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Nailfold Videocapillaroscopy in Pediatric Rheumatology: Established Roles, Emerging Applications, and Future Perspectives

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Nailfold videocapillaroscopy (NVC) provides direct, non-invasive access to the peripheral microcirculation and has become a central tool in adult rheumatology. Its primary clinical value lies in the evaluation of patients presenting with signs of Raynaud phenomenon (RP) and scleroderma-spectrum disorders. In particular, NVC facilitates the differentiation between primary and secondary (scleroderma-related) RP. The characteristic scleroderma pattern observed on NVC, including giant capillaries, capillary loss, microhemorrhages, and architectural disorganization, supports the diagnosis of scleroderma-spectrum disorders. International standardization efforts have further improved the reliability and interpretability of these findings, particularly in adult populations with RP and systemic sclerosis (SSc). In children, the clinical utility of NVC is now well established for similar indications; however, interpretation requires age-specific reference data. Pediatric capillary density, diameter, and length vary throughout childhood, and minor findings such as tortuosity, crossing, and isolated microhemorrhages may also be observed in healthy individuals. Without normative pediatric data, these features may be overinterpreted as pathological findings. Beyond RP and juvenile SSc, evidence supporting the use of NVC is accumulating across a range of pediatric rheumatic diseases. NVC abnormalities have been reported in juvenile dermatomyositis (JDM), mixed connective tissue disease, childhood-onset systemic lupus erythematosus, juvenile Sjögren disease, and juvenile localized scleroderma. Among these conditions, JDM demonstrates the strongest association between NVC abnormalities—including reduced capillary density, dilatation, abnormal morphology, and microhemorrhages—and disease activity. In contrast, findings in juvenile idiopathic arthritis remain non-specific. More recent studies involving pediatric patients with Behçet disease, psoriatic arthritis, and certain autoinflammatory and vasculitic disorders suggest that NVC may detect subtle microvascular alterations; however, disease-specific patterns have not been established outside the scleroderma spectrum. Selected non-rheumatologic conditions, particularly diabetes mellitus, severe acute respiratory syndrome coronavirus 2 infection, multisystem inflammatory syndrome in children, and long coronavirus disease, further highlight the potential of NVC to reflect systemic endothelial dysfunction and non-specific microvascular injury, although these applications remain exploratory. Despite its promise, several limitations continue to restrict the broader clinical application of NVC. Pediatric reference data remain heterogeneous, scoring systems are largely adapted from adult protocols, and longitudinal evidence linking capillaroscopic changes to disease progression, treatment response, or long-term outcomes remains limited. Standardized image acquisition and reporting, multicenter cohort studies, and validation of automated image-analysis techniques are essential next steps for translating descriptive observations into clinically actionable information. This review summarizes the contribution of NVC to pediatric rheumatology and explores potential areas for future expansion. The strongest indications remain RP and scleroderma-spectrum disorders, whereas the most promising emerging application is in JDM, where microvascular changes correlate with disease activity. Across other pediatric rheumatic diseases, NVC currently serves as a complementary descriptive tool for vascular phenotyping rather than a standalone diagnostic modality. With the development of age-specific reference standards, harmonized protocols, and longitudinal validation studies, NVC has the potential to evolve from a descriptive imaging technique into an integral component of risk stratification and disease monitoring in pediatric rheumatology.

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INTRODUCTION

Nailfold videocapillaroscopy (NVC) is a non-invasive imaging technique that enables *in vivo* assessment of the peripheral microcirculation within the nailfold.[1] Because nailfold capillaries run parallel to the skin surface, their morphology and architecture can be visualized in detail. Consequently, the nailfold serves as an accessible window for evaluating systemic microvascular status, particularly in diseases characterized by endothelial injury, vasculopathy, and immune-mediated vascular remodeling.[2]

The role of capillaroscopy in rheumatology has been largely established through its application in Raynaud phenomenon (RP) and systemic sclerosis (SSc). In patients presenting with RP, the detection of a scleroderma pattern provides an important clue for distinguishing primary RP from secondary RP associated with SSc or other scleroderma-spectrum disorders.[3] Furthermore, the inclusion of abnormal nailfold capillary findings consistent with a scleroderma pattern in the 2013 American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) classification criteria for SSc further reinforced the role of NVC in rheumatologic practice.[4] The scleroderma pattern is characterized by enlarged or giant capillaries, capillary loss, microhemorrhages, disruption of capillary architecture, and ramified capillaries indicative of neoangiogenesis. In the simplified fast-track assessment, the presence of giant capillaries or markedly reduced capillary density, particularly ≤ 3 capillaries/mm, supports the diagnosis of a scleroderma pattern.[3] This pattern-based approach has enhanced the clinical utility of NVC, particularly in the early recognition and risk stratification of SSc.

Interpretation of NVC findings in children requires additional caution because the pediatric nailfold microvascular architecture is not simply a smaller version of that observed in adults. Capillary density, dimensions, and morphological characteristics vary throughout childhood, and findings considered abnormal in adults may represent non-specific variants in healthy children.[2,5] Therefore, pediatric NVC assessment should be based on standardized image acquisition protocols, consistent terminology, and age-appropriate interpretation rather than isolated capillaroscopic findings.

Over the past decade, NVC has been increasingly incorporated into pediatric rheumatology. Its most established indications in children are RP and juvenile SSc; however, growing evidence supports its application in childhood-onset systemic lupus erythematosus (SLE), juvenile dermatomyositis (JDM), juvenile idiopathic arthritis (JIA), vasculitic diseases, autoinflammatory disorders, and selected non-rheumatologic conditions.[6] This state-of-the-art review aims to synthesize current evidence regarding the role of NVC in pediatric RP and scleroderma-spectrum disorders, evaluate its emerging applications across other childhood rheumatic diseases, identify existing methodological gaps, and outline future priorities for standardized and technology-supported implementation.[7]

LITERATURE SEARCH STRATEGY

This article is presented as a narrative review rather than a systematic review or meta-analysis. A structured literature search was conducted to identify relevant studies on NVC in pediatric rheumatology. PubMed/MEDLINE was searched using combinations of the terms “nailfold capillaroscopy,” “nailfold videocapillaroscopy,” “children,” and “pediatric.” In addition, the reference lists of key review articles, consensus statements, and original research papers were manually screened to identify additional relevant publications. Priority was given to pediatric studies, international standardization papers, consensus recommendations, systematic reviews, and recent investigations providing clinically relevant data. Adult studies were included when pediatric evidence was limited or when they provided essential background information for established concepts. The final selection of references was based on clinical relevance, methodological rigor, recency, and applicability to pediatric rheumatology.

TECHNICAL AND METHODOLOGICAL CONSIDERATIONS IN CHILDREN

Nailfold capillaroscopy can be performed using several devices, including an ophthalmoscope, dermoscope, smartphone attachments, USB microscope, stereomicroscope, and digital videocapillaroscope. Among these modalities, digital videocapillaroscopy is considered the gold standard because it provides high-resolution images, enables image storage, and allows quantitative assessment of capillary density and dimensions.[2] Dermoscopy is a more practical and accessible screening tool; however, it is inferior to NVC in terms of image quality and the ability to classify capillary abnormalities. When dermoscopic findings are inconclusive or there is clinical suspicion of a scleroderma-spectrum disorder, further evaluation with NVC is recommended.

Image acquisition in pediatric NVC should follow a standardized protocol to minimize false-positive findings and improve reproducibility. Ideally, the examination should be performed in a room maintained at approximately 21-22 °C, with an adequate acclimatization period before image acquisition to stabilize peripheral circulation. Hands should be clean, and nail polish should be removed if it interferes with image quality. In addition, recent manicures should be avoided. In children, factors such as nail biting, minor trauma, sports activities, manual hobbies, and the use of vasoactive medications should be considered, as they may influence capillary morphology and peripheral perfusion.[2]

Standard assessment typically includes eight fingers, excluding both thumbs. Although data regarding the minimum number of fingers required for reliable assessment in children are limited, adult studies suggest that examination of fewer than eight fingers may reduce the sensitivity for detecting capillary abnormalities.[8] When time constraints exist, evaluation of both ring fingers may serve as a practical alternative; however, it should not replace the standard eight-finger assessment when diagnostic accuracy is a priority. Most studies use 200× magnification, and an immersion fluid, commonly cedar oil or another vegetable oil, is applied to enhance image quality.[8] The probe should be positioned gently to avoid compression of capillary blood flow, which may create the artificial appearance of avascular areas.

Consistent terminology is essential to ensure comparability across studies.[9] The EULAR Study Group on Microcirculation in Rheumatic

Diseases recommends that capillaroscopic assessment be based on four principal variables: capillary density, apical diameter, capillary morphology, and microhemorrhages.[10] Capillary density should be reported as the number of capillaries per linear millimeter in the distal row. For the assessment of capillary size, apical diameter is the preferred parameter because measurements of afferent and efferent limb widths or intercapillary distance may be subject to greater interindividual variability.

PEDIATRIC NORMAL VALUES AND AGE-RELATED VARIABILITY

Accurate interpretation of NVC in children depends on a thorough understanding of normal pediatric nailfold findings. Pediatric capillaries may vary with age in terms of density, diameter, length, and morphology.[2,5] Therefore, NVC findings in children should not be interpreted solely on the basis of adult reference values, as age-related physiological variability must be taken into account.

Capillary density and apical diameter are key quantitative parameters in capillaroscopic assessment. In adults, reduced capillary density, particularly when accompanied by giant capillaries, microhemorrhages, and architectural disorganization, is a characteristic feature of the scleroderma pattern.[3] In children, however, capillary density may exhibit physiological variation according to age and developmental stage. In contrast, technical factors such as image quality, magnification, probe pressure, finger selection, ambient temperature, and motion artifacts primarily influence the accuracy and reproducibility of capillary density measurements. Consequently, mildly reduced capillary density should be interpreted with caution unless it is consistently observed across multiple fingers and accompanied by other pathological findings. Likewise, the presence of giant capillaries is a strong indicator of scleroderma-type microangiopathy in an appropriate clinical context, whereas mild capillary dilatation alone is less specific.[6]

Developmental stage is another important consideration in pediatric NVC interpretation. Throughout childhood and adolescence, age-related changes in capillary density, diameter, length, and morphology, together with variations in finger size and nailfold visibility, may influence quantitative capillary assessment.[2,5,6] Although pubertal hormonal changes may theoretically contribute to peripheral vascular variability, pediatric NVC data stratified by pubertal stage remain limited. Therefore, adult thresholds and pattern-recognition criteria should not be applied directly to pediatric populations. Instead, interpretation should rely on age-appropriate reference ranges, standardized image acquisition, and longitudinal assessment, particularly when findings are mild, isolated, or of uncertain clinical significance.[2,5,6]

Morphological findings require particular caution when interpreting NVC in pediatric populations. Tortuous and crossing capillaries may be observed in healthy children and should not automatically be regarded as disease-specific abnormalities.[11] Similarly, isolated microhemorrhages may result from local trauma, nail biting, or technical artifacts. In contrast, giant capillaries, significant capillary loss, numerous microhemorrhages, and progressive architectural disorganization are generally considered more clinically meaningful findings and, when interpreted within the appropriate clinical context, may indicate underlying microangiopathy. Examples of normal pediatric NVC findings, including regular hairpin-shaped capillaries and preserved capillary density, are presented in Figure 1.

The concept of a non-specific pattern is particularly important in pediatric NVC. This category encompasses findings that are not entirely normal but do not meet the criteria for a scleroderma pattern. In many pediatric rheumatic diseases, microvascular abnormalities may be present without a disease-specific capillaroscopic signature.[11] The use of this classification helps prevent overinterpretation of mild or isolated abnormalities in children who lack clinical evidence of connective tissue disease (CTD). Overall, NVC findings in children should be interpreted in conjunction with clinical symptoms, physical examination findings, autoantibody profiles, disease phenotype, and longitudinal follow-up. An overview of the principal NVC findings reported across pediatric rheumatic diseases and their clinical relevance is provided in Table 1.

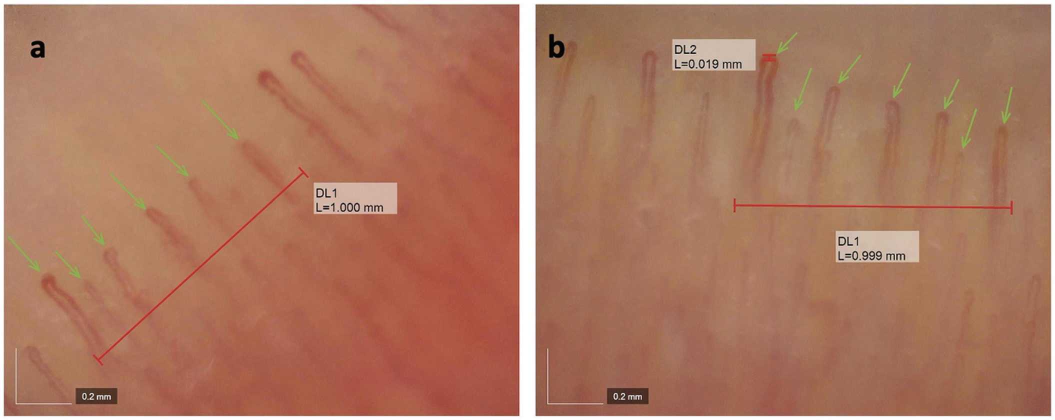

FIG. 1. Normal nailfold capillaroscopy findings in a healthy 13-year-old child. Images were obtained using a Dino-Lite digital capillaroscope at 200× magnification. (a) Capillary loops with normal hairpin morphology and preserved density are shown by green arrows. (b) Capillary loops with normal apical diameter (0.019 mm = 19 μm) and preserved density are shown by green arrows.

TABLE 1. Reported Nailfold Videocapillaroscopy Findings in Pediatric Rheumatic Diseases and Related Rheumatologic Conditions.

Disease/condition	Main reported NVC findings	Clinical interpretation/relevance	Key references
Raynaud phenomenon/ primary RP	• Increased apical diameter • Dilated and tortuous capillaries • Microhemorrhages • Density preserved	NVC is useful for distinguishing low-risk primary RP from children requiring closer evaluation for connective tissue disease, especially when interpreted with ANA, disease-specific autoantibodies, and clinical features.	3,12-15
Juvenile systemic sclerosis/ scleroderma-spectrum disorders	• Giant capillaries • Capillary loss • Avascular areas • Microhemorrhages • Neoangiogenesis	Core indication for NVC in pediatric rheumatology. Supports recognition of scleroderma-spectrum microangiopathy, diagnostic assessment, and longitudinal microvascular follow-up.	3,4,6,16,17
Juvenile localized scleroderma	• Morphological changes • Increased density, length, and capillary dimensions • Reduced intercapillary distance • No giant capillaries or avascular areas	May demonstrate microvascular involvement in localized disease, but findings should not be interpreted as a classic systemic sclerosis pattern. Current role remains exploratory.	24
Juvenile dermatomyositis	• Reduced density • Giant or bushy capillaries • Bizarre shapes • Microhemorrhages • Architectural disorganization	Reflects JDM-related microangiopathy and may complement assessment of vascular and cutaneous activity. Longitudinal changes in density, dilatation, and morphology may support follow-up.	25-28
Childhood-onset systemic lupus erythematosus	• Tortuous and crossing capillaries • Dilated capillaries • Microhemorrhages • Reduced density	Findings are heterogeneous and should not be used alone to define overlap disease. NVC may contribute to vascular phenotyping, particularly in patients with RP, digital symptoms, or suspected overlap features.	35,38
Juvenile Sjögren disease	• Reduced density • Dilated, bushy, meandering, and bizarre capillaries • Neoangiogenesis • Microhemorrhages	May reflect non-specific microvascular involvement accompanying glandular and systemic autoimmunity. Potentially useful for exploring links between glandular disease and microvascular changes.	36,39
Mixed connective tissue disease/ overlap syndromes	• Normal • Non-specific pattern • Scleroderma-like microangiopathy	NVC may help characterize vascular phenotype and identify scleroderma-spectrum features when RP, puffy fingers, digital ulcers, skin thickening, or disease-specific autoantibodies are present.	37,40
Kawasaki disease	• Irregular dilatation • Reduced density • Architectural disruption	May reflect systemic vascular inflammation and has been correlated with coronary involvement in limited data. It should not replace echocardiography and remains an exploratory adjunct.	41
Behçet disease	• Dilated and tortuous capillaries • Giant loops • Microhemorrhages • Neoangiogenesis • Reduced density • Reduced arterial/venous diameters, and length • Increased intercapillary distance and width	May show measurable microvascular changes even without overt vascular involvement. Potential role in phenotypic characterization, but not diagnostic as a standalone tool.	42-44
Deficiency of adenosine deaminase 2	• Non-specific dilatations • No giant capillaries • Density preserved	Does not currently provide a disease-specific diagnostic pattern. May be useful as an exploratory tool in monogenic vasculopathies.	45
Antineutrophil cytoplasmic antibody-associated vasculitis	• Microhemorrhages • Capillary loss • Neoangiogenesis • Tortuous or bizarre capillaries • Branching and morphological changes	Findings are variable and influenced by disease activity, treatment, selection, and comorbidities. Current role is descriptive and research oriented.	46,47

TABLE 1. Continued

Disease/condition	Main reported NVC findings	Clinical interpretation/relevance	Key references
Immunoglobulin A vasculitis	• Reduced density • Irregular architecture • Microhemorrhages, macrohemorrhages • Periungual edema	May reflect acute small-vessel inflammation and residual microcirculatory changes, but findings are not specific and do not show consistent clinical correlations.	48,49
Juvenile idiopathic arthritis	• Normal • Non-specific findings	No validated JIA-specific NVC pattern. Its use is mainly limited to selected cases with vascular or connective tissue disease features.	6,11
Juvenile psoriatic arthritis/psoriatic arthritis spectrum	• Tortuous and crossing capillaries • Microhemorrhages • Reduced density • Dilatation • Irregular capillaries • Neoangiogenesis	May reflect microvascular involvement of the nail-enthesis complex. More informative when integrated with nail ultrasonography, enthesitis assessment, and longitudinal follow-up.	50-52
Familial Mediterranean fever/ autoinflammatory diseases	• Tortuosity • Microhemorrhages • Density changes	Current evidence supports NVC as a research tool rather than a routine clinical method. Findings should not be interpreted as FMF-specific microangiopathy.	53,54

ANA, antinuclear antibody; ANCA, antineutrophil cytoplasmic antibody; FMF, familial Mediterranean fever; IgA, immunoglobulin A; JDM, juvenile dermatomyositis; JIA, juvenile idiopathic arthritis; NVC, nailfold videocapillaroscopy; RP, Raynaud phenomenon.

RAYNAUD PHENOMENON

RP is one of the most well-established indications for NVC in pediatric rheumatology.[3,12] Clinically, the primary objective is not merely to identify RP but to distinguish primary RP from secondary RP associated with evolving CTDs. This distinction is important because RP may represent the initial manifestation of scleroderma-spectrum disorders, mixed CTD, SLE, or other CTDs.[13] Therefore, children presenting with RP should be evaluated using a combination of clinical history, physical examination, autoantibody testing, and NVC.

The diagnostic value of NVC in RP lies in its ability to detect structural microvascular changes before systemic disease becomes clinically evident. In adult cohorts, the coexistence of abnormal nailfold capillaries and SSc-associated autoantibodies is strongly associated with an increased risk of progression to SSc.[14] Conversely, normal capillaroscopic findings reduce the likelihood of scleroderma-spectrum disease, particularly in the absence of additional clinical warning signs. However, these adult-derived observations cannot be directly extrapolated to children, as physiological variability and non-specific findings are more common in the pediatric population.[13,15]

Pediatric studies indicate that children with primary RP may exhibit microvascular differences compared with healthy controls. In a prospective study using the standardized EULAR microcirculation protocol, children with primary RP more frequently demonstrated increased apical diameter, dilated capillaries, abnormal capillary morphology, microhemorrhages, tortuous capillaries, and crossing capillaries than healthy controls.[15] The most common overall pattern in these patients was non-specific, whereas a normal pattern predominated in healthy controls. Importantly, markedly reduced capillary density did not differ significantly between the primary RP group and controls.[15] Thus, primary RP in children is not necessarily associated with a completely normal capillaroscopic profile.

The most important warning sign in a child with RP is a scleroderma pattern. Giant capillaries, marked capillary loss, avascular areas, and architectural disorganization should prompt careful evaluation for scleroderma-spectrum disease. However, pediatric evidence suggests that isolated giant capillaries or early scleroderma-like changes may not always be persistent or progressive. Therefore, diagnostic classification should not rely on a single examination alone; instead, reproducibility, clinical correlation, autoantibody profile, and longitudinal follow-up should be considered. The Pediatric Rheumatology European Society Juvenile Scleroderma Working Group recommends antinuclear antibody (ANA) testing and NVC in all children presenting with RP, with follow-up intensity guided by ANA status, disease-specific autoantibodies, and capillaroscopic abnormalities.[13]

JUVENILE SYSTEMIC SCLEROSIS AND SCLERODERMA-SPECTRUM DISORDERS

Juvenile systemic sclerosis (jSSc) is one of the pediatric rheumatic diseases in which NVC has the clearest diagnostic and clinical utility.[3] In SSc, microvascular damage represents one of the earliest and most fundamental pathological features, and NVC enables this process to be assessed directly, non-invasively, and repeatedly over time. Following the incorporation of abnormal nailfold capillary findings into the 2013 ACR/EULAR classification criteria for SSc, NVC has transitioned from an ancillary imaging tool to a core component of the diagnostic pathway.[4]

Recognition of the scleroderma pattern is central to capillaroscopic evaluation in SSc and scleroderma-spectrum disorders. This pattern is characterized by giant capillaries, reduced capillary density, microhemorrhages, disruption of capillary architecture, avascular areas, and ramified or abnormally shaped capillaries reflecting neoangiogenesis. In the classical classification, the scleroderma pattern is subdivided into early, active, and late phases. The early pattern typically shows preserved capillary density with giant capillaries and few microhemorrhages; the active pattern is characterized by frequent giant capillaries, microhemorrhages, and progressive capillary loss; and the late pattern demonstrates severe capillary loss, avascular areas, architectural disorganization, and prominent neoangiogenesis.[3] Figure 2 illustrates typical scleroderma pattern findings in an 11-year-old patient with juvenile SSc, including bushy capillaries reflecting neoangiogenesis, microhemorrhages, and avascular areas.

The fast-track algorithm provides a practical approach for distinguishing scleroderma and non-scleroderma patterns in routine clinical practice. According to this algorithm, images with preserved capillary density and absence of giant capillaries are considered non-scleroderma patterns, whereas the presence of giant capillaries or markedly reduced capillary density, together with abnormal capillary morphology, supports a scleroderma pattern.[16] This approach demonstrates high interobserver agreement across different levels of examiner experience and facilitates pattern-based decision-making. In pediatric populations, however, technical quality, age-related variability, and non-specific morphological findings must be carefully considered, and interpretation should rely on the overall capillaroscopic pattern rather than isolated abnormalities.[6]

NVC is also important for the very early recognition of scleroderma-spectrum disease. The Very Early Diagnosis of Systemic Sclerosis (VEDOSS) framework, developed through an EUSTAR Delphi consensus, identifies RP, puffy fingers, and ANA positivity as level-1 red flags, while SSc-specific autoantibodies and abnormal nailfold capillaroscopy are considered level-2 features that further strengthen diagnostic suspicion.[17] In a subsequent 5-year VEDOSS registry analysis, ANA negativity was associated with a low-risk of progression, whereas the combination of SSc-specific autoantibodies and puffy fingers conferred the highest risk.[14] These findings are derived from adult cohorts and should therefore be applied to jSSc only as a conceptual framework.[14,17]

Because jSSc is rare, pediatric NVC data remain limited compared with the adult SSc literature. Available studies suggest that the scleroderma pattern is frequently observed in jSSc and may resemble the microangiopathic changes seen in adult disease.[6] Therefore, NVC should be regarded as an important tool for diagnostic assessment and follow-up in children with suspected jSSc, although adult-based classification and prognostic data should be interpreted with caution.[6]

Adult SSc studies also support the prognostic value of NVC.[18,19] Reduced capillary density, active or late scleroderma patterns, and progressive capillary loss have been associated with skin fibrosis, digital ulcers, calcinosis, pulmonary hypertension, interstitial lung disease (ILD), gastrointestinal involvement, and overall disease progression.[19-23] Irregular capillary architecture and late NVC patterns have also been linked to declining pulmonary function and progression of ILD, whereas changes in capillary density may reflect disease course and treatment response in SSc-ILD cohorts.[20,21] In addition, diffuse cutaneous SSc tends to exhibit more pronounced capillary loss and late pattern features than limited cutaneous disease.[22] Collectively, these findings suggest a relationship between microvascular damage and the severity of cutaneous and systemic involvement, although pediatric longitudinal validation remains lacking.

Juvenile localized scleroderma (JLoS) is traditionally considered distinct from SSc because it primarily affects the skin and subcutaneous tissues; however, emerging evidence suggests measurable microvascular involvement in children. In a recent case-control study, patients with JLoS demonstrated more frequent capillary tortuosity, crossing, dilatation, and neoangiogenesis compared with age-matched controls, along with increased capillary density, capillary length, arterial limb width, apical loop width, and disorganization scores. Intercapillary distance was reduced relative to controls. Neither giant capillaries nor avascular areas were

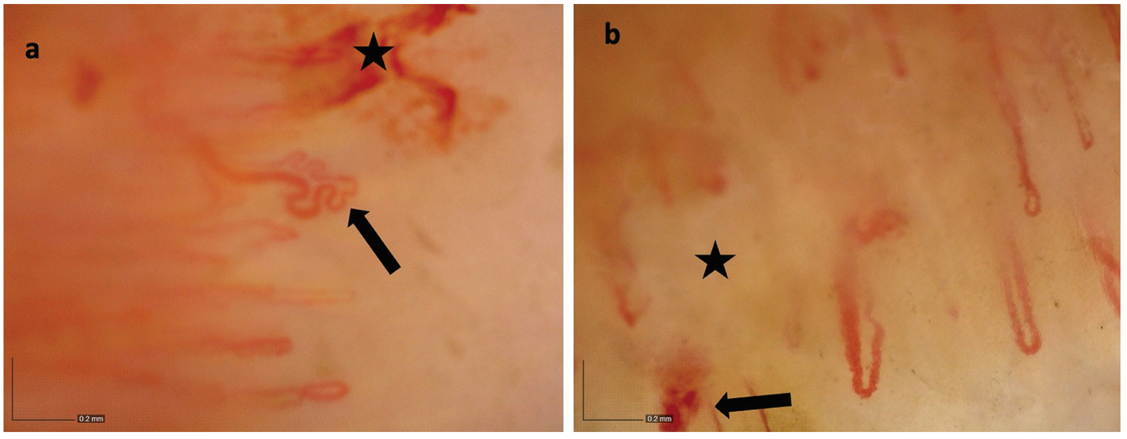

FIG. 2. Nailfold capillaroscopy images from an 11-year-old patient with systemic sclerosis at 200× magnification. (a) Bushy capillary/neoangiogenesis is shown by the arrow, and microhemorrhage is shown by the star. (b) Microhemorrhage is shown by the arrow, and an avascular area is shown by the star.

observed; therefore, these findings were interpreted not as a classic scleroderma pattern but as an early non-scleroderma microvascular pattern.[24] NVC may therefore be useful for characterizing microvascular changes in JLoS; however, larger longitudinal studies are required before its routine diagnostic or prognostic use can be recommended.

JUVENILE DERMATOMYOSITIS

JDM is one of the most clinically relevant non-scleroderma pediatric rheumatic diseases for NVC assessment, as microangiopathy plays a central role in its pathogenesis.[25,26] The disease is an immune-mediated disorder affecting the skin, muscle, and systemic vasculature. Endothelial injury and vasculopathy contribute to its clinical manifestations and long-term outcomes.

NVC has long been used as a supportive tool for demonstrating peripheral microvascular abnormalities in JDM. Early studies described reduced capillary density, while subsequent investigations showed that nailfold abnormalities may persist over time and are particularly associated with cutaneous involvement.[26] Commonly reported NVC findings include reduced capillary density, dilated or giant capillaries, bushy or ramified capillaries, bizarre capillary morphology, microhemorrhages, and disorganization of capillary architecture.[25,27,28] These findings do not necessarily correspond to the classic SSc pattern; rather, they reflect active microvascular remodeling and capillary injury in a distinct clinical and pathogenetic context.

Among NVC parameters, capillary density is particularly important. Evidence suggests that reduced capillary density may be associated with cutaneous disease activity as well as patient-reported and physician-assessed global disease activity scores.[27-29] Changes in capillary density and apical diameter over time may also help predict cutaneous disease activity.[28,30,31] In contrast, the relationship between NVC findings and muscle involvement appears more variable and less consistent.[32] Therefore, NVC should not replace muscle strength assessment, serum muscle enzymes, or validated disease activity indices but should instead be interpreted as a complementary tool for evaluating vascular and cutaneous disease activity.

The current clearest role of NVC in JDM is longitudinal follow-up rather than primary diagnosis.[26,28] Changes in capillary density, dilatation, and abnormal morphology may complement clinical examination and laboratory findings, and nailfold capillaroscopy is therefore included among baseline and follow-up assessments in the Childhood Arthritis and Rheumatology Research Alliance biologic consensus treatment plans.[33] Beyond disease monitoring, preliminary data suggest clinical heterogeneity within JDM. For example, anti-transcription intermediary factor 1-γ (anti-TIF1-γ)-positive patients have been reported to exhibit lower capillary density and wider apical diameter compared with other myositis-specific autoantibody subgroups.[27] NVC findings in an adolescent with anti-TIF1-γ-positive JDM, including giant capillaries, microhemorrhages, tortuosity, and avascular areas, are shown in Figure 3.

However, the supporting evidence remains limited and heterogeneous. Most studies involve small cohorts and vary with respect to disease duration, treatment exposure, imaging devices, and scoring methodologies.[25,34] To date, no JDM-specific capillaroscopic pattern has been defined or validated, and assessment continues to rely on frameworks originally developed for SSc. Whether NVC parameters can predict disease flares, treatment response, calcinosis, ulceration, or long-term damage remains to be determined in larger, prospective cohort studies.

CHILDHOOD-ONSET SLE, JUVENILE SJÖGREN DISEASE, MIXED CTD, AND OVERLAP SYNDROMES

NVC abnormalities have been reported in childhood-onset SLE, juvenile Sjögren disease, mixed connective tissue disease (MCTD), and overlap syndromes; however, these findings are less disease-specific than those observed in SSc.[35-37] In these conditions, NVC should primarily be considered a complementary tool reflecting immune-mediated endothelial injury, vascular inflammation, microvascular remodeling, or overlap features within the scleroderma spectrum, rather than a primary diagnostic modality.

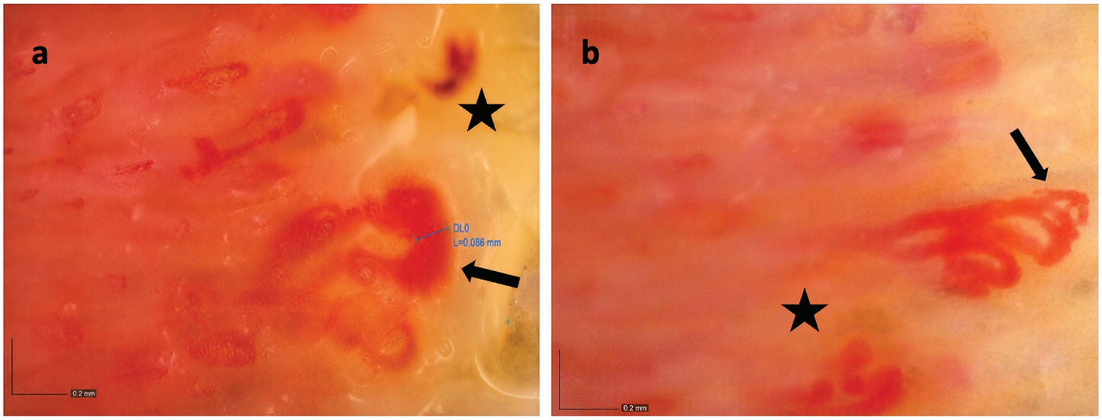

FIG. 3. Nailfold capillaroscopy images from a 15-year-old patient with anti–TIF1-γ-positive juvenile dermatomyositis at 200× magnification. (a) Giant capillary (0.086 mm = 86 μm) is shown by the arrow, and microhemorrhage is shown by the star. (b) Tortuous capillary is shown by the arrow, and an avascular area is shown by the star.

In SLE, both adult and pediatric studies have described tortuous and crossing capillaries, microhemorrhages, abnormal capillary morphology, reduced capillary density, dilated capillaries, and, in some cases, scleroderma-like changes.[35,38] Recent multicenter adult SLE studies indicate that most patients exhibit a normal NVC pattern, whereas a smaller subset demonstrates more pronounced abnormalities, including enlarged or giant capillaries, microhemorrhages, irregular nailfold architecture, and reduced capillary density. This more severe capillaroscopic profile has been associated in some studies with higher disease activity, arthritis, renal involvement, and glucocorticoid use.[38] However, pediatric SLE findings remain heterogeneous, and adult data should not be directly extrapolated to children. In particular, scleroderma-like findings do not necessarily indicate SSc overlap. Microhemorrhages may be relevant in childhood-onset SLE; however, potential confounding factors such as trauma and technical artifacts should always be considered.

Data on juvenile Sjögren disease are more limited, although pediatric evidence is gradually emerging. In a recent study, children with juvenile Sjögren disease demonstrated lower capillary density and more frequent dilated, bushy, meandering, and bizarre capillaries, along with neoangiogenesis and microhemorrhages, compared with healthy controls.[36] All patients exhibited at least two minor morphological abnormalities; however, no scleroderma pattern was observed, consistent with the non-specific microvascular profile reported in adult Sjögren cohorts.[39]

The biological rationale for NVC is stronger in MCTD, as RP and scleroderma-spectrum microangiopathy are more frequently observed within its clinical phenotype. Capillaroscopic findings in MCTD may range from normal patterns to non-specific changes and scleroderma-like microangiopathy.[37] Adult data suggest that a scleroderma-like pattern may be relatively frequent and has been associated in some studies with ILD, pulmonary hypertension, or a more severe clinical course.[40] However, these associations are not consistently demonstrated, and no specific NVC pattern unique to MCTD has been established to date.

Overlap syndromes require careful interpretation. When RP, disease-specific autoantibodies, puffy fingers, skin thickening, or digital ulcers are present, NVC findings may strengthen the suspicion of scleroderma-spectrum involvement.[11,37] In contrast, isolated non-specific abnormalities in the absence of compatible clinical features are insufficient to support an overlap diagnosis. In children, tortuosity, capillary crossing, and limited microhemorrhages may reflect physiological variation, minor trauma, or technical artifacts.

Thus, NVC in these conditions is best regarded as a tool for vascular phenotyping when integrated with clinical examination, autoantibody profiles, symptomatology, glandular involvement, digital manifestations, and longitudinal changes over time.

VASCULITIDES

Childhood vasculitides share vascular inflammation and endothelial injury as core pathogenic features, making the peripheral microcirculation a plausible target for capillaroscopic assessment. However, no disease-specific or validated capillaroscopic pattern has been established for pediatric vasculitis, and existing studies are largely exploratory. Accordingly, NVC is best regarded in this group as a descriptive tool for documenting microvascular involvement rather than a diagnostic instrument.

Kawasaki disease (KD) is of particular interest because it is an acute systemic vasculitis of childhood with major clinical relevance due to coronary artery involvement. In a pediatric case-control study, patients with KD showed more frequent capillary diameter abnormalities, irregular dilatation, reduced capillary density, and architectural disruption than healthy controls. A positive correlation between abnormal capillaroscopic findings and coronary involvement suggests that peripheral microvascular changes may reflect systemic vascular inflammation.[41] Nevertheless, these findings are not specific to KD, and current evidence does not support NVC as a routine diagnostic or prognostic tool. It should not replace echocardiography but may serve as a complementary exploratory method for assessing microvascular involvement.

Behçet disease (BD) is another notable example among pediatric vasculitides. This multisystem disorder may present with mucocutaneous, ocular, articular, gastrointestinal, vascular, and neurological manifestations, with endothelial dysfunction playing a central role in its pathogenesis. Adult studies have reported dilated and tortuous capillaries, giant capillary loops, microhemorrhages, and neoangiogenesis, although no disease-specific signature has been identified.[42,43] A recent pediatric study using the standardized EULAR microcirculation protocol provided the first systematic characterization of NVC findings in childhood-onset BD.[44] Children with the complete phenotype showed more pronounced microvascular alterations than those with the incomplete phenotype, including lower capillary density and higher scores for tortuous, dilated, and abnormal capillaries. Compared with age-matched healthy reference data, the patient cohort also demonstrated reduced arterial and venous diameters and shorter capillary length, along with increased intercapillary distance and capillary width.[44] Notably, microvascular changes were detectable even in patients without clinically overt vascular involvement and did not correlate with global disease activity scores, suggesting that NVC may reflect cumulative vascular burden rather than current inflammatory activity. Representative microvascular changes in an adolescent with BD, including dilated capillaries, microhemorrhage, and abnormally shaped capillaries, are illustrated in Figure 4.

Deficiency of adenosine deaminase 2 (DADA2) is a monogenic autoinflammatory vasculopathy characterized by systemic inflammation, vasculitis, livedo racemosa, digital ischemia, RP, and early-onset stroke. In the first case-control study of NVC in DADA2, all patients exhibited non-specific NVC changes, particularly capillary dilatations; however, capillary density was normal, giant capillaries were absent, and no scleroderma pattern was detected.[45] No significant differences were observed between DADA2 patients, primary RP patients, and healthy controls. Interpretation of these findings is limited by the rarity of DADA2, small sample size, and clinical heterogeneity. Therefore, current evidence supports NVC as an exploratory method for describing non-specific microvascular changes rather than as a tool for diagnostic discrimination or disease monitoring in DADA2.

Evidence on antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAV) is increasing but remains insufficient for routine clinical application. An adult study described microhemorrhages, capillary loss, neoangiogenesis, tortuous and bizarre capillaries, and architectural disruption, with possible associations with skin, lung, and kidney involvement as well as reduction in abnormalities after immunosuppressive therapy.[46] However, available adult data are not entirely consistent, suggesting that disease activity, treatment status, patient selection, and comorbidities may influence findings. Pediatric data are limited; in a small childhood AAV series, typical adult abnormalities such as dilated capillaries, capillary loss, and microhemorrhages were not observed, although branching and other morphological changes were noted in some patients.[47] The discrepancy between adult and pediatric observations may reflect differences in disease duration, organ involvement, treatment exposure, timing of examination, and capillaroscopy acquisition protocols. Standardized longitudinal studies are required before NVC can be used to assess vascular damage, treatment response, or relapse risk in pediatric AAV. Figure 5 shows NVC images from a 10-year-old patient with ANCA-AAV, illustrating preserved capillary morphology as well as areas of increased tortuosity and abnormally shaped capillaries.

Immunoglobulin A vasculitis is theoretically relevant to NVC because it is the most common small-vessel vasculitis of childhood. Pediatric studies have reported reduced capillary density, altered capillary length and morphology, irregular microvascular architecture, microhemorrhages, occasional macrohemorrhages, and periungual edema during the acute phase.[48] A subsequent longitudinal study extended these observations by reassessing children at six months. Most morphological abnormalities had resolved; however, periungual edema persisted in all patients, suggesting incomplete microvascular recovery despite clinical improvement.[49] Capillaroscopic findings did not correlate with laboratory parameters or clinical outcomes in either study, and overall, NVC in pediatric vasculitides should currently be viewed as a descriptive and research-oriented tool rather than a validated diagnostic test.

In childhood inflammatory arthritis, NVC findings are generally far less specific than in scleroderma-spectrum diseases.

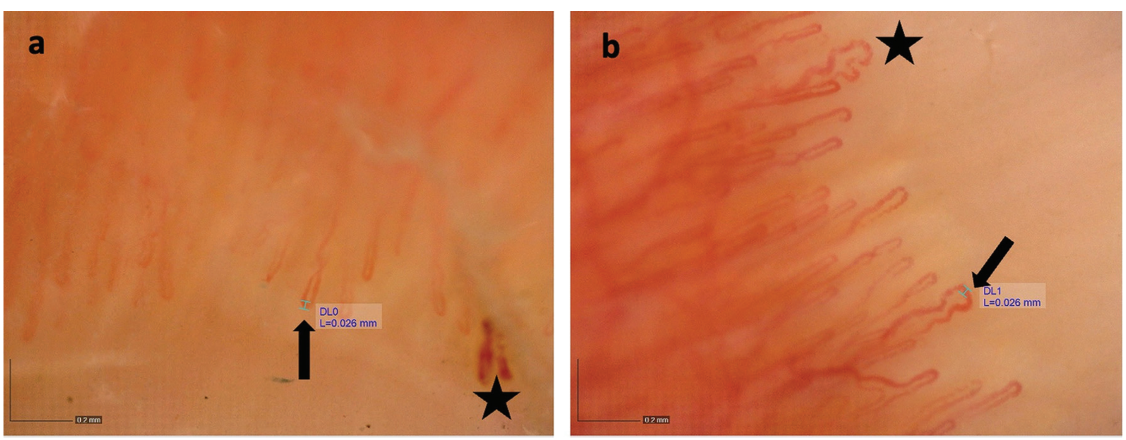

FIG. 4. Nailfold capillaroscopy images from a 16-year-old patient with Behçet disease at 200× magnification. (a) Dilated capillary (0.026 mm = 26 µm) is shown by the arrow, and microhemorrhage is shown by the star. (b) Dilated capillary (0.026 mm = 26 µm) is shown by the arrow, and abnormally shaped capillaries are shown by the star.

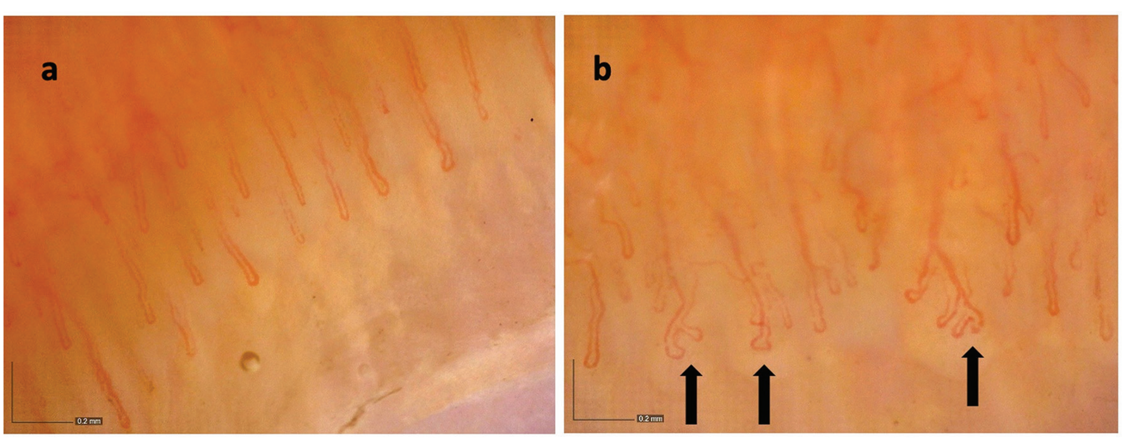

FIG. 5. Nailfold capillaroscopy images from a 10-year-old patient with ANCA-associated vasculitis at 200× magnification. (a) Normal capillary morphology with preserved density. (b) Increased capillary tortuosity and abnormally shaped capillaries are shown by arrows.

In JIA, capillaroscopic changes may be absent, mild, or non-specific, and no validated NVC pattern exists for routine diagnosis or classification of JIA subtypes.[6,11] This low specificity is clinically important because mild tortuosity, capillary crossing, or isolated microhemorrhages may also be observed in healthy children or in other inflammatory conditions. Therefore, in JIA, NVC should not be used for subtype classification, disease activity assessment, or treatment monitoring, except when vascular symptoms or CTD overlap is suspected.

Juvenile psoriatic arthritis (JPsA) has become an area of interest because of the close anatomical and biological relationship between the nail-enthesis complex, nailfold microcirculation, psoriasis, and psoriatic arthritis (PsA). Pediatric data on JPsA remain limited but noteworthy. In a recent cross-sectional study of children with JPsA and healthy controls, patients more frequently exhibited tortuous capillaries, capillary crossing, and microhemorrhages.[50] The increased frequency of tortuosity and microhemorrhages suggests possible microvascular involvement; however, no disease-specific capillaroscopic pattern was identified. Capillary density and apical diameter did not differ significantly between patients and controls, and NVC parameters were not associated with disease activity. A negative correlation between disease duration and capillary density raised the possibility that microvascular changes may become more evident over time.[50]

Adult PsA studies similarly report irregular and tortuous capillaries, reduced capillary density, dilated capillaries, microhemorrhages, and neoangiogenesis, although marked methodological heterogeneity limits comparability.[51] Combined ultrasound and NVC assessment of the nail-enthesis complex in adults suggests a continuum of microvascular and structural changes between psoriasis and PsA.[52] Therefore, in children with JPsA, NVC is best interpreted as a complementary tool for exploring nailfold microvascular involvement rather than as a diagnostic or disease activity marker.

FAMILIAL MEDITERRANEAN FEVER (FMF) AND OTHER AUTOINFLAMMATORY DISEASES

Autoinflammatory diseases remain an exploratory area for NVC in pediatric rheumatology. FMF is the prototype monogenic autoinflammatory disease, characterized by recurrent inflammatory attacks, serositis, arthritis, and an acute-phase response. Interest in NVC in FMF is based on the hypothesis that repeated systemic inflammation may be associated with endothelial activation and microvascular changes.[53,54] Nevertheless, nailfold capillary tortuosity, microhemorrhages, or mild capillary density changes should not be interpreted as FMF-specific microangiopathy. Current evidence supports the use of NVC in FMF as a research tool rather than a routine clinical diagnostic modality.[53,54] A major limitation of the current FMF literature is that reported abnormalities are mild and non-specific, and consistent associations with genotype, inflammatory burden, attack frequency, colchicine response, or vascular outcomes have not yet been established. These findings should therefore be interpreted as hypothesis-generating rather than as evidence of an FMF-specific capillaroscopic phenotype.

SELECTED NON-RHEUMATOLOGIC DISEASES

Although this review focuses on pediatric rheumatology, studies in non-rheumatologic diseases suggest that NVC may have broader potential for detecting systemic microvascular dysfunction. Diabetes mellitus has received the most attention in this regard.[55-57] A recent meta-analysis confirmed consistent associations between nailfold capillary abnormalities (e.g., abnormal morphology, microhemorrhages, and avascular areas) and diabetes, with stronger effects in type 2 disease.[56] Pediatric data remain limited but are emerging. In children with type 1 diabetes, capillaroscopic alterations have been described before the onset of clinical microvascular complications, and capillary density has been positively correlated with time-in-range on continuous glucose monitoring, suggesting that NVC may capture early relationships between microvascular structure and glycemic control.[57]

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection, multisystem inflammatory syndrome in children (MIS-C), and long coronavirus disease-19 (COVID-19) provide contemporary examples of inflammation-related microvascular injury. In children following COVID-19 infection and MIS-C, studies have reported mostly non-specific NVC changes, including tortuosity, capillary crossing, ramification, neoangiogenesis, microhemorrhages, reduced capillary density, and increased intercapillary distance.[58,59] These findings appear more pronounced in MIS-C, suggesting that systemic hyperinflammation, endothelial activation, and thromboinflammation may be reflected in the peripheral microcirculation. Giant capillaries, avascular areas, and a scleroderma pattern are generally absent; therefore, these changes should not be interpreted as evidence of scleroderma-spectrum disease.[59] In adult long COVID, similar non-specific changes may persist for months after acute infection.[60]

ARTIFICIAL INTELLIGENCE AND FUTURE PERSPECTIVES

The future clinical value of NVC in pediatric rheumatology will depend on standardized image acquisition, development of pediatric reference datasets, longitudinal validation, and more objective image analysis. Current limitations include operator dependence, variable image quality, inconsistent terminology, and age-related variability in normal pediatric capillaroscopic patterns.

Artificial intelligence and automated image analysis may help standardize the assessment of capillary density, apical diameter, loop morphology, microhemorrhages, and abnormal capillary shapes. These approaches may reduce interobserver variability and facilitate multicenter longitudinal studies. A recent machine learning model using NVC image features to distinguish adult rheumatoid arthritis, SLE, and healthy controls suggests that automated image analysis may eventually contribute to clinical decision support.[9,10,61] However, its role is to support expert clinical interpretation, not to replace it.

Future work will likely move beyond the search for a single disease-specific pattern toward the integration of standardized microvascular measurements into broader clinical models. In such frameworks, NVC could be combined with clinical scores, autoantibodies, biomarkers, ultrasound findings, and patient-reported outcomes to support vascular phenotyping and longitudinal monitoring in pediatric rheumatology.

METHODOLOGICAL LIMITATIONS

The interpretation of pediatric NVC studies is limited by methodological heterogeneity. Many studies are single-center, cross-sectional, and based on small cohorts, particularly outside RP, jSSc, and JDM. Differences in age, disease duration, disease activity, treatment exposure, and autoantibody profiles further limit direct comparability. Technical variability in devices, magnification, examined fingers, image selection, and acquisition conditions also reduces reproducibility. In addition, scoring systems and terminology remain incompletely harmonized, and non-scleroderma diseases are often interpreted using SSc-derived frameworks. Future studies should adopt standardized protocols, age-appropriate reference datasets, and longitudinal designs linked to clinically meaningful outcomes.

CONCLUSION

NVC is a non-invasive and valuable method for assessing peripheral microcirculation in pediatric rheumatology. The clearest indications remain RP and jSSc/scleroderma-spectrum disorders, where recognition of the scleroderma pattern contributes to early detection of scleroderma-spectrum microangiopathy. Beyond this core role, NVC is increasingly applied across other pediatric rheumatic conditions, including JDM, childhood-onset SLE, juvenile Sjögren disease, MCTD, JLoS, JPsA, vasculitides (e.g., BD, KD), and autoinflammatory diseases. In most of these settings, findings are non-specific and cannot serve as standalone diagnostic markers. Selected non-rheumatologic conditions, particularly diabetes mellitus and SARS-CoV-2 infection/MIS-C, further illustrate the potential of NVC to capture systemic endothelial and microvascular dysfunction, although these applications remain exploratory.

Pediatric interpretation of NVC must consider age-related physiological variability, technical factors, and clinical context. Adult thresholds and prognostic frameworks cannot be directly applied to children. Pediatric reference datasets must be established, and acquisition and reporting protocols should be standardized. Longitudinal multicenter cohorts and validation of automated image analysis will be essential to determine whether NVC can evolve from a descriptive tool into a more robust instrument for diagnosis, risk stratification, and disease monitoring in pediatric rheumatology.

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