

# VESSOMAG's Inside the VESSEL ...



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

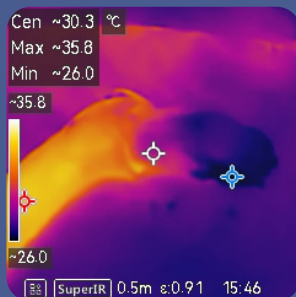


## THEME DIABETIC FOOT



### Must Reads ....

- Is This Foot Salvageable? – The First 5-Minute Clinical Assessment
- Why Every Diabetic Foot Needs a Vascular Assessment Early
- Infection vs Ischemia – What Should You Treat First?
- Why Wounds Don't Heal Despite Good Sugar Control
- Heel Ulcers vs Toe Ulcers – The Art of Offloading
- Needle to a Cut – What Determines the Choice of Management



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# TABLE OF CONTENTS

EDITOR : DR. ANIRUDDHA BHUIYAN



| No. | Section            | Topic                                                                                 | Author                                     | Pg. No |
|-----|--------------------|---------------------------------------------------------------------------------------|--------------------------------------------|--------|
| 1.  |                    | Secretary Speaks...                                                                   | Dr. Amish Mhatre                           | 5      |
| 2.  | EXAMINATION        | Is This Foot Salvageable?<br>– The First 5-Minute<br>Clinical Assessment              | Dr. Aniruddha Bhuiyan                      | 7      |
| 3.  |                    | Why Every Diabetic Foot<br>Needs a Vascular<br>Assessment Early                       | Dr. Simit Vora                             | 10     |
| 4.  | DECISION<br>MAKING | Infection vs Ischemia –<br>What Should You Treat<br>First?                            | Dr. Rohit Gupta /<br>Dr. Harshawardhan Oak | 13     |
| 5.  |                    | Why Wounds Don't Heal<br>Despite Good Sugar<br>Control                                | Dr. Archana Juneja /<br>Dr. Aneesha Panda  | 17     |
| 6.  |                    | Heel Ulcers vs Toe Ulcers<br>– The Art of Offloading                                  | Dr. Pravin Narkhede                        | 23     |
| 7.  | MANAGEMENT         | Needle to a Cut – What<br>Determines the Choice of<br>Management                      | Dr. Pranjal Melge Sawant                   | 28     |
| 8.  |                    | Editor's Closing Note :<br>Chitosan - Based Dressings :<br>Old Molecule, New Promise? |                                            | 35     |

# TABLE OF CONTENTS

EDITOR : DR. ANIRUDDHA BHUIYAN



| No. | Section | Topic              | Author | Pg. No |
|-----|---------|--------------------|--------|--------|
| 12. |         | COMICS             |        | 37     |
| 13. |         | VESSOMAG DIRECTORY |        | 41     |
| 14. |         | SPONSOR            |        | 43     |



# Secretary Speaks ...

**Dr. Amish Mhatre**  
Vascular & Endovascular Surgeon  
MUMBAI



## **Q: Why Should Diabetic Foot Disease Be Treated by a Vascular & Endovascular Surgeon?**

Ask a patient with a diabetic foot ulcer who should be treating them, and the answer often depends on which specialist they happened to see first.

A Vascular & Endovascular Surgeon is trained in the medical, endovascular and open surgical management of peripheral artery disease (PAD).

Diabetic foot disease refers to any condition arising from PAD and/or peripheral sensory neuropathy. Known as diabetic foot syndrome, it presents as infection, diabetic foot ulcer and neuropathic osteoarthropathy — which, in its advanced form, produces the characteristic bone deformity known as Charcot foot. Traditionally, diabetic foot disease was managed almost exclusively by General Surgeons, with treatment limited to debridement and when the disease progressed - AMPUTATION....

A better understanding of its pathophysiology has since widened this circle of care to include General Surgeons, Diabetic Foot Specialists, Plastic Surgeons, Interventional Radiologists and *Vascular & Endovascular Surgeons*.

### **Why a Vascular & Endovascular Surgeon?**

General Surgeons, Diabetic Foot Specialists and Plastic Surgeons do not perform procedures to restore blood supply. Interventional Radiologists perform peripheral endovascular procedures but do not perform open surgery. The Vascular & Endovascular Surgeon is the only specialist trained across the entire vascular system — arteries, veins, and lymphatics — offering the full spectrum of care, from lifestyle modification and medical management to minimally invasive intervention and complex open surgery.

## Secretary Speaks ...



**Glimpses of VESSOMAG's 'Amputation Free India' Walkathon, conducted in Maharashtra, November 2025**

Assessing perfusion objectively — through Ankle-Brachial Index (ABI), Toe-Brachial Index, or transcutaneous oxygen pressure (TcPO<sub>2</sub>) — and staging disease severity using systems such as Wifi (Wound, Ischemia, foot Infection), is core vascular surgical practice. It determines whether a wound will heal on the blood supply it already has, or whether revascularisation must come first.

Unlike specialists confined to a single organ system, a Vascular & Endovascular Surgeon evaluates how a blockage in the leg relates to circulation elsewhere in the body — a systemic view essential to managing atherosclerosis and diabetes, neither of which respects anatomical boundaries.

*Many vascular conditions are managed with medication and exercise alone; surgery is reserved for limb- or life-threatening complications.*

The Vascular & Endovascular Surgeon is equally skilled in endovascular technique — using balloons and stents to open blocked vessels without large incisions — and in open surgery, performing bypasses or aneurysm repairs where endovascular treatment falls short.

Not every blockage needs a stent or a bypass. Knowing which patient needs which treatment — and which needs neither — has always been the hallmark of the Vascular & Endovascular Surgeon.

**Regards**

**Secretary, VESSOMAG**



# Is this FOOT SALVAGABLE ?

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



## EXAMINATION



It's 9 a.m. The emergency physician calls: "Diabetic foot, looks bad, can you come take a look?" You already know what you're walking into before you've parked the car — a shuffling gait, a slipper stained with something best left unidentified, and a family in the corridor already bracing for the word "amputation."

Here's the good news: you don't need an hour, an angiogram or a crystal ball to know whether that foot has a fighting chance. You need five minutes and a system. Below is the one worth pinning on the department wall.

### **Minute 1: Look Before You Touch**

Strip the dressing, resist the urge to poke immediately, and just look. Note the colour — pink, dusky, mottled, black — the depth of the wound and whether it's weeping, dry or producing something that smells like a chemistry experiment gone wrong. Dry gangrene is a slow-burn problem. Wet gangrene is a today problem.

Check the wound's borders. A punched-out ulcer sitting neatly over a pressure point screams neuropathy. A necrotic, well-demarcated edge on an otherwise cold foot screams ischemia. Diabetic feet often insist on doing both at once — the classic neuroischemic wound nobody asked for.

### **Minute 2: Temperature Is a Vital Sign Too**

Run the back of your hand from mid-shin down to the toes. A sudden drop in warmth is the foot telling you, quite precisely, where its blood supply gave up. This costs nothing, needs no equipment and still gets skipped more often than it deserves to be.

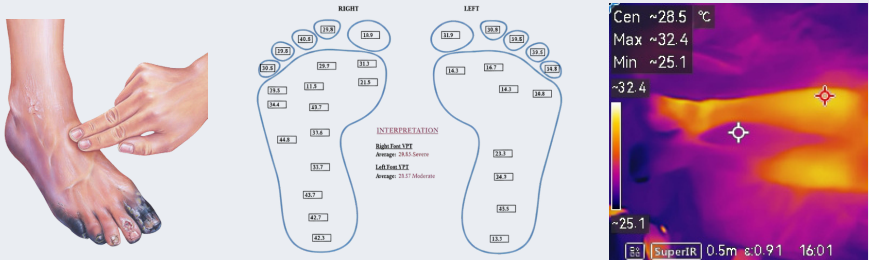
## Is this FOOT SALVAGABLE ?

If the foot is colder than the exam room, colder than your coffee and colder than the patient's opinion of the hospital food, that's three strikes — not a coincidence.

### Minute 3: Feel for Pulses, Then Confirm You Aren't Feeling Your OWN

Palpate the dorsalis pedis and posterior tibial pulses. If you find them, document it and move on with your life. If you don't, do not spend the next four minutes hunting for a pulse that simply isn't there out of sheer optimism — reach for the handheld Doppler instead.

Listen to the waveform, not just its presence. Triphasic is reassuring. Biphasic, make a note. Monophasic or silence and this foot's perfusion problem has just become the main character of your assessment.



### Minute 4: Sensation and Infection, Back to Back

Ten-gram monofilament, four sites, quick and standardised. If the patient can't feel it, they have likely been walking on damage for months without knowing – which, incidentally is exactly how diabetic feet end up in your clinic later rather than earlier.

While you're down there, screen for infection: warmth, spreading erythema, induration, purulence and the single most underrated test in all of surgery — probe-to-bone. If the probe hits bone, assume osteomyelitis until proven otherwise. It is right about as often as it needs to be, which is often enough to matter.

# Is this FOOT SALVAGABLE ?

## Minute 5: Put the Pieces Together

This is where the WIfI framework — Wound, Ischemia, Foot Infection — earns its keep. Grade each quickly in your head as you walk away from the bed. A deep wound with poor perfusion and active infection is not a “wait and watch” foot. It is a “get an imaging now” and salvage the cold foot.

A shallow wound with palpable pulses and clean margins, on the other hand, may need nothing more dramatic than offloading, glycaemic control and a firm conversation about footwear.

## The Five-Minute Cheat Sheet

For the resident who wants it on an index card:

- Look:** colour, depth, gangrene type, wound border
- Feel:** temperature gradient, mid-shin to toes
- Pulses:** palpate first, Doppler waveform if absent
- Sense and Infect:** monofilament, probe-to-bone, spreading erythema
- Stage:** WIfI in your head — wound, ischemia, infection — before you leave the bedside

If two or more of these are red flags, that foot does not wait for medical management

## The Real Skill Isn't Speed — It's Not Skipping Steps

Five minutes only works if you resist shortcutting it because the wound “looks fine” or the patient “seems okay.” **Diabetic feet are remarkably talented at hiding catastrophe behind a calm exterior.**

Salvage decisions made in these first five minutes set the tone for everything downstream — urgent revascularisation, same-day debridement or simply better shoes and a stern lecture. Get the five minutes right, and the rest of the pathway tends to write itself.





# Why Every Diabetic Foot Needs a Vascular Assessment Early

**Dr. Simit Vora**  
Vascular & Endovascular Surgeon  
MUMBAI, INDIA



## EXAMINATION

Early vascular assessment is important in every person with a diabetic foot problem because diabetes can silently reduce blood flow to the feet, making even small wounds difficult to heal. Peripheral arterial disease (PAD) is common in diabetes. Narrowed or blocked leg arteries often cause no typical symptoms, since diabetic neuropathy can mask pain.

Ulcers require adequate oxygen and nutrients to heal. Without sufficient circulation, wounds may persist despite good wound care and antibiotics.



Poor blood flow therefore delays wound healing. Early identification of poor circulation allows timely angioplasty or bypass, reducing the risk of amputation and improving limb salvage.

A diabetic foot ulcer may be:

- 1. Mainly neuropathic**
- 2. Mainly ischemic (poor blood supply)**
- 3. Neuro-ischemic (both)**



# Why Every Diabetic Foot Needs a Vascular Assessment Early

An early vascular assessment helps distinguish the cause of the foot problem, since treatment differs depending on which type is present. Infection is more dangerous when circulation is poor. Antibiotics do not reach infected tissue adequately, and infection can progress rapidly in an ischemic foot.

Vascular assessment helps in guiding and determining:

- 1.Healing potential**
- 2.Need for revascularization**
- 3.Whether compression therapy is safe (if edema is present)**
- 4.Appropriate level of debridement**

How to perform an early vascular assessment in the diabetic foot

- Clinical examination:
  1. Palpation of pedal pulses
  2. Capillary refill
  3. Skin temperature and color.
- Hand-held Doppler examination
- Ankle-Brachial Index (ABI), interpreted cautiously due to arterial calcification
- Toe-Brachial Index (TBI)
- Transcutaneous oxygen pressure (TcPO<sub>2</sub>) or skin perfusion pressure
- Duplex ultrasound if arterial disease is suspected.
- Further imaging (CT angiography, MR angiography)
- DSA or catheter angiography when planning revascularization

## **Guideline recommendations:**

Major organizations, including the International Working Group on the Diabetic Foot and the American Diabetes Association, recommend prompt vascular assessment for every diabetic foot ulcer, even when pedal pulses feel normal.

# Why Every Diabetic Foot Needs a Vascular Assessment Early

**"Time is tissue"** is a widely respected medical dictum. Early vascular assessment identifies impaired blood flow before irreversible tissue loss occurs, improving healing and reducing the risk of amputation.



**Fig 1. Timely intervention is needed to prevent such mishaps**



# Infection vs Ischemia – What Should You Treat First?

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



**Dr. Harshawardhan Oak**  
Vascular & Endovascular Surgeon  
PUNE



## /// DECISION MAKING

Every vascular surgeon has stood at the bedside of a diabetic patient with a foul-smelling, poorly perfused ulcer and silently asked the same question: antibiotics first, or the angiogram? It's a debate that resists a single protocol, because the honest answer is “it depends” — and it depends on things you can really only judge standing at the bedside.

For this issue, we put the question directly to two of our regular contributors, Dr. Rohit Gupta & Dr. Harshawardhan Oak. Their answers agree more often than the headline suggests — and disagree in exactly the places that matter.

**Q: When you walk into a room and see an infected, poorly perfused diabetic foot, what's actually running through your head first?**

**Dr. Oak:** “Honestly, the first thing I'm working out isn't infection versus ischemia — it's whether this patient is septic. That's the real fork in the road. Severe infection can tip into a systemic inflammatory response and multiorgan dysfunction faster than people appreciate, and if there's an abscess, necrotizing infection, or wet gangrene in front of me, that gets drained today, vascular status or not. But if the patient is stable and what I'm looking at is a chronic ulcer with poor perfusion, the whole calculus changes.”

# Why Wounds Don't Heal Despite Good Sugar Control

**Dr. Gupta:** “I'd frame it slightly differently. Before I even think about antibiotics, I want to know why this ulcer exists in the first place. In most arterial ulcers I see, infection isn't the disease — it's a complication of a disease that's already been running quietly for months, which is inadequate blood flow. So my first instinct is pulses, then Doppler. Not because infection doesn't matter, but because treating it without understanding perfusion is treating the symptom and missing the disease.”

**Q:** **Dr. Gupta, that's a fairly strong claim — infection as a downstream consequence rather than the primary problem. Can you unpack that?**

**Dr. Gupta:** “It's not really a bold claim, just an under-appreciated one. Peripheral arterial disease starves tissue of oxygen and nutrients. Minor trauma that would heal without incident in a well-perfused foot instead breaks down and ulcerates. That same ischemic environment weakens local immunity and blocks antibiotic delivery to the tissue, so bacteria happily colonize what's essentially dying tissue with no resistance left. That's why so many of these ulcers fail to heal despite round after round of antibiotics and debridement — you're fighting the infection while ignoring the reason it keeps coming back.”

**Dr. Oak:** “I won't argue with the mechanism — it's real, and it's exactly why ischemic tissue has such poor antibiotic penetration and blunted leukocyte function. Where I'd add a little nuance is that diabetics also carry neuropathy and impaired immunity running independently of the vascular disease. So yes, ischemia often sets the stage, but the two processes end up feeding each other — infection raises tissue metabolic demand at exactly the moment ischemia is restricting oxygen delivery. Calling it a one-way street undersells how vicious that loop actually gets.”

**Q:** **Is there a scenario where the two of you agree, without hesitation, that infection has to be dealt with first?**

# Why Wounds Don't Heal Despite Good Sugar Control

**Dr. Oak:** “Absolutely, and this is where the debate gets oversimplified in most discussions. Sepsis, necrotizing soft tissue infection, gas gangrene, wet gangrene — these are lifesaving surgical emergencies. Source control and broad-spectrum antibiotics take priority over everything else, including vascular imaging. Stabilise the patient first, full stop.”

**Dr. Gupta:** “Completely agree, no daylight between us here. If someone is septic or has wet gangrene tracking up the leg, this stops being a nuanced conversation. Drain it, debride what needs debriding — guillotine amputation if that's what source control demands — resuscitate, broad-spectrum antibiotics, and the angiogram waits until the patient is stable enough to actually benefit from one.”

**Q: So where does the real disagreement live — is it about debridement?**

**Dr. Gupta:** “Largely, yes. My biggest concern day to day isn't the septic patient — everyone manages that correctly. It's the stable patient with a chronic ulcer who gets aggressively debrided before anyone has checked the blood supply. You end up converting a manageable wound into a bigger one that needs even more perfusion to heal, in a limb that couldn't supply the original wound to begin with. I tell trainees to ask one question before they pick up the scalpel: does this limb have enough blood flow to heal the wound I'm about to create?”

**Dr. Oak:** “That's a fair challenge, and the evidence genuinely backs it — delayed revascularisation is consistently linked to higher rates of major amputation. My only caveat is that “let's wait for the angiogram” can't become an excuse to leave necrotic tissue or a walled-off collection untouched. The practical compromise most of us have settled on is minimal essential debridement for source control, followed by prompt revascularisation, and save the definitive wound reconstruction for after perfusion is restored.”

**Q: Once the patient is stable, how fast is “fast” for revascularisation?**

**Dr. Oak:** “Days, not weeks, wherever it's feasible. Endovascular options — angioplasty, stenting — have made this far more achievable even in frail patients who wouldn't tolerate an open bypass.”

# Why Wounds Don't Heal Despite Good Sugar Control

The data is fairly consistent: the longer perfusion is delayed, the higher the amputation rate.”

**Dr. Gupta:** “Early is the operative word, and I'd put it on equal footing with the antibiotics, not after them. Restoring inflow doesn't just help the wound heal directly — it improves antibiotic delivery and immune function in the tissue, which means the infection you're already treating responds better too. It isn't sequential. It's parallel.”

**Q: Last question — if a medical professional reads only one line from your article, what should it be?**

**Dr. Oak:** “It's not ischemia versus infection, it's ischemia and infection. Whichever one is more immediately life- or limb-threatening gets addressed first, but the other one is still on the clock — and pretending otherwise is what costs limbs.”

**Dr. Gupta:** “Check the blood supply before you pick up the blade. Treat the infection, yes — but if the limb is going to heal, you have to restore perfusion. And if the infection is the thing about to kill the patient, control that first, then go chasing the vessels.”

## Editor's Note :

Both Vascular Surgeons reach for source control the moment sepsis enters the picture, and both land on early revascularisation once the patient is stable. Where they genuinely differ is the default assumption walking into the room — Dr. Oak treats infection and ischemia as parallel threats racing each other; Dr. Gupta treats ischemia as the root cause hiding behind the infection. Worth deciding, before your next diabetic foot consult, which lens you reach for first.





# Why Wounds Don't Heal Despite Good Sugar Control

**Dr. Archana Juneja**

Dept. of Endocrinology  
KDAH, MUMBAI



**Dr. Aneesha Panda**

Dept. of Endocrinology  
KDAH, MUMBAI



**DECISION MAKING**

**Diabetic foot ulcers (DFUs)** represent one of the most devastating complications of diabetes mellitus. Approximately 15% of all people with diabetes will develop a DFU during their lifetime, and these ulcers precede 84% of diabetes-related lower-leg amputations. The mortality burden is staggering: the five-year survival rate for patients with diabetic wounds is only 50%—a prognosis comparable to many advanced cancers.

Despite optimal glycemic control, aggressive debridement, and infection management, up to 40% of wounds recur within one year and over 60% within five years due to persistent underlying pathophysiology. Impaired or delayed wound healing affects approximately 25% of diabetic people, with 1 in 3 to 1 in 5 diabetic individuals at risk of chronic non-healing wounds in their lifetime.

Understanding the mechanisms underlying impaired wound healing is essential for early identification of high-risk patients and timely referral.





# Why Wounds Don't Heal Despite Good Sugar Control

Wound healing is a complex, temporally synchronized process divided into four overlapping phases:

1. **Hemostasis** – clot formation and cessation of bleeding.
2. **Inflammation** – recruitment of inflammatory cells to clear debris and bacteria.
3. **Proliferation** – formation of granulation tissue, angiogenesis, and re-epithelialization.
4. **Remodeling** – maturation and strengthening of the wound.

In diabetes, every phase is disrupted simultaneously, creating a vicious cycle of non-healing.

## Poor Blood Supply: The Central Problem

Adequate tissue perfusion is fundamental to wound healing. Diabetes accelerates atherosclerosis, particularly in the arteries below the knee. Peripheral artery disease (PAD) is therefore significantly more common and often more severe in individuals with diabetes.

### Reduced arterial blood flow leads to:

- *Tissue hypoxia*
- *Reduced nutrient delivery*
- *Impaired leukocyte function*
- *Decreased collagen synthesis*
- *Delayed granulation tissue formation*

Importantly, diabetic PAD often involves distal tibial and pedal vessels, meaning that a foot may appear warm while still having critically impaired perfusion. The absence of classic symptoms such as claudication should not exclude vascular disease.

Any non-healing ulcer should prompt assessment of peripheral circulation through pulse examination and, when indicated, non-invasive vascular testing and referral to a vascular specialist.

# Why Wounds Don't Heal Despite Good Sugar Control

## **Neuropathy: The Silent Culprit**

Diabetic neuropathy contributes to wound formation and impaired healing through several mechanisms.

### **1) Sensory Neuropathy**

Loss of protective sensation prevents patients from recognizing trauma, pressure, burns, or poorly fitting footwear. Repetitive minor injuries go unnoticed and progress into ulcers.

### **2) Motor Neuropathy**

Weakness and muscle imbalance lead to foot deformities such as claw toes and prominent metatarsal heads. These deformities create abnormal pressure points that predispose to skin breakdown.

### **3) Autonomic Neuropathy**

Reduced sweating causes dry, cracked skin, compromising the skin barrier and providing portals of entry for microorganisms.

Thus, even a small injury can evolve into a chronic ulcer because the patient neither perceives the injury nor adequately protects the affected area.

## **Hyperglycemia Induced Molecular Toxicity**

Persistent hyperglycemia directly impairs multiple cellular processes required for tissue repair.

### **High glucose levels:**

- *Reduce fibroblast proliferation*
- *Impair collagen production*
- *Decrease keratinocyte migration*
- *Suppress angiogenesis*
- *Increase oxidative stress*
- *Promote formation of advanced glycation end products (AGEs)*

AGEs alter protein structure and function, stiffen tissues, and impair cellular signaling necessary for wound repair.

While local wound care remains important, optimization of glucose control is a critical component of treatment.

# Why Wounds Don't Heal Despite Good Sugar Control

## **Infection: Cause and Consequence**

Diabetic wounds are particularly susceptible to infection. Several factors contribute:

- **Impaired neutrophil chemotaxis and phagocytosis**
- **Reduced cellular immunity**
- **Tissue hypoxia**
- **Hyperglycemia, which favors bacterial growth**

Once infection develops, it further increases tissue destruction, inflammation, and metabolic stress, creating a vicious cycle.

Clinicians should maintain a high index of suspicion, as infection may present subtly in patients with diabetes. Increasing wound size, malodor, drainage, friable granulation tissue, or unexplained hyperglycemia may be the earliest clues.

## **Chronic Inflammation: A Wound Stuck in Neutral**

Normal healing requires a transition from inflammation to tissue repair. In diabetes, wounds often remain trapped in a prolonged inflammatory phase. Elevated levels of inflammatory cytokines and proteolytic enzymes lead to:

- **Destruction of growth factors**
- **Breakdown of extracellular matrix**
- **Reduced tissue regeneration**

As a result, wounds fail to progress toward closure despite standard wound care measures.

## **Impaired Angiogenesis**

Healing tissues require the development of new blood vessels. Diabetes disrupts angiogenesis through endothelial dysfunction, oxidative stress, and reduced responsiveness to growth factors.

The inability to generate an adequate microvascular network limits oxygen delivery and cellular repair, particularly in areas already affected by peripheral arterial disease.

# Why Wounds Don't Heal Despite Good Sugar Control

## **Red Flags Requiring Urgent Vascular Referral**

A diabetic foot ulcer should not be managed as a routine wound when any of the following features are present:

### **Signs of Limb Ischemia**

- Absent or markedly diminished foot pulses
- Rest pain, especially at night
- Dependent rubor or pallor on elevation
- Cold foot compared with the contralateral side
- Delayed capillary refill (>3 seconds)
- Toe discoloration, gangrene, or tissue necrosis

### **Non-Healing Wound**

- Failure to show signs of healing after 2–4 weeks of appropriate wound care
- Progressive increase in ulcer size or depth
- Recurrent ulceration at the same site

### **Severe or Spreading Infection**

- Cellulitis extending beyond 2 cm from the ulcer margin
- Purulent discharge
- Systemic features such as fever, tachycardia, hypotension or altered sensorium
- Suspected deep abscess or necrotizing soft tissue infection
- Visible bone or suspected osteomyelitis

### **Critical Limb-Threatening Features**

- Gangrene of toes or foot
- Rapidly progressive tissue loss
- Severe ischemic pain
- Combination of ulceration and peripheral

### **High-Risk Patient Factors**

- Previous major or minor amputation / Prior vascular intervention
- End-stage kidney disease or dialysis
- Severe peripheral neuropathy with foot deformity
- Recurrent diabetic foot ulcers

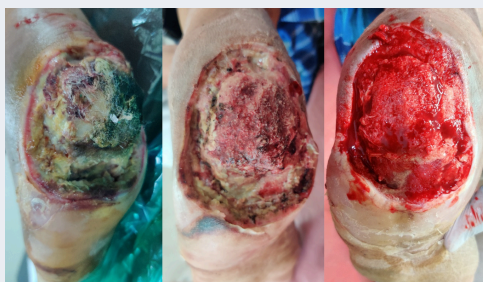
# Why Wounds Don't Heal Despite Good Sugar Control

Early recognition and intervention are critical.

**Key actions include:**

- Inspect feet routinely in all patients with diabetes.
- Assess for neuropathy using monofilament testing.
- Palpate peripheral pulses and evaluate for PAD.
- Encourage appropriate footwear and foot care.
- Optimize glycemic control and cardiovascular risk factors.
- Treat infection promptly.
- Offload pressure from ulcerated areas.
- Refer early when wounds fail to improve or when ischemia is suspected.

A diabetic foot ulcer should never be viewed as a simple skin problem. It is often a manifestation of underlying vascular disease and requires a multidisciplinary approach involving primary care physicians, endocrinologists, vascular surgeons, podiatrists and wound-care specialists.



**Fig 1. Healing heel ulcer post revascularisation**



# Heel Ulcers vs Toe Ulcers – The Art of Offloading

**Dr. Pravin Narkhede**  
Vascular & Endovascular Surgeon  
NASHIK, INDIA



## DECISION MAKING



To heal diabetic foot ulcer (DFU), you must completely eliminate mechanical pressure on the wound. This practice, known as offloading, is most critical factor in diabetic wound care, as repetitive weight-bearing causes nearly 90% of plantar healing failures.

### **Why Offloading is Essential ?**

DFU are driven by poor circulation (Vasculopathy), improper footwear, and nerve damage (Neuropathy). Neuropathy robs the patient of sensation, making it impossible to feel the friction and high-impact pressure of walking. Offloading redistributes these forces to give the tissue the environment it needs to close and heal.

### **Mechanical vs. Surgical Offloading**

International guidelines prioritize non-surgical mechanical devices as the frontline defence. Surgery is reserved for resistant cases or severe structural deformities.

### **Mechanical Options (First Line)**

First-choice treatment for non-infected, non-ischemic neuropathic plantar forefoot and midfoot ulcers

- Total Contact Cast (TCC): A non-removable fiberglass cast molded to the leg. It is the gold standard because it evenly distributes weight across the entire lower leg
- Instant Total Contact Cast (iTCC): A removable knee-high walker wrapped in fiberglass tape by medical staff to prevent the patient from removing it at home.
- Removable Knee-High Walkers: Sturdy, tall boots that transfer weight away from the foot and up into the calf muscles.

# Heel Ulcers vs Toe Ulcers – The Art of Offloading

- Removable Ankle-High Devices: Shorter boots used for stable, lower-risk wounds.
- Felted Foam Padding: Specialized foam adhered directly to the healthy skin surrounding the ulcer to transfer pressure to adjacent tissues.

## **Surgical Options (Second Line)**

When conservative, mechanical offloading options fail or severe structural foot deformity is present

- Digital Flexor Tenotomy: Cutting a tight tendon to flatten claw or hammer toes, relieving pressure on the tips.
- Metatarsal Head Resection: Removing a portion of bone beneath the ball of the foot to eliminate localized friction hotspots.
- Achilles tendon Lengthening: Elongating the calf tendon to reduce downward forces slammed into the front and middle of the foot during walking.

|                              | <u><i>Toe Ulcers (Forefoot)</i></u>                                     | <u><i>Heel Ulcers (Hindfoot)</i></u>                                                     |
|------------------------------|-------------------------------------------------------------------------|------------------------------------------------------------------------------------------|
| <b>Aetio-pathogenesis</b>    | Friction from tight shoes or pressure from claw/hammer toe deformities. | Repetitive heavy impact during walking or prolonged bed rest pressure.                   |
| <b>Blood Supply</b>          | Poor arterial circulation and micro-angiopathy                          | Poor arterial circulation                                                                |
| <b>Bone Infection Risk</b>   | Moderate<br>Phalanx/ toe bones                                          | <b>Extremely high;</b><br>Easily spreads to calcaneus (heel bone).                       |
| <b>Healing Timeline</b>      | Faster<br>Typically responsive to basic pressure shifts.                | <b>Slow and complex</b><br>Often takes many months to close.                             |
| <b>Major Amputation Risk</b> | Low<br>Localized to the digit.                                          | <b>Very High</b><br>Often requires major hind foot or below-knee amputation if infected. |



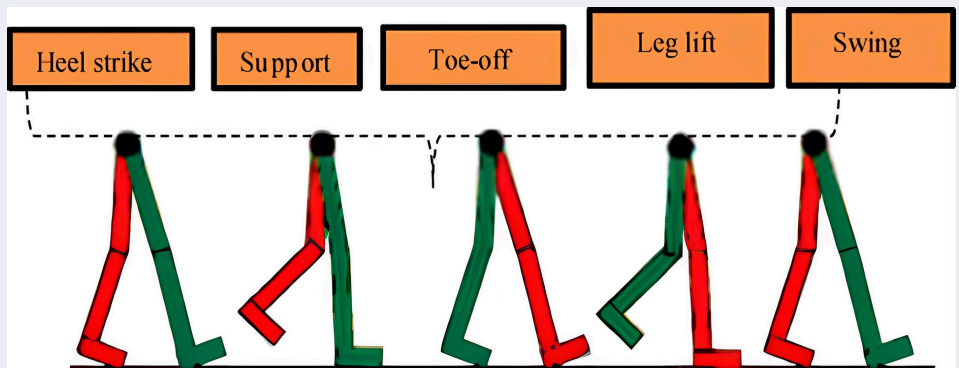
# Heel Ulcers vs Toe Ulcers – The Art of Offloading

## A Tale of Two Locations: Toe Ulcers vs. Heel Ulcers

The location of an ulcer dictates its severity, healing timeline and the risk of losing a limb.

### Targeted Offloading Strategies

The front and back of the foot absorb weight differently, requiring opposite technical designs to keep the wound bed suspended.



**Fig 1. Walking Gait Cycle**

## A Tale of Two Locations: Toe Ulcers vs. Heel Ulcers

The location of an ulcer dictates its severity, healing timeline and the risk of losing a limb.

### Targeted Offloading Strategies

The front and back of the foot absorb weight differently, requiring opposite technical designs to keep the wound bed suspended.

#### For Toe Ulcers (Forefoot Protection)

- Challenge : High pressure builds during the "toe-off" phase when pushing forward.

# Heel Ulcers vs Toe Ulcers – The Art of Offloading

- **Solution: Front Offloading Shoes (Ortho wedge) :** These feature a steep, 15-degree reverse slope where the thickest part sits under the heel. The sole cuts off entirely beneath the ball of the foot.
- **How it Works :** Shifts 100% of body weight backward, leaving the toes suspended in the open air.
- **Prevention :** Custom silicone toe spacers, specialized felted foam wraps, or wide, square-toed post-operative boots.

## **For Heel Ulcers (Hindfoot Protection)**

- **Challenge :** The heel absorbs the heavy, initial impact of every single step.
- **Solution: Back Offloading Shoes (Heel wedge) :** This is the exact geometric opposite of the front offload shoe, utilizing a thick front platform that drops off completely beneath the foot arch.
- **How it Works :** Forces the patient to bear weight exclusively on the toes and ball of the foot, protecting the heel bone from ground contact.
- **Prevention :** Knee-high walkers or customized moulded inserts with deep "cupping" under the heel.
- **Non-Ambulatory Care :** For bedridden patients, use heel-suspension boots or place pillows beneath the calves and ankles so the heels hang completely free from the mattress.



# Heel Ulcers vs Toe Ulcers – The Art of Offloading

## Essential Daily Mobility Rules

- When swelling or active infections require daily wound evaluation, removable offloading footwear becomes the centre of care. Patients must follow these guidelines:
- Wear a "Twin" Shoe : Offloading shoes have exceptionally thick, unyielding soles. You must wear a thick-soled sneaker on your healthy foot to match this height. Uneven shoes tilt your hips, causing severe lower back pain and instability.
- Walk Slowly : These devices completely alter your natural gait. Walk at a slower pace and always use a cane or walker to maintain balance.

## The Golden Rule

“You can put any medical ointment, solution, or dressing on a diabetic foot ulcer but never put the patient's weight on it”



# Needle to a Cut – What Determines the Choice of Management

**Dr. Pranjal Melge Sawant**  
Vascular & Endovascular Surgeon  
PUNE, INDIA



## MANAGEMENT

Antyllus performed the first proximal and distal artery ligation to treat an aneurysm during the Roman era. Since then, vascular surgery has evolved. But when John Murray successfully completed the first human vascular anastomosis in 1897, arterial repair really took off. In the 1950s, DeBakey began using dacron grafts to replace damaged arteries. Charles Dotter introduced minimally invasive angioplasty in the 1960s to treat clogged arteries. Since then, the field has advanced significantly, owing to the advent of catheters, balloons, stents, and stent-graft.

Now started the dilemma of which of the two accessible treatment modalities—surgical bypass and minimally invasive intervention—is superior emerged.

To put it simply, *do you prefer a needle or a knife?*

Evidence-Based Revascularization (EBR) originated here. The Global Vascular Guidelines (GVG) published the PLAN concept for EBR in 2020 with the goal of classifying each patient with an arterial blockage based on their overall fitness, comorbidities, wound status and the morphology of the arterial occlusion. This made it possible to tailor the treatment plan to the patient's need so as to facilitate clinician's decision-making.

### GOALS OF REVASCULARIZATION:

- 1.Limb Salvage and preservation
- 2.Ischemic pain relief
- 3.Wound healing
- 4.Improving quality of life

# Needle to a Cut – What Determines the Choice of Management

## Advantages of Surgical Bypass:

1. Greater patency
2. Increased durability

## Pitfalls of Surgical bypass:

1. Greater perioperative morbidity and mortality
2. Longer hospital stays
3. Dependence on availability of conduit

## Advantages of Endovascular Interventions:

1. Shorter hospital stays
2. Lesser morbidity and mortality

## Pitfalls of Endovascular Interventions:

1. Patency inferior to surgical bypass
2. Limited reconstruction capability

## PLAN Concept for Evidence-Based Revascularization:

PLAN is a three-step integrated approach based on

- A. **P**atient risk estimation
- B. **L**imb staging
- C. **AN**atomic pattern of disease

### **A.PLAN: Patient Risk Estimation:**

This entails assessing patient's candidacy for limb salvage, periprocedural risk and life expectancy. Multiple models have been proposed to assess this.

The most commonly used of these proposed models is the VQI risk assessment score that takes into account the patient's age, comorbidities, medications, and smoking habits.

Based on the risk assessment patient is classified in low-risk or high-risk categories.



# Needle to a Cut – What Determines the Choice of Management

| Tool         | End points                                                  | Critical factors                                                                                                                               | Reference                    |
|--------------|-------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------|
| Taylor et al | Mortality, ambulatory failure (median follow-up of 2 years) | Age, race, ESRD, CAD, COPD, DM, dementia, baseline ambulatory status                                                                           | Taylor, <sup>403</sup> 2006  |
| Finnvasc     | Perioperative (30-day) mortality, limb loss                 | DM, CAD, gangrene, urgent operation                                                                                                            | Biancari, <sup>63</sup> 2007 |
| PREVENT III  | AFS (1 year)                                                | ESRD, tissue loss, age >75 years, CAD, anemia                                                                                                  | Schanzer, <sup>64</sup> 2008 |
| BASIL        | Survival (2 years)                                          | Age, CAD, smoking, tissue loss, BMI, Bollinger score, serum creatinine concentration, AP (number measured and highest value), prior stroke/TIA | Bradbury, <sup>65</sup> 2010 |
| CRAB         | Perioperative (30-day) mortality, morbidity                 | Age >75 years, prior amputation or revascularization, tissue loss, ESRD, recent MI/angina, emergency operation, functional dependence          | Meltzer, <sup>66</sup> 2013  |
| Soga et al   | Survival (2 years)                                          | Age, BMI, nonambulatory status, ESRD, cerebrovascular disease, tissue loss, left ventricular ejection fraction                                 | Soga, <sup>225</sup> 2014    |
| VQI          | AFS (1 year)                                                | Age, tissue loss, DM, CHF, serum creatinine concentration, ambulatory status, urgent operation, weight, bypass conduit used                    | Simons, <sup>67</sup> 2016   |
| VQI          | Survival (30 days, 2 and 5 years)                           | Age, CKD, ambulatory status, CAD, CHF, COPD, tissue loss, diabetes, smoking, beta-blocker use                                                  | Simons, <sup>412</sup> 2018  |

AFS, Amputation-free survival; AP, ankle pressure; BASIL, Bypass vs Angioplasty in Severe Ischaemia of the Leg; BMI, body mass index; CAD, coronary artery disease; CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; CRAB, Comprehensive Risk Assessment for Bypass; DM, diabetes mellitus; ESRD, end-stage renal disease; MI, myocardial infarction; PREVENT III, Project of Ex-vivo Vein graft Engineering via Transfection III; TIA, transient ischemic attack; VQI, Vascular Quality Initiative.

## B.PLAN: Limb Staging:

EBR recommends the used of SVS Threatened Limb Classification (WIFI) which integrates:

I.Wound (W)

II.Ischemia (I)

III.foot Infection (fI)

### W: Wound:

SVS grades for rest pain and wounds/tissue loss (ulcers and gangrene): 0 (ischemic rest pain; no ulcer) 1 (mild) 2 (moderate) 3 (severe).

### I: Ischemia:

Hemodynamics/perfusion: Measure TP or TcPO<sub>2</sub> if ABI noncompressible (>1.3)

SVS grades 0 (none), 1 (mild), 2 (moderate), and 3 (severe).

### fI: foot Infection:

SVS grades 0 (none), 1 (mild), 2 (moderate), and 3 (severe: limb and/or life-threatening)

# Needle to a Cut – What Determines the Choice of Management

|     | Ischemia – 0 |     |     |     | Ischemia – 1 |     |     |     |  | Ischemia – 2 |     |     |     | Ischemia – 3 |     |     |     |
|-----|--------------|-----|-----|-----|--------------|-----|-----|-----|--|--------------|-----|-----|-----|--------------|-----|-----|-----|
| W-0 | VL           | VL  | L   | M   | VL           | L   | M   | H   |  | L            | L   | M   | H   | L            | M   | M   | H   |
| W-1 | VL           | VL  | L   | M   | VL           | L   | M   | H   |  | L            | M   | H   | H   | M            | M   | H   | H   |
| W-2 | L            | L   | M   | H   | M            | M   | H   | H   |  | M            | H   | H   | H   | H            | H   | H   | H   |
| W-3 | M            | M   | H   | H   | H            | H   | H   | H   |  | H            | H   | H   | H   | H            | H   | H   | H   |
|     | fl-          | fl- | fl- | fl- | fl-          | fl- | fl- | fl- |  | fl-          | fl- | fl- | fl- | fl-          | fl- | fl- | fl- |
|     | 0            | 1   | 2   | 3   | 0            | 1   | 2   | 3   |  | 0            | 1   | 2   | 3   | 0            | 1   | 2   | 3   |

Four classes: for each box, group combination into one of these four classes

Very low = VL = clinical stage 1

Low = L = clinical stage 2

Moderate = M = clinical stage 3

High = H = clinical stage 4

Clinical stage 5 would signify an unsalvageable foot

**Fig 1. Amputation risk according to Wifl**

## **C.PLAN: Anatomic pattern of disease (and conduit availability):**

The anatomic pattern and complexity of the arterial disease in CLTI is defined by GLASS i.e., Global Limb Anatomic Staging System.

Similar to the SYNTAX Score for Coronary artery disease, GLASS quantitatively characterizes the arterial vasculature of the lower limb quantitatively by taking into account the number, location, and complexity of the lesion as seen angiographically.

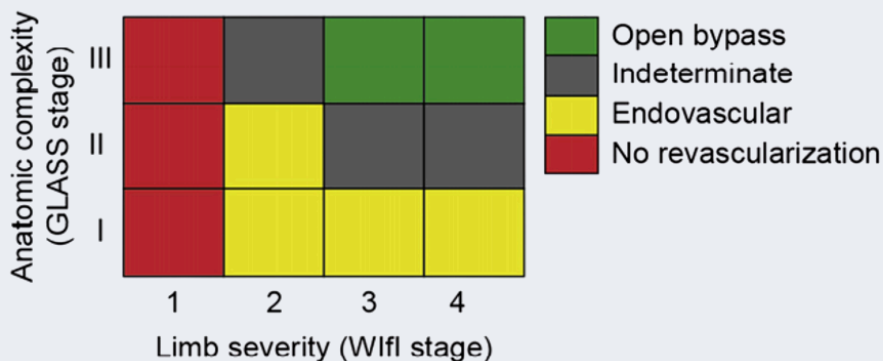
GLASS incorporates two novel and important concepts, the Target Arterial Path (TAP) and estimated limb-Based Patency (LBP). Based on appropriate angiographic imaging, the TAP is defined by the treating surgeon/interventionalist as the optimal arterial path to restore in-line flow to the ankle and foot, and resolve the clinical problem at hand.



# Needle to a Cut – What Determines the Choice of Management

## GLASS stages for the limb thus reflect a gradient of TAP complexity:

- Stage I: Average Complexity Disease: expected technical failure < 10% AND >70% 12- month LBP
- Stage II: Intermediate Complexity Disease: expected technical failure < 20% AND 12- month LBP 50-70% 24
- Stage III: High Complexity Disease: expected technical failure >20%; OR <50% 12-month LBP.

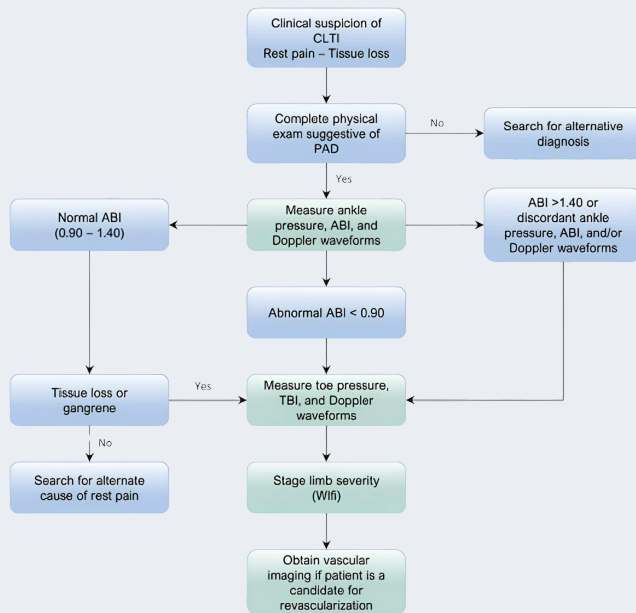


**Fig 2. Preferred Initial Revascularisation**

## Fig.3: PLAN FRAMEWORK FOR CLINICAL DECISION MAKING

Apart from the diseased arterial segments, it is to be noted that the burden of the wound and patient's comorbidities and longevity are also equally important considerations that need to be paid attention to, while planning a revascularization. The choice of management has to be individualized and catered to patient's needs. While doing this, it is of utmost importance to take into account, what kind of procedure the patient can tolerate while giving him/her the best possible outcome.

# Needle to a Cut – What Determines the Choice of Management



**Fig 3.1.** Flow diagram for the investigation of patients presenting with suspected chronic limb-threatening ischemia (CLTI). *ABI*, Ankle-brachial index; *PAD*, peripheral artery disease; *TBI*, toe-brachial index; *WIFI*, Wound, Ischemia, and foot Infection.

## Fig 3. Plan Framework for Clinical Decision Making

Apart from the diseased arterial segments, it is to be noted that the burden of the wound and patient's comorbidities and longevity are also equally important considerations that need to be paid attention to, while planning a revascularization. The choice of management has to be individualized and catered to patient's needs. While doing this, it is of utmost importance to take into account, what kind of procedure the patient can tolerate while giving him/her the best possible outcome. A high-risk patient may not tolerate a surgical intervention owing to longer procedure time, anaesthesia, post-operative morbidity, longer recovery time, extended hospital stay etc.

# Needle to a Cut – What Determines the Choice of Management

In contrast, a low or moderate risk patient, will benefit from a surgical intervention i.e., bypass which will give better long-term limb-based patency and lesser need of repeated interventions.

It is also essential to take into consideration the wound burden of the patient and the anatomy of the arterial disease. In a patient with a smaller wound burden with an angiogram showing multiple discrete lesions, an endovascular approach can be considered. In a patient with a larger wound and a long segment occlusion, bypass should be considered to give the best possible chance of limb salvage and better long-term results.

In conclusion, the choice between endovascular intervention and open bypass needs to be individualized for every patient depending on their risk profile, the condition of their wounds, and the type of vascular lesion while also taking into account the availability of a good-caliber conduit either from the same or the contralateral limb.

To put it simply, a high-risk patient with a small disease burden would benefit from an endovascular procedure, while a relatively younger patient with moderate risk and a long arterial blockage, is an ideal candidate for a bypass.

## CONCLUSION:

The concept of evidence-based revascularization in an extension of evidence-based medicine. It advocates treating each patient according to their unique circumstances, therefore there is no one treatment that works for everyone. Therefore, having both a needle and a knife in their toolbox makes vascular surgeons the most qualified to treat vascular disease.

*The best prognosis for the patient's limb and life is achieved through prompt referral and consequently, prompt action. This is vascular surgery's main objective.*



### Chitosan - Based Dressings : Old Molecule, New Promise?



As this issue closes, a smaller reflection than the rest. A colleague recently shared a set of papers on chitosan-based wound dressings, prompted by a newer product on the market, Velvert. It sent me back to the broader literature on chitosan itself, and I thought it worth sharing some of that reading with readers, since the underlying science is more interesting than any single product built on it.

Chitosan is a linear polysaccharide obtained by partial deacetylation of chitin, the structural material in crustacean shells. What sets it apart from the alginates and foams most of us reach for by default is its charge: chitosan is polycationic, and that single property appears to drive three separate effects relevant to complex wound management.

The first is antimicrobial. The positive charge binds to negatively charged components of microbial cell walls and disrupts membrane integrity. In vitro time-kill assays on chitosan dressings have reported multi-log reductions — in some cases exceeding 99% within 30 minutes — against MRSA, VRE, and carbapenem-resistant (NDM-1) *Klebsiella*. That is a striking number, though it is worth remembering it comes from laboratory testing, not clinical outcome data.

The second is hemostatic. Chitosan promotes red cell and platelet aggregation independently of the coagulation cascade, which is the more clinically interesting claim for those of us managing patients on anticoagulants or antiplatelet agents. This mechanism is reasonably well documented — a 2024 review in the *International Journal of Biological Macromolecules*, and earlier work in *Polymers and Eplasty*, all point the same way.

## Editor's Closing Note ....



The third is its role in tissue regeneration: a porous structure that absorbs exudate while keeping the wound bed moist, with some evidence of supporting fibroblast proliferation and epithelialization.

Taken together, these properties make a reasonable case for chitosan as a dressing material in venous and diabetic foot ulcers, pressure injuries, infected surgical wounds, and partial-thickness burns — the same categories of hard-to-heal wound that bring most of us back to the literature in the first place. Where I'd urge some caution is in how much of the strongest evidence is manufacturer-generated and in vitro. Velvert's own reported data are consistent with the wider chitosan literature, which lends it credibility, but I have not yet seen comparative clinical series against established dressings such as silver-impregnated foam or alginate. Before this becomes a routine choice rather than an interesting option, that is the gap that needs filling.

I raise this not to endorse a product, but because chitosan as a class seems to be maturing past its original role as a battlefield hemostatic agent, and it may be worth more of us watching this space. If any of you have used chitosan-based dressings — Velvert or otherwise — in venous, diabetic, or post-surgical wounds, I would genuinely welcome hearing about your experience, good or bad. Write in, and we'll open a Letters section with it in a future issue.

**Dr. Aniruddha Bhuiyan**  
**Editor, Inside the Vessel**





PAGE 1

### HEENA'S FOOT: THE JOURNEY TO HEALING

*Timely vascular assessment  
prevents avoidable amputations.*



**1** Meet Heena, a 58-year-old school teacher from Pune with diabetes for 15 years.

It's just a small black spot on my toe... It'll heal...

**2** Heena visits her local GP.

LOCAL CLINIC

Nothing serious. We'll do regular dressings.

Thank you, Doctor.

**3** Dressings continue... but the wound refuses to heal.

WEEK 2 WEEK 6 WEEK 12

**4** Doctor... it's been almost three months.

DIABETES CAN AFFECT YOUR FEET

Let's continue another week.

**5** Heena's son searches for answers.

Q Why isn't my diabetic foot wound healing?

Possible reasons:

- Poor blood flow
- Peripheral artery disease
- Neuropathy
- Infection

Could poor blood flow be the reason?

**6** Heena decides to visit a tertiary care hospital.

TERTIARY CARE HOSPITAL  
DEPARTMENT OF VASCULAR & ENDOVASCULAR SURGERY

Sometimes the wound isn't the problem... the circulation is.



*If a diabetic foot wound fails to improve within 2–4 weeks despite appropriate wound care, evaluate perfusion early. Timely referral to a vascular surgeon can be limb-saving.*

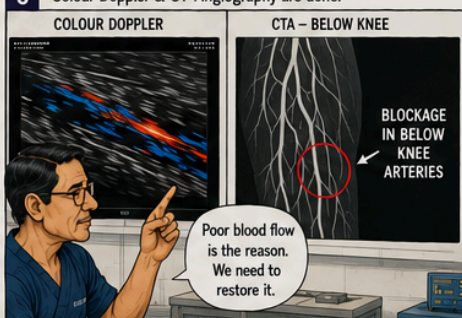


### PAGE 2

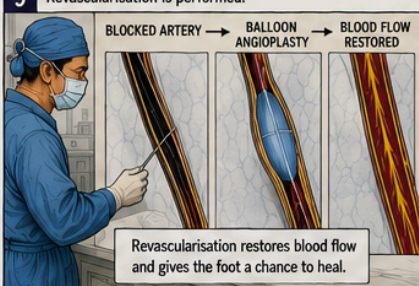
7 Vascular surgeon evaluates Heena.



8 Colour Doppler & CT Angiography are done.



9 Revascularisation is performed.



10 Debridement of right great toe performed.



11 Debridement of right great toe performed.



12 Follow-up - healed at 8 weeks.



13 The right doctor for the right job!



**EVERY DIABETIC FOOT WOUND DESERVES A VASCULAR ASSESSMENT.  
THINK BEYOND THE DRESSING. THINK CIRCULATION. SAVE LIMBS.**







**7 PREPARING THE WOUND**

- ✓ Infection controlled
- ✓ Regular wound care
- ✓ Appropriate antimicrobials
- ✓ Compression started

It already feels better.



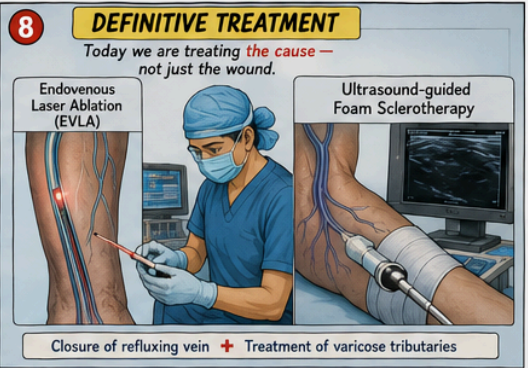
**8 DEFINITIVE TREATMENT**

Today we are treating **the cause** — not just the wound.

Endovenous Laser Ablation (EVLA)

Ultrasound-guided Foam Sclerotherapy

Closure of refluxing vein + Treatment of varicose tributaries



**9 RECOVERY**

I haven't walked this comfortably in months!

- ✓ Pain reduced
- ✓ Swelling reduced
- ✓ Wound getting smaller
- ✓ Back to daily activities



**10 HEALING**

After a few weeks...

**BEFORE**

The ulcer has healed beautifully.

**AFTER**

Treat the **cause**, and the wound heals.



**11 THE ADVICE**

Continue wearing compression stockings every day for the next **3 months**.

That helps prevent recurrence.

COMPRESSION STOCKINGS

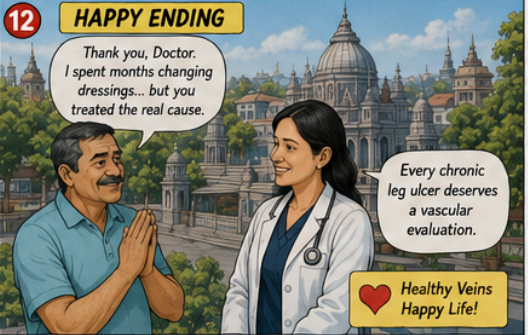


**12 HAPPY ENDING**

Thank you, Doctor. I spent months changing dressings... but you treated the real cause.

Every chronic leg ulcer deserves a vascular evaluation.

♥ Healthy Veins Happy Life!



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| 2.     | Dr. Paresh Rajanikant Pai | drpareshpai@gmail.com          | Mumbai |
| 3.     | Dr. Raghuram Sekhar       | drsekhar@gmail.com             | Mumbai |
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**Dr. David G Armstrong**

***"At the end of an anatomic peninsula,  
the foot in diabetes is prone to  
complications involving neuropathy,  
vascular disease and infection."***