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Acute renal failure and hypoglycaemia due to sulphadiazine poisoning

A. W. CRAFT
M.B. B.S., M.R.C.P.

J. T. BROCKLEBANK
M.B. B.S., M.R.C.P.

R. H. JACKSON
M.A., B.M., B.Ch., F.R.C.P.

Department of Child Health, Royal Victoria Infirmary, Newcastle upon Tyne NE1 4LP

Summary

A 3-year-old girl is described who became oliguric and hypoglycaemic whilst receiving an excessive dose of sulphadiazine. The mechanism of the hypoglycaemia is discussed.

Introduction

Acute renal failure due to sulphonamide administration is now uncommon. This report describes a patient who became oliguric and hypoglycaemic whilst receiving treatment with an excessively high dose of sulphadiazine for meningococcal septicaemia.

Case report

A 3-year-old girl weighing 13 kg was admitted to another hospital, having been generally unwell for 12 hr, and then became comatose and developed a purpuric rash. Meningococcal septicaemia was subsequently proved when the organism was grown from the blood, but not from the cerebrospinal fluid. She was treated with i.v. fluids, hydrocortisone, penicillin and sulphadiazine (450 mg/kg body-weight/24 hr). Her condition improved over the next 48 hr and the dose of sulphadiazine was reduced

to 300 mg/kg/24 hr. Urine output, having been normal, was noted to be much less on days 4 and 5 and because of the appearance of haematuria, the sulphadiazine was stopped. The blood urea was 12 mmol/l but the following day rose to 30 mmol/l and she was transferred to this hospital for further management. On admission she was found to be oedematous and semi-conscious. The blood urea was 30 mmol/l, the sugar less than 1 mmol/l, plasma insulin 22 mu./ml, plasma cortisol 800 nmol/l and the blood sulphonamide 700 mg/l (therapeutic level 120 mg/l). Urine examination showed red cells and typical sulphadiazine crystals. She did not regain consciousness after infusion of 10 g of glucose. Peritoneal dialysis was commenced and her clinical condition slowly improved, the blood urea, glucose and sulphonamide levels reverting to normal as in Fig. 1. However, her urine output did not improve and cystoscopy revealed aggregations of sulphonamide deposits around both ureteric orifices. Bilateral ureteric catheters were inserted and a 2.5% sodium bicarbonate solution was infused at the rate of 2.5 ml/hr in each renal pelvis. Shortly afterwards, she began to pass large volumes of urine, the catheters were removed and she continued with an uneventful convalescence from her illness. At follow-up 2 months later she was physically and mentally normal, a ⁵¹Cr-EDTA clearance was 120 ml/min/1.73 m² and i.v. pyelogram was normal.

Correspondence: Dr A. W. Craft, Department of Child Health, The Royal Victoria Infirmary, Newcastle upon Tyne NE1 4LP.

FIG. 1. Clinical data, laboratory results and treatment of the patient.

Discussion

Anuria due to sulphonamides has been attributed to direct renal toxicity, to a hypersensitivity reaction or to an obstructive uropathy due to precipitation of insoluble crystals in the urinary tract (Merril, 1971). The findings on cystoscopy in this patient, and the prompt diuresis which occurred when the crystals were removed, suggest that the anuria was due to the latter mechanism as in a similar case recently reported by Winterborn and Mann (1973). Dorfman and Smith (1970) have reviewed the urological aspects of this problem. Hypoglycaemia was an unexpected finding and its association with an inappropriately elevated plasma insulin level suggests that there may have been direct stimulation of the beta cells of the pancreas by the high levels of sulphadiazine in the blood. Hypoglycaemia may occur in diabetic patients treated simultaneously

with sulphonamides and a sulphonyl urea compound (Christensen, Hansen and Kristensen, 1963), but occurs rarely due to sulphonamides alone, there being only one previous report of a child becoming hypoglycaemic after taking sulphonamides (Alain *et al.*, 1966). An alternative explanation for the hypoglycaemia is that high levels of sulphadiazine may have interfered with the glucose oxidase-peroxidase method used for the estimation of the blood sugar, as may occur in some cases of paracetamol poisoning (Burn, 1973). This is unlikely as Sharp (1972) found no interference between sulphadiazine and the 'GOD-Perid' method of glucose determination used by the present authors. The dose of sulphadiazine given to the patient presented in this report was very high and if sulphonamides do have a place in the therapy of bacterial infections, then a soluble preparation, e.g. sulphadimidine, should be used in a maximum dose of 150 mg/kg/24 hr, and the maintenance of a high urine flow rate is recommended (Weinstein, 1970).

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