REGIONE DEL VENETO

ULSS 1 DOLOMITI

HERPES SIMPLEX VIRUS TYPE 1 ENCEPHALITIS AND BRAIN GLIOMA: CASE REPORT

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Introduction

Herpes simplex virus type 1 (HSV-1) is the most common cause of sporadic fatal encephalitis worldwide. Although herpes simplex encephalitis (HSE) is not regarded as an opportunistic infection, the occurrence of HSE in immunocompromised subjects has been documented in patients with cerebral neoplasm on chemo- or radiotherapy as well as in patients receiving continuous immunosoppressive therapies such as natalizumab and TNF-al22fa. On the other hand, HSE may mimic clinical and MRI presentation of malignant neoplasms such as lymphomas and gliomas.

Case report

We describe a case of HSE in a 68-year-old patient admitted to our neurological department for an amnesic syndrome, hypodense right temporal lesion at CT scan and hyperdensity in T2 flair in right temporal lobe at MRI. The lesion was consistent with glioma and corticosteroid therapy with desametazone 8 mg bis in die was initiated.

While waiting for the NCH approach, after 6 days of corticosteroid therapy the patient developed a sudden clinical syndrome characterized by rapid onset of fever, headache, seizures, impaired consciousness and coma. CSF examination typically showed a lymphocytic pleocytosis (120/µL), increased number of erythrocytes and elevated protein levels (56 mg/dl). EEG showed prominent intermittent high amplitude slow waves (delta and theta slowing) and occasionally continuous periodic lateralized epileptiform discharges in the affected region. MRI and CT controls showed enlargement of the hypodense lesion at CT, extension of T2 hyperdensity at right temporal lesion and a new small left temporal lesion. Antiviral therapy with acyclovir was initiated. CSF polymerase chain reaction (PCR) testing was positive for HSV-1. After a few days of fever, headache and soporous-comatose state the patient recovered completely to his previous amnesic status. At clinical follow-up the patient presented only a few seizures. After a transient improvement and demarcation of hypodensity at CT and MRI, a four-month control revealed radiological progression of oedema and extension of the right temporal lobe lesion. The patient was admitted to a neurosurgical department and underwent a resection of the brain tumor which proved to be a glioblastoma GIV according to WHO.

Conclusions

HSE may mimic cerebral neoplasms and may also occur after surgery in patients with brain tumors immunodepressed by chemo- and radiotherapy. We presented the case of a patient with simultaneous HSV type 1 encephalitis and glioma. Corticosteroid therapy might have promoted the onset of the infectious disease.

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XLVIII CONGRESSO NAZIONALE
14-17 OTTOBRE 2017 – NAPOLI