

Clinical Study

Temporal Lobe Epilepsy in the Elderly

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The incidence of epilepsy has bimodal distribution peaking at the extremes of life. Incidence is greater in younger and older age groups (Hauser et al., 1993, Sidenvall et al., 1993, Forsgren et al., 1996, and Olafsson et al., 2005). As the world population ages more elders with epilepsy will be identified. In the high-income countries with longer life expectancy, the number of elders with epilepsy will be even higher. CPSs account for 40% of all seizure types in the elderly (Hauser et al., 1992); however, the proportion with temporal lobe epilepsy (TLE) is uncertain.

1. Causes

The specific causes of TLE in the elderly have not been clearly disclosed. With advancing age underlying coexisting factors are more likely to be identified, and the proportion of people classified as idiopathic is less when compared to the younger age groups. Nonetheless, up to half of elderly patients with epilepsy go without an identified cause.

In the under 65-year age group, head trauma, brain tumors, and CNS infections are common associations. Etiology of seizures veers towards a cerebrovascular origin in the elderly [6]. Dementing illnesses have also taken their place as a significant etiology [7].

Idiopathic TLE has been reported in adults with familiar history of epilepsy (mean age of onset: 25.5; range: 11–45 years). This group was also found to have a better prognosis [8]. Similar reports in elderly patients are missing. There are however case reports of mesial temporal lobe sclerosis (MTS) in elderly persons with new onset seizures. These accounts highlight the challenging differential diagnosis overlap with primary cognitive disorders and nonparaneoplastic limbic encephalitis [9]. New onset TLE has also been reported without evidence of MTS [10].

The etiology of hippocampal damage is discussed in a retrospective study of 38 patients with adult onset TLE [11]. The median age at onset was 37.8 years (range: 21.0 to 78.7 years). A total of seven patients, 60 years and older, were included in this report. In all cases, the common fea-

tures encompass frequent CPSs (range <1 to 600 seizures per month) developing over one year (defined as the median time from the initial onset of seizures to the time of assessment). Based on MRI evidence of hippocampal atrophy, history of causative events, concomitant disease, cerebrospinal fluid results, and autoantibodies patients were classified in one of four etiologic categories; secondary hippocampal sclerosis (HS); idiopathic HS; definite limbic encephalitis (LE) and MRI defined possible LE. Eleven patients met secondary HS criteria. Three had presented prolonged seizures and one nonprolonged repetitive seizures. In this subgroup, 36% showed bilateral MRI hippocampal abnormalities. Two patients had experienced a severe head trauma; two, old infarct; one a porencephalic cyst and one neurofibromatosis type I. Only two patients in this subgroup were 60 years and older. One showed bilateral hippocampal atrophy after head trauma and the other was unilateral. The latter reported 500 seizures per month. In the idiopathic HS group, all seven subjects had unilateral hippocampal abnormalities. Four underwent selective amygdalohippocampectomy and histopathology confirmed HS. In this subgroup of idiopathic HS, none of the seven patients were 60 years or older. Definite LE included nine patients with 5 (56%) showing bilateral hippocampal abnormalities on MRI. Six of the nine patients had sequential MRIs with initial hippocampal swelling, followed by normalization and ultimately atrophy and high-intensity signal. In addition to seizures, memory impairment was clinically significant. Four patients had positive voltage

gated potassium channel antibodies (VGKC abs) and one anti-Hu antibodies. The remaining four patients were diagnosed with rectal, small cell lung cancer (SCLC), testicular tumor with Ma2 antibodies, and a uterine leiomyosarcoma, respectively. In this subgroup of definite LE, three patients were 60 years and older. All three showed bilateral MRI hippocampal abnormalities. VGKC abs, rectal cancer, and small cell lung cancer were documented in one patient each. Finally, 11 patients were classified as MRI defined possible LE based on repeated MRIs with initial hippocampal swelling, normalization and atrophy, and absence of malignancy or antibodies (one exception with atypical anti-Hu antibodies). Seven (63%) showed bilateral MRI changes. This subgroup also presents episodic memory impairment and affective disturbances. Two patients, 60 years and older, were included, both with unilateral hippocampal involvement. This report delineates idiopathic and secondary HS as well as paraneoplastic and non-paraneoplastic LE as causative factors of late onset TLE emphasizing bilateral HS in the latter two subgroups, however, not infrequent with secondary HS. Memory impairment was more common with bilateral HS and in all cases frequent CPSs developed over a relatively short period of time. The few elderly subjects included in this series appear to follow these general premises.

A potential etiologic factor of TLE in the elderly is represented in generalized convulsive status epilepticus (GCSE) recognized to occur more often in the elderly with an estimated incidence of 86 per 100000 [12, 13]. A complication of GCSE is subsequent damage to the hippocampus [14, 15]. Mortality after GCSE is significant. Survivors would be at risk of subsequent partial seizures originating after hippocampal damage [16].

2. Clinical Presentation

The clinical features of various partial seizure types have been recently reported comparing patients 55 years of age and older (mean 65.2 ± 8.53) to a group between 18 and 45 years of age (mean 33.6 ± 6.75); each group encompassed 55 consecutive subjects [17]. Diagnosis was based on clinical and/or EEG findings. Partial seizures with loss of awareness and lack of prominent automatisms were classified as dialeptic seizures and in the older group accounted for 60% of all focal seizures and 52% of the younger patients. Partial seizures with prominent mouth and/or hand automatisms happened in 18% of the older patients while in 14% of the younger group. These differences were not statistically significant. The authors did not specifically report on the localization of the origin of these seizures. In this series, baseline characteristics showed some relevant differences. Not surprisingly, the older group had a later age at seizure onset (53.6 ± 20.23 yrs versus 23.4 ± 12.12 yrs), seizure-free period greater than 1 year (20% versus 8%), and cerebral vascular disease as a risk factor (20% versus 6%). The younger group reported more than one seizure per month (54% versus 28%) and a history of febrile seizures (20% versus 6%). For the older and younger age groups, risk factors included trauma 30% in each group, CNS infection in 6% and 4% and CNS tumor in 8% and

6%. Among the 28 patients that had no recollection of their seizures, 17 (61%) were from the older age group. In total, 9 subjects (18%), all of the older age group, reported subtle perception of transient confusion ($P = 0.002$). The total number of subjects with aura (defined as partial seizures without loss of awareness) was less common in the older age group (54% versus 76% $P = 0.03$). No significant differences could be demonstrated in the comparison of symptoms consisting of auras (psychic, autonomic, abdominal, visual, somatosensory or gustatory, auditory, olfactory, and vertigo) or generalized tonic clonic seizures (GTCSs) occurrence. Approximately one-third of patients in each group had a normal EEG, while 26% of the older group and 20% of the younger group had nonspecific finding. Focal epileptiform discharges were shown in 42% of the older and 23% of the younger subjects. Video-EEG monitoring was limited to a total of 9 patients yielding an additional 10% of older and 8% of younger patients with focal epileptiform and/or focal seizures.

In patients with epilepsy, memory dysfunction is not an unusual complaint. Transient epileptic amnesia is an emerging concept described in middle-aged and older people with an evident response to antiepileptic drug treatment (AED). Subjects with transient epileptic amnesia attacks may go unrecognized. A characteristic feature is prolonged periods of ante- and retrograde memory impairment with a mean duration between 30 and 60 minutes and subsequent amnesia that occurs upon awakening. The male population is more frequently affected. Transient amnesia may be the sole clinical manifestation in approximately one-third of patients. Attacks recur on the average 15 times per year. Hallucinations and automatisms have been frequently reported, while GTCSs are rare. Repetitive questioning is present in up to half of patients requiring differentiation from transient global amnesia. Interictal epileptiform abnormalities may be demonstrated in one-third of patients and ictal electrophysiological correlates have been recorded unilaterally or bilaterally in the temporal lobes. Brain imaging is usually clear, or lesions involve the temporal lobe [18, 19].

Diagnosis is further challenged when symptoms of dementia compound the clinical picture. Alzheimer dementia may present with partial or generalized seizures and nonlocalizing EEG abnormalities. An overlap with memory complaints is obviously expected in this group of cases [9].

The diagnosis of TLE in the elderly poses a challenge (see Table 1). The clinical features are subtle, and patients are unaware of seizures. Memory lapses, brief gaps in the flow of conversation, and blank stare and confusion may be the only clinical manifestations. Clinical features resemble TIA, delirium, congestive heart failure, cardiac arrhythmia, orthostatic hypotension, vasodepressor episodes, metabolic dysfunction with hypoglycemia, hyponatremia, sepsis, or drug toxicity [30].

3. Pharmacological Treatment

A small number of randomized controlled trials (RCTs) have been conducted in elderly patients testing the efficacy of

TABLE 1: Differential diagnosis of temporal lobe epilepsy in the elderly.

Temporal lobe epilepsy [19–21]	CPSs are common while auras and automatisms are not as common as in younger age groups. Brief gaps in conversation or periods of confusion may be only manifestation. Patients are frequently not aware of having seizures. Stroke is the most common cause in this age group. However, idiopathic cases have been reported.
Limbic encephalitis (LE) [8, 10]	Rapid progressive short-term memory deficit, with psychiatric symptoms consisting of irritability, depression, sleep disturbances, and hallucinations. CPSs more often than any other seizure types. Repetitive questioning may happen. CSF with increased proteins and lymphocytic pleocytosis. Temporal lobe abnormalities on EEG and MRI. Bilateral MTS not infrequent in nonparaneoplastic or paraneoplastic LE. May evolve to encephalomyelitis, decreased level of consciousness and refractory seizures. Antineuronal antibodies have been associated to underlying malignancy; anti-Hu (SCLC), anti-Ma2 (testis or other), CV2/CRMP5 (SCLC, thymoma), antiampiphysin (breast, SCLC), anti-Ri (carcinoid), anti-VGKC (thymoma, SCLC, other), and anti-NMDA (ovary).
Dementia [22, 23]	Gradual cognitive decline interfering with independence due to; memory, abnormalities; personality or behavioral changes; reasoning and judgment abnormalities; impaired language functions or visual spatial skills. Uncommon partial seizure or GTCs. Intermittent memory lapse that may be confused with CPSs. EEG diffuse slowing more often than focal abnormalities.
Transient ischemic attack [24, 25]	Sudden focal neurological dysfunction resulting from cerebral or retinal ischemia with clinical symptoms lasting less than 24 hours but frequently resolving within one hour. No evidence of cerebral infarction. Early CPSs are very rare after TIA. EEG abnormalities with temporary speech dysfunction and amnesia may represent focal inhibitory seizures.
Transient global amnesia [26]	Sudden transitory anterograde and retrograde memory loss or forming of new memories. Episodes last for less than 24 hours. Same question repeated over and over. A precipitating factor is common. Permanent residual memory gap after recovery. Awareness is spared. No aphasia or apraxia or focal neurological deficits. No seizures and normal EEG.
Delirium [27, 28]	Acute confusional state, altered awareness, fluctuating course, cognitive disturbance and difficulty maintaining attention. In elders hypoactive delirium is more common than hyperactive type. Disorientation, language impairment, and memory deficits. Sleep cycle disturbances or reversal. Intermittent fear, paranoia, anxiety, depression, irritability, anger or euphoria. Common in older and/or hospitalized patients. Frequent multiple predisposing factors. Diffuse slowing on EEG.
Epileptic transient amnesia [10, 29]	Recurrent episodes of memory deficits with long-term forgetting and remote autobiographical memory loss. Oral automatisms and olfactory hallucinations. More common in middle-to-old-aged men. Medial temporal lobe atrophy on MRI and epileptiform abnormalities present. Impressive response to antiepileptic treatment.

AEDs. Studies do not specifically address TLE but serve to evaluate efficacy and safety issues of AEDs in the setting of epilepsy in the elderly.

Recently, a group of 77 people with a mean age of 68 years and partial onset seizures were randomized in a pilot study to topiramate (TPM) 50 mg or 200 mg doses as add-on or monotherapy. Etiology of seizures was identified in 52% of patients. Cerebrovascular causes and head trauma accounted for 40% and 33%, respectively. Seizure freedom was similar with the lower and higher doses (52% and 58%, resp.) as well as seizure frequency (0.26/month and 0.33/month, resp.). More than 60% in both study groups complained of adverse side effects with somnolence, dizziness, and headache being the most common. Overall 18% of patients had to discontinue TPM due to adverse effects [31].

One randomized controlled trial (RCT) tested carbamazepine (CBZ) versus lamotrigine (LMT) in a total of 64 subjects (mean age: 67 years) with partial seizures (simple or complex) with or without secondary generalization presenting on average between 8 and 12 months after stroke. Stroke was classified as cortical in 64% of patients allocated to LMT and 66.7% of the CBZ group. Subcortical strokes were diagnosed in 36% and 33.3%, respectively, in these study

groups. Localization of the origin of the seizure was not reported. Subjects allocated to CBZ reported significantly more adverse effects than LMT leading to study withdrawal (31% versus 3%, $P = 0.02$). On completion of the first year, 72% of patients on LMT and 44% on CBZ were seizure-free ($P = 0.05$) [32].

A total of 590 elderly patients (mean age: 72 years) with newly diagnosed epilepsy with any seizure type were randomly allocated to gabapentin (GBP), LMT, or CBZ. In total, 42% of patients experienced CPSs. The etiology was similar among groups with an approximate one-third due to cerebral infarction. The localization of the origin of CPSs was not accounted for. A mild cognitive impairment or memory problems were present in 35% and 25% of patients, respectively. At 12 months, 46.7% had completed the study. LMT was significantly best retained than CBZ ($P < 0.0001$) or GBP ($P = 0.015$). At one year, seizure freedom was not significantly different between groups (LMT 51.4%, GBP 47.4%, and CBZ 64.3%). The time to first, second, fifth, and tenth seizure in the first year was also similar and no significant statistical differences were demonstrated between groups. Similarly, the overall seizure-free retention rate at one year was 24.9%. Severe adverse effects were reported

in 8.1%. Weight gain was greatest with GBP, while hyponatremia and any rash with CBZ. Of the seven patients requiring hospital admission due to hypersensitivity reactions, one was due to LMT and six to CBZ. Thirty-nine subjects died during the study. In the one case, CBZ was stopped after a hypersensitivity reaction two weeks before death [33].

A total of 150 elderly people (mean age: 77 years) with any seizure type resulting from idiopathic, symptomatic, or cryptogenic epilepsies were included and randomized in a 2:1 ratio to either LMT or CBZ [34]. Cerebral infarction was disclosed in 30% of the LMT group and 38% in the CBZ group. In 79% the daily LMT median dose was 100 mg (range: 75–300 mg), and in 82% the daily CBZ median dose was 400 mg (range: 200–800 mg). Adverse effects forced 41% of dropouts with CBZ and 18% with LMT. Seizure freedom during the 16 weeks of the study period was significantly different favoring LMT over CBZ (39% versus 21%, $P = 0.027$).

4. Surgery

Seizure outcome in 16 patients 50 years and older (mean 55.5 yrs, range 50–72) was compared to 184 younger patients (mean: 32.9 yrs, range: 16–49) undergoing anterior temporal lobectomy (ATL) [35]. All patients had pathologically confirmed unilateral HS and MRI lacked evidence of any other pathology. None of the variables showed to be predictors of the outcome. Following ATL older patients were less often seizure-free than the younger patients (56% versus 79%, $P = 0.041$). Postsurgical complications were more frequent in the older patients (25% versus 4.4%, $P = 0.009$).

Boling et al. reviewed 18 patients 50 years and older with 61% having MTS. The mean age at surgery was 54 years and followed for up to 64 months. Seizure freedom was reached by 61%, and 72% were able to reduce or stop their antiepileptic drugs. This older set of patients underwent multiple comparisons with subjects grouped in decades from ages 10 to 49 years. In the four resulting groups no significant differences could be demonstrated regarding proportions of seizure-free patients [36].

Sirven et al. reviewed a total of 30 patients aged 50 years and older (mean 54.2 ± 4.7) and compared them to 340 subjects younger than 50 years (mean 32.8 ± 7.7). These groups were followed for 4.0 ± 2.7 years and 5.0 ± 2.9 years, respectively. Seizure freedom was 52% in the older group and 75% in the younger group ($P = 0.008$). A discriminant function analysis disclosed that age at surgery and duration of epilepsy explained the largest fraction of variance. Younger age and shorter duration of epilepsy favored a seizure-free outcome. The authors consider that results could have been impacted by the fact that surgery in the older group was performed in their early 50's with only 5 patients 59 years and older. In this retrospective analysis, MR images were not available for all patients [37].

5. Conclusions

As population grows, an increment is expected in the number of subjects 65 years and older identified with TLE. Countries

with a longer life expectancy are going to be impacted to a greater extent. More often than not a coexisting significant medical condition may be revealed as well as acute or chronic brain involvement. Clinical diagnosis is elusive with subtle presenting features such as recurrent memory lapses or periods of confusion. Nevertheless, the typical signs of CPSs are likely. The undisputable advantage of video-EEG monitoring recognized in the investigation of people with epilepsy is clearly applicable to elderly populations [20–22, 38, 39]. AEDs continue to be the foundation of the medical pharmacological treatment.

References

- [1] W. A. Hauser, J. F. Annegers, and L. T. Kurland, "Incidence of epilepsy and unprovoked seizures in Rochester, Minnesota: 1935–1984," *Epilepsia*, vol. 34, no. 3, pp. 453–468, 1993.
- [2] R. Sidenvall, L. Forsgren, H. K. Blomquist, and J. Heijbel, "A community-based prospective incidence study of epileptic seizures in children," *Acta Paediatrica*, vol. 82, no. 1, pp. 60–65, 1993.
- [3] L. Forsgren, G. Bucht, S. Eriksson, and L. Bergmark, "Incidence and clinical characterization of unprovoked seizures in adults: a prospective population-based study," *Epilepsia*, vol. 37, no. 3, pp. 224–229, 1996.
- [4] E. Olafsson, P. Ludvigsson, G. Gudmundsson, D. Hesdorffer, O. Kjartansson, and W. A. Hauser, "Incidence of unprovoked seizures and epilepsy in Iceland and assessment of the epilepsy syndrome classification: a prospective study," *The Lancet Neurology*, vol. 4, no. 10, pp. 627–634, 2005.
- [5] W. A. Hauser, J. F. Annegers, and L. T. Kurland, "Incidence of epilepsy and unprovoked seizures in Rochester, Minnesota: 1935–1984," *Epilepsia*, vol. 33, supplement 4, pp. S6–S14, 1992.
- [6] W. A. Hauser, J. F. Annegers, and L. T. Kurland, "Prevalence of epilepsy in Rochester, Minnesota: 1940–1980," *Epilepsia*, vol. 32, no. 4, pp. 429–445, 1991.
- [7] D. C. Hesdorffer, W. A. Hauser, J. F. Annegers, E. Kokmen, and W. A. Rocca, "Dementia and adult-onset unprovoked seizures," *Neurology*, vol. 46, no. 3, pp. 727–730, 1996.
- [8] G. Regesta and P. Tanganelli, "Temporal lobe epilepsy of adult age of possible idiopathic nature," *Seizure*, vol. 11, no. 2, pp. 131–135, 2002.
- [9] D. A. Lozsadi, D. W. Chadwick, and A. J. Larner, "Late-onset temporal lobe epilepsy with unilateral mesial temporal sclerosis and cognitive decline: a diagnostic dilemma," *Seizure*, vol. 17, no. 5, pp. 473–476, 2008.
- [10] C. A. O'Donovan, M. E. Lancman, and H. O. Lüders, "New-onset mesial temporal lobe epilepsy in a 90-year-old: clinical and EEG features," *Epilepsy and Behavior*, vol. 5, no. 6, pp. 1021–1023, 2004.
- [11] C. G. Bien, H. Urbach, J. Schramm et al., "Limbic encephalitis as a precipitating event in adult-onset temporal lobe epilepsy," *Neurology*, vol. 69, no. 12, pp. 1236–1244, 2007.
- [12] R. F. M. Chin, B. G. R. Neville, and R. C. Scott, "A systematic review of the epidemiology of status epilepticus," *European Journal of Neurology*, vol. 11, no. 12, pp. 800–810, 2004.
- [13] R. J. DeLorenzo, W. A. Hauser, A. R. Towne et al., "A prospective, population-based epidemiologic study of status epilepticus in Richmond, Virginia," *Neurology*, vol. 46, no. 4, pp. 1029–1035, 1996.

- [14] R. L. Macdonald and J. Kapur, "Acute cellular alterations in the hippocampus after status epilepticus," *Epilepsia*, vol. 40, supplement 1, pp. S9–S20, 1999.
- [15] U. C. Wiesmann, F. G. Woermann, L. Lemieux et al., "Development of hippocampal atrophy: a serial magnetic resonance imaging study in a patient who developed epilepsy after generalized status epilepticus," *Epilepsia*, vol. 38, no. 11, pp. 1238–1241, 1997.
- [16] D. C. Hesdorffer, G. Logroscino, G. Cascino, J. F. Annegers, and W. A. Hauser, "Risk of unprovoked seizure after acute symptomatic seizure: effect of status epilepticus," *Annals of Neurology*, vol. 44, no. 6, pp. 908–912, 1998.
- [17] D. C. Silveira, L. Jehi, J. Chapin et al., "Seizure semiology and aging," *Epilepsy and Behavior*, vol. 20, no. 2, pp. 375–377, 2011.
- [18] C. R. Butler and A. Z. Zeman, "Recent insights into the impairment of memory in epilepsy: transient epileptic amnesia, accelerated long-term forgetting and remote memory impairment," *Brain*, vol. 131, no. 9, pp. 2243–2263, 2008.
- [19] A. Z. J. Zeman, S. J. Boniface, and J. R. Hodges, "Transient epileptic amnesia: a description of the clinical and neuropsychological features in 10 cases and a review of the literature," *Journal of Neurology Neurosurgery and Psychiatry*, vol. 64, no. 4, pp. 435–443, 1998.
- [20] T. Keränen, S. Rainesalo, and J. Peltola, "The usefulness of video-EEG monitoring in elderly patients with seizure disorders," *Seizure*, vol. 11, no. 4, pp. 269–272, 2002.
- [21] A. E. McBride, T. T. Shih, and L. J. Hirsch, "Video-EEG monitoring in the elderly: a review of 94 patients," *Epilepsia*, vol. 43, no. 2, pp. 165–169, 2002.
- [22] M. Kawai, R. A. Hrachovy, P. J. Franklin, and P. J. Foreman, "Video-EEG monitoring in a geriatric veteran population," *Journal of Clinical Neurophysiology*, vol. 24, no. 6, pp. 429–432, 2007.
- [23] D. C. Hesdorffer, W. A. Hauser, J. F. Annegers, E. Kokmen, and W. A. Rocca, "Dementia and adult-onset unprovoked seizures," *Neurology*, vol. 46, no. 3, pp. 727–730, 1996.
- [24] N. Scarneas, L. S. Honig, H. Choi et al., "Seizures in Alzheimer disease: who, when, and how common?" *Archives of Neurology*, vol. 66, no. 8, pp. 992–997, 2009.
- [25] J. de Reuck and G. van Maele, "Transient ischemic attacks and inhibitory seizures in elderly patients," *European Neurology*, vol. 62, no. 6, pp. 344–348, 2009.
- [26] M. Giroud, P. Gras, H. Fayolle, N. Andre, P. Soichot, and R. Dumas, "Early seizures after acute stroke: a study of 1,640 cases," *Epilepsia*, vol. 35, no. 5, pp. 959–964, 1994.
- [27] J. R. Hodges and C. P. Warlow, "Syndromes of transient amnesia: towards a classification. A study of 153 cases," *Journal of Neurology Neurosurgery and Psychiatry*, vol. 53, no. 10, pp. 834–843, 1990.
- [28] S. Grover, B. N. Subodh, A. Avasthi et al., "Prevalence and clinical profile of delirium: a study from a tertiary-care hospital in north India. PGI Chandigarh," *General Hospital Psychiatry*, vol. 31, no. 1, pp. 25–29, 2009.
- [29] S. K. Inouye, "Delirium in older persons," *New England Journal of Medicine*, vol. 354, no. 11, pp. 1157–1165, 2006.
- [30] N. S. Collins, R. A. Shapiro, and R. E. Ramsay, "Elders with epilepsy," *Medical Clinics of North America*, vol. 90, no. 5, pp. 945–966, 2006.
- [31] R. E. Ramsay, B. Uthman, F. M. Pryor et al., "Topiramate in older patients with partial-onset seizures: a pilot double-blind, dose-comparison study," *Epilepsia*, vol. 49, no. 7, pp. 1180–1185, 2008.
- [32] R. Gilad, M. Sadeh, A. Rapoport, R. Dabby, M. Boaz, and Y. Lampl, "Monotherapy of lamotrigine versus carbamazepine in patients with poststroke seizure," *Clinical Neuropharmacology*, vol. 30, no. 4, pp. 189–195, 2007.
- [33] A. J. Rowan, R. E. Ramsay, J. F. Collins et al., "New onset geriatric epilepsy: a randomized study of gabapentin, lamotrigine, and carbamazepine," *Neurology*, vol. 64, no. 11, pp. 1868–1873, 2005.
- [34] M. J. Brodie, P. W. Overstall, and L. Giorgi, "Multicentre, double-blind, randomised comparison between lamotrigine and carbamazepine in elderly patients with newly diagnosed epilepsy. The UK lamotrigine elderly study group," *Epilepsy Research*, vol. 37, no. 1, pp. 81–87, 1999.
- [35] T. Srikiyvilakul, S. Lerdlum, S. Tepmongkol, S. Shuangshoti, and C. Lochareonkul, "Outcome of temporal lobectomy for hippocampal sclerosis in older patients," *Seizure*, vol. 20, no. 4, pp. 276–279, 2011.
- [36] W. Boling, F. Andermann, D. Reutens, F. Dubeau, L. Caporicci, and A. Olivier, "Surgery for temporal lobe epilepsy in older patients," *Journal of Neurosurgery*, vol. 95, no. 2, pp. 242–248, 2001.
- [37] J. I. Sirven, B. L. Malamut, M. J. O'Connor, and M. R. Sperling, "Temporal lobectomy outcome in older versus younger adults," *Neurology*, vol. 54, no. 11, pp. 2166–2170, 2000.
- [38] I. Drury, L. M. Selwa, L. A. Schuh et al., "Value of inpatient diagnostic CCTV-EEG monitoring in the elderly," *Epilepsia*, vol. 40, no. 8, pp. 1100–1102, 1999.
- [39] A. Abubakr and I. Wambacq, "Seizures in the elderly: video/EEG monitoring analysis," *Epilepsy and Behavior*, vol. 7, no. 3, pp. 447–450, 2005.