ≤9	**<0.001**	r(1) 342.1	r(1) 98.6-1187.4	**0.012**	r(1) 19.7	r(1) 1.9-201.1
Morning stiffness ~20 minutes ~5-15 minutes	**<0.001**	r(1) 65.2	r(1) 26.4-160.8	0.653	r(1) 2.7	r(1) 0.04-209
Symptom duration >10 years ≤10 years	**<0.001**	r(1) 14	r(1) 6.4-30.7	0.801	r(1) 0.55	r(1) 0.005-57.1
Symptomatic knee OA No Yes	**<0.001**	r(1) 132.9	r(1) 44.5-397.2	0.318	r(1) 0.15	r(1) 0.004-6
Osteophyte grades 3 0-2	**<0.001**	r(1) 18.1	r(1) 8.3-39.6	**0.016**	r(1) 80.1	r(1) 2.23-2878
Family history of DIPJ Yes No	**<0.001**	r(1) 17.7	r(1) 8.4-37.3	0.476	r(1) 0.18	r(1) 0.001-20.9
Erosion Yes No	**0.006**	r(1) 5.6	r(1) 1.6-19.2	0.357	r(1) 7.8	r(1) 0.098-615.3
Diabetes Yes No	**<0.001**	r(1) 12.6	r(1) 5.7-27.8	**0.001**	r(1) 77.2	r(1) 5.3-1115

AOR = Adjusted odds ratio. COR = crude odds ratio. CI = confidence interval. r(1) = reference category.

Table (4): Comparisons of psoriasis vs. non-psoriasis cases

Characteristic	Psoriasis N=31	Non-psoriasis N=289	p-value
Erosions	5 (16.1%)	6 (2.1%)	**0.002**
Morning stiffness around 20 minutes	31 (100%)	11 (3.8%)	**<0.001**
Symptom duration > 10 years	31 (100%)	66 (22.8%)	**<0.001***
Osteophyte grades 3	23 (74.2%)	15 (5.2%)	**<0.001**
Family history of DIPJ involvement	28 (90.3%)	42 (14.5%)	**<0.001***
Inflammatory LBP	25 (80.6%)	0 (0%)	**<0.001**
Psoriasis in 1st degree relative	4 (12.9%)	5 (1.7%)	**<0.001**
Dactylitis	23 (74.2%)	0 (0%)	**<0.001**
Symmetrical distribution	1 (3.2%)	205 (70.9%)	**<0.001**
Joint space narrowing	30 (96.8%)	205 (70.9%)	**0.002***

Notes: Data is N (%). Tests of significance are Fisher's exact test or Chi-Square test*.

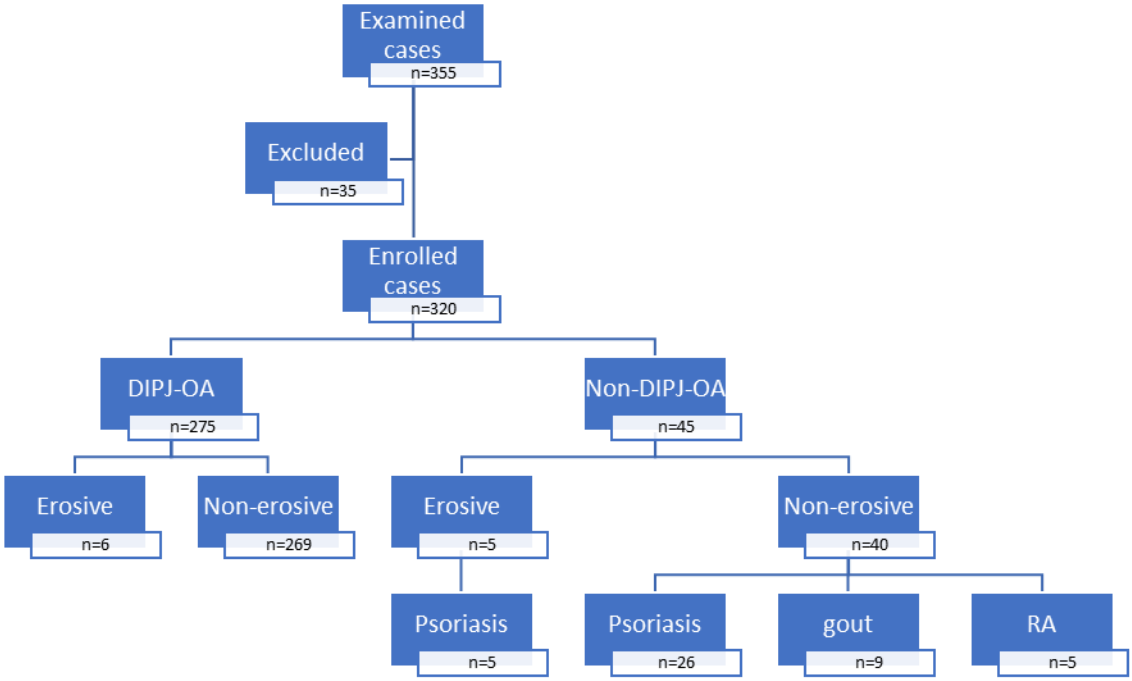

Figure (1): Flow chart of the cases in the study

3. **Gout:** This comes third in the ranking of DIPJ involvement diagnoses (9 cases).
4. **RA:** was the diagnosis in only 5 cases in this study.

DISCUSSION

Differentiating DIP-JOA from PsA and also from gout is one of the biggest challenges, as these diseases preferentially affect DIP-Joints with similar manifestations.[15]

As far as we are aware, there hasn't been any research done in Egypt to illustrate the epidemiological distribution and clinical characteristics of DIP-Joint Arthropathy. In our study DIPJ-OA was diagnosed in 275 cases (85.9%), with female predominance (53.1%).

Our results came consistent with previous studies since DIP Joint involvement was more prevalent in women and mostly caused by OA. According to the Beijing study, OA was the main DIP-Joint involvement factor.[35,36]

In the same line, OA was primarily observed in the DIP joint in a 20 to 23 year longitudinal Tecumseh, MI, USA investigation.[37]

A previous longitudinal investigation that looked at 286 persons with hand OA revealed that the DIP joint was frequently affected by the condition.[38]

In our study, most patients with DIPJ-OA had Heberden's node (74.1%). It was also revealed that all people with Haberdan's nodes had also symptomatic DIPJ- OA involvement had a substantial connection with knee OA (98%). This was consistent with the research done in Turkey, it was reported that DIPJ-OA was significantly related to knee OA. [39]

In a previous cohort trial involving 1,235 participants, it was discovered that those who had hand OA initially had a twofold elevated risk of developing knee OA and that 33.3% of people with DIP joint OA also had knee OA.[3]

On the contrary, some hospital based studies have reported DIPJ-OA prevalence was determined as 10.5% among those who are 50 years of age or older in Antalya,[39] 6.2% was the reported DIPJ-OA prevalence in the Spanish investigation.[40]

Such fluctuation of variations in the frequency of DIP-Joint involvement as a disease may be attributed to variations in quality and bias of the methodologic approaches.

In our patients, the most common pattern of PsA was predominant DIP involvement. Similarly, PsA can involve any joint, those most commonly involved are reported to be DIP- joints of the hands.[41]

PsA made up 36.4%of the population in the USA[42] and 34.8% in Greece. [43]

PsA is most common in Argentina (60.2%), but much less so in Brazil (13.7%) and Guatemala (10%)[44]; it was most widespread in Italy. [45]

In this study, gout was present in (2.8%) of patients with DIP-Joint Arthropathy.

A number of theories have been mentioned to explain the relation between the sites of acute attacks of gout and OA, including: cartilage and synovial proteoglycans changes,[47] mechanical shock,[46] formation of MSU crystal on cartilage fragments.[48]

This was in agreement with the case reports and small hospital-based case series report the incidence of gout at joint sites involved by OA especially DIP- joints;[17,18,49] Although, according to relevant literature, RA sufferers may not have damage to their DIP joints.[50] Our findings showed that RA was present in (1.5%) of patients with DIP-Joint Arthropathy.

The results were consistent with the study that investigated the association between DIP-Joint Arthropathy and illness activity in 10,038 patients with RA. DIP-Joint Arthropathy (based on the occurrence of DIP-Joint pain and/or swelling) was Diagnosed in 206 (2.1%) of 10,038 patients with RA.[19]

Strengths: The study is supported by the use of patients who were systematically selected from rheumatology clinics to represent the diagnostic conundrums in a clinical context. This increases the external validity of the study.

Some patients were receiving DMARD medication, which could be one of the study limits. Inflammatory activity-related findings may become less likely as a result. Given that the DIP-JOA group does not receive DMARD therapy, it may also help to explain the relatively higher frequency of these manifestations in this group. Additionally, the study's design prevented patients from being followed up on to determine the effectiveness of a recently introduced biological therapy, and the study's patient population was rather small.

In conclusion, Results obtained in this study provide a thorough categorization of various features of DIP Joint Arthropathy in Egyptian patients. Planning for future care and service requirements may benefit from having access to these data as they provide a better knowledge of the illness.

Conflict of interest: no conflict of interest.

Funding: No particular grant was given to this work by funding organizations in the public, private, or not-for-profit sectors.

Acknowledgements: The authors are thankful to the Rheumatology, Immunology and Radiology Departments, Faculties of Medicine in Helwan, Al-Azhar, Ain Shams and Kafrelsheikh Universities.

References

1. Hoxha A, Ruffatti A, Alberioli E, et al. (2016) Erosive osteoarthritis, psoriatic arthritis and pseudogout; a casual association? Clinical rheumatology 35 (7):1885-9
2. Jonsson H, Helgadottir GP, Aspelund T, et al. (2011) Hand Osteoarthritis Severity is Associated with Total Knee Joint Replacements Independently of BMI. The Ages-Reykjavik Study. The open rheumatology journal 5:7-12
3. Dahaghin S, Bierma-Zeinstra SM, Reijman M, Pols HA, Hazes JM, Koes BW (2005) Does hand osteoarthritis predict future hip or knee osteoarthritis? Arthritis and rheumatism 52 (11):3520-7
4. Hoeven TA, Kavousi M, Clockaerts S, et al. (2013) Association of atherosclerosis with presence and progression of osteoarthritis: the Rotterdam Study. Annals of the rheumatic diseases 72 (5):646-51
5. Peter JB, Pearson CM, Marmor L (1966) Erosive osteoarthritis of the hands. Arthritis and rheumatism 9 (3):365-88
6. Zhang W, Doherty M, Leeb BF, et al. (2009) EULAR evidence-based recommendations for the diagnosis of hand osteoarthritis: report of a task force of ESCISIT. Annals of the rheumatic diseases 68 (1):8-17
7. McGonagle D (2005) Imaging the joint and enthesis: insights into pathogenesis of psoriatic arthritis. Annals of the rheumatic diseases 64 Suppl 2 (Suppl 2):ii58-60
8. Ory PA, Gladman DD, Mease PJ (2005) Psoriatic arthritis and imaging. Annals of the rheumatic diseases 64 Suppl 2 (Suppl 2):ii55-7
9. Villani AP, Rouzaud M, Sevrain M, et al. (2015) Prevalence of undiagnosed psoriatic arthritis among psoriasis patients: Systematic review and meta-analysis. Journal of the American Academy of Dermatology 73 (2):242-8
10. Gladman DD, Antoni C, Mease P, Clegg DO, Nash P (2005) Psoriatic arthritis: epidemiology, clinical features, course, and outcome. Annals of the rheumatic diseases 64 Suppl 2 (Suppl 2):ii14-7
11. Kane D, Stafford L, Bresnihan B, FitzGerald O (2003) A prospective, clinical and radiological study of early psoriatic arthritis: an early synovitis clinic experience. Rheumatology (Oxford, England) 42 (12):1460-8
12. Ramonda R, Frallonardo P, Musacchio E, Vio S, Punzi L (2014) Joint and bone assessment in hand osteoarthritis. Clinical rheumatology 33 (1):11-9
13. Kodama R, Muraki S, Oka H, et al. (2016) Prevalence of hand osteoarthritis and its relationship to hand pain and grip strength in Japan: The third survey of the ROAD study. Modern rheumatology 26 (5):767-73
14. Kwok WY, Vliet Vlieland TP, Rosendaal FR, Huizinga TW, Kloppenburg M (2011) Limitations in daily activities are the major determinant of reduced health-related quality of life in patients with hand osteoarthritis. Annals of the rheumatic diseases 70 (2):334-6

15. Poletto E, Tinazzi I, Marchetta A, Smania N, Rossato E (2021) Hand Erosive Osteoarthritis and Distal Interphalangeal Involvement in Psoriatic Arthritis: The Place of Conservative Therapy. Journal of clinical medicine 10 (12):2630
16. Roddy E, Zhang W, Doherty M (2007) Are joints affected by gout also affected by osteoarthritis? Annals of the rheumatic diseases 66 (10):1374-7
17. Fam AG, Stein J, Rubenstein J (1996) Gouty arthritis in nodal osteoarthritis. The Journal of rheumatology 23 (4):684-9
18. Lally EV, Zimmermann B, Ho G, Jr., Kaplan SR (1989) Urate-mediated inflammation in nodal osteoarthritis: clinical and roentgenographic correlations. Arthritis and rheumatism 32 (1):86-90
19. Mizuuchi T, Sawada T, Nishiyama S, et al. (2022) Distal Interphalangeal Joint Involvement May Be Associated with Disease Activity and Affected Joint Distribution in Rheumatoid Arthritis. Journal of clinical medicine 11 (5):1405
20. Bossuyt PM, Reitsma JB, Bruns DE, et al. (2015) STARD 2015: an updated list of essential items for reporting diagnostic accuracy studies. BMJ (Clinical research ed) 351:h5527
21. Kroon FPB, Carmona L, Schoones JW, Kloppenburg M (2018) Efficacy and safety of non-pharmacological, pharmacological and surgical treatment for hand osteoarthritis: a systematic literature review informing the 2018 update of the EULAR recommendations for the management of hand osteoarthritis. RMD open 4 (2):e000734
22. Kroon FPB, Kortekaas MC, Boonen A, et al. (2019) Results of a 6-week treatment with 10 mg prednisolone in patients with hand osteoarthritis (HOPE): a double-blind, randomised, placebo-controlled trial. Lancet (London, England) 394 (10213):1993-2001
23. Kolasinski SL, Neogi T, Hochberg MC, et al. (2020) 2019 Management of Osteoarthritis of the Hand, Hip, and Knee. Arthritis care & research 72 (2):149-62
24. Kedor CC, Detert J, Rau R, et al. (2020) Ydorxychloroquine in patients with imflammatory and erosive osteoarthritis of the hand: Results of a randomized, doubleblind, placebo controlled, multi-centre, investigator-initiated trial (OA TREAT). Annals of the rheumatic diseases 79:115-6
25. Verbruggen G, Wittoek R, Vander Cruyssen B, Elewaut D (2012) Tumour necrosis factor blockade for the treatment of erosive osteoarthritis of the interphalangeal finger joints: 71 (6):891-8
26. Aitken D, Laslett LL, Pan F, et al. (2018) A randomised double-blind placebo-controlled crossover trial of HUMira (adalimumab) for erosive hand OsteoaRthritis - the HUMOR trial. Osteoarthritis and cartilage 26 (7):880-7
27. Fleischmann RM (2020) EULAR PsA management recommendations 2019: can the recommendations be improved? Annals of the rheumatic diseases 79 (6):700-10
28. Cañete JD, Ariza-Ariza R, Bustabad S, et al. (2018) Recommendations for the use of methotrexate in psoriatic arthritis. Reumatologia clinica 14 (4):183-90
29. Altman R, Alarcón G, Appelrouth D, et al. (1990) The American College of Rheumatology criteria for the classification and reporting of osteoarthritis of the hand. Arthritis and rheumatism 33 (11):1601-10
30. Taylor W, Gladman D, Helliwell P, Marchesoni A, Mease P, Mielants H (2006) Classification criteria for psoriatic arthritis: development of new criteria from a large international study. Arthritis and rheumatism 54 (8):2665-73
31. Liu H, Zhang XM, Wang YL, Liu BC (2014) Prevalence of hyperuricemia among Chinese adults: a national cross-sectional survey using multistage, stratified sampling. Journal of nephrology 27 (6):653-8
32. Aletaha D, Neogi T, Silman AJ, et al. (2010) 2010 Rheumatoid arthritis classification criteria: an American College of Rheumatology/European League Against Rheumatism collaborative initiative. Arthritis and rheumatism 62 (9):2569-81
33. Altman RD, Hochberg M, Murphy WA, Jr., Wolfe F, Lequesne M (1995) Atlas of individual radiographic features in osteoarthritis. Osteoarthritis and cartilage 3 Suppl A:3-70
34. Guldberg-Møller J, Mogensen M, Ellegaard K, et al. (2022) Multimodal imaging of the distal interphalangeal-joint synovio-entheseal complex in psoriatic arthritis (MIDAS): a cross-sectional study on the diagnostic accuracy of different imaging modalities comparing psoriatic arthritis to psoriasis and osteoarthritis. RMD open 8 (1):e002109
35. Zhang Y, Xu L, Nevitt MC, et al. (2003) Lower prevalence of hand osteoarthritis among Chinese subjects in Beijing compared with white subjects in the United States: the Beijing Osteoarthritis Study. Arthritis and rheumatism 48 (4):1034-40

36. Belhorn LR, Hess EV (1993) Erosive osteoarthritis. Seminars in arthritis and rheumatism 22 (5):298-306
37. Sowers M, Zobel D, Weissfeld L, Hawthorne VM, Carman W (1991) Progression of osteoarthritis of the hand and metacarpal bone loss. A twenty-year followup of incident cases. Arthritis and rheumatism 34 (1):36-42
38. Cvijetić S, Kurtagić N, Ozegović DD (2004) Osteoarthritis of the hands in the rural population: a follow-up study. European journal of epidemiology 19 (7):687-91
39. Kaçar C, Gilgil E, Urhan S, et al. (2005) The prevalence of symptomatic knee and distal interphalangeal joint osteoarthritis in the urban population of Antalya, Turkey. Rheumatology international 25 (3):201-4
40. Carmona L, Ballina J, Gabriel R, Laffon A (2001) The burden of musculoskeletal diseases in the general population of Spain: results from a national survey. Annals of the rheumatic diseases 60 (11):1040-5
41. Jajić Z, el Assadi G (2003) [Prevalence of psoriatic arthritis in a population of patients with psoriasis]. Acta medica Croatica : casopis Hravatske akademije medicinskih znanosti 57 (4):323-6
42. Weisman M, Learch TJ, Baraliakos X, et al. (2010) Current controversies in spondyloarthritis: SPARTAN. The Journal of rheumatology 37 (12):2617-23
43. Trontzas P, Andrianakos A, Miyakis S, et al. (2005) Seronegative spondyloarthropathies in Greece: a population-based study of prevalence, clinical pattern, and management. The ESORDIG study. Clinical rheumatology 24 (6):583-9
44. Saad CG, Gonçalves CR, Sampaio-Barros PD (2014) Seronegative arthritis in Latin America: a current review. Current rheumatology reports 16 (9):438
45. De Angelis R, Salaffi F, Grassi W (2007) Prevalence of spondyloarthropathies in an Italian population sample: a regional community-based study. Scandinavian journal of rheumatology 36 (1):14-21
46. Wilcox WR, Khalaf AA (1975) Nucleation of monosodium urate crystals. Annals of the rheumatic diseases 34 (4):332-9
47. Perricone E, Brandt KD (1978) Enhancement of urate solubility by connective tissue. I. Effect of proteoglycan aggregates and buffer cation. Arthritis and rheumatism 21 (4):453-60
48. Pascual E, Ordóñez S (1998) Orderly arrayed deposit of urate crystals in gout suggest epitaxial formation. Annals of the rheumatic diseases 57 (4):255
49. Simkin PA, Campbell PM, Larson EB (1983) Gout in Heberden's nodes. Arthritis and rheumatism 26 (1):94-7
50. England B, Mikuls T. Clinical features of rheumatoid arthritis. In: Firestein G, Budd R, Gabriel S, Koretzky G, McInnes I, O'Dell J, eds. Firestein & Kelly's Textbook of Rheumatology. 11th ed. Philadelphia, PA, USA: Elsevier; 2020: 1236–57