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LETTERS TO THE EDITOR

Non-Invasive Monitoring of Compression Therapy: A Report of Three Cases With Venous Insufficiency

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Sir,

Skin oedema is believed to have a negative effect on wound healing and to contribute to skin necrosis and the development of leg ulceration (1, 2). To understand the pathologic significance of oedema, it is important to be able to determine the quantity of fluid content and its localization in the skin.

High-frequency ultrasonography visualizes oedema as areas of reduced echogenicity in skin images (3, 4). In the post-thrombotic syndrome and lipodermatosclerosis, the drop in echogenicity is present mainly in the subepidermal region; in lymphoedema, echogenicity decreases in the entire dermis, whereas in cardiac insufficiency echogenicity decreases in the lower portion of the dermis (5, 6). Since the upper part of the dermis is a site of intensive metabolism (7), oedema localized in this layer might be particularly deleterious.

In this study, we used high-frequency ultrasonography to quantify the reduction of dermal oedema after use of two types of compression bandages in three patients with venous insufficiency.

PATIENTS AND METHODS

Three patients (patient 1, a 66-year-old woman, and patients 2 and 3, a 37-year-old man and a 38-year-old man, respectively) with venous insufficiency (in the posterior tibial vein (observed via bi-directional Doppler in patient 1 and in the vena saphena magna (observed via colour Doppler in patients 2 and 3)) and lipodermatosclerosis were examined. Patients 1 and 2 had an ulcer located medially in the gaiter area, measuring 20 cm^2 and 2 cm^2, respectively, while in patient 3 an ulcer had healed prior to this study. Systolic arterial pressure of the first toe in all three legs was >60 mmHg (8).

A B-mode 20 MHz ultrasound scanner (Dermoscan C, Cortex Technology, Hadsund, Denmark) was used to determine the presence of dermal oedema after 24 h use of either a short-stretch compression bandage (Comprilan, Beiersdorf, Hamburg, Germany) or a long-stretch compression bandage (Setopress, Seton Health Care, Oldham, UK). The efficacy of the short-stretch bandage was studied first, and a week later the efficacy of the long-stretch bandage.

Subbandage pressure was measured with an Oxford pressure monitor (Talley, Romsey, Hants, UK) connected to three sensor cells taped to the skin at three levels on the medial aspect of the lower leg (4 and 8 cm above the malleolus and at the level of the widest circumference of the lower leg) before application of the bandage. The bandages were applied as previously described (9). The extension used for both types of bandage aimed at obtaining a subbandage pressure of 40 mmHg in the dependent and upright positions 4 cm above the medial malleolus, the pressure decreasing up the leg.

The subbandage pressure measurements were performed with the patient in an upright position immediately after application of the bandage and just before removal of the bandage 24 h later, with the sensor cells remaining in unchanged position beneath the bandage.

Ultrasound images were performed with the patient in the supine position immediately after removal of the bandage. In patient 1, ultrasound images were taken approximately 10 cm above the medial malleolus within the lipodermatosclerotic area in the vicinity of the ulcer on the left lower leg and in the same area on the contralateral leg without compression bandage (control measurements). In patients 2 and 3, ultrasound images were taken 5 cm above the medial malleolus.

The measurements were performed in the same locations during the second rounds as during the first (previously marked with a pen).

Cross-sectional images of the skin were obtained and evaluated using image analysis software (Cortex Technology, Hadsund, Denmark). The number of low echogenic pixels (LEP) representing fluid (3, 4) and the total number of pixels (TP) were determined in the dermal region, between epidermis and interface with the subcutaneous tissue. The number of LEPs and total pixel number (TP) was quantified separately in the upper (LEP_u and TP_u) and in the lower dermis (LEP_l and TP_l) by dividing the dermal region into two parts of equal thickness. LEP/TP was then calculated to avoid possible influence of area size and was taken as an arbitrary value for the degree of oedema.

RESULTS

In lipodermatosclerotic skin of patient 1, use of the long-stretch bandage for 24 h resulted in a 30% decrease in LEP/TP_l compared to the short-stretch bandage, indicating a lower degree of oedema. The corresponding figures for patients 2 and 3 were 20% and 16%, respectively. The difference was most pronounced in the deep part of the dermis, 52%, 34% and 29%, respectively (Fig. 1).

In patient 1, LEP/TP in total dermis was 13% more pronounced in the lipodermatosclerotic area following treatment with the short-stretch bandage than in the contralateral control skin, suggesting dermal oedema, while following treatment with the long-stretch bandage the oedema was 18% less pronounced than in the control skin. In patients 2 and 3, LEP/TP_l for the short-stretch bandage was 133% and 24% higher than in the control skin, respectively, indicating more pronounced oedema. For the long-stretch bandage the corresponding figures were 54% and 6% more than in the control skin, respectively. After the long-stretch bandage treatment in patient 3, LEP/TP_l was 8% lower in the deep part of the dermis compared to control skin,

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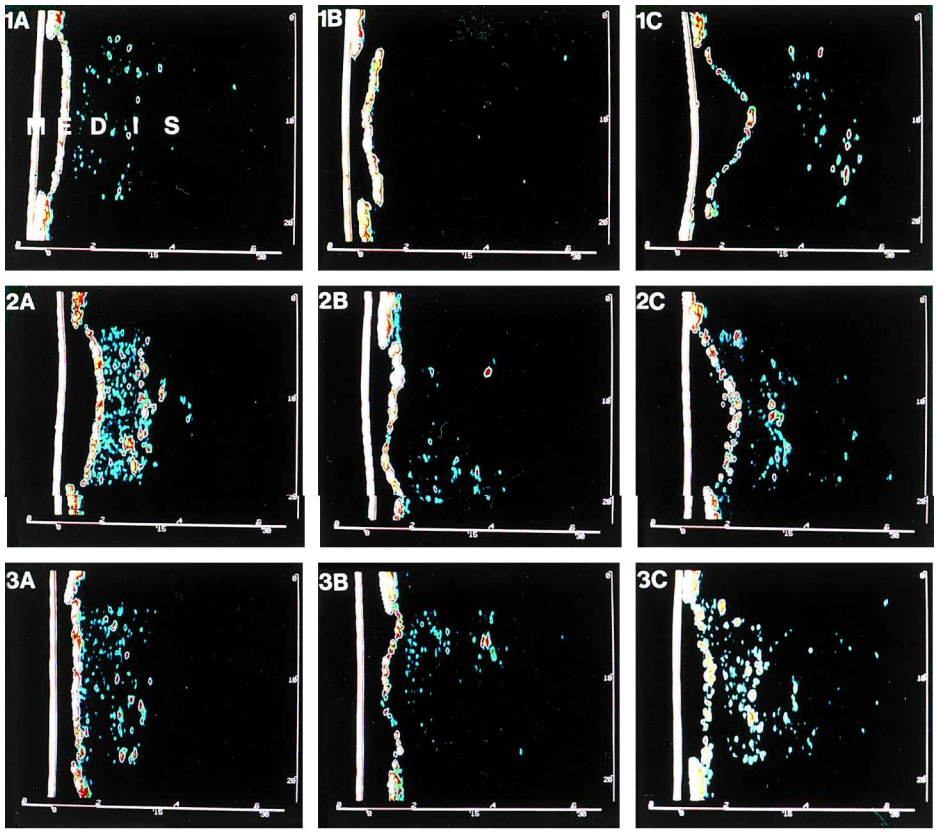

Fig. 1. Ultrasound images of the medial malleolar region in the three patients investigated (1–3). Normal skin of the contralateral leg (A); lipodermatosclerotic skin in the vicinity of an ulcer after treatment with the short-stretch bandage (B) and with the long-stretch bandage (C). M – membrane of the ultrasound probe; E – epidermal entrance echo; D – dermis; I – interface between dermis and subcutis; S – subcutis. Echogenicity: white > yellow > red > blue > black. Note the increase of echogenicity after compression therapy.

indicating less fluid content in the lower part of the dermis than in normal skin.

Measurements of the subbandage pressure showed the long-stretch bandage capable of maintaining a higher subbandage pressure than the short-stretch bandage. In the area 4 cm above the medial malleolus of patient 2, use of a short-stretch bandage resulted in a decrease of the subbandage pressure from 37 to 24 mmHg; the corresponding values for use of the long-stretch bandage were from 81 to 66 mmHg. In patient 3, use of the short-stretch bandage resulted in a decrease of the subbandage pressure from 39 to 20 mmHg, and of 39 to 33 mmHg with the long-stretch bandage. In patient 1 it was possible to measure only the subbandage pressure at the widest circumference because of the ulcer. Use of the short-stretch bandage resulted in a decrease in the subbandage pressure from 16 to 8 mmHg and of the long-stretch bandage from 16 to 14 mmHg.

DISCUSSION

Removal of oedema in skin and subcutaneous tissue together with prohibition of capillary leakage of macromolecules and cells by compression therapy has long been acknowledged as the major treatment modality in patients with leg ulceration and lipodermatosclerosis (1, 2, 10).

In this study, we have shown that effectiveness of various types of compression therapy can be individually compared by high-frequency ultrasonography. After 24 h of treatment, the long-stretch bandage was more effective in removing dermal oedema than the short-stretch bandage, particularly in the deep part of the dermis. It is remarkable that the long-stretch bandage in two patients was able to remove more oedema in the deep dermis than observed in the contralateral control skin, the skin probably being oedematous to some degree. These observations correlated well with the observed subbandage pressures underneath the two types of compression bandage, since the long-stretch bandage was able to maintain a higher pressure than the short-stretch bandage after 24 h. A subbandage pressure just below or above the 40 mmHg in the ankle area is suggested to be optimal during the upright position (10). The observed subbandage pressures are in agreement with a larger study showing Setopress maintaining a significantly higher subbandage pressure than Comprilan in the upright position and during passive dependency as well as during walking after both 2 h and 24 h (16). In the supine position, the subbandage pressure was found to decrease for both types of bandages, Setopress, however, obtaining a higher pressure (30 mmHg) than Comprilan (29–19 mmHg) (9).

None of the compression bandages applied for one day was able to totally remove the oedema of the upper dermis, when compared to the contralateral side. In two of the patients, however, less oedema was present following the long-stretch bandage than following the short-stretch bandage.

In the future, the non-invasive monitoring of compression therapy by high-frequency ultrasonography

could be helpful in evaluating the results of compression in individual patients. This method could also be useful for trials in which bandaging systems are compared.

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Atypical Autoimmune Blistering Dermatosis Associated with Sjögren's Syndrome

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Sir,

Sjögren's syndrome (SS) is a chronic autoimmune rheumatic disease characterized by dryness of the eyes and mouth resulting from lymphocytic infiltration of the lacrimal and salivary glands. In primary SS, these obligatory symptoms may be alone or accompanied by systemic manifestations (articular, renal, vascular, etc.), but no other well-defined connective tissue disease can be diagnosed on the basis of these systemic symptoms. In secondary SS, other rheumatic diseases, commonly rheumatoid arthritis or systemic lupus erythematodes (SLE), are coupled with the obligatory sicca symptoms (1, 2).

Immunopathologically, circulating autoantibodies, autoimmune inflammation, lymphocytic infiltration, and the consequent destruction of the affected tissues are the main features of the syndrome. Whereas bullous skin symptoms are commonly associated with generalized autoimmune diseases such as SLE (3, 4), we report here on a patient with primary SS in whom a blistering skin eruption developed, along with the presence of autoantibodies against type VII collagen.

CASE REPORT

A 56-year-old woman was admitted to the Department of Dermatology because of itchy, partly blistering skin eruptions in 1998. Her medical history included hypertension, thyroidectomy, subjective xerophthalmia and xerostomia, and since 1994 she had been followed for primary SS. At that time, ophthalmological examination indicated significantly reduced lacrimation (Schirmer's test: 1 mm/5 min) on both eyes. Because of the severe eye dryness, artificial stopping of the lacrimal ducts was performed. Salivary gland scintigraphy, together with the unstimulated and stimulated saliva production, revealed markedly decreased salivary function. The parotid gland sonography demonstrated a gross parenchymal inhomogeneity characteristic of primary SS (5). The abnormal laboratory findings included mild hypergammaglobulinaemia (21.1 g/l, normal value 7.5–18 g/l), IgM rheumatoid factor positivity (73.6 U/ml; normal value <14 U/ml), anti-SSA/Ro antibody positivity, and an elevated serum β_2-microglobulin level (13 μg/ml; normal value <2 μg/ml). Anti-dsDNA and anti-SSB/La autoantibodies were not detected. The diagnosis of primary SS was established in accordance with the European Economical Community Criteria (6, 7).

In 1998, she presented with itchy, partly blistering skin eruptions that had lasted for several weeks and showed no tendency to heal in response to local corticosteroid and antiseptic preparations. Because of her previously diagnosed diseases, she was regularly taking levothyroxine, nifedipine, chloroquine and bromhexine-hydrochloride, and she was using artificial tear.

On admission, physical examination revealed numerous 1–2 cm round or oval, reddish papules with or without tense blisters and excoriated erosions on her neck and upper trunk (Fig. 1). No exanthems or erosions were found on the mucous membranes, but a marked dryness was noted. The patient was otherwise in good general health. The clinical symptoms led