

while only 2.6% of the patients felt worse in autumn, in comparison of 10.3% in spring and 6.0% in summer. 24.4% of the patients felt relieved in summer, while surprising, only 2.7% felt better in spring, with a lowest rate in the four season. However, 48.1% of the patients believed there were no seasonal differences.

Conclusions: More patients had an onset of AS in summer, compared to other seasons. More patients felt worse in winter and better in summer. Nearly half of AS patients considered that there were no seasonal differences in the deterioration or improvement of the symptoms.

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SAT0432 CORRELATION BETWEEN THE SPINAL MRI FINDINGS AND NEW BONE FORMATION FACTOR (DKK-1) IN PATIENTS WITH SPONDYLOARTHRITIS

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Background: Recent prospective data suggest that spinal inflammatory damage in patients with ankylosing spondylitis will eventually convert into fat. In these complex inflammatory lesions, bone formation and inflammation are not synchronized. The molecular basis responsible for new bone formation in SpA patients is still unclear. Serum level of dickkopf-1 (Dkk-1), the natural inhibitor of WNT protein, is a main factor in limiting new bone formation.

Objectives: In this study, we aimed to assess the correlation between the secreted protein Dkk-1 and abnormal findings on spinal MRI through a prospective study of SpA.

Methods: Thirty patients with active axial SpA (axSpA) who fulfilled the ASAS axSpA criteria were enrolled. All patients received an injection of recombinant human TNF receptor-antibody fusion protein (YISAIPU) at a dosage of 50 mg/week for 6 months. Patient report outcome measure questionnaires and physical examination, blood tests were completed according to the study protocol. All patients were scored for bone marrow edema and fat infiltration on spinal MRI imaging. The spinal MRI imaging of the patients before and after the treatment were blindly reviewed and scored using the SPARCC scoring system by two individuals who were familiar with the system.

Results: There are 28 male and two female patients (mean age: 31±5.5 yrs, range: 22–41; mean duration: 93.5±75.8; HLA-B27(+): 96.7% (29/30)). In patients who finished the 6 month anti-TNF treatment, the ESR, CRP, BASDAI, BASFI, BASMI and ASDAS-CRP were significantly decreased ($P<0.01$). Serum Dkk-1 concentration was also significantly decreased ($P<0.05$), as were the edema measurements of spinal bone marrow ($P<0.05$), but not with the before and

Table 1. Clinical indexes, serum DKK-1 and spine imaging scores before and after treatment

	Before treatment	After treatment
ESR (mm/h)	23.78±22.27	5.03±4.63**
CRP (mg/dl)	2.59±2.90	0.40±0.52**
BASDAI	6.23±1.29	2.52±1.84**
BASFI	5.78±1.44	2.69±1.72**
BASMI	2.46±1.91	0.69±1.21**
ASDAS-CRP	3.77±0.83	1.58±0.74**
DKK-1 (ng/ml)	98.23±113.41	51.88±41.90*
Spine-BME	20.27±23.53	6.08±8.09**
Spine- FAT	10.08±10.38	13.81±15.34

* $p<0.05$; ** $p<0.01$.

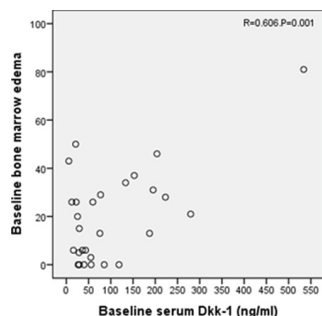


Figure 1. Serum Dkk-1 level is correlated with bone marrow edema at baseline

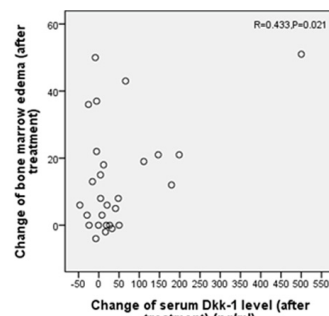


Figure 2. After treatment, the change of serum Dkk-1 level is correlated with change of bone marrow edema

after treatment differences in fat infiltration scores ($p>0.05$). (Table1). Correlation analysis found that serum Dkk-1 concentration before treatment was significantly correlated with spinal bone marrow edema scores ($P<0.01$). The differences in serum Dkk-1 levels significantly correlate with differences in spinal MRI bone marrow edema scores after treatment ($P<0.05$). (Figure 1 and 2).

Conclusions: Spinal marrow edema may have a role in predicting new bone formation in the spine, since the change of serum Dkk-1 level is correlated with change of spinal marrow edema. And Dkk-1 may participate in the molecular basis of the TNF inhibitor's blockade of new bone formation. Further research is needed on patients who have received long-term TNF antagonist treatment to find the time points when serum Dkk-1 level reaches a stabilized plateau. Increased knowledge in this area will be helpful when assessing a predictive marker for the timing of treatment withdrawal.

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Psoriatic arthritis

SAT0433 ANTI-TNF TREATMENT IN RHEUMATOID ARTHRITIS AND ANKYLOSING SPONDYLITIS PATIENTS IS ASSOCIATED WITH A STRONG INCREASE OF PALMOPLANTAR PUSTULOSIS BUT NOT OF PSORIASIS VULGARIS

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Background: The prevalence of paradoxical psoriasis developed with biological use is already studied. However, none of these studies discriminate between psoriasis vulgaris (PV) and palmoplantar pustulosis (PPP), while these might be different entities (1). The prevalence in general population is 2–4% for PV and 0.01–0.05% for PPP (1–3). Moreover, most reports in the literature imply only a role for anti-Tumor necrosis factors (anti-TNF), although, a few cases described paradoxical psoriasis in patients treated with biologicals other than anti-TNF.

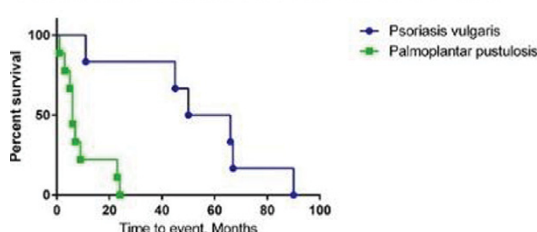
Objectives: To study the prevalence and incidence density of paradoxical psoriasis and palmoplantar pustulosis in patients with rheumatoid arthritis (RA) and ankylosing spondylitis (AS) treated with biological therapy. Second to investigate differences between paradoxical psoriasis and palmoplantar pustulosis.

Methods: Data were collected from the observational cohorts of AS and RA patients. 1499 consecutive patients were included for calculating prevalence and incidence density. Incidence density is calculated per 1000 person years. For calculating differences, only biological naïve patients ($n=830$) were included. Kaplan Meier curve was used to show the difference in time to onset.

Results: In all, 13 cases of PPP and 16 cases of PV were observed in both the RA as AS cohorts. In AS patients 1.73% developed PPP and 1.38% PV. In RA patients respectively 0.66% and 0.99%.

The incidence density of PPP in RA was 2.1 (95% CI 0.7–3.6), for PV 3.2 (95% CI 1.4–5.0). In AS, 4.7 (95% CI 0.6–8.8) for PPP and 3.7 (95% CI 2.3–12.7) for PV. Although not statistically significant, PPP was more prevalent in adalimumab (0.94%) compared to etanercept (0.34%). In contrast, PV occurs in 0.53% in adalimumab and 0.92% in etanercept treated patients. PPP was only observed in anti-TNF, PV was also observed in 1 patient treated with tocilizumab and 1 with abatacept. A difference was observed in the time to event, with a median of 6 months (IQR 4–16 months) for PPP and 50 months (IQR 11–67 months) for PV; $p=0.003$ (figure 1). Discontinuation of biological treatment was indicated in 80% of the PPP patients and 18.2% PV patients.

Figure 1. Difference between patients who developed psoriasis vulgaris versus palmoplantar pustulosis on biological therapy.



Conclusions: Our findings show that biological therapy in patients with RA or AS is associated with a 13 to 35 fold increase in prevalence of PPP. While the prevalence of biological-associated PV is lower than the prevalence of PV in the general population. In this study PV and PPP are different from each other regarding prevalence, time to onset and consequences for biological treatment, and therefore should be considered as separate entities.

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