Cerebral metastasis in general is associated with a relatively short survival time. However, brain deposits may occur rather late during follow up. Nine cases of solitary brain metastasis of renal cell carcinoma with a latency period of more than 10 years after nephrectomy have been reported in the literature so far. This is the first report of a case describing a second solitary brain metastasis which occurred 16 years after a first metastatic brain lesion. Complete microsurgical resection alone led to an excellent outcome in this particular case as the patient refused any adjuvant therapy at the same time. Regular nuclear morphology, a low mitotic index, and the absence of chromosomal abnormalities in tumour cells may be indicative for a relative benign clinical course.

CLINICAL PRESENTATION

A 61 year old man with a history of a single generalised seizure presented to our department with facial palsy, gait instability, and intermittent dysphasia. He had previously had a left nephrectomy and splenectomy due to renal cell carcinoma 19 years ago. There was no renal vein involvement, lymphatic dissemination, or other evidence of systemic disease at the time of nephrectomy. In 1985, 3 years after diagnosis of primary renal cell carcinoma, the patient developed symptoms of psychomotor deterioration accompanied by apraxia. At that time, imaging studies showed a cystic, right parieto-occipital cerebral metastasis (fig 1). This lesion was completely excised. In 1999, the patient developed a moderate thyroid mass. Diagnostic ultrasound and suppression scintigram indicated a second metastatic lesion in the thyroid gland and a partial right and complete left thyroidectomy was performed 18 years after primary surgery for the renal cell carcinoma.

On admission in 2000, CT and MRI showed a solitary left dorsofrontal lesion composed of solid and cystic compartments (fig 1). The serum ferritine concentration was mildly increased, but other laboratory indices including CEA, AFP, PSA, CA 125, CA 19–9, and CA 15–3 were within normal limits. Chest radiography and abdominal sonography detected no evidence of systemic disease. There was no weight loss. The patient underwent a left frontal craniotomy with intraoperative ultrasound guidance. The highly vascularised tumour was microsurgically removed and the patient’s symptoms resolved entirely.

PATHOLOGICAL EXAMINATION

Because secondary brain metastasis of renal cell carcinoma as long as 19 years after excision of the primary tumour has never been reported, histological sections from all previously obtained specimens were thoroughly reviewed. The kidney tumour which was removed in 1981 showed the typical cellular pattern of a clear cell carcinoma. The first brain metastasis showed features of a clear cell adenopapillary carcinoma, with hyperchromatic nuclei and pseudopapillary structures. The thyroid tumour removed in 1999 showed a bimodal differentiation. Primary thyroid carcinoma could be excluded. The second cerebral metastasis in 2000 again displayed highly differentiated clear cells with trabecular and tubular growth and moderate necrosis. No vascular invasion into peritumoural brain was seen in histological sections of both metastasis. A panel of immunocytochemical stains showed strong expression of epithelial membrane antigen (EMA) and vimentin, but negative results for Lu5, thyroglobulin, and CD 30. The MiB-1
labelling index was below 1%. There were no chromosomal abnormalities in tumour cells. The tissue of both brain metastases did not contain any of the characteristic changes for the known VHL gene mutations, so that haemangioblastomas could be excluded from the differential diagnosis.

**POSTOPERATIVE COURSE**

The patient made an unremarkable recovery, with improvement of facial palsy and gait disturbances. The Aachen aphasia test (AAT) showed no signs of aphasia. No tumour remnants were seen on postoperative CT. As before, the patient refused radiation therapy. Fourteen months after surgery clinical evaluation showed no neurological deficit and a Karoofsky score of 100 was ascribed.

**DISCUSSION**

The clinical course of renal cell carcinoma may vary considerably. Whereas 25%–40% of the patients harbour metastatic lesions at the time of diagnosis already, the rate of solitary late recurrence (>10 years) ranges from 4.7% to 11%. Besides the most preferred sites of metastasis from renal cell carcinoma—lung and bones—the tumour metastasises to different brain regions with no predilection site in 4% to 10% of cases. Brain metastasis in most cases occurs at an advanced stage, usually with evidence of widespread disease. Single brain metastasis of renal cell carcinoma is exceedingly rare with only nine cases described in the literature (table 1). 1,2

Metastatic brain lesions in general are associated with a short life expectancy. Average reported survival periods after the diagnosis of brain metastasis range from 5 to 9.5 months; 14.3% to 43.2% of the patients survive the first year and much shorter life expectancy. Average reported survival periods after metastatic brain lesions dedifferentiate in short periods of time and otherwise remain stable in their grade of malignancy are still to be determined.

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Table 1: Cases of renal cell carcinoma with brain metastasis occurring later than 10 years after the primary lesion

<table>
<thead>
<tr>
<th>Authors and year</th>
<th>Case No</th>
<th>Symptoms</th>
<th>Age (y), sex</th>
<th>Recurrence after primary (y)</th>
<th>Surgical treatment</th>
<th>Radiation dose</th>
<th>Survival (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Killebrew et al 1983</td>
<td>1</td>
<td>UN</td>
<td>55, F</td>
<td>13</td>
<td>Total removal</td>
<td>–</td>
<td>48</td>
</tr>
<tr>
<td>Ishikawa et al 1990</td>
<td>2</td>
<td>HA, ophasia, hemiparesis</td>
<td>46, F</td>
<td>14</td>
<td>Total removal</td>
<td>–</td>
<td>28</td>
</tr>
<tr>
<td>Cervoni et al 1993</td>
<td>3</td>
<td>HA, gait disturbance</td>
<td>61, M</td>
<td>13</td>
<td>en bloc resection</td>
<td>–</td>
<td>49</td>
</tr>
<tr>
<td>Kuroki et al 1999</td>
<td>4</td>
<td>HA</td>
<td>65, F</td>
<td>17</td>
<td>en bloc resection</td>
<td>–</td>
<td>55</td>
</tr>
<tr>
<td>Redley et al 1995</td>
<td>6</td>
<td>HA, ophasia, hemiparesis</td>
<td>78, M</td>
<td>18</td>
<td>Total removal</td>
<td>–</td>
<td>12</td>
</tr>
<tr>
<td>Jubelirer 1996</td>
<td>7</td>
<td>Confusion, ophasia</td>
<td>86, F</td>
<td>15</td>
<td>Debunking</td>
<td>–</td>
<td>1.5</td>
</tr>
<tr>
<td>Kuroki et al 1999</td>
<td>8</td>
<td>Alexia</td>
<td>86, F</td>
<td>12</td>
<td>Total removal</td>
<td>18 Gy</td>
<td>UN</td>
</tr>
<tr>
<td>Present case</td>
<td>10</td>
<td>Seizure, facial palsy</td>
<td>61, M</td>
<td>19</td>
<td>Total removal</td>
<td>18 Gy</td>
<td>Alive</td>
</tr>
</tbody>
</table>

HA: Headache; UN: unknown.

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**REFERENCES**