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Fulminant Type 1 Diabetes Mellitus Associated with Excessive Alcohol Use: A Case Report

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Abstract

We present a case of fulminant type 1 diabetes that is associated with excessive alcohol use. The patient was admitted due to our hospital due to refractory diarrhoea. His blood glucose level was 24.6 mmol/L and he was positive for urine ketone bodies. Laboratory examinations meets all the diagnostic criteria for fulminant type 1 diabetes. Immediate treatment actively reduced the blood glucose level and corrected his electrolyte disturbance and acid-base imbalance. Fulminant type 1 progresses rapidly and the prognosis is extremely poor. Therefore, early diagnosis and treatment are important to the outcomes.

Keywords: Diabetes; Autoimmunity; Pancreatic cells

Introduction

Fulminant type 1 diabetes is a new sub-type of type I diabetes. It is characterized by an intrinsic insulin deficiency resulting from a markedly and severe destruction of pancreatic cells. Because this disease progresses rapidly, early detection is critical for initiating timely interventions. Here we report a rare case of Fulminant type I diabetes associated with excessive alcohol use.

Case Presentation

A 34-year-old man Chinese man presented to our hospital due to diarrhoea for 8 days. He had no significant past medical history. There was no fever, abdominal pain, nausea or vomiting on admission. The patient has a normal body mass index (20.1 kg/m^2) with a height of 174 cm and a weight of 61.5 kg. After admission, a physical examination was performed and the results were as follows: Temperature (T), 37.1°C; Pulse Rate (PR), 102 bpm; Respiration Rate (R) 20/min; Blood Pressure (BP), 118/86 mmHg.

The patient was conscious but showed slow verbal responses. There were no obvious skin rashes or other symptoms. Clear breathing was heard in both lungs. The heart rhythm was irregular. Arrhythmia including audible and premature beats was detected. Routine admission laboratory tests performed immediately, and the results were summarized in Table 1. Peripheral blood examination revealed elevated high blood glucose (24.6 mmol/L), ketosis, elevated leukocyte count, metabolic acidosis with a pH level of 7.29, and elevation of liver function tests. The urinalysis showed a very high level of sugar (3+), presence of protein (+) and Ketone bodies (1+) in his urine. The patient was suspected as Fulminant Type 1 diabetes mellitus and received immediate treatment. Treatment includes administration of adequate liquid infusion, intravenous injection of regular insulin to reduce blood glucose, and correction of electrolyte disturbance, acid-base imbalance. 12 h after admission, the urinary ketone bodies were negative, and the blood Ph increased to 7.37. To further control his glucose level, the patient was admitted to our department. On day 2, the HbA1c was 5.2%. Laboratory examinations were repeated on day 2, day 4, week 1 and week 2 (Table 1). On week one, the patient received the Oral Glucose Tolerance Test (OGTT), the Insulin Releasing Test (IRT) and C-peptide release test (CRT) (Table 2). The results of these tests further confirmed the increased glucose level and

Cell dysfunction resulting in decreased insulin secretion in this patient. On week two, results from the blood chemistry tests showed normal liver and kidney function suggesting that the liver and kidney damages are transient and controlling the glucose level is able to reverse liver and kidney damages. Negative results were obtained from urine amylase test, blood lipase test, thyroid gland function test, blood coagulation test, stool culture, and measurement of the islet autoantibodies including Islet Cell Antibody (ICA), Glutamic Acid Decarboxylase (GAD) and Insulin Autoantibodies (IAA). After 16 days' treatment, the patient was discharged and continued with insulin (34 u/d) treatment for six months.

Discussion

Fulminant Type 1 diabetes mellitus is a novel subtype of Type 1 diabetes that was first reported in Japan in 2000 [1]. This subtype is characterized by its sudden onset of diabetic ketoacidosis, absence of insulin secretion and diabetes-related antibodies and high serum pancreatic enzyme concentrations [1,2]. In China, fulminant type 1 diabetes accounts for about 10% of the ketosis-onset type 1 diabetes cases [3]. The etiology and pathogenesis of Fulminant type 1 diabetes are still not fully understood. Several factors such as viral infection, genetic susceptibility, autoimmunity, pregnancy and others seem to be involved [4,5]. In our case, the patient has a long history of excessive alcohol use (200 g of pure alcohol a day for 8 years. The patient presented to our hospital with persistent diarrhoea without abdominal pains. According to the classification of diabetes mellitus by the American Diabetes Association or the World Health Organization [6,7], Fulminant type 1 diabetes has the following clinical characteristics:

(i) Duration of hyperglycemic symptoms is 4 days on average;

(ii) There is a high prevalence of preceding common-cold-like and gastrointestinal symptoms;

(iii) There is a near-normal level of glycated hemoglobin (HbA1c) in spite of very high plasma glucose levels associated with ketoacidosis;

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Test	Results					Normal Range
Routine Blood Analysis	**Day 1**	**Day 2**	**Day 4**	**Week 1**	**Week 2**	
WBC	11.22 × 10^9/L	10.32 × 10^9/L	8.4 × 10^9/L	NA	9.48 × 10^9/L	(3.5-9.5) × 10^9/L
N%	90%	89%	69%	NA	61.20%	40-75%
HGB	112 g/L	99 g/L	127 g/L	NA	109 g/L	110-160 g/L
PLT	68 × 10^9/L	58 × 10^9/L	149 × 10^9/L	NA	191	125-350 × 10^9/L
Blood Chemistry Tests						
ALT	401 U/L	305 U/L	184 U/L	116 U/L	27 U/L	9-50 U/L
AST	664/L	574 U/L	86 U/L	89 U/L	45 U/L	15-40 U/L
GGT	1423 U/L	1567 U/L	1354 U/L	1274 U/L	45 U/L	10-60 U/L
Urea	19.36 mmol/L	22.5 mol/L	6.6 mmol/L	2.4	NA	2.8-8.2 mmol/L
CRE	394.7 umol/L	347 umol/L	99 umol/L	48.4	NA	44-133 umol/L
GLU	24.46 mmol/L	20.46 mmol/L	8.3	6.38	6.38 mmol/L	3.89-6.11 mmol/L
TG	4.85 mmol/L	2.93 mmol/L	NA	NA	NA	0.23-1.7 mmol/L
LDH	815 U/L	NA	213	NA	NA	120-250 U/L
CK	4634 U/L	1402 U/L	230 U/L	57 U/L	22 U/L	50-310 U/L
CKMB	NA	29.3 U/L	13.0 U/L	13.4	9	0-24 U/L
CRP	257.25 mg/L	169.88 mg/L	68.36 mg/L	17.15 mg/L	13.93 mg/L	0-3 mg/L
Na+	141.3	129.6 mmol/L	NA	NA	NA	137-147 mmol/L
HbA1c	NA	5.20%	NA	NA	NA	5-6.3%
UA	1131 u/L	975 u/L	NA	239 u/L	237 u/L	208-428 u/L
ESR	41 mm/h	NA	NA	NA	32	0-20 mm/h
Blood Gas Analysis						
PH	7.29	7.4	NA	NA	NA	7.35-7.45
BE	-15.2	-10	NA	NA	NA	-2-3 mmol/L
Routine Urine Analysis						
PH	5.5	NA	5	NA	NA	5-7.5
Glucose	3+	NA	-	NA	NA	-
Protein	1+	NA	-	NA	NA	-
Ketone bodies	1+	NA	-	NA	NA	-

NA: Not Available

Table 1: Results of the laboratory tests.

Time (min)	OGTT (Glucose, mmol/L)	IRT (Insulin, uIU/mL)	CRT (C-peptide, ng/mL)
0	6.38	2.38	2.38
30	10.47	4.79	4.79
60	14.39	6.4	6.8
120	17.65	6.16	11.16
180	12.46	5.97	8.36

OGTT: Oral Glucose Tolerance Test; IRT: Insulin Release Test; CRT: C-Peptide Releasing Test

Table 2: Results of OGTT, IRT and CRT on week 1.

(iv) The disease is sometimes related to pregnancy; and

(v) There are increased serum pancreatic enzyme levels, absent C-peptide levels, but virtually no detectable autoantibodies against constituents of pancreatic beta cells.

In our case, the patient is:

(ι) A young male with normal BMI and no family history of diabetes;

(ii) Characterized by the rapid onset of disease;

(iii) Has signs of gastrointestinal infection caused by excessive drinking;

(iv) Finding on admission includes hyperglycemialevel but normal HbA1c level;

(v) ketoacidosis at onset and transiently increased liver and kidney function tests, muscle enzymes and uric acid;

(vi) After correction of ketoacidosis, symptoms improved;

(vii) The levels of C-peptide and insulin were significantly reduced with the progression of the disease;

(viii) During treatment with insulin, the blood glucose levels of the patient Fluctuated significantly.

Conclusion

Treatment of the disease is similar to autoimmune diabetes. Once diagnosed with ketoacidosis, it should be treated immediately. After the acute phase treatment, long-term glycemic control by insulin therapy is required. Because the multi-organ damages including liver, kidney, heart, muscle in these patients are fully reversible, it is extremely important monitor their liver and kidney function electrolytes, muscle enzymes, pancreatic enzymes and treat accordingly once the diagnosis is established. In our case, 4 days after treatment, results from routine blood test and kidney function tests were normal. One week after

treatment, his blood uric acid level returned to a normal value. Two weeks after treatment, the liver function was fully recovered. FT1D progresses rapidly and the prognosis is extremely poor. Compared with classic type 1 diabetes, these patients usually require high doses of insulin and are more prone to hypoglycemia and acute complications. Therefore, early cc and treatment are important to the outcomes.

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