Thyrotoxicosis Simulating Lymphoma
A Case Report

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Summary: We report a patient with unusual manifestations of hyperthyroidism which initially suggested lymphoma. The pathophysiology of these features in thyrotoxicosis is discussed. The need to consider thyrotoxicosis in an otherwise unexplained case of lymphoid hyperplasia will be stressed.

Introduction

Thyrotoxicosis is well known for its protein manifestations. We present a patient in whom the initial impression was one of a lymphoma.

Case Report

A 55-year-old man was admitted to the hospital because of a possibility of lymphoma.

He was well and active until six months earlier, when he experienced gradual loss of appetite and weight. He had lost 12 kg. in weight during this period. At the same time he also became breathless and on admission his effort tolerance was limited to a flight of stairs. Four weeks before admission he also complained of generalised prurities which progressively became unbearable. Foods, drugs, infections and emotional stress could not be incriminated. Benzyl benzoate and anti-histamines did not relieve the pruritus.

On examination, he was pale and had evidence of recent loss of weight. There was generalised lymphadenopathy involving the cervical, supraclavicular, axillary and inguinal regions. The lymph glands varied in size from a small to a large sized grape. They were soft, discrete, non-tender and mobile. Multiple skin excoriations were present on his extremities, chest and abdomen.

The pulse was 100/minute regular with good volume. His B.P. was 160/90 mm of hg. The thyroid gland was normal and there was no bruit. The examination was otherwise normal. There was no hepatosplenomegaly and the rectal examination was normal. There were no bruises, ecchymosis or purpuric spots.

The following investigations were done on admission: Haemoglobin 9.4 g/dl. The total white cell count, the differential count, platelets and the reticulocyte count were normal. The red cell indices were consistent with a microcytic hypochromic type of anaemia. The peripheral blood film showed normal neutrophils with a few eosinophils and atypical lymphoid cells. The platelets were adequate. The red cells were hypochromic with poikylocytes.

E.S.R. was 12 mm/1st. hour. Coomb's test (Direct and Indirect) was negative. Random blood sugar, blood urea, serum electrolytes, liver function tests including

alkaline phosphatase levels and urinanalysis were normal. The stool for occult blood was negative. The chest x-ray and electrocardiogram were normal. The bone marrow aspirate was essentially normal except for depletion of iron stores. In particular there were no abnormal cells seen infiltrating the marrow. The patient was discharged home after an axillary lymph node biopsy.

He was reviewed two weeks later when the clinic nurse observed the patient was restless and agitated. His pulse was 120/minute and he had fine tremors of the hand. Hyperthyroidism was suspected and confirmed by serum thyroxine level of 230/nmol/1 (normal range 59–130 nmol/1) and triiodothyronine level of 4.7 nmol/1 (normal range 1.5 – 3.2 nmol/1). Meanwhile the lymph node biopsy was reported as showing benign hyperplasia with no evidence of lymphoma or infiltration with abnormal cells.

He was treated with Carbimazole 45 mg. daily, Propranolol 20 mg. b.d., and ferrous sulphate 200 mg. b.d. In the course of six months clinical and biochemical euthyroid state was re-established. Propranolol was ceased after two months of treatment. The iron supplements were ceased three months after his hemoglobin became normal. The carbimazole was tailed off over fifteen months. His symptoms cleared rapidly as the antithyroid medication became effective. The lymphadenopathy disappeared completely upon his return to a euthyroid state. He remains well two years later.

Discussion

The initial clinical impression in this patient was one of a lymphoma. This would have adequately accounted for his presenting features namely, loss of appetite and weight, pruritus, anaemia and generalised lymphadenopathy. The preliminary investigations in this patient were therefore directed towards confirming a lymphoma. However, these tests including lymph node biopsy and bone marrow studies were negative for a lymphoma. Further attempts to prove lymphoma became unnecessary by the timely observation by the clinic nurse which led to the final diagnosis of thyrotoxicosis. Loss of appetite has occasionally been the presenting symptom of thyrotoxicosis as in our patient. Anorexia has been well documented in the so-called "masked hyperthyroidism" seen in elderly patients.[1]

The systemic causes of pruritus include lymphomas, occult malignancies, metabolic disorders, thyrotoxicosis and iron deficiency anemia. In our patient, both hyperthyroidism and iron deficiency anemia could have caused the pruritus. But as will be pointed later the anemia itself could have been secondary to the thyrotoxicosis.

The exact mechanism by which hyperthyroidism causes pruritus is not known. It is possibly the result of "vasodilatation and release of bradykinin in the tissues or potentiation of norepinephrine by excessive thyroxin hormone,"[4] Vasodilatation is known to lower the itch threshold and bradykinin can precipitate itching.

Generalised lymphadenopathy has occasionally been reported in hyperthyroidism. In extreme cases the signs and symptoms may be similar to that of leukemia or lymphoma.[2] The exact cause for this is not known. It may be due to margina-

tion and release of lymphocytes from bone marrow or to a longer than normal lymphocyte half-life.[2]

Anemia is rare even in the elderly thyrotoxic patient. This is because thyroid hormones promote erythropoiesis. But when anemia occurs in hyperthyroidism, it can present with the same morphological types as in hypothyroidism: (a) normocytic (b) hypochromic, microcytic and (c) macrocytic.[3] The red cell indices and morphology as well as the bone marrow aspirate findings in our patient were diagnostic of iron deficiency anemia. The reason for this type of anemia in hyperthyroidism is that more than one third of these patients have achlorhydria which impairs absorption of dietary iron.[3]

Our patient illustrates some of the more unusual manifestations of hyperthyroidism. Thyrotoxicosis needs to be considered in an otherwise unexplained case of lymphoid hyperplasia.

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