



Psychological Assessment in Children with Epilepsy

Thesis

Submitted for Fulfilment of Master Degree in Pediatrics

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2020

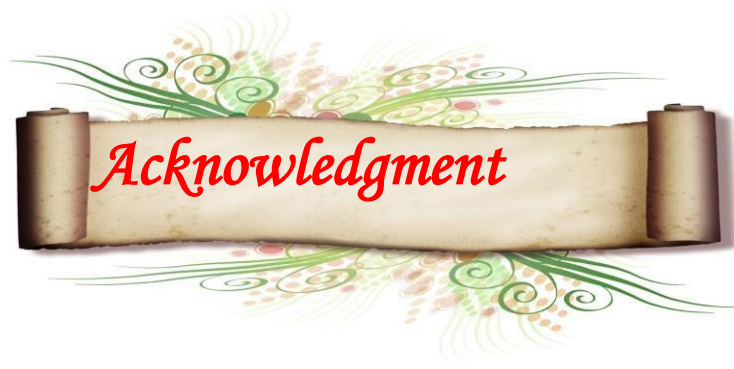


إِنْ أُرِيدُ إِلَّا الْإِصْلَاحَ مَا اسْتَطَعْتُ وَمَا
تَوْفِيقِي إِلَّا بِاللَّهِ عَلَيْهِ تَوَكَّلْتُ وَإِلَيْهِ أُنِيبُ

صدق الله العظيم

سورة مـود: الآية (88).





First and Foremost, I would like to give all my thanks to ALLAH the almighty.

I also extent my thanks and great appreciation to *Prof. Hesham Abd EL-Aziz Elghaiaty* Professor of Pediatrics Faculty of medicine - Benha university, *Dr. Tarek Mahmoud Arafa Khattab* Lecturer of pediatrics Faculty of medicine - Benha university, *Dr. Eman Gamal Abd EL-Rahman Amer* Lecturer of Pediatrics Faculty of medicine - Benha university For giving me the chance of finishing this work under their supervision and giving me much of their effort, work and time.

I also wish to express my thanks to all my patients for their patience and cooperation.

Yousra Mohsen Kamal Ali El-tabakhi

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LIST OF ABBREVIATION

<i>AEDs</i>	anti-epileptic drugs
<i>APA</i>	American Psychiatric Association
<i>BDNF</i>	Brain-derived neurotrophic factor
<i>BECTS</i>	Benign childhood epilepsy with centrotemporal spikes
<i>CBT</i>	Cognitive-behavioral therapy
<i>CI</i>	confidence interval
<i>CNS</i>	central nervous system
<i>COMT</i>	Catechol-O-methyltransferase
<i>COPD</i>	Chronic obstructive pulmonary disease
<i>CP</i>	cerebral palsy
<i>EEG</i>	Electroencephalogram
<i>EPC</i>	Epilepsia partialis continua
<i>FDA</i>	Food and Drug Administration
<i>GABA</i>	gamma-Aminobutyric acid
<i>GLUT-1</i>	Glucose Transporter Protein 1
<i>GTCS</i>	genelazied tonic clonic seizures
<i>Hz</i>	Hertz
<i>ILAE</i>	International League Against Epilepsy
<i>KD</i>	The Ketogenic Diet
<i>MR</i>	mental retardation
<i>OCD</i>	Obsessive compulsive disorder
<i>PET</i>	Positron Emission Tomography
<i>PLXNA2</i>	Plexin A2 Protein
<i>SD</i>	standerd deviation
<i>SMA</i>	Supplementary motor area
<i>SPECT</i>	Single-photon emission computed tomography

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Introduction

The epilepsies are chronic neurological disorders in which clusters of nerve cells, or neurons, in the brain sometimes signal abnormally and cause seizures. **Epilepsy is** a disorder of the brain **defined by** any of the following conditions: At least two unprovoked (or reflex) seizures occurring more than 24 hours apart ,One unprovoked (or reflex) seizure and a probability of further seizures similar to the general recurrence risk (at least 60%) after two unprovoked seizures, occurring over the next 10 years.(*Schraegle, et al., 2017*)

During a seizure, many neurons fire (signal) at the same time – as many as 500 times a second, much faster than normal. **epileptic seizure**, is defined as period of symptoms due to abnormally excessive or synchronous neuronal activity in the brain. Outward effects vary from uncontrolled shaking movements involving much of the body with loss of consciousness (tonic-clonic seizure), to shaking movements involving only part of the body with variable levels of consciousness (focal seizure), to a subtle momentary loss of awareness (absence seizure).[3] Most of the time these episodes last less than 2 minutes and it takes some time to return to normal. Loss of bladder control may occur.(*Delpisheh et al., 2014*).

Epilepsy affects patients of all ages, races, and ethnic backgrounds. According to the National Institute of Neurological Diseases and Stroke (NINDS), about 2.3 million adults and more than 450,000 children and adolescents in the United States have epilepsy(*Camfield, et al., 2015*).

Children with epilepsy also have a higher risk of developing depression and/or attention deficit hyperactivity disorder compared with their peers. Behavioral problems may precede the onset of seizures in some children. They are especially vulnerable to the emotional problems caused by ignorance or the lack of knowledge among others about epilepsy (*Pimentel et al.,2015*).

International classification of epileptic seizures

- PARTIAL SEIZURES (seizures beginning locally)
- Simple partial seizures (consciousness not impaired)
 1. With motor symptoms
 2. With somatosensory or special sensory symptoms
 3. With autonomic symptoms
 4. With psychic symptoms
- B. Complex partial seizures (with impairment of consciousness)
 1. Beginning as simple partial seizures and progressing to impairment of consciousness
 - (a) With no other features
 - (b) With features as in A 1–4
 - (c) With automatisms
 2. With impairment of consciousness at onset
 - (a) With no other features

(b) With features as in A 1–4

(c) With automatisms

C. Partial seizures secondary generalized

II. GENERALIZED SEIZURES (bilaterally symmetrical and without local onset)

A. 1. Absence seizures

2. Atypical absence seizures

B. Myoclonic seizures

C. Clonic seizures

D. Tonic seizures

E. Tonic-clonic seizures

F. Atonic seizures

III. UNCLASSIFIED EPILEPTIC SEIZURES (*Afzal, et al., 2017*)

Diagnosis of epilepsy is dependent on history, physical and neurologic examination, laboratory testing as indicated, and electroencephalography and neuroimaging findings. The history should include events directly preceding the seizure, number of seizures in the past 24 hours, length and description of the seizure, focal aspects, and length of the postictal period. The need for laboratory testing is based on clinical context and may include blood glucose, blood counts, electrolyte panels (particularly sodium), lumbar puncture in febrile patients, and urine toxicology. Electroencephalography should be used to confirm, but not to exclude, a diagnosis of epilepsy (*Samia et al., 2019*).

Treatment of epilepsy is include pharmacological and non-pharmacological treatment. **Pharmacotherapy** DRUG SELECTION Choice of AEDas(**carbamazepine, clonazepam, lacosamide, levetiracetam, lamotrigine, oxcarbazepine,.....**) should be individualized in consultation with a neurologist, and based on factors such as seizure type, presence of an epilepsy syndrome, other medications, comorbidities, lifestyle, and patient preference. Quality of evidence and treatment recommendations vary among seizure types. **Non pharmacological** treatment of epilepsy includes surgery, vagal nerve stimulation, ketogenic diet, and other alternative/complementary therapies, e.g., yoga, Ayurveda, electroencephalography (EEG) biofeedback technique, aerobic exercise, music therapy, transcranial magnetic stimulation, acupuncture, and herbal remedies (traditional Chinese medicine) (*Plavin et al.,2019*).

Aim of the work

The aim of the current work is to study the presence, possible factors related and consequences of psychological disorders in children with epilepsy.

Epilepsy

ILAE pediatric definition of epilepsy

In 2005, the **International League Against Epilepsy (ILAE)** released a conceptual definition of seizures and epilepsy, followed by an operational (practical) definition in 2014(*Falco et al., 2018*).

Epilepsy exists after at least one unprovoked seizure, when there is high risk for another, although the actual required risk is subject to debate. With a single unprovoked seizure, risk for another is 40–52%. With two unprovoked non-febrile seizures, the chance by 4 years of having another is 73%, with a 95% confidence interval (CI) of 59–87%, subsequently herein portrayed as approximately 60–90% (*Tinuper et al., 2016*).

The –two unprovoked seizure|| definition of epilepsy has served us well, but it is inaccurate in some cases. A patient might present with a single unprovoked seizure after a remote brain insult, such as a stroke, central nervous system (CNS) infection, or trauma (*Morse et al., 2016*)

In order to bring the practical clinical definition of epilepsy into concordance with how epileptologists think about epilepsy, the ILAE Task Force recommends broadening the definition of epilepsy and The Task Force also added a time limit to the definition. So, practical clinical definition of epilepsy of ILAE :

Epilepsy is a disorder of the brain defined by any of the following conditions. (*Sarecka et al., 2018*.)

At least two unprovoked (or reflex) seizures occurring more than 24 hours apart

One unprovoked (or reflex) seizure and a probability of further seizures similar to the general recurrence risk (at least 60%) after two unprovoked seizures, occurring over the next 10 years.

Diagnosis of an epilepsy syndrome.

Epilepsy is considered to be resolved for individuals who had an age-dependent epilepsy syndrome but are now past the applicable age or those who have remained seizure-free for the last 10 years, with no seizure medicines for the last 5 year

Status epileptics:

Status epilepticus could be defined as continuous seizure activity or recurrent seizures without recovery of consciousness for 30 minutes. The new definition is single seizure or recurrent seizure lasting for 5 minutes (*Hirsch et al., 2018*).

Epidemiology of epilepsy:

Ten and half million children worldwide are estimated to have active epilepsy Over the past fifteen years (*Camfield et al., 2015*.)

Age-Specific Incidence

In all studies providing separate information regarding the incidence by detailed age groups, the incidence is highest in the first year of life. The most recent studies report incidence in the first year of life of about 100 per 100,000 children. Incidence falls after age 1 year, although the rate of the fall varies. In developing countries, incidence may be higher in adolescence than in early childhood, whereas in developed countries,

incidence is lower in the second decade of life than in the first decade (Moshé et al., 2015).

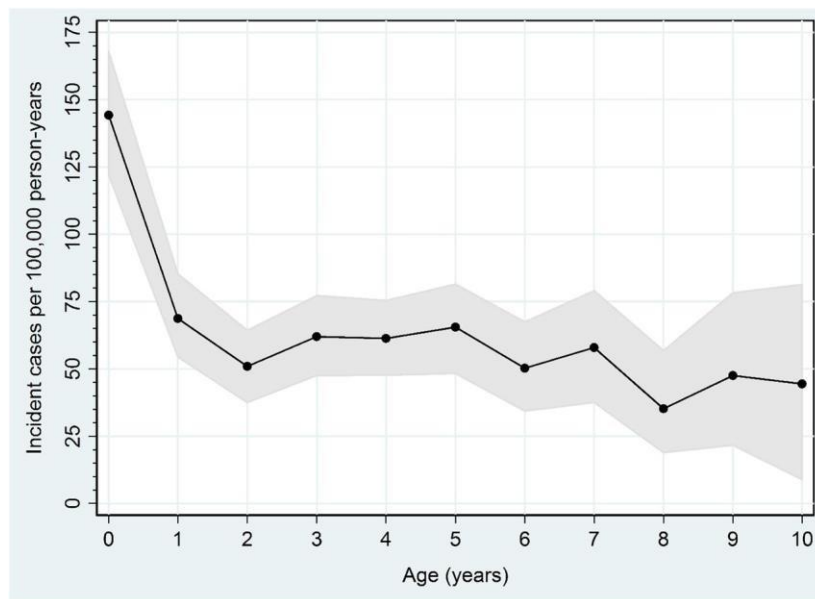


Fig (1): Gender-specific Incidence

Gender-specific Incidence

For most recent studies that report gender, gender-specific incidence in children is higher in males, although seldom significantly so. This seems to be true regardless of study definition (*Ackermann et al., 2019*).

Race

The incidence in African American children was higher than that in Caucasian children. This study did not control for socioeconomic status (*Camfield et al., 2015*)

Seizure Type

Most recent studies of epilepsy in developed countries report a slight predominance of partial seizure disorders over generalized seizure disorders. One must consider the age distribution being studied as well as

seizures classified, because generalized onset seem to predominate in the first year of life, after which partial seizures seem to predominate (*Ackermann et al., 2019*).

Etiology

Between 60% and 80% of all new cases in children have no obvious antecedent to explain the condition. This is true even with the use of magnetic resonance imaging (*Al-Qudah et al., 2017*).

A small proportion of new cases can be attributed to trauma, infection, postnatal vascular lesions, or CNS degenerative conditions, About 20% of cases are associated with neurologic handicaps presumed present from birth, mental retardation (MR), cerebral palsy (CP), or a combination (*Dilena et al., 2019*).

Familial (genetic) predisposition certainly plays a role in the risk for developing epilepsy the offspring or sibling of a person with epilepsy has a threefold increase in risk of developing epilepsy. Although there is no evidence of an association between adverse pre- and perinatal factors and the development of epilepsy. The concept of —birth trauma|| or of

pregnancy complications|| being a cause of epilepsy has not been supported in a variety of studies performed over the past 20 years. From a similar standpoint, febrile seizures are not –causal|| for epilepsy. They are more likely a marker for a preexisting susceptibility (*Sands et al., 2017*).

Classification& Clinical picture of epilepsy:

❖ according seizures category:

I. Focal Seizures (previously known as partial or local seizures)

A. Simple focal seizures (consciousness not impaired) 1- With motor symptoms

2- With somatosensory or special sensory symptoms 3- With autonomic symptoms.

3- With psychic symptoms

B. Complex focal seizures (with impairment of consciousness)

1) Beginning as simple focal seizures and progressing to impairment of consciousness

a) With no other features

b) With features as in A.1-4

c) With automatisms

2) With impairment of consciousness at onset

d) With no other features.

e) With features as in A.1-4.

f) With automatisms.

C. Focal seizures secondarily generalized

II. Generalized Seizures (convulsive or non-convulsive)

1. Absence seizures

2. Atypical absence seizures

3. Myoclonic seizures
4. Clonic seizures
5. Tonic seizures
6. Tonic-clonic seizures
7. Atonic seizures

III. Unclassified Epileptic Seizures (Scheffer et al.,2017)

Other classification by ILAE according seizures type:

I. Self-limited epileptic seizures

A. Generalized onset:

1. Seizures with tonic and/or clonic manifestations

- a. Tonic-clonic seizures
- b. Clonic seizures
- c. Tonic seizures

2.Absences

- a. Typical absences
- b. Atypical absences
- c. Myoclonic absences

3.Myoclonic seizure types

- a. Myoclonic seizures

- b. Myoclonic astatic seizures
- c. Eyelid myoclonia

4.Epileptic spasms

1.Atonic seizures

B. Focal onset:

2.Local

- a. Neocortical
- b. Without local spread
- c. With local spread
- d. Hippocampal and parahippocampal.

1. With ipsilateral propagation to:

- a. Neocortical areas
- b. Limbic areas

2. With contralateral spread to:

- a. Neocortical areas. Limbic areas

3. Secondarily generalized

- a. Tonic-clonic seizures
- b. Absence seizures
- c. Epileptic spasms
- C. Neonatal seizures

II. Status epilepticus

- A. Epilepsia partialis continua (EPC)
- B. Supplementary motor area (SMA)
- C. Aura continua
- D. Dyscognitive focal
- E. Tonic-clonic
- F. Absence
- G. Myoclonic
- H. Tonic

I. Subtle (Boride et al., 2018)

Typical seizures go through 3 phases:

Preictal:

About 20% of individuals with epilepsy experience a prodromal phase – patients feeling or sensation that can occur several hours or even days before the actual seizure. The most common symptoms of a prodrome include confusion, anxiety, irritability, headache, tremor, and anger or other mood disturbances. The prodromal period may serve as a warning sign of seizure onset for those who experience it, but, unlike an aura this phase is not part of the seizure (*Tran et al.,2017*).

Ictal:

The earliest sign of seizure activity is an *aura*. Although it has traditionally been thought of as a warning of an on-coming seizure, an

aura is actually the earliest sign of seizure activity and the beginning of the *ictal phase*. The ictal phase includes the time between the beginning (aura, if present) and the end of the seizure. Like the prodrome, not everyone with epilepsy has auras. For those who do, the specific symptoms vary depending on the seizure type, severity and affected brain region (*Tran et al.,2017*)

Post ictal:

Following a seizure, there is a recovery period called the post-ictal phase. Some people recover immediately, while others require minutes to days to feel like they're back at their baseline. The length of the post-ictal phase depends directly on the seizure type, severity, and region of the brain affected. Typical symptoms include the following:

Drowsiness – Confusion - Memory loss –Nausea - General malaise

(*Tran et al.,2017*).

Epileptic syndromes

International Classification of Epilepsies and Epileptic Syndrome:

1.0 Localization-related epileptic syndromes

1.1 Idiopathic (with age-related onset)

1.2 Symptomatic

1.3 Unknown as to whether the syndrome is idiopathic or symptomatic.

2.0 .Generalized epilepsies and syndromes:

2.1 Idiopathic (with age-related onset-listed in order of age) Benign

neonatal familial convulsions Benign neonatal convulsions Benign myoclonic epilepsy in infancy Childhood absence epilepsy (pyknolepsy) Juvenile absence epilepsy Juvenile myoclonic epilepsy (impulsive petit mal) Epilepsy with grand mal (GTCS) seizures on awakening Other generalized idiopathic epilepsies, if they do not belong to one of the above syndromes, can still be classified as generalized idiopathic epilepsies.

2.2 Cryptogenic (in order of age) West syndrome (infantile spasms, Blitz-Nick-Salaam Krampfe) Lennox-Gastaut syndrome Epilepsy with myoclonic-astatic seizures Epilepsy with myoclonic absences

2.3 Symptomatic

2.3.1 Nonspecific etiology Early myoclonic encephalopathy.

2.3.2 Specific syndromes Epileptic seizures may complicate many disease states. Under this heading are included those diseases in which seizures are a presenting or predominant feature.

3.0 Epilepsies and syndromes undetermined whether focal or generalized

3.1 With both generalized and focal seizures Neonatal seizures Severe myoclonic epilepsy in infancy Epilepsy with continuous spike-waves during slow wave sleep Acquired epileptic aphasia (Landau-Kleffner syndrome) **3.2** Without unequivocal generalized or focal features

4.0 Special syndromes

4.1 Situation-related syndromes (Gelegenheitsanfälle) Febrile convulsions Isolated seizures or isolated status epilepticus Seizures

occurring only when there is an acute metabolic or toxic event due to, for example, alcohol, drugs, eclampsia, non ketogenic hyperglycemia (*Pearl et al.,2018*).

Every type of epileptic syndrome has its own special character so in brief we lustrate most common types:

Febrile convulsion:

A febrile seizure is a convulsive episode occurring in association with an acute febrile illness. This is actually a subcategory of acute symptomatic seizure, differing only in that all children are exposed to the risk factor, between 2% and 4% of all children can be expected to experience a convulsion during a febrile illness by the age of 5 years, but there is striking variation in the frequency of occurrence of febrile convulsions worldwide. It is possible that a selective genetic predisposition for febrile convulsions most contemporary studies segregate febrile seizures from epilepsy, but a few series have included selected cases, primarily those with -complex|| features. Since such studies comprise 20% to 30% of all febrile convulsion cases, their inclusion may double the apparent incidence or prevalence of unprovoked seizures or epilepsy (*Hon et al., 2018*).

Benign Childhood Epilepsy with Centrotemporal Spikes Benign childhood epilepsy with centrotemporal spikes is a syndrome of brief, simple, focal, hemifacial motor seizures, frequently with somatosensory symptoms, which have a tendency to evolve into GTCS. Both seizure types are often related to sleep. Onset is between 3 and 13 years of age (peak 9–10), and recovery before ages 15 to 16. Genetic predisposition is frequent, and there is male predominance. The

EEG has blunt high voltage centrotemporal spikes, often followed by slow waves that are activated by sleep (*Dryzalowski et al., 2018*).

Benign Neonatal Familial Convulsions

Benign neonatal familial convulsions are rare, dominantly inherited disorders manifesting mostly on the second and third days of life, with clonic or apneic seizures and no specific EEG criteria. History and investigations reveal no etiological factors. Approximately 14 percent of these patients later develop epilepsy (*Zeng et al., 2018*).

Benign Myoclonic Epilepsy in Infancy

Benign myoclonic epilepsy in infancy is characterized by brief bursts of generalized myoclonus that occur during the first or second year of life in otherwise normal children who often have a family history of convulsions or epilepsy. The EEG shows generalized spike waves occurring in brief bursts during the early stages of sleep. These attacks are easily controlled by appropriate treatment. They are not accompanied by any other types of seizures, although GTCS may occur during adolescence. The epilepsy may be accompanied by a relative delay of intellectual development and minor personality disorders (*Caraballo et al., 2013*).

Childhood Absence Epilepsy (Pyknolepsy)

This syndrome of childhood absence epilepsy (pyknolepsy) occurs in children of school age (peak manifestation, age 6 to 7) with a strong genetic predisposition in otherwise normal children. It appears more frequently in girls than in boys and is characterized by very frequent (several to many per day) absences. The EEG reveals bilateral, synchronous symmetrical spike waves, usually three per second, on a

normal background activity. GTCS often develop during adolescence. Otherwise, absences may remit or more rarely, persist as the only seizure type (*Scheffer et al., 2019*).

Juvenile Myoclonic Epilepsy (Impulsive Petit Mal)

Juvenile myoclonic epilepsy appears around puberty and is characterized by seizures with bilateral, single or repetitive arrhythmic, irregular myoclonic jerks, predominantly in the arms. Some patients may suddenly fall from a jerk. No disturbance of consciousness is noticeable. The disorder may be inherited, and sex distribution is equal. Often, there are GTCS and, less often, infrequent absences. The seizures usually occur shortly after awakening and are often precipitated by sleep deprivation. Interictal and ictal EEG have rapid, generalized, often irregular spike waves and polyspike waves; there is no close phase correlation between EEG spikes and jerks. Frequently, the patients are photosensitive. Response to appropriate drugs is good (**Sánchez et al., 2012**)

West Syndrome (Infantile Spasms, Blitz-Nick-Salaam Krampfe)

West syndrome usually consists of a characteristic triad: infantile spasms, arrest of psychomotor development, and hypo arhythmia, although one element may be missing Spasms may be flexor, extensor, lightning, or nods but most commonly are mixed Onset peaks between 4 and 7 months and is always before 1 year. Boys are more commonly affected, and the prognosis is generally poor. West syndrome may be separated into two groups. The symptomatic group is characterized by the previous existence of brain damage signs (psychomotor retardation, neurologic signs, radiologic signs, or other types of seizures) or by a known cause. The smaller, idiopathic group is characterized by the absence of previous signs of brain damage and of known cause. The

prognosis is dependent upon etiology and response to treatment; a subgroup of infants with no prior brain damage and good response to treatment may have an excellent prognosis with normal development and no recurrence of seizures. Patients with symptomatic etiology and incomplete response to treatment do poorly and often have mental retardation and persistent seizure (*Song et al., 2017*).

Diagnosis

APPROACH TO THE PEDIATRIC EPILEPSY PATIENT

A. History

A complete medical history can provide clues as to whether the patients have generalized or localized epilepsy or syndrome. A careful history can further help the physician to further characterize the syndrome.(*Wilmshurst et al., 2015*)

1. Age of Onset Generalized Epilepsies

The age of onset varies in different types of generalized epilepsies. Childhood absence epilepsy is seen in children from age 2 to 13 years of age with the peak incidence between 6 and 7 years old. Benign neonatal familial convulsions are seen in the first week of life and benign myoclonic epilepsy is seen between 1 and 2 years of age. Benign neonatal convulsions are also known as fifth day fits. Lennox Gastaut syndrome begins between the ages of 1 and 6 years of age and persists during adulthood. Progressive myoclonic epilepsies begin in childhood but some forms do occur during adolescence. Juvenile absence epilepsy and Juvenile myoclonic epilepsy as the names suggest begin during adolescence between 12 and 18 years of age. Juvenile absence epilepsy can be seen in children as early as 10 years of age. Epilepsy with

generalized seizures on awakening is seen in children between 8 and 15 years of age. (Cornet et al., 2018)

Localization Related Epilepsies

Mesial temporal epilepsy with hippocampus sclerosis, familial temporal lobe epilepsy and autosomal dominant frontal lobe epilepsy are seen in childhood or early adolescence. Idiopathic childhood occipital epilepsy is seen in children from 3 to 16 years of age. Panayiotopoulos syndrome is seen between 1 and 14 years of age. The Panayiotopoulos syndrome occurs in children around 1 and 14 years of age. (Cornet et al., 2018)

2. Childhood Medical History

Birth history is important in localization related epilepsies. On careful interview of the parents or the caregivers, there is usually a history of febrile seizures, meningitis, stroke, head Pediatric Epilepsy 147 trauma, encephalitis, developmental delay or family history of seizures. This provides important information in case a surgical option is pursued later. (Holthausen et al., 2013)

3. Seizure Semiology Generalized Epilepsies

Careful history is important regarding the seizure semiology. **In Juvenile myoclonic epilepsy**, the adolescent gives a typical history of being -clumsy|| in the morning. Further history shows the presence of myoclonic jerks.

Generalized tonic clonic seizures are triggered by stress, alcohol intake, photosensitivity, sleep deprivation and fatigue. Generalized tonic

clonic seizures are also seen within 30 minutes of awakening with epilepsy with generalized tonic clonic seizures on awakening.

Complex partial seizures are also seen such as Lennox Gastaut syndrome, progressive myoclonic epilepsies, and west syndrome. (Eschbach et al., 2018)

Localization related Epilepsies

Focal motor seizures localized to the face and upper limbs with preserved consciousness are seen with **Benign childhood epilepsy with centrotemporal spikes** The seizures seen with this syndrome are usually nocturnal in nature and duration is 2 minutes.

Autonomic seizures are seen with Panayiotopoulos syndrome. These seizures begin with nausea, vomiting, flushing, hypersalivation, and urinary incontinence without alteration of awareness. The initial phase is followed by unresponsiveness and prolonged focal seizure which may last for 30 minutes (**autonomic status epilepticus**). (Celano., 2017)

Visual seizures are seen with idiopathic childhood occipital epilepsy with and without secondary generalization.(**Holthausen et al., 2013**)

Temporal lobe epilepsy with hippocampal sclerosis results in focal seizures. The seizures begin as simple partial seizures (aura) without alteration of awareness. The simple partial seizures are brief and are characterized by déjà vu sensation, rising epigastric sensation, intense fear, gustatory or olfactory sensations. These are followed by complex partial seizures with alteration of consciousness, oroalimentary automatisms, dystonic posturing of the limb and limb automatisms which include fumbling of the fingers and picking hand movements. Some

patients try to move aimlessly and become combative if attempts are made to restraint them. These seizures may also secondarily generalize. The duration of these seizures is 1 to 3 minutes followed by postictal confusion and amnesia which may range from 5-10 minutes to half an hour. (Nickels et al., 2018)

Lateral temporal seizures are seen with familial temporal lobe epilepsy. The auditory hallucinations (simple partial seizures) characterized by tinnitus or buzzing are typical for lateral temporal seizures followed by alteration of awareness (complex partial seizures). (Wilmshurst et al.,2015)

Frontal lobe epilepsy is characterized by brief nocturnal seizures with bizarre body movements. Frontal lobe seizures rapidly generalize and there is no postictal confusion. (Eschbach et al., 2018)

Supplementary motor area seizures show asymmetric posturing of the limbs, brief in onset and consciousness is preserved. (Eschbach et al., 2018)

Parietal lobe seizures begin with a simple partial seizure with somatosensory sensations which includes tingling, numbness, pain or shock like sensation contralateral to the seizure focus. (Wilmshurst et al.,2015)

4. Seizure Frequency

The parents and caregivers are advised to maintain a seizure diary. Appropriate medical management is possible only if the frequency of seizures is known. (Nickels et al., 2018).

5. Failed Anticonvulsants

A careful history is required regarding failed anticonvulsants before making any adjustments in the medication regimen. Patients failing more than two anticonvulsants and are still having seizures fulfill the criteria for intractability. These patients may prove to be good surgical candidates. (**Holthausen et al., 2013**)

B. Physical Examination

Careful complete physical examination is extremely important in the determination of a particular epilepsy syndrome based on the associated neurological findings. (**Patel et al., 2016**)

Focal neurological deficits are seen in localization related syndromes due to childhood strokes or tumors. (**Patel et al., 2016**)

Mental retardation is seen with Lennox –Gastaut syndrome, West syndrome, and Progressive myoclonic epilepsies which include Unverricht-Lundborg disease, Lafora’s disease, Neuronal Ceroid Lipofuscinosis and myoclonic epilepsy with ragged red fibres. (**Madaan et al., 2019**)

The neurological manifestations seen in Unverricht-Lundborg disease includes ataxia, intention tremor, dysarthria, and progressive decline in mental status. The myoclonus can be so severe that the patient develops difficulty with gait, speech and swallowing. (**Stafstrom et al., 2015**)

Lafora’s disease is characterized by visual loss, apraxia, dementia and ataxia. Visual loss, ataxia, dysarthria, severe myoclonus and extrapyramidal symptoms are seen in different types of neuronal ceroid

lipofuscinoses. Dementia, ataxia, myopathy, lactic acidosis, dystonia neuropathy, optic atrophy and lower motor neuron abnormalities are seen in Myoclonic epilepsy with ragged red fibres. Café au lait spots are seen with neurofibromatosis. Tuberous sclerosis can be diagnosed with careful physical examination based on the presence of adenoma sebaceum and hypomelanotic spots. **(Patel et al., 2016)**

C. Diagnostic Tests EEG

The EEG is an important diagnostic tool in epilepsy. The EEG test gives us a clue that the syndrome is either localized or generalized. **(Sheth., 2019)**

Localization Related Epilepsies as **Benign childhood epilepsy with centrotemporal spikes (BECTS)** is named so because of the characteristic electroencephalographic findings. Spikes are seen in the central and temporal regions which becomes more prominent during drowsiness and sleep. These spikes have characteristic morphology and are identified by the presence of a polyphasic dipole tangential to the rolandic region. **(Sheth., 2019)**



Fig.(2): EEG of Benign childhood epilepsy with centrotemporal spikes (BECTS)

- In Panayiotopolous syndrome, spikes are similar in morphology to BECTS but occipital spikes predominate in this syndrome. **(Denis et al.,2019)**
- High amplitude occipital spikes are seen idiopathic childhood epilepsy. **(Denis et al.,2019)**

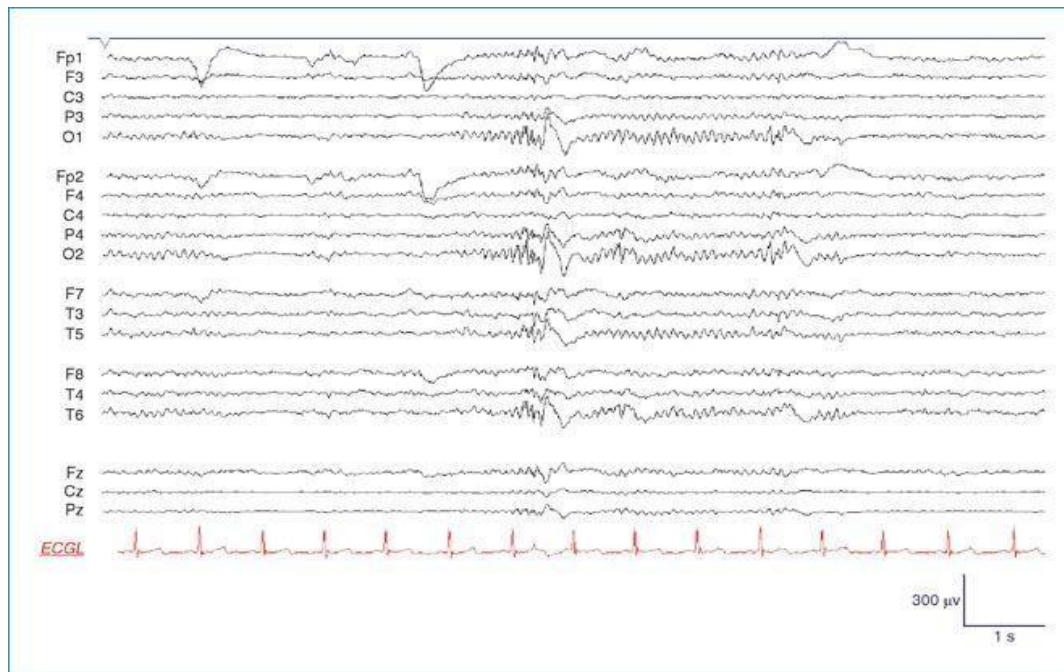


Fig. (3): EEG of idiopathic childhood epilepsy.

Mesial temporal epilepsy with hippocampal sclerosis and familial temporal lobe epilepsy are characterized by focal slowing and spikes in the temporal regions. Ictal EEG shows rhythmic 4-5 Hz theta activity in the anterior mid temporal regions. **(Peter et al., 2013)**

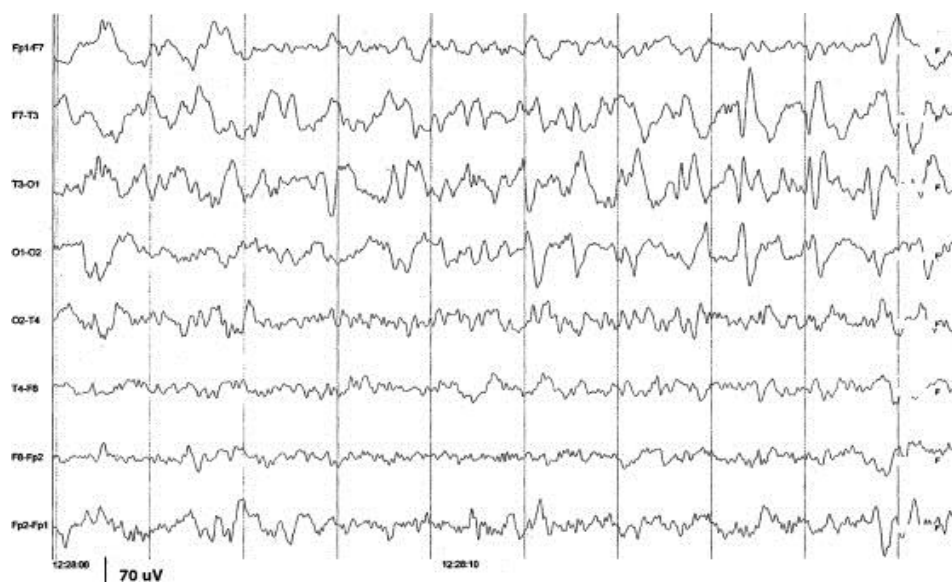


Fig. (4): EEG of Mesial temporal epilepsy with hippocampal sclerosis

The EEG is normal in frontal lobe epilepsies. **(Peter et al., 2013)**
Generalized Epilepsies Generalized discharges are seen in generalized epilepsies but the patterns differ depending on the type of syndrome. Childhood absence seizures are characterized by 2-4 Hz generalized frontally dominant spikes and wave discharges. **(Denis et al.,2019)**

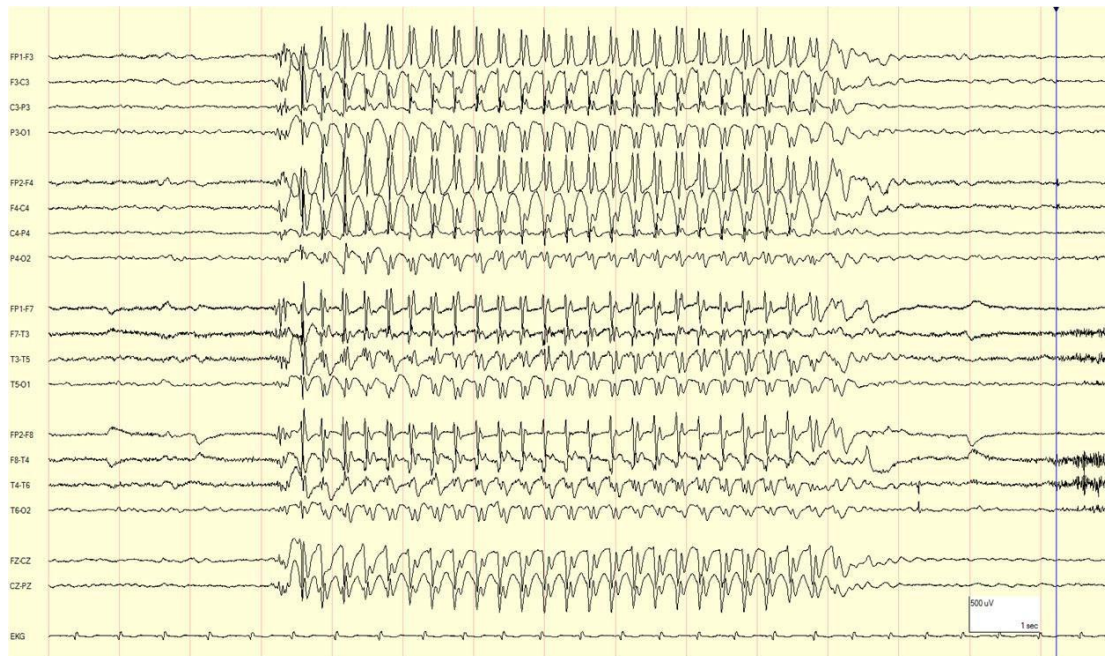


Fig. (5): EEG of Childhood absence seizures

- In Juvenile myoclonic epilepsy, 4-6 Hz frontally dominant spikes and polyspikes and wave discharges are seen. **(Koutroumanidis et al.,2017)**
- Generalized polyspike discharges are seen with myoclonic jerks on EEG in Benign myoclonic epilepsy of infancy. **(Isnard et al., 2018)**

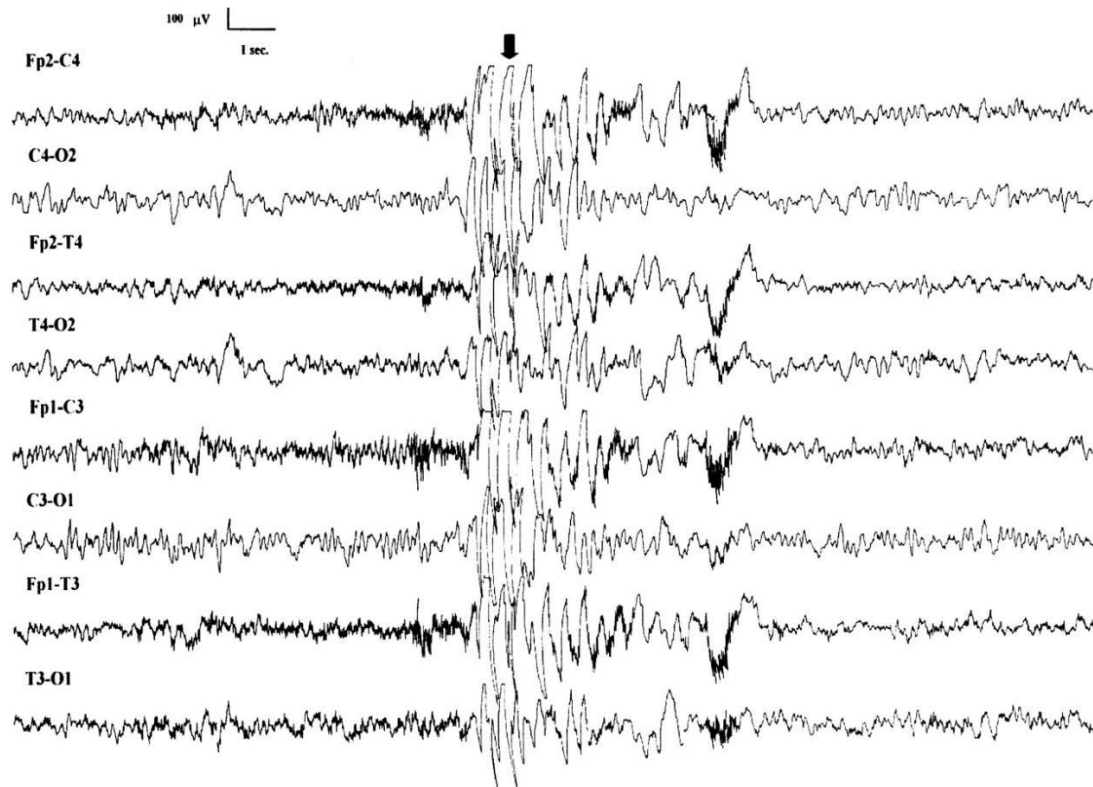


Fig. 1. Patient 2: the ictal EEG shows ~ 3 Hz generalized high-amplitude epia and wave discharges synchronous with bilateral arm myoclonias

Fig. (6): EEG of Benign myoclonic epilepsy of infancy

- Lafora body disease is characterized by generalized epilepsy with diffuse slowing in the earlier stage followed by extremely disorganized background with continuous multifocal epileptiform discharges. (Duez et al., 2019)
- EEG shows generalized epileptiform discharges in neuronal ceroid lipofuscinoses. (Isnard et al., 2018)

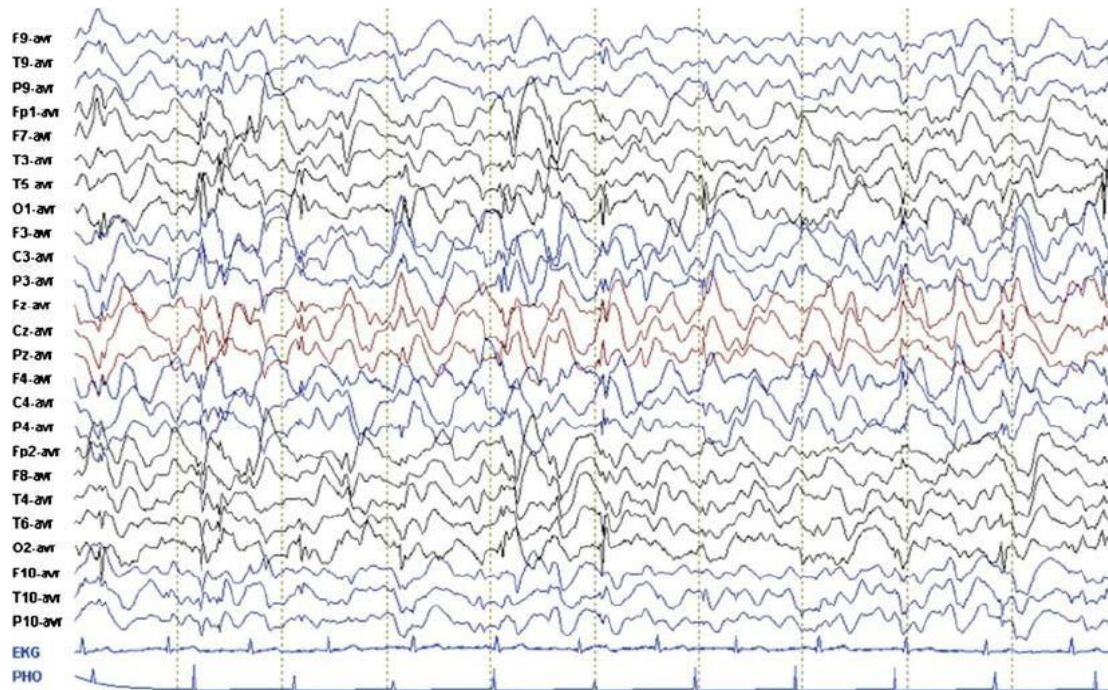


Fig. (7): EEG of neuronal ceroid lipofuscinoses

- Lennox –Gastaut syndrome has a typical EEG pattern characterized by the presence of generalized frontally dominant slow wave discharges in the range of 1.5 -2.5 Hz. (Koutroumanidis et al.,2017)

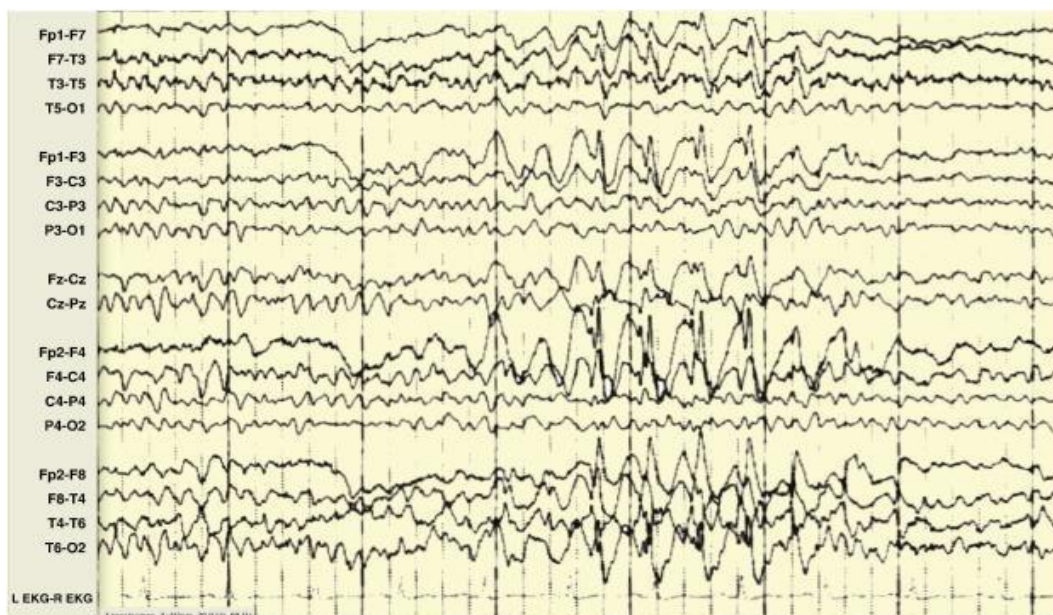


Fig. (8): EEG of Lennox –Gastaut syndrome Ambulatory EEG

Ambulatory EEG is important to rule out spells which are suspicious for seizures by the presence or absence of epileptiform discharges. Non-epileptic spells are diagnosed by the absence of epileptiform abnormalities. Patients with diagnosed epilepsy can also benefit from this test as this may ascertain the frequency of seizures if the parents or caregivers are not sure. This will help in the appropriate adjustment of anticonvulsant medications. (Mountz et al.,2017)

Long Term Video EEG Monitoring in the Epilepsy Monitoring Unit Video- EEG:

Monitoring is essential for localization and characterization of seizures for the epilepsy surgery workup. The advantage of this test over ambulatory EEG is that the patients can be withdrawn from their anticonvulsants so that adequate and reliable data are available to accurately diagnose the type of epilepsy and to rule out non-epileptic spells. The goal is to capture at least 4 to 5 seizures to determine the seizure onset. The EEG data under medication withdrawal determines the extent of interictal epileptiform abnormalities and ictal localization of the seizure focus. The semiology of seizures is determined by the video EEG monitoring. The localization of seizures determine the specific semiology.(Wilmshurst et al.,2017)

Intracranial EEG

Intracranial EEG is required in case of non localization of seizure onset on scalp recordings. Subdural grids, subdural strip and depth electrodes are used for this purpose for precise localization of the seizure focus. Intracranial EEG is also important in extratemporal and dominant hemisphere epilepsy cases to map the language, memory and frontal cortices to minimize deficits post surgery. The contacts on the subdural

grid electrodes are stimulated for functional brain mapping. (Ahmed et al.,2018)

Imaging Tests

Magnetic Resonance Imaging (MRI)

MRI Brain is the most important tool to detect the seizure focus and to rule out structural abnormalities. Special seizure protocols are used in the epilepsy centers to detect these lesions. T1 sequences with thin cuts are used to detect the lesions like cortical dysplasias, hamartomas, polymicrogyria, schizencephaly, arteriovenous malformations, cavernous hemangiomas and low grade tumors seen in younger children. (Duez et al ., 2019)

SPECT:

The SPECT scan measures the distribution of radioactive isotopes in the brain. There are two types of SPECT scans used for the epilepsy surgery work up. The interictal SPECT which measures the distribution of radioisotopes in the resting state as compared to ictal SPECT where the injection should be administered within 30 seconds of seizure onset. The ictal SPECT has much higher yield than interictal SPECT.Regions of hypometabolism are seen with ictal SPECT and hypometabolism with interictal SPECT. (Ergün et al.,2016)

Positron Emission Tomography (PET)

A PET scan detects changes in brain glucose metabolism. It has been shown that epileptic foci have decreased glucose metabolism as compared to the non-epileptogenic regions of the brain. PET scan can detect focal dysplasias which are common causes of pediatric epilepsy.

These dysplasias can be missed on magnetic resonance imaging. The most commonly employed tracer is ¹⁸F-fluorodeoxyglucose. (Yu et al.,2018)

Functional Magnetic Resonance Imaging

Functional MRI shows that metabolically active areas have high ratios of oxygenated to deoxygenated hemoglobin secondary to increased neuronal activity in the brain. The functional MRI is employed in the epilepsy centers to localize language as part of pre-surgical work up. It is used to supplement WADA testing. (Martinez et al.,2016)

Neuropsychological Testing

Neuropsychological testing in children is important in order to assess neurocognitive deficits due to chronic epilepsy. It includes a series of neuropsychometric testing. Neuropsychological testing is extremely important in the pre-surgical work up of epilepsy since it localizes the deficits in the areas of memory and language which are extremely important to predict post surgery outcome.(Berg et al.,2017)

Treatment of Pediatric Epilepsy

a. Medical Management

Acute Management of Children with epilepsy

Are frequently admitted to the hospital because of status epilepticus or acute exacerbation of seizures.The acute management starts with general measures which includes establishing the diagnosis of status epilepticus or acute repetitive seizures.(Laino et al.,2018)

- assess the patient's airway and oxygenation.

- circulation and obtain intravenous access.
- Once the above procedures are accomplished, etiology should be assessed by: laboratory testing including blood count with differential, serum electrolytes, blood urea nitrogen, creatinine, serum toxicology screen, anticonvulsant levels and blood alcohol levels. Thiamine should be given to prevent Wernicke's hypoglycemia. (Gasparini et al.,2019)

Anticonvulsant therapy should be initiated as soon as possible and continuous EEG should be arranged to rule out non-convulsive status epilepticus. The first line agents include benzodiazepines. These include intravenous lorazepam and diazepam. Lorazepam can be administered at a rate of 1-2 mg/min (0.1 mg/kg). The maximum dose is 8 mg. Rectal diazepam can also be used 0.5 mg/kg but is more effective in acute repetitive seizures. Consider loading with intravenous phenytoin or fosphenytoin 20 mg/kg. The above measures should be completed within 30 minutes. The other choices are midazolam and

Phenobarbital. Midazolam can be used in a intravenous dose of 0.1-0.3 mg/kg within 5-10 minutes. It has been shown to abort seizures within one minute. If the seizures still continue, the choices are pharmacologically induced coma by pentobarbital, propofol or thiopentane. The last choice is neuromuscular blockade by or halothane administration by an anesthesiologist.(Cornet et al.,2018)

Newer antiepilepticus in management of acute attack:

Valproic acid was found to be effective in controlling refractory status epilepticus as an alternative to diazepam in an open label,

randomized, controlled study conducted by Mehta and colleagues in children.

Intravenous levetiracetam has been found to be effective in the treatment of status epilepticus and acute repetitive seizures. Data have already been published in different age groups regarding the safety and efficacy of levetiracetam in children. It has been shown in several retrospective studies that intravenous levetiracetam has been effective in treating status epilepticus and acute exacerbation of seizures in children and neonates. (Gasparini et al.,2019)

Chronic Management

Chronic management with anticonvulsants is needed for adequate control of seizures in patients with a well diagnosed history of epilepsy. **the first generation anticonvulsants** which includes phenytoin, carbamazepine, Phenobarbital, primidone, valproic acid, ethosuximide and benzodiazepines. **The second generation** newer anticonvulsants include felbamate, lamotrigine, gabapentin, topiramate, levetiracetam, oxcarbazepine, tigabine, pregabalin **and newly approved anticonvulsant** lacosamide (Abou-Khalil.,2019).

First Generation Anticonvulsants:

Phenytoin is a sodium channel blocker and is used in the management of tonic clonic seizures and partial seizures. (Mudigoudar et al.,2016)

Carbamazepine

Is also a sodium channel blocker and used to treat simple partial, complex partial seizures with and without secondary generalization. It is

also used to treat generalized tonic clonic seizures but should not be used in patients with myoclonic and absence seizures. It has been shown in the literature that carbamazepine causes worsening of myoclonic and absence seizures. (Sathyaprabha et al., 2018)

Phenobarbital is also a sodium channel blocker like phenytoin and carbamazepine and is used to treat tonic clonic and complex partial seizures. Phenobarbital is effective in neonatal seizures and status epilepticus. (Verrotti et al., 2019)

Valproic acid acts on the GABA receptors and causes blockage of the sodium channel. It is the first line of treatment in primary generalized epilepsies. These include generalized epilepsy syndromes like juvenile myoclonic epilepsy, juvenile absence epilepsy and other syndromes associated with absence seizures. (Mudigoudar et al., 2016)

- **Ethosuximide** is a T-type calcium blocker and is effective in childhood absence seizures. (Kanner et al., 2018)
- **Clonazepam** is used as adjunctive therapy for generalized epilepsies but not as effective for partial seizures. but **Clorazepate** can be used for both generalized and absence seizures.. (Verrotti et al., 2019)

Second generation anticonvulsant:

- **Felbamate** is used for partial epilepsy and in Lennox gastaut syndrome. (Knupp et al., 2018)
- **Lamotrigine** is a broad spectrum second generation anticonvulsant used in the treatment of primary generalized and partial epilepsy. It has also been shown to be effective in seizures associated with Lennox Gastaut syndrome (Moavero et al., 2017)

- **Gabapentin** has been approved by FDA for adjunctive therapy in children aged 3 to 12 years for the treatment of partial epilepsy. (Kanner et al.,2018)
- **Topiramate** has been approved as adjunctive therapy in children aged 2 to 6 years for both partial and primary generalized epilepsies and seizures associated with Lennox Gastaut syndrome (Fariba et al., 2020)
- **Tigabine** is also effective in the treatment of partial epilepsy.(Moosa., 2019)
- **Levetiracetam** was first approved as adjunctive therapy for partial seizures but later received approval for primary generalized and juvemile mypclonic epilepsies.(Chen et al., 2013)
- **Zonisamide and pregabalin** are also used for partial seizures. The choice of the anticonvulsant depends on the patient’s history, type of epilepsy, other medical comorbidities or cost constraints. (Fariba et al., 2020)

b. Surgical Management

The type of epilepsy surgery depends on the type of lesions. Focal lesions such as low grade tumors and focal cortical dysplasias are treated by **lesionectomies**. Total resection of the lesion results in seizure freedom in 80% of the patients. Functional mapping is required prior to resction. **Hemispherectomy** is done in patients with Sturge-Weber syndrome, prenatal stroke or Rasmussen encephalitis. (Lee et al., 2019)

Anterior temporal lobectomy and amydalo-hippocampectomy are the common surgical procedures in patients with mesial temporal sclerosis which is seen in adolescents and adults.(**Shah et al., 2015**)

Callosotomy which is the division of anterior two thirds of the corpus callosum and posterior one thirds if needed to prevent secondary generalized seizures, Lennox Gastaut syndromes and drop attacks. (**Lee et al., 2019**)

Subpial transections are palliative procedures where respective surgery is not possible. In subpial transections, the lateral cortical connections are disconnected to prevent the spread of epileptiform discharges. This procedure is used with Landau-kleffner syndrome and is found to be beneficial. (**Jayalakshmi et al., 2017**)

Vagal nerve stimulation

Cerebral Stimulation This includes chronic stimulation of the anterior nucleus of the thalamus and the subthalamus. Deep brain stimulation is still awaiting approval from the FDA for use in epilepsy patients. Deep brain stimulation causes disruption of the cortical reticular network which results in seizures. It has been shown to be effective in both generalized and partial epilepsy. It has been reported that a 30-50% decrease in refractory partial and secondary generalized seizures is seen with vagal nerve stimulation.(**Tzadok et al., 2019**)

Ketogenic Diet

The Ketogenic Diet (KD) is a modality of treatment used as a treatment for intractable epilepsy. It has been proposed as a dietary treatment that would produce similar benefits to fasting.

The KD has a high fat content (90%) and low protein and carbohydrate. Evidence shows that KD and its variants are a good alternative for non-surgical pharmaco-resistant patients with epilepsy of any age, taking into account that the type of diet should be designed individually and that less-restrictive and more-palatable diets (*Boison., 2017*)

The CKD is rich in lipids (90%) and low in carbohydrates and protein, in order to produce ketosis, and simulates a starvation state. Ketone bodies, acetoacetate, and β -hydroxybutyrate (β OHB), are byproducts of fatty acid oxidation in the mitochondrial matrix of the hepatocytes. There are many theories about the role of KB, but the existence of an anticonvulsant effect is controversial. KB supplementation resulted in attenuation of electrographic seizure-like events (*Martin et al., 2016*)

It must also provide adequate vitamins and minerals. The shift in the energy metabolism from glycolytic energy production to energy generation through oxidative phosphorylation (fatty acid β -oxidation and ketone-body production) is part of the anticonvulsant mechanism of the KD (*van et al., 2016*)

Traditionally, the KD has been considered the gold standard for the treatment of metabolic diseases such as Glucose Transporter Protein 1 (GLUT-1) deficiency syndrome and Pyruvate Dehydrogenase Deficiency. At present, the KD has been consistently reported as more beneficial, with more than 70% patients showing positive responses, as opposed to the average 50% response in several conditions such as infantile spasms. The KD has also been used in other conditions with less evidence, but possible benefits (*Elia et al., 2017*)

Additionally, the KD is an important alternative treatment for patients with refractory epilepsy that are not surgery candidates (*Garci et al a., 2018*)

Psychological abnormalities

Body and mind influence each other and work as a unit. Most of the diseases are psychological, which involve both the mind and body. There may be physical effects from mental illness. Some physical diseases are thought to be especially prone to be worsened by mental factors such as anxiety and depression. It is thought that the actual physical part of the person's illness might be affected by mental factors; something that is hard to prove. Many people; however, with these and other physical diseases say their current mental state can affect how bad their physical disease is at any particular time. (**American Psychiatric Association (APA) ,2000**)

When a person is anxious or afraid; for example, they might develop:

- Tremor
- Nausea
- Sweating
- Dry mouth
- Headaches
- Chest pains
- Palpitations
- Increased heart rate

- A, 'knot,' in the stomach
- Increased breathing rate

(American Psychiatric Association (APA) ,2000)

Anxiety:

Anxiety is an emotion characterized by an unpleasant state of inner turmoil, often accompanied by nervous behavior, such as pacing back and forth, somatic complaints, and rumination. It is the subjectively unpleasant feelings of dread over anticipated events, such as the feeling of imminent death. Anxiety is not the same as fear, which is a response to a real or perceived immediate threat, whereas anxiety is the expectation of future threat **(American Psychiatric Association, 2013)**.

Anxiety is a feeling of uneasiness and worry, usually generalized and unfocused as an overreaction to a situation that is only subjectively seen as menacing. It is often accompanied by muscular tension, restlessness, fatigue and problems in concentration. Anxiety can be appropriate, but when experienced regularly the individual may suffer from an anxiety disorder **(Bouras and Holt, 2007)**.

People facing anxiety may withdraw from situations which have provoked anxiety in the past. There are various types of anxiety. Existential anxiety can occur when a person faces angst, an existential crisis, or nihilistic feelings. People can also face mathematical anxiety, somatic anxiety, stage fright, or test anxiety. Social anxiety and stranger anxiety are caused when people are apprehensive around strangers or other people in general. Furthermore, anxiety has been linked with physical symptoms such as IBS and can heighten other mental health

illnesses such as OCD and panic disorder. The first step in the management of a person with anxiety symptoms is to evaluate the possible presence of an underlying medical cause, whose recognition is essential in order to decide its correct treatment. Anxiety symptoms may be masking an organic disease, or appear associated or as a result of a medical disorder (**Bouras and Holt, 2007**).

Anxiety can be either a short term "state" or a long term "trait". Whereas trait anxiety represents worrying about future events, anxiety disorders are a group of mental disorders characterized by feelings of anxiety and fear. Anxiety disorders are partly genetic but may also be due to drug use, including alcohol, caffeine, and benzodiazepines (which are often prescribed to treat anxiety), as well as withdrawal from drugs of abuse. They often occur with other mental disorders, particularly bipolar disorder, eating disorders, major depressive disorder, or certain personality disorders (**Diagnostic and Statistical Manual of Mental Disorders American Psychiatric Association, 2013**).

Etiology:

Genetics:

Genetics and family history (e.g., parental anxiety) may predispose an individual for an increased risk of an anxiety disorder, but generally external stimuli will trigger its onset or exacerbation. Genetic differences account for about 43% of variance in panic disorder and 28% in generalized anxiety disorder (**American Psychiatric Association, 2013**).

Although single genes are neither necessary nor sufficient for anxiety by themselves, several gene polymorphisms have been found to correlate with anxiety: PLXNA2, SERT, CRH, COMT and BDNF.

Several of these genes influence neurotransmitters (such as serotonin and norepinephrine) and hormones (such as cortisol) which are implicated in anxiety. The epigenetic signature of at least one of these genes BDNF has also been associated with anxiety and specific patterns of neural activity (**Moser et al., 2015**). Medical conditions:

Many medical conditions can cause anxiety. This includes conditions that affect the ability to breathe, like COPD and asthma, and the difficulty in breathing that often occurs near death. Conditions that cause abdominal pain or chest pain can cause anxiety and may in some cases be a somatization of anxiety; the same is true for some sexual dysfunctions. Conditions that affect the face or the skin can cause social anxiety especially among adolescents, and developmental disabilities often lead to social anxiety for children as well. Life-threatening conditions like cancer also cause anxiety (**Moser et al., 2015**).

Furthermore, certain organic diseases may present with anxiety or symptoms that mimic anxiety. These disorders include certain endocrine diseases (hypo- and hyperthyroidism, hyperprolactinemia), metabolic disorders (diabetes), deficiency states (low levels of vitamin D, B2, B12, folic acid), gastrointestinal diseases (celiac disease, non-celiac gluten sensitivity, inflammatory bowel disease), heart diseases, blood diseases (anemia), cerebral vascular accidents (transient ischemic attack, stroke), and brain degenerative diseases (Parkinson's disease, dementia, multiple sclerosis, Huntington's disease), among others (**Vanfleteren et al., 2016**).

Substance-induced:

Several drugs can cause or worsen anxiety, whether in intoxication, withdrawal, or from chronic use. These include alcohol, tobacco, cannabis, sedatives (including prescription benzodiazepines), opioids

(including prescription pain killers and illicit drugs like heroin), stimulants (such as caffeine, cocaine and amphetamines), hallucinogens, **anticonvulsant** medication and inhalants (**American Psychiatric Association, 2013**).

While many often report self-medicating anxiety with these substances, improvements in anxiety from drugs are usually short-lived (with worsening of anxiety in the long-term, sometimes with acute anxiety as soon as the drug effects wear off) and tend to be exaggerated. Acute exposure to toxic levels of benzene may cause euphoria, anxiety, and irritability lasting up to 2 weeks after the exposure (**CDC, 2016**).

Psychological:

Poor coping skills (e.g., rigidity/inflexible problem solving, denial, avoidance, impulsivity, extreme self-expectation, affective instability, and inability to focus on problems) are associated with anxiety. Anxiety is also linked and perpetuated by the person's own pessimistic outcome expectancy and how they cope with feedback negativity. Temperament (e.g., neuroticism) and attitudes (e.g. pessimism) have been found to be risk factors for anxiety (**Gu et al., 2010**).

Cognitive distortions such as overgeneralizing, catastrophizing, mind reading, emotional reasoning, binocular trick, and mental filter can result in anxiety. For example, an overgeneralized belief that something bad "always" happens may lead someone to have excessive fears of even minimally risky situations and to avoid benign social situations due to anticipatory anxiety of embarrassment. Such unhealthy thoughts can be targets for successful treatment with cognitive therapy.

Psychodynamic theory posits that anxiety is often the result of opposing unconscious wishes or fears that manifest via maladaptive defense mechanisms (such as suppression, repression, anticipation, regression, somatization, passive aggression, dissociation) that develop to adapt to problems with early objects (e.g., caregivers) and empathic failures in childhood. For example, persistent parental discouragement of anger may result in repression/suppression of angry feelings which manifests as gastrointestinal distress (somatization) when provoked by another while the anger remains unconscious and outside the individual's awareness. Such conflicts can be targets for successful treatment with psychodynamic therapy (**Gu et al., 2010**).

Evolutionary psychology:

An evolutionary psychology explanation is that increased anxiety serves the purpose of increased vigilance regarding potential threats in the environment as well as increased tendency to take proactive actions regarding such possible threats. This may cause false positive reactions but an individual suffering from anxiety may also avoid real threats. This may explain why anxious people are less likely to die due to accidents (**Pinquart et al., 2011**).

When people are confronted with unpleasant and potentially harmful stimuli such as foul odors or tastes, PET-scans show increased bloodflow in the amygdala. The participants also reported moderate anxiety. This might indicate that anxiety is a protective mechanism designed to prevent the organism from engaging in potentially harmful behaviors (**American Psychiatric Association, 2013**)

Social:

Social risk factors for anxiety include a history of trauma (e.g., physical, sexual or emotional abuse or assault), early life experiences and parenting factors (e.g., rejection, lack of warmth, high hostility, harsh discipline, high maternal negative affect, anxious childrearing, modelling of dysfunctional and drug-abusing behaviour, discouragement of emotions, poor socialization, poor attachment, and child abuse and neglect), cultural factors (e.g., stoic families/cultures, persecuted minorities including the disabled), and socioeconomics (e.g., uneducated, unemployed, impoverished (although developed countries have higher rates of anxiety disorders than developing countries)). (O'Connell et al., 2009).

Gender socialization:

Contextual factors that are thought to contribute to anxiety include gender socialization and learning experiences. In particular, learning mastery (the degree to which people perceive their lives to be under their own control) and instrumentality, which includes such traits as self-confidence, independence, and competitiveness fully mediate the relation between gender and anxiety. That is, though gender differences in anxiety exist, with higher levels of anxiety in women compared to men, gender socialization and learning mastery explain these gender differences (Gu et al., 2010).

Depression:

Depression is a mood disorder that causes a persistent feeling of sadness and loss of interest. Also called major depressive disorder or

clinical depression, it affects feeling, thinking and behavior and can lead to a variety of emotional and physical problems (**Segal et al., 2018**).

Pediatric depression is a relatively common psychiatric condition that generally continues episodically into adulthood. Whether damaging experiences or biologic processes trigger depressive episodes remains subject to debate; however, the final common pathways to depression involve biochemical changes in the brain (**Hammen, 2018**).

Etiology:

Biologic versus nonbiologic causes:

Psychosocial stressors are posited to be mediators for the development of depression. The final common pathways to depression involve biochemical changes in the brain (**Bredt et al., 2015**).

Genetic factors:

Several studies of adults who are depressed, such as those reported by **Akiskal and Weller (1989)** and **Weissman et al (1984)** suggest a genetic component in the etiology of depressive disorders.

Parent-child relation model:

The parent-child relation model conceptualizes pediatric depression as the result of poor parent-child interaction. Adults with depression report low paternal involvement and high maternal overprotection during early childhood. Troubled relationships with parents, siblings, and peers are common in children and adolescents with affective illness (**Bang et al., 2012**).

A child who is affectively ill often has a parent who is affectively ill. It is not uncommon for children to report abuse and/or neglect by the parent or parents who are affectively ill. **Hammen et al (1991)** reported a significant temporal association between maternal and child stress and depression. They found that children with substantial stress exposure who also had a symptomatic mother were significantly more depressed than children who were exposed to comparable levels of stress only (**Hammen et al., 1991**).

Cohort effect:

Klerman and Weissman (1989) reported a progressive increase in the lifetime cases of major depression over the last 70 years. They found high rates of affective disorders among relatives, with a younger age of onset in successive cohorts.

Increased time spent using electronic devices (e.g., cell phones, tablets, computers, etc.) may have played a role in the increased rates of depression and suicide observed between 2010 and 2015, especially among girls, according to one study. Researchers found that adolescents who spent more time on new media (including social media) were more likely to report mental health issues, and adolescents who spent more time on nonscreen activities (in-person social interaction, sports/exercise, homework, print media, and attending religious services) were less likely. Although the study demonstrates a correlation between long hours of daily screen time and symptoms of alienation, it does not prove that one causes the other (**Twenge et al., 2017**).

Depression as a dimensional concept:

Depression as an emotion:

Depressive affect or feeling is a normal response to disappointment, loss or other painful events of human life. Depressive affects are self-limited and do not usually significantly interfere with a person's functional capacity, unless becoming longer lasting. Moreover, it has been postulated that in some situations the depressive mood might even be useful and have offered a selective advantage in humans' evolutionary history, by disengaging former goals and reallocating resources (Nesse, 2006).

Depression as symptoms:

Depressive symptoms include, among others, mood bias toward negative emotions (depressed mood), impaired reward function (anhedonia, lack of reactivity and loss of interest) and psychomotor symptoms which are probably heterogeneous with respect to etiology and pathophysiology (Hasler et al. 2004).

Moreover, depression itself has been found to be dynamic in nature, evolving on a continuous scale, ranging from no depressive symptoms, depressive symptoms, minor depression and finally to major depressive disorder (Kessing, 2007). In addition, the symptoms of depression measured cross-sectionally might change over time in the individual patient, fulfilling criteria for major depression, minor depression, dysthymia and subsyndromal states (Vuorilehto et al., 2005).

Depression as a clinical syndrome:

In most definitions of depression, a distinction is drawn between a feeling state of dejection, sadness, or unhappiness, which may be brief in duration, and a clinical syndrome characterized by persistent sadness, profound discouragement, or despair which persists two weeks or more and is associated with a change from previous functioning. This clinical syndrome invariably involves alterations in mood experienced by an individual as a feeling of sadness, irritability, dejection, despair, or loss of interest or pleasure. Associated neurovegetative or biological signs of depression include impairment in sleep, appetite, energy level, and psychomotor activity. Cognitive manifestations of the depressive syndrome include distortions about oneself, one's experience in the world and the future, accompanied by self-blame and indecision. These core symptoms of depression are evident in children or adolescents with MDD although the depressed mood may be manifested by irritability or social withdrawal (Oros et al., 2017).

Psychological assessment in children with epilepsy

The ultimate goal of contemporary Medicine is to overcome life-threatening barriers and offer superior survival chances to certain patient categories with terrible destinies. This idea is understood best, in its truest sense, by those who assist patients with epilepsy. (Schraegle et al.,2017)

Neurobehavioral disorders including fatigue, depression, anxiety, and psychosis commonly affect patients with epilepsy. People with epilepsy may also have cognitive problems that effect attention, memory, mental speed, and language, as well as executive and social functions. Cognitive and behavioral disorders often overshadow the seizures themselves and can be the greatest cause of impaired quality of life.

Furthermore, these problems often go unrecognized and, even when identified, are often under treated or untreated. Patients with epilepsy frequently suffer from cognitive and behavioral disorders that range from subtle to severe. Behavior changes occur during and immediately after most seizures. However, in some cases, cognition and behavior also change for prolonged periods after individual seizures or throughout the long interictal gaps. (**Reardon et al.,2015**)

Aggressive control of seizures, and possibly reduction of interictal epileptiform activity and epileptogenesis, may help prevent interictal cognitive and behavioral disorders. The late 19th century view of epilepsy as a progressive disorder—in terms of both seizures and cognitive- behavioral disorders—is finding support from modern studies (1). While the best therapy for cognitive and behavioral disorders may be prevention, there is little systematic study of the phenomenon either retrospectively or prospectively. (**Schraegle et al.,2017**)

❖ **Important factors in the developing psychological problems:**

Epilepsy is one of the most common chronic medical problems in childhood. Despite advances in epilepsy diagnosis and treatment, many children with epilepsy function poorly, with an excessive incidence of psychosocial difficulties and behavioral problems. (**Puka et al., 2017**)

Disability may be related to social stigma, school failure, or emotional problems, rather than to seizures, which may be infrequent or fully controlled. Parental anxiety about the child's condition may contribute to excessive dependency, social disability, and family dysfunction. Parents may unnecessarily or excessively restrict their epileptic child's activities and may lower their expectations for the child.6 Negative family perceptions of the epileptic child may adversely

influence psychosocial development. It has long been recognized that even with normal intelligence in the child, epilepsy increases the chance of school failure. However, we previously reported that indicators of social and economic status were more important than severity, duration, or treatment of seizures in determining academic achievement of children with epilepsy. Socioeconomic and family status may influence other aspects of the child's functioning as well. Social disability may be unrelated to the severity and controllability of seizures. For example, good seizure control and normal intelligence were associated with poor self-image, low achievement, and low expectations for the future in adolescents with generalized seizures. (**Schraegle et al.,2017**)

Stressful life events seem to increase health care utilization and psychosomatic and behavioral complaints. However, prospective studies have not supported the idea that stress alone is a strong determinant of chronic illness or poor functioning. The more important effect of stressful life events may be their influence on the family's ability to cope with and their attitudes toward illness. Cultural background, social power, and family resources may be important determinants of a child's medical and behavioral outcome, as well as of family attitudes. For example, ongoing seizures may exacerbate behavior problems or family anxieties, or vice versa. Unless these influences are considered simultaneously. (**Speechley et al., 2013**)

Seizure-Related Risk Factors.

Indicators of seizure-related risk included the type(s) of seizure and current and maximum seizure frequency, age at onset, duration of the seizure disorder, prior treatment (successful or unsuccessful), number of medications used in the past, and reasons for previous medication

changes. We hypothesized that children whose seizures began at a young age, who had not responded to treatment, or who had a prior adverse behavioral reaction to medication were more likely to do poorly at outcome. (**Ferro,2014**)

Developmental/Cognitive Risk Factors:

Depending upon age at enrollment and primary language, Children with intellectual deficits were expected to have poor outcome compared to children with normal cognitive function. Behavioral Risk Factors. A history of behavior problems, hyperactivity, or attention deficit disorder predicts poor functioning despite adequate seizure control and contributes to inappropriate family responses to the child’s epilepsy. (**Bompori etal., 2014**)

Family Structure and Socioeconomic Risk Factors:

children with fewer social supports were at higher risk of adverse psychosocial outcome. (**Bompori etal., 2014**)

Table (1): Emotional Reactions: Their manifestations and adverse effects

Emotional Reaction	Manifestation	Adverse Effects
Denial	Avoids the implication of the illness. Acts as if the illness is not severe or will go away. May deny even having the illness.	Interferes with the ability to monitor the condition, take initiative in seeking treatment or maintain a follow-up.
Anxiety	Is vigilant about every minor ache or pain. Feels anxious about the implication of the disease for the future and impact {work and leisure activities}.	Intrinsically distressing. Can interfere with good functioning. Not so receptive to therapy. Shows increased symptomatology
Depression	Catastrophizes about the situation and generalizes negatively from the situation. Manifests weight loss, fatigue, sleeplessness, crying spells, pessimism and suicidal attempts.	Intrinsically distressing. Has a negative impact on long term hospitalization, rehabilitation and recovery.

(**Alfstad etal.,2011**)

Anxiety:

Chronic medical illness is a significant risk factor for the development of psychiatric disorders, including anxiety. Moderate to severe anxiety affects symptom management, treatment adherence, medical outcome, and the ability of the child/adolescent to cope with the illness. Anxiety, a part of emotional status, is perceived as one of very important aspects of health-related quality of life. This is why anxiety should be spotted early and appropriately treated (**Speechley et al., 2013**).

Despite the increased significance of anxiety in somatic diseases, most common psychological disorder involves anxiety. Anxiety and epilepsy are so closely linked that seizures sometimes are mistaken for panic attacks in those who have never had seizures before.

- About 4% of the general population has generalized anxiety disorder, a constant state of tension or worry.
- The number is much higher for people with epilepsy. It's possible that this disorder is caused or made worse by seizures.
- Anxiety disorders have been associated with the amygdala, a structure in the front part of the temporal lobe. The seizures of temporal lobe epilepsy frequently affect the amygdala and cause it to act in different ways.
- Anxiety can also be directly related to the possibility of seizures. Not knowing when a seizure may occur can increase worry about having one in an embarrassing or dangerous situation.
- Certain kinds of people are more susceptible to anxiety disorders.

- Genetic influences and a person's response to stress may play a part in their development.
- Women are more likely than men to have anxiety disorders, and patients with nonepileptic seizures also have higher rates of anxiety.

The presence of auras involving fear has been linked to anxiety disorders. **(Siqueira et al.,2017)**

Depression:

The personality profile is very important for the survival rate of the epileptic patient. Psychological support is necessary in order to avoid or diminish the severity of the following depressive syndrome. Depression is generally accepted as the most common psychological problem in epileptic patient. the syndrome of clinical depression includes sadness, guilt, hopelessness, helplessness and changes in sleep, appetite and libido Depression in epileptic patients is seen as a predictable and frequent complication. **(Mendes et al., 2017)**

Monitoring for depression should be incorporated into routine care together with evaluative interventions that might enhance survival. **(Alfstad et al.,2011)**

The approach to treatment of depression is framed by the severity of depression, suicidal ideation, presence of social support and other chronic health conditions.. It may be highlighted that depression is common and may explain some of their physical symptoms relating to sleep, energy and appetite. Education tools such as pamphlets and trusted web-sites may augment the face-to-face education from a clinician. Patients should

be encouraged to adopt a healthy lifestyle by increasing exercise, reducing stress, and avoiding drugs and alcohol. (Vonneilich et al.,2016)

Mild depression may be addressed with psychotherapy in addition to lifestyle changes and close observation. These approaches can be used alone or in combination with anti-depressant medications. Psychotherapy includes a number of approaches such as cognitive therapy, behavioral therapy, family therapy, couples therapy, interpersonal therapy, and problem-solving approaches. There have been studies on using cognitive behavioral therapy as an approach to treating depression in patients with epilepsy (Borusiak et al., 2016)

Major depression should be treated with an anti-depressant. Some patient may benefit from drug therapy combined with psychotherapy. The classes of medications widely used to treat depression include selective serotonin reuptake inhibitors (SSRIs), tricyclic antidepressants, and inhibitors of the reuptake of both serotonin and norepinephrine (SNRIs). Most studies of these medications find similar side effect profiles and efficacy among depressives in the general population (Vonneilich et al.,2016)

Guidelines intervention for helping children with epilepsy

Having epilepsy influences a child's self-image and relationships with peers and family. It can lead to behaviour problems and make achieving goals more difficult. Being aware of these problems can help recognize that child may need some additional guidance or understanding at times (Reilly et al., 2014).

1. Family support:

Parents showed greater levels of anxiety and depression than a normal sample and more psychosomatic problems than a control group consisting of parents of children with other chronic physical conditions. Positive correlations were found between age on diagnosis of renal failure and fathers' depression and anxiety scores. Mothers' anxiety and depression scores were also positively correlated with those of father.

The child's illness was reported to have caused disruption in family life by most parents in the dialysis group (77%) significantly more often than by parents in the non-dialysis group (31%). Disturbance was commonly explained in terms of the restrictions imposed by the child's condition or treatment, including dialysis, which made family outings or holidays difficult to organize. Higher family conflict predicts also more externalizing symptoms and higher number of prescribed medications; higher family cohesion predicted fewer hospitalizations. Non-traditional family structure predicted higher number of prescribed medications (**Liu et al., 2015**)

In addition to 'normal' parental roles, being a parent of a child with epilepsy demands a high-level health care provider, problem solving, information seeking, and financial and practical skills at a time when the capacity to cope is threatened by physical tiredness, uncertainty, and disruption to peer support within and outside the family structure. Parents of children with epilepsy need multidisciplinary care, which may lead to improved outcomes for their children (**Michaelis et al., 2017**).

2. School:

Assuring a normal life is very important for epileptic child. Negative consequences include the impossibility of continuing to attend school. Learning problems could be the consequence of missing classes or the impossibility to focus during tasks. Data on school performance have shown that children with epilepsy are at risk for impairment. The aetiology of this deficit appears to be multifactorial, with the neurological side effects of the disease itself and the associated treatments, as well as school absences, all potentially playing a role. Tutoring or vocational rehabilitation could help (**Michaelis et al.,2017**)

3. Dealing with low self-esteem:

Learning problems and physical consequences of the illness contribute to a lower self-esteem. They could feel depressed and powerless. They often have poor self-esteem and a pervasive sense of losing their identity, body integrity, control, independence and opportunity. Interventions are needed to equip children with the capacity to manage their health, participate in community, engage in permissible recreational activities, progress in their studies, and remain vigilant in dialysis and treatment responsibilities, for improved health and treatment outcomes (**Michaelis et al.,2017**)

4. Following medical instructions:

Children can refuse the medical treatment. In case of noncompliance or non-adherence, the psychological therapy is a must. The teenagers are usually difficult patients, due to their age's psychological, physical and social needs (**Reilly et al., 2014**).

5. Engaging sports or physical activity:

Usual limited, the physical activity is important for two aspects: integrate the child in social activity (play, games, competition, fun etc) and having benefits on the physical and psychological life (feeling powerful and independent. **(Carona et al., 2014)**)

6. Making friends:

Children with epilepsy may have trouble in making friends. The physical activity restrictions, the drug treatment, the food and drinks restriction could limit them for socializing. **(Carona et al., 2014)**

7. Religious/spiritual coping:

Among other variables that influence the health behaviour, religious/spiritual coping mechanisms are an important strategy to cope with the disease. Religious practice (like praying) is influencing the quality of life of patients with epilepsy.

Positive religious coping was associated with better overall, mental and social relations Religious struggle was an independent correlate of worse overall, physical, mental, social relations and environment health quality of life. The effects of spirituality may be mediated by social support and social support is correlated with survival. **(Michaelis et al.,2017)**

Patients and Methods

I. Patients:

This cross sectional study will be carried out in neurological unit and clinic of the pediatric department at Benha university hospital from February 2020 to June 2020. It will comprise children with epilepsy on regular treatment of both sexes after obtaining informed consent from children's parents.

Sample:

It were comprised (30) children with epilepsy on antiepileptic drugs and other (30) with no chronic illness as control group of both sexes after obtaining informed consent from children's parents.

The study were under the following inclusion and exclusion criteria:-

Inclusion criteria:-

- Children with epilepsy on regular treatment.
- Both sexes.
- Children age from 6 years up to 18 years

Exclusion criteria:

- Children who had a suggestive history of congenital syndromes affecting psychology of patients.
- Children who diagnosed with epilepsy less 6 years.
- Children with organic brain lesion: brain hemorrhage, brain tumor or brain atrophy.

Ethical consideration:

Approval of the study protocol by an ethical committee of Benha university will be obtained and informed consent will be obtained from the parents before enrollment in the study.

Methods:

All patients were subjected to:

1-Full history taking:

- Personal history: age, sex, residence, birth order, level of education, socioeconomic status.
- Present history: onset, course of disease, frequency of seizures, type of seizures; generalized or focal. tonic or clonic or atonic description of preictal, ictal, post ictal period, investigation done to establish diagnosis, prescribed medication, to what extent seizures controlled after starting medication.
 - a) Nutritional habits, e.g., appetite change in desire or amounts, number of meals per day, and preferred restricted food and types of food prepared for the child at home.
 - b) Sleeping patterns, e.g., place of sleeping, number of hours of sleep per day, and naps and sleep problems.
 - c) School attendance and achievements
 - Past history: febrile convulsion, head trauma, drug intake.
 - Family history: family member with epilepsy, consanguinity, psychiatric conditions.

2-Clinical examination:

- General examination: vital signs, anthropometric measurements (weight, height, body mass index)
- Neurological examination
- Chest examination
- Heart examination.
- Local abdominal examination.

3-study investigations:

- Routine laboratory investigations
- CBC, serum electrolytes(Na,K,Ca,Po4), kidney function tests, liver function tests,.
- Specific investigations and imaging:
- EEG
- MRI
- Specific investigations:
 - Self reporting scales as:

(A) Children anxiety scale (El-Beblawy 1998):

- This scale was developed by Castaneda, McCandless and Palermo in (1956) then translated into Arabic language by Viola El beblawy (1998) this Arabic copy was used in the present study for asking the children.

- This scale consists of 42 items to measure anxiety for children which is considered group of symptoms which can be classified into 3 groups physiological, behavioral and verbal measures.
- 11 items were added as lie scale to ensure the reliability of answers of these children. These items are numbered (5,10,17,21,30,34,36,41,47,49 and 52)
- If the patient's answers more than 3 degrees on lie scale, this patient is excluded from the study.
- The total number of items in the scale are 53 and each item consists of one statement which has two answers yes or no.
- If the answer is Yes → score 1
- If the answer is No → score 0
- but in lie scale; one degree for answer (Yes) except items number 10 & 49 one degree for answer (No)
- Total score ranges from (0-53)
- According to their scores, they were classified into mild, moderate & severe degree of anxiety.

Table (2): Anxiety Score

Children anxiety score	Children anxiety status
<18	Mild
19-28	Moderate
>29	Sever

(B) Children depression inventory scale (CDI) (Ghareeb and Beshai):

- The CDI was developed by American clinical psychologist Maria Kovac, PhD, and was published in 1979. It was developed by using

the Beck Depression Inventory (BDI) of 1967 for adults as a model.

- The CDI is a widely used and accepted assessment for the severity of depression symptoms in children and youth, with high reliability. It also has a well-established validity using a variety of different techniques, and good psychometric properties.
- The arabic copy was used in this study. It was translated into arabic by Ghareeb Abdel-Fattah Ghareeb and J.A. Beshai.
- This scale is designed to assess mood disturbance, ability of enjoyment, growth functions, self esteem and behaviour with others.
- The scale includes 27 items; each item consists of 3 choices of answers and the patient should choose one.

According to the severity, the degrees ranges from 0-2 as follows:

No symptoms = 0

Mild to moderate = 1

Sever symptoms = 2

Total score ranges from (0-54)

Table (3): Depression Score

Depression	Gender	Depression status
0-9		Normal
9-14	Male	Mild
9-16	Female	Mild
14-18	Male	Moderate
16-22	Female	Moderate
>18	Male	Sever
>22	Female	Sever

Procedures:

- This questionnaire was applied individually.
- Every child had given 2 questionnaire sheets (one for anxiety and the other for depression) and a pen.
- The questionnaire was explained for all children and how to answer it after having their agreement to share in this study.
- 80%(47 children) answered the questionnaire by themselves.
- 20%(13 children) couldnot read well so i asked them and wrote their answers in the sheet.
- The average time for application of the questionnaire differs accordind to level of education of children and their ability to read and understand the questions.
- According to anxiety test it took about 15 to 40 minutes.
- According to depression test it took about 20 to 45 minutes.

Consent approvals:

Informed consent were taken from parents of children and hospital managers sharing in this study.

III. Statistical analysis

Statistical Analysis

The collected data were tabulated and analyzed using SPSS version 22.0 (IBM, Armonk, NY, USA) for Windows. Categorical data were presented as number and percentages, Chi square (χ^2) and Fisher's exact tests were used to analyze them. Quantitative data were tested for normality using Shapiro-Wilks test assuming normality at $P > 0.05$. Normally distributed variables were expressed as mean \pm standard deviation and analyzed student "t" for 2 independent groups. While non parametric data were presented as median and range, and analyzed by Mann Whitney U (ZMWU) and Kruskal Wallis (KW) test for 2 and more than 2 independent groups respectively. $P \leq 0.05$ was considered significant. Spearman's correlation coefficient (rho) was used to assess correlations involving non parametric variables. The accepted level of significance in this work was stated at 0.05 ($P < 0.05$ was considered significant),.

P value > 0.05 is non significant (NS)

$P < 0.05$ is significant (S)

$P \leq 0.001$ is highly significant (HS)

Mean =

Is the sum of the values in a set of data divided by the number of the values in the set. It is denoted by the sign \bar{X} (called X bar).

$$\bar{X} = \frac{\sum x}{n}$$

Where: X denotes any value of observation.

\sum the Greek capital letter sigma, means the sum of.

n The number of observations.

Standard deviation (SD):

It is the positive square root of the variance.

$$\text{Variance} = S^2$$

The sum of the squares of the deviation of each measurement in a series from the mean of the series, divided by the total number of the observation minus one.(The degree of freedom).

$$S^2 = \frac{\sum \text{Squared deviation of the mean}}{n - 1}$$

$$S^2 = \frac{\sum (X - \bar{X})^2}{n - 1}$$

Median = middle value of the ordred data

$(N+1)/2$ if sample size is odd

$n/2$ & $(n/2)+1$

if sample size is even

Chi square test $\sum (O - E)^2$
$$X^2 = \frac{\quad}{E}$$

Where O is the observed value

E is the expected value

It compares between 2 or more categorical groups (tables 2x2 or more)

$$Expected = \frac{col.total \times rowtotal}{Grand total}$$

Fisher's exact test is used when you have two nominal variables. Fisher's exact test is more accurate than the chi-squared test when the expected numbers are small.

Student “t” test compares between 2 means of 2 independent groups.

t-value is the ratio of the difference between the two means/calculated SD of this difference.

$$t = \frac{\bar{X}_1 - \bar{X}_2}{\sqrt{\frac{SD_1^2}{n_1} + \frac{SD_2^2}{n_2}}}$$

Where \bar{X}_1 = mean of group 1

\bar{X}_2 = mean of group 2

SD₁ = Standard deviation of group 1

SD₂ = Standard deviation of group 2

n_1 = sample size of group 1

n_2 = sample size of group 2

rho→**Spearman's correlation coefficient**: it evaluates the linear association between 2 quantitative variables (one is the independent var.X, and the other is the dependent var., Y).

value of "r" ranges from -1 to 1

0= no linear correlation

1= perfect positive correlation

-1 = perfect negative correlation

Positive= increase in the independent variable leads to increase in the dependent variable

Negative = increase in the independent variable leads to decrease in the dependent variable.

Mann Whitney U test:

Non parametric test used to compare 2 non parametric quantitative variables.

Kruskall Wallis test(KW test): non parametric test used to compare quantitative variables among more than 2 independent groups.

The most common post-hoc test for the Kruskal–Wallis test is the Dunn test.

Boxplots

A useful way of graphically representing the symmetry of data is the boxplot. This type of graph displays the median value by a horizontal bar surrounded by 50% of the scores shown within a box. This 50% of scores falls between the 25th and 75th percentile marks. The 25th percentile is at the bottom of the box and the 75th percentile is at the top. The whiskers extending from both ends of the box show the highest and lowest values that are not outliers. Outliers are scores in the distribution that are more than 1.5 box-lengths from the 25th or 75th percentile, and they are displayed by a circle; those that are more than 4 box-lengths away are shown by an asterisk.

Results

Table (4): Comparing the studied groups regarding socio-demographic characters

Variable		Patients (n=30)		Controls (n=30)		Test of significance	P
Age (ys)	Mean±SD	13.0±2.0		12.4±1.95		St “t”= 1.11	0.27 (NS)
	Range	10-16		10-16			
		No.	%	No.	%	χ^2	P
Sex	Female	17	56.7	15	50.0	0.26	0.61 (NS)
	Male	13	43.3	15	50.0		
Residence	Urban	18	60.0	20	66.7	0.29	0.59 (NS)
	Rural	12	40.0	10	33.3		
Family problems	No	16	53.3	23	76.7	3.59	0.058 (NS)
	Yes	14	46.7	7	23.3		
Socio-economic level	Poor	13	43.3	12	40.0	0.07	0.79 (NS)
	Good	17	56.7	18	60.0		

This table shows that there was no statistically significant difference between the studied groups regarding age, sex, residence, family problems or socio-economic level ($P > 0.05$ for all variables).

Table (5): Characters of patients group

Variable		No. (N=30)	% (100%)
Type of seizures	Absence	1	3.3
	Complex partial	3	10.0
	Focal	6	20.0
	GTC	20	66.7
	Mean ±SD	Minimum	Maximum
Age of onset (years)	10.1±1.94	6	13
Duration (m)	6.8±1.9	5	10
Frequency		No. (N=30)	% (100%)
	Non	15	50.0
	One	9	30.0
	Two	3	10.0
	Three	2	6.7
	Four	1	3.3

This table demonstrates that 2/3 of patients (66.75) suffered from GTC fits, 20% had focal type, 10% had complex partial and only 3.3% suffered from Absence, the age of onset of fits ranged from 6 to 13 years with mean value of 10.1 ± 1.9 . The average duration of the fit was 6.8 minutes ranging from 5 to 10. Thirty percent of patients had one fit per month, 10% had 2 fits, 6.7% had three and only one case (3.3%) had the fits 4 times per month.

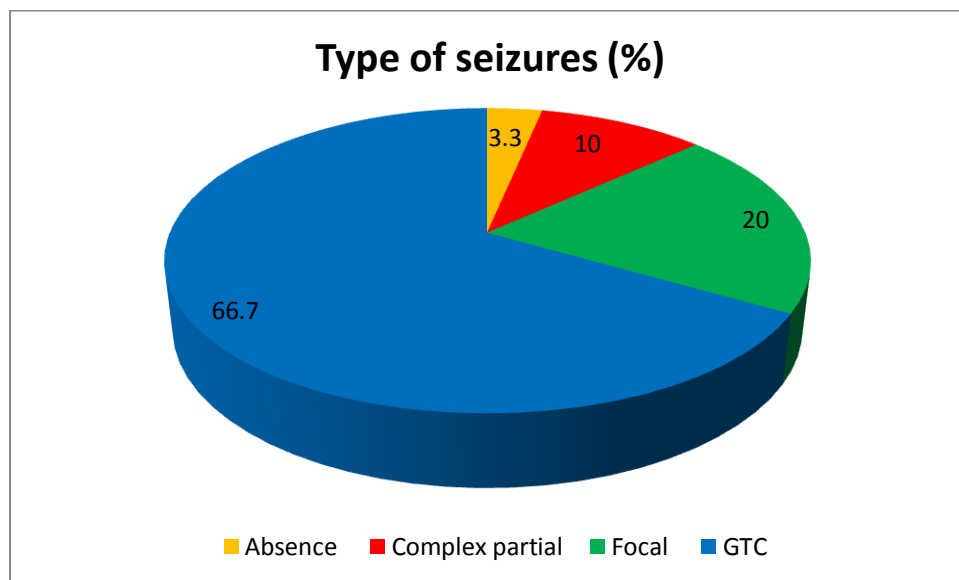


Fig (9): Pie chart showing the percentages of seizures types

Table (6): Treatment regimen and compliance among the studied patients

Variable		No. (N=30)	% (100%)
Number of drugs	Single	12	40.0
	Two	6	20.0
	Three	12	40.0
Type of treatment	Tegretol	26	86.7
	Topramate	16	53.3
	Tiratam	14	46.7
	Depakin	3	10.0
	Exthaomide	1	3.3
Compliance to treatment	No	12	40.0
	Yes	18	60.0

This table shows that 40% of patients are on single drug and another 40% are on three while 20% are on 2 drugs. Tegretol is the most commonly used drug (86.7%) followed by topramate (53.3%), Tiratam (46.7%), Depakin (10%) and Exthaomide (3.3%). Forty percent of children showed noncompliance to treatment.

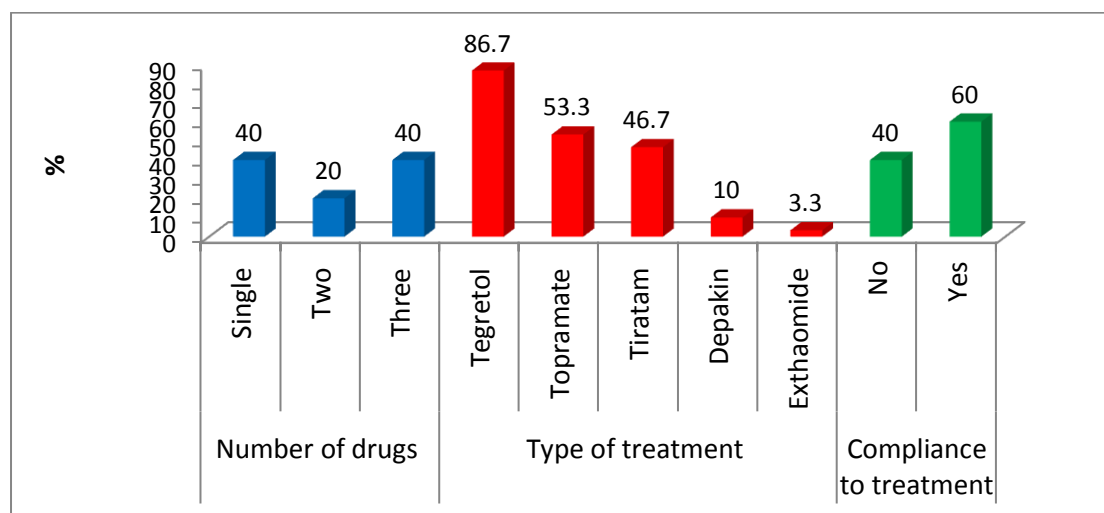


Fig (10): Bar chart showing regimen and compliance to treatment

Table (7): Comparing the studied groups regarding attention deficit

			Group		Total	χ^2 (P)
			Cases	Controls		
Attention deficit	No	Count	16	30	46	18.2 (<0.001 , HS)
		% within Group	53.3%	100.0%	76.7%	
	Yes	Count	14	0	14	
		% within Group	46.7%	0.0%	23.3%	
Total		Count	30	30	60	
		% within Group	100.0%	100.0%	100.0%	

This table illustrates that there was a significant association between attention deficit and being a case, where 46.7% of cases had attention deficit compared to non of the control group ($P<0.001$).

Table (8): Comparing the studied groups regarding school performance

Variable		Patients (n=30)		Controls (n=30)		χ^2	P
		No.	%	No.	%		
School attendance	Irregular	14	46.7	4	16.7	6.24	0.012 (S)
	Regular	16	53.3	25	83.3		
School performance	Poor	10	33.3	0	0.0	20.2	<0.001 (HS)
	Adequate	18	60.0	15	50.0		
	Good	2	6.7	15	50.0		

This table illustrates that there was a significant difference between the studied groups regarding scholastic attendance, where 46.7% of cases attended school irregularly compared to 16.7% of the control group ($P<0.05$). Also, 33.3% of patients had poor school performance compared to nill of the controls, and only 6.7% of them had good performance compared to 50% of the controls. these differences were statistically highly significance ($P<0.001$).

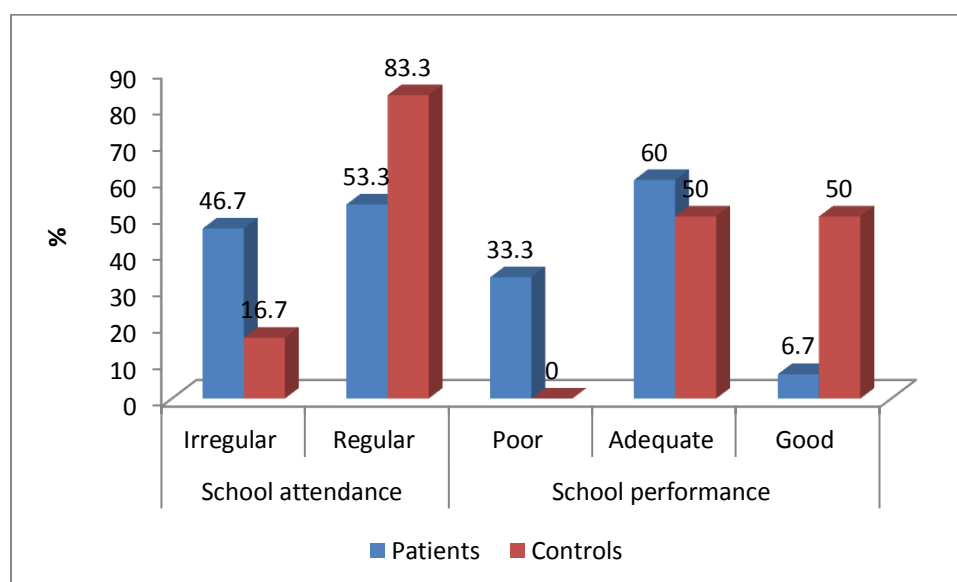


Fig. (11): Bar chart showing school attendance and performance among the studied groups

Table (9): Comparing the studied groups regarding social relationship

Variable		Patients (n=30)		Controls (n=30)		χ^2	P
		No.	%	No.	%		
Having friends	No	12	40.0	0	0.0	15.0	<0.001 (HS)
	Yes	18	60.0	30	100.0		
Social withdrawal	No	16	53.3	30	100.0	18.2	<0.001 (HS)
	Yes	14	46.7	0	0.0		

This table illustrates that there was a significant association between poor social relations and being a case, where 40% of cases had no friends compared to non of the control group ($P < 0.001$). Also, 46.7% of them suffered from social isolation compared to 0% of the controls ($P < 0.001$).

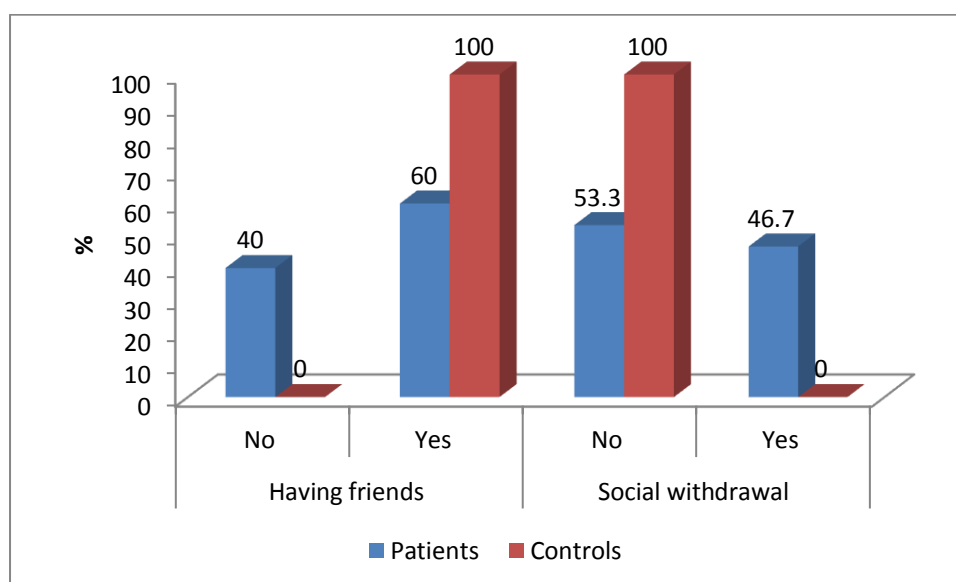


Fig. (12): Bar chart showing social relationship among the studied groups

Table (10): Comparing the studied groups regarding physical exercise

			Group		Total	χ^2 (P)
			Cases	Controls		
physical exercise	No	Count	17	6	23	8.53 (0.003, S)
		% within Group	56.7%	20.0%	38.3%	
	Yes	Count	13	24	37	
		% within Group	43.3%	80.0%	61.7%	
Total		Count	30	30	60	
		% within Group	100.0%	100.0%	100.0%	

This table illustrates that there was a significant association between lack of physical exercise and being a case, where 56.7% of cases reported no physical activity compared to 20% of the control group ($P < 0.05$).

Table (11): Comparing the studied groups regarding depression and anxiety scores

Variables	Patients (n=30)			Controls (n=30)			Z_{MWU}	P value
	Mean	\pm SD	Median (Range)	Mean	\pm SD	Median (Range)		
Depression score	20.4	9.19	18.0 (10-37)	7.3	2.35	7 (4-14)	6.35	<0.001 (HS)
Anxiety score	20.0	8.23	18.5 (10-36)	13.5	4.12	13.5 (7-22)	3.25	=0.001 (HS)

This table shows that the mean and median values of depression (20.4 and 18 respectively) and anxiety scales (20 and 18.5 respectively) were significantly higher in patients group than in controls (7.3 & 7 respectively for depression and 13.5 for both mean and median anxiety) $P < 0.001$ for both.

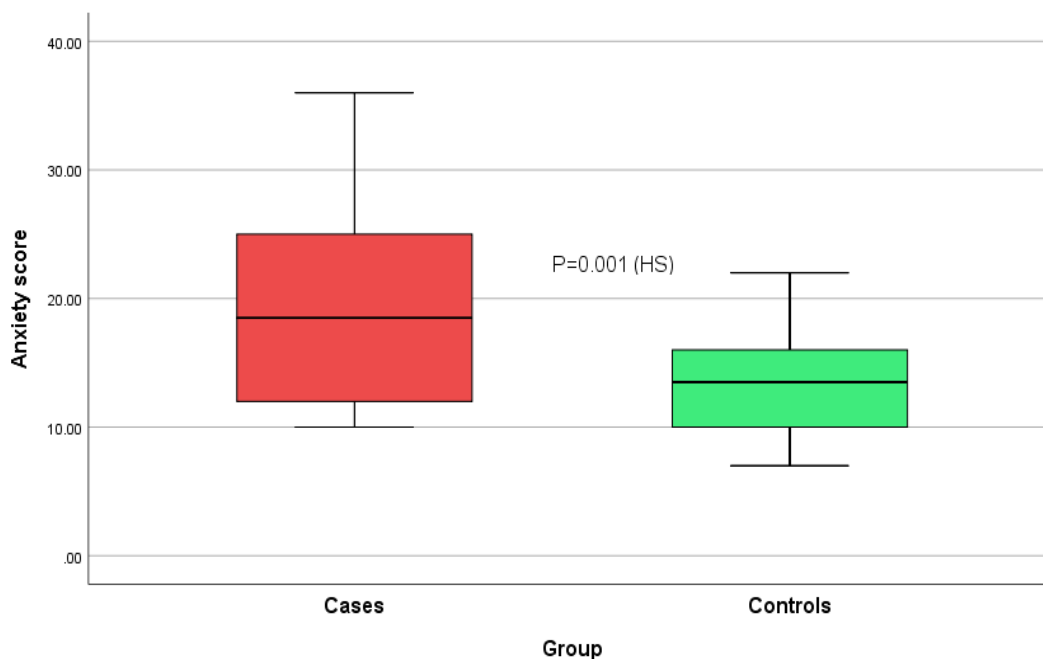


Fig. (13): Box plot showing median and range of anxiety score among the studied groups

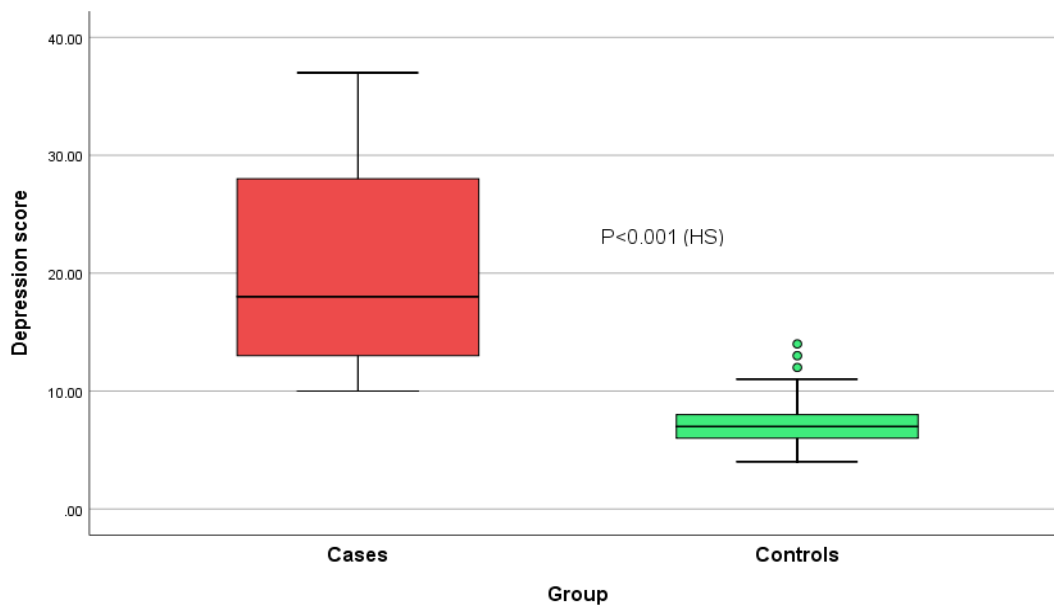


Fig. (14): Box plot showing median and range of depression score among the studied groups

Table (12): Comparing the studied groups regarding depression and anxiety grades

Variable		Patients (n=30)		Controls (n=30)		Fisher's exact test	P
		No.	%	No.	%		
Depression grade	No	0	0.0	26	86.7	55.4	<0.001 (HS)
	Mild	13	43.3	4	13.3		
	Moderate	9	30.0	0	0.0		
	Severe	8	26.7	0	0.0		
Anxiety grade	Mild	11	36.7	26	86.7	17.3	<0.001 (HS)
	Moderate	12	40.0	4	13.3		
	Severe	7	23.3	0	0.0		

This table illustrates that there was a significant difference between the studied groups regarding the depression and anxiety grades, where 86.7% of the controls had no depression compared to 0% of the patients, while only 13.3 % of them had mild grade compared to 43.3% of the patients. On the other hand, 30% and 26.7% of patients suffered moderate

and severe depression respectively compared to 0% of the controls. ($P < 0.001$).

Regarding anxiety, control group had mild anxiety at higher percentage (86.7%) than the patients (36.7%), while patients suffered moderate and severe anxiety (40% and 23.3% respectively) at higher percentages than the controls (13.3% and 0% respectively) these differences were significant ($P < 0.001$).

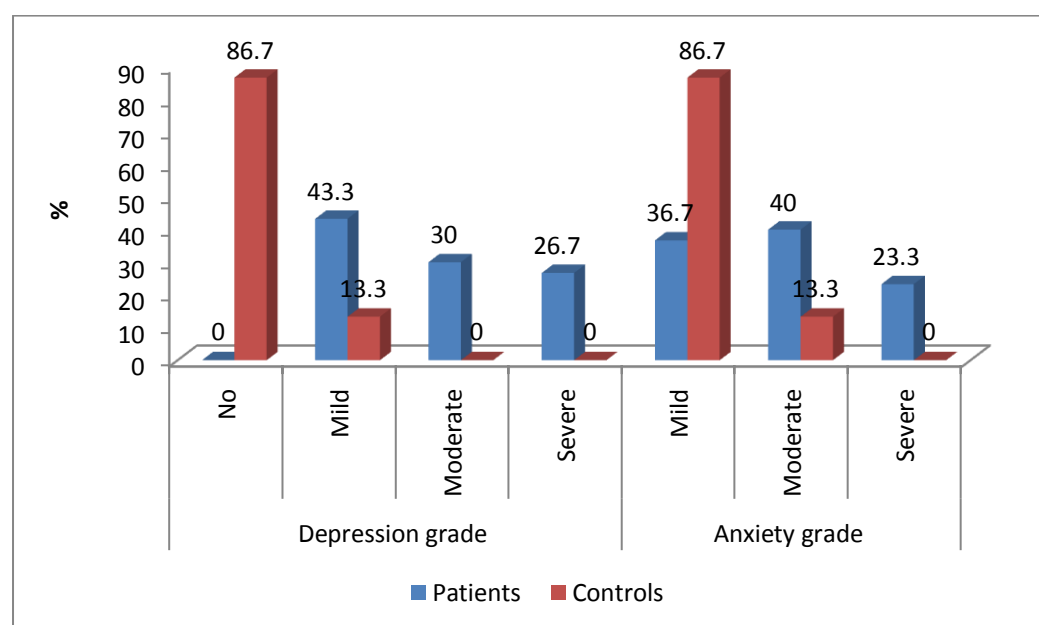
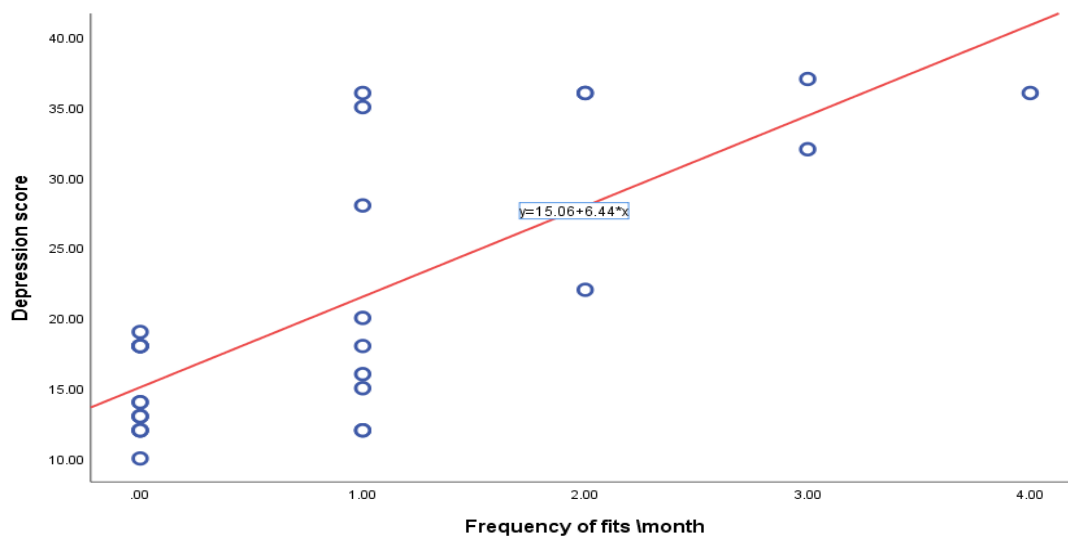


Fig. (15): Bar chart showing depression and anxiety grades among the studied groups
Underlying Factors of depression and anxiety among the studied patients

Table (13): Correlation between depression score and the studied continuous variables

With	Depression score	
	Patients group (N=30)	
	rho	P
Age (ys)	0.267	0.15
Socio-economic level	-0.599	<0.001 (HS)
Age of onset (ys)	-0.078	0.68
Duration (min)	0.742	<0.001 (HS)
Frequency/month	0.664	<0.001 (HS)
No. of drugs	0.766	<0.001 (HS)
Anxiety score	0.685	<0.001 (HS)

This table shows that there were significant positive correlation between depression scores and duration of fit, frequency/month, number of drugs and anxiety score ($P < 0.001$ for all). While there was a significant negative correlation between it and socio-demographic level ($P < 0.001$).



Scatter plot showing significant positive correlation between frequency and depression score, also shows the regression equation of both factors.

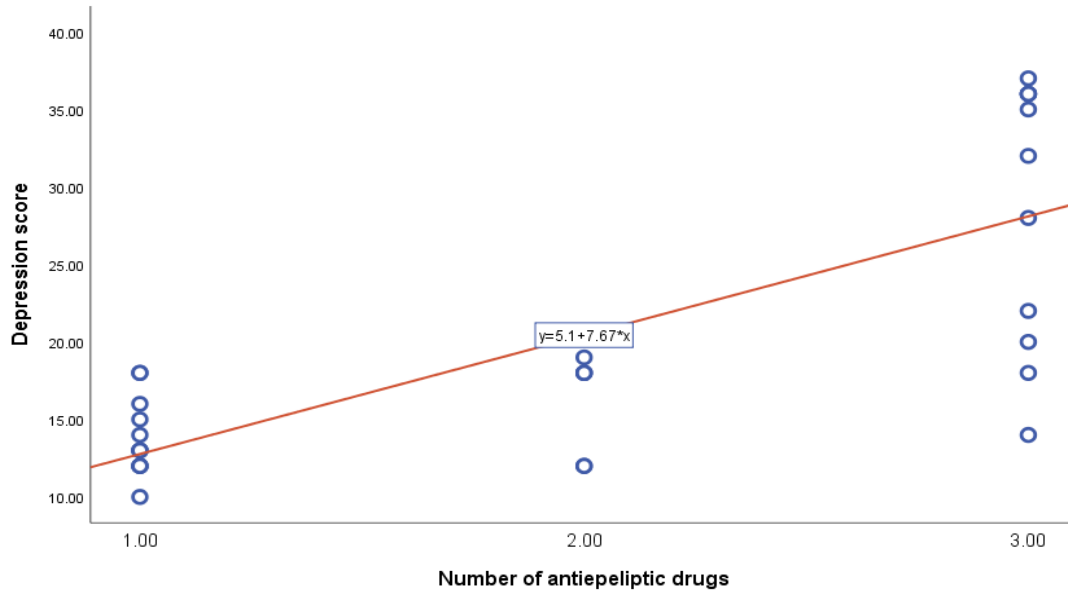


Fig. (16): Scatter plot showing significant positive correlation between no of drugs and depression score, also shows the regression equation of both factors.

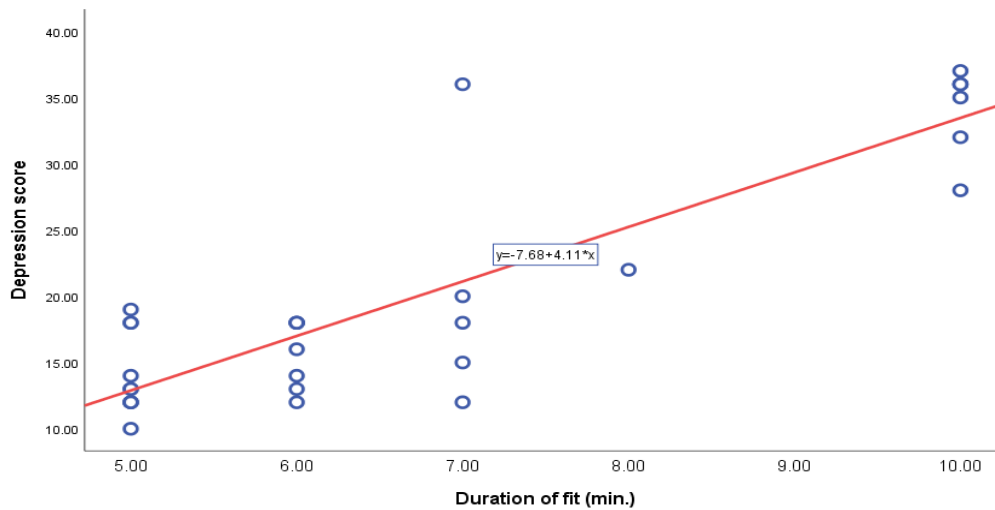


Fig. (17): Scatter plot showing significant positive correlation between duration of fit and depression score, also shows the regression equation of both factors.

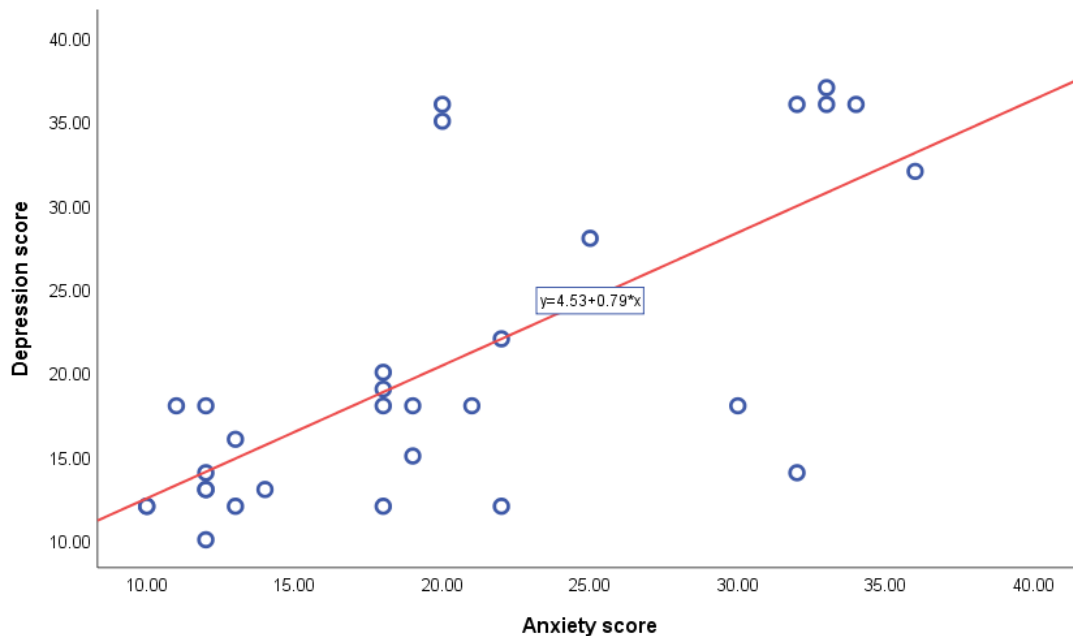
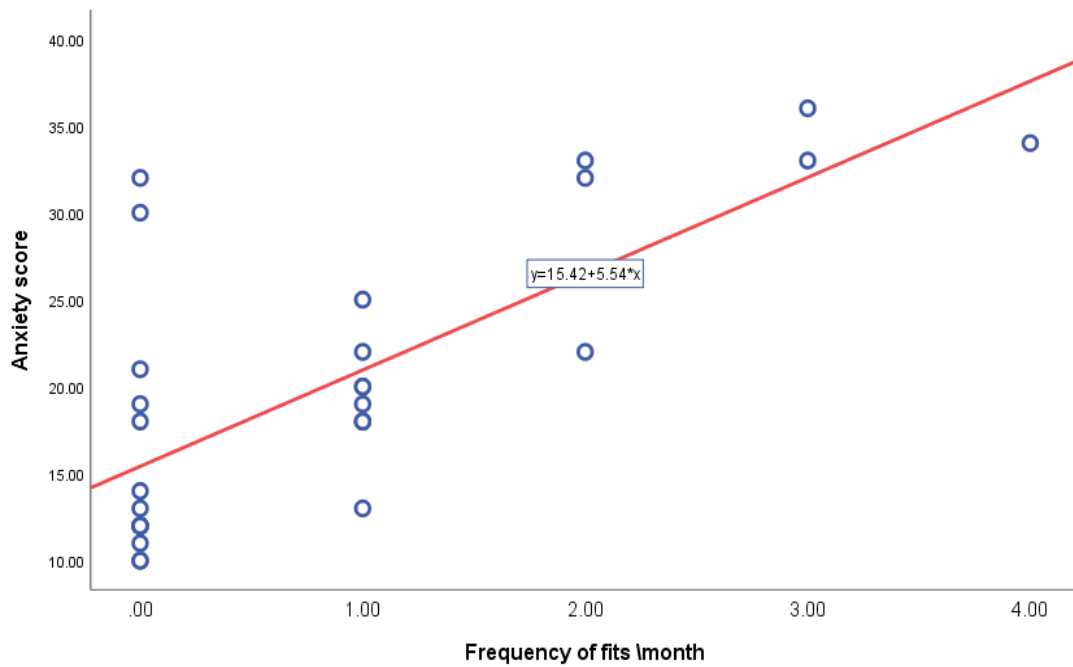
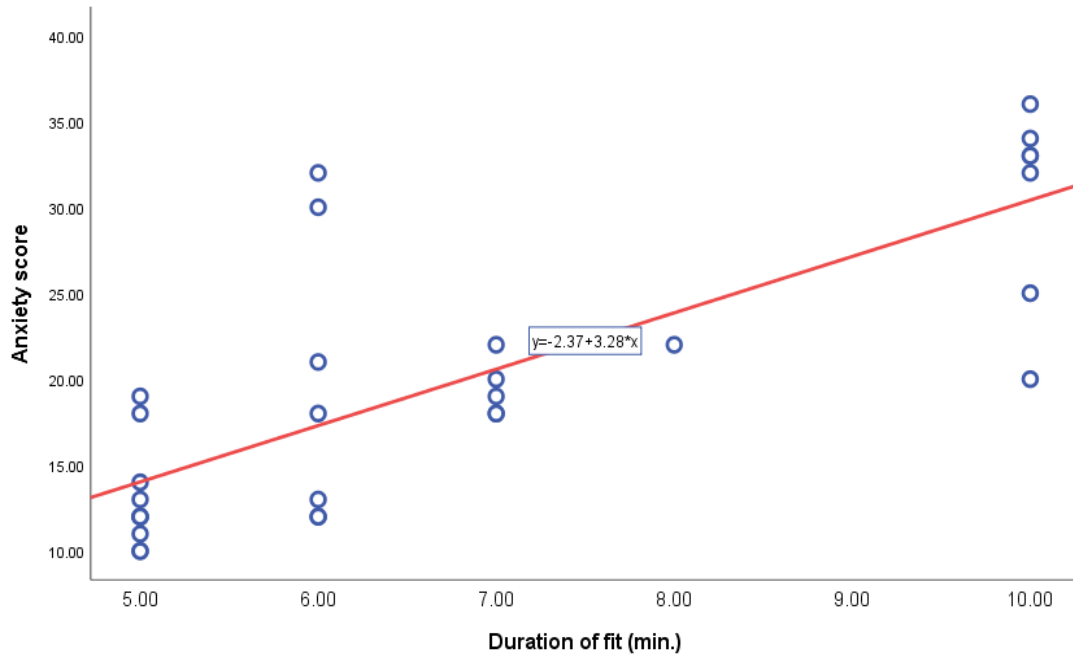


Fig. (18): Scatter plot showing significant positive correlation between anxiety and depression scores, also shows the regression equation of both factors.

Table (14): Correlation between depression score and the studied continuous variables

With	Anxiety score	
	Patients group (N=30)	
	rho	P
Age (ys)	0.386	0.035 (S)
Socio-economic level	-0.581	=0.001 (HS)
Age of onset (ys)	0.035	0.85
Duration (min)	0.794	<0.001 (HS)
Frequency/months	0.702	<0.001 (HS)
No. of drugs	0.811	<0.001 (HS)

This table shows that there were significant positive correlation between anxiety scores and age, duration of fit, frequency/month and number of drugs ($P < 0.001$ for all). While there was a significant negative correlation between it and socio-demographic level ($P < 0.001$).



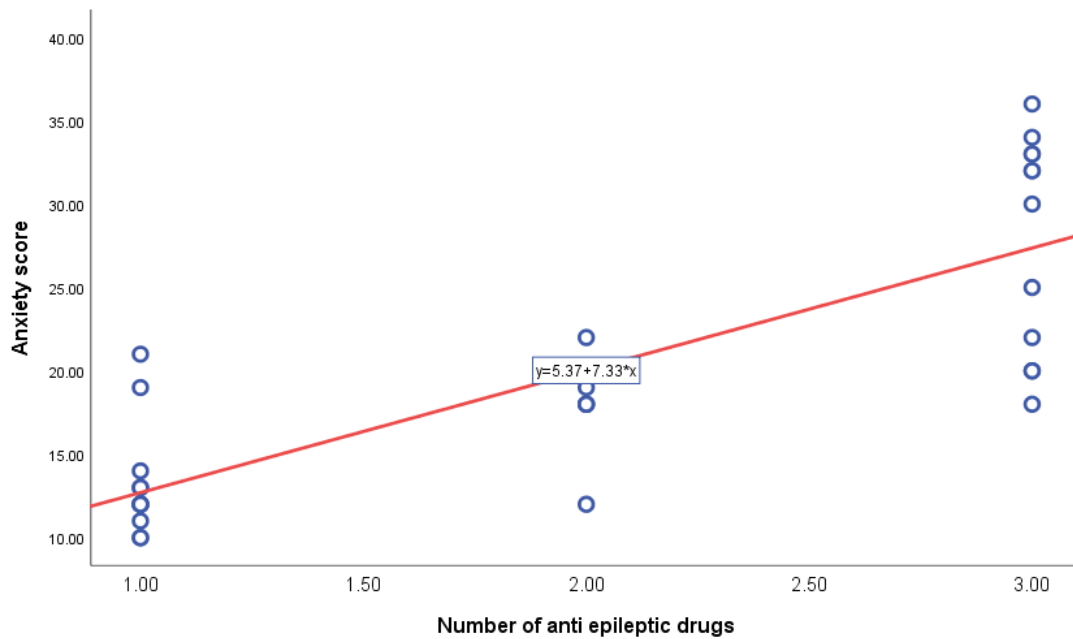


Fig. (19): Depression scores according to socio demographic factors

Table (15) : Depression scores according to socio demographic factors

Variable			Depression score		Z_{MWU}	P
			Median	Range		
Sex	Female	17	19	12-37	2.19	0.028 (S)
	Male	13	14	10-35		
Residence	Urban	18	15	10-28	3.18	0.001 (HS)
	Rural	12	33.5	13-37		
Family problems	No	16	14	10-35	2.74	0.006 (S)
	Yes	14	24	12-37		
Socio-economic level	Poor	13	22	14-37	3.23	0.001 (HS)
	Good	17	13	10-36		

This table demonstrates that the median depression scores were significantly higher among females than males (19 and 14 respectively), among rural (33.5) than urban residents (15.0), among individuals with family problems than those without (24 versus 14), among those with poor socio-economic level than those with good circumstances (22 versus 13). P values are < 0.05 for all variables.

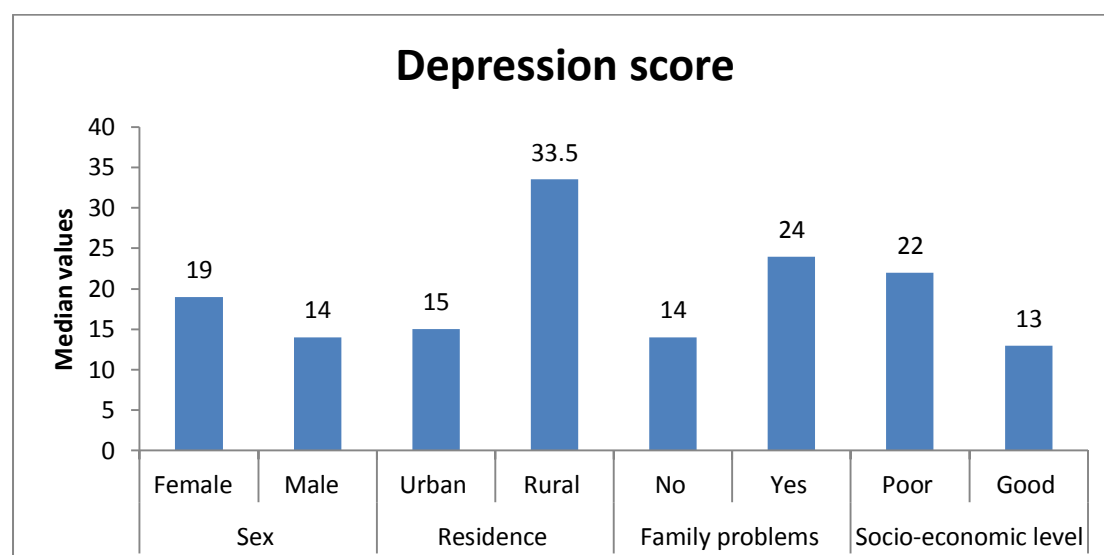


Fig. (20): Bar chart showing median values of depression score according to socio-demographic characters

Table (16): Anxiety scores according to socio demographic factors

Variable			Anxiety score		Z_{MWU}	P
			Median	Range		
Sex	Female	17	19	11-36	1.57	0.11 (NS)
	Male	13	18	10-30		
Residence	Urban	18	13.5	10-30	2.78	0.005 (S)
	Rural	12	26.0	12-36		
Family problems	No	16	13	10-22	3.34	=0.001 (HS)
	Yes	14	27.5	12-36		
Socio-economic level	Poor	13	25	12-36	3.13	0.002 (S)
	Good	17	13	10-32		

This table demonstrates that the median anxiety scores were significantly higher among rural than urban residents (26 versus 13.0), among individuals with family problems than those without (27.5 versus 13), among those with poor socio-economic level than those with good circumstances (25 versus 13). P values are < 0.05 for all variables. On the other hand there was no significant difference in the scores regarding sex ($P > 0.05$).

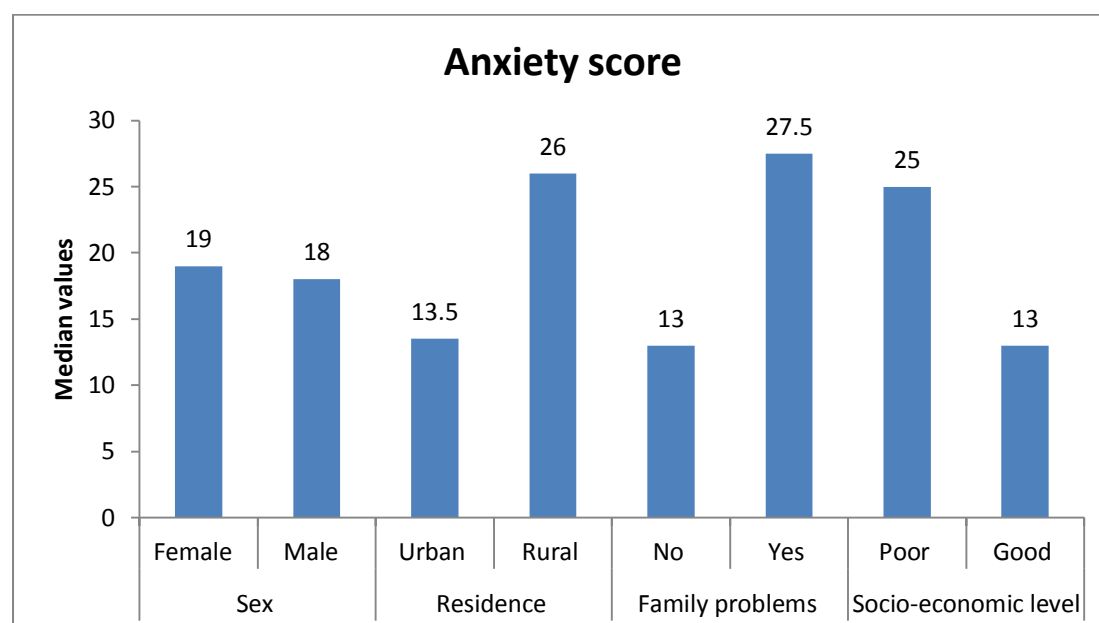


Fig. (21): Depression scores according to type of seizures and compliance to treatment

Table (17) : Depression scores according to type of seizures and compliance to treatment

Variable		n.	Depression score		KW test	P
			Median	Range		
Type of seizure	Absence	1	--	18-18	4.25	0.24 (NS)
	Complex partial	3	36	14-37		
	Focal	6	13	12-20		
	GTC	20	18	10-36		
Compliance to treatment	No	12	27	15-37	$Z_{MWU} = 3.22$	0.001 (HS)
	Yes	18	13.5	10-36		

This table demonstrates that the median depression scores were significantly lower among patients compliant to treatment than non (13.5

versus 27.0), (P=0.001). On the other hand there was no significant difference in the scores regarding type of seizures (P>0.05).

Table (18): Anxiety scores according to type of seizures and compliance to treatment

Variable	n.	Anxiety score		KW test	P
		Median	Range		
Type of seizure	Absence	1	--	6.84	0.077 (NS)
	Complex partial	3	20		
	Focal	6	15		
	GTC	20	20.5		
Compliance to treatment	No	12	21.5	Z _{MWU} =2.83	0.005 (HS)
	Yes	18	13.5		

This table demonstrates that the median anxiety scores were significantly lower among patients compliant to treatment than non (13.5 versus 21.5), (P=0.005). On the other hand there was no significant difference in the scores regarding type of seizures (P>0.05).

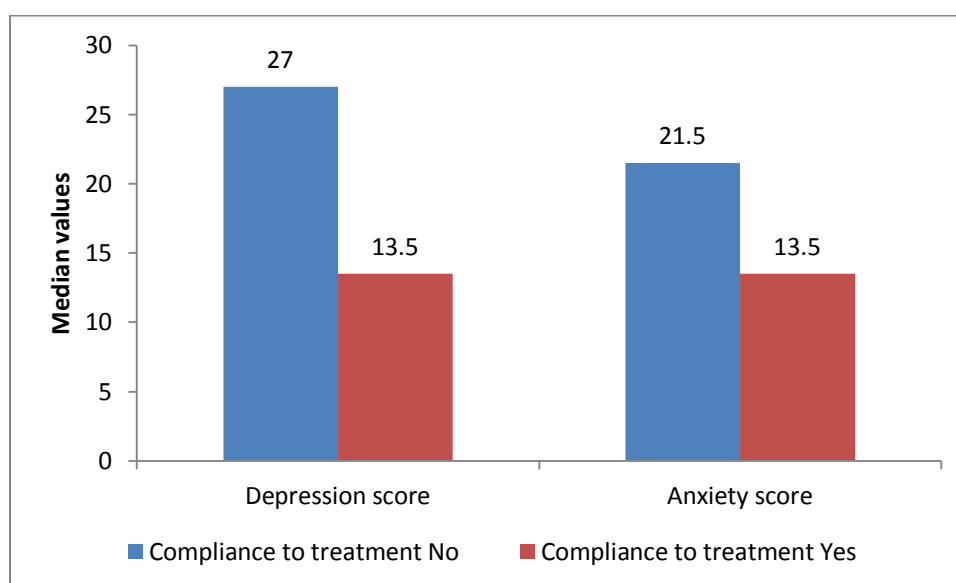


Fig.(22): Bar charts showing the median values of depression and anxiety scores according to compliance to treatment

Table (19): Consequences of depression and anxiety among the studied patients Relation between depression grade and attention deficit

			depression grade			Total	FET (P)
			Mild	Moderate	Severe		
Attention deficit	No	Count	10	6	0	16	13.08 (0.002, S)
		%	76.9%	66.7%	0.0%	53.3%	
	Yes	Count	3	3	8	14	
		%	23.1%	33.3%	100.0%	46.7%	
Total		Count	13	9	8	30	
		%	100.0%	100.0%	100.0%	100.0%	

This table shows that there was a significant association between grade of depression among patients and attention deficit, where 76.9% and 66.7% of patients with mild and moderate depression did not have attention deficit while 100% of those with severe depression suffered from attention deficit ($P < 0.05$).

Table (20): Relation between anxiety grade and attention deficit

			Anxiety grade			Total	FET (P)
			Mild	Moderate	Severe		
Attention deficit	No	Count	10	6	0	16	14.8 (< 0.001 , HS)
		%	90.9%	50.0%	0.0%	53.3%	
	Yes	Count	1	6	7	14	
		%	9.1%	50.0%	100.0%	46.7%	
Total		Count	11	12	7	30	
		%	100.0%	100.0%	100.0%	100.0%	

This table shows that there was a significant association between grade of anxiety among patients and attention deficit, where 90.9% and 50% of patients with mild and moderate depression did not have attention deficit while 100% of those with severe anxiety suffered from attention deficit ($P < 0.05$).

Table (21): Relation between depression grade and school attendance

			Depression grade			Total	FET (P)
			Mild	Moderate	Severe		
School attendance	irregular	Count	4	3	7	14	7.09 (0.03,S)
		%	30.8%	33.3%	87.5%	46.7%	
	Regular	Count	9	6	1	16	
		%	69.2%	66.7%	12.5%	53.3%	
Total		Count	13	9	8	30	
		%	100.0%	100.0%	100.0%	100.0%	

This table shows that there was a significant association between grade of depression among patients and school attendance, where 87.5% of patients with severe depression attended school irregularly compared to 30.8% and 33.3% of those with mild and moderate grades respectively. ($P < 0.001$).

Table (22): Relation between anxiety grade and school attendance

			anxiety grade			Total	FET (P)
			Mild	Moderate	Severe		
School attendance	irregular	Count	2	7	5	14	5.8 (0.06, NS)
		%	18.2%	58.3%	71.4%	46.7%	
	Regular	Count	9	5	2	16	
		%	81.8%	41.7%	28.6%	53.3%	
Total		Count	11	12	7	30	
		%	100.0%	100.0%	100.0%	100.0%	

This table shows that there was an insignificant association between grade of anxiety among patients and school attendance, ($P > 0.05$).

Table (23): Relation between depression grade and school achievements

			Depression grade			Total	FET (P)
			Mild	Moderate	Severe		
School achievement	Poor	Count	2	1	7	10	13.1 (0.002, S)
		%	15.4%	11.1%	87.5%	33.3%	
	Adequate	Count	10	7	1	18	
		%	76.9%	77.8%	12.5%	60.0%	
	Good	Count	1	1	0	2	
		%	7.7%	11.1%	0.0%	6.7%	
Total		Count	13	9	8	30	
		%	100.0%	100.0%	100.0%	100.0%	

This table shows that there was a significant association between grade of depression among patients and school achievement, where 87.5% of patients with severe depression reported poor achievement compared to only 15.5% and 11.1% of those with mild and moderate depression ($P < 0.05$).

Table (24): Relation between anxiety grade and school achievements

			anxiety grade			Total	FET (P)
			Mild	Moderate	Severe		
School achievement	Poor	Count	1	4	5	10	7.3 (0.075, NS)
		%	9.1%	33.3%	71.4%	33.3%	
	Adequate	Count	9	7	2	18	
		%	81.8%	58.3%	28.6%	60.0%	
	Good	Count	1	1	0	2	
		%	9.1%	8.3%	0.0%	6.7%	
Total		Count	11	12	7	30	
		%	100.0%	100.0%	100.0%	100.0%	

This table shows that there was a statistically non significant association between grade of anxiety among patients and school achievement, ($P > 0.05$).

Table (25): Relation between depression grade and having friends

			Depression grade			Total	FET (P)
			Mild	Moderate	Severe		
Having friends	No	Count	3	2	7	12	9.65 (0.009, S)
		%	23.1%	22.2%	87.5%	40.0%	
	Yes	Count	10	7	1	18	
		%	76.9%	77.8%	12.5%	60.0%	
Total		Count	13	9	8	30	
		%	100.0%	100.0%	100.0%	100.0%	

This table shows that there was a significant association between grade of depression among patients and having friends, where 87.5% of patients with severe depression did not have friends compared to only 23.1% and 22.2% of those with mild and moderate depression ($P < 0.05$).

Table (26): Relation between anxiety grade and having friends

			anxiety grade			Total	FET (P)
			Mild	Moderate	Severe		
Having friends	No	Count	2	3	7	12	13.5 (0.002, S)
		%	18.2%	25.0%	100.0%	40.0%	
	Yes	Count	9	9	0	18	
		%	81.8%	75.0%	0.0%	60.0%	
Total		Count	11	12	7	30	
		%	100.0%	100.0%	100.0%	100.0%	

This table shows that there was a significant association between grade of anxiety among patients and having friends, where all patients with severe anxiety did not have friends compared to only 18.2% and 25% of those with mild and moderate grades ($P < 0.05$).

Table (27): Relation between depression grade and social withdrawal

			depression grade			Total	FET (P)
			Mild	Moderate	Severe		
Social withdrawal	No	Count	10	6	0	16	13.08 (0.002, S)
		%	76.9%	66.7%	0.0%	53.3%	
	Yes	Count	3	3	8	14	
		%	23.1%	33.3%	100.0%	46.7%	
Total		Count	13	9	8	30	
		%	100.0%	100.0%	100.0%	100.0%	

This table shows that there was a significant association between grade of depression among patients and social withdrawal, where 76.9% and 66.7% of patients with mild and moderate depression did not suffer social withdrawal, while 100% of those with severe depression suffered from it ($P < 0.05$).

Table (28): Relation between anxiety grade and social withdrawal

			Anxiety grade			Total	FET (P)
			Mild	Moderate	Severe		
Social withdrawal	No	Count	11	5	0	16	19.9 (< 0.001 , HS)
		%	100.0%	41.7%	0.0%	53.3%	
	Yes	Count	0	7	7	14	
		%	0.0%	58.3%	100.0%	46.7%	
Total		Count	11	12	7	30	
		%	100.0%	100.0%	100.0%	100.0%	

This table shows that there was a significant association between grade of anxiety among patients and social withdrawal, where all patients with severe anxiety grade had social withdrawal compared to 0% and 58.3% of those with mild and moderate grades respectively. ($P < 0.001$).

Table (29): Relation between depression grade and physical exercise

			depression grade			Total	FET (P)
			Mild	Moderate	Severe		
Physical exercise	No	Count	6	3	8	17	9.13 (0.01,S)
		%	46.2%	33.3%	100.0%	56.7%	
	Yes	Count	7	6	0	13	
		%	53.8%	66.7%	0.0%	43.3%	
Total		Count	13	9	8	30	
		%	100.0%	100.0%	100.0%	100.0%	

This table shows that there was a significant association between grade of depression among patients and physical exercise, where all patients with severe depression grade did not practice exercise compared to 46.2% and 33.3% of those with mild and moderate grades respectively, (P=0.01).

Table (30): Relation between anxiety grade and physical exercise

			anxiety grade			Total	FET (P)
			Mild	Moderate	Severe		
Physical exercise	No	Count	3	8	6	17	6.39 (0.045, S)
		%	27.3%	66.7%	85.7%	56.7%	
	Yes	Count	8	4	1	13	
		%	72.7%	33.3%	14.3%	43.3%	
Total		Count	11	12	7	30	
		%	100.0%	100.0%	100.0%	100.0%	

This table shows that there was a significant association between grade of anxiety among patients and physical exercise, where 66.7% and 85.7% of patients with moderate and severe grades did not practice exercise compared to only 27.3% of those with mild grades respectively, (P<0.05).

Discussion

Epilepsy is the most common neurological disorder in children and adolescence. Childhood and Adolescence are periods of highly sensitive psychophysical development, and it involves intense changes in the biological, social, and psychological domains that can be especially challenging for children and adolescents with epilepsy.(*Kwong et al.,2016*)

Anxiety and depression in children and adolescents with epilepsy are common comorbidities which place a significant burden on patients and families and complicate the clinical management of epilepsy. Chronic illness itself can lead to anxiety and depression. This may be symptomatic of the illness itself; duration of illness, a consequence of the course and management of the illness, leading to a sense of lack of control and uncertainty or hopelessness and helplessness from poor prognoses; an increasing fear of death; stigma and ostracism from peers; overprotective parental behaviour; and side effects from treatment.(*Pinquart et al.,2011*)

Several factors have been found to be associated with increased risk of depression in the epileptic children.lower educational status, lower monthly income, frequency of seizure, polytherapy, the side effects of,anti-epileptic drugs and difficulties adhering to AEDs have all been found to be prominent risk factors for depression and anxiety.(*Tilahune et al., 2016*)

The broad goal of psychologists is to maximize quality of life and psychosocial functioning of epileptic patient. To do so, a psychologist must take into consideration the potential influences of the youth's

seizures, antiepileptic medication, compliance/adherence to medical treatment, familial/social environment, and comorbid mood/behavioral difficulties when considering diagnosis and course of treatment. **(Michaelis et al. 2016).**

it can be helpful to provide psychoeducation to families about the role of stress in everyday life, and the negative effects that avoidance can have on helping their child to learn effective stress management strategies. The goal of this psychoeducation is to shift families from a focus on avoiding stress to a goal of appropriately and effectively responding to stress using active coping strategies. Cognitive-behavioral therapy (CBT) techniques can be effective in helping families learn new strategies for responding to both everyday stress and stress specific to seizure activity. It has also been suggested that relaxation techniques such as visualization and deep breathing are useful tools when managing anxiety during pre-ictal or early ictal sensation. *(Michaelis et al. 2016).*

The objective of our study was to assess the presence, possible factors related and consequences of psychological disorders in children with epilepsy

This cross-sectional study was done on 30 epileptic children in neurological unit of the pediatric department at Benha university hospital and another 30 healthy children as a control group from February 2020 to June 2020.

This study group study has female predominance about 56.7% and that is in agreement with **Cianchetti et al.,2018** and **Vega et al.,2011** in which their study group has also female predominance represent 52.5%,66% respectively.

The mean age among patient group of our study is 13.0 years \pm 2.0 and that in agreement with **Stevanovic et al.,2011** in which mean age for his study group was 13.85 years \pm 2.76 And also in agreement with **Baki et al., 2004** in which mean age was 12.8 \pm 3.6

Our study demonstrated that 66.75% of study group patient suffered from GTC followed by simple partial seizures 20% fits.

This is in concordance with the results from **Schraegle et al., 2017** patients who suffered from generalized type was 35.6% as(Frontal lobe (31.8%) Temporal lobe (32.6%),Unlike the study **Stevanovic et al., 2011** and **Baki et al., 2004** showed partial seizures predominate(50%), 74.3% respectively.

In our study, mean of duration of seizures among patient study group is 6.8 minutes \pm 1.9, unlike **Stevanovic et al.,2011** and **Baki et al., 2004** in which their mean of duration of seizures were 5.24 ,2.4 minutes respectively.

Our study is in contrast with **Schraegle et al., 2017** in which Most patients experienced seizure onset at an early age (mean age = 5.64 years, SD = 4.23), in the other hand the age of onset among patient group in our study (mean age = 10.1, SD=1.94) ,also there is difference in frequency of seizures between two studies as **in our study**, 50% of patient suffered from monthly seizures(30%one fit per month, 10% had 2 fits, 6.7% had three and only one case (3.3%) had the fits 4 times per month).while **Schraegle et al., 2017** only 28% had monthly seizures due to lower socio-economic, Not committed to treatment and negligence of some parent who ignore regular visit to neurological clinic o adjust dose of AEDs .

In our study 56.7% of patient group showed good socioeconomic status and this in agreement with **Baki et al., 2004** in which 71.4 % of patient group have good socioeconomic status.

Our study revealed that 60% of our patient group was on polytherapy in contrast with **Cianchetti et al.,2018** and **Nathália et al., 2015**

In which 79.1%, 55% of patient group was on monotherapy respectively.

Most used drug among our studied patient group is tegretol 68.7% in contrast with **Cianchetti et al.,2018** valporate was most used drug 48.8%.

This study demonstrated regarding to anxiety score among all studied cases with Mean +SD was $20 \pm 8,23$ Regarding anxiety grade, mild were 11 (36.7%), moderate were 12 (40%) and sever were 7 (23.3%).

In our study age of patient studied group is in positive correlation with anxiety score(p value is lower than.035) and that **in agreement** with **Kwong et al., 2015** in which p value was.028, and **in contrast** with **Schraegle et al., 2017** and **LaGrant et al., 2019** in which their result show no significant correlation between age of patient and anxiety score that may be due to older children became more oriented to seizures and its impact on their life style or fear of that their seizures may witnessed by their friends which may lead to have Compassionate treatment as they thought.

In our study duration of seizures is in positive correlation with anxeity score and It's p value 0.001 and that in agreement with **Schraegle**

etal., 2017 in which p value 0.01. **in contrast** with our study **LaGrant etal., 2019** and **Kwong etal., 2015** who found That there is significant correlation between anxiety and depression score and both p value are 0.001.

Sometimes psychiatric symptoms are an emotional reaction to epilepsy. Frustration and discouragement about persistent seizures can lead to depression. Agoraphobia can develop from fear of having a seizure in a public place. But there is also evidence that dysfunction in the same brain structures can cause both seizures and interictal dysphoric disorder.

A condition called interictal dysphoric disorder occurs in some patients with epilepsy. The definition includes eight symptoms, of which the patient must have at least three: depression, lack of energy, pain, irritability, anxiety, fear, and, oddly, euphoria. The disorder is usually milder than major depression but treated similarly. Whether to blame it on epilepsy is a matter of dispute.

Disputed effect of epilepsy is the interictal behavior syndrome. People with this presumed disorder a minority of epileptic patients seem to feel everything, but especially sadness and fear, more intensely. They often have strong religious and philosophical interests and read deep significance into events and experiences that others regard as unremarkable. They are easily provoked to anger, especially by moral indignation. Other common symptoms are humorlessness, lack of interest in sex, an obsession with details, compulsive writing, and difficulty in ending conversations and other social encounters. Some specialists believe these personality traits develop when repeated focal seizures alter

brain function. Others suspect that a common underlying brain abnormality causes both the epilepsy and the behavioral syndrome.

This study demonstrated regarding to depression score among all studied cases with Mean +SD was 20.4 ± 9.19 Regarding depression grade, mild were 13 (43.3%), moderate were 9 (30%) and severe were 8 (26.7%).

In our study the median depression scores were significantly higher among females than males with p value.028 in contrast with **Kwong et al., 2015** in which demonstrate that there is no significant correlation between them.

In our study the number of anti-epileptic drugs used by patient studied group is in positive correlation with depression score (p value of our study is lower than.001) and that in agreement with **Schraegle et al., 2017** and **Kwong et al., 2015** in which p value more than.01,.005 respectively.

The antiepileptic drugs most closely associated with acute depression on initiation of treatment are vigabatrin, phenobarbitone, and topiramate. Depression with topiramate may be linked to abrupt cessation of seizures or drug toxicity. Patients starting tiagabine may develop symptoms of agitation, withdrawal, and mood disturbance suggestive of depression. so, depression is reported with tiagabine

Positive psychotropic effects of antiepileptic drugs are seen in many psychiatric disorders: carbamazepine and lamotrigine are used for bipolar disorders and valproate for acute mania. Unfortunately there is no good evidence to show that these drugs lift mood in epilepsy patients

Acute antiepileptic drug related depression beginning shortly after initiation of treatment resolves promptly in the majority of cases. Warning patients, their carers, and GPs of this potential side effect is important as is discontinuation of the offending drug. There is a dearth of evidence on the outcome of treatment of inter-ictal depression in epilepsy, but the sertraline study referred to above showed that 54% of 100 patients had total remission of their psychiatric symptoms. Since patients with depression attending neurology outpatients as a whole appear to have persistent depression, this result is encouraging and should prompt us actively to seek out and treat symptoms of depression in our patients. (**Carson.,2003**)

In our study anxiety score is in positive correlation with depression score and its p value is lower than.001 and that in agreement with **Kwong et al., 2015** and **LaGrant et al., 2019** in which their p value more than.003, lower than.001 respectively.in agreement with **Kwong et al., 2015** results shows there is positive correlation between duration of seizures and depression score p value of our study is lower than.001while p value of of other study is.005.

Depression and anxiety are frequently comorbid, and in clinical practice it is difficult to evaluate them separately since both involve negative affective symptoms. (**Olino et al.,2008**)

Therefore, the close relationship between depression and anxiety may be caused not only by their frequent comorbidity but also by the negative affective symptoms that they share. The importance of the comorbid occurrence of primary depression and anxiety disorders has led to specifiers for anxiety being were added to the diagnostic criteria for

depressive disorders included in the fifth edition of the Statistical Manual of Mental Disorders (DSM-5).(**Arlington., 2013**)

Our Results revealed that seizures frequency significantly correlated with anxiety and depression score(p value <.001) in contrast with **Kwong et al., 2015** who found that only depression score affected by seizures frequency with (p value.003).

Seizure frequency has been linked to psychological disturbances in a number of relevant studies. With regard to depression, Boylan.,et al 2004reported that 50% of inpatients undergoing video-EEG telemetry suffered from depression with 19% exhibiting suicidal ideation. Jacoby et al.,1996 observed in a community-based survey that 21% of patients with recurrent seizures were depressed versus 9% of controlled subjects and O'Donoghue et al.,1999 have similarly demonstrated at primary care level that 33% of patients with recurrent seizures versus 6% of those in remission had probable depression. Overall, the prevalence of depression has been reported to range from 20 to 55% in pharmacoresistant populations versus 3–9% in well controlled subjects as in Gilliam et al.,2004 Parenthetically, the occurrence of depression in epileptic patients, particularly those with high seizure counts, might seem paradoxical as one of the most powerful treatments for depression is electroconvulsive therapy, which is entirely based on the tenet of the anti depressive effects of convulsions.

Our Results are in agreement with **Schraegle et al., 2017** who revealed that no significant correlation between seizure type (i.e., generalized versus focal) with anxiety and depression score in contrast with **Thomé-Souza et al.,2004** who demonstrated that, in comparison to

generalized seizures, focal seizures were associated with a higher risk of ADHD and Depression in children.

Depression in epilepsy may be linked temporally to seizures, but the most common disorder is that of inter-ictal depression. In addition to the recognised symptoms of anhedonia (lack of enjoyment), reduced appetite, poor energy, and sleep disturbance, inter-ictal depression or dysphoria is more likely to be associated with agitation and psychotic features or impulsive self-harm than is depression in people without epilepsy; a fact worth remembering when faced with a restless or truculent patient in the clinic.

Pre-ictal depression may appear hours before a seizure; if this pattern can be recognised a short acting benzodiazepine such as clobazam may be used to abort seizures. Ictal depression is rare, much less common than ictal fear or anxiety, but can be profound. (**Lambert.,1999**)

Summary

Epilepsies are chronic neurological disorders in which clusters of nerve cells, or neurons, in the brain sometimes signal abnormally and cause seizures. Epilepsy is a disorder of the brain defined by any of the following conditions: At least two unprovoked (or reflex) seizures occurring more than 24 hours apart ,One unprovoked (or reflex) seizure and a probability of further seizures similar to the general recurrence risk (at least 60%) after two unprovoked seizures, occurring over the next 10 years.

During a seizure, many neurons fire (signal) at the same time – as many as 500 times a second, much faster than normal. epileptic seizure, is defined as period of symptoms due to abnormally excessive or synchronous neuronal activity in the brain. Outward effects vary from uncontrolled shaking movements involving much of the body with loss of consciousness (tonic-clonic seizure), to shaking movements involving only part of the body with variable levels of consciousness (focal seizure), to a subtle momentary loss of awareness (absence seizure).[3] Most of the time these episodes last less than 2 minutes and it takes some time to return to normal. Loss of bladder control may occur.

Epilepsy affects patients of all ages, races, and ethnic backgrounds. According to the National Institute of Neurological Diseases and Stroke (NINDS), about 2.3 million adults and more than 450,000 children and adolescents in the United States have epilepsy.

Children with epilepsy also have a higher risk of developing depression and/or attention deficit hyperactivity disorder compared with their peers. Behavioral problems may precede the onset of seizures in some children. They are especially vulnerable to the emotional problems

caused by ignorance or the lack of knowledge among others about epilepsy.

Our result revealed that:

The mean and median values of depression (20.4 and 18 respectively) and anxiety scales (20 and 18.5 respectively) were significantly higher in patients group than in controls (7.3 & 7 respectively for depression and 13.5 for both mean and median anxiety) $P < 0.001$ for both.

There was a significant difference between the studied groups regarding the depression and anxiety grades, where 86.7% of the controls had no depression compared to 0% of the patients, while only 13.3 % of them had mild grade compared to 43.3% of the patients. On the other hand, 30% and 26.7% of patients suffered moderate and severe depression respectively compared to 0% of the controls. ($P < 0.001$).

Regarding anxiety, control group had mild anxiety at higher percentage (86.7%) than the patients (36.7%), while patients suffered moderate and severe anxiety (40% and 23.3% respectively) at higher percentages than the controls (13.3% and 0% respectively) these differences were significant ($P < 0.001$).

There were significant positive correlation between depression scores ,anxiety score and duration of fit, frequency/month, number of drugs and anxiety score ($P < 0.001$ for all). While there was a significant negative correlation between it and socio-demographic level ($P < 0.001$).

Our study demonstrates that the median anxiety and depression scores were significantly higher among rural than urban residents among individuals with family problems than those without, among those with

poor socio-economic level than those with good circumstances). P values are < 0.05 for all variables . females were higher than males *in depression score* but, On the other hand there was no significant difference in the anxiety score regarding sex our study revealed that the median anxiety and depression scores were significantly lower among patients compliant to treatment than non .On the other hand there was no significant difference in the scores regarding type of seizures ($P>0.05$).

This study shows that there was a significant association between grade of depression and anxiety among patients and attention deficit Also, shows that there was a significant association between grade of depression an anxiety among patients and school attendance.

Conclusion

Our study concluded that

- Depression scores were higher among females, among rural residents, among individuals with family problems, and among those with poor socio-economic level.
- Anxiety scores were significantly higher among rural residents, among individuals with family problems, among those with poor socio-economic level.
- Both depression scores and Anxiety scores were lower among patients compliant to treatment.
- There was association between grade of depression and grade of anxiety among patients and attention deficit, school attendance, school achievement, having friends, and physical exercise.

Recommendations

- Any child diagnosed with epilepsy should be psychological assessment.
- Compliance on antiepileptic drugs very important.
- Parents of children with epilepsy should be trained to deal with their children and keep family problems away from children.
- Physical exercise help patients to deal with stress and depression.

Study Limitations:

1. The results were obtained from a single medical center, with a rather small sample size (30 patients).
2. Due to pandemic the questionnaire was delayed .
3. Few parents denied any negligence or family problems that may affect psychology of their children.

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الملخص العربي

الصرع عبارة عن اضطرابات عصبية مزمنة حيث تشير أحياناً مجموعات من الخلايا العصبية أو الخلايا العصبية في الدماغ بشكل غير طبيعي وتسبب نوبات. الصرع هو اضطراب في الدماغ يتم تحديده من خلال أي من الحالات التالية: نوبتان على الأقل غير مستقرتين (أو نوبات انعكاسية) تحدث بفارق يزيد عن ٢٤ ساعة ، نوبة غير مستثارة (أو انعكاسية) واحتمال حدوث نوبات أخرى مشابهة لخطر التكرار العام (٦٠٪ على الأقل) بعد نوبتين غير مستثارتين ، تحدث على مدى السنوات العشر القادمة.

أثناء النوبة ، تطلق العديد من الخلايا العصبية (إشارة) في نفس الوقت - ما يصل إلى ٥٠٠ مرة في الثانية، أسرع بكثير من المعتاد. تُعرف نوبة الصرع بأنها فترة من الأعراض بسبب النشاط العصبي المفرط أو المتزامن بشكل غير طبيعي في الدماغ. تختلف التأثيرات الخارجية من حركات الاهتزاز غير المنضبطة التي تنطوي على الكثير من الجسم مع فقدان الوعي (نوبة التوتر الارتجاجية) ، إلى حركات الاهتزاز التي تشمل جزءاً فقط من الجسم بمستويات متغيرة من الوعي (النوبة البؤرية) ، إلى فقدان الوعي اللحظي الدقيق (نوبة غيابية). تستمر هذه النوبات في معظم الأحيان أقل من دقيقتين وتستغرق بعض الوقت للعودة إلى وضعها الطبيعي. قد يحدث فقدان السيطرة على المثانة.

يؤثر الصرع على المرضى من جميع الأعمار والأجناس والخلفيات العرقية. وفقاً للمعهد الوطني للأمراض العصبية والسكتة الدماغية (NINDS) ، يعاني حوالي ٢,٣ مليون بالغ وأكثر من ٤٥٠,٠٠٠ طفل ومراهق في الولايات المتحدة من الصرع.

الأطفال المصابون بالصرع لديهم أيضاً مخاطر أعلى للإصابة بالاكتئاب و / أو اضطراب فرط الحركة ونقص الانتباه مقارنة بأقرانهم. قد تسبق المشكلات السلوكية ظهور النوبات عند بعض الأطفال. هم معرضون بشكل خاص للمشاكل العاطفية التي يسببها الجهل أو نقص المعرفة من بين آخرين حول الصرع.

وقد اوضحت دراستنا ما يلي:

ان القيم المتوسطة والوسيطه للاكتئاب (٤,٢٠ و ١٨ على التوالي) ومقياس القلق (٢٠ و ١٨,٥ على التوالي) أعلى بشكل ملحوظ في مجموعة المرضى عنها في المجموعة الضابطة

(٧,٣ و ٧ على التوالي للاكتئاب و ١٣,٥ لكل من القلق المتوسط والمتوسط) $P < 0.001$ لكليهما.

كان هناك فرق معنوي بين المجموعات المدروسة فيما يتعلق بدرجات الاكتئاب والقلق ، حيث لم يكن لدى ٨٦,٧٪ من المجموعة الضابطة الاكتئاب مقارنة بـ ٠٪ من المرضى ، بينما كان ١٣,٣٪ منهم فقط بدرجات خفيفة مقارنة بـ ٤٣,٣٪ من المرضى. من ناحية أخرى ، عانى ٣٠٪ و ٢٦,٧٪ من المرضى من اكتئاب متوسط وشديد على التوالي مقارنة بـ ٠٪ من مجموعة الشواهد ($P < 0.001$).

المجموعة الضابطة قلق خفيف بنسبة أعلى (٨٦,٧٪) من المرضى (٣٦,٧٪) ، بينما عانى المرضى من قلق معتدل وشديد (٤٠٪ و ٢٣,٣٪ على التوالي) بنسب أعلى من المجموعة الضابطة (١٣,٣٪ و ٠٪). على التوالي) كانت هذه الاختلافات معنوية ($P > 0,001$).

كانت هناك علاقة ارتباط موجبة معنوية بين درجات الاكتئاب ودرجة القلق ومدة اللياقة والتكرار خلال الشهر وعدد الأدوية ودرجة القلق ($P > 0,001$ للجميع). بينما توجد علاقة ارتباط سلبية معنوية بينه وبين المستوى الاجتماعي والديموغرافي ($P > 0,001$).

توضح دراستنا أن متوسط درجات القلق والاكتئاب كانت أعلى بشكل ملحوظ بين سكان الريف منها بين سكان الحضر وبين الأفراد الذين يعانون من مشاكل أسرية من أولئك الذين ليس لديهم مستوى اجتماعي واقتصادي ضعيف من أولئك الذين يعانون من ظروف جيدة). قيم $P > 0,05$ لجميع المتغيرات. كانت الإناث أعلى من الذكور في درجة الاكتئاب ولكن من ناحية أخرى لم يكن هناك فرق كبير في درجة القلق بين الذكر والانثى .

توضح دراستنا أن متوسط درجات القلق والاكتئاب كان أقل بشكل ملحوظ بين المرضى الممثلين للعلاج مقارنة بغيرهم. ومن ناحية أخرى لم يكن هناك فرق كبير في الدرجات فيما يتعلق بنوع النوبات. ($P > 0.05$)

تظهر هذه الدراسة أن هناك علاقة معنوية بين درجة الاكتئاب والقلق لدى المرضى ونقص الانتباه ، كما تبين أن هناك علاقة معنوية بين درجة الاكتئاب والقلق بين المرضى والحضور إلى المدرسة.



التقييم النفسي لدى الأطفال المصابين بالصرع

رسالة

توطئة للحصول على درجة الماجستير فى طب الأطفال

مقدمه

الطبيبه / يسرا محسن كمال علي الطباخ

بكالوريوس الطب والجراحة

كلية الطب

تحت إشراف

الأستاذ الدكتور / هشام عبد العزيز الغاياتى

أستاذ طب الأطفال

كلية الطب - جامعة بنها

الدكتور / طارق محمود عرفه خطاب

أستاذ مساعد طب الأطفال

كلية الطب - جامعة بنها

الدكتورة/إيمان جمال عبد الرحمن عامر

مدرس طب الأطفال

كلية الطب - جامعة بنها

كلية الطب

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