Subclinical Tonic-Clonic Epileptic Seizure Detected by an Implantable Loop Recorder

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Summary

A 73-year old man received an implantable loop recorder (ILR) for the evaluation of transient loss of consciousness (TLOC) spells. His medical history was without any epileptic convulsions or automatism. ILR recording during a spontaneous episode revealed the presence of a regular, narrow QRS complex tachycardia associated with low-amplitude, high-frequency, continuous or discontinuous artifacts, consistent with myopotentials. During the event, the regular, low-amplitude continuous signals gradually became discontinuous, with a prolongation of the inter-signal cycle length, until their disappearance after manual activation of the ILR. The patient was diagnosed as experiencing subclinical tonic-clonic epileptic seizures. Antiepileptic drug treatment was initiated, and the patient has remained free of TLOC symptoms during 13 months follow-up. (Int Heart J 2013; 54: 289-291)

Key words: Epilepsy, Syncope, Loss of consciousness

The implantable loop recorder (ILR) is a useful instrument for detection of heart rhythm disturbances in patients with transient loss of consciousness (TLOC) spells of unknown origin. For the most part ILRs have proven to be of greatest value in determining an arrhythmic basis for syncope,1,2 and on occasion have been helpful in demonstrating that certain patients believed to be suffering from epilepsy, but whose symptoms are ‘drug-refractory’, may in fact be suffering from syncope.3 However, the ability of an ILR to unmask a seizure disorder in patients in whom the cause of TLOC is undetermined despite the usual investigations has been largely overlooked.

Previous reports of ILR use in recurrent transient loss of consciousness (TLOC) in drug refractory epileptic patients have been published, and an association between ILR recorded myopotentials and clinically documented convulsive symptoms have been observed.4,5 The present case describes a patient presenting with TLOC in the absence of apparent convulsions, in whom ILR monitoring detected myopotentials consistent with a subclinical epileptic seizure.

Case Report

A 73-year-old man with a history of prior remote myocardial infarction and a 4-month-old hemorrhagic cerebral infarction was referred for evaluation of TLOC. The episode was characterized by sudden skin pallor followed by an abrupt fall onto the floor from a seating position in absence of convulsion. The episode lasted approximately 30 to 60 sec, and the patient regained consciousness immediately but sustained facial injuries from the fall. He denied having experienced palpitations or chest pain before or after the spell. His wife, who witnessed the episode, reported that the patient suddenly became pale and unresponsive to verbal stimuli; however, she did not observe any evidence of automatism or tonic-clonic movements. There was no report of epilepsy in the patient’s family history.

Blood pressure on admission was 114/82 mmHg and heart rate was 72 bpm and regular. A general physical examination and detailed neurologic assessment, including orthostatic vital signs, were normal. A 12-lead electrocardiogram (ECG) showed sinus rhythm at a rate of 82 bpm, with premature ventricular complexes and no abnormal Q wave. The PR interval was 0.16 sec, the QRS duration was 0.10 sec, and the QT interval was 0.37 sec. Transthoracic echocardiography showed diffuse, severe left ventricular (LV) hypokinesia consistent with ischemic cardiomyopathy, an LV ejection fraction of 30%, and LV end-diastolic/end-systolic diameters of 66/54 mm. Chest X-rays revealed a cardiothoracic index of 57%. The serum brain natriuretic peptide concentration was mildly elevated to 186 pg/mL (normal range < 20 pg/mL). A 24-h ambulatory ECG showed one episode of asymptomatic nonsustained ventricular tachycardia (average ventricular rate, 140 bpm). Computed tomography of the brain revealed the presence of a low-density area in the left frontal lobe, corresponding to the territory supplied by the left middle cerebral artery (Figure 1).

Two consecutive sleep electroencephalograms (EEG) were normal. Since the patient presented with an ischemic cardiomyopathy, a depressed LV ejection fraction and nonsustained ventricular tachycardia on 24-h ambulatory ECG, an ar-
rhythmic cause of TLOC was suspected and an implantable cardioverter defibrillator (ICD) was considered. However, inasmuch as the possibility of an epileptic seizure had not been excluded given the hemorrhagic cerebral infarction, ICD implantation was deferred.

To further investigate the cause of TLOC, a Reveal® DX 9528 ILR (Medtronic Inc., Minneapolis, MN) was implanted; ILR use in this setting was in accordance with the European Society of Cardiology syncope practice guidelines. One month later, a similar 1-min episode without convulsion occurred at home, and the ILR was manually activated by the patient’s wife. The ECG recording revealed the presence of a regular, narrow QRS complex tachycardia, at a rate of 120 bpm, associated with low-amplitude, high-frequency, continuous or discontinuous artifacts, consistent with myopotentials. Figure 2 shows the regular, low-amplitude, continuous high frequency signals gradually becoming discontinuous as the episode progressed, with prolongation of the inter-signal cycle length, until disappearance of the myopotential artifact after manual activation of the ILR. The duration of ILR recording, between onset of continuous and the end of discontinuous, low-amplitude, high-frequency signals, was 70 sec.

After review, it seemed most probable that the initially sustained high-frequency signals corresponded to myopotentials elicited by the tonic phase, while the following nonsustained bursts of lower-frequency signals were consistent with myopotentials elicited by the clonic phase of an epileptic seizure, despite the absence of overt visible abnormal muscular activity.

Treatment for the seizure disorder was initiated (levetiracetam, 500 mg/day). In addition, given his ischemic cardiomyopathy he underwent implantation of an ICD for primary prevention of sudden cardiac death. At a follow-up of 13 months, the patient was free from TLOC recurrence, and had not required ICD therapy.

**DISCUSSION**

ILRs are typically used in the investigation of unexplained syncope, and have been helpful for excluding an epilepsy diagnosis in some patients in whom that diagnosis had been incorrectly applied. However, the case presented here illustrates the potential utility of ILRs for documenting seemingly atonic epileptic spells; that is, spells in which bystander observers report no apparent tonic-clonic activity prior to or during the loss of consciousness episode.

The importance of the initial evaluation in TLOC, which includes a detailed medical history (with special attention being paid to symptoms preceding and following the episodes), physical examination, and ECG findings, has been emphasized in several professional practice guidelines. However, despite careful initial assessment by even experienced clinicians, a confident diagnosis is made in fewer than 50% of cases. On the basis of these observations, the Consensus Report of the European Heart Rhythm Association recommended the early use of ILR monitoring in patients presenting with unexplained TLOC. Such monitoring is primarily used to define an arrhythmic cause of syncope in TLOC patients. However, the detection, by the ILR, of myotonic electrical signals associated with tonic-clonic epileptic seizures, has been described previously and has been used to confirm an epilepsy diagnosis.

Convulsions may be observed during epileptic seizures as well as during syncope of cardiac or noncardiac origin. TLOC associated with convulsion, abnormal movement, or automatism is an important finding in the diagnosis of epilepsy, because the diagnosis of epilepsy is made by clinical symptoms with or without abnormal EEG findings. The differential diagnosis in elderly individuals presenting with TLOC is particularly challenging, as it often occurs in the context of underlying cardiovascular and potentially neurological structural disease.
and in many cases may present with symptoms that are atypical for either syncope or seizures.\textsuperscript{10,11} The previously reported incidence of epileptic seizures is as high as 10.9\% after subarachnoid hemorrhages\textsuperscript{12} and 7.7\% after hemorrhagic cerebral infarctions.\textsuperscript{13} In most clinical settings, the presence or absence of convulsions or abnormal movement is judged by the information from the patient’s clinical manifestations by the witness.

Specific symptoms such as convulsions, abnormal movements, or automatism and any arrhythmias except for sinus tachycardia in the ILR were not observed in the patient. It, therefore, might be difficult to confirm a diagnosis of epilepsy. However, sinus tachycardia, associated with regular continuous or discontinuous myopotentials on ECG during TLOC led us to confirm the diagnosis of tonic-clonic epileptic seizure.

In conclusion, to the best of our knowledge this is the first report of a subclinical tonic-clonic epileptic seizure detected by ILR. This observation suggests that, whereas ILR recordings have clearly unmasked over-diagnosis of epilepsy in TLOC patients,\textsuperscript{10} they may prove similarly useful in establishing the correct diagnosis of epilepsy in the apparently rare patient in whom there is absence of evident convulsions or automatism.

\section*{REFERENCES}


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