Thyrotoxicosis and asthma

A case report

M. A. K. OMAR, A. C. ASMAL, N. K. J. RANA

Summary

A 55-year-old woman with intrinsic asthma that was aggravated by the onset of thyrotoxicosis is described. The possible mechanism for such a relationship is discussed.

Since asthma and thyrotoxicosis are both relatively common conditions, they may on rare occasions coincide. The effect of thyroid function on the severity of asthma was first noted by Feinberg, who stated: 'removal of this hyperthyroid state may cure the asthma'. There have since been 4 reported cases illustrating the relationship between hyperthyroidism and severe asthma.

Case report

The patient was a 55-year-old postmenopausal Indian woman who presented with a 2-month history of excessive sweating, weight loss, poor appetite, palpitations and weakness of her arms (particularly when combing her hair). Five years previously she had been diagnosed as having intrinsic asthma, for which she was regularly taking salbutamol. However, over the past few months she had noticed a progressive deterioration in her condition despite more frequent use of bronchodilators, and this had necessitated more frequent visits to the hospital. Two years previously she had been given antacids for a duodenal ulcer. There had been no relapse since then, and there was nothing else of significance in her history.

On examination she was not in any obvious distress. There was no cyanosis, jaundice, clubbing or any significant lymphadenopathy. She was not in cardiac failure. There was a resting tachycardia of 112/min and her blood pressure was 120/70 mmHg. Heart sounds were normal. Examination of the chest showed a mild bronchospasm. She had a fine tremor when the hands were outstretched, and warm, moist palms. There was evidence of a proximal myopathy. She did not have any ocular signs of thyrotoxicosis, nor was pretibial myxoedema present.

There was a diffuse non-tender goitre (3 cm x 4 cm) with no bruit in her neck. Special investigations showed a serum thyroxine (T4) level of 18.5 μg/dl (normal 4 - 11 μg/dl), a free thyroxine index (FTI) of 23.4 μg/dl (normal 3.5 - 12.5 μg/dl), and a serum T3 level of 4.2 ng/ml (normal 1.6 - 2.1 ng/ml), thus confirming the presence of thyrotoxicosis. A thyroid scan showed a diffuse uptake of radiolabelled I131, which was increased to 65% (normal 20 - 50%). Antithyroglobulin antibodies were present in a weak dilution (1:20), but antimicrosomal antibodies were not detected.

She was treated with radio-iodine 5 mCi, and within 2 months she became euthyroid and the asthma had all but cleared. During the following 2 months she had no episodes of bronchospasm, but she now showed evidence of hypothyroidism, which was confirmed by biochemical testing (serum T4 level 1.6 μg/dl, FTI 1.2 μg/dl, thyroid-stimulating hormone level 56 μU/ml (normal 0 - 6 μU/ml)). She was given thyroxine, starting with 0.05 mg/d and gradually increasing to a maintenance dose of 0.2 mg/d. This was associated with a few episodes of bronchospasm (2 per month) which were easily relieved by means of oral salbutamol.

Discussion

There is little doubt that the onset of thyrotoxicosis in this patient was accompanied by aggravation of her asthmatic state.
and that control of the hyperthyroidism resulted in amelioration of bronchospasm. In fact, the patient required no treatment at all when she developed hypothyroidism as a result of radio-iodine therapy. Although replacement therapy did lead to bronchospasm, episodes were mild and infrequent and easily relieved by bronchodilators.

The precise mechanism whereby hypothyroidism aggravates the asthmatic state is not known. It is possible that a decrease in levels of adenosine triphosphate, which has been observed in hyperthyroidism, results in a decrease in intracellular levels of cyclic 3', 5'-adenosine monophosphate (cAMP), thereby predisposing to bronchospasm in susceptible individuals. However, no further studies on cAMP levels in hyperthyroidism do not support such a hypothesis.

Decreased catecholamine levels (which have been observed in hyperthyroidism) may also explain the exacerbation of bronchospasm in these patients. However, other studies on the relationship between catecholamines and hyperthyroidism have not corroborated these results.

According to Bush et al., a likely mechanism for the association between thyroid disease and the severity of asthma relates to altered corticosteroid metabolism. Hyperthyroidism appears to increase the conversion of hydrocortisone to its inactive 11-ketonic derivative, and in this way may aggravate bronchospasm, whereas the reverse is true for hypothyroidism.

Reduced breakdown of prostaglandins, which has been demonstrated in rats made hyperthyroid with thyroxine, could account for the increased bronchospasm seen in our patient. On the basis of studies showing that histamine is released from the thyroid mast cells of rats and mice in response to thyroid-stimulating hormone, it could be postulated that aggravation of the asthmatic state in association with hyperthyroidism is due to histamine release.

In conclusion, regardless of the mechanism involved in the relationship between hyperthyroidism and asthma, recognition of the interactions between the two diseases occurring in the same patient is essential for the proper management of these disorders.

REFERENCES


Spontaneous rupture of the bladder

A case report

Z. EL HAY, L. STEIN

Summary

A case of spontaneous rupture of the bladder is described. The patient was a 35-year-old woman presenting with an unusual clinical pattern of peri-

Spontaneous rupture of the bladder is rare. The diagnosis should be considered in patients who present with minor abdominal pain and an inability or a decreased ability to void urine, diarrhoea, abdominal distension, cough, pneumonia, post-cathe-

Department of Surgery, Coronation Hospital and University of the Witwatersrand, Johannesburg

Z. EL HAY, M.D. (ISRAEL), Registrar
L. STEIN, B.SC. HONS. F.R.C.S., Principal Surgeon

Reprint requests to: Mr L. Stein, Dept of Surgery, University of the Witwatersrand Medical School, Parktown, Johannesburg, 2195 RSA