Introduction

Mr O was referred to the hospital leg ulcer clinic due to non-healing ulceration which had been present for the last 4 months. He was presently being seen by the community nursing team who believed the ulcer was of venous aetiology and commenced treatment with appropriate 4-layer compression bandaging (Profore™). This had been well tolerated by Mr O but whilst in compression his ulcer had increased in size and depth so the second opinion of the Vascular Nurse Specialist was sought.

Mr O was 34 years old, married and not currently in employment. He had never smoked and his past medical history included arthritis, asthma, previous venous ulceration (healed in compression) and sleep apnoea. He was morbidly obese with a BMI of 56. On examination he had a full complement of pulses and an ABPI of 1.12 with a tri-phasic tone. He was currently in a 4-layer compression system appropriate for his limb size (30cm kit) and had been using this for the last 3 months, during which time the ulcer had increased in size and depth and this was causing great concern to Mr O. He complained of pain at times but stated that this was controlled with paracetamol.

Method

On initial review the ulcer was extensive in size, measuring 15cm x 10cm and approximately 1cm deep with steep cliff edges evident. The wound bed was clean but the tissue appeared bright red and extremely fragile to touch, there was moderate amounts of exudate but with no malodour. Also evident was haemosiderin staining and scar tissue from previous venous ulceration to both legs. The ulcer aetiology was confirmed as venous disease and vindicating the current management approach based on the use of compression bandaging. It was thought that wound bed colonisation might be the key factor in preventing healing and causing the noted increase in wound size. Given this Acticoat™ absorbent dressings were therefore commenced to reduce bacterial load and promote healing. Due to the unusual appearance of the ulcer edges a precautionary biopsy was taken.

First Review

Mr O was reviewed in clinic only 2 weeks later. The ulcer had dramatically reduced in size, with depth now reduced to be nearly at surface level. The wound bed had healthy granulation tissue, viable and advancing epithelising edges. The exudate had reduced to a minimal level as had Mr O’s pain. Due to exudate levels being reduced the primary dressing was changed from Acticoat™ absorbent to Acticoat™ 7. The wound biopsy taken at initial assessment identified no abnormality. Further review planned for in 2 weeks.

Second Review

At the subsequent review 4 weeks since commencing Acticoat™ the wound bed had again reduced in size and was now at surface level. Marginal epithelisation was continuing whilst pain and exudate was minimal.

Third Review

When Mr O was reviewed at 6 weeks after commencing Acticoat the ulceration which had been present and non-healing for the previous 4-months was nearly healed. Only a small area was awaiting re-epithelisation. The dressing regimen was changed but the Acticoat™ was changed to a non-adherent dressing and the ulcer went on to completely heal 8 weeks after initial presentation at the leg ulcer clinic.

Conclusion

This case study highlights the severe impact bacteria can have on causing increase in ulcer size and preventing healing. This gentleman was previously in appropriate compression the only treatment changed was the introduction of Acticoat™. Acticoat™ works speedily on reducing bacterial load therefore allowing healing to occur.

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**The use of Acticoat™ nanocrystalline silver dressing on a colonised venous leg ulcer**

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Leanne Cook, Vascular Nurse Specialist, Mid Yorkshire Hospitals NHS Trust