

Evidence that venous hypertension causes stasis dermatitis

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Abstract

A clinical model to examine the hypothesis that venous hypertension of the lower leg *per se* can cause lower leg stasis dermatitis is described. To prove this concept, we retrospectively studied a consecutive series of 38 patients with lower leg dermatitis who underwent phlebological examination at our consultation over a period of four years. Among those patients who had an insufficiency of the superficial veins only, without insufficiency of the deep veins, 22 had undergone patch testing to common allergens in phlebology. We found 10 patients with a stasis dermatitis of the lower leg and an incompetent great saphenous vein, six of whom had no detectable contact sensitization at all and another four exclusively to phlebologically irrelevant substances, e.g. nickel, cobalt, chromate or epoxid resin. All these 10 patients showed long saphenous vein incompetence from the groin to the medial aspect of the leg. All were operated by classical flush ligation and saphenectomy. Lower leg dermatitis healed in all 10 patients within 8–12 weeks and no recurrence was observed (1 year follow-up). These results support clinical experience that venous hypertension alone indeed can cause lower leg dermatitis.

Keywords: stasis dermatitis; chronic venous insufficiency; venous hypertension; varicose veins; contact dermatitis

Introduction

Most phlebologists would agree that lower leg dermatitis is a common condition among patients with chronic venous insufficiency (CVI). In the population-based cross-sectional Bonn Study, 3.7% of participating persons complained of eczema of the lower leg and 2.9% of examined persons showed lower leg skin changes in the context of CVI (stage C4, clinical, aetiological, anatomical and pathophysiological elements [CEAP] classification¹) including lower leg dermatitis.²

However, the existence of a pure form of stasis dermatitis with venous stasis as the exclusive

cause of lower leg dermatitis might be challenged. Lower leg dermatitis can be caused by several factors, such as CVI ('stasis dermatitis', corresponding to CEAP classification stage C4a),¹ but also by contact sensitizations ('allergic contact dermatitis'), 'xerosis cutis' (dry skin) corresponding to cumulative-toxic contact dermatitis in seborrheic skin, 'atopic dermatitis' and in many cases by combinations of several factors.

Patients with clinical stage C4–C6 (CEAP classification)¹ of CVI seem to carry a higher risk of contact sensitization, ranging from 37% to 85%.³ Three pathophysiological factors are likely to enhance sensitization rates in CVI patients: high penetration of allergens through the deteriorated epidermal barrier, particularly long and repetitive contact of potential allergens with the skin and application of particularly potent allergens in local therapy of dermatitis and chronic wounds.³ Venous ulcer patients (C5, C6) tend to have even higher sensitization rates, around 66%, a prevalence that remained

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