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 micro-circulation 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***).