Clinical features of Todd’s post-epileptic paralysis

Loren A Rolak, Paul Rutecki, Tetsuo Ashizawa, Yadollah Harati

Abstract
Two hundred and twenty nine patients with generalised tonic–clonic seizures were prospectively evaluated. Fourteen were identified who had transient focal neurological deficits thought to be Todd’s post-epileptic paralysis (PEP). Eight of these 14 patients had underlying focal brain lesions associated with the postictal deficits. All patients with PEP were weak, but there was wide variation in the pattern (any combination of face, arm, leg), severity (plegia to mild), tone (spastic, flaccid, or normal), and reflexes (increased, decreased, or normal). Significant sensory loss occurred in only one patient. The only other signs of PEP were aphasia (in five patients all with underlying lesions) and gaze palsy (in four patients). Post-epileptic paralysis persisted from half an hour to 36 hours (mean of 15 hours). Post-epileptic paralysis may occur with the first seizure or after many years of seizures and does not appear after every seizure. The clinical features of PEP are thus heterogeneous.

Bravais in 1827 first noted that paralysis may follow a unilateral seizure, a condition he termed hemiplegia epileptique.1 Todd used the same term (epileptic hemiplegia), apparently arrived at independently, in his 1854 description.2 He noted that paralysis may occur on only one side even when both sides had been convulsed and that in contrast to the spasticity expected from a central nervous system lesion, the limbs were flaccid. By 1890, Hughlings Jackson had extended these observations and described post-epileptic aphasia, sensory loss, stupor, and mania.3 Since then post-epileptic paralysis (PEP) has become a well accepted syndrome, and numerous post-epileptic symptoms have been reported, including hemihemnopsia,4 complete blindness,3 weakness,6–11 unilateral pupillary dilatation,12 aphasia,13 bulimia,14 and prolonged confusion.15 These descriptions, however, are all in the form of case reports, and there has never been a prospective, systematic analysis of PEP to describe its fundamental clinical features, such as incidence, physical findings, duration, and relation to underlying pathology. Focal findings are especially vexing after generalised (as opposed to partial) seizures because there is often no structural (focal) brain lesion to account for them. We studied the clinical characteristics of PEP after generalised tonic-clonic seizures.

Patients and methods
We prospectively evaluated all patients admitted to one hospital during a one year period with generalised tonic-clonic seizures, defined as the sudden onset of bilateral symmetrical tonic and clonic convulsive activity with loss of consciousness. Some patients could have had a very brief unwatched focal onset to their seizure (partial seizure with secondary generalisation), but every effort was made to exclude all patients with any focality in the beginning or subsequent course of their seizure. All patients had a thorough neurological examination immediately after the seizure and at about two hour intervals until all neurological deficits returned to baseline. All patients also had brain imaging with contrast CT scanning or MRI, EEG, and other appropriate diagnostic tests.

Results
Two hundred and twenty nine patients (all men, mean age 46 years) presented with generalised tonic-clonic seizures, of whom 14 (mean age 47 years) had transient focal post-ictal deficits (PEP). Their clinical features are summarised in the table. Eight of the 14 patients with PEP had an underlying structural lesion, which in each case was a pre-existing ischaemic stroke in the hemisphere contralateral to the paralysis. All strokes were in the middle cerebral artery territory, affecting the frontal or temporal lobe. Only 51 of the 215 patients without PEP had structural lesions (χ² 7.69, p < 0.01). Altogether, 69 of the 229 had structural lesions and eight had PEP. All patients with PEP had weakness, but it varied from very mild paresis to complete plegia. The weakness involved any combination of face or arm or leg. The tone could be flaccid, normal, or spastic, and the reflexes decreased, normal, or increased. Very minimal sensory deficits occurred in eight patients but only one complained of noticeable numbness. Five patients, all with pre-existing left hemisphere strokes, developed aphasia, which was fluent in one and non-fluent in four. The only other neurological deficit seen in PEP was gaze palsy, in four patients.

Post-epileptic paralysis persisted from half an hour to 36 hours, with a mean of 15 hours. The nature, duration, and severity of PEP were unrelated to the duration or severity of the seizures, the presence or absence of underlying lesions, or any changes on the EEG. Weakness always persisted longer than other symptoms. Post-epileptic paralysis occurred sporadically
and did not follow every seizure. Some patients had suffered recurrent seizures for years before their first episode of PEP, and most had subsequent seizures without associated PEP. There was no apparent reason why some seizures resulted in PEP and others did not. Six patients with PEP had a baseline abnormal EEG (see table), which in each case showed a slow wave (theta or delta) focus in the contralateral frontal or temporal lobe, corresponding to an underlying ischaemic stroke. Four of the tracings also had epileptiform spike activity accompanying the focal slowing. Only one patient (number 2, with idiopathic seizures) had an EEG recorded during his PEP, and it showed no abnormality.

Discussion

Transient focal neurological deficits after an epileptic seizure are often called Todd's paralysis in recognition of their description by the British neurologist Robert Todd. Since then, research has focused primarily on possible mechanisms of post-epileptic paralysis, but its clinical features have never been systematically studied, and there is almost no information about the nature, duration, or aetiology of the deficits that occur after a seizure. Our conclusions about PEP are limited by the population studied (adult male veterans) and the restriction to generalised tonic-clonic seizures without ictal focality. In this group, PEP was a heterogeneous syndrome encompassing a variety of neurological signs including aphasia, gaze palsy, weakness, and (rarely) numbness. The motor deficits were highly variable, from mild to severe, focal to hemiparetic. Abnormalities never persisted beyond 36 hours. Most patients with PEP had an underlying structural lesion but, interestingly, in many (43%) no cause was found. The aetiology of PEP is not clear. It may be due to neuronal exhaustion from hypoxia or substrate depletion because a localised region of the brain is already damaged or is more severely affected by the seizure, or because some underlying condition, such as vascular disease, predisposes to insufficient metabolism. Alternatively, it may result from inhibitory neuronal discharges, arterial venous shunting, or release of endogenous inhibitory (possibly opioid) substances. Our study, though not intended to address the aetiology of PEP, demonstrates its great clinical diversity and thus suggests that it may have multiple causes.

Table: Clinical features of patients with post-epileptic paralysis (PEP)

<table>
<thead>
<tr>
<th>Patients</th>
<th>Age (years)</th>
<th>Duration of epilepsy (years)</th>
<th>Cause of seizures</th>
<th>EEG</th>
<th>Weakness</th>
<th>Pattern</th>
<th>Tone</th>
<th>Reflexes</th>
<th>Extensor plantar</th>
<th>Other signs</th>
<th>Duration of PEP (hours)</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>50</td>
<td>10</td>
<td>Alcohol withdrawal</td>
<td>NL</td>
<td>0/5</td>
<td>F, A, L</td>
<td>?</td>
<td>?</td>
<td>Yes</td>
<td>Gaze palsy</td>
<td>8</td>
</tr>
<tr>
<td>2</td>
<td>28</td>
<td>1st seizure</td>
<td>Idiopathic</td>
<td>NL</td>
<td>4/5</td>
<td>A, L</td>
<td>?</td>
<td>?</td>
<td>None</td>
<td>None</td>
<td>36</td>
</tr>
<tr>
<td>3</td>
<td>25</td>
<td>15</td>
<td>Idiopathic</td>
<td>NL</td>
<td>4/5</td>
<td>A, L</td>
<td>?</td>
<td>?</td>
<td>None</td>
<td>Numbness</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>31</td>
<td>1</td>
<td>Idiopathic</td>
<td>NL</td>
<td>4/5</td>
<td>A, L</td>
<td>?</td>
<td>?</td>
<td>None</td>
<td>None</td>
<td>24</td>
</tr>
<tr>
<td>5</td>
<td>36</td>
<td>3</td>
<td>Idiopathic</td>
<td>NL</td>
<td>4/5</td>
<td>A, L</td>
<td>?</td>
<td>?</td>
<td>None</td>
<td>None</td>
<td>24</td>
</tr>
<tr>
<td>6</td>
<td>28</td>
<td>5</td>
<td>Idiopathic</td>
<td>NL</td>
<td>3/5</td>
<td>A, L</td>
<td>?</td>
<td>?</td>
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<td>None</td>
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<tr>
<td>7</td>
<td>51</td>
<td>1st seizure</td>
<td>Stroke</td>
<td>Focal theta</td>
<td>4/5</td>
<td>A, L</td>
<td>?</td>
<td>?</td>
<td>Yes</td>
<td>Gaze palsy</td>
<td>6</td>
</tr>
<tr>
<td>8</td>
<td>54</td>
<td>1st seizure</td>
<td>Stroke</td>
<td>Focal spikes + theta</td>
<td>0/5</td>
<td>A, L</td>
<td>?</td>
<td>?</td>
<td>No</td>
<td>Gaze palsy + aphasia</td>
<td>12</td>
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<tr>
<td>9</td>
<td>48</td>
<td>1</td>
<td>Stroke</td>
<td>Focal spikes + delta</td>
<td>4/5</td>
<td>A, L</td>
<td>?</td>
<td>?</td>
<td>No</td>
<td>Aphasia</td>
<td>24</td>
</tr>
<tr>
<td>10</td>
<td>61</td>
<td>10</td>
<td>Stroke</td>
<td>Focal theta</td>
<td>4/5</td>
<td>L</td>
<td>NL</td>
<td>NL</td>
<td>No</td>
<td>Aphasia</td>
<td>36</td>
</tr>
<tr>
<td>11</td>
<td>63</td>
<td>8</td>
<td>Stroke</td>
<td>Focal spikes + delta</td>
<td>4/5</td>
<td>F, A</td>
<td>?</td>
<td>?</td>
<td>No</td>
<td>Aphasia</td>
<td>8</td>
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<tr>
<td>12</td>
<td>70</td>
<td>6</td>
<td>Stroke</td>
<td>Focal spikes + theta</td>
<td>0/5</td>
<td>F, A, L</td>
<td>?</td>
<td>?</td>
<td>No</td>
<td>Gaze palsy</td>
<td>6</td>
</tr>
<tr>
<td>13</td>
<td>47</td>
<td>8</td>
<td>Stroke</td>
<td>Focal spikes + theta</td>
<td>4/5</td>
<td>A, L</td>
<td>?</td>
<td>?</td>
<td>No</td>
<td>None</td>
<td>0.5</td>
</tr>
</tbody>
</table>

F = face, A = arm, L = leg, ? = increased, | = decreased, NL = normal.

2 Todd RB. Clinical lectures on paralysis, certain diseases of the brain, and other affections of the nervous system. London: John Churchill, 1854;284-307.
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