Solitary cerebral metastasis from a papillary carcinoma of the thyroid

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Summary: A woman aged 52 was treated with radioactive iodine for a papillary carcinoma of the thyroid. Four years later she developed signs and symptoms of an intracranial space occupying lesion. A computed tomographic scan showed a mass in the right posterior frontal region. Although she was suspected of having metastatic disease a definite diagnosis was not established until she died 6 months later when post-mortem examination confirmed that she had a cerebral metastasis from a papillary carcinoma of the thyroid. There was no evidence of metastatic disease elsewhere in the body.

Cerebral metastases from papillary carcinoma of the thyroid are uncommon but may occur in patients who have metastases in bones or lungs. A search of the literature has revealed only two patients with solitary cerebral metastases.

Introduction

Papillary carcinoma of the thyroid is the commonest type of thyroid tumour constituting between 30 and 70% of all thyroid carcinomas. There is a wide age range. In younger patients the tumour is nearly always confined to the neck with or without spread to the cervical lymph nodes. The prognosis in general is good – better than for any of the other types of thyroid tumour – but in older patients papillary carcinoma behaves in a more aggressive manner and may give rise to distant metastases. Such metastases are commonly sited in the lungs or bones. We report here a patient with papillary carcinoma of the thyroid who had a solitary metastasis in the brain.

Case report

At the beginning of 1984 a woman aged 52 noticed increasing hoarseness of her voice. On examination she was found to have a hard mass on the left side of her neck and a paralysed left vocal cord. Surgical exploration in February 1984 revealed a tumour replacing the left lobe and isthmus of the thyroid gland. It was infiltrating the strap muscles and was firmly fixed to the trachea. Biopsy was performed but no attempt was made to remove the tumour. Histological examination showed a locally invasive papillary carcinoma of the thyroid which, as is frequently the case with papillary carcinoma, was predominantly of follicular pattern.

She was referred to the Royal Free Hospital for further management. A thyroid scan showed a normal right lobe but diminished activity in the region of the left lobe. Thyroid uptake at 24 hours was 15%. It was decided to treat her with radioactive iodine (131I). She was accordingly given 120 mCi (4.3 GBq) of 131I in March 1984 with the intent of first ablating normal thyroid tissue. She developed radiation thyroiditis 3 days after this treatment. Three months later a scan showed activity just above and to the left of the suprasternal notch with a 24-hour uptake of 0.3%. She was given a second therapeutic dose of 150 mCi (4.5 GBq) of 131I in June 1984. She was subsequently maintained on 200 μg of thyroxine daily.

She remained well for the next 4 years. Throughout this time there was some induration on the left side of her neck but no tumour could be identified. Her progress was monitored with serial measurements of serum thyroglobulin (Tg). This never became normal (<0.1 μg/l), varying between 11 and 27 μg/l. The source of the Tg was assumed to be residual primary tumour, but in retrospect could have been the metastasis.

When seen in March 1988 she complained of drowsiness, confusion and weakness of her left hand. On examination she had dysdiadochokinesis of the left hand and loss of power in the left hand and arm. The signs and symptoms suggested an intracranial space occupying lesion. A computed tomographic (CT) scan (Figure 1) showed a lobulated mass in the right posterior frontal region immediately above the anterior part of the lateral ventricle. The findings were thought to be compatible with metastatic disease. A chest X-ray was
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normal. Liver function tests were normal. A
mammogram showed no evidence of breast car-
cinoma. A test dose of 5.0 mCi (185 MBq) of $^{131}$I
was given 24 hours after an intramuscular injection
of 5 units of bovine thyroid stimulating hormone
(TSH) and a whole body scan was performed 72
hours later. The patient was given exogenous TSH
in preference to allowing her to become
hypothyroid because the retention of fluid which
occurs in hypothyroidism would undoubtedly have
resulted in a deterioration of her neurological
condition. Scanning (performed under optimal
conditions of timing and dose) showed no activity
in abnormal sites and in particular no activity in the
neck or brain. It was concluded that the patient had
metastatic disease but there was some doubt as to
whether this was secondary to the thyroid tumour.
It was clearly unsatisfactory not having a tissue
diagnosis but the neurosurgical opinion was that
biopsy carried some risk and that it was not
justified unless it was going to alter her treatment.
She was given a course of external irradiation in the
hope that this would retard the progress of her
disease. She received a dose of 3600 cGy in 12
fractions.

By July her condition had deteriorated. She
could do very little with her left arm and had largely
lost the use of her left leg. A CT scan showed the
ventricular system to be slightly larger than on the
previous examination but the metastatic lesion had
not changed. Her condition continued to
deteriorate. She died 6 months after presentation.

Post-mortem examination showed the cause of
death to be a massive embolus occluding the right
main pulmonary artery. The thyroid gland was
represented as a thin fibrous strip. There was no
evidence of regional lymph node involvement.
Sectioning of the brain showed a well circumscrib-
ed solid and cystic tumour mass within the
right fronto-parietal white matter. The mass con-
tained small areas of apparent necrosis. It
measured 3 cm in its maximum dimension and
extended into the outer margin of the right anterior
horn of the lateral ventricle. There was no evidence
of metastatic disease elsewhere in the body.

Histological examination showed the tumour to
be composed of multiple papillary processes show-
ing great variation in size. Small foci of calcification
(psammoma bodies) were noted. In some areas
there were small follicular structures containing
colloidal substance (Figure 2). Immunohisto-
chemical staining showed intracellular thyro-
globulin in some cells. The appearances were those
of metastatic papillary carcinoma of the thyroid.

Discussion

McConahy et al.\textsuperscript{1} reviewed all patients with papil-
lar carcinoma of the thyroid treated at the Mayo
Clinic between 1946 and 1970. In a series of 859
patients 11 (1.3\%) were identified as having cere-
bral metastases. Høe et al.\textsuperscript{2} used the Cancer
Registry in Norway to find out the incidence of
distant metastases in patients with papillary car-
cinoma of the thyroid. In a series of 731 patients,
registered between 1956 and 1978, 91 developed
distant metastases. Seventy three of these patients
had intrathoracic metastases, 17 skeletal metas-
tases and 9 (1.2\%) cerebral metastases. In both the
above series the patients who developed cerebral
metastases also had metastases in lungs or bones or
in both sites.

As Parker et al.\textsuperscript{3} have pointed out, cerebral
metastases from papillary carcinoma of the thyroid
are uncommon. These authors reported two
patients where the tumour had metastasized to the
brain. One had metastases elsewhere. The second
one did not, and this would appear to be the first
recorded instance of a solitary cerebral metastasis
secondary to a primary papillary carcinoma of the
thyroid. A search of the literature has revealed only
one other case, a patient with an occult primary
carcinoma of the thyroid.\textsuperscript{4}

The patient whose case history is described here
is another example of a solitary cerebral metastasis
from a papillary carcinoma of the thyroid. The
rarity of this condition made us reluctant to make
what turned out to be the correct diagnosis which

Figure 1 Computer tomogram of brain showing a
lobulated mass impressing the ventricular roof in the deep
right fronto-parietal region. This enhanced image sug-

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\caption{Computer tomogram of brain showing a
lobulated mass impressing the ventricular roof in the deep
right fronto-parietal region. This enhanced image sug-
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was only confirmed after a post-mortem examination had been carried out. Non-iodine-concentrating metastases have been identified by giving the patient labelled anti-thyroglobulin antibodies and subsequently doing a scan but this technique is not currently available.

Histological examination of the metastasis showed some areas where there were follicular structures containing colloidal substance and some cells were seen to be producing thyroglobulin, the latter probably accounting for the elevated levels of thyroglobulin in the serum. It might seem surprising that the metastasis did not concentrate iodine. However, although histological appearances provide some indication of whether or not a tumour is likely to take up iodine there is not always a good correlation between structure and function.

The primary tumour did presumably contain functioning thyroid tissue because it resolved with no treatment other than 131I. It might be expected that metastases from a functioning primary tumour would behave in the same way as the parent tissue with respect to iodine concentration but this is not always the case. Metastases are often less well differentiated than the primary. Comparison of sections of the primary tumour and the metastasis in this patient suggest that the follicular foci in the metastasis were much less prominent.

Treatment of a cerebral metastasis from a differentiated carcinoma of the thyroid is a problem if the metastasis does not concentrate 131I. This patient's tumour clearly did not respond to external irradiation. One of the patients described by Parker et al. was treated by surgical excision, an implant using 125I seeds and external irradiation but this method of treatment would be of questionable value in a patient who already had quite severe brain damage.

Acknowledgement

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References


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