Absence-to-bilateral-tonic-clonic seizure: a generalized seizure type

Short title: Absence-to-bilateral-tonic-clonic seizure

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Abstract

Objective: To test the hypothesis that absence seizures can evolve to generalized tonic-clonic seizures, we documented electro-clinical features of this novel seizure type.

Methods: In four large video-EEG databases, we identified recordings of seizures starting with impaired awareness and that, without returning to baseline interictal state, evolved to generalized tonic-clonic seizures. We extracted the detailed semiological and electrographical characteristics of these seizures, and we documented the clinical background, diagnoses and therapeutic responses in these patients.

Results: We identified 12 seizures from 12 patients. All seizures started with a period of impaired awareness and bursts of generalized spike or polyspike and slow-wave discharges – the hallmark of absence seizures. Without returning to baseline, the nonmotor (absence) phase was followed by tonic-clonic convulsions. We coined this novel generalized seizure type absence-to-bilateral-tonic-clonic seizure. Most patients had idiopathic generalized epilepsies, though with a high incidence of unusual features and poor therapeutic response.

Conclusions: Absence-to-bilateral-tonic-clonic seizures are a novel generalized seizure type. Clinicians should be aware of this seizure for correctly diagnosing the patients. This novel seizure type may further elucidate generalized ictogenesis.
Introduction

Identifying seizure types in patients with epilepsy is an important step in diagnosis, and is necessary for choosing the appropriate therapy\(^1\). Identifying the clinical phenomena during seizures (semiology) along with the EEG abnormalities contribute to identifying the various seizure types and may contribute to diagnosis of epilepsy syndrome. In the recently published position paper on seizure classification, the International League Against Epilepsy (ILAE) recognized four new generalized seizure types: absence with eyelid myoclonia, myoclonic absence, myoclonic–atonic seizures and myoclonic–tonic–clonic seizures\(^2\). Video-EEG monitoring is needed to document the electro-clinical features of these seizures. However, this resource-demanding method is mainly used in patients with drug-resistant focal epilepsy\(^3\). Therefore, detailed documentation of those generalized seizures types that occur less frequently, is challenging, leaving the possibility that some seizure-types remain unrecognized.

We noticed that many patients with generalized epilepsies described a sense of confusion preceding tonic-clonic seizures. This was in agreement with the experience of several epilepsy experts, who mentioned in review papers and textbook chapters, absence seizures or absence status epilepticus heralding generalized tonic-clonic seizures\(^4\text{-}6\). However, none of these provided video or EEG documentation of the seizures. Only one case report provided EEG documentation (without video) of a “petit mal seizure” with immediate transition to a “grand mal seizure”\(^7\). We hypothesized that the initial confusion reported by patients was an integral part of the seizure, rather than a simple prodrome. To investigate this hypothesis, and to provide evidence for this seizure type that has previously been observed\(^4\text{-}7\) but not systematically documented in a cohort of patients, we identified seizures starting with confusion and continuing with generalized tonic-clonic seizures in video-EEG databases of four epilepsy centers.
Although this peculiar seizure type has previously been described\textsuperscript{4-7}, here we provide detailed electro-clinical documentation of absence seizures evolving to tonic-clonic seizures, in a cohort of patients. We coined this generalized seizure type absence-to-bilateral-tonic-clonic seizure. Besides the diagnostic implication, this novel seizure type contributes to a better understanding of generalized ictogenesis.

**Methods**

*Patients and recordings*

We extracted cases from the video-EEG databases of four large epilepsy centers (Danish Epilepsy Center, Dianalund; Bellaria Hospital, Bologna; Children's Hospital Agia Sophia, Athens; Jefferson Comprehensive Epilepsy Center, Philadelphia). Inclusion criteria: seizures beginning with a period of impaired awareness evolving directly to a generalized tonic-clonic seizure (formerly known as primary generalized tonic-clonic seizures\textsuperscript{8}) without return to baseline (interictal) state between the impairment of awareness and the convulsive behavior. Impaired awareness was defined as video-documented semiology features consisting of motor arrest, lack of responsiveness or confusion, with ictal EEG correlate for at least 5 seconds, corresponding to generalized nonmotor (absence) seizure\textsuperscript{8,9}. There were no exclusion criteria. In the video-EEG databases, we searched first for the key word “absence”. This resulted in 106 patients. Then we screened the recordings for the occurrence of generalized tonic-clonic seizures. In addition, we added cases remembered by the authors.

For each patient, we extracted the following data: age, gender, family history, development, cognitive and neurological status, age at onset, reported seizure types, neuroimaging, medication,
current seizure frequency and diagnosis. Descriptive statistics were performed with Statistica software – version 13 (TIBCO Inc., Palo Alto, CA, USA).

Electro-clinical features

All patients underwent video-EEG monitoring. EEG was recorded with standard 10-20 system electrodes\textsuperscript{10} or the extended standard array of the International Federation of Clinical Neurophysiology\textsuperscript{11}. Video-recordings were synchronized with the EEG. For each included seizure, we extracted the semiological features\textsuperscript{12} and the EEG features\textsuperscript{13} that occurred during the ictal and postictal period.

Standard Protocol Approvals, Registrations, and Patient Consents

This non-interventional study was reviewed and obtained clearance from the Ethics Committees of the participating centers. Patients gave written informed consent for the retrospective use of the de-identified dataset, including post-processed video-EEG recordings, with blurred faces.

Data Availability

Individual de-identified EEG recordings in EDF format (European Data Format), video-files (AVI format) and self-extracting video-EEG files that include the reader (EXE format – available only for Microsoft Windows operation system) will be shared after publication. Unrestricted access to these data will be made available from the day of the online publication of the article, until 2030, using a publicly-accessible repository (https://doi.org/10.5061/dryad.tht76hdvj). The whole dataset can be downloaded using this link: https://doi.org/10.5061/dryad.tht76hdvj.
Results

We identified 12 patients (6 female) who had video-EEG documentation of a period with impaired awareness evolving to generalized tonic-clonic seizure. Table 1 summarizes the demographic and clinical data of the patients. Their age was between 12 and 56 years (mean: 32.67; median: 24; standard deviation: 17; interquartile range: 18-50). Age at onset of seizures was between 2 and 20 years (mean: 12.64; median: 13; standard deviation: 5.15; interquartile range: 11-17). Two patients had family history of seizures. In all patients, the interictal EEG showed bilateral synchronous (generalized) epileptiform discharges, consisting of spike-and-slow-waves and/or polyspike-and-slow-waves (Table 1). In one patient, MRI showed bi-frontal posttraumatic lesions (contusion) and in one patient a small (4 mm) periventricular nodular heterotopia, of uncertain significance for the patient’s seizures. Neuroimaging was not done in one patient and was normal in all other patients.

In one patient, the seizure was triggered by hyperventilation (patient 1) and in one patient the seizure was triggered by intermittent photic stimulation at 23 Hz (patient 12). In the remaining patients, seizures occurred spontaneously.

Figures 1 and 2 show examples with EEG recordings of the seizures. Video-EEG files are available on DRYAD (see section on data sharing in the Methods).

All 12 seizures started with an initial period of impaired awareness. In three patients, this was prolonged (6 minutes in patient-3 and > 30 minutes in patients 4 and 5); in the remaining patients, its duration was between 5 and 28 seconds (median 10 seconds; interquartile range: 7-15). In all patients, bursts of bilateral synchronous (generalized) epileptiform discharges were recorded during the initial period of impaired awareness: in four patients, generalized spike-wave discharges and in eight patients mixed spike and polyspike and slow-wave discharges. In six patients, the bursts were irregular and in six patients they were rhythmic (2.5 – 4 Hz). The electro-clinical features of this
initial phase of the seizures were similar to generalized nonmotor (absence) seizures or absence status epilepticus (in the patients with prolonged initial phase). In all patients, generalized tonic-clonic behavior directly followed the initial phase of impaired awareness, without return to baseline cognitive performance before convulsive movement occurred. In five patients (6-9, 11) forced head version was observed at the start of the tonic phase. In seven patients (1-7), the generalized spike/poly spike and slow-wave bursts continued until the EEG became obscured by muscle artifacts (Figure 1). In five patients (8-12), a high frequency (beta-alpha) ictal rhythm with evolution in time appeared at the beginning of the tonic phase (Figure 2).

Diagnosis was uncertain in two patients. Ten patients were diagnosed with Idiopathic / Genetic Generalized Epilepsy (IGE): seven with Juvenile Absence Epilepsy, two with Juvenile Myoclonic Epilepsy and one with IGE with generalized tonic-clonic seizures only, GTCS-only). Five of the 10 patients diagnosed with IGE had some unusual features: one had a very small periventricular nodular heterotopia (4 mm), one had infantile autism, one had mild cognitive impairment, two had a history of status epilepticus. Seven patients were drug-resistant (six of the 10 patients with IGE).

Three clinical patterns emerged in our cohort:

A. Patients with absences seizures that occasionally evolved to GTCS (eight patients);
B. Absence seizures and rare absence status epilepticus, terminating with GTCS (three patients);
C. Rare GTCS preceded by absences, but no isolated absence seizures (one patient)

Discussion

In this paper, we present a detailed video-EEG documentation of a generalized seizure type that we coined absence-to-bilateral-tonic-clonic seizures (ABTCS). This peculiar seizure has been
previously described by several authors\textsuperscript{4-7}, but a systematic electroclinical documentation (video and EEG) in a cohort of patients has been lacking. The initial phase of the seizure consists of impaired awareness with the electrographic correlate of generalized bursts of spike or polyspike and slow-waves, the hallmark of generalized nonmotor (absence) seizures\textsuperscript{2,5,8}. These directly evolve to tonic and then clonic activity without an intervening baseline interictal state.

While 42\% of our patients had forced head version at the beginning of the tonic phase, this does not necessary imply that these seizures are focal. A large study showed that lateralizing symptoms occurred in 52\% of patients with idiopathic generalized epilepsies\textsuperscript{14}. The appearance of lateralized symptoms in a generalized seizure is in line with the concept that these seizures originate at some point within, and rapidly engaging, bilaterally distributed networks, and that generalized seizures can be asymmetric\textsuperscript{15}.

Although our search strategy was useful in providing good quality video-EEG documentation of the seizures, from 12 patients, it does not allow estimation of the prevalence of this seizure type, for the following reasons: 1) Only patients who gave informed consent were included into the databases. 2) The referral pattern to long-term video-EEG monitoring is biased towards the more complicated patients. 3) This is not a population-based screening. Estimating the prevalence of this seizure type was beyond the goals and limitations of this study. Our aim was to collect compelling evidence from a group of patients (n>10) with well-documented electroclinical features of this seizure type. Although we only identified 12 patients with ABTC seizures, it seems probable that this seizure-type is more frequent, since the absences preceding the tonic-clonic seizure can be very short (as little as 5 seconds), so it might go unnoticed unless seizures are recorded with video-EEG. As patients with IGE usually respond well to therapy and are rarely evaluated with video-EEG monitoring, it is more difficult to detect this seizure type. How often it occurs remains to be determined.
Most patients in our cohort were diagnosed with generalized epilepsy syndromes encompassed within the IGE spectrum. One patient (11) was classified as IGE with GTCS only, in which no other generalized seizures (myoclonic or absences) occurred. We cannot exclude that this patient might present very brief absence episodes, i.e., phantom absences, as previously reported in some adult patients with IGE, and that would include our patient in the proposed syndrome of IGE with phantom absences. The other patients in our cohort had full-blown absence seizures in the history, which differentiates them from the previous study, in which such patients were excluded. Half of our patients diagnosed with IGE had some unusual clinical features and most were not seizure free, in spite of adequate choice of anti-seizure medication. This could indicate that some additional factors are needed for development of ABTC seizures, and that the therapeutic response is less favorable in patients who have ABTC seizures. The alternative explanation is that this seizure type is underestimated in the general population with IGE who respond well to the medication and do not undergo video-EEG monitoring, hence, video-EEG databases are biased towards those patients who have unusual clinical features, absence status epilepticus and inadequate therapeutic response.

Several generalized seizures consisting of combinations of other seizure types have been previously described for absence seizures (eyelid myoclonia with absences, myoclonic absence) and for tonic-clonic seizures (myoclonic-tonic-clonic seizure). It is important that clinicians are aware of these seizures when taking history, because questions targeting these details can contribute to the identification of the seizures and help in diagnosing the epilepsies in these patients. Combination of absence and tonic-clonic seizure, co-occurring in the same seizure-event has not been documented by video-EEG recordings before. This seems to combine two seizure categories involving different neuronal substrates and cardinally opposed mechanisms. In absences, ictogenesis is due to a predominance of inhibitory activity (aggravated by GABAergic drugs), in contrast to generalized tonic-clonic seizures in which an excess of excitatory activity is present.
How might absences trigger convulsive activity and how might it be prevented? Absence seizures reflect dysfunction of thalamocortical networks. Spike-wave bursts reflect synchronized firing of neurons in cortex and thalamus with widespread neuronal firing and synchrony\textsuperscript{17}. Recent studies in Genetic Absence Epilepsy Rats from Strasbourg suggest that seizures are sustained by a bidirectional activation of cortico-thalamo-cortical circuits, with cortical and reticular thalamic neurons driving a process dependent upon calcium T-channel activation\textsuperscript{18}. As the reticular cells target only thalamocortical neurons, spread of seizures must take place at the cortical level. While absences show primary cortical involvement in somatosensory cortex, activation of cortical projections beyond this somatosensory network likely accounts for the appearance of convulsive activity. Treatment approaches might best focus on preventing initiation of absences, which are the trigger for convulsive activity.

In conclusion, recognizing the generalized seizure type described in this paper is important both for diagnostic reasons and for understanding of generalized ictogenesis. Therapeutic implications can be inferred from the mechanisms noted above. Should tonic-clonic seizures prove refractory to ordinary therapy in these patients, one might consider targeting T-channel mechanisms; this approach would need to be tested to determine efficacy.
## Appendix 1: Authors

<table>
<thead>
<tr>
<th>Name</th>
<th>Location</th>
<th>Contribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sándor Beniczky</td>
<td>Danish Epilepsy Centre, Dianalund, Aarhus University Hospital, Aarhus, and Aarhus University, Aarhus, Denmark</td>
<td>Designed and conceptualized study; analyzed the data; interpreted the results and drafted the manuscript.</td>
</tr>
<tr>
<td>Guido Rubboli MD</td>
<td>Department of Neurology, Danish Epilepsy Centre, Dianalund; University of Copenhagen, Copenhagen, Denmark</td>
<td>Designed and conceptualized study; collected and analyzed the data; interpreted the results and revised the manuscript for intellectual content.</td>
</tr>
<tr>
<td>Athanasios Covavis MD</td>
<td>Department of Child Neurology, the Children’s Hospital ”Agia Sophia”, Athens, Greece</td>
<td>Designed and conceptualized study; collected and analyzed the data; interpreted the results and revised the manuscript for intellectual content.</td>
</tr>
<tr>
<td>Michael R. Sperling MD</td>
<td>Jefferson Comprehensive Epilepsy Center, Department of Neurology, Thomas Jefferson University, Philadelphia, Pennsylvania, PA, USA</td>
<td>Designed and conceptualized study; collected and analyzed the data; interpreted the results and revised the manuscript for intellectual content.</td>
</tr>
</tbody>
</table>
**Figure 1:** EEG recording (patient 6) showing the transition from the absence to the tonic phase of the ABTC seizure. The two channels in green are surface electromyography recorded from the left and right deltoid muscles (EMG 1/2). The generalized spike-wave bursts continue, while the tonic muscle activity builds up.
**Figure 2:** EEG recording (patient 10) showing the transition from the absence to the tonic phase of the ABTC seizure. The six channels in green are surface electromyography recorded from the left and right deltoid muscles (EMG 1/2), brachial biceps (EMG 3/4) and anterior tibial (EMG 5/6) muscles. The generalized spike-wave bursts are gradually replaced by evolving ictal rhythm (low amplitude fast activity).
Table 1: Demographic and clinical data

<table>
<thead>
<tr>
<th>Patient nr.</th>
<th>Age (years), gender</th>
<th>Family history</th>
<th>Development cognition &amp; neurologic exam</th>
<th>Age at onset &amp; seizure types</th>
<th>EEG</th>
<th>Neuroimaging</th>
<th>Medication and current seizure frequency</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>21, female</td>
<td>Younger sister had febrile seizures</td>
<td>Normal</td>
<td>12 years: AS and GTCS while playing computer game.</td>
<td>3 Hz GSWD, accentuated during sleep</td>
<td>ND</td>
<td>Levetiracetam; seizure-free</td>
<td>JAE</td>
</tr>
<tr>
<td>2</td>
<td>48, male</td>
<td>Negative</td>
<td>Head-trauma at 18 yo; normal cognition and exam.</td>
<td>20 years: AS and GTCS</td>
<td>Irregular GS/PWD, accentuated during sleep</td>
<td>MRI: bi-frontal contusions.</td>
<td>Valproate &amp; lamotrigine; no GTCS since 2013; 2-3 AS/week</td>
<td>Uncertain. Posttraumatic focal epilepsy or JAE or both.</td>
</tr>
<tr>
<td>3</td>
<td>25, female</td>
<td>Negative</td>
<td>Developmental delay (IQ ~ 65).</td>
<td>2 years: AS; 7 years: GTCS</td>
<td>GPSWD</td>
<td>Normal</td>
<td>Valproate; seizure-free.</td>
<td>Uncertain. Presumed genetic</td>
</tr>
<tr>
<td>4</td>
<td>56, male</td>
<td>Negative</td>
<td>Normal.</td>
<td>16 years: AS; 20 years: GTCS. Since 41 years: absence status terminated with GTCS (twice)</td>
<td>GS/PWD</td>
<td>Normal CT &amp; MRI</td>
<td>Phenytoin and phenobarbitone; sporadic absences; 2 GTCS /year.</td>
<td>JAE</td>
</tr>
<tr>
<td>5</td>
<td>55, male</td>
<td>Negative</td>
<td>Mild cognitive impairment (slowing); otherwise normal.</td>
<td>17 years: AS; 27 years: GTCS; 55 years: one absence status terminating with GTCS</td>
<td>GS/PWD</td>
<td>Normal CT &amp; MRI</td>
<td>Valproate &amp; phenobarbitone; daily AS; 4 GTCS/year.</td>
<td>JAE</td>
</tr>
<tr>
<td>6</td>
<td>17, male</td>
<td>Negative</td>
<td>Normal</td>
<td>13 years: AS &amp; GTCS</td>
<td>3 Hz GS/PWD</td>
<td>Normal MRI</td>
<td>Lamotrigine and levetiracetam; no JAE</td>
<td>JAE</td>
</tr>
<tr>
<td>#</td>
<td>Age</td>
<td>Gender</td>
<td>Status</td>
<td>Age at Onset</td>
<td>Seizure Type</td>
<td>MRI Findings</td>
<td>Treatment</td>
<td>Notes</td>
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<tr>
<td>7</td>
<td>12, female</td>
<td>Unknown (adopted)</td>
<td>Normal</td>
<td>11 years: AS &amp; GTCS</td>
<td>3 Hz GSWD</td>
<td>Normal MRI</td>
<td>Lamotrigine; seizure-free.</td>
<td>JAE</td>
</tr>
<tr>
<td>8</td>
<td>45, female</td>
<td>Uncertain</td>
<td>Normal</td>
<td>AS: uncertain age of onset; 37 years: GTCS.</td>
<td>3 Hz GS/PWD</td>
<td>Normal MRI</td>
<td>Valproate; 2-3 GTCS/year; several AS/week</td>
<td>JAE</td>
</tr>
<tr>
<td>9</td>
<td>18, male</td>
<td>Negative</td>
<td>Normal</td>
<td>6 years: AS &amp; GTCS</td>
<td>3 Hz GS/PWD</td>
<td>Normal MRI</td>
<td>Valproate &amp; lamotrigine; 3-4 AS/week; 1 GTCS/month</td>
<td>JAE</td>
</tr>
<tr>
<td>10</td>
<td>53, female</td>
<td>Younger brother, mother and maternal uncle have epilepsy</td>
<td>Normal</td>
<td>12 years: GTCS, myoclonic jerks; 15 yrs: AS</td>
<td>GSWD</td>
<td>MRI showed a very small nodular heterotopia, left lateral ventricle (frontal) - 4 mm.</td>
<td>PB, BRV, TPM. (She has tried also LTG, VPA, ZNS, LVT); 4-7 GTCS/month; 5-6 AS/year (probably underestimated); 2-3 myoclonic jerks/month.</td>
<td>JME</td>
</tr>
<tr>
<td>11</td>
<td>23, male</td>
<td>Negative</td>
<td>Normal</td>
<td>17 years: GTCS (in the morning); brief period of unresponsiveness preceding the GTCS.</td>
<td>3 Hz GS/PWD</td>
<td>Normal MRI</td>
<td>Lamotrigine; 1 GTCS/year (poor compliance)</td>
<td>IGE with GTCS-only</td>
</tr>
<tr>
<td>12</td>
<td>19, female</td>
<td>Negative</td>
<td>Infantile autism; otherwise normal</td>
<td>13 years: GTCS in the morning; AS, myoclonic jerks</td>
<td>3 Hz GS/PWD</td>
<td>Normal MRI</td>
<td>LEV and ZNS (LTG did not work); rare (&lt; 1/year) GTCS. No AS. No myoclonic jerks.</td>
<td>JME</td>
</tr>
</tbody>
</table>

AS = absence seizure; IGE = Idiopathic Generalized Epilepsy; JAE = Juvenile Absence Epilepsy; GSWD = Generalized Spike-Wave Discharges; GS/PWD = Generalized Spike/Polyspike and Wave Discharges; GTCS = Generalized Tonic-Clonic Seizure; ND = not done
Video 1- http://links.lww.com/WNL/B181
Video 2- http://links.lww.com/WNL/B182

References


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