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Electroclinical features of idiopathic generalized epilepsies in the elderly: A geriatric hospital-based study

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ABSTRACT

Purpose: Idiopathic generalized epilepsies (IGE) are age-related epileptic syndromes mainly described in children and adolescence. Our aim is to describe their electroclinical features in the elderly.
Methods: Patients aged 70 years or more were prospectively selected in a geriatric EEG laboratory on the basis of rhythmic generalized spikes and waves discharges. Their clinical data were then examined to ascertain the syndromic diagnosis.
Results: Among 1181 geriatric patients referred for EEG over a 30-month period, IGE were identified in 10 cases. Eight patients began seizures in childhood or adulthood (3 childhood absence epilepsies, 2 juvenile/adult myoclonic epilepsies and 3 epilepsies with-generalized-tonic–clonic-seizures alone (EGTCS)) and 2 very late in life with EGTCS. The early-onset IGE cases had usually experienced a quiescent long period in adulthood before relapsing late in life. This relapse, mostly severe, consisted of absence status, myoclonic status or repeated generalized tonic clonic seizures and was often not-situation related. Absence status and myoclonic status were stopped by Clonazepam. The two late-onset IGE cases had familial history of epilepsy. Inappropriate antiepileptic drugs (AED) previously given in four patients with two worsenings were corrected.
Conclusions: In this study, the non-negligible number of elderly cases observed over a short period of time suggests that IGE are frequent in the elderly but underestimated until recently. IGE may be lifelong with late severe exacerbations. A few very late-onset IGE cases exist. EEG remains useful in contributing to diagnose IGE and AED adjustment continues to be beneficial at extreme age.

1. Introduction

Idiopathic generalized epilepsies (IGE) are presumably genetic in nature and characterized by initially generalized seizures and generalized discharges on EEG. They affect approximately 30% of all patients with epilepsy[1,2] and have been typically described in childhood or adolescence[3] with an excellent prognosis with time.[4,5] Gastaut[4] found less than 1% of patients with IGE after the age of 65 and Loiseau et al.[6] failed to identify late-onset IGE cases and found only five cases in the literature. Recent studies indicate however a less favourable prognosis of absence epilepsies[7–9] and of epilepsies with generalized-tonic–clonic seizures[10] or a much more prolonged evolution of juvenile myoclonic epilepsies (JME).[11–13] IGE might be thus prolonged into adulthood and, presumably, lifelong,[9,12] might exacerbate at an advanced age[14–18] or might even appear after the age of 60.[14,19–28] These previous reports however are few, scattered and concern relatively young older patients. Long-term follow-up studies of IGE did not go beyond the age of 60[12] and cannot predict the frequency and the evolution of IGE at an extreme age. So it seems interesting to report IGE cases in older patients and their clinical features. Here we describe the characteristics of 10 cases, collected over a 30-month period, to illustrate their age of onset, their evolution with time, their seizure types, their diagnostic and therapeutic history up to a very advanced age (mean age of 79.4 years).

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Table 1
Summary of patients' electrical and clinical characteristics.

Pts	Sex/age (yrs)	Familial atcd of epilepsy	Age at onset of seizures	Seizures types and seizures frequency	EEG data	Subsyndromes	Treatment and outcome
1	F/85	No	Adolescence	GTCSs in adolescence treated by PB. No recurrence during decades. Relapse of repeated GTCSs followed by one AS from age 80 under PB.	Ictal EEG: continuous 2.5–3 Hz generalized SW and PSW discharges during absence status which were suppressed by intravenous Clonazepam. Interictal epileptic discharges presented on 24 hours EEG without activation during sleep.	EGTCS	Good control with VPA. Follow-up of 7 months.
2	F/77	No	Adolescence	Myoclonic jerks in adolescence. One GTCS at the age of 20. No recurrence during decades without treatment. Severe relapse with myoclonic status, at age 76 under no AED.	Ictal EEG: sub continuous generalized PSW discharges accompanied with myoclonias which were suppressed by intravenous Clonazepam. Interictal epileptic discharges persisted, accompanied by myoclonic jerks on IPS at 15 Hz on standard EEG.	JME	Good control with VPA but side effects. Worsening of seizures under CBZ, Myoclonus exacerbated by LTG, Complete control under LEV at age 77. Follow-up of 1 year.
3	F/81	Yes (sister)	8 yrs	GTCS at age 8. Rare recurrences under PB and no seizure during the last 20 yrs. Relapse of repeated GTCSs at age 80 under no AED.	3 Hz generalized SW discharges on standard EEG. SW, PSW were activated on drowsiness and on awakenings during 24 h EEG.	EGTCS	Good control with VPA. Follow-up 15 months.
4	F/75	Yes (little daughter)	40 yrs	GTCSs, myoclonic jerks at age 40. No recurrence without treatment. Relapse of repeated GTCSs and myoclonic jerks at age 75 under no AED.	4 Hz interictal generalized SW discharges on standard EEG.	JME	Persistence of myoclonias under PHT (prescribed before admission) which was replaced by LEV. Lost to follow-up.
5	F/74	No	Childhood	Absence seizures in childhood, then GTCSs. Persistence of absence seizures with 2 AS, GTCSs under GBP and PHT at age 71.	Ictal EEG: sub continuous, irregular 3 Hz-generalized SW during absence status which were stopped by Clonazepam per os. Interictal epileptic discharges persisted on standard EEG.	CAE	Good control with VPA but side effects. Rare seizures under LTG and LEV. Follow up of 2 yrs.
6	M/81	No	35 yrs	GTGSs at age 35. No recurrence without treatment. Relapse of seizures at age 81 under no AED.	Interictal 3.5 Hz generalized SW discharges activated by drowsiness. PSW presented during sleep on 24 h EEG.	EGTCS	Good control with LEV. Follow-up 12 months.
7	F/97	No	Childhood	Absence seizures, GTCSs at age 27 treated with PB. Absences and several GTCSs per yrs under PB. Last GTCS at age 93. No seizure during the last 4 yrs.	Interictal generalized SW, PSW discharges on standard EEG, activated by drowsiness.	CAE	Refused to change her PB. Lost to follow up.
8	F/70	No	9 yrs	Absence seizures, GTCSs. 2 GTCSs per year, daily absences seizures under CBZ, PHT and PB. Repeated GTCSs at age 69 without modification of treatment.	Ictal EEG: brief absence seizure with stopping of counting accompanied by a 3 Hz generalized SW discharge during hyperventilation. Interictal epileptic discharges were activated by drowsiness, PS discharges presented during sleep on 24 h EEG.	CAE	CBZ, PHT were replaced by LEV, Lost to follow-up.