Case report: Reversible splenial lesion syndrome in a patient with epileptic seizures during bupropion therapy

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Introduction: Reversible splenial lesion syndrome (RESLES) has been reported in patients with a broad spectrum of conditions. We report the case of a young girl assuming bupropion and neuroleptic drugs who presented a reversible splenial lesion after her first generalized epileptic seizure.

Materials and methods: A 24-year-old girl with a history of cyclothymic depression was admitted to us because of a generalized seizure with mild facial concussion. She was being treated with bupropion, clomipramine and olanzapine and she had no history of seizures. The patient underwent brain MRI and electroencephalographic studies. Bupropion was immediately suspended; AED therapy with levetiracetam was started.

Results: After the generalized seizure, the patient remained in prolonged postictal state clinically characterized by confusion and psychomotor slowing. EEG studies showed significant and persisting epileptiform abnormalities despite AED therapy. There were no blood test alterations. Brain MRI showed in the splenium of the corpus callosum an oval area of hypointense signal on T1 and hyperintense signal on T2 with restricted diffusion and no enhancement following gadolinium administration. On follow-up, two weeks after discharge from our Unit, the neuroimaging detected the completely resolution of the lesion and the EEG appeared clearly improved.

Discussion: Bupropion is known as a cause of seizure threshold lowering; it is probably responsible for the generalized seizure and epileptic abnormalities found in our patient. RESLES is a rare entity associated with a wide spectrum of conditions like AED withdrawal, recurrent epileptic seizures, metabolic and infectious disorders causing encephalopathies or mild encephalitis. In our case, there was no history of previous seizures or AED withdrawal and all other pathological conditions have been excluded in particular we did not find extracallosal injuries on brain MRI, neurological disorders or blood test alterations. During hospitalization bupropion was discontinued and AED was started. These therapeutic changes probably have determined the clinical and electroencephalographic improvement and the resolution of the splenial lesion.

Conclusions: Despite these considerations we could not define the precise role of bupropion in determining RESLES. We hypothesized that the splenial lesion was a transient cytotoxic oedema consequent to bupropion-induced epileptic activity in a predisposed patient.

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