Drug-resistant parietal lobe epilepsy: clinical manifestations and surgery outcome

Marjan Asadollahi, Michael R. Sperling, Amin H. Rabiei, Ali A. Asadi-Pooya
Jefferson Comprehensive Epilepsy Center, Department of Neurology, Thomas Jefferson University, Philadelphia, Pennsylvania, USA

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ABSTRACT – Aim. We reviewed a large surgical cohort to investigate the clinical manifestations, EEG and neuroimaging findings, and postoperative seizure outcome in patients with drug-resistant parietal lobe epilepsy (PLE).

Methods. All drug-resistant PLE patients, who were investigated for epilepsy surgery at Jefferson Comprehensive Epilepsy Center between 1986 and 2015, were identified. Demographic data, seizure data, EEG recordings, brain MRI, pathological findings, and postsurgical seizure outcome were reviewed.

Results. In total, 18 patients (11 males and seven females) were identified. Sixteen patients (88%) had tonic-clonic seizures, 12 (66%) had focal seizures with impaired awareness, and 13 (72%) described auras. Among 15 patients who had brain MRI, 14 patients (93%) had parietal lobe lesions. Only three of 15 patients (20%) who had interictal scalp EEG recordings showed parietal interictal spikes. Of 12 patients with available ictal surface EEG recordings, only three patients (25%) had parietal ictal EEG onset. After a mean follow-up duration of 8.6 years, 14 patients (77.7%) showed a favourable postoperative seizure outcome.

Conclusion. In patients with PLE, semiology and EEG may be misleading and brain MRI is the most valuable tool to localize the epileptogenic zone. Postsurgical seizure outcome was favourable in our patients with drug-resistant parietal lobe epilepsy.

Key words: epilepsy, parietal lobe, seizure, surgery

Parietal lobe epilepsy (PLE) accounts for approximately 5% of all focal epilepsies (Salanova, 2012). The parietal lobe has rich connections with the adjacent lobes (Akimura et al., 2003; Kamali et al., 2014) and as a consequence, clinical symptoms and seizure semiology in PLEs are diverse and the EEG findings are often mislocalizing (Salanova et al., 1995a; Binder et al., 2009; Ristic et al., 2012; Liava et al., 2014; Francione et al., 2015).

Like other focal epilepsy syndromes, a sizable number of patients with PLE may experience drug-resistant
seizures (Liava et al., 2014; Francione et al., 2015). Surgery is a potential therapeutic option for these patients (Liava et al., 2014; Francione et al., 2015). However, surgical treatment of drug-resistant PLE can be challenging. First, electroclinical presentations of PLE may be misleading. Moreover, many areas within the parietal lobe have critical functions (Kamali et al., 2014; Kim et al., 2004a; Binder et al., 2009). Based on previous studies, compared with temporal lobe epilepsy, success rates and seizure outcome for epilepsy surgery in drug-resistant PLEs is less promising (Binder et al., 2009; Harroud et al., 2012; Liu et al., 2015).

In this retrospective study, we reviewed a large surgical cohort to investigate the demographic and clinical characteristics, EEG and neuroimaging findings, and post-operative seizure outcome in patients with drug-resistant PLE. This may shed more light on presentations and surgical outcome in this uncommon syndrome of focal epilepsies.

**Materials and methods**

In this retrospective study, all patients with a diagnosis of drug-resistant PLE, who underwent epilepsy surgery at Jefferson Comprehensive Epilepsy Center, were recruited. Patients were prospectively registered in a database from 1986 to 2015. The diagnosis of PLE was made by the epileptologists working at this institution based on ictal semiology, MRI lesion, and EEG findings. There was no age limitation to enter the study. All patients underwent a comprehensive presurgical evaluation including brain MRI and prolonged video-EEG monitoring.

All patients underwent resective brain surgery. Office visits, telephone contacts, and letters were used to monitor seizure outcome periodically. Postsurgical seizure outcome was classified as Class 1 (seizure-free for one year or more after surgery), Class 2 (less than three seizures per year during the last year of follow-up or nocturnal seizures only), and failure. Aura was not considered as a relapse; only postoperative tonic-clonic seizures and focal seizures with impaired awareness were considered as relapse.

Age, gender, race, epilepsy risk factors (e.g., history of febrile seizures in childhood, any family history of epilepsy, etc.), age at seizure onset, seizure type(s) and history, date of surgery, date of the first relapse (if any), date of the last contact with all patients, and EEG and MRI findings were registered routinely.

No informed consent was required as this was a retrospective study. This study was conducted with the approval of the Thomas Jefferson University Institutional Review Board.

**Results**

Among 1,225 patients with epilepsy who were investigated for surgery between 1986 and 2015, we identified 19 patients (1.6%) with drug-resistant PLE. Sufficient data were available for 18 patients (11 males and seven females). Mean age (±standard deviation) at the epilepsy onset was 16.2 (±11.5) years and at the time of surgery was 29.7 (±14.2) years. The mean (±standard deviation) preoperative epilepsy duration was 13.13 (±10.22) years. None of our patients had a history of febrile convulsion at childhood. Three patients (16.6%) had a positive family history of epilepsy. Only one patient (5.5%) had a history of status epilepticus before surgery. The mean (±standard deviation) full scale IQ of the patients was 93.8±9.6.

Sixteen patients (88%) had a history of tonic-clonic seizure(s) before surgery. Twelve patients (66%) described focal seizures with impaired awareness and 13 patients (72%) described auras; seven (38.8%) patients had sensory auras contralateral to the resected side, two (11%) had contralateral simple motor signs (one hand tonic posture and one hand clonic jerks), two (11%) patients reported dizziness, one (5.5%) had auditory aura, one (5.5%) had olfactory aura, and one (5.5%) reported epigastric aura. All patients who had motor auras underwent intracranial EEG recordings to determine the epileptogenic zone. Brain MRI was available for 15 patients. MRI showed parietal tumours in five (33%) patients. Four (26.6%) patients had gliosis or encephalomalacia and one (6.6%) had dysplasia, one (6.6%) had multiple cavernous hemangioma, one patient (6.6%) had right medial parietal cavernous hemangioma, one patient (6.6%) had parietal lobe infarct, one patient (6.6%) had right parietal superficial sidenosis, and one patient (6.6%) had normal MRI.

Interictal scalp EEG recordings were available for 15 patients (83%). Two patients (13%) had right parietal, one (6.6%) had left parietal, five (33.3%) had right extra-parietal (T4, T6, F4, C4), and five (33.3%) had left extra-parietal (F7, T3, T5, F3) interictal spikes. All the unilateral interictal spikes were ipsilateral to the resected side. Two patients (13%) did not have any interictal spikes on surface EEG recordings. Ictal surface EEG recordings were available for 12 patients (66.6%). Of these, two (16.6%) had right parietal ictal onset, one (8%) had left parietal ictal onset, three (25%) had right extra-parietal (one right mid-temporal, one right posterior temporal, and one right fronto-central), four (33.3%) had left extra-parietal (two had left fronto-central, one left mid-temporal, and one left posterior temporal), and two (16.6%) had bilateral mid-temporal ictal onsets. Unilateral ictal EEG findings were ipsilateral to the resected side. Intracranial EEG recordings
Table 1. Clinical findings in patients with parietal lobe epilepsy.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Number of patients</th>
<th>Age at seizure onset (years)</th>
<th>Aura/Interictal EEG recording</th>
<th>Ictal EEG recording</th>
<th>Mean post-operative follow-up</th>
<th>Engel Class 1 outcome</th>
<th>Engel Class 2 outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Binder et al., 2009</td>
<td>40</td>
<td>25</td>
<td>not available</td>
<td>26.9% one ipsilateral focus; 42.3% multiple ipsilateral foci; 30.7% contralateral focus</td>
<td>45 months</td>
<td>57.5%</td>
<td>10%</td>
</tr>
<tr>
<td>Bartolomei et al., 2011</td>
<td>17 (10 had surgery)</td>
<td>7.3</td>
<td>82% aura; 58% somatosensory</td>
<td>Not specified</td>
<td>14% one ipsilateral focus; 66.6% multiple ipsilateral foci; 19% contralateral focus</td>
<td>2 years</td>
<td>60%</td>
</tr>
<tr>
<td>Salanova et al., 1995a</td>
<td>82</td>
<td>14.1</td>
<td>94% aura; 63.4% somatosensory</td>
<td>14% parietal spikes; 78.5% extraparietal ± parietal spikes; 7.5% no spikes</td>
<td>not well described</td>
<td>20.4 years</td>
<td>45.5%</td>
</tr>
<tr>
<td>Salanova et al., 1995b</td>
<td>34</td>
<td>28.4</td>
<td>79% aura; 62% somatosensory</td>
<td>16.6% parietal spikes, 66.8% extraparietal ± parietal spikes, 16.6% no spike</td>
<td>not specified</td>
<td>12.3 years</td>
<td>64%</td>
</tr>
<tr>
<td>Kim et al., 2004b</td>
<td>40 (27 had surgery)</td>
<td>27</td>
<td>67.5% aura; 32.5% somatosensory</td>
<td>not well described</td>
<td>not well described</td>
<td>&gt;1 year</td>
<td>53.9%</td>
</tr>
<tr>
<td>Kim et al., 2004a</td>
<td>38</td>
<td>10</td>
<td>78.9% aura; 31.6% somatosensory</td>
<td>68% lateralized; 8% localized; 8% falsely localized; 8% normal; 8% bilateral</td>
<td>81.5% lateralizing; 10.5% localizing; 7.8% non-conclusive</td>
<td>50.7 months</td>
<td>39.5%</td>
</tr>
<tr>
<td>Francione et al., 2015</td>
<td>40 (11 children, 29 adults)</td>
<td>5.9</td>
<td>80% aura; 62.5% somatosensory</td>
<td>25% localizing; 32.5% regional; 27.5% false-localizing; 5.5% only lateralizing; 5% false lateralizing; 2.5% normal</td>
<td>25% localizing; 53.1% regional; 3.12% false-localizing; 9.4% one lateralizing; 3.1% false-lateralizing; 6.2% diffuse</td>
<td>9.4 years</td>
<td>75%</td>
</tr>
<tr>
<td>Cascino et al., 1993</td>
<td>10</td>
<td>24</td>
<td>not available</td>
<td>30% no spikes; 70% parietal ± extraparietal spikes</td>
<td>not well described</td>
<td>31.4 months</td>
<td>90%</td>
</tr>
<tr>
<td>This study</td>
<td>18</td>
<td>16.2</td>
<td>72% aura; 38.8% somatosensory</td>
<td>20% parietal spikes; 66.6% extraparietal spikes; 13.3% no spike</td>
<td>25% parietal onset; 75% extraparietal onset</td>
<td>8.6 years</td>
<td>61.1%</td>
</tr>
</tbody>
</table>
were available for four (22%) patients. Of these, one had left parieto-occipital origin, two left parietal, and one right fronto-parietal ictal onset.

Seven patients (38%) underwent left parietal resection, 10 (55%) right parietal resection, and one patient (5%) underwent right parietal subpial transection. Pathological findings were available for 13 patients (72%). Four patients (30%) had low-grade tumours (astrocytoma, oligodendroglioma, ganglioglioma, and desmoplastic neuroepithelial tumour), one patient (7%) had brain infarction, three patients (23%) had gliosis, one patient (7%) had cortical dysplasia, and two patients (15%) had cavernous hemangioma. Two patients (15%) had normal pathological findings.

The mean (±standard deviation) follow-up duration after epilepsy surgery was 8.6 (±5.9) years (minimum of one year and maximum of 21 years). Fourteen (77.7%) patients had favourable seizure outcome. Eleven (61.1%) patients had Class 1 (seizure-free) and three (16.6%) patients had Class 2 (rare seizures) seizure outcome. Favourable surgical outcomes were not relevant to specific pathological findings.

Discussion

Parietal lobe epilepsy accounts for a small percentage of all focal epilepsies (Salanova, 2012; Siegel, 2003). In a large case series (Salanova et al., 1995a), only 6% of patients undergoing epilepsy surgery at Montreal Neurological Institute between 1929 and 1988 had PLE. In our study, PLE represented 1.6% of patients who were investigated for epilepsy surgery from 1986 to 2015. Clinical symptoms and seizure semiology are diverse in patients with PLE. In our study, 72% of patients reported aura. The most common reported aura was somatosensory feelings (38.8%). This finding is consistent with previous studies (Cascino et al., 1993; Salanova et al., 1995a; Salanova et al., 1995b; Kim et al., 2004a; Bartolomei et al., 2011). However, somatosensory auras are not specific to parietal lobe epilepsy (Perven et al., 2015). Similarly, other aura types, including those often attributed to other lobes (e.g. epigastric aura), could be seen in patients with PLE (Tufenkjian and Lüders, 2012; Liava et al., 2014; Francione et al., 2015). There is no pathognomonic clinical manifestation in patients with PLE.

Our study showed that scalp EEG monitoring is often misleading or non-localizing in patients with PLE. This finding has also been observed in previous studies (Cascino et al., 1993; Salanova et al., 1995a; Kim et al., 2004a; Binder et al., 2009; Bartolomei et al., 2011; Liava et al., 2014; Francione et al., 2015) (table 1). Interictal and ictal scalp EEG recordings in patients with PLE have lower localizing value compared with temporal lobe epilepsy patients (Ristic et al., 2012). In our study, 93.3% of patients had parietal lobe lesions on brain MRI, which is consistent with previous studies (Cascino et al., 1993; Salanova et al., 1995a; Kim et al., 2004a; Binder et al., 2009; Bartolomei et al., 2011; Francione et al., 2015). Therefore, compared to ictal semiology and EEG findings, brain MRI is a more reliable tool to localize the epileptogenic zone in patients with PLE.

Surgery is a beneficial therapeutic option for patients with drug-resistant PLE. In our study, more than three quarters of patients benefited from surgery. In previous studies, similar postsurgical outcomes were observed (Cascino et al., 1993; Salanova et al., 1995a; Kim et al., 2004a; Binder et al., 2009; Bartolomei et al., 2011; Francione et al., 2015) (table 1).

In conclusion, PLEs are rare causes of focal epilepsy syndromes. Clinical manifestations and EEG findings in patients with PLE may be unusual or even misleading, but brain MRI is the most valuable tool to localize the epileptogenic zone in these patients. In patients with drug-resistant PLE, surgery is a valuable therapeutic option.

Study limitations

This was a small and clinic-based series and may not represent the full spectrum of PLEs.

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References


Drug-resistant parietal lobe epilepsy


Kamali A, Sair HI, Radmanesh A, Hasan KM. Decoding the superior parietal lobule connections of the superior longitudinal fasciculus/arcuate fasciculus in the human brain. *Neuroscience* 2014; 277: 577-83.


