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Convulsion in Children with Down syndrome at Benghazi Children Hospital (2020-2021)

Dr. Mohamed Masoud Elferjani[1*], Dr. Najat Alrogig[2], Dr. Fatima Alsedawi[3], Dr. Abdalhamed Mohamed Alshiki[4]

[1]Consultant Neonatology and Genetics, Associated Professor Benghazi University, Libya
[2]Cardiologist Benghazi Children Hospital, Lecturer Benghazi University Libya
[3]Consultant Neonatology, Lecturer Benghazi University, Libya
[4]Consultant Pediatric, Assistant Professor Benghazi University, Libya

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***Corresponding author:** Dr. Mohamed Masoud Elferjani
Consultant Neonatology and Genetics, Associated Professor Benghazi University, Libya

Abstract **Original Research Article**

Introduction*:** Down syndrome (DS) (trisomy 21) is the most frequent chromosomal cause of mental retardation, with a frequency rate of about 1 in 700 births. The prevalence of epilepsy in subjects with DS is reported to range from 1% to 13%, higher than in the general population but lower than in patients with mental retardation. ***Aim: To find the prevalence of convulsion in Down syndrome patients attending pediatric hospital at Benghazi and to find the associated factors related to convulsion in Down syndrome patients. ***Methodology*:** cross –sectional study included all Down syndrome patients admitted to pediatric hospital during two years period (2020- 2021) , and Patients attending both genetic and neurological clinics during the same years. ***Results:*** Total number of cases was 150; History of convulsion was positive in (18)12% of Down syndrome patients. Majority (89%) of patients had generalized tonic clonic, 5.5% had focal convulsion, and 5.5 % had infantile spasm, all patients were in treatment and had good response. Males constitute to 61% of all patients with male to female ratio 1.6:1. Nearly half (50.4%) of male patients their age was 1- 5years, while 43.8% of females was in same age group. Majority of mothers age were > 32years, 88% of all mothers their age 30years or above. All patients with convulsion had no complication during delivery, while 9% of patients without convulsion had complications. More than half (61.5%) of the patients the convulsion started before the age of one year. Results of EEG were abnormal in 25% of patients with convulsion. ***Conclusion*:** Normal ECHO was found in 33% of all cases, Atrial septal defect was recorded in 27% of patients with convulsion and 19.5% of patients without convulsion, Ventricular septal defect and Atrial septal defect in 15.4% of patients with convulsion and 10.3% of patients without convulsion, Patent ductus arterious was in 11.5% of patients with convulsion and 3.2%of patients without convulsion. More than half (69.2%) of patients with convulsion had history of congenital heart disease, and 71.4% of patients without convulsion had history of congenital heart disease.

Keywords: Down syndrome (DS), mental retardation, genetic and neurological clinics, arterious.

INTRODUCTION

Down syndrome is a common congenital chromosomal anomaly which is found worldwide. The condition occurs when there is one extra copy of chromosome 21 in cells in the body. The extra chromosome 21 material may affect the physical developmental and learning abilities of people with Down syndrome [1].

Down syndrome is not a disease or an illness that can be cured. People with Down syndrome do not suffer from it, nor is it anybody faults. Down syndrome is one of the most leading causes of intellectual disability and millions of these patients face various health issues including learning and memory , congenital heart diseases ,Alzheimer's diseases, leukemia, cancers and Hirschprung diseases [2]. The incidence of trisomy is influenced by maternal age and differs in population. Down syndrome has high genetic complexity and phenotype variability. Trisomic features are at elevated risk of miscarriages and Down syndrome people have increased incidence of developing several medical conditions [3]. Recent advancement in medical treatment with social support has increased the life expectancy for Down syndrome population. In developed countries, the average life span for Down syndrome population is 55 years [4].

HISTORICAL BACKGROUND

Down syndrome was first described by Dr. John Langdon Down in 1866. While working in London in 1866, he noticed that some of his patients, though not related, have similar physical characteristics that made their condition different from other types of intellectual disabilities [5].

Langdon Down thought that their facial features reassembled those of the Mongolian people and so, he introduced the term Mongol. People with Down syndrome were once referred to as having "mongolism" or being "Mongols". These terms are inappropriate and are no longer used when referring to a person with Down syndrome [6]. It was not until 1959 that Dr. Jerome Lejeune, a French physician, made the discovery that Down syndrome was the result of a chromosomal anomaly. His research led him to the fact that the cells of people with Down syndrome had 47 chromosomes, compared to the typical 46 chromosomes. Shortly after, it was discovered that chromosome number 21 contained an extra partial or complete chromosome, thus the term trisomy 21 was born [7].

TYPES OF DOWN SYNDROME

There are three types of Down syndrome:

1. Trisomy 21(Non-disjunction) (97 % of cases (Down syndrome is caused by trisomy 21. A child with trisomy 21 has three copies of chromosome 21 instead of the usual two copies in all of his or her cells [8].
2. Translocation (2% of the cases) Down syndrome can also occur part of chromosome 21 becomes attached (translocated) onto another chromosome, before or at conception. Children with translocation Down syndrome have the usual two copies of chromosome 21, but they also have additional material from chromosome 21 attached to the translocated chromosome. In approximately half of the babies with this type of Down syndrome, it is a unique occurrence. In the other half, the translocation occurs because one or other parent happens to have a number 21 chromosome translocated or stuck onto another chromosome. Either parent can carry a translocation without showing any symptoms , because he or she still carries the correct amount of genetic material, although some of it is out of place (by translocation). In the latter situation, there is a reoccurrence of Down syndrome. In this situation chromosome studies would be carried out to ascertain the size of the risk [8].
3. Mosaic (1% of the cases) of Down syndrome has a mosaic pattern. They have a mixture of cells, some with an extra chromosome 21 and some normal cells. This mosaic of cells is caused by abnormal cell division after fertilization. Some of these babies may present with less of the physical characteristic typically associated with Down syndrome [8].

CHARACTERICTIS

Typical facial features:

1. Face tends to be round and flat.
2. Eyes slant upwards and have an extra fold of skin on the upper eyelid known as an epicanthic fold or the epicanthus. This skin fold covers the inner corner of the eye next to the nose.
3. The mouth cavity is slightly smaller than average and the tongue slightly larger causing the person with Down syndrome to sometimes protrude his \ her tongue. With speech and language therapy from an early age, tongue protrusion can be managed through exercises that strengthen the lips and tongue.
4. The back of the head is slightly flattened.
5. Nose may be small with a flat and low bridge.
6. Ears may also be small and slow set [9].

OTHER PHYSICAL FEATURES

The neck in new born babies may have excess skin, this called the nuchal fold. Older children and adults tend to have short broad necks.

A single creases across the palm of the hand.

Hands tend to be broader and shorter and the little finger can sometimes curve inwards.

A bigger than the normal space between the first and second toe (the sandal gap).

Low muscle tone or hypotonia is common among babies and very young children with Down syndrome. Hypotonia generally improves over times, and most children with Down syndrome will have physiotherapy in infancy and childhood to help improve their hypotonia.

Babies may have a low birth weight.

Down syndrome is the most common genetic cause of mental retardation. Although seizers were not mentioned in the original description of Down syndrome, the prevalence of epilepsy in children with Down syndrome is now known to be higher than in the general population, but lower than in patients with mental retardation [12].

Age is an important determinant of the prevalence of epilepsy in Down syndrome. A tri – phasic distribution of epilepsy in Down syndrome is now generally accepted and includes: infancy epilepsy, early adult hood epilepsy ,and a distinct epilepsy syndrome in patients over 50- 55 years [13].

Seizure onset is reported to occur within a year of birth for 1 – 13 % of epileptic individual with Down syndrome and in the third decade of life for another 40% [14] in the younger age group, primarily infantile spasm and tonic – clonic seizures with myoclonus are observed. However, older patients often have simple or complex partial seizures as well as tonic –clonic seizures.

A new syndrome of late onset myoclonic epilepsy in Down syndrome has been recognized, this syndrome is expected to be better delineated in the future as the life expectancy of Down syndrome patients increases [14].

Sex distribution for epilepsy in children with Down syndrome has not been uniformly reported. In studies which included different seizure types, males were reported to have a younger age at onset [12].

This impression is likely to be on founded by the significant male predominance in the infantile spasms group. Maternal age has also not been sufficiently reported and its relationship with incidence of epilepsy in Down syndrome remains unexplored.

EPILEPTOGENESIS IN DOWN SYNDROME

Seizure susceptibility in patients with Down syndrome is attributable both to inherent genetic differences in brain structure and to secondary complications. On a macroscopic level, neuroimaging has delineated certain brain differences between patients with Down syndrome and the general population. In a study of children with Down syndrome, compared to age –matched controls, hippocampal volumes were found to be significantly smaller in the Down syndrome group even when corrected for overall brain volume. Another study has shown a disproportionately smaller cerebellar volume [15]. The pathogenic significance of these findings has not been defined. Any abnormality that enhances net excitation or limits net inhibition would favour the development of a hyper – excitable, seizure –prone state. Down syndrome brains are characterized by a 20- 50 % decrease in the number of small granule cells, lower neuronal density and abnormal neuronal distribution, especially in cortical layers II and IV. These granule cells are inhibitory, gamma – amino – butyric acid (GABA) – containing cortical inters – neurons. Functionally, these morphological abnormalities will distort synaptic input such that voltage attenuation is greater in spines with short, thick, necks. Neuronal membranes in Down syndrome are abnormally hyper-excitable.

Another putative mechanism involves anomalous glutamate receptor function. The level of this excitatory transmitter is elevated in both Down syndrome and infantile spasms. Further- more; the gene for at least one glutamate receptor subunit is localized to the Down syndrome region on the distal arm of chromosome 21.

Recently, some nutritional similarities between epilepsy and Down syndrome with pathogenic significance were reported. Uncontrolled data from human and animal studies has led to the identification of altered levels of several nutrients including vitamins, trace metals and amino acids. It has been proposed that these alterations may affect neurotransmitter levels or function; however, these observations should be further substantiated before any conclusions can be made [17].

DIAGNOSTIC METHODS

Prevention of Down syndrome depends upon offering prenatal diagnosis to high risk pregnancies via amniocentesis and chorionic villous sampling. Amniocentesis and chorionic villous sampling are quite reliable but offers risk of miscarriage of between 0.5 to 1% [1]. Based soft markers like small or no nasal bone, large ventricles and nuchal fold thickness, the risk of Down syndrome for fetus can be identified through ultrasound generally at 14 to 24 weeks of gestation [18]. Increased fetal nuchal translucency indicates an increased risk of Down syndrome [19]. The other methods used for prenatal diagnosis in which traditional cytogenic analysis is still widely used in different countries .However some rapid molecular assays FISH (fluorescent in situ hybridization), QF – PC (quantitative fluorescence PCR), and MLPA (multiplex probe ligation assay) – also used for prenatal diagnosis [19].

ROUTINE KARYOTYPING

Cytogenetic analysis of metaphase karyotype remains the standard practice to identify not only trisomy 21, but also all other aneuploidies and balanced translocations [20].

RAPID ANEUPLOIDY TESTING METHODS

Over the past 10 years however, several other methods have been developed and used for the rapid detection of trisomy 21, either in fetal life or after birth. The most widely used is FISH of interphase nuclei, using Has 21 – specific probes or whole Has 21 [21]. An alternative method that is now widely used in some countries is QF- PCR, in which DNA polymorphic markers (microsatellites) on Has 21 are used to determine the presence of three different alleles [21].

This method relies on informative markers and the availability of DNA. Rapid diagnosis by PCR – based methods using polymorphic STR markers may reduce these difficulties using conventional approach. Using STR markers method we can detect trisomy in 86.67 % cases with only two markers. Using more number of markers can further increase the reliability of the test [21].

Simultaneously parental origin of the nondisjunction can also be detected [22, 23]. Additional method to measure copy number of DNA sequences include MLPA [24] which was first introduced in 2002 as a method of relative quantification in DNA [25].

MANAGEMENT OF THE DISEASE

One of the hallmarks of Down syndrome is the variability in the way that the condition affects people with Down syndrome. With the third 21st chromosome existing in every cell, it is not surprising to find that every system in the body is affected in some way. However, not every child with Down syndrome has the same problems or associated conditions. Parents of children with Down syndrome should be aware of these possible conditions so they can be diagnosed and treated quickly and appropriately [25].

Timely surgical treatment of cardiac defects during first 6 months of life may prevent from serious complications. Congenital cataracts occur in about 3 % of children and must be extracted soon after birth to allow light to reach the retina. A balance diet and regular exercise are needed to maintain appropriate weight. Feeding problems and failure to thrive usually improve after cardiac surgery. A Down syndrome child should have regular checkup from various consultants. These include:

1. Clinical geneticist – Referral to a genetics counseling program is highly desirable.
2. Cardiologist - Early cardiologic evaluation is crucial for diagnosing and treating congenital heart defects which occur in as many as 60 % of these patients.
3. Developmental pediatrician.
4. Pediatric pulmonologist - Recurrent respiratory tract infections are common in patients with Down syndrome.
5. Ophthalmologist.
6. Neurologist / Neurosurgeon - As many as 10 % of patients with Down syndrome have epilepsy; therefore, neurologic evaluation may be needed.
7. Orthopedic specialist.
8. Child psychiatrist - A child psychiatrist should lead liaison interventions, family therapies, and psychometric evaluations.
9. Physical and occupational therapist.
10. Speech - language pathologist.
11. Audiologist [25].

Aim of the Study

1. To find the prevalence of convulsion in Down syndrome patients attending pediatric hospital at Benghazi.
2. To find the associated factors related to convulsion in Down syndrome patients.

METHODOLOGY

Type of the Study: Cross –sectional study.

Population

All Down syndrome patients admitted to pediatric hospital during two years period (2020- 2021), and patients attending both genetic and neurological clinics during the same years.

Data Collection

Data were collected from the medical records of the patients and also phone call to their parents if some data was deficient in the record.

Data as age, sex, family history, history of convulsion … etc. Was recorded in the perform (Appendix I).

Statistical Analysis

Data were analyzed using statistical package for social science (SPSS) version 2**3**.

Descriptive statistics, as mean, standard deviation and median were used.

Inferential statistics were used when needed, as t – test to find the difference between the means of the two groups , and Chi-square(x^2) to find the difference in the distribution of the variables between the two groups, P-value were considered significant when ≤ 0.05.

Data were presented in form of tables and figures, were the figures done by Microsoft Excel 2010.

RESULTS

Table 1: Distribution of patients according to history of convulsion

History of convulsion	No.	%
Yes	18	12
No	132	88
Total	**150**	**100**

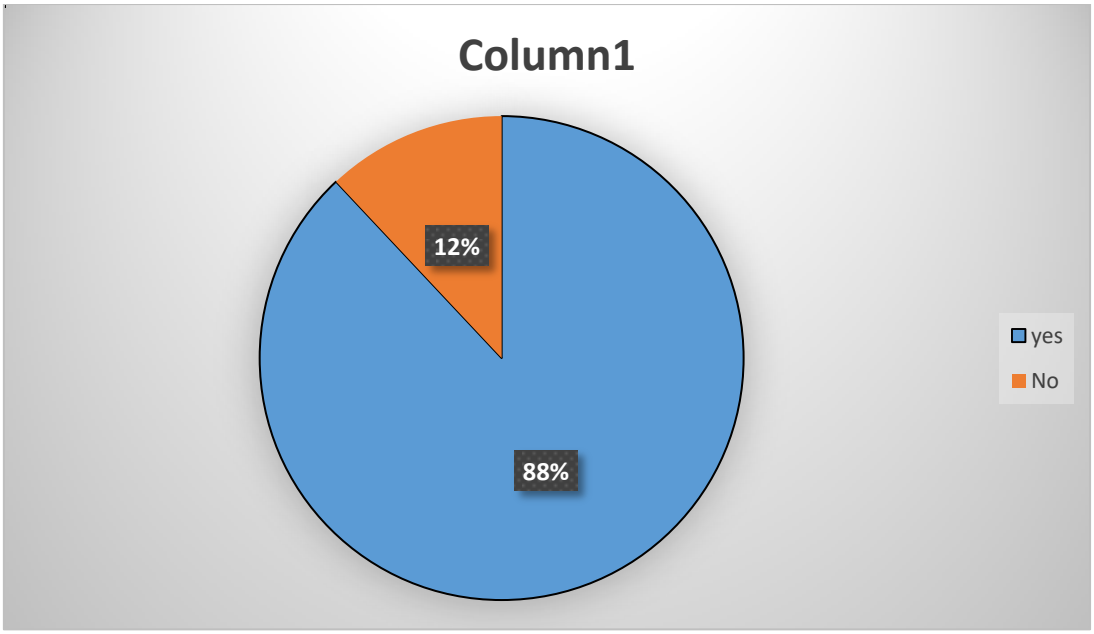

Fig.1 : Distribution of patients according to history of convulsion

Table 2: Distribution of patients according to type of convulsion

Type of convulsion	No.	%
Generalized tonic colonic	16	89
Infantile spasm	1	5.5
Focal convulsion	1	5.5
Total	**18**	**100**

*All in treatment and had good response

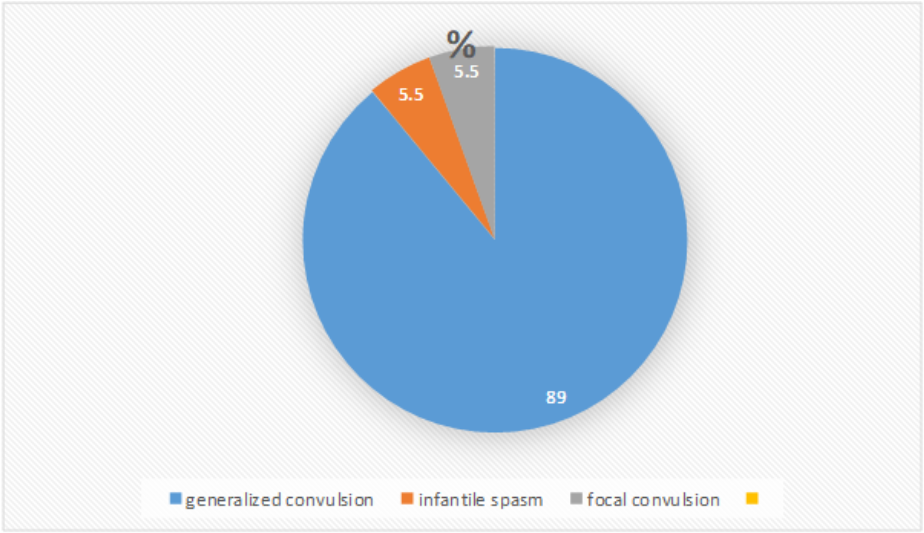

Fig. 2: Distribution of patients according to type of convulsion

Table 3: Distribution of patients according to history of convulsion and sex

	With convulsion		Without convulsion		Total	
Sex	**No.**	**%**	**No.**	**%**	**No.**	**%**
Male	12	66.7	79	60	91	61
Female	6	33.3	53	40	59	39
Total	**18**	**100**	**132**	**100**	**150**	**100**

X2 = 0.024 df =1 p=0.877(Not significant)
With convulsion: Male: Female =2:1
Without convulsion: Male: Female =1.5:1

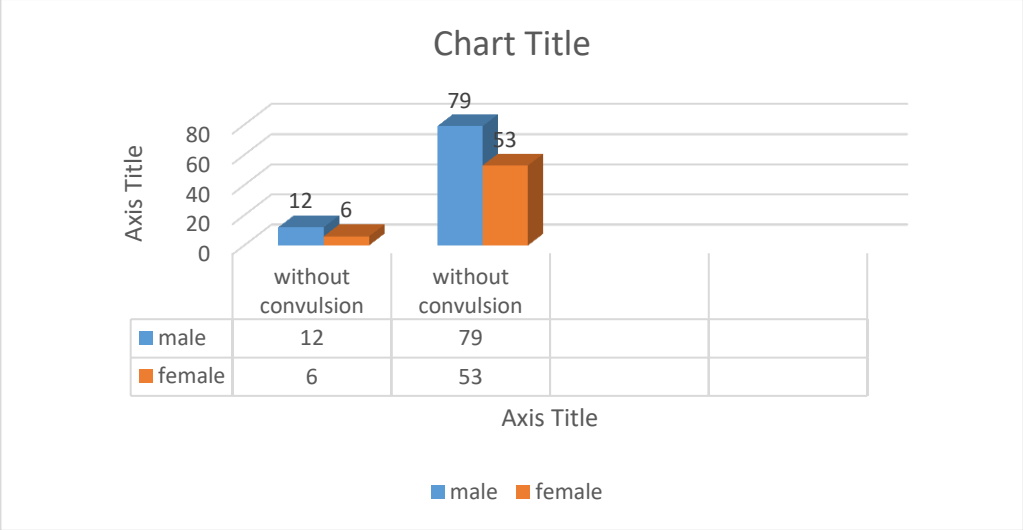

Fig. 3: Distribution of patients according to history of convulsion and sex.

Table 4: Distribution of patient's age

Age group/years	Male		Female		Total	
	No.	%	No.	%	No.	%
<1	29	31.8	26	44.1	55	36.7
1-5	50	55	22	37.3	72	48
6 - 10	**7**	7.7	7	11.8	14	9.3
>10	**5**	5.5	4	6.8	9	6
Total	**91**	**100**	**59**	**100**	**150**	**100**

Mean =3yars. Std. Deviation =3.3years. Median=2years. Minimum =One day. Maximum =13years.

Table 5: Distribution of patients according to history of convulsion and family history of convulsion

Family history of convulsion	With convulsion		Without convulsion		Total	
	No.	%	No.	%	No.	%
Yes	2	11.1	2	1.5	4	2.7
NO	16	88.9	130	98.5	146	97.3
Total	**18**	**100**	**132**	**100**	**150**	**100**

X^2=6.729 df= 1 p=0.009(Significant).

Table 6: Distribution of patients according to history of convulsion and birth weight

Birth weight/kg	With convulsion		Without convulsion		Total	
	No.	%	No.	%	No.	%
<2.5	**5**	27.8	25	18.9	30	20
2.5 – 4	**12**	66.7	102	77.3	124	82.6
>4	**1**	5.5	5	3.8	6	4
Total	**18**	**100**	**132**	**100**	**150**	**100**

With convulsion: Mean weight=2.81kg. Std. Deviation = 0.66kg. Median= 2.7kg Minimum weight= 2kg. Maximum = 5kg.

Without convulsion: Mean weight =2.86. Std. Deviation = 0.66 Median= 2.8. Minimum = 0.750kg. Maximum = 5kg.

X^2= 0.928, df=2, p= 0.629(Not significant).

Table 7: Distribution of patients according to age at onset of convulsion

Age at onset of convulsion/years	No.	%
<1	11	61.1
1-5	6	33.3
>5	1	5.6
Total	**18**	**100**

With convulsion: Mean age at onset =1.4years. Std. Deviation = 2years. Minimum age at onset= one day. Maximum = 8years.

Table 8: Distribution of patients with convulsion according to results of EEG

Results of EEG	With convulsion	
	No.	**%**
Normal	5	27.8
Abnormal	5	27.8
Not done	8	44.4
Total	**18**	**100**

Table 9: Distribution of patients with convulsion according to results of CT and MRI

Results	Results of CT		Results of MRI	
	No.	**%**	**No.**	**%**
Normal	2	11.1	5	27
Abnormal	**1**	5.5	**1**	5.5
Not done	15	83.2	12	66.5
Total	**18**	**100**	**18**	**100**

Table 10: Distribution of patients according to history of convulsion and results of USS brain

Results of USS brain	With convulsion		Without convulsion		Total	
	No.	**%**	**No.**	**%**	**No.**	**%**
Normal	16	**88**.9	130	98.5	146	97.6
Abnormal*	**2**	11.1	2	1.5	4	2.4
Total	**18**	**100**	**132**	**100**	**150**	**100**

Notes:

1. One case of abnormal result with convulsion was dandy worker male formation and second case dandy worker with hydrocephaly.
2. The 2 cases without convulsion with abnormal US brain were one of them was IVH intraventricular heg. and one cases were hydrocephalus.

Table 11: Distribution of patients according to history of convulsion and results of ECHO

Results of ECHO	With convulsion		Without convulsion		Total	
	No.	**%**	**No.**	**%**	**No.**	**%**
Normal	6	3**3.3**	45	34	65	30.8
Ventricular septal defect and Atrial septal defect	3	16.7	14	10.6	23	11
Atrial septal defect	3	16.7	26	19.7	43	20.4
Atrioventricular septal defect	**2**	11.1	21	16.0	32	15.2
Ventricular septal defect and Patent ductus arterious	**1**	5.5	5	3.8	7	3.3
Mitral regurgitation	**1**	5.5	1	0.76	1	0.47
Patent ductus arterious	**2**	11.1	5	3.0	9	4.**2**
Ventricular septal defect	**0**	**0**	13	9.8	18	8.5
Patent ductus arteriosus and tricuspid regurge	**0**	**0**	2	1.51	1	0.47
Tetralogy of follt	**0**	**0**	2	1.1	2	0.95
Patent formamen oval and Atrial septal defect	**0**	**0**	2	1.1	2	0.95
Patent ductus arterious and Atrial septal defect	**0**	**0**	7	3.8	7	3.3
Pulmonary hypertension	**0**	**0**	1	0.54	1	0.47
Total	**18**	**100**	**132**	**100**	**211**	**100**

Table 12: Distribution of patients according to history of convulsion and history of congenital heart disease

History of congenital heart disease	With convulsion		Without convulsion		Total	
	No.	**%**	**No.**	**%**	**No.**	**%**
Patent ductus arterious	3	16.7	11	8.3	14	9.3
Ventricular septal defect and Atrial septal defect	**4**	22.2	17	13	21	14
Atrial septal defect	**7**	38.9	37	28	44	29.3
Aterioventricular septal defect	**2**	11	31	23.5	33	22
Ventricular septal defect and Patent ductus arterious	**1**	5.6	7	5.3	8	5.3
Mitral regurgitation	**1**	5.6	0	0	1	0.7
Ventricular septal defect	**0**	**0**	18	13.6	18	12
Patent ductus arterious and tricuspid atresia	**0**	**0**	1	0.75	1	0.7
Tetralogy of follt	**0**	**0**	2	1.5	2	1.3
Patent formamen oval and Atrial septal defect	**0**		1	0.75	1	0.7
Patent ductus arterious and Atrial septal defect	**0**	**0**	7	5.3	7	4.7
Total	**18**	**100**	**132**	**100**	**150**	**100**

Table 13: Distribution of patients according to history of convulsion and history of hypothyroidism

History of hypothyroidism	With convulsion		Without convulsion		Total	
	No.	%	No.	%	No.	%
Yes	7	38.9	26	19.7	33	22
No	11	61.1	106	80.3	117	78
Total	**18**	**100**	**132**	**100**	**150**	**100**

X^2= 3.209, df=1 p=0.073(Not significant).

Table 14: Distribution of patients according to cause of convulsion

Cause of convulsion	No.	%
Fever	5	19.2
Hypocalcemia	3	11.5
Idiopathic	**8**	61.5
Brain defect (Dandy worker)	**2**	7.8
Total	**18**	**100**

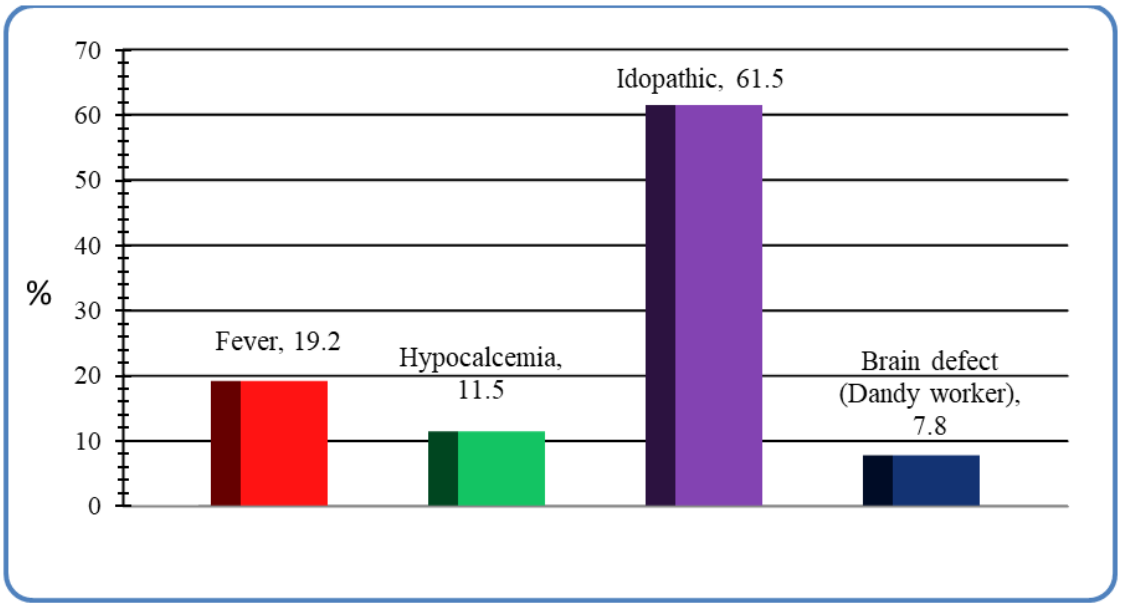

Fig. 4: Distribution of patients according to cause of convulsion

DISCUSSION

History of convulsion was positive in 12% of Down syndrome patients. Study in Italy found that the prevalence of seizures in Down syndrome patient was 13.2% [26] while other study documented that the prevalence of epilepsy in Down syndrome is overall around 5 to 6%, increasing up to 20% after the third decade [27]. Majority (89%) of patients had generalized tonic clonic, 5.5 % had infantile spasm and 5.5 % had focal convulsion, all patients were in treatment and had good response. Study in Italy recorded that 1.7% had generalized tonic clonic, 2.6% partial complex and 3.5% infantile spasms [26]. Other study found that about 8% of patients with Down Syndrome (DS) have seizure disorders: 47% of them develop partial seizures, 32% infantile spasms and 21% generalized tonic-clonic seizures [28]. Other author recorded that the pharmacological treatment of epilepsy in DS is no different from that of other patients diagnosed with epilepsy; the key is proper clinical and electrical classification to guide epilepsy treatment and thereby obtain good therapeutic results [29]. Males constitute to 61% of all patients with male to female ratio 1.6:1, patients with convulsion 66.7% of them were males and 33.3% females with male to female ratio: 2:1, and male constitute to 60% of patients without convulsion and females 40% with male to female ratio 1.5:1, this difference was not statistically significant p= 0.878. Nearly half (50.4%)of male patients their age was 1-5years, while 43.8% of females was in same age group, <1year 33.6% of male was in this age group, and 43.8% of female were in this age group, this difference was not statistically significant p = 0.471.

Mean age was 3 ±3.3years with minimum age one day and maximum age 14 years. Mean age of mothers of patients with convulsion was 36±6.7 years, and mean age of mothers of patients without convulsion was 35± 7.3years, this difference was not statistically significant p = 0.805.

Majority of mothers age were > 30years, 89.2% of all mothers their age 30 years.

History of consanguinity was positive in 15.4% of patients with convulsion and 12% of patients without convulsion and this difference was not statistically significant p = 0.850. Family history of convulsion was positive in 11.1% of patients with convulsion and 0.9% of patients without convulsion and this difference was statistically significant p = 0.008.

More than half (53.8%) of patients with convulsion were normally delivered and 46.2% delivered by C/S, patients without convulsion 55.7% delivered normally and 44.3% by C/S, this difference was not statistically significant p = 0.972.Whil in other study the rate of C/S was 60% in patients with convulsion and 68% in patients without convulsion [31]. Mean birth weight of patients with convulsion was 2.81±0.66kg, with minimum weight 2kg and maximum 5kg and 27% of them had low birth weight, while the mean of birth weight for patients without convulsion was 2.86±0.66 kg, with minimum weight 0.750kg and maximum weight 5kg and 18.9% of them had low birth weight, this difference was not statistically significant p = 0.929.In other study the mean birth weight of patients with convulsion was 725 ± 140 gm, while mean birth of patients without convulsion was 789 ± 138gm [31]. All patients with convulsion had no complication during delivery, while 9.2% of patients without convulsion had complication. Around one third (34.6%) of patients with convulsion were admitted to new natal ward and 30.3% of patients without convulsion were admitted to new natal ward, this difference was not statistically significant p = 0.824.

More than half (61.5%) of patients the convulsion started before the age of one year, 30.8% started at age 1-5 years and 7.7 % started at age >5 years, the mean age of onset of convulsion was 1.4 ±2years, with minimum age one day and maximum age 8 years. Other study shows that the age of seizure onset is bimodal: 40% occurs before 1 year of age and 40% occur in the third decade of life [32]. History of fever was recorded in 23% of patients with convulsion and 0.5% in patients without convulsion and this difference was statistically significant p = 0.0001.In Italy study 5.3% had febrile convulsion [26]. Mean level of calcium in patients with convulsion was 8.4±2.2, with minimum value 2.5 and maximum level 14.

Mean level of calcium in patients without convulsion was 9±0.73, with minimum value 6.3 and maximum level 12, there were no difference between the mean of both group p =0.295, while there were difference between the distribution of the level of calcium p = 0.003, 34.6% of patients with convulsion had calcium level <8.5, and 11.4% of patients without convulsion had level <8.5.

Majority of patients had normal level of blood pressure 88.5% of patients with convulsion and 80.6% of patients without convulsion. Blood sugar more than 120 mg/dl was recorded in 11.5% of patients with convulsion and 15.1% of patients without convulsion.

Mean of blood sugar in patients with convulsion was 99.1±24.7 mg/dl, with minimum level 70mg/dl and maximum level 163mg/dl.

Mean of blood sugar in patients without convulsion was 105.7±22.6 mg/dl, with minimum level 47mg/dl and maximum level 216mg/dl.

These difference was not statistically significant p = 0.606.

Results of EEG were abnormal in 30.8% of patients with convulsion. Other authors mentioned that individuals with DS have increased absolute power in all the EEG bands, independent of cognition functions [33].

Results of CT of patients with convulsion were abnormal in one patient (3.8%) of them and results of MRI were abnormal in one patient (3.8%). In Italy study CT scan was performed in 6 subjects, in 4 of them no anomalies were observed, in one patients showed slight dilatation of subarachnoid spaces and lateral ventricules and other patient's slight dilatation of subarachnoidal spaces, lateral ventricles and opercular portion of the Sylvain fissure [26].

Results of USS brain was normal in 92.3% of patients with history of convulsion and 98.4% of patients without convulsion, abnormal result was recorded in 2 patients with convulsion, one male was with dandy worker and the second case dandy worker with hydrocephaly, while the three cases without convulsion with abnormal US brain were one of them was (IVH) intraventricular heg, and two cases were hydrocephalus.

USS of abdomen was normal in all patients with history of convulsion and 96.2% in patients without convulsion, 1.6% of patients without convulsion had gall bladder stone, 1.1% had bilateral renal hydronephrosis and 1.1% had hernia. Results of ECHO 30.8% of patients with convulsion and without convulsion were with normal ECHO, Atrial septal defect was recorded in 27% of patients with convulsion and 19.5% of patients without convulsion, Ventricular septal defect and Atrial septal defect in 15.4% of patients with convulsion and 10.3% of patients without convulsion, Patent ductus arterious was in 11.5% of patients with convulsion and 3.2%of patients without convulsion. Other study shows mitral valve prolapse (MVP) was found significantly more frequently in the DS group (17.9%) as compared to a normal control group [34].

More than half (69.2%) of patients with convulsion had history of congenital heart disease, and 71.4% of patients without convulsion had history of congenital heart disease ,as Atrial septal defect, Ventricular septal defect and Atrial septal defect and Aterioventricular septal defect, etc. Congenital heart disease was recorded in 71.1% of Down's syndrome children, 69.2% of patients with convulsion had heart problem and 71.4% of patients without convulsion. The result of other study, Atrioventricular septal defect was the commonest defect 15(20.27%), followed by Ventricular septal defect 12(16.21%), Patent ductus arteriosus 11(14.86%), Atrial septal defect 7(9.46%), Tetralogy of Fallot 3(4.05%), Pentology of Fallot 2(2.70%) [5]. Also other study recorded that Down's syndrome children is associated with a high prevalence of cardiac, gastrointestinal, immunological, respiratory, sensory, and orthopedic anomalies [35]. While others recorded that the Seizures can also be regarded as complications of congenital cardiovascular anomalies in children with DS [36]. History of hypothyroidism was positive in 38.5% in patients with convulsion, and 20.5% in patients without convulsion, this difference was not statistically significant p = 0.073. Results of other study found that a high prevalence of thyroid dysgenesis in patients with DS and hypothyroidism [37]. Idiopathic was the cause of convulsion in 61.5%, fever was cause in 19.2%, hypocalcemia in 11.5% and brain defect in 7.8% of patients. Other author mentioned the cause of convulsion is likely due to structural and functional abnormalities in the brain caused by the extra chromosome 21. Associated medical complications such as cardiovascular abnormalities and recurrent infections might also influence the likelihood of getting epilepsy [12].

CONCLUSION

Concluded from this study that the 12.3% of Down syndrome patients attending the hospital during the two years had history of convulsion. Majority (92.3%) of patients had generalized tonic clonic. Male to female ratio was 1.6:1. Age of the mothers does not appear to be risk factor for convulsion. More than half of patients the convulsion started before the age of one year. EEG was abnormal in around third of patients with convulsion. Around two third of patients with convulsion had congenital heart disease.

RECOMMENDATION

1. Early detection of epilepsy is essential for successful treatment.
2. Awareness of the association between Down syndrome and epilepsy is important so that doctors can intervene early to maximize development and to improve the quality of life.

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