INTRODUCTION
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Chronic venous insufficiency of lower extremities is common (*Philips and Dover, 1991*). A common cutaneous finding in patients with venous disease is lipodermatosclerosis most typically of the lower extremities. It has been observed in up to 90% of patients who have had a thrombotic event leading to an abnormality of the deep venous system (*Kirsner et al., 1993*).

Many factors that contribute to the pathogenesis of stasis in patients with chronic venous insufficiency (CVI) are still unknown. However, trapping of leucocytes in the microcirculation of the lower limb may be important. As a result skin capillaries are obstructed, endothelial cells (ECS) are damaged and capillary permeability is increased (*Scott et al., 1991*).

Leucocyte trapping could be caused by:

1-An increased activation of leucocytes in CVI.

2-An increased expression of adhesion molecules on ECs and leucocytes. Among the many adhesion molecules expressed
on leucocytes and ECs, intercellular adhesion molecule-1 (ICAM-1), vascular cell adhesion molecule-1 (VCAM-1), leucocyte function-associated antigen-1 (LFA-1) and very late activated antigen-4, (VLA-4) appear to be of principal importance to regulate migration of leucocytes into tissue.

ICAM-1 is expressed on ECs and leucocytes and its expression is upregulated by cytokines produced during inflammation. ICAM-1 is a crucial factor in cellular adhesion events during cutaneous inflammatory responses by promoting leucocyte adhesion to ECs and their transmigration into tissues (Norris, 1990).

VCAM-1 a glycoprotein expressed by cytokine-activated ECs, is capable of mediating the adhesion of lymphocytes and monocytes (Elices et al., 1990).

Upregulation of ICAM-1 and VCAM-1 on ECs may contribute to the increased adherence and extravasation of leucocytes in chronic venous insufficiency (Weyl et al., 1996).