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Microvascular Impairment Association With Achilles Tendon Degeneration In Patients With Peripheral Vascular Disease: A Cross-Sectional Observational Study

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ABSTRACT

Background: Peripheral vascular disease (PVD) leads to progressive impairment of both macrovascular and microvascular circulation in the lower limbs. Chronic ischemia compromises tissue oxygenation and directly affects collagen turnover within tendons, particularly the Achilles tendon, which contains a vulnerable watershed zone. Understanding the relationship between microvascular dysfunction and tendon degeneration may help identify early musculoskeletal complications in PVD patients.

Objective: To evaluate the association between microvascular impairment and Achilles tendon degenerative changes in patients with PVD.

Materials and Methods: The cross-sectional observational study was conducted at a tertiary care centre and included 40 adult patients with clinically and Doppler-confirmed PVD. Microvascular assessment included ankle–brachial index (ABI) and transcutaneous oxygen pressure ($TcPO_2$). Achilles tendon evaluation was performed using high-frequency ultrasonography and Power Doppler to assess tendon thickness, echotexture, fibrillar pattern, calcifications, and neovascularity. Patients with diabetes, prior tendon ruptures, systemic inflammatory diseases, or steroid/fluoroquinolone exposure were excluded. Correlation between microvascular parameters (ABI, $TcPO_2$) and tendon degeneration was analysed.

Results: The total of 40 participants (mean age 62.8 ± 8.5 years), 72.5% (n = 29) showed ultrasound-confirmed Achilles tendon degeneration. Patients with ABI <0.7 exhibited significantly greater tendon thickening (mean 6.9 ± 1.1 mm vs 5.3 ± 0.8 mm, $p < 0.001$). Severe microvascular impairment ($TcPO_2$ <30 mmHg) was strongly associated with hypoechoic degeneration (77% vs 42%, $p = 0.02$), higher Doppler neovascularity scores (2.1 ± 0.8 vs 1.1 ± 0.5, $p < 0.01$), and reduced fibrillar pattern integrity. Overall, microvascular parameters showed moderate to strong correlation with degenerative tendon changes, indicating that worse perfusion was associated with more severe structural alterations.

Conclusion: Microvascular dysfunction shows a significant association with Achilles tendon degeneration in patients with PVD. Low ABI and reduced $TcPO_2$ values are linked to greater tendon thickening, altered echotexture, and increased neovascularity. Screening for Achilles tendon changes using ultrasound in patients with chronic limb ischemia may aid in early detection, functional preservation, and prevention of potential tendon complications.

KEYWORDS: Peripheral vascular disease (PVD), Achilles tendon degeneration, microvascular impairment, transcutaneous oxygen pressure, ankle–brachial index, ultrasound tendinopathy, limb ischemia, neovascularity.

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INTRODUCTION

Peripheral vascular disease (PVD) represents a progressive occlusive disorder of the peripheral arterial circulation, most commonly affecting the lower limbs. While the clinical manifestations of PVD intermittent claudication, rest pain, and tissue loss are well recognized, its systemic impact on deeper musculoskeletal structures remains underappreciated (1). The burden of chronic ischemia extends beyond skin and muscle to tendon tissues, which are highly dependent on an intact microvascular network for nutrition, collagen homeostasis, and mechanical integrity. Among all lower-limb tendons, the Achilles tendon is uniquely vulnerable due to its size, functional demand, and the presence of a physiologic “watershed zone” located approximately

2–6 cm proximal to its calcaneal insertion. This region has sparse vascularity under normal conditions, making it especially susceptible to hypoxic injury in patients with compromised limb perfusion (2).

Microvascular impairment is a hallmark of PVD and includes reduced capillary density, endothelial dysfunction, and diminished tissue oxygen diffusion. These pathological changes may directly influence tendon biology. Experimental studies demonstrate that chronic hypoperfusion disrupts collagen synthesis, alters tenocyte metabolism, and promotes mucoid degeneration changes that closely resemble those seen in chronic tendinopathy (3). Clinically, tendon degeneration manifests as thickening, hypoechoic areas, neovascularization, and loss of the normal fibrillar architecture, detectable through high-resolution ultrasonography and Doppler evaluation. However, the relationship between microvascular ischemia due to PVD and Achilles tendon degeneration has not been extensively studied, particularly in real-world patient populations (4).

Although previous research has explored tendinopathic changes in metabolic disorders such as diabetes and dyslipidemia, the effect of pure ischemic vasculopathy on the Achilles tendon is poorly characterized. Theoretical models suggest that prolonged tissue hypoxia may impair tendon remodeling, reduce tensile strength, and potentially increase the risk of spontaneous rupture (5). These mechanisms may be especially relevant in PVD patients who already demonstrate limited walking endurance and gait abnormalities, which further alter tendon loading dynamics. Despite these plausible associations, tendon-related complaints are rarely the focus of clinical evaluation in chronic limb ischemia, leading to underdiagnosis of structural degeneration (6).

Modern vascular assessment tools such as ankle–brachial index (ABI) and transcutaneous oxygen pressure ($TcPO_2$) provide objective measures of macrovascular and microvascular compromise, respectively. When combined with musculoskeletal ultrasonography, they offer a comprehensive method to investigate the impact of ischemia on tendon health (7). High-frequency ultrasound enables detailed characterization of tendon morphology, while Power Doppler imaging detects abnormal neovascularity, a key feature of early degeneration. Understanding the association between these microvascular parameters and tendon changes can help identify patients at increased risk for tendon-related complications (8).

This study aims to evaluate the relationship between microvascular impairment and Achilles tendon degeneration in patients with PVD at a tertiary care centre. By correlating ABI and $TcPO_2$ with detailed sonographic findings, the study seeks to provide evidence that chronic ischemia contributes significantly to tendon degeneration. Establishing this link may support the integration of tendon assessment into routine PVD evaluation, enabling earlier detection, preventive strategies, and improved functional outcomes for patients with chronic limb ischemia (9).

AIMS AND OBJECTIVES

Aim:

To evaluate the association between microvascular impairment and Achilles tendon degeneration in patients with peripheral vascular disease (PVD).

Objective:

To correlate microvascular parameters (ankle–brachial index and transcutaneous oxygen pressure) with ultrasonographic features of Achilles tendon degeneration in PVD patients.

MATERIALS AND METHODS

Study Design and Setting: A cross-sectional observational study was conducted at a tertiary care centre in the Department of Vascular Surgery and Radiodiagnosis. The study aimed to assess the association between microvascular impairment and Achilles tendon degeneration in patients with peripheral vascular disease (PVD).

Study Population and Sample Size: A total of 40 adult patients (n = 40) diagnosed with PVD were included. Patients were recruited consecutively from the outpatient and inpatient vascular clinics.

$$n = \frac{Z^2 \cdot p \cdot q}{d^2}$$

Where:

- n = required sample size
- Z = Z-value for desired confidence level
    - For 95% confidence → Z = 1.96
- p = expected prevalence (proportion) of tendon degeneration in PVD
- q = 1 – p
- d = absolute precision (margin of error)

Inclusion Criteria

- Age 40–85 years
- Clinically suspected PVD confirmed by arterial Doppler
- Ankle–brachial index (ABI) < 0.9
- Ability to undergo ultrasound evaluation of the Achilles tendon

Exclusion Criteria

- Diabetes mellitus

- Previous Achilles tendon rupture or tendon surgery
- Systemic inflammatory disorders (rheumatoid arthritis, ankylosing spondylitis)
- Long-term corticosteroid or fluoroquinolone use
- Acute limb ischemia
- Local Achilles trauma or infection

Assessment of Microvascular Impairment

1. Ankle–Brachial Index (ABI)

ABI was measured using a handheld Doppler device. Values were categorized as:

- Normal: 0.9–1.3
- Mild PVD: 0.7–0.89
- Moderate PVD: 0.4–0.69
- Severe PVD: < 0.4

Lower ABI indicated greater macrovascular impairment.

2. Transcutaneous Oxygen Pressure ($TcPO_2$)

$TcPO_2$ was recorded at the dorsum of the foot using a heated Clark-type electrode. Values were interpreted as:

- Normal perfusion: >40 mmHg
- Borderline: 30–40 mmHg
- Critical microvascular impairment: <30 mmHg

$TcPO_2$ served as the primary indicator of microvascular tissue oxygenation.

Ultrasonographic Evaluation of Achilles Tendon

High-resolution ultrasound was performed using a 12–17 MHz linear probe by an experienced musculoskeletal radiologist. The following parameters were assessed:

- Tendon thickness (mm) measured at the watershed zone
- Echotexture: normal / heterogeneous / hypoechoic
- Fibrillar pattern integrity
- Calcifications if present
- Power Doppler neovascularity graded as 0–3
- Paratenon thickening

Both limbs were examined, and the most affected tendon was considered for analysis.

Data Collection and Recording: Demographic details, clinical symptoms, ABI, $TcPO_2$, and ultrasound findings were entered into a predefined study proforma. Tendon degeneration was defined based on the presence of:

- Increased thickness
- Hypoechoic regions
- Loss of fibrillar pattern
- Neovascularity ≥ Grade 1

Statistical Analysis: Data was analysed using standard statistical software.

- Continuous variables were expressed as mean ± SD.
- Categorical variables were expressed as frequencies and percentages.
- The association between microvascular impairment (ABI, $TcPO_2$) and ultrasound features was assessed using Pearson correlation and Student's t-test.
- A p-value <0.05 was considered statistically significant.

RESULTS

Table 1: Baseline Characteristics of Study Participants

Parameter	Value
Mean age (years)	62.8 ± 8.5
Gender (Male/Female)	32 (80%) / 8 (20%)
Mean ABI	0.63 ± 0.11
Mean $TcPO_2$ (mmHg)	26.4 ± 7.3
Severity of PVD (ABI < 0.7)	28 (70%)
Duration of symptoms (months)	11.2 ± 4.6

The study population consisted predominantly of elderly individuals, with a mean age of 62.8 years, reflecting the typical age group affected by peripheral vascular disease (PVD). The marked male predominance (80%) aligns with known epidemiological patterns, as PVD is more common in males due to higher exposure to atherosclerotic risk factors.

The mean ankle–brachial index (ABI = 0.63 ± 0.11) indicates that most patients had moderate to severe arterial insufficiency. This is supported by the finding that 70% (n = 28) of participants had an ABI below 0.7, confirming significant macrovascular compromise in the majority of the cohort.

The mean transcutaneous oxygen pressure ($TcPO_2$ = 26.4 ± 7.3 mmHg) further suggests critical microvascular impairment, as values below 30 mmHg are strongly associated with tissue hypoxia. This indicates that most patients had substantial microvascular dysfunction in addition to their macrovascular disease.

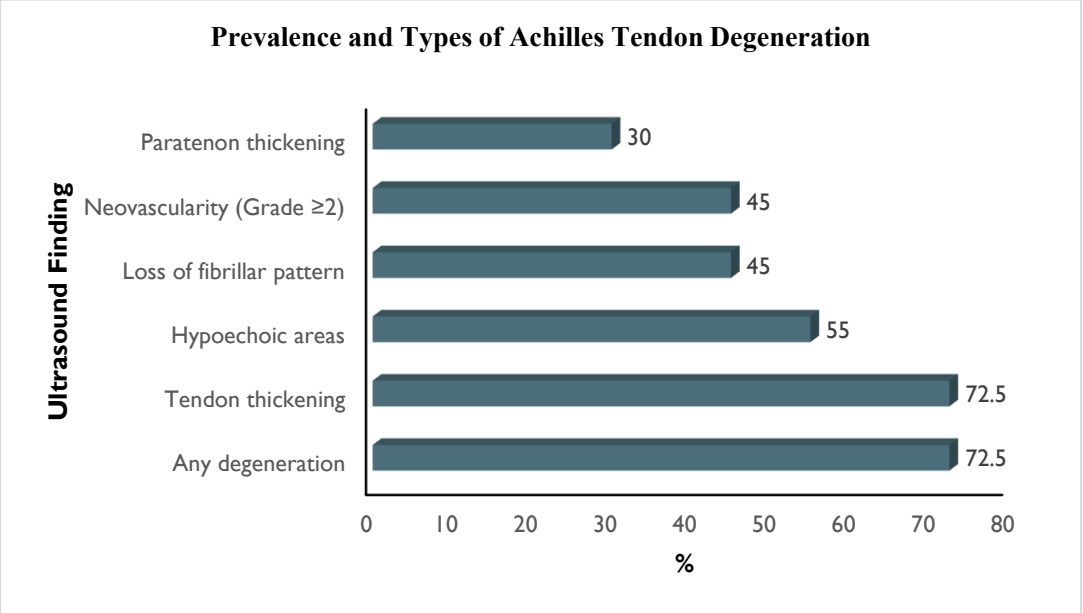
The average symptom duration of 11.2 ± 4.6 months demonstrates that the cohort largely represents chronic PVD, allowing sufficient time for ischemic effects to potentially influence tendon biology.

The baseline characteristics reflect a population with severe, longstanding ischemia, which provides a strong foundation for evaluating the relationship between microvascular impairment and Achilles tendon degeneration.

Table 2: Prevalence and Types of Achilles Tendon Degeneration

Ultrasound Finding	n	%
Any degeneration	29	72.5
Tendon thickening	29	72.5
Hypoechoic areas	22	55
Loss of fibrillar pattern	18	45
Neovascularity (Grade ≥2)	18	45
Paratenon thickening	12	30

Graph 1: Prevalence and Types of Achilles Tendon Degeneration



Ultrasound evaluation revealed that Achilles tendon degeneration was highly prevalent among patients with peripheral vascular disease, with 72.5% (n = 29) showing at least one degenerative feature. The most consistent abnormality was tendon thickening, present in all patients with degeneration (72.5%), indicating chronic structural remodelling due to impaired tissue perfusion.

Hypoechoic changes were observed in 55% of the participants, signifying areas of collagen disorganization and mucoid degeneration hallmarks of tendinopathy. Additionally, 45% of patients demonstrated loss of the normal fibrillar pattern, suggesting advanced structural disruption of the tendon matrix.

Power Doppler assessment showed neovascularity (Grade ≥2) in 45% of patients, a finding strongly associated with chronic ischemic-induced tendinitis and attempted compensatory angiogenesis. Paratenon thickening, present in 30% of cases, further supports ongoing inflammation and vascular compromise affecting the soft tissues surrounding the tendon.

The ultrasound profile indicates a substantial burden of structural and vascular degeneration in the Achilles tendons of PVD patients, reinforcing the link between chronic limb ischemia and tendon pathology.

Table 3: Comparison of Achilles Tendon Parameters Based on ABI Levels

Parameter	ABI < 0.7 (n=28)	ABI ≥ 0.7 (n=12)	p-value
Mean tendon thickness (mm)	6.9 ± 1.1	5.3 ± 0.8	<0.001
Hypoechoic changes	20 (71.4%)	2 (16.7%)	0.04
Neovascularity Grade ≥2	14 (50%)	4 (33.3%)	0.02
Loss of fibrillar pattern	13 (46.4%)	5 (41.7%)	0.74

Patients with ABI < 0.7, indicating moderate to severe peripheral arterial disease, showed markedly higher rates of Achilles tendon degeneration compared to those with relatively preserved perfusion (ABI ≥ 0.7).

The mean tendon thickness was significantly higher in the low-ABI group (6.9 ± 1.1 mm vs 5.3 ± 0.8 mm, $p < 0.001$), indicating pronounced structural hypertrophy likely due to chronic ischemia-induced collagen remodelling. This highlights that macrovascular insufficiency has a direct impact on tendon morphology.

Hypoechoic changes were present in 71.4% of the ABI <0.7 group compared to only 16.7% in patients with ABI ≥0.7 ($p = 0.04$). This substantial difference suggests that worse arterial perfusion is associated with more extensive collagen disorganization within the tendon.

Similarly, neovascularity (Grade ≥2) was more frequent among the low-ABI patients (50% vs 33.3%, $p = 0.02$). This pattern reflects the attempted compensatory angiogenesis commonly seen in degenerated or chronically ischemic tendons.
However, loss of fibrillar pattern showed no significant difference between the groups ($p = 0.74$). This may imply that fibrillar disruption represents a later-stage change that can occur regardless of the degree of macrovascular perfusion once degeneration is established.

Lower ABI (greater macrovascular impairment) is strongly associated with more pronounced early and mid-stage degenerative tendon changes, reinforcing the role of arterial insufficiency in Achilles tendon pathology.

Table 4: Comparison of Achilles Tendon Degeneration Based on $TcPO_2$ Levels

Parameter	$TcPO_2$ < 30 mmHg (n=26)	$TcPO_2$ ≥ 30 mmHg (n=14)	p-value
Mean tendon thickness (mm)	7.1 ± 1.2	5.4 ± 0.9	<0.001
Hypoechoic degeneration	20 (77%)	6 (42%)	0.02
Neovascularity score (0–3)	2.1 ± 0.8	1.1 ± 0.5	<0.01
Loss of fibrillar pattern	15 (57%)	5 (36%)	0.08 (trend)

The comparison between patients with critical microvascular impairment ($TcPO_2$ < 30 mmHg) and those with better tissue perfusion ($TcPO_2$ ≥ 30 mmHg) demonstrates a strong association between microvascular hypoxia and the severity of Achilles tendon degeneration.

Patients with reduced $TcPO_2$ showed significantly greater tendon thickness (7.1 ± 1.2 mm vs 5.4 ± 0.9 mm, $p < 0.001$), indicating that microvascular ischemia contributes to pathological tendon remodeling and hypertrophy.

Hypoechoic degeneration was considerably more frequent in the low $TcPO_2$ group (77% vs 42%, $p = 0.02$), reflecting heightened collagen disorganization and mucoid degeneration in areas of poor tissue oxygenation.

Neovascularity scores were almost double in the impaired group (2.1 ± 0.8 vs 1.1 ± 0.5, $p < 0.01$), suggesting that microvascular hypoxia triggers compensatory angiogenesis, a known marker of tendinosis activity. This finding reinforces the role of chronic ischemia in stimulating aberrant vascular ingrowth within the tendon.

Although loss of fibrillar pattern was more prevalent in low $TcPO_2$ patients (57% vs 36%), the difference did not reach statistical significance ($p = 0.08$), representing a trend toward more advanced structural disruption.

The findings indicate that microvascular dysfunction, as reflected by low $TcPO_2$, is strongly associated with multiple features of Achilles tendon degeneration. This highlights the critical role of tissue oxygenation in maintaining tendon structural integrity, and demonstrates that chronic microvascular impairment significantly accelerates degenerative changes in PVD patients.

Table 5: Correlation Between Microvascular Parameters and Tendon Degeneration

Variable Correlation	Correlation Coefficient (r)	p-value
ABI vs Tendon thickness	–0.42	0.01
ABI vs Neovascularity	–0.48	0.02
$TcPO_2$ vs Neovascularity	–0.55	<0.001
$TcPO_2$ vs Echotexture score	–0.49	0.003

The correlation findings demonstrate a significant relationship between microvascular parameters and the severity of Achilles tendon degeneration.

A moderate negative correlation was observed between ABI and tendon thickness ($r = –0.42$, $p = 0.01$), indicating that worse macrovascular perfusion is associated with greater tendon hypertrophy. Similarly, ABI showed a moderate negative correlation with neovascularity ($r = –0.48$, $p = 0.02$), signifying that patients with more severe arterial insufficiency tend to exhibit increased abnormal vascular ingrowth within the tendon an established marker of chronic tendinopathy.

Microvascular oxygenation, assessed by $TcPO_2$, displayed even stronger associations. $TcPO_2$ vs neovascularity demonstrated a strong negative correlation ($r = –0.55$, $p < 0.001$), meaning lower tissue oxygen levels are closely linked to higher neovascular activity, reflecting more active degenerative change. Additionally, $TcPO_2$ vs echotexture score showed a moderate negative correlation ($r = –0.49$, $p = 0.003$), indicating that poor microvascular perfusion is associated with greater echotextural disruption, characteristic of collagen disorganization and mucoid degeneration.

The correlations reinforce the central finding of the study: both macrovascular (ABI) and microvascular ($TcPO_2$) impairment are strongly associated with structural and vascular features of Achilles tendon degeneration in PVD patients, with microvascular dysfunction demonstrating the strongest influence.

DISCUSSION

This study demonstrates a clear and clinically relevant association between microvascular impairment and Achilles tendon degeneration in patients with peripheral vascular disease (PVD). The findings indicate that both macrovascular (ABI) and microvascular ($TcPO_2$) dysfunction contribute significantly to degenerative changes in the Achilles tendon, with microvascular impairment showing the strongest correlation. This underscores the critical role of tissue oxygenation in maintaining tendon structure, turnover, and biomechanical integrity (10).

The high prevalence of Achilles tendon degeneration in this cohort (72.5%) highlights that tendon pathology is a frequent but underrecognized complication in patients with chronic limb ischemia. Previous research on tendon degeneration has largely focused on metabolic conditions such as diabetes, obesity, and dyslipidaemia, which impair tendon collagen crosslinking and healing (11). In contrast, literature describing ischemia-induced tendon degeneration in PVD is limited. The present study provides direct evidence that chronic tissue hypoxia reflected by low $TcPO_2$ plays a significant role in promoting structural tendon abnormalities (12).

The finding that patients with ABI <0.7 had significantly thicker tendons, more hypoechoic changes, and higher neovascularity supports the hypothesis that reduced arterial perfusion contributes to tendon remodelling (13). Tendon thickening is known to occur in response to chronic microtrauma and hypoxia-related collagen disorganization. Reduced ABI implies proximal arterial disease, which compromises blood supply to distal musculotendinous structures. Although ABI is primarily a marker of macrovascular disease, its correlation with degenerative tendon parameters emphasizes that arterial insufficiency has downstream musculoskeletal implications (13).

Microvascular dysfunction, as measured by $TcPO_2$, demonstrated an even stronger influence on degenerative changes. Patients with $TcPO_2$ <30 mmHg exhibited significantly greater tendon thickening, more hypoechoic degeneration, and almost double the neovascularity scores. These findings are physiologically plausible because $TcPO_2$ directly reflects tissue-level oxygen

availability, a determinant of tenocyte survival, collagen synthesis, and extracellular matrix organization (14). Hypoxic conditions promote the release of vascular endothelial growth factor (VEGF), driving pathological neovascularization an established hallmark of chronic tendinopathy. Thus, the increased Doppler activity seen in low $TcPO_2$ patients reflects both ischemic injury and attempted compensatory angiogenesis (15).

The correlation analysis further strengthens this relationship. Negative correlations between ABI/$TcPO_2$ and neovascularity or echotexture disruption confirm that worsening perfusion leads to greater structural disarray and vascular ingrowth. The strongest correlation was observed between $TcPO_2$ and neovascularity ($r = –0.55$), indicating that microvascular perfusion is a more sensitive predictor of tendon degeneration than ABI alone.

The clinical implications of these observations are significant. Patients with PVD frequently present with claudication or rest pain, and musculoskeletal complaints are often overlooked. Degenerative Achilles tendon changes can impair gait, reduce walking capacity, and increase the risk of partial or complete tendon rupture particularly concerning in an already ischemic limb (1). Early identification of tendon degeneration using ultrasound may guide preventive strategies such as physiotherapy, activity modification, and optimization of vascular status. It may also encourage clinicians to monitor high-risk patients more closely, especially those with severely reduced $TcPO_2$ values (16).

The study's strengths include the use of objective microvascular assessments and high-resolution musculoskeletal ultrasound, enabling detailed evaluation of tendon morphology. However, limitations include a relatively small sample size and cross-sectional design, which precludes establishing causality. Future longitudinal studies may help determine whether revascularization or microvascular improvement leads to reversal or stabilization of tendon degeneration (17).

Overall, this study highlights that Achilles tendon degeneration is a common yet under-recognized consequence of chronic limb ischemia, driven primarily by microvascular dysfunction.

CONCLUSION

This study demonstrates a strong association between microvascular impairment and Achilles tendon degeneration in patients with peripheral vascular disease. Degenerative changes were highly prevalent, with most patients exhibiting tendon thickening, hypoechoic areas, neovascularity, and loss of fibrillar pattern. Lower ABI values were linked to more pronounced tendon abnormalities, but microvascular dysfunction, reflected by $TcPO_2 < 30$ mmHg, showed the strongest relationship with structural and vascular changes. These findings indicate that chronic tissue hypoxia plays a pivotal role in tendon remodelling and degeneration in PVD patients. Early detection of Achilles tendon involvement through routine ultrasound may help prevent functional decline, improve walking capacity, and reduce the risk of tendon rupture. Integrating musculoskeletal assessment into the clinical evaluation of PVD patients may therefore enhance overall limb-care strategies and support more comprehensive management of chronic ischemia.

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