mixed linear models. Binary logistic regression analyses were performed to determine the probability for PsA diagnosis based on capillaroscopy findings.

Results: Reduced capillary density and reduced nailfold blood flow in OCTA images distinguished PsA from both PsO ($p=0.004$ and $p=0.052$) and OA ($p=0.024$ and $p<0.001$). Qualitative analysis revealed that glomerular capillaries were found in only 3% of PsA patients but in 13% of PsO patients ($P=0.003$). Furthermore, coiled vessels were only seen in 55% of PsA patients and 71% in PsO patients ($P=0.043$). NVC microhaemorrhage was dominant in PsA patients (13 % and 17 %) and significantly different to OA patients ($p=0.05$). No capillary pattern was associated with an increased probability of the PsA diagnosis.

Conclusion: A pathognomic pattern for PsA diagnosis was not identified, however we demonstrated characteristics significant capillaroscopy findings for PsA such as decreased capillary density, reduced blood flow, and less coiled vessels in OCTA and presence of NVC microhemorrhages.

Table 1.

	NVC-rating	PsA (n=188)	PsO (n=42)	OA (n=48)	PsA vs PsO	PsA vs OA
NVC ad modum Cutolo	Reduced capillary density	51 %	31 %	51 %	$P=0.107$	0.770
	Irregularly enlarged capillaries	22 %	33 %	25 %	$P=0.082$	$P=0.988$
	Giant capillary	2 %	0	0	$P=0.400$	$P=0.375$
	Microhaemorrhages	13 %	7 %	2 %	$P=0.327$	$P=0.05$
	Capillary ramifications	14 %	24 %	29 %	$P=0.054$	$P=0.307$
	Capillary disorganisation	45 %	57 %	48 %	$P=0.170$	$P=0.685$
	OCTA-rating	PsA n=193	PsO n=48	OA n=52	PsA vs PsO	PsA vs OA
OCTA ad modum Cutolo	Reduced capillary density	53 %	21 %	33 %	$P=0.004$	$P=0.024$
	Irregularly enlarged capillaries	34 %	56 %	19 %	$p=0.015$	$P=0.178$
	Giant capillary	0	0	0	-	-
	Microhaemorrhages	0	0	0	-	-
	Capillary ramifications	0	0	0	-	-
	Capillary disorganisation	55 %	46 %	49 %	$P=0.209$	0.723
	Nail thickness*, mm., mean (SD)	0.62 (0.13)	0.73 (0.39)	0.59 (0.08)	$p=0.876$	$P=0.020$
OCT	OCTA blood flow percentage, mean (SD)	6.00 (2.94)	6.94 (3.08)	7.88 (3.30)	$P=0.052$	$P<0.001$
	Capillary morphology	PsA n=192	PsO n=48	OA n=52	PsA vs PsO	PsA vs OA
	Tortuous/Coiled capillaries	55 %	71 %	56 %	$P=0.043$	$P=0.890$
	Enlarged capillary diameter	17 %	21 %	17 %	$P=0.557$	$P=0.984$
Qualitative evaluation of NVC morphology	Ramified capillaries	13 %	15 %	19 %	$P=0.701$	$P=0.215$
	Microhaemorrhages	17 %	6 %	8 %	$P=0.068$	$P=0.106$
	Elongated capillaries	6 %	4 %	4 %	$P=0.669$	$P=0.592$
	Different capillary shapes, glomerular	3 %	13 %	2 %	$P=0.003$	$P=0.779$

The distribution of NVC and OCTA capillary morphology between PsA, PsO and OA, and qualitative evaluation of NVC morphology. Data are presented as percentage of capillary findings within the group. Differences between PsA and the other groups are presented as p-values. PsA: psoriatic arthritis, PsO: psoriasis vulgaris, OA: hand osteoarthritis, NVC nailfold video capillaroscopy, OCTA: angiographic optical coherence tomography. *: 1 patient (PsO group) was excluded from the analysis because of nail thickness>2.0mm exceeding the maximum penetration depth of the OCT laser, representing an outlier.

with psoriatic arthritis, psoriasis, and hand osteoarthritis. *: nail plate. α: cuticle. #: proximal nailfold. §: one-millimetre field.

Figure 1. Optical coherence tomography angiography of the nail and nailbed blood vessels in patients with psoriatic arthritis, psoriasis, and hand osteoarthritis. *: nail plate. α: cuticle. #: proximal nailfold. §: one-millimetre field.

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POS0145

PREDICTORS OF PSORIATIC ARTHRITIS DEVELOPMENT IN PSORIASIS PATIENTS: A SYSTEMATIC LITERATURE REVIEW AND META-ANALYSIS

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Background: Up to 30% of Psoriasis (PsO) patients are prone to develop Psoriatic Arthritis (PsA). Agreement on how to identify PsO patients at risk of developing PsA is still lacking (1).

Objectives: To identify predictors of PsA development in PsO patients through a systematic literature review (SLR) and meta-analyses (MA).

Methods: MEDLINE, EMBASE and COCHRANE databases were searched (up to February 22nd, 2020). The PICO framework (population = PsO patients; intervention = clinical, environmental, imaging and genetic features; comparator = not applicable; outcome = PsA development) was used to design searches and define the eligibility of studies for inclusion. The MA focuses on 3 major features as possible predictors: i) PsO skin and nail involvement; ii) musculoskeletal (MSK) complaints; iii) inflammation and structural damage detected by imaging (Table 1). The sample estimates of the relative risk were pooled only when the studies were homogeneous in terms of cohort of patients, follow-up, study design and metrics.

Results: 4698 articles were screened for eligibility, 110 underwent a full reading and 29 were finally included. Figure 1 shows the study flow-chart for article selection. Though pooling was judged not appropriate, 5/7 prospective studies significantly support a predictive value of PsA development in patients with severe PsO (n=174635). While the predictive value of nail involvement is not well defined (n=2202), the pooled estimate from two cohort studies (n=474) supports nail pitting as predictor of PsA (RR=2.14 [1.32; 3.46]). Moving to the MSK complaints, the risk of developing PsA is about two times greater in PsO with than without arthralgia (pooled RR: 2.15 [1.16, 3.99]) within 2 years. Lastly, PsO patients with inflammation or structural damage detected by imaging are nearly four times more likely to develop PsA within 1-2 years (pooled RR from 4 cohort studies (n=247): 3.72 [2.12; 6.51]) (Table 1). The incidence rate of PsA varies from 1.34 to 5.9/100 p-ys in PsO and from 10.9 to 12.5/100 p-ys in PsOAr.

Conclusion: Arthralgia and inflammation and/or structural damage detected by imaging are predictive features, likely prodromal, of PsA development.

PsO severity and nail pitting are clinical manifestations related to PsA development.

Figure 1.

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Table 1. Studies reporting the major features as possible predictors of PsA development.

PsO skin – severity PsO nail involvement PsO nail lesions	Variables	Relative Risk [95%CI]	Follow-up
Wilson, 2009	PsO nail involvement	HR _{adjusted} : 2.24 [1.26; 3.98]	13.1±8.8 ys
Faustini, 2015	PASI score +	/C= -0.18[-0.84; 0.48]	1.2±0.2 ys
	PsO nail involvement	RR: 0.95 [0.37; 2.47]	
Eder, 2016	PASI score	HR (>20vs<10): 5.39 [1.64; 17.7]	4.1±2.1 ys
	PsO nail involvement	HR _{unadjusted} : 1.36 [0.76; 2.45]	
Eder, 2017	Type of PsO nail lesions	HR _{unadjusted} : 2.21 [1.24; 3.92]	
	PASI score +	HR _{unadjusted} : 1.05 [1.01; 1.09]	3.8±2.1 ys
	Type of PsO nail lesions	HR _{unadjusted} : 1.98 [0.83; 4.74]	
Lewinson, 2017	PsO severity defined from medications	HR _{adjusted} (moderate/severe vs mild): 5.02 [4.18; 6.04]	5.1 ys
Egeberg, 2018	PsO severity defined from medications	RR (severe vs mild): 1.31 [1.18; 1.46]	18 ys
Elnady, 2019	PASI score +	/C= 0.79[-0.37; 1.95]	2 ys
	PsO nail Involvement	RR: 1.43 [0.38; 5.31]	
Green, 2020	PsO severity defined from medication	RR (severe vs mild): 2.79 [2.49; 3.13]	5.8 ys
MSK-complaints	Variables	Incident-PsA cases	Follow-up
Faustini, 2015	Arthralgia	RR: 1.98 [0.75; 5.19]	1.2±0.2 ys
Eder, 2017	Arthralgia	HR _{adjusted} : 2.59 [1.15; 5.88]	3.8±2.1 ys
Zabotti, 2019	Arthralgia	RR: 4.44 [0.54; 36.72]	1.6±0.5 ys
Simon D, 2020	Arthralgia	RR: 2.04 [0.86; 4.86]	2.4±1.5 ys
Sub-clinical inflammation or structural damage detected by imaging	Variables	Incident-PsA cases	Follow-up
Faustini, 2015	MRI	RR: 2.10 [0.75; 5.90]	1.2±0.2 ys
Elnady, 2019	MSK-US	RR: 5.38 [1.17; 24.63]	2 ys
Zabotti, 2019	MSK-US	RR: 6.86 [0.83; 56.63]	1.6±0.5 ys
Simon D, 2020	HR-pQCT	RR: 4.32 [1.96; 9.55]	2.3±1.4 ys

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POS0146

PERIPHERAL NEUROPATHY IN PSORIASIS AND PSORIATIC ARTHRITIS: A SINGLE BLIND, CONTROLLED, ELECTROPHYSIOLOGICAL STUDY

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Background: It is well known that both psoriasis (PsO) and psoriatic arthritis (PsA) causes morbidity and disability in patients' lives. Additional pathologies such as peripheral neuropathy (PN) may interfere with disease specific symptoms and may increase the burden of disease. Neuropathic complaints of patients with PsO and PsA are observed during patients' follow-up (1,2). However, the presence of PN in PsO and PsA is not well defined in clinical studies and clinicians might be neglecting this issue.

Objectives: Our aim was to investigate the presence of PN by electrophysiological methods and to examine its relationship with neuropathic pain and with treatments in patients with PsO and PsA.

Methods: 38 patients with PsA, 36 patients with PsO and 37 age&sex-matched healthy volunteers were enrolled into this prospective, single-blind study. Disease activity was assessed with Psoriasis Area Severity Index (PASI) for patients with PsO and with Disease Activity in Psoriatic Arthritis (DAPSA) score for patients with PsA. Leeds Assessment of Neuropathic Symptoms and Signs (LANSS) score was used to evaluate neuropathic pain. All participants were electrophysiologically examined in Electromyography (EMG) Laboratory by an experienced neurologist blinded for study groups.

Results: There were no significant differences on patients age, sex and body mass index among 3 study groups ($p>0.05$). However, disease duration was significantly longer in PsO group compared to PsA group (median 19.1[1-43] years in PsO vs median 7.5[2-29] years in PsA; $p<0.01$). PN was detected in 7 patients

(19.44%) in the PsO group, 14 patients (34.21%) in the PsA group, and in 1 volunteer (2.7%) in the control group (PsO vs control: $p<0.05$; PsA vs control: $p<0.001$, PsO vs PsA: $p>0.05$) (Table 1). Among patients with PsA, 1 patient had ulnar neuropathy and 1 patient had both carpal tunnel syndrome (CTS) and sensory polyneuropathy. All other PN detected electrophysiologically were CTS. Neuropathic pain was present in 11 patients (30.56%), 20 patients (52.63%) and in 2 volunteers (5.41%) in PsO, PsA and control groups consecutively (PsO vs control: $p<0.05$; PsA vs control: $p<0.001$, PsO vs PsA: $p>0.05$). PN did not show any significant correlation with disease activity or treatments including biological agents.

Conclusion: To our knowledge, this is the first study to investigate simultaneously the presence of neuropathic pain and PN by electrophysiological methods in patients with PsO and PsA. We observed PN and neuropathic pain high in patients with PsO&PsA and among them CTS appears as the most prominent neuropathy. Clinicians should keep this in mind during routine care of patients with PsO and PsA, which may improve patient-centred care. Large, prospective studies are needed to understand the characteristics of these neuropathies and its prognosis.

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POS0147

ANALYSIS OF VENOUS THROMBOEMBOLIC RISK AMONG PSORIATIC ARTHRITIS PATIENTS

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Background: Psoriatic arthritis (PsA) is a chronic, immune-mediated, systemic inflammatory arthritis associated with comorbidities including metabolic syndrome, cardiovascular risk factors and cardiovascular disease (CVD).

Objectives: To evaluate the prevalence of venous thromboembolism (VTE) in a PsA patient cohort using a large health care provider database.

Methods: A large health care provider database serving 4.7 million healthcare subscribers was interrogated for an adult patient cohort who were newly diagnosed with PsA between January 2005 (start date) and 31 December 2018 with date of diagnosis considered the index date. A risk set was employed to randomly select 4 controls without PsA as a comparator group to the PsA cohort matched by age, sex, ethnic group, and index date. Both groups were followed from the index date until the first occurrence of VTE event, death, or end of follow-up 31 December 2019, whichever came first. Marginal model with robust covariate estimate counting for the matching was used to estimate the crude and adjusted hazard ratio (HR) for the association between PsA and VTE. Within the group of PSA patients, Cox proportional hazard regression models was used to calculate the risk of having VTE given demographic variables, SES, smoking, selected comorbidities, and conventional vs biologic disease modifying anti-rheumatic drugs (c/bDMARD). Continuous variables were summarized with mean $\pm$ standard deviation, and categorical variables were presented as numbers and proportions. All tests were 2-sided; p values of ≤ 0.05 were considered statistically significant. All data were analyzed using SPSS, 24 (IBM SPSS Statistics for Windows, version 24.0, 2016, Armonk, NY) and SAS, 9.4 (SAS institute Inc, Cary, NC).

Table 1. Results of peripheral neuropathy and neuropathic pain in control group and patients with PsO and PsA

	Control Group (n=37)	PsO Group (n=36)	PsA Group (n=38)	p^a	p^+	p^{Δ}
CTS	1 (%2.70)	7 (%19.44)	13* (%34.21)	0.028 ^a	<0.001 ^a	0.153 ^a
Ulnar Neuropathy	0	0	1 (%2.63)			
Polyneuropathy	0	0	1* (%2.63)			
Total PN	1 (%2.70)	7 (%19.44)	14 (%36.64)	0.028 ^a	<0.001 ^a	0.097 ^a
LANSS Score	5 (0:13)	6.50 (0:16)	13 (3:19)	0.170 ^b	<0.001 ^b	0.004 ^b
According to LANSS likely neuropathic pain	2 (%5.41)	11 (%30.56)	20 (%52.63)	0.005 ^a	<0.001 ^a	0.054 ^a

*: Both polyneuropathy and CTS were detected in one patient in the PsA group; Data are given as n (%) and median (minimum: maximum); a: Chi-Square Test, b: Kruskal Wallis Test; p^{Δ} value: control vs PsO, p^+ value: control vs PsA, p^a value: PsO vs PsA