Original paper

Is there a correlation between serum and faecal calprotectin levels and the disease severity in patients with moderate-to-severe plaque psoriasis? A pilot study

Karina Polak[1,2], Tomasz Muszyński[3,4], Aleksandra Frątczak[1], Bartosz Miziołek[1], Beata Bergler-Czop[1]

[1]Chair and Department of Dermatology, Medical University of Silesia, Katowice, Poland
[2]Doctoral School of the Medical University of Silesia, Katowice, Poland
[3]Brothers Hospitallers of Saint John of God Hospital, Krakow, Poland
[4]Doctoral School of Medical and Health Sciences, Jagiellonian University, Krakow, Poland

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Abstract

Introduction: The role of calprotectin in psoriasis still remains unclear.
Aim: To elucidate the associations between the concentrations of serum and faecal calprotectin (CP) and the severity of psoriasis in 20 patients with moderate-to-severe plaque psoriasis and 20 healthy individuals.
Material and methods: The CP levels as well as the disease severity including Psoriasis Area and Severity Index (PASI), Body Surface Area (BSA) score and Dermatology Life Quality Index (DLQI) were assessed.
Results: The median serum CP level in the psoriasis group was notably higher at 112.24 µg/l (interquartile range, IQR: 81.39–206.82 µg/l) compared to 60.31 µg/l (IQR: 37.41–81.54 µg/l) in the control group (p = 0.001), while the median faecal CP levels were similar between the two groups: 15.00 µg/g (IQR: 6.20–36.13 µg/g) in psoriasis patients and 13.00 µg/g (IQR: 10.26–30.15 µg/g) in controls (p = 0.766). No significant correlations between serum and faecal CP level and the PASI, BSA and DLQI were found.
Conclusions: Despite the fact that patients with moderate-to-severe plaque psoriasis seem to present general higher blood calprotectin levels than healthy individuals, they are still within the population cut-off value. The elevated blood calprotectin levels are not conclusively linked to increased severity of psoriasis or to the deterioration in quality of life. Also, in individuals with psoriasis, gastrointestinal inflammation does not differ markedly from healthy controls.

Key words: serum calprotectin, faecal calprotectin, psoriasis.

Introduction

Calprotectin (CP) is a protein composed of S100A8 and S100A9 subunits [1]. CP is a calcium and zinc-binding protein, present in the neutrophil cytoplasm. It is also produced by the neutrophils, monocytes and macrophages or released from dead cells' debris [2]. Initially treated as an anti-microbial peptide, it was found to regulate the inflammatory processes, influencing the cell adhesion to the epithelium. It was also found to show elevated expression in the skin during wound healing or hyperproliferation of the dermis [3]. The role of calprotectin in psoriasis still remains unclear, although stratum corneum levels of calprotectin proteins S100A8/A9 were found to be higher than in healthy individuals or patients with atopic dermatitis and to correlate with disease activity [4, 5]. CP concentration can be marked inter alia in the blood serum or in the faeces; its level is recognized as a marker of the inflammatory state [1].

The role of serum CP in psoriatic patients was investigated, however mostly in the field of rheumatology, concerning psoriatic arthritis, which is a different disease entity with separate pathogenesis and methods of treatment. In psoriatic arthritis, the serum CP level was proved to be elevated and found as a strong, independent marker predicting the recurrence of polyarthritis in TNF-α-inhibitors-treated patients [6]. By far, very few studies concerning the link between the serum CP and the condition of the skin in psoriatic patients were conducted. The cut-off point for healthy individuals in most publications assumes a level of < 50 µg/g CP in faeces for adults [7]. As the mean serum CP level in a healthy individual may differ, a proposed cut-off level for diagnosing

Address for correspondence: Prof. Beata Bergler-Czop, Department of Dermatology, Medical University of Silesia, 20-24 Francuska St, 40-027 Katowice, Poland, phone: +48 32 25 91 200, e-mail: bettina2@tlen.pl
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inflammatory arthritis was > 900 µg/l [8]. Values among healthy individuals may differ, with ranges < 250–600 µg/l found normal [9, 10].

Aim

The study aims to elucidate the associations between the concentrations of serum and faecal calprotectin and the severity of psoriasis. We assumed the following hypotheses:

The calprotectin concentration in the peripheral blood serum and in the stool is increased in patients with plaque psoriasis.

There is a relationship between serum and faecal calprotectin levels and the intensity of skin lesions in plaque psoriatic patients.

Material and methods

The study was conducted as a part of the project "Bacterial dysbiosis in psoriasis vulgaris", according to the guidelines of the Declaration of Helsinki and approved by the local research Ethical Committee of the Medical University of Silesia with decision number PCN/0022/KB1/47/20 given on 30 June 2020. In this study, a total of 20 patients suffering from moderate-to-severe plaque psoriasis who were admitted to the Dermatology Clinic in the years 2022–2023 and 20 healthy controls who attended the outpatient clinic for dermoscopic evaluation of pigmental nevus but without diseases who met inclusion and exclusion criteria (Table 1) were recruited.

On the first day of patients' stay, their detailed characteristics including sex, age, disease duration, concomitant diseases, body mass index (BMI) and assessments using Psoriasis Area and Severity Index (PASI), body surface area (BSA), Dermatology Life Quality Index (DLQI) were taken.

The day after admission, the authors collected stool sample (10 g) to assess the concentration of calprotectin in the stool, and blood sample from peripheral vein to determine the concentration of calprotectin in the blood serum. Specimens were collected using sterile collection sets following the procedure for safely collecting samples; stool samples were immediately frozen at –20°C, while the blood samples were centrifuged in order to obtain serum (ca. 500 µl) and then serum was frozen at –20°C. Peripheral blood serum and stool sample calprotectin analysis was performed in Diagnostyka sp. z o.o. Central Laboratory of the Silesia Region. The faecal calprotectin concentration was measured using the LIAISON® XL instrument (CLIA Systems, DiaSorin) using chemiluminescence analysis method and expressed in µg/g (micrograms/grams), while the concentration of calprotectin in the peripheral blood serum was measured using CalproLab Calprotectin ELISA kit and the DRG Microplate Reader with concentration expressed in ng/ml (nanograms per millilitre) and later calculated into µg/l. Normal cut-off levels were consistent with literature data, for stool calprotectin the cut-off point was a level of < 50 µg/g and for serum calprotectin < 250 µg/l. In all cases, the quality and quantity of provided material was sufficient to perform analysis.

Statistical analysis

The obtained data underwent statistical analysis with the statistical significance threshold for the study set at $\alpha = 0.05$. To determine the distribution characteristics of numerical data, the Shapiro-Wilk test was used to assess normality. For numerical data that did not display normal distribution, descriptive statistics using the median (*Mdn*) accompanied by the first (*Q1*) and third (*Q3*) quar-

Table 1. Inclusion and exclusion criteria

Inclusion criteria	Exclusion criteria
1. Confirmed diagnosis of plaque psoriasis with no other psoriasis type ever recognized and PASI ≥ 10 and BSA ≥ 10 2. Age between 18 and 40 years old 3. No current or past (during the last 60 days) systemic therapies with antibiotics, probiotics, non-steroidal anti-inflammatory drugs	1. Lack of consent 2. Pregnancy or breastfeeding 3. Bacterial, viral, fungal infections of the skin, mucosae membranes, upper respiratory tract gastrointestinal tract during the last 60 days 4. Current tobacco smoking 5. Alcohol/drug addiction 6. Any systemic immunosuppressive therapy in the past 7. A diagnosis or suspicion of concomitant psoriatic arthritis 8. A diagnosis of inflammatory bowel disease 9. In females, menstruation, in order to avoid contamination of the faecal sample with blood 10. Any special dietary restrictions or dietary supplements 11. Any other concomitant disease or drug intake that in the opinion of the authors could affect the CP level 12. Any history of gastrointestinal tract disease that affects the frequency or may result in problems with defecation (e.g. irritable bowel syndrome (IBS), haemorrhoids etc.)

Table 2. Demographics and BMI characteristics by group

Characteristic	*N*	Total sample[a]	Group		*P*-value[c]
			Psoriasisa, $n^1 = 20$	Control[a], $n^2 = 20$	
Sex:	40				1.000
Female		14.00 (35.00%)	7.00 (35.00%)	7.00 (35.00%)	
Male		26.00 (65.00%)	13.00 (65.00%)	13.00 (65.00%)	
Age [years]	40	35.50 (32.75, 39.00)[b]	35.50 (32.00, 39.00)[b]	35.00 (33.75, 39.00)[b]	0.924[d]
BMI [kg/m^2]	40	28.77 (25.52, 31.17)[b]	29.24 (26.01, 30.72)[b]	28.22 (25.52, 31.46)[b]	0.808[d]

[a]n (%), [b]Mdn (Q1, Q3), [c]Pearson's χ^2 test, [d]Wilcoxon rank sum test, [e]Fisher's exact test. N – sample size, n – group size; Mdn – median; Q1 – the first quartile (25%); Q3 – the third quartile (75%); p – the p-value of statistical test.

tiles were used to illustrate central tendency and dispersion. For categorical variables, analysis involved reporting the counts (*n*) and percentages of each category. To compare differences between two independent groups with respect to numerical variables, the Wilcoxon rank-sum test was used. In cases where associations between two nominal (categorical) variables were examined, the χ^2 test was the primary method; when expected frequencies in any cell of the contingency table were less than five, making the χ^2 test less reliable, Fisher's exact test was used instead. For the analysis of correlation between two non-normal numeric variables, Spearman's rho correlation coefficients were used, with *p*-values calculated through the asymptotic *t* approximation to aid in assessing statistical significance. To ensure a thorough understanding of the examined relationships, both the 95% confidence intervals (*CI*) and *p*-values were estimated using the asymptotic *t* approximation.

Results

The study group consisted of 40 individuals, divided equally into two groups: 20 patients diagnosed with plaque psoriasis (psoriasis group) and 20 healthy individuals serving as controls (control group); each group contained 7 women and 13 men. The demographic characteristics of the analysed group are presented in Table 2. The average age of investigated individuals was 35.5 years. Medium BMI levels were 29.24 kg/m^2 in the psoriatic patients and 28.22 kg/m^2 in the healthy individuals. Table 3 provides a detailed overview of psoriasis-specific data for the study participants diagnosed with plaque psoriasis. This table encapsulates key clinical metrics that describe the severity and impact of psoriasis on these individuals. The parameters include the duration of psoriasis, stratified into different categories based on years; the PASI score along with its categorical breakdown; the BSA score which indicates the extent of skin involvement; and the DLQI score, reflecting the disease's impact on the patients' quality of life. In 45% of patients, the disease lasted for more than 10 years. The medium achieved PASI score was 11, BSA 14.5% and DLQI score 12 points.

The data concerning the investigated serum and faecal calprotectin levels are presented in Table 4. The median serum calprotectin level in the psoriasis group was notably higher at 112.24 µg/l (interquartile range, IQR: 81.39–206.82 µg/l) compared to 60.31 µg/l (IQR: 37.41–81.54 µg/l) in the control group. This difference was statistically significant ($p = 0.001$). When categorizing calprotectin levels into high (> 250 µg/l) and normal (≤ 250 µg/l), a higher percentage of psoriasis patients (25%) exhibited high serum calprotectin levels compared to just 5% of healthy controls, although this did not reach statistical significance ($p = 0.182$). The results showed that patients with moderate-to-severe plaque psoriasis exhibited higher blood calprotectin levels compared to healthy individuals. The elevated levels still remained within the population cut-off value.

Table 3. Characteristics of the psoriasis-specific data in plaque psoriasis patients

Characteristic	Psoriasis[a] *N* = 20
Psoriasis duration [years]	10.00 (4.75, 16.00)
Psoriasis duration categories:	
Below 5 years	6.00 (30.00%)[b]
5–10 years	5.00 (25.00%)[b]
Over 10 years	9.00 (45.00%)[b]
PASI score	11.00 (10.18, 15.00)
BSA score	14.50 (11.25, 34.25)
DLQI score	12.00 (6.00, 18.25)

[a]Mdn (Q1, Q3), [b]n (%), n – group size; Mdn – median; Q1 – the first quartile (25%); Q3 – the third quartile (75%).

Table 4. Comparative analysis of serum and faecal calprotectin levels in psoriasis patients and healthy controls

Characteristic	*N*	Total sample[a]	Group		*P*-value[c]
			Psoriasis[a], $n^1 = 20$	Control[a], $n^2 = 20$	
Blood calprotectin [µg/l]	40	81.26 (57.93, 121.95)	112.24 (81.39, 206.82)	60.31 (37.41, 81.54)	0.001
Blood calprotectin categories:	40				0.182[d]
> 250 µg/l		6.00 (15.00%)[b]	5.00 (25.00%)[b]	1.00 (5.00%)[b]	
≤ 250 µg/l		34.00 (85.00%)[b]	15.00 (75.00%)[b]	19.00 (95.00%)[b]	
Stool calprotectin [µg/g]	40	13.55 (9.30, 31.58)	15.00 (6.20, 36.13)	13.00 (10.26, 30.15)	0.766
Stool calprotectin categories:	40				1.000[d]
High (> 50 µg/g)		8.00 (20.00%)[b]	4.00 (20.00%)[b]	4.00 (20.00%)[b]	
Normal (≤ 50 µg/g)		32.00 (80.00%)[b]	16.00 (80.00%)[b]	16.00 (80.00%)[b]	

aMdn (Q1, Q3), bn (%), cWilcoxon rank sum test, dFisher's exact test, N – sample size, n – group size, Mdn – median, Q1 – the first quartile (25%), Q3 – the third quartile (75%), p – the p-value of statistical test.

Similarly, when examining faecal calprotectin, a marker predominantly used to assess gastrointestinal inflammation, the median levels were similar between the two groups: 15.00 µg/g (IQR: 6.20–36.13 µg/g) in psoriasis patients and 13.00 µg/g (IQR: 10.26–30.15 µg/g) in controls, with a non-significant *p*-value ($p = 0.766$). When categorizing calprotectin levels into high (> 50 µg/g) and normal (< 50 µg/g), 20% of the whole investigated group was within the high values. The number of individuals with high stool calprotectin values was equal in both psoriatic and healthy adults. The results showed no significant difference in faecal calprotectin levels between psoriatic patients and healthy individuals. The faecal calprotectin levels remained within general population cut-off values.

The correlation coefficients and associated *p*-values, detailing the relationships between blood calprotectin levels and the following indices: PASI, BSA, and DLQI are presented in Table 5. The analysis yielded correlation coefficients (*Rho*) that suggested negative associations between blood calprotectin levels and all three examined indices. Specifically, calprotectin levels correlated with PASI at $Rho = -0.27$ and DLQI at $Rho = -0.26$, indicating that higher levels of this inflammatory marker might be associated with milder disease severity and have less impact on the quality of life. However, the correlation with BSA was notably weaker ($Rho = -0.08$), suggesting that calprotectin levels were less reflective of the extent of the body surface area affected by psoriasis. Importantly, none of these correlations reached statistical significance as indicated by *p*-values (PASI: $p = 0.255$, BSA: $p = 0.767$, DLQI: $p = 0.268$). There were no statistically significant correlations between the serum calprotectin

Table 5. Differences in psoriasis severity and quality of life indices grouped by blood calprotectin levels (high vs. normal) (**A**) and correlation coefficients between blood calprotectin levels (µg/l) and psoriasis severity and quality of life indices (**B**)

A

Psoriasis severity and quality of life indices	*N*	Blood calprotectin levels		*P*-value[b]
		High (> 250 µg/l)[a] $n = 5$	Normal (≤ 250 µg/l)[b] $n = 15$	
PASI	20	10.40 (9.60, 12.60)	11.00 (10.30, 16.00)	0.205
BSA	20	20.00 (15.00, 23.00)	12.00 (11.00, 35.00)	0.827
DLQI	20	8.00 (6.00, 12.00)	12.00 (7.00, 18.50)	0.483

B

Psoriasis severity and quality of life indices	Blood calprotectin [µg/l]	
	Rho	*P*-value
PASI	–0.27	0.255
BSA	–0.08	0.767
DLQI	–0.26	0.268

[a]Mdn (Q1, Q3), [b] Wilcoxon rank sum test, n – group size, Mdn – median, Q1 – the first quartile (25%), Q3 – the third quartile (75%), p – the p-value of statistical test.

Table 6. Differences in psoriasis severity and quality of life indices grouped by faecal calprotectin levels (high vs. normal) (**A**) and the correlation coefficients between faecal calprotectin levels (µg/g) and psoriasis severity and quality of life indices (**B**)

A

Psoriasis severity and quality of life indices	*N*	Faecal calprotectin levels		*P*-value
		High (> 50 µg/g) *n* = 4	Normal (≤ 50 µg/g) *n* = 16	
PASI	20	13.40 (10.20, 19.30)	11.00 (10.18, 13.15)	0.705
BSA	20	32.50 (13.75, 52.50)	13.00 (11.25, 25.75)	0.394
DLQI	20	18.50 (16.50, 19.50)	10.00 (5.50, 13.00)	0.128

B

Psoriasis severity and quality of life indices	Faecal calprotectin [µg/g]	
	Rho	*P*-value
PASI	–0.03	0.915
BSA	–0.03	0.900
DLQI	0.10	0.666

n – group size, Mdn – median, Q1 – the first quartile (25%), Q3 – the third quartile (75%), p – the p-value of statistical test.

level and the disease severity or quality of life in psoriatic patients.

While analysing the correlations between stool calprotectin levels and the PASI, BSA, and DLQI scores as well as the correlation of the stool calprotectin level with variations in psoriasis severity and patient quality of life (Table 6), the results indicated very weak and statistically insignificant correlations between faecal calprotectin levels and both the PASI and BSA indices, with Rho values of –0.03 ($p = 0.915$) and –0.03 ($p = 0.900$), respectively. Similarly, the correlation between faecal calprotectin levels and the DLQI, an index measuring the impact of psoriasis on the quality of life, was also found to be weak and not statistically significant, with $Rho = 0.10$ ($p = 0.666$). There were no statistically significant correlations between faecal calprotectin level and the disease severity or quality of life in psoriatic patients.

Discussion

The uniformity in demographic and health status characteristics across both groups allowed for a focused investigation of plaque psoriasis without confounding influences from these variables. The strict inclusion and exclusion criteria enabled to eliminate most of the possible confounding factors. The group included only patients aged 18–40 in order to provide more homogenous population as the early psoriasis onset may be related to genetic factors.

The median duration of psoriasis among the patients was 10 years, which indicated a chronic condition with a wide interquartile range from 4.75 to 16 years, suggesting variable disease progression among individuals.

The PASI score had a median value of 11 with an interquartile range stretching from 10.18 to 15. This score placed the study participants in the moderate-to-severe psoriasis category. Correspondingly, the BSA score, which measures the percentage of body area affected by psoriasis, further supported the findings of considerable disease impact with a median score of 14.50. The DLQI, which quantifies the disease's impact on patients' lives, had a median score of 12, suggesting very large impairment in daily functioning and wellbeing.

Serum CP

Until now, only few studies concerning the link between the serum calprotectin and the condition of the skin in psoriatic patients with no arthritis have been conducted. A study by Guzel *et al*., performed in 2014, showed that the serum calprotectin level was elevated in psoriatic patients vs. healthy controls, and that serum calprotectin level was higher in patients with severe than with mild psoriasis (assessed with PASI score) [11]. In 2018, Qian and Song analysed the serum calprotectin levels in 72 psoriatic patients and found that it correlated with PASI score. It also decreased during treatment with TNF-a inhibitors, and the concentrations difference after the first month of treatment could predict the final response to it [12]. In 2019, Hamza *et al.* confirmed the finding concerning the correlation with the PASI score made by Qian and Song and found that the serum calprotectin level can well serve as a marker of response to the methotrexate treatment and predict a relapse of psoriasis after finishing the treatment course. In patients treated with methotrexate, the serum calprotectin concentration with a cut-off point > 60 µg/l could predict, with 82.35% sensitivity and 69.23% specificity, a positive response to the drug, whilst the serum concentration of calprotectin after treatment with a cutoff point > 56.5 µg/l, with 66.67% sensitivity and 100% specificity, let predict another flare [13]. However, in the study by Duvetorp *et al*., although narrowband-UVB treatment significantly

reduced S100A8, S100A9 gene expression and S100A8/A9 protein levels in lesional skin, its serum levels despite the applied treatment showed no significant change. Also, no correlation between PASI and serum S100A8/A9 protein levels was found [4]. Lack of the link between serum S100A8/A9 levels and psoriasis severity was also shown by Matsunaga *et al.* [5]. In a pilot study by Scali *et al*., which included 32 patients and 13 healthy controls, the authors found that psoriatic patients had higher serum calprotectin concentrations than healthy controls (5.68 ±2.86 µg/ml vs. 3.77 ±1.93, $p = 0.033$). However, because of the inclusion criteria, this study included 12 patients previously treated with monoclonal antibodies, 7 with other immunosuppressants and 13 that used only topical therapy. Also, the authors did not exclude patients both with psoriasis and psoriatic arthritis [14].

The results of mentioned studies remained inconsistent, they were all conducted on small, heterogeneous groups of patients with different comorbidities or methods of treatment. In our study, the median serum calprotectin level in the psoriasis group was notably higher at 112.24 µg/l compared to 60.31 µg/l in the control group ($p = 0.001$), yet none of these levels exceeded the population cut-off point. After categorizing calprotectin levels into high (> 250 µg/l) and normal (≤ 250 µg/l), despite the fact that a higher percentage of psoriasis patients (25%) exhibited high serum calprotectin levels compared to just 5% of healthy controls, the results did not reach statistical significance ($p = 0.182$), so no cut-off level that could distinguish between psoriatic patients and healthy controls could be established. The correlation coefficients and associated *p*-values, detailing the relationships between blood calprotectin levels and PASI, BSA, and DLQI show that while there might be a trend towards negative correlations, the data do not strongly support a definitive or robust relationship between blood calprotectin levels and the severity or quality of life measures in psoriasis under the conditions of this study. When analysing the differences in psoriasis severity and quality of life indices grouped by blood calprotectin levels (high vs. normal), they suggest that while there are observable differences in the median scores for psoriasis severity and quality of life between patients with differing levels of systemic inflammation (as indicated by calprotectin), these differences do not statistically confirm that higher levels of calprotectin are associated with more severe disease or lower quality of life. This could be due to the relatively small sample size, particularly in the high calprotectin group. Given these findings, it is prudent to conclude that, within this study, despite the fact that patients with moderate-to-severe plaque psoriasis seem to present general higher blood calprotectin levels than healthy individuals, they are still within the population cut-off value. The elevated blood calprotectin levels are not conclusively linked to increased severity of psoriasis or to a deterioration in quality of life.

However, the authors want to emphasize the limitations of their study. The limited size of the investigated and control group was caused by funding limitations and restrictive inclusion and exclusion criteria; however, it was still possible to find statistically significant differences between the groups. The other limitation was the fact that the measured parameter was blood serum calprotectin with no distinguishing between the subunits. It was recently found that alarmin S100A9, which can form CP heterodimers with S100A8, was mainly produced by keratinocytes and innate immune cells and in an animal model was found to play a regulatory role in psoriatic skin and joint disease [15]. It is assumed that, based on an animal model, S100A8 and S100A9 may regulate psoriasis by inhibiting production of IL-17A and –F [16]. In a study by Farag *et al*., the serum S100A8 level was significantly higher in psoriatic patients than controls and was positively correlated with PASI score ($r = 0.826$, $p < 0.001$). What is more, S100A8 (rs3806232) gene polymorphism with AA genotype and A allele significantly increased among psoriasis patients vs. controls ($p < 0.001$), seemed to influence the risk of disease development by about 5, 12, and 6 times more than AG, GG, and G alleles. The AA genotype was shown to be associated with psoriasis severity ($p = 0.005$) [17]. Comparing to psoriatic arthritis, psoriasis-only patients had significantly lower serum concentrations of S100A9, CP, VEGF, IL-6 and TNF-α, but only S100A9 and CP could efficiently discriminate between healthy controls, psoriasis and psoriatic arthritis patients. Therefore, S100A9/CP seemed to be a promising marker that could help in identifying patients with psoriasis at risk of developing psoriatic arthritis [15]. Also, serum S100A8/A9 correlated with the severity of atherosclerosis and the biological treatment with anti-TNF-α, anti-IL-12/23, and anti-IL-17 for 1 year improved S100A8/A9 levels and high-risk atherosclerotic plaques [18]. Similar results were obtained by Berg *et al.* [19]. Thus, in further studies, the authors would like to recommend not only measuring the general blood serum calprotectin level, but also focusing on the concentrations of S100A8 and S100A9.

Faecal CP

There is a strong relationship between the skin and gastrointestinal inflammation, with evidence showing a higher prevalence of psoriasis and inflammatory bowel disease (IBD) in the same patients. In the latest research, the prevalence of psoriasis in IBD patients was estimated around 1.2% [20]. In IBD, faecal CP levels are considered one of the most sensitive parameters for diagnosis. Also, the faecal CP level may serve as a marker of response to the therapy in IBD patients [21, 22]. However, most of the published studies focus on the relationship between the faecal CP level and the severity of skin and joint lesions in patients suffering not only from psoriasis, but also psoriatic arthritis. In a study by Adarsh *et al*., the authors tried

to evaluate the subclinical gut inflammation using faecal calprotectin levels and colonic mucosal biopsy in 100 patients with psoriasis, 50 patients with psoriatic arthritis and 30 with irritable bowel syndrome (IBS), finding that faecal CP was elevated in 58% of patients with PsA, 26% with PsO and 10% with irritable bowel syndrome. Sigmoidoscopic colonic biopsy showed normal results in all PsO patients [23]. In a study by Di Brizzi *et al*., including adult patients affected by moderate-to-severe psoriasis starting biological therapy, the authors found that the baseline faecal calprotectin levels were 74.7 µg/g. A statistically significant reduction was detected at week 24 of biological therapy in patients receiving IL-23 inhibitors (62.3 µg/g). However, in this group only 32% of patients were bio-naïve; also 16.15% were affected by psoriatic arthritis and 5.28% by IBD. The obtained CP faecal level at week 24 for IL-23 inhibitors, as well as general CP faecal level among patients treated with biologic therapy (57.5 µg/g) [24] still remained over the general cut-off value.

In our study, the median levels of faecal CP were similar between psoriatic patients (15.00 µg/g) and healthy individuals (13.00 µg/) with a non-significant *p*-value ($p = 0.766$). This suggests that the inflammatory processes in psoriasis may not significantly affect gastrointestinal inflammation as measured by faecal calprotectin. The analysis of faecal calprotectin categories showed that both groups have an equal proportion (20%) of individuals with high levels (> 50 µg/g), and this uniformity across groups was statistically supported ($p = 1.000$), reinforcing the notion that psoriasis-related inflammation is more systemic rather than localized to the gastrointestinal tract.

Also, there is no meaningful association between the levels of faecal calprotectin and the physical severity of psoriasis as measured by the extent of skin involvement or the intensity of lesions. This lack of correlation may imply that, within this study population, gastrointestinal inflammation as indicated by faecal calprotectin does not parallel the extent or severity of skin manifestations in psoriasis. Also, the results suggest that higher levels of faecal calprotectin are not associated with a greater impact of psoriasis on patients' life quality. These findings indicate that faecal calprotectin, while a valuable marker for gastrointestinal inflammation, may not be a relevant biomarker for evaluating the severity or the psychosocial impact of psoriasis. Our findings illustrate that in individuals with plaque psoriasis, gastrointestinal inflammation does not differ markedly from healthy controls and does not affect the course of the disease.

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Ethical approval

Approval number: PCN/0022/KB1/47/20.

Conflict of interest

The authors declare no conflict of interest.

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