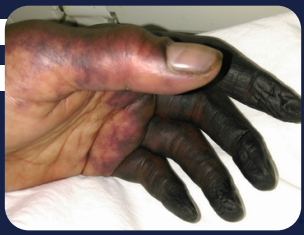
September 2025 | 2nd Edition

# VESSOMAG's Inside the VESSEL ...

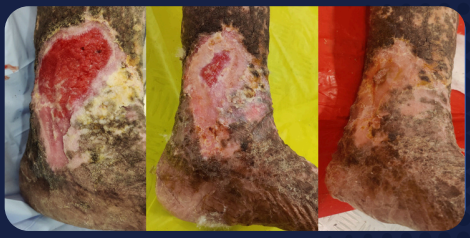
Official Quarterly Newsletter of the Vascular & Endovascular Surgeons of Maharashtra and Goa

## THEME WOUNDS



## Must Reads ....

- Cracking the Code in Chronic Vascular Wounds - an INTRODUCTION
- All Venous Ulcers are not due to VARICOSE VEINS
- Beyond Ischaemia : The Vasculitis Connection
- The Evolving Evidence of Anticoagulation in Wound Healing
- From Clock to CLINIC - Why Time is Tissue in Vascular Wounds
- Synergistic Approach in the management of CHRONIC WOUNDS
- The SORBACT® Technology



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# Secretary Speaks .....

**Dr. Amish P. Mhatre**
Vascular & Endovascular Surgeon
SECRETARY, VESSOMAG

Dear Colleagues,

It is my great pleasure to introduce the second edition of our quarterly newsletter, where VESSOMAG highlights various topics related to vascular diseases.

In this edition, we are focusing on wounds.

By definition, a wound occurs when the integrity of any tissue is compromised (e.g., skin breaks, muscle tears, burns, or bone fractures).

Wounds and ulcers—the latter being defined as a local defect or excavation on the surface of an organ or tissue, produced by the sloughing of necrotic inflammatory tissue—are terms often used interchangeably. We have all encountered and managed them since our MBBS training.

While they may appear straightforward to treat, in reality, they are often not. As clinicians, we treat the patient as a whole, not just the lesion. Looking beyond imaging reports, we aim to identify the underlying cause of the wound before planning appropriate management.

For example, a seemingly venous ulcer may turn out to be vasculitis; a diabetic wound may have an underlying venous or lymphatic pathology. Performing endovenous ablation of superficial veins post-DVT can become catastrophic for the patient—and equally distressing for us as clinicians. VESSOMAG, through its awareness team, is committed to updating fellow colleagues on various aspects of wound management related to arterial, venous, and lymphatic systems.

# Secretary Speaks .....

Looking beyond imaging, our members—drawing on their vast collective knowledge and experience—are happy to share both theoretical insights and practical understanding of this subject with fellow clinicians.

As the famous sentence by David Herbert Richards Lawrence (1885–1930) says:

**"What the eye doesn't see and the mind doesn't know, doesn't exist."**

This was paraphrased by Dr. Ashok Sarnaik (b. 1946) as:

**"If someone isn't familiar with something, such as a diagnosis, they won't be likely to notice it."**

(Source: Prologue – Chapter 2, What the Eyes Don't See, Bartleby)

Respected colleagues, VESSOMAG will continue bringing you a quarterly newsletter. Your inputs and suggestions will help us improve and make it more relevant.

We also invite you to share any specific topics related to vascular diseases that you would like to see included in future editions.

Please write to us at **vessomageducation@gmail.com**

# From the Editor's Desk .....

**Dr. Aniruddha Bhuiyan**
**Vascular & Endovascular Surgeon**
**MUMBAI**

Wounds are an everyday challenge for all of us in Vascular and Endovascular Surgery. Whether it's a diabetic foot ulcer, an ischemic limb wound or a post-operative non-healing site, wounds demand attention, patience and lots of effort. In this 2nd issue of INSIDE THE VESSEL, we bring together experiences and perspectives from vascular surgeons across VESSOMAG who regularly face this problem in their practice.

The aim of this edition is to offer practical insights rather than textbook theory. From tips on choosing the right plan of management, to updates on nuances in wound therapy and thoughts on when to revascularise - this issue is packed with cases, discussions and real-world strategies. We've also highlighted interesting cases and original articles for your perusal.

The contributors in this issue come from different parts of Maharashtra & Goa, from established centres to solo vascular practitioners in their infancy. But what unites them is a shared focus on healing, limb salvage and improved patient outcomes.

We hope this compilation not only helps you sharpen your clinical decision-making but also serves as a reminder that wounds are not just local problems - they often reflect deeper vascular issues. We hope an earlier referral of such wounds can overall improve clinical outcome and save the limb.

As always, we welcome feedback, suggestions and your own stories in future issues. Let's keep the conversation going.

Warm regards....

# Cracking the Code in Chronic Vascular Wounds - an INTRODUCTION

**Dr. Shahzad Bulsara**
Vascular & Endovascular Surgeon
DUBAI, UAE

## INTRODUCTION

Chronic wounds are defined as those that fail to progress through the usual, timely phases of healing and do not achieve anatomical and functional restoration within three months. These wounds become **"stuck"** in one of the physiological phases of healing—namely the **inflammatory**, **proliferative** or **remodeling** phases—due to various intrinsic and extrinsic factors. The common reasons for this arrest include cellular dysfunction from diabetes or smoking, ischemia-reperfusion injury, bacterial colonization triggering a chronic inflammatory state, depletion of local and systemic growth factors, changes in extracellular matrix, impaired fibroblast activity, biofilm formation and impaired circulation. Successful wound healing requires **optimal oxygen delivery, good nutrition**—including adequate protein, ascorbic acid and zinc—a moist wound bed and protection from trauma, infection and desiccation.

Chronic wounds can arise from pressure-related causes such as decubitus or neuropathic ulcers, vascular causes including venous, arterial or mixed insufficiency, autoimmune or primary cutaneous inflammatory conditions and miscellaneous factors such as radiation injury, malignancy or vasculitis. Their evaluation must begin with a detailed clinical history and physical examination that documents the wound's onset, duration, progression and associated systemic symptoms. **Identifying comorbidities** like diabetes, cardiac disease and immunodeficiencies is essential, as is understanding the patient's lifestyle, occupational posture, footwear and tobacco habits. A review of past treatments —both medical and surgical—often reveals valuable insights.

General examination should not overlook signs of anemia, edema, cardiac or respiratory dysfunction or nutritional deficiencies, while local wound assessment should carefully evaluate all parameters.

# Cracking the Code in Chronic Vascular Wounds - an INTRODUCTION

The surrounding skin should be checked for eczema, maceration, dermatitis and trophic changes. Bedside assessments such as **wound cultures, tissue biopsy** for suspected malignancy or vasculitis, vascular studies including Doppler ultrasound or ankle-brachial index and nutritional profiling (albumin, hemoglobin, weight trends) offer valuable guidance. Neuropathy assessments using nerve conduction velocity or Sudo scan are also helpful. Imaging such as X-rays or MRI may be required to rule out osteomyelitis, while duplex ultrasound and CT angiography assist in mapping vascular compromise. Lymphoscintigraphy helps in evaluating lymphedema.

Equally important is addressing the psychosocial aspect of chronic wound care. These patients have often seen multiple doctors and failed several treatments, leaving them fatigued and frustrated. Mental health support, social and economic assessment, counselling for adherence and efforts to preserve their quality of life are critical components of care. Management invariably benefits from a **multidisciplinary team** approach, incorporating expertise from vascular surgery, dermatology, endocrinology, plastic surgery, infectious diseases, physiotherapy and nutrition.

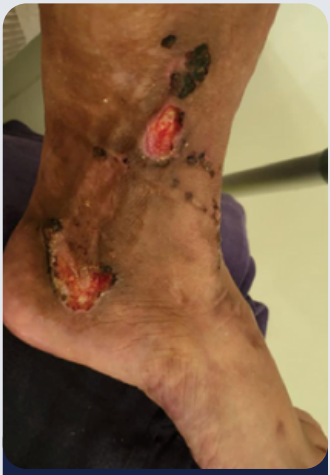


Fig 1. Venous Ulcer with Vasculitis (PAN)

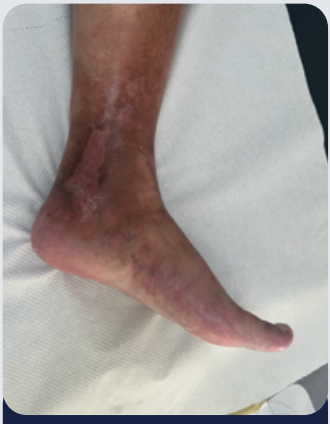


Fig 2. Healed Ulcer

# Cracking the Code in Chronic Vascular Wounds - an INTRODUCTION

Correcting negative influences is a cornerstone of treatment. Extrinsic factors like smoking, stress, prolonged standing or sitting and improper footwear must be addressed. Intrinsic factors such as uncontrolled diabetes, anemia, hypoalbuminemia, infections, autoimmune disorders and poor vascular flow must be medically optimized. Improving circulation may involve venous procedures like deep venous recanalization, ablation of varicosities or compression therapy for reflux; arterial interventions such as endovascular angioplasty, stenting or bypass; and lymphatic therapies including manual decompression, compression garments or surgical lymphovenous bypasses.

At the wound site, **moisture** plays a key role in enhancing granulation, cellular migration, nutrient delivery and epithelialization while minimizing necrosis and scar formation. **Wound bed preparation (WBP)** involves clearing necrotic tissue, reducing bacterial load, addressing hypoxia and removing senescent cells and biofilms. The choice of dressing should be based on wound characteristics. While traditional gauze may suffice for protection, **advanced options** like antimicrobial dressings (silver, iodine), autolytic debridement agents (films, hydrogels) and chemical agents (papain, collagenase) provide additional benefits.

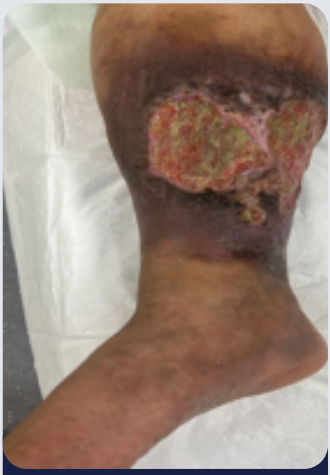


Fig 3. Venous Ulcer

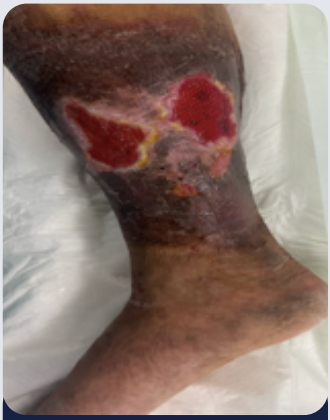


Fig 4. Healing Venous Ulcer

# Cracking the Code in Chronic Vascular Wounds - an INTRODUCTION

Absorbent dressings such as foams, alginates or hydrofibers help manage exudate depending on volume. Modern advances such as collagen dressings, super-oxidized solutions, vacuum-assisted closure (NPWT) and growth factors support enhanced healing. **Emerging therapies**—like skin substitutes, stem cell and gene therapy, topical insulin or hemoglobin and both hyperbaric and topical oxygen—offer exciting future avenues.

Several illustrative cases highlight the complexity of chronic wounds. A classic chronic wound may present with punched-out edges and pale granulation with biofilm. A more complex case, such as a combined arterial and venous ulcer secondary to polyarteritis nodosa, required endovascular venous ablation, anticoagulation and immunosuppression for healing. Post-thrombotic wounds are recurrent and demand long-term endovascular surveillance and compression therapy. Arterial ulcers, particularly in diabetic patients with poor cardiac output, may remain non-healing even after revascularization and benefit from negative pressure wound therapy and systemic optimization.

Fig 5. On VAC Dressing

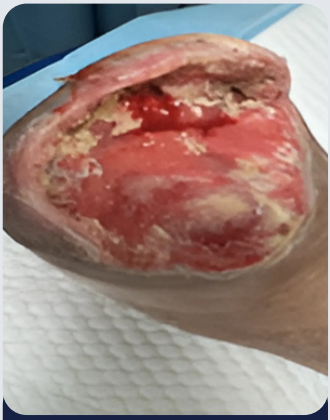
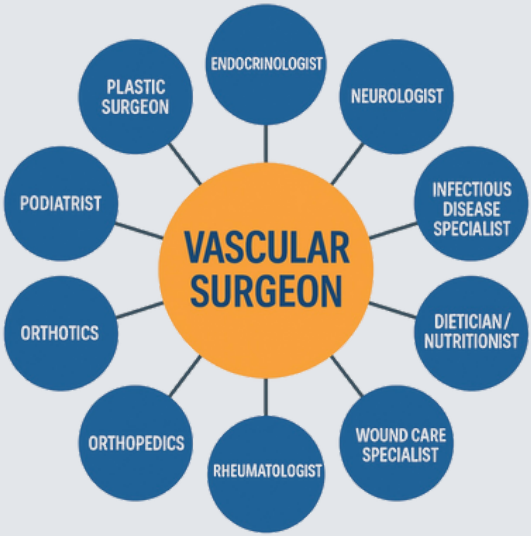


Fig 6. Improved Healing over Forefoot stump



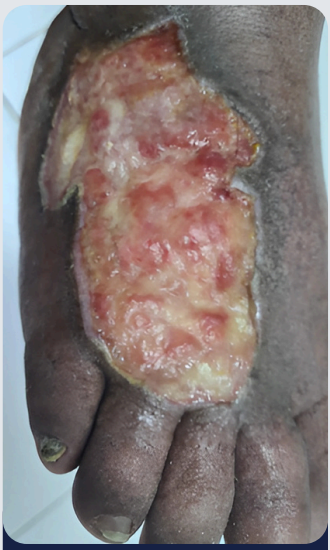



Fig 7. Mixed Ulcers

In conclusion, *managing chronic wounds goes far beyond dressing and debridement*. It requires an in-depth understanding of wound biology, patient comorbidities, psychosocial factors and access to a broad armamentarium of medical, surgical and supportive therapies delivered through a cohesive, multidisciplinary strategy. With vigilance, persistence and comprehensive care, healing can be achieved even in the most stubborn chronic wounds.

# Questions and Answers
# VASCULAR WOUNDS

**Dr. Shrikant Ghanwat**
Vascular & Endovascular Surgeon
PUNE

## Q & A

**Q1. Why do chronic wounds matter in vascular surgery?**

- They are not just skin problems but markers of systemic pathology.
- Often signal advanced vascular disease and predict poor outcomes.
- Central to:
  - Limb salvage
  - Preservation of function and mobility
  - Overall quality of life
- For vascular surgeons, wound care is not secondary—it is core to practice.

**Q2. What types of vascular wounds should we recognize?**

- Arterial ulcers – ischemic, punched-out edges, severe rest pain, due to PAD.
- Venous ulcers – shallow, exudative, gaiter region, linked to CVI or PTS.
- Mixed ulcers – features of both arterial and venous disease; high risk of mismanagement if one component is ignored.
- Diabetic foot ulcers – neuropathic, ischemic, or infective; common cause of amputation.
- Vasculitis ulcers – inflammatory, atypical sites, often resistant to conventional therapy.

**Q3. How should vascular wounds be assessed?**

Assessment is the foundation for management:

- Etiology identification – arterial, venous, neuropathic, or mixed.
- Perfusion evaluation – $TcPO_2$ (tissue oxygenation), Skin perfusion pressure & Duplex / handheld Doppler
- Wound characterization – Area and depth, Presence of infection or biofilm, Tissue viability and loss

**Case Capsule :**
58-year-old gentleman with a history of Trendelenburg's procedure 4 years ago presented with recurrent varicose veins and a medial gaiter-area ulcer. Venous Doppler revealed mid-thigh and above-ankle perforator incompetence, GSV reflux, and multiple below-knee varicosities. He underwent EVLA of the incompetent perforators and GSV in the thigh. Compression dressing was applied on an OPD basis. The ulcer healed completely within 6 weeks.

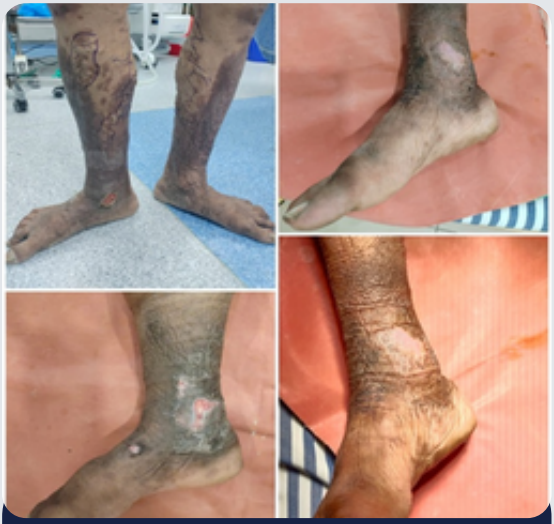


Fig 1. Phases of Healing of the Venous Ulcer

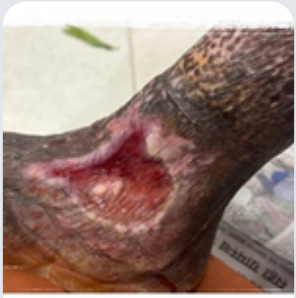


Fig 2. Venous Ulcer Pre-OP

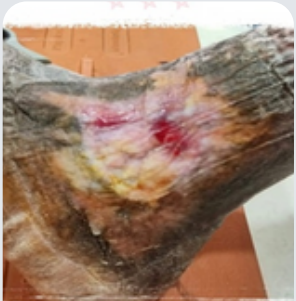


Fig 3. Venous Ulcer Post-OP

**Case Capsule :**
64F, chronic lateral ankle ulcer >1 year.
Duplex: superficial venous reflux.
IVUS: 90% left common iliac vein stenosis.
IVUS-guided venoplasty + stenting, RFA of superficial veins.
Healed: marked at 4 weeks, complete at 8 weeks

**Case Capsule :**
55/F, diabetic & hypertensive, multiple toe amputations done elsewhere.
Presented with non-healing wound & forefoot gangrene.
CTA: short SFA occlusion + tibial vessel disease.
Treated with SFA stenting + tibial angioplasty, then forefoot amputation.
NPWT + skin grafting → complete wound healing

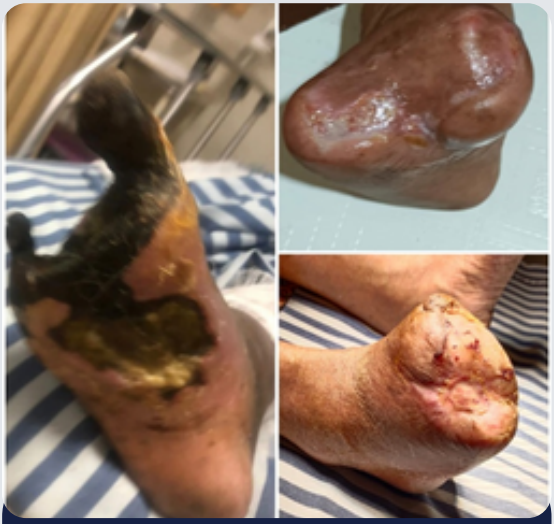


Fig 4. Phases of Diabetic Foot Ulcer

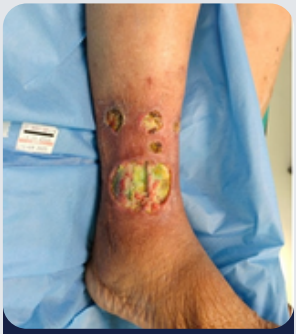


Fig 5. Vasculitis Ulcer

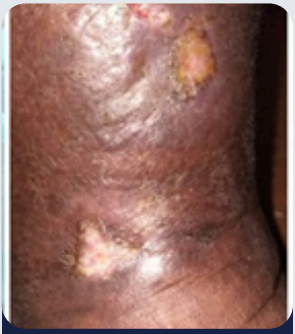


Fig 6. Healed Ulcer

**Case Capsule :**
Middle-aged female with multiple painful leg ulcers.
Arterial & venous Doppler: normal.
Suspicion of vasculitis → edge biopsy + debridement done.
ESR & CRP elevated, confirming vasculitis.
Medical management + debridement + dermal dressing → complete healing.

**Q4. What role does the vascular surgeon play?**

Vascular surgeons are uniquely positioned at the crossroads of:

- Diagnosis (differentiating wound types)
- Revascularization (restoring flow)
- Wound care (optimizing local environment for healing)

Key interventions:

- Arterial revascularization : Open bypass (fem-pop, fem-distal), Endovascular (angioplasty, stenting, atherectomy)
- Venous interventions : Superficial ablation – laser, RFA, glue, microwave, Deep venous stenting – IVUS-guided iliac vein stents for outflow obstruction

**Q5. What advanced wound therapies are available?**

- Negative Pressure Wound Therapy (NPWT): Promotes angiogenesis, Reduces edema and bacterial load, Enhances granulation tissue, Modern NPWT systems allow instillation therapy for irrigation
- Biological/Bioengineered therapies: Amniotic membrane allografts – rich in growth factors ; Skin substitutes (Apligraf, Dermagraft) – support re-epithelialization; Platelet-Rich Plasma (PRP) – autologous growth factors that stimulate healing

**Q6. What is the ultimate clinical takeaway?**

- Vascular wounds = systemic disease in disguise.
- They predict potential limb loss if not addressed early.
- Combining:
  - Revascularization (open or endovascular)
  - Advanced wound therapies
  - ensures not just healing but also restored dignity and independence for patients.

# The SORBACT® Technology

**Dr. Paresh Pai**
Vascular & Endovascular Surgeon
MUMBAI

## SPOTLIGHT

Patients and surgeons encounter wounds in many situations. These wounds can affect the outcome of the surgical procedure or disease condition, leading to varying degrees of morbidity, mortality, or speedy recovery.
There is no doubt that the age-old adage "**prevention is better than cure**" holds true for ensuring smooth and fast recovery. However, early detection and appropriate, adequate treatment of wounds also contribute significantly to a successful outcome.

Wound healing may be delayed due to local, focal, or systemic factors—either alone or in combination.

- Local factors include large or deep wounds, presence of necrotic tissue and exposed bone, foreign bodies, involvement of joints, areas previously exposed to radiation, and infections by organisms such as Acinetobacter baumannii, Pseudomonas aeruginosa, Enterobacteriaceae, Enterococcus faecium, and Staphylococcus aureus. These microbes may manifest as contaminants, colonizers, local infections, spreading infections, or systemic infections depending on their virulence and load.
- Focal factors include poor arterial inflow, inadequate venous or lymphatic drainage, and neuropathy, which make wounds more prone to repeated injury.
- Systemic factors include anemia, edema, poor nutrition, reduced immunity, use of immunosuppressive drugs or steroids, diabetes, and compromised function of vital organs such as the heart, lungs, kidneys, and liver.

**Antimicrobial resistance (AMR)** plays a major role in complicating wound treatment and adds significantly to the difficulty and cost of care.

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According to the WHO, AMR is one of the top global public health threats, rising to dangerously high levels worldwide. Without urgent action, we could be heading for a post-antibiotic era where common infections and minor injuries may once again become fatal. AMR is predicted to cause 10 million deaths annually by 2050. At least 30% of antibiotics prescribed in outpatient settings are unnecessary. Around 40% of chronic wounds develop infection, which may present as slow healing, halted improvement, deterioration of the wound, increased pain or discharge.

This article focuses on a **new option in wound care** that is cost-effective and serves as an adjunct to standard treatment. It supports the correction of local, focal, and systemic factors to improve outcomes.

The correct choice of dressing material and technique plays a vital role in successful and quick healing. This choice depends on the wound's condition, presence of microbes, amount of necrotic tissue, slough, and discharge. A balanced approach is needed—maintaining local moisture, controlling microbes, removing necrotic tissue and slough (surgically or non-surgically), eliminating discharge, and preventing maceration. This encourages the formation of healthy granulation tissue and new skin. Options include antibacterial ointments and creams, desloughing enzymes, hydrocolloid gels with or without foam, alginates with or without silver, agents to clear biofilms, and negative pressure wound therapy (NPWT), with or without irrigation. However, *some antimicrobial agents may release harmful endotoxins when killing bacteria*.

Wound dressings that work through physical methods of infection control are ideal in managing AMR and align well with antimicrobial stewardship. One such solution is dressings coated with Di-alkyl-carbamoyl chloride (DACC). These dressings allow bacteria to irreversibly bind to their surface for safe removal, providing an antibacterial effect without releasing active substances—thus not contributing to AMR.

Sorbact® Technology uses **DACC-coated dressings** to safely and effectively manage wound infections. The surface has unique hydrophobic properties that cause bacteria and fungi to bind irreversibly.

# The SORBACT® Technology

This includes binding of endotoxins that might otherwise impair healing. Once bound, these microbes and endotoxins are removed safely without being released into the wound, thus reducing bioburden and supporting natural healing.

Sorbact® dressings have also been shown to be effective against **MRSA** infections and may reduce the need for systemic antibiotics. They help accelerate healing, decrease wound size, are safe and easy to use, improve patient comfort, reduce pain during and after dressing changes, and minimize odour.

Cutimed® Sorbact® Technology is available in different forms for treating various types of wounds.

| |  Swab |  Ribbon gauze |  Dressing pad |  Round Swab |  Siltec |  Sorbion |  Leukomed |
|---|---|---|---|---|---|---|---|
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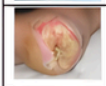
|  | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
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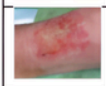
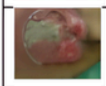
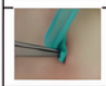
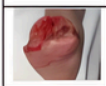
|  | ✓ | ✓ | ✓ | | ✓ | ✓ | ✓ |
|  | ✓ | ✓ | | ✓ | | | |
|  | | ✓ | | | | | |
|  | ✓ | | | | | | |

# The SORBACT® Technology

## Case report: Leg ulcers

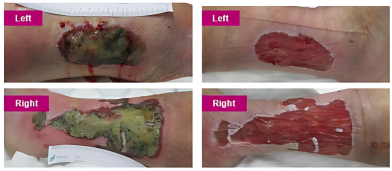
### The patient

An 89-year-old man suffering from hypertension with a long history of leg ulcers.

### The treatment

Treatment with silver dressing without sufficient effect. Changed to Cutimed® Sorbion® Sorbact® with focus on managing the exudate and lowering the bioburden.

Dressing changed every 3 days.


**Day 0**
Start of treatment with Cutimed® Sorbion® Sorbact®

**Day 21**
Result 3 weeks later

## Case report: Venous Leg Ulcers

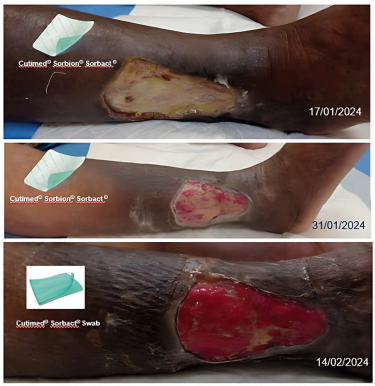
### The patient

A 73-year-old lady suffering from CVI with EVLT 10 years ago and now a long history of leg ulcers since 3 years that are very painful.

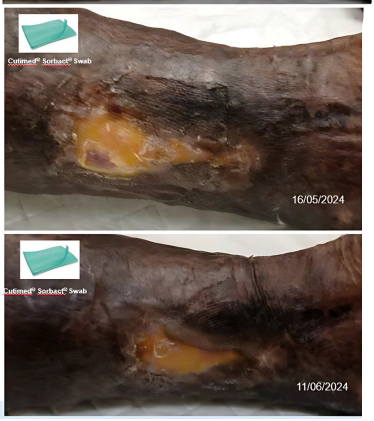
### The treatment

Treatment with silver dressing without sufficient effect. Changed to Cutimed® Sorbion® Sorbact® with focus on managing the exudate and lowering the bioburden.

Dressing changed every 7 days using Cutimed Sorbion Sorbact along with multilayer compression bandage




## Case report: Venous Leg Ulcers

## Case report: Arterial Leg Ulcers


### The patient

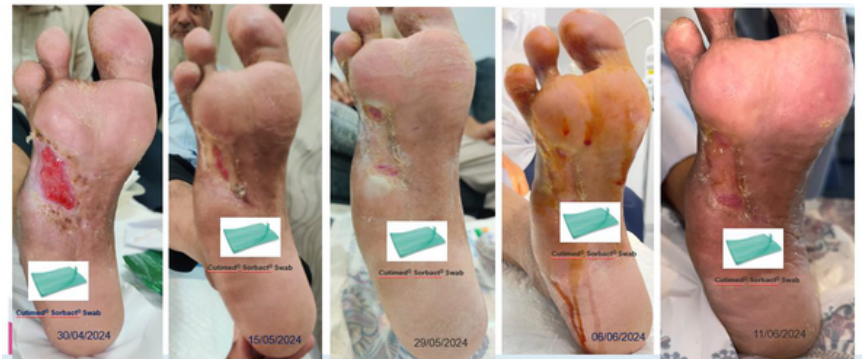
A 56-year-old man suffering from HT, NIDDM with diabetic neuropathy and diabetic foot with amputation of R) 4th & 5th toes with non-healing wounds since 1 month.

### The treatment

Treatment with Angioplasty and stenting of R) SFA, PTA and ATA with debridement of R) foot ulcer followed with once a week dressing with Veraflow Cleanse Choice using Microdyscn irrigation.

Once wound improved skin cover obtained with Polynovo dermal matrix kept in place for 3 weeks.

## Case report: Arterial Leg Ulcers



# Beyond Ischaemia : The Vasculitis Connection

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**Dr. Amit Rane**
Vascular & Endovascular Surgeon
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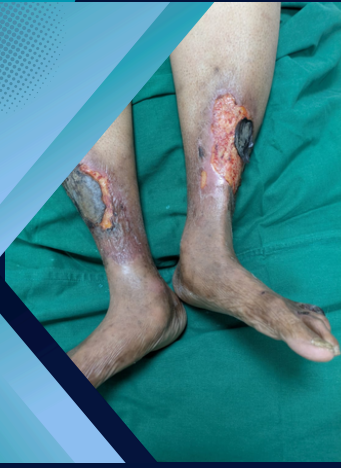

SPOTLIGHT

Vasculitis ulcers are among the most challenging manifestations of systemic and cutaneous vasculitis. They often indicate small- or medium-vessel involvement and may reflect active disease, requiring timely diagnosis and a coordinated multidisciplinary approach.

**Pathophysiology at a Glance**

Vasculitis ulcers result from inflammation and necrosis of blood vessels, most commonly involving post-capillary venules or small arterioles in the dermis or subcutis. Immune complex deposition, leukocytoclasia, and complement activation lead to endothelial damage, thrombosis, and tissue ischemia.

Common types of vasculitis associated with ulceration include:

- *Cutaneous leukocytoclastic vasculitis (CLV)*
- *Polyarteritis nodosa (PAN)*
- *Microscopic polyangiitis (MPA)*
- *Granulomatosis with polyangiitis (GPA)*
- *Cryoglobulinemic vasculitis*
- *Antiphospholipid syndrome (APS)-associated vasculitis*

# Beyond Ischaemia : The Vasculitis Connection

- **Clinical Features**

  Vasculitis ulcers typically present with:
  - Painful, necrotic ulcers with irregular borders
  - Location: often on lower extremities (especially the ankles, shins, and feet)
  - Accompanying purpura, livedo reticularis, or nodules
  - Systemic symptoms in systemic vasculitis (fever, weight loss, arthralgia)

Early differentiation from other causes of leg ulcers (venous, arterial, diabetic, infectious, neoplastic) is crucial.

- **Diagnostic Workup**

  Clinical + Histopathological + Laboratory correlation is essential.

  Skin Biopsy

  Histology: fibrinoid necrosis, neutrophilic infiltration, leukocytoclasia

  Direct immunofluorescence (DIF) for immune deposits

- **Laboratory Tests:**
  - CBC, ESR/CRP
  - ANA, ANCA, RF, complement levels
  - Antiphospholipid antibodies

- **Management Overview**

Treatment is guided by severity and extent (cutaneous vs systemic):

- **Topical / Local Measures (for isolated skin-limited disease):**
  - Non-adherent dressings and gentle debridement
  - Pain control
  - Compression therapy (if no arterial compromise)
- **Systemic Therapy:**
  - Mild cutaneous disease: Colchicine, dapsone or low-dose corticosteroids
  - Moderate to severe/systemic disease:
  - Systemic corticosteroids
  - Immunosuppressants: azathioprine, methotrexate, mycophenolate mofetil
  - Biologics (e.g. rituximab) in ANCA-associated vasculitis or refractory cases

- **Adjunctive Strategies:**
  - •Infection prevention (watch for secondary infection)
  - •Risk factor modification (smoking cessation, control of hypertension, etc.)
  - •Vascular support and wound care teams

**Case Capsule :**
A 65 year old gentleman with a diagnosed case of left lower limb chronic limb threatening ischemia with rest pain and great toe non healing ulcer with purulent discharge.
CT Angio showing left SFA chronic total occlusion with ABI- 0.6 and Pedal acceleration time category 4.
Underwent peripheral angioplasty after optimisation.

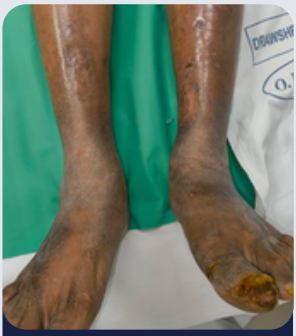

Fig 1. Initial Presentation

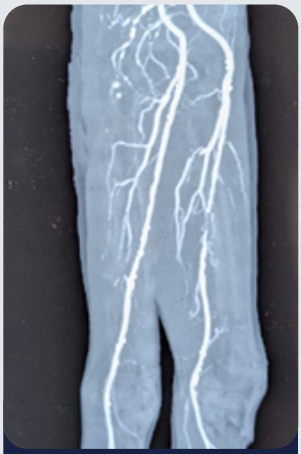

Fig 2. CT Angiogram

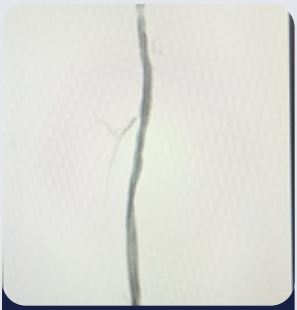

Fig 3. Post Angioplasty (SFA)

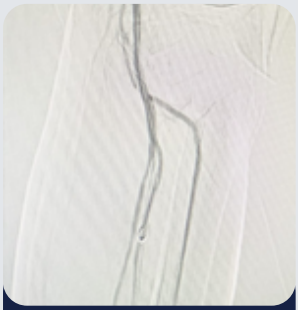

Fig 4. Post Angioplasty (Tibial)

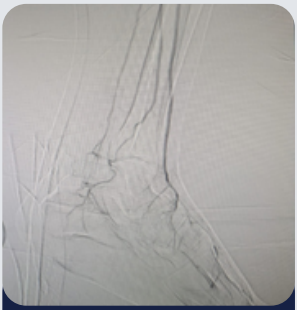

Fig 5. Post Angioplasty (Foot)

Post procedure ulcer showed signs of healing in terms of granulation but the ulcer was very painful. Hence ulcer edge biopsy was performed suggesting vasculitic ulcer . He was started on oral steroids , pain subsided in a week and ulcer healed within 2 weeks .

Fig 6. Painful Granulating Ulcer

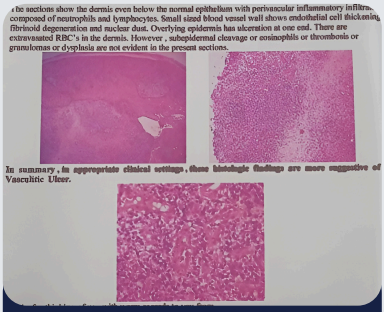


Fig 7. Vasculitic Ulcer : HPE

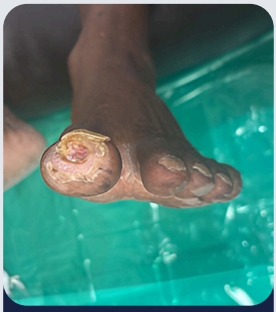

Fig 8. Pain Relief Post Steroids

**Key Takeaways**

- Always consider systemic vasculitis in patients with non-healing, painful ulcers and constitutional symptoms
- Early biopsy and immunologic workup are vital for diagnosis
- Treatment should balance inflammation control with wound healing support
- A multidisciplinary approach involving dermatology, rheumatology, wound care, and vascular surgery improves outcomes.
- Immune suppression is a double edged sword in such cases, unless proven with ulcer edge biopsy such ulcers may worsen after immune suppression.

# From Clock to CLINIC - Why Time is Tissue in Vascular Wounds

**Dr. Simit Vora**
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MUMBAI

SPOTLIGHT

Non-healing ulcers, particularly in the lower extremities, are often the result of chronic limb ischemia due to peripheral arterial disease (PAD). Commonest cause are Diabetes and smoking. Revascularisation is a key therapeutic strategy aimed at restoring adequate perfusion to promote wound healing and prevent limb loss. Timely and appropriate revascularisation is critical for improving clinical outcomes.

**When to Revascularise:**

Revascularisation should be considered when non-healing ulcers are associated with critical limb-threatening ischemia (CLTI) like :-

1. Rest pain lasting >2 weeks.
2. Non-healing wounds or ulcers on the lower limb.
3. Gangrene of toes or foot
4. Objective hemodynamic evidence of ischemia like:

· Ankle-Brachial Index (ABI) < 0.3
· Toe pressure < 30 mmHg
· Transcutaneous oxygen pressure ($TcPO_2$) < 30 mmHg.

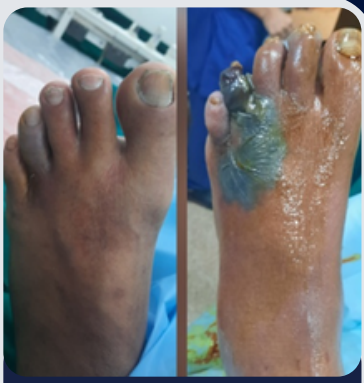


Fig 1. Initial Presentation (FOOT)

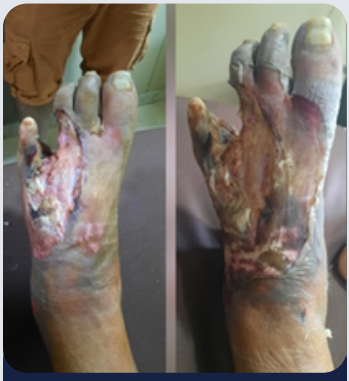


Fig 2. Post Debridement (OUTSIDE HOSPITAL)

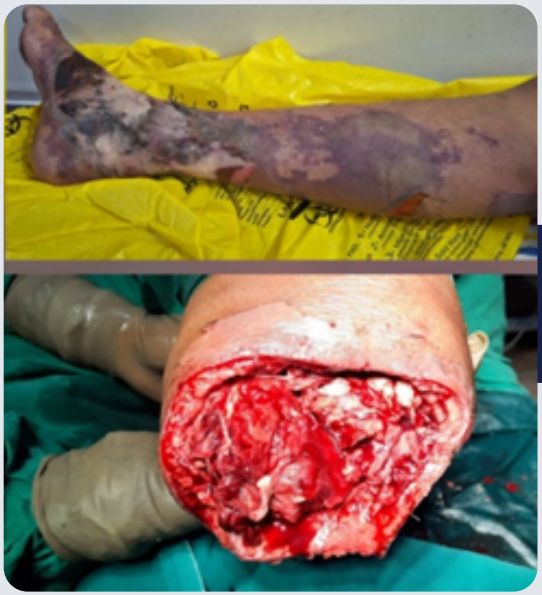


Fig 3. DEAD LIMB (Above Knee Amputation)

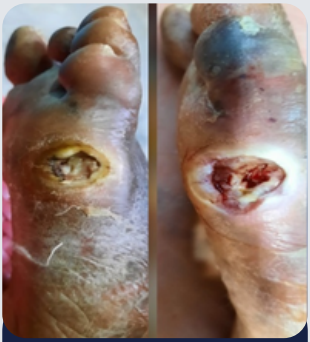


Fig 4. Presented to a Vascular Surgeon

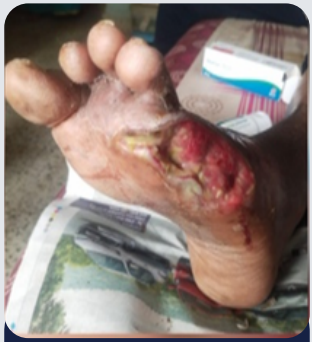


Fig 5. Post Left lower limb Angioplasty & 5th Toe Amputation

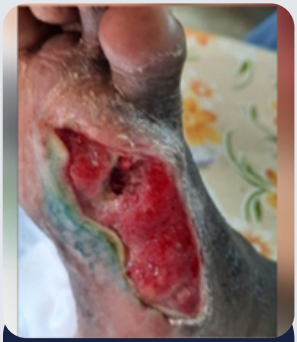


Fig 6. After 3 weeks of Surgery

**Assessment and Workup :**

1. Clinical examination: pulse examination, ulcer morphology as per WIFi classification.
2. Imaging: Duplex ultrasonography, CT angiography (CTA), MR angiography (MRA), or digital subtraction angiography (DSA).

**Timing :**

Revascularisation should be performed urgently in CLTI to prevent progression to limb loss.

In the setting of severe infection or wet gangrene, urgent or emergency revascularisation is often required as part of a multidisciplinary approach.

**How to Revascularise :**

**1. Endovascular Revascularisation (Minimally Invasive)**

**Indications:**

- First-line therapy in many patients, especially those with high surgical risk.
- Suitable for focal or segmental disease in inflow or outflow vessels.

**Techniques:**

- Angioplasty (balloon dilation)
- Stenting
- Atherectomy
- Drug-coated balloons

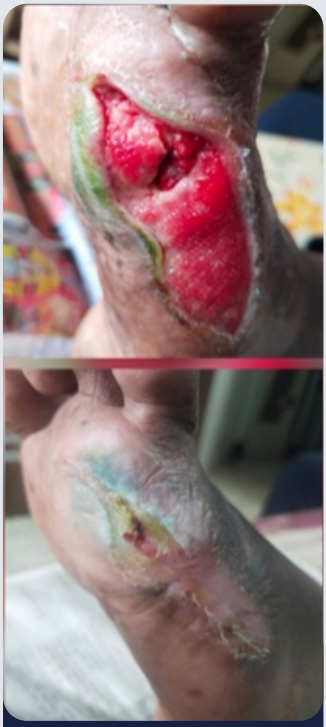


Fig 7. Post Wound Healing

**Advantages:**

- Minimally invasive
- Shorter recovery
- Repeatable

**Limitations:**

- Poor patency in long, complex lesions
- May require re-intervention

**2.Surgical Revascularisation (Bypass Surgery)**

**Indications:**

- Long, complex occlusions (e.g., long segment superficial femoral artery or tibial occlusions)
- Failed endovascular therapy
- Suitable anatomy for bypass

**Techniques:**

- Autogenous vein bypass (preferred)
- Prosthetic grafts if no vein available

**Advantages:**

- Durable long-term patency, especially with vein graft.

**Limitations:**

- Invasive with higher perioperative risk
- Requires good conduit and target vessel

**3. Hybrid Procedures:**

- Combination of endovascular and open surgery
- Tailored to complex, multilevel disease
- Increasingly used in tertiary vascular centers

**Post-Revascularisation Considerations:**

- Wound care and debridement are crucial adjuncts.
- Glycemic control, smoking cessation, and antiplatelet therapy optimize outcomes.
- Surveillance with clinical follow-up and imaging is important for early detection of restenosis.

**Guideline Recommendations:**

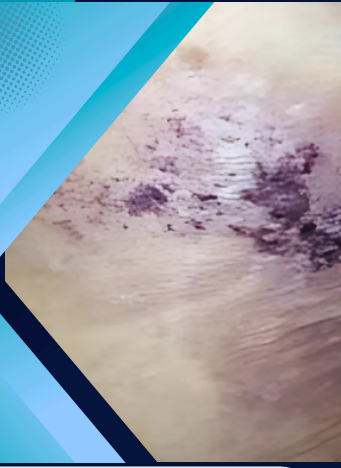
- The Society for Vascular Surgery (SVS) and American Diabetes Association (ADA): recommend that vascular assessment and intervention be conducted early in diabetic foot management.
- BASIL Trial (NEJM, 2005): Revascularization improves survival and limb outcomes.
- The Global Vascular Guidelines (2019) emphasize revascularization as the primary determinant of limb salvage in chronic limb-threatening ischemia (CLTI).

**Conclusion:**

- Revascularisation plays a central role in the management of non-healing ischemic ulcers.
- A comprehensive and multidisciplinary evaluation is essential to determine the timing and method of revascularisation.
- Endovascular approaches are increasingly first-line, but surgical bypass remains crucial in selected patients.
- Timely revascularisation is often limb-saving and improves the quality of life.

# CKD, DM & Ischaemia
# The Triple Threat

**Dr. Piyush Jain**
Vascular & Endovascular Surgeon
NAVI MUMBAI



## EVIDENCE

**Chronic Kidney Disease (CKD)** and **Diabetes Mellitus (DM)** often go hand-in-hand, creating a double challenge for both patients and healthcare providers. Diabetes is the leading cause of CKD and almost 4 in 10 people with diabetes will develop kidney problems over time. When these two conditions coexist, the body's ability to heal wounds—especially on the feet—is severely compromised. The reasons are many: weakened immunity, poor blood circulation, nerve damage and slower tissue repair all play a part.

One of the most serious complications is the Diabetic Foot Ulcer (DFU). These wounds often develop due to a mix of **nerve damage** (neuropathy), **reduced blood flow** (ischemia) and **minor injuries** that go unnoticed. Infections, including those reaching the bone (osteomyelitis) are common. The risk is highest for people who have had a previous ulcer or amputation, those on dialysis and anyone with both neuropathy and poor circulation. Unfortunately, CKD itself increases the likelihood of major amputation in diabetic patients, with dialysis patients facing the greatest danger.

Early and accurate assessment is key. We often use the WIfI system (Wound, Ischemia, and foot Infection) to classify ulcers and guide treatment. Blood flow can be checked with an **Ankle-Brachial Index (ABI)**, but because CKD often causes calcified arteries, this test can sometimes be misleading. In such cases, a **transcutaneous oxygen measurement ($TcPO_2$)** or **toe pressure** is more reliable. Regular lab checks—such as blood sugar (HbA1c), kidney function (eGFR), urine protein (albuminuria), and infection markers (CRP/WBC)—help track both the underlying disease and wound healing progress.

# CKD, DM & Ischaemia
# The Triple Threat

The best treatment plans involve a team approach. National and international guidelines (NICE, SVS, ESVS, IWGDF) recommend integrated foot care services, clear referral pathways and input from specialists in wound care, diabetes and vascular surgery. Local wound care still follows the golden rules: remove dead tissue (debridement), keep the wound moist but clean, control infection and reduce pressure on the foot through special footwear, casts or walkers. In selected cases, advanced therapies like Negative Pressure Wound Therapy (NPWT), revascularisation within 4–6 weeks, hyperbaric oxygen, growth factors and skin substitutes can improve healing.

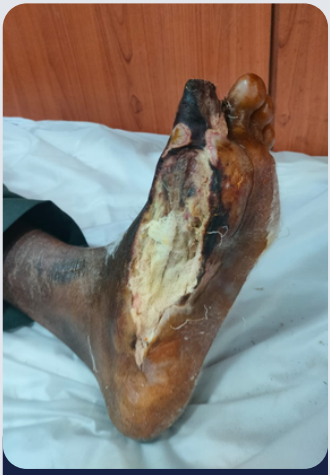


Fig 1. On Presentation Large Non Healing Wound

**Case Capsule :**
A 60-year-old Diabetic patient, on dialysis for 3 years presented with gangrene of the left Hallux with large non healing wound. He had non palpable foot pulses. He underwent an angioplasty of the tibial arteries and subsequent great toe amputation. With daily dressing, front foot offloading and strict sugar control, the wound healed in 3 months' time.

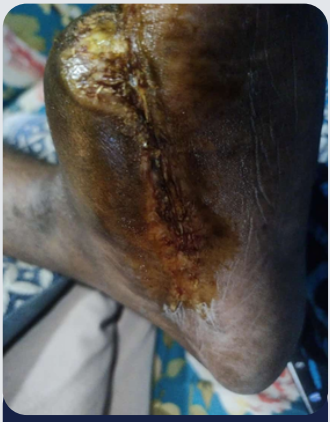


Fig 2. Post Intervention & Daily Dressing

The outlook for patients with both CKD and diabetes can indeed be challenging. Wounds tend to heal much more slowly due to the combined effects of poor circulation, nerve damage and reduced immune function.

# CKD, DM & Ischaemia
# The Triple Threat

Even when a diabetic foot ulcer heals, there is a significant risk of recurrence, especially if the underlying risk factors are not addressed. For patients on dialysis or those who have recently experienced a DFU, the stakes are even higher, as the likelihood of severe infection or amputation rises sharply.

However, this is not a hopeless scenario. With structured, proactive follow-up care, outcomes can be improved dramatically. Regular wound checks, close monitoring of blood sugar and kidney function, and timely intervention for any signs of infection can prevent minor issues from becoming major complications. **Preventive strategies—such as routine foot inspections, wearing appropriate protective footwear, controlling blood pressure and blood sugar and stopping smoking**—play a critical role in keeping patients out of the hospital and on their feet.

Patient education is equally vital. When individuals understand how to recognize early warning signs, practice good foot hygiene and seek medical attention promptly, the chances of avoiding serious outcomes increase significantly. Multidisciplinary care pathways, involving diabetologists, nephrologists, vascular surgeons, podiatrists and wound care specialists, ensure that treatment is not only timely but also comprehensive.

**Bottom line:** CKD and diabetes together create a perfect storm for foot complications. Yet, this storm can be navigated successfully with the right approach. Evidence-based guidelines, coordinated team-driven care and regular monitoring can protect the feet, promote faster and more complete healing and preserve mobility. By combining medical expertise with patient involvement and preventive planning, it is possible to reduce amputations, improve quality of life and keep patients active and independent for longer.

# The Evolving Evidence of Anticoagulation in Wound Healing ..

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## EVIDENCE

In the management of chronic wounds, especially those of vascular origin, our approach has traditionally centered around restoring perfusion, controlling infection and providing optimal wound care. However, emerging evidence in recent years has brought attention to another important factor in the healing process: thrombotic burden in microcirculation and how anticoagulation may help modulate it to improve outcomes.

**The Pathophysiological Link: Thrombosis and Impaired Healing**

Chronic wounds such as diabetic foot ulcers, venous leg ulcers and ischemic wounds from peripheral arterial disease often demonstrate microvascular thrombosis or sluggish blood flow in the wound bed. These microthrombi impair oxygen and nutrient delivery, increase local inflammation and delay tissue regeneration. In addition, platelet activation and fibrin deposition can create a persistent pro-thrombotic and inflammatory environment, further complicating healing.

This understanding has prompted exploration into the use of anticoagulants—not just for preventing deep vein thrombosis or treating peripheral arterial disease—but also as a supportive therapy in wound healing.

**Evidence Supporting Anticoagulation in Wound Management**

Several small-scale studies and observational reports have shown potential benefits of anticoagulation in selected patient populations with chronic wounds. Here are a few highlights:

# The Evolving Evidence of Anticoagulation in Wound Healing ..

- **Venous leg ulcers (VLU):** Patients with active VLUs often show evidence of hypercoagulability and venous stasis. A few randomized studies suggest that adding low-dose anticoagulation (especially with low molecular weight heparin) to compression therapy may accelerate healing rates, particularly in patients with underlying thrombophilia.
- **Diabetic foot ulcers (DFU):** In some cases of infected or non-healing DFUs with underlying peripheral arterial disease, anticoagulation (especially with rivaroxaban or apixaban) has been shown to improve microvascular flow and reduce major adverse limb events (MALE).
- **PAD-related ischemic ulcers:** The VOYAGER PAD trial (2020) was a game-changer. It studied low-dose rivaroxaban (2.5 mg twice daily) plus aspirin versus aspirin alone in patients with symptomatic PAD undergoing revascularization. The combination not only reduced cardiovascular and limb events but also appeared to lower the rate of wound complications and amputations in a subgroup analysis.

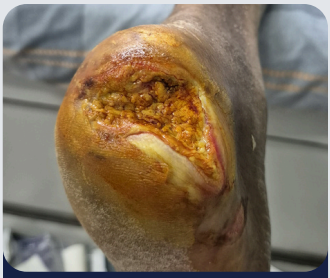


Fig 1. Non Healing Heel Wound

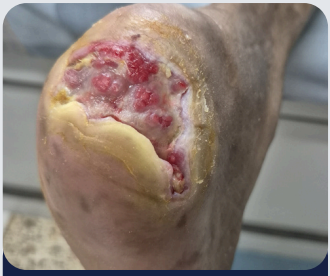


Fig 2. After 6 weeks of Anticoagulation and Wound Care

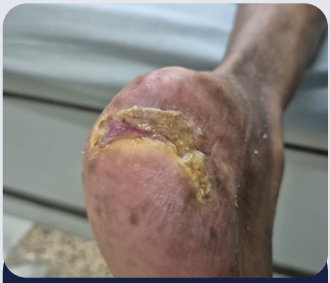


Fig 3. After 12 weeks of Anticoagulation and Wound Care

[Fig 1 : Right heel wound, desert foot with no direct blood supply going to the heel - no angioplasty performed]
[Fig 2,3 : Post 6 and 12 weeks of Regular Wound Care, Offloading, Oral Anticoagulation and Debridement]

- **Heparin and wound inflammation:** Unfractionated heparin and LMWHs have anti-inflammatory properties beyond anticoagulation. Topical heparin has been studied in animal models and small human trials, suggesting reduced periwound inflammation and improved granulation tissue.

**Practical Considerations: Who Should Get It?**

Anticoagulation is not indicated for all patients with wounds. However, it can be considered in the following situations:

1. **Wounds with underlying PAD after revascularization** – Low-dose anticoagulation (as in the VOYAGER protocol) may be used post-intervention to reduce thrombotic risk and support healing.
2. **Recurrent venous leg ulcers or VLUs with known thrombophilia** – A case can be made for long-term anticoagulation especially if compression therapy alone is insufficient.
3. **Diabetic foot ulcers with underlying ischemia or thrombosis** – In selected patients with moderate PAD not suitable for revascularization, oral anticoagulants may help improve microvascular circulation.
4. **Patients with history of VTE or atrial fibrillation who are already on anticoagulants** – Wound healing should be monitored as these patients may actually fare better in terms of healing outcomes than matched non-anticoagulated controls.

**Protocols and Recommendations (as per recent data)**

Though there is no unified guideline solely focused on anticoagulation for wound healing, several consensus statements and trial results help shape our protocols:

- Post-revascularization in PAD:
  **VOYAGER PAD protocol**
  - Rivaroxaban 2.5 mg BD + Aspirin 75 mg OD
  - Duration: Up to 36 months (with individual risk-benefit assessment)

- **Venous leg ulcers:**
  - In patients with recurrent ulcers and thrombophilia, long-term oral anticoagulation (DOACs preferred) may be considered, especially in cases where compression alone is not sufficient.
- **DFUs with ischemic component:**
  - In selected patients, especially those with concomitant atrial fibrillation or coronary artery disease, consider maintaining therapeutic anticoagulation using DOACs or LMWH.
- **Monitoring:**
  - Regular wound assessment, INR checks if on warfarin and vigilance for bleeding signs are essential.
  - Avoid combining full-dose anticoagulation with dual antiplatelets unless clearly indicated.

**Cautions and Contraindications**

It's critical to weigh the risk of bleeding against the potential benefit in wound healing. Anticoagulation should be avoided or used with great caution in:

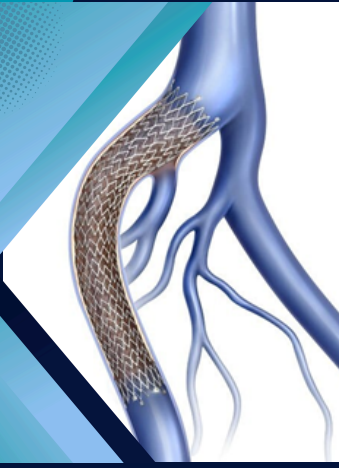
- **Patients with active wound bleeding or hematoma**
- **Those with recent major surgery or intracranial hemorrhage**
- **Severe thrombocytopenia or coagulopathy**
- **Patients on concurrent NSAIDs or dual antiplatelet therapy (unless justified)**

Also, topical anticoagulants like heparin gel are not standard of care and should not replace systemic therapy in appropriately selected patients.

The intersection of thrombosis and wound healing represents a promising but complex area of vascular care. As more evidence accumulates, especially in the form of large-scale randomized trials, we may see personalized protocols that use anticoagulants not only for thromboembolic prevention but also to improve wound outcomes.

# All Venous Ulcers are not due to VARICOSE VEINS...

**Dr. Ishita Jethwa**
Vascular & Endovascular Surgeon
MUMBAI



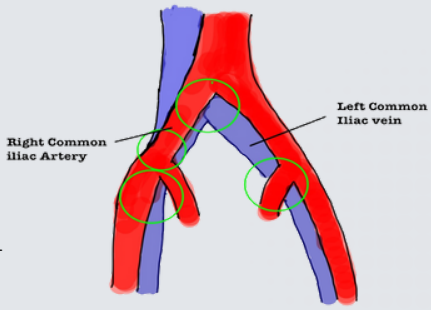
**PULSE POINT**

A 47-year-old gentleman presented to our OPD with a history of a non-healing wound just above the medial malleolus of his left leg, persisting for 8 years. The wound was not very painful but was associated with itching. He reported undergoing laser surgery for varicose veins on three separate occasions at three different centers, as well as one sitting of sclerotherapy at another center. Despite these interventions, the wound failed to heal.
He was otherwise healthy, with no comorbidities and a non smoker. Distal pulses were well palpable and no varicosities were visible in his legs apart from the pigmented lesion shown in Fig. 1. Duplex venous scanning was normal, as was the MR venogram performed to rule out any iliac vein lesion.

He subsequently underwent intravascular ultrasound (IVUS) under local anesthesia, which revealed a 77.5% area reduction in his left iliac vein due to compression by the overlying artery—a classical finding of May-Thurner Syndrome, also termed Non-thrombotic Iliac Vein Lesion (NIVL).

He was subjected to iliac vein stenting and compression bandages and the ulcer healed in 6 weeks and remained healed at 2 yrs.

A **Non-thrombotic Iliac Vein Lesion** (NIVL) is defined as an extrinsic compression or narrowing of the iliac vein—most commonly the left common iliac vein—without the presence of a blood clot (thrombosis).




Areas where artery can compress vein in pelvis.

# All Venous Ulcers are not due to VARICOSE VEINS...

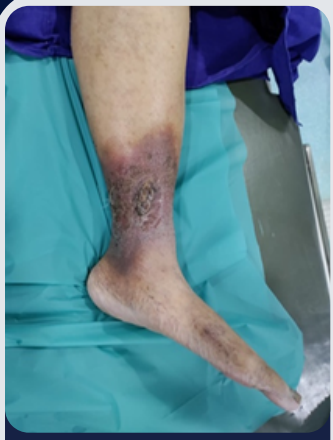


Fig 1. Venous Ulcer

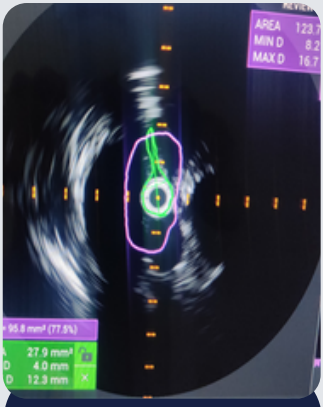



Fig 2. IVUS Showing Narrowing of Iliac Vein

This compression typically occurs where the iliac vein is pressed between the right common iliac artery and the lumbar spine, leading to venous lumen stenosis characterized by vessel wall changes such as fibrosis and intraluminal webs or spurs.

**Non-thrombotic iliac vein lesions (NIVLs)**—most often caused by extrinsic compression of the iliac vein, such as in May-Thurner syndrome—are a recognized and significant cause of chronic venous insufficiency (CVI) and can directly contribute to the development and persistence of venous leg ulcers.

- **Pathophysiology:** NIVLs lead to venous outflow obstruction without prior thrombosis, resulting in venous hypertension in the lower limb. This chronic elevation in venous pressure can cause symptoms ranging from leg swelling and pain to skin changes and, in severe cases, venous stasis ulcers.
- **Prevalence:** Studies report that NIVLs are present in 53% to 87% of patients with advanced (CEAP class 4–6) venous disease, which includes those with skin changes and ulcers.
- **Clinical Presentation:** Patients may be asymptomatic or present with a spectrum of symptoms including leg edema, pain, symptoms of pelvic congestion in young women, varicose veins, and venous ulcers. The severity of symptoms is influenced by the degree of obstruction and individual patient factors.

# All Venous Ulcers are not due to VARICOSE VEINS...

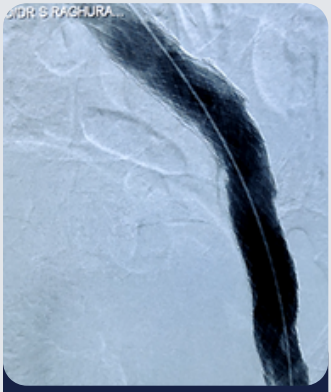



Fig 3. Left Iliac Vein Stented

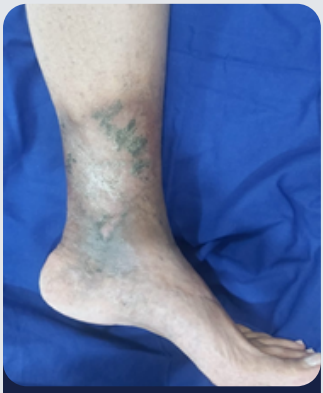


Fig 4. Healed Ulcer after Stenting

- **Classification of NIVL**

| | |
|---|---|
| Type 1 | NIVL limited to the iliac vein (classic extrinsic compression) |
| Type 2 | Chronic venous obstruction involving the iliac vein segment |
| Type 3 | Obstruction extending into the iliofemoral segment above the common femoral vein confluence |
| Type 4 | Obstruction involving one main inflow vein below the inguinal ligament (femoral vein or deep femoral vein) |
| Type 5 | Obstruction involving both main inflow veins (femoral and deep femoral veins) |

- **Diagnosis:** Diagnosis relies on a combination of clinical assessment and imaging (ultrasound, CT, MR venography, intravascular ultrasound), as anatomic compression can also be found in asymptomatic individuals.
- **Treatment:** In selected patients, endovascular stenting of the compressed iliac vein can relieve obstruction, which has shown to significantly improve ulcer healing rates, reduce healing time, and improve quality of life in patients with venous ulcers

In summary, NIVL are a crucial and often under-recognized cause of venous ulcers. Their identification and endovascular treatment, particularly stenting, can lead to substantial improvements in ulcer healing and patient outcomes.

# HEALing the HEEL WOUND THE RIGHT WAY ....


**Dr. Rohit Gupta**
Vascular & Endovascular Surgeon
NAGPUR



**Dr. Kartik Gupta**
Vascular & Endovascular Surgeon
NAGPUR


PULSEPOINT

A 64-year-old female from Rajasthan, known to have diabetes mellitus for the past 20 years and on insulin therapy, presented with a chronic, non-healing ulcer over her left heel. She had a significant cardiac history of coronary artery disease, with a severely reduced ejection fraction of 20%. She also suffered from peripheral arterial disease (PAD).

The heel ulcer had been severely painful and persistent for two years, during which she underwent **multiple debridements**, repeated **peripheral angioplasties** and a **free flap rotation** to cover the wound. In addition, she had received multiple sessions of platelet-rich plasma (PRP) and stem cell therapy, but her symptoms progressively worsened. She was eventually advised to undergo a below-knee amputation.

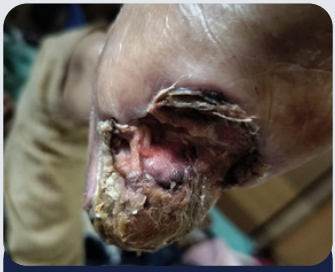


Fig 1. Heel Wound on Presentation

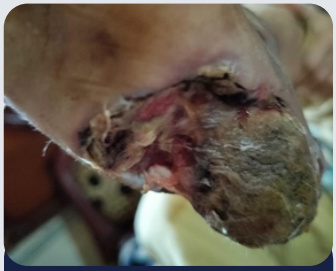


Fig 2. Heel Wound (Pre-Debridement)

When she presented to our outpatient department, the ulcer was ischemic, infected and severely painful. There was exposed calcaneal bone, a sloughed-out Achilles tendon and copious foul-smelling purulent discharge. The ulcer was classified as **Wagner Stage 3**.

On vascular examination, her left popliteal and bilateral pedal pulses were absent. The **left ankle-brachial index (ABI) was 0.5, the toe-brachial index was not recordable and transcutaneous partial oxygen pressure ($TcPO_2$) was only 20 mmHg.** Laboratory investigations showed anemia (hemoglobin 7 g/dL), leukocytosis (TLC 25,400/cu mm), elevated HbA1c of 9.0% and a CRP of 97 mg/L. X-ray of the foot revealed erosion of the calcaneus, and CT angiography showed severe infrapopliteal atherosclerotic occlusive disease with significant in-stent restenosis in the previously placed left popliteal artery stent.

We took up the case as a limb salvage challenge. Emergency minimal debridement was done to drain pus and reduce infection. Sepsis was controlled with intravenous antibiotics and tight glycemic management. Once stabilized, the patient underwent left popliteal and below-the-knee (BTK) angioplasty using drug-eluting balloons (DEBs). **Post-procedure, her $TcPO_2$ improved to 58 mmHg.**

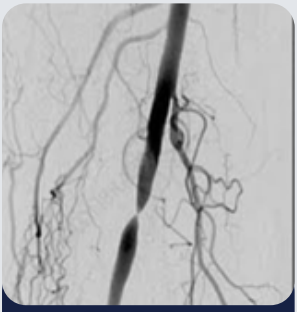


Fig 3. Pre Angioplasty (SFA)

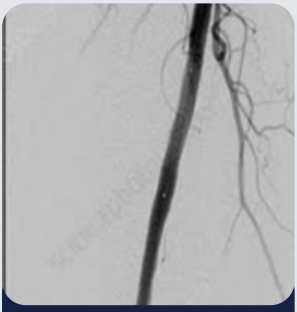


Fig 4. Post Angioplasty (SFA)

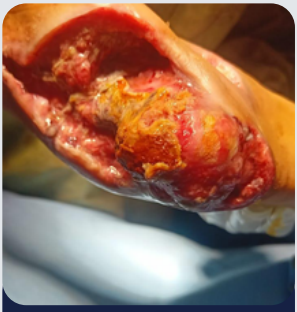


Fig 5. Post Debridement (Heel)

Wound care was continued by the podiatry team with serial debridements and negative pressure wound therapy (NPWT). As the wound began to granulate, the exposed bone was covered with a dermal matrix, followed by split-thickness skin grafting.

Over the course of 12 weeks, the limb was successfully salvaged. The patient is now ambulatory with the assistance of an ankle-foot orthosis (AFO).

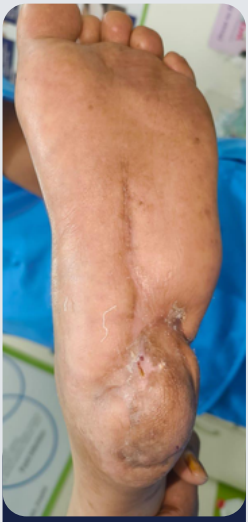
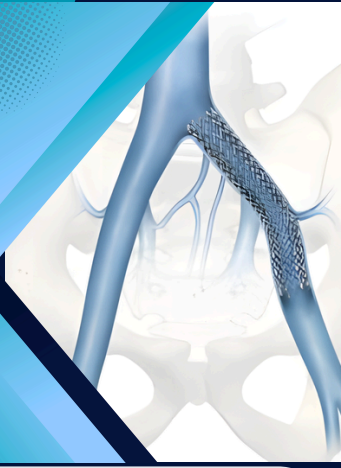


Fig 6. Healed Heel Wound

Fig 7. Walking with Ankle Foot Orthosis

# Young Man - OLD PROBLEM Approach to Intravascular Ultrasound Guided Venous Stenting

**Dr. Ritesh Gaikwad**
Vascular & Endovascular Surgeon
THANE



PULSE POINT

Chronic venous insufficiency in young adults is often underestimated or incorrectly attributed to superficial vein disease. Yet, for many patients, especially those with recurrent symptoms despite previous interventions, the real pathology lies deeper. This case highlights how a young patient with a seemingly routine venous ulcer revealed a much more complex underlying problem — one that could only be effectively managed using intravascular ultrasound (IVUS) and iliac vein stenting.

**Introduction: A Familiar Story with an Unfamiliar Ending**

Chronic leg ulcers in younger patients are uncommon but not unheard of. However, what's more unusual is when these ulcers persist or recur despite repeated superficial vein interventions. This is where the story of a 26-year-old man becomes a lesson in looking beyond the obvious.

The patient, a young male with a 4-year history of varicose veins, had undergone two sessions of endovenous laser ablation (EVLA) on both limbs and even an open surgical procedure on the right side. Despite all this, he continued to suffer from swelling, pain, and eventually a non-healing ulcer that brought him limping into our vascular surgery department.

The initial clinical suspicion was straightforward — recurrent superficial venous insufficiency. But the persistence and severity of symptoms, especially the ulceration, hinted at a more proximal cause that had likely been missed in previous assessments.

**Reassessing the Problem: The Role of Proximal Venous Outflow Obstruction**

# Young Man - OLD PROBLEM Approach to Intravascular Ultrasound Guided Venous Stenting

A thorough clinical examination revealed classic signs of chronic venous insufficiency — edema, varicosities, and a painful medial leg ulcer. However, the pattern and chronicity of his symptoms suggested an underlying outflow obstruction, particularly in the iliac veins, which often remains undiagnosed in such cases.

Initial non invasive test ( physiological testing) like *venous reflux time and MVO/ SVC ratio* and imaging with an MR venogram suggested iliac vein compression. However, MR and CT venography, although useful for screening, often fall short in evaluating iliac vein pathology due to the collapsible, thin-walled nature of veins. In this case, the imaging showed a **"pancaking" of the iliac vein** — a classic feature of May-Thurner syndrome (left iliac vein compressed by the overlying right iliac artery and spine) or non thrombotic iliac vein lesion (NIVL).

But as we know, conventional imaging provides only indirect clues and for this, IVUS proved indispensable. IVUS allows direct visualisation of the vessel lumen, wall, and surrounding structures. Unlike venograms, which rely on contrast filling and can miss non-occlusive lesions, IVUS gives real-time, high-resolution images that can quantify compression, determine cross-sectional area, and guide precise stent sizing and placement.

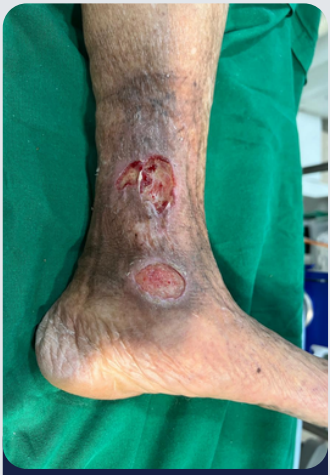


Fig 1. Venous Ulcer

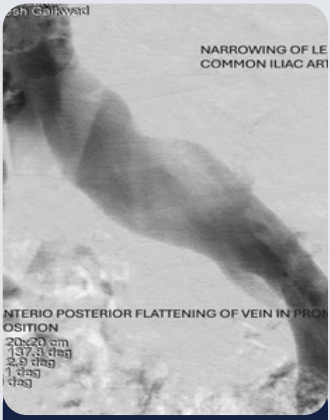



Fig 2. Conventional Venogram - NOT USEFUL

# Young Man - OLD PROBLEM Approach to Intravascular Ultrasound Guided Venous Stenting

We performed *IVUS in both prone and supine positions*, revealing significant extrinsic compression of the left iliac vein. The degree of narrowing was clearly underestimated on MR venography, reaffirming the superiority of IVUS in such cases. With this definitive diagnosis in hand, we proceeded to intervene.


**Fig 3. IVUS Showing Vein Compression**

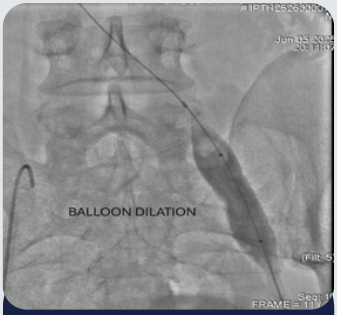



**Fig 4. Balloon Venoplasty**

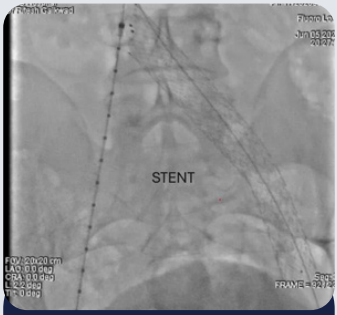



**Fig 5. Venous STENT**

The patient underwent balloon angioplasty followed by deployment of a dedicated self-expanding venous stent in the compressed segment of the left iliac vein. The procedure was uneventful, completed in under an hour, and the patient was mobilized within two hours postoperatively.

The immediate postoperative period was remarkable. By the next day, he reported over 90% reduction in swelling and pain. Patient was discharged with rapid wound healing already underway — a dramatic turnaround after years of ineffective treatment.

This case underscores a crucial point — young patients with recurrent varicose veins and leg ulcers may have a proximal venous outflow obstruction that is often overlooked by physician and treating physician.

In such scenarios, failing to consider iliac vein compression — often seen in variants of May-Thurner syndrome and NIVL — can delay definitive treatment. Even when suspected, conventional imaging may not detect it reliably. This is where IVUS becomes a critical diagnostic and therapeutic tool.

More importantly, treating the root cause with iliac vein stenting, particularly using modern, dedicated venous stents, offers excellent results. These stents are designed to handle the dynamic, compressive environment of the pelvis and provide long-term patency with minimal complications.

*As endovenous interventions for varicose veins become increasingly common, we must remain vigilant for underlying pathologies when patients present with recurrence or poor response to treatment.* Chronic venous ulceration in a young adult should never be taken lightly or assumed to be merely a failure of superficial ablation.

Instead, we need a shift in our diagnostic algorithm — one that routinely includes evaluation of the iliocaval venous system, especially in refractory cases. The cost and expertise required for IVUS may be higher, but so is the value it brings in clarity of diagnosis and precision of therapy. The key is in recognizing the problem early and not stopping at the superficial layer — quite literally.

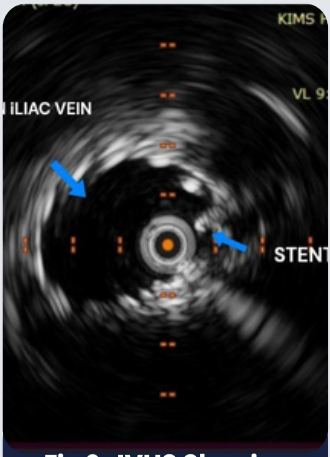



**Fig 6. IVUS Showing good opening up of the vein**

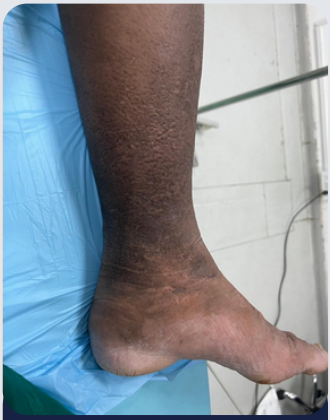


**Fig 7. Post OP Wound Healed**

**Conclusion: Looking Beneath the Surface**

This case reminds us that good outcomes in vascular surgery often depend not just on what we see, but on where we look. For this young man, years of pain and disability were resolved not by repeating previous treatments but by reevaluating the diagnosis and treating the true cause: outflow obstruction .

IVUS-guided venous stenting is more than just a technical advancement — it's a paradigm shift. And for patients like this, it means the difference between chronic suffering and rapid, lasting relief.

Let this be a reminder that even in the young, old venous problems can hide in plain sight — and that a deeper look can bring lasting healing.

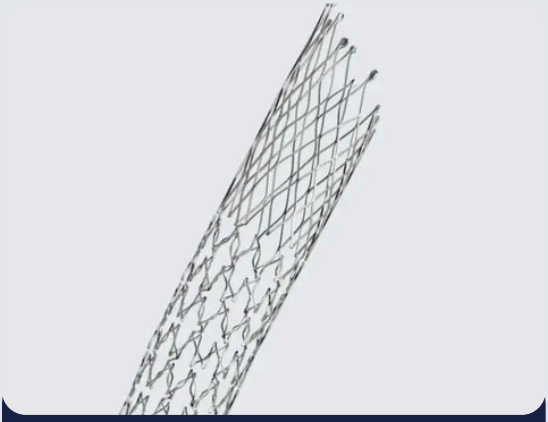


Fig 8. Example of a VENOUS STENT

# Treating Infection and Insufficiency TOGETHER

**Dr. Bhushan Shinde**
Vascular & Endovascular Surgeon
KOLHAPUR

PULSE POINT

**Introduction**

Venous ulcers represent a challenging and often frustrating stage in the continuum of chronic venous insufficiency (CVI). For many patients, the distress is not primarily due to pain, but rather the persistent exudative nature of the wound. This constant drainage not only interferes with daily activities but also creates a significant psychosocial burden.

As vascular surgeons, it is imperative to recognise that ulcer healing is not merely a matter of dressing techniques—it is the result of addressing both the underlying pathophysiology and superimposed complications such as infection.

**Case Presentation**

A 60-year-old male, a schoolteacher by occupation, was referred for the evaluation of a non-healing wound on the lateral aspect of the right leg.

- **Past history:** The patient had suffered cellulitis of the same leg in the past and underwent surgical debridement through two longitudinal incisions—one medial and one lateral.
- **Post-debridement course:** Both incision sites developed non-healing exudative wounds.
- First intervention: Considering the visible varicosities and non-healing nature, the patient underwent endovenous laser therapy (EVLT) of the long saphenous vein (LSV) at a vein centre. Following this, the medial wound healed completely, but the lateral wound persisted in an exudative state, requiring frequent dressings.

When the patient presented to our unit, his primary concern was persistent wound discharge from the lateral site.

# Treating Infection and Insufficiency TOGETHER

**Initial Assessment**

**On clinical examination:**

- The lateral wound appeared non-healing and moist, with moderate exudate.
- Few residual varicosities were visible in the distribution of both the LSV and the short saphenous vein (SSV).

**Investigations:**

- Wound swab for culture and sensitivity grew Methicillin-Resistant Staphylococcus Aureus (MRSA).
- Colour Doppler ultrasound at this stage was deferred until after infection control.

**Step 1 – Infection Control**

Given the MRSA-positive culture, targeted antibiotics were initiated based on sensitivity.

- Systemic antibiotics were prescribed and monitored.
- Local wound care involved absorbent dressings to control exudate.
- Compression therapy was applied in accordance with the degree of venous insufficiency to aid venous return and reduce oedema.

Following optimal antibiotic therapy and compression, there was a visible reduction in exudate and healthy granulation tissue began to appear.

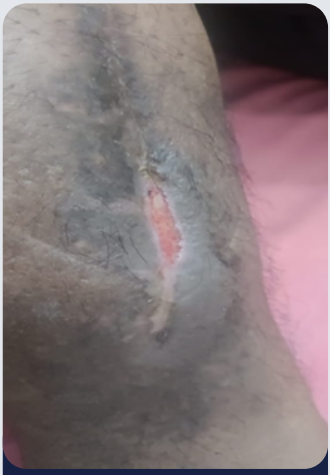


Fig 1. Venous Ulcer PRE - EVLT

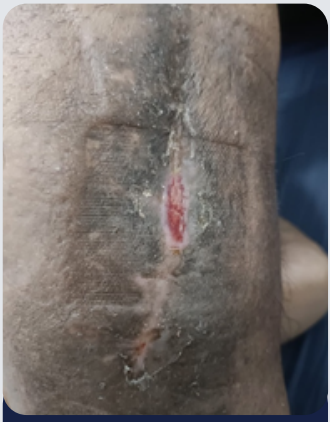


Fig 2. Venous Ulcer Immediately POST EVLT

# Treating Infection and Insufficiency TOGETHER

**Key Learning Point:**
A persistent infection is one of the most common reasons for delayed ulcer healing. In this patient, MRSA eradication was a crucial first step before proceeding to further venous interventions.

**Step 2 – Identifying Residual Venous Insufficiency**
Once the wound was cleaner and less exudative, a comprehensive colour Doppler evaluation performed showed the following:

- Main LSV: Previously ablated, no reflux.
- Accessory LSV: Significant reflux present.
- SSV: Reflux present.
- Deep venous system: No evidence of reflux or DVT.

Residual reflux in untreated segments was likely contributing to the persistence of ulceration and risk of recurrence.

**Step 3 – Definitive Venous Intervention**
Based on these findings, EVLT was planned for both the refluxing accessory LSV and SSV.

- The procedure was performed as day-care surgery under tumescent anaesthesia.
- The patient tolerated the intervention well, with early mobilisation encouraged.

Post-EVLT care included:

- Continued compression therapy to optimise venous return.
- Absorbent dressings until complete epithelialisation.

Over subsequent weeks, the wound healed fully, and the patient resumed normal activities.

**Discussion – Why This Case Matters**
This case underlines several important clinical principles in the management of venous ulcers:

# Treating Infection and Insufficiency TOGETHER

1. Dual Pathology – Infection and Reflux
    - Wound infection, especially with resistant organisms such as MRSA, can independently delay healing even if the venous pathology is addressed.
2. Comprehensive Care by a Single Clinician
    - When the same clinician is skilled in both wound management and endovenous interventions, treatment is streamlined and delays are minimised.
3. Role of Evidence-Based Practice
    - Landmark trials such as EVRA and ESCHAR have demonstrated that early treatment of superficial venous reflux reduces ulcer healing time and decreases recurrence rates when combined with optimal compression therapy.
4. Importance of Surgeon-Performed Duplex
    - A detailed colour Doppler examination by the treating surgeon allows for a targeted, personalised intervention plan.
5. Compression Therapy – The Cornerstone
    - Regardless of surgical intervention, compression remains the single most effective adjunct in managing venous ulcers.

**Conclusion**

The successful healing of a venous ulcer requires a multifaceted approach—infection control, correction of venous reflux, meticulous wound care, and sustained compression therapy. In this patient, targeted antibiotic therapy for MRSA and subsequent EVLT of residual refluxing veins led to complete healing and reduced recurrence risk.

The central lesson is clear: understanding the pathophysiology and addressing all contributing factors—both local and systemic—is essential for optimal outcomes in venous ulcer management.

# Whats New in Diabetic FOOT.... With VERAFLO ...

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Vascular & Endovascular Surgeon
PUNE

## TOOL TALK

**Case Summary**

**1. Personal History:**
A 58-year-old male patient with a history of right first toe amputation two years ago. He recently developed wet gangrene of the right second toe, for which a partial toe amputation was performed at an outside hospital.

**2. History of Other Relevant Medical Illness:**
Known case of diabetes mellitus.

**3. Drug History:**
On human insulin, adjusted as per blood sugar levels.

**4. Local Examination:**
The patient presented with necrotizing fasciitis of the right leg, showing early signs of septic shock. He had a non-healing ulcer on the right foot for the past 10 days. Pedal pulses were palpable.

**5. Relevant Investigations:**
CBC, coagulation profile, blood sugar levels, and wound culture. (Post 3 sessions of Veraflo therapy, wound culture was negative.)

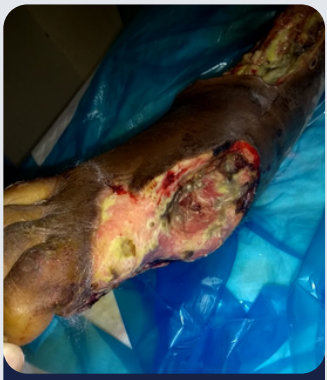


Fig 1. Initial Presentation (FOOT)

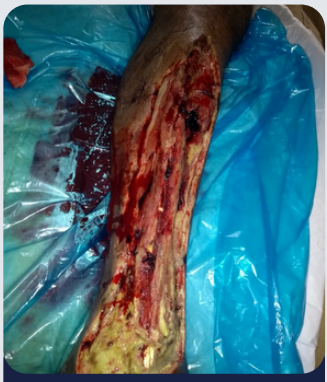


Fig 2. Initial Presentation (CALF)

**6. Wound Closure Method Used:**

The patient underwent a Ray's amputation of the right great toe, followed by multiple sessions of debridement. Initially, one session of VAC Granufoam dressing was applied. Although some progress was observed, a layer of slough persisted. Hence, VAC was converted to Veraflo therapy.

**Veraflo Settings:**

Cycle: 3.5 hours VAC with 6 minutes of instillation using 25 ml of normal saline.

After 4 sessions of Veraflo therapy, healthy granulation tissue developed over the right leg and the amputation sites of the first and second toes.

**Result:**

Wound bed was adequately prepared for skin grafting. The patient was referred for split-thickness skin grafting (STSG).

Fig 3. After 1st VAC (72 hrs)

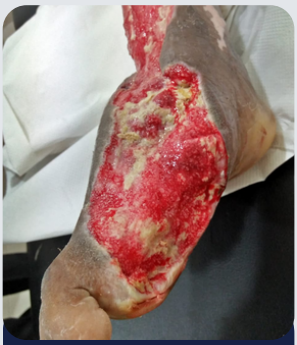


Fig 4. After 1st Veraflo (VAC) - FOOT

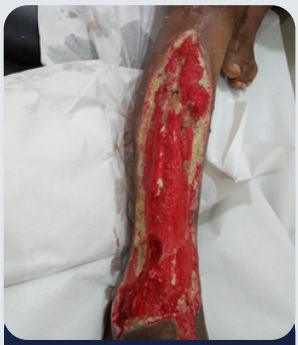


Fig 5. After 1st Veraflo (VAC) - CALF

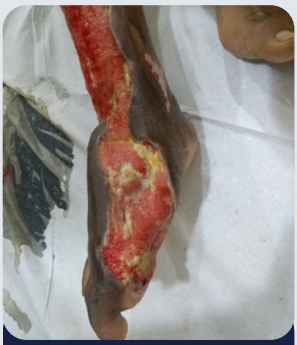


Fig 6. After 2nd Veraflo (VAC) - FOOT

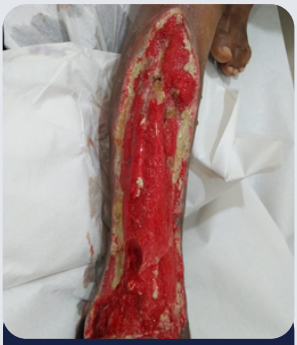


Fig 7. After 2nd Veraflo (VAC) - CALF

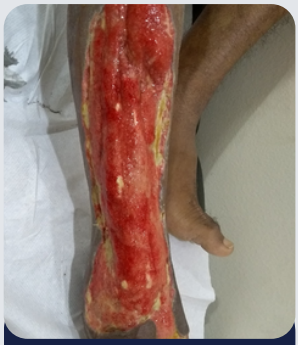


Fig 8. After 3rd Veraflo (VAC) - CALF

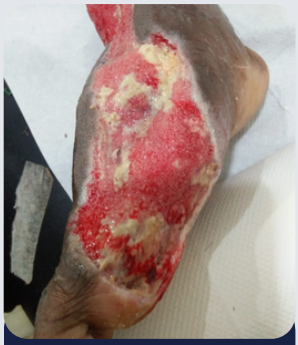


Fig 9. After 3rd Veraflo (VAC) - FOOT

**Background**

Many diabetic foot wounds, in the absence of arterial disease, tend to heal well with proper wound care—particularly with the use of negative pressure wound therapy (NPWT). In the case presented, the patient was advised above-knee amputation before approaching the vascular surgery team. With appropriate wound care, his limb was successfully salvaged.

India being the diabetes capital of the world, such scenarios are increasingly common. As vascular surgeons, we believe that amputation should not be offered for non-vascular diabetic wound infections—especially when managed by diabetologists, general surgeons, or other specialists—unless the patient is in septic shock. In most cases, meticulous wound care is sufficient for healing.

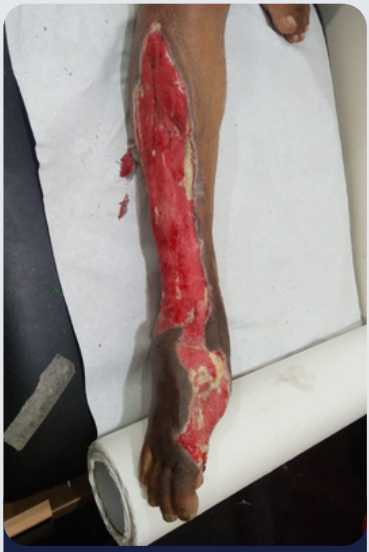


Fig 10. After 4th VAC PRE-Skin Grafting

**Advantages of Veraflo over Conventional VAC:**

The instillation of a topical solution followed by its removal through alternating negative pressure cycles represents a significant advancement in the NPWT concept. Negative Pressure Wound Therapy with Instillation and Dwell Time (NPWTi-d) involves the periodic delivery of a wound solution, which is allowed to dwell in the wound for a set time, facilitating thorough wound cleansing and preparation of the wound bed. This helps promote healing, particularly in complex wounds.

A newer NPWTi-d dressing with through-holes is also available. This design helps broaden the application of this therapy in wounds containing devitalized tissue or in patients who are not suitable candidates for immediate surgical debridement.


Fig 11. The Device

# Role of Biodegradable Temporising Matrix in Vascular Wounds

**Dr. R. Sekhar**
Vascular & Endovascular Surgeon
MUMBAI

**TOOL TALK**

**PolyNovo BTM (Biodegradable Temporising Matrix)** is a wound care and reconstructive product used frequently by vascular surgeons in the treatment of complex wounds, such as:

- Chronic wounds (e.g. diabetic ulcers , venous ulcers, pressure ulcers)
- Ischemic ulcers and wounds after revascularization

It is also popular amongst other speciality surgical branches for:

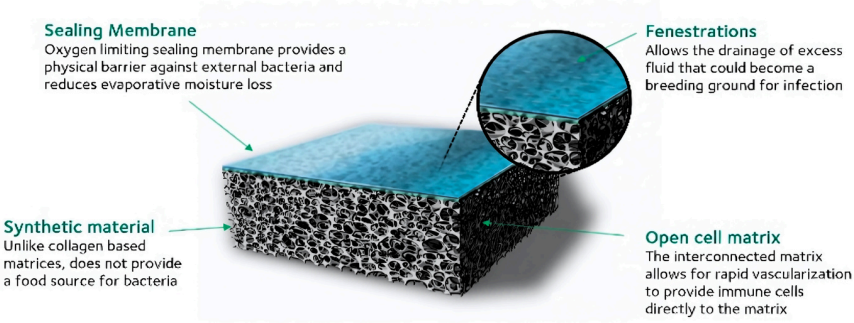
- Burns (especially full-thickness burns)
- Trauma injuries
- Surgical wounds (e.g., after tumour removal)

**BTM is made from NovoSorb®**, a biodegradable synthetic polymer developed by PolyNovo, an Australian biomedical company.

- It's fully synthetic, which means it doesn't use animal or human tissue (unlike some other wound matrices).
- It's biodegradable, so it gradually breaks down in the body as healing occurs.

BTM acts as a scaffold that temporarily replaces the skin's structure and allows the body to regenerate tissue.

1. Application: The surgeon places the BTM over the wound.
2. Integration: Blood vessels and tissue grow into the matrix.
3. Delamination: After the wound is integrated (typically after a few weeks), the outer sealing membrane is removed.
4. Skin grafting: A thin skin graft can then be applied over the healed tissue bed.




**Points to remember about BTM.**

1.BTM is a completely synthetic material which is robust in presence of infection as there is no biological component in it. BTM will often be retained while infection is treated.

2. Can be applied over the bone and tendons for neo-dermis regeneration

3. Designed to minimise contracture over functional important areas and improve cosmesis (Uniformity of texture)

4.More economical as compared with biological dermal matrix.

Fig 1. Diabetic Foot Wound PRE-ANGIOPLASTY

Fig 2. Diabetic Foot Wound POST-ANGIOPLASTY

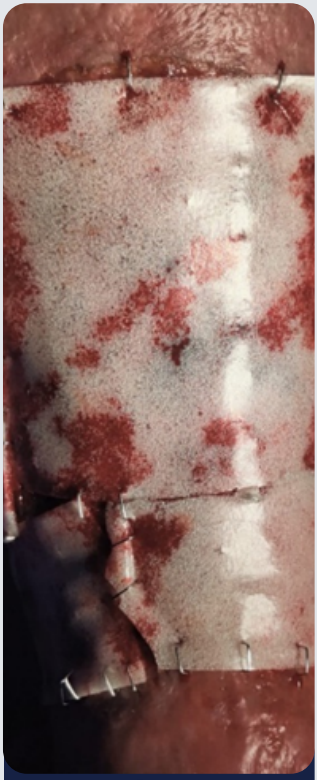


Fig 5.
BTM Application

Fig 3. Debridement + BTM + NPWT

Fig 4. Debridement + BTM + NPWT

Fig 6. Definitive Skin Cover

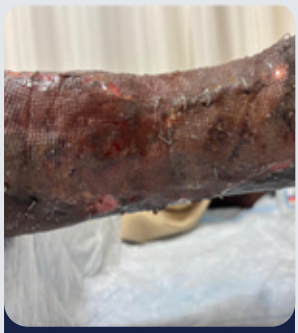


Fig 7. Fully Healed Wound

# What a Venous Ulcer Wants ...

**Dr. Pankaj Patel**
Vascular & Endovascular Surgeon
MUMBAI


**TOOL TALK**

The commonest ulcers in the leg are due to Venous diseases comprising 70% of all causes. Diagnosis of these ulcers, are based on venous duplex scan and MR/CT Venography. Management of these ulcers have always been a challenge. The ulcer is the effect and venous abnormality the cause. A lot of energy is spent on treating the effect and very little effort to find the cause. Generally small ulcers are due to primary varicose veins. The bigger, recurrent and more resistant to heal ulcers are due to deep venous problems or Secondary varicose veins. The commonest cause being post thrombotic legs due to previous Deep vein thrombosis.

**Pathophysiology of venous ulcer**

Increased cap/venous pressure leading to Leakage of fibrinogen into interstitial spaces and formation of pericapillary fibrin layer (White cell trapping theory) Anoxia, Malnutrition and accelerated by trauma causes ulcer

**Treatment**

Following extensive randomised controlled studies, involving different dressing types used in conjunction with the four layer bandage, it was found that dressings had little influence on ulcer healing. Therefore, a simple low adherent dressing such as Bactigras, Paraffin Gauze is used

Four layer compression bandaging

The four layer bandage system is developed to apply 40 mm Hg pressure at the ankle graduated to 17 mm Hg at the knee using bandages of differing properties.

Graduated compression increase blood velocity in the deep veins, reduce oedema and therefore reduce pressure differential between the capillaries and the tissues.

# What a Venous Ulcer Wants ...

It also reduces distention of the superficial veins and reverse venous hypertension. This is the basis for Improving healing rate of chronic venous ulcers.

**Layer I: Soft cotton roll**
**Layer II: Cotton crepe Bandage**
**Layer III: Elasticated crepe bandage**
**Layer IV: Coban**

**Contraindications**

Peripheral vascular disease.
Oedema secondary to heart failure.
Thin calves and narrow ankles

Once healing is achieved it is mandatory to treat the cause and correct the venous abnormality. With advent of endovenous ablation therapy the treatment can be done as daycare. In majority of cases the cause is simple to treat and good long term results can be achieved. The most difficult to treat are post thrombotic legs where the recurrence is high especially when the Ilio-femoral veins are occluded.

These cases can nowadays be treated with endovascular approach with newer venous stents and intravascular ultrasound. All patients need to wear class II compression stockings.

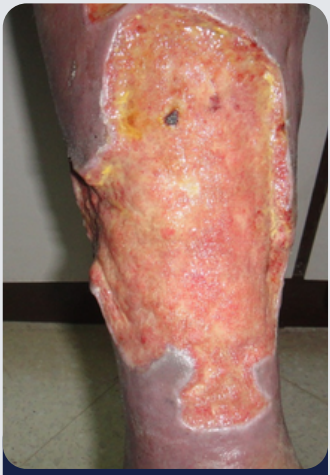


Fig 1. Venous Ulcer

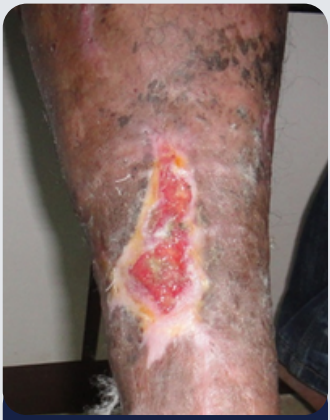


Fig 2. After 10 weeks of 4 layer bandage

# Synergistic Approach in the management of CHRONIC WOUNDS

**Dr. Paresh Pai**
Vascular & Endovascular Surgeon
MUMBAI

## TOOL TALK

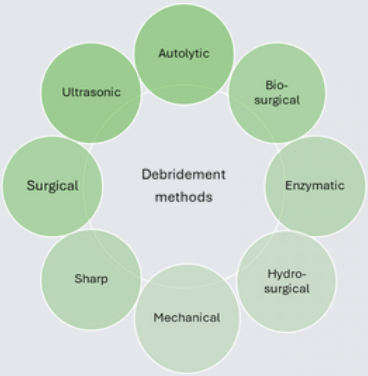
**1.Integral debridement for Wound Bed Preparation (WBP)**

This article focuses on the importance of integrated local management of chronic wounds and the role of newer technology that can play a game changing role to support & augment the efficacy of other standard treatment and correction of local, focal and systemic factors that is irreplaceable.

Local debridement is a crucial part of WBP that is important for wound healing and involves removal of dead non-viable tissue including necrotic material, slough, micro-organisms and biofilm that interferes with wound healing by increasing risk of infection, regeneration of biofilm and impedes growth of healthy granulation tissue and skin cover.

60-100% of chronic wounds have biofilm that regenerates within 24 hours of removal. Frequent periodic debridement of wounds promotes wound healing by 4-fold. Presence or increase in pain besides presence of biofilm, inflammation, stagnant or deterioration of wounds helps indicate the need for repeated debridement or escalation of treatment.

Selective sharp debridement performed at the bed side and Surgical Debridement performed in the OT under anaesthesia form the gold standard of debridement, along with other methods such as Hydro-surgical, Ultrasonic, Mechanical, Bio-surgical, Enzymatic and Autolytic done selectively in isolation or with various permutations and combinations to provide the best & most effective therapy.

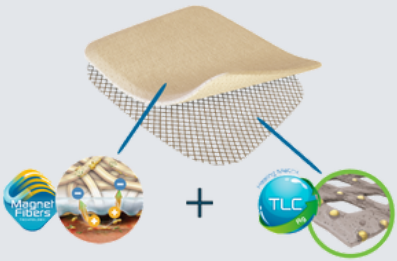


# Synergistic Approach in the management of CHRONIC WOUNDS

**INTEGRAL DEBRIDEMENT** is the combined use of different but complementary methods of debridement on the same wound, as required, to achieve an optimal outcome. It also allows for a tailored and patient-centered approach in WBP and over all wound care that highlights the need to consider patient's comfort and preferences when selecting a debridement method.

**Continuous Autolytic Debridement (CAD)** is a very effective, safe and patient friendly option that adheres to the principle of Integral Debridement by using a dressing material that is suitable to use in the post-debridement period when you aim for maintenance debridement for the better outcome of healing process. Integral Debridement can be used along with sharp debridement, when sharp debridement is contraindicated or risky, forms part of the anti-biofilm protocol, and is suitable for use in most clinical settings bridging limitations of different levels of expertise of the healthcare professionals by providing step up or step-down options.

**URGOCLEAN AG** is one such Anti-biofilm silver dressing material that incorporates two technologies to provide CAD. There are negatively charged Polyabsorbent fibres (Magnet fiber technology) that attract & bind to slough, necrotic wound residue, micro-organisms & fibrin that forms a gel to promote moist wound healing & support CAD maintaining a clean wound bed for optimizing silver efficacy. It also has Anti-microbial TLC-Ag healing matrix that provides fast anti-microbial activity against resistant strains like MRSA, VRE, destroys biofilm & prevents regeneration and provides pain free atraumatic removal by maintaining a moist environment.



**Case Capsule :**
83 year lady
Obese
HT + IHD
MVA + R) femur fracture – ORIF, Hypothyroid,
OA knees,
L) leg DVT + PE with temp IVC filter
Ulcer – posted for SSG
Had swelling in post-thrombotic leg

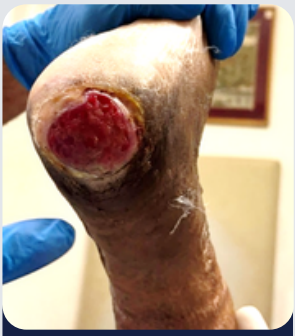


Fig 1. Urgoclean Ag Initiated

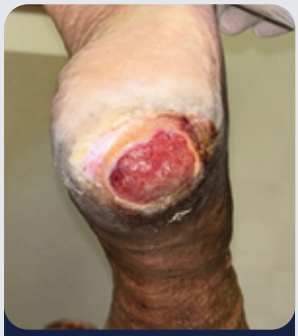


Fig 2. Urgoclean Ag Followup Dressings

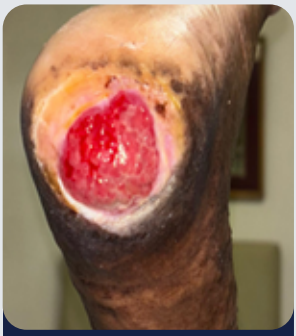


Fig 3. Urgoclean Ag Followup Dressings

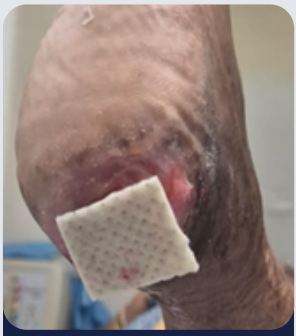


Fig 4. Application of Urgoclean Ag

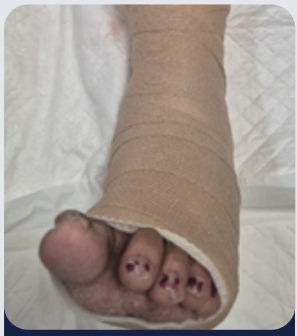


Fig 5. Urgoclean Ag Followup Dressings

**2. Sucrose Octasulfate dressing for closing the wounds sooner**

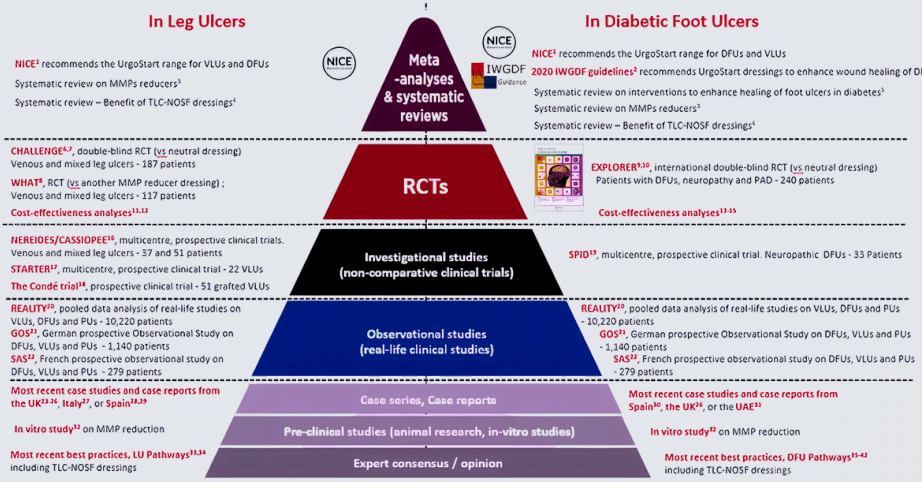
Matrix Metalloproteinases (MMPs) are enzymes produced during the inflammatory phase by activated macrophages. They help clean wounds by breaking down damaged proteins and extracellular matrix (ECM), promoting granulation. In normal healing, MMP levels decline, but in chronic wounds, reduced Tissue Inhibitors of Metalloproteinases (TIMPs) lead to excess MMPs.

# Synergistic Approach in the management of CHRONIC WOUNDS

High MMPs destroy ECM, damage growth factors, and impair repair, with MMP-9 most abundant, followed by MMP-8, MMP-2, and MMP-1. Elevated MMPs lower VEGF, further delaying healing. MMP-9 levels correlate with ulcer severity and poor healing in diabetic foot ulcers (DFUs), venous leg ulcers (VLUs), and pressure injuries (PUs).

While treating the underlying cause is vital, local therapy is essential. Sucrose Octasulfate dressings reduce MMPs, enhance growth factor activity, and promote healing. After infection, biofilm, and slough are reduced with UrgoClean Ag, UrgoStart should be initiated for chronic wounds to shorten healing time, improve quality of life, avoid complications, and reduce costs.

UrgoStart's jellified hydrocolloid (CMC) and lipophilic matrix with Sucrose Octasulfate lowers MMPs, supports fibroblast proliferation, and allows painless dressing changes. It restores balance and accelerates closure; earlier use yields better outcomes.




IWGDF 2023 guidelines recommend UrgoStart for non-infected neuro-ischaemic DFUs alongside standard care, and NICE 2023 supports its use in DFUs and VLUs for faster healing and cost-effectiveness.

**Case Capsule :**

A 63-year-old man
Type 2 diabetes x 6 years
Peripheral vascular occlusive disease status post (S/P) Left anterior tibial artery (ATA) and peroneal artery (PTA) angioplasty, critical limb ischaemia Rutherford VI

The comprehensive approach of Integral debridement and Closing wounds sooner has been shown here (Wounds international 2024):

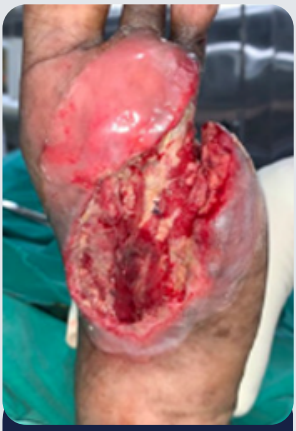


Fig 6. Urgoclean Ag Initiated

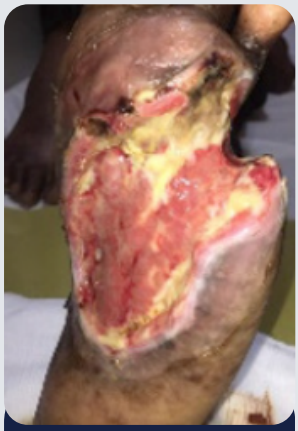


Fig 7. WEEK 3

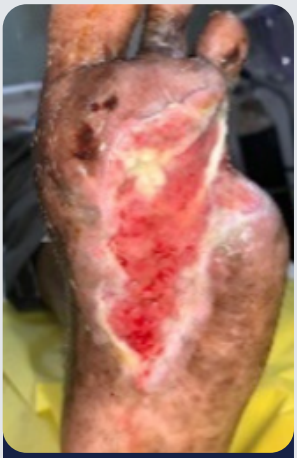


Fig 8. WEEK 7 Shifted to URGOStart

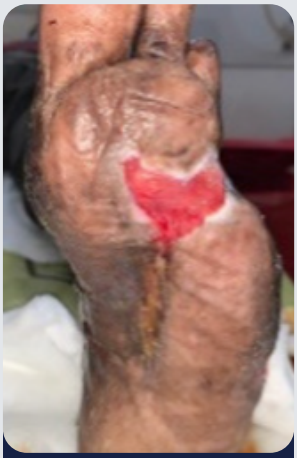


Fig 9. WEEK 9

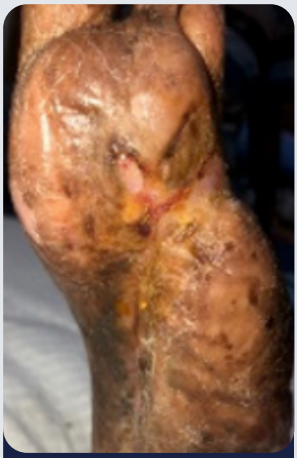


Fig 10. WEEK 10 : Healed

**Case Capsule :**

78-year-old woman
Type 2 diabetes x 18 years, with systemic hypertension, Alzheimer's disease, hypothyroidism and dyslipidaemia.
Right calcaneal pressure injury x 5 months, which was not healing and increasing in size.

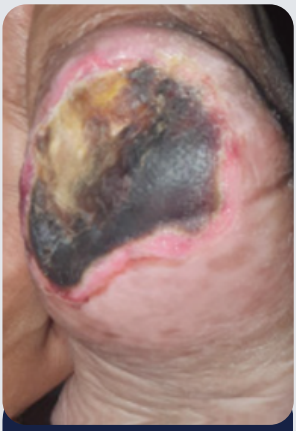


Fig 11. On Presentation

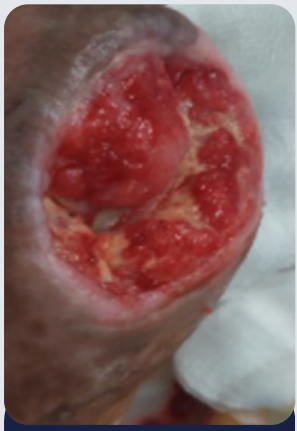


Fig 12. Week 3 - Initiated on Urgoclean Ag Post Debridement

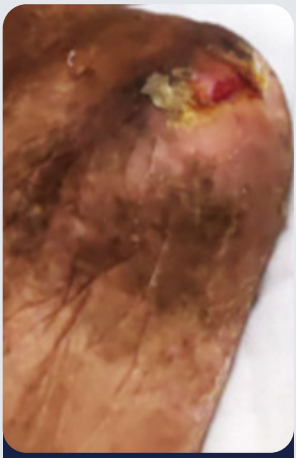


Fig 13. WEEK 6
Shifted to URGOStart

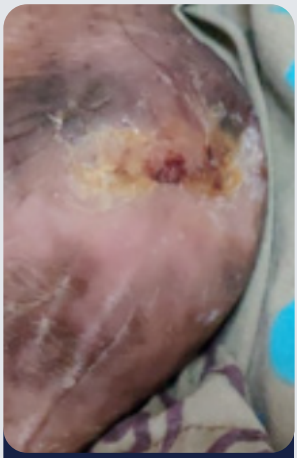
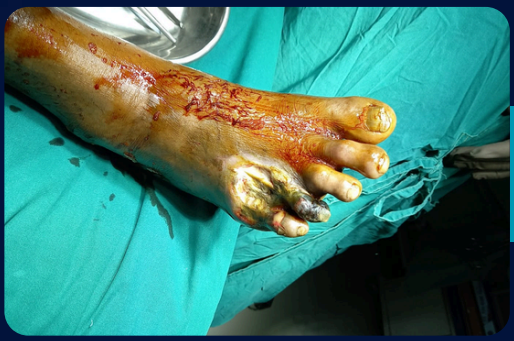


Fig 14.
WEEK 7 : Healed

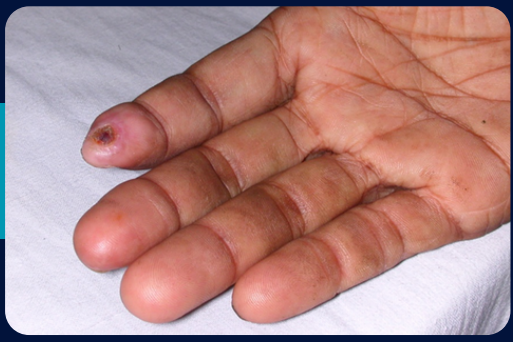
# Vascular SPOTTERS PEARLS



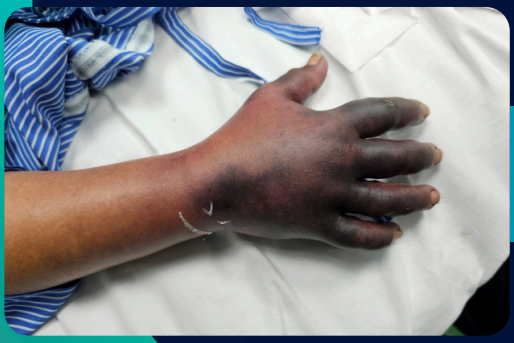
Courtesy : Dr. Vinit Paliwal

Arterial Ulcer with Toe Gangrene
Treatment : Revascularisation of the Right lower limb with amputation of 4th & 5th toe

Courtesy : Dr. R Sekhar



Right Index Finger tip Ischaemia
Treatment : Right Cervical Rib showering distal embolus : Cervical Rib resection

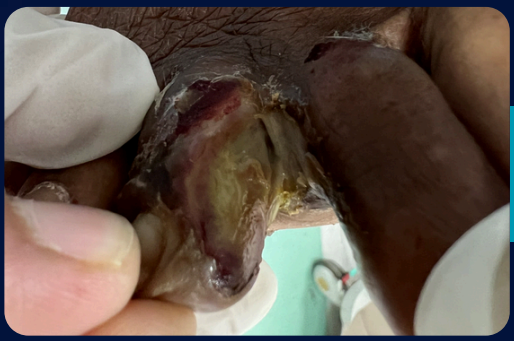


Courtesy : Dr. Aniruddha Bhuiyan

Right Hand Ischaemia with Compartment Syndrome
Treatment : Right upper limb thrombectomy with Fasciotomy and Debridement

# Vascular SPOTTERS

PEARLS

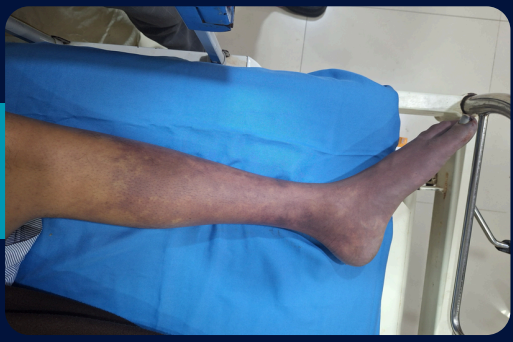


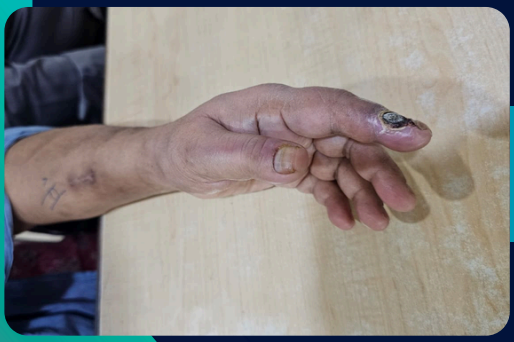
Courtesy : Dr. Ritesh Gaikwad

Interdigital Ulcer with PVD
Treatment : Peripheral Angioplasty with Wound Debridement

Courtesy : Dr. Simit Vora

DEAD LIMB
Treatment : Delayed Presentation with SEPSIS ; ABOVE KNEE AMPUTATION

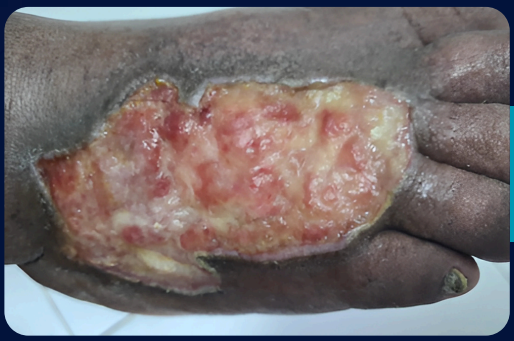




Courtesy : Dr. Piyush Jain

Left Upper Limb AVF Steal Syndrome
Treatment : AVF Closure on Left upper limb

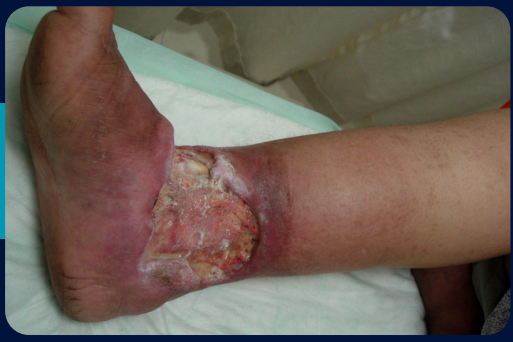
# Vascular SPOTTERS PEARLS



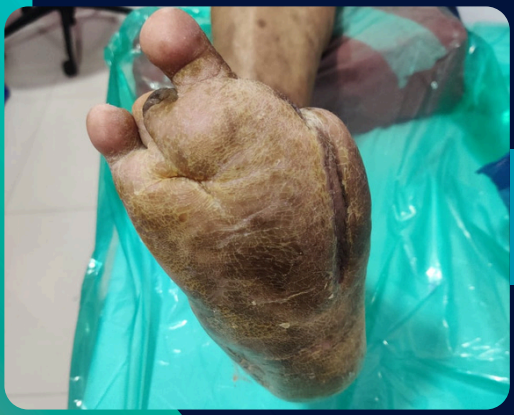
Courtesy : Dr. Shahzad Bulsara

Mixed Ulcer (Art + Venous)
Treatment : Peripheral Angioplasty with Treatment of Venous Reflux

Courtesy : Dr. Paresh Pai



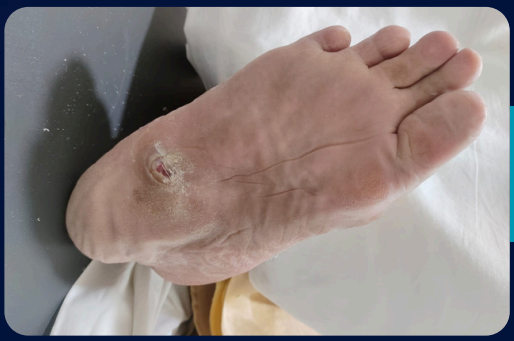
Lymphatic Ulcer
Treatment : Compression Therapy with Local Debridement and Wound Care



Courtesy : Dr. Amish Mhatre

Charcots Foot
Treatment : Right lower limb angioplasty with debridement & Total Contact Cast

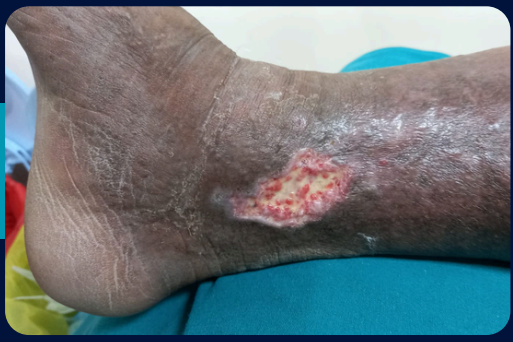
# Vascular SPOTTERS PEARLS



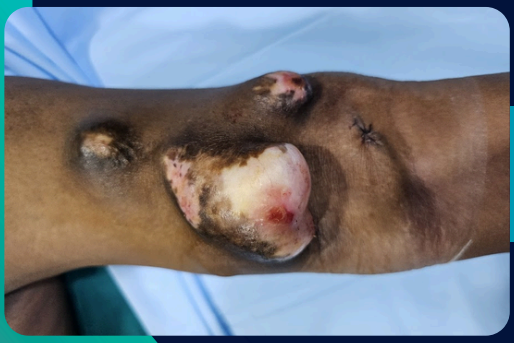
Courtesy : Dr. Shrikant Ghanwat

Diabetic Trophic Ulcer
Treatment : Local Wound Care with Offloading Footwear

Courtesy : Dr. Vinit Paliwal



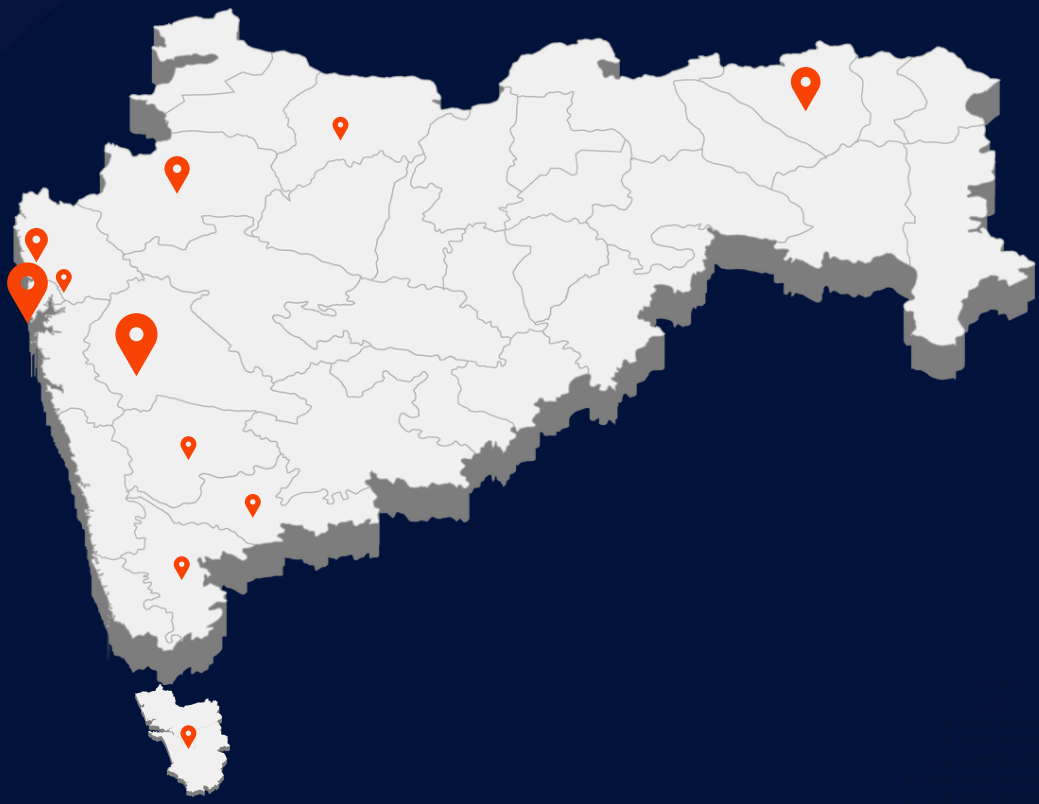
Venous Ulcer
Treatment : Endovenous Laser Ablation with 4 layer Compression Bandaging



Courtesy : Dr. Aniruddha Bhuiyan

AVF Aneurysms
Treatment : AVF Closure with excision of aneurysmal sacs

# Availability of Vascular & Endovascular Surgery Services



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**Dr. Alexis Carrel**

**"The cell is immortal. It is merely the fluid in which it floats that degenerates. Renew this fluid at regular intervals, give the cells what they require for nutrition, and as far as we know, the pulsation of life can go on forever".**