Interesting image

Primary spinal leptomeningeal gliomatosis in a 3-year-old boy revealed with MRI and FDG PET/CT mimicking tuberculosis meningitis

Gliomatosis leptomeningea medular primaria en un niño de 3 años de edad, identificada por RM y FDG PET/TC imitando meningitis tuberculosa

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A 3-year-old boy was referred to neurologist for inward deviation of his left eye, weakness, lack of appetite, headache and behavioral change. On physical examination, the findings of bilateral abducens nerve paralysis were noted. Cranial magnetic resonance imaging (MRI) revealed diffuse leptomeningeal contrast enhancement at the supra-infratentorial regions predominantly involving the basal cisterns and also hydrocephalus and initially suggesting basilar meningitis. Cerebrospinal fluid (CSF) studies showed normal glucose and elevated protein levels but no tumor cells. No growth was detected in any of the bacterial cultures including acid-resistant bacteria. The patient was started on oral anti-tuberculosis medication and corticosteroid treatment with the presumed diagnosis of tuberculosis meningitis and a ventriculoperitoneal (VP) shunt was inserted due to hydrocephalus. After 4 months of treatment, the patient was re-admitted to hospital with vomiting, convulsion and confusion. Cranial MRI showed the similar findings to previous one, and spinal MRI depicted diffuse leptomeningeal contrast enhancement as well as microcystic changes along the spinal cord (Fig. 1). Histopathology of the dural biopsy material revealed widespread glioma infiltration of the subarachnoid space suggestive of leptomeningeal gliomatosis. This patient with unknown primary was referred for FDG PET/CT. Under child sedation, brain and whole body non-contrast enhanced PET/CT images were obtained 90 min after the intravenous injection of 148MBq (4mCi) 18F FDG using a Biograph 6 PET/CT. The scan depicted slightly increased FDG uptake (SUVmax 2.1) along the spinal cord without any other FDG-avid primary tumor. The patient was started on chemotherapy including vincristine and carboplatin (Figs. 2 and 3). His symptoms were relieved after completion of chemo.

Primary diffuse leptomeningeal gliomatosis (PDLG) is a rare disease thought to arise from isolated astrocytic cell nests also known as heterotopic neuroglial tissue within the leptomeninges (HLNT) in the subarachnoid space. The HLNT can transform into two

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Fig. 1. A and B. T1-weighted spin-echo MR images showed diffuse pathologic contrast enhancement at all spinal levels (A). Turbo inversion recovery magnitude (TIRM) with T2-weighted MR images revealed hyperintensity at these surfaces and multiple microcystic lesions (B).
patterns, and the diffuse type has poorer prognosis than solitary type.\textsuperscript{1} PDLG initially was misdiagnosed as tuberculosis meningitis because of characteristic clinical findings and CSF profile. Clinical symptoms and signs of PDLG include headache, cranial nerve involvement, meningismus, and decreased mental status. PDLG has to be differentiated from secondary diffuse gliomatosis which represent leptomeningeal infiltration by a primary parenchymal glioma, particularly medulloblastoma and glioblastoma, spreading along CSF pathways. However, the diagnosis of PDLG should only be established in the absence of intraparenchymal tumor. When contrast enhanced MRI of the brain and spinal cord fails to demonstrate a primary tumor, a clinical antemortem diagnosis can be made. FDG PET has been used in leptomeningeal gliomatosis to search for an underlying systemic malignancy.\textsuperscript{2} Tripathi et al. showed diffuse leptomeningeal metastases in a case of medulloblastoma by using FDG PET/CT.\textsuperscript{3} The usefulness of FDG-PET in differentiating benign from malignant disease and searching for the primary disease are well established but certain infections including tuberculosis show FDG uptake equivalent to that of tumors. However, as in our case, the presence of non-improving symptoms despite anti-tuberculosis and corticosteroid medication let us consider the diagnosis of malignant processes rather than inflammatory diseases.

**Conflicts of interest**

The authors have no conflicts of interest to declare.

**References**