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Late Post-Traumatic Epilepsy In The Algerian Population

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Abstract:
Background*: Post-traumatic epilepsy is defined by the recurrence of seizures following a head trauma, apart from early seizures, thus occurring beyond the first week following the trauma. Post-traumatic epilepsy occurs in 50% of cases during the first year following the head injury, in 75% of cases within 2 years, 85% of cases within 5 years, and only 3% occur after 10 years. The objective of our study was to determine and analyse post-traumatic 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: *Post-traumatic causes represent 10 cases. The distribution by age group shows a predominance of head trauma for the groups of subjects aged (30-34 years).*
Conclusion: *Our study confirms the presence of head trauma in the etiologies of late onset epilepsy with a percentage of 6.1%.*
Key Words*: Late onset epilepsy, post-traumatic causes, head trauma, Algerian population.*

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I. Introduction

Head trauma account for 4% of all etiologies of late-onset epilepsies (Annegers, 2000) [1].Post-traumatic epilepsy is defined by the recurrence of seizures following a head trauma, apart from early seizures, thus occurring beyond the first week following the trauma.

Post-traumatic epilepsy occurs in 50% of cases during the first year following the head injury, in 75% of cases within 2 years, 85% of cases within 5 years, and only 3% occur after 10 years.

The risk of developing epilepsy after a head injury is 03 times higher than in the general population. The risk of developing epilepsy is directly correlated with the severity of the trauma. The severity of the trauma directly influences the onset of epilepsy.

A head trauma is considered mild if the loss of consciousness is less than 30 minutes, without fracture, without contusion, or intracerebral hematoma. For a moderate trauma, the loss of consciousness is less than 24 hours, a fracture can be found but there is no contusion or hematoma.

Finally, a severe trauma associated with a loss of consciousness for more than 24 hours, the presence of contusion and intracerebral haemorrhage, embarrure, a cranioencephalic wound.

Risk factors for developing epilepsy after trauma:
1/ The severity of the trauma:
- Open trauma
- The presence of projectiles
- Presence of a hematoma
- Bruise
- Cranio-cerebral wound with cerebro-meningeal infection
- Subdural hematoma
- Glasgow score between 3 and 8
- Initial neurological deficit
- Cortico-subcortical lesions

2/Early seizures in adults
3/ Age > 65

3/ Loss of consciousness of long duration >24 hours
4/ Post-ictal amnesia
5/ Trauma topography: frontal, parietal, biparietal, temporal
6/ Abnormal EEG persisting beyond 01 month after the trauma (risk increased to 3 times).

Pohlman-Eden and Bruckmeir indicate that the risk increases if 3 elements characterizing severe trauma are present.Epilepsy occurring in half of the cases during the 1st year, after a moderate to severe head trauma.

Roger et al, retain the following criteria to establish the imputability of trauma to the development of epilepsy:
- Criteria of certainty:
- *Cranioencephalic wound*
- *Cortical involvement*
- *Immediate post-traumatic complication*
- *Durable neurological sequelae of cortical type (aphasia, hemiplegia)*
- Suspicion criteria:
- *Coma and/or amnesia for more than 24 hours*
- *Skull fracture with embrure*
- *Early epileptic seizures*

Post traumatic epilepsy is a partial epilepsy expressed by partial seizures which can be simple, complex and secondarily generalized. It is often seen in head injuries of frontal and central locations. From a medico-legal point of view, to retain imputability, at least one criterion of certainty is required, otherwise two criteria of suspicion. One of the main elements to retain a post-traumatic origin of epilepsy: is the observation of post-traumatic lesions on imaging. Imaging consists of performing a cerebral CT supplemented by a cerebral MRI. Radiological examinations are essential: To characterize the sequelae of head trauma and to establish the correlation between the lesion and epilepsy Imaging is now a very useful tool for determining imputability. The epileptic abnormalities on the EEG are very tiled with the loss of cerebral volume, the presence of seizures, and diffuse cerebral involvement.

EEG abnormalities can be observed during the first month and are related to the trauma and will then disappear, which is why it is necessary to repeat an EEG at more than 1 month in order to see whether the abnormalities persist. These abnormalities persisting beyond the first month are primarily sequel elements of the trauma, slow theta or delta waves and flap rhythm, this rhythm is observed during a bone breach and is characterized by polyrhythmic elements which intertwine, which are ample and sometimes acute. The EEG has a predictive value if it is abnormal within 24 hours after the trauma, epileptic abnormalities on the EEG are highly correlated with the loss of brain volume, the presence of seizures, and diffuse brain damage. Anterior temporal and Rolandic epileptic foci are always associated with post-traumatic epilepsy (Jabbari B et al 1986) [2]. Standard EEGs are sometimes devoid of epileptic activity, whereas EEGs performed after 24 hours of sleep deprivation detect more abnormalities. Epileptic seizures on the EEG can be focal or generalized.

Post-traumatic epilepsy is the most frequent pathology in subjects whose age group is between 25-64 years in the study carried out in Iceland (Olafsson et al, 2005) [3], moreover it represents a by (3 – 4%) in subjects over 60 (Luhdorf, 1986 [4]; Loiseau, 1990 [5]). While in the survey conducted in Texas in 1999 (Annegers et al) [6] found a percentage of 20% among subjects aged 25-44, 10% in the group aged 45-64, and less than 10% among subjects over 65 years old.

Early onset seizures assisted in 10% of adult severe head injuries (50% within the first day) and only 0.4% of mild injuries; early seizures are normally related to cerebral contusion; immediate attacks can worsen the clinical picture, the prognosis and the subsequent risk of attacks.

Late seizures in post-traumatic epilepsy mainly result from sequelae of cerebral contusions. These seizures are accompanied after a free interval which is normally less than 2 years. The risk of seizure increases with the severity of the trauma and of the immediate brain damage, but also appears to be linked to the topography of the initial attack: the sequelae of parietal contusion are more epileptogenic than temporal, occipital or frontal lesions. In the acute phase, the scan identifies the acute complications (pericerebral hematomas, cerebral contusions, fractures of the vault and the base of the skull, trauma to the facial bone). In the late phase, CT shows the most severe sequelae of contusion in the form of cerebral atrophy associated with hypodensity of adjacent brain tissue; these lesions predominate at the fonto-basal and frontopolar level as well as at the temporo-polar level; parieto-occipital damage is rarer and leads to repercussion lesions. If the scan does not show any lesion likely to explain the clinical picture, recourse to MRI is essential. MRI allows a complete lesional assessment, thanks to two main sequences; the FLAlR sequence visualizes the adjacent parenchymal lesions in a hyperintense signal which reflects gliosis and demyelination; the gradient echo T2 sequence identifies hemorrhagic sequelae in the form of hypointense cortical foci (sequelae of cortical contusions) and within the supratentorial white matter, of the corpus callosum (diffuse axonal lesions).

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.

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).

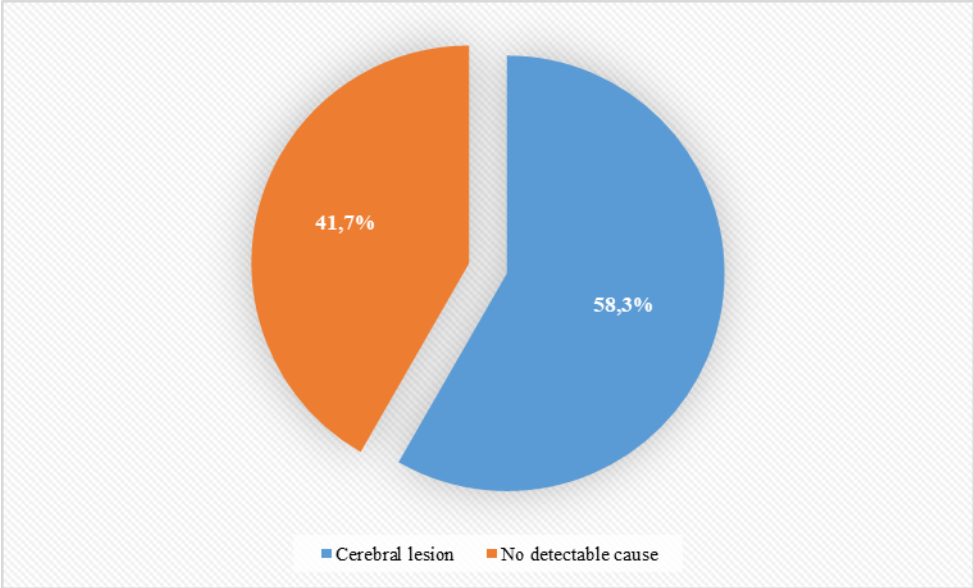

Figure 1. Frequency of cerebral lesion in the study population

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.

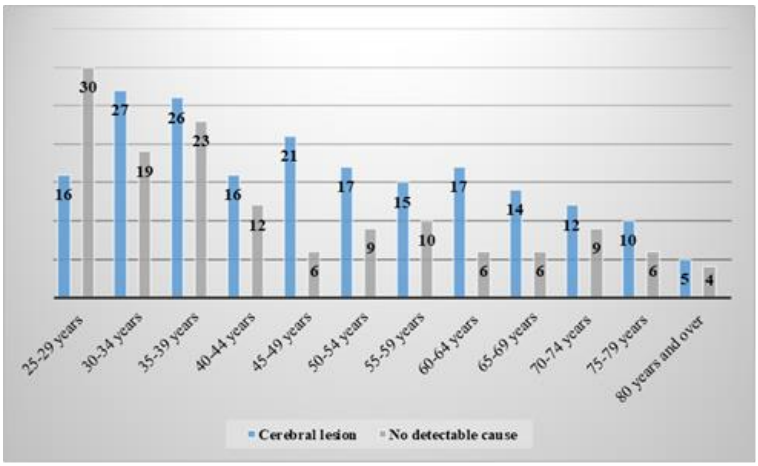

Figure 2. Distribution of cerebral lesion according to age groups

2. Post-traumatic causes (Head trauma):

Table 3. Distribution of head trauma by age group

	Head Trauma
25-29 years	0
30-34 years	5
35-39 years	1
40-44 years	0
45-49 years	2
50-54 years	0
55-59 years	0
60-64 years	1
65-69 years	1
70-74 years	0
75-79 years	0
80 years and over	0
Total	**10**

The distribution by age group shows a predominance of head trauma for the groups of subjects aged (30-34 years).

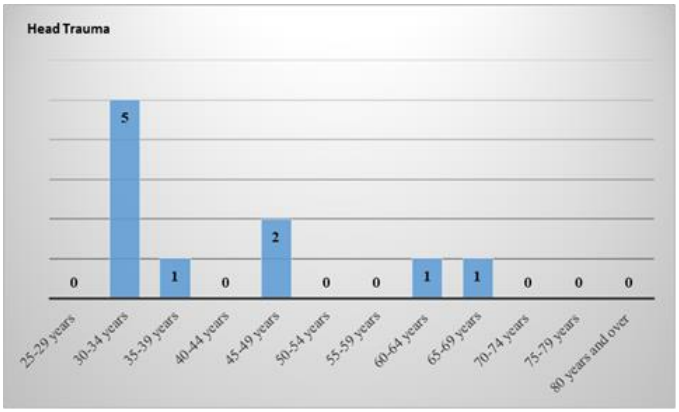

Figure 3. Distribution of head trauma according to age groups

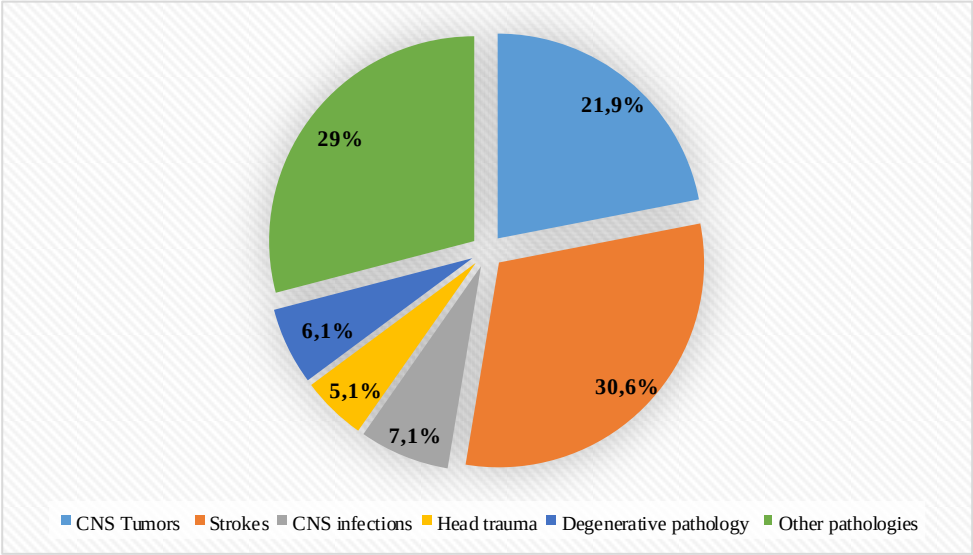

Figure 4. Frequency of head trauma compared to other etiologies

Our study confirms that post-traumatic causes represent 5.1% of cases (10 cases).

IV. Discussion

A cause was found in 58.3% of cases. This situation has been observed in several studies (José Luis Perez Lopez, 1985 [7] - Roberto Suastegui et al, 2009 [8] - Lars Forsgren, 1990 [9]) with respectively 50.8%, 51%, and 49%.

Regarding traumatic causes, we still find a rate of 5.1%. Our results are consistent with data from the literature (R.Sridharan et al, 1986 [10]; Lars Forsgren, 1990 [9]; Christian Napon et al, 2009 [11]).

The distribution by age group shows a predominance of head trauma in the group of subjects (30-34 years old).

Our results agree with the literature data. In the work of Lars Forsgren, 1990 [9], a cause was found in 49% of cases, head trauma was in 7% of cases.

José Lwis Lopez et al, 1985 [7], had shown that head trauma was 11.2%. Agnet Mouritzen Dam et al, 1985 [12] found that head trauma was 4%. The work of Belaidi et al, 1986 [13], shows that head trauma represents 18.7%.

In the study by Marcelo Rigatti et al, 1999 [14], the most frequent etiologies were: Neurocysticercosis 20%, head trauma 15%.The results of the work of Andre Oun et al, 2003 [15], show that head trauma was by far the dominant cause with a rate of 13.4%.). It is especially in the group of (40-59 years) that the cranial trauma was dominant.

Sridharan et al, 1986 [10] found that head trauma was the most frequent cause with a percentage of 50.1% of cases.In the work of Christian Napon et al, 2009 [16], we note that head trauma was the cause of late onset epilepsy in 6.8% of cases. The etiological data concerning the study Ewan Hunter et al, 2012 [17] show that traumatic causes represent 9.8% of cases.

Table 4. Literature review of head trauma in late onset epilepsy

Study	Country	Head trauma
José lwis Perez Lopez, 1985	Spain	11.2%
Agnete Mouritzen Dam, 1985	Denmark	4%
R.Sridharan et al, 1986	Libya	5.1%
Basim A.Yakoub et al, 1987	Saudi Arabia	ND
Anthony Hopkins et al, 1988	United Kingdom	ND
Lars Forsgren, 1990	Sweden	7%
Daniel Arbaiza 1995	Peru	ND
Lars Forsgren et al, 1996	Sweden	ND
Marcelo Rigatti et al, 1999	Brasil	15%
Andre Oun et al, 2003	Estonia	13.4%
GCY Fong et al, 2003	Hong Kong	ND
David Ortega Rivero et al, 2003	Ecuador	ND
Christian Napon et al, 2009	Burkina Faso	6.8%
Robero Suastegui et al, 2009	Mexico	ND
Ewan Hunter et al, 2012	Tanzania	9.8%
Sudhir Chasani et al, 2015	India	ND

Our series	**Algeria**	**5.1 %**

V. Conclusion

Etiologically, our study confirms the presence of head trauma in the etiologies of late onset epilepsy with a percentage of 5.1%. The distribution by age group shows a predominance of head trauma for the group of subjects aged 30-34 years.

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