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Research Article

Assessment of the Impact of Apremilast on Levels of IL-17, IL-23, and Lipids in Obese Psoriatic Patient

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Abstract

Background: Psoriasis is an immune-mediated inflammatory disease with unknown aetiology that may be associated with the defect in proliferation and differentiation of the keratinocytes related to inflammatory cell infiltration. According to published reports, it is universal in occurrence; its prevalence in different populations varies from 0.1% to 11.8%. Receiving Apremilast resulted in a strong reduction in interleukin 17 and interleukin 23, as well as reduced expression of other inflammatory cytokines and improvement of psoriatic lesions.

Objectives: This study aimed to assess the impact of Apremilast on levels of IL-17, IL-23, and lipids in obese psoriatic patients.

Methods: Thirty obese patients with psoriasis were included in this prospective interventional study to measure serum levels of lipid profile, IL-17, and IL-23, before and after receiving Apremilast treatment. A t-test was used to compare between means.

Results: The mean age of the participants was 38 years. The most common age group was 30–40 years. The levels of IL-17 before the administration of Apremilast were 225.55 ± 7.70 pg/mL. After six months of treatment with Apremilast, a statistically significant reduction was seen, with the value decreasing to 183.41 ±2.33 pg/ml. IL-22 levels before the administration of Apremilast were measured to be 76.42 ± 4.03 pg/mL. After six months of treatment with Apremilast, these levels exhibited a non-significant decrease to 67.15 ± 5.40 pg/ml. Modest alterations were noted in the lipid profile.

Conclusion: The use of Apremilast is effective in decreasing IL-17 levels, which have pro-inflammatory effects; this leads to improvement in psoriatic lesions. Moreover, receiving Apremilast in obese psoriatic individuals led to a reduction in TG levels and an elevation in HDL-C levels. Additionally, a rise in TC levels and LDL-C was seen.

Keywords: Apremilast; IL-17; IL-23; Obese; Psoriasis.

Introduction

Psoriasis is an immune-mediated inflammatory disease with unknown etiology that may be associated with the defect in proliferation and differentiation of keratinocytes associated with inflammatory cell infiltration particularly consisting of T-lymphocytes, macrophages, and neutrophils (1). Psoriasis is a common disease characterized by highly proliferating keratinocytes and extensive leukocyte infiltration (2). These skin lesions are typically covered with dry, fragile, loosely attached, silvery, or greyish-white scales with a micaceous appearance (3).

The most characteristic lesions consist of red, scaly, well-demarcated plaques (4). It is universal in occurrence; its prevalence in different populations varies from 0.1% to 11.8%, according to published reports (5). Psoriasis is a common disease with unknown etiology (6). It is characterized by inflammation, autoimmune responses, and abnormal proliferation of skin cells (7). The disease involves inflammation and scaling of the skin as epidermal cells come to the surface prematurely before Completing their maturation process (8). Apremilast phosphodiesterase-4 inhibitors (PDE-4 inhibitors) inhibit the degradation of cAMP, thus increasing the concentration of cAMP and ultimately reducing the expression of pro-inflammatory mediators, including IFN-γ, TNF-α, and IL-2, IL-12, and IL-23 (9).

In the pharmacodynamic analyses in patients with moderate to severe psoriasis, Apremilast demonstrated partial inhibition of key cytokines that regulate inflammation in psoriasis, including IL-23 and IL-17 (10). Furthermore, it led to a decrease in the infiltration of myeloid dendritic cells and T lymphocytes into the epidermis and dermis of psoriatic lesions (11). Additionally, IL17- plays a pivotal role in the pathogenesis of psoriasis. Dysregulation in the production of IL-17 induces chronic inflammation and autoimmune disorders (12). The induction of immunological responses at skin surfaces is mostly dependent on IL17- cytokines, in particular on IL-17A. As a result, neutrophils migrate into the surrounding tissue (13, 14). The IL-17/23 axis is central to psoriasis pathogenesis, and the efficacy of monoclonal antibodies targeting IL-17

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supports the importance of this cytokine in the disease. Inhibiting antibodies against IL-17A have shown success in treating psoriasis (15, 16). In addition, IL-23 is involved in the pathophysiology of psoriasis, and by inhibiting the function of IL-23, inflammation that produces psoriasis symptoms can be reduced (17).

Previous studies have demonstrated that during phase 3 clinical trials, modest elevations in total cholesterol, HDL cholesterol, and triglyceride levels were detected following 24-52 weeks of Apremilast administration. Nevertheless, it should be noted that these increments were deemed to lack clinical significance (18). According to Gualtierotti et al. (2019), Apremilast has beneficial effects on the metabolic profile, in their case study, they reported that Apremilast was associated with an improvement in the lipid profile of the patient (19). In addition to improvements in psoriatic disease activity, Apremilast has been reported to be associated with weight loss (20).

Although Apremilast has been employed as a treatment for psoriasis, the existing body of research, pertaining to its effects on inflammatory cytokines such as IL-17 and IL-23 in patients, remains limited. Hence, the primary objective of this research effort was to examine the impact of Apremilast on the levels of IL-17 and IL-23, along with the lipid profile, in the serum of obese patients with psoriasis.

Patients, Materials, and Methods

This prospective interventional study was conducted at the Dermatology Center, Medical City in Baghdad, Iraq, between November 2021 and December 2022. Participants in the current study were informed about the aim of the study, the nature of the illness, course prognosis, and Apremilast treatment as well as its potential complications by a Dermatologist. Also, formal consent was obtained from each patient before starting the study. Ethical approval No. 145 on 10-11-2022 was obtained from the Development Department at the Medical City Directorate in Baghdad, Iraq.

A total of 30 psoriatic patients (19malles and 11 females) attending the outpatient clinic were enrolled in the study. These patients received Apremilast (Aprezo®), administered twice daily after food (approximately 12 hours apart) without food or drink restrictions and with titration over the first week to mitigate gastrointestinal side effects, started with 10 mg morning dose with a daily increment of 10 mg until day 6 when the recommended dose (30 mg bid) is reached which is continued at thereafter for six months. Of the 30 patients enrolled, 6 did not complete the study for various reasons.

Blood samples were obtained from all patients before receiving Apremilast at the beginning of the study. A second sample was obtained from each patient after they had been receiving Apremilast for 24 weeks.

These samples were employed for the purpose of quantifying IL17-, IL23-, TC, HDL-C, and TG. The levels of IL17- and IL23- were quantified using commercially available human ELISA kits obtained from ELK Biotechnology-China. The lipid profile tests were performed using a colorimetric assay kit supplied by Linear Company.

The BMI of each patient was calculated according to the international standard equation "[BMI = weight (kg) / (height (m))2]". Baseline BMI was calculated and monitored monthly.

The Statistical Analysis System- SAS (2018) program was used to detect the effect of difference between the two groups in study parameters, the t-test was used to compare between means, Chi-squared test was used to compare between percentages. The P value ≤0.05 is considered significant (21).

Results

Demographic characteristics of participants

In the present study, data showed that nineteen (63.3%) of the participants were males and eleven (36.7%) were females. Moreover, cigarette smokers were only four (13.3%) out of 30 participants, while twenty-six (86.7%) were non-smokers. In addition, the average age of the group was 38 years. The most common age group was 30-40 years (60% of participants) followed by the >40 years' age group (26.6% of participants) (Table 1).

Table 1: Demographic characteristics of the study sample

	No.	Percentage
Sex		
Male	19	63.33
Female	11	36.67
Total	30	100%
P-value	---	0.074 NS
Smoking		
Yes	4	13.33
No	26	86.67
Total	30	100%
P-value	---	0.0001 **
** ($P \leq 0.01$)		
Age group (year)		
<30	4	13.33
30-40	18	60.00
>40	8	26.67
Total	30	100%
P-value	---	0.0052 **

** ($P \leq 0.01$), NS: Non-Significant

Interleukins-17 and Interleukins-23 (IL-23): In the current study, prior to receiving Apremilast, the mean IL-17 levels were 225.55 pg/ml. After six months of treatment, the mean IL-17 level decreased significantly to 183.41 pg/ml ($p < 0.01$). In addition, the mean IL-23 level prior to Apremilast was 76.42 pg/ml. After six months of treatment, the mean IL-23 level decreased to 67.15 pg/ml. However, this decrease was not statistically significant ($P>0.05$), as illustrated in Table 2.

Table 2: Comparison between Interleukins-17 and Interleukins-23 levels before and after receiving Apremilast

Group	Mean ± SE	
	IL-17 (pg / ml)	IL-23 (pg / ml)
Patients: Before treatment	225.55 ±7.70	76.42 ±4.03
Patients: After treatment	183.41 ±2.33	67.15 ±5.40
P - value	0.0001	0.166
	(p < 0.01).	

IL-17: Interleukin-17, IL-23: Interleukin-23.

Lipid profile: In the present study, before the administration of Apremilast, the mean levels of total cholesterol were recorded as 152.86 mg/dl. Following a period of six months of treatment, the observed value rose to 167.17 mg/dl. However, this observed rise did not reach statistical significance (p>0.05). The average triglyceride level prior to the administration of Apremilast was recorded as 190.80 mg/dl. Following the intervention, the observed value reduced to 150.39 mg/dl; however, this reduction did not yield statistically significant results (p> 0.05). Moreover, HDL cholesterol before the administration of Apremilast was recorded as 22.17 mg/dl. After a period of six months, there was a notable increase to 32.82 mg \ dl, with statistical significance shown by a *P*-value of less than 0.01. Ultimately, the mean of LDL-C levels prior to the administration of Apremilast was recorded as 92.53 mg/dl, which subsequently rose to 104.36 mg/dl following the completion of the treatment. However, the observed increase did not reach statistical significance (P> 0.05). as shown in Table 3.

Table 3: Comparison between lipid profile data before and after receiving Apremilast

Group	Mean ± SE of Lipid profile (mg/dl)			
	Cholesterol	Triglyceride	HDL-C	LDL-C
Patients: Before treatment	152.86 ±5.62	190.80 ±19.12	22.17 ±1.04	92.53 ±4.94
Patients: After treatment	167.17 ±4.71	150.39 ±16.02	32.82 ±4.78	104.36 ±7.14
p-value	0.649	0.126	0.010	0.166
	* (p-< 0.05), ** (p-< 0.01).			

HDL-C: High Density Lipoprotein cholesterol, LDL-C: Low Density Lipoprotein cholesterol.

Body mass index Participants in the current study were considered obese according to international standards (22). However, their mean BMI after 6 months of treatment with Apremilast did not significantly differ from that before treatment (P>0.05; Table 4).

Table 4: Comparison of participants' BMI before and after treatment with Apremilast for 6 months

Group	BMI (Mean ± SE) (kg/m^2)
Patients: Before treatment	32.97 ±1.07
Patients: After treatment	30.48 ±1.14
p-value	0.349

Discussion:

Psoriasis is a chronic disease of the immune system that is characterized by increased inflammation cytokine production, including IL-17 and IL-23. To the best of our knowledge, this is the first study to assess the effects of Apremilast on IL-17 and IL-23 in psoriatic patients in Iraq.

The findings showed that after six months of Apremilast treatment, there was a decrease in the serum levels of IL-17 and IL-23. These findings point to its efficacy in reducing inflammation. According to *Strober et al. (19*), this study was consistent with their previous findings, which also showed a decrease in mean levels of IL-17. Apremilast significantly decreased plasma IL-17A, IL-17F, and IL-22 levels among patients with moderate plaque psoriasis (10). Additionally, this further corroborates the Apremilast involvement in modulating IL-17-related pathways. It also plays a role in modulating the release of inflammatory cytokines by immune and non-immune cells (23). In T cells, Apremilast inhibited various T-cell-derived cytokines, including IL-2, IL-5, IL-13, and IL-17 (24).

In addition, this study was parallel with *Parab et al. (2022),* which found that Apremilast decreased the production of IL-12 and IL-23; however, in patients with mild plaque psoriasis who were innocent to systemic therapy, it did not significantly change IL-23 levels (25). Also, another study by *Ilowite et al*. (*2016*), showed that Apremilast is an oral targeted PDE-4 inhibitor that affects many inflammatory mediators involved in psoriasis and psoriatic arthritis. These mediators include inducible TNFα, IL-23, and IL-10 expression, which are all decreased, and IL-10, which is increased (26).

This study elucidated novel effects of Apremilast on blood lipids, demonstrating its efficacy in beneficially modulating lipids. Specifically, Apremilast elevated HDL-C while decreasing circulating TG in psoriatic patients. According to a recent study, found an increase in HDL-C serum, as well as a progressive and constant decrease in TC, LDL-C, and TG over the first month of receiving Apremilast (27, 28). Moreover, according to *Blum et al. (2019),* Apremilast may provide additional advantage due to its ability to increase lipolysis, decrease hepatic triglyceride synthesis, and improve cholesterol flux, all of which can lead to improvements in the lipid profile (29).

Moreover, according to Ferguson LD et al. (2022), There were reductions in weight and BMI with Apremilast treatment across all time points compared with baseline, with a mean weight loss of 2.2 kg and a mean BMI decrease of 0.8 kg/m2 by the end of the study (30).

Conclusion

Apremilast as monotherapy is effective in reducing the inflammatory cytokines and is effective in decreasing IL17- and IL23- levels, which have pro-inflammatory effects, this leads to improvement in psoriatic lesions. Additionally, Apremilast exhibits

favorable modulation of the lipid profile, decreasing triglycerides and increasing HDL cholesterol. Though total and LDL cholesterol increased

Limitation:
The study was conducted at a single centre, the Dermatology Centre, Medical City in Baghdad, and the study had a limited sample size of 30 patients initially, with 6 participants withdrawing before completion.

Authors' declaration:
We confirm that all the Figures and Tables in the manuscript belong to the current study. Besides, the Figures and images, which do not belong to the current study, have been given permission for re-publication attached to the manuscript. Authors sign on ethical consideration's Approval-Ethical Clearance: The project was approved by the local ethical committee in (the College of Medicine, University of Baghdad) according to the code number (145 on 1/11/2023). In addition, approval of the Baghdad Hospital was obtained. While verbal agreement was obtained from patients to participate.
Conflict of Insert: None.
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Authors' contributions:
Study conception & design: (Haitham M. Saad, Adil A. Noaimi, Halla Gh. Mahmood). Literature search: (Haitham M. Saad, Adil A. Noaimi, Halla Gh. Mahmood). Data acquisition: (Haitham M. Saad, Adil A. Noaimi, Halla Gh. Mahmood). Data analysis & interpretation: (Haitham M. Saad, Adil A. Noaimi, Halla Gh. Mahmood). Manuscript preparation: (Haitham M. Saad, Adil A. Noaimi, Halla Gh. Mahmood). Manuscript editing & review: (Haitham M. Saad, Adil A. Noaimi, Halla Gh. Mahmood).

Adil A. Noaim is an **Editorial board member** but did not participate in the peer review process other than as an author.

Reference:
1. *Rajguru JP, Maya D, Kumar D, Suri P, Bhardwaj S, Patel ND. Update on psoriasis: A review. Journal of Family Medicine and Primary Care. 2020;9(1):20.* https:doi.org/10.4103/jfmpc.jfmpc_689_19
2. *Thamer SM, Yahya MQ. The effect of lenalidomide ointment on TNF-α tissue levels in mice with imiquimod-induced psoriasis. JFacMedBaghdad. 2022;64(4):252-60.* https://doi.org/10.32007/jfacmedbagdad.6441959
3. *Ahmad A. A critical review of Daus-Sadaf (Psoriasis): Unani & modern perspectives. Int J Creat Res Thoug. 2020;8(7):4570-82.* http://dx.doi.org/10.13140/RG.2.2.25897.83040
4. *Al-Bidri KZ, Salman HA, Al-Hassan Y, Hasan MS. Fibromyalgia Syndrome in a sample of Iraqi patients with psoriasis. Journal of the Faculty of Medicine Baghdad. 2014;56(1):49-52.* https://doi.org/10.32007/jfacmedbagdad.561425
5. *Sharquie KE, Noaimi AA, Alobaidi MH. A New Regimen in the Treatment of Psoriasis Using Oral Methotrexate. Journal of Cosmetics, Dermatological Sciences and Applications. 2019;9(02):165.* https://doi.org/10.4236/jcdsa.2019.92014
6. *Al-Ammari AM, Al-Attraqhchi AA, Al-Jibouri M. Species of Malassezia associated with psoriatic patients. Journal of the Faculty of Medicine Baghdad. 2012;54(4):356-60.* https://doi.org/10.32007/jfacmedbagdad.544704
7. *AL-Sariay AH, Al-Ahmer SD, Muslim AM, Abood ZH, Haleem H. Genetic study of psoriasis disease: a review. Plant Archives. 2021;21(1):2046-8.* https://doi.org/10.51470/PLANTARCHIVES.2021.v21.S1.335
8. *Afra T, Razmi TM, Dogra S. Apremilast in psoriasis and beyond: big hopes on a small molecule. Indian dermatology online journal. 2019;10(1):1.* https://doi.org/10.4103/idoj.idoj_437_18
9. *Milakovic M, Gooderham MJ. Phosphodiesterase-4 inhibition in psoriasis. Psoriasis: Targets and Therapy. 2021:21-9.* https://doi.org/10.2147%2FPTT.S303634
10. *Strober B, Alikhan A, Lockshin B, Shi R, Cirulli J, Schafer P. Apremilast mechanism of efficacy in systemic-naive patients with moderate plaque psoriasis: pharmacodynamic results from the UNVEIL study. Journal of Dermatological Science. 2019;96(3):126-33.* HTTPS://DOI.ORG/10.1016/j.jdermsci.2019.09.003
11. *wang A, Bai Y. Dendritic cells: The driver of psoriasis. The Journal of dermatology. 2020;47(2):104-*
13. *HTTPS://DOI.ORG/ 10.1111/1346-8138.15184*
12. *Mohammed RM, Hamid ZA. Assessment of Interleukin-17 levels in patients with hepatitis C Viral Infection. Journal of the Faculty of Medicine Baghdad. 2024;66(1):39-44.* https://doi.org/10.32007/jfacmedbagdad.2157
13. *Mosca M, Hong J, Hadeler E, Hakimi M, Liao W, Bhutani T. The role of IL-17 cytokines in psoriasis. ImmunoTargets and therapy. 2021:409-18.* https://doi.org/10.2147/ITT.S240891
14. *Branisteanu DE, Cojocaru C, Diaconu R, Porumb EA, Alexa AI, Nicolescu AC, et al. Update on the etiopathogenesis of psoriasis. Experimental and Therapeutic Medicine. 2022;23(3):1-13.* https://doi.org/10.3892/etm.2022.11124
15. *Bugaut H, Aractingi S. Major role of the IL17/23 axis in psoriasis supports the development of new targeted therapies. Frontiers in immunology. 2021; 12:621956.* https://doi.org/10.3389/fimmu.2021.621956
16. *Lauffer F, Eyerich K, Boehncke WH, Asadullah K, Beissert S, Ghoreschi K, et al. Cytokines*

of the IL-17 family in psoriasis. JDDG: Journal der Deutschen Dermatologischen Gesellschaft. 2020;18(7):675-
81. https://doi.org/10.1111/ddg.14124
17. Lé AM, Puig L, Torres T. Deucravacitinib for the treatment of psoriatic disease. American Journal of Clinical Dermatology. 2022;23(6):813-22. https://doi.org/10.1007/s40257-022-00720-0
18. Mikhaylov D, Hashim PW, Nektalova T, Goldenberg G. Systemic psoriasis therapies and comorbid disease in patients with psoriasis: a review of potential risks and benefits. The Journal of clinical and aesthetic dermatology. 2019;12(6):46. PMCID: PMC6624011
19. Gualtierotti R, De Lucia O. Efficacy and Metabolic Effect on Serum Lipids of Apremilast in Psoriatic Arthritis: A Case Report. 2019;8(3). https://doi.org/10.3390/jcm8030398
20. Edwards CJ, Blanco FJ, Crowley J, Birbara CA, Jaworski J, Aelion J, et al. Apremilast, an oral phosphodiesterase 4 inhibitor, in patients with psoriatic arthritis and current skin involvement: a phase III, randomised, controlled trial (PALACE 3). Ann Rheum Dis. 2016;75(6):1065-73. https://doi.org/10.1136/annrheumdis-2015-207963
21. SAS S. Statistical Analysis System, User's Guide. Statistical. Version 9.6. SAS Inst Inc Cary NC USA. 2018.
22. Woolcott OO, Seuring T. Prevalence trends in obesity defined by the relative fat mass (RFM) index among adults in the United States: 1999–2018. MetabClin Exp 2022; 128:155027. https://doi.org/10.1016/J.METABOL.2021.155027
23. Schafer P, Parton A, Capone L, Cedzik D, Brady H, Evans J, et al. Apremilast is a selective PDE4 inhibitor with regulatory effects on innate immunity. Cellular signalling. 2014;26(9):2016-29. https://doi.org/10.1016/j.cellsig.2014.05.014
24. Schafer P. Apremilast mechanism of action and application to psoriasis and psoriatic arthritis. Biochemical pharmacology. 2012;83(12):1583-90. https://doi.org/10.1016/j.bcp.2012.01.001
25. Parab S, Doshi G. An update on emerging immunological targets and their inhibitors in the treatment of psoriasis. International Immunopharmacology. 2022; 113:109341. https://doi.org/10.1016/j.intimp.2022.109341
26. Ilowite NT, Laxer RM. Pharmacology: biologics. Textbook of pediatric rheumatology: Elsevier; 2016. p. 161-75. e6. https://doi.org/ 10.5114/reum.2020.102001
27. Wu C, Rajagopalan S. Phosphodiesterase-4 inhibition as a therapeutic strategy for metabolic disorders. obesity reviews. 2016;17(5):429-41. https://doi.org/10.1111/obr.12385
28. Gualtierotti R, De Lucia O. Efficacy and metabolic effect on serum lipids of Apremilast in psoriatic arthritis: a case report. Journal of Clinical Medicine. 2019;8(3):398. https://doi.org/10.3390/jcm8030398
29. Blum S, Altman D. Treatment of generalized granuloma annulare with Apremilast: a report of 2 cases. JAAD case reports. 2019;5(11):976-8. https://doi.org/10.1016/j.jdcr.2019.09.015
30. Ferguson LD, Cathcart S, Rimmer D, Semple G, Brooksbank K, Paterson C, et al. Effect of the phosphodiesterase 4 inhibitor Apremilast on cardiometabolic outcomes in psoriatic disease-results of the Immune Metabolic Associations in Psoriatic Arthritis study. 2022;61(3):1026-34. https://doi.org/10.1093/rheumatology/keab474

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تقييم تأثير أبريميلاست على مستويات الإنترلوكين-17 والإنترلوكين-23 والدهون لدى مرضى الصدفية المصابين بالسمنة

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الخلاصة

خلفية البحث: الصدفية هو مرض التهابي مناعي مجهول السبب قد يرتبط بخلل في تكاثر وتمايز الخلايا الكيراتينية المرتبطة بتسلل الخلايا الالتهابية. هوا مرض عالمي الحدوث؛ ويتراوح معدل انتشاره بين المجموعات السكانية المختلفة من 0.1% إلى 11.8%، وفقا للتقارير المنشورة. أدى تلقي ابريميلاست إلى انخفاض كبير في IL-23 وIL-17A، كما أدى إلى انخفاض التعبير عن السيتوكينات الالتهابية الأخرى وتحسين من مرض الصدفية
الهداف: الغرض من هذه الدراسة هو تقييم تأثير ابريميلاست على مستويات IL-23 , IL-17 والدهون لدى الأشخاص المصابين بالصدفية و يعانون من السمنة المفرطة.
الطرق: تم تضمين ثلاثين مريضا يعانون من السمنة والصدفية في هذه الدراسة التدخلية المستقبلية لقياس المستويات المصلية لملف الدهون، والإنترلوكين-17 والإنترلوكين-23، قبل وبعد تلقي العلاج بأبريميلاست. تم استخدام اختبار "اختبار T" للمقارنة بين المتوسط
النتائج: كان متوسط عمر المشاركين 38 عاما. وكانت الفئة العمرية الأكثر شيوعا هي (30-40 عاما). وكانت مستويات الإنترلوكين-17 قبل إعطاء أبريميلاست 225.55 ±7.70 بيكوغرام/مل. بعد ستة أشهر من العلاج بأبريميلاست، لوحظ انخفاض ذو دلالة إحصائية، حيث انخفضت القيمة إلى 183.41 ±2.33 بيكوغرام/مل. تم قياس مستويات الإنترلوكين-23 قبل إعطاء أبريميلاست بـ 76.42 ±4.03 بيكوغرام/مل. بعد ستة أشهر من العلاج بأبريميلاست، أظهرت هذه المستويات انخفاضا غير ملحوظ إحصائيا إلى 67.15 ±5.40 بيكوغرام/مل (P > 0.05). كما لوحظت تغيرات طفيفة في ملف الدهون (P > 0.05).
الاستنتاجات: أدى استخدام ابريميلاست إلى انخفاض ملحوظ إحصائيا في مستويات السيتوكينات الالتهابية IL-17 و IL-23, أدى استخدام عقار ابريميلاست لدى الأشخاص المصابين بالصدفية الذين يعانون من السمنة المفرطة إلى انخفاض مستويات الدهون الثلاثية(TG) وارتفاع مستويات البروتين الدهني عالي الكثافة (HDL) . بالإضافة إلى ذلك، لوحظ ارتفاع في مستويات الكوليسترول الكلي(TC) والبروتين الدهني منخفض الكثافة (LDL) .
الكلمات المفتاحية: الصدفية, السمنه, أبريميلاست، IL-17, IL-23.