The Medical Journal of Tikrit University (2025) 31 (2): 228-238
DOI: http://dx.doi.org/10.25130/mjotu.31.2.24

IRAQI Academic Scientific Journals

ISSN: 1813-1638

The Medical Journal of Tikrit University

Available online at:www.mjotu.com

MJTU

The Medical Journal of Tikrit University

Frequency of Birth Asphyxia among Full-Term Infants in the Neonatal Care Unit at Tikrit Teaching Hospital

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***Keywords*:** *Asphyxia, Neonatal, Newborn, Infants.*

ARTICLE INFO

Article history:

Received 01 Jul 2025
Accepted 01 Sep 2025
Available online 31 Dec 2025

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http://tikrit-medicine.tripod.com/ id10.html

Citation:

ABSTRACT

Background: Birth asphyxia is the most important preventable cause of cerebral injury in the neonatal period, leading to very high neonatal mortality and morbidity in developing countries.

Aim of study: The aim of the current study is to assess the frequency of neonates with birth asphyxia admitted to the neonatal care unit at Tikrit Teaching Hospital.

Patients and methods: This observational cross-sectional study involved the study of 320 newborn infants (165 males and 155 females) done in the period between the first of May 2021 to the end of July 2021. Total of 2240 live birth infants were born in Tikrit Teaching Hospital, 320 births were attended by a convenient sampling and 54 newborn infants had Apgar scores < 7 at five minutes and with no obvious congenital abnormalities were diagnosed to have asphyxia.

Results: The frequency of birth asphyxia was (16.9%), various stages of hypoxic-ischemic encephalopathy occurred in 21/54 infants (38.9%) of asphyxiated infants, and the incidence was higher in the low Apgar score group. There was a statistically significant relationship between birth asphyxia, and birth weight, mode of delivery, parity of mothers, antenatal care, and other maternal risk factors as pregnancy-induced hypertension, Oligohydramnios, and breech presentation. There were 12/54 infants (22.2%) mortality during the hospital stay, correlated with the severity of hypoxic-ischemic encephalopathy.

Conclusion: It is concluded that some causes of birth asphyxia are currently unavoidable. It is, however, widely accepted that some cases of birth asphyxia may be prevented by the delivery of high-risk pregnancies in obstetric facilities with appropriate intervention and by good neonatal resuscitation.

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INTRODUCTION

Seven million perinatal deaths occur annually, mostly in developing countries. According to the World Health Organization (WHO), four to nine million cases of newborn asphyxia occur each year. In spite of major advances in monitoring technology, obstetric care, and knowledge of fetal and neonatal pathologies, asphyxia remains a serious condition causing significant mortality and long-term morbidity. More than a million newborns that survive asphyxia at birth develop long-term problems such as cerebral palsy, mental retardation, speaking, hearing, visual and learning[1]. disabilities

The term "perinatal asphyxia", "asphyxia neonatorum" and "hypoxic–ischemic encephalopathy" are often used synonymously, more precisely, perinatal asphyxia refers to the disturbed exchange of O_2 and CO_2 while HIE refers to the deprivation of O_2 to the brain by the combined effect of hypoxemia (decrease oxygenation of blood) and ischemia (decreased blood flow)[2].

MATERIALS AND METHODS

The study has been conducted in the Neonatal Care Unit (NCU) at Tikrit Teaching Hospital in where all cases admitted were delivered inside a hospital, over a 3-month period.

The current work represented an observational cross-sectional study, which was conducted during the period extending from the first of May 2021 to the end of July 2021, with regular working hours. A convenient sampling technique which has been used to collect samples from the operative and labour theatre, with regular morning working hours of 10:00a.m to 02.00p.m and regular evening working hours of 07:00p.m to 11:00 p.m., with 5 days per week. During the study period, there were 2240 live births, 320 of which were attended, representing more than 10% of the total number of live births.

Demographics, duration of study as well and inclusion criteria played an important role in determining the size of study population. The total sample size chosen is suspected to be suitable to this study for showing the expected degree of difference regarding different variables. The questionnaire was developed to collect all data relevant to the aim of study. Data collection would thus comprise of two aspects, First aspect is description of the risk factors associated with asphyxia and the second aspect is birth asphyxia assessment and find out clinical presentation of birth asphyxia. The study includes two components: interviewer administration of questionnaire & birth weight. Prior to the interview, the purpose of data collection was explained & consent was obtained. The mothers were interviewed before or after labour or operation according to urgency of each case. Labour ward records and antenatal investigations were reviewed.

Live born full term babies (Gestational age ≥ 37 completed weeks) (i.e. ≥259 days) were included [3]. Birth asphyxia is considered on the basis of the following criteria:

1. Death in the first 24 hours of life in the absence of obvious congenital abnormality.
2. The major criteria for diagnosis of birth asphyxia in this study was having an

Apgar score of < 7 at 5 minutes with one or more of the following criteria[4]:

- Signs of fetal distress (heart rate of less than 100 beats per minute, late decelerations, or an absence of heart rate variability).
- Presence of Meconium stained liquor.
- Abnormal neurological manifestations within 48 hours of birth with hyperirritability, alteration in muscle tone or conscious level, convulsions or abnormal primitive reflexes.
- Need for assisted ventilation by face mask and oxygen or endotracheal intubation.

RESULTS

A total of (320) live birth infants were attended and assessed during the study period (91) infants (28.4%) had delayed initiation of breathing and Apgar score <7 at first minute and need resuscitation. At five minute(43) infants (11.5%) Apgar score become >7 with no criteria of asphyxia and (54) infants (16.9%) Apgar score remain <7 with clinical signs of birth asphyxia and admitted in NCU at Tikrit teaching hospital as shown in figures (1, 2). The characteristics of the sample size according to gender were (165) male (51.6%) and (155) female (48.4%). According to residence of mothers (149) mothers were living in urban areas (46.6%) and (171) mothers were living in rural areas (53.4%) as shown in table (1). Regarding gender of newborn infants birth asphyxia was found to be more in males than females. Out of the newborn infants with birth asphyxia (55.6%) were males and (44.4%) were females. There was no statistical significant difference regarding gender as shown in table (2). Regarding the residency of mothers of the asphyxiated newborn infants (44.4%) mothers resided in urban areas and (55.6%) mothers resided in rural areas. There was no statistical significant difference regarding residency as shown in table (3). As regard to birth weight of the full term asphyxiated newborn infants enrolled in the study (43) newborn infants had normal birth weight and (11) newborn infants had low birth weight representing (20.4%) of asphyxiated infants. There was statistical significant difference regarding birth weight and birth asphyxia as shown in table (4). Newborn infants were delivered by C/S representing high percentage about (70.4%) of the asphyxiated infants (by elective C/S and emergency C/S equally). Whereas in nonasphyxiated newborn infants (44.4%) newborn infants were delivered by C/S (28.6% by elective C/S and 15.8% by emergency C/S) ,newborn infants were delivered by normal vaginal delivery 16(29.6%) of the asphyxiated infants . There was stagtistical significant difference regarding method of delivery by C/S as shown in table (5) .As regard to maternal age, the majority of mothers of asphyxiated neonates were between 20-35 years about (42) representing (77.8%). There was statistical significant difference regarding maternal age as shown in table (4.8). Regarding to maternal risk factors, most cases of birth asphyxia (75.9%) associated with one or more risk factors whereas (40.6%) of cases of no asphyxia associated with risk factors as shown in figure (3). Among associated maternal risk factors obstructed labour, pregnancy induced hypertension, meconium stained liquor and history of birth asphyxia among previous babies were seen in high percentages about (24.1%), (20.4%), (18.5%) and (14.8%) respectively. There was statistical significant difference

regarding pregnancy induced hypertension, Oligohydramnios and breech presentation. Maternal smoking was not recorded among the studied sample as shown in table (4.9). In comparison between Apgar scoring at first minute and five minute. Asphyxiated infants at first minute were (91) infants whereas at five minute (54) infants were asphyxiated. At first minute (28) Infants (30.8%) had Apgar score < 3 and at five minute (3) infants (5.6%). Also at first minute (35) infants (38.4%) had Apgar score 3-5 and at five minute (12) infants (22.2%) had Apgar score 3-5. There was statistical significant difference regarding Apgar scoring as shown in table (4.10). In comparison between Apgar scoring at first minute and five minute. Asphyxiated infants at first minute were (91) infants whereas at five minute (54) infants were asphyxiated. At first minute (28) Infants (30.8%) had Apgar score < 3 and at five minute (3) infants (5.6%). Also at first minute (35) infants (38.4%) had Apgar score 3-5 and at five minute (12) infants (22.2%) had Apgar score 3-5. There was statistical significant difference regarding Apgar scoring as shown in table (4.10). Among asphyxiated newborn infants, 7(2.2%) newborn infants had no cry, 26(8.1%) had delayed cry, grunting was found in 45(14.1%), cyanosis was found in 15(4.7%), respiratory distress was present in 42(13.1%), lethargy was found in 44(13.8%), whereas irritability was found in 4(1.3%), decreased tone was present in 21(6.6%), whereas increased tone was present in 3(0.9%), Meconium staining was noticed in 8(2.5%), convulsions were noticed in 6(1.9%), whereas all of them had difficulty in feeding and need assisted ventilation by facemask and O_2 as shown in table (4.11). According to Sarnat and Sarnat classification, hypoxic-ischemic encephalopathy staging among asphyxiated newborn infants; 33 infants (61.1%) had no encephalopathy and 21infants (38.9%) had HIE. Among them 4 infants (7.4%) were in stage -1, 9 infants (16.7%) were in stage - 2 and 8(14.8%) were in stage - 3 as shown in figure (4).

Among asphyxiated newborn infants (24.1%) had normal blood urea levels between (1.1-4.3)mmol/l and (61.1%) had normal serum creatinine levels between (27-88) mmol/l. whereas (24.1%) had high blood urea levels >6.7 mmol/l and (9.3%) had high serum creatinine levels >100 mmol/l. as shown in tables (4.13, 4.14) .

Discussion: Apparently because of varying definitions the true incidence of asphyxia is unknown. The incidence of birth asphyxia depends largely on the definition used to diagnose the condition as well as the gestational age of the infant [4-7]. In fact, the real incidence of birth asphyxia in our population may be higher than the obtained figure because the present study included only hospital deliveries in Tikrit city. However, the relatively high figure can be explained by the fact that (61.8%) of mothers involved in this study do not have regular antenatal care, and there was a delay in seeking hospital care, as many pregnant mothers attend to hospital late, and signs of fetal distress were already present. In addition to the unavailability of facilities important for early detection of fetal distress, like blood sampling for pH estimation, and inadequate health services like Doppler and echocardiography. Gestational age as a risk factor will not be discussed, as only infants with a gestation of≥ 37 weeks were included in the study.

The incidence of birth asphyxia increases with the decrease in gestational age as

premature infants are more vulnerable to cytokine-induced damage due to the immaturity of their blood-brain barrier, and the outcome is poorest [8,9]. Although birth asphyxia is higher in males than females, it was statistically not significant, which may be due to sample selection and duration of study. In a comparison study by Sitthivuddhi F. et al [10]. Studies by Hubacek JA. et al[11] and Futrakul S. et al [12], which mentioned that the male gender is highly vulnerable to threatening factors such as increasing the risk of sepsis, bronchial hyperesponsiveness, atopy, and mortality of RDS. In the current study, mothers of the asphyxiated infants resided in rural areas higher than mothers resided in urban areas without significant relationship between birth asphyxia and the residency of mothers which may be related to the size of the population, duration and location of study. In comparison with studies by Khreisnat and Habahbeh in Jordan [13] and Pattinson R. et al in South Africa [14] which indicate that mothers in rural areas often have no or infrequent antenatal care and reduced utilization of antenatal facilities may also be attributed to charges or service fees that have recently been introduced at various levels of care. In current study, there was significant relationship between birth asphyxia and birth weight and all infants with low birth weight had birth asphyxia which may be due to that most of them (9/11) infants (81.1%) had associated maternal risk factor of hypertensive disease of pregnancy with subsequent intrauterine growth restriction. These results are different from that obtained by Khreisat and Habahbeh [13] which showed no significant relationship between birth asphyxia and birth weight. In Nigerian study by Airede [15] also showed that an infant with low birth weight plays a significant role in occurrence of severe type of asphyxia most of them had associated maternal risk factors of either hypertensive disease of pregnancy or alcohol use during pregnancy with subsequent intrauterine growth restriction. In current study, asphyxiated infants delivered by C/S (Elective and Emergency C/S equally) more than those delivered vaginally. Tikrit teaching hospital is a referral centre receives the pool of cases from the surrounding urban and suburban province, so we expect that the cases received were of high risk group with complicated situation in addition, the effect of drugs of anesthesia which may increase the risk of birth asphyxia. This result is identical to study result that obtained by Bashir AI. et al [16] in Madina Al-Munawara in 2003 and study by Khreisat and Habahbeh [13] showed significantly higher proportion of asphyxiated cases were delivered by C/S and consider C/S itself is an important factor in producing asphyxia especially when associated with high risk indications like fetal distress, prolapsed cord, abruptio placentae, IUGR, maternal toxemia, maternal diabetes, prolonged rupture of membrane [17].

Other important maternal risk factors encountered more frequently among the asphyxiated infants like obstructed labour, pregnancy induced hypertension, meconium stained liquor and history of birth asphyxia among previous babies. There were significant association with pregnancy induced hypertension, Oligohydramnios and breech presentation. These findings are consistent with Indian study by Aga AS. et al [18] in which antepartum haernorrhage and pregnancy induced hypertension carried high risk for birth asphyxia. Other studies by Khatoon SA. et al [19] and Daga AS. et al [20] which

showed that important maternal risk factors encountered more frequently among the asphyxiated babies were prolonged 2nd stage of labour, PROM, toxaemia of pregnancy, hypertension and APH.

In current study, Infants had low Apgar scores at first minute higher than those had low Apgar scores at five minute and this is related to the early intervention and resuscitation by suctioning and ventilation in form of bag and mask with chest compressions. Ellenberg JH. et al [21] showed that large group of term infants depressed immediately after birth who rapidly improve after resuscitation. Apgar score at birth is not much important but is much valuable in terms of outcome determination if done at 5 minutes [22]. Apgar score at five minutes was evaluated in current study;

Infants had Apgar score of < 3 are (3/54) (5.6%): The first case presented with thick meconium aspiration and severe prolongs prenatal asphyxia and apnoea, the second case presented obstructed labour and rupture uterus and the third case presented with poly hydramnios and history of asphyxia among previous babies and the cause may be subtle congenital anomaly that need autopsy to reveal it which is not done in Tikrit teaching hospital. Infants had Apgar score between 3-5 are (12) (22.2%): Of them 4 cases presented with pregnancy induced hypertension, diabetes mellitus and history of asphyxia among previous babies; 6 cases presented with obstructed labour, meconium and fetal distress and the last 2 cases one presented with PROM, maternal fever and fetal distress while the other one had no risk factor. The most common presentation of birth asphyxia was delayed cry, grunting, respiratory distress, lethargy, unable to feed and need for assisted ventilation. Most common complication of birth asphyxia is hypoxic-ischemic encephalopathy (HIE), and this aspect was also evaluated in this study. Most of infants that developed HIE presented with in HIE stage-2 or stage-3. It is different from the study done by Muhammad A. et al [23] in Pakistan where most infants present within HIE stage-1 or stage-2 and study done in Nigeria where half of the infants present within HIE stage-3[24].

The rate of HIE among asphyxiated infants was (28.9%) which is comparable with study by Shrestha M. et al [25] and other studies in which they have also found the incidence of HIE to be around 30% in infants born with low Apgar score[10, 26].

The low glomerular filtration rate of newborn kidneys limits postnatal renal function adaptation to endogenous and exogenous stresses [27]. As kidneys are very sensitive to oxygen deprivation, renal insufficiency may occur within 24 hours of a hypoxic ischemic episode, which if prolonged, may even lead to irreversible cortical necrosis. However renal injury in birth asphyxia is a potential consequence of adaptive mechanism. Among the recognized complications *i.e.*, acute tubular necrosis, renal vein thrombosis and acute renal failure, ARF is the commonest and carries a poor prognosis and may even result in permanent renal damage in up to 40% of survivors [28].

In current study, blood urea and serum creatinine levels had been done in 45 of asphyxiated infants after 24 hours and 9 missing cases because death or discharge occur within less than 24 hours. It was observed that blood urea and serum creatinine levels were increased in (59.3%)

and (22.3%) of cases respectively after 24 hours. Jayashree et al [29] from India showed (43.3%) of cases developed acute renal impairment. Jones et al study founded (62.5%) of cases developed acute tubular necrosis as a result of perinatal asphyxia, hypoxemia and shock [30].

Obstruction of tubular lumen and back leak mechanism contributed to increase in urea and creatinine levels in asphyxiated neonates with renal damage [31]

CONCLUSION

The incidence of birth asphyxia 16.9% was relatively high due to absence of regular antenatal care in high proportion of pregnant mothers. Low birth weight infants, multiparity and delivery by caesarean section were associated with higher incidence of asphyxia.Other associated risk factors were obstructed labour, pregnancy induced hypertension, meconium stained liquor and history of birth asphyxia among previous babies. `Among the serious complications HIE was commonest in term asphyxiated neonates and mortality was higher in neonates with HIE. This study concludes that 5 minutes Apgar score could be a good predictor for HIE.

REFERENCES

1. World Health Organization, Facts and figures on birth asphyxia2005. http://wwww.eho/int/entity/whr/2005/media_centre/facts_en.pdf>

2. Bax M, Nelson KB. Birth asphyxia: a statement. Dev Med Child Neutrol 1993; 35: 1015-24.

3. Parkin JM, Hey EN, JS C. Rapid assessment of gestational age at birth. Arch Dis Child 1976; p. 51: 259-263.

4. Day P, Lancaster P, Huang J. Perinatal statistic series. No. 6. Australia's mothers and babies 1995. Sydney: National Perinatal Statistics Unit (NPSU). Australian Institute of Health and Welfare; 1997.

5. Thornberg E, Thiringer K, Odeback A, Milsom I. Birth asphyxia: incidence, clinical course and outcome in a Swedish population. Acta Paediatr 1995; 84: 927-32.

6. Hall DR, Smith M, Smith J. Maternal factors contributing to asphyxia neonatorum. J Trop Pediatr 1996; 42: 192-5.

7. Chandra S, Ramji S, Thirupuram S. Perinatal asphyxia: multivariate analysis of risk factors in hospital births. Indian Pediatr 1997; 34: 206-12.

8. Bona E, Andersson AL, Blomgren K, Gilland E, Puka-Sundvall M, Gustafson K, et al. Chemokine and inflammatory cell response to hypoxia-ischemia in immature rats. Pediatr Res 1999 Apr; 45(4 Pt 1):500-9.

9. Fotopoulos S, Pavlou K, Skouteli H, Papassotiriou I, Lipsou N, Xanthou M. Early markers of brain damage in premature low-birth-weight neonates who suffered from perinatal asphyxia and/or infection. Biol Neonate 2001; 79(3-4):213-8.

10. Sitthivuddhi F, Pramot P, Pimorlat T; Risk Factors Hypoxic-Ischemic Encephalopathy in Asphyxiated Newborn Infants. J Med Assoc Thai 2006; 89(3): 322-8.

11. Hubacek JA, Stuber F, Frohlich D, Book M, Wategrove S, Ritter M, et al. Gene variants of the bactericidal/permeability increasing

protein and lipopolysaccharide binding protein in sepsis patients: gender-specific genetic predisposition to sepsis. Crit Care Med 2001; 29: 557-61.

12. Futrakul S, Deerojanawong J, Prapphal N. Risk factors of bronchial hyperesponsiveness in children with wheezing associated respiratory infection. Pediatr Pulmonolo 2005; 40: 81-7.

13. Khreisat, W.H. & Habahbeh, Z. Risk factors of birth asphyxia: A study at Prince Ali Ben Al-Hussein Hospital, Jordan. Pakistan Journal of Medical Science 2005; 21 (1): pp. 30 –32.

14. Pattinson, R., Woods, D., Greenfield, D. & Velahpi, S. Improving survival rates of newborn infants in South Africa. Reproductive Health 2005; 2(4): p. 1-13.

15. Airede AI. Birth asphyxia and hypoxic-ischemic encephalopathy: incidence and severity. Ann Trop Paediatr J 1991; 11: 331-5.

16. Bashir AI., Zakaria M., Anwar HK. Hypoxic-ischemic encephalopathy Incidence and risk factors in North Western Saudi Arabia. Saudi Medical Journal 2003; 24 (2): 147-153.

17. Casey BM, McIntire DD, Leveno KJ. The continuing value of the Apgar score for the assessment of newborn infants. N Engl J Med 2001; 344; 467-71.

18. Aga AS, Daga SR, Patole SK. Risk assessment in birth asphyxia. J Trop Paed 1990; 36(1): 34-9.

19. Khatoon SA, Kawser CA, Talukder M Q-K. Clinical spectrum and outcome of birth asphyxiated babies in neonatal unit of IPGMR: A study of 122 cases. BJCH 1989; 13: 7-15.

20. Daga AS, Daga ER, Patole SK. Risk Assessment of Birth Asphyxia. J Trop Pediatr 1990; 36: 34- 39.

21. Ellenberg JH, Nelson KB. Cluster of perinatal events identifying infants at high risk for death or disability. J Pediatr 1988; 113: 546-552.

22. Nelson KB, Ellenberg JH. Apgar scores as predictors of chronic neurologic disability. Pediatrics 1981; 68:36-44.

23. Muhammad A, Pervaiz AK, Fiaz AM. Birth Asphyxia; Risk Factors. The professional 2004 Oct-Dec; 11(4).

24. Simon N. Birth asphyxia. Emory Development Continuity Programme Atlanta, Georgia. 1998.

25. Shrestha M., Shrestha L., Shrestha PS. Profile of Asphyxiated Babies. J. Nepal Paediatr. Soc. 2008; 29(1).

26. Shah GS, Singh R, Das BK. Outcome of Newborns with Birth Asphyxia. J Nepal Med Assoc. 2005 Apr-Jun; 44(158):44-6.

27. Toth-Heyn P, Drukker A, Guignard JP. The stressed neonatal kidney: from Pathophysiology to clinical management of neonatal vasomotor nephropathy. Pediatr Nephol 2000; 14:227-239.

28. Singh M. Care of Newborn, 5th Ed. New Delhi: Sagar Publication; 1999: pp 113-114.

29. Jayashree G, Dutta AK, Sarna MS, Saili A. Acute renal failure in asphyxiated newborn. Indian Pediatrics 1991; 29:19-23.

30. Jones AS,James L,Blard H, Groshong T. Renal failure in newborn. Clin pediatr 1979, 5: 286-291.

31. Bailie MD. Renal function and disease. Clin Perinatol 1992; 19:91-92.

TABLES

Table 1: Distribution of the sample size according to sex & residence.

Sample	No.	%
Sex		
male	165	51.6%
female	155	48.4%
Total	320	100.0%
Residence		
Urban	149	46.6%
Rural	171	53.4%
Total	320	100.0%

Table 2: Relationship between birth asphyxia and gender.

Sex	Cases					
	Asphyxia		No asphyxia		Total	
	No.	%	No.	%	No.	%
Male	30	55.6	135	50.8	165	51.6
Female	24	44.4	131	49.2	155	48.4
Total	54	100	266	100	320	100

X^2 =0 .313, p value =0.576 > .0.05, df= 1 (not significant)

Table 3: Relationship between residence and birth asphyxia.

Residence	Cases					
	Asphyxia		No asphyxia		Total	
	No.	%	No.	%	No.	%
Urban	24	44.4	125	47.0	149	46.6
Rural	30	55.6	141	53.0	171	53.4
Total	54	100	266	100	320	100

X^2 =1.513, p value =0.219 > 0.05, df= 1 (not significant)

Table 4: Relationship between birth weight and birth asphyxia.

Birth weight	Cases					
	Asphyxia		No asphyxia		Total	
	No.	%	No.	%	No.	%
Normal birth weight	43	79.6	266	100	309	96.6
Low birth weight	11	20.4	0	0	11	3.4
Total	54	100	266	100	320	100

X^2 = 277.513, p value =0 .00 < 0.05, df= 1 (significant)

Table 5: Relationship between mode of delivery and birth asphyxia.

Mode of delivery	Cases					
	Asphyxia		No asphyxia		Total	
	No.	%	No.	%	No.	%
Normal vaginal delivery	16	29.6	148	55.6	164	51.3
Elective C/S	19	35.2	76	28.6	95	29.7
Emergency C/S	19	35.2	42	15.8	61	19.0
Total	54	100	266	100.0	320	100.0

X^2 = 51.644, p value =0.00 < 0.05, df= 2 (significant)

Table 6: Relationship between maternal age and birth asphyxia.

Maternal age	Cases					
	Asphyxia		No asphyxia		Total	
	No.	%	No.	%	No.	%
<20 yr.	8	14.8	21	7.9	29	9.1
20-35 yr.	42	77.8	210	78.9	252	78.7
>35 yr.	4	7.4	35	13.2	39	12.2
Total	54	100	266	100	320	100

$X^2 = 297.494$, p value = 0.00<0.05, df= 2 (significant)

Table 7: Risk factors related with birth asphyxia.

Other risk factors	Cases						P value
	Asphyxia		No asphyxia		Total		
	No.	%	No.	%	No.	%	
Ante partum hemorrhage	0	0	9	3.4	9	2.8	0
Pregnancy induced hypertension	11	20.4	31	11.7	42	13.1	0.002
Eclampsia	2	3.7	2	0.8	4	1.3	1.000
Poly hydramnios	3	5.6	5	1.9	8	2.5	0.480
Oligo hydramnios	2	3.7	15	5.6	17	5.3	0.002
Diabetes mellitus	4	7.4	3	1.1	7	2.2	0.705
Twin pregnancy	1	1.9	6	2.3	7	2.2	0.059
Breech presentation	2	3.7	10	3.8	12	2.8	0.021
Transverse lie	2	3.7	4	1.5	6	1.9	0.414
Obstructed labour	13	24.1	10	3.8	23	7.2	0.532
Prolong rupture of membranes	4	7.4	8	3.0	12	3.8	0.248
Me conium stained liquor	10	18.5	10	3.8	20	6.3	1.000
Cord around neck	1	1.9	0	0	1	0.3	0
Fetal distress	7	13.0	9	3.4	16	5.0	0.617
Maternal fever	2	3.7	4	1.5	6	1.9	0.414
Midwifery interference	0	0	3	0.9	3	0.9	0
History of asphyxia among previous babies	8	14.8	12	4.5	20	6.3	0.371
Severe anemia of mothers	5	9.3	9	3.4	14	4.4	0.285

Table 8: Relationship between Apgar scoring and birth asphyxia.

Apgar scoring	Birth asphyxia at first minute		Birth asphyxia at five minute	
	No.	%	No.	%
< 3	28	30.8	3	5.6
3-5	35	38.4	12	22.2
5-7	28	30.8	39	72.2

Total	91	100	54	100

X^2 = 344.247, P value = 0.00<0.05, df=9 (significant)

Table 9: Clinical presentation of birth asphyxia.

Clinical presentations	Cases of birth asphyxia	
	No.	%
No cry	7	13.0
Delayed cry	26	48.1
Grunting	45	83.3
Central cyanosis	11	20.4
Peripheral cyanosis	4	7.4
Respiratory distress	42	77.8
Lethargy	44	81.5
Irritability	4	7.4
Decreased tone	21	38.9
Increased tone	3	5.6
Unable to feed	54	100
Meconium staining	8	14.8
Convulsion	6	11.1
Need for assisted ventilation	54	100