Aggravation of type 2 diabetes mellitus triggered by acute exacerbation of chronic thyroiditis (Hashimoto’s disease)

A 73-year-old woman who had 25-year history of type 2 diabetes and 1-year history of chronic thyroiditis (Hashimoto’s disease) consulted Kawasaki Medical School, Okayama, Japan, for loss of appetite and weight loss (4.0 kg/1 month). Three days earlier, she had fever and left neck pain, and she started taking antibiotics. To examine her clinical condition, she was hospitalized in our institution. Her body temperature was 36.6°C, and blood pressure was 122/50 mmHg.

Laboratory data were as follows: thyroid-stimulating hormone, <0.010 μIU/mL (normal range 0.400–6.000 μIU/mL); free T3, 9.53 pg/mL (2.50–4.20 pg/mL); free T4, 3.79 ng/dL (0.80–1.60 ng/dL); anti-thyroid-stimulating hormone receptor antibody, <0.1 IU/L (<0.1 IU/L); thyroid-stimulating antibody, 102% (0–120%); anti-thyroid peroxidase antibody, 248.2 IU/mL (<16.0 IU/mL); anti-thyroglobulin antibody, 912.8 IU/mL (<28.0 IU/mL); decreased radioactive 99mTcO4 uptake to the thyroid gland (0.2%); white blood cell count, 7030/μL (neutrophil 69.8%); red blood cell count, 401 × 10^6/μL; hemoglobin, 11.5 g/dL; platelet, 29.6/μL; and C-reactive protein, 0.34 mg/dL. Her thyroid was slightly enlarged in both lobes (right > left), and slightly hard at the tender region. Thyroid ultrasonography showed an inhomogeneous pattern and hypervascular pattern with an adenomatous goiter and non-toxic nodular goiter, but no cysts in the entire thyroid area. We did not carry out a thyroid biopsy because of the hypervascular pattern in the thyroid gland. However, the C-reactive protein level and neutrophil count were almost normal, and the patient did not have hypoechoic lesions in the painful portion of the thyroid. Thereby, we finally diagnosed her as acute exacerbation of chronic thyroiditis. Although at first she was considered to have Graves’ disease and she had 15 mg/day of thiamazole for 5 days, we stopped thiamazole, and 2 weeks later her thyroid hormone levels became normal. After then, she developed hypothyroidism, and she required 25 μg/day of levothyroxine (Figure 1).

On admission, the patient’s height, bodyweight, and body mass index were 159.0 cm, 47.0 kg and 18.59 kg/m², respectively. Her glycemic control was poor (hemoglobin A1c (HbA1c), 8.1%; glycoalbumin, 24.3%), although her HbA1c levels were approximately 7% for over 1 year with the treatment of 25 mg/day of alogliptin, 750 mg/day of metformin, and 0.9 mg/day of voglibose. Fasting and postprandial plasma glucose were 168 and 230 mg/dL, respectively, just before starting insulin therapy. Her insulin secretory capacity was preserved; serum C-peptide immunoreactivity index was 1.15, and urinary C-peptide immunoreactivity excretion was 56.6 μg/day before starting insulin therapy. During the acute exacerbation of chronic thyroiditis, we treated her with insulin therapy. As she had the same oral

Figure 1 | Clinical time-course. Increased serum thyroid hormones and decreased thyroid-stimulating hormone (TSH) were observed on admission. Two weeks later, the patient’s thyroid hormone levels were within the normal range, and she developed hypothyroidism approximately 1 month later. Her hemoglobin A1c (HbA1c) level and glycoalbumin level decreased for approximately 4 weeks, which was very much correlated with the improvement of chronic thyroiditis. FT3, free T3; FT4, free T4; GA, glycoalbumin.
hypoglycemic agents and her glycemic control was good for over 1 year, we believe that the aggravation of type 2 diabetes was triggered by acute exacerbation of chronic thyroiditis. Anti-glutamic acid decarboxylase antibody and anti-insulin antibody were negative. Anti-nuclear antibody, myeloperoxidase-anti-neutrophil cytoplasmic antibody (ANCA) and proteinase 3-ANCA were within the normal range. Her glycoalbumin level decreased from 24.3 to 18.5% for approximately 4 weeks, which very much correlated with the improvement of chronic thyroiditis (Figure 1). These data support that aggravation of type 2 diabetes was triggered by acute exacerbation of chronic thyroiditis.

It is known that type 1 diabetes is induced or aggravated after the onset of Graves’ disease as a result of an abnormal immune response in pancreatic β-cells. In addition, type 2 diabetes is induced or aggravated after the onset of Graves’ disease presumably as a result of an increase of glucose uptake in the intestine and/or increase of insulin resistance in insulin target tissues. In patients with Graves’ disease, hyperthyroidism plays a central role in the aggravation of glycemic control. Therefore, we believe that in the present case, hyperthyroidism worsened her glycemic control. The present case clearly shows that glycemic control in type 2 diabetes is possibly related to a transient change in hyperthyroidism in patients with chronic thyroiditis.

Taken together, we should keep in mind that acute exacerbation of chronic thyroiditis could lead to the disturbance of glycemic control in patients with type 2 diabetes.

**DISCLOSURE**

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