

Clinical Audit on Management of Neonatal Convulsion in Neonatology Unit of Assiut University Pediatric Hospital

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Abstract: Objective: Is to investigate how much the protocol of diagnosis and management of neonatal convulsions is practically implemented in the NICU of AUCH. **Methods:** The target population of this retrospective study were all neonates who were admitted to NICU with neonatal seizures during the period between the first of January 2015 to 31th of December 2015. The study included all newborn infants with convulsions occurring during the neonatal period, within the first 4 weeks of life. Cases of neonatal convulsions were diagnosed according to clinical suspicion from history, physical examination, neurological examination and laboratory investigation. **Results:** The study included 175 patients with neonatal convulsions. 117 cases were males and 58 were females. Data of the study showed that AUCH partially followed the reference standard of the study.

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1. Introduction:

The word *Audit* borrowed from economics and stands for the examination of records or financial accounts with the purpose of checking their accuracy. In a wider sense, an *audit* can be described as an inspection of the accounting procedures and records by a trained accountant, as it happens in business management or information technology (Simon, 2008).

Clinical Audit is a term which has acquired different meanings over time in relation to health care quality. Ten years ago the National Institute for Clinical Excellence (NICE, 2002) published the first manual of Clinical Audit, with the classical definition "Clinical audit is a quality improvement process that seeks to improve patient care and outcomes through systematic review of care against explicit criteria and the implementation of change". (NICE, 2002).

The most prominent feature of neurologic dysfunction in the neonatal period is the occurrence of seizures. Determining the underlying etiology for neonatal seizures is critical. Etiology determines prognosis and outcome and guides therapeutic strategies (Volpe, 2000).

The neonatal period is limited to the first 28 days of life in a term infant. For premature infants, this term usually is applied until gestational age 44 weeks; ie, the age of the infant from conception to 44 weeks (ie, 4 wk after term). Neonatal seizures by definition occur within the first 4 weeks of life in a full-term infant and up to 44 weeks from conception for premature infants. Seizures are most frequent during the first 10 days of life (Volpe, 2000).

Neonatal seizures are paroxysmal (EEG) activity often with motor manifestations and sometimes with autonomic or behavioural clinical manifestations including effects on respiration, heart rate and blood pressure. Frequent or prolonged seizures may contribute to a worsening of brain injury (Glass and Wirrell, 2009).

Neonatal seizures are abnormal electrical discharges in the CNS of neonates and usually manifest as stereotyped muscular activity or autonomic changes. Diagnosis is confirmed by EEG; testing for causes is indicated. Treatment depends on the cause. Some apparent clinical seizure-like activity, e.g. jitteriness and irritability, is not associated with EEG abnormality. These are not seizures and do not require treatment (Bassanet al., 2008).

Seizures in neonates are relatively common, with variable clinical manifestations. Their presence is often the first sign of neurologic dysfunction, and they are powerful predictors of long-term cognitive and developmental impairment (Lombroso, 2007).

Seizures occur in up to 1.4% of term infants and 20% of premature infants. Seizures may be related to a serious neonatal problem and require immediate evaluation. Most neonatal seizures are focal, probably because generalization of electrical activity is impeded in neonates by lack of myelination and incomplete formation of dendrites and synapses in the brain. Some neonates undergoing EEG to assess seizures or other symptoms of encephalopathy (eg, hypoactivity, decrease responsiveness) are found to have clinically silent seizures (≥ 20 sec of rhythmic epileptiform electrical activity during an EEG but

without any clinically visible seizure activity). Occasionally, clinically silent electrical activity is continuous and persists for > 20 min; at that point, it is defined as electrical status epilepticus (**Margaret and McBride, 2013**).

2. Methods:

The aim of this clinical audit study is to assess how much the adapted protocols of diagnosis and management of neonatal seizures are applied in NICU in AUCH, it was carried out in Neonatal Intensive Care Unit (NICU) in Assuit University Children Hospital during the period between the first of January 2015 to 31th of December 2015. The study included all newborn infants with convulsions occurring during the neonatal period, within the first 4 weeks of life in a full-term infant and up to 44 weeks from conception for premature infants, whatever the underlying cause of convulsions which may be due to IEM, Hypoglycemia, Electrolyte disturbance, HIE, Cerebral hemorrhage, Meningitis, Benign neonatal syndromes, Neonatal jaundice (kernicterus) and Neonatal sepsis. Any newborn infant with abnormal movements that mimic convulsions and convulsions occurring beyond the neonatal period were excluded from the study. Cases of neonatal convulsions were diagnosed according to clinical suspicion from history, physical examination, neurological examination and laboratory investigation.

History included, Gestational age (preterm, fullterm), Family history of convulsion, Previous sick baby with convulsions, Maternal diseases (diabetes mellitus or immune disorders), Maternal drugs (sulfonamides, aspirin, antimalarials), Mode of delivery (NVD, CS), and Birth trauma.

Examination included:, Gestational age assessment, Head circumference if there is microcephaly or not, Cephalohematoma, Bruises and petechiae, Signs suggestive of intracranial hemorrhage (apnea, changes in blood pressure and heart rate, decreased muscle tone, decreased reflexes, excessive sleep, lethargy, weak suck, seizures and other abnormal movements, pallor), Pallor, Eye examination:e.g. Cataract or signs of chorioretinitis, Chest examination, Cardiac examination, Abdominal examination: Hepatosplenomegaly, Neurological examination:-Primitive reflexes, Muscle ton.,. Activity, and abnormal. Movements.

Lab investigations, First line investigation for all children, included CBC (mainly for Hb and Hct but also WBC as a non-specific marker of infection) and Peripheral blood smear. Bilirubin (total, direct, indirect). Maternal blood group and Rh status. Infant blood group and Rh status. BGL. Serum electrolytes, and CSF. Second line investigations included, CT and MRI, EEG, Liver function tests (if biphasic or

conjugated hyperbilirubinemia is diagnosed), TORCH (if congenital infection is suspected) PT, PC (if there is liver disease), Reducing substances in urine (if metabolic disorder is suspected), Metabolic screening (if metabolic disorder is suspected) and abdominal Ultrasonography (if clinically indicated, e.g. baby has organomegaly).

Regarding the Management, the steps of management were: Resuscitation and supportive measures, Assessment and treatment of the underlying cause, and Anticonvulsant therapy according to (**WHO, 2012**).

3. Results:

Table (1) shows that 175 children with neonatal seizures were included in this study. one hundred and seventeen (66.9%) were males and fifty eight (33.1%) were females, with highest age group (49.4%) were 1 - 5 days and majority of children were full term (81.1%).

Table (1): Personal data of the studied neonates

	No. (n= 175)	%
Name:		
Asked	175	100.0
Not asked	0	0.0
Postnatal age:		
Asked	164	93.7
Not asked	11	6.3
Age groups:		
< 1 day	18	11.0
1 - 5 days	81	49.4
> 5 days	65	39.6
Sex:		
Asked	175	100.0
Not asked	0	0.0
Sex distribution:		
Male	117	66.9
Female	58	33.1
Gestational age:		
Full-term	142	81.1
Pre-term	33	18.9

Table (2) demonstrates that 100% of the studied neonates were normal heart rate whereas 59.4% were abnormal respiratory rate. About 44% were abnormal blood pressure, more than 22% were abnormal temperature and 13.7 were abnormal heart examination. Vast majority of cases (80.6%) were abnormal neurological examination.

Table (3) shows that the 27.4% of neonates were having maternal risk factors, most of them 27.1% were PROM.

Table (4) illustrates Obstetrical risk factors among studied patients, 36% of them were positive

obstetric risk factors. More than one third of them 38.1% were obstructed labour.

Table (5) shows the clinical types and characteristics of neonatal convulsions. With more than one half of neonates (50.6%) with subtle convulsions, most of them (83.1%) were frequently with convulsions, Only 5.7% of neonates were positive Family history of neonatal convulsions.

Table (2) Clinical examination data of the studied neonates.

6.5	No. (n= 175)	%
Heart rate:		
Normal	175	100.0
Abnormal	0	0.0
Not done	0	0.0
Respiratory rate:		
Normal	71	40.6
Abnormal	104	59.4
Not done	0	0.0
Blood pressure:		
Normal	98	56.0
Abnormal	77	44.0
Not done	0	0.0
Temperature:		
Normal	136	77.7
Abnormal	39	22.3
Not done	0	0.0
General examination:		
Normal	142	81.1
Abnormal	33	18.9
Not done	0	0.0
Chest examination:		
Normal	175	100.0
Abnormal	0	0.0
Not done	0	0.0
Heart examination:		
Normal	151	86.3
Abnormal	24	13.7
Not done	0	0.0
Abdominal examination:		
Normal	160	91.4
Abnormal	15	8.6
Not done	0	0.0
Neurological examination:		
Normal	34	19.4
Abnormal	141	80.6
Not done	0	0.0

Table (6) reveals Laboratory investigations of the studied neonates. About 37.1% of them were abnormal RBS and 28% were abnormal RBCs. 25.1 were abnormal coagulation profile.

Table (7) illustrates the Imaging studies of the studied neonates. Most of them were abnormal TICUS where as 22.3% were abnormal EEG.

Table (8) reveals the causes of neonatal convulsions. With highest percentage (30.9%) was due to hypoglycemia.

Table (9) shows the management of neonatal convulsions.

Table (10): Showing the Anti-convulsant drugs were used, most of them (68%) received phenobarbital with nearly equal halves between Drug withdrawal and starting Start oral drugs.

Table (11): highlight on the number of anti-convulsant drugs used, most of them (57.7%) of the neonates received single drug therapy.

Table (12): showing the antibiotics that were used, vast majority of neonates (98.9%) received antibiotics.

Table (3): Maternal risk factors

	No. (n= 175)	%
Maternal risk:		
Yes	48	27.4
No	124	70.9
Not asked	3	1.7
Type of maternal risk:		
Antepartum-Hge.	4	8.3
DM	6	12.5
DM-HTN	2	4.2
DM + infertility	1	2.1
Eclampsia	1	2.1
HTN	5	10.4
HTN + infertility	1	2.1
Incompetent cervix	1	2.1
Infertility	4	8.3
Maternal drug intake	3	6.3
Placenta previa	1	2.1
Pre-eclampsia	1	2.1
PROM	13	27.1
RHD	2	4.2
Thrombocytopenia	1	2.1
Toxoplasmosis	2	4.2

Table (4): Obstetrical risk factors.

	No. (n= 175)	%
Obstetrical risk:		
Yes	63	36.0
No	108	61.7
Not asked	4	2.3
Obstetrical risk:		
Bleeding	4	6.3
Cord prolapse	4	6.3
Fetal bradycardia	13	20.6
Fetal deceleration	2	3.2
Meconium aspiration	1	1.6
Obstructed labour	24	38.1
Prolonged labuar	5	7.9
PROM	5	7.9
Rupture uterus	5	7.9

Table (5): Clinical types and characteristics of neonatal convulsions.

	No. (n= 175)	%
Convulsion:		
Yes	175	100.0
No	0	0.0
Asking about type of convulsion:		
Asked	158	90.3
Not asked	17	9.7
Type of convulsion:		
Tonic	73	46.2
Clonic	61	38.6
Myclonic	5	3.2
Subtle	80	50.6
Asking about number of convulsions:		
Asked	166	94.9
Not asked	9	5.1
Number of convulsions:		
One	14	8.4
Two	10	6.0
Three	4	2.4
Frequent	138	83.1
Asking about duration of convulsion:		
Asked	151	86.3
Not asked	24	13.7
Duration of convulsion:		
≤ 1 min	76	50.3
> 1 min	75	49.7
Asking about conditions mimic convulsion:		
Asked	138	78.9
Not asked	37	21.1
Conditions mimic convulsion:		
Yes	15	10.9
No	123	89.1
Family history of neonatal convulsions:		
Positive	10	5.7
Negative	124	70.9
Not asked	41	23.4

Table (6): Laboratory investigations of the studied neonates.

	No. (n= 175)	%
RBS:		
Normal	104	59.4
Abnormal	65	37.1
Not done	6	3.4
Calcium:		
Normal	125	71.4
Abnormal	45	25.7
Not done	5	2.9
Potassium:		
Normal	129	73.7
Abnormal	41	23.4
Not done	5	2.9
Magnesium:		
Normal	6	3.4

Abnormal	0	0.0
Not done	169	96.6
Sodium:		
Normal	138	78.9
Abnormal	32	18.3
Not done	5	2.9
CBC:		
Normal	120	68.6
Abnormal	49	28.0
Coagulation profile: (PT, PC, PTT)		
Normal	72	41.1
Abnormal	44	25.1
Not done	59	33.7
ABG:		
Normal	65	37.1
Abnormal	63	36.0
Not done	47	26.9
CSF analysis:		
Normal	1	0.6
Abnormal	17	9.7
Not done	157	89.7
CSF culture:		
Normal	4	2.3
Abnormal	1	0.6
Not done	170	97.1
Blood culture:		
Normal	81	46.3
Abnormal	67	38.3
Not done	27	15.4
TORCH:		
Normal	22	12.6
Abnormal	0	0.0
Not done	153	87.4
Serum ammonia:		
Normal	33	18.9
Abnormal	24	13.7
Not done	118	67.4

Table (7): Imaging studies of the studied neonates.

	No. (n= 175)	%
TCUS:		
Normal	21	12.0
Abnormal	104	59.4
Not done	50	28.6
CT:		
Normal	10	5.7
Abnormal	49	28.0
Not done	116	66.3
MRI:		
Normal	3	1.7
Abnormal	5	2.9
Not done	167	95.4
EEG:		
Normal	8	4.6
Abnormal	39	22.3
Not done	128	73.1

Table (8): Causes of neonatal convulsions.

	No. (n= 175)	%
Hypoglycemia	54	30.9
Hypocalcemia	34	19.4
HIE	31	17.7
Sepsis	27	15.4
Hemorrhage	23	13.1
Hyponatremia	20	11.4
Inborn error of metabolism	19	10.9
Meningitis	17	9.7
Kernicterus	17	9.7
Brain anomalies	15	8.6
Hypomagnesemia	0	0.0

Table (9): Management of neonatal convulsions.

	No. (n= 175)	%
Basic life support:		
Done	175	100.0
Not done	0	0.0
Glucose10% IV:		
Taken (Hypoglycemia)	65	100.0
Not taken	0	0.0
Results of IV glucose10% intake:		
Well-corrected	63	96.9
Not -corrected	2	3.1
Calcium:		
Taken (Hypocalcemia)	45	100.0
Not taken	0	0.0
Results of calcium intake:		
Well-corrected	37	82.2
Not -corrected	8	17.8
Magnesium:		
Taken	0	0.0
Not taken	175	100.0
Pyridoxine 50mg IV:		
Taken	0	0.0
Not taken	175	100.0

Table (10): Anti-convulsant drugs.

	No. (n= 175)	%
Phenobarbital	119	68.0
Phenytoin	40	22.9
Midazolam	40	22.9
Diazepam	3	1.7
Drug withdrawal	91	52.0
Start oraldrug	88	50.3

Table (11): Number of anti-convulsant drugs used.

Number ofdrugs	No.	%
No drugs	31	17.7
Single drug therapy	101	57.7
Multiple drug therapy	43	24.6

Table (12): Antibiotics.

Antibiotics	No. (n= 175)	%
Yes	173	98.9
No	2	1.1

Conclusion:

Neonatal seizures are a manifestation of neurological dysfunction. They are paroxysmal electroencephalographic (EEG) activity often with motor manifestations and sometimes with autonomic or behavioural clinical manifestations.

The aim of this retrospective clinical audit study is to investigate how much the protocol of diagnosis and management of neonatal convulsions is practically implemented in the NICU of AUCH.

The study included 175 patients with neonatal convulsions. 117 cases were males and 58 were females. Data of the study showed that AUCH partially followed the reference standard of the study.

Recommendations:

To improve the process of diagnosis, management and therefore the outcome of neonatal convulsions in newborn infants attending to the Neonatology Unit of AUCH the following recommendations are suggested:

1. A full history of antenatal care, maternal and obstetric risk factors must be taken for all cases in details.

2. Inquiring about family history of neonatal convulsions in previous siblings.

3. Imaging studies of the cranium should not be performed to determine the presence or absence of clinical seizures or to evaluate the efficacy of treatment with AED in neonates. These may be done as apart of the comprehensive evaluation of the etiology of neonatal seizures or to determine prognosis in neonates with seizures.

4. If available, all clinical seizures in the neonatal period should be confirmed by EEG. EEG should not be performed for the sole purpose of determining the etiology in neonate with clinical seizures.

5. In absence of hypoglycaemia, hypocalcaemia, meningitis or other obvious underlying etiology such as HIE, intracranial hemorrhage or infarction, pyridoxine treatment may be considered before antiepileptic drug treatment.

6. Phenobarbital should be used as first-line for treatment of neonatal seizures, and it should be made readily available in all settings.

7. In neonates who continue to have seizures despite administering the maximum tolerated dose of Phenobarbital, either benzodiazepine, phenytoin or lidocaine may be used as second-line for control of seizures.

8. In neonate with normal neurological examination and/or normal EEG, consider stopping AED if neonate has been seizure-free for >72 hours.

The AED should be reinstated in case of recurrence of seizures.

9. Neonates in whom seizures control was achieved with a single AED, the drug can be discontinued abruptly without any tapering of the doses. In neonates requiring more than one antiepileptic drug for seizure control, the drugs may be stopped one by one, with Phenobarbital being the last drug to be withdrawn.

10. In the absence of clinical seizures, neonates with HIE need not be given prophylactic treatment with Phenobarbital.

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