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Late Epilepsy Secondary To Mesial Sclerosis

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Abstract:
Background*: Williamson et al and then Engel defined the syndrome of the medial side of the temporal lobe which associates hippocampal sclerosis with a relatively typical electroclinical picture. In the medial aspect of the temporal lobe syndrome, there is often a reproducible chronological sequence of events. The objective of our study was to determine and analyse late epilepsy secondary to mesial sclerosis in the Algerian population.*
Materials and Methods*: The study population includes all Algerian patients whose age of onset of the first seizure is 25 years or more, recruited during the period from January 2008 to December 2016 at ALI AIT IDIR Hospital in Algiers.*
Results: *In our study, we find 5 cases of mesial sclerosis. The distribution by age group shows that mesial sclerosis is predominant in the group of subjects (30-34 years).*
Conclusion: *Our study confirms the presence of mesial sclerosis in the etiologies of late onset epilepsy with a percentage of* 2.5%.
Key Words*: Late onset epilepsy, Mesial sclerosis, Hippocampal sclerosis, Temporal lobe epilepsy, Algerian population.*

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I. Introduction

Hippocampal sclerosis has been known to neuropathologists since the original description by Bouchet and Cazauvieilh. These two pathologists had described as early as 1825 neuronal loss specifically affecting certain hippocampal fields during post-mortem studies of epileptic patients.

Sommer subsequently established a relationship between the existence of this hippocampal lesion and seizures. More recently, Williamson et al and then Engel defined the syndrome of the medial side of the temporal lobe which associates hippocampal sclerosis with a relatively typical electroclinical picture. In the medial aspect of the temporal lobe syndrome, there is often a reproducible chronological sequence of events: history of an early event (complicated febrile seizures or head trauma). Asymptomatic free interval of a few years then onset of clinical crises in childhood or adolescence. This chronology of events raises many questions and led to think that hippocampal sclerosis could be the cause and not the consequence of epilepsy. The debate remains open even if many elements suggest that hippocampal sclerosis is both the cause and the consequence of epilepsy in predisposed subjects.

II. Epilepsy Of The Medial Aspect Of The Temporal Lobe (According To Angel)

Background

-Higher frequency of complicated febrile seizures than in other types of epilepsy.
-Family history of epilepsy.
-Beginning in the second half of the first decade. Auras occurring in isolation.
- Rare secondarily generalized seizures.
-Seizures disappearing for a few years at the time of adolescence
- Crises often becoming drug resistant.
-Possibility of developing behavioral problems, usually depression.

Clinical characterization of seizures

An aura is usually present. The most frequent is epigastric, often associated with other dysautonomic or psychic symptoms of an emotional type (eg fear). Olfactory or taste sensations may also occur. The duration of auras is usually a few seconds.Complex partial seizures often start with a stop reaction. Oro¬alimentary and

complex automatisms are frequent.Abnormal posture of one arm, contralateral to the epileptogenic discharge, may occur.The seizure usually lasts 1 to 2 minutes.The post-ictal phase usually includes disorientation, short-term memory deficit, seizure amnesia and dysphasia if the epileptic discharge originates in the dominant hemisphere. This phase usually lasts a few minutes.

Neurological examination and paraclinical data

Neurological examination usually normal, except for memory deficits. Uni or bilateral anterior temporal spikes on the EEG with maximum amplitude on the basal electrodes.Ictal activity on the surface EEG only contemporary with the symptoms of the complex partial phase: initial or delayed onset of a focal rhythmic EEG pattern of 5 to 7 cycles per second, with maximum amplitude on one of the basal temporal leads. Memory problems specific to an impairment of the temporal lobe during neuropsychological tests.

In the strict sense of the term, the definition of hippocampal sclerosis can only be pathological. This is a neuronal loss specifically affecting certain hippocampal sectors: CA1 and CA3 (rarity of pyramidal cells). CA4 (rarefaction of polymorphic cells in the hilum region) and to a lesser degree the dentate gyrus (rarefaction and dispersion of granule cells). Other regions such as CA2 or the SUBICULUM are relatively preserved by neuro¬nal loss. (Babb and Brown) [1] demonstrated through their cell counting work that the distribution of neuro¬nal loss within the hippocampal sectors could vary, either predominating at the level of CA1 (sclerosis of Ammon's horn) or CA3, CA4 and the hilum of the dentate gyrus (sclerosis of the terminal folium or end folium sclerosis), either by globally affecting all the hippocampal sectors (hippocampal sclerosis stricto sensu), or by affecting. Besides the hippocampus, other internal temporal structures such as the parahippocampal gyrus, the peritonsillar cortex and the amygdala (medial temporal sclerosis).

The radiological expression of hippocampal sclerosis is hippocampal atrophy, which can now be visualized in vivo using MRI thanks to the work of (Jackson et al 1990) [2]. For this, we must use the appropriate plane, the plane perpendicular to the long axis of the hippocampus. The MRI aspect is that of both a decrease in hippocampal volume reflecting atrophy and a modification of the hippocampal signal in a T2-weighted sequence. Hippocampal atrophy is the morphological consequence of hippocampal sclerosis and therefore of neuronal loss. It can be objectified either by visualizing a decrease in volume of one hippocampus compared to the other, or by a method of volumetric quantification. Another characteristic of hippocampal sclerosis on MRI is the existence of a signal anomaly on the T2-weighted sequences in the form of a hypersignal either localized precisely in the hippocampus, or extended to the entire medial temporal region. This hypersignal in weighted T2 sequences is interpreted as evidence of a change in tissue composition (in particular gliosis reactions accompanying neuronal loss).

One of the main interests of MRI, in addition to the essential contribution it represents in the diagnosis of this hippocamic sclerosis, is to be able, through repeated and longitudinal follow-up studies in epileptic patients, to better understand the physiopathology of the latter. We know today, thanks to studies MRI, that hippocampal sclerosis is a dynamic process that can evolve in stages. Over the years and with the repetition of the seizures, the sclerosis of the hippocampus will worsen, thus constituting an autonomized epileptogenic focus contributing to the recurrence of the seizures, this repetition of the seizures causing in turn an aggravation of death cellular and therefore hippocampal sclerosis. Mathern et al [3], thus re-examined the surgical specimens of 572 patients suffering from temporal lobe epilepsy and demonstrated that the presence of hippocampal sclerosis was closely linked to the existence of an initial precipitating factor and to an extended duration. Seizures with a direct correlation between the neuronal loss of the different ammonian fields and the duration of the seizures. Other specific observations report the appearance of hippocampal sclerosis in the aftermath of repeated generalized seizures, prolonged febrile seizures or status epilepticus. Interestingly, early transient MRI abnormalities such as increased volume of hippocampal structures and hypersignal were often described in these patients, giving way to sclerosis and suggesting the existence of an initial cytotoxic phenomenon. All these facts therefore suggest that hippocampal sclerosis may appear following an initial precipitating factor and may be the consequence of seizures. However, not all patients with complicated febrile seizures in early childhood, or recurrent generalized seizures will develop hippocampal sclerosis. Moreover, while the initial trauma is most often diffuse throughout the brain, hippocampal sclerosis tends to be unilateral or at least to predominate on one side. Finally, we do not always find an initial precipitating factor in patients with hippocampic sclerosis.

MRI of the hippocampus

• Found hippocampal sclerosis more frequently encountered in temporal lobe epilepsy (60-85%). Usually unilateral, but can be bilateral

• Atrophy associated with hypersignal from the hippocampus on T2 and FLAIR weighted sequences.

• Other signs:

– an enlargement of the ipsilateral temporal horn,

– gliosis of the temporal pole resulting in dedifferentiation white matter – gray matter
– loss of digits of the head of the hippocampus, which reflects the architectural disorganization induced by neuronal destruction.

III. Material And Methods

The study population includes all Algerian patients whose age of onset of the first seizure is 25 years or more, recruited at ALI AIT IDIR Hospital in Algiers.

Inclusion criteria:
1. The age of the patients must be greater than or equal to 25 years at the time of inclus.
2. Patient presenting with his first epileptic seizure at the age of 25 years or older.
3. Clinically and electrically confirmed diagnosis of epilepsy.

Exclusion criteria:
1. Age less than 25 years

IV. Results

Our study population includes 336 patients, recruited during the period from January 2008 to December 2016. This figure corresponds to the number of patients selected according to the inclusion criteria.

1. Etiological diagnosis:

Table 1. Etiological diagnosis in the study population

	Cases	%
Cerebral lesion	196	58,3
No detectable cause	140	41,7
Total	336	100

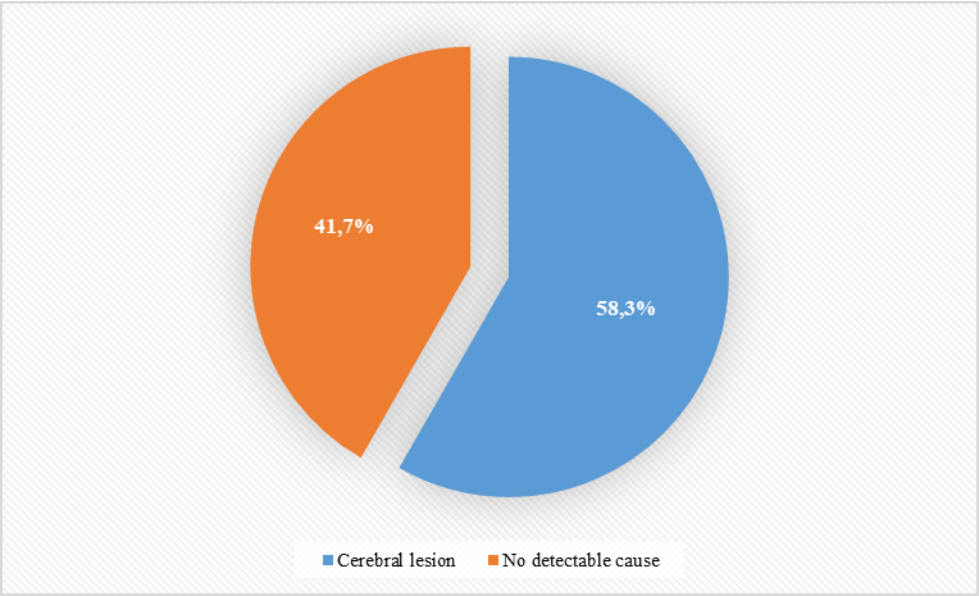

Figure 1. Frequency of cerebral lesion in the study population

A cerebral lesion was found in approximately 58.3% of cases (196 cases).

Table 2. Distribution of cerebral lesion by age group

	Cerebral lesion		No detectable cause	
	Cases	%	Cases	%
25-29 years	16	5	30	9
30-34 years	27	8	19	6
35-39 years	26	8	23	7
40-44 years	16	5	12	3
45-49 years	21	6	6	2
50-54 years	17	5	9	3

55-59 years	15	4	10	3
60-64 years	17	5	6	2
65-69 years	14	4	6	2
70-74 years	12	3	9	3
75-79 years	10	3	6	2
80 years and over	5	1	4	1
Total	**196**	**57**	**140**	**43**

The distribution by age group shows a predominance of cerebral lesion for all age groups except for the group of subjects aged (25-29 years) where the patients had no detectable cause.

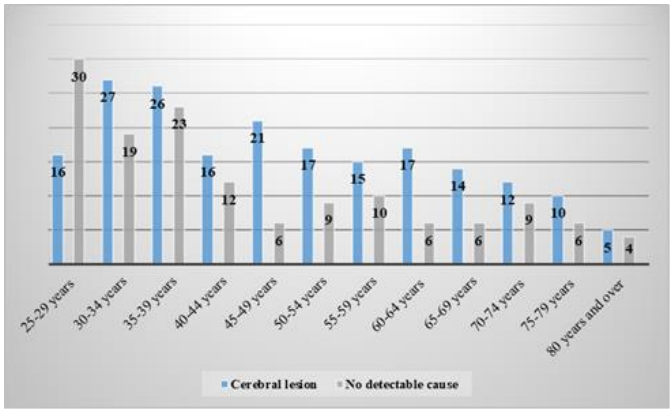

Figure 2. Distribution of cerebral lesion according to age groups

2. Mesial sclerosis:

Table 3. Mesial sclerosis in the study population

		Cases	**%**
Other cerebral pathologies		56	16,6
Inflammatory			
	Multiple sclerosis	15	4,5
	Neuro-Behçet's disease	5	1,5
	Neurolupus	1	0,3
	Mixed connective tissue disease	1	0,3
Brain arteriovenous malformations		19	5,6
Basal ganglia calcification		1	0,3
Mesial temporal sclerosis		5	1,5
Cerebral venous thrombosis		7	2
Arachnoid cyst		2	0,6
Total		**336**	**100**

In our study we find 5 cases of mesial sclerosis.

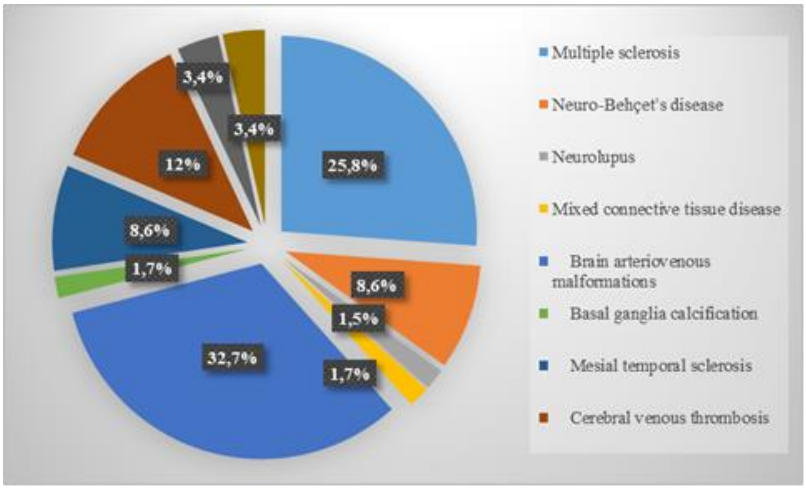

Figure 3. Frequency of mesial sclerosis in the study population
Mesial sclerosis represent 2.5% of cases.

Table 4. Mesial sclerosis by age group

	Mesial sclerosis
25-29 years	0
30-34 years	2
35-39 years	0
40-44 years	0
45-49 years	1
50-54 years	1
55-59 years	1
60-64 years	0
65-69 years	0
70-74 years	0
75-79 years	0
80 years and over	0
Total	**5**

The distribution by age group shows that mesial sclerosis is predominant in the group of subjects (30-34 years).

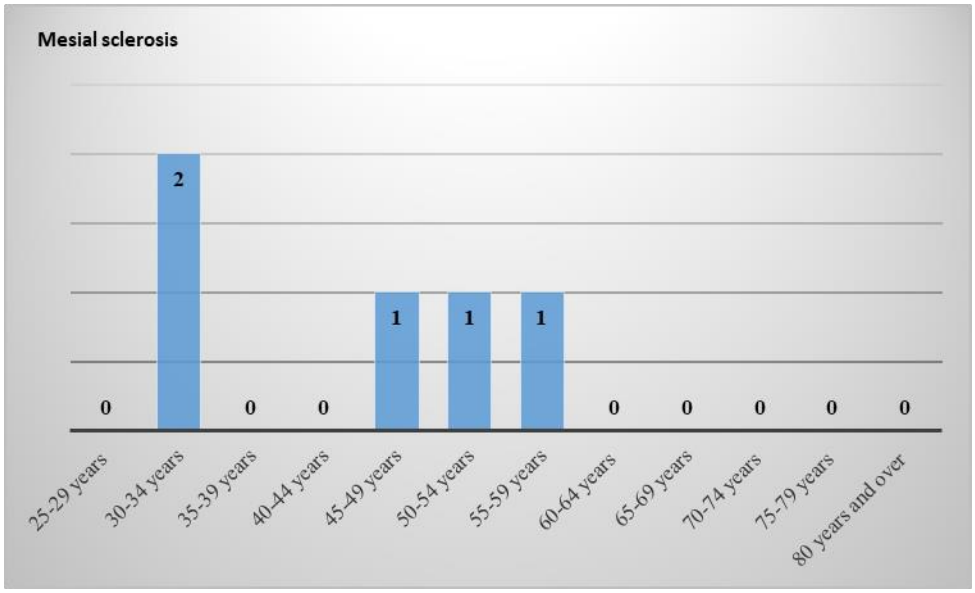

Figure 4. Distribution of mesial sclerosis according to age groups

V. Discussion

A cause was found in 58.3% of cases. This situation has been observed in several studies (José Luis Perez Lopez, 1985 [4] - Roberto Suastegui et al, 2009 [5] - Lars Forsgren, 1990 [6]) with respectively 50.8%, 51%, and 49%.

Our study confirms the presence of mesial sclerosis in the etiologies of late onset epilepsy with a percentage of 2.5%.

Mesial sclerosis is common in subjects whose age group (30-34 years).

Our results agree with the literature data. In the work of Lars Forsgren, 1990 [6], a cause was found in 49% of cases.

GCY Fong et al, 2003 [7], had reported a frequency of hippocampal sclerosis (20.3%).

Mesial sclerosis is another cause found in our results, but which is not very frequent in our study, with a figure of 2.5% of cases.

Our results are however different from those of the literature. GCY Fong et al, 2003[7], noted a rate of 20.3%, this could be explained by the frequency of febrile seizures in the history of the study population.

Table 5. Literature review of mesial sclerosis in late onset epilepsy

Study	Country	Mesial sclerosis
José lwis Perez Lopez, 1985	Spain	ND
Agnete Mouritzen Dam, 1985	Denmark	ND
R.Sridharan et al, 1986	Libya	ND
Basim A.Yakoub et al, 1987	Saudi Arabia	ND
Anthony Hopkins et al, 1988	United Kingdom	ND
Lars Forsgren, 1990	Sweden	ND
Daniel Arbaiza 1995	Peru	ND
Lars Forsgren et al, 1996	Sweden	ND
Marcelo Rigatti et al, 1999	Brasil	ND
Andre Oun et al, 2003	Estonia	ND
GCY Fong et al, 2003	Hong Kong	20.3%
David Ortega Rivero et al, 2003	Ecuador	ND
Christian Napon et al, 2009	Burkina Faso	ND
Robero Suastegui et al, 2009	Mexico	ND
Ewan Hunter et al, 2012	Tanzania	ND
Sudhir Chasani et al, 2015	India	ND
Our series	Algeria	2.5%

VI. Conclusion

On the etiological level, our study confirms the presence of mesial sclerosis in the etiologies of late onset epilepsy with a low percentage of 2.5%. The distribution by age groups shows that mesial sclerosis is dominant in the group of subjects (30-34 years old).

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