Nailfold capillaroscopy in idiopathic inflammatory myopathies

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Abstract

Nailfold videocapillaroscopy (NVC) is a non-invasive technique that enables the evaluation of peripheral microcirculation by visualising nailfold capillaries. Although traditionally used in systemic sclerosis, NVC may also be useful in other systemic autoimmune rheumatic diseases, and particularly idiopathic inflammatory myopathies (IIMs). This review aims to summarise the current evidence on the prevalence of NVC findings in IIMs and their correlation with clinical and serological characteristics, as well as diagnostic, follow-up, and prognostic implications.

Keywords: Idiopathic Inflammatory Myopathy; Nailfold Videocapillaroscopy; Myositis Antibodies; Systemic autoimmune rheumatic diseases

Introduction

Nailfold videocapillaroscopy (NVC) is an easily accessible, low-cost, noninvasive technique that enables the direct evaluation of peripheral microcirculation through the visualisation of nailfold capillaries. In systemic autoimmune rheumatic diseases (SARDs), NVC has traditionally been used in the assessment of Raynaud's phenomenon (RP), facilitating the distinction between primary and secondary RP, particularly in association with systemic sclerosis (SSc)[1]. In SSc, NVC abnormalities are part of the 2013 classification criteria by the American College of Rheumatology / European League Against Rheumatism, reinforcing its role in this disease[2]. Certain NVC findings, such as altered capillary density, morphology, and dimension, form the basis for defining a capillaroscopic scleroderma pattern[3]. Although more characteristic of SSc, similar microvascular changes may also be observed in other SARDs, including inflammatory idiopathic myopathies (IIMs)[4].

IIMs are a heterogeneous group of SARDs primarily characterised by skeletal muscle inflammation and weakness, but frequently involving other organs such as the skin, joints, and lungs[5, 6]. IIMs are currently classified into six major subgroups: dermatomyositis (DM) (including clinically amyopathic DM), antisynthetase syndrome (ASyS), immune-mediated necrotising myopathy (IMNM), inclusion body myositis (IBM), polymyositis (PM), and overlap myositis (OM)[7].

Given the possibility of microvascular dysfunction, IIMs may lead to detectable abnormalities on NVC. In 2023, a multicentric Portuguese study reported that 19.1% of IIM patients had some type of capillaroscopic abnormality[8]. Nonetheless, different forms of IIMs may show a higher likelihood of specific capillaroscopic abnormalities, an aspect that is further explored in this work and may suggest different degrees of vasculopathy among subtypes.

In this review, we aim to summarise the current knowledge on the usefulness of NVC in IIMs, highlighting its potential value in disease characterisation and monitoring.

Search strategy

This study was conducted as a narrative review of the literature. A literature search was performed in the MEDLINE database, including articles published up to November 2025, without restrictions on the start date. Publications in English and/or Portuguese were considered. The search strategy used Boolean operators (AND, OR) and combined the following MeSH terms searched in the title and/or abstract: (("nailfold videocapillaroscopy") OR ("capillaroscopy")) AND (("idiopathic inflammatory myopathies") OR ("dermatomyositis") OR ("juvenile

dermatomyositis") OR ("antisynthetase syndrome") OR ("polymyositis") OR ("necrotising myopathies") OR ("overlap myositis") OR ("inclusion body myositis")). Given the limited available literature on this topic, a broad range of study designs – including systematic reviews, cohort studies, and case series – was considered. Reference lists of selected articles were also screened to identify additional relevant publications not indexed in the database. EndNote software (version 20, Clarivate, Philadelphia, USA) was used for reference management.

Titles and abstracts were screened for relevance, and full-text articles were reviewed when available, with particular attention to the presence of NVC findings across different IIM subtypes. Studies clearly unrelated to IIMs or NVC, articles not published in English or Portuguese, and papers without accessible full-text content were not considered. Study selection and data extraction were performed by a single reviewer.

Information from the included publications was organised according to IIMs subtypes. For each subtype, data were summarised in relation to the prevalence, definitions, and characteristics of NVC findings, as well as their reported associations with clinical phenotype, autoantibody profiles, and patient prognosis.

Prevalence and types of nailfold videocapillaroscopy findings in idiopathic inflammatory myopathies

Dermatomyositis

In DM, NVC abnormalities are common and tendentially more severe than in other forms of IIMs[9] (Table I). A systematic review published in 2024 found that approximately 65% of DM patients exhibited enlarged capillaries, 57.5% decreased vascularity, 45.1% microhaemorrhages, 31.2% disorganised capillary architecture, and 16.5% neovascularization[10]. Of note, this study included data not only from NVC but also from microscopy or dermatoscopy findings.

Regarding NVC patterns, DM may course with a “scleroderma-like” (ScL) pattern, in which mixed microvascular markers of the scleroderma capillary patterns coexist, although not fully fitting the definition for the single early, active, or late scleroderma pattern[11]. Manfredi et al. found that 69% of DM patients showed a ScL pattern on NVC evaluation; this pattern was not detected in PM patients or controls with primary RP[12]. This pattern was observed in 32.7% of DM patients[13] in the study by Kubo et al., and in 34% in a recent work by Leclair et al.[14]

Other NVC patterns can also be found. For instance, Kubo et al. documented a scleroderma pattern in a total of 32.6% of DM patients (19.2% early, 9.6% active, and 3.8% late patterns)[13]. Similarly, in the series by Torres-Ruiz et al., 45.2% of DM patients displayed non-specific

abnormalities on NVC, and 51.5% showed a scleroderma capillaroscopic pattern, with the active form being particularly prevalent[15].

Importantly, in DM, NVC alterations may correlate with disease activity, specific myositis autoantibodies, and clinical manifestations, as will be discussed later.

Juvenile Dermatomyositis

In juvenile dermatomyositis (jDM), a scleroderma capillaroscopic pattern is common, reported in more than 80% of patients in a study by Barth et al.[16] Potential abnormalities included capillary loss (36%), dilated capillaries (33%), and microhaemorrhages (28%)[16]. Similarly to adult DM, a ScL pattern can also be observed[17].

Antisynthetase Syndrome

NVC abnormalities are also frequent in ASyS. An Italian multicentric study published in 2019, including 190 patients, demonstrated NVC abnormalities in 62.1%, a proportion significantly higher than in healthy controls[18]. Giant capillaries (31.1%) and avascular areas (20.5%) were reported exclusively in the ASyS group[18]. Other common NVC alterations included ramified capillaries (49.5%) and a ScL pattern (35.3%). Microhaemorrhages, however, were rarer, detected in 23.2% of patients[18].

In line with these findings, Torres-Ruiz et al. reported that 80.7% of analysed ASyS patients exhibited non-specific abnormalities on NVC, while 15.3% presented a scleroderma pattern, namely the active subtype[15].

Immune-Mediated Necrotising Myopathies

In contrast, NVC abnormalities are less frequently reported in IMNM, although most available studies are limited by small cohort sizes. In 2019, Soubrier et al. evaluated NVC changes in a mixed cohort of patients including DM, ASyS, OM, and IMNM[19]. Significant capillaroscopic changes, such as giant capillaries, severe disorganisation, and marked capillary loss, were absent in the IMNM subgroup. Less relevant changes, such as ramified (13.1%) or tortuous capillaries (33.3%), and microhaemorrhages (16.6%), were documented[19]. More recently, a study reported that 83.3% of IMNM patients displayed non-specific capillaroscopic abnormalities, with a single patient exhibiting an early scleroderma-like pattern[15].

Inclusion Body Myositis

Data regarding NVC changes in IBM is limited. Available evidence suggests that abnormalities are uncommon, with one study reporting a normal capillaroscopic pattern in 85.8% of patients[15].

Overlap Myositis

OM rarely courses with a normal NVC. In a study by Soubrier et al., several NVC abnormalities were documented, including disorganised architecture (62.5%), giant capillaries (50%), tortuous or ramified capillaries (36.9% and 18.67%, respectively), reduced capillary density, and microhaemorrhages (25%)[19]. Torres-Ruiz et al. reported non-specific abnormalities and a scleroderma pattern (mostly the active form) in 50% of patients[15]. Apart from myositis, these studies did not specify the other SARD involved, although Soubrier et al. included information on detected autoantibodies[19]. A recent study reported that patients with scleromyositis presented more frequently with a non-specific or ScL capillaroscopic pattern[14].

Polymyositis

Similarly, PM rarely presents with a normal NVC. In a study by Manfredi et al., tortuous (52.2%) and enlarged capillaries (17.4%) were observed[12], consistent with the findings of Selva-O'Callaghan et al.[20] Rarer findings included ramified capillaries (8.7%), microhaemorrhages (8.7%), and capillary disorganisation (4.3%)[12]. Giant capillaries, avascular areas, and a ScL pattern were absent, contrasting with the NVC findings in DM[12]. Concerning NVC patterns, non-specific abnormalities tend to be consistently reported[15, 21, 22].

Correlation between nvc abnormalities and clinical phenotype

Microvascular abnormalities identified by NVC in IIMs patients may provide insight into disease phenotype, severity, and organ involvement (Table 2). Despite accumulating evidence, results across studies remain heterogeneous and sometimes limited by small cohort sizes. Therefore, conflicting data persist regarding the strength and specificity of most associations[23].

Disease Duration

The relationship between NVC findings and disease duration has been primarily investigated in DM. Manfredi et al. reported that patients with a disease duration less than six months exhibited lower mean capillary density and frequent giant capillaries, whereas those with longer disease duration more often displayed a ScL capillaroscopic pattern[12]. These observations suggested that NVC findings may evolve, reflecting the transition from early inflammatory to chronic microvascular changes[24, 25].

Additionally, it seems that adequate disease activity control, based on immunosuppressive therapy, may improve DM microangiopathy. For instance, Kubo et al. demonstrated that a cohort of IIMs patients, including DM and PM, showed that scleroderma-spectrum

capillaroscopic abnormalities tended to diminish after one year of immunosuppressive treatment[13]. Consistently, in a study by Mugii et al., involving DM patients, the scores of irregularly enlarged capillaries, microhaemorrhages, and loss of capillaries were significantly reduced after disease stabilization[26]. The potential regression of NVC findings seems also to be linked to certain autoantibodies, as will be developed later.

Disease Activity

Several studies have examined whether NVC abnormalities correlate with disease activity in IIMs.

In DM, Johnson et al. demonstrated that capillary density was independently associated with disease activity, as measured by the Myositis Intention-to-Treat Activity Index (MITAX)[24]. Similarly, Mugii et al. found that DM patients with a scleroderma pattern had higher scores on the muscle disease activity visual analogue scale (VAS) and a tendency toward elevated serum creatine kinase (CK) levels[26]. In an earlier study, the extent of NVC abnormalities in DM patients correlated with greater disease activity on the Myositis Disease Activity Assessment Tool (MDAAT) and with a higher severity score on the Myositis Damage Index (MDI)[20]. More recently, Tang et al. reported that greater NVC scores tended to be associated with MITAX in patients with anti-melanoma differentiation-associated protein 5 (MDA5) or anti-aminoacyl-tRNA-synthetase (ARS) antibodies, although this trend did not reach statistical significance, possibly due to a limited sample size[9]. In contrast, Torres-Ruiz et al. found no significant correlation between NVC abnormalities and clinical or laboratory markers of disease activity in a cohort formed by several subtypes of IIMs, including DM[15].

In jDM, Barth et al. observed that active disease, according to the Pediatric Rheumatology International Trials Organization criteria[27], was associated with reduced capillary density[16].

Finally, in OM, the presence of a scleroderma capillaroscopic pattern has been linked to higher global VAS scores[21].

Cutaneous Involvement

Given the prominent cutaneous manifestations of DM and jDM, several studies have explored correlations between NVC changes and skin disease in these diseases.

In a mixed cohort of IIMs patients, Bogojevic et al. associated the presence of a scleroderma pattern with diverse mucocutaneous features, including Gottron's sign, heliotrope rash, periungual erythema, and RP[22].

In DM, dilated or giant capillaries and avascular areas were significantly associated with Gottron's sign, while microhaemorrhages correlated with both Gottron's sign and heliotrope rash[21].

In jDM, Bica et al. demonstrated an inverse correlation between the skin Disease Activity Score (DAS) and mean capillary number, while higher skin VAS scores were associated with the presence of giant capillaries[28]. Furthermore, periungual telangiectasias and Gottron's papules were significantly linked to reduced mean capillary density and giant capillaries[28]. In another study, Smith et al. reported an association between skin DAS and end-row capillary loss[29].

In OM, a scleroderma capillaroscopic pattern and other NVC abnormalities, including abnormal shapes, giant capillaries, avascular areas, and haemorrhages, were associated with a higher skin VAS[21].

These data suggest that cutaneous involvement and NVC abnormalities frequently coexist, reinforcing the idea that nailfold microvasculature may reflect skin pathology in certain IIM subtypes.

Pulmonary Involvement

Regarding pulmonary involvement, most available evidence pertains to interstitial lung disease (ILD). In fact, given the prognostic relevance of ILD in IIMs, a possible correlation with NVC findings is a key area of interest.

A recently published study found an association between a scleroderma capillaroscopic pattern and the presence of ILD in a cohort of IIMs patients[22]. Sieiro Santos et al. studied a cohort with several subtypes of IIMs, finding that certain capillary findings, such as avascular zones, low capillary density, and an active scleroderma pattern, acted as predictors of ILD[30].

Wakura et al. found that, in patients positive for anti-MDA5 or anti-ARS antibodies, ILD was more lethal in those with higher median scores of microhaemorrhage[31].

In DM, a study published in 2010 demonstrated that ILD correlated with a higher number of NVC abnormalities[20].

In jDM, Barth et al. described a weak correlation between reduced capillary density and smaller lung capacity, reduced diffusion capacity, and airway disease detected on high-resolution computer tomography[32].

In ASyS, NVC abnormalities, especially ramified capillaries, were more frequent in patients with ILD[18].

Globally, these findings suggest that capillaroscopic changes may act as a window into pulmonary involvement, particularly as ILD, although their predictive or prognostic utility requires further validation.

Other Organ Involvements

Analysing a cohort with several subtypes of IIMs, Sieiro Santos et al. found an association between dysphagia and low capillary density, avascular zones, and enlarged capillaries on NVC[30]. Additionally, an international study published in 2023 concluded that NVC abnormalities, specifically enlarged capillaries and capillary loss, tended to be more frequent in IIMs patients with dropped head/bent spine syndrome[33].

Recently, Xu et al. reported a significantly higher incidence of RP in DM patients with abnormal NVC[34].

Correlations between myositis-specific autoantibodies and nailfold capillaroscopy findings

Autoimmunity is considered a key mechanism in the pathogenesis of IIMs, and myositis-specific autoantibodies (MSAs) are relevant for diagnosis, clinical phenotyping, and prognosis[35-37]. Increasing evidence suggests that certain MSAs may also correlate with distinct microvascular profiles identifiable by NVC (Table III). Importantly, the lack of large, antibody-stratified studies limits definite conclusions regarding distinct NVC aspects of each MSA.

Anti-MDA5: Severe Microangiopathy

Anti-MDA5 autoantibodies are typically associated with both mild muscle involvement and severe cutaneous and lung manifestations in DM patients[36]. NVC studies in anti-MDA5-positive patients consistently demonstrate marked microvascular injury, characterised by enlarged capillaries, capillary disorganisation, neoangiogenesis, and microhaemorrhages[25, 30, 31]. Notably, it has been suggested that these abnormalities may be reversible following immunosuppressive therapy[25]. A normal NVC pattern is rare in anti-MDA5 positive-DM; most patients exhibit non-specific or scleroderma patterns, particularly the active subtype[15]. These findings are consistent with a severe microangiopathic process, which appears linked to the pathogenesis of anti-MDA5-DM, involving a type I interferonopathy signature[38, 39] and activation of type I interferon signalling in blood vessels[40]. Therefore, the endothelial injury observed through NVC may represent a consequence of interferon-mediated vasculopathy, a hallmark of the anti-MDA5-DM phenotype[38].

Anti-TIF1γ: Chronic Capillary Rarefaction

Anti-transcriptional intermediary factor 1-gamma (TIF1γ) antibodies define a subset of DM often characterised by extensive cutaneous involvement and a higher malignancy risk in adult patients[36]. The corresponding NVC phenotype is dominated by enlarged capillaries, capillary

rarefaction, and microhaemorrhages[26]. Similar to anti-MDA5 antibodies, anti-TIF1γ-positive patients often present non-specific or scleroderma patterns on NVC, particularly the active subtype[15].

In a longitudinal analysis by Mugii et al., anti-TIF1γ–positive patients exhibited persistent capillary rarefaction and enlargement (no definitions provided) throughout follow-up, contrasting with the partial reversibility observed in anti-MDA5-positive patients[26, 41].

Anti-Mi-2, Anti-NXP2, and Anti-SAE: Subtle Findings

Data regarding the capillaroscopic features of patients with less frequent MSAs remain scarce, and comparative analyses between antibody-defined subgroups are particularly limited.

In DM, anti-Mi-2 is typically associated with the classic cutaneous features, higher serum creatine kinase levels, lower prevalence of ILD and malignancy, and an overall favourable prognosis[36]. On NVC, both scleroderma patterns and non-specific abnormalities have been observed, and a reduced capillary density is also possible[30]. Capillary disorganisation has also been described[42].

Similarly, anti-nuclear matrix protein 2 (NXP2) autoantibody, common in jDM and in association with calcinosis, has been linked to the same NVC patterns, although the available evidence is limited[15]. Enlarged capillaries are a possible capillaroscopic finding[30].

Anti-small ubiquitin-like modifier activating enzyme (SAE) autoantibody, one of the rarer MSAs in DM[36], has also been linked to scleroderma patterns in the cohort analysed by Torres-Ruiz et al[15].

Anti-synthetase Antibodies: Nonspecific Abnormalities and Chronic Vascular Damage

Anti-ARS form a group of autoantibodies directed against an aminoacyl-tRNA synthetase, essential to RNA transcription and protein synthesis[43]. Presently, this group includes anti-Jo-1 (more common, with an estimated prevalence of 25-30%) and non-Jo-1 antibodies (rarer, individual estimated prevalence <5%) – anti-PL7, anti-PL12, anti-EJ, anti-KS, anti-OJ, anti-Zo, anti-Ha, anti-Ly, and anti-VRS antibodies[35, 43, 44].

These autoantibodies are the hallmark of ASyS, characterised by classical clinical manifestations including myositis, fever, inflammatory arthritis, RP, mechanic's hands, and ILD[6, 45]. Particularly, anti-PL12 positivity was linked to microhaemorrhages, while the detection of anti-Jo1 was related to enlarged capillaries[30]. Anti-Jo1, anti-PL12, anti-PL12, anti-OJ, and anti-EJ have all been associated with a non-specific capillaroscopic pattern[15]. Most available evidence, however, doesn't differentiate between specific anti-ARS antibodies. Ramified or tortuous capillaries,

giant capillaries, microhaemorrhages, and reduced capillary density are potential findings[18, 31, 41]. These results suggest that microangiopathy is milder in anti-ARS than in pure DM[31].

Anti-SRP and anti-HMGCR: Mild, Non-Specific Findings

Data on NVC findings in IMNM are also limited, but suggestive of milder abnormalities in comparison with other forms of IIMs. The two major serological subsets are defined by anti-signal recognition particle (SRP) and anti-3-hydroxy-3-methylglutaryl-coenzyme A reductase (HMGCR) autoantibodies[36]. In a multicenter study by Soubrier et al., IMNM patients exhibited only tortuous and ramified capillaries, having no giant capillaries, major capillary loss, or severe capillary disorganisation[19]. In the analysis by Torres-Ruiz et al., including eight anti-HMGCR and five anti-SRP-positive patients, a predominance of non-specific NVC abnormalities was noted[15]. These results suggest that microangiopathy is not as significant in IMNM as in DM, although this concept remains to be validated by further research.

Nvc findings in idiopathic inflammatory myopathies in comparison with other systemic autoimmune rheumatic diseases

Most comparative studies of NVC abnormalities between IIMs and other SARDs have focused on SSc[11], although there is also some data emerging from comparative analyses, including a broader spectrum of SARDs[46].

A large multicenter study published in 2023, comprising 1,181 patients with SSc, undifferentiated connective tissue disease (CTD), mixed CTD, DM, systemic lupus erythematosus (SLE), Sjögren's disease, and primary antiphospholipid syndrome (APS), showed that capillary enlargement, microhaemorrhages, and reduced mean capillary density were significantly more frequent in SSc and DM than in other SARDs[46].

In 2016, Manfredi et al. made a longitudinal evaluation of NVC findings in patients with DM and SSc[47]. At baseline, SSc patients had more giant capillaries and severe capillary loss, whereas ramified capillaries were more frequent in DM[47]. Interestingly, giant-ramified capillaries were almost exclusively observed in patients with DM. During a 30-month follow-up, divergent evolutions were noted. In DM, there was a significant reduction in giant capillaries and a recovery of capillary density, whereas in SSc, capillary loss worsened slightly[47].

Similarly, Pizzorni et al. reported higher capillary density, ramification, and disorganisation at baseline in DM, especially in patients with shorter disease duration (<1 year); SSc patients displayed more giant capillaries and microhaemorrhages[48].

Apart from SSc and specific abnormalities such as the "comblike" haemorrhages in APS[49], NVC findings are nonspecific in most SARDs, justifying the lack of comparative studies with IIMs. Importantly, the microvasculature (and consequently, NVC findings) may also be affected by non-rheumatic comorbidities, such as diabetes mellitus[50] or hypertension[51].

Prognostic significance of nvc findings in idiopathic inflammatory myopathies

Certain NVC abnormalities seem to have prognostic value in IIMs, although most evidence pertains to DM and jDM.

Wakura et al. reported that DM patients who died due to ILD exhibited higher median microhaemorrhage and neoangiogenesis scores on NVC, suggesting a link between the severity of capillary damage and adverse pulmonary outcomes[31]. Also in DM, patients presenting with abnormal NVC showed a greater likelihood of being managed with triple combination immunosuppressive therapy[34].

In jDM, baseline NVC abnormalities have been associated with a higher probability of developing calcinosis during follow-up, emphasising the potential of NVC findings as early indicators of long-term disease damage[52].

Discussion

Current evidence supports the role of NVC as a valuable tool in the diagnosis, management, and assessment of IIMs.

Microvascular injury plays a central role in the pathogenesis of IIMs, and NVC provides valuable insight into the microvascular involvement. This is especially relevant for DM, where the deposition of complement on the endothelial wall of endomysial vessels leads to destruction of endothelial cells[53]. NVC abnormalities are frequent and tendentially more severe in DM, compared to other forms of IIM[9, 10, 15], suggesting a special contribution of microangiopathy in its pathogenesis. Severe microvascular alterations may be present early in the course of DM, with partial regression over time – an evolution not typically observed in other SARDs, such as SSc[13, 47]. Importantly, the identification of ramified giant capillaries at early stages may provide a useful clue for distinguishing DM from SSc[47].

In line with this thought, DM-associated autoantibodies appear to be more frequently linked to sclerodermic capillaroscopic patterns compared to other MSAs, more often associated with non-specific abnormalities (Table 3). This suggests that the serological profile may influence the severity of microvascular involvement, with DM-related autoantibodies reflecting a more

pronounced microangiopathy. Such association also supports a potential role of NVC as a complementary tool for phenotypic stratification in IIMs.

Apart from DM, NVC abnormalities are also frequent in other forms of IIMs, including ASyS. Patients with ASyS may present with cutaneous features resembling those of DM[6, 54], raising ongoing debate as to whether these individuals should be classified as having ASyS or as a form of overlap with DM. This distinction is particularly relevant when interpreting capillaroscopic findings, as certain abnormalities commonly associated with DM – such as giant capillaries and a ScL pattern – have been reported in ASyS. Heterogeneity in disease definition and classification may partially explain the variability of capillaroscopic findings reported in this population. Possibly, the development of refined classification criteria may help to standardise patient categorization and harmonise the reported capillaroscopic findings across future studies[55].

NVC also represents a valuable tool in the monitoring of patients with IIMs, given its character as an inexpensive, rapidly performed, widely accessible, and non-invasive examination. Particularly in DM, capillaroscopic abnormalities correlate with disease activity[20, 24, 26]. Moreover, as previously mentioned, these microvascular changes are not static; they may fluctuate over time and improve with adequate disease control. Consequently, in situations where a flare is suspected – such as in a patient with mild muscle weakness and a modest elevation in creatine kinase –, worsening of capillaroscopic findings may provide additional supportive evidence of a flare, prompting rapid therapeutic adjustment. Furthermore, NVC may also contribute to risk stratification for ILD in IIMs[22, 30], serving as an adjunctive tool to identify patients at higher risk of pulmonary involvement, potentially prompting earlier diagnostic evaluation and careful pulmonary monitoring.

Despite the growing interest in the application of NVC in IIMs, several limitations remain, opening the opportunity for future research. First, most available studies are based on small, single-centre cohorts, which restricts statistical power and hinders the generalisability of findings. Future research would benefit from the development of large, multicentric registries that systematically collect detailed and standardised NVC parameters alongside clinical data.

A second challenge relates to the lack of standardisation in NVC image acquisition and interpretation. Considerable variability persists regarding the definition and reporting of capillaroscopic abnormalities, complicating cross-study comparisons, as highlighted in Table 4. The same capillaroscopic finding can be referred to by several definitions, and distinct classification systems are applied to categorise similar abnormalities across studies. Such heterogeneity complicates the synthesis of evidence and substantially limits the feasibility of meta-analyses with pooled data. Therefore, harmonised definitions and criteria, such as the

EULAR-recommended protocols for NVC image acquisition, scoring, and terminology[3], are essential to strengthen the robustness and comparative value of future research.

Evidence regarding the association between NVC abnormalities and rare MSAs – such as anti-SAE, anti-NXP2, or anti-HMGCR – also remains limited. It is not yet clear whether the combination of specific NVC abnormalities and MSAs can inform the clinician about prognosis or risk of specific clinical manifestations. Targeted studies or stratified analyses focusing on serological subsets are therefore needed to clarify these potential associations.

Finally, while the prognostic relevance of NVC abnormalities is increasingly recognised in DM, data for other forms of IIMs remain sparse. Prospective studies assessing the evolution of NVC patterns and their association with organ involvement and clinical outcomes in non-DM IIMs are essential to determine the broader utility of NVC as a prognostic prediction tool for IIMs.

Conclusion

NVC provides a valuable window into the microvasculature of people living (or suspected of having) IIMs. Distinct findings may reflect underlying pathophysiology and hold diagnostic, prognostic, and follow-up (disease activity) relevance. Further research, including longitudinal, MSA-stratified studies, will permit the validation of the clinical impact of microvascular changes in IIMs patients.

Table I - Heat map chart representing the main nailfold videocapillaroscopy findings in different studies for the subtypes of idiopathic inflammatory myopathies.

			NVC Findings									Capillaroscopic Patterns			
IIMs	**Study**	*N*	Tortuous	Ramified	Giant	Enlarged	Disorganised	Haemorrhages	Neo	Reduced	Avascular	No	NS	Sc	ScL
DM	Selva-O'Callaghan et al.[20]	34	94%	79%		65%		67%		20%					
	Mugii et al.[26]	50												74%	
	Manfredi et al.[12]	29	58.6%	58.6%	65.5%	69%	72.4%	37.9%			31%				69%
	Manfredi et al.[47]	29		55.2%	58.6%			41.4%							
	Kubo et al.[13]	52												32.6%	32.7%
	Soubrier et al.[19]	17	36%	21.2%	17%		11.8%	29.4%		11.8%	11.8%			0%	
	Johnson et al.[24]	15						80%		80%					
	Shenavandeh et al.[21]	81			46.9%	49.4%		64%		49%	37%	0%	27.2%	72.8%	
	Bogojevic et al.[22]	37			67.6%	75.7%				70.2%		2.7%	16.2%	81.1%	
	Torres-Ruiz et al.[15]	95										3.1%	45.2%	51,5%	
	Leclair et al.[14]	41										17%	37%	12%	34%
	Xu et al.[34]	63			1.7%	41.7%		15.9%							
jDM	Barth et al.[16]	58			9%	33%		28%	41%	36%				84%	
	Shenavandeh et al.[21]	25			48%	52%		54%		72%	32%	0%	27%	72%	
	Bogojevic et al.[22]	5			20%	80%				80%		0%	20%	80%	
	Bica et al.[28]	28			39%	100%		50%		71.4		3.6%	14.2%		82.1%
	Doğantan et al.[17]	14			21.4%	57.1%		35.7%	50%		21.4%				
ASyS	Soubrier et al.[19]	12	41%	20.2%	0.09%		0%	33.3%		0%	0%			0%	
	Sebastiani et al.[18]	190		49.5%				23.2%			20.5%				35.3%
	Torres-Ruiz et al.[15]	26										3.8%	80.7%	15.3%	
	Leclair et al.[14]	16										38%	19%	37%	6%
	Xu et al.[34]	26			0%	30.4%		3.8%							
PM	Selva-O'Callaghan et al.[20]	17	88%	64%		24%		6%		0%					
	Manfredi et al.[12]	23	52.2%	8.7%	0%	17.4%	4.3%	8.7%			0%				0%
	Kubo et al.[13]	18												5.6%	22.2%
	Shenavandeh et al.[21]	19			15.8%	53.7%		43%		16%	16%	11%	47%	42%	
	Bogojevic et al.[22]	35			2.8%	2.8%				37.1%		48.6%	48.6%	2.9%	
	Torres-Ruiz et al.[15]	2										0%	100%	0%	
IMNM	Soubrier et al.[19]	6	33.3%	13.1%	0%		0%	16.6%		0%	0%			0%	

	Torres-Ruiz et al.[15]	12										8.3%	83.3%	8.3%	
IBM	Torres-Ruiz et al.[15]	7										85.8%	14.2%	0%	
OM	Soubrier et al.[19]	8	36.9%	18.7%	50%		62.5%	25%		25%	62.5%			75%	
	Torres-Ruiz et al.[15]	10										0%	50%	50%	
	Leclair et al.[14]	24										13%	13%	33%	42%

ASyS: antisynthetase syndrome. Disorganised: disorganised capillaries. DM: dermatomyositis. Enlarged: enlarged/dilated capillaries. Giant: giant capillaries. IBM: inclusion body myositis. IIMs: inflammatory idiopathic myopathies. IMNM: immune-mediated necrotising myopathy. jDM: juvenile dermatomyositis. *N:* number of patients. Neo: Neoangiogenesis. No: normal. NS: non-specific. NVC: nailfold videocapillaroscopy. OM: overlap myositis. PM: polymyositis. Ramified: ramified capillaries. Reduced: reduced capillary density. Sc: scleroderma. ScL: scleroderma-like. Tortuous: tortuous capillaries

Table II - Main clinical correlations with nailfold videocapillaroscopy findings in idiopathic inflammatory myopathies.

Clinical Domain	IIMs Subtype	Main Findings
Disease Duration	**DM**	Disease duration < 6 months associated with lower capillary density and giant capillaries[22] Longer disease duration linked to scleroderma patterns[22]
Disease Activity	**DM**	Capillary density correlated with MITAX[24] Scleroderma patterns associated with higher muscle VAS and serum CK levels[26] More NVC abnormalities correlated with greater activity on MDAAT/MDI[20] NVC abnormalities potentially resolved after adequate immunosuppressive treatment[13, 26]
	jDM	Active disease associated with lower capillary density[16]
	OM	Scleroderma pattern linked to higher global VAS[21]
Cutaneous Involvement	**IIMs**	Scleroderma patterns associated with certain mucocutaneous manifestations[22]
	DM	Giant capillaries and avascular areas linked to Gottron's sign[21] Haemorrhages associated with Gottron's sign and heliotrope rash[21]
	jDM	Skin DAS inversely correlated with mean capillary number[28] Giant capillaries linked to higher skin VAS[28]
	OM	Scleroderma patterns and other abnormalities (giant loops, avascular areas, haemorrhages) associated with higher skin VAS[21]
Pulmonary Involvement	**IIMs**	Scleroderma patterns associated with ILD[22]
	DM	ILD correlated with a greater number of NVC abnormalities[20] In anti-MDA5/ARS IIMs, fatal ILD more frequent if higher microhaemorrhage scores[31]
	jDM	Weak correlation between capillary density and reduced lung function/airway disease[32]
	ASyS	NVC abnormalities (especially ramified capillaries) more frequent in ILD[18]

ASyS: antisynthetase syndrome. DAS: disease activity score. DM: dermatomyositis. IIMs: idiopathic inflammatory myopathies. ILD: interstitial lung disease. jDM: juvenile dermatomyositis. MDAAT: Myositis Disease Activity Assessment Tool. MDI: Myositis Damage Index. MITAX: Myositis Intention-to-Treat Activity Index. NVC: nailfold videocapillaroscopy. OM: overlap myositis. VAS: visual analogue scale.

Table III - Main nailfold videocapillaroscopy findings for distinct myositis-specific antibodies.

Autoantibody	Main NVC Findings
Anti-ARS	Ramified or tortuous capillaries and reduced capillary density[18, 31] Non-specific patterns[15]
Anti-MDA5	Enlarged capillaries, capillary disorganisation, neoangiogenesis, and microhaemorrhages[25, 30, 31] Non-specific or scleroderma (active subtype) patterns[15]
Anti-Mi-2	Reduced capillary density[30] Non-specific or scleroderma patterns[15]
Anti-NXP2	Enlarged capillaries[30] Non-specific or scleroderma patterns[15]
Anti-SAE	Scleroderma patterns[15]
Anti-TIF1γ	Enlarged capillaries, capillary rarefaction, and microhaemorrhages[26] Non-specific or scleroderma (active subtype) patterns[15]
Anti-SRP **Anti-HMGCR**	Tortuous and ramified capillaries[19] Non-specific patterns[15]

Anti-ARS: anti-aminoacyl-tRNA-synthetase antibodies. Anti-MDA5: anti-melanoma differentiation-associated protein 5 antibody. Anti-HMGCR: anti-3-hydroxy-3-methylglutaryl-coenzyme A reductase antibody. Anti-NXP2: anti-nuclear matrix protein 2 antibody. Anti-SAE: anti-small ubiquitin-like modifier activating enzyme antibody. Anti-SRP: anti-signal recognition particle antibody. Anti-TIF1γ: anti-transcriptional intermediary factor 1-gamma antibody. NVC: nailfold videocapillaroscopy.

Table IV - Nailfold videocapillaroscopy findings and their different definitions across studies.

NVC Findings	Definition Across Studies
Tourtous Capillary	Capillary with a single or multiple crossovers[12, 19, 47]
Ramified Capillary	Branching or bushy interconnected capillary, originating from a single capillary[12, 18, 19, 30, 47, 48]
Dilated Capillary	Internal capillary diameter between 25-50 µm[16, 17] or 20-50 µm[9, 16, 17, 21, 28, 46, 48]
Enlarged Capillary	Increase of capillary diameter ≥20 and <50 µm[12, 25, 30, 34, 47] Increase of capillary diameter > three times the normal capillary loop diameter for the patient[23]
Giant Capillary	Homogeneously enlarged capillary loop with a diameter ≥ 50 µm[12, 16-19, 25, 28, 30, 34, 46-48] Increase of capillary diameter > ten times the normal capillary loop diameter for the patient[23]
Capillary Disorganisation	Loss of capillaries parallelism[9, 19] Irregular capillary distribution and orientation, along with heterogeneity in the loop shape[30]
Low Capillary Density	<7 capillaries/mm[9, 20, 21, 46] <6 capillaries/mm[32] 4–6 capillaries/mm[19, 34] < mean minus 2 standard-deviations, calculated from the control group[16]
Capillary Loss	<7 capillaries/mm[28, 30] <7–10 capillaries/mm[12] Reduction of the number of capillaries[48]
Capillary Rarefaction	≤ 3 capillaries/mm[19, 34]
Avascular Areas	Intercapillary distance > 500 µm[12, 18, 19, 21] Two or more capillaries missing, as compared with the areas of low capillary density in the rest of the row[30] Loss of two consecutive loops of the nail bed[23]

NVC: nailfold videocapillaroscopy. mm: milimiter. µm: micromete

FIGURES

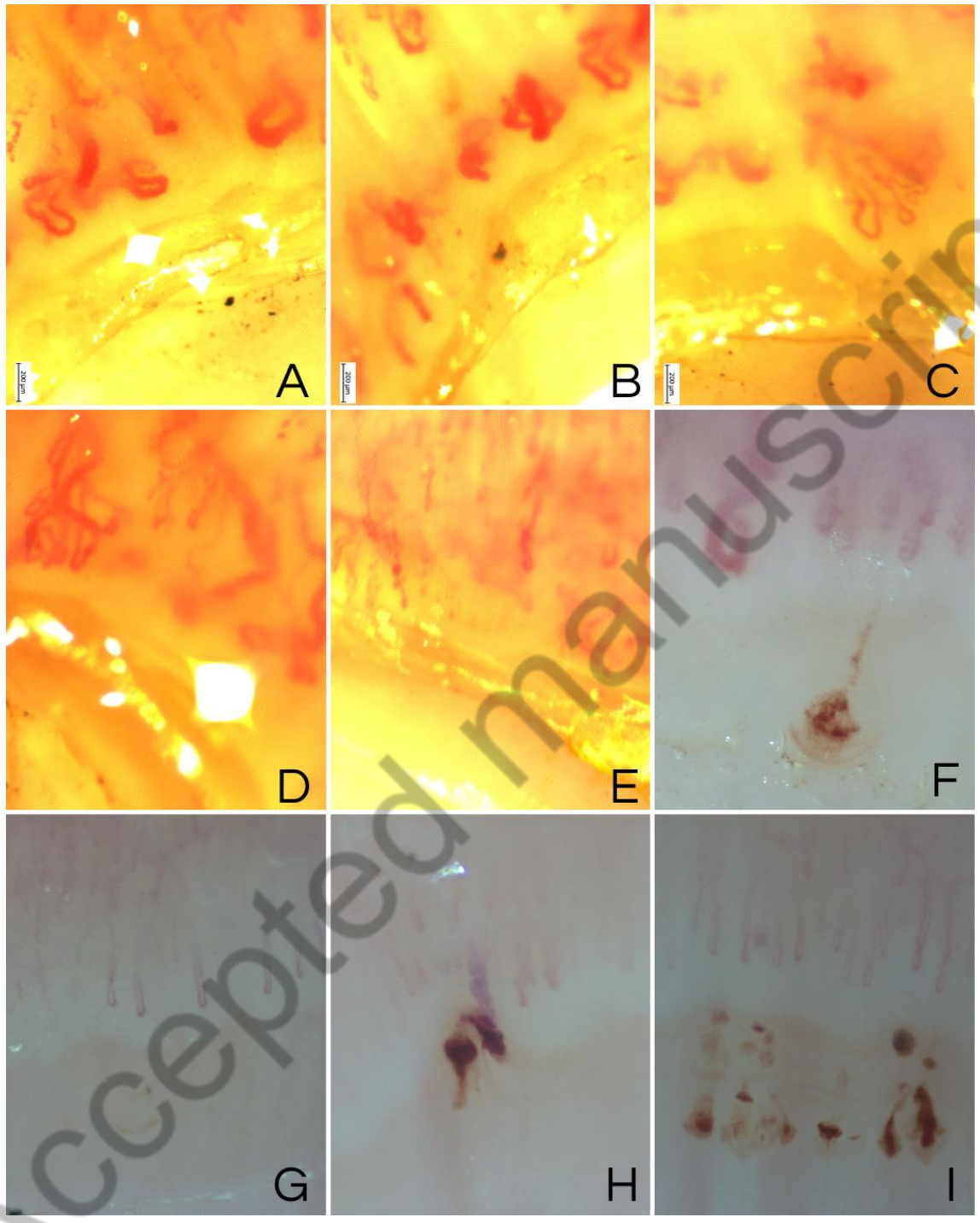

Figure 1. Examples of nailfold videocapillaroscopy findings found in people living with inflammatory idiopathic myopathies, followed at our Myositis Clinic. Images obtained with the ZEISS Stemi 508 stereo microscope, at 200x magnification.

Panels A, B, F: giant capillaries and abnormal capillaries with >50 µm. C, D: abnormally shaped capillaries in areas of reduced capillary density, suggesting neoangiogenesis. E: capillary disorganisation. G: reduced capillary density. F, H, I: microhaemorrhages.

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