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HENOCH – SCHÖNLEIN NEPHRITIS ASSOCIATED WITH SUBACUTE THYROIDITIS*

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SUMMARY: Öner A, Demircin G, Tınaztepe K, Akıncı A, Teziç T. (Department of Nephrology, Dr. Sami Ulus Children's Hospital, Ankara, Turkey). Henoch-Schönlein nephritis associated with subacute thyroiditis. Turk J Pediatr 1986; 38: 131-135.

We present a 12-year-old boy who developed subacute thyroiditis during the course of rapidly progressive glomerulonephritis due to Henoch-Schönlein purpura (HSP) proven by clinical findings and percutaneous renal needle biopsy. The thyroid gland of the patient suddenly enlarged with mild tenderness while he was on steroid and dipyridamole therapy. Thyroid hormone levels revealed T_3 0.31 ng/ml (nl: 0.52-1.75 ng ml), T_4 2.53 ug/dl (nl: 4.8-12.8 ug/dl), free T_3 0.80 pg/ml (nl: 2.14-5.34 pg/ml), free T_4 0.2 ng/dl (nl: 0.73-1.95 ng/dl) and TSH 1.02 U/ml (nl: 0.36-3.25 U/ml). Antimicrosomal antibody was negative while antithyroglobulin antibody was slightly positive (1/80+). Hypoactivity with a spotty pattern was demonstrated by thyroid scanning. Serologically proven mumps infection was detected and may have been a triggering factor in the development of both HSP and subacute thyroiditis. *Key words: Henoch-Schönlein purpura, rapidly progressive glomerulonephritis, subacute thyroiditis, mumps.*

Henoch-Schönlein purpura (HSP) is a clinical syndrome of unknown etiology which is predominantly seen in childhood and is characterized by generalized vasculitis that affect small vessels[1]. Arthritis, gastrointestinal manifestations and nephritis usually accompany the characteristic skin lesions of HSP[2]. Clinical evidence of renal disease, the most serious feature of HSP, occurs in 20 to 75 percent of patients[1-2]. Involvement of other organs and systems such as the nervous system, eyes, endocrine glands and heart have rarely been reported in HSP[2-4], while thyroid involvement associated with HSP has not been reported before.

We present here a 12-year-old boy who developed subacute thyroiditis during the course of rapidly progressive glomerulonephritis (RPGN) due to HSP. The laboratory data revealed: Hb 17.8 g/dl, white blood cell count 23.200/mm^3 with 84% neutrophils and 16% lymphocytes. The erythrocyte sedimentation rate was 30 mm/hour. Urine examination showed 3 (+) proteinuria (6 g/m^2/day) and

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hematuria with granular casts. The serum sodium level was 119 mEq/L while the other electrolytes, renal and liver function tests were within normal limits. The creatinine clearance was 100 ml/min/1.73 m^2. The stool guaiac test was positive. Microbiological examinations were negative for a pathogen microorganism. Serum immunoglobulins and complement levels were normal. Abdominal ultrasonography revealed increased renal parenchymal echogenicity. The patient was treated with intravenous fluids for dehydration. On the third day of admission he was started on oral prednisolone therapy (60 mg/m^2/day) because of gastrointestinal system hemorrhage.

The patient developed scalp edema on the seventh day of hospitalization and hypertension on the tenth day. On the 11th day his renal functions were impaired with blood urea nitrogen 78 mg/dl, serum creatinine 2.8 mg/dl and proteinuria increased to 11 g/m^2/day. Percutaneous renal needle biopsy was performed, and endo- and extracapillary proliferative glomerulonephritis was demonstrated. The patient was given pulse therapy with methyl prednisolone on three consecutive days, and oral prednisolone (45 mg/m^2/day) was given together with dipyridamole and captopril on the following days. The steroid dose was tapered gradually after one month to a maintainence dose of 10 mg every other day.

On the 27th day, the patient's thyroid gland was diffusely enlarged with slight tenderness. He had also lost eight kilograms of weight during this time. Measurement of thyroid hormones revealed T_3 0.31 ng/ml (nl: 0.52-1.75 ng/ml), T_4 2.53 ug/dl (nl: 4.8-12.8 ug/dl), free T_3 0.80 pg/ml (nl: 2.14-5.34 pg/ml), free T_4 0.2 ng/dl (nl: 0.73-1.95 ng/dl) and TSH 1.02 U/ml (nl: 0.36-3.25 U/ml). Antimicrosomal antibody was negative while antithyroglobulin antibody was slightly positive (1/80+). Thyroid ultrasonography revealed diffuse glandular hyperplasia with enlarglement of the right lobe to 40 x 17 x 14 mm and left lobe to 40 x 14 x 13 mm. Scanning of the thyroid gland with Tc 99-m pertechnetate showed hypoactivity with a spotty pattern (Fig. 1). These clinical and laboratory data led to the diagnosis of subacute thyroiditis. Needle aspiration of the thyroid gland was performed, but adequate material could not be obtained to confirm the diagnosis. The patient was started on L-thyroxine therapy.

The serological tests performed five days after presentation of the first symptoms of subacute thyroiditis in order to find a viral etiological agent were negative for rubella, rubeola and Epstein Barr virus, while IgG for mumps was 1/10 (+) and increased to 1/40 positivity two weeks after the initial measurement.

Two weeks after therapy with methyl prednisolone, pulse renal function tests were normal and the proteinuria was decreased to 2 g/m^2/day. The patient's thyroid gland was diminished in 15 days and thyroid scanning returned to normal in three weeks with L-thyroxine treatment (Fig. 2). At the end of the third month, the proteinuria disappeared. In the sixth month the thyroid hormone levels were normal with T_3 1.47 ng/ml, T_4 10.25 ug/dl, free T_3 4.49 pg/ml, free T_4 1.27 ng/dl and TSH 1.43 U/ml.

The therapy with L-thyroxine and prednisolone was discontinued. In the seventh month the patient's antimicrosomal and antithyroglobulin antibodies were still normal. He was followed up for 22 months without any clinical or laboratory abnormalities.

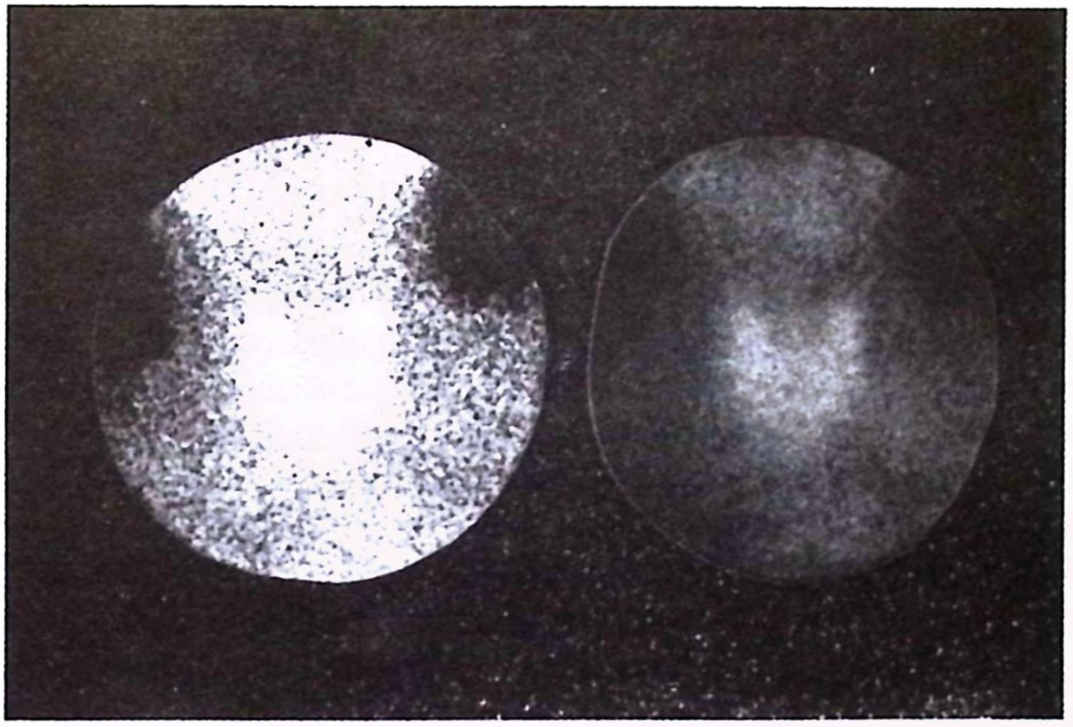

Fig. 1: Tc-99-m pertechnetate thyroid scanning demonstrates hypoactivity and spotty pattern in the gland.

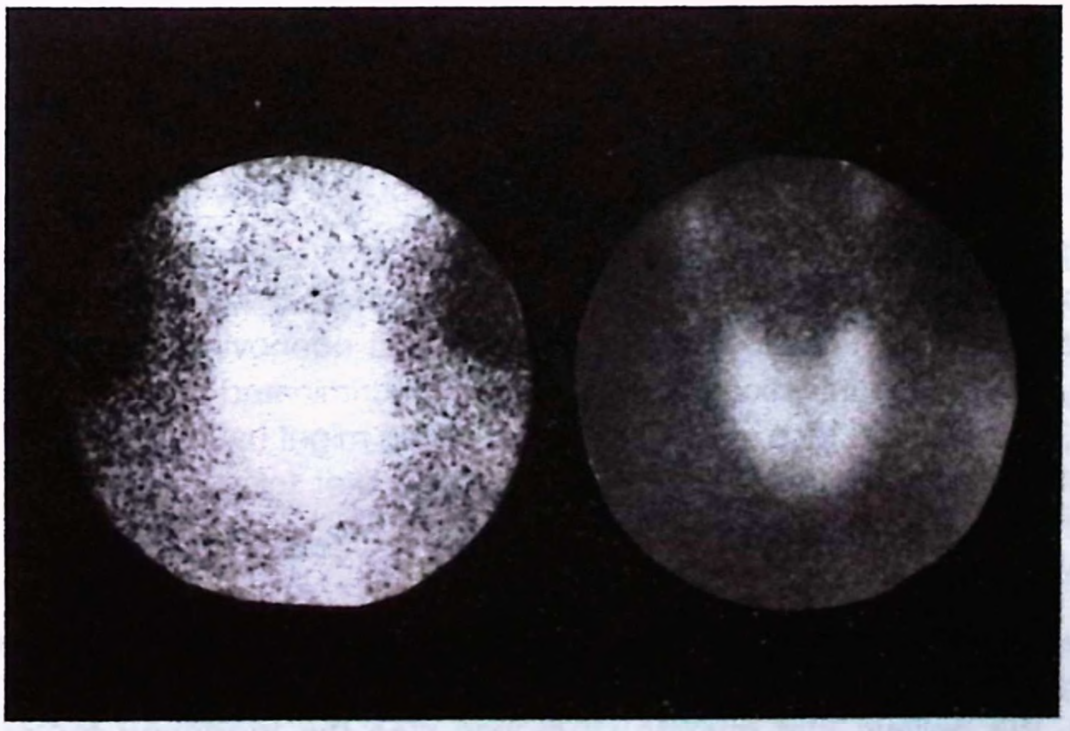

Fig. 2: Tc-99-m pertechnetate thyroid scanning shows normal thyroid gland after therapy.

Discussion

There are reports about the involvement of certain endocrine glands including the pancreas, adrenals, parotis glands and testes in HSP[2-4]. However, there is no report in the literature about thyroid pathology during the course of HSP. In this case report, a patient whose Henoch Schönlein nephritis was proven by characteristic clinical findings and percutaneous renal needle biopsy suddenly developed a diffuse, mildly tender goiter on the 27th day of steroid therapy, suggesting a diagnosis of thyroiditis. Development of symptoms under steroid therapy and tenderness of the thyroid gland with the findings on thyroid scanning suggested a diagnosis of subacute thyroiditis rather than autoimmune Hashimoto thyroiditis. The negative antimicrosomal antibody titers and slightly positive titer of antithyroglobulin antibodies which are increased significantly in 90 to 95 percent of patients with Hashimoto thyroiditis also supported the diagnosis of subacute thyroiditis[5]. Although we were not able to obtain adequate material for pathological diagnosis, the clinical improvement of the goiter, the disappearance of pathological findings on thyroid scanning in three weeks, and maintenance of clinical and laboratory remission long after therapy was discontinued confirmed the diagnosis.

Subacute thyroiditis is a self-limited, transient inflammation of the thyroid gland with a duration of one month to one year[5]. The thyroid gland is generally sensitive to palpation or only mildly tender[6]. It classically evolves through four consecutive phases: the hyperthyroidic phase lasting for 1-4 weeks, the euthyroidic, hypothyroidic and recovery phases[5, 6]. The thyroiditis of this patient was detected in the hypothyroidic phase when the thyroid gland was suddenly enlarged. The symptoms in the first two phases were masked by severe symptoms of Henoch Schönlein nephritis and by the effect of steroid therapy. Administration of prednisolone also inhibited the elevation of TSH which was expected to be high because of the positive feedback mechanism of low thyroid hormones[7].

Although the etiology of subacute thyroiditis remains unknown, identification of many patients with evidence of viral diseases such as mumps, coxsackie virus, influenza, measles, infectious mononucleosis and adenovirus suggests a viral etiology[5]. Since the infectious agents are also incriminated in the development of HSP, we searched for a single viral agent which might have triggered the HSP and subacute thyroiditis together. The initial IgG titer for mumps was found to be slightly positive, and increased significantly in two weeks, demonstrating that the patient had recently had mumps infection.

Review of the literature revealed eleven cases of thyroiditis in patients with serologically proven mumps, two cases of which grew mumps virus from thyroid tissue[5]. We believe that mumps virus was also the triggering factor in the developmenat of both HSP and subacute thyroiditis in this case. To our knowledge

this patient is the first in the literature to develop subacute thyroiditis during the course of HSP. The serologically proven mumps infection which might be the cause of the whole clinical picture would also support the infectious etiology in HSP and subacute thyroiditis.

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