Metastatic follicular carcinoma of the thyroid presenting with thyrotoxic induced impaired control of diabetes mellitus

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Summary: We report a patient with pulmonary and bony metastases due to follicular carcinoma of the thyroid, occurring 12 years after the initial diagnosis. This was brought to light by worsening diabetic control due to thyrotoxicosis from functioning malignant thyroid tissue. Following radio-active iodine therapy, she remains well with good control of her diabetes.

Introduction

Thyroid cancer is known to present in diverse ways, but thyrotoxic symptoms are rare (Staunton & Greening, 1973). We report a case of metastatic follicular carcinoma of the thyroid, causing thyrotoxicosis and impaired diabetic control.

Case report

A 66 year old woman presented in 1969 with a goitre of 35 years' duration. Over the last year, she had noticed some increase in the size of the goitre and had also experienced some dysphagia. She was clinically euthyroid and had a large smooth goitre with retrosternal extension. There was no lymphadenopathy. She underwent a partial thyroidectomy for pressure symptoms and histology of the gland removed was reported as follicular carcinoma of the thyroid. She did not receive post-operative radio-iodine. Six months later she appeared clinically hypothyroid, confirmed biochemically, and was started on replacement therapy with L-thyroxine 0.2 mg/day.

She remained well for the next 10 years, until 1980 when she developed diabetes mellitus. This was controlled on diet alone. Two years later, her diabetes was out of control and she had developed clinical features of thyrotoxicosis. Plasma thyroxine (T_4) was 180 nmol/l and triiodothyronine (T_3) 6.1 nmol/l (normal range, T_4 54–142 nmol/l, T_3 0.8–2.5 nmol/l). L-thyroxine was stopped but 2 months later she had worsened clinically and biochemically. Thyrotrophin (TSH), thyroid stimulating antibodies, thyroglobulin and thyroid microsomal antibodies were undetectable.

At this stage she also developed a hard subcutaneous nodule in the left chest wall which on histology was reported as showing thyroid glandular spaces with infiltration by tumour cells, consistent with secondary deposits from the previously reported thyroid tumour. A chest X-ray showed multiple metastases, and a whole body radio-iodine scan showed an equal degree of activity in the thyroid gland, lungs, liver and iliac crests indicative of metastatic disease.

She required oral tolbutamide for the control of her diabetes, and was given an ablation dose of radio-iodine. She was subsequently started on a replacement dose of T_3 . About 10 weeks later she appeared clinically euthyroid with a serum T_4 of 56 nmol/l, T_3 2.8 nmol/l and TSH 2.6 mU/l (normal < 6 mU/l). At the same time, she began to have symptoms of hypoglycaemia and the tolbutamide was stopped. At recent follow-up she remains well and asymptomatic. She is clinically and biochemically euthyroid, and on diet therapy her glycosylated haemoglobin is within the normal range, indicative of good diabetic control.

Discussion

Staunton & Greening (1973) have recently reviewed the clinical forms of presentation of thyroid cancer. Both toxic symptoms and metastases are relatively uncommon and occur in less than 5% of cases. Concomitant thyrotoxicosis in diabetes mellitus is known to affect diabetic control and result in increased insulin requirements (Ganda & Sveldner, 1983). We feel that our patient is very rare in that altered control of her diabetes was the first indication of metastatic disease due to thyroid cancer.

Other causes of thyrotoxicosis in theory, would

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Accepted: 15 August 1985

include Graves' disease, an autonomous toxic adenoma (Plummer's disease) and toxic multinodular goitre (Ingbar & Woeber, 1981). We feel that our patient's age, the absence of other clinical features especially ophthalmopathy and the absence of thyroid stimulating antibodies would exclude the diagnosis of Graves' disease. The absence of goitre and cardiovascular manifestations and the absence of patchy thyroid uptake on a scan would be against a toxic multinodular goitre. Plummer's disease occurs at a younger age, is generally associated with a palpable nodule, may show normal levels of serum T_4 but a raised serum T_3 , and on a radioactive scan shows suppression of surrounding thyroid tissue. These features were not present in our patient. Our patient had an equal degree of radioactive iodine uptake in the lungs, liver and bones and we feel that the most probable cause of her thyrotoxicosis was functioning metastases. The fact

that the thyroid was not suppressed would suggest autonomous activity. In view of the previous histology, this would probably represent a functioning primary or secondaries in the thyroid.

It is debatable whether or not she should have received radio-iodine therapy initially, after the diagnosis was suspected. In conclusion we feel that a case such as this is of interest because it demonstrates that thyroid malignancy may have many unusual and diverse aspects.

Acknowledgements

We are grateful to Dr A.G. Ackerley, Consultant Pathologist, for reviewing the histology and to Dr B.P. O'Malley for assaying thyroid stimulating antibodies.

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