SUMMARY

Chronic venous insufficiency of lower extremities is common. Many factors that contribute to the pathogenesis of stasis 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.

Leucocyte trapping could be caused by an increased activation of leucocytes in (CVI) and 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) appear to be of principal importance to regulate migration of leucocytes into tissue.

The aim of our work was to investigate dermatoliposclerotic skin with established CVI for the expression of the adhesion molecule ICAM-1. Thirty biopsy specimens of inflamed dermatoliposclerotic skin adjacent to venous leg ulcers were stained immunohistochemically with monoclonal antibodies against ICAM-1. Staining intensity was compared with that of normal skin.
SUMMARY

The results show that \textbf{ICAM-1} expression was seen in 17 cases out of 30 cases. The expression was present in various sites, in the endothelial cells of the capillaries, keratinocytes and inflammatory cells. It was seen mostly in capillaries at the edge of the ulcer and to a lesser degree in the capillaries away from the ulcer.

At the edge of the ulcer, it gave mostly a moderate degree of \textbf{ICAM-1} expression in 10 cases out of 17 cases and weak degree in 5 cases and high degree of expression in the other 2 cases. The capillaries in the skin away from the ulcer gave weak to negative expression similar to normal control.

In keratinocytes 11 cases out of 17 cases gave positive \textbf{ICAM-1} expression. In the inflammatory cells 8 cases gave positive \textbf{ICAM-1} expression.

In conclusion intercellular adhesion molecule-1 (ICAM-1) is important in the pathogenesis of chronic venous insufficiency disease and decreasing intercellular adhesion molecule-1 (ICAM-1) on endothelial cells and leucocytes could be a target for future pharmacological intervention in chronic venous insufficiency.