Dermatology Practical & Conceptual

Therapeutic Modulation Of Peripheral Blood Cells And Inflammatory Indices During 52 Weeks Of Risankizumab In Responder Patients With Moderate-to-Severe Psoriasis: Results From A Multicenter Prospective Study

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Key message: Biologic therapies act on specific immune-pathogenic pathways and potentially on systemic inflammation as well. Risankizumab modulates relevant peripheral inflammatory indices such as neutrophils-to-lymphocyte-ratio, pan-immune-inflammation-value, systemic-immune-inflammation-index, and systemic-immune-response-index, especially in super-responder patients, with potential impact on long-term psoriatic patients' prognosis.

Key words: Psoriasis, Risankizumab, SII, SIRI, NLR

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ABSTRACT **Introduction:** Whole blood cells and derived indices can be used as a read-out of the inflammatory pathogenic processes in many human diseases, including psoriasis. Indeed, systemic treatments with special regard to anti-IL-23 agents could exert anti-inflammatory and disease modifying effects; however, there is still a lack of conclusive data.

Objectives: We aimed to assess the therapeutic effect of risankizumab on whole blood cells and inflammatory indices in psoriatic patients.

Methods: We performed a prospective multicenter observational study on adult patients with moderate-to-severe psoriasis who underwent risankizumab therapy for at least 52 weeks. Blood cell count, CRP, and ESR were prospectively recorded for each patient included in the study, at routine visits up to week 52. The ratio between peripheral cells, namely NLR, PLR, MLR, SII, and PIV, were calculated and compared at time 0 and at weeks 12, 24, and 52 of treatment. At different timepoints, modulation of laboratory values and derived indices and their correlation with disease severity and response to treatment were analyzed. Subanalysis of very early responders and late responders was performed as well.

Results: We observed a progressive reduction in inflammatory indices such as CRP, ESR, NLR, and SII during treatment, with prominent modification involving SIRI as well, in patients who had a very early and almost complete response to risankizumab. Reduction in neutrophils and MPV, a transient increase in eosinophils at week 12, and a progressive increase in peripheral basophils were associated with therapeutic response.

Conclusion: Risankizumab promotes a progressive anti-inflammatory effect, more prominent in patients with a faster response in the early phases of treatment.

Introduction

Despite consistent data on disease pathogenesis and on the efficacy of different biologic therapies, there is still a need for manageable and accessible tools to optimize psoriasis treatment. Innate and acquired immune cells such as lymphocytes and neutrophils are key drivers in psoriasis onset and maintenance, and their ratio (NLR) is a recognized marker of systemic inflammation [1–4].

Other blood-derived indices, such as pan-immune-inflammation value (PIV) and systemic immune inflammation index (SII), have been evaluated as risk factors for disease development, prognostic markers of evolution, or of treatment response in human diseases and inflammation [5–10]. Recently, two metanalyses summarized the potential application of systemic inflammatory indices in psoriasis compared to the general population, with special regard to NLR (neutrophil-to-lymphocyte ratio) and PLR (platelet-to-lymphocyte ratio) and their possible correlation to disease severity. Some studies retrospectively investigated the effect of systemic treatment on peripheral blood cells and their derived parameters, including SII and SIRI (systemic immune response index) [11–14].

For these reasons, we performed a prospective observational study on patients with moderate-to-severe cutaneous psoriasis treated with risankizumab in order to assess therapeutic effect overtime on whole blood cell count and derived indices with regard to clinical efficacy of the drug.

Materials and Methods

Patients with moderate-to-severe psoriasis, defined as PASI ≥10 or PASI <10 but BSA >10 and involvement of special sites/difficult-to-treat areas (i.e., scalp, genitals, face, and palmoplantar regions), eligible to therapy with risankizumab were prospectively enrolled in the study. Patients were prescribed risankizumab SC 150 mg at T0, week 4, and every 12 weeks thereafter.

Baseline demographic and clinical features as well as complete hematologic data, including whole blood cell count, c-reactive protein (CRP), and erythrocyte-sedimentation rate (ESR), were recorded at baseline and during risankizumab treatment at weeks 12, 24, and 52. Patients were considered responders to risankizumab if they reached at least PASI 75 within three risankizumab injections, with clinical evaluation at week 24. Patients with incomplete data or who did not reach week 52 and discontinued risankizumab, either due to primary failure (one patient) or to adverse events or for personal reasons (i.e., moved to another town, three patients) were excluded from the analysis (Figure 1). At each time point, calculation of NLR, PLR, MLR (monocyte-to-lymphocyte ratio), PIV, SII, and SIRI was performed.

The formula for SII, PIV, and SIRI were, respectively: i) [neutrophil (103/mmc) × platelet (103/mmc)]/lymphocyte (103/mmc); ii) [neutrophil (103/mmc) × platelet (103/mmc) × monocyte (103/mmc)]/lymphocyte (103/mmc) count; iii) neutrophil (103/mmc) × monocytes (103/mmc)]/lymphocyte (103/mmc) count.

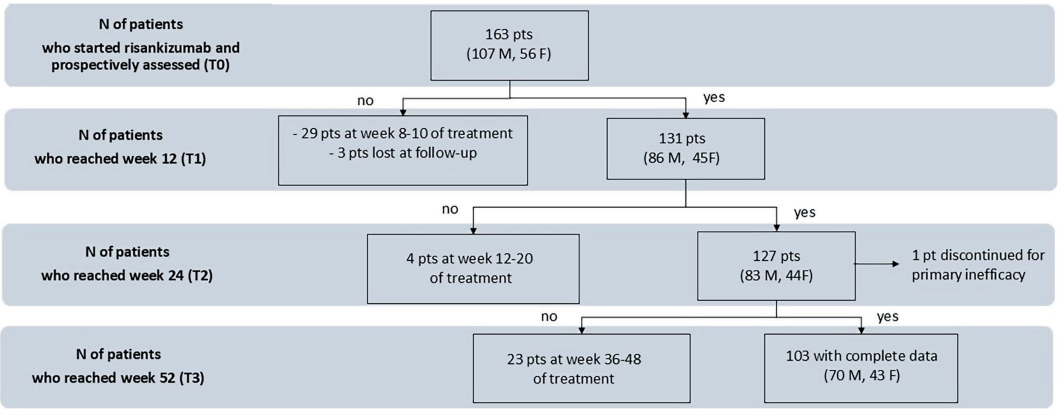

Figure 1. Flow diagram depicting the number of patients (pts) who were screened and started on risankizumab and therefore enrolled in the prospective study at baseline (T0), and the number of patients who completed each follow-up time point (T12, T24, and T52). Only patients who reached week 52 (N=103) of continuous risankizumab therapy with complete hematologic data were included in the analysis.

The primary aims of the study were to analyze the modulation of laboratory values and derived indices at different time points, and their correlation with disease severity.

The co-primary endpoint was the assessment of differences between patients who reached PASI ≥90 as early as week 12 (super-responders, SR) versus patients who reached PASI ≥75 from week 24 (late responders, LR).

Statistical Analysis

Descriptive statistics were used to summarize the characteristics of the study population. Normal distribution was tested for all samples and not parametric tests were used where appropriate. For continuous variables, the significance of the difference between medians of the groups was investigated by using Mann-Whitney test. One-way ANOVA test for independent measures (Kruskal Wallis test, plus Dunn's correction) was used to compare the means of ≥3 independent samples simultaneously. Treatment effect was studied by comparing medians of values at different time points either with Wilcoxon matched-pairs signed-rank test or with matched Friedman ANOVA test with Dunn's post-test for ≥3 samples simultaneously. Categorical variables were analyzed with Pearson's chi-square test. Fisher exact test was also used when appropriate. Correlation between variables was assessed with non-parametric Spearman rank coefficient test. All p-values cited are two-sided, and values of *P* less than .05 were considered statistically significant.

Results

Total Population and Baseline Clinical Features

In total, 103 patients (70 M, 43 F; median age: 54, IQR: 45–67) were included in the study (Figure 1). Patients were all affected with cutaneous plaque psoriasis (median PASI 13.7, IQR 9.7–16.8), and 18.4% (19/103) had concomitant arthritis (PsA). Special areas including scalp, nail, palmoplantar, genital, and face regions were involved in 55.3% (57/103), 35.9% (37/103), 33.98% (35/103), 25.2% (26/103), and 6.8% (7/103) of patients, respectively. Patients had all been previously treated with conventional systemic agents: (69/103; 66.9%) cyclosporin A, 41/103 (39.8%) methotrexate, 27/103 (26.21%) acitretin, and 1/103 (0.97%) dimethyl-fumarate or phototherapy (44/103; 42.7%), nbUVB and/or PUVA; 16/103 (15.5%) were naïve to biologics. Among bio-experienced patients, 37/103 (35.9%) had failed one, 34/103 (33%) two, 7/103 (6.8%) three, 6/103 four (5.85), 2/103 five (1.9%), and 1/103 (0.97%) six biologic agents, with a median number of one biologic agent (IQR 1–2/patient).

Patients had a median body mass index (BMI) of 26.54 (IQR: 24.14–29.41), and 21/103 (20.4%) patients were obese (BMI ≥30). Comorbidities were reported for 65/103 (63%) patients, the most common being systemic hypertension (35/103; 33.98%), dyslipidemia (24/103; 23.3%), and type 2 diabetes (9/103; 8.74%). Latent TB, previous HCV infection, and inactive HBV infection were recorded in 13/103 (12.6%), 3/103 (2.9%) and 8/103 (7.7%) patients, respectively. Baseline laboratory values of the overall population and derived indices are reported in Table 1.

A significant correlation between age and BMI was found (*P*=0.015; r= 0.2391), mainly linked to lower BMI in females (M: *P*=0.24 r=0.142; F: *P*= 0.014, r=0.424). Actually, male patients (BMI: 27.28; IQR: 25.36–29.76) were significantly overweight compared to females (BMI: 24.14, IQR: 21.14–28.19) (*P*=0.0015), while no gender difference in disease severity (PASI M= 13.75; IQR: 9.7–16.23; PASI F= 13.9; IQR 10.8–17.4; *P*=0.9089) or age at observation (age M=53; IQR: 45.75–64.25); Age F=57; IQR 41–71; *P*=0.9089) was found.

Table 1. Whole Blood Cells and Derived Indices at Baseline and their Therapeutic Modulation during Risankizumab Treatment.

	T0	Week 12	Week 24	Week 52	p
WBC mmc/10^3	6.95 (3.58-8.91)	7.18 (5.86-8.41)	7.18 (5.67-8.690)	6.91 (5.72-8.17)	0.3746° 0.0869@
RBC mmc/10^6	5.120 (4.82-5.4)	5.11 (4.76-5.38)	5.13 (4.77-5.37)	5.11 (4.66-5.39)	0.6545 0.9586@
PLT mmc/10^3	261 (228-306)	253 (219-316)	269 (215-319)	265 (217-318)	0.3588° 0.2444@
MPV Fl	10** (8.45-10.75)	10.03** (8.5-10.7)	10.13 (9.02-11.1)	10.13** (9.11-11.07)	**0.0016°** **0.0004@**** T0-W52, W12-W52
Neutrophils mmc/10^3	5.2 (3.98-6.15)	5.01 (3.98-6.05)	5.07 (3.73-6.11)	4.61 (3.59-6)	**0.0364°** 0.0562@
Lymphocytes mmc/10^3	2.1 (1.53-2.41)	2.15 (1.7-2.39)	2.14 (1.73-2.36)	2.15 (1.76-2.34)	0.9821° 0.6090@
Monocytes mmc/10^3	0.58 (0.39-0.86)	0.58 (0.41-0.85)	0.56 (0.39-0.8)	0.57 (0.4-0.77)	0.4397° 0.1156@
Eosinophils mmc/10^3	0.23** (0.16-0.41)	0.27** (0.18-0.45)	0.24* (0.15-0.35)	0.24* (0.15-0.34)	0.3751° **0.0007@*** T0-W12,**W12-W24nd 52
Basophils mmc/10^3	0.07 (0.03-0.11)	0.08* (0.04-0.14)	0.08* (0.04-0.13)	0.08* (0.04-0.14)	**0.0439°** **0.0008@***W12-24; *W24-52
ESR mm/h	27.0**** (15.25-38)	22 (13.25-29.75)	25* (13.25-29)	19****,* (13-23)	**<0.0001°** **<0.0001@****** T0-W52; *W24-W52
CRP mg/dL	0.47***,**** (0.34-0.53)	0.36***,** (0.27-0.45)	0.31****,** (0.21-0.39)	0.26****,*** (0.2-0.33)	**<0.0001°** **<0.0001@***** T0- W12, W12-W52, **W12-W24, ****T0-W24 and 52
NLR	2.44 (1.81-3.18)	2.42 (1.79-2.82)	2.35 (1.74-2.89)	2.31 (1.79-2.67)	**0.0063°** 0.2394@
PLR	123.2 (100.9-61.6)	129.5 (101.-162.3)	127.8 (102.6-61.4)	126.3 (100-159.6)	0.3840° 0.4252@
MLR	0.29 (0.196-0.43)	0.29 (0.21-0.41)	0.28 (0.19-0.38)	0.27 (0.2-0.38)	0.4093° 0.6665@
PIV	340.9 (175-710.5)	325.6 (175-568.1)	329.7 (179.4-497.5)	274.9 (178.2-439.5)	0.0971° 0.0719@
SII	626.2 (447.3-69.4)	592.7 (390.0-804.2)	603.1 (444.3-823.7)	578.2 (392.3-822.4)	**0.038°** 0.0889@
SIRI	1.35 (0.75-2.28)	1.26 (0.84-2.3)	1.15 (0.74-2)	1.13 (0.80-1.74)	0.0698° 0.3421@

Results are shown as median with IQR; °Wilcoxon matched-pairs signed rank test; @ ANOVA matched Friedman test with Dunn's post-test.

However, neither disease severity measured as PASI score nor BMI, age, presence of PsA, special sites involvement, previous exposure to biologics, or concomitant comorbidities affected results in terms of median values or correlation to disease severity (correlation matrix, Spearman rank test for each marker and subgroup with $P>0.05$, data not shown).

ESR positively correlated with CRP ($P=0.006$; r=0.29), and both values were positively correlated to neutrophils (ESR $P=0.004$, r=0.29; CRP $P=0.001$, r=0.33), NLR (ESR $P=0.001$, r=0.29; CRP $P=0.001$, r=0.32), PIV (ESR $P=0.002$, r=0.31; CRP $P=0.005$, r=0.29), SII (ESR $P<0.001$, r=0.40; CRP $P=0.001$, r=0.32), and SIRI (ESR $P=0.012$, r=0.252; CRP

P=0.008, r=0.27). ESR was also related to RBC (*P*=0.001, r=0.32), PLT (*P*=0.009, r=0.26), eosinophils (*P*=0.004, r=0.29), basophils (*P*=0.013, r=0.25), and PLR (*P*=0.01, r=0.26), with a negative correlation only with MPV (*P*=0.012, r=-0.28).

Therapeutic Modulation of Whole Blood Cells and Derived Indices

Total Population

From T0 to week 52, risankizumab modulated different parameters, with an increase in MPV (*P*=0.0016) and basophils (*P*=0.0439) and a decrease in neutrophils (*P*=0.0364), ESR (*P*<0.0001), CRP (*P*<0.0001), and SII (*P*=0.038) (Table 1).

A dynamic increase in MPV and basophils and a transient increase in eosinophils were detected (Table 1).

A positive correlation was found between changes in PASI and NLR (ΔPASI versus ΔNLR, from T0 to week 52; *P*=0.02, r=0.23).

Super responders (SR) versus late responders (LR)

Overall, 64 patients were defined as SR (45 M, 19 F), while 39 (25 M, 14 F) patients were LR (Table 2).

Baseline median differences were observed only for neutrophils levels (SR: 4.52; IQR: 3.96–5.69 versus LR: 5.65; IQR: 4.01–6.35; *P*=0.0482) and BMI (SR: 26.09; IQR: 23.68–28.97 versus LR: 27.77; IQR: 24.98–31.64; *P*=0.0497, which were higher in LR patients. Normal weight patients did not display any baseline or over-time difference in blood-derived indices compared to patients with BMI >25 (data not shown). Moreover, BMI did not impact baseline disease severity or the overall response to risankizumab since all the patients reached PASI 75 at week 24, and no difference in PASI score at week 52 was found between LR and SR (Figure 2).

In LR patients, PASI score inversely correlated to lymphocytes (*P*=0.02; r= -0.372) and directly correlated to

Table 2. Baseline Demographic, Clinical, and Hematologic Features in SR versus LR patients.

	SR N=64	LR N=39	p
Sex (Male, N)	45	25	0.5223§
Age (years) median (IQR)	52 (42.5-63.75)	57 (49-71)	0.1038*
BMI median (IQR)	26.09 (23.68-28.97)	27.77 (24.98-31.64)	**0.0497***
PASI median (IQR)	14 (10.8-15.95)	12.30 (7-17.4)	0.2925*
Special sites involvement, N			
Scalp	37	20	>0.9999§
Nail	25	12	0.5257§
PP	16	19	0.5457§
Genital	13	13	0.1645§
Face	3	2	0.7069§
PsA, N	11	8	0.7943§
Comorbidities, N	39	27	0.5257§
HPT	12	10	0.4686§
Diabetes	4	5	0.2937§
Dyslipidemia	11	9	0.6083§
Previous systemic therapies, N			
Cyclosporin A	43	26	>0.9999§
Methotrexate	25	16	>0.9999§
UV (nbUVB-PUVA)	29	15	0.5425§
Acitretin	11	16	**0.0109§**
Number of previous biologics			
Median number (IQR)	1 (1-2)	2 (1-2)	0.6390*
0 (N)	8	8	0.4006§
1 (N)	26	11	0.2898§
2 (N)	23	11	0.5184§
3 (N)	4	3	>0.9999§
4 (N)	2	4	0.1965§
5 (N)	0	2	--
6 (N)	1	0	--

§fisher exact test *Mann Whitney t-test, N=number of patients

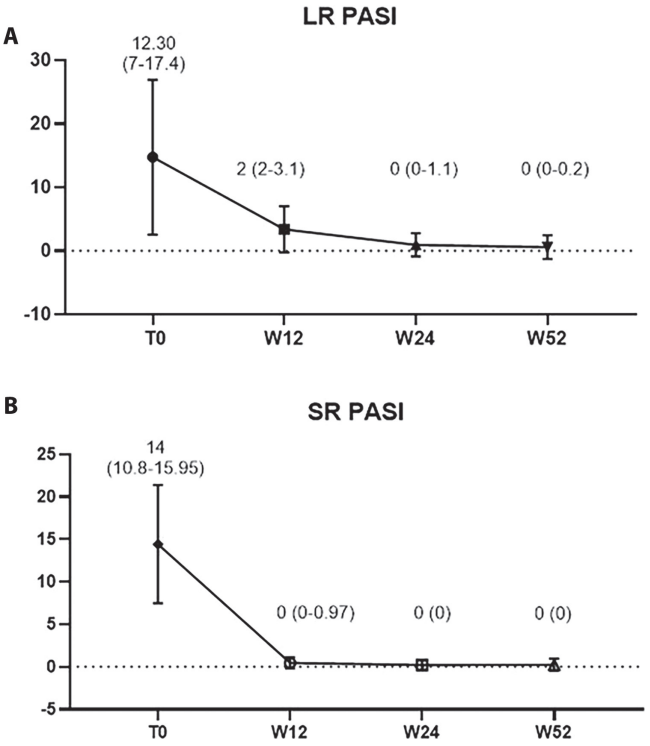

Figure 2. Efficacy of risankizumab on PASI score over time in LR (A) and SR (B) patients.

Changes in PASI score were significant as early as week 12 of treatment in both SR and LR subgroups; however, despite the fact that no difference at baseline was observed in SR versus LR patients (P >0.9999), at week 12 LR patients had significantly higher values of PASI (P<0.0001), which turned into similar scores at week 24 (SR versus LR, P>0.9999) and at week 52 (SR versus LR, P>0.9999) (multiple comparison, Kruskal Wallis test with Dunn's correction).

NLR (P=0.016, r=0.383), PLR (P=0.032, r=0.343), and SII (P=0.047; r=0.320) at baseline.

During treatment, the median number of neutrophils normalized as early as week 12 (SR: 4.625; IQR: 3.98–5.47 versus LR: 5.18; IQR: 3.98–6.32; P=0.1525).

In LR there was only a slight reduction in the inflammatory indices such as ESR (mm/h) (T0: 23; IQR: 15.5–36.5 versus Week 52: 18; IQR: 13.5–22.5; P= 0.0398), CRP (mg/dL) (T0: 0.45; IQR: 0.41–0.58 versus Week 52: 0.25; IQR: 0.22–0.35; P=0.0021), NLR (T0: 2.59; IQR: 1.81–3.61 versus Week 52: 2.48; IQR: 1.79–2.9; P=0.2491), SII (T0: 653.3; IQR: 348.4–822.1 versus Week 52: 611.5; IQR: 392.3–859.9; P=0.3872, PIV (T0: 359.3; IQR: 147.4–830.7 versus Week 52: 284.9; IQR: 178.2–575.3; p=0.6240) and SIRI (T0: 1.58; IQR: 0.67–2.24 versus Week 52: 1.28; IQR: 0.8–2.1; P=0.5759).

SR patient reached a significant reduction in ESR (T0: 28; IQR: 14–39 versus Week 52: 19; IQR: 12–23; P<0.0001), CRP (T0: 0.46; IQR: 0.31–0.52 versus Week 52: 0.27; IQR: 0.20–0.33; P<0.0001), NLR (T0: 2.23; IQR: 1.83–2.97 versus Week 52: 2.20; IQR: 1.78–2.54; P=0.0124), SII (T0: 596.3; IQR: 452.3–898.3 versus Week 52: 568.4; IQR: 388–815.5); P=0.0477), and SIRI (T0: 1.32; IQR: 0.76–2.36 versus Week 52: 0.99; IQR: 0.76–1.62; P=0.0462). PIV slightly reduced over time (T0: 327.1; IQR: 192.3–700.1 versus Week 52: 269.3; IQR: 178.6–408.8; P=0.0768).

Furthermore, at week 52, NLR was significantly lower in SR patients (SR: 2.2; IQR: 1.78–2.53 versus LR: 2.48; IQR: 1.79–2.91; P=0.0495), despite the fact that ΔNLR was positively correlated to ΔPASI only in LR patients (P=0.022, r=0.37).

The effect on transient increase in eosinophils was maintained in both LR and SR patients: for LR, T0: 0.26; IQR: 0.15–0.43; Week 12*: 0.32; IQR: 0.16–0.47; Week 24: 0.28; IQR: 0.16–0.40; Week 52: 0.26; IQR: 0.14–0.38; P= 0.0099. For SR, T0: 0.22; IQR: 0.16–0.4; Week 12*: 0.25; IQR: 0.18–0.42; Week 24: 0.22; IQR: 0.14–0.33; Week 52: 0.24; IQR: 0.16–0.32; P=0.0262), while in SR patients, only basophils (T0: 0.06; IQR: 0.03–0.10; Week 12: 0.10; IQR: 0.05–0.15; Week 24: 0.09; IQR: 0.04–0.14); Week 52: 0.09; IQR: 0.04–0.14; P=0.0001, increase) and MPV (T0: 9.8; IQR: 8.3–10.6; Week 12: 10; IQR: 8.3–10.6; Week 24: 10.11; IQR: 8.4–11.20; Week

52: 10.13; IQR: 8.9-11.04; *P*=0.0001, reduction) modified overtime.

Discussion

The role of cutaneous and systemic inflammation in psoriasis has been extensively studied and demonstrated [15–17]. However, understanding the potential effect of systemic treatment at the peripheral level is still under investigation.

Recently, many authors have focused on the application on simple indices such as the ratio between blood cells in order to identify disease severity and activity markers in many human diseases, from cancer to inflammatory disorders, including psoriasis [5,12,18–22].

Data in the literature suggest that patients with cutaneous psoriasis and PsA have significantly increased NLR, MLR, PLR, and SII compared to controls. Moreover, NLR and PLR positively correlate with the mean PASI scores being significant indicators of psoriasis severity [11,12,23,24]. Tiuca OM et al.[25] suggested that patients with mild psoriasis could be defined by WBC<6.25, neutrophils <3.64, NLR <2.35, d-NLR <1.49, and SII <408.8. In our opinion, these data need to be carefully validated and correlated to clinical cutaneous and general features of each patient.

In our patients, no correlation was found between inflammatory indices and disease severity, with the exception of patients that had a lower response to risankizumab. This fits with the concept that NLR might serve as marker of global disease severity. Indeed, LR patients had higher NLR levels at week 52 compared to SR group, and changes in NLR were correlated to changes in PASI score.

Modulation of inflammatory indices during systemic and biologic therapies has been reported, suggesting that treatment efficacy could impact the total inflammatory overload in patients who are at risk or are affected with several comorbidities such as diabetes, dyslipidemia, or hypertension. Indeed, all the above mentioned diseases have been linked to increases in such parameters [26–32].

Risankizumab is one of the latest molecules introduced for psoriasis and PsA, one of the anti-IL-23p19 agents, which have been proposed as having a disease-modifying effect [33–36]. Understanding the ability of risankizumab to obtain general anti-inflammatory effect could add relevant information for therapeutic decision making and timing. Indeed, it has been reported that MLR, NMLR (neutrophils+monocytes)/lymphocytes), and SIRI positively correlate with mortality in patients with psoriasis [37].

Albayrak H. [38] described NLR, MLR, PLR, SII, and neutrophil-to-monocyte ratio (NMR) variation during methotrexate, acitretin, and anti-TNF agents, suggesting that biologics are more effective in reducing inflammation.

In a retrospective study, anti-IL-17 agents (number of patients, 314) were more effective in reducing NLR, PLR, MLR, and SII compared to anti-TNF (N=49) and also to anti-IL-12/23+IL-23 agents (N=72), which exerted a significant effect on NLR and SII. However, no subanalysis of the single molecules was performed [39].

Morariu et al. [13] retrospectively looked at the effects of anti-TNF (adalimumab, N=22; etanercept, N=18; infliximab, N=6; certolizumab; N=6), anti-IL-17 (ixekizumab, N=61; secukinumab, N=41), anti-IL-23p19 (tildrakizumab, N=11; guselkumab, N=22; risankizumab, N=44), and anti-IL-23p40 (ustekinumab, N=17) biologics. A reduction in NLR, PLR, derived NLR/dNLR, SII, SIRI, AISI, MLR, and platelet-to-monocyte ratio/PMR was observed at week 52, with differences between molecules, but without class effect. Similar results were shown by Kulakli S et al. [14], while another retrospective study suggested that lower baseline levels of NLR could be predictive of response to anti-TNF agents [40]. A retrospective analysis [41] of the Voyage I and II and Eclipse studies showed that the ability to modulate NLR, MLR, and PLR of biologic agents such as guselkumab, secukinumab, and adalimumab was higher compared to placebo-treated patients during the first 16 weeks of treatment, while two other studies showed discording results on the superiority of anti-IL17 versus anti IL-23 agents in reducing systemic inflammation [42,43].

This study offers a prospective evaluation on hematologic indices in psoriasis, and it includes the largest population of patients treated with risankizumab analyzed for blood cells and inflammatory values.

The results of this study confirmed the potential ability of risankizumab to reduce systemic inflammation, lowering ESR, CRP, NLR, and SII overtime. It also demonstrated the effect of systemic treatment on PIV in psoriasis, which has been reported in other cutaneous diseases such as hidradenitis suppurativa [5,44–46].

Another new finding of our study is the transient increase in eosinophils during the first weeks, and their progressive restoration to baseline levels; this is potentially relevant, since the relative risk of psoriasis incidence increases with the increase in neutrophils, monocytes, and eosinophils. Eosinophils can have a potential impact on psoriasis severity [15].

We have also shown that risankizumab increases peripheral basophils, with special regard to SR patients. Recently, Zhang et al. showed that eosinophils and basophils positively correlate to PASI and that, although not significantly, they increase during treatment [39].

We understand that our results need to be implemented and compared with data on cutaneous samples; however, due to the very low levels of cutaneous and peripheral basophils, it may be difficult to dissect this pathogenetic aspect. Indeed, a next step could be to prospectively evaluate

class effect of hematologic markers, with a direct comparison between risankizumab and other anti-IL-23 agents and anti-TNF agents and IL-17 inhibitors.

An increase in MPV without a significant impact on platelet and PLR in our patients needs to be further assessed, as data on baseline MPV levels and their modulation in psoriasis and PsA are conflicting [47–50].

In our study, risankizumab lowered the number of neutrophils, but also restored them to the baseline difference in patients with initial slower response. Biologics are known to increase the risk of non-serious infections both in short- and long-term treatment. However, risankizumab profile is very safe [33,51], and in our patients the number of neutrophils was always within normal range at each time point, thus suggesting overall rebalancing of circulating and re-circulating cutaneous WBCs [52].

Activated neutrophils and NETosis have been associated with early phases of psoriasis development; thus, it is not surprising that higher levels of circulating cells could influence therapeutic response, as in the LR patients in our study [53–55].

It is well known that BMI represents a negative prognostic factor to therapeutic success [56–61]; of note, some authors have highlighted that weight reduction could reduce IL-23 levels [62]. Indeed, we and others have previously shown that risankizumab is effective regardless of baseline BMI. However, BMI could affect the time needed to reach the result within week 36, despite the fact that, in our patients, differences were resolved as soon as week 24, and no impact of BMI was observed in baseline disease severity [63–65].

Finally, the variation in PASI score significantly correlated to the variation of NLR, as reported by Zhang et al. [39].

Conclusions

Risankizumab treatment results in progressive modulation of hematologic parameters, with improvement in general systemic inflammation. Further research is needed to understand whether this modulation relates to cutaneous amelioration and whether the exerted anti-inflammatory effect lasts and contributes to disease modification, potentially offering the opportunity of treatment discontinuation or dose tapering in patients with stable response.

Ethics Approval: This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of Azienda USL Toscana Centro, Firenze (CEAVC 19799).

Ethics Statement: Informed consent was obtained from all individual participants included in the study. The patients in this manuscript gave their written informed consent to the publication of their case details.

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