20 Plasma Exchange in Macroglobulinaemia

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20.1 Introduction

In 1944, Waldenström described a new syndrome in three patients who presented with a variety of haemorrhagic and ocular disorders associated with marked increases in both serum viscosity and in a serum globulin which had a very high molecular weight on ultracentrifugation; an underlying lymphoproliferative disorder involving the bone marrow was subsequently found in all three patients. In a recently published study of 40 cases of Waldenström's macroglobulinaemia (WM), one-third of the patients had clinical evidence of this hyperviscosity syndrome, (Mackenzie and Fudenberg, 1972). Some authors (Fahey et al., 1965; Mackenzie et al., 1970) have claimed that individual patients have a threshold value for blood viscosity above which they become symptomatic, while others (Pronk et al., 1969) could find no clear relation between either serum viscosity or paraprotein level and the clinical symptomatology. There is a similar divergence of opinion on the relation between IgM level and viscosity. Some workers have found a highly significant linear correlation between serum viscosity and IgM (Somer, 1966; 1975; Rosenblum and Asofsky, 1968), yet others have found a steep non-linear increase in serum viscosity, particularly with IgM levels above 40 g/litre (Preston et al., 1978). This discrepancy may in part be explained by the observation of Mackenzie and Babcock (1975) that a steep non-linear relationship is found in patients whose monoclonal paraprotein is markedly asymmetrical in configuration, while those with a more normal spheroidal IgM tend to exhibit a linear relationship. In our experience, there is a highly significant linear correlation between whole-blood viscosity and IgM level, at least up to 40 g/litre paraprotein (Fig. 20.1).
20.2 Methods

We have treated six patients with WM using an Aminco cell separator. There were three male and three female patients ranging in age from 62 to 80 years (mean 69.3) and on each occasion, 2.5-4.0 litres plasma was exchanged for stable Plasma Protein Solution (43 g/litre albumin solution). These patients all satisfied the criteria for WM in having serum IgM levels greater than 10 g/litre at presentation, along with characteristic marrow findings. The indications for plasmapheresis were symptoms referable to hyperviscosity, e.g. tiredness, exertional dyspnoea, early congestive cardiac failure and characteristic fundal changes.

Before and after each plasma exchange, measurements were made of immunoglobulins, by radial immunodiffusion, and of plasma and whole-blood viscosity at 37°C, using a Wells-Brookfield microviscometer. As fibrinogen levels are usually normal or slightly low in WM (Waldrenström, 1944) and as physiological variations in fibrinogen have a negligible effect on whole-blood viscosity unless at ultra-low shear rates (Begg and Hearns, 1966; Weaver et al., 1969) fibrinogen was not routinely assayed. All whole-blood viscosity measurements were corrected to a haematocrit of 0.30 (the mean Hct. value for the series) using Cokelet’s (1963) equation for red-cell suspensions,

$$\frac{\mu_s}{\mu_p} = \frac{1}{(1-H)^{2.5}}$$

where $\mu_s$ = viscosity of suspension; $\mu_p$ = viscosity of plasma; and $H$ = haematocrit.