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Atypical leg ulcers after sclerotherapy for treatment of varicose veins: Case reports and literature review

Bibombe P. Mwipatayi[a,b,*], Catherine E. Western[a], Jackie Wong[a], Donna Angel[a]

[a] Department of Vascular Surgery, Royal Perth Hospital, Perth, Australia
[b] School of Surgery, Faculty of Medicine, Dentistry and Health Sciences, Western Australia, Australia

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ABSTRACT

INTRODUCTION: Skin necrosis is a rare complication of foam sclerotherapy, a common form of treatment for varicose veins.
PRESENTATION OF CASE: Both patients presented to the outpatient clinic within 2–14 days after foam sclerotherapy with Aethoxysklerol® 1%, with severe soft tissue and skin necrosis. Further aggressive treatment of the ulcer was required to resolve the necrosis, resulting in marked residual scar and well granulated leg ulcer respectively.
DISCUSSION: Foam sclerotherapy is a common and usually well-tolerated treatment modality for varicose veins. The aetiology of skin necrosis is conventionally related to extravasation of sclerosant. In order to minimise the risk of necrosis, the lowest concentration and lowest volume of sclerosant necessary to achieve adequate treatment of the target vein should be used.
CONCLUSION: We would like to emphasise that whilst skin and soft tissue necrosis is a rare complication of foam sclerotherapy, it is a complication that is highly disfiguring and requires aggressive treatment. As such, it should be adequately discussed with the patient prior to obtaining informed consent.

1. Introduction

Ultrasound-guided foam sclerotherapy involves injecting a chemical agent (a sclerosant) to induce blood vessel scarring and closure. In foam sclerotherapy, air is mixed with the liquid sclerosant to create foam. When this is injected into the varicose vein (under ultrasound guidance), it displaces the blood within the vein and fills the vein, causing the vein to spasm and scar. Foam sclerotherapy has a good success rate, with 80–90% of veins remaining closed after 3 years. As reported in the consensus document of the American Society of Dermatologic Surgery, the advantage of foam is that the sclerosing power of the solution is increased three-fold, while the toxicity is decreased four-fold [1]. Reported complications include bleeding, bruising, thrombophlebitis, infection at the injection site, deep vein thrombosis, pulmonary embolism, skin staining, and skin necrosis [2]. Whilst skin necrosis is rare, with a reported incidence of 0.2–1.2% [1], it is a complication that is highly disfiguring and requires aggressive treatment. Here, we report two cases of patients who experienced atypical extensive skin necrosis following sclerotherapy using liquid Aethoxysklerol® 1% (polidocanol, laureth-9).

2. Case report

1st Patient was a 32-year-old woman who presented to the vascular outpatient clinic in June 2011 for treatment of some prominent varicosities in her right leg. The patients' history of mild Von Willebrand's disease (vWD type IIA) resulted in varicosities secondary to an arterio-venous malformation. Von Willebrand's disease was confirmed by the measurement of the ristocetin cofactor (RCoF) activity below 30 IU/d. She had right leg ulceration after minor trauma in the past, which left her with marked scarring.

In July 2014, she re-presented with a 4 mm subcutaneous lump on her right anterior shin. After review, the lump was deemed secondary to thrombophlebitis and managed conservatively. She re-presented a few weeks later with varicosities that were secondary to her well-known AV malformation, confirmed by duplex scanning. She was offered foam sclerotherapy of the varicosities and underwent treatment with an unknown dosage of Aethoxysklerol® 1% (polidocanol, laureth-9) in September 2014 in a private clinic. Two days following treatment, she presented to the wound clinic with an area of tissue necrosis on the medial aspect of the right knee (Fig. 1). On clinical and duplex examination, it was established that there was no arterial compromise and a mepilex® border dressing (Mölnlycke Health Care AB, Gothenburg,

* Corresponding author at: University of Western Australia, School of Surgery, Royal Perth Hospital, Department of Vascular Surgery, Level 2, MRF Building, Royal Perth Hospital, Australia.
E-mail address: bibombe@iinet.net.au (B.P. Mwipatayi).

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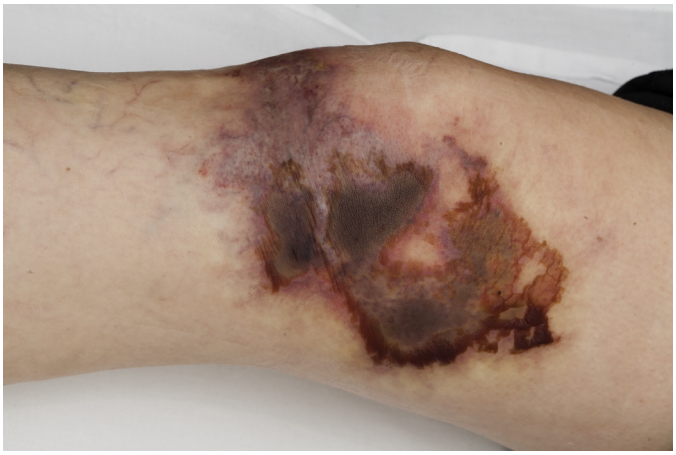

Fig. 1. Patient 1: area of tissue necrosis on the medial aspect of the right knee that appear two days following Aethoxysklerol® 1% foam sclerotherapy.

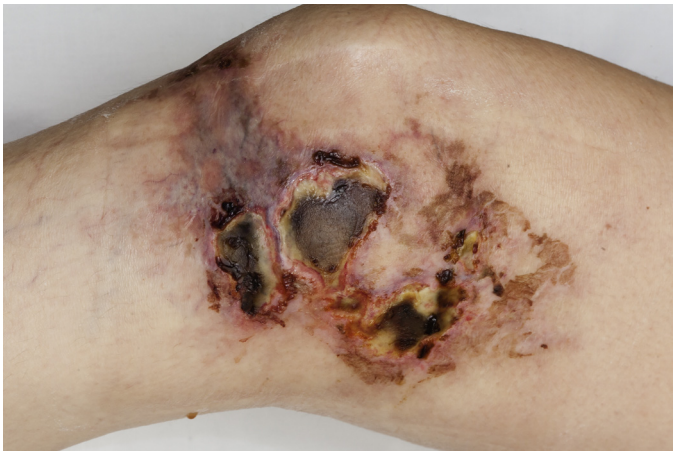

Fig. 2. Patient 1: picture showing that the necrotic areas increasing in size and began to demarcate and lift, without any clinical evidence of infection.

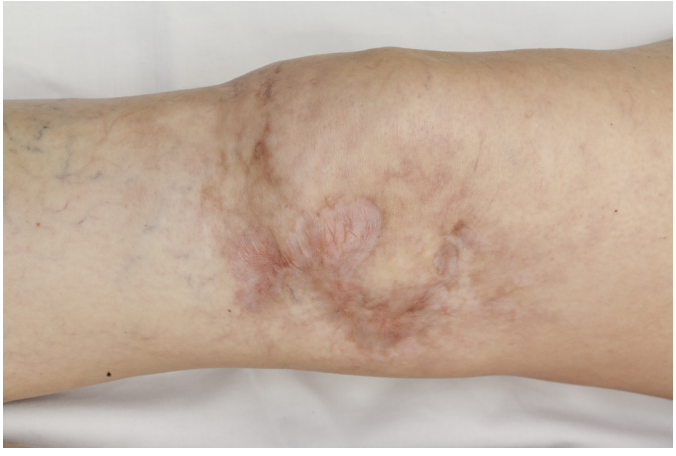

Fig. 3. Patient 1: five months post-treatment, the ulcers healed with marked residual scarring.

Sweden) was applied. Over the subsequent months, the necrotic areas increased in size and began to demarcate and lift, without infection (Fig. 2). Five months post-treatment, the ulcers healed with marked residual scarring (Fig. 3). On discharge from the wound clinic, the patient was advised to continue moisturising the area to improve scarring.

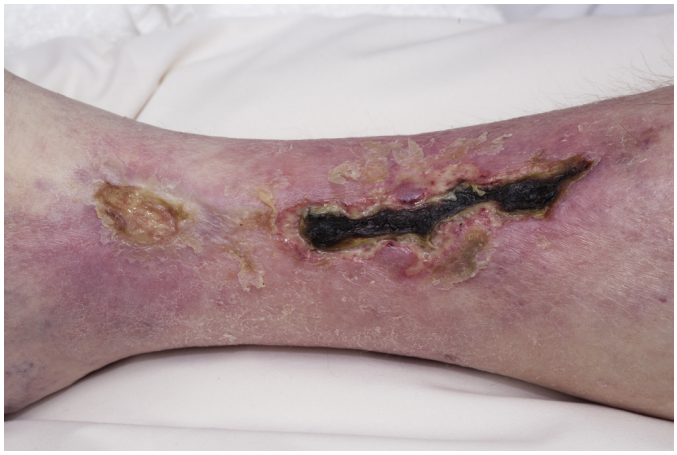

Fig. 4. Patient 2: the ulcer developed 4 weeks after treatment of an incompetent short saphenous vein and lateral aspect varicosities with Aethoxysklerol® 1% foam sclerotherapy.

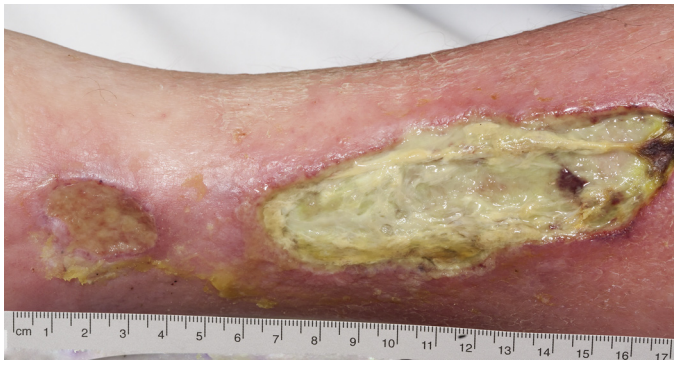

Fig. 5. Patient 2: large lateral sloughy left leg ulcer (11 × 4 cm) in December 2015.

2nd Patient was a 69-year-old woman who had suffered from chronic venous disease for 10 years and presented at a private clinic with leg oedema (CEAP class C3). The ulcer developed 4 weeks after treatment of an incompetent short saphenous vein and lateral aspect varicosities with Aethoxysklerol® 1% foam sclerotherapy (Fig. 4). She was admitted to the vascular ward in December 2015 after developing a large lateral sloughy left leg ulcer 11 × 4 cm (Fig. 5). The left leg arterial duplex scan was normal and the results from the microbiology samples were clear from infection. We decided to proceed with debridement of the ulcer. She underwent 4 sessions of out patient low-frequency ultrasound debridement (LFUD) using the SONOCA-185® Machine (MediGroup Australia Pty Ltd, Melbourne, Australia). EMLA® cream (Eutectic Mixture of Local Anaesthetics, AstraZeneca Pty Ltd, North Ryde, NSW, Australia) with intrasite conformable was used 45–60 min prior to each of the LFUD treatments for pain control during the procedure.

Due to the size of the wound and the amount of devitalised tissue, the initial LFUD treatment was 55 min in duration and the hoof sonotrobe head was chosen for the directional spray. Patient pain was minimal; hence the amplitude was set to a maximum effect of 100% for debridement with the normal saline flow (lavage) set at 20%.

The second LFUD treatment was 45 min in duration, using the hoof sonotrobe head with concurrent suction. The amplitude was decreased to 80% and the saline flow was increased to 40% due to the patient experiencing moderate pain. The third LFUD treatment was 30 min in duration, with simultaneous suction (with an amplitude of 100%) and flow adjusted to 40% according to the patient's tolerance. The final LFUD treatment was 20 min in duration using