

BIONICA-MDI Therapy as the Novel Therapeutic Strategy in the Management of Complex Diabetic Foot Ulcers (DFUs): A Case Report of a Salvaged Leg

Dr. Pradeep Mahajan¹, Dr. Harsadbhai D Patel², Dr. Anilkumar Daxini³, Dr. Masira Khan⁴, Marilyn Dsouza⁵

¹Professor in Surgery, Faculty in Regenerative Medicine Maharashtra University (MUHS) & Consultant in Regenerative Medicine & ABRM, StemRx Biosciences Solutions, Pvt Ltd, India

²Consultant & Gastroenterologist, MDI, Houston, United States of America

³Consultant Physician, StemRx Biosciences Solutions, Pvt Ltd, & Fortis Hospital, India

⁴Head of Department, Quantum Energy Medicine, StemRx Biosciences Solutions, Pvt Ltd, India

⁵Medical Writer, StemRx Biosciences Solutions, Pvt Ltd, India

***Corresponding Author:** Dr. Pradeep V. Mahajan, Professor in Surgery, Faculty in Regenerative Medicine Maharashtra University (MUHS) & Consultant in Regenerative Medicine & ABRM, StemRx Biosciences Solutions, Pvt Ltd, India. Email: drpvmahajan@gmail.com

Abstract:

Background & Objective: This case report presents an 80-year-old male with a chronic, non-healing diabetic foot ulcer (DFU) with severe peripheral neuropathy, peripheral vascular disease, Charcot joint and osteomyelitis, for whom amputation was recommended.

Methods: The patient was treated with a comprehensive protocol that included wound debridement, infection control, antibiotics, metabolic stabilization, and BIONICA-MDI. 45 BIONICA-MDI sessions were administered across 4 months, alongside growth factors, peptides, and antibiotics.

Results: A significant clinical improvement was observed with, new osteogenesis, chondrogenesis in Charcot joint deformities and new vascularization, transmitting into resolution and infection and wound healing.

Conclusion: This case highlights the potential of BIONICA-MDI therapy in managing complex diabetic foot, Charcot joint and deep necrotic ulcer disease, underscoring its promise as an adjunct to conventional diabetic wound care strategies, waiting for amputation as fate.

Keywords: Diabetic Foot ulcers (DFUs), Diabetic Neuropathy, BIONICA-MDI, Diabetes Mellitus, Charcot joint

1. INTRODUCTION

The spectrum of diabetic foot ulcer (DFU) ranges from cellulitis to a full-grown gangrene. The four major causes are peripheral neuropathy, autonomic neuropathy, ischemia, nutritional failure, and infection [1]. Fifteen percent of all diabetic patients suffer from DFU causing major morbidity, mortality, and financial burden [2], [3].

Newer therapies are vacuum, wound dressing, hyperbaric oxygen therapy, and growth factor formulations [3],[4],[5],[6]. Mitochondrial dysfunction has been identified as an essential

pathophysiological procedure contributing to nerve injury in diabetic neuropathy [7]. BIONICA-MDI treatment improves mitochondrial functions.

2. METHODS

2.1. Case Processing

An 80-year-old male patient with history of Diabetes Mellitus, Hypertension, and ischemic heart disease; status post percutaneous transluminal coronary angioplasty (PTCA). Patient also has a history of Peripheral Vascular Disease S/A right great toe amputation (17

years), severe axonal sensorimotor neuropathy affecting both lower limbs, and chronic kidney disease presents with diabetic foot disease since several months. Patient was advised amputation of feet; however, he was unwilling for the same.

Patient came to StemRx Biosciences Solutions Pvt Ltd, Navi Mumbai; for further treatment. Cultures of soft tissue and bones were taken. Patient was treated with wound debridement, antibiotics. Sugar was controlled with insulin and oral drugs. Patient was also started on BIONICA-MDI treatment additionally. Patient's stool occult blood was positive for which he underwent upper GI-scopy and colonoscopy with showed gastritis negative for *H. Pylori*. Patient was also treated with intravenous ferric carboxymaltose.

MRI foot report showed destruction of navicularis, cuboid, cuneiform bones with significant irregularity and destruction involving part of talus, and base of 1st and 2nd metatarsal. Extensive soft tissue swelling and thickening involving subtalar, talonavicular and inter-tarsal joints, which insinuating into joint spaces and adjacent tissue. Intramedullary extension of the collection into 1st metatarsal. Marrow edema noted involving the tarsal and proximal metatarsal with extensive surrounding soft tissue and subcutaneous edema. Bone remodeling

noted involving the proximal aspect of metatarsal, head of 1st metatarsal with diffusely reduced bone mineralization.

Tendoachilles insertional ossification. Hyperintensity involving plantar fascia suggesting plantar fasciitis at attachment of calcaneum. Fluid noted along peroneal tendon and tibialis posterior. There is associated subluxation of multiple tarsometatarsal joint. Visualized anterior body of talus shows T1 hypo intensity representing bone necrosis. Heterogenous signal involving metatarsal shafts diabetic foot/ neuropathic Charcot deformity.

2.2. Treatment Protocol

Patient received a total of 45 BIONICA-MDI sessions over a period of 4 months. By targeting mitochondrial function, BIONICA-MDI therapy improves energy metabolism at the injury site, mitochondrial function, reduces inflammation, and accelerates healing. Alongside, patient was given growth factors, peptides, anti-biotics and rehabilitation programs.

3. RESULTS

Patient's wound gradually improved, discharge decreased, ulcer healed up completely with residual scarring. Also, patient had fever regularly which resolved completely.



Fig 1. Diabetic Foot ulcer (DFU)



Fig 2. *During BIONICA-MDI treatment effect on DFU*



Fig 3. *Effect of BIONICA-MDI in wound healing*



Fig 4. *Post treatment of a Diabetic healed foot*

DISCUSSION

“No evidence glucose-lowering drugs help ward off long term complications” [8].

BIONICA-MDI mimics normal pancreas stimulation of the liver (with insulin); which is essential for the release of enzymes that mediate the adenosine triphosphate (ATP) production for each organ. It normalizes carbohydrate and lipid metabolism. It stops and reverses complications by reducing the damage from high lipid metabolism & restoring missing cellular energy.

Pulsatile Insulin has a greater hypoglycemic effect, maintains peripheral insulin receptor activity and peripheral glucose uptake and upregulates metabolic enzymes of glycolysis and the Krebs cycle.

Mitochondria play a central role in regulating energy metabolism. In metabolic syndrome and insulin resistance, mitochondrial dysfunction contributes significantly to the underlying pathology, particularly through impaired energy production, excess reactive oxygen species (ROS), and altered lipid metabolism [9].

In diabetes mellitus mitochondria are unable to function optimally. This alters their ability to respond to external stressors and distorts the mechanisms that maintain cell viability [10].

Mitochondrial dysfunction has emerged as a pivotal factor contributing to the complexity of diabetic complications [11].

The intricate relationship between these pathophysiological pathways and mitochondrial health highlights the necessity of addressing mitochondrial dysfunction in therapeutic strategies for diabetic peripheral neuropathy [10]; [12]. For diabetic patients, this metabolic disease leads primarily to peripheral neuropathy. The rising prevalence of diabetic neuropathy impacts quality of life; including pain, disability, and increased risk of amputations; necessitate effective treatment [13].

A key component of this treatment was the use of the BIONICA-MDI, which greatly enhanced cellular repair and tissue regeneration. BIONICA-MDI therapy boosted mitochondrial function, reducing inflammation and speeding up healing. Its ease of administration also supports greater patient compliance and more effective healthcare delivery.

4. CONCLUSION

The integration of BIONICA-MDI therapy in the management of this complex diabetic foot case provided a significant turning point in the patient’s recovery. BIONICA-MDI therapy, conventional wound care and systemic management, facilitated tissue regeneration,

infection control, and functional improvement; ultimately avoiding amputation. This case highlights the potential of BIONICA-MDI as a therapeutic strategy in managing chronic, non-healing wounds, particularly in high-risk, diabetic patients with multiple comorbidities.

INFORMED CONSENT

Consent from the patient was obtained prior to the treatment and for necessary images.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

ACKNOWLEDGEMENT

The authors thank BIONICA-MDI, Dr. Harsadbhai D Patel, Houston (Patent Issued) for his contribution with the treatment and in literature. We also thank all of the staff and colleagues who have helped in contributing with the treatment.

REFERENCES

- [1] Murphy-Lavoie HM, Ramsey A, Nguyen M et al (2025). Diabetic Foot Infections. StatPearls.
- [2] Zhang P, Lu J, Jing Y et al (2017). Global epidemiology of diabetic foot ulceration: a systematic review and meta-analysis. *Ann Med* 49:106-116. doi: 10.1080/07853890.2016.1231932
- [3] Akkus G, Sert, M (2022) Diabetic foot ulcers: A devastating complication of diabetes mellitus continues non-stop in spite of new medical treatment modalities. *World journal of diabetes* 13(12), 1106–1121. doi: 10.4239/wjd.v13.i12.1106
- [4] Harries RL, Harding KG (2015). Management of diabetic foot ulcers. *Curr Geriatr* 4:265–276. doi:10.7454/jki.v21i2.634
- [5] Chen CE, Ko JY, Fong CY et al (2010) Treatment of diabetic foot infection with hyperbaric oxygen therapy. *Foot Ankle Surg* 16:91–95. doi: 10.1016/j.fas.2009.06.002
- [6] Martí-Carvajal AJ, Gluud C, Nicola S et al (2015) Growth factors for treating diabetic foot ulcers. *Cochrane Database Syst Rev*. CD008548. doi: 10.1002/14651858.CD008548.pub2
- [7] Sriwastawa K, Kumar A (2024). Mitochondrial dysfunction in diabetic neuropathy: Impaired mitophagy triggers NLRP3 inflammasome. *Mitochondrion*. 101972, Volume 79, ISSN 1567-7249. doi: 10.1016/j.mito.2024.101972
- [8] Crowe K (2016) New study questions Type 2 diabetes treatment. *CBC News*.
- [9] Hellman B (2009) Pulsatility of insulin release-- a clinically important phenomenon. *Upsala journal of medical sciences* 114(4), 193–205. doi: 10.3109/03009730903366075
- [10] Espinoza N, Papadopoulos V (2025) Role of mitochondrial dysfunction in neuropathy. *International Journal of Molecular Sciences*. 26(7), 3195. doi: 10.3390/ijms26073195
- [11] Yaribeygi H, Atkin SL, Sahebkar A (2019) Mitochondrial dysfunction in diabetes and the regulatory roles of antidiabetic agents on the mitochondrial function. *Journal of cellular physiology* 234(6), 8402–8410. Doi: 10.1002/jcp.27754
- [12] Bai R, Luo Y (2024) Exploring the role of mitochondrial-associated and peripheral neuropathy genes in the pathogenesis of diabetic peripheral neuropathy. *BMC Neurology* 24(1). doi: 10.1186/s12883-024-03589-0
- [13] Yang Y, Zhao B, Wang Y et al (2025) Diabetic neuropathy: cutting-edge research and future directions. *Sig Trans- duct Target Ther* 10, 132. doi: 10.1038/s41392-025-02175-1

Citation: Dr. Pradeep Mahajan, et al. *BIONICA-MDI Therapy as the Novel Therapeutic Strategy in the Management of Complex Diabetic Foot Ulcers (DFUs): A Case Report of a Salvaged Leg*. *ARC Journal of Diabetes and Endocrinology*. 2025; 10(1):18-22. DOI: <https://doi.org/10.20431/2455-5983.1001004>.

Copyright: © 2025 Authors. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.