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Multidisciplinary Integrative Approach for Non- Healing Ulcer in Chronic SCI

Sane HM[1], Sayyed HS[2], Hamdule TT[1*], Khubchandani PV[2], Kulkarni PP[1], Gokulchandran N[3] and Badhe PB[4] and Sharma AK[3]

[1]Department of Research and Development, NeuroGen Brain and Spine Institute, Navi Mumbai, Maharashtra, India
[2]Department of Integrative therapy, KLS wellness and NeuroGen Annexe Hospital, Navi Mumbai, Maharashtra, India
[3]Department of Medical Services, NeuroGen Brain and Spine Institute, Navi Mumbai, Maharashtra, India
[4]Department of Regenerative Laboratory, NeuroGen Brain and Spine Institute, Navi Mumbai, Maharashtra, India

[*]**Corresponding author:** Hamdule TT, Research Associate, Department of Research and Development, NeuroGen Brain and Spine Institute, Plot 19, Sector 40, Nerul West, Navi Mumbai 400706, Maharashtra, India, Tel: +9122-41136565; E-mail: publications@neurogen.in

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Abstract

Background: Spinal cord injury (SCI) often leads to severe complications which includes non-healing Stage 4 decubitus ulcers due to immobility, impaired sensation, and poor tissue perfusion. These chronic wounds significantly increase patient morbidity and strain healthcare resources, with traditional management often yielding limited success.

Case Presentation: In this study, we present the case of a 26-year-old male with quadriplegia secondary to an incomplete C5-C6 SCI with chronic advanced Stage 4 sacral and Stage 4 bilateral trochanteric pressure ulcers. He was treated with integrative multidisciplinary regimen including Hyperbaric Oxygen Therapy (HBOT), Ozone Therapy, Platelet-Rich Plasma (PRP) therapy, Intravenous (IV) Micronutrient supplementation, deep tissue mobilization, enema-based gut cleansing, structured neurorehabilitation and intrathecal administration of autologous Bone Marrow Derived Mononuclear Cells (BMMNCs).

Results: Clinical progress was evaluated at 4, 10, 13 and 24 months using ASIA, SCIM, FIM, EPUAP, PUSH and SPSSS scores. The patient demonstrated progressive and complete ulcer healing at sacral and Right trochanteric region by 13 months, with PUSH tool 3.0 scores improving from 16 to 0 and 17 to 2 respectively, EPUAP and SPSSS grading indicating complete resolution at both the regions. Functional improvements included enhanced posture, increased sitting tolerance, absence of postural hypotension, and partial independence in daily activities. No adverse effects were reported throughout the intervention period.

Conclusion: This case illustrates the potential benefits of combining multidisciplinary integrative and rehabilitative treatments along with administration of autologous BMMNCs to enhance wound healing and functional recovery in SCI patients with chronic pressure ulcers, offering an effective alternative to conventional wound management strategies.

Keywords: ***Pressure ulcer; Integrative therapy; PRP; Spinal cord injury***

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1. Abbreviations

SCI: Spinal cord injury; HBOT: Hyperbaric Oxygen Therapy; PRP: Platelet-Rich Plasma Therapy; BMMNCs: Bone Marrow Derived Mononuclear Cells; RTA: Road Traffic Accident; FIM: Functional Independence Measure; SCIM: Spinal Cord Independence Measure; ASIA: American Spinal Injury Association; PUSH Tool 3.0: Pressure Ulcer Scale for Healing; SPSSS: Stirling Decimal Based Pressure Ulcer Scale; IV Micronutrient: Intravenous Micronutrient MNCs: Mononuclear Cells; G-CSF: Granulocyte Colony Stimulating Factor; EPUAP: European Pressure Ulcer Advisory Panel

2. Introduction

Spinal cord injury (SCI) results in loss of motor and sensory functions of the limb and body which further affects the quality of life of the patient. Recovery from SCI is difficult since the injured spinal cord loses its ability to regenerate lost or damaged cells and re-establish neural connections leading to severe functional impairments. One of the significant complications in SCI is the occurrence of non-healing decubitus ulcers. Development of these non-healing or decubitus ulcers in SCI can be attributed to a number of factors, mainly impaired sensation, lack of mobility, prolonged pressure, friction, accumulation of moisture which can lead to reduced blood flow, tissue necrosis and localized ischemia. These ulcers commonly develop in the sacral region, left or right trochanteric region, back, heels, glutes, etc. [1].

Decubitus ulcer healing is a complicated process that involves the interaction of several cell types, growth factors, and extracellular proteins to repair the damage to the skin's dermal and epidermal layers. Sometimes, the tissue's repair system is unable to cure the ulcers on time [2]. Non-healing wounds and sores can pose a medical challenge, especially when motor as well as sensory nerves are affected as in the case of spinal cord injury [3]. Recurrent hospitalizations, multiple surgeries are some challenges that prove to be devastating for the patients and caregivers [4].

The conventional treatment for pressure ulcers in SCI patients can be non-operative local wound care (antiseptic solutions, ointments, creams, dressings, topical or mechanical debridement, and electrical stimulation, nutrition, oral antibiotics) and surgery (surgical debridement, direct wound closure, skin grafts, and skin, fasciocutaneous, or myocutaneous flaps). While Stage III and IV pressure ulcers necessitate surgical interventions, Stage I and II ulcers typically require non-operative wound management [4].

Hyperbaric oxygen therapy (HBOT), ozone therapy and cell therapy are some new treatment strategies that have shown promise in enhancing the healing of decubitus ulcers and improving symptoms in patients with spinal cord injuries. HBOT, by providing high oxygen concentrations under pressure, increases oxygen diffusion into ischemic tissues, reducing edema, promoting neovascularization, and supporting collagen synthesis, which accelerates ulcer healing [5,6].

Ozone therapy, with its anti-inflammatory, antimicrobial and oxygenating effects, can further enhance wound healing by improving local oxygen delivery, modulating immune responses, and stimulating antioxidant defense [7]. When combined, these therapies work synergistically to improve both ulcer healing and overall cellular environment, offering a supportive approach in management of spinal cord injury.

Here, we report a case of a patient with incomplete spinal cord injury who developed a sacral pressure sore 15 days post injury and bilateral greater trochanteric sores 5-6 months post SCI who underwent multidisciplinary integrative therapy combined with rehabilitation and cell therapy, demonstrating rapid healing of his decubitus ulcer along with functional improvements.

3. Case Report

We describe a case of a 26-year-old male diagnosed with quadriplegia due to incomplete C5-C6 traumatic spinal cord injury following a Road Traffic Accident (RTA) who received multidisciplinary integrative therapy combined with rehabilitation and autologous BMMNC treatment 2 years later to the accident. He was admitted to the hospital following the RTA and had no history of seizures, emesis, or unconsciousness. Three days following the accident, decompression and stabilization surgery was performed at C4-C6 level. The recommended rehabilitation was done post-surgery. The sacral pressure sore developed after 15 days and bilateral trochanteric pressure sores developed at around 6 -7 months (FIG. 1).

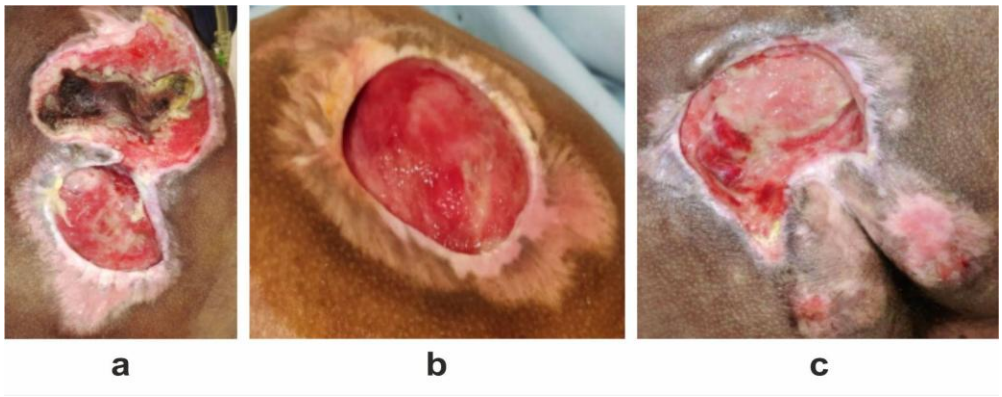

FIG. 1. **Photographic evidence of bed sore before treatment. a. right trochanteric bed sore; b. left trochanteric bed sore; c. sacral bed sore.**

Prior to treatment, the patient was presented with bilateral lower-limb weakness and stiffness and was completely bedridden. He was entirely reliant on his caregiver for all daily activities and transitions between positions. He was unable to write, eat, or hold objects due to poor hand function. Bladder and bowel sensation and control were impaired. Stage 4 pressure sores were noted over the sacral area and bilateral greater trochanteric regions.

His static and dynamic sitting balance were poor, and he exhibited symptoms of postural hypotension. At admission, the Functional Independence Measure (FIM) score was 49, the Spinal Cord Independence Measure (SCIM) score was 19/100, and American spinal injury association (ASIA) impairment scale grade was "B" for incomplete injury.

Pressure sores on the right and left greater trochanter regions and sacrum received a Grade 4 on European Pressure Ulcer Advisory Panel (EPUAP) and Stage 4 on Stirling Decimal Based Pressure Ulcer Scale (SPSSS) with dimension as 11 cm × 15 cm, 6.5 cm × 6 cm, 7.5 cm × 7 cm respectively; photographic evidence is mentioned in FIG. 1. The Right greater trochanter ulcer received a score of 17, Left greater trochanteric and sacral ulcers received a score of 16 on the Pressure Ulcer Scale for Healing (PUSH Tool 3.0). (TABLE 1 & 2).

TABLE 1. **Ulcer dimensions.**

	On admission a	**4th month f**	**10th month g**	**13th month h**
Right greater trochanter area	11 cm × 15 cm	2.5 cm × 3 cm	1 cm × 1 cm	0.2 cm × 0.1 cm
Left greater trochanter area	6.5 cm × 6 cm	4 cm × 4 cm	3cm × 2 cm	1.5 cm × 1 cm
Sacral area	7.5 cm × 7 cm	3.5 cm × 4 cm	2cm × 1 cm	Healed

Table 1 summarizes the changes in ulcer dimensions from admission to the 13-month follow-up. All three pressure ulcers showed progressive reduction in size over time. The right greater trochanteric ulcer decreased from 11 cm × 15 cm at admission to 0.2 cm × 0.1 cm at 13 months, indicating near-complete healing. The left greater trochanteric ulcer reduced from 6.5 cm × 6 cm to 1.5 cm × 1 cm, while the sacral ulcer decreased from 7.5 cm × 7 cm and achieved complete healing by the 13-month follow-up.

TABLE 2. **Changes in ulcer healing were documented using the EPUAP, PUSH and SPSSS scales.**

		On admission a	**4th month f**	**10th month g**	**13th month h**
EPUAP	Right Trochanteric	Grade 4	Grade 2	Grade 2	Grade 1
	Left Trochanetric	Grade 4	Grade 3	Grade 2	Grade 2
	Sacral	Grade 4	Grade 3	Grade 1	Healed completely
PUSH tool 3.0	Rt Trochanric	17	10.5	4	2
	Lt Trochantric	16	14	10	6
	Sacral	16	13	7	0
SPSSS	Rt Trochantric	Stage 4	Stage 2.3	Stage 2	Stage 0.3
	Lt Trochantric	Stage 4	Stage 3.2	Stage 3.1	Stage 2.3
	Sacral	Stage 4	Stage 3.1	Stage 0.2	Stage 0.2

TABLE 2 presents progression of pressure ulcer healing as assessed by the EPUAP, PUSH Tool 3.0, and the SPSSS. Progressive improvement was observed across all three assessment tools throughout the follow-up period. EPUAP grades improved from Grade 4 at admission to Grade 1 for the right trochanteric ulcer, Grade 2 for the left trochanteric ulcer, and complete healing of the sacral ulcer. Similarly, PUSH scores demonstrated a steady decline, reflecting progressive wound healing, with the sacral ulcer achieving a score of 0 by 13 months. SPSSS staging also showed consistent improvement, indicating marked reduction in ulcer severity over time.

3.1 Intervention

On surgical evaluation, the plastic surgery team noted extensive ulcer size with associated infection, rendering the patient unsuitable for conventional flap surgery. Consequently, the patient was considered for integrative therapy to promote wound healing. The intervention included a holistic combination of integrative treatment followed by cell therapy. Integrative therapy included Hyperbaric oxygen therapy, ozone therapy, Platelet rich plasma therapy locally on the wound, IV micronutrient therapy, deep tissue mobilization, gut cleansing with enemas, rehabilitation along with cell therapy which involved intrathecal administration of autologous bone marrow mononuclear cells (BMMNCs).

HBOT - The intervention was administered using the Tekna Hybrid 3200/32 Monoplace Hyperbaric Chamber, a medically certified, high-quality, economically designed HBOT system engineered for single-patient use. The chamber delivers pressurized treatment with 100% medical-grade oxygen, enabling enhanced oxygen delivery for therapeutic purposes. Over the course of 2.5 months, the patient underwent 66 HBOT sessions, conducted once daily. The pressure was initiated at 1.5 ATA and gradually increased to 2.2 ATA over the first 14 days. The pressure of 2.2 ATA was maintained until the 25th session, after which it was increased to 2.25 ATA at the 26th session. The pressure was further escalated to 2.35 ATA over the subsequent 4 sessions until the 30th session. Following this, the pressure was reduced to 2.2 ATA and maintained consistently through the 66th session.

Ozone therapy - The intervention was conducted using Ozone's generator (Medical Ozone Therapy Machine), a high-quality, medically engineered system designed for the safe and controlled delivery of medical ozone (O_3) generated from medical grade oxygen. The machine allows precise control of ozone concentration and flow rate, ensuring consistent and safe therapeutic application for the patient.

Rectal ozone was given one session daily for a total 44 sessions over 2.5 months. After lubricating the tip with ozonated oil, the rectal tube was inserted 4" into the rectum. After connecting the rectal bag containing 300 ml of medical ozone to the rectal tube, the ozone was gradually given over 10 minutes.

Additionally, he got 25 sessions of intravenous ozonized saline every alternate day over 50 days. After 15 minutes of bubbling, 200 milliliters of 0.45% normal saline were injected into the scalp vein over the course of 40-45 minutes.

The patient underwent 127 local ozone funneling sessions over bilateral trochanteric and sacral regions over a period of 4 months (First 3 months he received twice daily then once daily). Over the pressure ulcer on the sacral region and bilateral greater trochanter area, a medium-sized ozone funnel was positioned. To stop ozone from escaping, a damp towel was placed around the funnel's base.

Medical ozone gas was administered locally through a funnel to both the ulcers for 15 minutes each; the ozone concentration was adjusted based on the kind of ulcer. For improved diffusion, the funnel was left without ozone for an additional fifteen minutes. The concentration was set at 60 mcg for 14 days when it looked infected; the concentration was reduced to 40 mcg for another 18 days; and the reduced to 35-30 mcg for 50 days when infection was reduced and wound showed signs of healing and slight granulation where the concentration was further reduced to 20 mcg.

Platelet rich plasma therapy - The ulcer was treated with platelet rich plasma treatment once a week for 5 sessions when the wound started showing signs of healthy granulation. Two 10-milliliter ACD tubes were used to collect 40 milliliters of blood from the cubital vein, which was then centrifuged for 20 minutes at 1500 RPM. Under aseptic circumstances, PRP was extracted in insulin syringes. Three pressure ulcer sites- the sacrum, left lateral greater trochanter, and right lateral greater trochanter were treated with PRP injections. The muco-cutaneous junction, the base of the wound, and the borders of the wounds were all injected with PRP. Gauze and a Duoderm CGF dressing were applied to the wound for 24 hours.

IV micronutrient therapy - The patient received a total of 105 intravenous micronutrient drips at an interval of five times per week for 3.5 months. 39 sessions of Myers drip which is a combination of 5 gm IV Vitamin C, 500 mg of Magnesium chloride and 2 ml of Vitamin B Complex in 100 ml Normal Saline over 40 mins. 33 sessions of a drip consisting of 1.2 gms IV Glutathione in 50 ml Normal saline for 15 mins followed by 33 sessions of a drip consisting of 1 gm N Acetyl cysteine in 100 ml Normal saline over 30 mins.

Deep tissue mobilization - The patient had deep tissue mobilization which included oil massage and acupressure treatment. Acupressure was administered twice daily for the first month and then reduced to once daily for subsequent 2 months. Oil massage was given once daily for the initial two months, followed by alternate sessions during the third month. In total, 95 deep tissue mobilization sessions were completed. Slow strokes were used to apply deep, continuous pressure.

Gut cleansing, enema - The patient underwent 15 enema sessions every alternate day for gut cleansing in the first month. In order to clear the bottom portion of the colon, a neem and herb decoction was administered rectally.

Nutritional Management - The patient was initially vegetarian, but considering the increased protein requirements for wound healing, dietary modifications were implemented under the supervision of a nutrition specialist. A structured high-protein diet (~1850 kcal, 65 g protein/day) was introduced, including milk, pulses, paneer, and nuts. As per dietary guidance, eggs were gradually incorporated as an initial non-vegetarian protein source, followed by the inclusion of chicken and fish over time to further enhance nutritional status and support tissue repair.

Rehabilitation - The patient also received comprehensive rehabilitation therapy, which included occupational therapy, physiotherapy, psychological support and standard medical treatments.

BMMNCs Administration - The Institutional Committee for Stem Cell Research and Therapy provided ethical approval, and the patient signed an informed consent form for the procedure. Granulocyte colony stimulating factor (G-CSF), which aids in cell survival and proliferation, was given to the patient in doses of 300 mcg 72 and 24 hours prior to cell therapy. A bone marrow aspiration needle was used to extract 110 milliliters of bone marrow from the left anterior superior iliac bone in heparinized tubes while under local anesthetic and sedation.

The density gradient technique was then used to extract the mononuclear cells (MNCs) from the bone marrow. Trypan blue vital dye was used to assess the viability and total cell count of the separated MNCs. The Invitrogen Attune NXT flow cytometer was used to determine the proportion of CD34+ cells.

The isolated MNCs, which had an average of 1.06 × 108 cells and 97% viability, were administered by lumbar puncture at the level between the fourth and fifth lumbar vertebrae using a 25G spinal needle. To reduce inflammation and increase the survival of the injected cells, 500 milliliters of isolyte P containing 1 gram of solumedrol was injected intravenously.

Ten months following the initial treatment, the patient received an additional dose of cell therapy alone, following the same procedure as previously used.

Follow up: To evaluate the impact of the intervention on functional recovery and ulcer healing, a thorough follow-up assessment was conducted for the patient at 4, 10, 13 and 24 months. At each follow-up, the ulcer's size was measured until 13 months.

Outcome measures: Objective scales namely, The Functional Independence Measure (FIM) score, Spinal Cord Independence Measure (SCIM) score, American spinal injury association (ASIA) impairment scale, European Pressure Ulcer Advisory Panel (EPUAP) scale (TABLE S1), The Pressure Ulcer Scale for Healing (PUSH) tool 3.0 and Stirling Decimal Based Pressure Ulcer Scale (SPSSS) (TABLE S2) were used to evaluate the effect of intervention on patient's functional recovery and the healing of decubitus ulcer.

4. Results

4.1 Section A: Improvements related to Ulcers

Following multidisciplinary integrative therapy combined with rehabilitation and cell therapy, the patient's bed sores of sacral region and right trochanteric region were completely healed at 13 months period and left trochanteric sore was in the healing phase at stage 2. The left trochanteric ulcer got completely healed at 15 months.

At admission, all three pressure ulcers were severe, indicating extensive tissue damage and poor wound status. At 4 months, clinical improvement was observed across all ulcer sites, the dimension of ulcers at the Right trochanteric, left trochanteric and sacral region got reduced to - 2.5 cm × 3 cm, 4 cm × 4 cm, 3.5 cm × 4 cm respectively which is considered approximately 70% reduction in wound size along with improvement in wound bed and reduction in exudate. Consistent with these findings, EPUAP, PUSH tool 3.0 and SPSSS (TABLE 2) demonstrated significant improvements. The previously visible underlying bony structures were no longer exposed, accompanied by a reduction in wound dimensions and exudate, and an improvement in tissue type, indicating active wound healing.

At 10 months, the dimension of ulcers at the Right trochanteric, left trochanteric and sacral region got reduced to 1 cm × 1 cm, 3 cm × 2 cm, 2 cm × 1 cm representing approximately 90% wound healing. EPUAP, PUSH tool 3.0 and SPSSS scores (TABLE 2) also showed further improvements with a reduction in depth of the ulcer, increased soft tissue coverage, and the presence of healthy granulation tissue and epithelialization visualized at all three sites, a clear indication of healing.

At 13 months, the right trochanteric bed sore was almost healed with re-epithelialization, indicating near complete healing with only minimal superficial tissue damage (dimension measuring 0.2 cm × 0.1 cm). The left trochanteric bed sore further reduced in size to 1.5 cm × 1 cm demonstrating substantial wound contraction and healing while sacral region bed sore healed

completely with complete epithelialization. EPUAP, PUSH tool 3.0 and SPSSS also showed remarkable improvement indicating advanced wound healing in the Right trochanteric and sacral region and no presence of exudate in the ulcer of Left trochanteric region, reflecting on improved bed sores. The Left trochanteric ulcer healed completely in 15 months of time.

At 24 months, all 3 Right trochanteric, Left Trochanteric and sacral ulcers remained completely healed, no evidence of development of new bed sores or recurrence of the healed bed sores.

Overall, the results demonstrate significant and continuous wound healing over time across all pressure ulcer sites. These findings indicate effective wound management and sustained tissue recovery during the follow-up period.

4.2 Section B: Functional improvements

The patient exhibited consistent, progressive and sustained functional and clinical improvements over a 24-month follow-up period. No side effects were observed post-treatment.

At 10 months, in addition to wound healing, functional improvements were also observed. There was marked improvement in postural hypotension, standing tolerance was improved previously from few seconds to 2-3 hours using a standing frame and sitting balance improved upto 3-4 minutes with hand support. Sensory gains were noted, with pin-prick sensation improving from absent to partial bilaterally from T11 to S4-S5 on the right side, and light touch improving from absent to partial from T6 to S4-S5 on the left side. A reduction in both flexor and extensor spasms was also observed. Neurological status remained ASIA grade B, while functional scores remained SCIM: 19 and FIM: 49.

At 13 months, Postural hypotension further showed marked improvements. Standing tolerance improved from 2-3 hours to 4-5 hours a day using standing frames. Flexor and extensor spasm further reduced compared with the previous follow-up, and overall stamina improved. Fine motor function improved further enabling him to eat using a modified spoon, wear a shirt with partial assistance and use a mobile phone independently. Neurological status remained ASIA grade B, while functional scores improved to SCIM: 20 and FIM: 50.

At 24 months, sustained autonomic symptoms were observed, with the patient no longer experiencing postural hypotension or autonomic dysreflexia. Functional abilities continued to improve. He was able to eat approximately one-quarter of a meal with moderate assistance, hold a glass and drink independently, wear a T-shirt with partial assistance, and use a mobile phone independently for activities such as online shopping and financial transactions. He became partially independent in bed mobility. While bladder and bowel control remained absent, vague bowel sensations were reported. Throughout the follow up period, Neurological status remained unchanged at ASIA grade B, Functional outcome measures remained stable, with a SCIM: 20 and a FIM: 50 at the 24-month assessment.

Changes in ulcer healing were documented using the ulcer dimensions in TABLE 1, EPUAP, PUSH and SPSSS scales in TABLE 2, and photographic evidence of Right greater trochanteric ulcer healing in FIG. 2 and FIG. 5, photographic presentation of Left lateral ulcer healing in FIG. 3 and FIG. 6, photographic presentation of Sacral ulcer healing in FIG. 4 and FIG. 7.

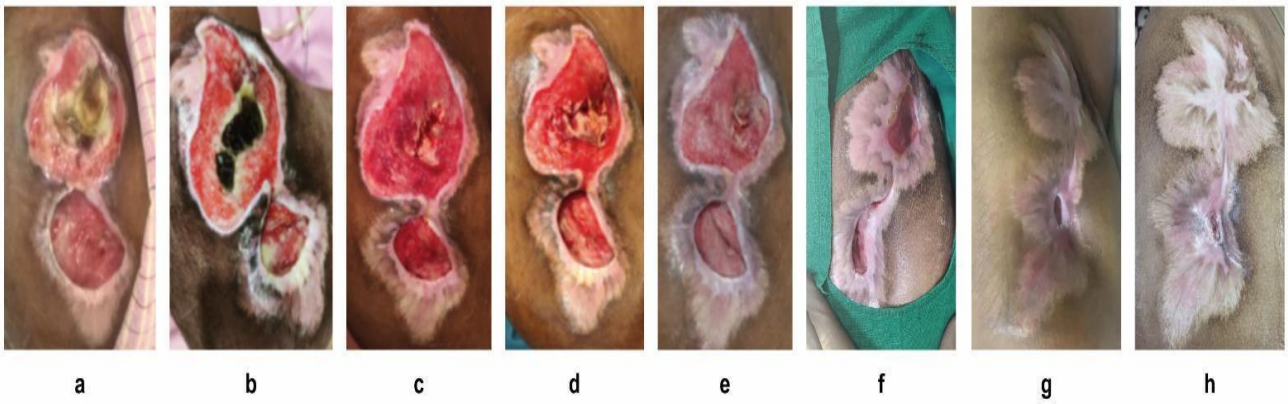

FIG. 2. **Photographic evidence of Right greater trochanteric bed sore healing in 7 follow up images.**

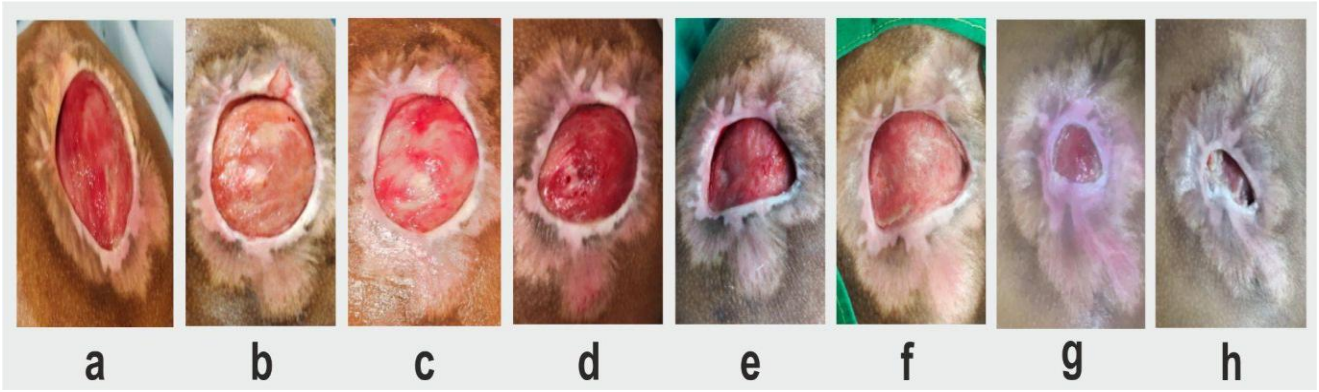

FIG. 3. **Photographic evidence of Left later ulcer healing in 7 follow up images.**

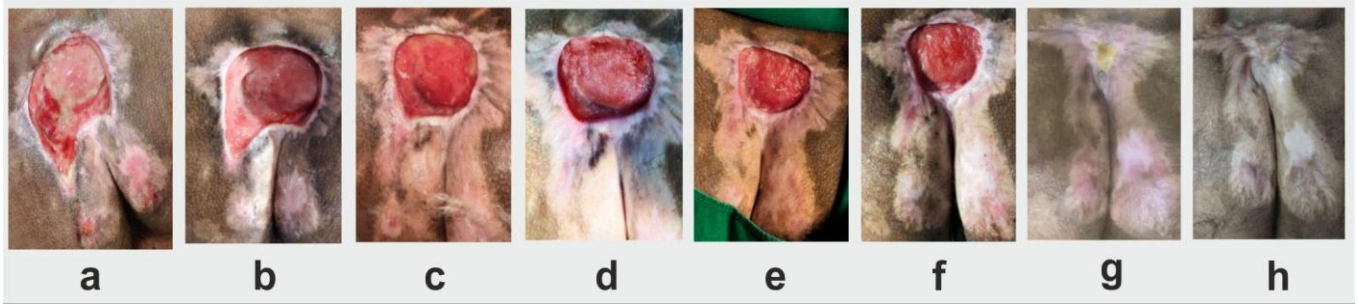

FIG. 4. **Photographic evidence of Sacral ulcer healing in 7 follow up images.**

a. Patient provided the image before the admission
b. After 10th sessions of HBOT and ozone therapy.
c. After 15th sessions of HBOT and ozone therapy and Debridement.
d. After 23rd sessions of HBOT and ozone therapy.
e. After 31st sessions of HBOT and ozone therapy.
f. At 4 months follow up. 4 sessions of PRP were done from the 49th session of HBOT until now.
g. At 10 th month Follow up.
h. At 13 th month Follow up.

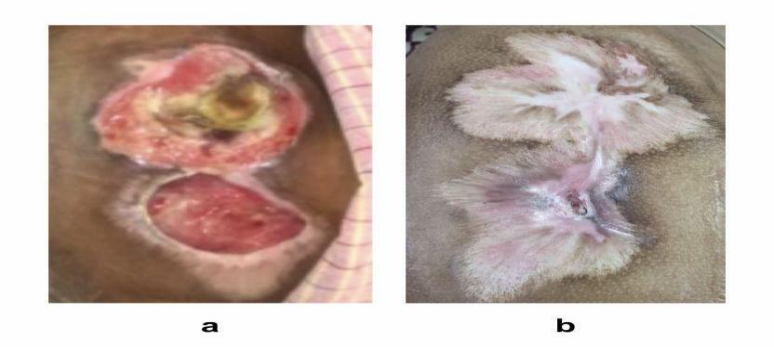

FIG. 5. **Photographic presentation of Right greater trochanteric ulcer before and after ulcer healing.**

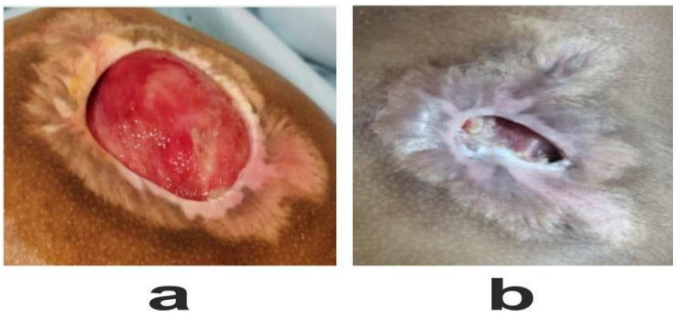

FIG. 6. **Photographic presentation of Left lateral ulcer before and after ulcer healing.**

FIG. 7. **Photographic presentation of Sacral ulcer healing before and after ulcer healing.**

a. Before the start of the treatment
b. At 13 months follow up.

5. Discussion

Pressure ulcers, also known as bedsores or decubitus ulcers are a significant concern in patients with spinal cord injuries (SCI). These ulcers are a major cause of morbidity, adversely affecting the quality of life and complicating the management of SCI patients.

The development of pressure ulcers can be attributed to factors such as friction, shear, moisture, and poor nutritional status. This pressure impairs blood flow, leading to ischemia. Furthermore, the ischemic tissues accumulate toxic metabolites. Subsequently causing tissue ulceration and necrosis. In SCI patients, the absence of sensation below the level of injury prevents them from feeling or reacting to pressure, resulting in prolonged pressure on susceptible areas. This lack of sensation, combined

with immobility and reduced blood flow due to decreased muscle activity, creates an ideal environment for ulcer development [2]. Pressure ulcers can significantly affect a patient's quality of life and increase healthcare costs due to recurrent hospitalisation caused by chronically infected wounds, increased risk of osteomyelitis, prolonged intravenous antibiotic treatments, amputation, and eventually the need for supportive care measures [3,4].

Wound healing involves three main phases - an initial inflammatory phase where platelets release clotting factors, cytokines, and growth factors; a proliferative phase (around days 5-7) marked by fibroblast migration, granulation tissue formation, and angiogenesis; and a long remodeling phase involving wound contraction and matrix reorganization, resulting in scar formation [8]. Effective healing requires a consistent supply of growth factors, nutrients, and oxygen. Factors like infection, malnutrition, chronic disease, or diabetes can delay healing, leading to chronic wounds [9]. Conventional treatments for decubitus ulcers include non-surgical approaches such as pressure relief, moist dressings (e.g., hydrocolloids, hydrogels), antiseptics, debridement, electrical stimulation, nutritional support, and antibiotics to promote healing and prevent infection. Surgical options may involve debridement, wound closure, skin grafts, or flap reconstruction [10].

In spinal cord injury (SCI) patients, pressure ulcer management is particularly challenging and requires a multidisciplinary approach due to impaired circulation, reduced cellular regeneration, and increased susceptibility to infection. Therefore, there is an urgent need to explore innovative treatment strategies which can aid in faster healing of the ulcers in SCI patients. HBOT an ozone therapy has emerged as a promising therapeutic approach for chronic wound healing. Integrating them with deep tissue mobilization, IV multivitamins, Rehabilitation and Cell therapy further amplifies its beneficial healing effects.

Despite conventional treatment the patient had stayed with nonhealing pressure ulcers for 1.5 years. After receiving multidisciplinary integrative treatment combined with rehabilitation and cell therapy, the bedsores completely healed within 13 months, compared with the approximately 24 months typically required with conventional treatment.

Hyperbaric oxygen therapy (HBOT) involves exposing the body to 100% oxygen at higher pressures. There is high level of evidence that it promotes healing of problem wounds and pressure ulcers [6]. Studies have also shown that HBOT results in significant sensory and motor improvements in spinal cord injury patients [11].

Hyperbaric oxygen therapy enhances healing through hyperoxygenation and bubble size reduction. Increased plasma oxygen, as per Henry's law, aids in conditions like crush injuries, compartment syndrome, flap salvage, anemia, and ulcers [12,13]. It reduces edema, boosts collagen synthesis, promotes wound healing, and aids in bacterial killing, especially anaerobes, through oxygen free radicals [14].

Non-healing wounds often result from underlying hypoxia. Hyperbaric oxygen therapy (HBOT) promotes healing by enhancing oxygen-dependent processes like fibroblast proliferation, collagen synthesis, and angiogenesis, supported by growth factors like VEGF. Its antimicrobial properties boost leukocyte activity, while systemic vasoconstriction reduces edema, improving oxygen diffusion and relieving vessel pressure [12,13]. HBOT now plays a vital role in plastic surgery, aiding in the treatment of ulcers, ischemic and traumatic wounds, necrotizing infections, failing grafts, radiation injuries, and burns [14]. Our findings reinforce the previous evidence that suggest HBOT plays vital role in non-healing wounds.

Ozone therapy has shown to have tremendous improvements in chronic wounds, potentially acting by eliciting mild oxidative stress or disinfection [15,16]. Systemic ozone therapy irreversibly damages viral RNA [17] and bacterial growth [18] by oxidizing pathogen lipoproteins and phospholipids. Its decomposition in blood generates reactive oxygen species (ROS) like superoxide, hydroxyl radicals, and nitric oxide, which act as vasodilators and stimulate growth factors essential for healing and repair [19].

Ozone, at 30-60 mcg, exhibits strong antimicrobial properties and promotes wound healing by disrupting microbial structures and generating oxygen free radicals. It activates the nuclear factor erythroid 2-related factor 2 (Nrf2) which in turn activates complex cascade of events, enhancing cell growth, immune response, and proinflammatory processes promoting tissue repair and recovery [20]. Ozone therapy may accelerate chronic wound healing compared to standard care [15]. Our findings support the conclusion of the study that ozone therapy may accelerate wound healing as compared to standard one.

Studies in rodents show it enhances vascularity, speeds healing, and reduces neuronal damage [21] and enhanced vascular growth factor and cyclin D1 expression when ozonated sesame oil was used as a local application for wound repair [22].

Platelet-rich plasma (PRP). Plasma that is rich in platelets and growth factors promotes tissue regeneration and repair. Insulin-like growth factor-1 (IGF-1) and vascular endothelial growth factor (VEGF) is particularly important for promoting re-epithelialization and healing in wounds, burns and nerve injury [23,24,25]. Platelet-rich plasma (PRP) plays a significant role in wound healing by promoting epidermal stem cell (ESC) activity, angiogenesis, and tissue remodeling [26]. When a wound occurs, ESCs rapidly form clones at the wound's edge and then expand toward the center, gradually differentiating into daughter cells to repair the skin defect [27,28]. This process is enhanced by PRP through the IGF-1/IGF-1R pathway [24]. PRP also boosts VEGF secretion, facilitating angiogenesis and the formation of new blood vessels that supply nutrients and oxygen to the wound [24/29]. In our case, wound healing was significantly accelerated with the addition of PRP sessions, thereby supporting the validity of the above references.

It increases myofibroblast differentiation, marked by α-SMA expression, which contributes to wound contraction [24,30]. Numerous studies show PRP significantly enhances healing time and collagen organization, either alone or with biomaterial scaffolds [31,32]. A systematic review and Meta-analyses of randomized clinical trials demonstrate PRP's efficacy and safety, with a higher likelihood of complete wound closure compared to control groups [33,34].

IV Micronutrient therapy - High dose vitamin C [35-37] magnesium [38], B-complex vitamins [39,40], glutathione [41], and N-acetylcysteine [42] enhance collagen synthesis, reduce inflammation, boost antioxidant defenses and supports wound healing. Although our study did not include specific biomarkers or tests to directly quantify wound-healing outcomes, clinical observations suggested faster tissue recovery, improved immune status, and enhanced overall wellbeing in patients receiving this integrative intervention.

Deep tissue mobilization/ massages can enhance wound healing in pressure ulcers by improving blood circulation, reducing tissue tension, and promoting the delivery of nutrients to the affected area [43]. In cervical-level spinal cord injuries, it may also aid in maintaining tissue integrity and enhancing functional outcomes by preventing further complications like contractures

and pressure sore progression [44,45]. In our case, the contribution of this treatment to wound healing could not be clearly established, although it appeared to provide pain relief in areas with preserved sensation.

Rehabilitation programs like physiotherapy, occupational therapy, and psychological support play crucial roles in managing spinal cord injury (SCI) cases with decubitus ulcers. Physiotherapy aids in improving mobility, muscle strength, and circulation, which are vital for preventing further ulcer development and promoting wound healing [46]. Occupational therapy focuses on enhancing functional independence by assisting patients in adapting to daily activities and using adaptive equipment, which helps reduce pressure on vulnerable areas [47]. Psychological support addresses mental health challenges, fostering coping strategies and improving overall well-being, thereby enhancing patient adherence to treatment and rehabilitation plans [48]. Our findings are consistent with those of studies, that demonstrated clear benefits of the rehabilitation program in enhancing functional mobility and quality of life, thereby helping prevent wound recurrences, although its direct impact on wound healing could not be confirmed.

Cell therapy - Several clinical studies have shown that cell therapy reverses neurological damage in SCI and helps in functional recovery thereby improving the quality of life of the patients [49-58]. In case of SCI patients with decubitus ulcers, it may also further enhance wound healing. Bone marrow-derived mononuclear stem cells (BMMNCs) are one of the most feasible and effective due to their ability to support neural repair and enhance chronic ulcer healing through angiogenic factor secretion [59]. This further leads to functional improvements, including better sensation, mobility, in spinal cord injury cases. Consistent with prior research, our patient also gained benefit with improved bed mobility, decreased flexor and extensor spasms, improved standing endurance on standing frame and settling of postural hypotension which further helped to bed sore healing and improved quality of life [49-58].

In contrast to the findings that Stage 4 pressure ulcers in spinal cord injury cases often require around 2 years to heal [60], with many necessitating surgical intervention. In our case, however, the holistic intervention combining integrative treatments, rehabilitation and cell therapy resulted in complete healing of two pressure ulcers within 13 months, a considerably shorter duration than the typical healing time frame for such wounds in spinal cord injury patients. Notably, there was no recurrence at the 24-month follow-up. This healing was further accompanied by significant functional improvements and improved quality of life of the patient.

6. Limitations

Because multiple integrative therapies were administered alongside rehabilitation interventions, the independent efficacy of any single treatment modality could not be evaluated in this study.

7. Conclusion

This case demonstrates the beneficial effects of a holistic intervention of multidisciplinary integrative therapy combined with rehabilitation and cell therapy, in chronic spinal cord injury with complication of multiple bed sores. This intervention resulted in complete healing of the bed sores faster as compared to conventional treatments, and remarkable functional recovery thereby improving the quality of life of the patient significantly.

8. Data Availability

The data supporting the findings of this case report are not publicly available due to patient confidentiality and institutional privacy restrictions. Relevant clinical information and data may be obtained from the corresponding author upon reasonable request and with appropriate permission from the NeuroGen Brain and Spine Institute.

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11. Author Contributions

Dr. Hemangi M. Sane: Conceptualization, Validation, Resources, Critical review of the manuscript, Supervision and Principal Investigator.

Dr. Heena S. Sayyed: Conceptualization, Methodology development, Validation, Data Acquisition, Data Analysis, Statistical Analysis, Writing of the Original Draft.

Tahreem T. Hamdule: Conceptualization, Methodology development, Validation, Data Acquisition, Data Analysis, Statistical Analysis, Writing of the Original Draft, Editing of the manuscript.

Dr. Pallavi V. Khubchandani: Methodology development, Validation, Data Acquisition.

Pooja Kulkarni: Resources, Critical review of the manuscript, Supervision, Project Administration.

Dr. Nandini Gokulchandran: Critical review of the manuscript.

Dr. Prerna Badhe: Critical review of the manuscript.

Dr. Alok Sharma: Resources, Critical review, final approval of the manuscript, Supervision, Funding.

12. Competing Interests

The authors declare that they have no competing interests.

13. Consent Statement

Written informed consent was obtained from the participants parents or legal guardians prior to inclusion in the study.

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Supplementary Information

The European Pressure Ulcer Advisory Panel (EPUAP) classification system is used to grade the severity of pressure ulcers based on observable clinical features.

TABLE S1. **EPUAP Pressure Ulcer Classification Table.**

Category / Grade	Title	Clinical Description	Key Identifying Features
Category 1	**Non-blanchable erythema**	Intact skin with non-blanchable redness of a localised area, usually over a bony prominence.	Area does **not** turn white when pressed; may be painful, firm, soft, warmer, or cooler compared to adjacent tissue.
Category 2	**Partial thickness skin loss**	Loss of dermis presenting as a shallow open ulcer with a red-pink wound bed.	May also present as an intact or open/ruptured **serum-filled blister**.
Category 3	**Full thickness skin loss**	Subcutaneous fat may be visible, but bone, tendon, or muscle are **not** exposed.	Slough (dead tissue) may be present; may include undermining and tunnelling.
Category 4	**Full thickness tissue loss**	Extensive destruction with exposed bone, tendon, or muscle.	Slough or eschar (scab) may be present on some parts of the wound bed; often includes undermining and tunnelling.

While the official EPUAP/NPIAP classification uses whole numbers (1–4), the decimal-based "Detailed Progression" system is typically found in the Stirling's Pressure Ulcer Severity Scale (SPSSS). This 2-digit scale is used in clinical research to provide finer detail on wound characteristics.

TABLE S2: **Stirling Decimal-Based Pressure Ulcer Scale.**

Stage	Sub-Stage	Clinical Description & Characteristics
0 (No Clinical evidence of a Pressure sore)	**0.1**	**Normal appearance**; intact skin with no signs of damage.
	0.2	**Healed**; area shows scarring from a previous ulcer.
	0.3	**Tissue damage** present, but not clinically assessed as a pressure sore.
1 (Discolouration of Intact Skin)	**1.1**	**Non-blanchable erythema** (redness) with increased local heat.
	1.2	**Blue/purple/black** discoloration of intact skin (early deep tissue damage).
2 (Partial thickness skin loss or carnage involving epidermis or dermis)	**2.1**	**Blister** (intact or ruptured) involving the epidermis/dermis.
	2.2	**Abrasion**; superficial scraping of the skin surface.
	2.3	**Shallow ulcer** without undermining of the adjacent tissue.
	2.4	Any Stage 2 injury with underlying **discoloration** or induration.
3 (Full thickness skin loss involving damage or necrosis of subcutaneous tissue but not	**3.1**	**Crater**; full-thickness loss without undermining.

extending to underlying bone, tendon or joint capsule)		
	3.2	**Crater with undermining**; tissue destruction extends under the skin edges.
	3.3	**Sinus**; a narrow tract whose full extent is unknown.
	3.4	**Necrotic/Slough-covered**; wound bed is masked by black eschar or yellow slough.
4 (Full thickness skin loss with Extensive destruction and tissue necrosis extending to underlying **bone, tendon, or joint capsule**)	**4.1**	Visible Extensive destruction extending to **bone, tendon, or joint capsule**.

Unstageable	**Depth Unknown**	Full thickness tissue loss in which the base of the ulcer is covered by slough and/or eschar.	The true depth cannot be determined until enough slough/eschar is removed to expose the wound bed.
SDTI	**Deep Tissue Injury**	Purple or maroon localised area of discoloured intact skin or blood-filled blister.	Caused by damage of underlying soft tissue from pressure and/or shear; the area may be preceded by tissue that is painful, firm, or mushy.