The Utilization of an Autologous Blood Clot Tissue Matrix in the Treatment of Chronic Wounds in Patients with Diabetes

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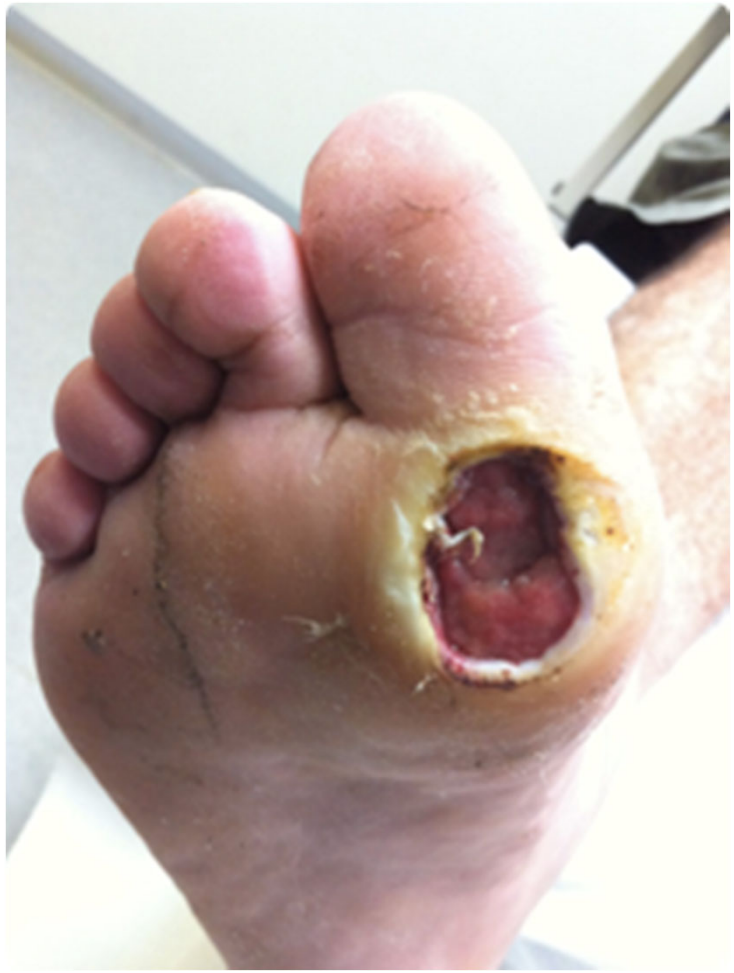
Barry University School of Podiatric Medicine

Disclosures

- I am a consultant for RedDress Ltd

A neuropathic ulcer in a patient with diabetes…

What would you do?



Wound Bed Preparation

Identify and
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Address patient-
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Debride d
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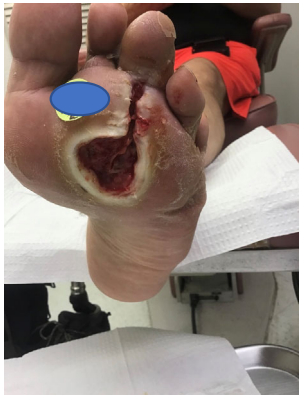
balance



Sibbald RG, et al (2011) Adv Skin Wound Care. 24:415-36
Schultz GS, Sibbald RG, Falanga V et al. Wound bed preparation: a systemic approach to wound management. Wound Repair and Regeneration, 2003;11:1-28

Objectives

- To explain the epidemiology of the diabetic foot
- To list the differences between acute and chronic wound biochemistry and the extracellular matrix
- To learn about an autologous blood clot tissue wound matrix (RD1) that has been shown to accelerate wound healing
- To hypothesize the mechanism of action(MOA) of topically applied blood clot tissue

Diabetes Mellitus: Overview

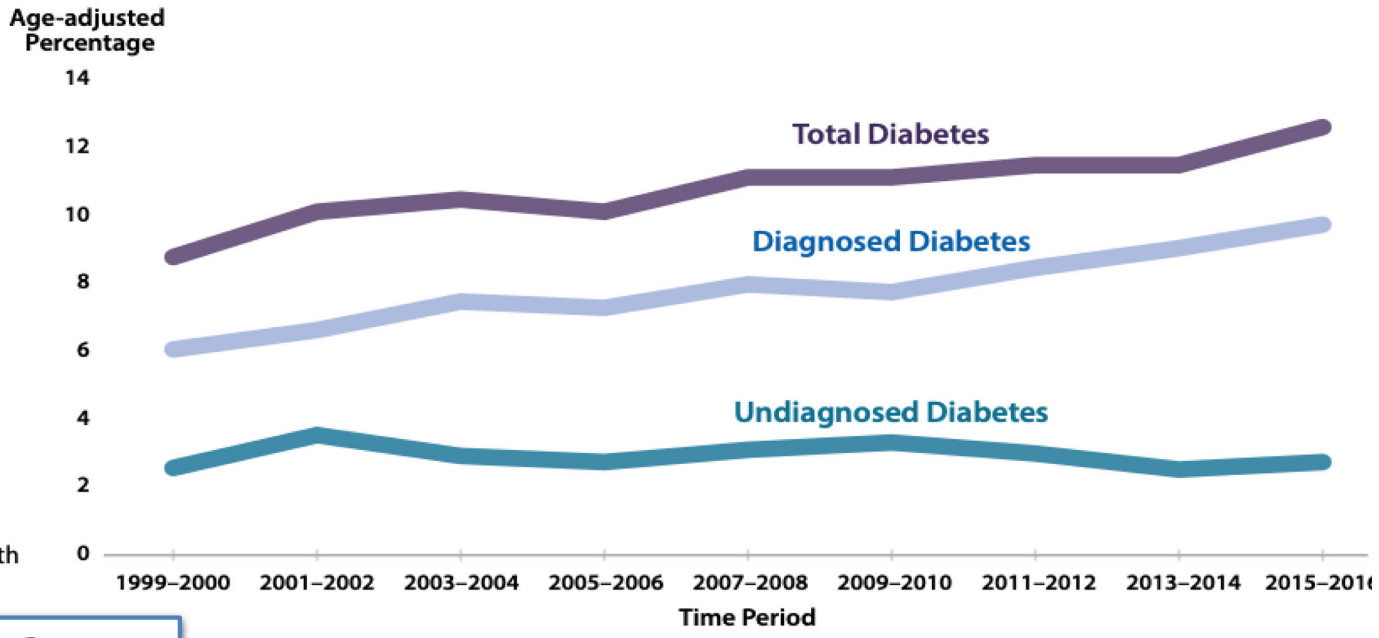
- In 2015, diabetes mellitus affected 30.3 million people in the United States[1] and is projected to affect more than 12% of the population by the year 2050[2]
- Currently 9.4% of the population
- In 2007, diabetes was the 7th leading cause of death in the United States, causing more than 71,000 deaths[3]

1. Diabetes.org, 2015 ;Centers for Disease Control and Prevention. National diabetes fact sheet: national estimates and general information on diabetes and prediabetes in the United States, 2011. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, 2011. 2. Narayan KMV, et al. *Diabetes Care*. 2006;29(9):2114-2116. 3. Xu JQ, Kochanek KD, Murphy SL, Tejada-Vera B. Deaths: Final data for 2007. National vital statistics reports; vol 58 no 19. Hyattsville, MD: National Center for Health Statistics. 2010.

Trends in Prevalence of Diagnosed Diabetes, Undiagnosed Diabetes, and Total Diabetes

- During 1999–2016, the age-adjusted prevalence of total diabetes significantly increased among adults aged 18 years or older (Figure 1).
- Prevalence estimates were 9.5% in 1999–2002 and 12.0% in 2013–2016 (Appendix Table 2).
- During this period, the age-adjusted prevalence significantly increased for diagnosed diabetes. No significant change in undiagnosed diabetes prevalence was detected (Figure 1; Appendix Table 2).

Figure 1. Trends in age-adjusted prevalence of diagnosed diabetes, undiagnosed diabetes, and total diabetes among adults aged 18 years or older, United States, 1999–2016.



Notes: Diagnosed diabetes was based on self-report. Undiagnosed diabetes was based on fasting plasma glucose and A1C levels among people self-reporting no diabetes.

Data source: 1999–2016 National Health and Nutrition Examination Surveys.

National Diabetes Statistics Report 2020, CDC

An Epidemic of Diabetes

By 2030 the number of people with diabetes globally will rise to an estimated:

552 MILLION[1]

1. The International Diabetes Foundation, IDF Diabetes Atlas, 5th ed.: http://www.idf.org/diabetesatlas/5e/the-global-burden. Accessed February 23, 2012.

Diabetic Foot Ulcer(s)

- One of the most common complications of diabetes
- Annual incidence 1% t
- Lifetime risk 15
- ~15% of diab tation
- ~85% of lowe re proceeded by
- Peripheral neuropat in diabetic foot ulcers
  - Other factors: foot deformity, callus, trauma, infection, and peripheral vascular disease

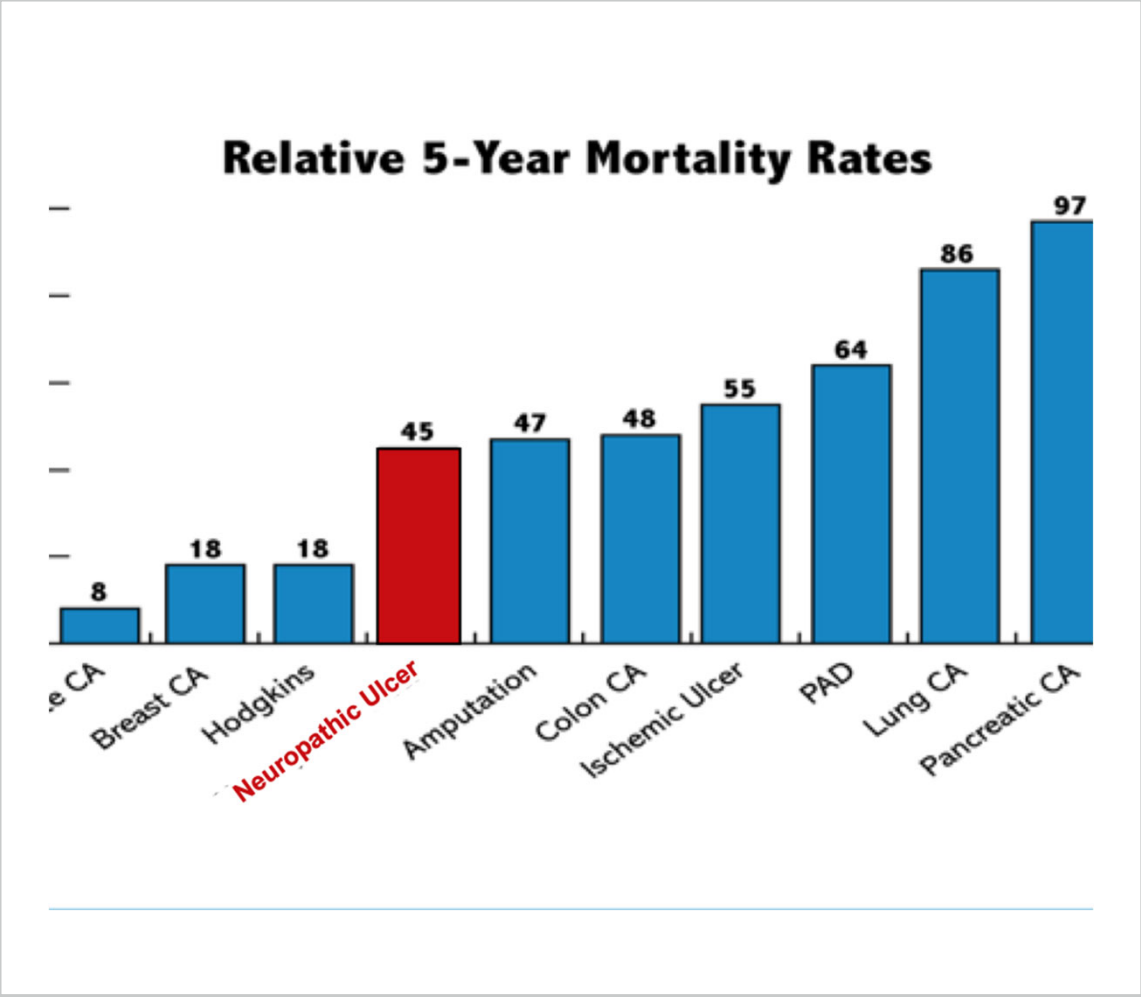
1 million amputations globally
in patients with diabetes
(every 30 seconds)
In the United States,
1200 amputations weekly

Reiber and Ledoux. In The Evidence Base for Diabetes Care. Williams et al, eds. Hoboken, NJ: John Wiley & Sons; 2002:641-665. Boulton AJ, et al. N Engl J Med. 2004;351(1):48-55.Sanders LJ, et al. J Am Podiatry Med Assoc. 1994;84(7):322-328. Boulton AJ, et al. Lancet. 2005;366(9498):1719-1724. Ramsey SD, et al. Diabetes Care. 1999;22(3):382-387. Pecoraro RE, et al. Diabetes Care. 1990;13(5):513-521. Apelqvist J, et al. Diabetes Metab Res Rev. 2000;16(Suppl 1):S75-S83. Moulik PK, et al. Diabetes Care. 2003;26(2):491-494.

Consequences of Unhealed Neuropathic Ulcers

- Nearly half of all neuropathic ulcers result in death within 5 years

- Armstrong DG, Int Wound J (2007);4(4): 286-287



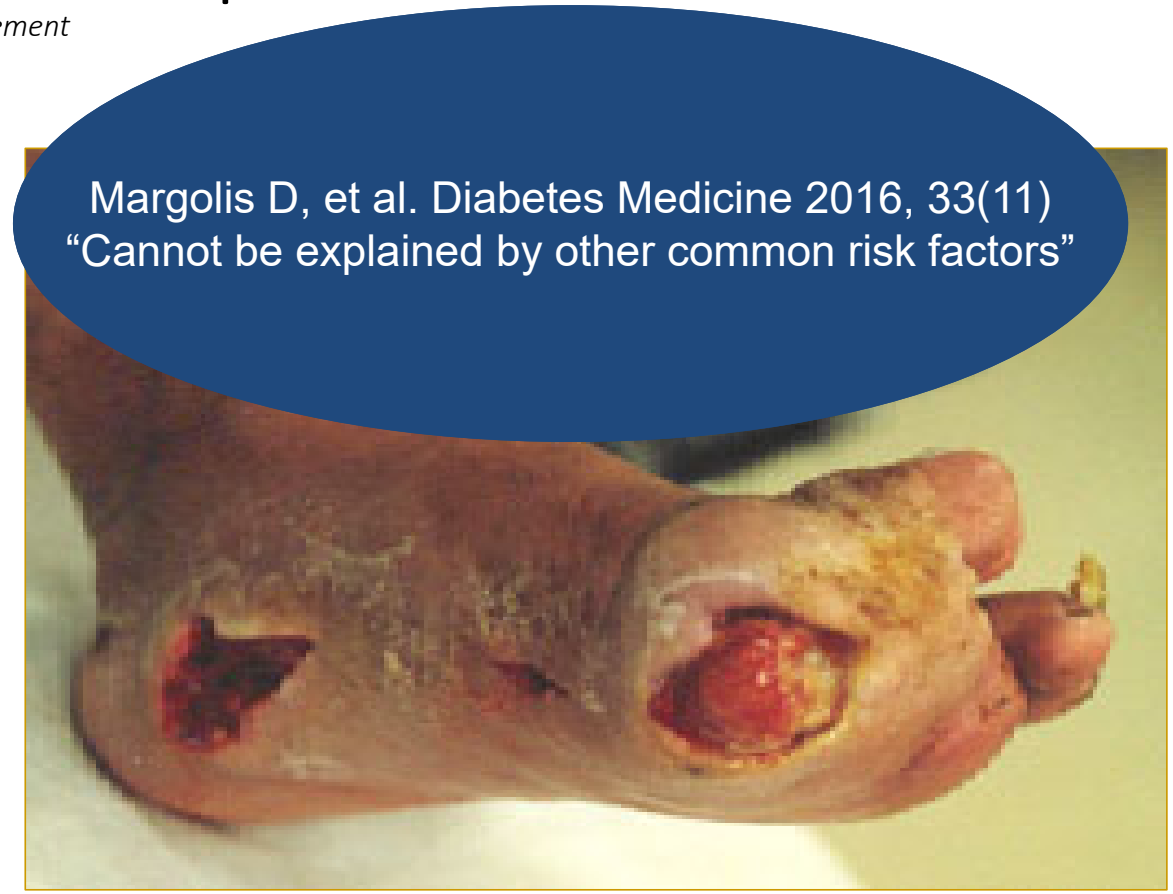
History of Foot Ulcer Increases Mortality Among Individuals with Diabetes

- **Ten Year Follow-up of the Nord-Trøndelag Health Study, Norway**
  - A large population based study examined the association between foot ulcers in patients with diabetes and mortality risk while controlling for disease factors
  - Foot ulcers were independently associated with increased mortality risk
    - Patients with diabetes and a foot ulcer had an increased mortality risk of 2.3-fold (229%) compared to non-diabetic subjects
    - In patients with diabetes, presence of a foot ulcer alone increased mortality risk by 47%

Iversen et al. Diabetes Care. 2009;32:2193-9.

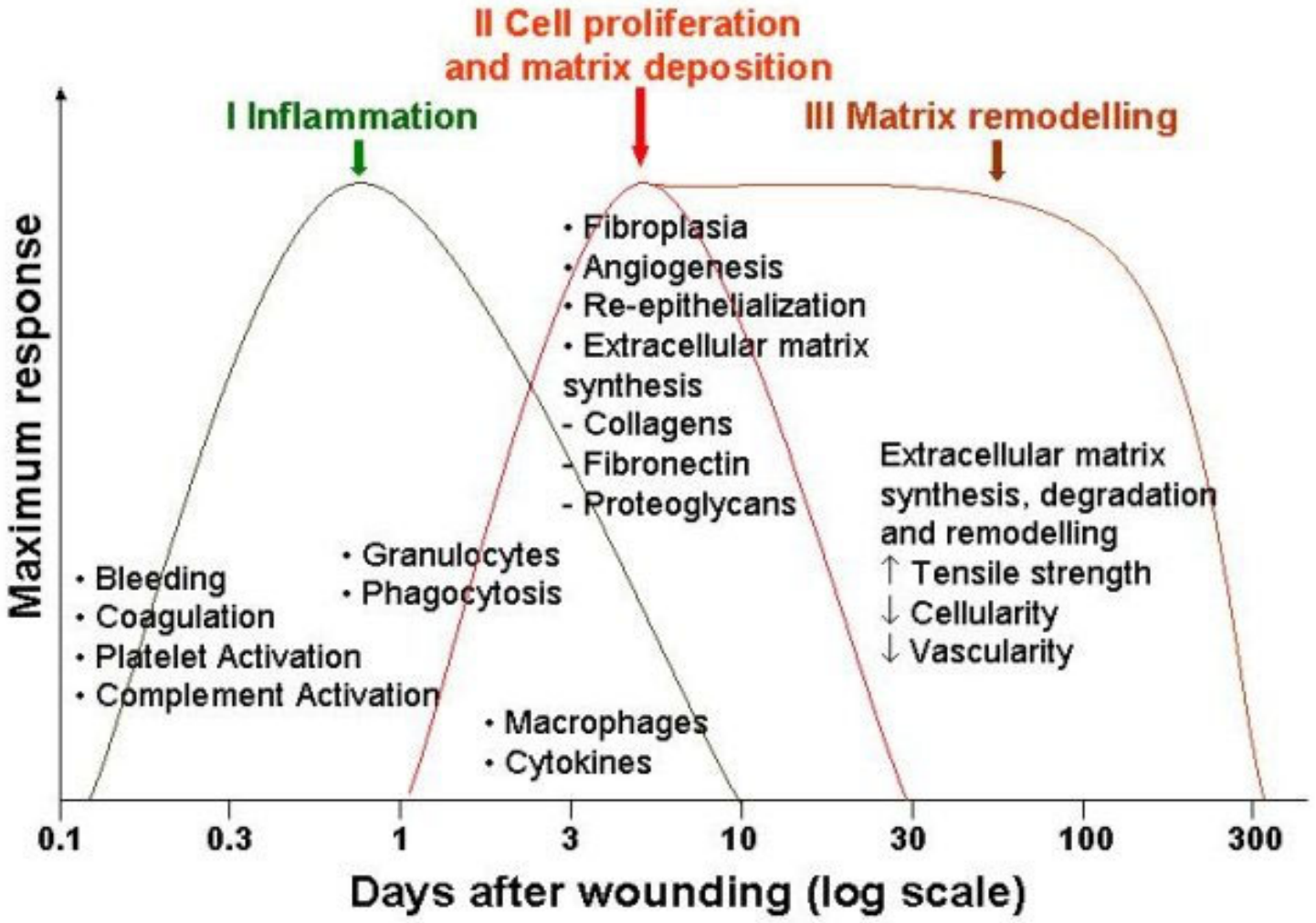
Just Having a Neuropathic Foot Ulcer is a Marker for Death!

Snyder RJ (2010) Podiatry Management



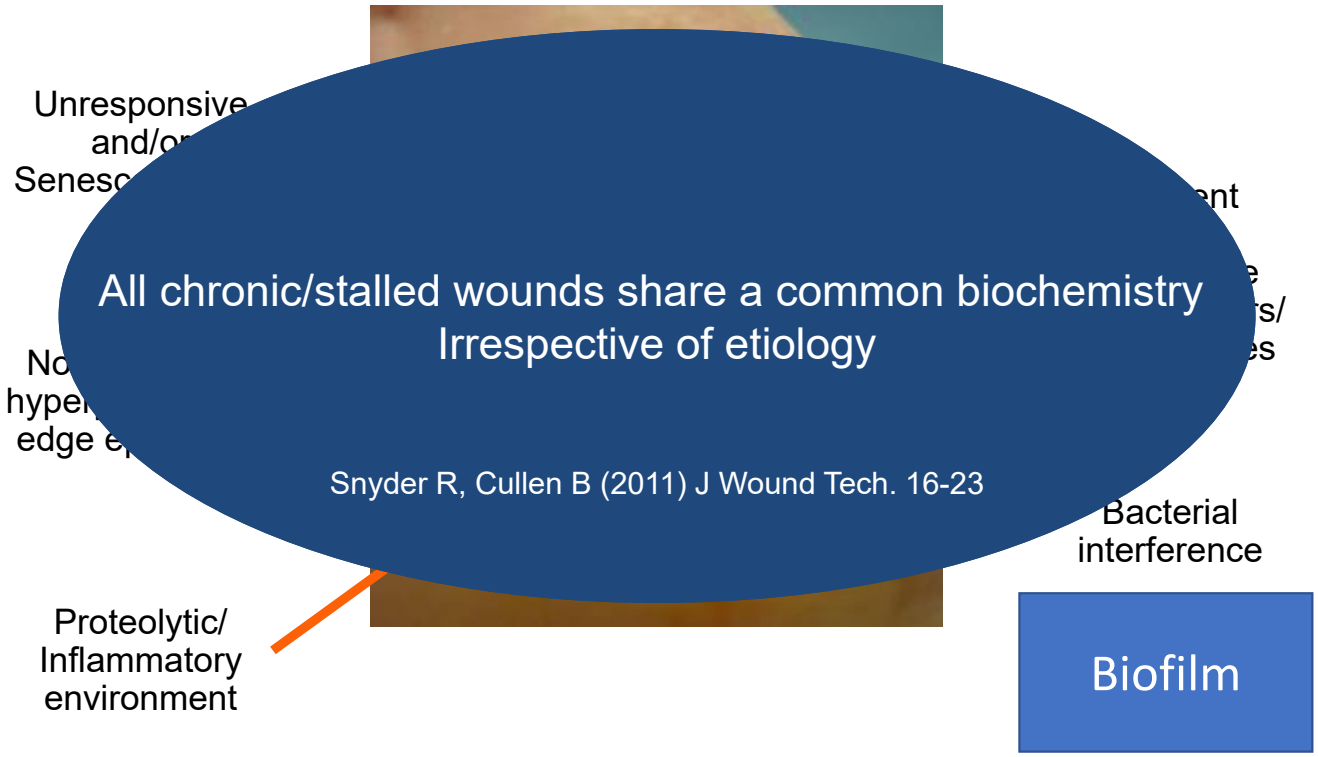
What is the problem?

Wound H


II Cell proliferation
and matrix deposition
I Inflammation
III Matrix remodelling
Maximum response
• Fibroplasia
• Angiogenesis
• Re-epithelialization
• Extracellular matrix
synthesis
- Collagens
- Fibronectin
- Proteoglycans
Extracellular matrix
synthesis, degradation
and remodelling
↑ Tensile strength
↓ Cellularity
↓ Vascularity
• Granulocytes
• Phagocytosis
• Bleeding
• Coagulation
• Platelet Activation
• Complement Activation
• Macrophages
• Cytokines
0.1
0.3
1
3
10
30
100
300
Days after wounding (log scale)

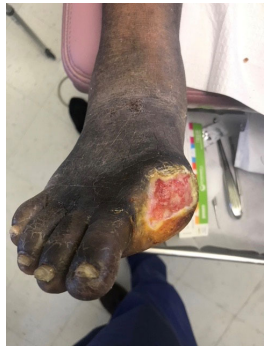
Proposed Mechanisms for Chronicity in Diabetic Foot Ulcer

Robert Kirsner, 2010



Falanga V. The Chronic Wound: Impaired Healing and Solutions in the Context of Wound Bed Preparation. *Blood Cells and Diseases*, 2004;32:88-94

Healing Neuropathic Ulcers: Results of a Meta-Analysis



Recent Meta-analysis
16 Randomized clinical trials
Healing rates:
14.9 % at week 6
33.4% at week 12
43% at week 20

Parks et al (2020) Diabetes 2020 Jun (Supplement 1)

0

12-Week End Point (N=450)

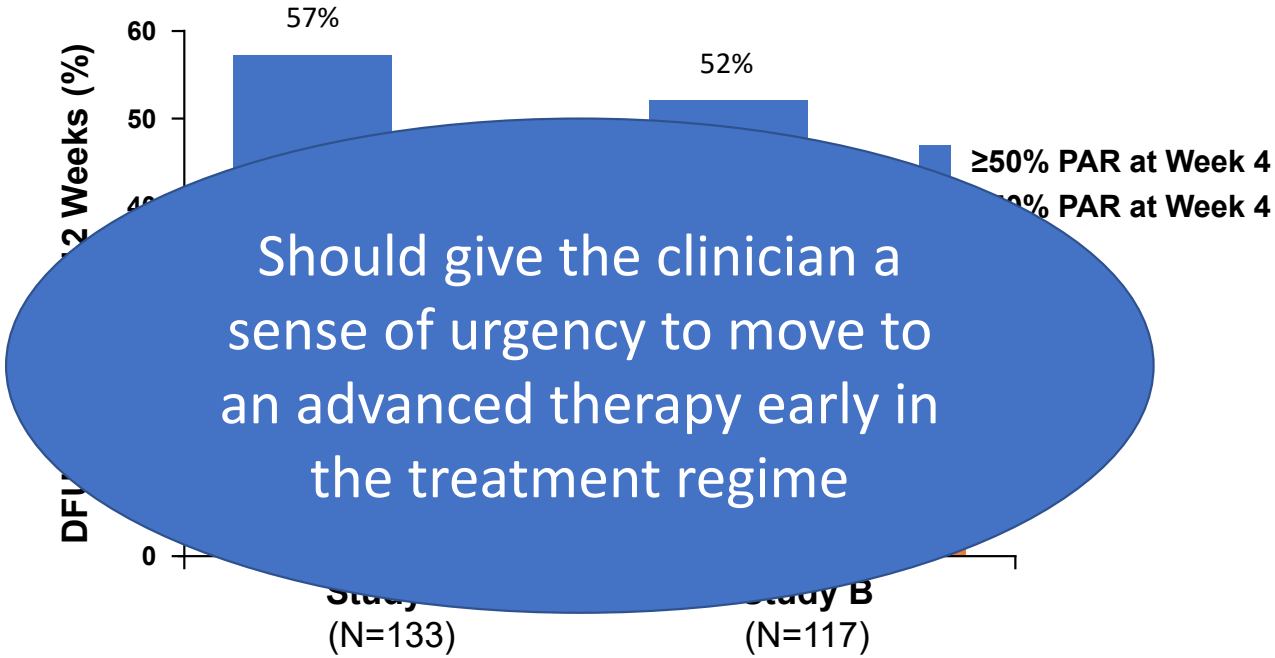
20-Week End Point (N=172)

Results have improved over the past 20 years however endpoints remain low

- The
  hea
- Even with good, standard wound care, healing neuropathic ulcers in patients with diabetes continues to be a challenge

Margolis, et al. *Diabetes Care*. 1999;22:692.

Association Between PAR at Week 4 & DFU Closure at Week 12



Should give the clinician a sense of urgency to move to an advanced therapy early in the treatment regime

- Data was dichotomized by PAR of <50% or ≥50% by week 4 to assess the association of PAR with DFU closure by 12 weeks

Snyder R, et al. 2010 Ostomy Wound Management

ORIGINAL ARTICLE

Differentiating diabetic foot ulcers that are unlikely to heal by 12 weeks following achieving 50% percent area reduction at 4 weeks

Robert A Warriner, Robert J Snyder, Matthew H Cardinal

Warriner RA, Snyder RJ, Cardinal MH. Differentiating diabetic foot ulcers that are unlikely to heal by 12 weeks

following achieving 50% percent area reduction at 4 weeks. Int W

ABSTRACT

This retrospect foot ulcer (DFU) trials in an effort to differentiate u ion (PAR) at 4 weeks]. Predicted and actual wound d compared for ulcers that did and did not heal at

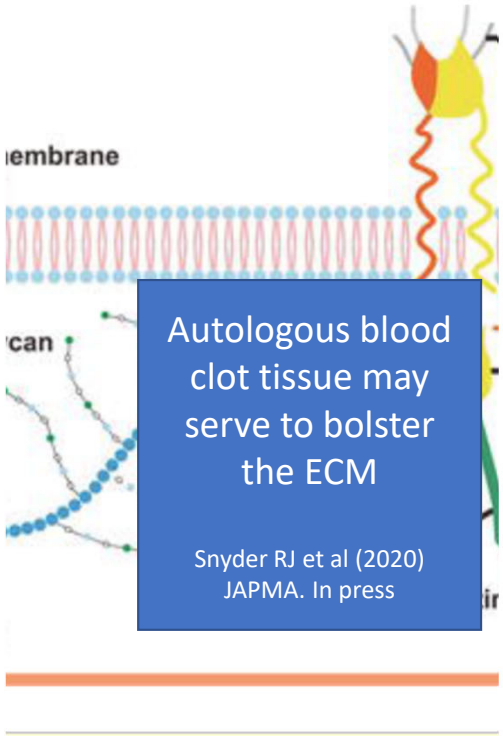
Deviations fro ealing significantly lowered healing rates (P = 0·00 ieving ≥90% versus <90% PAR at 8 weeks had a 2·7-fold higher healing rate at 12 weeks (P = 0·001). A PAR of <90% at 8 weeks provided a negative predictive value for DFU healing at 12 weeks of 82%.

Ulcers that fail to progress or worsen from weeks 4 to 6
and those that fail to achieve 90% PAR at week 8
are unlikely to heal by week 12

Key words: Diabetic foot ulcer • Healing rates • Percent area reduction • Predicting failure to heal

Int Wound J. 2011 Dec;8(6):632-7

The Extracellula



Autologous blood clot tissue may serve to bolster the ECM

Snyder RJ et al (2020) JAPMA. In press

Wound Repair and Regeneration

Dynamic reciprocity in the wound microenvironment

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2. Department of Pathology, Vanderbilt University Medical Center and Research Service, VA Tennessee Valley Healthcare System, Nashville, Tennessee,
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DOI:10.1111/j.1524-475X.2011.00673.x

ABSTRACT

Here, we define dynamic reciprocity (DR) as an ongoing, bidirectional interaction among cells and their surrounding microenvironment. In this review, we posit that DR is especially meaningful during wound healing as the DR-driven biochemical, biophysical, and cellular responses to injury play pivotal roles in regulating tissue regenerative responses. Such cell–extracellular matrix interactions not only guide and regulate cellular morphology, but also cellular differentiation, migration, proliferation, and survival during tissue development, including, e.g., embryogenesis, angiogenesis, as well as during pathologic processes including cancer, diabetes, hypertension, and chronic wound healing. Herein, we examine DR within the wound microenvironment while considering specific examples across acute and chronic wound healing. This review also considers how a number of hypotheses that attempt to explain chronic wound pathophysiology may be understood within the DR framework. The implications of applying the principles of DR to optimize wound care practice and future development of innovative wound healing therapeutics are also briefly considered.

Normal wound healing is characterized by a well-coordinated, progressive series of events designed to restore the barrier function and mechanical integrity of the skin. Like other developmental processes and tumor growth, wound healing involves interactions between cells and their microenvironment, of which the extracellular matrix (ECM) is the primary component.[1–3] It is largely through these interactions that cells are directed to differentiate or dedifferentiate, proliferate, or remain quiescent, and assume the architecture and function of the skin vs. that of some other organ.[1,4]

More than 25 years ago, it was noted that interactions between cells and the ECM occur both ways—that is, they are reciprocal.[5,6] Moreover, it was noted that these interactions were dynamic, continuously changing in response to cues from the microenvironment.[5,6] These observations were collectively termed "dynamic reciprocity" (DR) indicating the ongoing, bidirectional interactions between cells and the ECM (Figure 1).

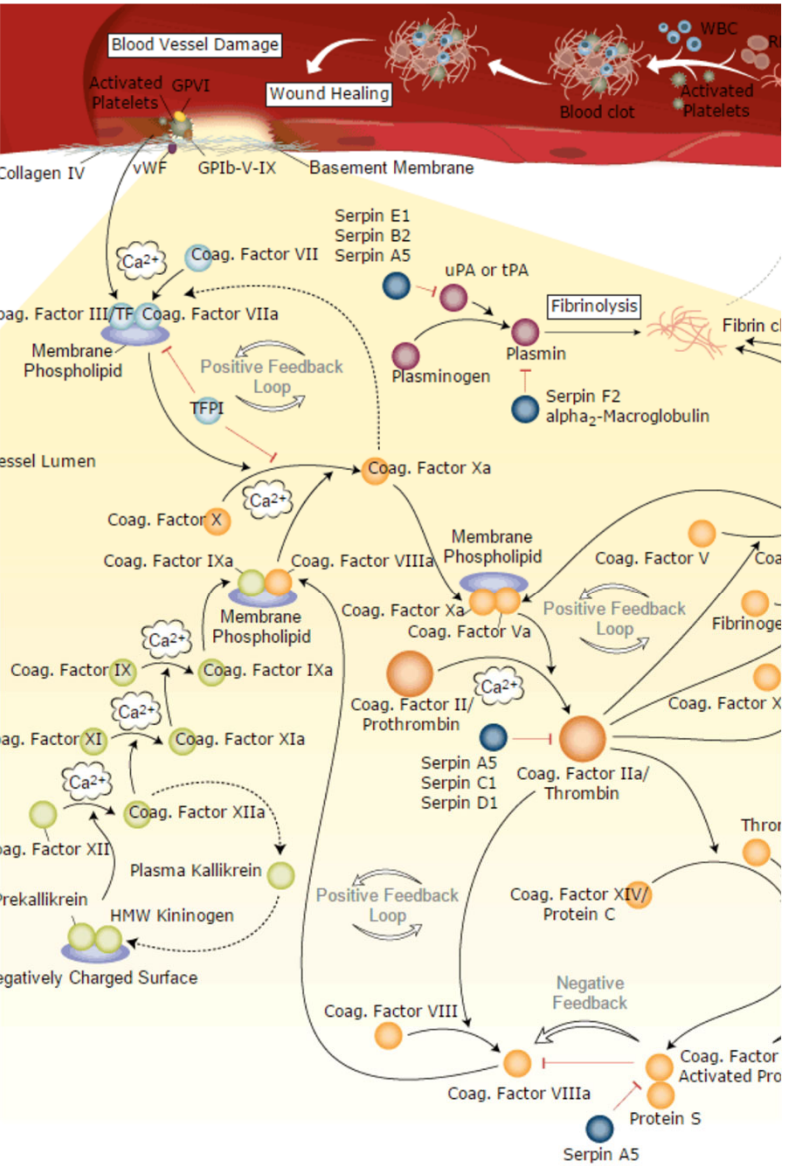
DR conceptually encompasses many types of cell–ECM interactions, which embrace a number of fields of basic and clinical study, including developmental biology and the pathobiology of human disease. Indeed, DR is likely to be relevant during tissue development, reparative and regenerative processes, and human pathogenesis, including embryogenesis, angiogenesis, lactogenesis, cancer, and wound healing. Within each of these basic and clinical research domains, there have been numerous reports that reflect upon the biochemical and biomechanical effects that cells and their interacting surrounding microenvironments or ECM have on tissue or organ-specific responses during human development or disease. Reciprocally, responsive biochemical and mechanochemical interactions between cells and ECM during wound healing and angiogenesis have been demonstrated and discussed by a number of authors.[7–17] These interactions represent integral features of DR,[9,10] which link a deepened understanding or awareness of how cell–matrix interactions modulate cellular responses to injury and wound healing in vivo.

One goal of this review is to explore the relevance of DR as it relates to the wound microenvironment. First, we

CTGF	Connective tissue growth factor
DR	Dynamic reciprocity
EC	Endothelial cells
ECM	Extracellular matrix
FGF	Fibroblast growth factor
GP	Glycoprotein
Hb-EGF	Heparin-binding epidermal growth factor
HSPG	Heparan sulfate proteoglycan
IL	Interleukin
MMP	Matrix metalloprotease
PDGF	Platelet-derived growth factor
TGF	Transforming growth factor
TIMP	Tissue inhibitor of metalloprotease
VEGF	Vascular endothelial growth factor
Vn	Vitronectin
vWF	von Willebrand factor

Wound Rep Reg (2011) 19 134–148

- Proteases are protein-degrading enzymes
- 2 categories of proteases
  - Serine proteases eg. Elastase
  - Matrix metalloproteases eg. MMPs
- Function optimally under physiological conditions
- Collectively, can degrade all soft tissue components of the extracellular matrix
- Normally controlled at the tissue level by natural inhibitors eg. TIMPs, AAT
- Synthesized and stored as inactive pro-enzymes

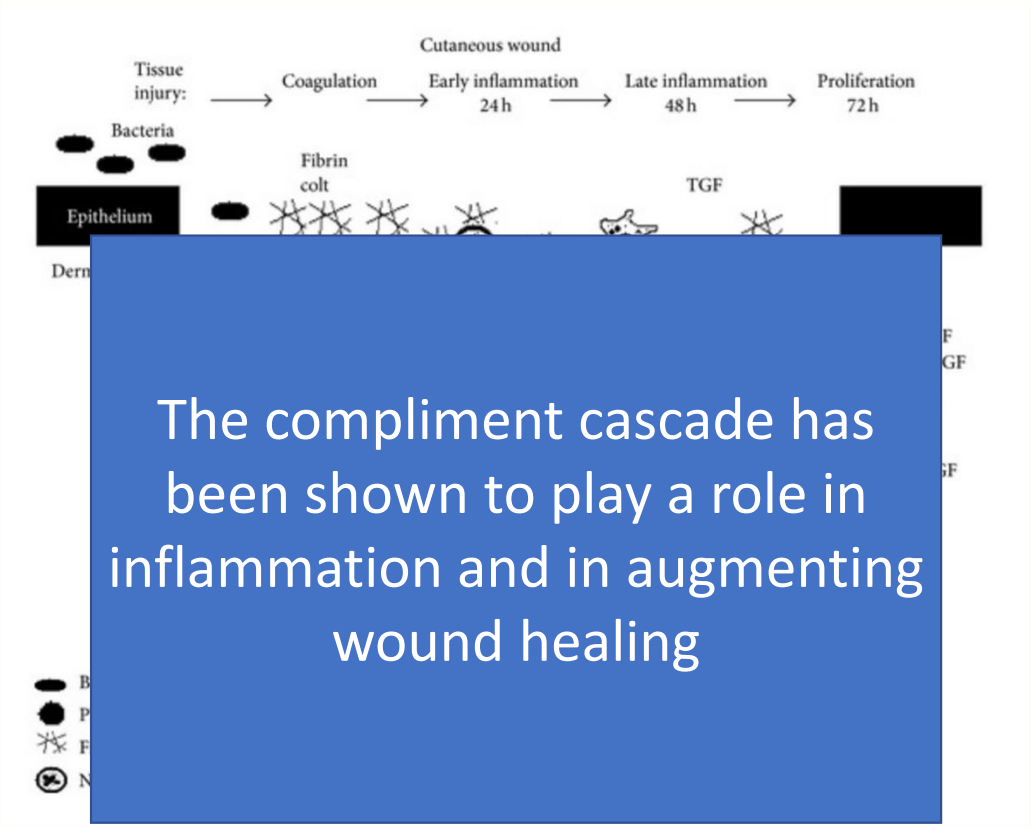


The Clotting Cascade

- Complex
- 13 clotting factors
- After blood vessel damage, the underlying collagen is exposed to circulating platelets
- Platelets bind directly to the collagen creating a ‘platelet-plug’
- Intrinsic pathway= injury pathway
- Extrinsic pathway=everything pathway

The Clotting Cascade Made Easy (2016, Apr 5). Retrieved 2/22/2020

Sinno, H & Prakash, S. (2013) Plast Surg Int. Doi: 10.10155/2013/146764



The compliment cascade has been shown to play a role in inflammation and in augmenting wound healing

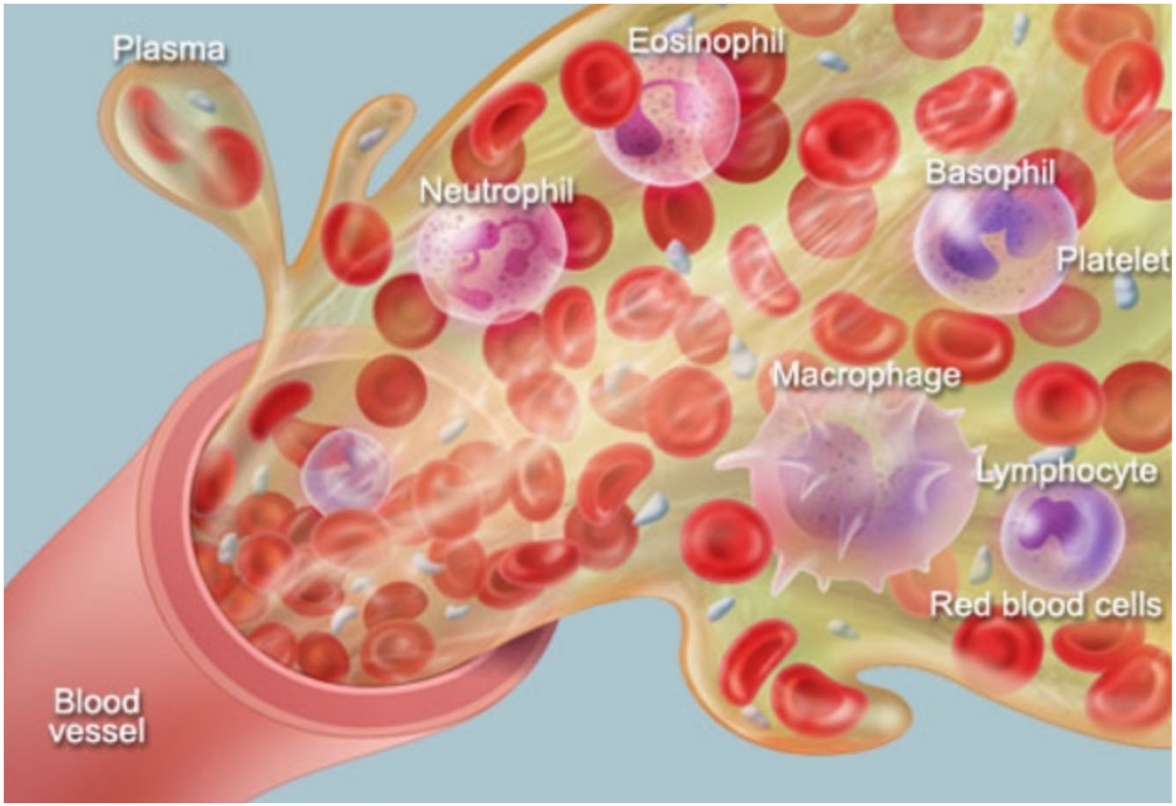
Figure 3

Cutaneous wound healing in time. A schematic representation of cutaneous wound healing and the growth factors and cellular participants in the first 72 hours of injury. The complement cascade appears to be involved in many stages of the wound healing. Platelets, macrophages, fibroblasts, and the formation of the fibrin clot are the major cellular players in early cutaneous, tendon, ligament, muscle, and bone healing.

The Importance of Macrophages

Snyder RJ et al. JAPMA, in press

Components of Blood



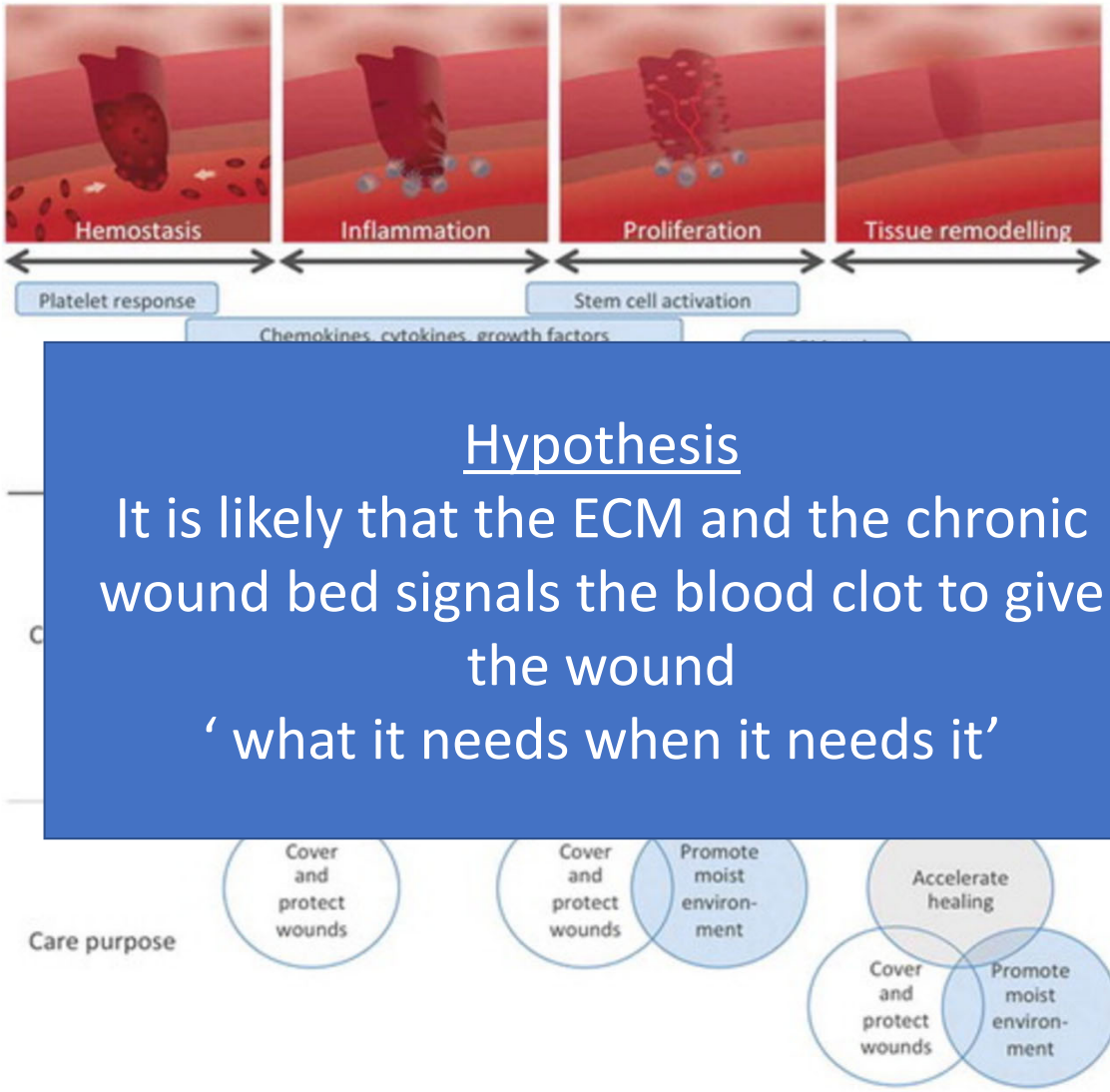
Hoffman, M (2019). Picture of Blood. Webmd.com

It can be hypothesized that autologous blood clot tissue:

- Functions as a tissue sealant and drug delivery system
- Produces ***multiple*** growth factors which stimulate tissue vascularity via an increase in angiogenesis
- Prevents infection via macrophages
- Acts as a scaffold to bolster or replace the ECM
- Fosters wound contraction
- Stimulates pluripotent stem cell recruitment

Lacci KM & Dardik. Yale J Bio Med. 2010 Mar, 83(1): 1-9
Kuhn, DFM et al. Transfus Med Hemother 2006; 33: 307-313
Snyder RJ et al (2020). JAPMA. In press

Lacci KM & Dardik. Yale J Bio Med. 2010 Mar, 83(1): 1-9
Kuhn, DFM et al. Transfus Med Hemother 2006; 33: 307-313
Snyder RJ et al (2020). JAPMA. In press



Hypothesis

It is likely that the ECM and the chronic wound bed signals the blood clot to give the wound

‘ what it needs when it needs it’

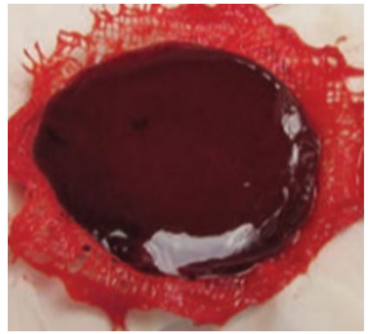


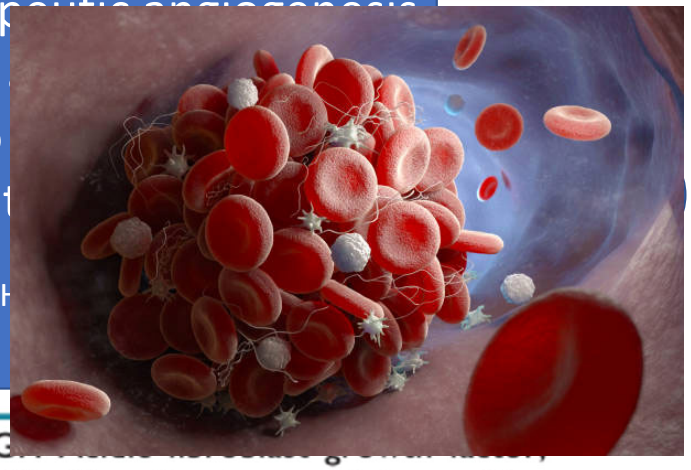
Table 1: Angiogenic stimulators and inhibitors

Stim
aFG
bFG
TGF
TGF
PGE
TNF
VEG
EGF

FGF: Fibroblast growth factor, aFG
bFGF: Basic fibroblast growth factor, TGF-α: Transforming growth factor-alpha, TGF-β: Transforming growth factor-beta, VEGF: Vascular endothelial growth factor, EGF: Endothelial growth factor, PGE2: Prostaglandin E2

The concept of therap
is aimed at locally
factors in excess to
inhibi

Kuhn, DFM et al. Transfus Med H



Kumar, P. Plastic and Aesthetic Research. 2015: 243-248

Hypothesis

Topical blood products have been shown to produce growth factors that could stimulate angiogenesis

Cell and Tissue Based Products

- Autologous (i.e. STSG, EBG, PRP)
- Allogenic (i.e. LSE, Cad., Amniotic)
- Xenographic (i.e. Porcine, Bovine, Bladder, SIS Equine)

Autologous Blood Clot Tissue
Protective covering
Biologic scaffold
Simulates the normal healing cascade
Unlimited resource
Cost effective

Benefits of Autologous therapies
Biocompatibility
Reduced risk associated with disease transmission
Minimal chance of rejection

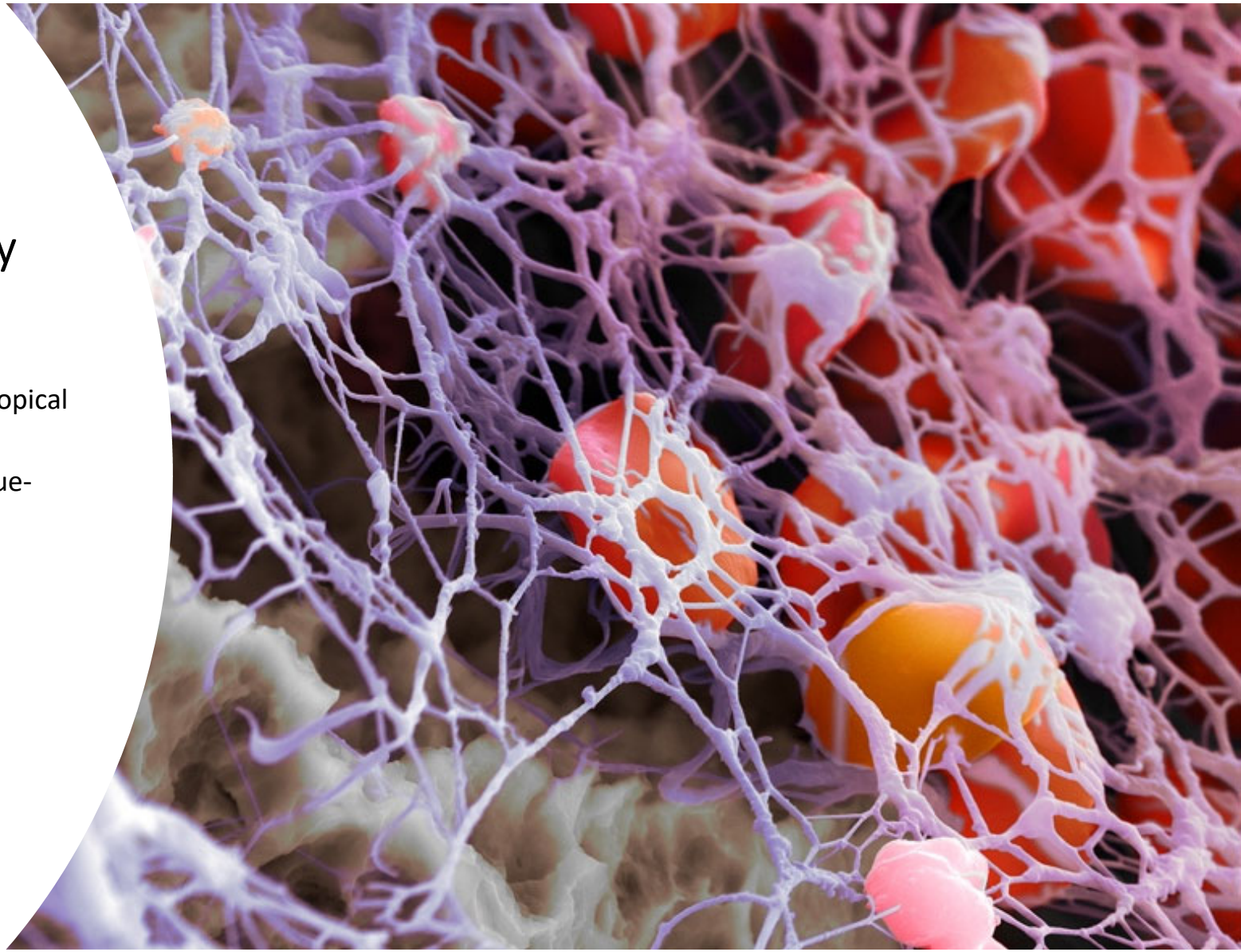
Snyder RJ et al (2020). JAPMA. In press

Electron Microscopy

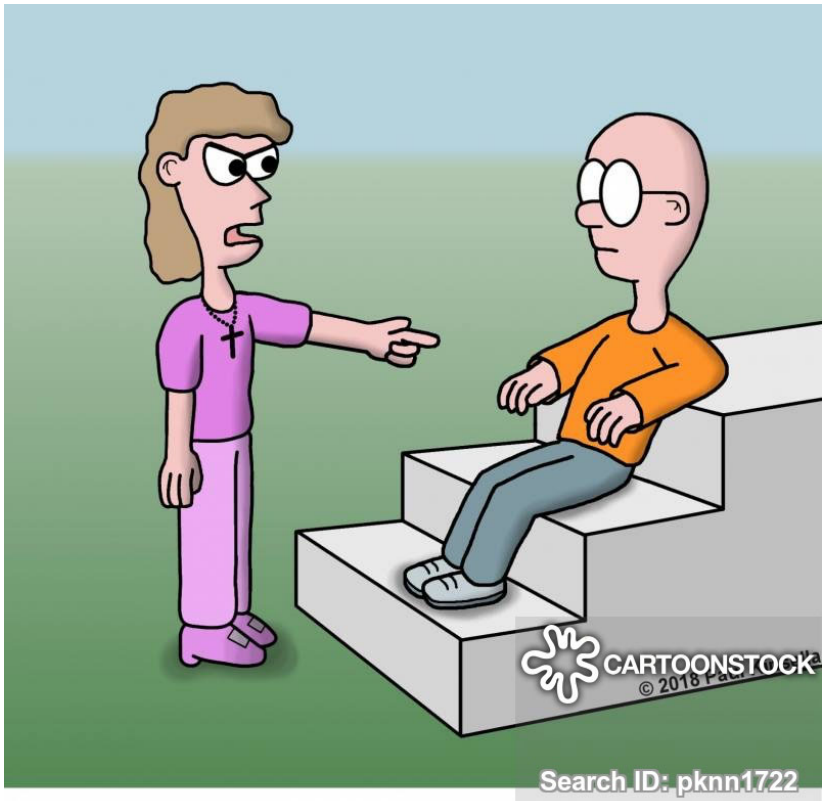
- Stromal matrix observed in topical autologous blood clot tissue
- Represents true cell and tissue-based therapy that allows proliferation of cells
- Creates wound contraction

From: Oeggerli, M, with permission

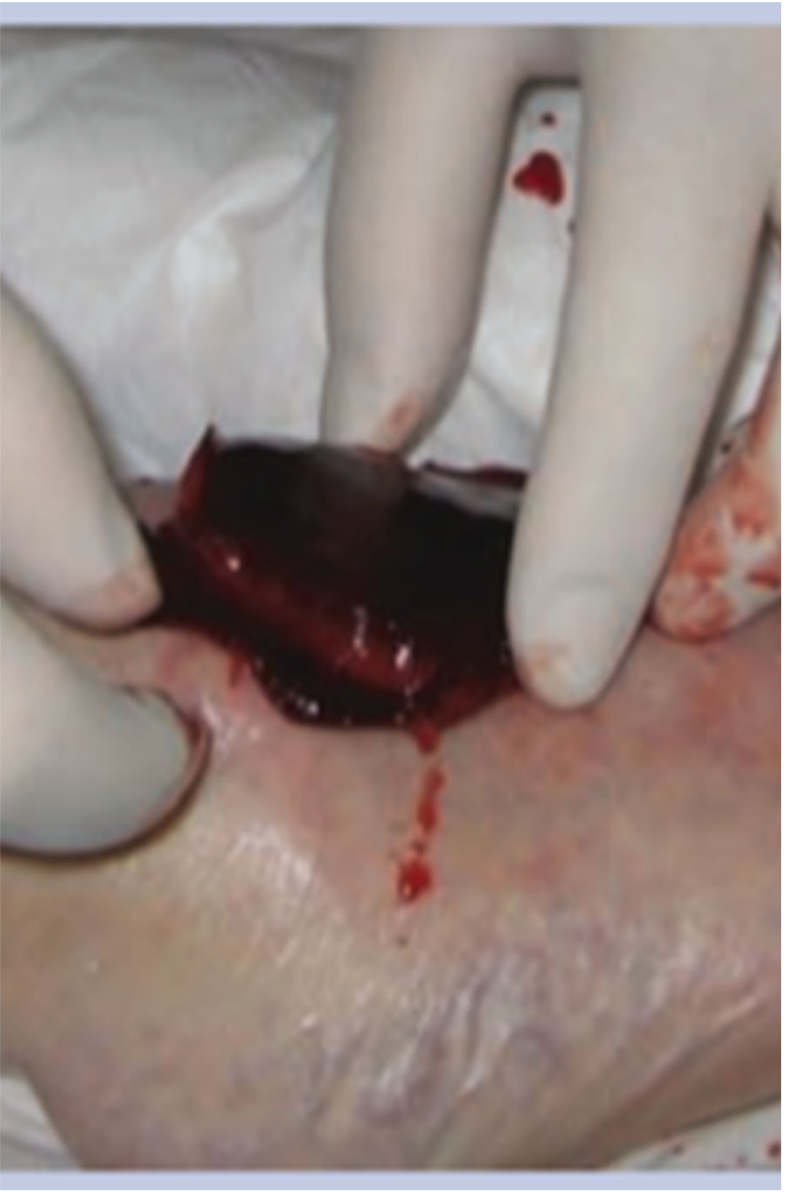
Snyder RJ et al (2020). JAPMA. In press



Current Evidence



"I have a right to disagree!
You can't force me to use logic."



Efficacy and Safety of a Novel Autologous Wound Matrix in the Management of Complicated, Chronic Wounds: A Pilot Study

Kushnir I et al (2016). Wounds. 28(9):317-327

- **Results:** Seven patients with multiple and serious comorbidities and 9 chronic wounds were treated with 35 clot matrices.
- Complete healing was achieved in 7 of 9 wounds (78%).
- No systemic adverse events occurred.
- **Conclusions:** This pilot study demonstrates the in vitro autologous whole blood clot matrix is effective and safe for treating patients with chronic wounds of different etiologies
- A larger clinical trial is needed

Wounds. 2018 Jul;30(7):84-89. Epub 2018 Apr 20.

Safety and Efficacy of an Autologous Blood Clot Product in the Management of Texas 1A or 2A Neuropathic Diabetic Foot Ulcers: A Prospective, Multicenter, Open Label Pilot Study.

Snyder RJ, Kasper MA, Patel K, Carter MJ, Kushnir I, Kushnir A, Serena TE.

Abstract

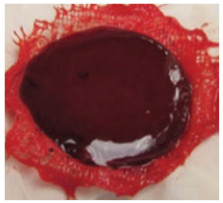
OBJECTIVE: This pilot study evaluates safety
as the efficacy in terms of complete wou
neuropathic diabetic foot ulcers (DFU

MATERIALS AND METHODS: Partic
clinic. Up to 10 mL of blood drawn
Within 12 minutes, the blood clot pr
covered with primary and secondary
applications every 5 to 9 days for up to

RESULTS: Twenty patients were enrolled; 2
were in the per-protocol (PP) population. Thirty-two
The mean AE rate for both the ITT and PP populations was
healed in the ITT and PP populations was 13 out of 20 (65%) and 13 out of 18 (72.2%), respectively. Percentage area reduction (PAR) for the ITT population at 4 and 12 weeks was 61.6% and 67.1%, respectively; the PARs for the PP population were 60.3% and 76.2% at 4 and 12 weeks, respectively. Mean times to wound healing were 59 days and 56 days in the ITT and PP populations, respectively.

CONCLUSIONS: This study demonstrates that the blood clot product is safe and efficacious for treating DFUs.

A pivotal randomized controlled trial comparing autologous blood clot tissue to standard of care (Debridement, CAM Walker and foam) is currently ongoing



Summary of Results

20 patients enrolled

Proportion of patients healed over 12 weeks

ITT: 13/20 (65%)

PP: 13/18 (72.2%)

Percent area reduction:

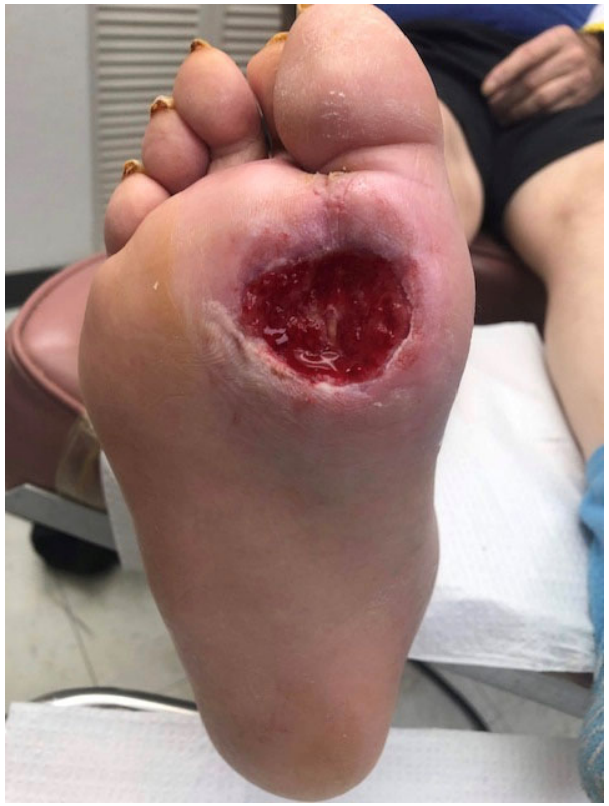
ITT at 4 and 6 weeks: 61.6%/67.1%

PP at 4 and 12 weeks: 60.3%/76.2%

Mean times to wound healing:

ITT: 59 days

PP: 56 days



Analysis of Three Prospective, Open-Label, Clinical Trials with Autologous Blood Clot in Chronic Wounds

Donner B., DO, Patel K,; DO, Serena Thomas E., MD

Background: The RD1 (RedDress Ltd.) was cleared by FDA in July 2018, for creating autologous blood clots for management of cutaneous wounds, was the subject of three prospective studies. Data from the three studies was analyzed and results are reported.

Tools and Method: All studies were approved by IRB. All studies were monitored by 3rd party CRO. Studies were conducted in 5 centers: 3 wound care clinics and 2 specialized nursing facilities (4 USA, 1 Israel). Wounds were treated weekly with RD1 for up to 12 weeks.

Results: Total of 42 subjects enrolled, number of subjects and their wound was: 20 DFUs, 9 PUs, 7 VLUs, 6 Skin Tears (ST). 42 subjects were analyzed for the ITT (intent-to-treat) population, including all patients that did not complete more than 1 treatment or withdrawn. The PP (per protocol) population that completed at least 4 weeks of treatment included 38 subjects. The proportion of wounds completely healed in the ITT populations was 27/42 (64%) and 27/38 (71.1%) for the PP population. Percentage area reduction (PAR) for the ITT population at 4 and 12 weeks was 62% and 79.%, respectively.

Conclusion: The analysis is consistent with the efficacy results of the RD1 in literature, demonstrating the high effectiveness of healing chronic ulcers with the use of Autologous blood clot.

References:

[1] Kushnir I, Serena T, Garfinkel D, Efficacy and Safety of a Novel Autologous Wound Matrix in the Management of Complicated, Chronic Wounds: A Pilot Study. WOUNDS ;28(9):317-327.

[2] RJ, Snyder, MA. Kasper, K Patel, MJ. Carter, I Kushnir, A Kushnir, TE. Serena. Efficacy of an Autologous Blood Clot Product in the Management of Texas 1A or 2A Neuropathic Diabetic Foot Ulcers: A Prospective, Multicenter, Open Label Pilot Study. Wounds 2018;30(7):205–212.

[3] Razzell W, Wood W, Martin P. Swatting flies: modelling wound healing and inflammation in Drosophila. Dis Model Mech. 2011;4:569–574.

[4] Laurens N, Koolwijk P, de Maat MP. Fibrin structure and wound healing. J Thromb Haemost. 2006;4(5):932–939.

[5] Tonnesen MG, Feng X, Clark RA. Angiogenesis in wound healing. J Investig Dermatol Symp Proc. 2000;5(1):40–46.

Summary of Results

Analyzed 3 prospective, open labeled trials utilizing autologous blood clot tissue

Demonstrated efficacy of healing acute and chronic wounds both in vitro and in vivo

PREP AND APPLICATION

Step 1: Blood Draw

- Standard phlebotomy procedure
- Blood can be stored in ambient temp for up to 24h
- Blood draw via vacu-tubes (18 mL)

Step 2: Clot Preparation

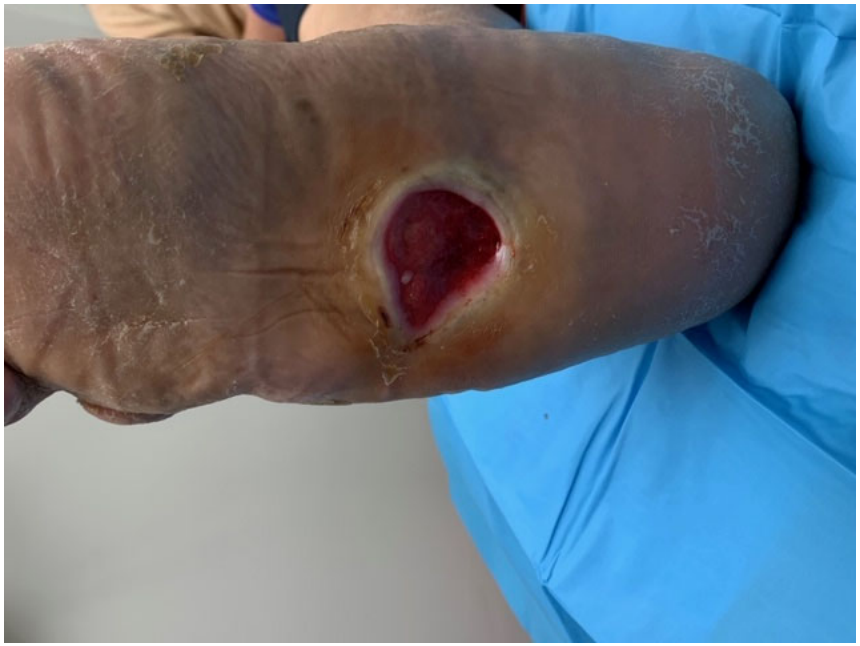
- Clotting time averages 10 mins, slightly longer for patients on blood thinners
- Clot is 6 cm diameter, but may be cut for smaller or tunneling wounds

Step 3: Clot Application

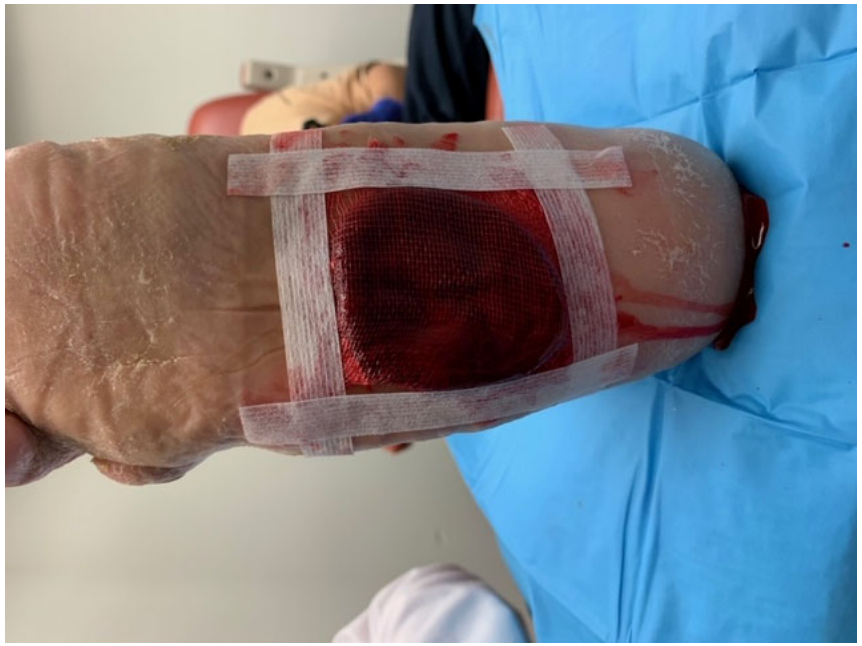
- Clot is applied in a “clean technique”
- Clot is stabilized by a gauze matrix on the outward facing side
- Secured with steri-strips, covered with foam bandage and over-wrapped with gauze

Application of topically applied blood clot tissue to the plantar foot ulcer in a patient with diabetes

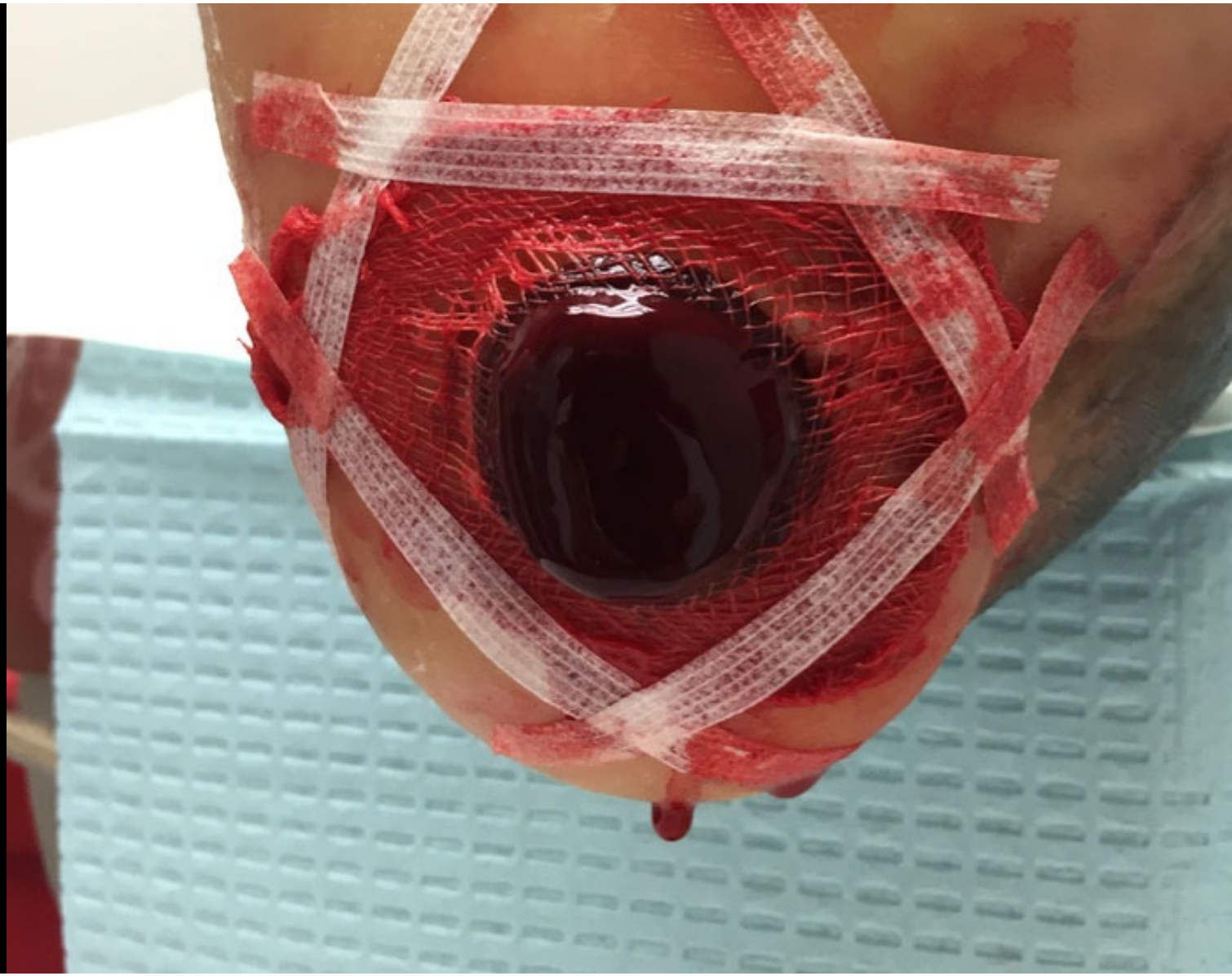
Pre-application



Post application



Application of topically applied blood clot tissue to a plantar heel ulcer in a patient with diabetes



CASE STUDY

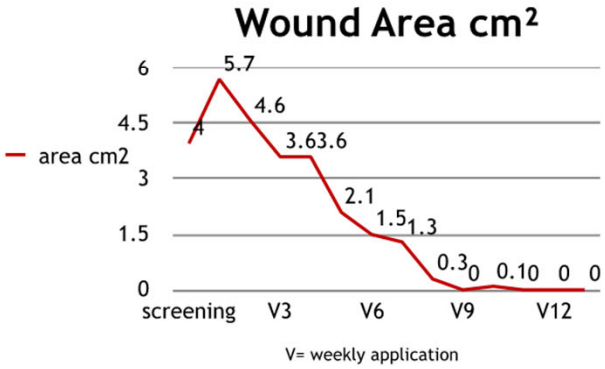
- **Patient:** 77 year old white male
- **PMH:** DM2, Neuropathy, PVD, Gout, Afib, HTN, HPL , OA, History of smoking/15 pack years
- **Labs:** HbA1C 9.7, WBC 10.8, RBC 3.61, HgB/HCT 10.3/31.7, MCV 79.8, MCH 25.8, RDW 19.8, PT 14.7

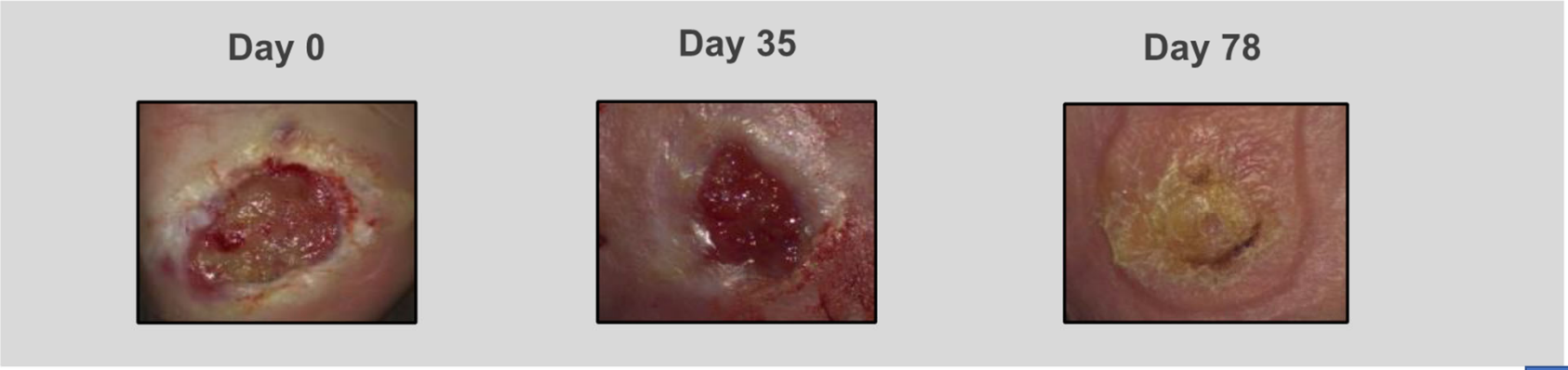
Wound History:

- Diabetic Ulcer 2A, Right heel, onset
- 30 week old ulcer
- 3.96cm2 x 0.5 cm, moderate amounts of serous/sanquinous drainage
- ABI Results for the affected leg =1.11

Past Treatments: wet-to-dry, foam, silver alginate, Anbx agents , HBO, skin graft, skin substitute, regular surgical debridement

Offloading: boot, TCC





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CASE STUDY

- **Patient:** 98 year old white male
- **PMH:** CHF, Afib (long term coumadin), Stage 4 right heel, chronic osteomyelitis right ankle and foot, Venous Stasis Disease, Vit d def, Malnourished, Anemia.

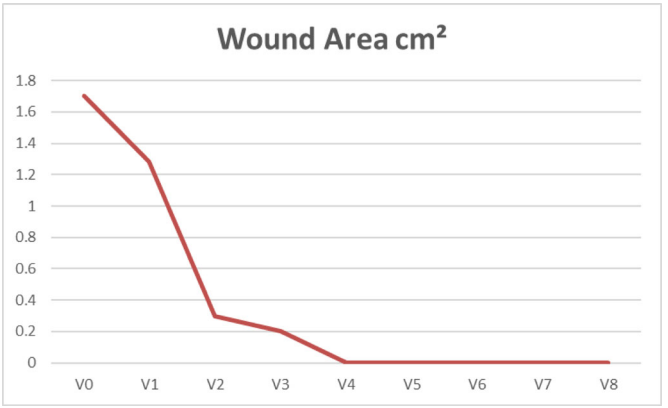
- **Labs:** (12.4.18) H/H 8.9/31.5, albumin 2.4

Wound History:

- Admitted after hospitalization for L femur fracture
- 8 month old ulcer
- Boggy heels assessed upon admission
- DTI injury developed into a Stage 4 with exposed bone
- MRI showed osteomyelitis
- ID consult with no further Anbx recommended

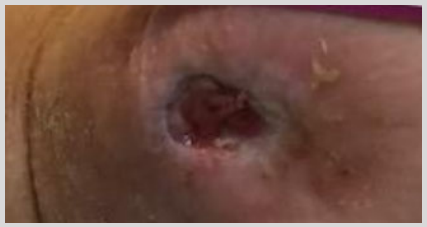
Past Treatments: Santyl and mesalt, VAC, optifoam, puracol, iodoform and dry dressing with crushed Flagyl for odor, VAC

Offloading: heel offloading boots worn 24/7, he was up in his W/C most of the day

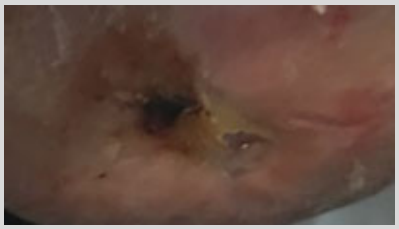


V= weekly application

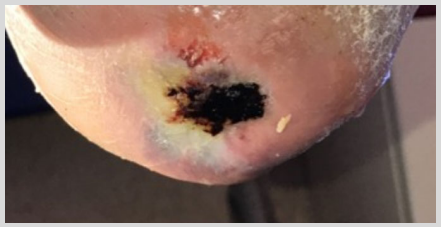
Day 1



Day 13



Day 19



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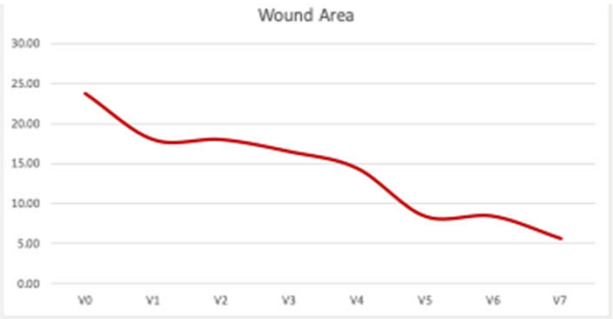
PATIENT:
67 year-old, female.

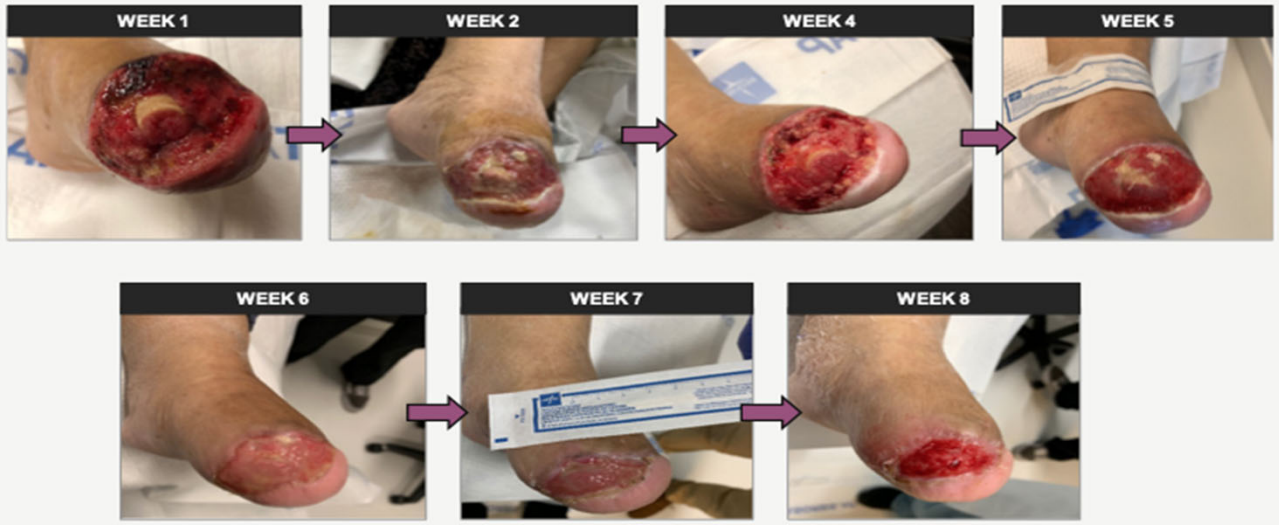
WOUND HISTORY:
- ESRD
- DM2
- PVD
- Gangrene of the great toe
- Failed ray amputation an TMA
- Refused BKA



PAST TREATMENTS:
Wound Vac & HBO

OFFLOADING:
N/A

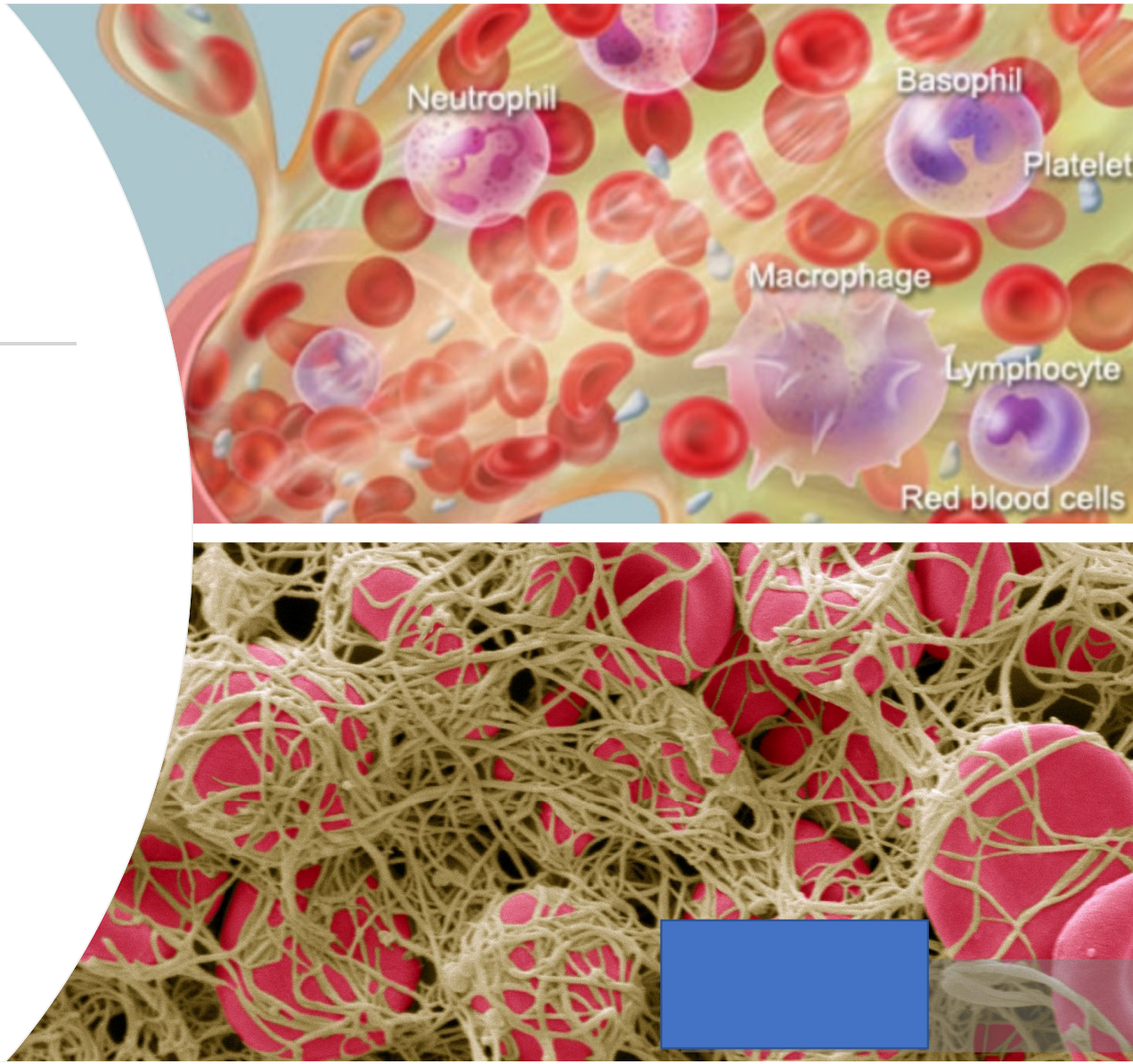
TREATMENT PROGRESSION:



Case courtesy of Dr. Naz Wahab

Summary

- Topically applied autologous blood products augment healing
- Stimulate growth factor production
- Mitigate infection
- Bolster of replaces the ECM
- Evidence-based
- Easy to use
- Cost effective



Thank You

Questions?