International Journal of Dermatology, Venereology and Leprosy Sciences

E-ISSN: 2664-942X
P-ISSN: 2664-9411
www.dermatologypaper.com
Derma 2024; 7(1): 21-26
Received: 08-11-2023
Accepted: 15-12-2023

Mai Tarek
Department of Dermatology & Venereology, Faculty of Medicine, Tanta University, Tanta, Egypt

Mohamed Gamei
Department of Dermatology & Venereology, Faculty of Medicine, Tanta University, Tanta, Egypt

Hossam Hodeib
Department of Pathology, Faculty of Medicine, Tanta University, Tanta, Egypt

Ramy Atlam
Department of Cardiology, Faculty of Medicine, Tanta University, Tanta, Egypt

Yasmina EL Attar
Department of Dermatology & Venereology, Faculty of Medicine, Tanta University, Tanta, Egypt

Corresponding Author:
Hossam Hodeib
Department of Pathology, Faculty of Medicine, Tanta University, Tanta, Egypt

Serum Interleukin-17 in psoriatic patients implication for cardiovascular comorbidities

Mai Tarek, Mohamed Gamei, Hossam Hodeib, Ramy Atlam and Yasmina EL Attar

DOI: https://doi.org/10.33545/26649411.2024.v7.i1a.172

Abstract
Psoriasis is a multifactorial autoimmune chronic relapsing skin disorder whose pathogenesis is not exactly known. It is caused by complex interaction between genetic, immunological, psychological and environmental factors. Interleukin-17 (IL-17, also known as IL-17A) is a cytokine that has been recognized in promoting a variety of chronic inflammatory and autoimmune disorders. IL-17 is a 155-amino acid protein that is a disulfide linked, homodimeric, secreted glycoprotein with a molecular weight of 35 KD. IL-17 is suggested to be involved in neutrophil accumulation followed by the formation of epidermal microabcesses in psoriasis. Together with other Th 17 cytokines, IL-17 also upregulates the production of several cytokines that are implicated in psoriasis pathogenesis. An association of increased cardiovascular comorbidity with psoriasis has been postulated. Psoriatic patients are more likely to have an increased body mass index, dyslipidemia, hypertension and diabetes mellitus which are well known risk factors for cardiovascular comorbidities. Even though, studies support the existence of an independent relationship between psoriasis and cardiovascular diseases. Some reports indicated that IL-17 may represent one of the main links between cardiovascular disease manifestation and psoriatic inflammation. IL-17 has been suggested to play an important role in the pathogenesis and prognosis of cardiovascular comorbidity that may be associated with psoriasis. Biologic anti-IL-17 therapy may be recommended not only to improve skin manifestations of psoriasis, but also to help minimize the potential risk of developing cardiovascular accidents.

Keywords: Serum Interleukin, cardiovascular accidents, cardiovascular comorbidities, skin disorder

Introduction
Psoriasis is a multifactorial autoimmune chronic relapsing skin disorder that requires lifelong treatment. It is caused by complex interaction between genetic, immunological, psychological and environmental factors. It is characterized by erythematous scaly plaques covered with silvery white lamellar scales that can be present on extensor aspect of extremities, trunk and scalp [1].

Clinical types of psoriasis [2].
Plaque psoriasis: This is the most common type of psoriasis manifested as erythematous scaly plaques covered with silvery white lamellar scales.

Guttate psoriasis: Lesions appear as small erythematous scaly papules on trunk and proximal extremities, usually after streptococcal infection and disappear spontaneously as the infection regresses. This type is more common in children and young adults.

Erythrodermic psoriasis: There is widespread erythema affecting more than 90% of the body surface area in this type of psoriasis. Hypothermia, hypoproteinemia and systemic complications such as cardiac and renal failure may occur.

Pustular psoriasis: This type is characterized by multiple sterile pustules which can be localized or generalized.

Arthropathic psoriasis: This type usually involves painful inflammation of the joints most commonly affecting joints of fingers and toes resulting in sausage-shaped swelling.

Skin lesions seem to occur before arthritis in about 75% of cases.

Palmo-planter psoriasis: There are well defined erythematous scaly plaques symmetrically affecting palm and sole characterizing this type of psoriasis.

Scalp psoriasis: This type is characterized by well-defined red scaly lesions in scalp that often extend slightly below the hair line. It can be a single patch or several and can even affect your entire scalp. It can also spread to the forehead, the back of neck, and behind and inside ears.

Psoriasis of nail: Psoriasis of the nails is manifested as nail pitting, subungual hyperkeratosis, oil spots under the nails, thickening and discoloration of nail plate and onycholysis. Fingernails are more commonly affected than toenails.

Inverse (flexural) psoriasis: This is an uncommon type of psoriasis that affects skin folds such as axilla, groin and under breast. The eruption is characterized by severe erythema with less scaling. This type occurs more in obese individuals and more resistant to treatment [2].

Assessment of severity of psoriasis:

The assessment of severity of psoriasis is mainly based on two scoring systems: Psoriasis Area and Severity Index (PASI) and Dermatology Life Quality Index (DLQI). Psoriasis Area and Severity Index (PASI) is calculated from body surface area involvement (7-point score for each of four anatomical areas including head, trunk, upper extremities and lower extremities) and score of erythema, scaling and induration (5-point score for each from 0-4). The score range from 0 to 72, where 0 means no disease, and 72 means maximal disease. Dermatology Life Quality Index (DLQI) is a questionnaire designed to measure how much skin problems have affected a patient's life. It consists of 10 questions concerning patient perception of impact of skin disease on different aspects of his quality of life over the last week [3].

Treatment of psoriasis

1. **Topical treatment:** Topical corticosteroid, vitamin D analogues, topical immunomodulators (Calcineurin inhibitors), emollients and moisturizers, coal tar, salicylic acid, anthralin (dithranol) and topical retinoids (Tazarotene) [4].
2. **Phototherapy:** Ultraviolet B (UVB) and psoralen plus ultraviolet A (PUVA): UVB therapy is frequently combined with one or more topical treatments. The Goeckerman protocol has been demonstrated to result in disease remission in more than 80% of patients when coral tar is combined with UVB exposure [5].
3. **Systemic therapy:** For those with body surface involvement of more than 10% to 20%, pustular psoriasis, erythrodermic psoriasis, psoriatic artheritis, and more localized, recalcitrant psoriasis should be considered for systemic therapy [6].

Five classes of biologics are currently available

Blockers of tumor necrosis factor-alpha (Certolizumab pegol, entanercept, adalimumab, infliximab, and golimuab). Interleukin 12 and 23 (IL-12/23) inhibitors (Ustekinumab). IL-17 inhibitors (Secukinumab, brodalumab, and ixekizumab). T-cell inhibitors (Abatacept, alefacept, and efalizumab). IL-23 inhibitors (Tildrakizumab-asmn, risankizumab-rzaa and guselkumab). Each of these molecules is a specific messenger in the immune system involved in the development of psoriasis [7].

Comorbidity of psoriasis

Autoimmune disorders: Psoriasis, as an immune mediated inflammatory disorder, may be more likely to be associated with other immune related diseases such as rheumatoid arthritis, inflammatory bowel diseases including crohn's disease and ulcerative colitis [8].

Tumor association: Psoriatic patients are supposed to have increased risk of developing cutaneous T-cell lymphoma and Hodgkin's disease [9].

Psychological comorbidity: Disturbance in social and occupational activity is more common in psoriasis. (10)

Metabolic comorbidities: The link with psoriasis can be explained as metabolic syndrome is characterized by increased activity of Th1 cells [11].

Cardiovascular comorbidities in psoriasis

Psoriasis, being a multisystem chronic inflammatory disease, is supposed to be associated with a higher prevalence of cardiovascular risk factors contributing to accelerated atherosclerosis, hypertension, coronary artery disease and higher relative risk of myocardial infarction. Psoriatic patients are at increased risk of cardiovascular comorbidities and are at greater risk for subsequent major cardiovascular events compared with the general population. (12) Atherosclerosis is likely driven by a combination of traditional risk factors which occur more frequently in psoriasis and by systemic inflammation with associated proinflammatory cytokines production. Psoriasis is known to be associated with several risk factors, such as smoking, alcohol, diabetes, hypertension and dyslipidemia, taking in consideration that psoriasis itself may be an independent risk factor for cardiovascular diseases [13].

Atherosclerosis is a chronic inflammatory cardiovascular disease characterized by lipid deposition in the arterial wall with smooth muscle cell and fibrous matrix proliferation, resulting in gradual development of atherosclerotic plaque. This plaque leads to platelet aggregation and thrombosis with subsequent stenosis or occlusion of blood vessels resulting in acute cardiovascular events such as myocardial infarction. Endothelial dysfunction with oxidative stress and proinflammatory cytokines production such as TNF-α, IL-6 and IL-17 are clinically involved in pathogenesis of chronic inflammatory processes related to psoriasis and atherosclerosis. Some carotid ultrasound studies have reported a higher prevalence of carotid artery plaques and vascular inflammation in psoriatic patients compared to healthy controls. Carotid duplex ultrasound is a non-invasive imaging technique that can identify the extent of atherosclerosis by measuring the intimal medial thickness, internal diameter and arterial wall mass. This is valuable for early detection of subclinical atherosclerosis in such patients [14]. Some reports have suggested that the prevalence of hypertension is higher in psoriatic patients compared to that in the general population, especially in patients with psoriatic arthritis. Moreover, the chance of having

uncontrolled blood pressure is increased in patients with more severe psoriasis, and this association may be still significant even after controlling other hypertension risk factors such as diabetes mellitus, hypercholesterolemia and high body mass index (BMI), suggesting that psoriasis can be an independent risk factor for hypertension [15]. On the other hand, patients with hypertension were reported to have a higher risk of developing psoriasis, possibly related to use of beta blockers [16]. Hypercholesterolemia is an established risk factor for cardiovascular diseases. Moreover, hypercholesterolemia, through involvement of immune system, can induce inflammation in the vessel wall. T-lymphocytes play an important role in psoriasis pathogenesis, and it has been proved that one of the early immune cell types activated by hypertension is T-lymphocyte. Several studies have reported lipid alteration in psoriatic patients including changes in concentrations of total cholesterol, triglycerides, low density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C). Specific immune cells, called CD1 restricted self-lipid reactive T-cells have been identified in the blood and skin of humans, which may be the missing link between psoriasis and dyslipidemia. It has been postulated that insulin resistance possibly explains the cardiovascular comorbidity associated with systemic inflammatory diseases such as psoriasis. Insulin resistance contributes to endothelial cell dysfunction, subsequently leading to atherosclerosis and finally to end organ damage, stroke or myocardial dysfunction. There is growing evidence that insulin resistance, metabolic syndrome and atherosclerosis have a common inflammatory basis. Moreover, these conditions have been suggested to be significantly associated with severity of psoriasis [17].

Studies which have suggested that psoriasis is associated with subclinical atherosclerosis and high risk of cardiovascular diseases have been increased. Arterial stiffness has been recognized as a factor contributing to cardiovascular morbidity and mortality. Some reports have revealed increased arterial stiffness in psoriatic patients compared to controls, as shown by arterial pulse wave velocity (aPWV) which is a standard measurement of arterial stiffness. Also, higher coronary artery plaque burden in psoriasis has been shown by some studies, and reported to be associated with underlying disease severity. Finally, a number of epidemiologic studies have also suggested that psoriatic patients have an increased risk of myocardial infarction independent of other established risk factors, but this conclusion remains controversial [18].

Interleukin-17

Interleukin-17 (IL-17, also known as IL-17A) is a cytokine that has been recognized in promoting a variety of chronic inflammatory and autoimmune disorders. IL-17 is a 155-amino acid protein that is a disulfide linked, homodimeric, secreted glycoprotein with a molecular weight of 35 KD. The IL-17 family contains six isoforms and includes 6 structure related cytokines (A-F) [19].

Interleukin-17 and skin

Interleukin-17 is an essential proinflammatory cytokine which is suggested to be associated with the pathogenesis of several inflammatory skin diseases including psoriasis, atopic dermatitis, hidradenitis suppurativa, alopecia areata, pitryasis rubra pilaris, pemphigus vulgaris and systemic sclerosis. As IL-23 plays a pivotal role in stimulating the production of IL-17 by activating Th17 cells, the IL-23/IL-17 axis is now becoming an important pathway for targeted therapy for such inflammatory diseases. The role of IL-23 is crucial in the differentiation of IL-17 expressing phenotypes via activating the transcription factor retinoid-related orphan receptor γ and signal transducer and activator of transcription 3 (STAT-3) [20].

Atopic dermatitis (AD)

Essentially, atopic dermatitis is considered a Th2 immune response with elevated levels of IgE. However, studies have revealed that Th1, Th2, Th22 and Th17 cells are involved in the pathogenesis of atopic dermatitis. It has been demonstrated that Th22 and Th17 immune responses contribute to chronic skin lesions of atopic dermatitis, especially in children. IL-17E level is increased in the epidermis of atopic dermatitis patients, associated with skin barrier dysfunction [21].

Hidradenitis suppurativa (HS)

The data on the efficacy and safety of biologics targeting IL-17 for treating hidradenitis suppurativa are limited. However, secukinumab has been reported to be effective in treatment of hidradenitis suppurativa when administered in a dose of 300 mg once a week for 5 weeks. Also, brodalumab was demonstrated to be effective in hidradenitis suppurativa treatment with good improvement as early as week 2 [22].

Alopecia areata (AA)

The Th17 cell frequencies and IL-17 levels are significantly increased both in the peripheral blood and scalp lesions in patients with alopecia areata. However, studies have reported that patients with alopecia areata don't show significant improvement with anti-IL-17 therapy secukinumab. Therefore, the contribution of Th17/IL-17 in the pathogenesis of alopecia areata is still controversial [23].

Pityriasis rubra pilaris (PRP)

The IL-23/IL-17 axis seems to play a role in the pathogenesis of pitryasis rubra pilaris as shown by clinical and histopathological improvement by biologics targeting this axis. Patients with pitryasis rubra pilaris who received ustekinumab or secukinumab showed significant decrease in erythema, follicular hyperkeratosis, and scaling during a 15-month follow up period [24].

Pemphigus vulgaris

The levels of IL-17 and IL-23 have been shown to be increased in serum and lesional skin of patients with pemphigus vulgaris and are significantly correlated with disease severity. Treatment of pemphigus vulgaris with biologics targeting IL-17 is still under investigation [25].

Systemic sclerosis (SSc)

Quantitative analysis of Th17 cytokines in lesional skin of systemic sclerosis showed that the expression of IL-17A mRNA is higher compared with healthy controls. Also, serum IL-17 level was elevated in patients with systemic sclerosis and correlated with disease severity and collagen overproduction. The elevated levels of IL-17A act on dermal vascular smooth muscle cells to promote vascular fibrosis in patients with systemic sclerosis, via activating extracellular signaling pathway [26].

The role of Interleukin-17 in psoriasis

The immunologic cascade mediated by IL-17 pathway has been suggested to play a critically important role in the pathogenesis of psoriasis. IL-23-IL-17 axis is believed to be the predominant pathway in psoriasis pathogenesis. IL-23 supports the survival, differentiation and activation of Th-17 cells that secrete IL-17 cytokines. Moreover, neutrophils and mast cells release IL-17 through IL-23 activation. IL-23 is produced by dendritic cells and macrophages and binds to IL23R on activated Th-17 cells leading to the production of effector IL-17 cytokines. This may explain why therapies targeting IL-23 are effective in psoriasis [27]. (Figure 1)

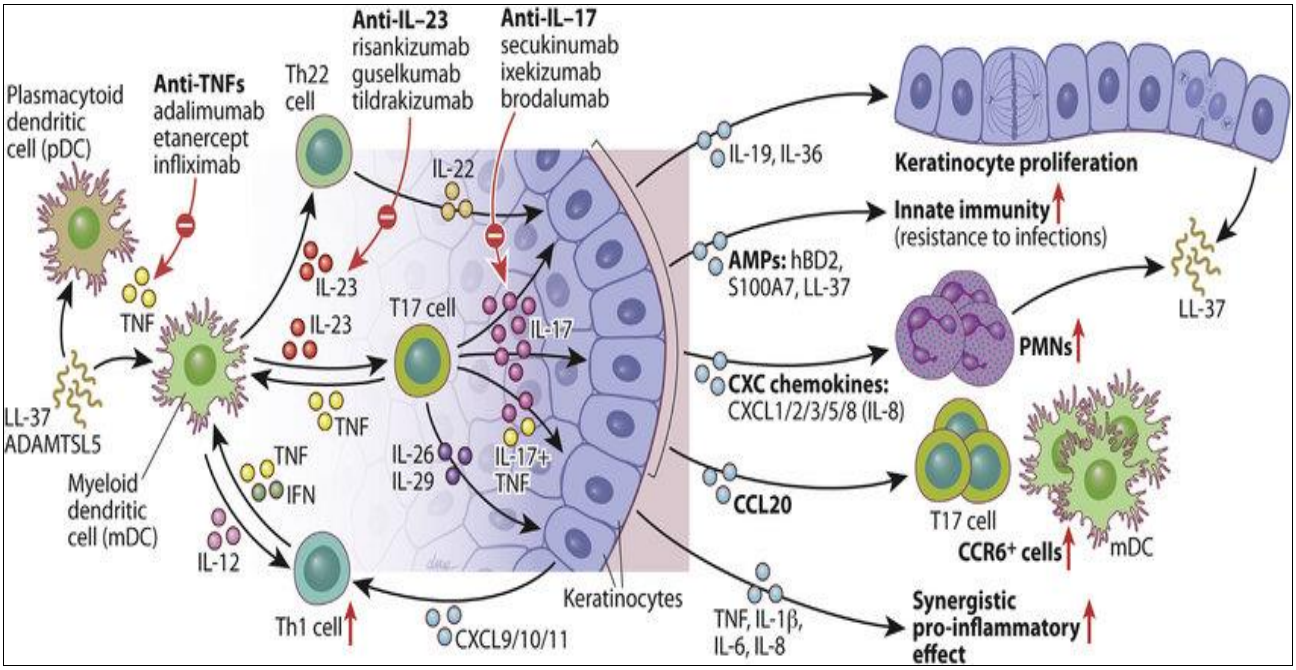

Fig 1: IL-23/T17-mediated effects on epidermal keratinocytes (KCs) in psoriatic skin [28]

Keratinocytes, endothelial cells and immune cells are all targets of IL-17 in the psoriasis pathway. IL-17 acts directly on keratinocytes resulting keratinocyte proliferation as well as upregulation of chemokines such as IL-6, IL-8 and stimulation of angiogenesis. The IL-17 pathway is also hypothesized to modulate the inflammatory responses linking comorbid systemic diseases with psoriasis. Furthermore, the clinical response seen with current and emerging therapies targeting IL-17 emphasizes the importance of IL-17 cytokines in the pathogenesis of psoriasis [29].

The IL-17 and psoriasis comorbidities

Psoriasis is associated with several comorbidities including cardiovascular diseases, metabolic syndrome, psychological illness, obesity and inflammatory bowel diseases, although the exact mechanism behind this relationship is unknown. Alterations and dysfunction of the immune system are proposed to be the key mechanisms linking these disease states. Given the fundamental role it plays in psoriasis, the IL-17 pathway is hypothesized to modulate the inflammatory response seen in both primary psoriatic shin disease and comorbid systemic diseases. Studies suggest that IL-17 may play a pathogenic role in psoriasis-associated cardiovascular dysfunction, depressive disorder and obesity [30].

Several studies support the role of IL-17 in psoriasis and cardiovascular dysfunction. Studies on mice demonstrated that overexpression of IL-17A in keratinocytes resulted in psoriasis-like skin changes together with increased vascular oxidative stress, endothelial dysfunction and arterial hypertension. Also, patients with carotid atherosclerosis were noted to have significantly increased IL-23 plasma levels, compared to controls, and increased levels of IL-23 and IL-23R within atherosclerotic plaques, suggesting a role of IL-23/Th-17 axis in pathogenesis of atherosclerosis. (31) Furthermore, it has been reported that serum IL-17 is increased threefold in hypertensive patients, and may mediate the critical hypertensive response to angiotensin II [32].

Interleukin-17 may mediate the feed-forward inflammatory cycle associated with obesity. Adipocytes and macrophages in adipose tissues exacerbate the inflammatory state of psoriasis by inducing several proinflammatory cytokines, including IL-6 which mediates Th-17 production by naïve T cells. This leads to elevated levels of plasma IL-17 observed in obese patients when compared to controls. It has been hypothesized that IL-17 mediates the link between obesity and psoriasis through maintaining inflammation in adipose tissues and stimulating lipolysis of adipocytes [33].

Conclusion

Interleukin-17, besides its role in the pathogenesis and severity of psoriasis, can be considered as one of the main links between cardiovascular comorbidity and psoriasis. Biologic anti-IL-17 therapy may be recommended not only to improve skin manifestations of psoriasis, but also to help minimize the potential risk of developing cardiovascular accidents.

Conflict of Interest

Not available

Financial Support

Not available

References

1. Bejarano JJR, Valdecantos WC. Psoriasis as autoinflammatory disease. Dermatologic Clinics. 2013;31(3):445-60.
2. Khan S, Das A, Uddin KM, Zia S, Khan A, Memon MS, *et al*. Psoriasis and its Variants. National Journal of Health Sciences. 2022;7(4):181-92.
3. Prevezas C, Katoulis AC, Papadavid E, Panagakis P, Rigopoulos D. Short-term correlation of the psoriasis area severity index, the nail psoriasis area severity index, and the dermatology life quality index, before and after treatment, in patients with skin and nail psoriasis. Skin Appendage Disorders. 2019;5(6):344-9.
4. Maul JT, Anzengruber F, Conrad C, Cozzio A, Häusermann P, Jalili A, *et al*. Topical treatment of psoriasis vulgaris the Swiss treatment pathway. Dermatology. 2021;237(2):166-78.
5. Branisteanu DE, Dirzu DS, Toader MP, Branisteanu DC, Nicolescu AC, Brihan I, *et al*. Phototherapy in dermatological maladies. Experimental and Therapeutic Medicine. 2022;23(4):1-6.
6. Raharja A, Mahil SK, Barker JN. Psoriasis: A brief overview. Clinical Medicine. 2021;21(3):170-3.
7. Smith C, Lopez JZ, Yiu Z, Bale T, Burden A, Coates L, *et al*. British association of dermatologists guidelines for biologic therapy for psoriasis 2017. British Journal of Dermatology. 2017;177(3):628-36.
8. Halling ML, Kjeldsen J, Knudsen T, Nielsen J, Hansen LK. Patients with inflammatory bowel disease have increased risk of autoimmune and inflammatory diseases. World Journal of Gastroenterology. 2017;23(33):6137-46.
9. Davis MS, Spencer RK, Johnson CE, Elhage KG, Jin JQ, Hakimi M, *et al*. Risk of Cutaneous T Cell Lymphoma with Psoriasis Biologic Therapies. Dermatology and Therapy. 2024;14(1):15-30.
10. Leman J, Walton S, Layton AM, Ward KA, McBride S, Cliff S, *et al*. The real world impact of adalimumab on quality of life and the physical and psychological effects of moderate-to-severe psoriasis: A UK prospective, multicenter, observational study. Journal of Dermatological Treatment. 2020;31(3):213-21.
11. Branisteanu DE, Pirvulescu RA, Spinu AE, Porumb EA, Cojocaru M, Nicolescu AC, *et al*. Metabolic comorbidities of psoriasis. Experimental and Therapeutic Medicine. 2022;23(2):1-7.
12. Weber B, Merola JF, Husni ME, Di Carli M, Berger JS, Garshick MS. Psoriasis and cardiovascular disease: novel mechanisms and evolving therapeutics. Current Atherosclerosis Reports. 2021;23:1-11.
13. Szentpetery A, Haroon M, FitzGerald O. Cardiovascular comorbidities in psoriatic disease. Rheumatology and Therapy. 2020;7:5-17.
14. El-Mongy S, Fathy H, Abdelaziz A, Omran E, George S, Neseem N, *et al*. Subclinical atherosclerosis in patients with chronic psoriasis: A potential association. Journal of the European Academy of Dermatology and Venereology. 2010;24(6):661-6.
15. Tablazon IL, Al-Dabagh A, Davis SA, Feldman SR. Risk of cardiovascular disorders in psoriasis patients: Current and future. American Journal of Clinical Dermatology. 2013;14:1-7.
16. Masson W, Lobo M, Molinero G. Psoriasis and cardiovascular risk: A comprehensive review. Advances in Therapy. 2020;37(5):2017-33.
17. Wakkee M, Thio H, Prens E, Sijbrands E, Neumann H. Unfavorable cardiovascular risk profiles in untreated and treated psoriasis patients. Atherosclerosis. 2007;190(1):1-9.
18. Garshick MS, Ward NL, Krueger JG, Berger JS. Cardiovascular risk in patients with psoriasis: JACC review topic of the week. Journal of the American College of Cardiology. 2021;77(13):1670-80.
19. McGeachy MJ, Cua DJ, Gaffen SL. The IL-17 family of cytokines in health and disease. Immunity. 2019;50(4):892-906.
20. Topal FA, Zuberbier T, Makris MP, Hofmann M. The role of IL-17, IL-23 and IL-31, IL-33 in allergic skin diseases. Current Opinion in Allergy and Clinical Immunology. 2020;20(4):367-73.
21. Nakahara T, Nakahara KM, Tsuji G, Furue M. Basics and recent advances in the pathophysiology of atopic dermatitis. The Journal of Dermatology. 2021;48(2):130-9.
22. Kashetsky N, Mufti A, Alabdulrazzaq S, Lytvyn Y, Sachdeva M, Rahat A, *et al*. Treatment outcomes of IL-17 inhibitors in hidradenitis suppurativa: A systematic review. Journal of Cutaneous Medicine and Surgery. 2022;26(1):79-86.
23. Tomaszewska K, Kozłowska M, Kaszuba A, Lesiak A, Narbutt J, Janowska ZAM. Increased serum levels of interleukin-17 in patients with alopecia areata and non-segmental vitiligo. Advances in Dermatology and Allergology/Postępy Dermatologii I Alergologii. 2022;39(1):195-9.
24. Boudreaux BW, Pincelli TP, Bhullar PK, Patel MH, Brumfiel CM, Li X, *et al*. Secukinumab for the treatment of adult-onset pityriasis rubra pilaris: A single-arm clinical trial with transcriptomic analysis. British Journal of Dermatology. 2022;187(5):650-8.
25. Anguti F, Raziloo H, Fununi AM, Sereshteh S, Rajabpour M, Mohebbi A. Enhancing the Expression of Interleukin-17 Level in Pemphigus Vulgaris Patients. Biomedical Research. 2023;1(1):15-8.
26. Ahmed S, Misra DP, Agarwal V. Interleukin-17 pathways in systemic sclerosis-associated fibrosis. Rheumatology International. 2019;39(7):1135-43.
27. Gaffen SL, Jain R, Garg AV, Cua DJ. The IL-23–IL-17 immune axis: From mechanisms to therapeutic testing. Nature Reviews Immunology. 2014;14(9):585-600.
28. Hawkes JE, Chan TC, Krueger JG. Psoriasis pathogenesis and the development of novel targeted immune therapies. Journal of Allergy and Clinical Immunology. 2017;140(3):645-53.
29. Akhtar T, Wani WY, Kamal MA, Kaur R. Role of angiogenic growth factors in psoriasis: A review. Current Drug Metabolism. 2018;19(11):910-6.
30. Ni C, Chiu MW. Psoriasis and Comorbidities: Links and Risks. Clinical, Cosmetic and Investigational Dermatology. 2014;17:119-32.
31. Samotij D, Nedoszytko B, Bartosińska J, Baran BA, Czajkowski R, Dobrucki I, *et al*. Pathogenesis of psoriasis in the "OMIC" era. Part I. Epidemiology, clinical manifestation, immunological and neuroendocrine disturbances. Advances in Dermatology and Allergology/Postępy Dermatologii I Alergologii. 2020;37(2):135-53.
32. Nguyen H, Chiasson VL, Chatterjee P, Kopriva SE,

Young KJ, Mitchell BM. Interleukin-17 causes Rho-kinase-mediated endothelial dysfunction and hypertension. Cardiovascular Research. 2013;97(4):696-704.
33. Paroutoglou K, Papadavid E, Christodoulatos GS, Dalamaga M. Deciphering the association between psoriasis and obesity: Current evidence and treatment considerations. Current Obesity Reports. 2020;9:165-78.

How to Cite This Article
Tarek M, Gamei M, Hodeib H, Atlam R, Attar YEL. Serum Interleukin-17 in psoriatic patients implication for cardiovascular comorbidities. International Journal of Dermatology, Venereology and Leprosy Sciences. 2024;7(1):21-26.