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Nailfold videocapillaroscopy as a diagnostic clue in interstitial lung disease: differentiating idiopathic from autoimmune patterns in a Caucasian cohort

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Contributions: Daniele Vito Andrea Sabella, Marco Fornaro: conception and design of the study. Daniele Vito Andrea Sabella, Marco Fornaro, Laura Coladonato, Ester Boniello, Luciana Labate, Paola Piarulli, Anna Violante: patient recruitment and data collection. Daniele Vito Andrea Sabella: statistical analysis and draft of the manuscript. Marco Fornaro, Florenzo Iannone: critical revision of the manuscript for important intellectual content. All authors: data interpretation, review of the manuscript, and approval of the final version to be published. Florenzo Iannone is the guarantor of the study and takes responsibility for the integrity of the data and the accuracy of the analysis.

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Ethics approval and consent to participate: the study was conducted in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki Declaration and its later amendments. The study was approved by the local Ethics Committee (INflammatory MYositis Registry: study no. 6229, approval no. 84762,2020/11/06; comitatoetico@policlinico.ba.it).

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Abstract

Objective. Interstitial lung diseases (ILDs) include idiopathic pulmonary fibrosis (IPF) and autoimmune-related forms such as interstitial pneumonia with autoimmune features (IPAF) and connective tissue disease-associated ILD (CTD-ILD). Nailfold videocapillaroscopy (NVC) is well established in systemic sclerosis (SSc) but remains underused in ILD characterization. This study aimed to evaluate NVC findings in IPF, IPAF, and CTD-ILD.

Methods. This cross-sectional study included 67 Caucasian ILD patients (16 IPF, 13 IPAF, and 38 CTD-ILD, including 21 SSc-ILD). NVC was performed using a digital videocapillaroscope and scored semi-quantitatively (0–3) across six parameters (total score 0–18). Patterns were classified as normal, nonspecific abnormalities, or scleroderma pattern (early, active, or late). Continuous variables were analyzed with one-way analysis of variance and Bonferroni correction; categorical variables with χ^2 or Fisher's exact test.

Results. The total NVC score increased progressively from IPF (3.2 ± 1.4) to IPAF (4.5 ± 3.2) and CTD-ILD (6.5 ± 3.1 ; $p=0.001$). Capillary loss was the only item of NVC significantly different among groups ($p=0.006$), although all items showed a gradual increase across the spectrum. Scleroderma patterns were observed in 6.3% of IPF, 23.1% of IPAF, and 65.7% of CTD-ILD patients, particularly in SSc-ILD (95.2%). Notably, 81.3% of IPF patients showed nonspecific abnormalities, suggesting subtle microvascular involvement even in idiopathic disease.

Conclusions. NVC demonstrates potential discriminatory value between IPF and autoimmune-related ILD, especially SSc-ILD. The progressive increase in microvascular abnormalities observed from IPF to SSc-ILD suggests a possible contribution of immune-mediated microvascular involvement across different ILD phenotypes. These findings should be considered exploratory and warrant confirmation in larger prospective studies. NVC may represent a useful adjunct to serological and radiological assessment in ILD diagnostic pathways.

Introduction

Interstitial lung disease (ILD) is a manifestation that can occur in a diverse group of disorders affecting the alveolar–interstitial compartment (1). Among these, idiopathic pulmonary fibrosis (IPF) is a persistently progressive fibrotic lung disease with a poor prognosis and response to immunosuppressive therapy, whereas connective tissue disease-associated ILD (CTD-ILD) often follow a different pathophysiologic and therapeutic trajectory, with a better prognosis (2). A third group, interstitial pneumonia with autoimmune features (IPAF), describes patients with ILD who exhibit autoimmune serology or clinical findings without fulfilling diagnostic criteria for a defined connective tissue disease (CTD). Distinguishing between idiopathic and autoimmune ILD is clinically crucial, as the presence of autoimmunity may indicate potential responsiveness to immunomodulatory therapy (3). Given that CTD are characterized by immune-mediated vasculopathy (4), nailfold videocapillaroscopy (NVC), a noninvasive method that visualizes microvascular morphology, may serve as a useful adjunct for identifying autoimmune involvement. Although NVC is a cornerstone in systemic sclerosis (SSc) diagnosis (5), its potential diagnostic role in ILD remains insufficiently characterized (6). This study aimed to evaluate NVC findings in patients with IPF, IPAF, and CTD-ILD, and to explore differences in microvascular patterns across these ILD subgroups.

Materials and Methods

Study design and population

This cross-sectional study was conducted at the Rheumatology Unit, University Hospital Policlinico di Bari, Italy. Sixty-seven consecutive Caucasian patients with established ILD were enrolled between November 2024 and September 2025 and classified into three groups: the IPF group included 16 patients diagnosed according to the ATS/ERS/JRS/ALAT 2018 guidelines and recruited from the Respiratory Diseases Unit at the University Hospital Policlinico di Bari (1); the IPAF group, which included 13 patients classified according to the ERS/ATS 2015 criteria (7); and the CTD-ILD group, composed of 38 patients, of whom 21 with SSc-ILD and 17 with non-SSc CTD-ILD, including antisynthetase syndrome (ASyS), dermatomyositis (DM), and overlap myositis (PM-Scl), each classified according to the current classification criteria (5, 8-10). Inclusion criteria were: age ≥ 18 years; a confirmed diagnosis of ILD among the three predefined groups (IPF, IPAF, or CTD-ILD); availability to undergo periungual NVC at our centre according to a standardized protocol; and availability of complete clinical, serological, and instrumental data at the time of assessment. Exclusion criteria included rheumatoid arthritis-associated ILD (RA-ILD) and indeterminate ILD diagnosis. RA-ILD was excluded to reduce clinical and pathophysiologic heterogeneity, as rheumatoid arthritis is associated with distinct serological and capillaroscopic characteristics that may influence NVC findings and complicate comparisons across ILD subgroups. All participants provided written informed consent.

All patients underwent high-resolution computed tomography (HRCT) for pattern classification, including usual interstitial pneumonia (UIP), nonspecific interstitial pneumonia (NSIP), organizing pneumonia (OP), and lymphocytic interstitial pneumonia (LIP), as well as complete pulmonary function testing, including forced vital capacity (FVC), forced expiratory volume in one second (FEV1), and diffusing capacity of the lungs for carbon monoxide (DLCO).

HRCT examinations were reviewed as part of routine multidisciplinary ILD assessment by experienced pulmonologists and thoracic radiologists. Patterns were classified according to current ATS/ERS recommendations as UIP, probable UIP, NSIP, OP, or LIP (11). Quantitative radiological scoring of disease extent was not available. Autoimmune serology included ANA (HEp-2 IIF), anti-ENA panel (anti-SSA/Ro, anti-SSB/La, Scl70, ACA, Jo1, Sm, RNP), anti-MPO, anti-PR3, RF, and ACPA. Myositis-specific antibodies (MSA) (Jo-1, PL7, PL12, EJ, OJ, Mi-2 a/b, TIF1gamma, MDA-5, NXP2, SAE1/2, SRP) and myositis-associated antibodies (Ku, PM-Scl100/75, Ro-52) were searched by the same line blot assay, performed according to the manufacturer's recommendations (Euroline Autoimmune Inflammatory Myopathies, Euroimmun, Germany).

Nailfold videocapillaroscopy

Patients were acclimatized at room temperature (20–24°C) for at least 15 minutes before examination. Immersion oil was applied to the nailfold. The second to fifth fingers of both hands were examined using a 200× videocapillaroscope. At least four consecutive fields were acquired for each finger and subsequently evaluated by an experienced rheumatologist trained in nailfold videocapillaroscopy. Six parameters were semi-quantitatively scored (0–3) according to a validated scoring method (12): dilated capillaries ($>20\ \mu\text{m}$), megacapillaries ($>50\ \mu\text{m}$), microhemorrhages, capillary loss (<7 loops/mm), disorganization of the capillary array, and ramified/neoangiogenic loops. A total score ranging from 0 to 18 was obtained. Patterns were classified according to the EULAR/CTC definitions as normal, nonspecific (distinct patterns), or as a scleroderma pattern (early, active, late) (13).

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics 21. Continuous variables were expressed as mean $\pm$ standard deviation. Comparisons between groups were conducted using one-way analysis of variance or the Student's t-test, as appropriate. Categorical variables were compared using the χ^2 test or Fisher's exact test. A p value <0.05 was considered statistically significant. A post hoc analysis was subsequently performed with pairwise group comparisons using Bonferroni correction.

An exploratory multivariable linear regression analysis was then performed using total NVC score as the dependent variable. Age, sex, smoking status, Raynaud's phenomenon, disease duration, UIP pattern, and diagnostic group were included as covariates.

Results

Population

Characteristics of our population are reported in Table 1 and *Supplementary Table 1*. The cohort included 67 ILD patients. Age was significantly higher in IPF (73.3 ± 7.7 years) than IPAF (64.1 ± 10.0) and CTD-ILD (61.5 ± 10.8 ; $p < 0.001$). The age at disease onset showed the same decreasing trend, being highest in IPF (69.9 ± 9.1 years), intermediate in IPAF (59.3 ± 11.4 years), and lowest in CTD-ILD (51.2 ± 10.8 years, $p < 0.001$), further supporting the distinct demographic profiles of idiopathic and autoimmune ILD. Disease duration differed significantly among groups, being shorter in IPF (3.3 ± 3.1 years) and IPAF (4.8 ± 3.9 years) compared with non-SSc CTD-ILD (10.0 ± 7.8 years) and SSc-ILD (10.6 ± 6.3 years) ($p < 0.001$). Raynaud's phenomenon was observed in 12.5% of IPF patients, 15.4% of IPAF patients, 41.2% of non-SSc CTD-ILD patients, and 71.4% of SSc-ILD patients ($p < 0.001$). Regarding HRCT patterns, UIP predominated in IPF (87.5%), whereas NSIP was most frequent in IPAF (92.3%) and CTD-ILD (92.1%). No significant differences were found in FVC, FEV1, or DLCO among the three groups ($p > 0.05$). High-titer ANA positivity ($>1:320$) was observed in 0% of IPF, 46.2% of IPAF, and 42.1% of CTD-ILD ($p < 0.0001$). Anti-Scl70 antibodies were found in 36.8% (all SSc-ILD), anti-Ro/SSA in 53.8% of IPAF and 26.3% of CTD-ILD, and anti-synthetase antibodies in 31.6% of CTD-ILD (non-SSc). The majority of IPF patients (87.5%) were seronegative.

Nailfold videocapillaroscopy scores and scleroderma patterns

At NVC, a progressive increase in microvascular abnormalities was observed across the ILD spectrum (Figure 1). The mean total NVC score showed a significant increasing trend from IPF (3.2 ± 1.4) to IPAF (4.5 ± 3.2) and CTD-ILD (6.5 ± 3.1) ($p = 0.001$). When the CTD-ILD group was further subdivided, this gradient was confirmed, with progressively higher values observed in non-SSc CTD-ILD (5.3 ± 3.2) and SSc-ILD (7.5 ± 2.8) ($p < 0.0001$) (Figure 2). Post-hoc comparisons demonstrated statistically significant differences between IPF and CTD-ILD ($p = 0.001$) and particularly between IPF and SSc-ILD ($p < 0.0001$). The difference between IPAF and SSc-ILD also reached significance ($p = 0.019$). Among the six semi-quantitative parameters, capillary loss emerged as the most discriminative feature, with mean values increasing from IPF (0.1 ± 0.3) to IPAF (0.3 ± 0.6), non-SSc-ILD (0.4 ± 0.7), and finally SSc-ILD (1.5 ± 1.3) ($p < 0.0001$). Post-hoc analysis confirmed

significant differences for capillary loss between SSc-ILD and all other subgroups (vs. IPF $p<0.0001$; vs. IPAF $p=0.002$; vs. non-SSc-ILD $p=0.003$). Other single items (dilated loops, megacapillaries, microhemorrhages, disorganization, and ramified loops) showed an increasing trend from IPF to SSc-ILD but did not reach statistical significance (*Supplementary Table 2*). Qualitatively, most IPF patients showed nonspecific capillaroscopic alterations (81.3%), whereas normal patterns were observed in 12.5%, and one case (6.3%) exhibited an early scleroderma pattern. Nonspecific abnormalities included isolated ectatic loops, sparse microhemorrhages, and minimal architectural disorganization. In the IPAF group, scleroderma patterns were observed in 23.1% of patients (early 7.7%, active 7.7%, late 7.7%). Overall, scleroderma patterns were present in 65.7% of CTD-ILD cases (early 10.5%, active 28.9%, late 26.3%) (Figure 3). When stratified, scleroderma patterns were observed in 95.2% of patients with SSc-ILD (early 14.3%, active 33.3%, late 47.6%), compared with 29.4% in non-SSc CTD-ILD (early 5.9%, active 23.5%, late 0.0%) (Figure 4). The distribution of scleroderma patterns demonstrated a statistically significant stepwise increase from IPF to IPAF and CTD-ILD ($p<0.001$). Post-hoc analysis confirmed that active and late scleroderma patterns were significantly more frequent in SSc-ILD compared with IPF ($p<0.001$) and IPAF ($p=0.042$). In an exploratory multivariable linear regression model adjusted for age, sex, smoking status, Raynaud's phenomenon, disease duration, and UIP pattern, SSc-ILD remained independently associated with a higher total NVC score compared with IPF ($\beta=6.32$, 95% confidence interval 1.60-11.03, $p=0.010$). No significant independent association was observed for age, sex, smoking status, Raynaud's phenomenon, disease duration, or UIP pattern.

Discussion and Conclusions

This study evaluated the role of NVC as a diagnostic and potentially discriminative tool across the ILD spectrum, focusing on IPF, IPAF, and CTD-ILD. We observed a clear quantitative and qualitative progression of microvascular abnormalities from IPF to SSc-ILD, with progressively higher NVC scores and increasing prevalence of scleroderma patterns across the different ILD subgroups. The main finding is the stepwise increase in total NVC score and capillary loss, significant in both overall and post-hoc analyses, confirming prior data from Lee *et al.* and Umashankar *et al.* (14, 15). Importantly, in an exploratory multivariable linear regression model adjusted for age, sex, smoking status, Raynaud's phenomenon, disease duration, and UIP pattern, SSc-ILD remained independently associated with higher total NVC scores compared with IPF. Although these findings should be interpreted cautiously because of the limited sample size, they suggest that the observed capillaroscopic differences are not entirely explained by these potential confounders.

Our study adds new insights into the intermediate phenotype of IPAF and confirms that even idiopathic forms show mild, nonspecific abnormalities, which might hold diagnostic relevance. Notably, many IPF patients displayed isolated dilated loops, rare microhemorrhages, or minimal architectural disorganization, almost never determining a scleroderma pattern, except for a single early pattern (6.3%). The only IPF patient showing an early scleroderma pattern was a 57-year-old male with a UIP radiological pattern, negative ANA and extractable autoantibodies, absence of Raynaud's phenomenon, and no clinical features fulfilling IPAF classification criteria. Therefore, despite the capillaroscopic findings, the patient remained classified as IPF according to current ATS/ERS criteria. These findings suggest that IPF is not entirely devoid of microvascular involvement. The mild and predominantly nonspecific capillaroscopic abnormalities observed in IPF may reflect chronic hypoxia, fibrotic remodeling, and endothelial dysfunction rather than systemic autoimmunity, consistent with evidence supporting endothelial-to-mesenchymal transition and vascular rarefaction in fibrotic lung disease (16-18). However, the relatively high frequency of these mild abnormalities reduces the specificity of NVC for distinguishing IPF from IPAF. Indeed, IPAF patients showed higher NVC scores than IPF patients but largely maintained nonspecific patterns, with only a minority displaying scleroderma-type features. This intermediate profile likely reflects the heterogeneous nature of IPAF, in which capillaroscopic abnormalities may depend on the relative contribution of autoimmune features, specific autoantibody profiles, Raynaud's phenomenon, and overlap with myositis- or antisynthetase-related phenotypes, which are known to exhibit

capillaroscopic abnormalities resembling those observed in systemic sclerosis (19, 20). In contrast, the marked capillary loss, giant capillaries, microhemorrhages, and architectural disorganization observed in SSc-ILD are consistent with the well-established systemic microangiopathy that characterizes the disease. Thus, while NVC is a useful supportive tool, its ability to differentiate IPF from IPAF is limited. It remains particularly useful, however, at separating the extremes of the ILD spectrum, clearly distinguishing IPF from CTD-ILD, particularly SSc-ILD, where active and late scleroderma patterns with marked capillary loss dominate. These findings may be compatible with the hypothesis of a microangiopathic continuum across different ILD phenotypes: minimal, fibrosis-related endothelial dysfunction in IPF; intermediate patterns in IPAF; and overt autoimmune vasculopathy in CTD-ILD, especially SSc-ILD. However, this interpretation remains speculative and requires validation in larger longitudinal studies. IPAF's intermediate behavior further highlights its heterogeneity, spanning early autoimmune phenotypes to idiopathic-like disease. Clinically, NVC can serve as a low-cost, noninvasive adjunct in evaluating newly diagnosed ILD. Nonspecific findings do not exclude autoimmunity, but a clear scleroderma pattern, particularly active or late, should prompt immediate rheumatologic assessment and targeted serology. Our data align closely with previous reports. Lee *et al.* found more capillary loss and megacapillaries in CTD-ILD than in IPAF or IPF (14), while Umashankar *et al.* reported scleroderma patterns in 13.8% of IPF, 27.4% of IPAF, and 80.4% of CTD-ILD, figures like ours (15). Despite the monocentric design and modest sample size, this consistency strengthens our findings. Importantly, while Lee *et al.* investigated an Asian cohort, our findings provide consistent data in a Caucasian population, supporting the reproducibility of these capillaroscopic differences across different ethnic backgrounds.

Several limitations should be acknowledged. First, the cross-sectional design precludes any assessment of temporal relationships between capillaroscopic abnormalities and ILD progression. Second, the relatively small sample size, particularly in the IPF and IPAF groups, limits the robustness of adjusted estimates despite the exploratory multivariable analysis and increases the risk of type II error. In addition, although HRCT patterns were systematically recorded, quantitative assessment of ILD extent or severity using validated scoring systems was not available, preventing a comprehensive evaluation of the potential impact of disease burden on capillaroscopic findings. Finally, capillaroscopic abnormalities were classified according to established EULAR definitions, which do not specifically account for the "scleroderma-like" microvascular changes described in inflammatory myopathies and antisynthetase syndrome. As a result, the full spectrum of microvascular abnormalities in some non-SSc autoimmune ILD patients may have been underestimated. Despite this, NVC remains highly effective in identifying the two extremes of ILD, IPF and clear immune-mediated vasculopathy such as SSc-ILD. As a simple and noninvasive tool, NVC enriches the multidisciplinary assessment of ILD. A prospective study design with NVC assessment performed at the time of ILD diagnosis could provide additional clinical and prognostic information. In particular, longitudinal evaluation of capillaroscopic abnormalities might help identify patients developing overt connective tissue disease, clarify the evolution of IPAF phenotypes, and determine whether specific NVC patterns are associated with disease progression, pulmonary function decline, or treatment response. Future adequately powered multicenter and longitudinal studies are needed to confirm these observations and to determine whether NVC abnormalities have diagnostic, prognostic, or treatment-monitoring value in ILD.

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Online supplementary material

Supplementary Table 1. Serological data according to interstitial lung disease subgroups. Values are expressed as n (%) unless otherwise specified.

Supplementary Table 2. Nailfold videocapillaroscopy scores and patterns.

Table 1. Demographic and clinical characteristics of interstitial lung disease population.

Variable (total 67 pt)	IPF (n 16)	IPAF (n 13)	Non-SSc-ILD (n 17)	SSc-ILD (n 21)	p-value
Female sex (%)	5 (31.3)	8 (61.5)	12 (70.6)	18 (85.7)	0.007
Age, years (mean ± SD)	73.3±7.7	64.1±10.0	60.9±12.7	61.9±9.2	0.003
Age at disease onset, years (mean ± SD)	69.9±9.1	59.3±11.4	51.1±12.5	51.3±9.7	<0.0001
Disease duration, years (mean ± SD)	3.3±3.1	4.8±3.9	10.0±7.8	10.6±6.3	<0.001
Smoking, n (%)	7 (43.8)	4 (30.8)	0 (0)	1 (4.8)	0.002
Raynaud's phenomenon, n (%)	2 (12.5)	2 (15.4)	7 (41.2)	15 (71.4)	<0.001
HRCT patterns					
NSIP, n° (%)	0 (0)	12 (92.3)	16 (94.1)	19 (90.5)	<0.0001
UIP, n° (%)	14 (87.5)	0 (0)	0 (0)	2 (9.5)	<0.0001
Probable UIP, n° (%)	2 (12.5)	0 (0)	0 (0)	0 (0)	0.087
NSIP + OP, n° (%)	0 (0)	1 (7.7)	1 (5.9)	0 (0)	0.453
Spirometry					
FVC, % (mean ± SD)	81.1±16.8	83.8±20.9	89.4±16.8	86.5±25.8	0.720
FEV1, % (mean ± SD)	82.1±21.4	85.7±20.4	90.3±17.1	82.6±26.1	0.699
DLCO, % (mean ± SD)	54.5±22.8	49.4±16.6	57.7±18.5	58.4±17.9	0.569

pt, patients; SD, standard deviation; ILD, interstitial lung disease; IPF, idiopathic pulmonary fibrosis; IPAF, interstitial pneumonia with autoimmune features; SSc-ILD, systemic sclerosis-associated interstitial lung disease; HRCT, high resolution computed tomography; NSIP, nonspecific interstitial pneumonia; UIP, usual interstitial pneumonia; OP, organized pneumonia; FVC, forced vital capacity; FEV1, forced expiratory volume in the 1 second; DLCO, diffusing capacity of the lungs for carbon monoxide.

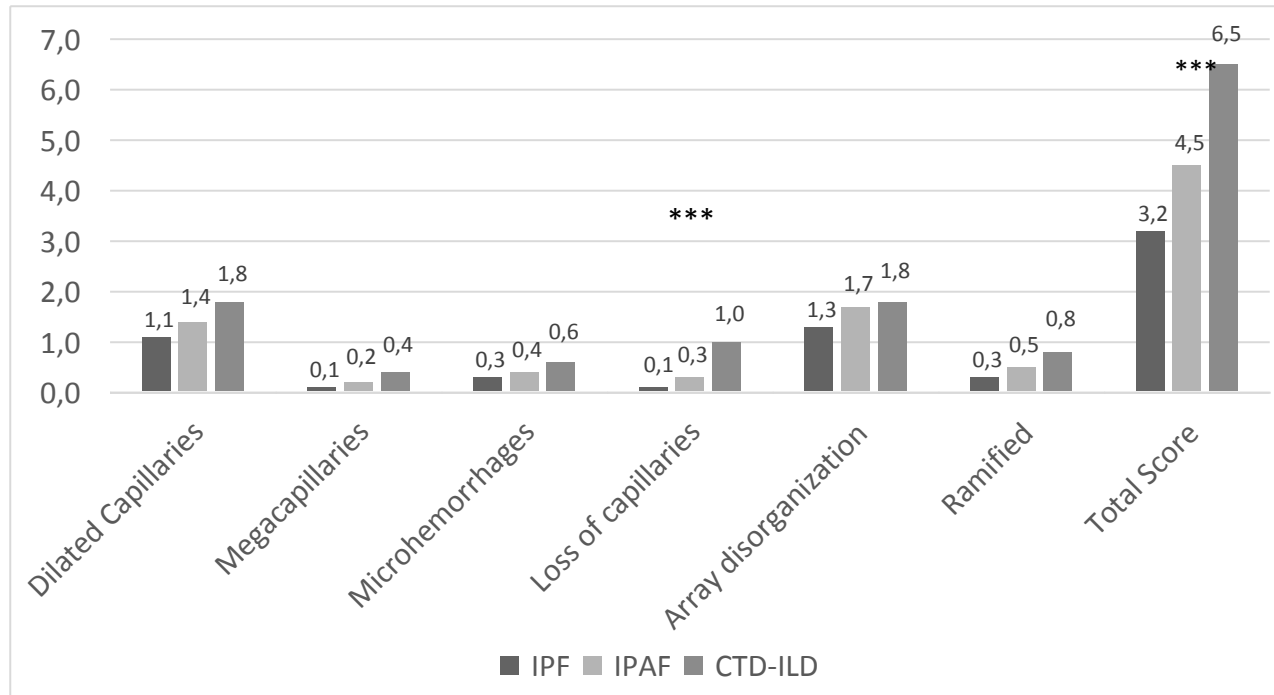


Figure 1. Mean nailfold videocapillaroscopy scores in IPF, IPAF and CTD-ILD. * $p<0.05$. IPF, idiopathic pulmonary fibrosis; IPAF, interstitial pneumonia with autoimmune features; CTD-ILD, connective tissue disease-associated interstitial lung disease.**

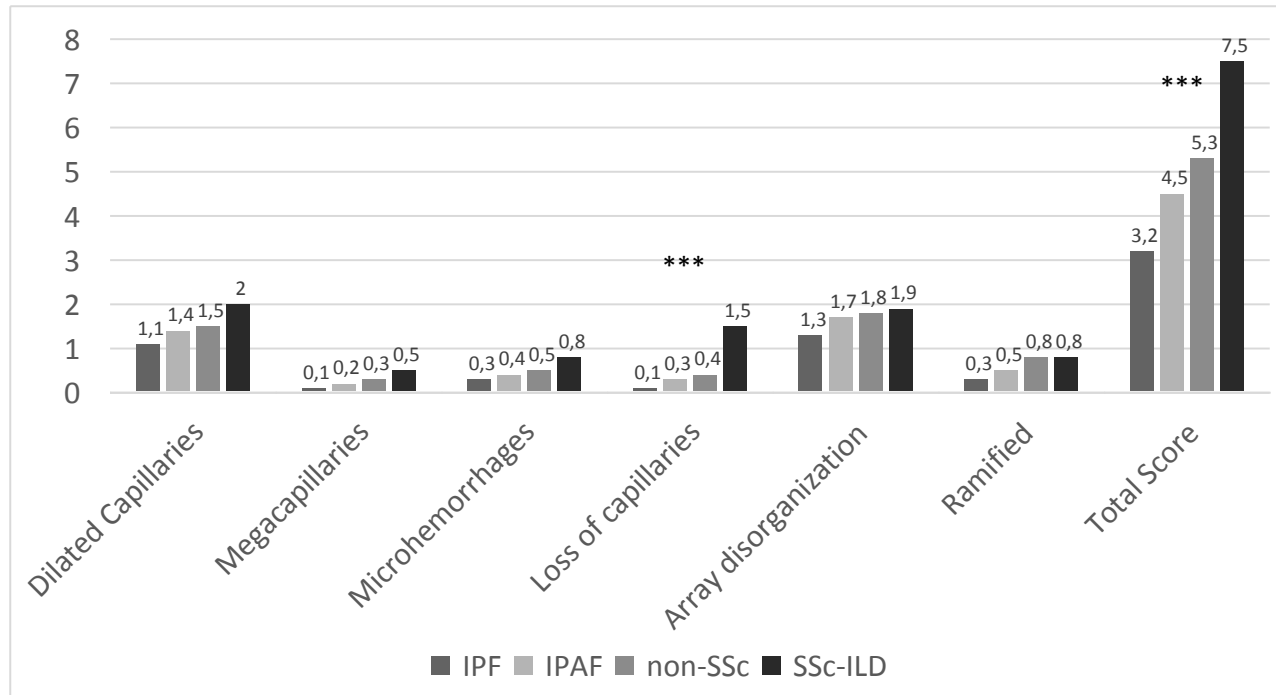


Figure 2. Mean nailfold videocapillaroscopy scores in IPF, IPAF, non-SSc-ILD and SSc-ILD. * $p < 0.05$. IPF, idiopathic pulmonary fibrosis; IPAF, interstitial pneumonia with autoimmune features; SSc-ILD, systemic sclerosis-associated interstitial lung disease**

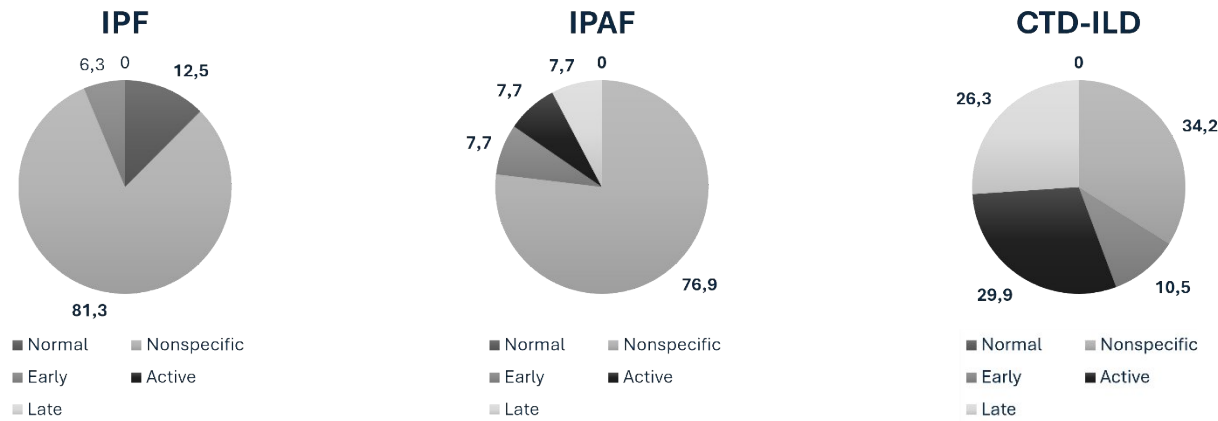


Figure 3. Frequency of nailfold videocapillaroscopy patterns in IPF, IPAF and CTD-ILD. IPF, idiopathic pulmonary fibrosis; IPAF, interstitial pneumonia with autoimmune features; CTD-ILD, connective tissue disease-associated interstitial lung disease.

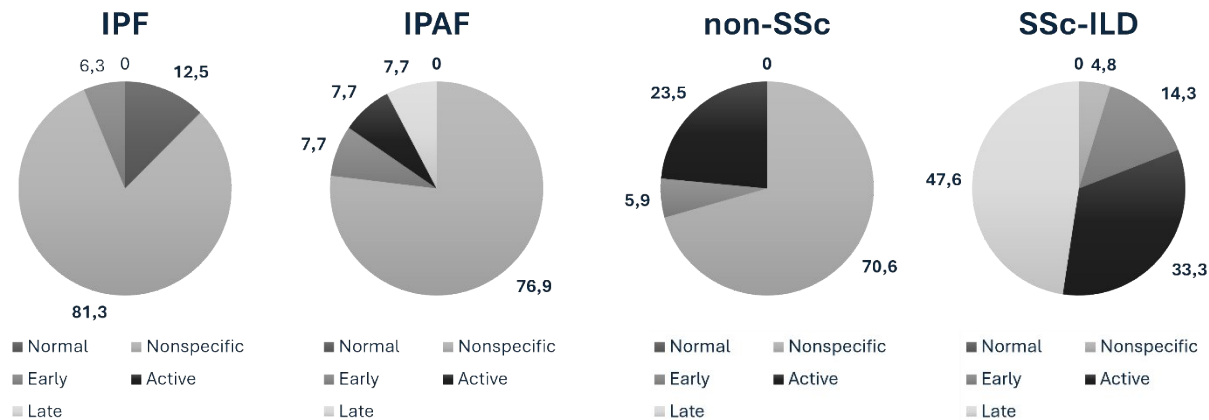


Figure 4. Frequency of nailfold videocapillaroscopy patterns in IPF, IPAF, Non-SSc-ILD and SSc-ILD. IPF, idiopathic pulmonary fibrosis; IPAF, interstitial pneumonia with autoimmune features; SSc-ILD, systemic sclerosis-associated interstitial lung disease.