International Journal of Current Advanced Research
ISSN: O: 2319-6475, ISSN: P: 2319-6505, Impact Factor: 6.614
Available Online at www.journalijcar.org
Volume 8; Issue 12 (D); December 2019; Page No. 20810-20818
DOI: http://dx.doi.org/10.24327/ijcar.2019.20818.4075

Research Article

A PROSPECTIVE OBSERVATIONAL STUDY ON INCIDENCE AND MANAGEMENT OF THROMBOCYTOPENIA IN TERM NEWBORNS WITH BIRTH ASPHYXIA ADMITTED IN SNCU, GGH, KURNOOL

ReddypoguJonaMethusala,* Aluri Dinesh Kumar, Sayila Keerthana, Damam Sivaleela, and Agraharam Radhika

Department of Pharmacology,Dr. K.V.Subba Reddy Institute of Pharmacy, Kurnool, Andhra Pradesh, India

A R T I C L E I N F O

Article History:
Received 10th September, 2019
Received in revised form 2nd October, 2019
Accepted 26th November, 2019
Published online 28th December, 2019

Key words:

Thrombocytopenia, Birth asphyxia, Term Newborns, Incidence and Platelets count

A B S T R A C T

Background: Thrombocytopenia is a deficiency of platelets (thrombocytes), which increases the risk of bleeding. Aim: To study the incidence and management of Thrombocytopenia in Birth asphyxia of all term neonates admitted in SNCU, GGH Kurnool. **Objectives:** The main objective of the study is to collect data regarding the thrombocytopenia cases in the Sick New Born Care Unit. To assess the outcome of the management in the thrombocytopenia of the newborns. **Methods:** The data from medical records of all the cases of birth asphyxia admitted in SNCU, GGH, KURNOOL. Over the study period of six months were assessed and documented further study. The newborns were selected based on inclusion and exclusion criteria. **Results:** Total admissions to the SNCU over the study period was 1750 with 150 reported cases of birth asphyxia and hence the incidence of birth asphyxia 60% and thrombocytopenia 40%. The newborns with thrombocytopenia as well as birth asphyxia involves 55(36.6%) males and 35(23.3%) females. The gestational age at delivery of cases ranged from 37<42 weeks. The total discharged thrombocytopenia cases in the overall population are 68 and the remaining 20 cases were expired. The good management was observed in this study. **Conclusion:** This study concludes that the management methods included in SNCU, GGH, Kurnool were innovative. Thus good management was recorded in this study.

INTRODUCTION

Perinatal asphyxia, neonatal asphyxia or birth asphyxia is the medical condition resulting from deprivation of oxygen to a newborn infant that lasts long enough during the birth process to cause physical harm, usually to the brain. Hypoxic damage can occur to most of the infant's organs (heart, lungs, liver, gut, kidneys), but brain damage is of most concern and perhaps the least likely to quickly or completely heal. In more pronounced cases, an infant will survive, but with damage to the brain manifested as either mental, such as developmental delay or intellectual disability, or physical, such as spasticity.[1]Perinatal asphyxia, neonatal asphyxia or birth asphyxia is the medical condition resulting Neonatal thrombocytopenia is defined as a concentration of platelets in the blood falling below the "reference range" for gestational age. In adults, non-neonatal children, and term neonates, this lower reference range limit is 150,000/μL, and therefore thrombocytopenia is defined as a platelet count Perinatal asphyxia, neonatal asphyxia or birth asphyxia is the medical condition resulting <150,000/μL.[2]

Corresponding author:* **ReddypoguJonaMethusala
Department of Pharmacology,Dr. K.V.Subba Reddy Institute of Pharmacy, Kurnool, Andhra Pradesh, India

Perinatal asphyxia, neonatal asphyxia or birth asphyxia is the medical condition resulting Based on Platelet count thrombocytopenia is classified as :

Perinatal asphyxia, neonatal asphyxia or birth asphyxia is the medical condition resulting Mild –

1,00,000 to 1,50,000/μL
Moderate – 50,000 to 99,000/ μL
Severe - < 50,000

Perinatal Hypoxic-ischemic encephalopathy (HIE) is an important cause of brain injury in the newborn and can result in long-term devastating consequences. Perinatal hypoxia is a vital cause of long-term neurologic complications varying from mild behavioural deficits to severe seizure, mental retardation, and/or cerebral palsy in the newborn.

Epidemology

A 2008 bulletin from the World Health Organization estimates that 900,000 total infants die each year from birth asphyxia, making it a leading cause of death for newborns.[3] In the United States, intrauterine hypoxia and birth asphyxia was listed as the tenth leading cause of neonatal death.[4] Perinatal hypoxic-ischemic encephalopathy (HIE) occurs in one to three per 1000 live full-term births [5]. Of affected newborns, 15%–

20% of affected newborns will die in the postnatal period, and an additional 25% will develop severe and permanent neuropsychological sequelae, including mental retardation, visual motor or visual perceptive dysfunction, increased hyperactivity, cerebral palsy, and epilepsy [6].

Etiology

In term newborns, asphyxia can occur in utero and during labor and delivery as a result of impaired placental gas exchange. Preconceptional risk factors for asphyxia are maternal age ≥ 35 years, social factors, family history of seizures or neurologic disease, infertility treatment, previous neonatal death etc.

Antepartum risk factors include maternal prothrombotic disorders and proinflammatory states, maternal thyroid disease, severe preeclampsia, multiple gestation, chromosomal/ genetic abnormalities, congenital malformations, intrauterine growth restriction, trauma, breech presentation and antepartum hemorrhage.

Numerous intrapartum risk factors for asphyxia are recognized, including abnormal fetal heart rate during labor, chorioamnionitis/maternal fever, thick meconium, operative vaginal delivery, general anesthesia, emergency cesarean delivery, placental abruption, umbilical cord prolapse, uterine rupture, maternal cardiac arrest, and fetal exsanguination.

Asphyxia can also occur in the immediate postnatal period, usually secondary to pulmonary, neurological or cardiovascular abnormalities. It should be noted that, in many cases, the timing of asphyxia cannot be established with certainty.[7]

Clinical Manifestations

Apgar Score

The Apgar score is a clinical indicator commonly used to describe the newborn's physical condition at birth. A hypoxic-ischemic insult, but also many other non-asphyxial factors such as maternal analgesia, prematurity and infection, may cause depression of the Apgar score. A prolonged depression of the Apgar score has been shown to be related with death or severe neurodevelopmental outcome [8].

Neonatal Encephalopathy

Neonatal encephalopathy (NE) has been defined as "a clinically defined syndrome of disturbed neurological function in the earliest days of life in the term infant, manifested by difficulty with initiating and maintaining respiration, depression of tone and reflexes, subnormal level of consciousness, and often by seizures" [9] . In the past, it was assumed that the primary etiology of NE was hypoxia-ischemia. Indeed, the term NE is simply a clinical description of disturbed neurological function, irrespective of etiology or pathogenesis. Currently, NE is considered a nonspecific response of the brain to injury that may occur through multiple causal pathways [10].

Hypoxia-ischemia represents one of these pathways, and therefore the term hypoxic-ischemic encephalopathy (HIE) should be reserved for the sub-set of cases of NE with a good evidence of a recent hypoxic-ischemic cause. Robertson *et al*. [11] define HIE as "an acute non-static encephalopathy caused by intrapartum or late antepartum brain hypoxia and ischemia". This clinical condition evolves during the first days of life after significant hypoxic-ischemic insult, and is a leading predictor of neurodevelopmental disability [12]. Sarnat and Sarnat [13] classified HIE into 3 clinical stages: mild (stage 1), moderate (stage 2) and severe (stage 3) encephalopathy. Infants who develop HIE show alterations in the level of consciousness and in the behavior ranging from hyper-alertness/irritability through lethargy/ obtundation to stupor/coma. Disorders of tone ranging from an increase to a marked decrease, and a spectrum of abnormal movements from tremors and jitteriness to frank seizures may be observed. Other clinical manifestations of HIE include apnea with bradycardia and oxygen desaturation, feeding difficulty, shrill cry, exaggeration of the Moro reflex, increased deep tendon reflexes, and decorticate or decerebrate posturing. The severity of HIE symptoms reflects the timing and duration of the insult [14]. In contrast to other etiologies of NE (genetic disorders, brain malformations, metabolic defects etc.), HIE is a potentially modifiable condition, and therefore it is of crucial importance to ascertain the presence or absence of hypoxia-ischemia. The estimation of incidence of NE and HIE and the identification of their risk factors are problematic due to the lack of universal agreed definitions.

The incidence of NE has been estimated to be 2.5 to 3.5 per 1,000 live births); on the other hand, the incidence of HIE has been estimated to be 1.3 to 1.7 per 1,000 live births. It has been estimated that 30% of cases of NE in developed countries and 60% of cases in developing countries have evidence of intrapartum hypoxia-ischemia [15].

Clinical Manifestations of HIE Stages

Severity of hie	Mild	Moderate	Severe
Level of consciousness	Alert/Hyperalert	Lethargy	Stupor or coma
Tone	Normal/Hyperton ia	Hypotonia (focal or general)	Flaccidity
Tendon Reflexes	Increased	Increased	Decreased/Absent
Sucking	Weak	Weak to Absent	Absent
Moro Reflexes	Exaggerated	Incomplete	Absent
Pupils	Dilated	Constricted	Diviated,Dilated, Non-Reactive to light
Heart Rate	Tachyardia	Bradycardia	Variable
Respiration	Normal	Periodic breathing	Apnea
Others	Irritability, Jitterness	Brain stem dysfunction	± Elevated intracranial pressure
Seizures	Absent	±	Frequent, Often Refractory to anti-convulsants
EEG background	Normal	Low voltage, Periodic/Paroxysmal	Periodic /Isoelectric

Pathogenesis of Hypoxic Ischemic Encephalopathy

After perinatal hypoxia-ischemia, different sequences of pathologic events may occur, culminating in brain injury (Fig.). In newborn animals, the phases of primary and secondary energy failure have been recognized, based on characteristics of the cerebral energy state [16]. In the phase of primary energy failure, reductions in cerebral blood flow, in O2 /substrates and in highenergy phosphorylated compounds (ATP and phosphocreatine) have been observed; furthermore, tissue acidosis is prominent. This phase represents an essential prerequisite for all subsequent pathologic events.

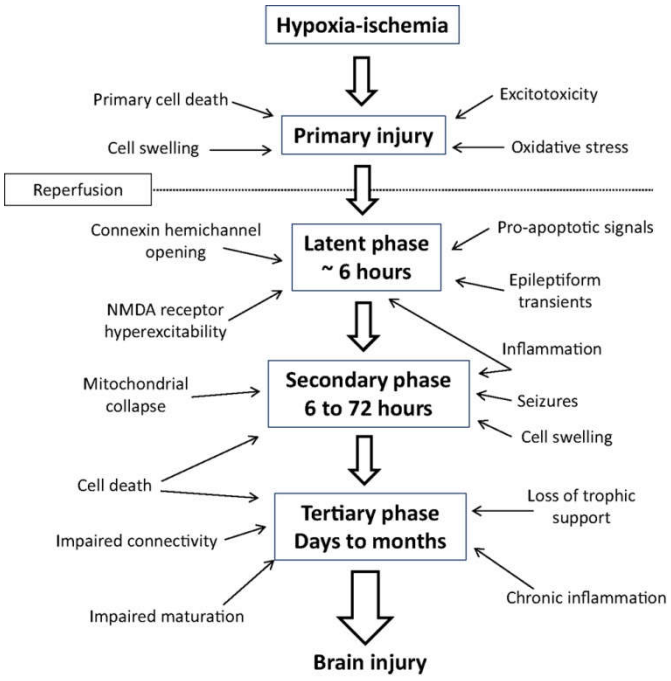


Figure 1 Schematic representation of primary and secondary energy failure in the brain following perinatal asphyxia.

Management of Thrombocytopenia

Risk of Bleeding

The risk of bleeding is increased where there is

- Decreased production rather than increased destruction
- Platelet function defect plus thrombocytopenia
- The platelet count < 50 x 10^9/L.

Platelet Transfusion

There is no accepted 'safe' level of platelets in neonates. Platelet transfusions need to be considered for each individual based on:

- The platelet count
- Clinical circumstances
- The presence or absence of bleeding treat the underlying cause if possible
- This should transiently increase the platelet count by 50-100 x 109/L. Transfuse over 30 minutes and monitor platelet count one hour post transfusion.
- The recommended volume of platelets to be transfused is 10-20 mL/kg.
- Potential triggers for platelet transfusion (see the Table below).

Potential Triggers for Platelet Transfusion

Platelet count	clinical condition
Normal platelet count	Platelet dysfunction and clinical bleeding Major bleeding Surgery
Platelet count < 100	DIC and bleeding Sepsis with rapid deterioration Minor bleeding
Platelet count < 50	Exchange transfusion Preterm
Platelet count < 30	Asymptomatic term infant

Ensure that the Platelets are

- ✓ Leukocyte-depleted
- ✓ CMV-negative (if unknown, filtering may be required before transfusion)
- ✓ Where possible, ABO compatible plasma
- ✓ Irradiated if infant is immune-compromised.

MATERIALS AND METHODS

Study design : A Prospective observational study.
Study site : SNCU,
Department of Pediatrics,
Government General Hospital, Kurnool.
Study duration : The study will be performed for 6 months.
Sample size : 150 babies.
Inclusion criteria: The birth asphyxic newborn's of either sex with a full-term gestation period and thrombocytopenic newborns along with birth asphyxia.

Exclusion criteria

- Preterm babies
- Congenital abnormalities
- Any neonates with term gestation with no history of delayed cry at birth.

Sampling

The clinical profile of 150 term newborns admitted with birth asphyxia and thrombocytopenia with birth asphyxia was selected based on inclusion and exclusion criteria. In the present study, the newborns admitted in SNCU with BIRTH ASPHYXIA [HIE - I, II, III] are selected. All the newborns with Birth Asphyxia and thrombocytopenia with birth asphyxia were systematically evaluated by Pediatrician.

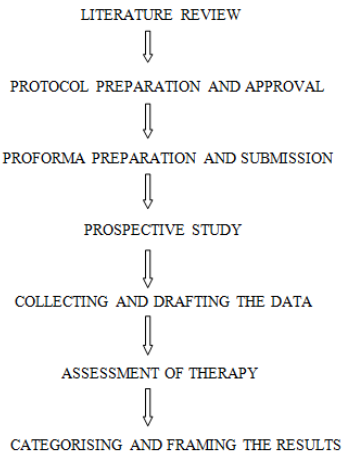
Newborns with Eligibility Criteria

- ✓ The present observational prospective study involves the newborns from the pediatric department who are diagnosed with Thrombocytopenia along Birth asphyxia, hypoxic-ischemic encephalopathy {BA-HIE }.
- ✓ Only the full term gestational age babies were selected for the study.

The subjects were selected based on the exclusion and inclusion criteria

Method of study

The method of study involves a sequence as,



RESULTS

In the first step, literature review was done on various studies of birth asphyxia and thrombocytopenia with birth asphyxia. The second step involves the preparation and approval of the protocol. The protocol was approved by the Institutional Review Board with IRB number - KVSP/IRB/2018-2019/Pharm.D/PROJ/04.

The next step was preparation and submission of proforma for the study.

Next prospective study was carried out by consulting the PG's, House surgeons, and other health care professionals.

MATERIALS

The data was collected including all baseline parameters of the newborns which are as following

- Demographic details
- Indication for Admission
- Provisional Diagnosis
- Mother's Information
- Baby's Information
- General Examination of the newborn
- Systemic examination
- Specific treatment and management methods
- Investigations
- Final outcome

RESULTS

Table 1 Incidence of Birth Asphyxia and Thrombocytopenia with Birth Asphyxia (n=150)

Incidence	Frequency	Percentage (%)
Birth asphyxia with thrombocytopenia	90	60
Birth asphyxia without thrombocytopenia	60	40
Total	150	100

The study comprises 150 clinical profiles of newborns of which 90 cases are thrombocytopenia along with birth asphyxia and the remaining 60 cases are birth asphyxia alone

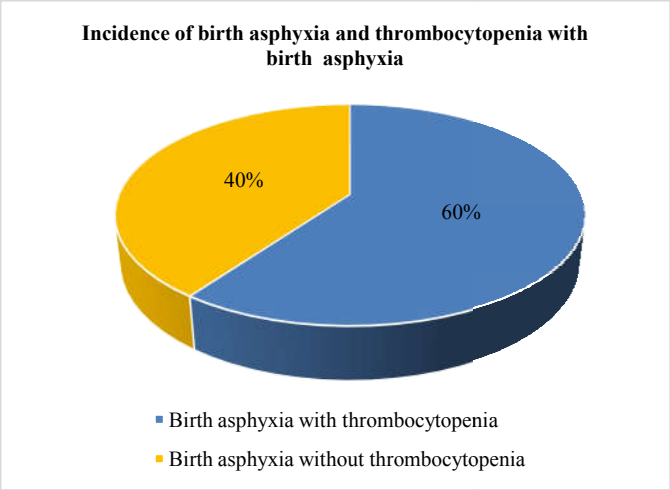


Figure 1 Incidence of Birth Asphyxia and Thrombocytopenia With Birth Asphyxia

Table 2 gender distribution (n=150)

Gender	Frequency	Percentage (%)
Male	91	60.7
Female	59	39.3

A total of 150 newborn cases were collected. In these 91 (60.7%) babies are male, remaining 59 (39.3%) babies are female. In our study the majority of newborns are male babies.

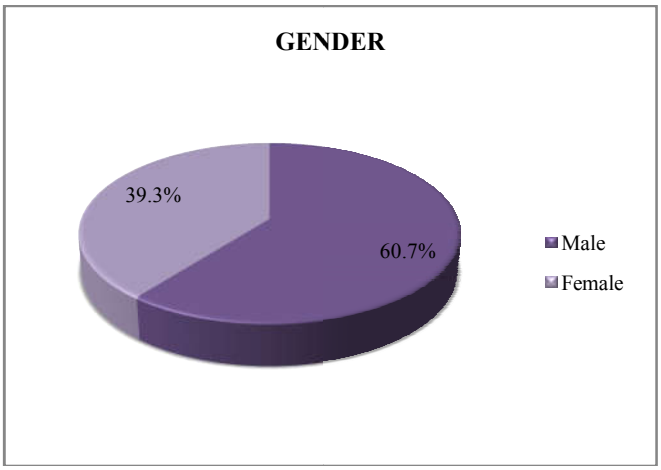


Figure 2 Gender Distribution

Table 3 Gender Distribution in Thrombocytopenia with Birthasphyxia (n=90)

Gender	Frequency	Percentage
Male	55	61.1
Female	35	38.9

In the gender distribution of thrombocytopenia, the total number of cases were 90. Among those 55 (61.1%) newborns are male and the remaining 35 (38.9%) newborns are female. The male thrombocytopenia babies are more among the evaluated case profiles.

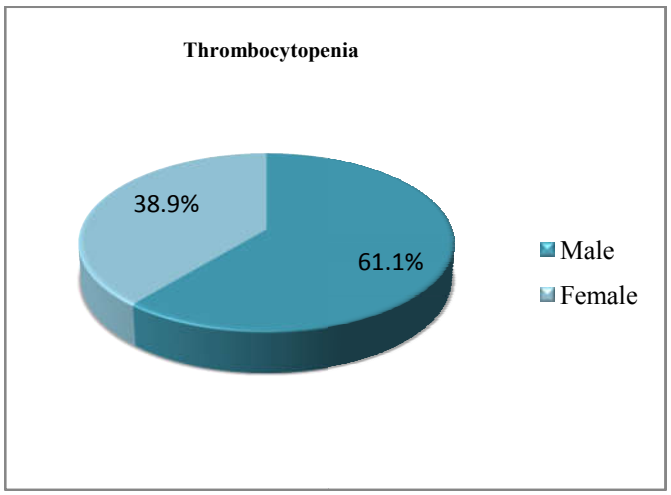


Figure 3 Gender Distribution in Thrombocytopenia with Birth Asphyxia

Table 4 stages of birth asphyxia (n= 150)

Birth asphyxia	STAGE-I	STAGE-II	Stage-III
Frequency	46	90	14
Percentage	30.7	60	9.3

Among the 150 newborns profiles evaluated, 46 (30.7%) newborns were diagnosed as stage- I birth asphyxia, 90 (60%) newborns were diagnosed as stage- II birth asphyxia and 14 (9.3%) newborns were diagnosed as stage III birth asphyxia.

So, it was observed that majority of babies are with stage-I birth asphyxia and stage-III was seen in only 14 babies i.e. 9.3% of total incidence.

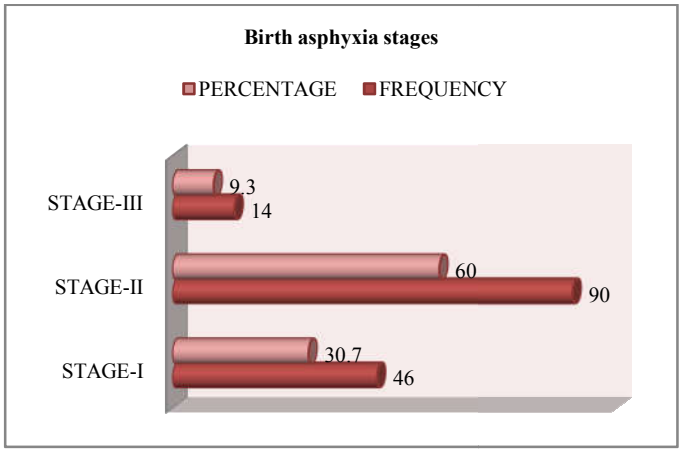


Figure 4 Stages of Birth Asphyxia

Table 5 Thrombocytopenia with birth asphyxia (n=90)

Birth asphyxia	Stage-I	Stage-II	Stage-III
Thrombocytopenia	21	55	14
Percentage	23.3	61.1	15.6

In our study the incidence of thrombocytopenia among three stages of birth asphyxia is as above. In stage-I birth asphyxia 21 (23.3%) babies are with thrombocytopenia , in stage-II birth asphyxia 55 (61.1%) babies are with thrombocytopenia and in stage-III birth asphyxia 14 (15.6%) babies are with thrombocytopenia.

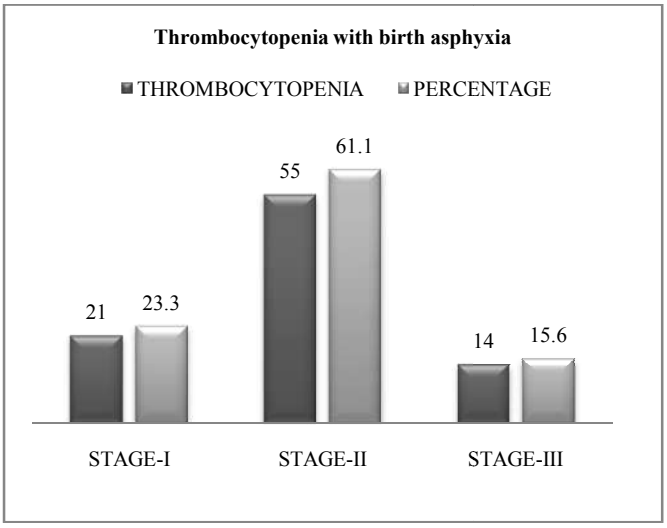


Figure 5 Thrombocytopenia with Birth Asphyxia

Table 6 Categories of Thrombocytopenia with Birth Asphyxia (N=90)

Thrombocytopenia	Mild	Moderate	Severe
Frequency	33	40	17
Percentage	36.7	44.4	18.9

The frequency of mild, moderate and severe thrombocytopenia with birth asphyxia was analysed. Among those 33(36.7%) babies had mild thrombocytopenia. 40 (44.4%) babies had moderate thrombocytopenia. 17 (18.9%) babies had severe thrombocytopenia. So, the incidence of moderate thrombocytopenia is more among the evaluated.

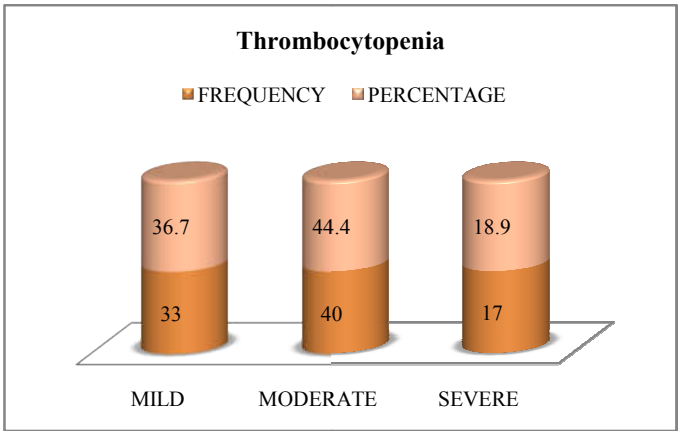


Figure 6 Categories of Thrombocytopenia With Birth Asphyxia.

Table 7 Mild Thrombocytopenia with Birth Asphyxia

Platelet count	No.of cases	Discharged	Expired
1,00,000-1,10,000	5	4	1
1,10,000-1,20,000	6	5	1
1,20,000-1,30,000	5	5	0
1,30,000-1S,40,000	7	7	0
1,40,000-1,50,000	10	10	0
Total	33	31	2
Percentage		93.93	6.06

The outcome of mild thrombocytopenia cases based on platelet count were evaluated. Among them 31 cases were discharged with standard deviation of 2.38,mean of 6.2 while 2 deaths with standard deviation of 0.54, mean of 0.4 were recorded.

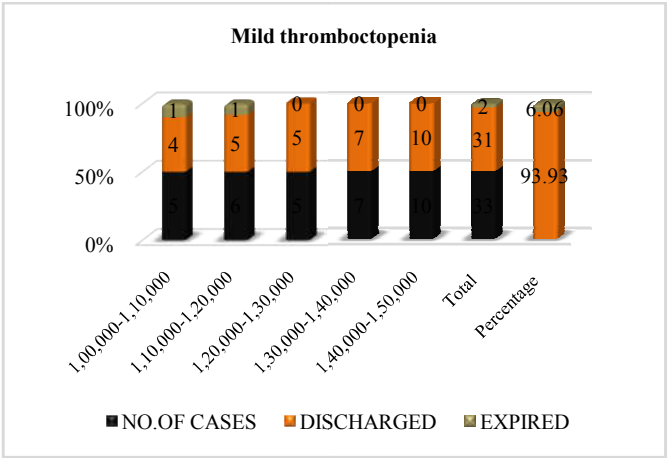


Fig no 7 mild thrombocytopenia

Table 8 Moderate Thrombocytopenia with Birth Asphyxia

Platelet count	No.of cases	Discharged	Expired
50,000-60,000	3	3	0
60,000-70,000	6	5	1
70,000-80,000	11	8	3
80,000-90,000	10	7	3
90,000-1,00,000	10	7	3
Total	40	30	10
Percentage		75	25

The outcome of moderate thrombocytopenia along with birth asphyxia based on platelet count were evaluated. Among them 30 cases were discharged with standard deviation of 2.0, mean of 6.0 while 10 deaths with standard deviation of 1.41,mean of 2.0 were recorded.

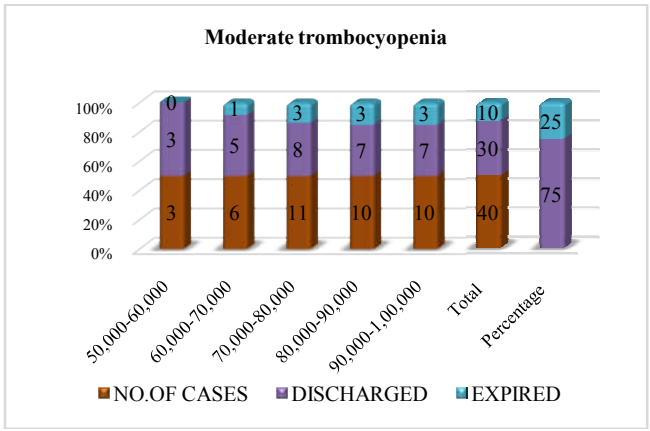


Fig no 8 moderate thrombocytopenia

Table 9 Severe Thrombocytopenia with Birth Asphyxia

Platelet count	No.of cases	Discharged	Expired
10,000-20,000	8	6	2
20,000-30,000	2	1	1
30,000-40,000	5	3	2
40,000-50,000	2	1	1
Total	17	11	6
Percentage		64.7	35.2

The outcome of thrombocytopenia along with birth asphyxia cases based on the platelet count were evaluated. Among them 11 cases were discharged with standard deviation of 2.36, mean of 2.75, while 6 deaths with standard deviation of 0.57, mean of 1.5 were recorded.

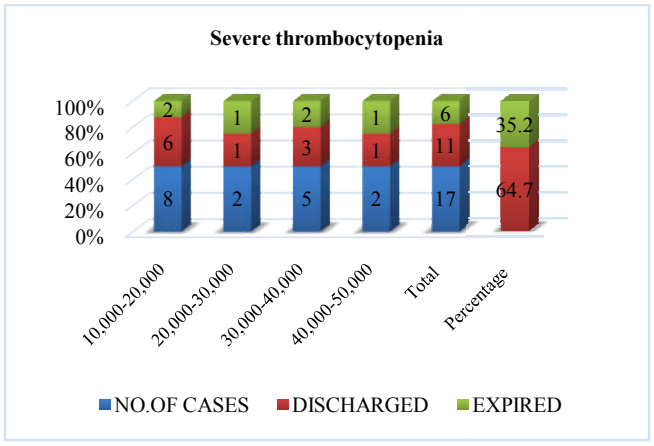


Fig no 9 severe thromboctopenia

Table 10 Incidence of Bleeding Manifestations in Newborns (n=42)

Type of bleeding	No. of cases	Percentage (%)
Skin bleeds	8	19.1
Umbilical cord bleeds	9	21.4
Mucosal bleeds	25	59.5
Total	42	100

In our study, total of 90 thrombocytopenia cases 42 babies presented with bleeding manifestations. Among them 8 (19.1) babies were presented with skin bleeds, 9 (21.4) babies were presented with umbilical cord bleeds and 25 (59.5) babies were presented with mucosal bleeds.

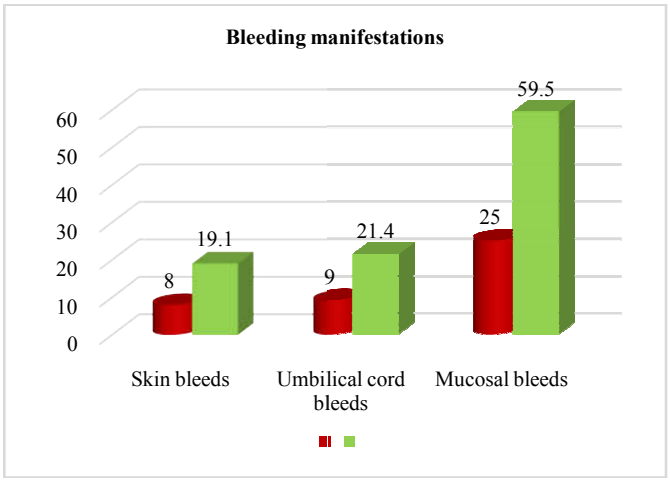


Figure 10 Incidence of Bleeding Manifestations In Newborns

Table 11 Incidence of Bleeding Manifestations In Different Categories of Thrombocytopenia **(n=42)**

Type of bleeding	Mild thrombocytopenia	Moderate thrombocytopenia	Severe thrombocytopenia
Skin bleeds	2	6	0
Umbilical cord bleeds	1	3	5
Mucosal bleeds	0	6	19
Total	3	15	24

In each category of thrombocytopenia the bleeding manifestations was analysed as above.

In mild thrombocytopenia 2 babies were presented with skin bleeds and only 1 baby presented with umbilical bleeds. In moderate thrombocytopenia 6 babies were presented with skin bleeds, 3 babies were presented with umbilical cord bleeds and 6 babies were presented with mucosal bleeds. In severe thrombocytopenia 5 babies were presented with umbilical cord bleeds and 19 babies were presented with mucosal bleeds. So, it was observed that high number of bleeds were observed in severe thrombocytopenia among the evaluated case profiles.

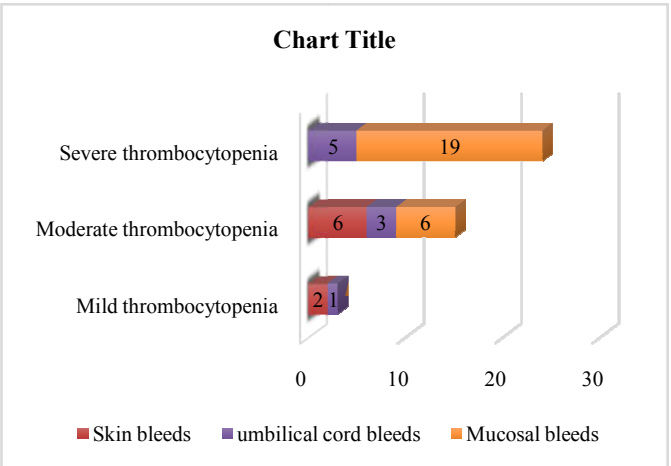


Figure 11 Incidence of Bleeding Manifestations in Different Categories of Thrombocytopenia

Table 12 Platelet and Packed Cell Transfusions in Severe Thrombocytopenia (n=17)

Transfusions	No. of cases	Percentage (%)
Platelet transfusion	4	23.5
Packed cell transfusions	5	29.4

Among the 17 babies with severe thrombocytopenia, 4 (23.5) babies have undergone platelet tansfusions and 5 babies have undergone packed cell transfusions.

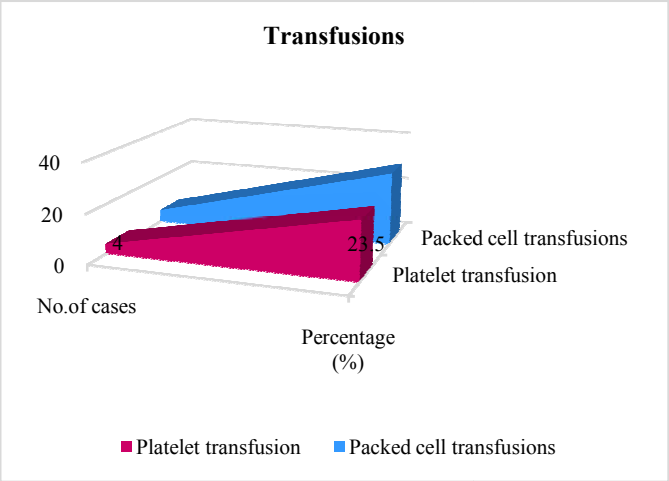


Figure 12 Platelet and Packed Cell Transfusions in Severe Thrombocytopenia

Table 13 Final Outcome of Thrombocytopenia with Birth Asphyxia (n=90)

Outcome	No. of Cases	Discharged	Percentage (%)	Expired	Percentage (%)
HIE Stage – I	21	21	23.3	0	0
HIE Stage – II	55	44	48.9	9	10
HIE Stage – III	14	3	3.3	11	12.2

The final outcome in three stages of birth asphyxia with thrombocytopenia was evaluated. Among them 21 (23.3%) babies were discharged in stage-I with no mortality. In stage-II, 44 (48.9%) babies were discharged and 9 (10%) babies expired. In stage-III, 3 (3.3%) babies were discharged and 11 (12.2%) babies expired. The mortality rate was high in third stage of HIE. The standard deviation was found to be 23.9 and the mean is 22.6.

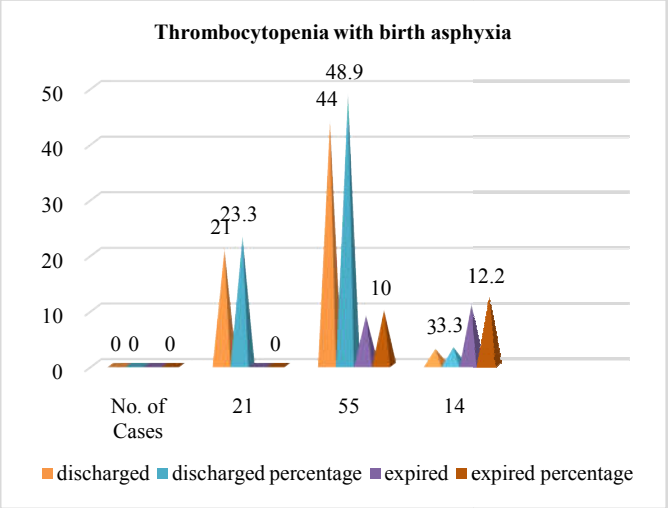


Figure 13 Final Outcome of Thrombocytopenia With Birth Asphyxia

DISCUSSION

The observational study of Birth Asphyxia contributes to the significance of morbidity and mortality. This study recorded an incidence of Birth asphyxia thrombocytopenia 60% and birth asphyxia alone is 40% which is higher than the 21.1% reported by IIah *et al.*,[98] .However, our incidence rate for severe birth asphyxia was significantly lower (9.3%) than what was obtained by Okechukwu and Achonwa (12.7%)[100] in Abuja, and higher(9.3%) than 2.6% by Kinoti[101] in East, central and Southern Africa.

The study comprises 150 clinical profiles of newborns of which 90 cases are thrombocytopenia along with birth asphyxia and the remaining 60 cases are birth asphyxia without thrombocytopenia.

In our study about 150 term newborns with Birth asphyxia were included. Among the 150 term newborns, 91(60.7%) are males and 59(39.3%) are females. The occurrence of thrombocytopenia in babies with Birth asphyxia is more in males (61.1%) than compared to females (38.9%) with a prevalence total of 60%.

According to the HIE classification, birth asphyxia was classified into 3 stages, in our study their frequency range in each stage of birth asphyxia are Stage I – 46(30.7%), Stage II – 90(60%) and Stage III – 14(9.3%), and the frequency range in each stage of birth asphyxia with thrombocytopenia are Stage I – 21(23.3%), Stage II – 55(61.1%) and Stage III – 14(15.6%).

In the present study, thrombocytopenia is analyzed into 3 groups ie,. Mild, Moderate and Severe with a frequency range of mild-33(36.7%) moderate-40(44.4%) and severe-17(18.9%). It is found that the majority of the cases had moderate thrombocytopenia.

In our study, total of 90 thrombocytopenia cases 42 babies presented with bleeding manifestations. Among them 8 (19.1%) babies were presented with skin bleeds, 9 (21.4%) babies were presented with umbilical cord bleeds and 25 (59.5%) babies were presented with mucosal bleeds. We observed that majority of mucosal bleeds were observed in severe thrombocytopenia babies.

In the present study, total of 42 babies presented with bleeding manifestations 2 babies were presented with skin bleeds and only 1 baby presented with umbilical bleeds in mild thrombocytopenia, 6 babies were presented with skin bleeds, 3 babies were presented with umbilical cord bleeds and 6 babies were presented with mucosal bleeds in moderate thrombocytopenia. Also 5 babies were presented with umbilical cord bleeds and 19 babies were presented with mucosal bleeds in Severe thrombocytopenia. We observed that majority of bleeds were observed in severe thrombocytopenia among the evaluated case profiles.

In our study, among 17 babies with severe thrombocytopenia 9 babies had undergone transfusions in which 4 (23.5%) babies had undergone platelet transfusions and 5 (29.4%) babies had undergone packed cell transfusions

In the present study the final outcome of thrombocytopenia in Birth asphyxia stages was determined where 21 (23.3%) babies were discharged and none of the babies were expired in stage-I, 44 (48.9%) babies were discharged and 9 (10%) babies expired out of 55 babies in stage-II, 3 (3.3%) babies were discharged and 11 (12.2%) babies expired out of 14 babies in stage – III admitted in SNCU.

In this study, the platelet count ranges from the three categories as we noticed that the outcome of severe

thrombocytopenia where 11(64.7%) babies discharged and 6(35.2%) babies expired. Whereas the outcome of moderate thrombocytopenia is 30(75%) babies discharged and 10(25%) babies expired. As well as the outcome of the mild thrombocytopenia is recorded as 31(93.93%) of babies discharged and 2(6.06%) babies expired.

It was noticed that the newborns with the complication of bleeding were taken the utmost care with complete observation and management. However, the lower mortality is attributable to the lower incidence of Stage – III HIE cases, but still severe thrombocytopenia contributed to significant mortality rate among babies with stage – III Birth asphyxia and hence can be concluded as a poor prognostic indicator.

CONCLUSION

Birth asphyxia still remains a major concern among neonatal morbidities. The reported mortality rate is about 18% in full-term birth asphyxiated infants. Around 18% of death rate more number of mortality was seen in stage III birth asphyxia. Low mortality rate was observed in stage-II birth asphyxia. Good outcome was reported in stage-I birth asphyxia with no mortality.

Birth asphyxia is associated with early onset of neonatal thrombocytopenia, So Thrombocytopenia is a common clinical condition in newborns. In this study we found that in Birth asphyxia, about 40% of newborns developed thrombocytopenia. The severity of thrombocytopenia in SNCU are mild, moderate and severe. Most episodes of neonatal thrombocytopenia are mild and moderate which resolve spontaneously without clinical sequelae. Rare episodes of neonatal thrombocytopenia are severe, few incidences were resolved by platelet transfusion. Major mortality rate was observed in newborns with severe thrombocytopenia along with birth asphyxia.

Moreover, the platelet count in thrombocytopenic cases shows that there is no significant difference among the three categories of thrombocytopenia along with birth asphyxia, which is determined by the p-value, that is greater than 0.005, hence the platelet count is not statistically significant among the thrombocytopenia with birth asphyxia cases.

Newborns with birth asphyxia have impaired platelet production, so the Birth asphyxia is a major risk factor for thrombocytopenic newborns. So, well care should be taken for the newborns with birth asphyxia.

Neonatal thrombocytopenia is a treatable and reversible condition. Hence it is important to identify newborns at risk, initiate appropriate therapy to prevent severe bleeding and potentially significant morbidity & mortality. Even though providing the desirable management to the newborns with birth asphyxia and thrombocytopenia, mortality was observed in our study. We conclude that more management methods should be developed innovatively. Harmonised guidelines are necessary to standardise the treatment and support clinical management decisions.

Acknowledgement

We wish to express our gratitude to a number of people who made important contributions to the accomplishment of this thesis.

First and foremost, we gratefully acknowledge Mr. R. Jona Methusala, M. Pharm., Associate Professor, Department of Pharmacology, for his guidance, benevolence, valuable admonition and continuous help throughout the completion of this thesis work.

It is a pleasure to convey our gratitude to Dr. K. Aruna Jyothi, MD., Associate Professor, Department of Paediatrics, KMC-Government Hospital for her supervision, admonition and guidance from the commencement of our project as well as her forby support, encouragement throughout our thesis.

It's pleasure to pay hommage to our principal Dr.G. Nagarajan, M. Pharm.,Ph. D., for providing this great opportunity to do our project work and also helped us in all possible way for completion of our thesis work.

It is our privilege and a wonderful experience to be a part of this Institution and we would like to express our gratitude to the management including Honourable Chairman Dr. K. V. Subba Reddy, Correspondent K. Vijaya Lakshmi Dr. KVSR College of Pharmacy for providing us all the facilities & support.

We thank all the P.G's, House surgeons and all Nursing staff for allowing us to collect the data from the case sheets in the ward.

It is our privilege to be thankful to Mrs. Sumalatha Lecturer in Statistics for helping us in the statistical studies.We also express our special thanks to our classmates for their support and timely help.

References

1. Barkovich AJ, Truwit CL. Brain damage from perinatal asphyxia: *American Journal of Neuroradiology*. 1990 Nov
2. Robert Christensen. Severe thrombocytopenia in neonates. Neonatal thrombocytopenia 2009
3. Spector JM, Daga S. Preventing those so-called stillbirths. Bulletin of the World Health Organization. 2008.
4. National Center for Health Statistics
5. Graham EM, Ruis KA, Hartman AL, Northington FJ, Fox HE. A systematic review of the role of intrapartum hypoxia-ischemia in the causation of neonatal encephalopathy. *American journal of obstetrics and gynecology*. 2008 .
6. Nelson KB, Chang T, Ghadini A, Aloja E, Muller M, Paribello F, Demontis R, Faa A,Locatelli A, Incerti M, Ghidini A. A systematic review of the role of intrapartumhypoxia-ischemia in the causation of neonatal encephalopathy. The Journal of Maternal-Fetal & Neonatal Medicine. 2011 May 1.
7. Vannucci RC, Perlman JM. Interventions for perinatal hypoxic–ischemicencephalopathy. Pediatrics. 1997 Dec 1.
8. Moster D, Lie RT, Markestad T. Joint association of Apgar scores and early neonatalsymptoms with minor disabilities at school age. Archives of Disease in Childhood-Fetaland Neonatal Edition. 2002 Jan 1.Nelson K, Ellenberg J. Apgar scores as predictors of chronic neurologic disability. Pediatrics. 1981.

9. Nelson KB, Leviton A. How much of neonatal encephalopathy is due to birth asphyxia. American journal of diseases of children. 1991 Nov 1. Badawi N, Kurinczuk JJ, Keogh JM, Alessandri LM, O'Sullivan F, Burton PR, Pemberton PJ,Stanley FJ. Intrapartum risk factors for newborn encephalopathy: the western Australiancase-control study. BMJ. 1998.
10. Robertson CM, Perlman M. Follow-up of the term infant after hypoxic-ischemic encephalopathy. Paediatrics & child health. 2006 May 1 .Ferriero DM.Neonatal brain injury. N Engl J Med. 2004 1985-95.
11. Sarnat HB, Sarnat MS. Neonatal encephalopathy following fetal distress: a clinical and electroencephalographic study. Archives of neurology. 1976 Oct 1. Rees S, Inder T. Fetal and neonatal origins of altered brain development. Early Hum Dev. 2005.
12. Kurinczuk JJ, White-Koning M, Badawi N. Epidemiology of neonatal encephalopathy and hypoxic–ischaemic encephalopathy. Early human development. 2010 Jun.
13. Lorek A, Takei Y, Cady EB, Wyatt JS, Penrice J, Edwards AD, Peebles D, WylezinskaM, Owen-Reece H, Kirkbride V, Cooper CE. Delayed ("secondary") cerebral energyfailure after acute hypoxia-ischemia in the newborn piglet: continuous 48-hour studies by phosphorus magnetic resonance spectroscopy. Pediatric research. 1994 Dec.
14. Johnston MV, Trescher WH, Ishida A, Nakajima W. Neurobiology of hypoxic-ischemicinjury in the developing brain. Pediatric research. 2001 Jun.
15. Siesjö BK, Bengtsson F. Calcium fluxes, calcium antagonists, and calcium-related pathology in brain ischemia, hypoglycemia, and spreading depression: a unifying hypothesis. *Journal of Cerebral Blood Flow &amp*; Metabolism. 1989 Apr.
16. Lapointe A, Barrington KJ. Pulmonary hypertension and the asphyxiated newborn. *The Journal of pediatrics*. 2011 Feb.

How to cite this article:

ReddypoguJonaMethusala *et al* (2019) 'A Prospective Observational Study on Incidence and Management of Thrombocytopenia in Term Newborns with Birth Asphyxia Admitted in Sncu, Ggh, Kurnool', *International Journal of Current Advanced Research,* 08(12), pp. 20810-20818. DOI: http://dx.doi.org/10.24327/ijcar.2019. 20818.4075

****