INTRODUCTION

Asphyxia can lead to multi-organ dysfunction and a redistribution of cardiac output to maintain perfusion of Brain, Heart, Adrenal gland while Potentially compromising the perfusion of Kidney, Gastrointestinal tract, Skin,[1-4] Kidneys are very sensitive to oxygen deprivation, renal insufficiency may occur within 24 hours of a hypoxic ischemic episode.[5] Incidence of acute kidney injury (AKI) in asphyxiated neonates, ranges between 30-60% in term babies.[6-9] The incidence of hypoxic cardiomyopathy in asphyxia varies from 24-31% which includes. 10 Shock, Valvular dysfunction, Rhythm abnormalities, Congestive cardiac failure.Hence this study is done to find out the impact of shock causing AKI in asphyxiated neonates. With the objectives to know the incidence of shock in perinatal asphyxia, to know the incidence of AKI in perinatal asphyxia and to find the incidence of AKI with and without shock in perinatal asphyxia.

MATERIALS AND METHODS

This is Prospective observational study was conducted at NICU, Department of Pediatrics, Katihar Medical College, Katihar. Study Participants were 53 asphyxiated neonates who met the inclusion criteria during the period of 1st January to 31st December 2021. Institutional ethical committee approval was taken before starting the study. An informed consent was obtained from subjects willing to participate in the study.

Inclusion Criteria

All inborn, term neonates who were diagnosed as perinatal asphyxia. (Apgar score of < 7 at 1 minute as defined by WHO).[11]

Exclusion Criteria

Preterm
Neonates with perinatal history of maternal kidney disorders.
Congenital anomalies of kidney or urinary tract (as detected by antenatal or postnatal ultrasonography).
Neonates with other factor which may change kidney function tests such as: Early onset neonatal
sepsis (EONS), Respiratory distress syndrome (RDS), Necrotizing enterocolitis (NEC), Major congenital anomalies

**Methodology**

53 term asphyxiated neonates who fulfilled the inclusion criteria were enrolled in the study. A predesigned and pretested proforma was used to collect data such as gestational age, birth weight and relevant perinatal history. Findings on physical examination and systemic signs were also recorded. Assessment of the neurological status was made by Sarnat & Sarnat staging for HIE along with assessment of Anterior Fontanel, tone, seizure, pupillary size & reaction every 12 hourly.[12] All enrolled babies were subjected to ultrasonography during the course of hospital stay to rule out any congenital malformations of the urinary tract. Hemodynamic adequacy was monitored by assessing: Pulse volume, Heart rate, Capillary filling time, Temperature, Colour, Cold extremities, Blood pressure by neonatal BP cuff. Renal function parameters – blood urea, serum creatinine, urine output were monitored initially after 48 hours of life. Abnormal renal functions 72 hrs after birth, had their laboratory parameters monitored every day till recovery or death. AKI was diagnosed according to proposed KDIGO definition for neonatal AKI.[13]

Statistical analysis- Analysis of results was done using SPSS 20.0 software. Chi-square test was used to find the significance of study parameters on categorical scale between two or more groups. Microsoft Excel have been used to generate tables. P-value of <0.05 was considered significant.

**RESULTS**

Out of the 53 enrolled new-born, 19 baby develop AKI [Figure 1], and out of 53, 11 baby develop shock [Figure 2]. Out of 11 asphyxiated neonates who develop shock, 63.63% [n=7] develop AKI. Out of 42 asphyxiated neonates who does not develop shock, 28.57% [n=12] develop AKI and this is statistically significant (p=0.031) [Figure 3]. Among 53 asphyxiated neonates, majority of them had HIE stage 2 [50.9%, n=27], followed by HIE-1 [32.1%, n=17] and then HIE stage-3 [17%, n=9], which is almost similar to many studies [girish el at.][Figure 4]. In HIE stage-1 2/17 cases [11.76%] develop AKI. In HIE stage-2 12/27[44.4%] develop AKI.In HIE stage-3 5/9 [55.5%] develop AKI.This is statistically significant p=0.036 [Figure 5]. In HIE stage-1, 1/17 [5.8%] develop shock.In HIE stage-2, 4/27 [14.8%] develop shock.In HIE stage-3, 3/9 [66.6%] develop shock and this is statistically significant (p=0.001) [Figure 6].
DISCUSSION

Incidence of acute kidney injury is asphyxiated neonates ranges between 30 – 60% in term babies, which was found comparable in our study [Figure 7]. The incidence of shock and AKI in asphyxiated term baby was found comparable to study done by Aslam et al. and Girish et al. [Figure 8,9]. The incidence of AKI with shock in our study was 63.6%, which was comparable to Aslam et al, but was less than Girish et al (91.6%), however the incidence of AKI without shock (28.5%) was found lower than the studies done by Aslam et al and Girish et al. [Figure 10]. In our study we found that incidence of shock and AKI increases as the stage of HIE advances and this was found to be statistically significant.

CONCLUSION

Shock is one of the major determinants which increases the incidence of AKI by approximately 2.5-fold in perinatal asphyxia. 
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REFERENCES


