Results

Results

Table (1) and Fig (1) show the age distribution of the patients. Six patients (3.6%) were at the age of 0-1 month with a mean 28.7 and S.D \pm 1.5 days. 29 patients (17.6%) were >1-6 months of age with a mean 4.0 and SD \pm 1.6 months. 49 patients (29.8%) were >6-12 months with a mean 9.5 and SD \pm 1.9 months. 80 patients (48.7%) were >12-24 months with a mean 18.1 and SD \pm 3.5 months. For all patients the mean age was 11.2 and SD \pm 7.2 months.

Table (2) and Fig (2) show the sex distribution of the patients. 103 patients (62.8%) were males and 61 patients (37.1%) were females. The $\frac{male}{female}$ ratio = $\frac{103}{61}$

Table (3) shows the distribution of cases according to their aetiological diagnosis. 164 infants enrolled in this study were classified into three main Categories. The epileptic group including 137 patients (83.5%) has been further subdivided into 3 subgroups (symptomatic 97 patients (70.8%), idiopthic 34 patients (24.8%) and cryptogenic 6 patients(4.3%)}. The febrile seizures group included 15 patients (9.1%). The acute symptomatic group entailed 12 patients (7.3%) including those with head trauma and hypocalcemia.

Table (1). Age distribution of patients (N=164).

Age classes (month)	Number	%	Mean±SD
0 - 1	6	3.6	28.7±1.5 d
> 1 - 6	29	17.6	4.0+1.6 m
> 6 - 12	49	29.8	9.5±1.9 m
>12 - 24	80	48.7	18.1+3.5 m
Total	164	100	11.2+7.2 m

Table (2). Sex distribution of patients (N=164).

Sex	No.	%
Male	103	62.8
Female	61	37.1
Total	164	100

Table (3). The distribution of cases according to their aetiological diagnosis.

uiugiiosis.	· · · · · · · · · · · · · · · · · · ·	
Group	No.	%
I- Epilepsy	137	83.5
a) Symptomatic	97	70.8
- Cerebral palsy	38	39.1
- TORCH	21	21.6
- Postmeningitic	12	12.3
- Neurodegenerative	8	8.2
- Aminoacidopathy	6	6.1
- Brain malformations	6	6.1
- Autosomal recessive microcephaly	2	2.06
- Down syndrome	1	1.03
- Brain ischaemia	1	1.03
- Brain granuloma	1	1.03
- Mitochondrial encephalomyopathy	1	1.03
b) Cryptogenic	6	4.3
- West syndrome	5	83.3
- Otahara syndrome	1	16.6
c) Idiopathic	34	24.8
II- Febrile	15	9.1
III- Acute symptomatic	12	7.3
- Head trauma	4	33.3
- Hypocalcaemia	8	66.7
Total	164	

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ı	able (4). Age of occurrance of first attack of convulsion in relation to diagnosis	
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2	To			E	-II					Γ	Π					Γ						F	T	Ą
	Total	- Hypocalcemia	- Head trauma	III- Acute symptomatic (No.= 12)	II- Febrile	c) Idiopathic	- Otahara syndrome	- West syndrome	b) Cryptogenic (No.=6)	- Mitochondrial encephalomyopathy	- Brain granuloma	- Brain ischaemia	- Down syndrome	- Autosomal recessive microcephaly	- Brain malformations	- Aminoacidopathy	- Neurodegenerative	- Postmeningitic	- TORCH	- Cerebral palsy	a) Symptomatic (No.= 97)	I- Epilepsy		Aetiological diagnosis
-	58					4	,	<u> </u>							3	4	1		7	36		,	No.	
l" year	35.4	1.7				6.8	1.7	1.7							5.1	6.8	1.7		12.0	62.0		,	%	0-1 m
ar	54	6	3		2	13		4					1	2	ω		3	5	11	1			No.	≥1.
145 (88.4%)	32.9	11.0	5.5		3.7	24.0		7.4					1.8	3.7	5.5		5.5	9.2	20.0	1.8	1		%	>1-6 m
3.4%)	33	1			10	12			*	1	1					1	1	3	3				No.	ķ
	20.1	3.0			30.0	36.3				3.0	3.0					3.0	3.0	9.0	9.0				%	>6-12·m
19 (1	19		1		3	5						1				1	3	4		-			No.	>12
1.5%)	11.5		5.2		15.7	26.3			•			5.2				5.2	15.7	21.0		5.2			%	2-24 m
	164	90	4		15	34	_	5		1	1	1	1	2	6	6	∞	12	21	38				Total

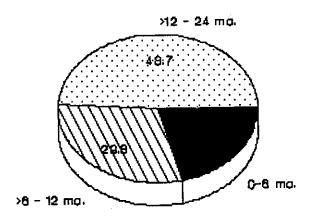


Fig (1) Age distribution of patients

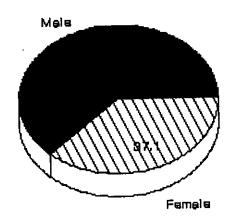


Fig (2) Sex distribution of patients

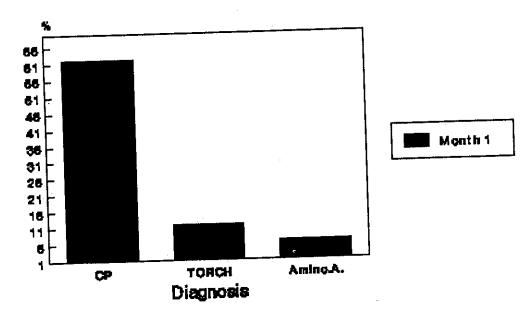


Fig (3) Groups of patients with convulsion in the 1st mo.

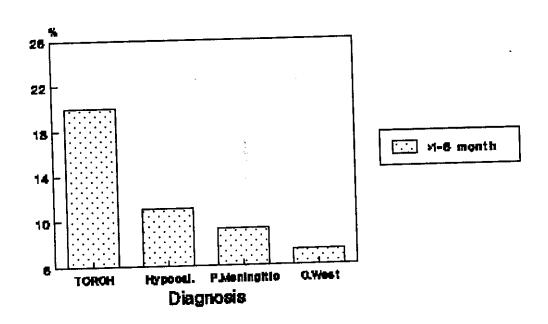


Fig (4) Groups of patients with convulsion in the age >1-6 mo.

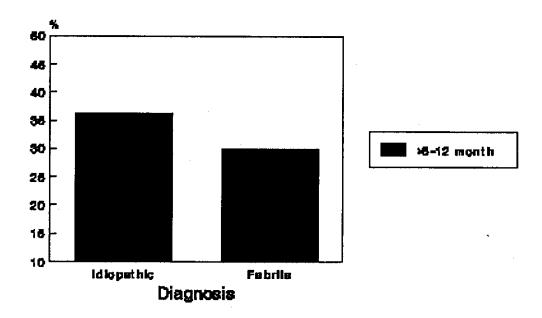


Fig (5) Groups of patients with convul. in the age >6-12 mo.

- Table (4) and Fig (3, 4, 5) represents the age at onset of first attack of convulsion in the different groups.
- * 58 patients (35.3%) presented with a first attack of convulsion in the neonatal period. (1-29 days). The largest group was due to patients with cerebral palsy secondary to ischemic hypoxic insult or kernicterus (No. 36 (62.0%) followed by the group of patients with congenital infection (No.7 (12.0%) and lastely those with aminoacidopathy (6.8%).
- * 54 patients (32.9%) presented with a first attack of convulsion in the first 6 months of life. 24% of patients included in this group were idiopathic as no definite aetiology could be found and had a normal motor and mental development. 20% had an evidence of TORCH infection and 11% were hypocalcemic.
- * In the second half of the first year (>6-12 months) 33 patients (20.1%) convulsed for the first time. 36% were assigned to the idiopathic group and 30% had febrile seizure.
- * 19 patients (11.5%) had the onset of their first seizure in the second year (>12-24 months) of life.

Statistical analysis had shown highly significant difference between the various groups concerning the age of occurrance of first attack with a chi-square value = 160.606 and P value = (0.0001).

Table (5) and Fig (6) demonstrate the types of convulsion in the main three clinical groups. In the epilepsy group, generalized tonic clonic seizures (25.5%) and generalized tonic seizures (25.5%) were the most frequent types of convulsions. While generalized tonic convulsions (46.6%) and generalized clonic (58.3%) were the most common types of seizures encountered in the febrile and acute symptomatic groups respectively.

Statistical analysis shows a significant difference between the epileptic, febrile and acute symptomatic groups concerning the generalized type of convulsion as the chi square value = 15.457 and P value = (0.05). There were no significant difference between the 3 groups concerning the partial type of convulsion.

Table (6) shows the distribution of patients presenting with myoclonic convulsion (No.=21 patients) according to the undrlying aetiology. 16 patients (76.1%) were symptomatic; and their aetiology were cerebral palsy in 6 patients (28.5%), congenital TORCH infection in 4 patients (19%), aminoacidopathy in 2 patients (9.5%), neurodegenerative

Table (5). The types of convulsion in the main three groups.

							٠,			cant)	Signifi	0.05 (alue=	P_v.	15.457,	value=1	Chi_square value=15.457, P_value= 0.05 (Significant)
			7%)	11 (6.7%)							2%)	153 (93.2%)					
162	9.0	1	18.1	2	72.7	∞	3.9		13.7	21	27.4 21 13.7 6		28.7 42	44	26.1	40	Total
12					16.6	2					58.3	7	16.6	2	8.3	_	Acute Symptomatic
15											26.6	4	46.6	7	26.6	4	Febrile
137	0.7	_	1.4	2	4.3	6	4.3		21 15.3 6	21	22.6	31	25.5	35	25.5	35	Epilepsy
	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	
	nic.	clo															
	т́с	tonic		tonic		clc	atonic	atc	myoclonic	myoc	clonic	clo	ис	tonic	clonic	tonic cionic	
Total		Partial	11	Partial	Partial	Pau	ralized	Generalized		Gener	<u>&</u>	Gener	alized	Generalized	alised	Generalised	Group
													ļ				•

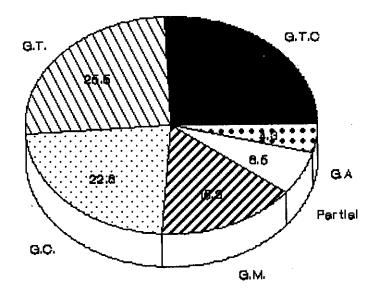


Fig (6) The type of convulsion in the epileptic group

Table (6). Distribution of patients presenting with myoclonic convulsion according to etiology (No. = 21).

Diagnosis	No.	%
I- Symptomatic	16	76.1
Cerebral palsy	6	28.5
TORCH	4	19.0
Aminoacidopathy	2	9.5
Neurodegenerative	1	4.7
Postmeningitic	1	4.7
Brain malformation	1	4.7
Autosomal recessive microcephaly	1	4.7
II- Cryptogenic		
West syndrome	5	23.8

disorder, postmeningitic, brain malformations and autosomal recessive microcephaly in 4 patients (4.7%) each. No etiolgy was found in 5 cases (23.8%) and diagnosed as cryptogenic West syndrome.

Table (7) shows the perinatal and maternal data in different groups of patients. Concrening the perinatal data, cyanosis and hypoxia at birth were found mainly in 32 patients with cerebral palsy (84.2%). As regard maternal data; recurrent abortions were predominant in the group of patients with congenital TORCH infection (33.3%) and those with brain malformations (33.3%), while positive consanguinity was most frequent in patients with neurodegenerative disorders (75%), and those with aminoacidopathy (66.6%). Positive family history was present mainly in the febrile group (26.6%), neurodegenerative group (25%) and patients with idiopathic convulsion (20.5%).

Statistical analysis shows highly significant difference between the various groups of patients concerning maternal data as the chi square value =40.375 and P value = (0.004).

Table (8) and Fig (7) show the head circumference in the three main groups of patients. In the group of epilepsy 53 patients (38.6%) were microcephalic with a head circumference <3rd centile for age. They were belonging to the symptomatic

Table (7). Perinatal and maternal data in different groups of patients.

	1011112121		ups of pat	CIIIO.				
Cyanosis	Perinatal Dat Pathological	C.N.S	 		Materna	il data		
Hypoxia	jaundice	infection	Abortion	%	+ve Conseng-unity	%	Other sibs	%
		 			1	25.0		╁
		 	1	20			1	+-
ļ					1	50.0	† <u></u>	20
2		<u> </u>	<u> </u>		_}	00.0		
			2	33.3	2	33 3	 	├
32	5	1	1	16.6	4	_	1	
	·	11		13.1	8			16
			7	33.3	10			
					3			14.
					1		-	
			2	25.0	6	75.0	(
		=	1	6.6	4			25.0
								26.6
	<u>, </u>	<u> </u>	19	11.5	40			20.5 10.9
	2 32 34	Hypoxia jaundice	Hypoxia jaundice infection	Hypoxia jaundice infection	Hypoxia jaundice infection 76 1 20 1 16.6 32 5 1 5 13.1 7 33.3 2 25.0 1 6.6 34 5 1 19 11.5	Hypoxia jaundice infection Hypoxia jaundice infection Hypoxia Hypoxia jaundice infection Hypoxia Hypoxia Hypoxia Jaundice Hypoxia Hypoxia Hypoxia Hypoxia Hypoxia Hypoxia Hypoxia Hypoxia Hypoxia Hy	Hypoxia jaundice infection Abortion	Hypoxia jaundice infection Abortion % +ve consang-unity % Other sibs affected

Chi-square value = 40.375, P- value = 0.004 (Highly significant)

Table (8). Head circumference in the main three groups of patients.

Group	MICE	cephaly	Macro	cephaly		rmal
Epilepsy (137)	No.	%	No.	%	No.	%
Febrile (15)	53	38.6	3	2.1	81	59.1
			1 <u></u>			
Acute symptomatic (12)				02	15	100
Total (164)	53	32.3	 	8.3		91.6
Chi- square value= 17 403	D - 1	32.3	4	2.4	107	65.2

Chi- square value= 17.403, P- value= 0.002 (Highly significant)

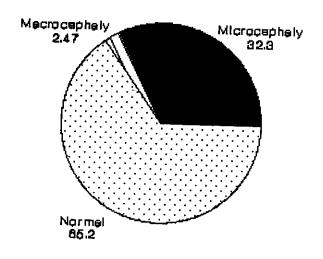


Fig (7) Head circumference distribution in all patients

and cryptogenic groups mainly. Three patients (2.1%) were macrocephalic with a head circumference >95th centile for age.

In the febrile group all patients had a normal head circumference. Wherease in the acute symptomatic group; 11 patients (91.6%) had a normal head circumference and only 1 patient (8.3%) was macrocephalic.

Statistical analysis shows highly significant difference between the epileptic, febrile and acute symptomatic groups regarding the changes in head circumference as the chi square value = 17.403 and the P value = (0.002).

Table (9) and Fig (8) demonstrate a comparison of the head circumference in different groups of patients. Microcephaly was predominating in patients with cerebral palsy (45.2%) followed by those suffering from congenital infection (30.1%) espescially those with cytomegaloviral infections. Macrocephaly was pronounced in infants with post meningitic sequalae (50%) followed by those who were subjected to head trauma (25%).

Stastical analysis shows highly significant difference between the various groups of patients regarding head circumference changes as the chi square value =99.888 and the P value = (0.0001).

Table (9). Head circumference in different groups of patients.

Diagnosis			cut gr		patien I.C	<u>ts.</u>	·
		centile	per	h-95th rcentile		95th centile	Total
Cerebral palsy	No.	%	No.	%	No.	1%	
Idiopathic	24	45.2	14	13.4			38
TORCH	16	1201	34	32.6			34
Febrile	16	30.1	5	4.8			21
Postmeningitic	4	120	15	14.4			15
Hypocalcemia	+4	7.8	6	5.7	2	50.0	12
Neurodegeneration	 	1.0	8	7.6			8
Brain malformation	$\frac{1}{2}$	1.9	7	6.7	-		8
Aminoacidopathy		3.9	3	2.8	1	25.0	6
Cryptogenic West			6	5.7			6
lead trauma	2	3.9	3	2.8	7-		5
Autosomal recessive microcephaly			3	2.8	1	25.0	4
otal		3.9					2
Chi-square value=99.888,	51	T	104	ighly sic	4		159

Chi-square value=99.888, P-value= 0.0001 (Highly significant)

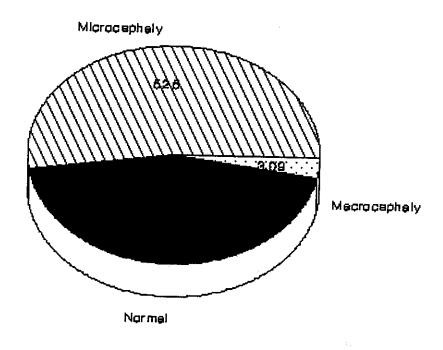


Fig (8) Head circumference in the symptomatic epilepsy group

The total number of patients was 159 as 5 patients were considered as miscellanous and described separetely.

Table (10) represents the EEG pattern in different types of convulsions. Generalised tonic seizures had a normal EEG pattern (Fig. 9) in 15 patients (34%), while 11 patients (25%) had a bilateral synchronous epileptogenic discharge. The predominant EEG pattern in patients presenting with tonic clonic fits was diffuse high voltage slow delta waves (32.5%) followed in order of frequency by a bilateral epileptogenic activity (25%). In cases suffering from generalised clonic seizures, a bilateral synchronous epileptogenic discharge (Fig. 10) was the main EEG pattern in (38 %), and a chaotic hypsarrhythmic pattern (Fig. 11) predominated in patients with myoclonic fits in 12 patients (57%).

Statistical analysis shows highly significant difference between the various groups of patients with generalised convulsions concerning the EEG pattern. as the chi square value = 107.363 and the P value = (0.0001).

Table (11) and Fig (12) show the number and percentage of patients with normal and delayed mentality. Out of 164 patients presenting with convulsions, 104 (63.5%) had global developmental delay. The majority of them were belonging to the epileptic group.

Table (10). EEG-pattern in different types of convulsions.

Type Co. EEG-pat	Normal Nor	
Type of Convulsion	The different types of a	
- crtatOII	Normal Normal Normal	
1	A.	
0	Focal Abnormal pattern Bilateral Diffuse	
Generalized	discharge Bilateral Diffuse Multifect IV	
Tonic	1 ***** 0/ 1 T *****************************	·
	No No South discharge	ic Total
Tonic clonic	1 9 204	
Clonic	6 15.0 No %	
	9 22.5 10 250 2 4.5	1
Myoclonic	3 7.1 6 140 25.0 13 32.5	44
	1 47 16 38.0 11 2 3.0	+44
Atonic	7 0 5 1 1 20.0 1	. 40]
Partial	3 3 142 1	T
	2 33.3 1 166	42
Clonic	1 16.6 2 33.3 12 57.0	21
Tonia		
Tonic clonic	3 37.5 2 25.0 1 12.6	_ 6
Tonic 1	1 125 2 2	
Total	1 30.0 - 1	J
		8 1
26	1 100	
· · · · · · · · · · · · · · · · · · ·		2
For Generalized convulsion:	30 11.9 16 97	17
For partial convulsion:	Chi_square value= 107.363, P value= 0 000 1	161
		164
	Chi_square value= 0.165, P_value= 0.0001 (N)	64

For Generalized convulsion: Chi_square value= 107.363, Chi_square value= 9.167, P_value= 0.0001 (Highly significant)
P_value= 0.328 (Non significant) 9.167,

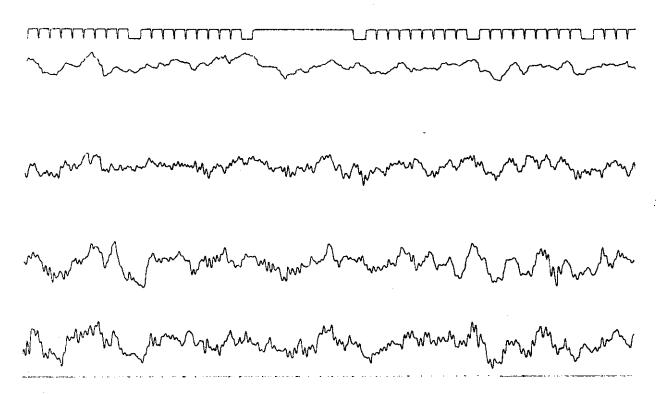


Fig (9) Normal EEG pattern.

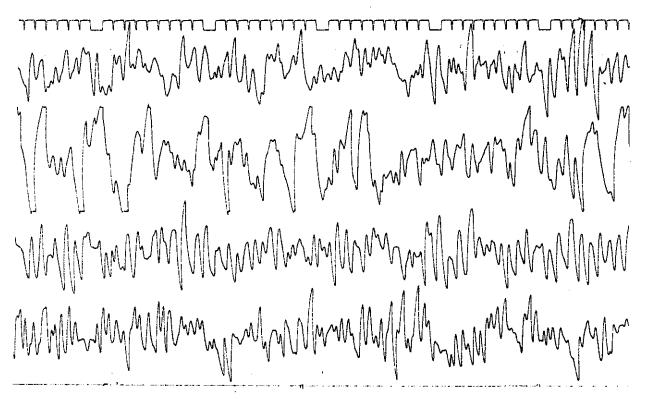


Fig (10) EEG pattern with bilateral epileptogenic discharge.

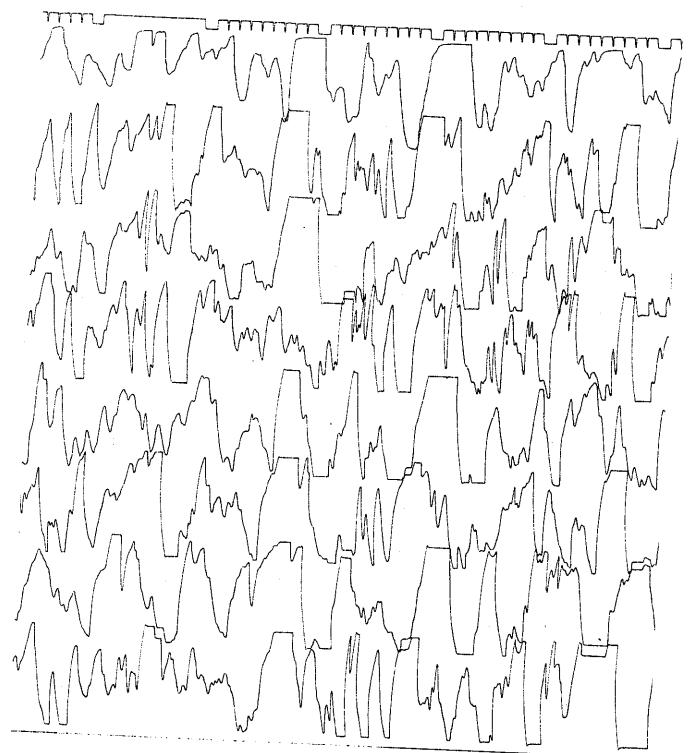


Fig (11) EEG with Chaotic Hypsarrhythmic pattern

Table (11). Number and percentage of patients with normal and delayed mentality.

Group	Global de	th normal and delivelopmental elay	Normal n	
Epilepsy (137)	No.	%	No.	%
Febrile (15)	102	74.4	35	25.5
Acute symptomatic (12)			15	100
otal (164)	2	16.6	10	83.3
Chi-square value= 44.498	104	63.5	60	36.5

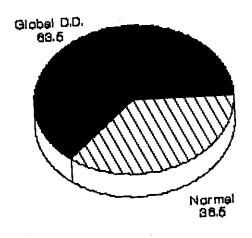


Fig (12) Percent of patients with normal & delayed mentality

Statistical analysis was showing highly significant difference between the epileptic, febrile and acute symptomatic groups regarding the normal and delayed development as the chi square value =44.498 and the value = (0.0001).

Table (12) and Fig (13) show the clinical, EEG and C.T findings in patients with the etiological diagnosis of cerebral palsy (No. =38). In this group, the predominant type of convulsion was generalised clonic (31.5%) followed by generalised tonic (26.3%). (63.1%) of patients were microcephalic. All patients had psychomotor retardation. A diffuse slowing pattern was the main EEG finding in this group (36.8%) and atrophic brain changes were the predominant picture in the C.T of (65.7%) of patients with cerebral palsy.

The clinical, EEG and C.T findings in the idiopathic group (No. =34) are shown in table (13) and Fig (14). Generalized tonic seizures were the commonest occurring in 44.1% of patients. Positive family history was elicited in (20.5%), normal motor and mental development in (100%). Bilateral epileptogenic discharge was found in (38.2%) and normal EEG pattern in (29.4%). Normal C.T was detected in (97%) of patients.

Table (14) and Fig (15) show the clinical data in the group of patients with congenital TORCH infection (No.=21). There was no predominance of any specific type of seizures.

Table (12). Clinical data in the group of cerebral palsy (N= 38).

Variable	Table (12). Clinical data in the g	roup of ce	rebral nalsy (N= 29	٥١
Spiral Canada Spiral Canad		No		<u>).</u>
Generalized tonic 12 31.5	Type of Convulsion			
Generalized tonic 10 26.3	Generalized clonic	12		
Generalized Myoclonic 6	Generalized tonic			
Generalized Myoclonic Generalized Atonic Seneralized Atonic Central Coloric Central	Generalized tonic clonic		<u> 26.3</u>	
Activity Activity	Generalized Myoclonic			_
Partial clonic 2 5.2	Creneralized Atonic			
Perinatal History 32 84.2 At birth cyanonis and hypoxia 32 84.2 Pathologic jaundice 5 13.1 C. N.S. infection in the first 1 2.6 Head Circumferance 5th - 95th percentile 14 36.8 ≤ 5th percentile 24 63.1 1 Tone Examination Normal 1 81.5 Hypertonia (spastic C.P.) 31 81.5 Hypertonia (a tonic diplagia) 7 18.4 Reflex Examination 7 18.4 Normal 7 18.4 Hyper reflexia 31 81.5 Hyporeflexia Motar Development Normal Normal Delayed 38 100 Mental Development Normal Normal 1 2.6 Focal 3 7.8 Diffuse slowing 14 36.8 Bilateral 12 31	Partial clonic			{
At birth cyanonis and hypoxia 32 84.2 Pathologic jaundice 5 13.1 C N S. infection in the first 1 2.6 Head Circumferance 5th - 95th percentile 14 36.8 ≤ 5th percentile 24 63.1 Tone Examination Normal 1 81.5 Hypertonia (spastic C P) 31 81.5 Hypertonia (a tonic diplagia) 7 18.4 Hyper reflexia 31 81.5 Hyporeflexia Motor Development Normal Delayed 38 100 Mental Development Normal 1 2.6 Focal 3 7.8 Diffuse slowing 14 36.8 Bilateral 12 31.5 Multifocal 5 13.1 Hypsarrhythmic 3 7.8 CT Normal 1 2.6 Central & Cortical (Brain 25 65.7 Ischaemic	Perinatal History		5.2	{
C.N.S. infection in the first	At birth evanonis and hypoxia	22		
C.N.S. infection in the first 1 2.6	Pathologic jaundice			
Sth - 95th percentile 14 36.8	- C.N.S. infection in the first			4
Sth percentile	Head Circumferance		2.6	_
Sth percentile 24 63.1	5th - 95th percentile	14		4
Normal	5th percentile			4
Normal	Tone Examination	- - 24 -	63.1	_[
Hypertonia (spastic C.P.) 31 81.5 Hypotonia (a tonic diplagia) 7 18.4 Reflex Examination	Normal			_
Hypotonia (a tonic diplagia) 7		+		_
Normal	Hypotonia (a tonic diplocio)			4
Normal	Reflex Examination		18.4	_
Hyper reflexia 31 81.5 Hyporeflexia	Normal			╛
Hyporeflexia				
Motor Development		$\frac{31}{1}$	81.5	
Normal	Motor Development			╛
Delayed 38 100	Normal		 	_]
Mental Development 38 100 Normal Delayed 38 100 EEG 3 78 Normal 1 26 Focal 3 78 Diffuse slowing 14 36.8 Bilateral 12 31.5 Multifocal 5 13.1 Hypsarrhythmic 3 7.8 CT. Normal 1 2.6 Central & Cortical (Brain 25 65.7 Ischaemic changes 4 10.5 Encephalomalacia 4 10.5 Basal Ganglia Calcification 1 2.6 Infarction (Parafalacine) 2 5.2 Thalamic Calcifications 1 2.6 Special defects 3 2 5.2				_]
Normal	Mental Development	38_	100]
Delayed 38 100	Normal		<u> </u>]
Normal		 	<u> </u>]
Normal	EEG	38_	100]
Focal 3 7.8 Diffuse slowing 14 36.8 Bilateral 12 31.5 Multifocal 5 13.1 Hypsarrhythmic 3 7.8 C.T.]
Diffuse slowing		 	2.6]
Bilateral]
Multifocal 5 13.1 Hypsarrhythmic 3 7.8 C.T. 7.8 7.8 Normal 1 2.6 Central & Cortical (Brain 25 65.7 Ischaemic changes 4 10.5 Encephalomalacia 4 10.5 Basal Ganglia Calcification 1 2.6 Infarction (Parafalacine) 2 5.2 Thalamic Calcifications 1 2.6 Special defects 2 5.2 Blindness 2 5.2	Rilateral		36.8]
Hypsarrhythmic			31.5	
C.T.			13.1]
Normal	CT	3	7.8	ŀ
Central & Cortical (Brain 25 65.7 Ischaemic changes 4 10.5 Encephalomalacia 4 10.5 Basal Ganglia Calcification 1 2.6 Infarction (Parafalacine) 2 5.2 Thalamic Calcifications 1 2.6 Special defects 3 2.6 Squint 4 10.5 Blindness 2 5.2		 -		
Ischaemic changes	Central & Cortical (D:	 		:
Encephalomalacia 4 10.5 Basal Ganglia Calcification 1 2.6 Infarction (Parafalacine) 2 5.2 Thalamic Calcifications 1 2.6 Special defects 2 5.2 Squint 4 10.5 Blindness 2 5.2	Ischaemic charges		65.7	
Basal Ganglia Calcification 1 2.6 Infarction (Parafalacine) 2 5.2 Thalamic Calcifications 1 2.6 Special defects Squint 4 10.5 Blindness 2 5.2	Encephalomatoria			
Thataction (Parafalacine) 2 5.2 Thatamic Calcifications 1 2.6 Special defects Squint 4 10.5 Blindness 2 5.2	Basal Ganglia Coloisanti			
Thalamic Calcifications 1 2.6 Special defects Squint 4 10.5 Blindness 2 5.2	Infarction (Parofalaria)		2.6	
Squint 4 10.5 Blindness 2 5.2	Thalamic Coloifoceiana	2		
Squint 4 10.5 Blindness 2 5.2	Special defects		2.6	
Blindness 2 5.2	Squint			
Speech discardant	Rlindnese			
Special disorder 2 5.2	Speach disorder			
	- incom district	2	5.2	

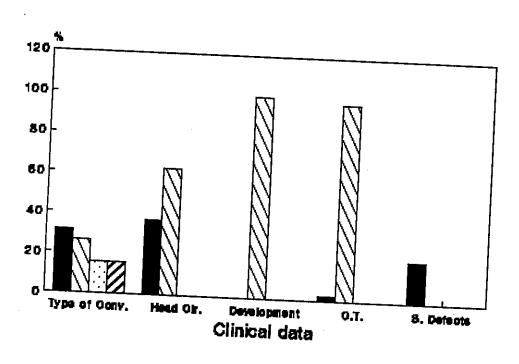


Fig (13) Clinical data in the group of cerebral palsy (No. 38)

Table (13). Clinical, EEG and CT findings in the group of Idiopathic Convulsion (N= 34).

of Idiopathic Convulsion (N= 34).				Jup
variable		lo.	%	_
Type of Convulsion				\dashv
Generalized clonic		6	17.6	\dashv
Generalized tonic	1	5	44.1	
Generalized tonic clonic		7	20.5	\dashv
Generalized Atonic		3	8.8	\dashv
Partial tonic			2.9	ᅱ
Partial clonic			2.9	\dashv
Partial tonic clonic			2.9	ㅓ
Perinatal and maternal data	!		4.7	\dashv
+ve Consanguinity	1.	2	35.2	\dashv
+ve Family history	7	_	20.5	\dashv
Head Circumference measurmen	ıt			\dashv
5th - 95th percentile	34	1	100	\dashv
< 5th percentile	 _	-	100	\dashv
Tone Examination				\dashv
Normal	34	ī	100	-
Hypertonia	 	-	100	\dashv
Hypotonia	 	\dashv		┥
Reflex Examination	<u> </u>			\dashv
Normal	34	T	100	┥
Hyperreflexia	+	╅	100	┨
Hyporeflexia	† <u>. </u>	+		1
Motor Development	<u>. I</u>			$\frac{1}{1}$
Normal	34	\neg	100	1
Delayed	† 	+	100	ł
Mental Development	<u>.l</u> .			
Normal	34	Τ	100	
Delayed		+	100	
EEG findings		Щ.		
Normal	10	T	29.4	
Bilateral epileptogenic activity	13	╁┈	38.2	
Focal epileptogenic activity	8	+-	23.5	
Multifocal epileptogenic activity	3	 	8.8	
C.T. findings	<u> </u>	1_	-0.0	
Normal	33	Τ	97	
Multiple frontal encephalomalacia	1	 	2.9	
		<u></u>	4.7	

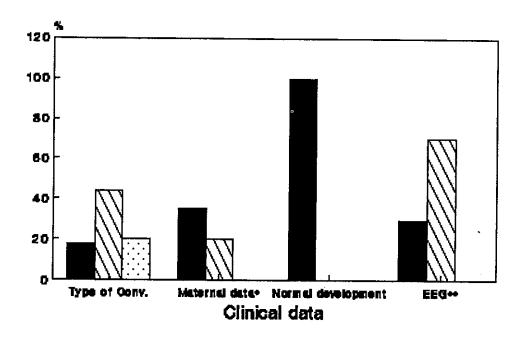


Fig (14) Clinical data in the group of idiopath. conv. (No.=34)

Table (14). Clinical data in the group of TORCH Infection (N=21).

Table (14). Clinical data in the group of	t TORCH Int	ection (N= 21)
Variable	No.	%
Type of Convulsion		
Generalised Clonic	5	23.8
Generalised Tonic	5	23.8
Generalised Tonic Clonic	6	28.5
Generalised Myoclonic	4	19.0
Generalised Atonic	1	4.7
Maternal Data		
+ve Consangunity	10	47.6
Other sibs affected	3	14.2
Abortion in mothers	7	33.3
Head Circumference		
5 th - 95 th percentile	5	23.8
<5 th percentile	16	76.1
Tone Examination		
Normal	3	14.2
Hypertonic	17	80.9
Hypotonia	1	4.7
Reflex Examination		
Normal	3	14.2
Hyperreflexia	17	80.9
Hyporeflexia	1	4.7
Fundus Examination		
Normal	21	100
Motor Development		
Normal		
Delayed	2:1	100
Mental Development		
Normal		
Delayed	21	100
EEG Findings		
- Normal	2	9.5
- Diffuse slowing	9	42.8
- Bilateral epileptogenic activity	6	28.5
- Multifocal epileptogenic activity	2	9.5
- Hypsarrhythmic pattern	1	4.7
- Focal epileptogenic activity	1	4.7
C.T.		
Normal	. 5	23.8
Brain Atrophy	13	61.9
Intracerebral Calcification	3	14.2
Spescial Defect		
Deafness	2	9.5
Blindness	2	9.5
Microphthalmia	1	4.7
Cataract	1	4.7

Abortion was present in (33.3%) of patients. (76.1%) were microcephalic. Global developmental delay was found in (100%) of patients. The EEG was characterized by slowing pattern in (42.8%). Brain atrophy was demonstrated in (61.9%) of patients and intracerebral calcification (Fig 16) in (14.2%) of patients on brain CT scan. Spescial defects as deafness and blindness were present in (9.5%) of patients each.

Table (15) shows the distribution of cases with congenital TORCH infection (No.=21) according to the specific aetiology. In an order of frequency, congenital CMV infection was diagnosed in 11 patients (52.3%), Toxoplasmosis and congenital Rubella in 10 patients (23.8% each).

Table (16) and Fig (17) show the clinical data in the group of febrile convulsions (N=15 patients). This group was characterised mainly by generalized tonic convulsions occurring in (46.6%) of patients, positive family history which was elicited in 26.6% and a normal psychomotor development (100%). The EEG pattern was normal in (46.6%) of patients, and C.T was normal in all patients.

Table (17) and Fig (18) show the clinical data in the group of postmeningitic epilepsy (No. =12 patients). In this group, the type of convulsion was mainly of the generalised tonic clonic type (41.6%). Other types of convulsions were also present. History of C.N.S infection could be traced in (100%)

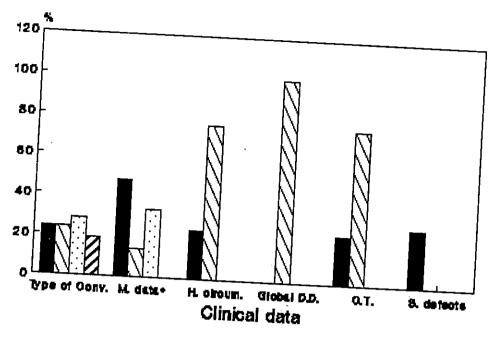


Fig (15) Clinical data in the group of TORCH (N0.=21).

Fig (16) Brain CT showing intracerebral calcification.

Table (15). Distribution of cases with TORCH

Type of congenital infection No.			
C.M.V.+	No.	%	
Toxoplasma	11	52.3	
Rubella	5	23.8	
Herpes simplex	5	23.8	
Total			
+ C.M.V = Cytomacal	21	100	

+ C.M.V. = Cytomegalo virus.

Table (16). Clinical data in the group of febrile convulsion (N= 15).

Table (16). Clinical data in the Variable	group	of febrile co	onvu	lsion (N= 1	
Type of Convulsion		No		%	
Generalized clonic					
Generalized tonic	<u>-</u> -	4		26.6	
Generalized tonic clonic		7		46.6	
Motor I Ti		4			
Maternal History		<u>'</u>		26.6	
+ ve Consanguinity		4		26.6	
+ ve family history		4		26.6	
Head Circumference measurm	ent	<u> </u>		26.6	
percentile		15			
Tone Examination		13		100	
Normal	•	15			
Hypertonia		15	_	100	
Hypotonia					
Reflex Examination					
Normal		r			
Hyperreflexia		15		100	
Hyporeflexia					
Motor Development			_		
Normal					
Delayed		15	7-	100	
	_ T				
Mental Development Normal			ــــــــــــــــــــــــــــــــــــــ		
Delayed		15	7	100	
 -			 	100	
EEG findings Normal					
		7	τ	16.6	
Bilateral epileptogenic activity			46.6		
Focal epileptogenic activity		$\frac{4}{2}$	26.6		
Multifocal epileptogenic activity		2		13.3	
Diffuse slowing				6.6	
.T. findings		1		6.6	
Normal					
		15		00	

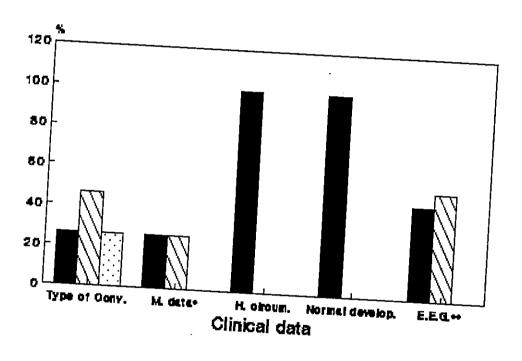


Fig (17) Clinical data in the group of febrile conv. (No.=15)

Table (17). Clinical data in group of Postmeningitic Epilepsy (N= 12). Type of Convulsion No Generalized clonic Generalized tonic 3 25.0 Generalized tonic clonic 2 167 Generalized Myoclonic 5 41.7 Partial Tonic 8.3 Generalized atonic 1 8.3 History of C.N.S. infection (Fever, loss of consciousness) Head Circumference measurment 12 100 5th - 95th percentile < 5th percentile >95th percentile 6 50.0 4 33.3 Tone Examination 2 16.7 Normal Hypertonia 4 33.3 Hypotonia 8 66.7 Reflex Examination Normal Hyperreflexia 4 33.3 Hyporeflexia 8 66.7 Motor Development Normal Delayed Mental Development 12 100 Normal Delayed EEG findings 12 100 Diffuse slowing Focal epileptogenic activity 6 50.0 Bilateral epileptogenic activity 3 25.0 Multifolical epileptogenic activity 2 16.7 C.T.findings 8.3 Brain atrophy Ventricular dilatation and 8 66.7 Bilateral subarachnoid haemorrhage 2 16.7 Left occipital pariatal infarction 1 8.3 Special Defects 1 8.3 Deafness Hemiplegia 8.3

16.6 8.3

Squint

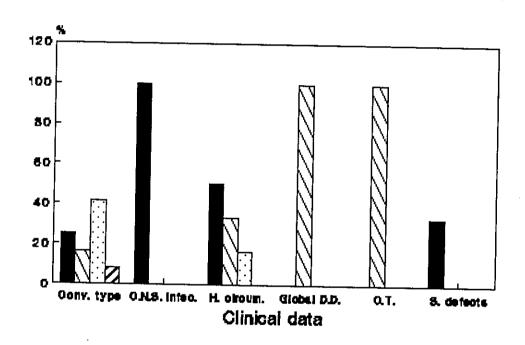


Fig (18) Clinical data in the group of postmeningite epilepsy (No.=12)

of patients. (33.3%) of patients were microcephalic, (50%) with normal head circumference and (16.6%) macrocephalic. All patients (100%) had global developmental delay. The EEG pattern was mainly diffuse encephalopatly in (50%) of patients. Atrophic brain changes were found in the CT of (66.6%) of patients.

Table (18) and Fig (19) show the clinical data in the group of neurodegenerative disorders (No.= 8 patients). In this group, generalized tonic clonic serizures were present in (62.5%) of patients. Positive consanguinity was present in (75%), microcephaly in only 1 patient (12.5%). All patients suffered from psychomotor retardation and regression. Fundus examination had revealed optic atrophy in (62.5%) of patients, cherry red spot of the macula in (25%) of patients. The C.T findings were brain atrophy in 4 patients (50%), and demylinating disease of the C.N.S in (50%) of patients. The final diagnosis was metachromatic leukodystrophy (Fig. 20), which was diagnosed in 4 patients after biochemical assay for aryl sulfatase enzyme in blood and demonstrated a reduced level. Tay sachs disease was diagnosed in 2 cases (25%) by the presence of a reduced level of hexosaminidase enzyme.

One patient was diagnosed as Schilder's disease after assay for plama and urine amino acids as well as organic acids, methylmalonic and methyl propionic acidemia were negative in blood and urine. One patient was diagnosed as infantile

Table (18). Clinical data in the group of Neurodegenerative disorders (N=8).

disorders $(N=8)$.		
Variable	No.	7
Type of Convulsion		%
Generalized Clonic		10.5
Generalized tonic	1	12.5
Generalized tonic clonic	5	12.5
Generalized Myoclonic		62.5
Maternal History		12.5
+ ve Consangunity	6	7-5-
Other sibs affected	2	75.0
Abortion		25.0
Head Circumference measurment 5 th - 95 th peercentile <5 th percentile		12.0
5 th - 95 th peercentile	7	7 05.5
<5 th percentile		87.5
- running examination		12.5
Normal		10.5
Optic atrophy	5	12.5
Cherry red spot of the macula		62.5
Tone Examination		25.0
Normal	5	7
Hypertonia	1 1	62.5
Hypotonia	2	12.5
Reflex Examination		25.0
Normal	-	
Hyperreflexia	5 2	62.5
Hyporeflexia		12.5
Motor Development	<u></u>	25.0
Normal		
Delayed	 	
Mental Development		100
Normal	T	
Delayed		
EEG	8	100
Normal	1 1	
Focal epileptogenic activity	4	12.5
Bliateral epileptogenic activity	2	50.0
Diffise slowing	1 - 4 - 1	25.0
<u>C.T.</u>		12.5
Brain Atrophy	7 4 1	
Demylination of the white matter	4 4	50.0
The final diagnosis	- 4	50.0
Leukodystrophy (Metachromatic)	4	
lay sachs disease (Hexosamindage	1 1	50.0
_ Schilder's disease	1 1	12.5
Neurodegenrative brain disease		12.5
Lipid storage disease (Gaucher	 	12.5
- Cadoner	<u> </u>	12.5

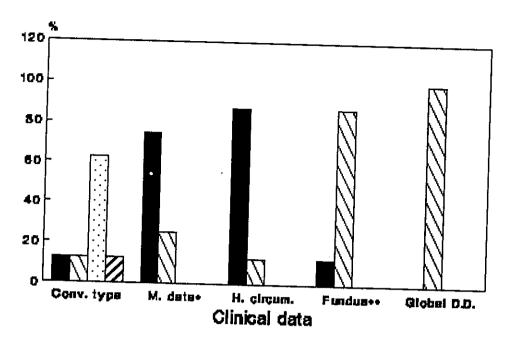


Fig (19) Clinical data in the group of neurodegenerativ disorders (N0.=8)

Fig (20) Brain CT scan showing white matter degeneration (Leukodystrophy)

Gaucher disease by the presence of hepatosplenomegaly and Gaucher cells in bone marrow.

Table (19-a) and Fig (21) represent the clinical data, EEG and C.T in the group of hypocalcemia (No. =8 patients). In this group generalized clonic convulsion was traced in (37.5%) of patients followed by generalised tonic in (25%) of patients. Delayed motor development was found in one patient only with rachitic manifistations while normal mental development was found in (100%) of patients. Normal C.T was found in (100%) of patients.

Estimation of parathormone level in blood had revealed normal level in (50%) of patients, increased and decreased level in (25%) each.

Table (19-b) shows the individual data of patients with hypocalcemia and their control group.

Table (20-a) and Fig (22) show the clinical, EEG and C.T in the group of aminoacidopathy (No. = 6patients). In this group, generalised tonic clonic convulsion was traced in (50%) of patients and generalised myoclonic in (33.3%) of patients. Positive consongunity was elicited in (66.6%) of patients.

Psychomotor retardation was found in (100%) of patients. C.T scan had revealed normal pattern in (50%) of patients and

Table (19-a). Clinical data, EEG and	CT in the group of hype	ocalcemia (N= 8).
Variable	No.	%
Type of Convulsion		
Generalized clonic	3	37.5
Generalized tonic	2	25.0

Type of Convulsion		
Generalized clonic	3	37.5
Generalized tonic	2	25.0
Generalized tonic clonic	1	12.5
Partial clonic	2	25.0
Head Circumferance		
5 th - 95 th percentile	8	100
Tone Examination		
Normal	8	100
Hypertonia		
Hypotonia		
Reflex Examination		
Normal	7	87.5
Hyper reflexia	1	12.5
Hyporeflexia		
Motor Development		
Normal	7	87.5
Delayed	1	12.5
Mental Development		
Normal	8	100
Delayed		<u></u>
EEG		
Normal	1	12.5
Focal epileptogenic activity	4	50.0
Bilateral epileptogenic activity	2	25.0
Multifocal epileptogenic activity	1	12.5
C.T. findings		
Normal	8	100
Parathormone level		
Normal	4	50.0
Increased	2	25.0
Decreased	2	25.0

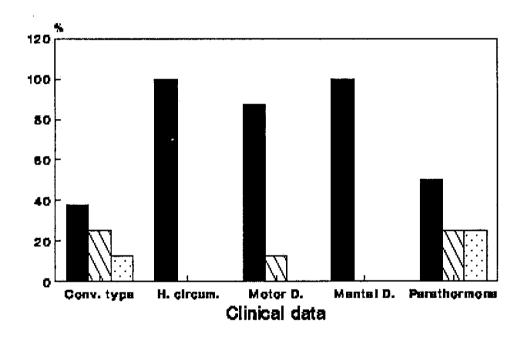


Fig (21) Clinical data in the group of hypocalcemia (No.=8).

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a of cases with hypocalcem
f cases wi
l data o
). Individual
ole (19-b). Inc
Table (1

	J. The state of the state of posting			74/1-1	77077	77		į			
Patient No.	1	2	3	4	S	9	L	8	Range	Mean	SD
Hypocalcemia group (8)	group (8)										
Serum Ca (mg/dl)	7.8	7.2	5.3	3.5	3.5 5.7	5.5	5.5 5.9	6.1	3.5-7.8	5.8	1.3
Ionized Ca++ (mmol/L)	0.82	0.71	0.57	0.30	0.57 0.30 0.58 0.48	0.48	0.73	0.73	0.3-0.82	0.61	0.17
Parathormone level (Pg/dl)	5.8	3.2	11.5	6.5	11.5 6.5 1.16 5.7	5.7	1.18	15.0	15.0 1.16-15.0	6.2	4.9
Control group (13)	(13)										
Serum Ca (mg/dl)									9.1-11.0	76.6	0.53
Ionized Ca++ (mmol/L)							; ;		1.0-1.2	1.08	0.07
Parathormone level (Pg/dl)		:							2.4-26.0	11.04	80.6
											-

Tale (20-a). Clinical data, EEG and CT in the group of aminoacidopathy

N	=	۸١	١.
ITA	_	U,	١.

(N=0).		
Variable	No.	%
Type of Convulsion		
Generalized clonic	1	16.6
Generalized tonic clonic	3	50.0
Generalized myoclonic	2	33.3
Perinatal and maternal data		
+ve consangunity	4	66.6
other sibs affected	1	16.6
Head Circumference measurment		
5 th - 95 th percentile	6	100
Tone Examination		
Normal	1	16.6
Hypertonia	4	66.6
Hypotonia	1	16.6
Reflex Examination		
Normal	2	33.3
Hyper reflexia	4	66.6
Hyporeflexia		
Motor Development		
Normal		
Delayed	6	100
Mental Development		
Normal		
Delayed	6	100
EEG		
Normal	1	16.6
Bilateral epileptogenic activity	2	33.3
Diffuse slowing e modified	2 2	33.3
hypsarrhythmic pattern		
Multifocal epileptogenic activity	1	16.6
C.T. findings		
Normal	3	50.0
Brain atrophy	2	33.3
Diffuse hypodensity of cerebral	1	16.6
and cerebelar white matter		<u></u>
Special features		
Irritability and hyperactivity	4	66.6
Intractable epilepsy	3	50.0
Fair complexion	1	16.6

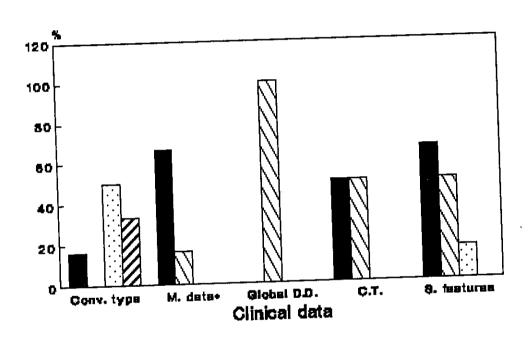


Fig (22) Clinical data in the group of aminoacidopathy (No=6)

brain atrophic changes in (33.3%) of patients. Special features in the form of irritability and hyperactivity were elicited in (66.6%) of patients. Intractable epilepsy was found in 50% of patients.

Table (20-b) show the distribution of Cases with aminoacid disorder. MSUD and PKU were found in (33.3%) of patients each. Urea cycle defects and isovaleric acidaemia were found in (16.6%) of patients each.

Table (21) and Fig (23) show the clinical data in the group of brain malformations (No. =6 patients). The generalized tonic and generalized tonic clonic serizures were found in (33.3%) of patients each. Microcephaly was elicited in (33.3%) of patients while, (16.6%) of patients had macrocephaly. 100% of patients had psychomotor retardation and regression. Brain CT scan had revealed Dandy walker malformations (Fig 24), and lissencephaly (Fig 25), in (33.3%) of patients each. Agenesis of corpus callosum (Fig 26) with and without arachnoid cyst in (16.6%) of patients each.

One patient of those with agenesis of corpus callosum was diagnosed later as Zellweger syndrome as he had intractable progressing macrocephaly, mongoloid facies, bilateral cystic kidneys, hepatomegaly, mental retardation and optic atrophy.

Table (20-b). Distribution of cases with aminoacid disorder.

Type of aminoacid	No.	%
disorder		
MSUD ⁺	2	33.3
PKU++	2	33.3
urea cycle defect	1	16.6
Isovaleric acidaemia	1	16.6
Total	6	100

^{*}MSUD = Maple syrup urine disease.
++PKU = Phenylketonuria.

Table (21). Clinical data, EEG and CT in the group of Brain Malformations (N= 6).

Malformations (N= 6).		
Variable	No.	%
Type of Convulsion		
Generalized tonic	2	33.3
Generalized tonic clonic	2	33.3
Generalized Myoclonic	1	16.6
Partial clonic	1	16.6
Perinatal and maternal data		
+ve Consangunity	2	33.3
+ve family history	44	
Head Circumferance measurment		
5 th - 95 th percentile	3	50.0
< 5 th percentile	2	33.3
>95 th percentile	1	16.6
Tone Examination	· · · · · ·	
Normal	2	33.3
Hypertonia	3	50.0
Hypotonia	1	16.6
Reflex Examination		
Normal	3	50.0
Hyperreflexia	3	50.0
Hyporeflexia		
Motor Development	, <u></u>	•
Normal	T	
Delayed	6	100
Mental Development		· · · · · · · · · · · · · · · · · · ·
Normal		
Delayed	6	100
EEG findings	1	<u> </u>
Diffuse slowing	3	50.0
Focal epileptogenic activity	2	33.3
Hypsarrhythmic pattern	1	16.6
C.T.findings	<u> </u>	!
Hypoplasia of cerebellar vermis	7 2	33.3
, capacious 4th venticle (Dandy-	_	
Walker variant)		
Lissencephaly, diffuse white matter	2	33.3
disease, brain atrophy		
Agenesis of corpus callosum	ī	16.6
Agenesis of corpus callosum with	i	16.6
arachnoid cyst+		

⁺ this case was diagnosed later as Zellweger syndrome.

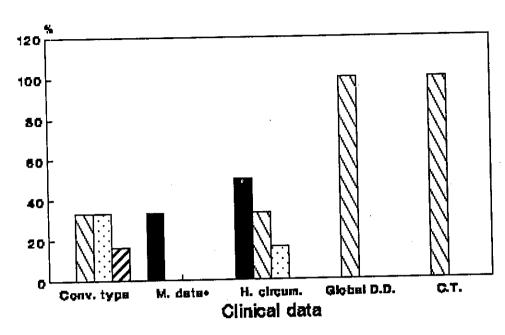


Fig (23) Clinical data in the group of brain malform. (No.=6)

(Fig 24) Brain CT scan of Dandy Walker variant with hypoplasia of the cerebellar vermis, and capacious 4th venticle.

Fig (25) Brain CT of a case with Agyria (Lissencephaly).

Fig (26) Brain CT showing Agenesis of corpus callosum.

Table (22) shows the clinical, EEG and CT findings in the group of cryptogenic West syndrome (No. =5 patients). In this group, no definite aetiology was found, so the term cryptogenic. All the patients had the common features of generalised myoclonic convulsion, global developmental delay and hypsarrhythmic EEG pattern, so the term (west syndrome). 80% of patients had bilateral frontoparietal brain atrophy.

Cyryptogenic otahara syndrome was found in one neoborn female baby with generalised tonic clonic convulsion, global developmental delay, failure to thrive and burst of complex paroxysmal activity separated by periods of suppression on EEG. TORCH profile was negative, aminogram was normal and C.T was normal. So no definite etiology was found in these cases.

Table (23) shows the clinical picture in 4 patients presenting with generalized clonic covulsion following head trauma. 75% of patients had normal head circumference and (25%) were macrocephalic. Psychomotor retardation was found in (50%) of patients. 50% of patients had focal epileptic discharge. Brain haematoma was found in (75%) of patients and subglial hematoma with cerebral atrophy in (25%) of patients on brain CT scanning.

	- Assanic We	st Syndrome (N= 5).
Table (22). Clinical, EEG and CT in the group of (Pryptogenie	%
Table (22). Clinical, DDS	No	L

- L d - group Of	f Crypto	genic West	Syndre	2/	
Table (22). Clinical, EEG and CT in the group of	1	Vo.		%	-
Variable					
Convulsion		5		100	
Camprol 1760 19190010222					
Head circumference measurment		3		60	
5th - 95th percentile		2		40	
< 5 th percentile					
Tone Examination		3		60	1
Normal		$-\frac{3}{2}$	1	40	1
Hypertonia			1-		1
Hyptonic					4
Reflex Examination		3		60	4
Normal		$-\frac{3}{2}$	_	40	4
Hyperreflexia			_		_
Hyporeflex	1				4
Motor Development					_
Normal		5		100_	_
Delayed					
Mental Development		T			
Normal		5		100_	
Delayed					
TOTAL Findings		T 5		100	
Hypsarrhythmic pattern					
C.T		-\-\-_1		20	
				· 80	
Bilateral frontopariatal brain atr	ophy	11 investion	tions (TORCH, amin	ogram
of patients have been subject	ected to	SII IIIAESIIR		•	

This group of patients have been subjected to all investigations (TORCH, aminograme) and proved all to be -ve

Table (23). Clinical, EEG and CT in the Variable	No.	<u>%</u>
Type of Convulsion		
Generalized clonic	4	100
Head Circumference measurmen	nt	
5 th - 95 th percentile	3	75.0
> 95 th percentile	1	25.0
Tone Examination		
Normal	. 4	100
Hypertonia		
Hypotonia		
Reflex Examination		
Normal	4	100
Hyper reflexia		
Hyporeflexia		
Motor Development		
Normal	2	50.0
Delayed .	2	50.0
Mental Development		
Normal	2	50.0
Delayed	2	50.0
EEG		
Normal	1	25.0
Focal	2	50.0
Diffuse slowing	1	25.0
СТ		
Brain Hematoma	3	75.0
Subglial hematoma and diffuse cerebral atrophy	1	25.0

Table (24) shows the clinical picture of two patients with autosomal recessive microcephaly. One of them had generalized clonic convulsion while the other one had generalized myoclonic convulsion. They had microcephaly and psychomotor retardation. Focal epileptogenic EEG pattern was found in one patient while the other one had diffuse high voltage slow wave discharge. Both patients had a normal brain CT scan.

Our study included four miscellaneous cases in the symptomatic epileptic group. They were: one case with Down syndrome, one case with brain ischaemia, one with mitochondrial encephalomyopathy and one with brain granuloma. This one with Down syndrome had the peculiar clinical picture of the syndrome in addition to generalized clonic convulsion, hypotonia and hyporeflexia, normal head circumference, psychomtor retardation with diffuse slowing EEG pattern and brain atrophic changes on brain CT scan.

The patient with brain ischaemia had a history of gasteroenteritis, dehydration, epilepsia partialis continua, microcephaly, global developmental delay, diffuse high voltage slow wave EEG pattern and brain atrophic changes on brain CT scan.

Table (24). Clinical, EEG and CT in the group of autosomal recessive micorcephaly (N= 2).

(14-2).	
No.	%
· · · · · · · · · · · · · · · · · · ·	
1	50.0
1	50.0
Perinatal and maternal data	
1	50.0
2	100
2	100
2	100
Motor Development	
	
2	100
2	100
1	50.0
1	50.0
2	100
	No. 1 1 1 2 2 2 2 1 1 1 1

The patient with mitochondrial encephalomyopathy had generalised clonic convulsion, maternal history of perivious abortion, negative consanguinity microcephaly, hypotonia, hyporeflexia, global developmental delay, bilateral epileptic discharge, and normal brain CT. Muscle biobsy had revealed mitochondrial myopathy.

The patient with brain granuloma, had partial clonic convulsion, normal head circumference, normal development, focal central epileptic focus on EEG (Fig 27). Brain CT had revealed brain granuloma (Fig 28) and MRI also had revealed this granuloma.

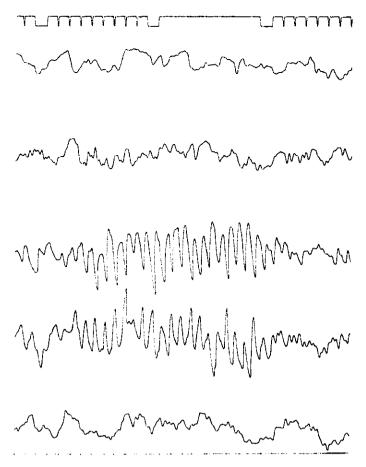


Fig (27) EEG pattern with focal epileptic discharge

(Fig 28) Brain CT scan delineating a brain granuloma.