Transient Appearance of Lactate Dehydrogenase (LDH)-linked Immunoglobulin and Thyroid Dysfunction at the Postpartum Period

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Abstract

Here, we report a 28-year-old woman who transiently showed lactate dehydrogenase (LDH)-linked immunoglobulin during postpartum thyroiditis. She demonstrated high levels of serum LDH (794 IU/*l*) and thyroid hormones 7 months after delivery. Electrophoretic isoenzyme analysis of LDH showed an abnormal broadband caused by LDH-linked immunoglobulin (IgG-κ). Transient thyrotoxicosis due to postpartum thyroiditis improved without any specific treatment, and elevated serum concentration of LDH decreased to the normal level (395 IU/*l*) with disappearance of LDH-linked IgG. LDH-linked immunoglobulin may also appear at the postpartum period.
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Key words: LDH anomaly, postpartum thyroiditis, autoimmune thyroid disease

Introduction

Lactate dehydrogenase (LDH) is distributed in various organs. The level is affected by cell injury, and usually increases in the serum. However, an elevated level of serum LDH has been found in some cases without tissue injury, and is often associated with LDH anomaly. Most cases of LDH anomaly have been reported to be induced by LDH-linked immunoglobulin (1). Although LDH anomaly has been found in patients with various diseases including thyroid diseases (1, 2), there is no report describing LDH anomaly in patients with postpartum thyroiditis.

Here, we describe the first case of transiently LDH-linked immunoglobulin in the patient who showed postpartum transient thyroid dysfunction and briefly review the literature related to LDH-linked immunoglobulin.

Case Report

The patient was a 28-year-old woman who had given birth to her first child on January 14, 1999. When she consulted a hospital for a common cold in August 1999, high levels of serum LDH (794 IU/*l*) and thyroid hormones were detected. Therefore, she was referred to our hospital for further examination in September 1999. She had not consumed excess iodine in food, medicine or contrast media, and had not taken any thyroid hormones.

On physical examination at the first consultation, the thyroid gland was slightly enlarged without neck pain or tenderness. Serum concentrations of free thyroxine, free triiodothyronine and thyroid stimulating hormone (TSH) were 1.87 ng/dl (normal range between 0.7 and 1.48 ng/dl), 4.91 pg/ml (normal range between 1.71 and 3.71 pg/ml) and less than 0.1 μU/ml (normal range between 0.35 and 4.94 μU/ml), respectively. There was neither anti-thyroid peroxidase, anti-thyroglobulin nor anti-TSH receptor antibodies. Although thyroid scintigraphy was not performed because she was lactating, she was diagnosed as having transient thyrotoxicosis induced by postpartum thyroiditis. Her thyroid function was normalized without a hypothyroid state in December 1999.

The serum LDH level at the first consultation was also elevated at 899 IU/*l* (normal range between 100 and 450 IU/*l*). Other serum enzymes were within normal limits (Table 1). Electrophoretic isoenzyme analysis of LDH showed an abnormal broad band caused by the formation of LDH-linked immunoglobulin (IgG-κ, Fig. 1). Thereafter, the serum LDH level decreased to the normal level (395 IU/*l*) at the euthyroid state in September 2000, and then LDH-linked

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Table 1. Laboratory Data (8 and 20 Months after Delivery)

1) 8 months after delivery (Sept. 1999)

WBC	7,800/μl	LDH	899 IU/*l*
RBC	470×10^4/μl	Amylase	201 IU/*l*
Plt	23.7×10^4/μl	T Cho	134 mg/dl
T Bil	0.3 mg/dl	BUN	15.1 mg/dl
TTT	3.2 MU	Cre	0.5 mg/dl
ZTT	7.5 KU	Na	141 mmol/*l*
AST	18 IU/*l*	K	4.2 mmol/*l*
ALT	7 IU/*l*	Cl	109 mmol/*l*
ALP	224 IU/*l*	Ca	9.4 mg/dl
FT4	1.87 ng/dl	FT3	4.91 pg/ml
TSH	<0.1 μIU/ml		

2) 20 months after delivery (Sept. 2000)

LDH	395 IU/*l*		
FT4	0.93 ng/dl	FT3	1.65 pg/ml
TSH	2.01 μIU/ml		

WBC: white blood cell, RBC: red blood cell, Plt: platelet, T Bil: total bilirubin, TTT: thymol turbidity test, ZTT: zinc sulfate turbidity test, AST: aspartate aminotransferase, ALT: alanine aminotransferase, ALP: alkaline phosphatase, LDH: lactate dehydrogenase, BUN: blood urea nitrogen, Cre: creatinine, Na: sodium, K: potassium, Cl: chloride, Ca: calcium, FT4: free thyroxine, FT3: free triiodothyronine, TSH: thyroid stimulating hormone.

IgG could not be detected by the same assay, although electrophoretic isoenzyme analysis showed a slightly abnormal broad band.

Discussion

The present patient did not have any symptoms of thyrotoxicosis before the first examination (7 months after delivery), and the onset of postpartum thyroid dysfunction was obscure. As she had no anti-TSH receptor antibody and transient thyrotoxicosis rapidly normalized without any special treatment, she might not have Grave's disease. However, there have been reported cases of painless thyroiditis with prolonged thyrotoxicosis (3). Therefore, she was diagnosed as having postpartum thyroiditis, painless thyroiditis at the postpartum period.

It has been reported that LDH-linked immunoglobulin (autoantibody to LDH) is observed in various diseases, especially in liver diseases and malignancies, and even in healthy subjects (1). In addition, patients with various types of autoimmune diseases are known to have LDH-linked immunoglobulin, and it was reported that LDH-linked immunoglobulin is also present in patients with autoimmune thyroid diseases (1, 2). However, there have not been any reported cases associated with postpartum thyroiditis. In the present patient, serum levels of LDH had been elevated during thyrotoxicosis due to postpartum thyroiditis, as she had demonstrated LDH-linked immunoglobulin (IgG-κ). This case is the first case of postpartum thyroiditis with LDH-linked immunoglobulin. It is apparent that the maternal immune mechanism changes greatly after childbirth (4). Various autoantibodies could appear or increase remarkably in the postpartum period, leading to the development of the various autoimmune diseases (5, 6). Postpartum thyroiditis is a common autoimmune disease. It usually occurs in patients

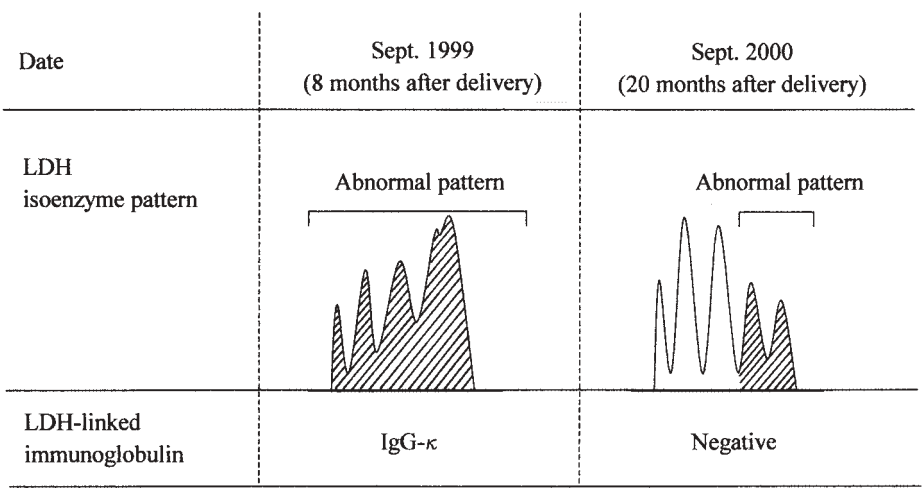

Figure 1. LDH isoenzyme pattern. LDH-linked immunoglobulin was measured by immunofixation electrophoresis assay (SRL Inc., Tokyo). In September, 1999 (8 months after delivery), electrophoretic isoenzyme analysis of LDH showed an abnormal broad band caused by LDH-linked immunoglobulin (IgG-κ). However, by September, 2000 (20 months after delivery), LDH-linked IgG could not be detected by the same assay, although electrophoretic isoenzyme analysis showed a slightly abnormal broad band.

with subclinical autoimmune thyroid diseases and the symptoms may become clinically apparent in the postpartum period. Since LDH-linked immunoglobulin is also one of these autoantibodies (1, 7, 8), it is suggested that LDH-linked immunoglobulin could appear or increase in association with postpartum thyroiditis.

LDH-linked immunoglobulin usually appears constantly in healthy subjects, however, the levels of this immunoglobulin could change or disappear (1). Nabeshima et al reported a case of LDH-linked IgG (IgG-κ, λ) with chronic hepatitis whose LDH levels had increased during interferon therapy and decreased after the cessation of interferon (9). Fujishima et al demonstrated that LDH-linked IgG (IgG-κ) with idiopathic interstitial pneumonia had disappeared by immunosuppressive therapy (10). Concerning autoimmune thyroid diseases, Gemma et al reported a case of Graves' disease with a transient appearance of LDH-linked IgG (IgG-κ) after treatment with methimazole (2). It is suggested that the transient appearance of LDH-linked immunoglobulin may appear transiently in patients with autoimmune disease or unusual immune conditions. In the present case, LDH-linked IgG returned to undetectable levels with recovery from postpartum thyroiditis, an abnormal immune condition. It has been interesting that the appearance of LDH-linked IgG coincided with the presence of postpartum thyroiditis, as there has not been any previous report concerning the alteration of serum levels of LDH-linked immunoglobulin in the postpartum period. We propose that one possible trigger for the appearance of LDH-linked IgG was postpartum hormonal changes. Further examination of LDH-linked immunoglobulin in patients with postpartum thyroiditis is needed.

In the present case, LDH-linked immunoglobulin disappeared without any treatment and the electrophoretic pattern of the LDH dramatically changed. However, there had been a slight abnormal pattern in a different band of LDH isoenzyme. It was reported that different types of LDH-linked immunoglobulin recognized many antigenic determinants on LDH molecules and showed different patterns of electrophoretic isoenzyme analysis (1, 11). There may have been two determinant sites of LDH-linked immunoglobulins in the present case.

In summary, we reported a patient with transient appearance of LDH-linked immunoglobulin coexisting with postpartum thyroiditis. It is suggested that LDH-linked immunoglobulin may have appeared or increased in association with postpartum thyroid dysfunction. It should be considered carefully to examine not only thyroid autoantibodies but also many types of enzyme-linked immunoglobulins during the postpartum period.

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