World Journal of Diabetes

Online Submissions: http://www.wjgnet.com/1948-9358office
wjd@wjgnet.com
doi:10.4239/wjd.v1.i4.135

World J Diabetes 2010 September 15; 1(4): 135-136
ISSN 1948-9358 (online)

Autoimmune polyglandular syndrome type 3 complicated by mineralocorticoid-responsive hyponatremia of the elderly

Hidekatsu Yanai, Seiko Okamoto, Junwa Kunimatsu

Hidekatsu Yanai, Seiko Okamoto, Junwa Kunimatsu, Department of Internal Medicine, Kohnodai Hospital, National Center for Global Health and Medicine, Chiba 272-8516, Japan
Author contributions: Yanai H contributed to the conception, writing and editing of the letter; Okamoto S and Kunimatsu J treated the patient presented in the letter, and contributed to the data collection.
Correspondence to: Hidekatsu Yanai, MD, PhD, FACP, Department of Internal Medicine, Kohnodai Hospital, National Center for Global Health and Medicine, Chiba 272-8516, Japan. dyanai@hospk.ncgm.go.jp
Telephone: +81-47-3733501 Fax: +81-47-3721858
Received: July 20, 2010 Revised: August 24, 2010
Accepted: August 31, 2010
Published online: September 15, 2010

Abstract

We experienced the first case with autoimmune polyglandular syndrome type 3 (anti-thyroid peroxidase antibody-positive hypothyroidism and anti-glutamic acid decarboxylase antibody-positive diabetes) complicated by mineralocorticoid-responsive hyponatremia of the elderly. This case is also a rare slowly progressive insulin-dependent diabetes mellitus (SPIDDM) case, for which the patient has been treated for many years with sulfonylurea or glinide. Our observation also demonstrated that glucose metabolism in autoimmune diabetes such as SPIDDM is influenced by appetite, thyroid function and glucocorticoid effect.

Key words: Anti-glutamic acid decarboxylase antibody; Autoimmune polyglandular syndrome; Mineralocorticoid-responsive hyponatremia of the elderly; Slowly progressive insulin-dependent diabetes mellitus

Peer reviewer: Hendrik-Jan Schuurman, PhD, Professor, University of Minnesota, Schulze Diabetes Institute, 101 Marquette Avenue South, #3103, Minneapolis, MN 55401, United States

Yanai H, Okamoto S, Kunimatsu J. Autoimmune polyglandular syndrome type 3 complicated by mineralocorticoid-responsive hyponatremia of the elderly. *World J Diabetes* 2010; 1(4): 135-136 Available from: URL: http://www.wjgnet.com/1948-9358/full/v1/i4/135.htm DOI: http://dx.doi.org/10.4239/wjd.v1.i4.135

TO THE EDITOR

Slowly progressive insulin-dependent diabetes mellitus (SPIDDM) occurs generally in adulthood, and usually progresses to the insulin-dependent stage within several years[1]. Since SPIDDM shows a better preserved residual beta-cell function, it is often misclassified as type 2 diabetes[2]. Autoimmune diabetes such as SPIDDM is due to the destruction of beta cells by an immune-mediated process that may be modified by the interaction of genetic and environmental factors, but this remains unknown. We experienced a SPIDDM patient having experienced many years of the insulin-independent stage, whose glucose metabolism was modified by complications including chronic thyroisitis and mineralocorticoid-responsive hyponatremia of the elderly (MRHE) and the treatments for these diseases.

An 83-year-old woman, who developed diabetes at 60 years of age, was at first treated with glibenclamide (2.5 mg/d) but has been treated with nateglinide (60 mg/d) for the last three years. Her hemoglobin A1c level was 5.9% in March, 2009. She complained of general fatigue and appetite loss in October, 2009. Laboratory data showed hypothyroidism and also showed positive for the anti-thyroid peroxidase antibody, suggesting the existence of chronic thyroiditis. She showed euthyroid by levothyroxine (25 μg/d), however, she still could not eat. She did not take nateglinide for two months, and her hemoglobin A1c level decreased to 5.7% in December, 2009. She showed hyponatremia (129 mmol/L), and was admitted to our hospital. Her body height, body weight,

and body mass index were 126 cm, 26 kg, and 16.4 kg/m^2, respectively. Hypotonic (serum osmolarity, 265 mOsm/L) hyponatremia, elevated urine osmolality (urine osmolarity, 469 mOsm/L), normal cardiac, hepatic, renal and adrenal functions all suggested the existence of a syndrome of inappropriate secretion of the anti-diuretic hormone (SIADH). However, mild dehydration and deterioration by water restriction indicated the development of MRHE[3]. Hydrocortisone (10 mg/d) improved her appetite, general condition and hyponatremia (140 mmol/L), and she left the hospital in March, 2010. She was again admitted to our hospital due to a fracture in May, 2010. Laboratory data revealed hyponatremia (129 mmol/L), euthyroid, and elevated hemoglobin A1c (6.3%) and she tested positive for the anti-glutamic acid decarboxylase antibody (GADab) (3.2 U/mL, normal range < 1.5 U/mL). Urinary C-peptide levels had decreased to 9.1 μg/d. After we found that she was GADab-positive, we changed her medication from hydrocortisone to fludrocortisones (0.02 mg/d) for the treatment of MRHE because the ratio of glucocorticoid effect to mineralocorticoid effect in hydrocortisone and fludrocortisones is 1 : 1 and 1 : 12.5, respectively. This change ameliorated blood glucose levels [119, 218, 135, 185, 152, 139 (hydrocortisone use), 101, 167, 104, 159, 167, 135 (fludrocortisones use) mg/dL at before and after breakfast, before and after lunch, after lunch and before bed, respectively], and hyponatremia (135 mmol/L) after a week. She was finally diagnosed as having autoimmune polyglandular syndrome (APS) type 3 (autoimmune thyroid diseases and type 1 diabetes)[4], and MRHE.

To our knowledge, our patient is the first case with APS type 3 complicated by MRHE, and is also a rare case having been treated with insulin secretagogues for many years of insulin-independent state. Further, our observation demonstrated that glucose metabolism in SPIDDM is largely influenced by appetite, thyroid function and glucocorticoid effect.

REFERENCES

1 **Kobayashi T**, Tamemoto K, Nakanishi K, Kato N, Okubo M, Kajio H, Sugimoto T, Murase T, Kosaka K. Immunogenetic and clinical characterization of slowly progressive IDDM. *Diabetes Care* 1993; **16**: 780-788

2 **Zimmet P**, Turner R, McCarty D, Rowley M, Mackay I. Crucial points at diagnosis. Type 2 diabetes or slow type 1 diabetes. *Diabetes Care* 1999; **22** Suppl 2: B59-B64

3 **Ishikawa S**, Saito T, Fukagawa A, Higashiyama M, Nakamura T, Kusaka I, Nagasaka S, Honda K, Saito T. Close association of urinary excretion of aquaporin-2 with appropriate and inappropriate arginine vasopressin-dependent antidiuresis in hyponatremia in elderly subjects. *J Clin Endocrinol Metab* 2001; **86**: 1665-1671

4 **Neufeld M**, Maclaren N, Blizzard R. Autoimmune polyglandular syndromes. *Pediatr Ann* 1980; **9**: 154-162

S- Editor Zhang HN **L- Editor** Herholdt A **E- Editor** Liu N