Clinical Case Report

Hypertensive crisis in a patient with thyroid cancer

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ABSTRACT
Phaeochromocytomas may be discovered incidentally when patients present with hypertensive crisis during general anaesthesia. A 49-year-old man underwent thyroidectomy 25 years ago and was diagnosed to have spindle cell carcinoma of the thyroid. He presented with recent onset of hoarseness of voice and was found to have a vocal cord nodule. He developed a hypertensive crisis during surgery. He was subsequently evaluated and found to have bilateral phaeochromocytoma. Further evaluation revealed a RET proto-oncogene mutation at codon 634 consistent with multiple endocrine neoplasia (MEN)-2A.

INTRODUCTION
It is well known that unrecognized phaeochromocytomas can cause hypertensive crisis when patients harbouring these tumours undergo unrelated surgery under general anaesthesia. We report a patient who had surgery and radiation therapy for thyroid cancer 25 years ago and developed a hypertensive crisis when he underwent excision of a vocal cord nodule under general anaesthesia.

THE CASE
A 49-year-old man noticed a right-sided thyroid swelling in 1981. He underwent total thyroidectomy in 1983. The tumour was confined to the thyroid with no cervical lymphadenopathy. The biopsy was reported as spindle cell carcinoma thyroid with no amyloid deposits, and he was subjected to 5000 cGy of external beam radiotherapy to the neck. He had no family history of thyroid malignancy, hypertension, renal stones or fractures. He noticed a change in his voice in 2008 and was found to have a right vocal cord nodule for which he was advised surgery.

His blood pressure was well controlled with losartan 50 mg and hydrochlorothiazide 12.5 mg once daily orally. Fentanyl, propofol and atracurium were used for induction of anaesthesia, followed by mask ventilation with oxygen, nitrous oxide and isoflurane. He developed severe hypertension during induction followed by acute pulmonary oedema which required intubation and ventilation. Frustramide and morphine were administered and he was shifted to the intensive care unit. The possibility of a phaeochromocytoma was considered. He was then started on phenoxybenzamine and prazosin and his symptoms gradually improved. Histopathology revealed the vocal cord lesion as a benign vocal nodule. His 24-hour urinary vanillylmandelic acid (VMA) levels were 19.1, 24.2 and 31.6 mg/day, respectively on three consecutive days (normal range 1–8 mg/day). Alpha blockers increase the urinary excretion of norepinephrine without influencing the other urinary metabolites of catecholamines. Hence the elevated VMA levels were considered to truly reflect catecholamine excess. Computed tomography scan revealed bilateral adrenal masses—right 4×3.2×5 cm and left 3×2×2.5 cm. A meta-iodobenzylguanidine (MIBG) scan confirmed the diagnosis of bilateral phaeochromocytoma. Serum basal calcitonin was normal (3.8 pg/ml, reference <50 pg/ml). A review of his previous pathology slides showed spindle cell carcinoma thyroid with no amyloid. However, the paraffin block was not available for calcitonin immunostaining. Slit-lamp examination showed corneal nerve thickening. The presence of eunuchoid body habitus, previous thyroidectomy for thyroid cancer, bilateral phaeochromocytoma and corneal nerve thickening led to a diagnosis of multiple endocrine neoplasia (MEN)-2A. Genetic testing revealed RET proto-oncogene mutation in codon 634 consistent with MEN-2A.

After adequate alpha blockade and control of his heart rate, he underwent bilateral cortical sparing adrenalectomy. Post-operatively he was on prednisolone and fludrocortisone. Both these drugs were slowly tapered and stopped one year after surgery. He is doing well and has not had a hypoadrenal crisis on follow-up.
DISCUSSION

Our patient developed a life-threatening hypertensive crisis and pulmonary oedema during general anaesthesia. Subsequent evaluation confirmed the presence of bilateral phaeochromocytoma. Fentanyl and atracurium used for induction of anaesthesia could have triggered the hypertensive crisis. The presence of LVH despite mildly elevated blood pressure and previous thyroid carcinoma should have alerted us to the possibility of MEN-2 syndrome with medullary thyroid carcinoma and phaeochromocytoma. About one-third of individuals with phaeochromocytoma have labile hypertension which can lead to LVH. This patient had limited local disease at the time of thyroidectomy and also received postoperative external beam radiotherapy, both of which could have cured the medullary thyroid carcinoma. If the possibility of MEN-2 had been considered even before surgery, we could have averted this life-threatening emergency.

Medullary thyroid cancer can be misreported as spindle cell tumour in the absence of demonstrable amyloid. In 1983, when this patient underwent thyroidectomy, immunohistochemistry was not available at our centre. The corneal nerve thickening noted in this patient is commonly seen in MEN-2B, but has also been reported in MEN-2A.

In conclusion, any patient with unexplained LVH who has had prior treatment for thyroid cancer should be evaluated for phaeochromocytoma, particularly if she/he is due for a surgical procedure under general anaesthesia. Slit-lamp examination to look for corneal nerve thickening is a simple and cost-effective method to identify patients with MEN-2. If any thyroid cancer shows unusual histological features, serum calcitonin should be measured and immunostaining should be done to rule out medullary thyroid carcinoma.

REFERENCES