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Carbamazepine-induced non-epileptic myoclonus and tic-like movements

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ABSTRACT – Carbamazepine-induced abnormal movements have been reported in children and adult patients, and both non-epileptic myoclonus and tic-like movements have been reported in the same patient. Although a pathogenetic mechanism underlying carbamazepine-induced epileptic negative myoclonus has been proposed, a causative role of carbamazepine for positive myoclonus has not been fully identified. Here, we describe the video-documented case of an adult patient with non-epileptic myoclonus and tic-like movements persisting for 21 years, which appeared after he started carbamazepine treatment at 10 years of age. [*Published with video-sequences*]

Key words: carbamazepine, tics, non-epileptic myoclonus

Carbamazepine (CBZ)-induced movement disorders have been reported in children and adult patients (Neglia *et al*., 1984; Aguglia *et al*., 1987; Dhuna *et al*., 1991; Robertson *et al*., 1993; Holtmann *et al*., 2000), and both non-epileptic myoclonus and tic-like movements have been reported in the same patient (Aguglia *et al*., 1987). Epileptic negative myoclonus (ENM) has been described in patients with benign childhood epilepsy with centrotemporal spikes (BECTS) (Parmeggiani *et al*., 2004). We describe an adult patient presenting with non-epileptic myoclonus and tic-like movements persisting for 21 years, which appeared after he started CBZ treatment at 10 years of age.

Case report

A 31-year-old male was affected by idiopathic generalised epilepsy. Generalised tonic-clonic seizures started at 10 years of age. Soon after the first seizure, CBZ, at 200 mg/d, was started and increased three months later up to 800 mg/d because of recurrence of seizures. He continued taking CBZ until the age of 31 years although he had been seizure-free since the age of 15 years. We first observed the patient at 31 years of age. During the visit, he showed involuntary movements mainly in the right thumb and shoulders. His mother reported that these movements had appeared after CBZ initiation at 10 years of age and never disappeared. Plasma CBZ levels

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were within the reference range (6.3 μg/mL). Neurological examination and MRI were normal. The interictal EEG showed multifocal and diffuse sharp waves. Video-polygraphic recording showed repetitive rapid abduction of the right thumb related to a brief burst of rhythmic myoclonic jerks on the EMG tracing and tic-like movements (shrugging of the right shoulder, sniffing, snorting, facial gestures, and repetitive touching). Involuntary movements were increased by emotional stimuli and attenuated by voluntary movements or posturing, and ceased during sleep. No epileptic discharges appeared during movements. A causative role of CBZ was hypothesized and it was stopped within 15 days. A slow titration of levetiracetam (LEV), at 1,500 mg/d, was performed. The abnormal movements disappeared within one week following CBZ discontinuation and did not recur. Thus, it was not possible to perform jerk-locked averaging to investigate the type of myoclonus. After three months, when he continued treatment with LEV (at 1,500 mg/d), CBZ readministration (400 mg/d for two weeks) was followed by reappearance of involuntary movements. CBZ was stopped and the movements disappeared within a few days.

Discussion

Our patient, affected by idiopathic generalised epilepsy, presented with myoclonic jerks and tic-like movements at 10 years of age which persisted until the age of 31 years and disappeared a few days after CBZ discontinuation. A child with a benign occipital epilepsy treated with CBZ presenting with non-epileptic myoclonus and tics, disappearing a few days after CBZ discontinuation, was previously reported (Aguglia *et al*., 1987). In our case, CBZ was stopped and LEV introduced. Although the disappearance of the non-epileptic myoclonus could have been due to the anti-myoclonic effect of LEV (Magaudda *et al*., 2004), the latter could not explain the disappearance of tic-like movements. Moreover, the reappearance of involuntary movements after the reintroduction of CBZ and the disappearance after CBZ withdrawal supported the causative role of CBZ. It has been hypothesized that CBZ may itself cause tic disorders due to its dopaminergic action (Holtmann *et al*., 2000). Although a pathogenetic mechanism underlying CBZ-induced ENM has been proposed (Parmeggiani *et al*., 2004), a causative role of CBZ for positive myoclonus has not been fully identified. CBZ-induced non-epileptic abnormal movements are sporadically reported and potentially misdiagnosed. Careful diagnostic evaluation may be supported by further video-documented cases. □

Disclosures.
None of the authors has any conflict of interest to disclose.

Legend for video sequence

The patient shows repetitive abduction of the right thumb. Tic-like movements consist of facial gestures involving the mouth, eyes, and chin, repetitive touching, sniffing, and shrugging of the right shoulder.

Key words for video research on www.epilepticdisorders.com
Syndrome: idiopathic generalized not specified
Etiology: AED aggravation
Phenomenology: tics; myoclonus (non-epileptic)
Localization: not applicable

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