Journal of Pharmaceutical Research International

Volume 34, Issue 61, Page 1-7, 2022; Article no.JPRI.95399
ISSN: 2456-9119
(Past name: British Journal of Pharmaceutical Research, Past ISSN: 2231-2919, NLM ID: 101631759)

Overview on Juvenile Psoriatic Arthritis: A Review

Ahmed Abdelsamie Fadl [a++,#*], Sultan Abdullah Hassan Alsuwaidani [b†], Sukainah Mohammed Al Batti [c†], Zahrah Hatim Saleh Al Saleh [c‡], Fatimah Abdullah AL Ghareeb [d^], Zakiah Hussain Ali Alrebh [e‡], Louloah Abdullah Al Omran [c†], Mohammad Salman ALRadhwan [b†], Fatema Hussain Abdullah Almarhoon [c^], Zahra Abdulkarim Saeed Almutlaq [f†] and Hussain Taher Al Aysh [b†]

[a] Doctor Samir Abbas Hospital, Department of Pediatrics, Alazhar University Hospitals, Cairo, Egypt.
[b] Qatif Preventive Medicine Center, Qatif City, Saudi Arabia.
[c] Al Reef Primary Health Care Center, Qatif City, Saudi Arabia.
[d] Qatif Health Network, Qatif City, Saudi Arabia.
[e] Alawmia Primary Health Care Center, Qatif City, Saudi Arabia.
[f] Alshowaika Primary Health Care Center, Qatif City, Saudi Arabia.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/JPRI/2022/v34i617274

Open Peer Review History:
This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://www.sdiarticle5.com/review-history/95399

[++]Pediatric Senior Registrar;
[#]SCFHS number: 12JM0036858;
[†]Health Assistant Nursing;
[‡]Nursing Technician;
[^]Nursing Specialist;
**Corresponding author: E-mail: Dr_fadl844@yahoo.com;*

Review Article

Received: 20/10/2022
Accepted: 28/12/2022
Published: 29/12/2022

ABSTRACT

Psoriatic juvenile idiopathic arthritis is a subtype of juvenile idiopathic arthritis that is characterized by both arthritis and psoriasis. juvenile idiopathic arthritis (JIA) is a heterogeneous category of idiopathic inflammatory arthritis that affects children under the age of 16 and lasts six weeks or more as it represents approximately 5% of the whole Juvenile Idiopathic Arthritis (JIA) population. The most prevalent rheumatic illness in children from Western countries is JIA. Chronic arthritis in JIA has an unknown etiology and causes. It is hypothetical if abnormal immune reactions may be brought on by the interplay of environmental elements in a person with a predisposed genetic makeup. Some environmental variables, such exposure to antibiotics and C-section births, provide significant dangers. If there are no characteristic psoriatic lesions present. According to International League of Associations of Rheumatology (ILAR) classification, JPsA is defined by the association of arthritis and psoriasis or, in the absence of typical psoriatic lesions, with at least two of the following: dactylitis, nail pitting, onycholysis or family history of psoriasis in a first-degree relative. In this review we will be looking at the disease etiology, epidemiology, treatment as well as some literature review.

Keywords: Juvenile psoriatic arthritis; epidemiology; psoriatic arthritis; treatment; idiopathic inflammatory arthritis.

1. INTRODUCTION

A heterogeneous category of idiopathic inflammatory arthritis that affects children under the age of 16 and lasts six weeks or more is known as juvenile idiopathic arthritis (JIA). Since 1995, the terms juvenile rheumatoid arthritis (JRA) and juvenile chronic arthritis (JCA) have been replaced with juvenile idiopathic arthritis (JIA). There are seven JIA classifications, according to the International League of Associations for Rheumatology's (ILAR) consensus meeting in 2001: Systemic arthritis, psoriatic arthritis, enthesitis-related arthritis, oligoarthritis, rheumatoid factor (RF) positive polyarthritis, RF negative polyarthritis, oligoarthritis, and undifferentiated arthritis are all forms of arthritis [1].

At least 5% of people with psoriasis develop psoriatic arthritis, a chronic inflammatory arthritis. The link between psoriasis and arthritis was discovered in the middle of the 19th century, but it took until the 1960s for psoriatic arthritis to be clinically separated from rheumatoid arthritis (RA). Patients with psoriasis are at risk for developing psoriasis arthritis if they have nail, inverse, or scalp psoriasis, a family history of psoriatic arthritis, or if their skin condition is severe. An examination of 120,523 psoriasis patients revealed that the overall 5-year prevalence of psoriatic arthritis was 9.9% in those with mild to moderate psoriasis, 35.0% in those with moderate to severe psoriasis, and 54.9% in those with severe psoriasis [2].

The prevalence of juvenile psoriatic arthritis (JPsA), which makes up around 5% of all cases of juvenile idiopathic arthritis (JIA), is quite low in children. If there are no characteristic psoriatic lesions present, JPsA is classified by the International League of Associations of Rheumatology (ILAR) as if at least two of the following are present: dactylitis, nail pitting, onycholysis, or a first-degree relative with a history of psoriasis. Recent research has however revealed that this categorization approach may hide more homogenous patient groupings that vary in terms of onset age, clinical traits, and prognosis. Particularly in the juvenile population, little is known about the genetic determinants and pathogenetic pathways that set JPsA apart from other JIA subtypes or from isolated psoriasis without joint involvement [3].

There is a female prevalence, with a median age of onset of 4.5 years in females and 10 years in males. Although it can occasionally be severe and damaging and proceed into adulthood, the sickness is mostly modest. Monoarticular arthritis affects 50% of children; DIP joint involvement happens at a similar incidence. Thirty percent of

children have tenosynovitis, and 71 percent have nail involvement, with pitting being the most prevalent but least precise symptom. Disordered bone development with subsequent shortening may occur in 47% of children when unfused epiphyseal growth plates are involved in the inflammatory process. 28 percent of juveniles get sacroiliitis, which is often correlated with HLA-B27 positive. HLA-B17 is often linked to a moderate type of psoriatic arthritis, but HLA-B8 may be a sign for a more severe condition. Compared to adults, children are more likely to experience the simultaneous beginning of psoriasis and arthritis, with 52 percent of children experiencing arthritis first [2].

Juvenile psoriatic arthritis (JPsA) has gained more recognition within the group of paediatric inflammatory arthritides since the ILAR criteria for juvenile idiopathic arthritis were introduced. According to the ILAR classification, patients are considered to have JPsA if they have persistent arthritis that has lasted longer than 6 weeks and developed before the age of 16, and they meet at least 2 of the following minor criteria: a first-degree relative with psoriasis, nail pitting or onycholysis, and dactilytis. Another categorization system, the Vancouver Criteria, was employed before the ILAR criteria [4-6].

The symptoms, genetic susceptibilities, pathophysiology, test results, illness history, and prognosis of the subgroups of JIA are unique. Even though chronic arthritis is a need for all subtypes, each individual subtype was distinguished by its extraarticular and systemic symptoms. The Pediatric Rheumatology International Trial Organization has just suggested and is formally validating a new preliminary data-driven categorization for JIA [7].

Compared to other JIA subtypes, JPsA patients have fewer publications outlining their features and long-term outcomes, and none have used the ILAR Criteria exclusively. Some also examined overlapping patient subgroups, were review papers, or tiny case series. Comparing various research can be challenging due to the usage of many JPsA categorization systems, and as recently as 2008, no single classification was agreed upon or widely acknowledged [4,8-19].

2. ETIOLOGY

Chronic arthritis in JIA has an unknown aetiology and trigger. It is hypothetical if abnormal immune reactions may be brought on by the interplay of environmental elements in a person with a predisposed genetic makeup. Some environmental variables, such exposure to antibiotics and C-section births, provide significant dangers; nursing and living with siblings may mitigate these effects. There is considerable debate over the functions of microorganisms including Parvovirus B19, Epstein-Barr virus, intestinal bacteria, Chlamydophila pneumoniae, and streptococcal infections [7].

Psoriasis and psoriatic arthritis susceptibility is largely influenced by genetic factors; over 40% of individuals with either of these disorders have a first-degree relative who has them. In first-degree relatives of patients with psoriatic arthritis, the recurrence risk ratio—a measure of the disease's heritability—is estimated to be 30-55, whereas for those with psoriasis, it is 8–10. Genetic variables that underlie illness susceptibility are more likely to be present in diseases that have a greater heritability. Although the precise mechanism of the relationship between HLA and psoriatic arthritis is still unclear, the following significant genetic susceptibility loci have been identified: [2,20-30].

- HLA-Cw6, HLA-B57, HLA-DR7, and HLA-B17 are all related with early-onset psoriasis, with the HLA-Cw*0602 variation being particularly significant
- Psoriasis: HLA-Cw6 and six additional psoriasis susceptibility loci (PSOR2, PSOR3, PSOR4, PSOR5, PSOR6, and PSOR7), as well as the transcription factor RUNX1.
- HLA-B7, HLA-B27, HLA-DR4, HLA-38, and HLA-DR7 all have psoriatic arthritis.
- HLA-Cw6, HLA-B13, HLA-B17, HLA-B57, and HLA-B39 are associated with psoriasis and psoriatic arthritis.
- HLA-B39, HLA-B27 in the presence of HLA-DR7, and HLA-DQ3 in the absence of HLA-DR7 are predictors of disease progression.

3. EPIDEMIOLOGY

The most prevalent rheumatic illness in children from Western countries is JIA. Depending on research methods, illness categories, and geographic regions, the incidence and prevalence range from 1.6 to 23 new cases per 100,000 children and 3.8 to 400 cases per 100,000 children, respectively. According to research from the US and Canada, JIA affects

between 0.04 and 0.06 of every 1000 kids. The frequency in white populations is 1.2 per 1000, according to the Utah Population Database. Similar to the relative risk of type 1 diabetes, the relative risk of JIA in siblings ranges from 15 to 30 [7].

In a population-based study of 440 children with JIA, having psoriasis or a rash that was similar to psoriasis and having at least two of the following conditions significantly reduced the likelihood of achieving remission after eight years: dactylitis, nail pitting, enthesitis, first-degree relative with psoriasis, or PsA3. Additionally, children with PsA had a higher risk of illness flare after developing an inactive disease and ceasing therapy compared to children with other types of JIA, with the exception of systemic JIA4. The disease course and treatment of children with PsA, as well as a description of the overall risk of arthritis in children with psoriasis and the time period during which psoriasis patients are most at risk of developing arthritis, still need to be better characterized [31].

50 to 60 percent of cases of oligoarthritis, 11 to 28 percent of cases of RF-negative polyarthritis, 2 to 7 percent of cases of RF-positive polyarthritis, 10 to 20 percent of cases of systemic arthritis, 2 to 15 percent of cases of psoriatic arthritis, and 1 to 7 percent of cases of enthesitis-related arthritis are the different subtypes that are most common. Some geographical areas have higher prevalences of particular subtypes. In North America, RF-negative polyarthritis is more prevalent than oligoarthritis, which is more prevalent in southern Europe. Southeast Asia has a higher prevalence of systemic arthritis and arthritis brought on by enthesitis. Northern and southern Europe have the greatest rates of uveitis, whereas Latin America, Africa, the Middle East, and Southeast Asia have the lowest rates. Except for enthesitis-related arthritis, which primarily affects men, and systemic JIA, which affects both sexes equally, the majority of JIA subtypes are predominately female [7].

4. DISCUSSION

In order to compare patient outcomes between juvenile psoriatic arthritis (PsA) and other juvenile idiopathic arthritis (JIA) subtypes, as well as to assess the traits and genetic markers that may set PsA apart from other JIA subtypes, research was conducted. Psoriasis in the patient or a first-degree relative, dactylitis, and ankle/toe arthritis during the first six months were predictors of PsA. PsA may be distinguished from either oligoarthritis or polyarthritis based on HLA-DRB1*11/12 status and onset beyond age 6 years. Patients with PsA showed worse physical health than the general population after 15 years**.** After 23 years, PsA patients had lower SF-36 physical ratings than people with oligoarthritis or polyarthritis. 33 percent of PsA patients required disease-modifying antirheumatic medications and/or anti-tumor necrosis factor medicines, compared to 8 percent of patients with oligoarthritis and 13 percent of individuals with either oligoarthritis or polyarthritis. Hence, a history of psoriasis, dactylitis, ankle or toe arthritis, and DRB1*11/12 in children with JIA also suggest the possibility of PsA, a subtype linked to a poor prognosis [32].

Retrospective analysis was performed on 60 kids who were thought to have juvenile psoriatic arthritis; the average follow-up time was 10.8 years. There was a 3:2 female to male ratio. Psoriasis and arthritis both started on average between 8 and 9 years ago. Nearly half had a family history of psoriasis, which helped doctors identify the 26 of 60 patients who had arthritis at initially. The majority of cases presented as monoarticular, often of the knee. In both the upper and lower limbs, additional joints often started to become sporadicly affected in an asymmetric pattern, resulting in 87 percent of patients having polyarticular illness. This course of arthritis in children differs from the typical one. Six patients required bilateral hip replacements, four of whom did so within the first five years of the development of arthritis, despite the fact that 40% of them remained asymptomatic at follow-up. Sixteen individuals got slow-acting medications, mostly in the form of gold; eight of them had polyarticular onsets and seven had ANA test results that were positive [33].

In another study 35 youngsters were evaluated for the presence of definite or probable juvenile psoriatic arthritis (JPsA) using a suggested definition of the condition. Having at least three of the following minor criteria—dactylitis, nail pitting, a rash that resembles psoriasis, or a family history of psoriasis—plus arthritis was considered to be definitive JPsA (24 cases). A total of 11 individuals had probable JPsA, which was characterised as arthritis plus two minor criteria. 33 of 35 patients had pauciarticular arthritis at the time of diagnosis, but 23 of 35 went on to develop polyarticular arthritis. Antinuclear antibodies (22 of 35), anticollagen

antibodies (10 of 35), chronic anterior uveitis (6 of 35), HLA-DR4 (2 of 28), and HLA-DR8 (5 of 28) all occurred at rates comparable to those found in individuals with juvenile rheumatoid arthritis. JPsA may resemble juvenile rheumatoid arthritis more than the seronegative spondylarthropathies that it has historically been linked to [34].

5. TREATMENT

Anti-inflammatory and immunomodulatory medications, physical therapy, and maybe surgery, dietary assistance, and psychological support are all required for the treatment of JIA. The selection of pharmacological therapy is influenced by the disease's subtypes, its severity and damage, any concomitant diseases, and the support of the patient's family. For all subtypes, nonsteroidal anti-inflammatory medications (NSAIDs) are the go-to medication for early symptomatic therapy. With the development of powerful contemporary therapies like methotrexate and biologics, the usage of NSAIDs in JIA has gradually reduced. With little stress on joints, physical therapy places an emphasis on range of motion. Swimming is frequently a wise choice. Patients should engage in light stretching, strengthening, and fitness activities [7].

6. CONCLUSION

Juvenile idiopathic arthritis (JIA) is a heterogeneous category of idiopathic inflammatory arthritis that affects children under the age of 16. There is no clear classification of this disease and is catogrized into 7 subtypes. There are no clear etiology for this disease. However, genetic factors plays a key role. Moreover, according to research , a history of psoriasis, dactylitis, ankle or toe arthritis, and DRB1*11/12 in children with JIA also suggest the possibility of PsA, a subtype linked to a poor prognosis. Treatment of this disease mainly depend on immunomodulatory medications and physical therapy.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Petty RE, Southwood TR, Manners P, Baum J, Glass DN, Goldenberg J, He X, Maldonado-Cocco J, Orozco-Alcala J, Prieur AM, Suarez-Almazor ME, Woo P. International league of associations for rheumatology. international league of associations for rheumatology classification of juvenile idiopathic arthritis: second revision, edmonton, 2001. J Rheumatol. 2004;31(2):390-2.
2. Anwar Al Hammadi, et al. Psoriatic Arthritis. Medscape; 2022. Available:https://emedicine.medscape.com/article/2196539-overview#a3
3. Brunello F, Tirelli F, Pegoraro L, Dell'Apa F, Alfisi A, Calzamatta G, Folisi C, Zulian F. New Insights on Juvenile Psoriatic Arthritis. Front Pediatr. 2022;10:884727. DOI: 10.3389/fped.2022.884727. PMID: 35722498; PMCID: PMC9199423.
4. Butbul Aviel Y, Tyrrell P, Schneider R, Dhillon S, Feldman BM, Laxer R, Saurenmann RK, Spiegel L, Cameron B, Tse SM, Silverman E. Juvenile Psoriatic Arthritis (JPsA): juvenile arthritis with psoriasis? Pediatr Rheumatol Online J. 2013;11(1):11. DOI: 10.1186/1546-0096-11-11. PMID: 23497068; PMCID: PMC3622582.
5. Petty RE, Southwood TR, Manners P. International league of associations for rheumatology classification of juvenile idiopathic arthritis: second revision, Edmonton, 2001. J Rheumatol. 2004;31: 390–392.
6. Southwood TR, Petty RE, Malleson PN. Psoriatic arthritis in children. Arthritis Rheum. 1989;32:1007–1013. DOI: 10.1002/anr.1780320810
7. Thatayatikom A, Modica R, De Leucio A. Juvenile Idiopathic Arthritis. [Updated 2022 Jan 25]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022. Available:https://www.ncbi.nlm.nih.gov/books/NBK554605/
8. Southwood TR, Petty RE, Malleson PN. Psoriatic arthritis in children. Arthritis Rheum. 1989;32:1007–1013. DOI: 10.1002/anr.1780320810

9. Lambert JR, Ansell BM, Stephenson E, Wright V. Psoriatic arthritis in childhood. Clin Rheum Dis. 1976;2:339–352.
10. Sills EM. Psoriatic arthritis in childhood. Johns Hopkins Med J. 1980;146:49–53.
11. Shore A, Ansell BM. Juvenile psoriatic arthritis–an analysis of 60 cases. J Pediatr. 1982;100:529–535.
DOI: 10.1016/S0022-3476(82)80747-6
12. Hafner R, Michels H. Psoriatic arthritis in children. Curr Opin Rheumatol. 1996;8: 467–472.
DOI: 10.1097/00002281-199609000-00013
13. Hamilton ML, Gladman DD, Shore A, Laxer RM, Silverman ED. Juvenile psoriatic arthritis and HLA antigens. Ann Rheum Dis. 1990;49:694–697.
DOI: 10.1136/ard.49.9.694
14. Burgos-Vargas R. Spondyloarthropathies and psoriatic arthritis in children. Curr Opin Rheumatol. 1993;5:634–643.
DOI: 10.1097/00002281-199305050-00012
15. Ansell BM. Juvenile psoriatic arthritis. Baillieres Clin Rheumatol. 1994;8:317–332.
DOI: 10.1016/S0950-3579(94)80021-9
16. Roberton DM, Cabral DA, Malleson PN, Petty RE. Juvenile psoriatic arthritis: followup and evaluation of diagnostic criteria. J Rheumatol. 1996;23:166–170.
17. Stoll ML, Zurakowski D, Nigrovic LE, Nichols DP, Sundel RP, Nigrovic PA. Patients with juvenile psoriatic arthritis comprise two distinct populations. Arthritis Rheum. 2006;54:3564–3572.
DOI: 10.1002/art.22173
18. Stoll ML, Lio P, Sundel RP, Nigrovic PA. Comparison of vancouver and international league of associations for rheumatology classification criteria for juvenile psoriatic arthritis. Arthritis Rheum. 2008;59:51–58.
DOI: 10.1002/art.23240
19. Flato B, Lien G, Smerdel-Ramoya A, Vinje O. Juvenile psoriatic arthritis: Longterm outcome and differentiation from other subtypes of juvenile idiopathic arthritis. J Rheumatol. 2009;36:642–650.
DOI: 10.3899/jrheum.080543
20. Nair RP, Duffin KC, Helms C, Ding J, Stuart PE, Goldgar D, et al. Genome-wide scan reveals association of psoriasis with IL-23 and NF-kappaB pathways. Nat Genet. 2009;41(2):199-204.
21. Huffmeier U, Uebe S, Ekici AB, Bowes J, Giardina E, Korendowych E, et al. Common variants at TRAF3IP2 are associated with susceptibility to psoriatic arthritis and psoriasis. Nat Genet. 2010; 42(11):996-9.
22. Sun LD, Cheng H, Wang ZX, Zhang AP, Wang PG, Xu JH, et al. Association analyses identify six new psoriasis susceptibility loci in the Chinese population. Nat Genet. 2010;42(11): 1005-9.
23. Ellinghaus E, Ellinghaus D, Stuart PE, Nair RP, Debrus S, Raelson JV, et al. Genome-wide association study identifies a psoriasis susceptibility locus at TRAF3IP2. Nat Genet. 2010;42(11):991-5.
24. Cargill M, Schrodi SJ, Chang M, Garcia VE, Brandon R, Callis KP, et al. A large-scale genetic association study confirms IL12B and leads to the identification of IL23R as psoriasis-risk genes. Am J Hum Genet. 2007;80(2):273-90.
25. Rozenblit M, Lebwohl M. New biologics for psoriasis and psoriatic arthritis. Dermatol Ther. 2009;22(1):56-60.
26. Huffmeier U, Lascorz J, Bohm B, Lohmann J, Wendler J, Mossner R, et al. Genetic variants of the IL-23R pathway: association with psoriatic arthritis and psoriasis vulgaris, but no specific risk factor for arthritis. J Invest Dermatol. 2009;129(2): 355-8.
27. Al-Heresh AM, Proctor J, Jones SM, Dixey J, Cox B, Welsh K, et al. Tumour necrosis factor-alpha polymorphism and the HLA-Cw*0602 allele in psoriatic arthritis. Rheumatology (Oxford). 2002;41(5): 525-30.
28. Choy E. T cells in psoriatic arthritis. Curr Rheumatol Rep. 2007;9(6):437-41.
29. Ho PY, Barton A, Worthington J, Plant D, Griffiths CE, Young HS, et al. Investigating the role of the HLA-Cw*06 and HLA-DRB1 genes in susceptibility to psoriatic arthritis: comparison with psoriasis and undifferentiated inflammatory arthritis. Ann Rheum Dis. 2008;67(5):677-82.
30. Nickoloff BJ, Nestle FO. Recent insights into the immunopathogenesis of psoriasis provide new therapeutic opportunities. J Clin Invest. 2004;113(12): 1664-75.
31. Brandon TG, Manos CK, Xiao R, Ogdie A, Weiss PF. Pediatric psoriatic arthritis: A population-based cohort study of risk factors for onset and subsequent risk of

inflammatory comorbidities. J Psoriasis Psoriatic Arthritis. 2018;3(4):131-136.
DOI: 10.1177/2475530318799072. Epub 2018 Sep 18. PMID: 31355354; PMCID: PMC6660180.

32. Flatø B, Lien G, Smerdel-Ramoya A, Vinje O. Juvenile psoriatic arthritis: Longterm outcome and differentiation from other subtypes of juvenile idiopathic arthritis. J Rheumatol. 2009;36(3): 642-50.
DOI: 10.3899/jrheum.080543. Epub 2009 Feb 4. PMID: 19208605.

33. Shore A, Ansell BM. Juvenile psoriatic arthritis- An analysis of 60 cases. J Pediatr. 1982;100(4):529-35.
DOI: 10.1016/s0022-3476(82)80747-6. PMID: 7199570.

34. Southwood TR, Petty RE, Malleson PN, Delgado EA, Hunt DW, Wood B, Schroeder ML. Psoriatic arthritis in children. Arthritis Rheum. 1989;32(8): 1007-13.
DOI: 10.1002/anr.1780320810. PMID: 2765001.

Peer-review history:
The peer review history for this paper can be accessed here:
https://www.sdiarticle5.com/review-history/95399