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Selective IgA Deficiency in a Patient with Hashimoto's Thyroiditis

Case Report

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ABSTRACT

We describe a case of Hashimoto's thyroiditis in which, eight years after initial diagnosis, an isolated (selective) IgA deficiency was detected. It is not clear which of these two conditions followed the other (both aetiologically and chronologically); indications and arguments for and against both hypotheses are discussed.

INTRODUCTION

Hashimoto's thyroiditis represents an autoimmune reaction against thyroidal antigens resulting in thyrocyte destruction by autoantibodies and lymphocytes. As a part of the immunological disturbance leading to the disease, it is accompanied by other disorders of autoimmune nature more often than can be accounted by chance; however, a combination of this disease with selective IgA deficiency (congenital or acquired) has not been reported so far.

CASE REPORT

A 39-year-old Greek female teacher was referred to this Department for a further evaluation; two months prior to her presentation, an investigation revealed isolated IgA deficiency; her serum immunoglobulin levels had not been determined before. There was no history of allergy or severe/recurrent infections apart from a history of a severe varicella infection at the age of 30 and rubella at the age of 33. She was married, had two healthy children and her menses were regular. The family history was unremarkable. Some eight years (at least) before her present admission she had experienced symptoms of unexplained fatigue, anorexia, irritability and difficulty in swallowing. An initial investigation by the family physician revealed no abnormality; nevertheless, her symptoms deteriorated without any diagnosis being put forward for the next three years; finally, a clinical and laboratory investigation indicated primary hypothyroidism on the basis of a Hashimoto's thyroiditis; hence, low serum T3/T4, elevated serum TSH and high titers of thyroid antibodies were found. Therefore, she was commenced on thyroxin replacement therapy (100 μg daily) and since then she has been feeling well.

On examination she looked normal; no abnormal signs or findings were found and the liver, spleen and thyroid gland were unpalpable.

The peripheral blood count was normal. A biochemical evaluation of her thyroid function revealed normal thyroid hormone levels (T3: 106 ng/dL range: 60-190; T4: 8.9 ng/dL, range: 5.2-12.5); the levels of TSH were also normal (1.2 μU/mL, range: 0.4-4.6). The anti-TPO levels were very high: 78 U/mL (normal <2) and the anti-TG antibodies were also high: 14.7 U/mL (normal <2).

The serum immunoglobulin IgA level was 29.5 mg/dL (range: 85-840) whereas the other immunoglobulin classes were found within normal limits; namely IgM was 364 mg/dL (range 60-370), IgG 1680 mg/dL (range 800-1700) and IgE 31.8 U/L (normal <150).

The serum test for parietal cell (APCA) antibodies was positive; the complement levels were normal; no anti-nuclear, anti-DNA or AMA antibodies were detected. Hepatitis (A, B, C) and HIV serology tests were also negative. The HLA type was as follow: HLA-A: 33,10; -B: 14,15, w4, w6; -DR: 1,4, w53, DQw3.

The ultra-sound investigation of the neck area revealed a very small thyroid gland, thus precluding any attempt for a needle aspiration biopsy.

Siblings were not available for investigation but serum immunoglobulins in the two children of the patient were already tested and found to be normal.

DISCUSSION

The detection of an isolated IgA deficiency in a patient with a known Hashimoto's thyroiditis raises several interesting speculations as to the putative association between the two conditions.

In our case, it is not clear whether the IgA deficiency preceded or followed the development of Hashimoto's thyroiditis. Isolated IgA deficiency may be either congenital or acquired; congenital IgA deficiency is often familial though mainly asymptomatic. In our case there was not history of anaphylactic reactions, respiratory infections or chronic diarrhoea all of which may characterise congenital IgA deficiency. Besides, the HLA types were not those described for this entity (1) and the family members tested had a normal serum immunoglobulin profile.

Patients with a congenital IgA deficiency are prone to autoimmune diseases; acquired IgA deficiency also coincides with a family history of organ-specific autoimmunity (3). However, thyroid disorders of autoimmune nature are not encountered within IgA deficient subjects (2). As the autoimmune character of Hashimoto's thyroiditis is generally accepted (6), one should also remember that autoimmune diseases such as juvenile rheumatoid arthritis is reported to be complicated by an acquired IgA deficiency which is attributed to aspirin therapy (5).

It is also worth mentioning that recently an isolated IgM deficiency was described in a case of Hashimoto's thyroiditis (4). On the other hand we must note that although in this case the clinical and laboratory diagnosis of a long-standing Hashimoto's thyroiditis seems clear, a cytological proof albeit unnecessary is lacking due to the gland atrophy. Besides, the HLA type is not typical for this disease (7).

In conclusion, we present an isolated IgA deficiency detected in a case of a long-standing Hashimoto's thyroiditis. The combination seems to be immunologically mediated; however, its precise nature remains elusive and the simple co-incidence can not be excluded.

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