

Evaluation of Renal Function in Intramural Neonates with Birth Asphyxia in a Tertiary Care Hospital



Medical Science

KEYWORDS : Birth asphyxia, Acute Kidney Injury, AKIN Abbreviations

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ABSTRACT

Aims: To study the incidence and evaluate the severity of renal dysfunction in neonates with moderate to severe birth asphyxia.

Setting & Design: Prospective cohort study done at Neonatal unit, Department of Pediatrics, B J Medical College, Ahmedabad.

Methods & Material: Subjects were inborn neonates of ≥ 34 completed weeks of gestation with an APGAR score < 7 at 1 min after birth. Renal function tests including serum electrolytes were measured at 24 hour interval until 96 hrs of life along with urinary output monitoring. Staging of Acute Kidney Injury was done using Acute Kidney Injury Network criteria (AKIN).

Results: A total of 3012 neonates were born during the study period (January 2015 to October 2015), 49 of them met the inclusion criteria. Data from 41 neonates were available for final analysis. AKI developed in 11% ($n=3$) infants with moderate asphyxia (26) and 53.3% ($n=8$) infants with severe asphyxia (15), making a total incidence of 26.8%. AKI persisted in 12.1% ($n=5$) neonates at 96 hours of life. Ten neonates (27.7%) had serum creatinine levels > 1.5 mg/dl. One infant died and one who was critically ill was discharged against medical advice; both had AKI.

Introduction

Birth asphyxia is an eventuality having far reaching consequences in the neonatal period. Overall incidence of asphyxia is reported to vary from 1 to 1.5% at various centres (1) and is related to birth weight and gestational age of the baby. Hypoxia and ischemia can cause damage to almost every tissue and organ of the body and various target organs involved have been reported to be kidneys in 50% followed by CNS in 28%, CVS in 25% and lungs in 23% cases (2).

As kidneys are very sensitive to oxygen deprivation, renal insufficiency may occur within 24 hours of a hypoxic ischaemic episode, which if prolonged, may even lead to irreversible cortical necrosis. Early recognition of renal failure is important in babies with Birth asphyxia to facilitate appropriate fluid and electrolyte management as a stable biochemical milieu is vital. Diagnosis of renal failure is difficult in neonates as many of the established clinical and biochemical parameters are unreliable in this age group. We performed this study to determine the incidence of renal failure in birth asphyxia and to correlate the severity and type of renal failure with Apgar score and AKIN criteria.

Materials and Methodology

Setting & Design: Prospective cohort study done at Neonatal unit

Study period: January 2015 to October 2015

Inclusion Criteria: All newborns born during the study period in civil hospital with apgar score < 7 at 1 min were enrolled in study after taking informed consent.

Exclusion Criteria: Extramural newborns and Renal failure due to other causes like septicemia, congenital anomalies were excluded.

Methodology

On the basis of Apgar score at 5 minutes the asphyxiated babies were further grouped into mild (score of 6 or 7) moderate (score 5 or 4) and severe asphyxia (score 3 or

less). Relevant data were recorded on a predesigned pre-tested proforma. 24 hours urine output measurement done by applying plastic collection bags. Renal function tests including serum electrolytes were measured daily until 96 hrs of life along with urinary output monitoring. First renal function test at birth taken as baseline. Staging of AKI was undertaken using Acute Kidney Injury Network criteria (AKIN). All neonates are treated as per NNF protocols.

- Renal failure according to AKIN criteria(3)
- Abrupt (within 48 h) reduction in kidney function currently defined as an absolute increase in serum creatinine of 0.3 mg/dL or more or
- A percentage increase in serum creatinine of 50% or more (1.5-fold from baseline) or
- A reduction in urine output (documented oliguria of < 0.5 mL/kg/h for > 6 h)

Staging of AKI according to AKIN Criteria (3)

	S. Creatinine	Urine output
Stage 1	1.5-1.9 times baseline ≥ 0.3 mg/dL increase	< 0.5 mL/kg/hr for 6 hr
Stage 2	2- 2.9 times baseline	< 0.5 mL/kg/hr for 12 hr
Stage 3	3 times baseline or Increase in serum creatinine to ≥ 4 mg/dL or Initiation of renal replacement therapy	< 0.3 mL/kg/h for 24 h or Anuria for ≥ 12 h

Results:

Total numbers of newborns- 3012

Newborns with birth asphyxia-41(moderate birth asphyxia-26 and severe birth asphyxia-15)

Total incidence of birth asphyxia- 1.3 per 1000 live birth.

Total Newborns having both asphaxia & AKI- 11

Total incidence of AKI among asphyxiated newborns is 26.8%

Table 1 Serum creatinine value as per day of life.

S. Creatinine	At birth	DOL 1	DOL 2	DOL 3	DOL 4	DOL 5
<0.5 mg/dl	2	0	0	0	0	0
0.5-1 mg/dl	5	0	0	0	3	2
1-1.5 mg/dl	4	5	1	3	4	2
1.5-2 mg/dl	0	2	4	5	2	3
2-2.5 mg/dl	0	3	3	1	1	2
2.5-3 mg/dl	0	1	2	1	0	0
>3 mg/dl	0	0	1	1	0	0

AKI persisted in 12.1%(n=5) neonates at 96 hours of life.

Ten neonates (27.7%) had serum creatinine levels >1.5 mg/dl.

Table 2 Classification According to AKIN criteria

	No of patients developed AKI			Total	% of patients
	Stage I	Stage II	Stage III		
Moderate birth asphyxia (Apgar 4-6 at 1 min) n=26	2	1	0	3	11.5%
Severe birth asphyxia (Apgar 0-3 at 1 min) n=15	2	4	2	8	53.3%
Total	4	5	2	11	

Outcome:

One infant(9%) died and one who was critically ill was discharged against medical advice(9%); both had AKI. Rest 9(82%) were discharged.

Discussion

Renal injury in birth asphyxia is a potential consequence of adaptive mechanism. Amongst the recognized complications *i.e.*, acute tubular necrosis, renal vein thrombosis and renal failure, AKI is the commonest and carries a poor prognosis and may even result in permanent renal damage in up to 40% of survivors (4).

Urinary output was slightly less in neonates with severe birth asphyxia but it was statistically insignificant when compared with cases of mild and moderate asphyxia. But oliguria has been reported in higher number of neonates by other authors with figures ranging from 25% to 69.2% babies (5)

In our study no neonate remained oliguric by day 4-6 of life, which compares well with observations of Pertman, *et al.* (5) who reported transient oliguria on 1st day in 23% of newborns and urine output increased to normal values on 3rd day of life.

A reduction in number of functional nephrons caused by asphyxia and leading to ARF evokes compensatory hypertrophy of the residual nephrons thus leading to improved renal functions in early months of life. But whether subtle defects may persist, can be said only after long term follow-up and one must be cautious in prognosticating these neonates.

References

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