Hyperthyroidism—an unusual feature of thyroid carcinoma

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Case description

A 53-year-old woman presented with a painless swelling of the left parieto-occipital scalp region of 6 months’ duration. Painless thyromegaly had been noted a month before presentation. Besides significant weight loss, there were no overt symptoms of thyrotoxicosis. There was no personal or family history of autoimmune disease, and prior head and neck irradiation was not elicited.

Examination revealed marked cachexia, sinus tachycardia of 105/min and a non-tender, soft left parieto-occipital scalp mass measuring 10 x 12 cm. She had moderate thyromegaly (30 g), which was hard in consistency, with a bruit and lymphadenopathy of the bilateral anterior triangles of the neck. There were no features of Graves’ ophthalmopathy or dermopathy. An additional finding was left lower lung consolidation.

Thyroid function test results were consistent with frank primary hyperthyroidism: free thyroxine (FT$\textsubscript{4}$) 46.3 pmol/l (normal 10.3 - 21.9 pmol/l) and thyrotropin (TSH) 0.01 IU/l (normal 0.35 - 4.5 IU/l). Anti-thyroglobulin and anti-thyroid peroxidase autoantibodies were negative. A Trucut biopsy of the scalp mass was performed. Histological review demonstrated well-formed thyroid follicles with occasional nuclear atypia and pleomorphism indicating metastatic follicular thyroid carcinoma.

A whole-body technetium scan revealed uptake in multiple bony sites, liver, lung parenchyma and the rim of the scalp mass but minimal uptake in the thyroid gland. The patient had received Lugol’s iodine for the control of hyperthyroidism prior to the technetium scan, which interfered with the interpretation of this investigation. A chest radiograph showed full-thickness erosion of the left parietal and occipital skull bones with intracranial extension of the scalp mass (Fig. 2).

The diagnosis of functional metastatic thyroid carcinoma was based on the finding of follicular carcinoma on histological examination of the scalp mass and hyperthyroidism.

Hospital course

We attempted to render the patient euthyroid to permit a safe total thyroidectomy. She was commenced on 40 mg daily of carbimazole and Lugol’s iodine was initiated 6 hours after the first dose of carbimazole. A Jod-Basedow effect (induction or exacerbation of thyrotoxicosis following administration of iodine in the presence of a goitre and iodine deficiency) occurred within 5 days of commencing Lugol’s iodine, with FT$\textsubscript{4}$ levels surging from 46.3 pmol/l to 80 pmol/l. This response to Lugol’s iodine was unexpected. In response to the dramatic
The common causes of thyrotoxicosis in thyroid malignancy are coexisting Graves’ disease, nodular goitre and thyroid carcinoma per se. The presence of the bruit over the thyroid gland, a characteristic feature of Graves’ disease, was the sole suggestion of this latter diagnosis; however, as the thyroid gland was hard and craggy, this diagnosis was less likely. In addition the patient exhibited no associated ophthalmopathy, dermopathy or positive antithyroidperoxidase and antithyroglobulin antibodies. Thyroid autoantibodies are, however, only positive in 75 - 80% of cases. The assay for TSH receptor antibody, which is a much more specific marker of Graves’ disease, is not available in our setting. Graves’ disease as a cause of thyrotoxicosis in metastatic thyroid carcinoma is remarkable because the TSH receptor antibodies resulting from autoimmune dysregulation still retain the ability to stimulate differentiated metastatic thyroid cancer cells after a total thyroidectomy. The presence of a bruit over the thyroid gland and its hard consistency also made the diagnosis of a toxic multinodular goiter highly unlikely.

Although functional thyroid metastases may cause low thyroid iodine uptake, the low uptake of technetium in our patient is confounded by prior treatment with Lugol’s iodine. Follicular destruction of the thyroid gland by the malignant process may also result in diminished thyroidal technetium uptake. In one series of 48 cases of toxic thyroid carcinoma, large tumour bulk is the factor most consistently associated with toxicity. The multiple sites of technetium uptake in our patient not only suggest functional metastases but are indicative of large tumour burden.

Iodopate inhibits both thyroidal uptake of iodine and release of thyroid hormones, in addition to suppressing the conversion of FT\(_2\) to free thyroxine (FT\(_3\)). It would have represented the treatment of choice, but its lack of availability in our setting precluded its use. Dexamethasone and propranolol suppress the conversion of FT\(_3\) to FT\(_2\). Propranolol has the additional benefit of non-selective beta-receptor blockade. Lithium inhibits the release of thyroid hormone from the thyroid gland and cholestyramine interferes with the enterohepatic circulation, of FT\(_3\) and FT\(_4\). Radioactive iodine therapy in the excessively thyrotoxic phase is not advisable, as mortality has been reported from further exacerbation of thyrotoxicosis. The occurrence of the Jod-Basedow effect shortly after starting Lugol’s iodine was unexpected, despite prior carbimazole administration. Multiple metastatic sites competing for iodine probably precipitated this.

This patient’s age of over 40 years, the large size of the primary tumour and extensive metastases, in particular intracranial metastases, conferred an extremely poor prognosis. Brain metastasis from...
thyroid malignancy is not only unusual but extremely ominous.

**Conclusion**

Thyrotoxicosis is an unusual manifestation of thyroid malignancy and usually occurs with differentiated follicular carcinoma. It is a recognised cause of thyrotoxicosis with low thyroidal uptake of iodine on nuclear uptake scanning. A large tumour burden is the single factor most commonly associated with thyrotoxicosis. The complexities in the management of toxic thyroid carcinoma have been highlighted in this very unusual clinical presentation.