Intracranial Castleman’s disease

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RESUMEN

ENFERMEDAD DE CASTLEMAN INTRACRANEAL

La enfermedad de Castleman es una entidad lipoproliferativa de etiología no conocida que afecta primordialmente al mediastino y fue informada por primera vez por Castleman en 1954, la afección intracranal es poco frecuente. Se presenta un caso con síntomas clínicos y signos radiológicos, así como los aspectos patológicos. La paciente de 39 años con historia de esplenectomía por un pseudo tumor en 1995 espondiloarteriopatía en 2000, linfadenitis en 2001 y un embarazo molar. Presentó astenia y adinamia, cefalea, náusea y vómito que mejoraron con antiinflamatorios no esteroides, crisis convulsivas, hemiparesia y hemianestesia fiebre con periodos de agravamiento y mejoría. Presentó cráneo hipertensivo la resonancia magnética y tomografía mostraron un tumor que parecía un meningioma. En la cirugía se mostró un tumor sólido amarillo que fue extirpado, el estudio patológico mostró compatibilidad con enfermedad de Castleman. El posoperatorio mostró ligera mejoría pero persistieron las crisis que se controlaron con oxicarbamazepina 900 mg al día y lesiones cutáneas que mostraron vasculitis que se controlaron con esteroides, a la fecha está controlada.

Palabras clave: enfermedad de Castleman, meningioma, tumor intracraneal, tratamiento.

Objective and importance: the Castleman’s disease is a benign lipoproliferative disorder of unknown etiology mostly involving the mediastinum and the first report was by Benjamin Castleman in 1954; intracranial involvement in Castleman’s disease is very unusual. We report clinical, radiological, and pathological features of intracranial involvement in a case of Castleman’s disease. Clinical presentation. a female patient of 39 years old, with history of splenectomy 75 g for an inflammatory pseudotumor in 1995, spondiloartropaty in the 2000, linfadenitis in the 2001 and a molar pregnant. She began with asthenia, adinamia, low feeding, holocranial headache 7/10, nauseas and vomiting, improvement with anti-inflamatory no steroids, partial seizures secondary generalized, left hemi paresia, hemianesthesia and fever, with periods of aggravate and improvement in three years period; in the neurological examination with papiledema and paresis of the left thoracic limb. Intervention: in the computed tomography (CT) and magnetic resonance imaging (MRI), with a neoplastic lesion extra-axial in plaque and wide base of dural attachment, right fronto-parietal and homogeneous enhancement, mimicking a meningioma; in laboratory studies with platelets in 1 132 000 x mm³. By surgery a resection of the tumor with wide dural attachment, solid, yellow - brown color and pale core; studies pathologic was a lesion meningeal inflammatory pseudotumor compatible with intracranial Castleman’s disease. Evolution of the patient had neurological improvement but with a intermitent febril syndrome, low feeding and pleural effusion, monitoring by hematology and neurology last control May 15, 2007 where she...
presents purple injuries in both arms is made biopsy that reports vasculitis is left with minimum doses of steroids neurologically stays free of focal motor seizures refers parestesias brachial lefts that can correspond to somatosensory seizures leaves with oxcarbazepine 900 mg/day. \textbf{Conclusion:} we presented a patient with intracranial Castleman’s disease with symptoms of elevated intracranial pressure and neurologic focalization, with antecedent of linfoploriferative systemic disease; by neuroimagen in the CT and MRI we find mimicking with a meningioma in plaque. The patient had improvement possurgical resection of the fronto-parietal lesion.

\textbf{Key words:} Castleman’s disease, meningioma, intracranial tumor.

\textbf{Figure 1.} Computed tomography, axial, simple (A) and with contrast (B) with low density in the white matter right fronto-parietal, and compression the ipsilateral ventricular system, and a lesion extraaxial right frontal, respective; axial T1 – weighted MRI (C), T2 – weighted (D) and T1-weighted with gadolinium (E) with cerebral edema fronto-parietal, compression of the ipsilateral ventricular system and the lesion that a homogeneous enhancement extra-axial in surface the right fronto-parietal, respective and sagittal in T1-weighted with gadolinium the lesion in the surface of the right fronto-parietal (F).

\textbf{Figure 2.} Pathologic studies. The specimen with diameters 7 x 5 x 1.5 cm, solid, wide dural attachment (A), microscopy with multiples follicles with interfollicular zones, and contain numerous small sclerotic vessels, by hematoxylin-eosin (B) and Masson stain (C).

\textbf{Figure 3.} MRI scan axial T1-weighted (A), T2-weighted (B), and T1-weighted post-gadolinium (C) and sagittal T1-weighted with gadolinium (D), to 10 months post-surgery, without the lesion in the surface of the right fronto-parietal, neither cerebral edema, and system ventricular normal, with hyper intensity in the surface right frontal.

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