CASE REPORT

Prolonged Survival after Neurosurgical Resection of Lung Cancer Metastasis

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SUMMARY

Metastatic tumours in the brain occur more frequently than primary neoplasms. Despite the generally dismal prognosis, neurosurgical resection is indicated in certain patients and can yield prolonged survival. Here we describe an 82-year-old male with a history of neurosurgical resection of a single brain metastasis 6 years ago. Tumour immunophenotype disclosed lung adenocarcinoma with low proliferation fraction. However, the primary tumour remained occult then. At present, 3 new brain metastases were identified by computed tomography. Repeated resection was performed in 2 stages, resulting in removal of 2 metastases. Lung mass was now evident as well. The final diagnosis was lung adenocarcinoma with metachronous brain metastases, stage IV. In conclusion, prolonged survival, in this case 6 years, can be reached even in patients with metastatic cancer by successful selective application of neurosurgical treatment. The biological properties of the tumour including low proliferation also contributed to longer survival and demonstrated surgery as a successful treatment option.

Key words: brain metastasis, neurosurgical resection, lung cancer, prolonged survival

AIM OF THE DEMONSTRATION

In order to broaden the awareness of up-to-dated neurosurgical treatment possibilities, here we report a well-documented case of prolonged survival due to neurosurgical resection of brain metastasis in a patient with stage IV lung cancer.

CASE REPORT

An 82-year-old male was transferred to a clinical university hospital for elective brain metastasis resection. The patient complained of weakness in the right hand and leg as well as tightness in left side of face during the previous 8 months. Ten days before the admission, the patient felt dizzy and collapsed. He was admitted to a regional hospital. Computed tomography (CT) of the brain revealed a mass lesion in left parietal lobe (Fig. 1A) thus the patient was transferred for neurosurgical treatment. He had a history of neurosurgical resection of a single brain metastasis in right frontal lobe 6 years ago. Tumour immunophenotype yielded lung adenocarcinoma with low proliferation fraction. However, the primary tumour remained occult then as the chest CT was negative at that time. The medical history included also brain infarction in the left middle cerebral artery basin 6 years ago and moderate primary arterial hypertension.

At present, the general condition was estimated as average. The Glasgow coma scale score was 15. Neurological examination revealed lower reflexes and positive Babinski’s sign on the right side. The arterial pressure was 150/85 mmHg, the heart rate – 74 times per minute. Breathing sounds were normal having the frequency of 17 times per minute.

The contrast-enhanced CT of head and brain showed 3 mass lesions in the left hemisphere: in parietal lobe, measuring 2x1.5 cm; in frontal lobe, measuring 5 mm and in the basal part of temporal lobe, measuring 3 mm. The chest CT showed pathologic mass in right upper pulmonary lobe, measuring 3.4x2.8 cm (Fig. 1B). Craniotomy was performed in 2 steps. At first, left frontal bone craniotomy provided access for resection of metastasis from frontal lobe using neuronavigation. The second step included resection of parietal lobe metastasis, using neuronavigation from different approach. The first step lasted 70 minutes, the second – 115 minutes. Metastasis in temporal lobe was considered not accessible. The post-operative period was smooth. The histological examination of surgical material showed high-grade adenocarcinoma (Fig. 2A) expressing the following immunohistochemistry (IHC) markers: cytokeratins CK7 (Fig. 2B) and CKAE 1/3 as well as TTF-1, diagnostic of primary lung cancer. Six years ago histological examination of metastasis from right frontal lobe showed intermediate-grade papillary adenocarcinoma expressing the same IHC markers. The proliferation fraction of cancer was 9.6% (Fig. 2C) in the first event, increased now to 36.7% (Fig. 2D). Thus, the cancer has transformed to higher grade and significantly higher proliferation fraction. The higher grade was characterised by increased nuclear polymorphism and by loss of papillary architecture (Fig. 2E) resulting in solid sheets of neoplastic cells (Fig. 2F). The final diagnosis was lung adenocarcinoma with metachronous brain metastases, stage IV. The patient was transferred back to regional hospital for further recovery. Oncologist council recommended symptomatic therapy under the guidance of family doctor.

DISCUSSION

Both primary and metastatic tumours can affect the brain. Brain metastases outnumber primary neoplasms by at least 10 to 1, and they occur in 20% to 40% of cancer patients. About 80% of metastases are located in...
the cerebral hemispheres, 15% in the cerebellum, and 5% in the brainstem. In a retrospective neurosurgical review, 45.6% of the patients had solitary brain metastasis without other systemic metastases, 26.5% had single brain metastasis along with other metastases, and the rest had two or more brain metastases. The most common primary cancer metastasizing to the brain is lung cancer that is responsible for 50% of all metastasis (Patchell, 2003; El Kamar and Posner; 2004; Stark et al., 2011). Neurosurgical resection is recommended for patients with a single accessible brain metastasis, especially when the tumour size is large causing a considerable mass effect or obstructive hydrocephalus. Surgery is also favoured in patients with good performance status, who are functionally independent and in whom systemic disease is limited or absent and for patients with radioresistant primary tumour (Kalkanis et al., 2010). Multiple brain metastases in most cases represent a contraindication for neurosurgical treatment and resection is recommended only for the dominant lesion (Paek et al., 2005). Multiple metastases can be handled in a single operation if they are located in the same hemisphere and are close to each other (Fujimaki, 2005). Recurrent brain metastases develop in 31–48% of neurosurgically treated patients, the median survival is 4 months and 2-year survival is less than 6% (Bindal et al., 1992; Nussbaum et al., 1996; Lutterbach et al., 2002). Despite the generally short survival of patients affected by brain metastases of lung cancer (D’Antonio et al., 2014), longer survival has been reported as well (Kanou et al., 2014).

The known prognostic factors for prolonged survival after surgery in patients with non-small cell lung cancer and synchronous brain metastasis include small size of primary tumour and lack of lymph node involvement (Kanou et al., 2014). As the primary cancer in our patient remained occult by CT, such characteristics can be hypothetically assumed. In addition, the presented case was characterised also by initially low proliferation fraction that could supposed to be associated with limited tumour spread. However, conversion to high-grade (Sica et al., 2010) adenocarcinoma with notably higher proliferation fraction followed.

In conclusion, here we show a patient benefiting from prolonged survival of 6 years after successful neurosurgical resection of solitary lung cancer metastasis. The biological properties of the tumour including low proliferation at the first occurrence could contribute to longer survival and demonstrated surgery as a successful treatment option. The case is also notable for successful application of IHC detecting the tumour origin before it was radiologically visible.

Conflict of interest: None

REFERENCES

Fig. 2. Morphological and immunohistochemical characteristics of the tumour. 2A, Tissue structure of the recurrent tumour. Haematoxylin-eosin, original magnification (OM) 100x; 2B, Intense expression of cytokeratin (CK) 7. Immunoperoxidase (IP), anti-CK7, OM 100x; 2C, Low proliferation fraction by Ki-67 in the initial neurosurgical operation material. IP, anti-Ki-67, OM 400x; 2D, Increased proliferation fraction in the recurrent tumour. IP, anti-Ki-67, OM 400x; 2E, Expression of TTF-1 in the initial neurosurgical material. IP, anti-TTF-1, OM 100x; 2F, Expression of TTF-1 in the recurrent tumour. IP, anti-TTF-1, OM 100x. Note the loss of architecture as well as increased nuclear polymorphism.