hosts due to prematurity, low birth weight and birth asphyxia probably contributed to the systemic spread rather than the colonization of the GI tract. Out of five cases of *S. senftenberg septicemia* reported prior to this study, four survived. However, 4 other newborns where the organism could be isolated from CSF alone, died. In the present study, one child who developed meningitis died within 72 hours of starting therapy. Since a high incidence of complications such as shock, apneic spell and meningitis were observed in the present series, an aggressive approach towards the management of *S. senftenberg* sepsis is warranted.

Follow-up of neonates who had suffered from salmonellosis is necessary as it has been shown that smaller and younger neonates are likely to excrete organism for a longer time and antibiotic therapy tends to increase the duration of carrier state(9). Such carriers maintain infection in the community and may act as source for starting fresh epidemics.

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Citrobacter Septicemia in Neonates

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A major cause of neonatal mortality in India is bacterial sepsis(1). Neonatal infections acquired in the hospital are noso-

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comial. Because most early onset infections are acquired intrapartum, infections that develop later than 48 to 72 hours after birth are usually considered nosocomial. However, exceptions include late onset infections with organisms acquired from the mother's genital tract and nosocomial infections acquired from the delivery room, in which signs of infection develop soon after birth(2). Nosocomial infections may be sporodic or occur as epidemics, and they may occur in hospital or after discharge. The list of organisms causing nosocomial infection is long and extensive. Multidrug resistant Gram negative bacilli like *Salmonella typhimurium,* Klebsiella and Citrobacter species are gaining importance. Nosocomial outbreak of citrobacter infection due to umbilical colonization has been described earlier(3). Although earlier reports of citrobacter infections in infants have been suspected to be nosocomial in origin, common source of outbreaks have not been identified and mode of transmission have not been well defined. This communication presents clinical profile and outcome of a citrobacter outbreak in the delivery room unit of this hospital between the period July to August, 1992.

Case Reports

During the period there were 113 births of which five were preterms. Of the ten neonates included in the study, eight were full term and two preterm. Two neonates were LSCS born. History of premature rupture membranes were noted in two cases. Only one neonate required active resuscitation at birth. There were no other risk factors observed either antenatally or intranatally.

The clinical profile of these ten neonates is given in the *Table.* There was no correlation between culture studies of the mother's and that of neonates. No asymptomatic carriers were detected during epidemiological surveillance. Culture studies of equipments confirmed contamination of the suction apparatus and its tubing. This was secondary to contamination of delivery room unit as a result of drainage pipe leakage. The surveillance confirmed the occurrence of early onset of nosocomial neonatal septicemia as a result of *Citrobacter freundi.*

Discussion

Increasing use of life supportive measures and improved survival of small sick neonates has resulted in a rise in nosocomial infections. However, occurrence of early onset nosocomial infection is directly related to the sterility of delivery room unit, prevalence of asymptomatic carrier state, and emergence of multidrug resistant strains of bacteria poses a threat to intensive care units.

Citrobacter is a potential pathogen and has been shown to be a primary invader during early infancy with a propensity for producing severe necrotizing meningo encephalitis(4). Two recent studies have highlighted the occurrences of multidrug resistant citrobacter septicemia(5,6). However, both studies were related to late onset sepsis. Also, in these studies the source of infection and mode of transmission have not been defined.

All the neonates presented with classified description of early onset sepsis with dominant sign being tachypnea. Retrospectively, it could be surmized that onset of tachypnea in these neonates was a very important sign and a warning signal for possibility of citrobacter septicemia. Such a correlation has not been reported. The importance of careful peripheral blood smear study to look for significant leucopenia,

TABLE–*Clinical Profile of Neonates with Citrobacter Septicemia*

Features	Patient number									
	1	2	3	4	5	6	7	8	9	10
Onset (h)	24	10	18	36	24	18	24	12	36	18
Signs										
Tachypnea	+	+	+	+	+	+	–	–	+	+
Fever	+	+	–	–	–	–	+	–	+	–
Icterus	–	–	–	–	+	–	–	–	–	+
Hypothermia	–	–	–	–	–	+	–	+	–	+
Reflexes	N	N	N	Poor	N	N	N	N	Poor	N
Seizures	–	–	–	+	–	–	–	–	+	–
Peripheral blood smear study										
Leucocytosis	+	–	–	–	+	–	–	–	–	–
Neutropenia	–	+	+	+	–	+	+	+	+	+
Band cell (%)	0.2	0.4	0.3	0.4	0.2	0.3	0.4	0.4	0.3	0.3
Toxic granulations	+	+	+	+	–	–	+	+	–	+
Platelets	N	N	N	°	N	N	°	°	N	N
Culture study										
Blood	+	+	+	+	+	+	+	+	+	+
Stool	–	–	+	–	+	–	–	–	–	–
Pus	–	+	+	+	–	+	+	–	+	–
Umbilicus	–	+	+	+	–	+	+	–	–	–
CSF	–	–	–	–	–	–	–	–	–	–
Antibiotic sensitivity pattern										
Ampicillin	R	R	R	R	R	R	R	R	R	R
Chloramphenicol	R	R	R	R	R	R	R	R	R	R
Gentamicin	S	S	R	R	R	S	S	S	S	R
Cloxacillin	R	R	R	R	R	S	R	S	R	R
Trimethoprim	S	S	R	R	R	S	S	R	S	R
Norflox	S	S	S	S	S	S	R	S	R	R
Netromycin	S	S	S	S	S	S	S	S	S	R

(Contd.)

Features	Patient number									
	1	2	3	4	5	6	7	8	9	10
Complications										
Abscesses	–	+	+	+	–	+	–	+	+	–
DIC	+	–	–	+	–	–	+	–	–	+
Hepatitis	–	–	–	–	–	+	–	–	–	+
Sclerema	–	+	+	–	–	+	–	–	–	–
Antibiotics used										
Amplicillin	✓	–	–	✓	–	–	–	–	–	–
Gentamicin	✓	–	–	✓	–	–	–	–	–	–
Cefatoxime	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Norflox	–	–	–	–	✓	–	–	–	–	–
Netromycin	–	–	–	–	–	–	–	✓	✓	✓
Outcome	Died	–	–	–	–	–	–	–	–	Died

toxic granulation, cytoplasmic vaculation, and band forms (>0.2%) is amply illustrated in this study. This simple and rapid test permits clinician to suspect neonatal sepsis and based on experience and existing antibiotic sensitivity pattern an appropriate antibiotic can be started at the earliest.

Culture studies revealed *Citrobacter freundi* as the causative organism for the septicemia. In six of the neonates the same organism was also grown from pus sample. Based on the antibiogram, the organisms were considered similar. The antibiotic sensitivity pattern suggested that the species to be multidrug resistant to the commonly exhibited antibiotics in the hospital. Emergence of such resistant organism is alarming in view of the previous study(5).

Among the complications, multiple subcutaneous abscesses and sclerema deserve mention. In addition to the extreme invasive properties of the organism, repeated venepuncures in these neonates could have contributed to occurrence of multiple abscesses. Sclerema, a sign considered ominous sign was seen in three neonates. With treatment sclerema disappeared in all three neonates. Probably the disappearance of sclerema is directly related to the recovery in a given case.

Epidemiological surveillance confirmed the outbreak of citrobacter early onset neonatal septicemia. The source for repeated infection was traced to the contaminated suction apparatus. This is considered secondary to contamination of the delivery room unit as a result of drainage pipe leak. Further, the screening of mother's of all neonates, and correlation between culture studies of neonates and that of mother's excluded the possibility of vertical transmission. The outbreak was contained once the delivery room unit was closed and alternate arrangements made. It is to be reiterated that though less common early

onset nosocomial infection is a potential danger in any delivery room unit whenever there is breakdown in asepsis and hygienic measures.

Nosocomial infections are preventable and an 'Infection Control Committee' should be instituted in all major hospitals to monitor and review cases of infection, establish an antibiotic policy, to create awareness and to educate medical and paramedical staff about it.

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Vitamin A and Post Measles Complications

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Measles a common childhood disease, predisposes to vitamin A deficiency(1,2). Such a deficiency can modify the outcome and the complication of the disease. Barclay and Hussey(1,3) have observed that the mortality due to measles was much higher in those who did not receive vitamin A supplementation. We studied 107 children with measles and its various complications, in an attempt to correlate their pattern and its severity with vitamin A status.

Material and Methods

One hundred and seven consecutive patients, admitted in JIPMER Hospital, Pondicherry, South India, during a one year period from June 1989 for various complications of measles, were included in this study. On admission, a diagnosis of vitamin A deficiency was made by the presence of night blindness/conjunctival xerosis/Bitot's spots/corneal xerosis/ulcer/keratomalacia. The relevant data were entered on a preplanned proforma and the details analysed

Results

Out of the 107 children, 71 were boys and 36 were girls. Sixty-nine (64.5%) were below 3 years, 26 (24.3%) between 3 and 5 years and 12 (11.2%) more than 5 years of age. No child was below 6 months of age, 29 (27.1%) were well nourished. Sixty-seven (62.6%) had no clinical evidence of vitamin A deficiency. The various complications seen in the groups with and without vitamin A deficiency are shown in the *Tables I and II.* The respiratory complica-

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