Original Article

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Clinical Spectrum of Acute Renal Failure in Septicemic Neonates

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Abstract

Background: In a full-term neonate, kidney functions are not fully mature and functional maturation continues postnatally. Normally they are able to cope up with most of the rapidly changing functional demands of the body and adapt to various endogenous and exogenous stresses. However in stressful conditions like septicemia, this capacity may be overcome leading to renal dysfunction. Neonatal septicemia or Sepsis neonatorum is a clinical syndrome resulting from the pathophysiologic effects of local or systemic infection in the first month of life. **Subjects and Methods:** All normal, full term, appropriate for gestational age neonates, delivered normally or by Caesarean section, who were without antenatal, perinatal or immediate postnatal complications and without congenital anomalies, admitted after the first day of life to the N1CU, during the above mentioned period, with a clinical suspicion of septicemia were evaluated. **Results:** Feeble cry (or the shrill cry of meningitis in some cases) was present in all cases (100.0%). Next important symptom was not sucking at breast which was present in all cases where breast feeding was initiated (In two babies it was not initiated) giving a total incidence of 92.6%. Other major symptoms included decreased urine output (70.4%), hurried respiration (70.4%) and vomiting (63.0%). Other symptoms more commonly found were convulsions (48.1%), hypothermia (48.1%) and fever (37.0%). **Conclusion:** Major differences between intrinsic and prerenal failures in septicemia in their presenting complaints were hurried respiration, cyanosis, grunting, convulsions, vomiting, fever and oedema were more in intrinsic renal failure where asoligoanuria, hypothermia, and loose stools were more in prerenalfailure.

Keywords: Acute Renal failure, Septicemic neonates, Prerenalfailure.

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Introduction

With the changes that occur in the transition from fetal to post natal life, the kidney must immediately assume the placenta's role of fluid, electrolyte and biochemical homeostasis. The newborn's kidney is well equipped to do so, reflected in his ability to thrive and grow under variable circumstances of fluid and solute intake. There are however, certain inherent immaturities in neonatal renal function that need specialized consideration in many medical and surgical situations. Diagnosis and management of abnormal renal functioning in neonates must consider physiologic and pathologic events particular to the perinatal period.[1]

Kidneys are responsible for body fluid and electrolyte homeostasis, in addition to being the principle excretory organ of the body. Acute renal failure in its broadest sense is the relatively abrupt cessation of kidney function, the result of which is the inability to maintain nitrogen,fluid and electrolyte balance.

Acute renal failure is increasingly recognized in neonates now a days which is attributed to the following factors, (i) Rapid advances in technology and better understanding of neonatal physiology have resulted in vast improvements in the quality of care of critically ill neonates which improved the survival (ii) Increased awareness of acute renal falure and importance of anticipatory monitoring (iii) Antenatal diagnosis of renal anomalies by ultrasonography (iv) Increased incidence of sepsis and use of nephrotoxic drugs and (v) Increased survival of premature neonates.[2]

In a full-term neonate, kidney functions are not fully mature and functional maturation continues postnatally. Normally they are able to cope up with most of the rapidly changing functional demands of the body and adapt to various endogenous and exogenous stresses. However in stressful conditions like septicemia, this capacity may be overcome leading to renal dysfunction. Neonatal septicemia or Sepsis neonatorum is a clinical syndrome resulting from the pathophysiologic effects of local or systemic infection in the first month of life.

Primary site of invasion in septicemia is most often the blood stream with the spread to meninges and other organs in some cases. Systemic bacterial disease occurs in one to ten cases per thousand live births. The incidence rates vary from nursery to nursery and depend on conditions predisposing to infection.[3]

Neonatal bacterial septicemia is the commonest cause of neonatal admissions and neonatal mortalityin India. In a developing country like India, high incidence of septicemia may be due to contaminated in utero environment, infected birth passages, infection at birth, procedures without due

regard to asepsis, the practice of prelacteal or top feeding, poverty and ignorance.[4]

Acute renal failure in neonatal septicemia is known entity. It should be considered in an anticipatory manner in all septicemic newborns, because it a life threatening medical emergency and demands the utmost skill of the pediatrician in its diagnosis and management. The seriousness of this disease is attested by the high mortality and morbidity that continues to be reported which can be brought down only by early diagnosis and management.[5]

Subjects and Methods

The study includes the clinical profile of 100 cases of normal full-term neonates with septicemia admitted to the medical college

All normal, full term, appropriate for gestational age neonates, delivered normally or by Caesarean section, who were without antenatal, perinatal or immediate postnatal complications and without congenital anomalies, admitted after the first day of life to the N1CU, during the above mentioned period, with a clinical suspicion of septicemia were evaluated.

A detailed antenatal history was elicited to rule out familial renal malformations, complications like oligohydramnios, hemorrhage, infections, hypertension and antenatal ultrasonographic abnormalities. Natal history was taken to find out the type of delivery, indications of intervention, drugs used and other complications. Postnatal history comprising of perinatal asphyxia and other complications, time the neonate first voided urine, initiation of breast feeding or prelacteal feeds and details of examination at birth wherever possible were noted.

The presenting complaints with special reference to the aetiology of septicemia, possible factors to the development of acute renal failure, clinical features of presentation and complications if any were recorded. A detailed clincal examination was also carried out.

A sample of blood was taken for Blood urea (Diacetylmonoxime method) , Serum creatinine (Modified Jaffe's method), Serum electrolytes (Auto analyser/Flame photometry), Hb% (Sahli'smethod), Serum bilirubin (colorimetric), Peripheral smear, Total count, Differential count, and Blood culture. 24 hrs urine output was measured using condom catheters or empty disposable syringe sachets in males and perineal urine collection bags in females. Urine samples were sent for Albumin (sulphosalicylic acid method), Microscopy, Urine creatinine (modified Jaffe 's method), Urine sodium (Flame photometry/ Autoanalyser) and Urine culture wherever possible..

A chest roentgenogram, Electrocardiograph, Nephrosonogram and CSF study were done wherever indicated.

Diagnostic criteria used for septicemia was blood culture positivity. Out of the 185 cases initially evaluated, first consecutive 100 blood culture positive cases were included in the study and analysed in detail.

A diagnosis of acute renal failure was based on 3 parameters - Blood urea >40 mg% or Serum creatinine>1.0 mg% or Urine output < 0.5 ml/kg/Hr.

All cases thus identified as having acute renal failure were divided into two groups as intrinsic renal failure and prerenal failure. The values of Fractional excretion of sodium percentage (FeNa%) and Renal failure index (RFI) were taken into consideration for this division.

An intravenous fluid challenge was given to neonates in both the groups, and furosemide challenge to some. Both groups were treated according to standard conservative regimen, proper antibiotics, other supportive measures, transfusion, peritoneal dialysis and ventillator care according to indications.

Blood urea, Serum creatinine and Serum electrolytes were measured on admission, after 24 hrs and then serially according to each case. A daily follow up of each case with detailed examination was carried out. Recovery was defined as return of biochemical values to normal, normal urine output, regaining of normal physical activity and breast feeding.

Results

Table 1: Incidence of Septicemia

Age of Onset	Intrinsic Renal Failure	Pre-renal Failure	Total	Total no. Of Cases studied In this group	Percentage
Early onset septicemia	07 (58.3%)	06 (40.0%)	13	44	29.5%
Late onset septicemia	05 (41.7%)	09 (60.0%)	14	56	25.0%

Table 2: Incidence and type of delivery

Type of Delivery	Intrinsic Renal Failure	Pre-renal Failure	Total	Total no. Of Cases studied In this group	Percentage
Normal vaginal	09 (75.0%)	11 (73.3%)	20	69	29.0%
Caesarean section	03 (25.0%)	04 (26.7%)	07	31	22.6%

Table 3: Common Presenting Symptoms in Septicemia with Acute Renal Failure

Symptoms	Number Of Cases And Percentage		
	Int. Renal Failure	Prerenal Failure	Total
Weak cry	12 (100.0%)	15 (100.0%)	27 (100.0%)
Refusal of feeds	11 (91.7%)	14 (93.3%)	25 (92.6%)
Oligoanuria	07 (58.3%)	12 (80.0%)	19 (70.4%)
Hurried respiration	11 (91.7%)	08 (53.3%)	19 (70.4%)

Vomiting	08 (66.7%)	09 (60.0%)	17 (63.0%)
Convulsions	07 (58.3%)	06 (40.0%)	13 (48.1%)
Cold body & Extremities	03 (25.0%)	10 (66.7%)	13 (48.1%)
Fever	05 (41.7%)	05 (33.3%)	10 (37.0%)
Cyanosis	04 (33.3%)	04 (26.7%)	08 (29.6%)
Grunting	06 (50.0%)	00 (00.0%)	06 (22.2%)
Swelling of limbs/ puffiness of face	04 (33.3%)	01 (06.7%)	05 (18.5%)
Loose stools	01 (08.3%)	04 (26.7%)	05 (18.5%)

Feeble cry (or the shrill cry of meningitis in some cases) was present in all cases (100.0%). Next important symptom was not sucking at breast which was present in all cases where breast feeding was initiated (In two babies it was not initiatied) giving a total incidence of 92.6%. Other major symptoms included decreased urine output (70.4%), hurried respiration (70.4%)and vomiting (63.0%). Other symptoms more commonly found were convulsions (48.1%), hypothermia (48.1%) and fever (37.0%).

Major differences between intrinsic and prerenal failures in septicemia in their presenting complaints were hurried respiration, cyanosis, grunting, convulsions, vomiting, fever and oedema were more in intrinsic renal failure where asoligoanuria, hypothermia, and loose stools were more in prerenalfailure.

Table 4: Common Physical Signs in Septicemia with Acute Renal Failure

Physical Signs No.		Number Of Cases And Percentage		
		Int. Renal Failure	Prerenal Failure	Total
01	Lethargy	12 (100.0%)	15 (100.0%)	27 (100.0%)
02	Weak reflexes	12 (100.0%)	15 (100.0%)	27 (100.0%)
03	Abdominal distension	09 (75.0%)	10 (66.7%)	19 (70.4%)
04	Respiratory distress	11 (91.7%)	07 (46.7%)	18 (66.7%)
05	Dehydration	06 (50M%)	11 (73.3%)	17 (63.0%)
06	Anemia	09 (75.0%)	07 (46.7%)	16 (59.3%)
07	Hepatomegaly	09 (75.0%)	07 (46.7%)	16 (59.3%)
08	Jaundice	06 (50.0%)	07 (46.7%)	13 (48.1%)
09	Sclerema	05 (41.7%)	07 (46.7%)	12 (44.4%)
10	Acidosis	08 (66.7%)	02 (13.3%)	10 (37.0%)
11	Apnoea	05 (41.7%)	03 (20.0%)	08 (29.6%)
12	Oedema	04 (33.3%)	01 (06.7%)	05 (18.5%)
13	Bleeding manifestations	03 (25.0%)	02 (13.3%)	05 (18.5%)
14	Hypertension	04 (33.3%)	00 (00.0%)	04 (14.8%)
15	Cardiac failure	02 (16.7%)	00 (00.0%)	02 (07.4%)
16	Cardiac arrhythmias	02 (16.7%)	00 (00.0%)	02 (07.4%)

Most important physical signs in septicemia with acute renal failure were lethargy (100.0%), and weak reflexes (100.0%). Abdominal distension (70.4%), respiratory distress (66.7%) and dehydration (63.0%) constituted next important signs. Anemia (59.3%), hepatomegaly (59.3%) and jaundice (48.1 %) were present in about half of the patients. Other features included sclerema (44.4%), acidosis breathing (37.0%), apnoea (29.6%), oedema(18.5%), bleeding manifestations (18.5%), hypertension (14.8%) and cardiac manifestations (14.8%).

Major differences between intrinsic and prerenal failures in septicemia in clinical presentation were hypertension, respiratory distress, anemia, oedema, acidosis, apnoea, cardiac manifestations and abdominal manifestations were comparitively higher in intrinsic renal failure whereas dehydration and sclerema were more in prerenal failure.

Eventhough there was no association between the aetiological organism and type of acute renal failure precipitated, some of the organisms like E. Coli, Klebsiella, Group-B. Streptococci and Acinetobacter produced septicemia early, whereas Coagulase positive staphylococci, Proteus and Pseudomonas produced septicemia late.

Discussion

Neonatal bacterial septicemia is the commonest cause of neonatal admissions and neonatal mortality in India . In a developing country like India, this may be due to contaminated in utero environment, infected birth passages, infection at birth, procedures without due regard to asepsis, the practice of prelacteal or topfeeding, poverty and ignorance. In the present study, incidence of septicemia in normal fullterm neonates was 18.6% among all NICU admissions during the study period.

Blood culture positivity in septicemia was 54.1% in this study, whereas Somu .N, et al found 75.0% and Monga. K, et al found 63.0% of culture positivity in their study groups. The difference may be due to technical lapses.[6]

In the present study incidence of acute renal failure in septicemic neonates was 27.0% with a 12.0% incidence of intrinsic renal failure and 15.0% incidence of prerenalfailure. This was comparable with Griffin, et al who showed that 16.0% of septicemic neonates developed intrinsic renal failure. Jayashree, et al in a similar study showed 20.0% incidence. Incidence of acute renal failure in neonates admitted to NICU in a study by Norman and Asadi was 23.0% in which 17.0% was prerenalfailure and 6.0% was intrinsic renal failure. These figures were comparable

with the present study in the incidence of intrinsic renal failure and prerenal failure in acute renal failure in neonates.[7]

Different authors showed a higher incidence of acute renal failure in septicemia in different clinical settings like Meeks and Sims - 30.0%, Nammalwar.BR, et al - 41.7%, Unni .JC, et al - 50.0% and Pereira .S, et al - 90.0%. These were in contrast to the present study and may be due to difference in selection of cases and methods.[8]

In the present study. Intrinsic renal failure was seen more (58.3%) in early onset septicemia where asprerenal failure was seen more (60.0%) in late onset septicemia.

This may be because early onset septicemia, commonly due to Group-B. streptococci and Gram negative organisms are more extensive and complications like endotoxic shock, sclerema, necrotizing enterocolitis, disseminated intravascular coagulation etc are more common . Griffin, et al studied 25 neonates with septicemia of which 4 infants with E. coli septicemia had intrinsic renal failure.

Neonatal sepsis is twice as frequent in the male infants as compared to female infants. This observation was seen also by Somu .N, et al.

In the present study male to female ratio in septicemia was 57:43.

There was no difference in the incidence of intrinsic renal failure between males and females in the present study. This was in contrast to Pereira .S, et al series where male to female ratio in acute renal failure was 12:8.

The present study showed a higher incidence of septicemia (69.0%) in vaginally delivered babies compared to babies delivered by Caesarean section (31.0%) . This may be due to infected birth passages, infection at birth or procedures without due regard to asepsis as stated earlier because a good number of normal deliveries in this part of the world are conducted at home by untrained Dhais.

The incidence of acute renal failure was also more (29.0%) in vaginally delivered septicemic neonates as compared to the other group (22.6%). The incidences of intrinsic and prerenal failures were high, 75.0% and 73.3% respectively in vaginal delivery compared to the other group.

Clinical features of acute renal failure in septicemic neonates were a combined picture of both septicemia and acute renal failure. The presenting symptoms in this study were weak cry, refusal of feeds, oliguria, hurried respiration, vomiting, convulsions, hypothermia, fever, cyanosis, grunting, oedema and loose stools. These were comparable with other studies and data in published literature.[9]

Oliguria was present in majority of acute renal failure cases (70.4%) in this study and incidence was more in prerenal failure (80.0%). This was comparable with 71.4% incidence of oliguria in Anand .SK, et al study, 80.0% in Pereira .S, et al series, 50.0% in Chevalier .RL, et al series, 100.0% in Griffin, et al series and data in literature.

Common physical signs in this study were a combination of signs in sepsis, acute renal failure and their complications. They were lethargy, weak reflexes, abdominal distension, respiratory distress, dehydration, anemia, hepatomegaly, jaundice, sclerema, acidosis, apnoea, oedema, bleeding manifestations, hypertension and cardiac manifestations. These were comparable to other studies and data available in published literature.[10]

Incidence of hypertension in this study was 14.8% and was seen only in intrinsic renal failure. This was comparable to Meeks and Sims (79) where the study showed 11.1% incidence.

Conclusion

Incidence of septicemia in normal, fullterm, appropriate for gestational age neonates was 18.6% of all NICU admissions in the present study. 27.0% of these cases developed acute renal failure. 12.0% of septicemic neonates developed intrinsic renal failure. In the present study intrinsic renal failure was seen more in early onset septicemia (58.3%). Normal vaginally delivered babies were more prone to develop septicemia (69.0%) and intrinsic renal failure (75.0%).

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