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Wound management with compression therapy and topical hemoglobin solution in a patient with Budd-Chiari Syndrome

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Abstract

Background: Although the underlying primary cause of chronic wounds may vary, a common etiology of this is a hypoxic or ischemic status of the affected tissue of the lower extremities. In particular, for rare diseases associated with disturbed blood flow a correlation between cause and effect is often diagnosed inappropriately. As a consequence, chronic wounds may develop and persist for years.

Main observations: We present a case of a patient with chronic venous insufficiency due to an occlusion of the inferior caval vein. Initially, a Budd-Chiari syndrome was diagnosed which is a thrombotic obstruction of the hepatic venous outflow. In addition, the patient developed an obstruction of the inferior caval vein and subsequently a chronic venous insufficiency. As a consequence, chronic leg ulcers developed with a history of more than 7 years.

Various wound care approaches were performed without success in wound closure. Finally, a combination of compression therapy and topical application of a hemoglobin solution successfully led to fast and persistent wound closure.

Conclusions: Chronic ulcers of the lower limb such as venous leg ulcers, even for patients with rare disorders like Budd-Chiari syndrome, are associated with oxygen supply disturbances resulting in a hypoxic status of the affected tissue. Therefore, an adequate oxygen supply to chronic wounds plays a pivotal role in successful wound healing.

Compression therapy in combination with enhancement of the local oxygen supply by topically applied hemoglobin showed marked improvement of wound healing in the presented patient. (*J Dermatol Case Rep.* 2014; 8(1): 20-23)

Introduction

The Budd-Chiari syndrome (BCS) is a rare and life threatening disorder, occurring in 1 in 100,000 of the population worldwide.¹ It is related to obstruction of the hepatic venous outflow tract while the etiology of outflow obstruction can be heterogeneous.^{2,3}

In case the obstruction to hepatic venous outflow is associated with thrombosis or stenosis^{1,3} BCS is classified as primary, while an obstruction to venous outflow caused by an abscess, tumor, cyst, or hyper plastic nodules^{2,3,4} is classified as secondary BCS.^{2,5}

In contrast to BCS, chronic venous insufficiency (CVI) is a common blood flow disorder in many cases leading to poor healing of leg ulcers and a frequent rate of recurrence^{6,7,8,9} without adequate diagnosis and therapy. One important factor of chronic leg ulcer is the hypoxic status of the tissues affected by the underlying abnormal venous physiology^{9,10,11} in chronic venous insufficiency and probably in BCS.

We present the successful treatment of chronic leg ulcer from a patient with Budd-Chiari syndrome and chronic venous insufficiency.

Case report

A 42-year-old male patient was diagnosed with a rare clinical Budd-Chiari syndrome in combination with further

complications like portal hypertension and media sclerosis. Most recently, a complete occlusion of the inferior caval vein and chronic venous insufficiency was determined. As a consequence the patient developed edema and venous leg ulcers.

The ulceration started 15 years ago with several periods of relapse and remission. During the past 8 years only temporary improvement of ulcers but no complete healing was observed.

Compression therapy was performed over a period of 4 years on both lower extremities by using compression stockings.

When the patient was presented to the home care nursing service in 2012, the leg ulcer on the right leg had a dimension of approx. 110 cm^2 (9.5 x 11.5 cm) and had existed for more than 6 months.

When wound treatment was started compression therapy was re-adjusted to compression with four short stretch



Figure 1
Wound Care. In addition to compression therapy (A), the wound was cleansed and hemoglobin solution applied to the wound bed (B) before wound dressing.



Figure 2

Course of wound healing of chronic ulcer.

A) Wound at starting point of wound treatment, B) 3 weeks, C) 6 weeks, D) 12 weeks and E) 15 weeks after treatment. Wound was closed after 16 weeks. F) The followed up visit 8 weeks after wound closure showed no recidivism.

bandages for 8 weeks (Rosidal® Sys, Fig. 1A) to reduce the edema status of the leg. The compression bandage was reapplied on a daily base by the home care nurse. Thereafter the compression therapy was switched back to compression stockings (category II) which were applied by the patient.

The wound was mechanically cleansed three times a week with sterile forceps and a compress pad, soaked with a wound irrigation solution (Octinisept®). After approx. one minute incubation the wound was rinsed with a physiological saline solution.

After each cleansing, a hemoglobin solution (Granulox®, porcine hemoglobin, 10g/dl) was applied equally onto the wound bed as a thin layer (spray for 2-3 seconds » 500µl/10cm²) (Fig. 1B). The wound was covered during the first 8 weeks of wound treatment with a sterile primary dressing comprising of an activated charcoal cloth and an absorbent compress (Actisorp® Silver + sorbion sachet S) fixed with a gauze bandage due to a high exudate level of the wound.

After this period, a significant reduction of exudation was achieved and the wound dressing was changed to adhesive air-permeable absorbent soft silicone foam (Mepilex®) wrapped with a gauze bandage. No further concurrent wound therapy was applied.

Figure 2 summarizes the course of wound healing at day 0 (Fig. 2A), 3 weeks (2B), 6 weeks (2C), 12 weeks (2D) and 15 weeks (2E). Within the first 3 weeks only marginal improvements of the wound were observed. Within the next 3 weeks a significant improvement of wound tissue and reduction of wound size was obtained as shown in Fig. 2C. During the next 6 weeks the course of healing progressed and wound size decreased constantly (Fig. 2D, E). 16 weeks after the start of treatment, a complete wound closure was achieved. No recidivism was obtained at a follow up inspection 2 months later (Fig. 2F).

Furthermore, the patient reported that he had achieved a significant improvement in quality of life as he was capable of going outside with only compression stockings even to the swimming bath for the first time in 10 years.

Discussion

BCS is a rare and life threatening disease related to a hepatic venous outflow obstruction. 1,2,3,4 In the case of additional obstruction affecting the caval vein it may be associated with leg ulcers of the lower extremities. In contrast, chronic venous insufficiency is a frequent health problem with venous leg ulcers as one of the most severe complications. 6,7,13 Some population studies describe that 20 percent of venous leg ulcers failed to heal within two years, and 66 percent of patients had episodes of ulceration lasting longer than five years. Although conventional compression therapy is established as the basic concept for treatment of venous leg ulcers, 20 more than 25% of patients are refractory to wound healing within one year. 14

Venous leg ulcers are known to be associated with a hypoxic and ischemic status of the affected tissue^{11,15,16} due to disturbed macro- and microcirculation.¹⁰ As it is known that oxygen plays a crucial role in all phases of wound healing,^{17,18}

the improvement of systemic and local oxygen supply to the affected tissue is seen as one major factor in wound management. To interfere with the primary cause of venous leg ulcers a treatment like compression therapy or surgery with the aim to prevent venous hypertension, reflux, and inflammation is the first choice for extenuating symptoms of chronic venous insufficiency and reduce the risk of ulcers.

In the presented case, several adjustments of the wound care were performed as the most recent diagnosis revealed a chronic venous insufficiency in addition to BCS.

Various studies have revealed that venous ulcers heal more rapidly with compression than without and that the type of compression used has a further impact on the speed of wound healing.²⁰ Therefore, compression therapy was adapted from compression stockings to short stretch bandages. Furthermore, regular wound treatment three times a week was performed by a home care nurse to achieve a high level of compliance.

As a third component a hemoglobin solution was applied after cleansing of the wound bed. Recent studies suggested that locally applied hemoglobin has a beneficial effect on the wound healing of venous leg ulcers, ²¹ as the hemoglobin is capable of improving the oxygen supply in aqueous solutions, e.g. wound exudates, by facilitated diffusion.²²

The presented result showed that the combined therapeutic efforts were successful in achieving wound healing. It further indicates that the improvement of the hypoxic status of the affected tissue may be an important beneficial factor on wound healing and recurrence as no new ulceration was observed.

Conclusion

A case report of a 45-year-old male patient with Budd-Chiari syndrome and chronic leg ulcer was presented. Appropriate diagnosis combined with an adequate treatment procedure and wound care management resulted in wound healing and overall significant improvement of his quality of life.

This case highlighted that, in particular for rare diseases like the Budd-Chiari Syndrome, a multi-disciplinary effort starting with a comprehensive diagnosis and monitoring of the patient as well as a consistent disease and home care management is of upmost importance in improving the overall quality of life of such patients.

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