A Chinese Patient Presenting with Clinical Signs of Fulminant Type 1 Diabetes Mellitus

Kazumi KATSUMATA and Kazuo KATSUMATA

Abstract

A 31-year-old Chinese woman suffering from excessive thirst and appetite loss consulted our hospital. Laboratory examination revealed a high blood glucose level and evidence of diabetic ketoacidosis, however, the serum HbA1c was normal. The 24-hour urinary excretion of C-reactive protein and the glucagon stimulation test indicated the loss of endogenous insulin secretion. The patient satisfied the criteria for the diagnosis of fulminant type 1 diabetes mellitus. However, she tested positive for serum anti-IA2 antibody even though the titer was low. We considered it worthwhile reporting this case, because very few cases of fulminant type 1 diabetes mellitus among Chinese people have been published.

Key words: Chinese, fulminant type 1 diabetes mellitus

Introduction

Recently, the concept of fulminant type 1 diabetes mellitus has been proposed as a new subtype of type 1 diabetes mellitus (1, 2), in which sudden destruction of the pancreatic β cells results in the loss of endogenous insulin secretion. These patients are usually negative for serum islet cell-related antibodies. Fulminant type 1 diabetes mellitus has been reported to account for about 20% of all Japanese cases of type 1 diabetes mellitus (3), however, only very few non-Japanese subjects with this condition have been reported to date. We recently encountered a Chinese patient showing clinical signs of fulminant type 1 diabetes mellitus, and report the case in this paper.

Case Report

The patient was a 31-year-old woman who presented with the chief complaints of vomiting and fatigue. Her past and family medical history was unremarkable. She had been born in Fujian Province, China, and had moved to Japan two years previously. On May 30, 2004, she began to suffer from headache, fatigue, sensation of heat, excessive thirst, and loss of appetite. On June 1, 2004, she developed frequent vomiting, and difficulty in eating. She consulted our hospital on June 2, 2004.

Physical findings on admission

She was 156 cm tall and weighed 50.8 kg, her BMI was 20.9 kg/m², her blood pressure 92/50 mmHg, her pulse 109/min (regular), her respiratory rate 20/min, and her body temperature was 36.5°C at this time. She was fully conscious and alert. Physical examination did not reveal any thyroid enlargement, and the chest and abdominal examinations were unremarkable. Her breathing was normal, with no indication of Kussmaul sign. Neurological examination also did not reveal any abnormalities.

Laboratory findings on admission (Table 1)

Urine was positive for both glucose and ketone bodies. Arterial blood gas analysis revealed a pH of 7.203 and HCO₃⁻ of 6.8, and the B.E. was –18.8, indicative of metabolic acidosis. Blood biochemical examination revealed a high blood glucose level (547 mg/dl), although the HbA1c was normal (5.6%). The serum amylase level (62 IU/l, reference range 40 to 150 IU/l) was within normal range. Slight elevation of the enzyme levels secreted from the exocrine pancreas was noted (lipase 144 IU/l, reference range 40 to 150 IU/l, elastase 440 IU/l, reference range 100 to 400 IU/l). Abdominal CT revealed no significant abnormality. As for autoantibodies, the serum was negative for anti-GAD antibody and anti-ICA antibody; however, anti-IA2 antibody was positive (2.5 U/ml, reference range <1.0 U/ml) even

From the Department of Internal Medicine, Katsumata Hospital, Nagoya
Received for publication December 1, 2004; Accepted for publication April 16, 2005
Reprint requests should be addressed to Dr. Kazumi Katsumata, the Department of Internal Medicine, Katsumata Hospital, 32-22 1-chome, Shinsakae, Naka-ku, Nagoya 460-0007

Internal Medicine Vol. 44, No. 9 (September 2005) 967
Clinical course after admission (Fig. 1)

After admission, indicating the loss of endogenous insulin secretion. The fasting serum CPR was 0.12 ng/ml before stimulation, and 0.36 ng/ml six minutes after glucagon stimulation. In the present case, diabetic ketoacidosis developed 4 days after the appearance of symptoms of hyperglycemia, and endogenous insulin secretion was almost completely undetectable throughout the patient’s hospital stay. The HbA1c at admission was within the normal range despite hyperglycemia. Thus, this patient satisfied the above-mentioned criteria for the diagnosis of fulminant type 1 diabetes mellitus, like the present case (4). On investigation of a nationwide survey in Japan, anti-GAD-antibody-positive cases have been reported although the titer was low. The fasting serum CPR was 0.12 ng/ml, and the 24-hour urinary excretion of CPR on August 4, 2004 was 4.4 µg/day, indicating the continuous loss of endogenous insulin secretion. Because she was not covered by National Health Insurance in Japan, she wished to return home for further treatment, and left Japan for China on August 20, 2004.

Discussion

Imagawa et al recently proposed the concept of fulminant type 1 diabetes mellitus as a subtype of idiopathic type 1 diabetes mellitus, which seems unlikely to involve autoimmune mechanisms (1, 2). The committee of Japan Diabetes Society has proposed the following criteria for the diagnosis of patients of fulminant type 1 diabetes mellitus: 1) development of ketosis or ketoacidosis within about one week after the appearance of symptoms of diabetes mellitus, 2) blood glucose of more than 288 mg/dl and HbA1c less than 8.5% during the first examination, and 3) urinary CPR at disease onset of less than 10 µg/day, or serum CPR of less than 0.3 ng/ml before glucagon stimulation, and less than 0.5 ng/ml after glucagon stimulation. In the present case, diabetic ketoacidosis developed 4 days after the appearance of symptoms of hyperglycemia, and endogenous insulin secretion was almost completely undetectable throughout the patient’s hospital stay. The HbA1c at admission was within the normal range despite hyperglycemia. Thus, this patient satisfied the above-mentioned criteria for the diagnosis of fulminant type 1 diabetes mellitus. Other characteristics of this case included slight elevation in the serum levels of enzymes secreted from the exocrine pancreas and a positive test for the anti-IA2 antibody (even though the titer was low). Chuo et al reported an anti-GAD-antibody positive case showing the clinical symptoms of fulminant type 1 diabetes mellitus, like the present case (4). On investigation of a nationwide survey in Japan, anti-GAD-antibody-positive cases have been reported to account for about 5% of fulminant type 1 diabetes mellitus (3). Although the mechanism underlying the sudden injury of the pancreatic β cells associated with fulminant type 1 diabetes mellitus remains unknown, the presence of autoantibody-positive cases among these subjects suggests that autoimmunity may be involved in some types of fulminant type 1 diabetes mellitus. It is well known that the incidence of type 1 diabetes mellitus differs greatly among different districts or races (5). However, few reports of fulminant type 1 diabetes mellitus have been published to date from countries other than Japan (e.g., Europe and USA) (6). Chinese people are known to have a relatively low incidence of type 1 diabetes mellitus, like the Japanese people. However, our literature search revealed no published report of fulminant type 1 diabetes mellitus from China, except for one report of a doubtful case (7). It is unknown whether the small number of cases of fulminant type 1 diabetes mellitus reported from abroad reflects the low incidence of this condition in these countries or the fact that the diagnosis tends to be overlooked despite a comparable incidence to that in Japan. It would,
therefore, be desirable to define better diagnostic criteria for fulminant type 1 diabetes mellitus, so that a reliable comparison of its prevalence can be made between Japan and other countries.

References