

Ictal asystole mimicking seizure deterioration in temporal lobe epilepsy

To the Editor,

I read with great interest the article by Guldiken *et al.* (2015) in which they report an interesting patient with temporal lobe epilepsy, in whom the change in seizure semiology was firstly interpreted as secondary generalization of seizures. However, after video-EEG monitoring (VEEGM), ictal asystole was detected and implantation of a pacemaker provided a total cure of the newly developed seizures, characterized by falls and myoclonic jerks (Guldiken *et al.*, 2015). I appreciate their effort in illustrating such a rare and interesting patient.

However, I would like to comment on a number of aspects of the article. I believe that the main issue mentioned in this report is a very crucial point. While cardiac syncope has been reported several times to be one of the most common misdiagnosis of epilepsy (Scheepers *et al.*, 1998; Smith *et al.*, 1999), the coexistence of epilepsy and ictal systole has also been reported, although as case reports (Howell and Blumhardt, 1989; van Rijckevorsel *et al.*, 1995; Isrel Tezer and Saygi, 2011; Strzelczyk *et al.*, 2011). In this case, pointing out new seizures manifesting as fall attacks, associated with coincidental cardiac syncope, is of great interest to clinicians. However, in this patient the cause-effect relationship between seizures and ictal asystole was not discussed, which probably also constitutes another important issue among neurologists. VEEGM had revealed six habitual seizures from the left temporal origin and in four of them, asystole (lasting from 22 to 35 seconds) was recorded. I surely agree with the indication of a pacemaker in this patient, however, I would like to emphasize that further data of cardiac evaluation for the patient was not mentioned which might give some perspective regarding the pathophysiology of asystole. To date, several other mechanisms have been suggested to account for the occurrence of arrhythmia in epilepsy, including, in particular, the lateralization hypothesis (Oppenheimer *et al.*, 1991; Tinuper *et al.*, 2001) and, more recently, another in which sympathetic and parasympathetic discharges are triggered interictally and ictally (Sevcencu and Struijk, 2010). I would like to point out that ictal asystole in this patient (Guldiken *et al.*, 2015) had occurred soon after the appearance of the left temporal seizure pattern, which may support a mechanism of secondary ictal asystole due to the ictus. There are also a

limited number of reports in which ictal activity preceding ictal asystole is illustrated by VEEGM (Katz *et al.*, 1983; Agostini *et al.*, 2012). Of note, in the patient reported by Katz *et al.*, further cardiac investigations were reported to be normal, leading to the consideration of a mechanism of secondary cardiac asystole (Katz *et al.*, 1983). However, to the best of our knowledge, in the majority of previous reports ictal asystole has been reported to occur simultaneously (van Rijckevorsel *et al.*, 1995) or precede EEG seizure onset (Howell and Blumhardt, 1989; Isrel Tezer and Saygi, 2011). These study results indicate that cardiac pathology is the primary responsible origin of asystole. In addition, an hypothesis of decreasing seizure threshold caused by the hypoxic syncopal episodes of an unproven cardiac problem has also been suggested (Bergey *et al.*, 1997; Isrel Tezer and Saygi, 2011).

In conclusion, I believe that this case provides valuable data to enhance our knowledge of the pathophysiology of cardiac asystole in epilepsy, as this (cardiac asystole following an ictal pattern) has been reported extremely rarely (Katz *et al.*, 1983; Agostini *et al.*, 2012). This report supports the plausibility of secondary cardiac asystole (Novak *et al.*, 1999; Isrel Tezer and Saygi, 2011) and argues that asystole is a contributing factor for the severity of seizure attacks (Howell and Blumhardt, 1989), considering the total recovery from fall attacks and myoclonic jerks in this patient (presented by Guldiken *et al.* [2015]) after pacemaker implantation. Considering that there is no consensus on this issue (the cause-effect relationship between ictus and cardiac asystole), I suggest that possible ictal seizure onset before asystole might not have been demonstrated in the previously mentioned reports (cardiac asystole preceding ictal activity) (Howell and Blumhardt, 1989; van Rijckevorsel *et al.*, 1995; Isrel Tezer and Saygi, 2011), as intracranial EEG monitoring was not conducted in any of these studies. Another hypothesis could also be based on the different mechanisms of cardiac asystole among patients. However, future studies, including intracranial EEG recordings, need to be conducted for clarification of these crucial arguments. □

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Disclosures.

The author has no conflict of interest to disclose.

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Author response**To the editor,**

We read with great interest the comments of Onder regarding our recent case report (Guldiken *et al.*, 2015). He addressed that the cause-effect relationship of seizures and asystole has remained unclear in some previously published case reports where asystole preceded the onset of ictal activity on scalp EEG (Irsel Tezer and Saygi, 2011; Howell and Blumhardt, 1989). A primary cardiac pathology has been discussed as a probable cause of asystole in these patients. Ictal EEG seizure patterns on scalp EEG may frequently occur after clinical seizure onset. In some of these patients, invasive EEG evaluation may demonstrate earlier seizure onset relative to surface evaluations if the invasive electrodes are placed in the seizure onset zone.

In our patient, however, the asystole was beyond any doubt secondary to the epileptic seizure activity. She did not have any cardiac disorder. She had had a left temporal lobe epilepsy due to cavernoma and we could demonstrate that the ictal EEG seizure pattern in the left temporal lobe preceded the asystole by 30 seconds. Thus, the asystole in our patient resulted from the temporal lobe seizure activity and her recurrent falls were due to syncope-associated atonia, rather than an expression of secondary seizure generalization. □

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