Original article

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De novo late-onset absence status epilepticus or late-onset idiopathic generalized epilepsy? A case report and systematic review of the literature

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ABSTRACT – *Aim*. Idiopathic (genetic) generalized epilepsies (IGEs) are age-related epileptic syndromes with typical age onset in childhood or adolescence. We report a patient with *de novo* late-onset absence status epilepticus (ASE) occurring at the age of 64 years, with clinical and EEG features suggestive of late-onset IGE. We also discuss the relationship between *de novo* late-onset ASE and late-onset IGE, and provide a comprehensive and critical review of the available literature on late-onset (*i.e.* onset ≥60 years) IGE.

Methods. MEDLINE (1966-2016 [23th April]) was systematically searched in order to identify reports of patients with late-onset IGE. Grey literature was also comprehensively searched.

Results. We identified nine patients with electroclinical features suggestive of late-onset IGE. Median age at seizure onset was 71 years (range: 60-80), with a female prevalence (67%). A family history of epilepsy was reported in 67% of cases. All patients had generalized tonic-clonic seizures, and 44% also had myoclonic seizures. Treatment and outcome were reported for six patients; all of whom reached seizure freedom under monotherapy with valproic acid (83%) or lamotrigine (17%) (range of follow-up: 3 to 24 months).

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Conclusion. Late-onset IGE are entities with unknown prevalence and incidence, and should be differentiated on the basis of late-onset reactivation of previous IGE. Late-onset IGEs are probably unrecognized or misdiagnosed, based on a common misconception that all elderly individuals with first-ever seizures have focal symptomatic epilepsy. Late-onset IGE should be actively investigated by accurate history taking aimed at identifying seizures, which may have been unnoticed, and familial antecedents of epilepsy. In elderly patients presenting with *de novo* late-onset ASE, a diagnosis of late-onset IGE should be considered in the differential diagnosis, particularly in atypical cases (*e.g.* absence of triggering factors, coexistence of generalized tonic-clonic or myoclonic seizures, and interictal generalized epileptiform discharges).

Key words: idiopathic generalized epilepsies, genetic generalized epilepsies, absence status epilepticus, late-onset, family history, generalized tonic-clonic seizure, myoclonic seizure

Idiopathic (or genetic) generalized epilepsies (IGEs) are age-related epileptic syndromes with typical age at onset in childhood or adolescence (Caraballo and Dalla Bernardina, 2013). Based on the predominant seizure type, the International League Against Epilepsy (ILAE) has recognized three IGE syndromes with typical onset in adolescents or young adults: juvenile absence epilepsy (JAE), juvenile myoclonic epilepsy (JME), and epilepsy with generalized tonic-clonic seizures (GTCS) only (Commission on Classification and Terminology of the International League Against Epilepsy, 1989; Engel, 2001; Scheffer *et al*., 2017).

We report a patient with atypical *de novo* late-onset absence status epilepticus (ASE) manifesting at the age of 64 years, with clinical and EEG features suggestive of late-onset IGE (*i.e.* IGE starting after the age of 60 without any previous history or signs of epileptic seizures). We also discuss the clinical difficulties encountered in daily practice in the differential diagnosis between *de novo* late-onset ASE and late-onset IGE, and provide a comprehensive and critical review of the available literature on late-onset IGE.

Materials and methods

Systematic search of the literature

We carried out a comprehensive and systematic search of the available literature on late-onset IGE. MEDLINE (1966-2016 [23th April]) and databases of grey literature, Opengrey (available at: http://opengrey.eu/) and Grey Literature Report (available at: http://www.greylit.org/library/search), were systematically searched using the following search strategy: ("idiopathic generalized epilepsy" AND [adult* OR elderly]). All resulting titles and abstracts were evaluated, and any relevant article was considered. References quoted in the included articles were hand-searched to identify further studies. No language restrictions were adopted.

We included only articles reporting information on patients with unprovoked generalized seizures (GTCS, myoclonic, or absence seizures) with an onset at or beyond age 60, and with generalized spike-wave discharges (SWD) on EEG. For each patient, the following data were extracted: gender, age at onset of seizures, seizure type, EEG and neuroimaging data, familial antecedents of epilepsy, final diagnosis, and treatment and outcome (including length of follow-up).

Results

Case description

A 64-year-old, previously healthy woman was admitted to our department due to a single GTCS. Her previous medical history was unremarkable, and she was not taking any drugs. There was no history of alcohol abuse or benzodiazepine intake, and no family history of epilepsy. The woman did not report any previous paroxysmal events. On the day of admission, very early in the morning (3.00 a.m.), she had been awoken by a phone call from a relative, who informed here about the sudden death of her elderly mother. Five hours later, after having her breakfast, she experienced a GTCS, which was witnessed by her husband. Two hours after recovering consciousness, she was still mildly confused, slow, and drowsy. Because of the persistence of these symptoms, she was referred to our hospital. Routine laboratory testing yielded normal results, and CT of the head was unremarkable. The EEG showed generalized 3-Hz SWD with prevalent amplitude over frontal regions, sometimes mixed with polyspike-wave discharges (PSWD) (*figure 1A*). During the recording, she was confused, slow, and unable to follow a simple conversation because of slow mentation and lack of attention. Performing simple arithmetic operations

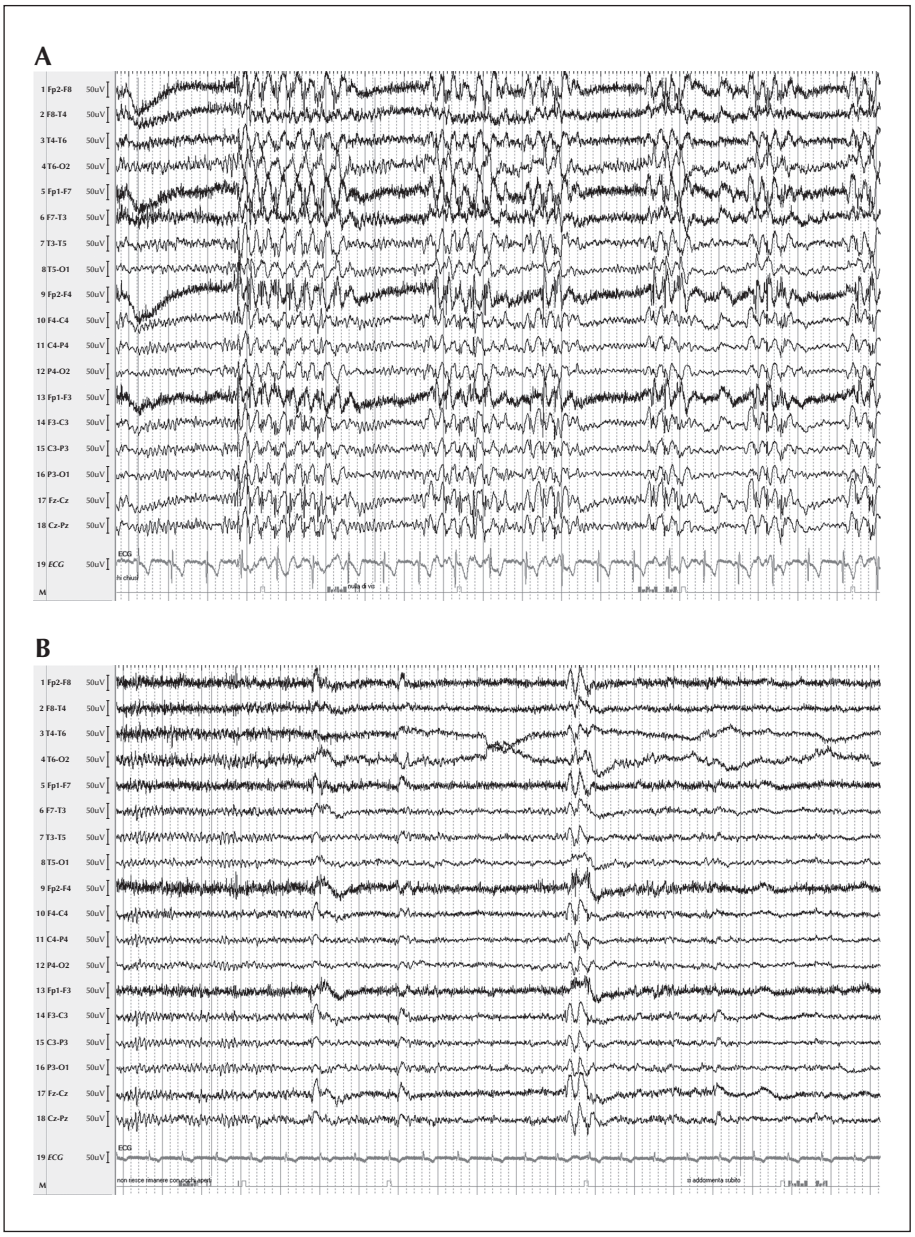

Figure 1. (A) Ictal EEG recording showing 3-Hz, somewhat irregular, bilaterally synchronous (generalized) spike-wave discharges, sometimes intermixed with polyspike-wave discharges; epileptiform abnormalities show a prevalence of amplitude over frontal regions, with normal background activity in between. (B) Routine EEG recorded the day after the episode of absence status, showing normal background activity with appearance of interictal bursts of spike-wave discharges activated by drowsiness.
Band pass filter: 0.53-70 Hz; no 50-Hz notch filter.

was impossible. Neurological examination was otherwise normal and no myoclonic jerks were observed. A diagnosis of *de novo* late-onset absence status without triggering factors was made. After administering 4 mg lorazepam intravenously, both the EEG and the patient's condition completely normalized. The EEG performed the day after showed normal background activity, with rare interictal bursts of generalized SWD, especially during drowsiness (*figure 1B*). No photosensitivity was found on intermittent photic stimulation. Two subsequent EEG recordings, performed after two weeks and 22 months, still showed rare interictal bursts of generalized SWD, especially during hyperventilation. Neurological and cognitive examination were completely normal, and the patient had only partial memory of what happened the day before. Brain MRI was unremarkable; more specifically, it did not show any sign of cortical atrophy or cerebrovascular lesions. The patient was carefully questioned about paroxysmal episodes of lapses of consciousness, clumsy

movements, or jerking in the past (including during childhood and in her youth), which she decisively denied. To reduce recall bias (*i.e.* the possibility that the patient might have forgotten absence seizures or mild myoclonic seizures during her childhood or youth), her sister and husband (who had been known to her for at least 47 years, having married her when she was 17) were also questioned. Both of them reported no previous history or signs of paroxysmal events. Treatment with valproic acid (VPA) (800 mg/day) was started and no further seizures occurred (length of follow-up: 22 months).

Systematic search of the literature

The search strategy described above yielded 467 results (MEDLINE: 461; Opengrey: 0; Grey Literature Report: 0; and six additional articles identified in reference lists of articles). After reading the titles and abstracts, eight articles were considered for possible inclusion. Having read the full-texts, one study (Hiyoshi and Yagi, 2000) was excluded on the basis that only one patient within the age range of 60-69 years at onset of IGE was included, without providing other electroclinical information. Another study (Yenjun *et al*., 2003) was excluded for similar reasons; three patients were reported to have had onset of seizures after 60 years, but no further details were provided.

Hence, six articles published between 1970 and 2011, reporting data on nine patients, were eventually included.

Median age at seizure onset was 71 years (range: 60-80), with a female prevalence (67%). A familiarity of epilepsy was reported in 67% of cases. All patients had GTCS, and four out of nine (44%) also had myoclonic seizures. One patient was reported to have "confusional status with myoclonia", followed by GTCS with bilateral PSWD on EEG (Gemignani *et al*., 1977). Data on neuroimaging for the three older reports was not available (Courjon *et al*., 1970; Vercelletto and Delobel, 1970; Gemignani *et al*., 1977). In more recent reports, MRI and/or CT yielded normal results (67%) or showed vascular lesions (33%), in one case associated with generalized brain atrophy. Treatment and outcome were reported for six patients, all of whom reached seizure freedom under monotherapy with VPA (83%) or lamotrigine (LTG) (17%) (range of follow-up: 3 to 24 months). The detailed electroclinical characteristics of patients included are presented in *table 1*.

Discussion

Our patient represents a peculiar case of *de novo* late-onset ASE and is atypical due to a lack of triggering factors, as well as neurophysiological and clinical features. Brain MRI showed neither cortical atrophy nor vascular encephalopathy, indicative of an identifiable cause of her seizures. Furthermore, the patient had no previous history of alcohol abuse or consumption of benzodiazepines, or other seizure triggering factors such as metabolic imbalance, systemic infections, or dehydration, which have been reported to typically occur in association with *de novo* late-onset ASE (Thomas *et al*., 1993; Thomas and Andermann, 1994; Bilo *et al*., 2014).

Late-onset ASE occurs as a reactivation of IGE in adults and elderly individuals, in whom epilepsy has remitted after puberty or juvenile age (Andermann and Robb, 1972; Thomas *et al*., 1992; Fernández-Torre and Díaz-Castroverde, 2004; Trinka, 2005; Zambrelli *et al*., 2006; Bauer *et al*., 2007; Fernández-Torre and Rebollo, 2009; Pro *et al*., 2011). These patients have a previous IGE resolved after puberty or juvenile age, which reactivates in older age after many years of seizure freedom without any AED treatment and in the absence of any identifiable triggering factors.

Late-onset ASE can also occur *de novo*, *i.e.* without any history of prior seizures. These cases have been more frequently described in women, are typically provoked by benzodiazepine withdrawal, alcohol abuse, or psychotropic drug initiation (Thomas *et al*., 1992; Thomas *et al*., 1993; Thomas and Andermann, 1994; Fernández-Torre and Díaz-Castroverde, 2004; Bilo *et al*., 2014), and are associated with EEG recordings with interictal irregular background activity (Pro *et al*., 2011).

Our patient would appear to add to the list of *de novo* late-onset ASE in the literature, however, the patient had no triggering factors, and her EEGs showed a persistence of interictal generalized epileptiform discharges on a normal background activity several weeks and months after the ASE. Furthermore, the lack of a previous history of IGE argues against a reactivation of IGE in older age, hence ruling out a diagnosis of late-onset ASE associated with previous IGE. Overall, the present case seems to be indicative of an IGE, manifesting for the first time in life, in a patient with a GTCS followed by atypical *de novo* late-onset ASE. Such diagnosis is further supported by: certain seizure types (GTCS and ASE); a specific EEG pattern (generalized 3-Hz SWD with interictal generalized epileptiform discharges and normal background activity); normal neurological examination, cognition, and neuroimaging; and a lack of previous history or signs of epileptic seizures. Sleep deprivation and emotional stress were probably responsible for lowering the seizure threshold, as typically described in IGE with typical age at onset (Nordli, 2005).

Based on a comprehensive search of the literature, we identified only nine patients with electroclinical features suggestive of late-onset IGE. However, this is

Table 1. Electroclinical characteristics of patients with late-onset IGE reported in the literature.

Author	Patient	Gender	Family history of epilepsy	Age at onset of seizures	Seizure type	EEG	Neuroimaging	Final diagnosis	Treatment and outcome
Courjon *et al.*, 1970*	1	F	Son (GTCS since 25 years, similar EEG)	64	2 GTCS	Bilateral SWD	NR	NR	NR
Vercelletto and Delobel, 1970*	2	F	Mother (GTCS at 36 years), brother (myoclonic and GTCS in puberty)	60	GTCS	3-Hz bilateral SWD	NR	NR	NR
Gemignani *et al.*, 1977*	3	F	No	71	Confusional status with myoclonia followed by GTCS; GTCS	Bilateral PSWD	NR	NR	NR
Marini *et al.*, 2003	4	F	No	75	Myoclonic seizures, one GTCS	Generalized 3-5-Hz SWD and PSWD	Normal (MRI/CT not specified)	Adult-onset myoclonic epilepsy	Seizure-free with VPA (follow-up NR)
	5	M	Twin sister and distant cousin (epilepsy not further specified)	69	GTCS	Generalized 4-5-Hz generalized SWD and PSWD	Normal (MRI/CT not specified)	Adult-onset tonic-clonic epilepsy	Seizure-free with VPA (follow-up: 12 months)

Table 1. Electroclinical characteristics of patients with late-onset IGE reported in the literature. (*Continued*)

Author	Patient	Gender	Family history of epilepsy	Age at onset of seizures	Seizure type	EEG	Neuroimaging	Final diagnosis	Treatment and outcome
Tóth *et al.*, 2007	6	F	No	73	Myoclonic seizures, GTCS	Generalized SWD	MRI: mild leukoaraiosis	JME	Seizure-free with VPA (follow-up: 6 months)
	7	M	Sister and sister's son (epilepsy not further specified; onset during adolescence)	72	2 GTCS; myoclonic seizures after carbamazepine	Generalized SWD	MRI: generalized brain atrophy, leukoaraiosis, multiple lacunar infarcts	JME	Seizure-free with VPA (follow-up: 3 months)
Michel *et al.*, 2011	8	F	Brother, daughter	61	At least 2 GTCS	4-Hz generalized SWD during drowsiness; PSD during sleep	CT: normal	Epilepsy with generalized tonic-clonic seizures only	Seizure-free with VPA (follow-up: 2 years)
	9	M	Father	80	2 GTCS	3-3.5-Hz generalized SWD during sleep; PSD during sleep	MRI, CT: normal	Epilepsy with generalized tonic-clonic seizures only	Seizure-free with LTG (follow-up: 1 year)

*Reported by Loiseau *et al.* (1998).
CT: computed tomography; GTCS: generalized tonic-clonic seizures; JME: juvenile myoclonic epilepsy; LTG: lamotrigine; MRI: magnetic resonance imaging; NR: not reported; PSD: polyspike discharges; PSWD: polyspike-wave discharges; SWD: spike-wave discharges; VPA: valproic acid.

in contrast with the view of Loiseau *et al*. (1998) who stated that "convulsive idiopathic generalized epilepsy with an onset over 60 years of age is comparable to the Loch Ness monster: aside from a handful of authors, no one has seen such an entity".

Identifying patients who truly have late-onset IGE, as opposed to those with onset during childhood or adolescence experiencing seizure recurrence after several years of seizure freedom (late-onset "reactivated" IGE) (Trinka, 2005; Bauer *et al*., 2007), may be extremely challenging. When asked, patients may have completely forgotten that they had had absences or mild myoclonic seizure during their childhood or adolescence (recall bias) (Bauer *et al*., 2007). Furthermore, particularly among older patients, absences or myoclonic jerks may be unrecognized or misdiagnosed (Bauer *et al*., 2007). Accurate history taking is therefore essential to diagnose "true" late-onset IGE and exclude late exacerbation of a previous IGE. Conversely, the risk of underdiagnosing late-onset IGE is probably high.

Based on our comprehensive analysis of the literature, we found that all patients with elderly onset of IGE had GTCS, associated in 44% of cases with myoclonic seizures and in one case (Gemignani *et al*., 1977) with absence status (a patient with confusional status and myoclonia).

The relatively high frequency of myoclonic seizures reported in these patients highlights the importance of a detailed interview aimed at identifying previous occurrence of myoclonic jerks or lapses of consciousness. Unfortunately, these signs of epileptic seizures may go completely unnoticed, as elderly people with late-onset seizures are rarely referred to epilepsy specialists (Tóth *et al*., 2007).

It is also important to ask about family history of epilepsy, as this may provide additional clues to the diagnosis of IGE. In the literature, a familial antecedent of epilepsy was reported in slightly more than half of cases (67%). Therefore, in contrast to IGE with a typical age at onset (Nordli, 2005), epilepsy in general is relatively well recalled in the elderly population. This is somewhat surprising, as elderly patients may be expected not to report accurately on their family history of epilepsy, because relatives may be dead or information about them may be incomplete (Tóth *et al*., 2007). In other words, one might expect recall bias leading to under-reporting of a positive family history of epilepsy more frequently among the elderly. However, the high rate of familial antecedents of epilepsy among these subjects is probably legitimate and not the consequence of recall or detection bias.

Apart from difficulties related to obtaining information about the occurrence of absences or myoclonic seizures, or familial antecedents of epilepsy, most first-ever seizures occurring in elderly patients may be automatically attributed to an underlying vascular encephalopathy. This simplistic conclusion may be further strengthened by the presence of vascular risk factors or asymptomatic vascular lesions on MRI, which may be observed relatively commonly in this age group and which may incorrectly shift the diagnosis towards symptomatic epilepsy (Tóth *et al*., 2007).

A correct diagnosis of late-onset IGE is further complicated by the suboptimal diagnostic sensitivity of routine EEG, which may not show the typical IGE-related epileptiform discharges. To increase the diagnostic sensitivity, sleep-deprived and sleep EEG recordings may be performed, however, these are usually avoided in elderly patients (Tóth *et al*., 2007).

Reaching an accurate syndromic diagnosis of IGE has relevant therapeutic consequences, as a misdiagnosis may lead to treatment with narrow-spectrum antiepileptic drugs (such as carbamazepine or phenytoin), which may be ineffective or result in persistence or even worsening of seizures (Hiyoshi and Yagi, 2000).

Our patient lacked a previous history or signs of epileptic seizures, thus a late relapse of IGE, remitting after puberty or juvenile age, seems extremely unlikely. A reactivation of IGE in elderly people, mostly women manifesting with absence status, has been reported (Andermann and Robb, 1972; Thomas *et al*., 1992; Fernández-Torre and Díaz-Castroverde, 2004; Trinka, 2005; Zambrelli *et al*., 2006; Bauer *et al*., 2007; Fernández-Torre and Rebollo, 2009; Pro *et al*., 2011). These patients experienced IGE which had resolved after puberty or juvenile age and reactivated in older age after many years of seizure freedom without any AED treatment and in the absence of any identifiable triggering factors. The cause of such reactivation of IGE in the older age group is an enigma (Trinka, 2005).

An explanation for this phenomenon, as well as for late-onset IGE in elderly people, is the increase in epileptogenic cerebrovascular lesions, which may alter cortical excitability and lower seizure threshold in genetically predisposed patients (Marosi *et al*., 1994; Tóth *et al*., 2007). Age-associated increased ventricular cerebrospinal fluid and white matter hyperintensities, which are commonly observed on MRI in non-demented elderly individuals, are associated with increased cortical excitability (Silbert *et al*., 2006) and might also be involved in the (re)activation of IGE. These factors might have played a role in the late manifestation of epilepsy in our patient.

Far from being "comparable to the Loch Ness monster", late-onset IGE in elderly people, which is clinically indistinguishable from IGE with typical age at onset, are real entities. Although no data on prevalence or incidence is currently available, it is reasonable to hypothesize that most late-onset IGEs are unrecognized or misdiagnosed, based on a common misconception that all elderly individuals with

first-ever seizures have a focal symptomatic epilepsy, usually due to cerebrovascular lesions or age-related cortical atrophy. The possibility of a late-onset IGE or reactivation of a previous IGE should be taken into consideration even among elderly subjects, and should be actively investigated by accurate and detailed history taking in order to identify seizures which may have been unnoticed (absence seizures or mild myoclonic jerks) and a positive family history. To confirm clinical suspicion, sleep-deprived or sleep EEG can be performed. If the epilepsy syndrome remains unclassified, broad-spectrum antiepileptic drugs, such as VPA (which in the elderly carries an increased risk of parkinsonism, encephalopathy, accelerated brain volume loss, and drug interactions [Marson and Sills, 2016]) or levetiracetam (Trinka *et al*., 2013), should be used.

In elderly patients presenting with *de novo* late-onset ASE, a diagnosis of late-onset IGE should be considered in the differential diagnosis, particularly in atypical cases (*e.g*. absence of triggering factors, ASE associated with GTCS or myoclonic seizures, and interictal generalized epileptiform discharges with normal background activity). *De novo* late-onset ASE (Thomas *et al*., 1992; Thomas *et al*., 1993) should be differentiated from both late-onset IGE manifesting with atypical *de novo* ASE and late-onset ASE in patients with a previous history of IGE (Fernández-Torre and Rebollo, 2009).

Although no studies on seizure recurrence for these three conditions have been conducted so far, it seems reasonable to hypothesize that late-onset IGE manifesting with atypical *de novo* late-onset ASE (as in our case) or late-onset ASE in patients with a previous history of IGE carry a higher risk of seizure recurrence relative to typical *de novo* late-onset ASE (Pro *et al*., 2011). Compared to the latter, late-onset IGE manifesting with ASE in the elderly may be associated with a higher risk of seizure recurrence because of the lack of triggering factors, the coexistence of different seizure types other than absence (*e.g*. GTCS or myoclonic seizures), and the presence of interictal generalized epileptiform discharges. Further studies are, however, required to assess the risk of seizure recurrence for different types of late-onset ASE and definitively ascertain whether atypical *de novo* late-onset ASE should be classified under late-onset IGE. □

Disclosures.
None of the authors have any conflict of interest to declare.

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