Comment

In this case conventional investigations⁴ did not show any aetiological cause for the patient's adverse reaction, but a penicillinase-sensitive penicillin was detected in the donor blood unit.

The patient's history, the symptoms that appeared after the injection of cephazolin, and the results of the rat mast-cell degranulation test leave little doubt about the diagnosis of hypersensitivity to the β -lactam antibiotics. The symptoms appearing after the transfusion of the blood unit containing penicillin were identical with those observed after the injection of cephazolin and consistent with an allergic reaction to penicillin.^{2,3} These findings indicate that the reaction in our patient was related to the presence of penicillin in the transfused blood unit.

Screening of donors at our blood bank includes questioning about the use of drugs. This case shows that this method is not reliable enough and that the presence of penicillins in blood units might induce reactions after transfusion. We suggest that blood units are tested for penicillins, especially when the recipient is known to be allergic to them. Testing is also warranted when conventional methods fail to show any reason for an adverse reaction after transfusion.

¹ Michel J, Sharon R. Detection of penicillins in the sera of "healthy" blood donors. *Vox Sang* in press.

² Cluff, LE, Johnson JE. Antimicrobial therapy. In: *Clinical concepts of infectious diseases*. Baltimore: Williams and Wilkins, 1972:296-314.

³ Pratt WB. The inhibitors of cell wall synthesis. In: *Fundamentals of chemotherapy*. London: Oxford University Press, 1973:84-118.

⁴ Grumet FC, Yankee RA. Non-cell reactions. In: *New approaches to transfusion reactions*. Washington: American Association of Blood Banks, 1974:39-52.

⁵ Perelmutter L, Khera K. A study on the detection of human reagins with rat peritoneal mast cells. *Int Arch Allergy Appl Immunol* 1970;39:27-44.

(Accepted 12 October 1979)

Hadassah University Hospital, Mount Scopus, Jerusalem, Israel

J MICHEL, MD, senior lecturer, department of clinical microbiology

R SHARON, PHD, lecturer in haematology, blood bank

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 failure¹ regular haemodialysis (RDT) remains a valuable holding therapy. Diabetic patients on RDT face numerous problems,² 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 dialyses² 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

interdialytic 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,³ involving dextran sedimentation of erythrocytes. Trapped extracellular fluid was measured using a ⁵¹Cr EDTA marker. After isolation cells were dried 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 $\pm$ SD = 143.4 $\pm$ 68.6 mmol/kg dry cell weight v 76.2 $\pm$ 30.3 mmol/kg, $p < 0.025$).

Comment

These observations confirm that the interdialytic 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,⁵ but this has clearly not occurred in the diabetics we have studied since their intracellular sodiums remain high. It has been suggested that hyperglycaemia and possible high concentrations of circulating angiotensin and aldosterone are responsible for increased thirst and weight gain in diabetics on dialysis.² 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.

¹ Sommer BG, Sutherland DER, Simmons RL, Howard RJ, Najarian JS. Prognosis after renal transplantation: cumulative influence of combined risk factors. *Transplantation* 1979;27:4-7.

² Watkins PJ, Parsons V, Bewick M. The prognosis and management of diabetic nephropathy. *Clin Nephrol* 1977;7:243-249.

³ Baron DN, Ahmed SA. Intracellular concentration of water and of the principal electrolytes demonstrated by the analysis of isolated human leucocytes. *Clin Sci* 1969;37:205-219.

⁴ Edmondson RPS, Hilton PJ, Jones NF, Patrick J, Thomas RD. Leucocyte sodium transport in uraemia. *Clin Sci Mol Med* 1975;49:213-216.

⁵ Patrick J, Jones NF. Cell sodium, potassium and water in uraemia and the effect of regular dialysis as studied in the leucocyte. *Clin Sci Mol Med* 1974;46:583-590.

(Accepted 23 October 1979)

Renal and Liver Units, King's College Hospital, London SE5

ROGER JONES, MA, MRCP, research fellow

LUCILLA POSTON, BSC, research biochemist

HUMBERTO HINESTROSA, MD, research fellow

VICTOR PARSONS, DM, FRCP, consultant physician

ROGER WILLIAMS, MD, FRCP, consultant physician

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

Shortt and Garnham's¹ discovery in 1948 of exoerythrocytic schizonts 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 review²) suggested that this idea was invalid, and the alternative theory of dormancy was substituted. In fact, in 1946 Shute³ 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 exoerythrocytic schizonts which have continued