ABSTRACT

Haemorheological and Blood Flow Abnormalities in Neuropathic Diabetic Subjects with and without Peripheral Vascular Disease

Jacqueline Eleanor Vigilance

Previous studies in diabetic patients have measured blood flow in the skin and foot and the rheological properties of blood independent of blood flow. No study has attempted to relate segmental blood flow in the diabetic neuropathic leg to rheological properties of blood. This study was designed to determine the role of vascular and haemorheological mechanisms in the pathogenesis of diabetic neuropathy. The relationship between blood flow in the leg and rheological determinants were investigated in an attempt to explain the development of diabetic neuropathy.

Arterial blood flow was measured by venous occlusion plethysmography. Flow was measured at the calf, ankle and great-toe in neuropathic diabetics with
(N = 14) and without (N = 28) peripheral occlusive arterial disease (POAD) and non-neuropathic diabetics with (N = 4) and without (N = 34) POAD. Ankle blood flow at rest and after reactive hyperaemia was used to derive the vasodilatory reserve (VDR) and debt recovery curve, which were used as indices of vascular abnormality. Blood flow, VDR and debt recovery in diabetics were compared with age and sex-matched non-diabetic controls (N = 21). Haemorheological variables were compared among the groups studied. Glycated haemoglobin levels were determined to assess glycaemic control in the diabetic patients.

The results showed a significant increase (p < 0.05) in ankle arterial blood flow, and plasma fibrinogen concentration (PFC), in all groups of diabetic patients studied. PFC was also significantly higher in diabetics with POAD than in those without POAD. Relative plasma viscosity (RPV) was significantly higher in diabetics with neuropathy or POAD than in C. There was a significant correlation between arterial blood flow and whole blood viscosity in neuropathic diabetics with and without POAD (p < 0.05).
Arterial blood flow, RPV and PFC were significantly different among diabetics with good, moderate and poor glycaemic control compared with each other and non-diabetic subjects. These variables were also significantly different in diabetics with disease of short (≤ 10 years) or long (> 10 years) duration.

VDR was significantly impaired in diabetics without POAD as compared with C (p < 0.05). The pattern of recovery was different in neuropathic diabetics without POAD compared with non-diabetic controls and other diabetic sub-groups. The findings of the present study suggest that vascular abnormalities, independent of POAD and neuropathy occur in diabetes. Peripheral vasodilatation occurs in diabetics, in the absence of neuropathy and in conditions of good glycaemic control. This vasodilatation may be attenuated as glycaemic control worsens. Additionally, rheological abnormalities, namely high PFC and RPV influence arterial blood flow and may have a role in the development of diabetic neuropathy. Finally, although POAD is associated with neuropathy, it does not contribute to the pathogenesis of diabetic neuropathy.