

ABNORMAL INSULIN RESPONSE IN A URAEMIC PATIENT TREATED WITH HYPERTONIC PERITONEAL DIALYSIS—A CASE REPORT

By P. H. Feng

SYNOPSIS

Disturbances in carbohydrate metabolism are known to occur in uraemia. Both hyperglycaemia and hypoglycaemia are seen occasionally after peritoneal dialysis. A patient is described in whom hyperglycaemia developed during hypertonic peritoneal dialysis. The exhibition of insulin resulted in prolonged hypoglycaemia which was not aborted with intravenous glucose. Possible mechanisms are discussed.

INTRODUCTION

Peritoneal dialysis is an accepted method for the treatment of acute and chronic renal failure. Hypertonic glucose solutions are frequently used in peritoneal dialysis to remove oedema. Hyperglycemia, hyperosmolar coma and fits have been described in some of these patients. (Boyer *et al*, 1967; Raja *et al*, 1970; Whang, 1967).

We report below a patient in whom hyperglycemia and fits developed during the course of peritoneal dialysis with hypertonic glucose solutions. The exhibition of insulin resulted in prolonged hypoglycaemia which was not aborted by large doses of 50% glucose or a continuous infusion of 10% dextrose and this persisted for 24 hours. Spontaneous hypoglycaemia after both peritoneal dialysis (Greenblatt, 1972) and haemodialysis (Gutman *et al*, 1967; Rigg et Bercu, 1967) have also been reported.

CASE REPORT

A massively oedematous 31-year old Chinese male with chronic renal failure secondary to nephrotic syndrome was admitted on 4.6.72 with oliguria and acute pulmonary oedema. 400 mgm. of intravenous frusemide and 200 mgm. of oral ethacrynic acid were given with total urine output of only 290 ml. Peritoneal dialysis was instituted on 6.6.72 for removal of excess fluid. 1 litre of dialysis fluid (500 ml. containing 1.5% glucose and 500 ml. containing 7% glucose) was used at each exchange. Because of technical difficulties only about 1.5 litres of fluid were removed at the

end of 15 exchanges. Nevertheless there was some subjective and objective improvement. Before dialysis the blood sugar was 142 mgm. % and blood urea was 426 mgm. %. After 15 exchanges the blood urea had fallen to 285 mgm. % and blood sugar rose to 300 mgm. %. On 7.6.72 at the end of 23 exchanges patient developed repeated fits which could only be controlled with intravenous diazepam (Valium). The sudden development of fits was thought to be either a reverse urea effect or a hyperosmolar state. Prior to this, patient had complained of severe thirst, nausea and vomiting. A repeat blood sugar was 420 mgm. %. Serum osmolarity was 339 mOs/L and C.S.F. osmolarity was 343 mOs/L. A single dose of 32 units of soluble insulin was given. Peritoneal dialysis was continued with 1.5% glucose solution. In order to prevent further hyperglycemic episodes 16 units of soluble insulin were added to each litre of dialysis fluid. A total of 22 such exchanges were carried out. A blood sugar done 6 hours later was 214 mgm. %. At this time the patient was semi-conscious and could take fluids orally. The fits had stopped.

The next morning (8.7.72) repeat blood sugar was 152 mgm. %. Patient's condition showed further improvement. At about mid-night on 9.7.72 he was found in coma and sweating profusely. Blood sugar done was 40 mgm. % and blood urea 159 mgm. %. Peritoneal dialysis was terminated. 100 ml. of 50% glucose was given intravenously and a 10% dextrose drip was set up. Despite a total of 200 ml. of hypertonic glucose intravenously and continuing 10% dextrose infusion, the blood sugar remained at 30-40 mgm. % for the next 24 hours. He remained in coma and developed respiratory infection followed by respiratory embarrassment. The trachea was intubated and assisted ventilation utilizing the Bennet PR2 respirator was undertaken. He failed to respond to vigorous therapy and died 16 days after admission. The blood sugar had returned to normal

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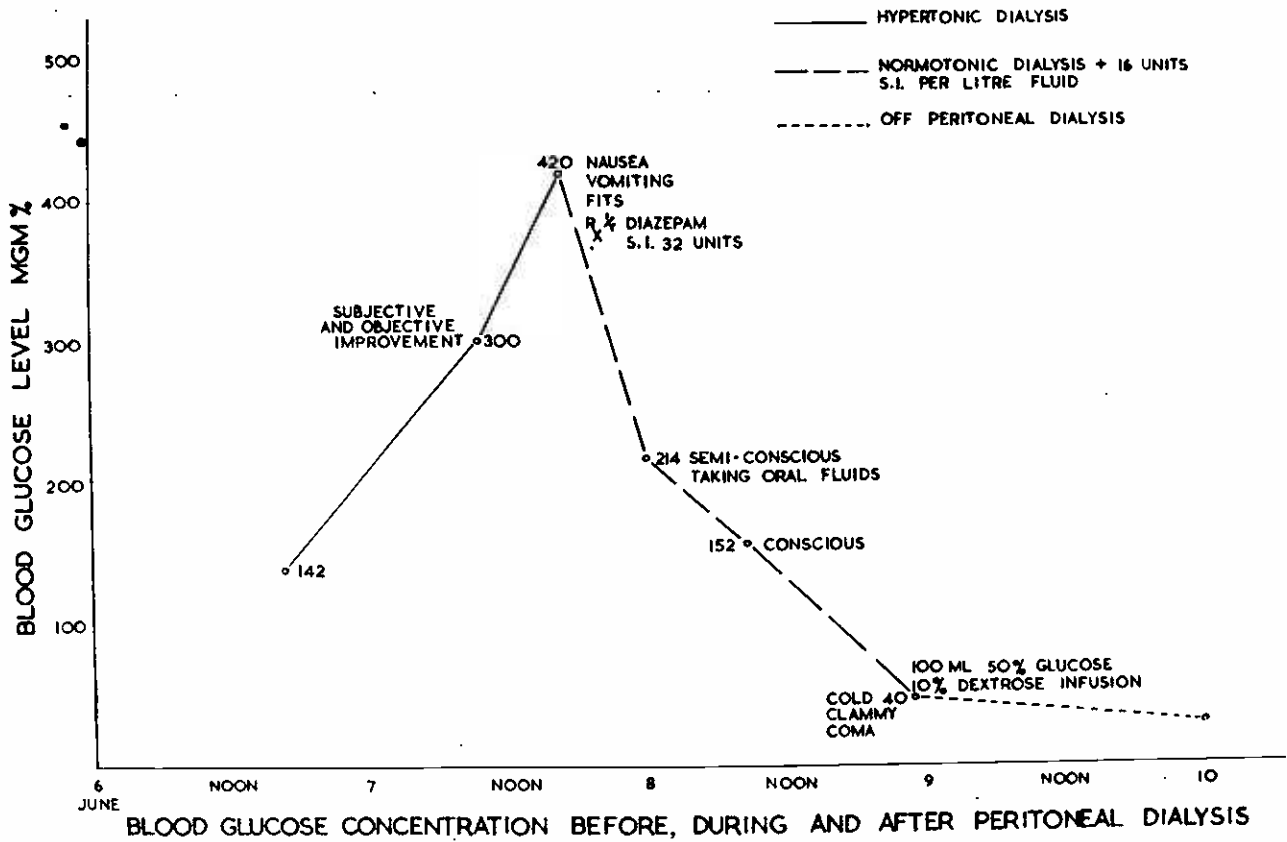


Fig. 1.

2 days after termination of peritoneal dialysis. Permission for post mortem was refused. Details of blood glucose concentration before, during and after peritoneal dialysis are shown in Fig. 1.

COMMENTS

Abnormal carbohydrate metabolism is known to occur in renal insufficiency (Perkoff *et al*, 1958). A number of theories have been put forward to explain this deficit. Horton (1968) and Rabkin (1970) have demonstrated that the azotaemic patient responds to carbohydrate loading with normal or supranormal plasma insulin levels. Westervelt (1969) implicated peripheral insulin resistance as the cause of this metabolic defect. The metabolism of insulin itself is also altered in renal insufficiency (Rubenstein and Spitz, 1968). In the healthy individual the kidney is the major site of degradation of insulin once it has passed through the liver. When functioning renal tissue is destroyed to the degree that severe azotaemia occurs, degradation of insulin is severely impaired resulting in abnormally prolonged hypoglycaemic effect. This could possibly be the explanation in our case. Nolph *et al* (1970) in a number of elegant studies demonstrated a wide range of glucose absorption rates from peritoneal dialysis solutions in 13 uraemic patients. Hence it is not possible to prognosticate the blood sugar level in any individual patient on hypertonic peritoneal dialysis.

Hyperglycaemia is also a fairly common complication in peritoneal dialysis in diabetic patients (Ribot *et al*, 1966; Chazan *et al*, 1969). This would result in early symptoms of thirst, nausea, vomiting and subsequently fits, coma and finally death. Crossley (1971) recommended the use of intraperitoneal insulin for control of blood sugar in such patients. It would appear therefore at the present stage of knowledge constant clinical observation and repeated blood sugar monitoring especially in diabetic patients or patients subjected to hypertonic peritoneal dialysis are mandatory.

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