Weight gain between dialyses in diabetics: possible significance of raised intracellular sodium content

Although well-timed renal transplantation, preferably using living related donors, offers the best chance of survival for diabetic patients who develop renal failure, regular haemodialysis (RDT) remains a valuable holding therapy. Diabetic patients on RDT face numerous problems, including difficulty in obtaining reliable access to the circulation and increased prevalence of sepsis related to shunts and fistulae. These patients also gain excessive weight between dialyses and, because of commonly-associated autonomic neuropathy, the ultrafiltration required with dialysis to correct this may cause undesirable hypotension. We have investigated weight gain between dialyses in a group of eight diabetic patients on RDT over a three-month period and compared them with a matched group of non-diabetic patients treated with RDT over the same period. After discovering grossly abnormal intracellular electrolyte concentrations in one of the diabetic patients we also studied leucocyte intracellular sodium content in a group of diabetic patients on RDT and peritoneal dialysis and compared them with a matched group of non-diabetic uraemic patients also on RDT. Six of the patients from the first part of the study were included in the second part.

Patients, methods, and results

The increase in weight between successive dialyses, separated by two to three days, was recorded over a three-month period. This was then expressed as a percentage of the patient's weight at the end of the previous dialysis and the mean obtained by dividing the total by the number of dialyses. Eight diabetic patients and eight non-diabetics were not significantly different in mean age, number of square metre hours of dialysis per week, urine output, or diuretic therapy. There was, however, a significantly greater intradialytic weight gain in the diabetic group (4-6% of body weight compared with 2-4%, p < 0.01). Intracellular sodium content was measured on peripheral blood leucocytes obtained immediately before dialysis. Leucocytes were separated from about 30 ml venous blood by the method of Baron and Ahmed, involving dextran sedimentation of erythrocytes. Trapped extracellular fluid was measured using a 14C EDTA marker. After the leucocytes were washed at 100°C to constant weight, treated with 0.1N HNO3 and electrolyte concentrations determined by flame photometry. Eight diabetic patients who had been on regular dialysis for at least three months, including two patients on peritoneal dialysis, were compared with eight non-diabetics on maintenance haemodialysis. The patients were matched in the same way as in the first group, and in particular there was no significant difference in sodium intake or serum albumin concentration between the two groups. A significantly higher intracellular sodium was found in the diabetic patients (mean ± SD = 143 ± 6 86 mmol/kg dry cell weight v 76 ± 30.3 mmol/kg, p < 0.025).

Comment

These observations confirm that the intradialytic weight increases in diabetics on dialysis, although this report is the first accurately to quantify this. High intracellular sodium content and concentration accompanied by a raised cell water and low intracellular potassium has been described in uraemia and ascribed to impairment of the ouabain-sensitive sodium pump. These abnormalities return to normal with regular dialysis therapy, but this has clearly not occurred in the diabetics we have studied since their intracellular sodium remain high. It has been suggested that hyperglycaemia and possibly high concentrations of circulating angiotensin and aldosterone are responsible for increased thirst and weight gain in diabetics on dialysis. But our results suggest an alternative explanation, since possibly the high white cell sodium content may be mirrored in the central nervous system and acts as a "false signal" to the thirst centres. The failure of regular haemodialysis to reverse this abnormality in diabetics remains unexplained.

Relapses in primate malaria: discovery of two populations of exerythrocytic stages. Preliminary note

Shortt and Garnham's discovery in 1948 of exerythrocytic schizosporonts of Plasmodium cynomolgi in the liver of the rhesus monkey 102 days after the inoculation of sporozoites and just before the onset of a relapse led them to postulate that successive cycles of development in the liver were responsible for the "true" relapse. Further work (see review) suggested that this idea was invalid, and the alternative theory of dormancy was substituted. In fact, in 1946 Shute had already suggested that the sporozoite, or "x body," into which it grew might remain inactive as a resting stage in the host. Apart from the nearly mature exerythrocytic schizosporonts which have continued