Basal Ganglia Hyperintensity on T1-weighted Imaging of a Patient with Central Nervous System Metastasis Producing Carcinoembryonic Antigens

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Abstract

We herein report unusual basal ganglia hyperintense lesions on noncontrast T1-weighted magnetic resonance imaging in a patient with central nervous system metastasis from lung adenocarcinoma that was treated with gefitinib. T2*-weighted magnetic resonance imaging showed no hypointense lesions, thereby excluding the possibility of calcification or haemorrhage. A stereotactic brain biopsy of the left basal ganglia lesions revealed atypical cells, some of which formed a glandular lumen with a micropapillary pattern. These cells were immunopositive for markers of lung adenocarcinoma, thereby confirming the diagnosis of metastasis. We speculate that proteins, including carcinoembryonic antigens from the adenocarcinoma cells in the basal ganglia, may have contributed to the hyperintensity observed on noncontrast T1-weighted magnetic resonance imaging.

Key words: carcinoembryonic antigen, central nervous system metastasis, gefitinib, lung adenocarcinoma, magnetic resonance imaging


Introduction

The diagnosis of central nervous system (CNS) metastasis depends on the findings of magnetic resonance imaging (MRI) and the results of cerebrospinal fluid (CSF) analyses (1). Leptomeningeal or parenchymal enhancement on gadolinium contrast T1-weighted MRI (T1WI) is a hallmark of CNS metastasis (1). In contrast, we herein report a case of CNS metastasis with basal ganglia hyperintense lesions on noncontrast T1WI.

Case Report

We admitted a 64-year-old woman with a five-year history of lung adenocarcinoma to our department due to convulsions in the right limbs. Gefitinib, an inhibitor of epidermal growth factor receptor (EGFR) tyrosine kinase, had been administered to the patient for 1.5 years because conventional chemotherapy was ineffective and a mutation in EGFR correlated with the clinical responsiveness to gefitinib (2) was observed. The patient presented with slurred speech and subsequent clumsiness in the right upper limb seven months after gefitinib treatment was initiated (approximately one year before admission). Brain MRI showed hyperintense granular lesions in the bilateral basal ganglia on T1WI (Figure A) with mild surrounding contrast enhancement (Figure B). The lesions also showed slight hyperintensity on T2-weighted MRI (Figure C), but they were unremarkable on diffusion-weighted MRI. Gadolinium contrast T1WI also revealed mildly enhanced leptomeningeal lesions on the cerebral surface (Figure B). The patient gradually developed dysphagia, gait disturbance and cognitive impairment, and her mini-mental state examination score was 18/
A neurological examination performed on admission revealed the following findings: the patient’s conscious state was alert; her speech was nonfluent, although verbal communication was possible; no headaches or nuchal stiffness were noted; clonic convulsions were observed in the right palpebra and right limbs; the patient’s tendon reflexes were brisk in the limbs, and pathological reflexes were absent. The basal ganglia lesions were mildly enlarged and confluent on follow-up MRI (Figure D, E). Leptomeningeal lesions were also observed on T1WI (Figure D) and were more evident on contrast-enhanced T1WI (Figure E). The lateral ventricles were dilated. T2*-weighted MRI showed no hypointense lesions, thereby excluding the possibility of haemorrhage (Figure F). The basal ganglia appeared normal on computed tomography. The cancer lesions outside the CNS were adequately controlled.

The serum carcinoembryonic antigen (CEA) titre was 3.4 ng/mL, the CSF pressure was 140 mm H2O, the cell count was 1/μL, the total protein level was 24 mg/dL, the glucose
level was 79 mg/dL, the CEA titre was 0.5 ng/mL in the CSF, the CSF/serum ratio of CEA was 0.15 (cutoff = 0.01) (3) and repeated CSF cytology indicated class II disease. A stereotactic brain biopsy of the left basal ganglia lesions revealed atypical cells, some of which formed a glandular lumen with a micropapillary pattern (Figure G, H). These cells were immunopositive for markers of lung adenocarcinoma, thyroid transcription factor 1 (Figure I), napsin A (Figure J) and CEA (Figure K), thereby confirming the diagnosis of metastasis.

Discussion

Proteins, including CEAs from the adenocarcinoma cells in the basal ganglia, may have contributed to the T1WI hyperintensity observed in the present case. Other possible causes of hyperintense basal ganglia lesions on T1WI include the presence of lipids, calcification or paramagnetic substances (4). However, the possibilities of calcification and paramagnetic substances were excluded based on the findings of T2*-weighted MRI. Metastatic melanoma can also produce hyperintensity on T1WI due to the presence of blood products and melanin (5, 6); however, the biopsy performed in the present case did not detect either any haemorrhage or melanoma cells.

The CNS is a frequent site of recurrence in patients with non-small cell lung carcinoma who develop lesions after showing an initial response to gefitinib (7). However, recent prospective studies have suggested that gefitinib may be effective for treating brain metastases in patients with non-small cell lung carcinoma (8, 9). Disruption of the blood-brain barrier in the presence of CNS metastasis is likely to lead to locally increased concentrations of gefitinib (10). We speculate that gefitinib may have beneficially affected the CNS lesions in the present case, considering the patient’s slow progressive course and mild CSF abnormalities.

In conclusion, basal ganglia hyperintensity on noncontrast T1-weighted MRI is a rare but important finding for the differential diagnosis in patients with malignancy. MRI abnormalities can suggest the presence of metastatic lesions, especially in patients with lung adenocarcinoma who are treated with gefitinib, even when the original pulmonary lesions are controlled.

The authors state that they have no Conflict of Interest (COI).

References