A rare case of inflammatory CLOCC in a young woman

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Background

Cytotoxic lesion of the corpus callosum (CLOCC) is a MRI alteration of the white matter, associated with different conditions such as malignancies, infections, metabolic disorders, trauma, and other entities.[1-2] We describe a case of reversible inflammatory CLOCC diagnosed in a young woman.

Case report

A 23-year-old woman, with negative clinical history, was admitted to the emergency department for episodic visual alterations of few minutes, characterized by phosphenes or distortions of the enviromental contours, not followed by headache. The episodes were persistent in the last 2 months.
Neurological and ophthalmological examination did not show any pathological finding. Brain MRI showed a round, well-defined alteration of corpus callosum, suggestive of CLOCC. A lumbar puncture was performed: CSF analysis was negative for infections, paraneoplastic antibodies, anti-AQP4 and anti-MOG antibodies. Also the citological analysis was normal; the only relevant finding was the presence of type IV oligloconal bands (BOCs).

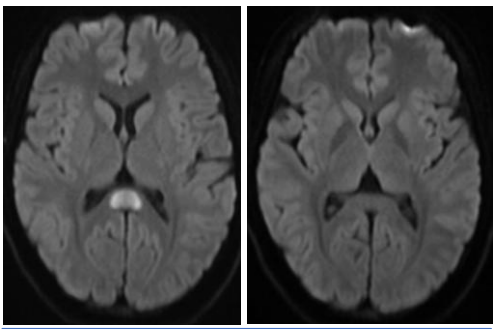

Fig.1 Brain MRI (DWI) showing a well-circumscribed, small, oval lesion in the splenium (A) and its resolution a month later (B).

The hematochemical tests excluded metabolic alterations, the reumathological panel was normal, while infectious screening was doubtful for EBV-IgM. This result was probably due to cross-reaction, given the complete absence of mononucleosis symptoms or signs: normal transaminases and indices of inflammation, no lymphadenopathies or hepatosplenomegaly. Patient's clinical picture remained stable and the visual symptoms resolved after a trial of i.v. steroids.
She was discharged with the diagnosis of "possible inflammatory CLOCC".
Brain MRI perfomed a month later showed a normal aspect of corpus callosum, as in most CLOCCs reported in literature.[1-2]

Conclusion

CLOCC clinical presentation relates to the underlying pathology rather than to the callosal lesion itself. Unlike many other lesions of the corpus callosum, CLOCCs do not demonstrate signs or symptoms of hemispheric disconnection. The underlying pathophysiological process is not known. The most widely accepted hypothesis considers CLOCCs as a consequence of a systemic inflammatory process, that affects the normal homeostasis of corpus callosum cells, determining a cytokine-induced cytotoxic damn.[1] In our case, we hypothesize an inflammatory origin of the condition for the presence of type IV BOCs, but we could not predict if this finding could have a prognostic value.

References
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