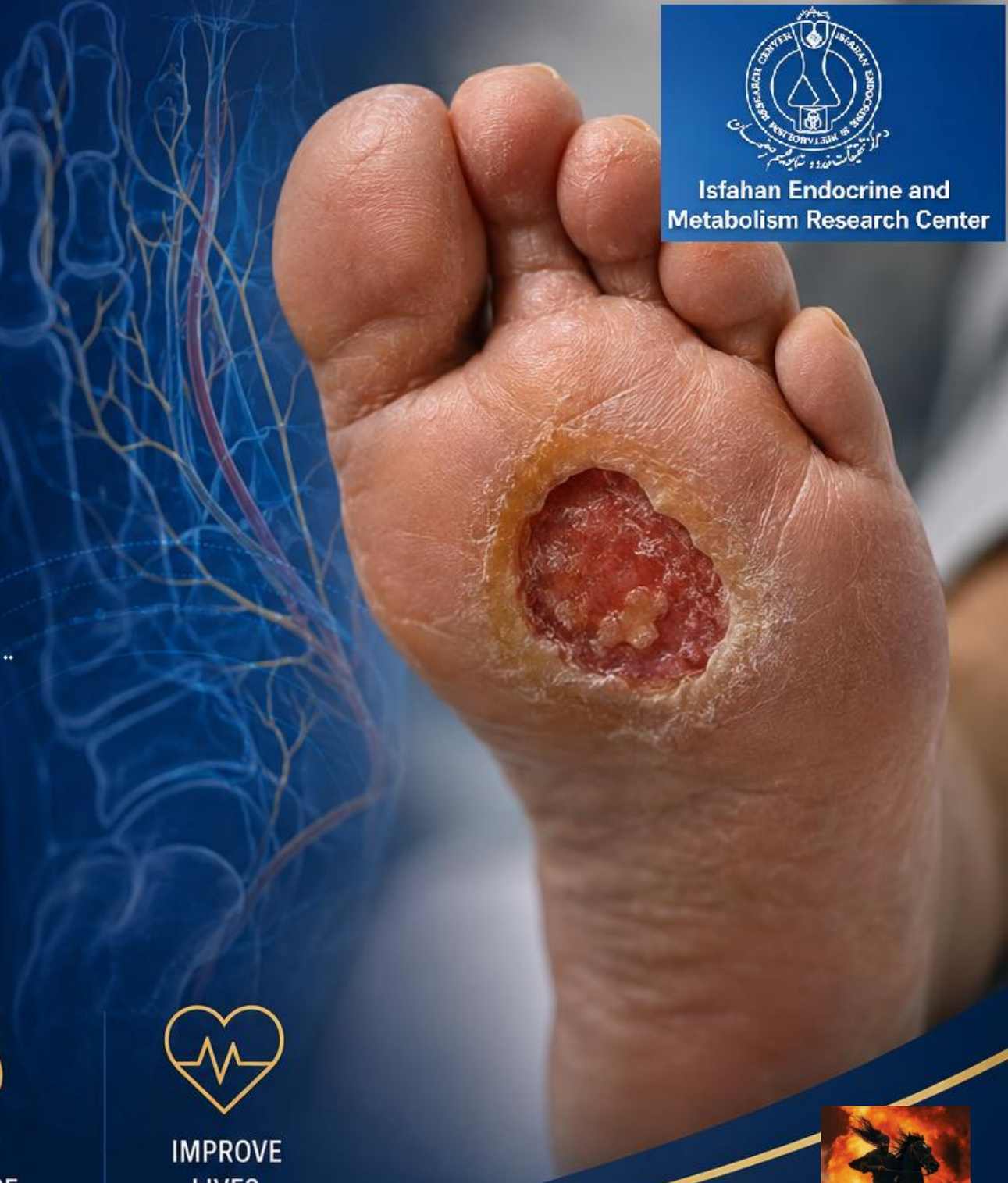
DIABETIC FOOT ULCERS

From Wound Assessment to Limb Preservation

Mansour Siavash, MD,
Professor of Endocrinology and Metabolism

Isfahan Endocrine and Metabolism Research Center



ASSESS ACCURATELY

TREAT THE CAUSES

PROMOTE HEALING

PREVENT RECURRENCE

IMPROVE LIVES

A CLINICAL JOURNEY THROUGH THE DIABETIC FOOT

Five decisions that organize the lecture—and the bedside encounter

01 **UNDERSTAND** — Why ulcers occur—and why they recur

02 **ASSESS** — Mechanism, wound, infection, bone and perfusion

03 **TRIAGE** — Recognize limb- or life-threatening disease

04 **TREAT** — Apply the six pillars in parallel

05 **ESCALATE** — Dressings and advanced therapies—at the right time

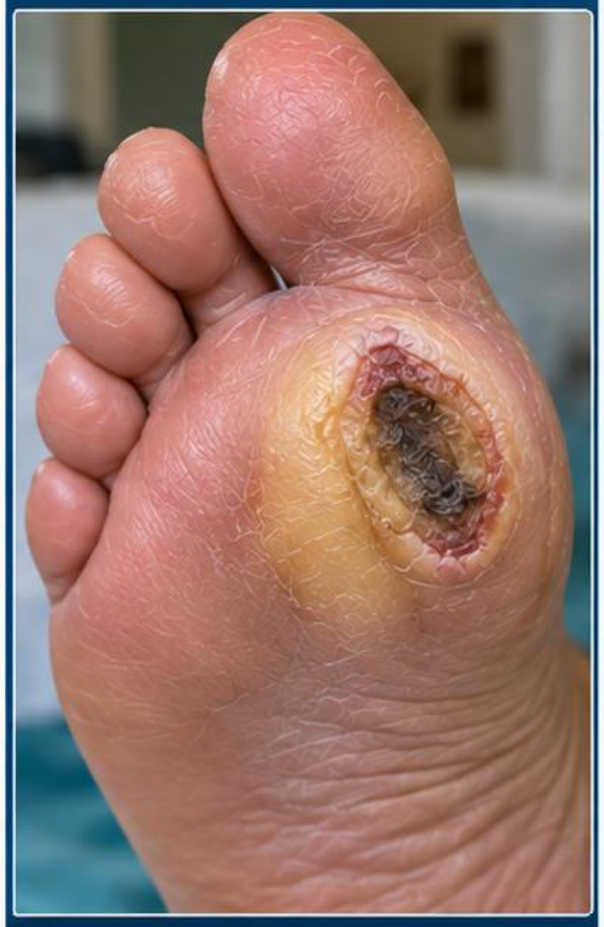
Case 1: A Complex Patient with Diabetes and Peripheral Artery Disease (CLTI)

Initial Presentation (1 of 2)

Isfahan Endocrine and Metabolism Research Center (IEMRC)
Isfahan University of Medical Sciences

Right foot
(of the patient)

Right foot – plantar view
(enlarged view of the same foot)



Plantar ulcer under the 1st metatarsal head
(approx. 3 × 2 cm)

Patient Profile

Age / Sex: 68-year-old man

Chief Complaint

- Non-healing right foot ulcer for 3 months, increasing pain, foul discharge

Past Medical History

- Type 2 diabetes mellitus (20 years)
- Hypertension
- Chronic kidney disease stage 3 (eGFR ~ 45 mL/min)
- Ischemic heart disease (PCI 5 years ago)
- Ex-smoker (40 pack-years, quit 5 years ago)

Medications

- Insulin, statin, aspirin, ACE inhibitor, β-blocker

Functional Status

- Limited walking (< 50 meters) due to leg/foot pain

Social

Lives with his wife, needs support for daily activities

Is this PAD — and how would you assess limb perfusion?

A multidisciplinary approach can save limbs and lives

Case 1: A Complex Patient with Diabetes and Peripheral Artery Disease (CLTI)

Vascular Assessment (2 of 2): Don't Be Reassured by the ABI

Isfahan Endocrine and Metabolism Research Center (IEMRC)
Isfahan University of Medical Sciences

Clinical Examination (Right Foot)

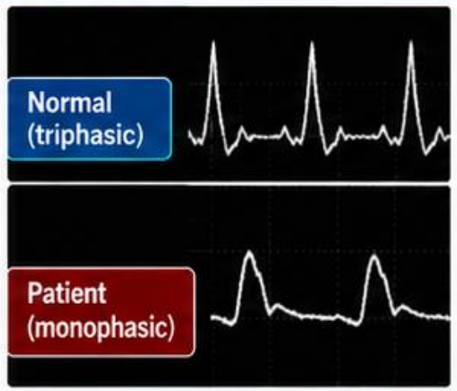
Foot temperature:	Cool
Capillary refill:	> 4 seconds
Dorsalis pedis pulse:	Not palpable
Posterior tibial pulse:	Not palpable
Sensory exam:	Reduced (diabetic neuropathy)
Skin changes:	Dry skin, callus, distal discoloration

Right foot – plantar view
(same patient as previous slide)

Laboratory Data

HbA1c:	8.2 %
Creatinine:	1.6 mg/dL (eGFR ≈ 45 mL/min)
Hemoglobin:	10.8 g/dL
CRP:	4.5 mg/dL

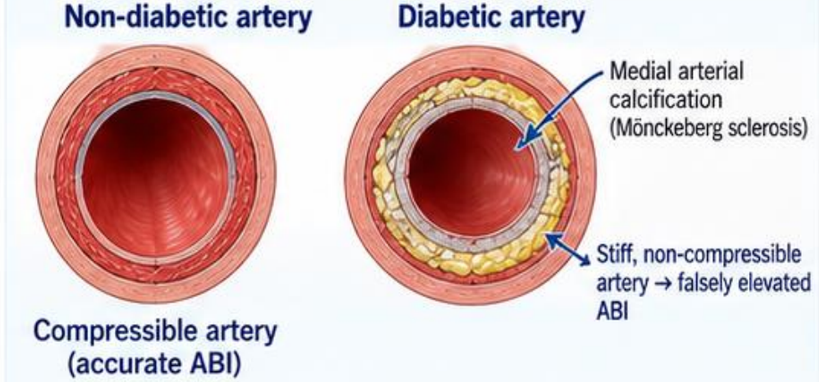
Pedal Doppler Waveforms (Right Leg)



Non-invasive Vascular Testing (Right Leg)

ABI:	1.28	Deceptively reassuring (may be falsely elevated due to medial arterial calcification)
Toe-brachial index (TBI):	0.28	Abnormal
Toe pressure:	28 mmHg	Markedly reduced (< 30 mmHg)
Transcutaneous oxygen pressure ($TcPO_2$, dorsum):	28 mmHg	Reduced (< 30 mmHg)
Pedal Doppler waveform (DP/PT):	Monophasic	Abnormal

Why ABI Can Be Misleading in Diabetes



Key Teaching Point

In diabetes, a normal or high ABI **does not exclude PAD.**

When clinical suspicion remains high, evaluate toe pressure/TBI and pedal Doppler waveforms.

What is the limb threat in this patient?

Next steps: → Stage the limb (WIfI) → Define anatomy (CTA/angiography) → Assess complexity (GLASS) → Plan (revascularization strategy)

From assessment to action ...

2

Why Diabetic Foot Ulcers Still Matter

A Global Health Burden – A Personal Catastrophe

GLOBAL & HEALTH-SYSTEM BURDEN

A major challenge for health systems worldwide

19–34%
Lifetime risk of developing a foot ulcer among people with diabetes

~2–4% / year
Approximate annual DFU incidence reported in higher-income settings; greater in many low-income countries

>1 MILLION
Amputations of the lower limb each year among people with diabetes
≈ 1 limb every 20 seconds

MAJOR HEALTHCARE-RESOURCE CONSUMPTION
Drives repeated outpatient care, hospitalization, surgery, antibiotics, vascular procedures, rehabilitation and long-term care

In low-income settings, treatment of a complex DFU may cost the equivalent of **5.7 YEARS** of annual income

DFU is a leading cause of hospitalization and amputation and consumes a substantial portion of diabetes care budgets.

DFU is not simply a wound.

It is a limb-threatening, life-threatening and economically consequential complication of diabetes.

THE BURDEN TO THE PATIENT

A life-changing journey with high risk of recurrence and death

DFU → Infection → Hospitalization → Amputation → Disability

40% Recurrence within **1 year** after healing

~65% Recurrence within **3 years** after healing

≈50% 5-year mortality

UP TO 85% Major lower-limb amputation preceded by an ulcer

- Loss of mobility
- Loss of independence
- Impaired quality of life
- Psychological burden
- Financial burden on patient and family
- High risk of re-ulceration and re-amputation

DFU profoundly affects every dimension of a person's life, and repeatedly threatens the limb and life.

PREVENT THE ULCER › SAVE THE LIMB › PROTECT THE LIFE

KEY POINT Effective prevention, early detection, and optimal management of DFU are essential to reduce amputations, deaths, suffering and costs.

SOURCES:
- IWGDF Guidelines 2023 (www.iwgdfguidelines.org)
- IWGDF Global Burden Methodology 2023
- Armstrong DG, et al. Diabetes Metab Res Rev. 2023;39:e3652.
- Zhao Y, et al. Diabetes Obes Metab. 2021;23:1782–1791.
- ADA Standards of Care in Diabetes—2024/2025.

3

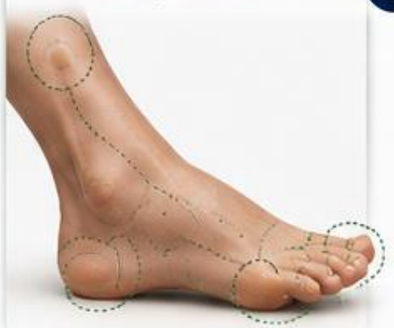
The Lifetime Journey of the Diabetic Foot

From At Risk to Remission – and Back Again

DFU is not a single event – it is a chronic, recurrent condition.
Our goal: Keep the foot in remission for life.

1 AT RISK FOOT

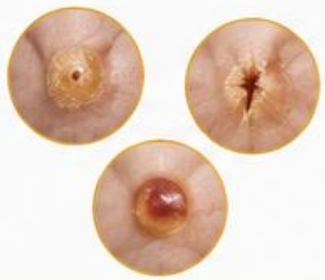
Neuropathy, PAD, deformity, previous ulcer or amputation



High risk of ulceration requires lifelong preventive care

2 PRE-ULCERATIVE LESION

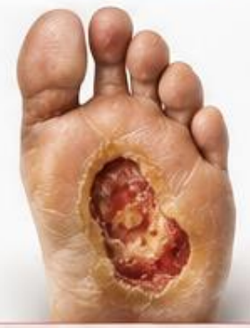
Callus, fissure, blister, hemorrhage, redness, or minor trauma



Early detection and offloading can prevent ulceration

3 DIABETIC FOOT ULCER

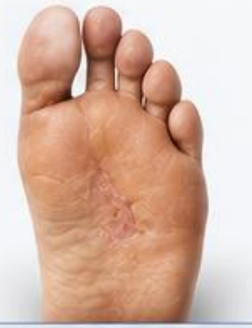
Breakdown of skin and tissue – may be infected and/or ischemic



Needs comprehensive assessment and evidence-based care

4 HEALING

Wound closure and tissue repair



Monitor healing trajectory and address underlying causes

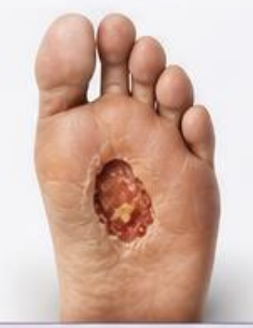
5 REMISSION (HEALED FOOT)

No open ulcer, but underlying risks remain

Prevent recurrence with offloading, footwear, skin care and education

6 RECURRENCE (VERY COMMON)

New ulcer can occur at the same site or elsewhere



Recurrence risk is high without continuous risk management

WHAT THE EVIDENCE TELLS US

19–34%
Lifetime risk of DFU among people with diabetes[1]

40%
Ulcer recurrence within 1 year after healing[2]

~65%
Ulcer recurrence within 3 years after healing[2]

~50%
5-year mortality after a DFU episode[3]

DFU is a leading cause of amputation, hospitalization, disability and reduced quality of life worldwide.

KEY MESSAGE: Our mission is not only to heal the ulcer, but to keep the foot in remission for life.

ASSESS RISK REGULARLY | PROTECT THE FOOT | REDUCE PRESSURE | DETECT EARLY | EDUCATE & EMPOWER

PAD: Peripheral Arterial Disease DFU: Diabetic Foot Ulcer

1. IWGDF Guidelines 2023 2. Van Netten JJ, et al. Diabetologia. 2021;64:2109–2118. 3. Armstrong DG, et al. Diabetes Care. 2020;43:991–1000.

4

The "Perfect Storm" of DFU

Multiple Interacting Factors Converge to Cause Ulceration

1 NEUROPATHY

Loss of protection, altered biomechanics, and poor autonomic regulation

Sensory loss
- No pain perception
- Repetitive trauma

Motor dysfunction
- Muscle imbalance
- Deformity (claw toes, prominent metatarsal heads, Charcot foot)

Autonomic dysfunction
- Dry, cracked skin
- Abnormal sweating
- Reduced skin perfusion

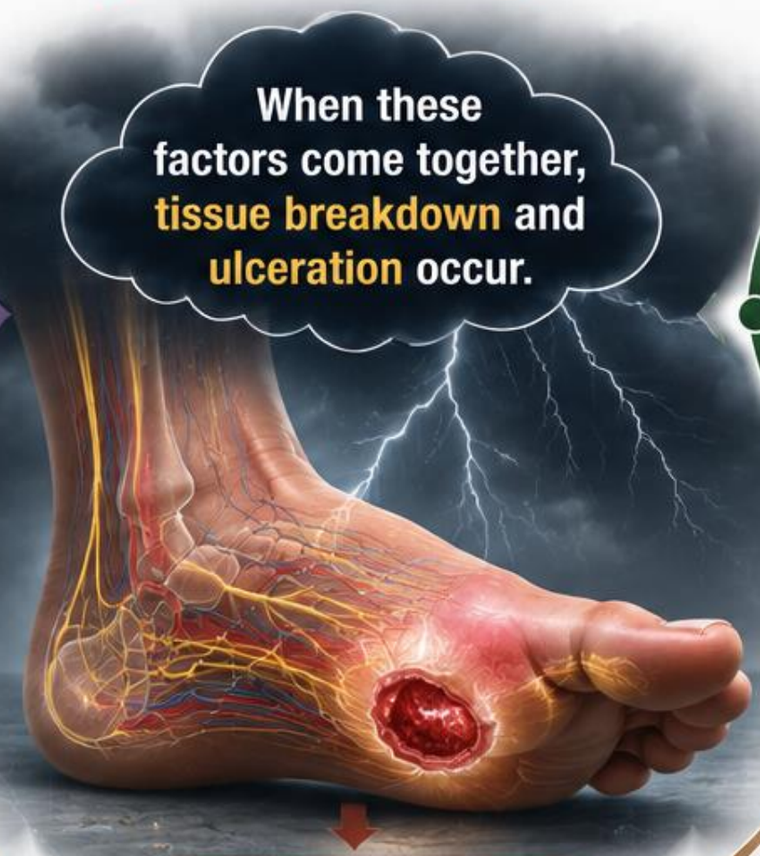
2 MECHANICAL STRESS

Excessive and repetitive plantar pressure leads to tissue injury

Repetitive plantar pressure
- High pressure points
- Prolonged standing/walking

Shear and friction
- Skin damage
- Blister, callus formation

Footwear & environment
- Inappropriate footwear
- Foreign body, minor trauma



3 PERIPHERAL ARTERY DISEASE (PAD)

Impaired blood flow and oxygen delivery impair healing and defense

Reduced perfusion
- Atherosclerosis
- Microvascular disease

Tissue hypoxia
- Delayed wound healing
- Increased risk of tissue loss

Impaired host defense
- Poor immune cell function
- Increased infection risk

⚠ THE RESULT

- ☑ Impaired tissue integrity and skin breakdown
- ☑ Ulceration
- ☑ Infection
- ☑ Impaired healing
- ☑ Risk of amputation and death

4 DIABETES & SYSTEMIC FACTORS

A hostile systemic environment impairs healing and immunity

Hyperglycemia
- Glycation end-products
- Oxidative stress

Inflammation
- Chronic low-grade inflammation
- Impaired cytokine response

Comorbidities
- CKD, anemia, malnutrition
- Smoking, dyslipidemia, obesity

THE PATHWAY TO COMPLICATIONS

Repetitive trauma → Callus formation → Subcutaneous hemorrhage → Skin breakdown / Ulceration → Infection → Tissue loss / Amputation → Increased mortality

Understanding and addressing ALL these factors is essential for healing the ulcer and preventing recurrence.

5

Neuropathy + Pressure: The Ulcer Generator

Loss of Protective Sensation Turns Repetitive Pressure into Ulceration

The same pressure is harmless in a normal foot — but dangerous in a neuropathic foot.

NORMAL FOOT (WITH PROTECTIVE SENSATION)

1. **PRESSURE** — Nociceptors activated
2. **PAIN PERCEPTION** — Pain perceived by the brain
3. **BEHAVIORAL RESPONSE** — Unload, shift weight, protect the area
4. **PRESSURE RELIEF** — Tissue remains healthy
5. **TISSUE INTACT**

NEUROPATHIC FOOT (LOSS OF PROTECTIVE SENSATION)

1. **PRESSURE** — Nerve damage blunts or abolishes pain sensation
2. **NO PAIN NO WARNING** — Continued pressure and microtrauma
3. **REPETITIVE TRAUMA** — • Callus formation • Subcutaneous hemorrhage • Fat pad atrophy • Tissue ischemia
4. **TISSUE DAMAGE PROGRESSES** — Breakdown of tissue → Ulceration
5. **ULCER**

Key Point
Pain is a warning system.
It protects the foot from damage.

Take-Home Message
Without pain, the foot does not know it is being injured.
Repetitive pressure ***silently*** destroys tissue and creates an ulcer.

Key Contributors

- Peripheral neuropathy (sensory loss)
- Motor neuropathy (deformity, muscle imbalance)
- Autonomic neuropathy (dry skin, ↓ sweating, cracked skin)
- Poor footwear & inappropriate insoles
- Foot deformities (claw toes, prominent metatarsal heads)
- High body weight and sedentary behavior

CLINICAL IMPLICATION

The cornerstone of prevention and healing is **offloading** to eliminate repetitive pressure.

BREAK THE CYCLE – PROTECT THE FOOT

Identify at-risk foot early → Educate the patient & family → Remove pressure (offloading) → Inspect daily → Prevent ulcer, prevent amputation

A small ulcer begins with repetitive pressure.

Prevention starts today.

NO PAIN ≠ NO PROBLEM. Protect the insensate foot as diligently as the painful one.

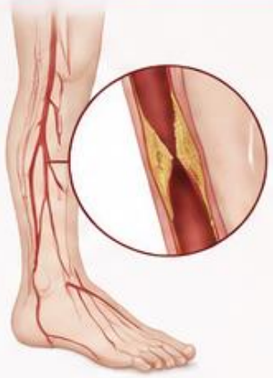
6

PAD Changes Everything

Poor Perfusion Is Both a Cause and an Amplifier of Diabetic Foot Ulcers

Adequate Blood Flow Is the Foundation of Healing – Without It, Every Other Therapy Is Compromised

PAD CAN ITSELF CAUSE ISCHEMIC ULCERS



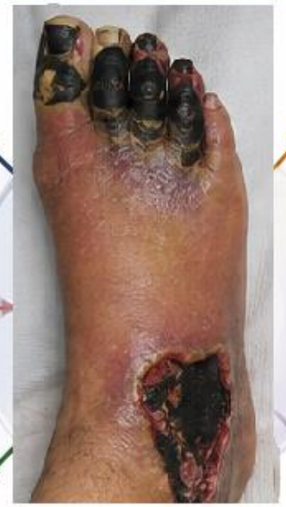
Mechanism

- Atherosclerotic blockage reduces blood flow
- ↓
- Tissue ischemia (hypoxia, acidosis, nutrient deprivation)
- ↓
- Tissue necrosis and ulceration

Typical Features

- Often on toes, pressure-free areas
- Pain (especially at rest or at night)
- Pale, cool skin, thin shiny appearance
- Weak or absent pulses

WHEN DFU COEXISTS WITH PAD



SLOWER HEALING
Impaired oxygen delivery and collagen synthesis delay tissue repair

ULCER RECURRENCE AND CHRONICITY
Poor perfusion predisposes to recurrent ulceration and non-healing

GREATER INFECTION RISK
Ischemic tissue is more susceptible to infection and spreads faster

MORE COMPLICATIONS
Higher rates of osteomyelitis, gangrene, and **hospitalization**

HIGHER RISK OF AMPUTATION
Critical ischemia is the strongest predictor of major amputation

WORSE PROGNOSIS
Increased mortality and cardiovascular events

ISCHEMIA SEVERITY MATTERS

From Claudication to Critical Limb-Threatening Ischemia (CLTI)

INCREASING SEVERITY
INCREASING RISK

No PAD
Normal perfusion –
healing potential preserved

Intermittent Claudication
Exercise pain – early PAD
Risk of DFU is increased

Chronic Limb-Threatening Ischemia
Rest pain, non-healing ulcer,
or gangrene
High risk of limb loss

Critical Ischemia
Very low perfusion,
tissue loss inevitable
without urgent revascularization

EVALUATE PERFUSION IN EVERY DFU PATIENT

Clinical Assessment
Pulses, skin, temperature, capillary refill, rest pain

Doppler Waveform
Triphasic / Biphasic / **Monophasic**

Ankle-Brachial Index (ABI)
>0.9 normal
0.5–0.9 PAD
<0.5 severe PAD

Toe Pressure or TBI
Toe pressure <50 mmHg or TBI <0.7 indicates poor healing potential

Advanced Tests
$TcPO_2$, SPP, imaging (duplex, CTA, MRA) as needed

MANAGEMENT PATHWAY

Confirm PAD Severity
Stratify limb threat (e.g., WIfI)

Imaging
Define anatomy and target vessels

Revascularization When Indicated
Endovascular or surgical to restore direct blood flow

Optimize Wound Care
After revascularization, healing potential is restored

Secondary Prevention
Antiplatelet, statin, BP & glucose control, smoking cessation, exercise

TAKE-HOME MESSAGE

If you do not assess perfusion, you cannot heal the ulcer.

- ✓ PAD is common in DFU (>50% in many series).
- ✓ Ischemia increases the risk of major amputation up to 10–30-fold.
- ✓ Timely revascularization saves limbs and lives.

MULTIDISCIPLINARY VASCULAR CARE = Limb Preservation

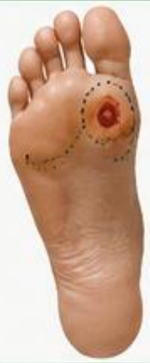
Sources: IWGDF PAD Guideline 2023 • IWGDF Practical Guidelines on the Management and Prevention of DFU 2023 • Faglia E, et al. Diabetes Care. 2018;41:2229–2236. • Aziz Z, et al. J Vasc Surg. 2022;75:200–208.

7

Let the Ulcer Tell Its Story

Six Questions at the First Encounter

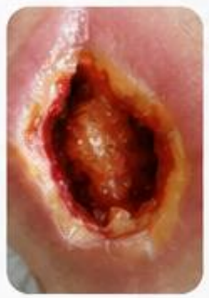
Before we choose a treatment, we must understand the wound, the foot, and the patient.



1 WHERE is it?

Location often suggests the underlying mechanism.

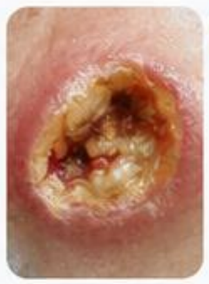
- Pressure points (plantar, heel) → neuropathic
- Distal toes / lateral foot → ischemic
- Between toes / plantar web spaces → infectious
- Anywhere / geometric pattern → traumatic
- Unexpected location → think atypical



2 HOW DEEP is it?

Depth determines prognosis and need for further evaluation.

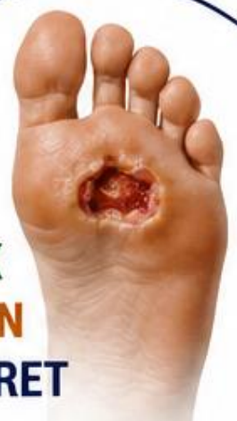
- Superficial (skin only)
- Subcutaneous tissue
- Tendon / joint capsule
- Bone (suspected or exposed)



3 IS IT INFECTED?

Identify clinical infection, not just colonization.

- Local signs: erythema, warmth, swelling, pain, purulent discharge, delayed healing
- Systemic signs (if present): fever, chills, leukocytosis, malaise



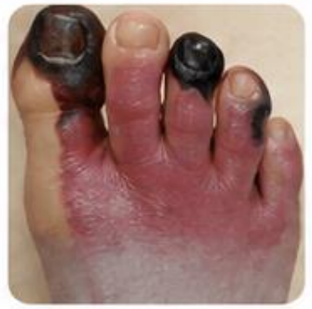
LOOK
LISTEN
INTERPRET

The ulcer often reveals the mechanism that created it.

4 IS IT ISCHEMIC?

Adequate blood flow is essential for healing.

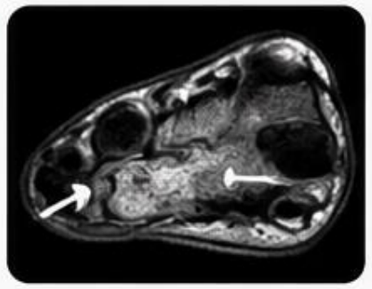
- Look: color, temperature, hair loss, atrophy, dependent rubor
- Palpate: pulses (DP, PT), capillary refill
- Assess: ABI / TBI, toe pressures, $TcPO_2$ / SPP if available



5 IS BONE INVOLVED?

Always consider osteomyelitis in:

- Deep ulcers
- Ulcers over bony prominences
- Chronic or recurrent ulcers
- Ulcers that probe to bone or have exposed bone



6 WHY did it occur—and what is preventing healing?

Identify and correct all contributing factors.

Pressure / Offloading · Ischemia · Infection · Deformity / Biomechanics · Systemic factors · Other / Atypical causes

A SYSTEMATIC FIRST LOOK
Answer these six questions every time you see a DFU.

THE GOAL
Determine the cause(s) and barriers to healing—then eliminate them.

CORRECT THE CAUSE. REMOVE THE BARRIER. HEAL THE PATIENT.

Treating the wound without answering these questions is treating the wrong problem.

★ **Every diabetic foot ulcer has a story. Our job is to read it—and act on it.**

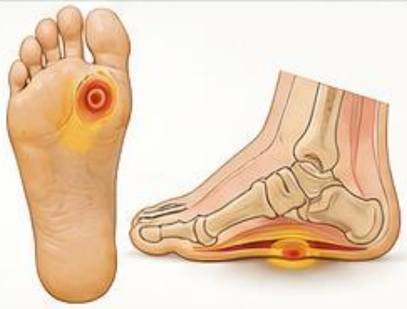
SLIDE 7 COMPLEMENTARY

BEYOND SEVERITY — DISCOVER WHY THE ULCER OCCURRED

Khorshid Hospital Mechanism-Based Classification of Diabetic Foot Ulcers

Assessing the underlying mechanism is essential—because removing the cause is often the key to healing.

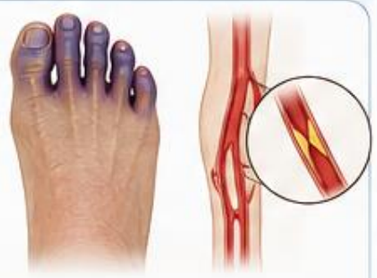
1 CHRONIC PRESSURE / NEUROPATHIC



- Usually painless
- Callus-associated
- High-pressure plantar site, especially metatarsal heads
- Pedal pulses often preserved
- Surrounding skin usually normal / pink or yellowish

Key mechanism:
Repetitive pressure + loss of protective sensation

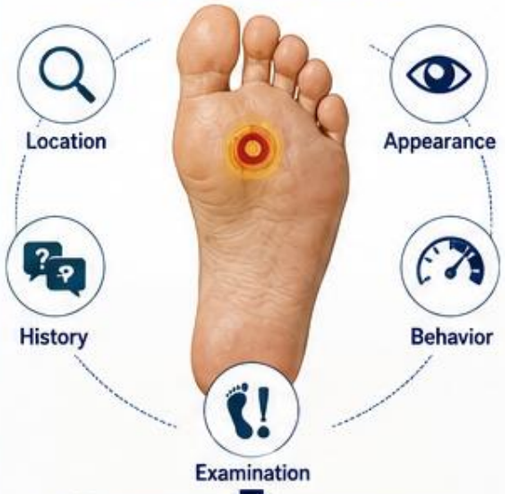
2 ISCHEMIC



- Often painful and distal, particularly toes
- Pedal pulses markedly reduced or absent
- Cyanotic / deep-purple appearance may occur
- May initially appear as a blister or small distal lesion
- Associated signs: thin/shiny skin, atrophy, hair loss

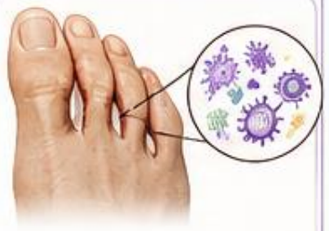
Key mechanism:
Inadequate arterial perfusion

Every ulcer has a story.
Our job is to read it.



LOOK at the ulcer
RECOGNIZE the pattern
ASK the history
IDENTIFY the mechanism
REMOVE the cause

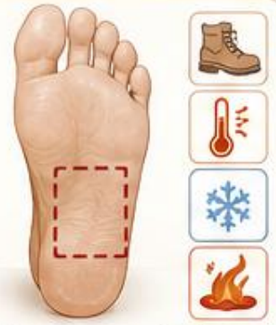
3 PRIMARY INFECTIOUS



- While any DFU may become secondarily infected, in this phenotype infection is an initiating mechanism
- Often interdigital or beneath the toes
- May begin with fungal maceration or skin-barrier disruption
- Can subsequently extend into deeper tissues
- Usually purulent, deep and destructive

Key mechanism:
Initial infection → skin breakdown → deeper bacterial involvement

4 ACUTE TRAUMATIC



- May occur at any location
- Morphology and location may provide clues to the external cause
- Consider repetitive mechanical exposure, object-related injury, heat, cold, scalding, occupational or behavioral exposures
- Sometimes self-inflicted

Key principle:
Ask what repeatedly contacts this part of the foot.

5 ATYPICAL

- Unexpected location, morphology, clinical course, or response to appropriate treatment
- Should trigger diagnostic reconsideration
- Consider uncommon infection, malignancy, inflammatory / dermatologic disease, unusual trauma, and other rare causes

Key message:
Unexpected behavior → reconsider the diagnosis.

TWO COMPLEMENTARY PERSPECTIVES

Severity-Based Assessment
"How bad is the ulcer?"
(Depth, infection, ischemia, extent, risk to limb)

↔

Mechanism-Based Assessment
"Why did the ulcer occur?"
(Etiology, provoking factors, underlying pathogenesis)

These approaches are complementary, not competing.

KEY MESSAGES

Severity tells us how bad the ulcer is.
Pathogenesis tells us **why** it happened—and may tell us **what must be corrected** for healing.

The ulcer often tells its story
through its location, appearance, behavior, and history.

Khorshid Hospital DFU Classification —
mechanism-, feature-, and behavior-based clinical framework (Siavash et al.).

Recognizing the cause is often the most powerful step toward healing.
Treat the mechanism. Remove the cause. Help the ulcer heal.

DIABETIC FOOT CARE
PREVENT • DETECT • HEAL

SLIDE 8

DESCRIBE THE WOUND SYSTEMATICALLY

A Structured Bedside Assessment of Every Diabetic Foot Ulcer

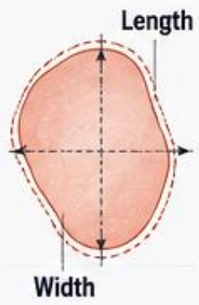
A systematic description is the foundation of correct management and meaningful follow-up.

1 SITE — Where is it?

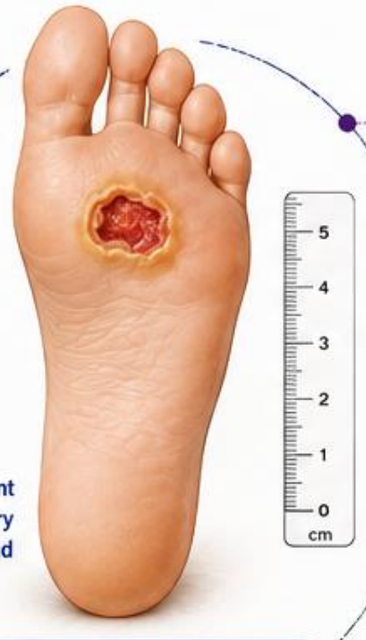
- Plantar vs dorsal
- Toe / interdigital
- Heel
- Medial / lateral border
- Over a bony prominence
- Pressure-bearing vs non-pressure-bearing

Location may provide the first clue to etiology.

2 SIZE — How large is it?

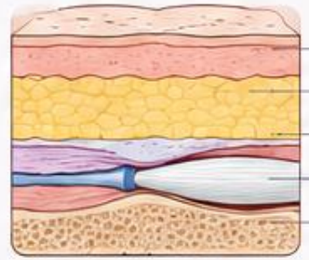


- Length × Width (cm)
- Surface area (when appropriate)
- Maximum depth (cm)
- Use same method every time
- Standardized photography with scale



Use a consistent method at every assessment and follow-up.

3 DEPTH — What structures are involved?



Depth often matters more than surface appearance.

4 WOUND BED — What is in the wound?

Granulation (tissue) | Slough | Necrotic / nonviable tissue | Exposed deeper structures

5 EDGE & PERIWOUND — What surrounds it?

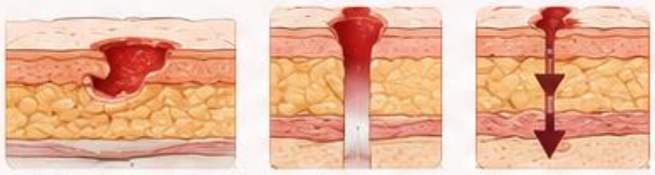
Callus / hyperkeratosis | Maceration | Undermining | Erythema | Edema | Dry / fissured skin | Ischemic changes

The tissue surrounding the ulcer is part of the diagnosis.

6 EXUDATE — What is coming from it?

- Character (serous, serosanguinous, purulent, etc.)
- Purulence (if present)
- Odor (supportive finding only)

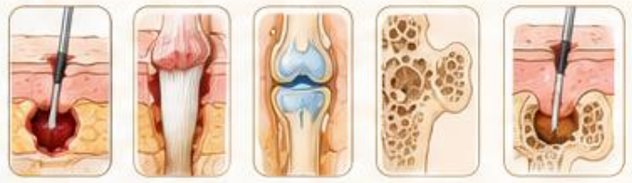
7 UNDERMINING / SINUS / TRACKING



Undermined edge | Sinus tract | Deep extension

The visible opening may underestimate the true extent of disease.

8 PROBE & DEEP STRUCTURES



Probe to bone (PTB) | Exposed tendon | Joint involvement | Exposed bone | Suspicion for osteomyelitis

DO NOT ASSESS THE ULCER IN ISOLATION.

Wound description must be integrated with:

Perfusion | Infection | Neuropathy | Pressure / Offloading | Deformity | Comorbidities & Patient Factors

- SITE
- SIZE
- DEPTH
- BED
- EDGE / PERIWOUND
- EXUDATE
- UNDERMINING / TRACKING
- DEEP STRUCTURES

DOCUMENT → PHOTOGRAPH → MEASURE → COMPARE OVER TIME

AT EVERY FOLLOW-UP

- Is it smaller?
- Is it shallower?
- Is the wound bed improving?
- Are the edges advancing?
- Is infection controlled?
- Has perfusion / offloading been addressed?

MEASURE WHAT YOU SEE—BUT INVESTIGATE WHAT YOU CANNOT SEE.

A reproducible wound description establishes the baseline, guides management, and allows objective assessment of healing.

The ulcer often tells its story through its location, appearance, behavior, and history.

*Khorshid Hospital DFU Classification — mechanism-, feature-, and behavior-based **clinical framework** (Siavash et al.).*

SLIDE 9

CLASSIFY WITH PURPOSE

Choose the Classification That Answers Your Clinical Question

Classify when the result changes communication, prognosis, or management.

There is no single "best" DFU classification—because different systems answer different questions.

1 WHY DID IT OCCUR?

Khorshid Mechanism-Based Classification

Identify the underlying mechanism and remove the cause

1. Chronic Pressure / Neuropathic	Repetitive pressure, loss of protective sensation
2. Ischemic	Inadequate arterial perfusion
3. Primary Infectious	Initial infection → skin breakdown → deeper involvement
4. Acute Traumatic	External injury: mechanical, thermal, chemical, or other
5. Atypical	Unexpected location, morphology, course or response

The ulcer's location, appearance, and behavior tell a story. Find the cause—or the ulcer will return.

2 HOW SHOULD I DESCRIBE OVERALL ULCER SEVERITY?

SINBAD Classification

Standardized communication and outcome comparison

	Domain	Definition	Score
S	Site	Forefoot	0
		Midfoot or hindfoot	1
I	Ischemia	Pedal blood flow intact: ≥1 pulse palpable	0
		Clinical evidence of reduced pedal blood flow	1
N	Neuropathy	Protective sensation intact	0
		Protective sensation lost	1
B	Bacterial infection	None	0
		Present	1
A	Area	Ulcer <1 cm²	0
		Ulcer ≥1 cm²	1
D	Depth	Ulcer confined to skin and subcutaneous tissue	0
		Ulcer reaching muscle, tendon, or deeper	1

Total SINBAD score: 0–6

Higher score = more severe ulcer

DIFFERENT QUESTIONS REQUIRE DIFFERENT TOOLS

QUESTION → CHOOSE THE TOOL → CLASSIFY → ACT

3 HOW SEVERE IS THE INFECTION?

IWGDF/IDSA Infection Classification

Guides diagnosis and infection management

Uninfected	No signs or symptoms of infection
Mild	• Local infection • ≤2 cm erythema • Superficial
Moderate	• Erythema >2 cm OR depth / abscess OR cellulitis
Severe	• Systemic toxicity • OR deep / extensive infection

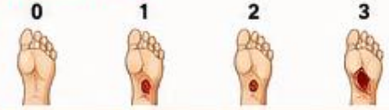
Essential for antibiotic decisions, hospitalization, and escalation of care.

4 HOW THREATENED IS THE LIMB?

SVS WIfI Classification

Estimates amputation risk and potential benefit of revascularization

W Wound (0–3)



I Ischemia (0–3)

fI foot Infection (0–3)

Risk of Amputation at 1 year (combine W + I + fI)

Low (0–2%)	Moderate (2–7%)	High (7–25%)	Very High (>25%)

Indispensable in PAD: helps predict limb outcomes and revascularization benefit.

WHAT ABOUT OTHER SYSTEMS?

Wagner Classification
Simple depth-based system (0–5).
Historically important and still widely recognized.

Useful in many settings, but limited by lack of assessment of ischemia, infection, and area.

University of Texas (UT) Classification
Depth + infection + ischemia (stages 1–4).
Historically important and still widely recognized.

Still used in clinical practice, but does not capture all key domains used today.

HOW TO USE CLASSIFICATION WISELY

- Start with mechanism (Why did it occur?)
- Describe the wound systematically (*What do I see?*)
- Assess infection and perfusion
- Choose the classification that answers your clinical question
- Reassess repeatedly and adapt management
- Document consistently to track progress

KEY PRINCIPLES

- Classifications complement each other—not compete.
- The goal is better decisions and better outcomes for our patients.
- Use the result—don't just collect a score.

DO NOT CLASSIFY FOR THE SAKE OF CLASSIFICATION.

Classify when it clarifies diagnosis, guides treatment, improves communication, or predicts prognosis.

Khorshid Hospital DFU Classification — mechanism-, feature-, and behavior-based clinical framework (Siavash et al.).

SLIDE 10A

INFECTION IS A CLINICAL DIAGNOSIS

Treat infection—not colonization or a positive culture.

The right diagnosis leads to the right action.

KEY MESSAGE

Every open DFU is colonized.
A positive swab or culture
DOES NOT establish infection.

COLONIZATION ≠ **INFECTION**

- ✓ Microorganisms present without host response
- ✓ No signs or symptoms of inflammation
- ✓ Microorganisms present WITH host response
- ✓ Local or systemic inflammatory signs

Do not treat colonization. Treat infection.

DIAGNOSIS BASED ON CLINICAL SIGNS AND SYMPTOMS

Local signs of infection

- Erythema
- Warmth
- Swelling / induration
- Tenderness or pain
- Purulent discharge
- Wound bed deterioration
- Malodor

Systemic signs of infection

- Fever or chills
- Malaise
- Tachycardia
- Hypotension
- Leukocytosis / leukopenia
- Elevated inflammatory markers (CRP, ESR, PCT)*

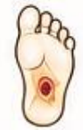
* In equivocal cases, inflammatory markers (CRP, ESR, PCT) can support the diagnosis, but they **DO NOT** replace clinical judgment.

SPECTRUM OF DIABETIC FOOT STATUS

1 UNINFECTED

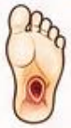
No clinical signs or symptoms of infection

2 MILD



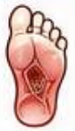
Local infection involving only skin and subcutaneous tissue

3 MODERATE



Local infection with erythema >2 cm, or deeper structures, or abscess, or systemic risk factors

4 SEVERE



Systemic toxicity or life-threatening infection (e.g., sepsis, necrotizing fasciitis)

Clinical severity guides the need for hospitalization, urgency of source control, and antimicrobial therapy.

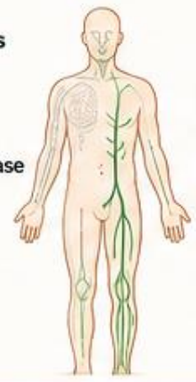
IMPORTANT PRINCIPLES

- A positive superficial swab reflects colonization. It does NOT prove infection.
- A clinically uninfected DFU should NOT receive antibiotics merely to promote healing or prevent infection.
- Use antibiotics ONLY when there is clinical evidence of infection.
- Early, accurate diagnosis improves outcomes and helps prevent unnecessary antibiotic use and resistance.

THINK BROAD: DIFFERENTIAL DIAGNOSIS

Conditions that may BLUNT inflammatory signs

- Neuropathy
- Peripheral arterial disease
- Chronic edema / venous disease
- Older age
- Immunosuppression
- CKD / ESRD
- Severe diabetes
- Malnutrition



Conditions that may MIMIC infection

- Charcot neuroarthropathy
- Gout / pseudogout
- Acute limb ischemia
- Deep vein thrombosis
- Hematoma / trauma
- Contact dermatitis
- Inflammatory disorders
- Post-surgical inflammation

When in doubt, reassess frequently and follow the clinical course.

ASSESSMENT CHECKLIST

- ☑ Careful history (onset, pain, systemic symptoms, previous antibiotics, comorbidities)
- ☑ Complete foot examination (inspection, palpation, vascular and neurologic assessment)
- ☑ Look for local AND systemic signs
- ☑ Consider factors that blunt or mimic infection
- ☑ Classify severity: Uninfected / Mild / Moderate / Severe
- ☑ Decide setting of care and need for urgent source control

A CLINICALLY UNINFECTED DFU SHOULD NOT RECEIVE ANTIBIOTICS MERELY TO PROMOTE HEALING OR PREVENT INFECTION.
(Best Practice Statement – IWGDF/IDSA)

DIAGNOSE CLINICALLY • CLASSIFY SEVERITY • RECOGNIZE THE NEED FOR SOURCE CONTROL • TREAT APPROPRIATELY

Successful DFI treatment is infection control—not simply antibiotic administration.

DFI: diabetic foot infection; DFU: diabetic foot ulcer; CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; PCT: procalcitonin.
Source: IWGDF/IDSA Guidelines on the Diagnosis and Treatment of Diabetes-related Foot Infections, 2023.

SLIDE 10B

THE FIRST DECISION IS NOT "WHICH ANTIBIOTIC?"

It Is: "How Sick Is the Patient—And Does This Foot Need Urgent Source Control?"

Antibiotics alone cannot drain an abscess, remove necrotic tissue, relieve pressure, or restore blood flow.

INITIAL TRIAGE

DFI DETECTED

- Assess clinical severity
- Assess patient stability and comorbidities
- Look for need of urgent source control or revascularization

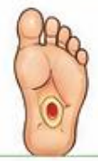
DECIDE SETTING AND URGENCY

- Outpatient care
- Hospitalize
- Urgent action

CLASSIFY INFECTION SEVERITY (IWGDF/IDSA)

1 MILD

Local infection involving only skin and subcutaneous tissue. Erythema ≤2 cm around ulcer; no systemic signs.

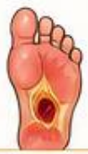


Usually managed as OUTPATIENT

Reassess in 24–48 h

2 MODERATE

Local infection with erythema >2 cm, or deeper structures/organs (e.g., abscess, osteomyelitis, septic arthritis) or systemic risk factors.*



Assess need for HOSPITALIZATION and/source control

- Important comorbidities (e.g., PAD, CHF, CKD)
- Extensive infection, deep abscess, osteomyelitis
- Inability to manage as outpatient
- Poor social support or adherence concernts

3 SEVERE

Any infection with systemic toxicity or metabolic instability or life-threatening infection (e.g., sepsis, severe infection with organ dysfunction).

- SIRS or sepsis
- Hemodynamic instability
- Mental status changes

HOSPITALIZE

Urgent evaluation for **SOURCE CONTROL** and supportive care

ICU care may be required

RED FLAGS – URGENT SURGICAL CONSULTATION

Do NOT delay for imaging or antibiotics when these are present:

- Extensive gangrene

- Necrotizing soft tissue infection

- Deep abscess

- Compartment syndrome

- Severe limb ischemia

Consider **hospitalization** for ALL severe infections and for **moderate** infections with important **morbidities**, especially PAD.
Osteomyelitis alone does NOT automatically require hospitalization if the patient is clinically stable.

DECIDE: SOURCE CONTROL AND REVASCULARIZATION

Source Control

Remove the source of infection.

- Debridement of necrotic tissue
- Incision & drainage
- Remove foreign bodies
- Relieve pressure

Revascularization

Restore blood flow when needed.

- PAD assessment
- Endovascular intervention
- Surgical bypass

DFI + PAD or gangrene → Urgent coordination between surgical and vascular teams.
Source control (drainage/debridement) and **revascularization** may need staged or combined strategies.

KEY PRINCIPLES BEFORE STARTING ANTIBIOTICS

Identify and remove the source.

Assess and correct ischemia.

Offload pressure.

Optimize glycemic control and comorbidities.

Then choose appropriate antibiotics.

EARLY RECOGNITION • EARLY SOURCE CONTROL • EARLY REVASCULARIZATION • APPROPRIATE ANTIBIOTICS
= LOWER AMPUTATION RISK • BETTER OUTCOMES • LOWER MORTALITY

Multidisciplinary team approach improves outcomes:
Endocrinology • Infectious Disease
Surgery • Vascular • Podiatry • Nursing

*Systemic risk factors: PAD, poor glycemic control, immunosuppression, renal failure, heart failure, advanced age, malnutrition, previous antibiotic therapy, large or chronic ulcer.

IWGDF/IDSA Guidelines on the Diagnosis and Treatment of Diabetes-related Foot Infections, 2023.

SLIDE 10C

EMPIRIC ANTIBIOTICS: TREAT THE PATIENT'S CONTEXT, NOT JUST THE ULCER

Match Spectrum to Clinical Context — Then Narrow Based on Cultures and Response

Right drug, right spectrum, right dose, right route, right duration.

CLINICAL SETTING	LIKELY PATHOGENS TO COVER	EMPIRIC ANTIBIOTIC OPTIONS* (Examples from IWGDF/IDSA Table 4, 2023)	KEY POINTS
1. MILD DFI Uncomplicated (antibiotic-naïve)	• Gram-positive cocci (Streptococci, MSSA)	**NARROW SPECTRUM FOR GPC** • Cloxacillin (or flucloxacillin) • 1st-generation cephalosporin: Cephalexin	✓ Outpatient care in most cases ✓ Shortest effective duration ✓ No need for broad coverage
2. MILD DFI β-lactam allergy or intolerance	• Gram-positive cocci	**ALTERNATIVES (GPC COVERAGE)** • Clindamycin • Doxycycline • TMP-SMX • Levofloxacin or Moxifloxacin	✓ Choose based on: severity, local resistance, comorbidities, drug interactions
3. MILD DFI Recent antibiotics (within 90 days)	• Gram-positive cocci • Gram-negative rods	**BROADEN TO INCLUDE GNR** • Amoxicillin–clavulanate • Ampicillin–sulbactam; Alternatives: • 2nd/3rd-gen cephalosporin (e.g., Cefuroxime) • Levofloxacin or Moxifloxacin	✓ Recent antibiotic use increases risk of resistant organisms ✓ Consider local antibiogram
4. ANY SEVERITY MRSA risk factors †	• **MRSA** (Add to regimen or use as primary agent)	**ADD OR SELECT MRSA-ACTIVE AGENT** • Vancomycin • Linezolid • Daptomycin • Clindamycin or Doxycycline (if susceptible) • TMP-SMX	✓ Use if MRSA colonization/history, prior MRSA infection, or high local prevalence
5. MODERATE / SEVERE DFI Uncomplicated	• Gram-positive cocci • ± Gram-negative rods • ± Anaerobes	**BROADER COVERAGE** • Amoxicillin–clavulanate or Ampicillin–sulbactam • 2nd/3rd-gen cephalosporin: Cefuroxime, Cefotaxime, or Ceftriaxone	✓ Consider IV therapy initially ✓ Adjust for severity, allergy, renal function, local data
6. SEVERE / DEEP / NECROTIC DFI Or recent antibiotics	• Gram-positive cocci • Gram-negative rods • Anaerobes	**BROADEST COVERAGE** • Piperacillin–tazobactam (Preferred in many settings); Alternatives: • Carbapenem (e.g., Imipenem, Meropenem, or Ertapenem§) • 3rd-gen cephalosporin (Cefotaxime/Ceftriaxone) + Metronidazole or Clindamycin	✓ Hospitalize ✓ Obtain cultures & source control urgently
7. PSEUDOMONAS RISK (Do NOT cover routinely)‡	• Include *Pseudomonas aeruginosa*	**INCLUDE ANTIPSEUDOMONAL COVERAGE** • Piperacillin–tazobactam • Ceftazidime or Cefepime • Antipseudomonal carbapenem (e.g., Meropenem, Imipenem)	✓ Recent *P. aeruginosa* isolation from the affected site AND moderate/ severe DFI, especially in Asia or North Africa
8. RESISTANT GNR RISK (ESBL / CRE / MDR-GNR)	• Resistant Gram-negative organisms	**INDIVIDUALIZE BASED ON PRIOR CULTURES & LOCAL ANTIBIOGRAM** • Carbapenem (e.g., Meropenem) often required; Other options guided by culture & susceptibility	✓ Prior resistant isolate, recent healthcare exposure or severe infection increases risk

IMPORTANT PRINCIPLES

- Obtain cultures appropriately BEFORE antibiotics, when feasible.
- Achieve source control (debridement, drainage, revascularization) as needed.
- Reassess at 48–72 hours: review cultures, clinical response, labs.
- **NARROW** to the narrowest effective regimen.
- **Shorter is better:** 1–2 weeks for soft-tissue DFI; adjust based on response and severity.

Antibiotics are an adjunct to, not a substitute for, infection control.

* Not an exhaustive list. Choose based on allergy, renal function, severity, susceptibility, availability and cost.

† MRSA risk factors: prior MRSA, known colonization, high local prevalence, recent hospitalization or dialysis, IV drug use, previous antibiotics.

‡ Do not use empiric antipseudomonal therapy in temperate climates unless risk factors are present (see key points).

§ Ertapenem: no *Pseudomonas* activity.

START APPROPRIATELY → CULTURE CORRECTLY → REVIEW RESPONSE → NARROW WHEN POSSIBLE

Right decision. Right drug. Better outcomes. Less harm.

SLIDE 10D

CULTURE SMART → START SMART → NARROW SMART

Right Specimen • Right Therapy • Right Duration

IWGDF/IDSA Guidelines 2023

WHEN TO OBTAIN CULTURE
(Before antibiotics, when feasible)

- ✔ Moderate or severe DFI.
- ✔ Severe infection or systemic illness.
- ✔ Failure to respond to initial therapy.
- ✔ Previous MRSA or resistant Gram-negative.
- ✔ Hospital-acquired or recently treated infection.

THE RIGHT SPECIMEN

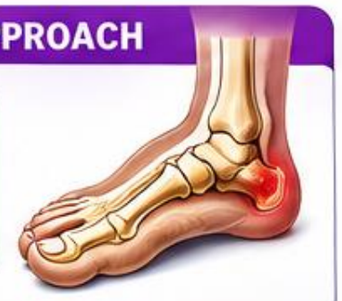
✔ **Preferred:**

Aseptically collected tissue by curettage or biopsy (rather than a superficial swab).

Tissue culture is more reliable than swab.

SUSPECTED OSTEOMYELITIS: STEPWISE APPROACH

Probe-to-bone test + Plain X-ray – ESR/CRP/PCT → If still uncertain: **MRI** → When microbiological diagnosis is needed: **Bone specimen** rather than soft-tissue culture



EMPIRIC THERAPY (Practical Options According to Clinical Context – IWGDF/IDSA 2023)

Clinical setting	Likely pathogens / coverage	Practical IWGDF empiric options
Mild, uncomplicated (community, antibiotic-naïve)	GPC (Streptococci, MSSA)	• 1st-generation cephalosporin (e.g., cephalexin) • OR antistaphylococcal penicillin
Mild + β-lactam allergy or intolerance	GPC	• Clindamycin OR doxycycline OR TMP-SMX • Levofloxacin or moxifloxacin
Mild + recent antibotics	GPC + GNR	• Amoxicillin/clavulanate OR ampicillin/sulbactam • Selected alternatives
MRSA risk	MRSA	• Add or select MRSA-active therapy according to susceptbiity and context
Moderate/severe, uncomplicated	GPC ± GNR	• Amoxicillin/clavulanate or ampicillin/sulbactam • Or 2md/3rd-gen cephalosporin (cefuroxime, cefotaxime or ceftriaxone)
Moderate/severe + recent antibiotics	Broader GPC ± GNR	• Piperacillin/tazobactam • Selected cephalosporin or ertapenem
Ischemia / necrosis / gas	GPC + GNR + anaerobes	• β-lactam/β-lactamase inhibitor or carbapenem OR cefuroxime/ceftriaxone/ cefotaxime + metronidazole or clindamycin
Pseudomonas risk	Include *P. aeruginosa*	• Piperacillin/tazobactam, ceftazidime-based regimen, or selected antipsudomonal carbapenem
Resistant GNR / ESBL risk	Resistant GNR	• Individualize from previous cultures and local antibiogram; carbapenem is among Table 4 options.

ANTIMICROBIAL STEWARDSHIP

Key Points:

- ✔ *Pseudomonas* coverage is NOT routine in temperate climates.
- ✔ Use antipseudomonal therapy only if *P. aerugioosa* was recently isolated or in moderate/severe DFI in Asia or North Africa.
- ✔ Tailor therapy to severity, setting and prior antibiotic exposure.
- ✔ De-escalate to the narrowest effective regimen.

DURATION

- Soft-tissue DFI: 1–2 weeks.
- Extend to 3–4 weeks if infection is extensive, resolving unusually slowly, or with severe PAD.
- Reassess (do not automatically prolong therapy) if no improvement.

SUCCESSFUL DFI TREATMENT IS INFECTION CONTROL — NOT SIMPLY ANTIBIOTIC ADMINISTRATION.

SLIDE 10E (1/2)

DIABETIC FOOT OSTEOMYELITIS: DIAGNOSE IT, THEN DECIDE—MEDICAL, SURGICAL, OR BOTH?

Integrated assessment → Microbiologic diagnosis → Individualized treatment → Long-term outcome

GOAL:
Eradicate infection with the least harm, preserve the foot, maximize function.

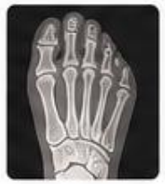
1. DIAGNOSIS: CONFIRM OSTEOMYELITIS

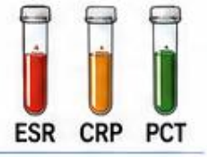
Clinical suspicion

- Probe-to-bone positive in a high-risk ulcer
- Ulcer >2 weeks, large/deep, or over bone
- Wound not improving

Initial evaluation

- Document depth, size, ischemia, and neuropathy
- Plain X-ray (all patients)
- Inflammatory markers: ESR, CRP ± PCT
- Assess PAD (ABI/toe pressure)

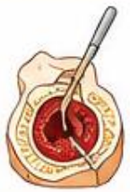




If diagnosis still uncertain

MRI (preferred) or other advanced imaging

Microbiologic diagnosis (when needed)



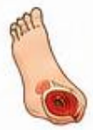
- Obtain bone specimen (biopsy or bone obtained at surgery) for culture and histopathology.

Bone culture is the reference standard for identifying the causative organism in DFO.

2. DECIDE: MEDICAL THERAPY, SURGERY, OR BOTH?

WHEN CAN ANTIBIOTIC THERAPY WITHOUT BONE RESECTION BE CONSIDERED?

ALL OF THE FOLLOWING MUST BE PRESENT:

- Forefoot osteomyelitis 
- No immediate need for incision & drainage 
- No peripheral arterial disease (PAD) 
- No exposed bone 

If any of the above are NOT met → Prefer surgical resection of infected bone (with or without soft-tissue surgery) plus systemic antibiotic therapy.

WHEN IS SURGERY INDICATED?

Need for rapid source control (abscess, extensive necrosis, sepsis)

Extensive bone destruction or instability

Persistent infection despite appropriate medical therapy

Compromised soft tissues requiring debridement/flap

Ischemia requiring revascularization first or in combination

Early and adequate debridement of all infected/necrotic bone is key to success.

PRINCIPLES WHEN SURGERY IS PERFORMED

Resect all infected and necrotic bone until healthy bleeding bone remains.

Obtain bone from the resection margin for culture ± histology.

Combine with appropriate antibiotics and optimal wound care.

SUCCESSFUL DFO TREATMENT = CONFIRMATION + SOURCE CONTROL + APPROPRIATE ANTIBIOTICS + HOST OPTIMIZATION + CLOSE FOLLOW-UP

DFO: diabetic foot osteomyelitis; PAD: peripheral arterial disease; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein; PCT: procalcitonin.

Source: IWGDF/IDSA Guidelines on the Diagnosis and Treatment of Diabetes-related Foot Infections, 2023.

SLIDE 10E (2/2)

DIABETIC FOOT OSTEOMYELITIS: DIAGNOSE IT, THEN DECIDE—MEDICAL, SURGICAL, OR BOTH?

Integrated assessment → Microbiologic diagnosis → Individualized treatment → Long-term outcome

GOAL: Eradicate infection with the least harm, preserve the foot, maximize function.

3. ANTIBIOTIC THERAPY FOR DFO

ROUTE

- Start IV if needed (e.g., severe infection), then switch to oral when clinically appropriate and effective agents are available.

DURATION (GENERAL GUIDANCE)

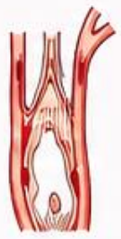
- DFO treated WITHOUT bone resection or amputation: ~6 weeks
- Positive bone-margin culture after minor amputation: up to 3 weeks

ASSESS REMISSION

- Assess remission after ≥6 months following completion of antibiotics (not merely when the wound heals).

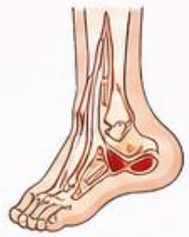
4. SPECIAL SITUATIONS

SEVERE ISCHEMIA (PAD)



- Coordinate with vascular team.
- Revascularization and infection control may need careful sequencing.

CHARCOT FOOT

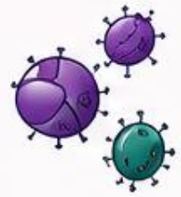


- Inflammation and osteomyelitis can coexist.
- Use imaging judiciously and rely on the entire clinical picture.

RECURRENT DFO

- Search for and correct all predisposing factors (pressure, ischemia, footwear, biofilm, infection).

COMPLEX POLYMICROBIAL INFECTIONS



- Use culture results and clinical response to guide and narrow therapy.

Multidisciplinary Foot-Care Team Approach:
Endocrinologist • Infectious Disease • Podiatrist • Vascular Surgeon • Orthopedic Surgeon • Radiologist • Wound-Care Specialist

5. KEY TAKE-HOME MESSAGES

- Osteomyelitis is a serious but treatable complication of DFI.
- Confirm the diagnosis before committing to therapy.
- Choose the right strategy: medical, surgical, or both.
- Adequate source control + appropriate antibiotics + offloading ± revascularization (if needed).
- Long-term follow-up is essential to confirm remission and prevent recurrence.

DFO MANAGEMENT ALGORITHM (AT A GLANCE)

1. **SUSPECT DFO** — Probe-to-bone (+), chronic ulcer, not healing
2. **INITIAL WORK-UP** — X-ray; ESR, CRP (± PCT); PAD assessment
3. **UNCERTAIN?** — MRI preferred (or other advanced imaging)
4. **CONFIRM NEED FOR MICROBIOLOGY** — Obtain bone specimen for culture ± histology
5. **DECIDE STRATEGY** — Medical only vs. Surgery vs. Surgery + Antibiotics
6. **IMPLEMENT TREATMENT** — Source control + Appropriate antibiotics + Offloading
7. **MONITOR & REASSESS** — Clinical signs, labs, imaging as needed
8. **CONFIRM REMISSION** — ≥6 months after stopping antibiotics

SUCCESSFUL DFO TREATMENT = CONFIRMATION + SOURCE CONTROL + APPROPRIATE ANTIBIOTICS + HOST OPTIMIZATION + CLOSE FOLLOW-UP

DFO: diabetic foot osteomyelitis; PAD: peripheral arterial disease; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein; PCT: procalcitonin.

Source: IWGDF/IDSA Guidelines on the Diagnosis and Treatment of Diabetes-related Foot Infections, 2023.

SLIDE 11A

PERFUSION ASSESSMENT – PART A

Does This Foot Have Peripheral Arterial Disease (PAD)?

We use a stepwise approach: ❶ Clinical clues → ❷ Pulses → ❸ Pedal Doppler → ❹ ABI & TBI

GOAL:
Identify ischemia early by combining clinical assessment with simple, non-invasive tests.

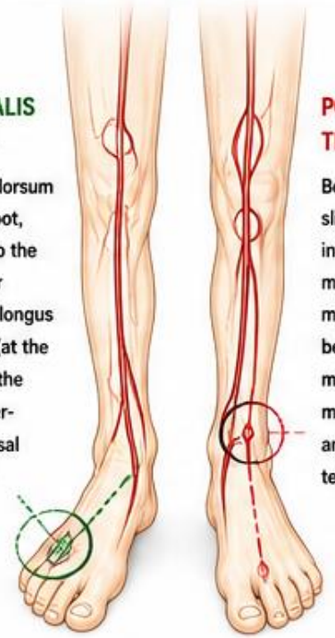
❶ CLINICAL ASSESSMENT
Look for signs of ischemia

- Rest pain (especially at night)
- Nonpalpable or weak pedal pulses
- Skin changes: thin, shiny, hairless
- Delayed capillary refill (> 3 seconds)
- Dependent rubor, cool skin
- Slow or nonhealing ulcer

ⓘ These findings increase suspicion, but do not confirm PAD.

❷ PALPATE PULSES (BILATERAL)

DORSALIS PEDIS
On the dorsum of the foot, lateral to the extensor hallucis longus tendon (at the level of the first inter-metatarsal space).



POSTERIOR TIBIAL
Behind and slightly inferior to the medial malleolus, between the medial malleolus and Achilles tendon.

✋ If pedal pulses are absent or diminished → proceed to Doppler assessment.

❸ PEDAL DOPPLER WAVEFORMS
Assess dorsalis pedis & posterior tibial arteries

TRIPHASIC (NORMAL)
Sharp systolic upstroke, brief reverse flow, late forward component. ➡ PAD less likely (normal high-resistance flow)

BIPHASIC (REDUCED COMPLEXITY)
Forward systolic with reverse component; loss of one normal phase. May still be compatible with adequate perfusion. PAD less likely. Interpret with ABI, TBI and clinical context.

MONOPHASIC (ABNORMAL)
Single forward component; often broad and damped. Abnormal waveform. Suggests PAD. Further vascular assessment indicated.

ABSENT SIGNAL (ABNORMAL)
No detectable arterial signal. Strongly abnormal. Consider significant arterial disease / ischemia.

⚠ **Never interpret the waveform alone.**
Always combine pedal Doppler findings with ABI + TBI/toe pressure and the clinical picture.

❹ ABI AND TBI
(Simple Hemodynamic Tests)

ABI (Ankle–Brachial Index)

ABI	INTERPRETATION
1.00 – 1.40	Normal
0.91 – 0.99	Borderline
≤ 0.90	Abnormal (PAD likely)
> 1.40	Noncompressible (arterial calcification common in diabetes)

TBI (Toe–Brachial Index)

TBI	INTERPRETATION
≥ 0.70	Normal
0.40 – 0.69	Borderline
< 0.40	Abnormal (PAD likely)

★ Use ABI and TBI together.
ABI may be falsely normal/high in arterial calcification. TBI is more reliable in diabetes.

KEY TAKE-HOME MESSAGE

PAD is **less likely** when:
- ✓ Pedal Doppler is triphasic or biphasic AND
- ✓ ABI 0.9 – 1.3 AND TBI ≥ 0.70

If results are abnormal or the patient is symptomatic (rest pain, tissue loss, nonhealing ulcer, etc.), proceed to perfusion adequacy (healing potential) assessment and consider vascular referral.

Source: IWGDF/ESVS/SVS Guideline on the Management of Peripheral Arterial Disease in Patients with Diabetes, 2023.

SLIDE 11B

PERFUSION ASSESSMENT – PART B
Does This Foot Have Enough Blood Flow to Heal?

After PAD is assessed (Slide 11A), estimate the potential for wound healing.

GOAL:
Identify patients with adequate perfusion to heal vs. those at high risk of poor healing or limb loss.

1. KEY NON-INVASIVE TESTS THAT PREDICT HEALING POTENTIAL

TOE PRESSURE (Great Toe)

Measured with small digital cuff and PPG/Doppler

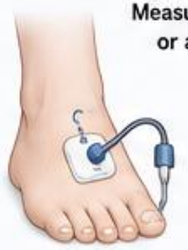
≥ 30 mmHg
Increases probability of healing

20 – 29 mmHg	Uncertain / Borderline
< 20 mmHg	Low probability of healing

Toe pressure ≥ 30 mmHg is associated with a higher likelihood of healing.

TRANSCUTANEOUS OXYGEN PRESSURE ($TcPO_2$)

Measure at dorsum of foot or at the wound site



≥ 25 mmHg
Increases probability of healing

15 – 24 mmHg	Uncertain / Borderline
< 15 mmHg	Low probability of healing

Reflects tissue oxygenation and microvascular perfusion.

SKIN PERFUSION PRESSURE (SPP)

Measured at dorsum of foot or toe

≥ 40 mmHg
Increases probability of healing

30 – 39 mmHg	Uncertain / Borderline
< 30 mmHg	Low probability of healing

Reflects nutritive capillary perfusion at the skin level.

2. INTERPRETATION: HEALING POTENTIAL

ADEQUATE PERFUSION
(Higher probability of healing)

- Toe pressure ≥ 30 mmHg
- $TcPO_2$ ≥ 25 mmHg
- SPP ≥ 40 mmHg

→ Optimize wound care and monitor closely

BORDERLINE / UNCERTAIN
(Intermediate probability of healing)

- Toe pressure 20–29 mmHg
- $TcPO_2$ 15–24 mmHg
- SPP 30–39 mmHg

→ Optimize care and reassess perfusion

POOR PERFUSION
(Low probability of healing)

- Toe pressure < 20 mmHg
- $TcPO_2$ < 15 mmHg
- SPP < 30 mmHg

→ High risk of nonhealing and limb loss – urgent vascular evaluation

Interpret results in the context of clinical assessment, Doppler waveforms, ABI and TBI.

3. COMPLEMENTARY TESTS (When available or when results are uncertain)

CT / MR ANGIOGRAPHY
Defines arterial anatomy and lesion characteristics.

DUPLEX ULTRASOUND
Assesses stenosis, occlusion and blood flow distally.

DIGITAL SUBTRACTION ANGIOGRAPHY (DSA)
Gold standard for planning endovascular or surgical intervention.

BLOB FLOW VELOCITY (Duplex)
Low flow velocities suggest significant arterial disease.

IMPORTANT REMINDER

- No single test is perfect.
- Always combine perfusion tests with the clinical picture, wound severity, infection and patient factors.
- Repeat testing if the clinical situation changes.

KEY TAKE-HOME MESSAGES

- Adequate perfusion is essential for wound healing.
- Toe pressure ≥ 30 mmHg, $TcPO_2$ ≥ 25 mmHg and SPP ≥ 40 mmHg are associated with higher healing probability.
- Values below these thresholds indicate increased risk of poor healing and limb loss – act early.
- Reassess and refer when perfusion is uncertain or poor.

PERFUSION ASSESSMENT HIERARCHY (Healing Potential)

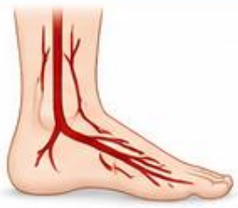
Clinical assessment → Key tests (Toe pressure, $TcPO_2$, SPP) → Interpret in context (ABI, TBI, Doppler, clinical picture) → Decide & act (Optimize care or refer for revascularization)

ABBREVIATIONS

ABI:	Ankle–Brachial Index
TBI:	Toe–Brachial Index
$TcPO_2$:	Transcutaneous Oxygen Pressure
SPP:	Skin Perfusion Pressure
PPG:	Photoplethysmography

GOOD PERFUSION HEALS WOUNDS. POOR PERFUSION DRIVES AMPUTATION. ASSESS – INTERPRET – ACT – SAVE LIMBS.

Source: IWGDF/ESVS/SVS Guidelines on the Management of Peripheral Arterial Disease in Patients with Diabetes, 2023.



TOE–BRACHIAL INDEX (TBI) MEASUREMENT TECHNIQUE

Simple • Accurate • Reproducible • Clinically Valuable

PRINCIPLE: TBI = Toe systolic pressure ÷ Higher brachial systolic pressure

WHY TBI?

Toe arteries are less likely to calcify. TBI is reliable when ABI is normal, borderline or noncompressible.

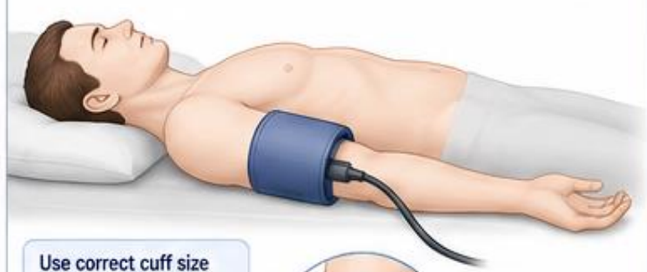
BEFORE MEASUREMENT

- Patient supine and resting for ≥ 5 minutes
- Warm, quiet room (22–25 °C / 72–77 °F)
- Feet and toes warm and uncovered
- No smoking, caffeine, or exercise for ≥ 2 hours
- Avoid measurement during acute pain, vasoconstrictive drugs or immediately after vascular procedures

Explain the procedure and ensure comfort.

① BRACHIAL PRESSURE MEASUREMENT

Measure systolic blood pressure in **BOTH** arms.
Use the **HIGHER** systolic value for TBI calculation.



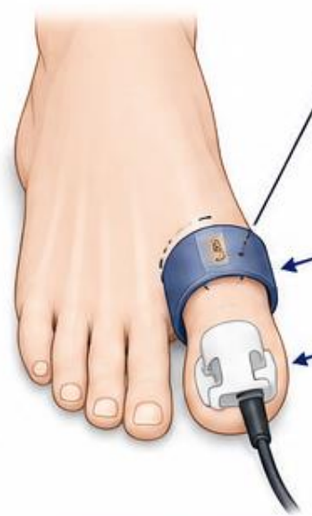
Use correct cuff size (bladder width ≈ 40% of arm circumference)

Place cuff on upper arm (brachial artery) and locate pulse with Doppler probe.

Record the higher brachial systolic pressure (mmHg) as the denominator for TBI.

② TOE PRESSURE MEASUREMENT (Great Toe)

Use a small digital cuff and a photoplethysmography (PPG) probe to measure great-toe systolic pressure.



Digital cuff placed at the **PROXIMAL** base of the great toe (just distal to the metatarsophalangeal joint).

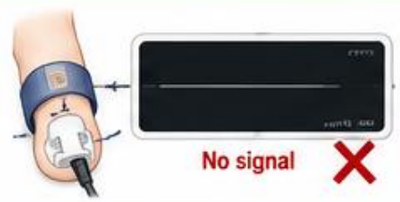
PPG sensor on the toe pulp, **DISTAL** to the cuff.

Ensure a good PPG signal before inflation.

③ MEASUREMENT SEQUENCE

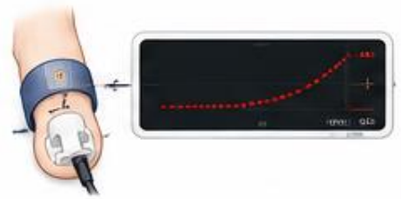
1 INFLATE TO OCCLUSION

Inflate the toe cuff until the PPG waveform disappears (arterial flow occluded).



2 SLOW DEFLATION

Deflate the cuff slowly (2–3 mmHg per second) while watching the PPG waveform.



3 PPG WAVEFORM RETURNS

The pressure at which the PPG waveform returns is the **TOE SYSTOLIC PRESSURE.**

TBI FORMULA & INTERPRETATION

$$TBI = \frac{\text{Toe systolic pressure}}{\text{Higher brachial systolic pressure}}$$

TBI VALUE	INTERPRETATION	CLINICAL SIGNIFICANCE
≥ 0.70	Normal	PAD less likely
0.40 – 0.69	Borderline	Possible PAD – correlate with clinical picture
< 0.40	Abnormal	PAD likely

TBI is more reliable than ABI in diabetes and when arteries are calcified.

KEY POINTS FOR ACCURATE TBI

- Use the correct cuff size for the arm and the toe.
- Toe must be warm; cold toes give falsely low values.
- Good PPG signal quality is essential.
- Allow adequate rest before measurement.
- Repeat measurement if signal is poor or inconsistent.

QUALITY CHECK & DOCUMENTATION

Record and report:

- Toe systolic pressure (mmHg)
- Higher brachial systolic pressure (mmHg)
- Calculated TBI value
- Limb (right/left) and toe (great toe)
- Patient position and conditions

NEVER INTERPRET TBI ALONE.
Always combine TBI with clinical assessment, pedal Doppler waveform and other tests.

ABNORMAL OR SYMPTOMATIC PATIENT?
Proceed to perfusion adequacy (healing potential) assessment and consider referral to the vascular team.

GOOD PERFUSION HEALS WOUNDS.
Timely recognition and management save limbs and improve lives.

Source: IWGDF/ESVS/SVS Guidelines on the Management of Peripheral Arterial Disease in Patients with Diabetes, 2023.

SLIDE 12

THE SIX PILLARS OF DFU HEALING

A Comprehensive, Patient-Centered Approach

1 OFFLOAD

Remove mechanical stress and redistribute pressure.

- Relieve pressure from the ulcer
- Use guideline-recommended offloading devices
- Promote mobility safely

2 RESTORE PERFUSION

Provide adequate blood flow for tissue healing.

- Assess for ischemia early
- Optimize medical therapy
- Revascularize when indicated

3 CONTROL INFECTION

Treat infection promptly and achieve source control.

- Diagnose clinically
- Determine severity
- Culture appropriately
- Antibiotics + source control when needed
- Do not treat colonization

4 DEBRIDE & OPTIMIZE WOUND BED

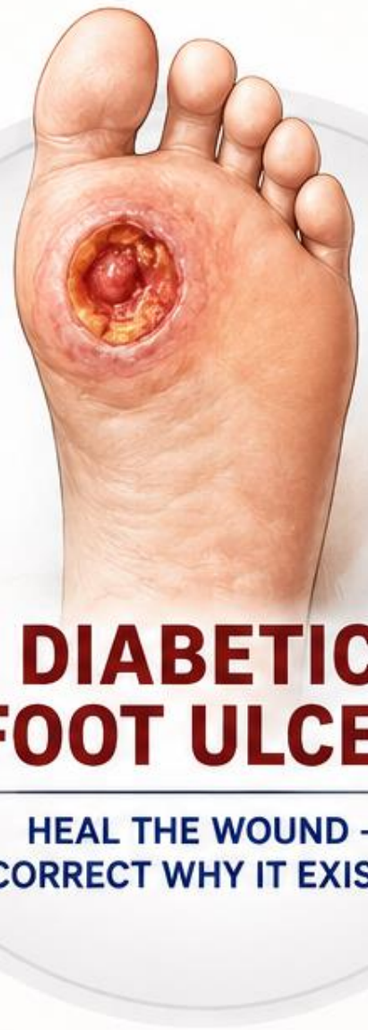
Create and maintain a wound environment conducive to healing.

- Remove devitalized tissue / callus
- Manage exudate and bioburden
- Choose appropriate dressings
- Reassess and adapt

5 OPTIMIZE THE HOST

Correct systemic and local factors that impair healing.

- Glycemic control
- Nutrition
- Treat edema
- Stop smoking
- Manage comorbidities
- Address anemia, medications, etc.
- Psychosocial & functional optimization



DIABETIC FOOT ULCER

HEAL THE WOUND – CORRECT WHY IT EXISTS

ASSESS → TREAT → REASSESS → PROTECT

6 PREVENT RECURRENCE

Healing is not the end— protect the foot for life.

- Appropriate footwear / offloading
- Regular surveillance
- Callus management
- Patient education & self-inspection
- Address the original cause

ALL SIX PILLARS MATTER – FAILURE OF ONE MAY PREVENT HEALING.

OFFLOAD the cause • PERFUSE the tissue • CONTROL infection • PREPARE the wound • OPTIMIZE the person • PROTECT the healed foot

A multidisciplinary team and an engaged patient are essential.

Multidisciplinary Care for Diabetic Foot Ulcer

A TEAM AROUND THE PATIENT

Together for a Healthier Foot and a Brighter Future

Many Skills
One Goal
Healthy Steps

Isfahan Endocrine and Metabolism Research Center
Isfahan University of Medical Sciences

Endocrinologist
- Optimize glycemic control
- Manage comorbidities

Vascular Specialist
- Assess and restore perfusion
- Endovascular / surgical options

Infectious Diseases Specialist
- Diagnose and treat infection
- Guide antibiotic therapy

Surgeon
- Surgical debridement
- Manage complicated cases
- Limb salvage

Podiatrist
- Foot assessment and care
- Callus and nail management
- Preventive foot care

Orthotist / Pedorthist
- Offloading devices
- Custom footwear / insoles
- Pressure redistribution

Physiotherapist
- Mobility and balance training
- Gait assessment
- Fall prevention

Wound Care Nurse
- Wound assessment and dressing
- Debridement support
- Patient education and follow-up

Dietitian / Nutritionist
- Medical nutrition therapy
- Weight management
- Support wound healing

Diabetes Educator
- Self-care skills
- Foot care education
- Empowerment for lifelong care

Social Worker / Case Manager
- Psychosocial support
- Access to services and supplies
- Coordinate care

Family / Caregivers
- Daily foot inspection
- Support adherence
- Be part of the solution

Different Expertise. A Shared Purpose.

Prevent Ulcers. Heal Wounds. Save Limbs. Improve Lives.

Patient-Centered Care

Communication — Early Action

Teamwork — Better Outcomes

Limb Preservation — *Higher Quality of Life*

Healthy People
Stronger Communities

SLIDE 12A

OFFLOAD: Treat the Pressure, Not Just the Wound

Offloading is the MOST important intervention for neuropathic plantar ulcers.

1 OFFLOAD

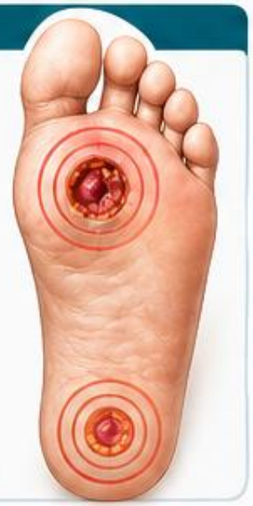
Remove the cause of pressure so the wound can heal.

GOAL: Eliminate or redistribute pressure on the ulcer to allow healing and prevent recurrence.

KEY MESSAGE
You can have the best dressing, but without offloading, the wound will not heal.

WHY OFFLOAD?

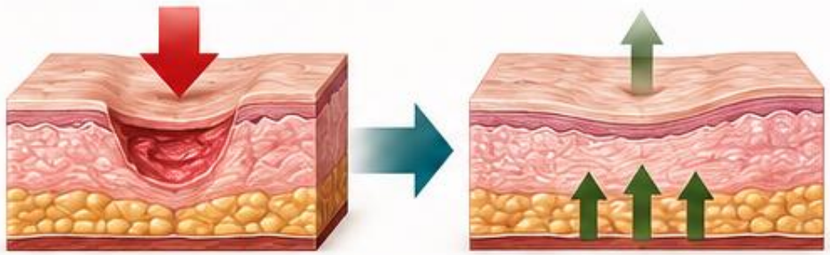
- Neuropathy → loss of pain → repetitive pressure and shear cause tissue breakdown.
- Plantar pressure at the ulcer site must be reduced to near zero to achieve healing.
- Consistent use is essential—"part-time" offloading does not work.



HOW PRESSURE CAUSES ULCERS

HIGH PRESSURE
Tissue breakdown

REDUCED PRESSURE
Tissue recovery



Pressure > Tissue tolerance
= Ulcer develops

Pressure ≤ Tissue tolerance
= Healing occurs

PRINCIPLES TO REMEMBER

- Target the wound, not just the foot.
- The more you offload, the better the outcome.
- Removable devices work only when worn.
- Total contact devices are the gold standard for plantar neuropathic ulcers.
- Reassess pressure, fit and adherence at every visit.

OFFLOADING OPTIONS (FOR PLANTAR NEUROPATHIC ULCERS)

TOTAL CONTACT CAST (TCC)

Gold standard
Most effective
Healing rates up to 90%*

NON-REMOVABLE WALKER (IRW)

Very effective
Alternative to TCC
when casting not possible

REMOVABLE WALKER (RW)

Effective only if worn
> 90% of the time
Educate and monitor

HEEL OFFLOADING DEVICES

Useful for heel ulcers
Relieve pressure
on the heel

OFFLOADING FOOTWEAR (Healing sandals / shoes)

For less severe ulcers
or after initial healing
Proper fit is essential

FELTED FOAM (Adjunct)

Use with appropriate
footwear/devices
Not a stand-alone solution

*Based on consistent use in appropriate patients with neuropathic plantar ulcers.

WHEN NOT TO USE TCC / IRW

- Active infection that cannot be managed under the device
- Critical limb ischemia not yet revascularized
- Severe edema / heart failure
- Severe balance issues / high fall risk
- Allergy/intolerance to cast materials
- Non-ambulatory patients

KEY ACTIONS

- ☐ Assess plantar pressure and gait.
- ☐ Select the most effective offloading device for this patient.
- ☐ Ensure correct application and fit.
- ☐ Reinforce adherence at every visit.
- ☐ Downgrade to footwear only after healing is achieved.

OFFLOAD THE PRESSURE TODAY → HEAL THE WOUND TOMORROW → PREVENT RECURRENCE FOR LIFE

No pressure, no ulcer. No offloading, no healing.

Consistent offloading is the single most important factor you can control.

SLIDE 12B

RESTORE PERFUSION: Blood Flow is Treatment

Adequate perfusion is essential for wound healing and limb salvage.

GOAL: Identify and treat clinically significant ischemia to deliver oxygen, nutrients and immune cells to the wound.

KEY MESSAGE

No blood flow = no healing.
Assess early, intervene promptly, and follow the patient.

1. WHY PERFUSION MATTERS

- Delivers oxygen and nutrients
- Supports immune cell function
- Removes waste products
- Enables repair, granulation and tissue remodeling

Even the best wound care fails without adequate perfusion.

2. UNDERSTAND THE PROBLEM

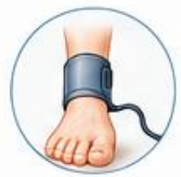
Atherosclerotic PAD reduces blood flow.

RESULT: ↓ Tissue perfusion → ↓ Oxygen delivery → Impaired healing → Infection & ulcer progression → Amputation risk

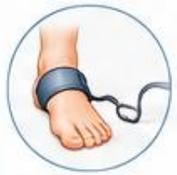
3. HOW TO ASSESS PERFUSION

CLINICAL

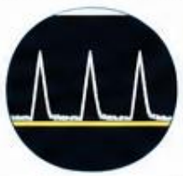
- Absent/weak pulses
- Cool skin, pale/cyanotic
- Delayed capillary refill
- Dependent rubor
- Rest pain, non-healing ulcer
- Gangrene



ABI
Ankle–brachial index
< 0.90
suggests PAD



TOE PRESSURE (TP)
More reliable in diabetes
< 30 mmHg
suggests poor healing potential



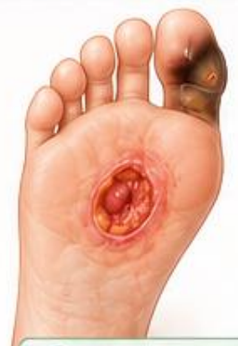
DOPPLER WAVEFORM
Monophasic, dampened or absent signals suggest ischemia

ADVANCED IMAGING
Duplex ultrasound (first-line)
CTA / MRA
Conventional angiography (gold standard) when revascularization is planned

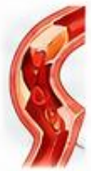
4. WHEN TO CONSIDER VASCULAR INTERVENTION

Consider revascularization when:

- Sustained non-healing ulcer > 4–6 weeks despite optimal care
- Ischemic rest pain
- Gangrene
- Severe perfusion abnormality (e.g., TP < 30 mmHg or ABI < 0.5)
- Multilevel disease with poor distal perfusion



Revascularization options



ENDOVASCULAR

- Angioplasty
- Stent placement
- Atherectomy
- Drug-coated devices

SURGICAL

- Bypass surgery
- Endarterectomy

Choose the best strategy for the patient, limb and anatomy.

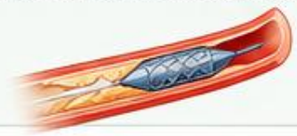
5. TARGETS FOR HEALING

Toe pressure (TP)	≥ 30 mmHg
Skin perfusion pressure (SPP)	≥ 40 mmHg
Transcutaneous oxygen ($TcPO_2$)	≥ 25 mmHg

These values are associated with improved wound healing potential.

AFTER REVASCULARIZATION:

- Confirm improved perfusion
- Optimize medical therapy
- Continue wound care
- Monitor regularly
- Address risk factors aggressively



TAKE-HOME MESSAGE

- Assess perfusion early in every DFU.
- Do not delay referral for vascular evaluation.
- Restoring blood flow saves limbs and lives.
- Perfusion + Offloading + Infection control + Wound bed preparation + Host optimization = HEALING.

THE PERFUSION PATHWAY

SUSPECT Ischemia → ASSESS Perfusion → DECIDE Revascularize? → INTERVENE Restore flow → CONFIRM Adequate perfusion → HEAL & PROTECT

WARNING SIGNS = URGENT ACTION

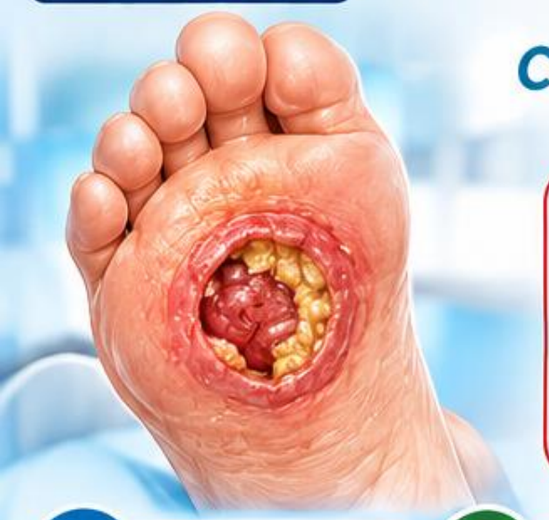
- Rest pain
- Rapidly progressive ulcer
- Gangrene
- Severe infection with ischemia

SLIDE 12C

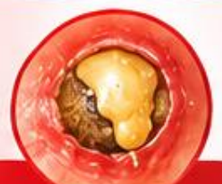
CONTROL INFECTION

Control the Source – Treat the Pathogens – Protect the Limb

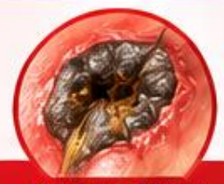
Infection control is a coordinated strategy, not just antibiotics.



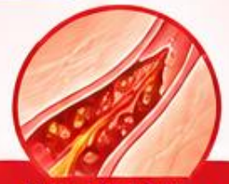
URGENT INDICATIONS FOR SOURCE CONTROL



DEEP ABSCESS



NECROSIS / GANGRENE



INFECTION + ISCHEMIA

URGENT SOURCE CONTROL
Surgical / Vascular Assessment

1 DIAGNOSE

- Clinical signs of infection
- Do not treat colonization or positive superficial culture

2 GRADE

Use IWGDF/IDSA classification

- MILD
- MODERATE
- SEVERE

Determines urgency and site of care (outpatient vs. hospitalization).

3 SOURCE CONTROL

- Drain abscess
- Remove necrotic tissue
- Address deep infection
- Revascularize if needed
- Consider bone resection for osteomyelitis

4 OBTAIN CULTURES

- Deep tissue / bone culture (not superficial swab)
- Guides targeted therapy

5 TREAT

- Dmpiric therapy based on severity and risk factors
- Cover likely pathogens
- Consider local antibiogram

6 NARROW

- Adjust to culture results
- Use the narrowest effective spectrum
- Shortest appropriate duration

7 REASSESS

- Monitor clinical response
- If not improving, look for:
  - missed abscess
  - osteomyelitis
  - ischemia
  - inadequate debridement
  - resistant organism
  - poor adherence / offloading

ANTIMICROBIAL STEWARDSHIP
Right patient • Right specimen • Right spectrum • Right duration

SUCCESSFUL DFI TREATMENT =

SOURCE CONTROL +

APPROPRIATE ANTIBIOTICS +

PERFUSION +

OFFLOADING +

HOST OPTIMIZATION

Antibiotics treat bacteria. They do not drain an abscess, remove necrotic tissue, or restore blood flow.

SLIDE 12D

DEBRIDE & OPTIMIZE THE WOUND BED

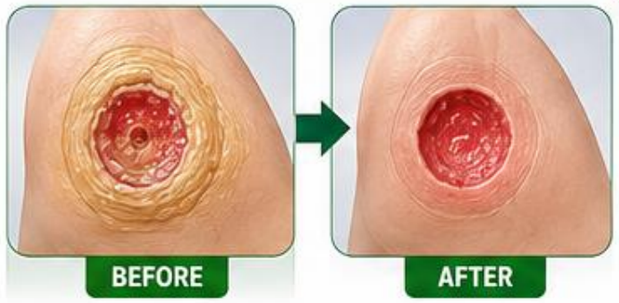
Prepare the Wound to Heal

GOAL: Create an environment in which the wound can heal.

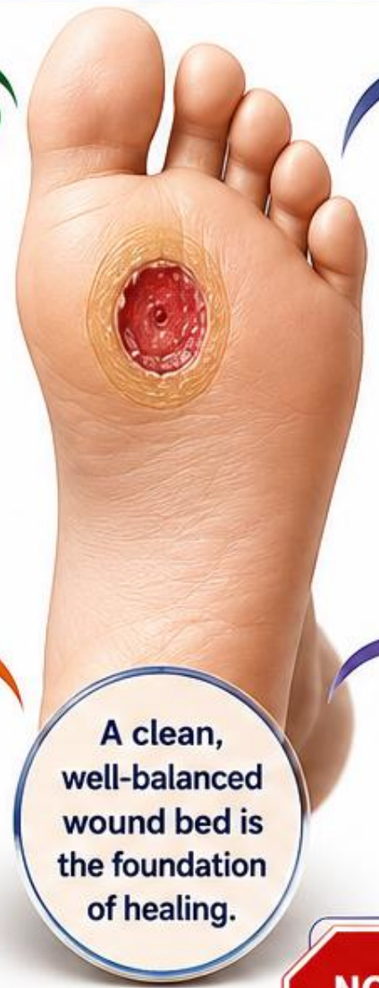
1 DEBRIDE

Remove barriers to healing

- Remove callus and nonviable tissue when clinically appropriate.
- Sharp debridement is the standard method when appropriate.
- Reassess the need and frequency regularly.



⚠ Ischemic dry stable eschar/gangrene without infection → assess perfusion before aggressive debridement.



2 CLEAN

Create an appropriate wound enviorment

- Cleanse appropriately at dressing changes.
- Remove loose debris and excess exudate.
- The goal is wound-bed preparation—not sterilization.

Colonization ≠ infection.

3 BALANCE MOISTURE & EXUDATE

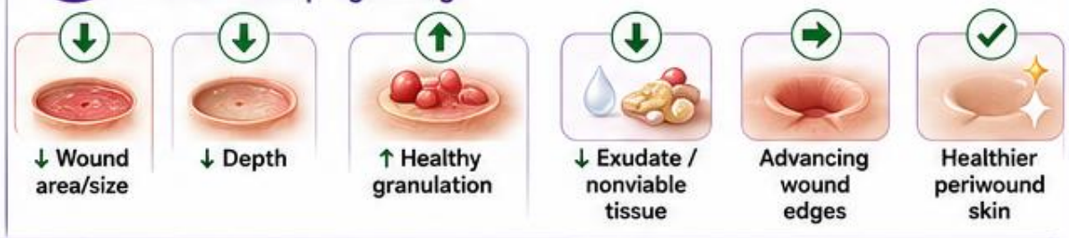
Choose the dressing for what the wound needs

Protect the periwound skin

The dressing supports healing—
it does not correct ischemia, pressure, or uncontrolled infection.

4 REASSESS THE TRAJECTORY

Is the wound progressing?



NOT HEALING?

REASSESS THE BARRIERS

WHEN A DFU IS NOT HEALING,
DON'T JUST CHANGE THE DRESSING — FIND THE BARRIER.

Evidence Based:
IWGDF 2023 Wound Healing Interventions and Practical Guidelines

Fundamentals first — advanced therapies come later when standard care has been optimized.

★ Active treatment, not passive dressing. | Multiple factors ≥ one cause. | ✓ Optimize the wound bed. Treat the cause. Support healing.

SLIDE 12D-MDT

MAGGOT DEBRIDEMENT THERAPY (MDT)

Biological Debridement: Interesting Tool, Selective Role

GOAL
Selective removal of devitalized tissue to prepare the wound bed and support healing.

MDT uses sterile fly larvae that selectively debride necrotic tissue, help reduce bioburden and biofilm, and create a healthier wound bed.

HOW IT WORKS

1. SELECTIVE DEBRIDEMENT
Larvae secrete enzymes that liquefy devitalized tissue, which is then ingested.

2. LIQUEFACTION & REMOVAL
Necrotic material is liquefied and removed, leaving viable tissue largely unharmed.

3. ANTIMICROBIAL EFFECTS
Secretions and mechanical activity may reduce bacterial burden and help disrupt biofilm.

HOW IT IS APPLIED

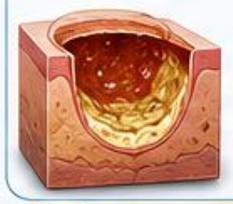
- Larvae are placed on the wound and covered with a permeable dressing or a biobag.

KEY POINTS

- Living organisms – not a drug
- Use sterile, quality-controlled larvae
- Single-use; remove larvae before they reach pupation
- Provides a moist environment inside the dressing
- Typically left in place for 24–72 hours, then replaced as needed

MDT IS AN ADJUNCT – NOT A REPLACEMENT FOR STANDARD CARE

BEFORE



- Necrotic tissue / slough
- High bioburden biofilm

AFTER

- Cleaner wound bed
- Less slough / bioburden
- Ready for healing

POTENTIAL ROLE – WHEN TO CONSIDER

- DFU with slough or necrotic tissue that is difficult to remove by repeated sharp debridement
- Patients who are poor candidates for frequent sharp debridement
- Wounds with high bioburden / biofilm
- As part of a comprehensive wound care plan (offloading, perfusion, infection control, etc.)

ADVANTAGES

- Selective debridement – spares viable tissue
- Effective in removing slough and necrosis
- May reduce bacterial load and biofilm
- Simple, minimally invasive, relatively low cost
- Can be used in various wound settings

LIMITATIONS & CONSIDERATIONS

Evidence for improved healing outcomes is limited

- Not for dry, clean wounds or stable eschar without proper assessment
- Patient acceptance (psychological / cultural)
- Pain or discomfort in some patients
- Need for trained application and availability
- Remove larvae before pupation (max ~7 days)
- Not a substitute for needed surgery or perfusion

Size: ~8–12 mm when mature

EVIDENCE & GUIDELINE POSITION

IWGDF 2023

IWGDF 2023 Recommendation:
DO NOT USE BIOSURGICAL DEBRIDEMENT OVER STANDARD OF CARE.

Strength: Strong Recommendation
Quality of Evidence: Low

Standard care includes appropriate sharp debridement and comprehensive wound management according to clinical need.

KEY MESSAGE: MDT MAY ASSIST SELECTIVE DEBRIDEMENT IN SELECTED WOUNDS, BUT IT SHOULD NOT REPLACE GUIDELINE-BASED STANDARD WOUND CARE AND APPROPRIATE SHARP DEBRIDEMENT.

MDT is one tool in a comprehensive plan: Debridement • Infection Control • Perfusion • Offloading • Moisture Balance • Host Optimization

References: IWGDF Guidelines on the Diagnosis and Treatment of Diabetes-related Foot Infections and IWGDF Guidelines on Wound Healing Interventions in Diabetes, 2023.

SLIDE 12D-1

WOUND DRESSING IN DIABETIC FOOT CARE

"Dress the Wound You See — Match the Dressing to the Wound's Needs

ASSESS the wound → **IDENTIFY** the dominant need → **SELECT** the dressing → **REASSESS** and adjust

GOAL
Select the most appropriate dressing to create the optimal environment for healing and prevent complications.

ASSESS BEFORE YOU DRESS

- Exudate amount
- Moisture balance
- Wound depth / cavity
- Periwound condition
- Infection status
- Perfusion / ischemia
- Location and mechanical pressure

The dressing decision begins with wound assessment.

THERE IS NO UNIVERSALLY "BEST" DFU DRESSING

Choose according to what the wound needs.

WHAT DOES THIS WOUND NEED?

DRY / LOW EXUDATE
Protect + maintain appropriate moisture

MODERATE EXUDATE
Maintain moisture balance + absorb excess fluid

HEAVY EXUDATE
Increase absorption + protect surrounding skin

FRAGILE / MACERATED PERIWOUND
Protect the surrounding skin

WHAT SHOULD A GOOD DRESSING DO?

MANAGE EXUDATE
Avoid excessive wetness and maceration

MAINTAIN APPROPRIATE MOISTURE
Support a favorable healing environment

PROTECT THE WOUND & PERIWOUND
Minimize trauma during dressing changes

BE PRACTICAL FOR THE PATIENT
Comfort, ease of use, frequency of changes, and cost

A DRESSING CANNOT CORRECT:

- **PRESSURE** — Inadequate offloading
- **ISCHEMIA** — Inadequate perfusion
- **UNCONTROLLED INFECTION**
- **SYSTEMIC BARRIERS TO HEALING** (e.g., poor glycemic control, malnutrition, comorbidities)

Do not respond to a non-healing DFU simply by changing the dressing.
Reassess the barriers to healing.

ANTIMICROBIAL ≠ AUTOMATICALLY BETTER

- Do not suggest that colonization alone requires antimicrobial treatment.
- Select antimicrobial approaches only for appropriate clinical indications — not simply because a DFU is colonized.

KEY MESSAGE

MATCH THE DRESSING TO THE WOUND — NOT THE WOUND TO THE DRESSING.
The dressing supports healing; offloading, perfusion, infection control, wound-bed preparation, and host optimization make healing possible.

Use the simplest effective dressing • Review regularly • Document response • Adjust as the wound evolves

Evidence-based principles from IWGDF Guidelines

SLIDE 12D-2A

CHOOSING THE DRESSING:
Start With the Wound's Dominant Need

Assess → Identify the dominant need → Select the dressing family → Reassess

1. DRY / MINIMALLY EXUDATIVE

GOAL: PROVIDE MOISTURE & PROTECT

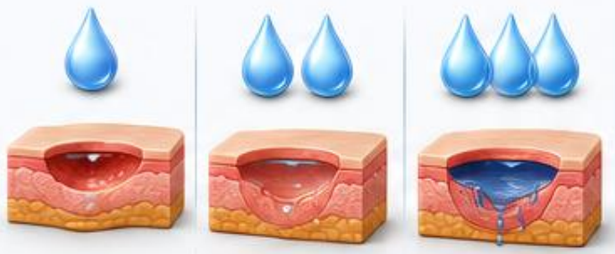
When the wound is dry, thick, or has minimal drainage.

SUITABLE DRESSING FAMILIES

- Hydrogel
- Moisture-donating dressings

2. EXUDATIVE (LOW–MODERATE TO HEAVY)

GOAL: MANAGE EXUDATE & MAINTAIN MOISTURE BALANCE



SUITABLE DRESSING FAMILIES

- Foam
- Alginate / Hydrofiber
- Highly absorbent dressings

3. CAVITY / DEAD SPACE OR SPECIAL STRUCTURES

GOAL: FILL SPACE, CONFORM & PROTECT STRUCTURES

Cavities, undermining, tunnels, or exposed tendon/bone.

SUITABLE DRESSING FAMILIES

- Alginate / Hydrofiber (rope/strip)
- Foam (conformable)
- Non-adherent contact layer

4. PERIWOUND / INFECTION CONCERNS

GOAL: PROTECT SKIN & ADDRESS THE UNDERLYING PROBLEM

Fragile or macerated periwound, or infection risk / presence.

SUITABLE APPROACH

- Protect periwound (barrier film / cream)
- Non-adherent contact layer
- Consider antimicrobial dressings for appropriate indications*

KEY REMINDERS

- ✔ No one dressing is best for all wounds.
- ✔ Match the dressing to the wound's dominant need.
- ✔ Reassess regularly and change as the wound evolves.

A DRESSING CANNOT CORRECT:

* Antimicrobial dressings should be used only when clinically indicated—not simply because a wound is colonized.

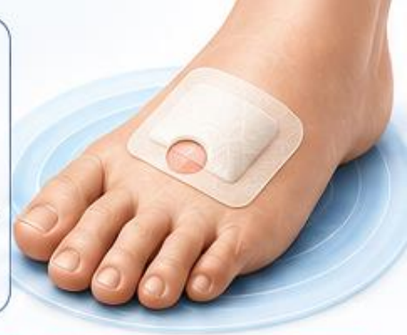
Based on IWGDF 2023 Guidelines & Best Practice Recommendations

SLIDE 12D-2B

DRESSING MATERIALS:
Know What Each One Does

Different materials. Different properties. Choose based on the wound's needs.

GOAL
Use the right **dressing material** to create the right environment for healing, protect the skin, and improve outcomes.



	HYDROGEL	FOAM	ALGINATE / HYDROFIBER	HYDROCOLLOID	FILM / TRANSPARENT	COLLAGEN	ANTIMICROBIAL DRESSINGS*
DRESSING MATERIAL							
BEST USED WHEN	Dry wounds or minimal exudate; necrotic tissue (e.g., slough autolysis).	Low to moderate exudate; superficial wounds; pressure areas.	Moderate to heavy exudate; cavity wounds; bleeding tendency.	Low exudate wounds; epithelializing wounds; as secondary cover.	Superficial, minimally exudative wounds; protect intact or fragile skin.	Clean, non-infected wounds with low to moderate exudate.	Clinically infected wounds or high risk of infection (as appropriate).
USE WITH CAUTION / AVOID WHEN	• Heavy exudate • Deep cavities • Infected wounds (as primary therapy)	• Heavy exudate without high absorption • Infected wounds without systemic management	• Dry wounds • Use with caution in patients with sensitivity to alginates	• Moderate–heavy exudate • Infected wounds • Macerated periwound	• Moderate–heavy exudate • Deep wounds • Infected wounds • Fragile / moist periwound	• Heavily exudative wounds • Infected wounds • Necrotic tissue present	• Not for colonization alone • Long-term use without indication • Replace need for debridement, offloading, or infection control
KEY PEARL	• Adds moisture to dry tissue and supports autolytic debridement.	• Versatile and cushioning—good balance of absorption and protection.	• Forms gel on contact with exudate—locks in fluid and supports hemostasis.	• Occlusive—promotes moist environment and autolytic debridement.	• Breathable barrier—protects from friction, bacteria, and contamination.	• Provides scaffold for tissue repair and supports granulation.	• Adjunct—not a substitute—for appropriate systemic management.

REMEMBER THE 5 PILLARS OF HEALING

1 OFFLOADING 

2 PERFUSION 

3 INFECTION CONTROL 

4 WOUND BED PREPARATION 

5 HOST OPTIMIZATION 

ADVANCED DOES NOT AUTOMATICALLY MEAN BETTER.

Match the material to the wound, the patient, and the goals of care.

PRACTICAL TIPS

- ✓ Protect periwound skin at every dressing change.
- ✓ Change dressings at the right frequency—not too often, not too late.
- ✓ Use the simplest effective dressing that works.
- ✓ Monitor and reassess regularly.

* Examples: Silver, Iodine, PHMB, Honey, Octenidine, etc. | Use only when clinically indicated—not simply because a wound is colonized.

Based on: IWGDF 2023 Guidelines & Best Practice Recommendations

SLIDE 12D-3

WHEN STANDARD CARE IS NOT ENOUGH: WHEN SHOULD WE ESCALATE?

GOAL
Promote healing, prevent complications, and improve outcomes through the right therapy—at the right time.

ADVANCED WOUND THERAPY IS AN ADJUNCT, NOT A SUBSTITUTE FOR OPTIMAL STANDARD CARE

1. PROVIDE OPTIMAL STANDARD CARE


Offloading (pressure relief)


Debridement & wound-bed preparation


Assess & optimize perfusion (revascularize when needed)


Infection management


Moisture balance with appropriate dressings


Optimize the host

2. REASSESS PROGRESS

AT ~4 WEEKS

Has the DFU area decreased by

≥50%?

Compared with baseline
(Use reliable wound measurement)

YES
Adequate response

CONTINUE CURRENT PLAN
Continue standard care and monitor regularly

NO
Inadequate response

3. IF INADEQUATE PROGRESS

REASSESS & ADDRESS BARRIERS

- Is pressure adequately offloaded?
- Is perfusion sufficient?
- Is infection controlled?
- Is osteomyelitis addressed?
- Has adequate debridement occurred?
- Is moisture balance appropriate?
- Are systemic / patient factors optimized?

CONSIDER ADVANCED WOUND THERAPY
in addition to standard care*

WHEN TO CONSIDER

Chronic DFU that fails to reduce in area by ≥50% after ~4 weeks of appropriate standard management.
(ADA Recommendation 12.32)

FOUNDATION THAT MUST BE IN PLACE BEFORE ESCALATION


Effective offloading


Adequate perfusion


Controlled infection


Adequate debridement


Balanced moisture


Optimized host factors

Advanced therapies work best when the foundation is strong.

Advanced does not mean automatic.
Right patient. Right wound. Right timing.
The right therapy makes the difference.

* Modalities with the best evidence (e.g., NPWT, placental membranes, bioengineered skin substitutes, selected acellular matrices, autologous fibrin/leukocyte-platelet patches, topical oxygen therapy) are preferred.

Based on: ADA Standards of Care in Diabetes—2025
Recommendations 12.31–12.32

SLIDE 12D-4

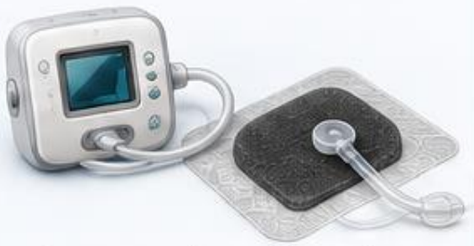
ADVANCED WOUND THERAPIES: WHAT ARE OUR OPTIONS?

For chronic DFUs that fail to heal with optimal standard care, consider these adjunctive therapies supported by clinical evidence.*

GOAL

Use the right advanced therapy for the right patient at the right time—as an adjunct to optimal care.

1 NEGATIVE-PRESSURE WOUND THERAPY (NPWT)



Removes exudate, reduces edema, improves perfusion, promotes granulation tissue.

Examples: NPWT systems (foam or gauze)

2 CELLULAR / TISSUE-BASED PRODUCTS & PLACENTAL MEMBRANES

Provide cells, growth factors, and extracellular matrix to support healing and tissue regeneration.

Examples: Human placental membranes, umbilical tissue products, amniotic membrane products

3 ACELLULAR MATRICES (BIOENGINEERED SKIN SUBSTITUTES)

Provide a scaffold and biological signals to support tissue in-growth and wound healing.

Examples: Dermal matrices, composite skin substitutes

4 GROWTH FACTOR / BIOLOGIC APPROACHES

Deliver growth factors or biologically active agents to stimulate cell proliferation and healing.

Examples: PDGF (becaplermin gel), others in development

5 AUTOLOGOUS BLOOD–DERIVED PRODUCTS

Use the patient's own blood components to promote healing and angiogenesis.

Examples: Platelet-rich plasma (PRP), leukocyte- and platelet-rich fibrin (L-PRF), autologous fibrin/leukocyte-platelet patches

6 OXYGEN THERAPIES

Improve tissue oxygenation, support angiogenesis, and enhance healing.

Examples: Topical oxygen therapy (Topical O_2 systems)

(Evidence stronger for topical oxygen than for hyperbaric oxygen in DFUs)

7 BIOPHYSICAL & ELECTRICAL STIMULATION

Electrical or electromagnetic energy may stimulate cells and enhance healing.

Examples: Electrical stimulation, capacitive coupling, ultrasound therapy

8 OTHER BIOACTIVES & ADJUNCTIVE AGENTS

Selected topical agents may support healing in appropriate clinical settings.

Examples: Medical-grade honey, pH-modulating therapies, enzymes

9 EMERGING & INVESTIGATIONAL THERAPIES

Novel therapies under investigation with promising preclinical or early clinical data.

Examples: Gene therapy, stem cell therapies, microbiome-based therapies, novel biologics

IMPORTANT CONCEPTS

- Advanced therapies are ADJUNCTS, not substitutes, for optimal standard care.
- Evidence quality varies—prefer therapies supported by high-quality RCTs and systematic reviews.
- Patient selection, wound bed preparation, and addressing barriers are essential for success.
- Cost, accessibility, patient preference, and feasibility matter.

The right therapy for the right patient at the right time.

FOUNDATION FIRST

Offloading • Perfusion • Infection control
Debridement • Moisture balance • Host optimization

Build the foundation, then add advanced therapy.

Advanced wound therapies can accelerate healing and improve outcomes when used appropriately and with the right patient.

*Based on: ADA Standards of Care in Diabetes—2025 Recommendations 12.32 and related discussion. (See Table 12.5 in the Standards for full list of categories and evidence levels.)

SLIDE 12D-5

ADVANCED DOES NOT MEAN APPROPRIATE FOR EVERYONE

Before using advanced wound therapy, make sure the FOUNDATION is solid and the patient is the RIGHT ONE.

GOAL

Use advanced therapies appropriately to add value—**NOT** to replace optimal standard care.

1. HAVE WE OPTIMIZED THE ESSENTIALS?

OFFLOADING

Is pressure adequately offloaded?

- Total contact cast
- Removable walker
- Custom device
- Patient adherence

PERFUSION

Is perfusion adequate?

- Pulses / Doppler
- TBI / toe pressure
- $TcPO_2$
- Revascularization when needed

INFECTION CONTROL

Is infection identified and controlled?

- Clinical diagnosis
- Source control
- Appropriate antibiotics

DEBRIDEMENT & WOUND BED

Has adequate debridement been done?

- Remove non-viable tissue
- Manage biofilm
- Prepare wound bed

MOISTURE BALANCE

Is moisture balance appropriate?

- Match dressing to exudate
- Protect periwound
- Avoid maceration

OSTEOMYELITIS MANAGEMENT

Has osteomyelitis been evaluated and addressed?

- Probe-to-bone
- Imaging / labs
- Antibiotics ± surgery

HOST OPTIMIZATION

Are systemic factors optimized?

- Glycemic control
- Nutrition
- Anemia
- Smoking cessation

IF THE ANSWER IS "NO" TO ANY OF THESE...

Fix the problem first. Advanced therapy placed on an unstable foundation will not succeed.

2. IS THE WOUND TRULY STALLED?

Has the wound area decreased by ≥50%?

- **YES** → Continue current plan and monitor.
- **NO** → Reassess barriers and consider advanced therapy.

3. DO WE HAVE THE RIGHT INDICATION AND EXPECTATION?

- Is the patient a good candidate for this therapy (comorbidities, life expectancy, ability to adhere)?
- Is there good evidence for this modality in this type of wound?
- What is the realistic expected benefit in this case (healing, healing time, limb preservation, QoL)?
- Are there contraindications, risks, or potential adverse effects?

4. CONSIDER PRACTICAL FACTORS

- Cost, insurance coverage, and overall value.
- Availability, access to skilled providers, and follow-up.
- Patient preference, lifestyle, and burden of treatment.
- Resource stewardship: use what is needed—not simply what is available.

KEY MESSAGE

Advanced wound therapy is a powerful **ADJUNCT** when used at the right time, in the right patient, on the right wound, and with the right foundation.

The goal is not more technology.
The goal is **better healing.**

* Categories and examples based on ADA Standards of Care in Diabetes—2025 (Recommendation 12.32 and Table 12.5). Apply clinical judgment and local expertise.

Based on: ADA Standards of Care in Diabetes—2025, Section 12. Retinopathy, Neuropathy, and Foot Care.

SLIDE 12E

OPTIMIZE THE HOST

"The Wound Belongs to a Patient"

Wound healing depends not only on local care, but also on the patient's biological, medical, functional, and social environment.

GOAL

Create the best internal environment for healing and prevent recurrence and amputation.

1 GLYCEMIA

Optimize glucose safely.

- Avoid severe hyperglycemia
- Avoid hypoglycemia
- Individualize targets
- Coordinate with the diabetes care team

2 NUTRITION & HYDRATION

Fuel tissue repair and immunity.

- Adequate calories, protein, vitamins and minerals
- Assess for weight loss and malnutrition
- Correct deficiencies (iron, B12, vitamin D, etc.)
- Ensure adequate hydration

3 SMOKING & CARDIOVASCULAR RISK

Stop smoking – it impairs blood flow and wound healing.

- Smoking cessation for all
- Control blood pressure
- Manage lipids
- Antiplatelet therapy when indicated
- Promote physical activity as tolerated

4 COMORBIDITIES

Identify, stabilize and coordinate care.

- Chronic kidney disease / dialysis
- Anemia – evaluate and treat
- Heart failure and venous disease
- Edema management
- Liver disease / immune dysfunction

THE PATIENT

- ✓ Unique
- ✓ Complex
- ✓ More than a wound

5 MEDICATIONS & ACUTE ILLNESS

Review and adjust to support healing and safety.

- Review all medications
- Adjust for renal function
- Consider effects on glucose, nutrition, bleeding, edema, and immunity
- Adjust diabetes therapy during infection or acute illness

6 FUNCTIONAL, PSYCHOLOGICAL & SOCIAL CONTEXT

Make the plan realistic and achievable.

- Mobility, offloading and fall risk
- Vision, dexterity and cognition
- Depression, motivation and coping
- Adherence capacity and education
- Family / caregiver support
- Affordability, access to care and supplies
- Occupation and daily living demands

KEY MESSAGE

Treat the ulcer locally – optimize the patient systemically.

A perfectly treated wound may still fail if the patient is not optimized.

REMEMBER

- ✓ The host determines the healing potential.
- ✓ Small improvements in multiple domains lead to meaningful healing.

An interdisciplinary team approach gives the patient the best chance for healing and limb preservation.

Endocrinology → Nursing → Nutrition → Nephrology → Cardiology → Psychology → Physiotherapy → Social Work

SLIDE 12F

PREVENT RECURRENCE:

A Healed DFU Is a Foot in Remission—Not a Cured Foot

GOAL
Prevent the next ulcer, protect function, and improve quality of life.

Wound closure ≠ elimination of risk. Ongoing prevention protects the foot for life.

1 FIND & REMOVE THE ORIGINAL CAUSE

Identify and correct the factor(s) that led to the first ulcer.

- Repetitive pressure
- Footwear problems
- Deformity / biomechanics
- Trauma or thermal injury
- Behavior / occupation
- Ischemia

2 PROTECT FROM PRESSURE

Offloading continues after healing.

- Therapeutic footwear with demonstrated pressure relief at the high-risk area
- Custom orthoses / insoles
- Rocker soles, accommodative footwear when indicated
- ADHERENCE is essential

3 DETECT & TREAT PRE-ULCERATIVE LESIONS

Act early—intercept before breakdown.

- Callus (especially with hemorrhage)
- Blisters
- Fissures / cracks
- Pressure erythema
- Nail pathology
- Maceration / skin problems

4 DAILY SELF-SURVEILLANCE & FOOT SELF-CARE

Look daily. Protect always. Act early.

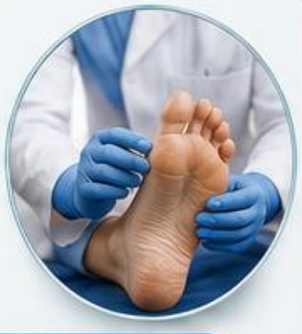
- Inspect daily (use mirror or assistance if needed)
- Wash and dry, especially between toes
- Moisturize dry skin (not between toes)
- Trim nails straight across
- No barefoot walking
- Check inside footwear
- Report problems early

Involve family/caregivers when needed (vision, mobility, dexterity, cognition).

5 LIFELONG PROFESSIONAL SURVEILLANCE

High-risk foot needs ongoing expert care.

- Integrated foot care: professional foot care + adequate footwear + structured self-care education
- Foot examination by trained professional at every visit
- For high-risk patients: reassess approximately every 1–3 months (or as needed)*
- Early identification of new risk factors



HEALED → NOT DISCHARGED
HEALED → ENTER THE PREVENTION PROGRAM

EMERGING TOOL TEMPERATURE MONITORING

Smart mats • Smart insoles • Smart socks

Detects inflammation (↑ temperature) before ulceration. May help intercept risk early.

6 CONTROL THE HOST & STRUCTURAL RISKS

Optimize overall health and address remaining risks.

Glycemic control | Smoking cessation | PAD management | Renal health | Nutrition & weight | Deformity correction | Activity & function

Consider surgical correction of deformity for recurrent ulcers not controlled by appropriate footwear/offloading (selected patients).

HEALING IS NOT THE END OF DFU CARE—IT IS THE BEGINNING OF RECURRENCE PREVENTION.
A healed ulcer needs continued protection, early detection, and lifelong attention.

Every contact is an opportunity to prevent the next ulcer.

* Frequency should be individualized based on risk level, wound history, comorbidities, and clinical judgment.

Sources: ADA Standards of Care in Diabetes—2026, Section 12.
IWGDF 2023 Guidelines on the Prevention of Foot Ulcers in Persons with Diabetes.

SLIDE 13

MASTER DFU MANAGEMENT ALGORITHM:
From First Encounter to Healing—and Beyond

A systematic, stepwise, patient-centered approach for the best outcomes

GOAL
Heal the ulcer, save the limb, and improve quality of life.

1 DFU PRESENTS
(Any new wound, blister, callus with bleeding, or drainage)

2 INITIAL ASSESSMENT
Answer the 6 key questions

- ✓ Where is the wound?
- ✓ How deep is it?
- ✓ Is it infected?
- ✓ Is it ischemic?
- ✓ Is bone involved?
- ✓ Why did it occur and Why is it not healing?

3 IDENTIFY URGENT CONCERNS
- Severe infection / sepsis
- Critical limb ischemia
- Deep abscess / necrotizing process
- Suspected osteomyelitis
- Need for urgent surgical / source control

Stabilize, treat, ad refer appropriately.

4 CLASSIFY SEVERITY
(e.g., SINBAD / IDSA-IWGDF)

MILD	No infection, no ischemia
MODERATE	Infection ± ischemia (not severe)
SEVERE	Severe infection ± ischemia
LIMB-THREATENING	Any urgent concern (see Step 3)

5 START THE 6 PILLARS IMMEDIATELY
- OFFLOAD the pressure
- RESTORE PERFUSION
- CONTROL INFECTION
- DEBRIDE & OPTIMIZE WOUND BED
- OPTIMIZE THE HOST
- PREVENT RECURRENCE

6 CHOOSE & APPLY APPROPRIATE DRESSING

Match dressing to wound needs: exudate, infection risk, tissue type, peri-wound condition. Reassess at each visit.

7 MEASURE & FOLLOW TRAJECTORY
Use planimetry or digital measurement & photography

BASELINE (Day 0)

Track progress at regular intervals (e.g., every 1–2 weeks)

Look for:
- ✓ Decrease in area
- ✓ Decrease in depth
- ✓ Improved wound bed
- ✓ Healthier peri-wound

8 IS THE WOUND HEALING APPROPRIATELY?
Reassess around ~4 weeks

YES
✓ ≥50% reduction in area*.

Continue current plan and monitor.

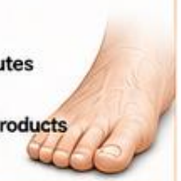
NO
✗ <50% reduction in area*.

Go to Step 9

9 REASSESS WHY & CORRECT BARRIERS
- Inadequate offloading?
- Inadequate perfusion?
- Persistent infection?
- Inadequate debridement / wound bed?
- Moisture imbalance?
- Host factors?

Fix the problems, reinforce adherence, and optimize care.

10 CONSIDER ADVANCED WOUND THERAPY
(If appropriate after Step 9)
- NPWT
- Placental membranes
- Bioengineered skin substitutes
- Acellular matrices
- Autologous blood-derived products
- Topical oxygen / others



Use as an adjunct, not as a substitute.*

11 WOUND HEALED
Complete epithelialization with no drainage.

Congratulations— but the job is not done!

12 ENTER RECURRENCE PREVENTION PROGRAM
(Slide 12F: Prevent Recurrence)

- Therapeutic footwear & offloading
- Find & treat pre-ulcerative lesions
- Daily self-inspection & foot care
- Regular professional follow-up
- Control systemic & structural risk factors
- Education, support, adherence

13 ONGOING SURVEILLANCE—LIFELONG
High-risk foot requires continuous care.

FOLLOW-UP INTERVAL (as needed)

VERY HIGH RISK (previous ulcer / amputation)	HIGH RISK (LOPS + deformity / PAD / callus)	MODERATE RISK (LOPS without deformity)
Every 1–3 months	Every 3–6 months	Every 6–12 months

AT ANY POINT: IF NEW ULCER OR RED FLAGS APPEAR

RETURN TO STEP 1
Don't wait. Act early.

PREVENT • PROTECT • HEAL • MONITOR
The right care, at the right time, for the right patient.

> "Healing closes the wound. Prevention protects the limb."

* Compared with baseline (use reliable wound measurement).

Based on: ADA Standards of Care in Diabetes—2026, Section 12.
IWGDF 2023 Guidelines on the Prevention and Management of Diabetic Foot Disease.

SLIDE 14

TEN RULES THAT SAVE THE DIABETIC FOOT

Take-Home Messages

Isfahan Endocrine and Metabolism Research Center
Isfahan University of Medical Sciences

1 

FIND WHY THE ULCER OCCURRED.

Identify and correct the underlying cause(s) of the first ulcer—pressure, footwear, deformity, trauma, ischemia, or behavior. Mechanism matters.

2 

DESCRIBE AND CLASSIFY— BUT DON'T COLLECT CLASSIFICATIONS.

Use a systematic approach and appropriate tools (e.g., SINBAD, IDSA/IWGDF) to guide management—not to label the patient.

3 

INFECTION IS A CLINICAL DIAGNOSIS.

Look, probe, smell, and assess the patient.
Treat the source, not just the culture.
Use antibiotics wisely.

4 

NEVER FORGET PERFUSION.

Assess pulses, Doppler, and advanced tests when needed. Ischemia must be recognized and revascularized when possible.

5 

OFFLOAD PRESSURE—NOT MERELY THE WOUND.

Offloading is the cornerstone of healing.
Use the right method, and ensure adherence—at every step.

6 

DEBRIDE AND CREATE THE RIGHT WOUND ENVIRONMENT.

Remove non-viable tissue, control biofilm, and optimize the wound bed.
Prepare it to heal.

7 

MATCH THE DRESSING TO THE WOUND'S NEEDS.

Manage exudate, protect the wound, maintain moisture balance, and prevent maceration and injury.

8 

OPTIMIZE THE PATIENT—NOT JUST THE FOOT.

Glycemic control, nutrition, anemia, smoking cessation, renal care, mobility, and comorbidity management all matter.

9 

IF HEALING STALLS, REASSESS THE BARRIERS FIRST.

Don't escalate technology before ensuring the basics are perfect. Fix what is preventing healing.

10 

AFTER HEALING, PREVENT THE NEXT ULCER.

Protect from pressure, treat pre-ulcerative lesions, educate and empower, ensure lifelong surveillance.
Healing closes the wound; prevention protects the foot.

THE RIGHT CARE, AT THE RIGHT TIME, FOR THE RIGHT PATIENT.

The best DFU treatment is not the most advanced treatment— it is the right intervention, for the right mechanism, at the right time.

A multidisciplinary, patient-centered approach delivers the best outcomes.

Save the foot. Save the limb. Save the life.

Key References: ADA Standards of Care in Diabetes—2026, Section 12 (Retinopathy, Neuropathy, and Foot Care) • IWGDF 2023 Guidelines on the Prevention and Management of Diabetic Foot Disease

SIX TAKE-HOME MESSAGES

A practical checklist for every diabetic foot ulcer

1

FIND THE CAUSE

Mechanism guides treatment and prevents recurrence.

2

TRIAGE THREATS

Infection, ischemia and deep tissue involvement set urgency.

3

MEASURE PERFUSION

Pulses alone do not establish healing capacity.

4

TREAT IN PARALLEL

Apply all six healing pillars; audit them when progress stalls.

5

CHOOSE BY NEED

Dressings support the wound; they do not replace foundational care.

6

KEEP REMISSION

After closure, protect pressure, footwear and surveillance for life.

THE GOAL: A HEALED, FUNCTIONAL FOOT—KEPT IN REMISSION

SELECTED GUIDELINES & CORE REFERENCES

Detailed source cues are also included in the speaker notes for each slide.

IWGDF 2023 Practical Guidelines

Core diagnosis, organization of care and treatment principles.

IWGDF/IDSA 2023 DFI Guideline

Diagnosis, severity, culture, antibiotics, surgery and osteomyelitis.

IWGDF 2023 Prevention Guideline

Risk stratification, education, surveillance and recurrence prevention.

IWGDF 2023 Wound-Healing Guideline

Debridement, dressings and adjunctive wound-healing interventions.

IWGDF 2023 Offloading Guideline

Device selection and hierarchy for neuropathic plantar ulcers.

ADA Standards of Care in Diabetes—2026, Section 12

Foot assessment, treatment, referral and advanced-therapy escalation.

IWGDF/ESVS/SVS 2023 PAD Guideline

Diagnosis, prognosis and revascularization in diabetes-related foot disease.

Siavash et al.—Khorshid classification & MDT work

Local mechanism-based classification and refractory atypical DFU research.

Use local antimicrobial resistance data, vascular expertise, product availability and patient context when applying guideline recommendations.

Thank You

FOR YOUR ATTENTION

The best DFU treatment is not the most advanced treatment—
it is the right intervention, for the right mechanism,
at the right time.

PREVENT • PROTECT • HEAL • MONITOR

Isfahan Endocrine and Metabolism Research Center

Isfahan University of Medical Sciences
Isfahan, Iran

Healing closes the wound.
Prevention protects the foot.

Mansour Siavash, MD, FACE
Professor of Endocrinology and Metabolism

Isfahan Endocrine and Metabolism Research Center (IEMRC)
Isfahan University of Medical Sciences
Isfahan, Iran

Together,
let's save the foot
and save the limb.

THANK YOU

Questions & clinical discussion

“The right treatment begins with the right question.”

Mansour Siavash, MD
Isfahan Endocrine and Metabolism Research Center

Assessment • Cause • Healing • Remission

ASSESS ACCURATELY

TREAT THE CAUSES

PROMOTE HEALING

PREVENT RECURRENCE

IMPROVE LIVES