IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 23, Issue 5 Ser. 2 (May. 2024), PP 25-31
www.iosrjournals.org

Infectious Causes Of Late Onset Epilepsy In The Algerian Population

Abdellaoui Walid, Louanchi Malika, Atroune Lynda,Djaafer Miloud , Ait-Oukaci Wassila, Toubal Nadia, Sadibelouiz Mustapha, Ait-Kaci-Ahmed Mahmoud

[1]*neurology Department, Mostaganem Hospital, University Of Mostaganem*
[2]*neurology Department, Ibn Sina Hospital, University Of Annaba*
[3]*neurosurgery Department, Mustapha Pacha Hospital, University Of Algiers*
[4]*neurophysiology Department, Ait Idir Hospital*
[5]*neurology Department, Ait Idir Hospital, University Of Algiers*

Abstract:
Background*: Infections are one of the most common causes of seizures. Central nervous system infections can cause subsequent seizures. Parenchymal infections (viral encephalitis, bacterial meningoencephalitis, brain abscess) are particularly epileptogenic.The objective of our study was to determine and analyse infectious 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: *Infectious causes represent 7.1% of cases. The distribution by age group shows a predominance of the infectious pathology for the groups of subjects aged (30-34 years, 35-39 years, and 55-59 years). The infectious causes are dominated by encephalitis (6 cases) and meningo-encephalitis (5 cases).We find that encephalitis is the predominant cause in the group of infectious pathologies with the percentage of 43%, followed respectively by meningo-encephalitis 36% and cerebral abscess 21%.*
Conclusion: *Our study found a percentage of 7.2% of cases for infectious pathology.We find a percentage of 3% for encephalitis and 2.5% for meningoencephalitis, and finally cerebral abscess 1.5%.*
Key Words*: Late onset epilepsy, Infectious pathology, Encephalitis, Meningo-encephalitis, Cerebral abscess, Algerian population.*

Date of Submission: 27-04-2024 Date of Acceptance: 07-05-2024

I. Introduction

Infections are one of the most common causes of seizures. Central nervous system infections can cause subsequent seizures. Parenchymal infections (viral encephalitis, bacterial meningoencephalitis, brain abscess) are particularly epileptogenic.

Neurocysticercosis is one of the main causes of late onset epilepsy in tropical areas. Infectious causes are strongly represented in epidemiological studies carried out in developing countries. Cysticercosis accounts for 50% of adult epilepsies in Latin America (Weber, 1998 [1]). On the other hand, they are less represented in surveys conducted in developed countries, with a rate of 10% (Hauser et al, 1993 [2]) in Rochester (Minnesota) during the period 1935-1984, and 10-20% (Annegers et al, 1999 [3]), in the group of subjects whose age group is between 25-65 years.

Viral encephalitis in adults is due to HSV-1. This encephalitis can occur as a primary infection or after reactivation of the virus. The clinical presentation is not pathognomonic, the focal encephalitic signs (aphasia, hemiparesis, confusion, behavioral changes) are associated with a febrile syndrome. Epileptic seizures can be partial. The state of mal is not uncommon. Electrical signs often important, nonspecific but often very evocative. The earliest signs are polymorphic slow waves lateralized or on the temporal regions, the most typical aspect observed in two thirds of the cases is that of a pseudo periodic complex (aspect of acute waves repeating every 1-3 seconds) on the temporal regions and which appears in a delayed way (a few hours to a few days). EEG abnormalities often very suggestive but not specific, the earliest signs are polymorphic slow waves lateralized or on the temporal regions. The most typical aspect observed in two thirds of the cases is that of a pseudoperiodic complex on the temporal regions and which appears in a delayed manner (a few hours to a few days). No clinical sign can predict that the encephalitis is of herpetic origin, the EEG is the most valuable and the earliest

examination to identify internal temporal abnormalities, it is almost always abnormal (Gaches J and Malon, 1982 [4]). The earliest signs are polymorphic slow waves lateralized or on the temporal regions. The most typical aspect observed in two thirds of the cases is that of pseudoperiodic complexes (aspect of acute waves repeating themselves every 1-3 seconds on the temporal regions and which appear in a delayed manner from a few hours to a few days).The MRI shows signs of bitemporal involvement (T2 hyper signal).

HIV has been recognized as a risk factor for the onset of epileptic seizures and the development of epileptic disease. Seizures can be isolated without other causes, they are due to infection of the brain by HIV itself . Epileptic seizures can reflect opportunistic infections (such as meningitis, progressive multifocal leukoencephalitis, cerebral toxoplasmosis) or metabolic changes (hyponatremia) (Persidsky et al 200689, Shiu et al 2009 [6]). Seizures are described as generalized tonic-clonic or partial. Possibility of continuous partial epilepsy, generalized status epilepticus. The EEG may show generalized epileptic abnormalities (Modi G et al, 2002 [7]). Status epilepticus has been reported (Bartolomei F et al, 1991 [8]).

All bacterial infections likely to cause meningitis and cerebral sequelae can be the cause of epileptic seizures and sequelae epilepsy. The most frequent bacterial agents encountered are generally meningococci and pneumococci, but also Koch's bacillus (tuberculosis).During bacterial meningitis, the vital prognosis is generally put into play and sequelae have been brought about in nearly 23% of survivors, including the appearance of epileptic manifestations in nearly 4% of them (Edmond et al. al, 2010 [9]).

Neurocysticercosis is due to the infestation of the organism by the larvae of teania sodium, and it is the encysted forms in the central nervous system which are responsible for this disease.It is the most common parasitic disease in developing countries. Epilepsy is the most common manifestation of this disease. Partial seizures are the most common.The EEG can show isolated epileptic anomalies or an association of epileptic anomalies and slow anomalies (theta-delta anomalies) (Diagana M et al, 2005 [10]).Diagnosis is based on cerebral imaging, in particular cerebral CT, showing isolated or calcified nodules. Some cysts have a less well-defined hypo or isodense appearance. When they are associated with cerebral edema, they correspond to cysts being eliminated by the immune system.

Neuromalaria is caused by the sequestration in the cerebral capillaries of red blood cells parasitized by plasmodium falciparum. Epileptic seizures are frequent in cerebral malaria. It is useful to perform serial EEGs because the patient's condition can vary rapidly (Thumasupapong S. et al, 1995 [11]. EEGs preferentially show spikes in the temporoparietal regions.

Cerebral toxoplasmosis Occurs in immunocompromised subjects, particularly in HIV infection.

Creutzfeld-Jakob disease is the most common human prion encephalopathy. Sporadic Creutzfeld-Jakob disease (85 to 90% of cases) most often begins between the ages of 60 and 70 and the course is fatal in 6 months. The clinical picture variably combines dementia, myoclonus, visual disturbances, cerebellar or extrapyramidal syndrome. Continuous partial epilepsy is indicative of the condition. Partial or generalized states of illness have been reported. The EEG is suggestive when it shows periodic complexes, usually bi or triphasic, these complexes are present in 60% of cases. EEG shows generalized periodic spike discharges. It is difficult to distinguish initially between confusional status, especially frontal, and impaired cognitive status due to disease and typically associated with periodic EEG abnormalities. MRI, in particular FLAIR sequences and diffusion MRI, can visualize lesions of the central gray nuclei or of the cortex. The presence in the CSF of protein 14.3.3 is a non-constant and non-specific marker of the disease. NSE (Neuronal Specific Enolase) is another indicator of massive neuronal destruction.

The infectious pathology may be associated with epilepsy in the acute or sequel phase. 10 to 20% of meningoencephalitis are associated with epileptic seizures in the acute phase; a history of infection such as meningitis or encephalitis multiplies the risk of subsequent epilepsy by seven; seizures appear in 90% of cases during the first year. Parasitic sequelae are rare but often epileptogenic.

the sequelae of cysti¬cercosis remain the leading cause of epilepsy in Central America and South America; many Asian and Indian Ocean countries are also risk regions; epilepsy can reveal parasitosis; The parasitic sequelae of cysticercosis result in nodular cortico-subcortical calcifications, the identification of which is easy on CT scans but difficult on MRI; only gradient-echo T2 MRI can identify these calcifications in the form of punctiform hypointensities.

The clinic and the results of biological analyzes are also decisive for the diagnosis of infectious lesions. Early-stage abscess is characterized by a hypodense parenchymal area on CT, then, when collected, by typical but nonexclusive peripheral annular enhancement after contrast. Herpetic encephalitis is characterized by its temporo-limbic tropism manifesting on CT as hypodensity. However, the lesion may go unnoticed on CT scans. In this case, MRI is essential and will make it possible to objectify the lesions in TI and FLAIR hypersignal..

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).

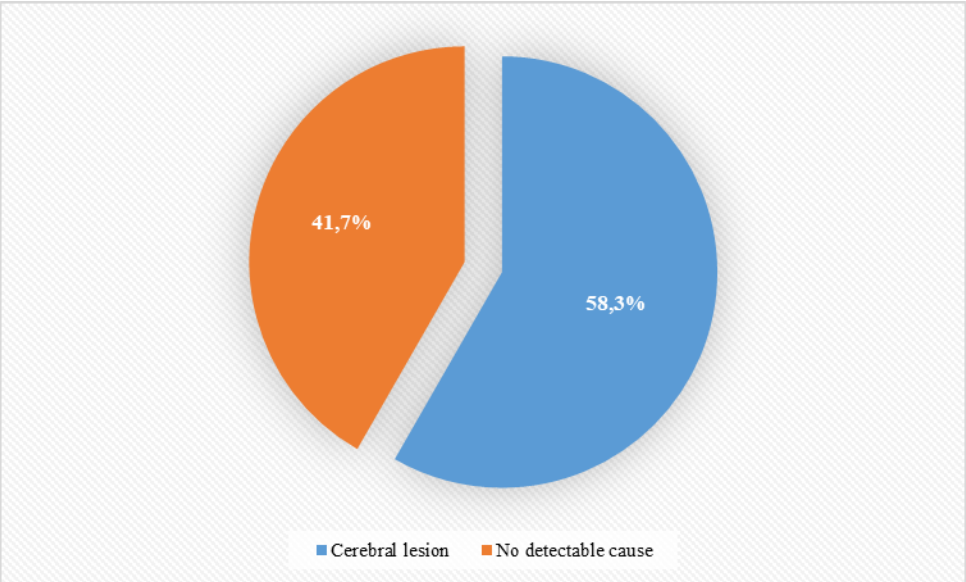

Figure 1. Frequency of cerebral lesion in the study population

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.

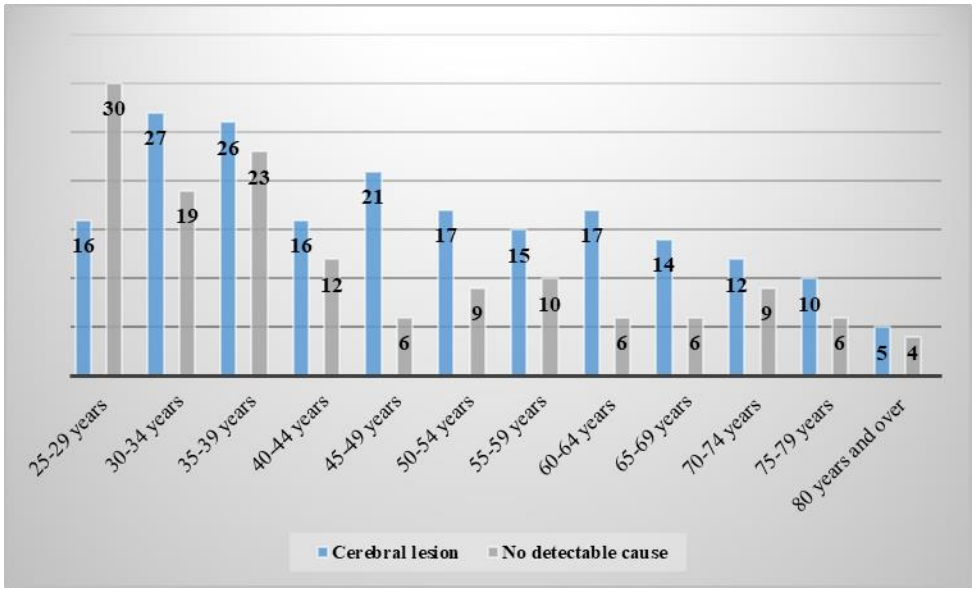

Figure 2. Distribution of cerebral lesion according to age groups

2. Infectious pathology:

Table 3. Distribution of infectious pathology by age group

	CNS Infections
25-29 years	0
30-34 years	3
35-39 years	3
40-44 years	0
45-49 years	1
50-54 years	2
55-59 years	3
60-64 years	1
65-69 years	1
70-74 years	0
75-79 years	0
80 years and over	0
Total	**14**

The distribution by age group shows a predominance of the infectious pathology for the groups of subjects aged (30-34 years, 35-39 years, and 55-59 years).

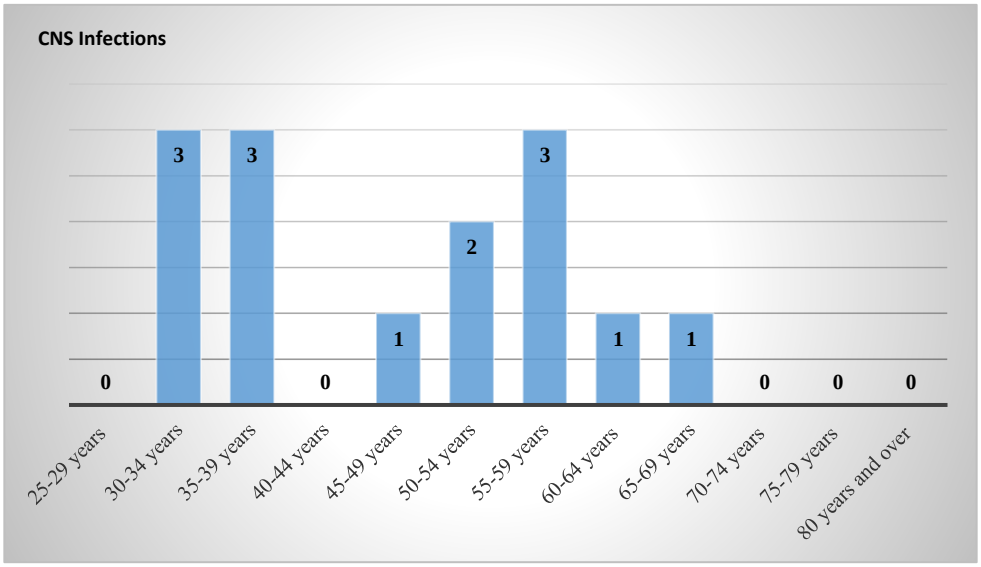

Figure 3. Distribution of infectious pathology according to age groups

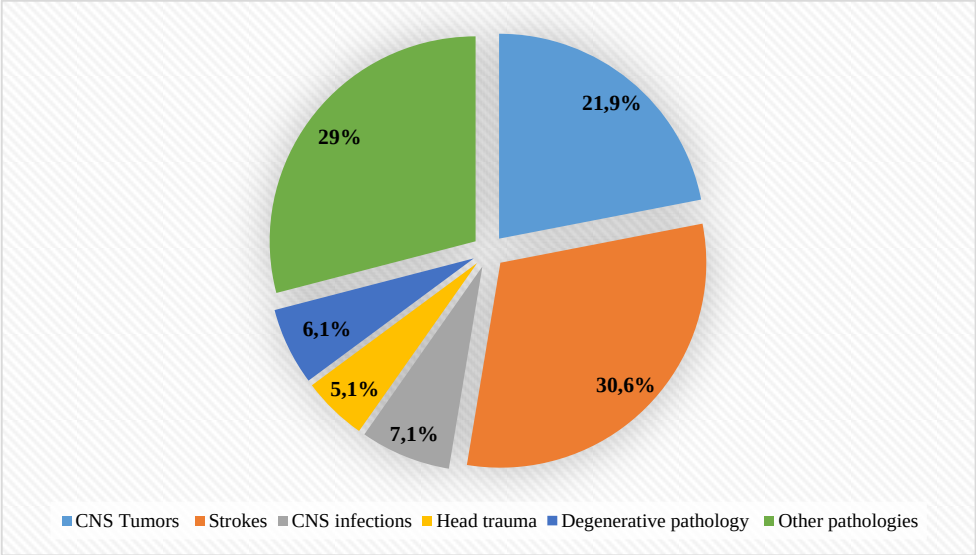

Figure 4. Frequency of infectious pathology compared to other etiologies

In our study, infectious causes represent 7.1% of cases.

Table 3. Distribution of central nervous system infections

	Cases	%
SNC Infections :	**14**	**4**
Meningitis	0	0
Encephalitis	6	3
Meningoencephalitis	5	2,5
Abscess	3	1,5
Total	**196**	**100**

The infectious causes are dominated by encephalitis (6 cases) and meningo-encephalitis (5 cases).

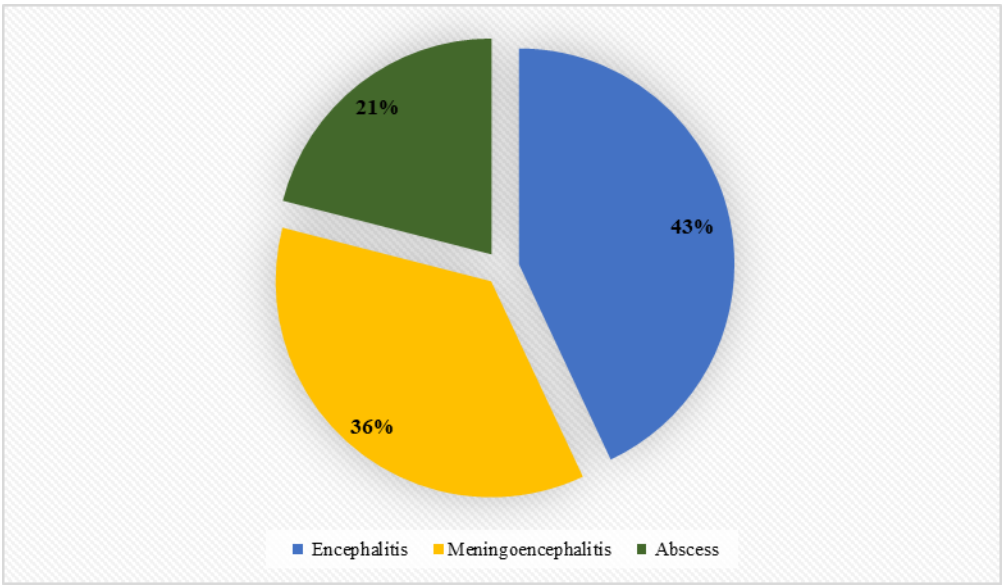

Figure 3. Frequency of central nervous system infections in the study population

We find that encephalitis is the predominant cause in the group of infectious pathologies with the percentage of 43%, followed respectively by meningo-encephalitis 36% and cerebral abscess 21%.

IV. Discussion

A cause was found in 58.3% of cases. Our results agree with data from the literature This situation has been observed in several studies (José Luis Perez Lopez, 1985 [1] - Roberto Suastegui et al, 2009 [13] - Lars Forsgren, 1990 [14]) with respectively 50.8% , 51%, and 49%.

In our study, infectious causes represent 7.1% of cases. We find a percentage of 3% for encephalitis and 2.5% for meningo-encephalitis, and finally brain abscess 1.5%. Infectious pathology is frequent in the group of subjects (30-34 years old) and (35-39 years old). [19]).

The work of Belaidi et al, 1986 [15], shows that the main etiologies are of infectious origin in 20.8% of cases.

Basim Ayaqub and Panayiotopoulos, 1987 [16], reported the main causes including CNS infection in 7% of cases.

Daniel Arbaiza et al, 1995 [17], found an infectious cause in 37% of cases.

In the study by Marcelo Rigatti et al, 1999 [18], among the most frequent etiologies Neurocysticercosis 20%.

The results of the work of Andre Oun et al, 2003 [19], show that central nervous system infections were identified in 4.5%.

David Ortega Rivero et al, 2003 [20], found that infection of the central nervous system represents 15% of cases (neurocysticercosis 6% of cases).

In the work of Christian Napon et al, 2009 [21], we note that infectious pathology was the dominant etiology with the rate of 12.6%. Neurocysticercosis was the most observed aspect (10.8%).

Concerning the etiologies, the analysis by Roberto Suastegui et al, 2009 [13], shows that the primary cause was neurocysticercosis in 21% of cases.

The etiological data concerning the study Ewan Hunter et al, 2012 [22] show that infectious etiologies (neurocysticrosis) represent 15.7% of cases.

The results of Sudhir Chasani et al, 2015 [23], showed that among all the causes of late-onset epilepsy, infections of the central nervous system were the main cause found, representing 39.7%.

Among the central nervous system infections, neurocysticercosis was the leading cause with 46.15%, in the younger age group (21-40 years).The most common cause is neurocysticercosis which accounts for 30.4% of all causes in this age group.

The low frequency of the infectious pathology is not unanimous in the literature, this could be explained by the accumulation of risk factors in certain countries (Neurocysticercosis).

This is how ; Daniel Arbaiza, et al. 1995 [17] found in their studies that infectious causes were the most important 37% of cases (Cysticercosis 24%, Cryptococosis 21%, Cerebral tuberculosis 12%)

The work of Marcelo Rigatti et al, 1999 [18] confirms the place of infectious pathology dominated by Neurocysticercosis in 20% of cases.

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

Study	Country	Infectious pathology
José lwis Perez Lopez, 1985	Spain	0%
Agnete Mouritzen Dam, 1985	Denmark	0%
R.Sridharan et al, 1986	Libya	0%
Basim A.Yakoub et al, 1987	Saudi Arabia	7 %
Anthony Hopkins et al, 1988	United Kingdom	0%
Lars Forsgren, 1990	Sweden	0%
Daniel Arbaiza 1995	Peru	37 % Cysticercosis Cryptococosis Tuberculosis
Lars Forsgren et al, 1996	Sweden	0%
Marcelo Rigatti et al, 1999	Brasil	20 % (Neurocysticercosis)
Andre Oun et al, 2003	Estonia	4.5 %
GCY Fong et al, 2003	Hong Kong	0%
David Ortega Rivero et al, 2003	Ecuador	15 % (Neurocysticercosis 6%)
Christian Napon et al, 2009	Burkina Faso	12.6 % (Neurocysticercosis)
Robero Suastegui et al, 2009	Mexico	21 % (Neurocysticercosis)
Ewan Hunter et al, 2012	Tanzania	15.7 % (Neurocysticercosis)
Sudhir Chasani et al, 2015	India	39.7 %
Our series	**Algeria**	**7.1 %**

V. Conclusion

Etiologically, our study found a percentage of 7.2% of cases for infectious pathology. Infectious pathology is common in the group of subjects (30-34 years) and (35-39 years).

Among the infectious causes, we find a percentage of 3% for encephalitis and 2.5% for meningoencephalitis, and finally cerebral abscess 1.5%.

References

[1]. Weber M. Epilepsie De L'adulte : Etiologie, Diagnostic, Evolution, Pronostic, Traitement. Rev Prat. 1998, Vol. 48 : 1249-55.
[2]. Hauser WA, Annegers JF, Kurland LT. Incidence Of Epilepsy And Unprovoked Seizures In Rochester, Minnesota: 1935-1984. Epilepsia. 1993, Vol. 34(3) :453-68.
[3]. Annegers JF, Dubinsky S, Coan SP, Et Al. The Incidence Of Epilepsy And Unprovoked Seizures In Multiethnic, Urban Health Maintenance Organizations. Epilepsia. 1999, Vol. 40(4) :502–506.
[4]. Gaches J, Malon S. Herpes Simplex Encephalitis In Neuro-Surgery. Electro-Encephalographic Contribution To Early Diagnosis (Author's Transl). Sem Hop. 1982. Vol. 25; 58(12):748-53.
[5]. Persidsky, Y., & Poluektova, L. Immune Privilege And HIV-1 Persistence In The CNS. Immunological Reviews. 2006, Vol. 213(1), 180–194.
[6]. Shiu C, Cunningham CK, Greenough T, Muresan P, Sanchez-Merino V, Carey V, Jackson JB, Ziemniak C, Fox L, Belzer M, Ray SC, Luzuriaga K, Persaud D. Identification Of Ongoing Human Immunodeficiency Virus Type 1 (HIV-1) Replication In Residual Viremia During Recombinant HIV-1 Poxvirus Immunizations In Patients With Clinically Undetectable Viral Loads On Durable Suppressive Highly Active Antiretroviral Therapy. J Virol. 2009, Vol. 83(19) :9731-42.
[7]. Modi G, Modi M, Martinus I, Vangu M. New Onset Seizures In HIV-Infected Patients Without Intracranial Mass Lesions Or Meningitis--A Clinical, Radiological And SPECT Scan Study. J Neurol Sci. 2002, Vol. 15 ; 202 (1-2):29-34.
[8]. Bartolomei F, Pellegrino P, Dhiver C, Quilichini R, Gastaut JA, Gastaut JL. Crises D'épilepsie Au Cours De L'infection Par Le VIH. Cinquante-Deux Observations. Presse Med. 1991, Vol. 20 : 2135-8.
[9]. Edmond K, Clark A, Korczak VS, Sanderson C, Griffiths UK, Rudan I. Global And Regional Risk Of Disabling Sequelae From Bacterial Meningitis: A Systematic Review And Meta-Analysis. Lancet Infect Dis. 2010, Vol. 10 (5):317-28.
[10]. Diagana M, Nsengiyumva G, Tuillas M, Druet-Cabanac M, Bouteille B, Preux PM, Tapie P. Electroencephalograms (EEG) In 250 Patients With Epilepsy In A Cysticercosis Endemic Area In Burundi. Neurophysiol Clin. 2005, Vol. 35(1):1-10.
[11]. Thumasupapong S, Tin T, Sukontason K, Sawaddichi C, Karbwang J. Electroencephalography In Cerebral Malaria. Southeast Asian J Trop Med Public Health. 1995, Vol. 26(1):34-7.
[12]. José Luis Péres Lopez, Jesus Longo, Fernando Quintana, Consuelo Diez And José Berciano. Late Onset Epileptic Seizures. Acta Neurol Scand. 1985, Vol. 72: 380-384.
[13]. Suástegui R, Gutiérrez J, Ramos R, Bouchan S, Navarrete H, Ruiz J, Plascencia N, Jauri S, León C, Castillo V, Ojeda EA. Caracteristicas Clinicas De La Epilepsia De Inicio Tardio En Mexico Al Principio Del Nuevo Milenio: 455 Casos. Revista De Investigacion Clinica. 2009, Vol. 61(5) : 354-363.
[14]. Lars Forsgren. Prospective Incidence Study And Clinical Characterization Of Seizure In Newly Referred Adults. Epilepsia.1990, Vol. 31 (3), 292-301.
[15]. M. Bélaidi, M. Baldy-Moulinier, M. Billard. Anomalies EEG De L'épilepsie Tardive. Rev. E.E.G. Neurophysiol. Clin. 1986, Vol.16, 303-309.
[16]. Basim A Yaqub, Chrysostomos Panayiotopoulos, Mansour Al-Nozha, Wajdi Qreishat, Abdullah Al-Dalaan. Causes Of Late Onset Epilepsy In Saudi Arabia: The Role Of Cerebral Granuloma. Journal Of Neurology, Neurosurgery, And Psychiatry. 1987, Vol. 50 : 90-92.
[17]. Daniel Arbaiza, Patricia Campos. Etiologia De La Epilepsia De Inicio Tardio Un Estudio Prospectivo. Rev. Per. Neurol. 1995, Vol.1(2).
[18]. Marcelo Rigatti, Trevisol-Bittencourt Paulo Cesar. Causas De Epilepsia Tardia Em Uma Clinica De Epilepsia Do Estado De Santa Catarina. Arq Neuropsiquiatr. 1999, Vol. 57(3B): 787-92.
[19]. Andre Oun, Haldre Sulev, Mägi Matt. Prevalence Of Adult Epilepsy In Estonia. Epilepsy Research. 2003, Vol. 52: 233-242.
[20]. David Ortega Rivero, Maria Gabriela Acuna Chong , Natalia Orellana Manzano. Factores Etiologicos Mas Frecuentes De Epilepsia Tardia. Revista Medicina. 2003, Vol. 9(3
[21]. Christian Napon, Yacouba Tamboura, Jean Kabore. Epilepsie Des Sujets De Plus De 14 Ans Au Centre Hospitalier Universitaire De Ouagadougou (Burkina Faso). Epilepsies. 2009, Vol. 21(1) : 93-7.
[22]. Hunter Ewan, Rogathi J, Chigudu S, Jusabani A, Jackson M, Mcnally R, Gray W, Whittaker RG, Iqbal A, Birchall D, Aris E, Walker R. Prevalence Of Active Epilepsy In Rural Tanzania: A Large Community-Based Survey In An Adult Population. Seizure. 2012, Vol. 21(9):691-8.
[23]. Sudhir Chalasani, M. Ravi Kumar. Clinical Profile And Etiological Evaluation Of New Onset Seizures After Age 20 Years. Journal Of Dental And Medical Sciences. 2015, Vol. 14(2) : 97-101.