An unconscious diabetic patient

Y Ozawa, H Maruyama, S Nakano, T Saruta

A 49-year-old man was transferred to our hospital by ambulance on 25th December 1995, for unconsciousness, hypotension, and hypothermia. The patient was unemployed and living alone. He had been well until a year earlier, when polyuria, polydipsia and weight loss (15 kg/4 months) developed. He had experienced a sore throat and a cough productive of yellowish sputum 4 days earlier, but had not consulted any doctors. His mother had suffered from non-insulin-dependent diabetes mellitus (NIDDM). On his admission, physical examination showed him to be in deep coma and his rectal temperature was 29.2°C. His body mass index was 20, blood pressure was 54/26 mmHg, heart rate was 62 beats/min, and respiratory rate was 24 breaths/min. His pupils were dilated and right reflex was sluggish, but his deep tendon reflexes were normal. Laboratory data revealed that his plasma glucose (48.3 mmol/l), acetoacetate (4540 µmol/l, normal 18–83 µmol/l), and 3-hydroxybutyrate (10500 µmol/l, normal 0–106 µmol/l) were markedly elevated. His urine was positive for ketone bodies. Arterial blood gas analysis showed severe metabolic acidosis (pH 6.783, pO₂ 133.7 mmHg, pCO₂ 10.9 mmHg, HCO₃ 3.4 mEq/l). His plasma potassium and phosphate levels were raised to 6.2 mmol/l and 2.9 mmol/l, respectively. There were no abnormal findings in his chest X-ray. His electrocardiogram (ECG) is shown in the figure.

**Department of
Internal Medicine,
Keio University School
of Medicine, Tokyo,
Japan**

Y Ozawa
H Maruyama
S Nakano
T Saruta

**Department of
Internal Medicine,
National Okura
Hospital, Tokyo, Japan**
Y Ozawa

Correspondence to
Yukako Ozawa, Department
of Internal Medicine,
National Okura Hospital,
2-10-1 Okura, Setagaya,
Tokyo 157, Japan

Accepted 10 March 1998

Figure Electrocardiogram

Questions

- 1 What is the diagnosis and appropriate treatment?
- 2 What does the ECG show?
- 3 What further examinations are necessary?

Answers

QUESTION 1

The patient was diagnosed as suffering from diabetic ketoacidosis associated with severe hypothermia. Proper treatment consists of active external and core rewarming, hydration, and continuous intravenous administration of insulin. When the patient's temperature rises above 32°C, the activity of insulin will be normalised. Therefore, after rewarming, precautions against hypoglycaemia should be taken. Antibiotic treatment is also indicated.

QUESTION 2

The figure shows a positive deflection at the end of the QRS complex which is called J wave, a typical manifestation of hypothermia. It was apparent in V3-V5 leads in this case. The J wave is seen when the body temperature drops below 32°C.²

QUESTION 3

The following further examinations are necessary to determine the type of diabetes:

- glucagon test to examine insulin secretion by the pancreas
- immunological markers such as cytoplasmic islet cell antibodies (ICA), auto-antibodies to insulin (IAA), glutamate decarboxylase 65 (GAD65 Ab) and islet cell antigen 512 (ICA512 Ab)
- HLA typing.

Discussion

Patients with diabetic ketoacidosis sometimes have a low body temperature, even when infection is present, although hypothermia rarely occurs. Hypothermia associated with diabetic ketoacidosis may arise for several reasons,¹ such as peripheral vasodilatation caused by acidosis, abnormal plasma osmolality (due to hypothalamic interference), or inability to maintain body temperature in cold ambient temperatures.

In a hypothermic state, catecholamine and cortisol secretions increase, which leads to insulin resistance. Insulin release and its activity are markedly reduced when a patient's body temperature drops below 32°C,¹ therefore, hypothermia itself is considered to aggravate diabetic ketoacidosis. Hypothermia may also irritate the myocardium. Premature atrial contractions occurred in our patient (data not shown) whose rectal temperature was 29.2°C. Hypothermic patients with diabetic ketoacidosis must be rewarmed carefully, as well as treating the diabetic ketoacidosis. Severely hypothermic patients may have hypovolaemic hypotension due to cold diuresis, and there may be a temperature

Resuscitation of hypothermic patients

- prevention of additional heat loss
- maintenance of tissue oxygenation
- rewarming: passive external, active external, and active core
- treat precipitating and underlying disease

Diagnosis of IDDM

- evaluation of islet function
- glucagon test
- immunological markers: ICA, IAA, GAD65 Ab, ICA512 Ab
- genetic markers: HLA typing

gradient between the core and periphery of the body. If we rewarm only the periphery, vasodilatation of the periphery will occur which may aggravate hypotension. In addition, rapid shunting of cold blood from the periphery to the core, as the result of the vasodilatation, may cause a drop of the core temperature. Therefore, proper hydration and rewarming the core as well as the periphery are important; we used heated intravenous fluid and gastric lavage with heated water (active core rewarming), in addition to a heated blanket (active external rewarming).

After recovering from the critical state, we performed a glucagon test which revealed that the patient's ability to secrete insulin was preserved; the serum C-peptide level 6 min after the glucagon injection was 1 nmol/l. He had neither GAD65Ab nor ICA512Ab; ICA, IAA, and HLA typing were not examined. Although ICA and IAA are the classical immunological markers for a diagnosis of IDDM, the combination of the new markers, GAD65Ab and ICA512Ab, is gradually replacing the classical markers.³ The methods of detection of these antibodies are much easier than that of ICA, and GAD65 Ab is also useful for diagnosis of slowly progressive IDDM.⁴ When treating patients with slowly progressive IDDM who initially did not need insulin therapy but are slowly progressing into an insulin deficiency state, it is considered better to avoid using oral antidiabetic agents. This patient was diagnosed as NIDDM clinically and serologically, and discharged with good glycaemic control using an oral antidiabetic agent.

Final diagnosis

Diabetic ketoacidosis associated with severe hypothermia.

Keywords: diabetic ketoacidosis; hypothermia; resuscitation

1 Krentz AJ, Natrass M. Diabetic ketoacidosis, non-ketotic hyperosmolar coma and lactic acidosis. In: Pickup J, Williams G, ed. *The textbook of diabetes*. Oxford: Blackwell Scientific Publications, 1991; pp 479–94.

2 Miller JW. Hypothermia. In: Kravis TC, Warmer CG, Jacobs LM, eds. *Emergency medicine*, 3rd edn. New York: Raven Press, 1993; pp 661–77.

3 Wiest-Ladenburger U, Hartmann R, Hartmann U, Berling K, Böhm BO, Richter W. Combined analysis and single-step

detection of GAD65 and IA2 autoantibodies in IDDM can replace the histochemical islet cell antibody test. *Diabetes* 1997;46:565–71.

4 Kasuga A, Ozawa Y, Maruyama T, Ishihara T, Amemiya S, Saruta T. Autoantibody against ICA512 did not improve test sensitivity for slowly progressive IDDM in adults. *Diabetes Care* 1997;20:679–80.