# Antithrombin-III Plasma Levels in Patients with Venous Leg Ulcer Disease

*Sir,*

In the development of venous leg ulcers, insufficiency of the deep leg veins is of primary pathogenic importance. The insufficiency often results from previous episodes of deep venous thrombosis, which phlebographically is demonstrable in over 80% of venous leg ulcer patients (1). Inherited deficiencies of anticoagulant proteins of the coagulation cascade, such as the protein C-protein S system, are among the risk factors which significantly predispose the affected individual to develop deep venous thrombosis and subsequently venous ulcers (2–5).

The significance of the anticoagulant protein antithrombin-III has not been investigated before in venous leg ulcer patients. Deficiency of antithrombin-III could be of primary importance, since antithrombin-III is a very potent physiological inhibitor of activated clotting factors IIa, Xa, IXa, XIa and XIIa, an action which is significantly amplified when heparin or other heparan sulphates are present (2–4).

During a 6-month inclusion period, we collected plasma samples from 46 unselected, consecutively admitted venous leg ulcer patients. Plasma concentrations of antithrombin-III were determined using a functional assay (3, 4), and concentrations were expressed in percentages of pooled, normal plasma. The mean plasma antithrombin-III concentration was 98% (SD 17%) in the 46 patients. None of the patients had antithrombin-III concentrations below 50%, which is indicative of inherited antithrombin-III deficiency (3, 4). Antithrombin deficiency does thus not appear to be a common risk factor for the development of venous leg ulcer disease.

## REFERENCES


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