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Does newest immunomodulative treatment in patients with psoriasis impact growth and transformation of melanocytic nevi in patients with psoriasis? Preliminary results from a prospective cohort study

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Abstract: Introduction: Patients with psoriasis treated with biologic therapy are considered to have increased risk of developing skin neoplasms. The impact of novel therapies on the development and biology of melanocytic lesions is not completely known. The aim of the study was assess if the treatment with biologics exerts effects on the melanocytic nevi.
Material and Methods: Dermoscopy and videodermoscopic imaging of 276 melanocytic lesions were performed at the initial visit and after one year in 13 patients with psoriasis treated with IL-12/23 inhibitors, IL-23 inhibitors or IL-17 inhibitors.
Results: The nevi size significantly increased at the one-year follow-up in the whole study group and in patients treated either with IL-23 inhibitors or IL-12/23 inhibitor. 2% of melanocytic nevi showed an increase in any of the scores (ABCD score, 7-Point Checklist score, CASH score) or at least 1-mm change in the diameter at the one-year follow-up; however, these changes were not statistically significant between the different therapy groups.
Conclusions: Although treatment with novel groups of biologics leads to an increase in nevi size, it does not contribute to significant structural or color changes in melanocytic nevi or to the development of dysplastic nevi or melanoma at the one-year follow-up.

Keywords: psoriasis, melanoma, melanocytic nevus, biologic therapy, dermoscopy.

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Introduction

Psoriasis is a chronic, immune-mediated skin disease affecting approximately 2% of the European and North American populations. [1] A significant proportion of individuals with psoriasis require systemic treatment, such as phototherapy, non-biologic systemic agents (methotrexate, acitretin, ciclosporin A) or biologic therapies. Utilization of these methods of treatment has been associated with an increased risk of skin malignancy in patients with psoriasis, particularly non-melanoma skin cancer (NMSC). [2, 3, 4] Additionally, a higher risk of certain malignancies, including melanoma and NMSC, cannot be ruled out in patients treated with tumor necrosis factor α (TNF-α) inhibitors. [5–7] These risks are much less known in patients treated with novel groups of biologics (interleukin (IL)-23 inhibitors, IL-17 inhibitors). Nevertheless, according to recently published meta-analysis, the risk of melanoma and NMSC seems to be slightly higher in patients treated with targeted therapies when compared to the general population. [8]

Melanoma is an aggressive malignancy that derives from melanocytes and approximately 30% of melanomas arise from a pre-existing nevus. [9] Patients with psoriasis have been reported to have a lower number of melanocytic nevi compared to healthy controls. [10] This phenomenon has been linked to the inhibitory effect of pro-inflammatory cytokines (IL-1α, IL-6, TNF-α and transforming growth factor-β1) on tyrosinase activity, which hinders melanogenesis and melanocytic growth. [10]

Up until now, only few studies have assessed if there are any changes in melanocytic nevi in patients treated with biologics and immunosuppressants and showed conflicting results. In 2015, Koseaglu *et al.* conducted a prospective case-control trial on patients treated with immunosuppressive therapy (including TNF-α inhibitors, cyclosporine, methotrexate and azathioprine) and found that such therapies were associated with increased number of nevi and dermoscopic changes compared to the group of healthy controls. [11] Nevertheless, another study including only patients treated with TNF-α inhibitors and ustekinumab, an IL-12/23 inhibitor, found no significant changes in nevi or development of new dysplastic naevi when compared to controls. [12] To the best of our knowledge, there are no corresponding studies on patients with psoriasis treated with newer biologics, such as IL-17 and IL-23 inhibitors.

Our article presents preliminary results of the study investigating whether treatment with novel groups of biologics (IL-12/23 inhibitors, IL-23 inhibitors, IL-17 inhibitors) leads to changes in the size or dermoscopic scores of existing melanocytic nevi in patients with psoriasis.

Materials and Methods

This prospective cohort study funded by the research grant (WSUB/2023/07/00009) at Andrzej Frycz Modrzewski Krakow University is being conducted at a single academic hospital since October 2023. The recruitment of patients took place between December 2023 and April 2024. The preliminary analysis was limited to patients from the study group who had completed follow-up before the end of January 2025. The inclusion criteria were: 1) age above 18 years, 2) diagnosis of psoriasis (with or without psoriatic arthritis), 3) treatment with IL-12/23 or IL-23 inhibitor (ustekinumab, guselkumab, risankizumab, tildrakizumab) or IL-17 inhibitor (bimekizumab, ixekizumab, secukinumab) for at least 3 months. Patients were excluded, if they concomitantly use other biologic drugs, immunosuppressants (for example methotrexate, cyclosporin A) or phototherapy. Previous use of TNF-α inhibitors or immunosuppressive agents was permitted. In the

control group we included healthy participants without a history of psoriasis or other conditions that necessitated prior or ongoing immunosuppressive treatment, including biologics. Data regarding demographic characteristics, onset of the therapy and previous treatment were collected from all participants in the study and control groups. Additionally, all patients completed questionnaires concerning sun exposure during specific life periods, history of sunburns, clothing and history of sun protection, personal and family history of skin cancer, frequency of previous dermoscopy check-ups. Patients with missing data or those who were lost to follow-up were excluded from the analysis. The ethical committee of Andrzej Frycz Modrzewski University approved the study. Informed consent was obtained from all individual participants included in the study in accordance with the Declaration of Helsinki.

At the initial visit and after one year total body pictures and photographs of each melanocytic nevi were taken by FotoFinder Medicam 1000 (FotoFinder Systems GmbH, Bad Birnbach, Germany). Dermoscopic evaluation at each visit was conducted using Dermlite DL4 (3GEN LLC, San Juan Capistrano, CA) handheld dermoscope. Dermoscopy and videodermoscopic imaging of the melanocytic lesions were performed by a dermatologist trained in dermoscopy. Any doubtful cases were discussed with a senior physician. In a given patient the assessement was performed by the same investigator at all time points. Dermoscopic parameters of each melanocytic nevi assessed during the initial visit and after 12 months were: 1) the largest dimension expressed in millimeters, 2) score according to the asymmetry, border sharpness, colors, different structural components (ABCD) rule, [13] 3) score according to 7-Point Checklist (atypical pigment network, grey-blue areas, atypical vascular pattern, radial streaming, irregular diffuse pigmentation, irregular dots and globules, regression pattern), [14] 4) score according to the color, architecture, symmetry, homogeneity (CASH) algorithm. [15] Changes in scores were defined as any increase in the total score of each rule/algorithm after one year of follow-up. Change in the diameter was defined as at least 1-mm increase or decrease in the largest dimension of the nevi after one-year of follow-up. Suspicious lesion were histopathologically examined or dermoscopically controlled at shorter intervals depending on the clinical evaluation. Melanocytic lesions surgically excised after first assessment were not included in the analysis.

Statistical analysis

STATISTICA 14.0 software (StatSoft Inc., Tulsa, OK, USA) was utilized for the statistical analysis. Categorical variables are presented as numbers and percentages, whereas continuous variables are expressed as mean ± standard deviation (SD) or median and interquartile range (IQR), as appropriate. The Wilcoxon signed-rank test compared continuous variables between related groups. ANOVA was used for normally distributed data with homogeneous variances, while the Kruskal-Wallis test was used for non-normally distributed data. Associations between categorical variables were tested using the Pearson chi-square test. Statistical significance was defined as p-values below 0.05.

Results

In this preliminary analysis a total of 13 patients (8 male and 5 female) with psoriasis were included. Three patients have been treated with ustekinumab (IL-12/23 inhibitor), 5 patients with risankizumab (IL-23 inhibitor), 1 patient with guselkumab (IL-23 inhibitor), 1 patient with tildrakizumab

(IL-23 inhibitor), 1 patient with bimekizumab (IL-17 inhibitor), 1 patient with ixekizumab (IL-17 inhibitor) and 1 patient with secukinumab (IL-17 inhibitor). A cumulative number of analyzed nevi during the initial assessment was 378. Two nevi in a male patient treated with risankizumab were surgically excised after first visit due to melanoma-specific dermoscopic findings (histopathologic reports revealed compound nevi); therefore, the number of nevi included in the analysis was 376. The mean time from the onset of the therapy to the initial visit was 28.5 ± 36.8 months. No significant differences were found between the different therapy groups in terms of baseline characteristics, including age, sex, duration of therapy and follow-up, number of nevi per person. Characteristics of study participants and nevi count are set out in Table 1 and Table 2.

Size of the melanocytic lesions was measured at the initial visit and after one year. Mean and median diameters of all nevi and in subgroups are presented in Table 3. We found a significant increase in the size of nevi in one-year follow-up in the whole study group ($p < 0.001$) and in patients treated either with IL-23 inhibitors ($p = 0.003$) or IL-12/23 inhibitor ($p = 0.02$). The increase was not statistically significant in patients treated with IL-17 inhibitors ($p = 0.76$).

Table 1. Baseline characteristics of the study participants.

Number of patient	Sex	Age (years)	Drug group	Drug name	Therapy duration (months)	Prior biologic therapy	Prior immunosuppressive therapy	Previous phototherapy	Number of nevi analyzed (n)
1	M	38	IL-23 inhibitor	Risankizumab	19	No	No	Yes	17
2	M	36	IL-23 inhibitor	Risankizumab	6	No	Yes	Yes	35
3	M	58	IL-17 inhibitor	Bimekizumab	6	No	Yes	Yes	10
4	F	52	IL-23 inhibitor	Risankizumab	6	Yes	Yes	Yes	84
5	F	50	IL-23 inhibitor	Risankizumab	13	Yes	Yes	No	35
6	F	33	IL-12/23 inhibitor	Ustekinumab	93	Yes	Yes	Yes	14
7	M	38	IL-23 inhibitor	Tildrakizumab	10	Yes	Yes	No	43
8	F	72	IL-17 inhibitor	Ixekizumab	25	Yes	Yes	Yes	24
9	M	46	IL-12/23 inhibitor	Ustekinumab	123	Yes	Yes	No	24
10	M	38	IL-12/23 inhibitor	Ustekinumab	12	Yes	Yes	Yes	46
11	M	63	IL-23 inhibitor	Guselkumab	21	Yes	Yes	No	6
12	M	38	IL-17 inhibitor	Secukinumab	3	Yes	Yes	No	22
13	F	30	IL-23 inhibitor	Risankizumab	32	No	Yes	Yes	16

M — male, F — female, IL — interleukin

Results reporting changes in the analyzed scores and diameter of nevi are presented in Table 4. We found no statistically significant differences in the number of nevi that showed significant changes in any of the scores (p-values 0.92, 0.86, 0.94 for ABCD score, 7-Point Checklist score, CASH score, respectively) or diameter (p-value 0.94) between the different therapy groups. Two lesions

were excised due to enlargement or high dermoscopy scores. Both were described in histopathology reports as compound nevi. Lesions showing minimal or insignificant changes were not surgically removed. Figure 1 presents examples of the changes observed in the nevi in the study group.

Table 2. Characteristics of study participants and number of nevi.

Parameter	All patients (n = 13)	IL-17 inhibitors (n = 3)	IL-23 inhibitors (n = 7)	IL-12/23 inhibitors (n = 3)	p-value
Age (years), mean ± SD	45.5 ± 12.7	56 ± 17.1	43.9 ± 11.5	39 ± 6.6	0.24
Sex, n (% male)	8 (61.5)	2 (66.7)	4 (57.1)	2 (66.7)	0.94
Duration of therapy (months), mean ± SD	28.5 ± 36.8	11.3 ± 11.9	15.3 ± 9.4	76 ± 57.4	0.18
Follow-up time (days), median [IQR]	343 [336–395]	365 [336–410]	336 [333–343]	395 [384–416]	0.09
Total number of nevi, n	376	56	236	84	
Number of nevi per patient, mean ± SD	28.9 ± 20.6	18.7 ± 7.6	33.7 ± 25.8	28 ± 16.4	0.65

IL — interleukin, SD — standard deviation, IQR — interquartile range

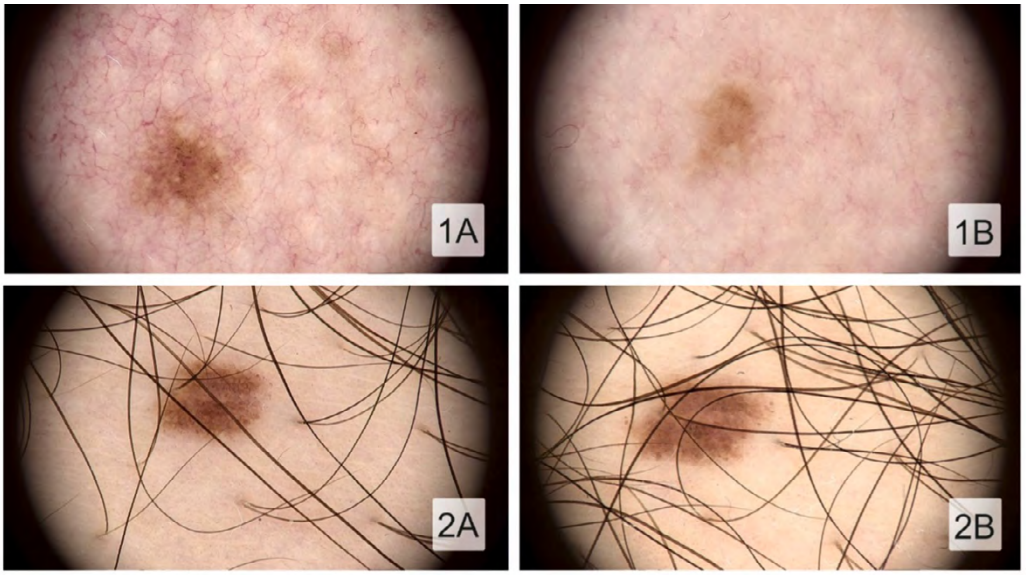

Fig. 1. Examples of the changes observed in the nevi in the study group.
1A and 1B: 34-year old male treated with risankizumab — regression in the size of the nevus, loss of brown globules, lighter appearance.
2A and 2B: 38-year old male treated with tildrakizumab — enlargement of the nevi with the presence of peripheral brown globules, loss of parts of the pigment network replaced by a light brown pigmentation. Histopathology report revealed compound nevus.

Table 3. Changes in nevi size in treatment groups.

	Diameter at baseline		Diameter in 1-year follow-up		p-value
	median [IQR]	mean ± SD	median [IQR]	mean ± SD	
All nevi (n = 376)	2.6 [2.0–3.4]	2.9 ± 1.4	2.6 [2.1–3.6]	3.0 ± 1.4	<0.001
IL-17 inhibitors (n = 56)	2.3 [1.9–2.8]	2.4 ± 0.9	2.4 [1.8–2.8]	2.5 ± 1.0	0.76
IL-23 inhibitors (n = 236)	2.8 [2.2–3.6]	3.1 ± 1.3	2.8 [2.2–4.0]	3.2 ± 1.4	**0.003**
IL-12/23 inhibitors (n = 84)	2.3 [1.8–3.2]	2.7 ± 1.8	2.4 [1.7–3.1]	2.8 ± 1.8	**0.02**

IQR — interquartile range, SD — standard deviation, IL — interleukin

Table 4. Changes in analyzed parameters at one-year follow up.

	ABCD score*		7-Point Checklist*		CASH score*		Nevi diameter**	
	Number of nevi changed, n (%)	p-value	Number of nevi changed n (%)	p-value	Number of nevi changed n (%)	p-value	Number of nevi changed n (%)	p-value
All nevi (n = 376)	7 (1.9)		7 (1.9)		6 (1.6)		6 (1.6)	
IL-17 inhibitors (n = 56)	1 (1.8)	0.92	1 (1.8)	0.86	1 (1.8)	0.78	1 (1.8)	0.94
IL-23 inhibitors (n = 236)	4 (1.7)		5 (2.1)		3 (1.3)		4 (1.7)	
IL-12/23 inhibitors (n = 84)	2 (2.4)		1 (1.2)		2 (2.4)		1 (1.2)	

* Change is defined as any increase in the total score after one-year of follow-up.
** Change is defined as at least 1-mm increase or decrease in the diameter of the nevi after one-year of follow-up.
IL — interleukin, ABCD — asymmetry, border sharpness, colors, different structural components, CASH — color, architecture, symmetry, homogeneity

Discussion

Current evidence suggests that patients with psoriasis are at increased risk of developing melanoma and NMSC due to the usage of specific therapies and environmental factors. The recent introduction of biologics has raised concerns about safety of their use with respect to tumorigenesis. In a recently published meta-analysis, the incidence rates of melanoma and NMSC in patients with psoriasis and psoriatic arthritis treated with IL-12/23 inhibitors, IL-23 inhibitors, IL-17 inhibitors

and Janus kinase inhibitors were 0.08 and 0.45 events per 100 patient-years, respectively, and appeared to be slightly higher than those observed in the general population. [8] Therefore, regular dermoscopic check-ups in patients treated with biologics appear to be reasonable for the early detection of suspicious changes in melanocytic nevi and the development of new lesions.

There is strong evidence that healthy immune system restrains melanocyte proliferation, while immunosuppression promotes the development of eruptive nevi and skin malignancies. [16, 17] Eruptive nevi have been reported in transplant recipients receiving cyclosporin A, prednisolone and azathioprine and been characterized by the presence of symmetrical rim of peripheral brown globules in dermoscopy. [18] This phenomenon is most likely related to the immunosuppressive action of drugs. There are few theories to explain the mechanisms of melanocyte proliferation. Immunosuppression can promote melanocyte-stimulating hormone and melanoma growth-stimulatory activity, which act as growth factors for melanocytes. [16] Another proposed mechanism involves alterations in IL-12 and IL-10 expression leading to inhibition of mitogen-activated protein kinase pathway involved in melanocyte proliferation. [16]

Eruptive nevi have also been described in patients with Crohn disease treated with biologics (infliximab, etanercept and alefacept). [19] Data on the behavior of melanocytic nevi in patients with psoriasis undergoing biological treatment are limited and focus mainly on TNF-α inhibitors. [11, 12, 20] According to Choi *et al.*, treatment with TNF-α inhibitors and ustekinumab was not associated with significant changes in melanocytic nevi in patients with psoriasis when compared to controls. [12] The results of *in vitro* studies on the effects of IL-12, IL-23 and IL-17 on tumorigenesis and the development of nevi are inconsistent. [21] Bernice *et al.* found increase immune activation in Th1/Th2/Th17 axes during the melanomagenesis from common melanocytic nevi to dysplastic nevi to malignant melanoma. [22] On the other hand, IL-23 was found to maintain melanocyte homeostasis and regulate melanomagenesis by activating DNA repair in melanocytes, whereas IL-12 promoted nevi development. [23]

To our knowledge, this is the first study to present changes in melanocytic nevi in patients treated with newest groups of biologics utilized in the treatment of psoriasis (IL-17 inhibitors, IL-23 inhibitors, IL-12/23 inhibitors). We found statistically significant increase in the size of nevi in the whole study group and in patients treated either with IL-23 inhibitors or IL-12/23 inhibitor. This finding is consistent with the results of the study conducted by Koseoglu *et al.*, which indicated that immunosuppressive therapy led to an increase in nevi size. [11] However, according to recent studies, most nevi in the general adult population tend to increase in size rather than decrease. [24] Therefore, an analysis incorporating healthy population is essential to validate this finding. Two percent of melanocytic nevi included in our study demonstrated an increase in any of the scores (ABCD score, 7-Point Checklist score, CASH score) or at least 1-mm change in the diameter at the one-year follow-up. These changes were not statistically significant between the different therapy groups.

The study has several limitations. Firstly, a longer follow-up period would be necessary to reliably assess changes in nevi and the incidence of malignancy. Secondly, since the study presents only preliminary findings from a larger ongoing research, it is limited to a small group of patients and does not include a healthy control group for comparison between the biologics receiving and biologics non-receiving people. On the other hand, this is interim analysis on first patient group included in the study. The final results will include 28 patients in both the study and control groups. What is more, the study does not account for participants' sun protection behaviors or their history of sun exposure, both of which could influence the observed outcomes.

To conclude, preliminary results of our study suggest that treatment with inhibitors of IL-17, IL-12/23 and IL-23 does not cause significant adverse changes in melanocytic nevi in patients with psoriasis. However, we have observed significant increase in nevi size in majority of patients over time. Further larger studies with longer follow-up period may be needed to validate this finding.

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Conflict of interest

M.K. has received honoraria and/or consultation fees from AbbVie, Amgen, Pfizer, Janssen and Eli Lilly. P.B. has received honoraria and/or consultation fees from AbbVie, Amgen, Concert Pharmaceuticals, Eli Lilly, Innovaderm Research, LEO Pharma, Pfizer, Sanofi Genzyme, Samsung, Janssen, and Novartis. W.M.W. has received honoraria and/or consultation fees from Roche, Novartis, Pfizer, Sanofi, BMS, Pierre-Fabre, PCI, J&J.

Authors' contribution

Conceptualization and Study Design: W.M.W. and M.K.; Data Collection: M.K., M.M., P.B.; Statistical Analysis: M.K., M.M., A.K., K.R. and W.M.W.; Data Interpretation: W.M.W., M.K., M.M., A.K., K.R. and P.B.; Manuscript Preparation: W.M.W., M.K., A.K., K.R. and P.B.; Literature Search: M.K., M.M., A.K., K.R., P.B. and W.M.W. All authors have read and agreed to the published version of the manuscript.

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