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Degenerative Causes Of Late Onset Epilepsy In The Algerian Population

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Abstract:
Background*: 10% of patients with Alzheimer's disease have epilepsy. Frontotemporal dementia with parkinsonism linked to chromosome 17 (FTDP - 17q) initially described in 1994 has been repeatedly associated with the presence of epilepsy. A degenerative dementia process occurring after the age of 30 is frequent in trisomy 21 and associated with neuropathological changes of the Alzheimer type, it is readily accompanied by epileptic seizures. In Huntington's disease, clinical semiology includes a choreo-athetotic syndrome, myoclonus and epileptic seizures.The objective of our study was to determine and analyse degenerative causes of late onset epilepsy 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: *Degenerative causes represent 12 cases (Alzheimer's disease).The distribution by age group shows a predominance of degenerative pathology for the group of subjects aged 65-69 years.*
Conclusion: *Our study confirms the presence of degenerative etiologies (represented by Alzheimer's disease) in the etiologies of late onset epilepsy with a percentage of 6.1%.*
Key Words*: Late onset epilepsy, Degenerative pathology, Alzheimer's disease, Algerian population.*

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I. Introduction

Among subjects over 65, degenerative causes represent a rate of 10% in the survey carried out in Rochestrer -Minnesota during the period 1935-1984 (Hauser et Al, 1993) [1] and (10-20%) in Texas (Annegers et al, 1999) [2] Whereas (Olafsson et al, 2005) [3] found a rate of 25%.

1. Alzheimer's disease

In Alzheimer's disease 10% of patients with Alzheimer's disease have epilepsy. The risk of seizures is multiplied by six in this population.The risk factors for seizures are represented by drugs (neuroleptics and procholinergics), and associated cardiovascular pathologies.Epileptic seizures must be differentiated from myoclonus of cortical origin, distal and of low amplitude, producing a pseudo-tremor appearance (mini polymyoclonus).

Seizures can be partial or more often described as generalized tonic-clonic, occurring at an advanced stage of the disease. Partial seizures limited to a subjective aura, or to a break in contact accompanied by food automation can also be difficult to diagnose, including for those around you, given the intellectual deterioration. However, a complex partial illness was reported in two subjects aged 71 and 85.

The background rhythms are replaced by theta activity, first on the temporal lobes then diffusely. When the disease worsens, these are delta rhythms which are recorded first on the temporal lobes then diffused and which are poorly reactive. Finally, bursts of very slow, intermittent rhythmic activity may appear (Gordon EB and Sim M, 1967) [4]. Patients are not equal when it comes to the appearance of EEG abnormalities. Thus, in patients whose disease progresses slowly, it is possible that no abnormality is recorded for several years (Soininen H et al, 1991) [5].

Dementia pathology is well represented in the group of subjects over 60 with a rate of 2% in the study by (Luhdorf, 1986) [6], 6% in the survey by (Loiseau, 1990) [7], and a rate of 4% for (Ettinger, 1993) [8].

2. Frontotemporal Dementia (FTD)

Frontotemporal Dementia constitute a group of conditions brought together on the basis of a semiology based on the predominant attack of the frontal and anterior temporal lobes, with a neuropathological attack distinct from Alzheimer's disease. Frontotemporal dementia with parkinsonism linked to chromosome 17 (FTDP - 17q) initially described in 1994 has been repeatedly associated with the presence of epilepsy. Frontotemporal dementias are the only ones that practically do not alter the EEG when intellectual deterioration is already advanced.

3. Trisomy 21 (Down Syndrome)

A degenerative dementia process occurring after the age of 30 is frequent in trisomy 21 and associated with neuropathological changes of the Alzheimer type, it is readily accompanied by epileptic seizures.The prevalence of epilepsy increases rapidly with age, reaching 46% of this population after 50 years. The late-onset epilepsy of Down syndrome is well characterized, associating myoclonus and generalized tonic-clonic seizures (senile myoclonic epilepsy).

4. Dento-rubro-palido-luysian atrophy (DRPLA)

Dento-rubro-palido-luysian atrophy is a rare pathology, autosomal dominant, initially described in Japan where the incidence remains the highest. Characterized by ataxia, abnormal movements, dementia syndrome, and epilepsy.The EEG can highlight epileptiform abnormalities such as atypical complex wave spikes, bursts of slow waves, photosensitivity, abnormal background rhythms (Inazuki G et al, 1989) [9].The DRPLA gene is characterized by the repetition of a CAG triplet in position 12 P13, 3, allowing the diagnosis to be made.

5. Huntington's disease

Huntington's disease is an autosomal dominant hereditary disease, it is characterized by the association of a choreo-athetotic syndrome and progressive dementia in an adult subject.The condition is due to an unstable mutation of a gene located on the short arm of chromosome 4, responsible for the synthesis of a protein called Huntington whose physiological role is not known. The mutation will lead to an abnormal repetition of a CAG triplet coding for glutamine and, therefore, to the formation of a pathological protein carrying an abnormal expansion of polyglutamines. The number of CAG trinucleotide repeats is less than 30 (on average 19) at the level of the normal gene, it is greater than 36 in patients and in the majority of cases, it is between 42 and 48. This number is correlated inverse with the age of clinical onset of the disease.

The clinical semiology includes a choreo-athetotic syndrome, myoclonus and epileptic seizures, we also note the presence of intellectual deterioration with behavioral disorders. In the advanced stages of the disease, the EEG is often normal, the background rhythms are of reduced amplitude, the alpha activity is poorly drawn and slow low or moderately volute activity is recorded diffusely (Scott DF et al , 1972) [10], this disappearance of alpha rhythms was correlated with the severity of the disease (Pokrovskaia ZA and Insarova NG, 1988) [11]. The percentage of alpha and theta powers is reduced, the percentage of delta and beta powers is increased. The frequency of theta rhythms is reduced by 1 C/S. These modifications are correlated with the severity of the neurological impairment (Bylsma FW et al, 1994) [12].

II. 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

III. 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

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

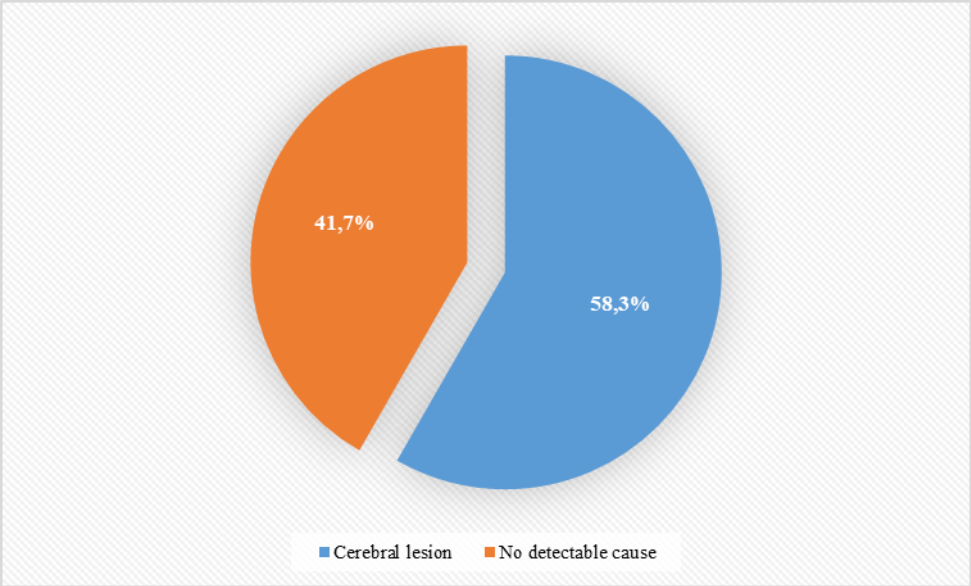

Figure 1. Frequency of cerebral lesion in the study population

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.

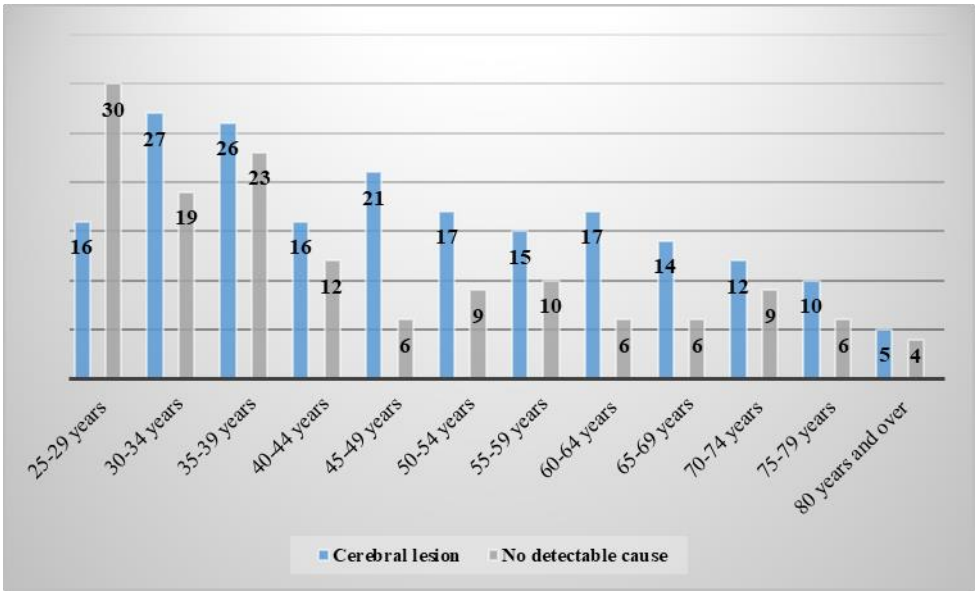

Figure 2. Distribution of cerebral lesion according to age groups

Degenerative pathology:

Table 3. Distribution of degenerative pathology by age group

	Degenerative pathology
25-29 years	0
30-34 years	0
35-39 years	1
40-44 years	2
45-49 years	0
50-54 years	0
55-59 years	0
60-64 years	2
65-69 years	3
70-74 years	2
75-79 years	1
80 years and over	1
Total	**12**

The distribution by age group shows a predominance of degenerative pathology for the group of subjects aged 65- 69 years.

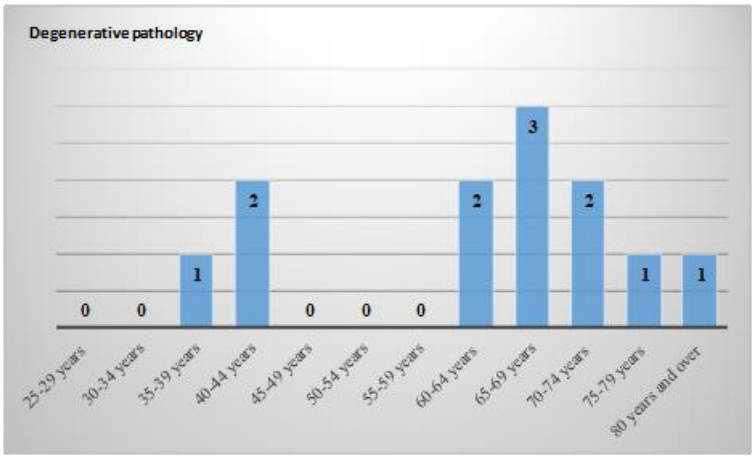

Figure 3. Distribution of degenerative pathology according to age groups

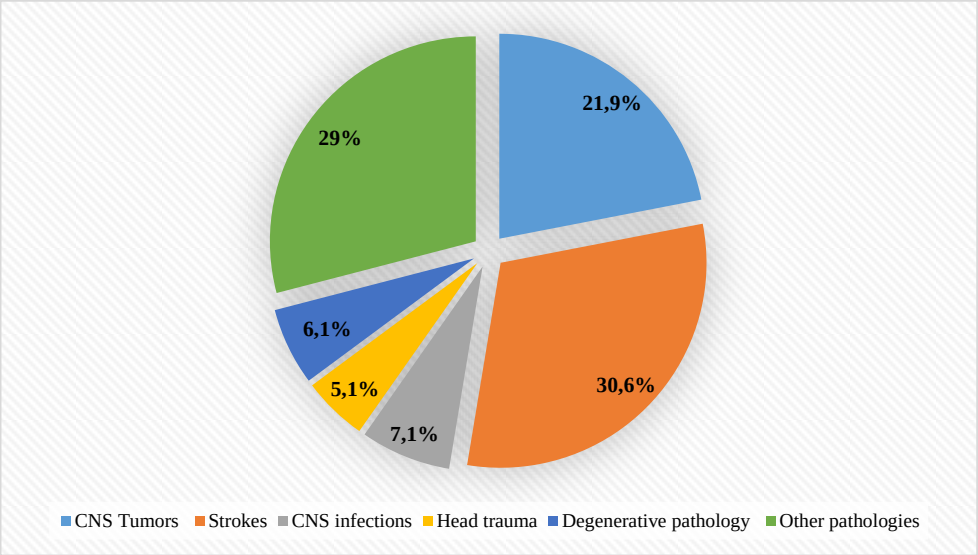

Figure 4. Frequency of degenerative pathology compared to other etiologies

Our study finds a percentage of 12 cases of degenerative pathologies (6.1%) represented by Alzheimer's disease.

IV. Discussion

A cause was found in 58.3% of cases. This situation has been observed in several studies (José Luis Perez Lopez, 1985 [13] - Roberto Suastegui et al, 2009 [14] - Lars Forsgren, 1990 [15]) with respectively 50.8%, 51%, and 49%.

Our study finds a percentage of 6.1% of degenerative pathologies.As for the degenerative causes which represent 6.1% of cases (Alzheimer's disease), which is consistent with certain studies (Anthony Hopkins et al, 1988[16]; Lars Forsgren et al, 1996 [17]; Christian Napon et al , 2009 [18]; Robrto Suastegui et al, 2009[14])

Our results agree with the literature data. In the work of Lars Forsgren, 1990[15], a cause was found in 49% of cases. It was also found that in Lars Forsgren's study, Alzheimer's disease was detected in 7% of cases.

Antony Hopkins et al, 1988[16] had observed that degenerative pathology represents 6.4%.The results of the work of Andre Oun et al, 2003[19], show that Alzheimer's disease was found in 0.3% of cases.

In the work of Christian Napon et al, 2009[18], we note that the pathology degenerative pathology represents 5.4% of cases.

Concerning the etiologies, the analysis by Roberto Suastegui et al, 2009[14], shows that the degenerative causes represents 6%.The etiological data concerning the Ewan Hunter et al study, 2012 [20] show that the degenerative causes represents a percentage of 2%.

Table 4. Literature review of degenerative pathology in late onset epilepsy

Study	Country	Degenerative Pathology
José lwis Perez Lopez, 1985	Spain	19.2 %
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	6.4%
Lars Forsgren, 1990	Sweden	ND
Daniel Arbaiza 1995	Peru	ND
Lars Forsgren et al, 1996	Sweden	7% (Alzheimer's disease)
Marcelo Rigatti et al, 1999	Brasil	ND
Andre Oun et al, 2003	Estonia	ND
GCY Fong et al, 2003	Hong Kong	ND
David Ortega Rivero et al, 2003	Ecuador	3%
Christian Napon et al, 2009	Burkina Faso	5.4%
Robero Suastegui et al, 2009	Mexico	6%
Ewan Hunter et al, 2012	Tanzania	2%
Sudhir Chasani et al, 2015	India	ND
Our series	**Algeria**	6.1%

		(Alzheimer's disease)

V. Conclusion

On the etiological level, our study confirms the presence of degenerative etiologies (represented by Alzheimer's disease) in the etiologies of late onset epilepsy with a percentage of 6.1%. The distribution by age group shows a predominance of degenerative pathology for the group of subjects aged 65-69 years.

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