Lesson of the Week

Association of hyperglycaemia with hyponatraemia

EWAN WALTERS, LAVINIA HALLAM

Hyperglycaemia is a well documented cause of hyponatraemia.^{1 2} Patients with poorly controlled diabetes with a plasma glucose concentration greater than 11.0 mmol/l (198 mg/100 ml) have a lower mean plasma sodium concentration than diabetics with a plasma glucose concentration less than 11.0 mmol/l.³ We noted that hyperglycaemia is rarely considered to be a possible cause during the initial investigation of hyponatraemia. We therefore determined the incidence of hyperglycaemia (plasma glucose concentration greater than 11.0 mmol/l) associated with hyponatraemia (plasma sodium concentration less than 130 mmol (mEq)/l) in samples received for estimation of urea and electrolyte concentrations in the laboratory of a teaching hospital.

Methods and results

Plasma glucose concentrations were measured in the blood samples (a total of 3319) received for estimation of urea and electrolyte concentrations during three non-consecutive weeks that had a plasma sodium concentration less than 130 mmol(mEq)/l. Those with pseudohyponatraemia resulting from visible hypertriglyceridaemia were excluded. Plasma glucose concentrations were also measured in a control group of 256 samples selected at random from those with plasma sodium concentrations between 130 and 144 mmol/l. All samples were collected into heparinised tubes and separated within three hours to minimise any decrease in plasma glucose concentration due to utilisation of glucose by red cells.⁴ Plasma sodium concentrations were measured with either a Technicon SMA 6-60 or a Beckman electrolyte 2 ion selective electrode. These use the principles of flame photometry and indirect potentiometry respectively, and the results obtained with both methods are therefore comparable.⁵ Plasma glucose concentrations were measured with an Analox GM-6 glucose analyser. The difference in plasma glucose concentrations between the control and hyponatraemic samples was assessed with an unpaired Student's *t* test after logarithmic transformation of the data.

Of the 3319 samples, 148 (4.5%) had a plasma sodium concentration less than 130 mmol/l. These came from 108 patients, of whom 19 (17.6%) had a glucose concentration greater than 11.0 mmol/l (198 mg/100 ml) (table). In contrast, only 17 patients (6.6%) in the control group had a plasma glucose concentration greater than 11.0 mmol/l. The difference in the incidence of hyperglycaemia between the two groups was significant (*p* < 0.001).

Clinical details were obtained for the 19 patients with combined

Plasma concentrations of glucose in 19 patients with hyponatraemia and hyperglycaemia (figures in parentheses are numbers of patients with previously unsuspected diabetes)

Glucose (mmol/l):	11-15	15.1-20	20.1-25	25.1-30	> 30
No of patients:	9 (2)	5	3	1	1 (1)

Conversion: SI to traditional units—Glucose: 1 mmol/l $\approx$ 18 mg/100 ml.

Department of Medical Biochemistry, University Hospital of Wales, Heath Park, Cardiff CF4 4XW

EWAN WALTERS, MB, CHB, registrar
LAVINIA HALLAM, MB, CHB, senior house officer

Correspondence to: Dr Ewan Walters.

Hyperglycaemia is common in patients with hyponatraemia

hyperglycaemia and hyponatraemia: nine of them were receiving intravenous fluids. Of these nine patients, five were known to have diabetes mellitus, and three of these were taking diuretics. Two patients had plasma glucose concentrations of 13.2 and 12.2 mmol/l (238 and 220 mg/100 ml) respectively while receiving infusions of 5% dextrose after surgical procedures and were subsequently shown not to have diabetes mellitus: one sample had been taken from the patient's drip arm, and the remaining patient died before hyperglycaemia could be confirmed.

The 10 other patients were not receiving intravenous fluids. In this group eight patients had diabetes mellitus, which was previously unsuspected in three. None of the new diabetics was clinically dehydrated or had ketonuria, but one was taking diuretics. Three of the five known diabetics were also taking diuretics. Of the two others, one was subsequently shown to have a normal fasting glucose concentration and the other died before hyperglycaemia could be confirmed.

Comment

As a raised plasma glucose concentration by itself can be a major cause of hyponatraemia,³ and as unsuspected diabetes mellitus is common in the general hospital population,⁶ it is perhaps surprising that the importance of measuring plasma glucose concentrations in the initial assessment of hyponatraemia is not more frequently emphasised.

Nineteen patients (17.6%) in our group with hyponatraemia had hyperglycaemia, and of these, 13 (68.5%) had diabetes mellitus, which was previously unsuspected in three.

Our report suggests that the measurement of plasma glucose concentration should always be part of the initial investigation of patients with hyponatraemia so that the correct treatment for both the hyponatraemia and hyperglycaemia may be started without unnecessary delay.

We thank R G Newcombe (department of medical statistics, Welsh National School of Medicine) for his help.

References

- Welt LG. *Clinical disorders of hydration and acid base equilibrium*. London: J & A Churchill, 1955:118-22.
- Flear CTG, Gill GV, Burn J. Hyponatraemia: mechanisms and management. *Lancet* 1981;ii:26-31.
- McNair P, Madsbad S, Christiansen C, Christiansen MS, Transbøl I. Hyponatraemia and hyperkalaemia in relation to hyperglycaemia in insulin-treated diabetic out-patients. *Clin Chim Acta* 1982;120:243-50.
- Sazama K, Robertson EA, Chesler RA. Is antiglycolysis required for routine glucose analysis? *Clin Chem* 1979;25:2038-9.
- Levy GB. Determination of sodium with ion selective electrodes. *Clin Chem* 1981;27:1435-8.
- Northam BE, Smith JH, Fitzgerald MG, Natrass M, Wright AD. Value of serum glucose assay as part of the biochemical profile for screening for diabetes. *Ann Clin Biochem* 1982;19:412-5.

(Accepted 7 September 1983)