A case of thyroid carcinoma metastasis within a cerebellar meningioma: Case and a review of the literature

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Abstract
A 60-year-old woman was discovered to have a new iodine avid cerebellar lesion on post-treatment whole body scintigrapy (WBS) after receiving 5.53 Gigabecquerels (GBq) of Iodine-131 (131I) for management of progressive thyroid carcinoma with bone and pulmonary metastases. At the time she demonstrated mild right-sided dysmetria and gait ataxia, consistent with her cerebellar lesion.

Magnetic resonance imaging (MRI) confirmed the presence of a right extra-axial posterior fossa lesion (20x23x25mm), with radiological characteristics of a meningioma compressing the cerebellar hemisphere with associated oedema and some mass effect on posterolateral pons.

Due to concern of a cerebral metastatic lesion, the patient was referred for excision of the lesion and histopathology confirmed a World Health Organisation (WHO) grade 1 meningioma with infiltrates of metastatic poorly differentiated thyroid carcinoma cells. Immunohistochemistry stained strongly positive for thyroglobulin, thyroid transcription factor 1 (TTF1) and cytokeratin MNF116. The surrounding meningioma tissue was negative for these markers and the Ki67 proliferation index was 2-3%.

Following metastectomy, external beam radiotherapy to the posterior fossa (30 Gray in 10 fractions) was administered concurrently with dexamethasone therapy. Her previously noted cerebellar signs subsequently resolved.

Six years earlier our patient was discovered to have multifocal thyroid carcinoma following total thyroidectomy for a multinodular goitre. Histology demonstrated multiple subtypes of thyroid cancer including a 65mm minimally-invasive follicular carcinoma, with a 22mm solid focus of poorly differentiated carcinoma in the centre. In addition, there was a separate 8.5mm papillary carcinoma involving the surgical margin with no vascular invasion.

Post-thyroidectomy, in view of the focus of poor histology, a fluorodeoxyglucose positron emission tomography (FDG-PET) was performed, showing uptake in the thyroid bed but no evidence of locoregional or distant metastatic disease. A non-
contrast whole body computed tomography scan revealed a poorly defined thyroid gland and a multi-fibroid uterus but no other abnormalities.

The WBS following the remnant ablation similarly demonstrated uptake in the surgical bed without evidence of metastatic disease. Levothyroxine at a dose of 100 micrograms daily to achieve thyrotropin (TSH) suppression was initiated.

In the first 2 years following diagnosis, the diagnostic $^{131}$I WBS demonstrated physiological uptake only. However 6 months later, a recombinant-TSH (Thyrogen™) stimulated plasma thyroglobulin level rose to 203 mcg/L but a diagnostic WBS was negative. An empiric dose of 3.64 GBq of $^{131}$I was administered. The post treatment $^{131}$I WBS combined with single-photon emission computed tomography demonstrated iodine-avid metastases to the right sacrum, left lower lobe of the lung and mediastinal lymph nodes.

Despite a cumulative $^{131}$I dose of 29.68 GBq administered over 6 years, her disease continued to progress with further pulmonary, bone metastases and a subsequent cerebellar metastasis. Due to this refractory nature of her disease, she was recently commenced on Sorafenib obtained on a compassionate basis.

Thyroid cancer presenting with neurological symptoms with an initial radiological diagnosis of meningioma but subsequent histological findings of cerebral metastasis from differentiated thyroid carcinoma (in the absence of meningioma on histology) is rare but has been reported. The coexistence of two separate primary tumors: thyroid cancer and meningioma, in the same individual has also been recognised, although the pathogenic link is unclear.

Meningioma has been reported as the most common primary intracranial tumor to harbour metastases, the majority of which arise from breast and lung carcinomas, with cases of melanoma, renal and thymic carcinoma also reported. Several hypotheses have been postulated to explain this occurrence, including haemodynamic, metabolic, hormonal and molecular factors, but the exact mechanisms responsible for the development of metastases within meningiomas are not known. Also of interest, a single case of primary meningioma metastasising to the thyroid gland was previously reported.

Iodine avidity without the presence of metastatic thyroid tissue has been described in some meningiomas, possibly but not necessarily related to brain oedema. Despite the suggestion of physiological uptake of iodine within meningiomas in patients treated with $^{131}$I for thyroid cancer, our case highlights the need to first exclude a cerebral metastasis.

At the time of writing, this is the first reported case of metastatic undifferentiated thyroid cancer coexisting within a meningioma, although there was one reported case of a presumed follicular thyroid cancer metastasising in a meningioma.

Clinical question: At what point should external beam radiotherapy, tyrosine kinase inhibitors and conventional chemotherapy be considered for patients with metastatic but iodine avid thyroid carcinoma?
Figure 1. Radiology
Cerebral MRI demonstrating an extra-axial enhancing lesion (22x18x23mm) based on the tentorium extending into right posterior fossa compressing the right cerebellar hemisphere associated with oedema.

Figure 2. Histopathology
B: Metastatic thyroid carcinoma within the meningioma tissue – X 200. Neoplastic thyroid follicles lined by rather bland follicular cells.
C: TTF1 – X 200. Metastatic thyroid carcinoma cells positive for TTF1 by immunoperoxidase method.

Figure 3. Thyroglobulin and radioactive iodine treatment
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