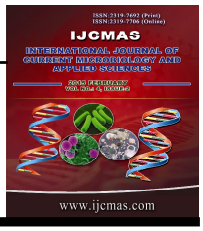
Int.J.Curr.Microbiol.App.Sci (2015) 4(2): 172-182

International Journal of Current Microbiology and Applied Sciences
ISSN: 2319-7706 **Volume 4 Number 2 (2015) pp. 172-182**
http://www.ijcmas.com



Original Research Article

Bacteriological profile of neonatal septicemia

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A B S T R A C T

Septicemia continues to be a major cause of neonatal mortality and morbidity. A very wide spectrum of organisms has been described for cases of neonatal septicemia and this spectrum is subject to geographical alterations. Moreover, the organisms isolated are often resistant to multiple antimicrobials which make the treatment difficult and grave sequelae ensue. The present study was undertaken to study the incidence of neonatal septicemia, clinical and bacteriological profile of neonatal septicemia and their antibiotic susceptibility pattern from June 2008 to December 2010 in a Government medical college and hospital. Male, preterm and low birth weight neonates were more prone for septicemia and the incidence of clinically diagnosed neonatal septicemia was 13.86/1000 live births in the present study. *Klebsiella pneumoniae*, *Pseudomonas aeruginosa* and *Acinetobacter baumannii* were the common isolates from neonatal septicemic cases. There was maximum resistance to the commonly used cephalosporins and aminoglycosides among gram negative organisms. Considering the burden of mortality resulting from septicemia, better diagnostic facilities should be employed for early detection of septicemia and rational use of narrow spectrum antibiotics is recommended.

Keywords

Septicemia, Neonatal, Incidence, Bacteriological profile, Antibiotic susceptibility

Introduction

According to National Neonatal Perinatal Database (NNPD) 2002–03 collected from 18 centers from various parts of India, Neonatal mortality rate has been reported to be 25.3 per 1000 intramural live births. Neonatal septicemia was one of the commonest causes of neonatal mortality contributing to 18.6% of intramural deaths. The reported incidence of nosocomial septicemia in neonates from India ranges from 1.5% to 37%. Every year an estimated 4 million babies die in the first 4 weeks of life. Globally, the main direct causes of neonatal death are estimated to be preterm birth (28%), severe infections (26%), and asphyxia (23%) (Lawn *et al.*, 2005)

Various laboratory tests are utilised to diagnose septicemia like total leucocyte count, C- reactive protein level, erythrocyte sedimentation rate, Acridine orange stained buffy coat smear examination, Nitroblue tetrazolium test, etc. The advantages of these tests are that they are sensitive indicators of

sepsis, less expensive and rapid. However, these tests never tell us anything about the aetiology of septicemia, whether the sepsis is unimicrobial or polymicrobial and their antimicrobial susceptibility pattern. The gold standard for diagnosis of septicemia is the isolation of the bacteria from a blood culture which takes at least 48 hours to confirm the diagnosis; a delay of which a neonate can ill afford for initiation of appropriate therapy.

The bacteriological profile of neonatal septicemia is constantly under change with advances in early diagnosis and treatment. In developed countries, group B streptococci and coagulase negative *Staphylococci* are the most common aetiologic agents for early onset and late onset sepsis (EOS & LOS) respectively. However, in the developing countries, these organisms are rare with an entirely different bacterial spectrum (Mahapatra *et al.,* 2002). Resistance to commonly used antibiotics is increasing. Establishment of appropriate rational antibiotic policy is essential to control this growing problem. There is an urgent need to do longitudinal surveillance of the microbial flora in every hospital (Deorari, 2006).

With this background, the present study was undertaken to study the incidence of neonatal septicemia, the clinical and bacteriological profile of neonatal septicemia and their antibiotic susceptibility pattern in a Government Medical college and hospital.

Material and Methods

The present study was carried out in a Government Medical College and hospital during the period from June 2008 to December 2010.

Inclusion criteria: As per the criteria by Integrated management of Childhood illness (IMCI) and WHO young infant study group (Vergnano *et al.,* 2005), all neonates presenting with convulsion, respiratory rate>60breaths/min, severe chest indrawing, nasal flaring, grunting, bulging fontanelle, redness around umbilicus extending to skin, temperature>37.7°C or <35.5°C, lethargic or unconsciousness, reduced movements, not able to feed, not sucking at all, crepitations, cyanosis, reduced digital capillary refill time (>3 seconds) were included in the study.

Exclusion criteria: Neonates who had undergone surgery because of risk of wound infection, congenital anomalies, birth weight less than 1000 gms and age more than 28 days at the time of diagnosis were excluded from the study.

Case definition: Neonatal septicemia is a clinical syndrome characterized by systemic signs of infection in first month of life (Vinodkumar *et al.,* 2008). It is classified as early onset sepsis (EOS) within 72 hours of birth and late onset-sepsis (LOS) after 72 hours of birth.

Sample collection (Koshi Grace., 1986): Blood sample was collected before starting antimicrobial therapy. In patients already on antibiotic therapy, blood sample was taken just before the next dose of antimicrobial agents. 2 ml of blood was drawn using a sterile syringe, out of which 1 ml of the blood sample was inoculated aseptically into a blood culture bottle containing 10 ml of Brain heart infusion broth in a ratio of 1:10. Clotting of blood was prevented by addition of 0.025% Sodium Polyanethol Sulphonate (SPS). 1 ml of the blood was allowed to clot in a sterile bottle to collect serum for the estimation of C-reactive protein. The bottles were immediately brought to the Microbiology laboratory for incubation at 37°C. Blood culture bottles were carefully

examined for macroscopic evidences of growth such as hemolysis of RBC, gas in medium, uniform or surface turbidity, surface pellicle and white grains on the surface or deep in the blood layer. Gram stained smear was made if the broth showed visible signs of growth. First subculture was made after 6–18 hours. Thereafter daily subculturing was done for 10 days.

After identification on basis of colony morphology, isolates were subjected to following biochemical reactions for confirmation along with positive and negative control: Carbohydrate fermentation test, Indole production, methyl red test, Voges Proskauer Test, Citrate utilisation test, Urease test, Nitrate reduction test, triple sugar iron test, catalase test, coagulase test, oxidation-fermentation glucose test, Optochin sensitivity test, bile solubility test, aesculin hydrolysis, hanging drop preparation and phosphatase test.

Antimicrobial susceptibility testing was performed for all the bacterial isolates as per CLSI guidelines by modified Kirby Bauer method. For all the tests, positive and negative controls were kept. Every batch of Mueller Hinton agar and antibiotic discs were tested by using ATCC control strains. The plates were incubated at 37°C. After overnight incubation, the diameter of clear zone around the disc was measured and interpreted as sensitive or resistant according to the zone diameter as per CLSI. Cefoxitin was used as a surrogate for detecting methicillin resistant *Staphylococcus aureus* (MRSA) and methicillin sensitive *Staphylococcus aureus* (MSSA). B lactamase production was detected by chromogenic cephalosporin method. For detecting high level aminoglycoside resistance; gentamicin (120µg) and streptomycin (300µg) discs were used.

All the isolates belonging to Enterobacteriaceae group were tested for extended spectrum beta lacatmases (ESBL) by CLSI phenotypic confirmatory test and double disc method (Figure 1).

Double disc method: For testing ESBL, a lawn culture of test strain was exposed to discs of amoxyclav (20 µg + 10 µg) and cefotaxime (30 µg) placed at a distance of 1.5 cm from edge to edge. After overnight incubation, there was extension of zone of inhibition of cefotaxime disc towards the disc of amoxyclav in case of ESBL producer organisms.

CLSI phenotypic confirmatory test: ESBL was tested by applying the discs of ceftazidime (30 µg) and ceftazidime and clavulinic acid (30 µg + 10 µg) to the lawn culture of the test organism. If the zone of inhibition around ceftazidime – clavulinic acid was ≥ 5 mm than the zone of inhibition around ceftazidime disc, then the test organism was considered as ESBL producer.

Metallo-β-Lactamase (MBL) production: *Pseudomonas* and *Acinetobacter* isolates found resistant to imipenem were screened for metallo-β-lactamase production by combination disc test (Yong *et al.*, 2002). A colony of the suspected isolate was suspended in Mueller Hinton broth and turbidity was adjusted to 0.5 McFarland opacity standards. Lawn culture was prepared on Mueller Hinton agar and combination disc test was put. The combinations used were imipenem (I) and imipenem – EDTA (I-EDTA).Two 10 µg imipenem discs were placed 10 mm apart from edge to edge on the plate and 750 µg of 0.5 M –EDTA solution was then applied to one of the imipenem disc.The inhibition zones of the imipenem (I) and imipenem – EDTA(I-EDTA) discs were compared after overnight incubation. In case of

Pseudomonas spp, if the zone of inhibition around I-EDTA was ≥ 7mm than the zone of inhibition around Imipenem disc, then the test organism was said to be MBL producer. In case of *Acinetobacter spp*, if the zone of inhibition around I-EDTA was ≥ 17mm than the zone of inhibition around Imipenem disc, then the test organism was said to be MBL producer.

CRP estimation was done with rapid slide agglutination test using C-Reactive protein reagent.

The data accrued on all neonatal sepsis was analyzed using SPSS version 17.0.Chi-square test was used in assessing the associations between categorical variables. A p-value of 0.05 or less was considered statistically significant.

Result and Discussion

Out of the 14,280 live births during the study period from June 2008 to December 2010, 198 neonates were diagnosed clinically as septicemic according to WHO infant study group and IMCI criteria giving an incidence of 13.86/1000 live births. This was comparable to the study done by Karthikeyan and Premkumar (2001).

Out of 198 cases studied, 71 (35.86%) neonates had positive blood culture.125 (63.13%) cases were of early onset and 73 (36.87%) cases were of late onset septicemia. 36 % of early onset and 35.61% of late onset septicemia cases were culture positive.118 (59.6%) of the 198 cases studied were males. Males contributed to 60.56% of the culture positive cases. Khatua et al (1986) postulated that the factors regulating the synthesis of gamma globulins are probably situated on the X chromosome. Presence of one X chromosome in the male infant thus confers less immunological protection compared to female counterpart. Out of 198 cases studied, 113 cases (57.07%) were preterm. Among 71 culture positive cases, 46 (64.78%) were preterm. 66.67% of early onset and 61.54% of late onset culture positive septicemia babies were preterm. Premature infants are extremely vulnerable to infection because of their inherent compromised immunity, vulnerable skin and mucosal barrier, prolonged in hospital stay and extensive interventions for other complication of prematurity (Gerdes, 2004).

In the present study, 143 (72.22%) cases were below 2.5 kg. Among 71 culture positive cases, 54 (76.04%) were below 2.5 kg. 35 of 45 (77.78%) early onset culture proven septicemia cases had their birth weight below 2.5 kg. 19 (73.07%) of 26 late onset culture proven septicemia cases were below 2.5 kg. Mondal *et al.* (1991) stated that LBW infants have low IgG and they are more susceptible to infections. In the present study, 55 of 71 (77.47%) culture positive cases were delivered spontaneously without assistance. Other 16 (22.53%) were born by assisted delivery.

Preterm (52.8%), birth asphyxia (64.8%), prolong duration of labour (48%), unclean vaginal examination (78.4%) and foul smelling liquor (30.4%) were the common predisposing factors among early onset septicemia cases. Fanaroff *et al.* (1998) observed that birth asphyxia was significantly associated with neonatal sepsis. In the present study, birth asphyxia was seen in 64.8 % of cases. Higher proportion of late onset septicemia babies were preterm (60.27%) and low birth weight (53.42%). More often they had prelacteal feeds (20.54%) and some focus of infection like GIT Infection (19.17%), Omphalitis (8.21%) and IV site infection (10.95%) as risk factors for sepsis. Afterbirth skin and umbilical cord

become important alternative routes for the entrance of bacteria in to the systemic circulation. The umbilical cord stump is frequent site for cutaneous infection leading to septicemia; however, cellulitis may occur at sites of injections, intravenous fluid administration and capillary or venous blood sampling. The insertion of umbilical venous and arterial catheters deserves increased attention as potential route of entry for bacteria (Gotoff *et al.,* 1970).

In the present study, higher proportion of neonatal sepsis cases had intrapartum risk factors like duration of labor more than 24hrs (48%), unclean vaginal examination (78.4%) and foul smelling liquor (30.4%). Septicemia is more common among those whose mother had PROM with increased risk of contamination of amniotic fluid by organisms from birth canal before delivery (Saxena *et al.,* 1980). In the present study, maternal fever was seen in 12% of cases. Higher proportions of septic babies had vague symptoms like refusal of feeds (66.67%), lethargy (73.24%), diarrhea (65.65%), abdominal distension (25.75%), jaundice (28.28%), etc.

Gram negative bacilli were found to be the commonest cause of neonatal septicemia (69.01%). The fetus is frequently exposed to enteric bacteria during the course of maternal peripartal infections. In those instances in which the same agent was recovered from mother and infant, gram negative bacteria were predominant. Postnatally the infant may be exposed to Gram negative organisms through humidification apparatus, resuscitation equipment or articles used in daily care. Because of the neonates lack of normal flora at birth he/she may become heavily colonized with Gram-negative bacteria in sites where these agents are not normally found. The newborn infant has been shown to have a lack of serum bactericidins against Gram-negative bacilli. These antibodies against somatic or "O" antigens in Gram-negative bacteria are in the gamma-M fraction which is not passed transplacentally from mother to fetus. Another factor may be the recent widespread use of antibiotics both in the mother and the infant, which would tend to select out the relatively more resistant Gram-negative organisms. Hence; Gram-negative organisms are commonest cause of neonatal septicemia (Overall James, 1970). In present study, Gram positive organisms were found in 30.99% of total cases and consisted of *Staphylococcus aureus,* Coagulase negative *Staphylococcus species, Enterococcus fecalis* and *Streptococcus pneumoniae* (Table.1). Gram negative bacilli consisted of *Klebsiella pneumoniae, E. coli, Acinetobacter baumannii, Acinetobacter lwoffii, P. aeruginosa, Citrobacter freundii* and *Enterobacter aerogenes* (Table 9). *Klebsiella pneumoniae* (22.53%), *Pseudomonas aeruginosa* (21.12%) and *Acinetobacter species* (15.49%) were the common isolates from neonatal septicemia cases. *Klebsiella pneumoniae* (35.55%) was the predominant pathogen in early onset septicemia while Coagulase negative *Staphylococci* (34.61%) was the predominant pathogen in late onset septicemia cases (Graph 1). Hammerberg *et al.* (1992) stated that after the careful cleaning of vein puncture site, the growth of Coagulase negative *Staphylococci* (CONS) in blood culture of specimens of premature neonates indicates bacteremia rather than the skin contaminant in vast majority of cases.

All the Enterobactericeae isolates were sensitive to piperacillin+tazobactum and imipenem. Among the *Klebsiella pneumoniae* isolates, 56.25% were sensitive to ciprofloxacin and amikacin while there was complete resistance to ampicillin, amoxyclav, cefazolin, cephalothin, cefuroxime and cefoperazone (Table 2).

62.5% of the *Klebsiella* isolates and 20% of *E. coli* isolates showed ESBL production while there was no ESBL production in *C. freundii* and *E. aerogenes*. The high percentage of ESBL producing *Klebsiella* spp. may be due to selective pressure imposed by extensive use of antimicrobials. Intensive care unit in which antibiotic usage is heaviest and the potential for patient to patient transmission of organisms is greatest is an important factor for ESBL production (Jain *et al.,* 2007). In the present study, ESBL detection was done by two methods viz. double disc method and CLSI phenotypic confirmatory test. CLSI phenotypic confirmatory test method was able to detect 11 ESBL producers as compared to double disc method that detected 9 ESBL producers.

80% of *Acinetobacter baumannii* and *Pseudomonas spp.* were sensitive to piperacillin + tazobactum while 90% of *Acinetobacter baumannii* and 86.67% of *Pseudomonas spp*. were sensitive to imipenem, respectively (Table 3). Two isolates of Pseudomonas and one isolate of *Acinetobacter spp.* were tested for metallo-β-lactamase production. MBL was detected in one isolate of *Pseudomonas aeruginosa* while it was not detected in the isolate of *Acinetobacter baumannii*.

All the gram positive organisms were sensitive to vancomycin, pristinomycin and linezolid. Methicillin resistance was seen in all isolates of *Staph. aureus*. While it was seen in 33.33% isolates of coagulase negative *Staphylococcus spp* (Table 4). The high resistance rates found may be associated with the frequent use of antimicrobial drugs for both prophylactic and therapeutic treatment of hospitalized newborns (Loureiro *et al.,* 2002). A coordinated effort to limit inappropriate use of broad spectrum antibiotics, efficient hospital policies, vigilant detection of resistant species, rigorous surveillance and infection control protocols are needed to control the increasing incidence of antimicrobial resistant organisms.

CRP test was positive in 92.95% of culture positive cases. There was significant statistical association between culture positivity and CRP test.CRP test was positive in 64.34% of early onset septicemia cases and 35.66% of late onset septicemic cases (Table 5). Sensitivity and specificity of CRP test was 92.96% and 50.39% respectively. Foetal distress, hyperbilirubinaemia, intraventricular haemorrhage can result in elevation of CRP. So it should not be used as a sole indicator for starting or stopping of therapy but viewed in context of blood culture report, clinical findings and other laboratory studies (Datta *et al.,* 2006).

Mortality was 29.6% in early onset septicemic cases and 15.06% in late onset septicemic cases. There was significant statistical association between mortality and the age of onset of septicemia. The greater incidence of mortality in EOS may be due to lower host resistance, under weight babies, associated birth trauma and anoxia. The level of compliment in blood of newborn is less so also the immunoglobulins like IgM and IgA (Lokeshwar *et al.,* 1988)

Mortality was 46.47% in culture positive cases and 11.81% in culture negative cases. There was significant statistical association between mortality and culture positivity (Table 6). Mortality was more due to Gram negative bacteria (69.7%). *Klebsiella pneumomiae* had the highest mortality among the gram negative isolates (52.17%) and was responsible for 36 % of the total mortality (Table 7).

Table.1 Distribution of organisms with respect to early and late onset sepsis

Organisms	EOS (%)	LOS (%)	Total
Gram positive organisms	9 (20)	13(50)	22(30.99)
Methicillin sensitive Staphylococcus aureus	0	0	0
Methicillin resistant Staphylococcus aureus	1(2.22)	4(15.38)	5(7.04)
Coagulase negative Staphylococcus species	3(6.67)	9(34.61)	12(16.9)
Enterococcus fecalis	4(8.89)	0	4(5.63)
Streptococcus pneumoniae	1(2.22)	0	1(1.4)
Gram negative organisms	**36 (80)**	**13(50)**	**49(69.01)**
Klebsiella pneumoniae	16(35.56)	0	16(22.53)
Escherichia coli	3(6.67)	2(7.69)	5(7.04)
Acinetobacter baumannii	8(17.78)	2(7.69)	10(14.08)
Acinetobacter lwoffii	0	1(3.84)	1(1.4)
Pseudomonas aeruginosa	8(17.78)	7(26.92)	15(21.12)
Citrobacter freundii	1(2.22)	0	1(1.4)
Enterobacter aerogenes	0	1(3.84)	1(1.4)
Total	**45**	**26**	**71**

Table.2 Antimicrobial sensitivity of Enterobacteriacae isolates

Drugs	***Klebsiella pneumoniae*** **(n=16)%**	***E. coli*** **(n=5)%**	***Citro. freundii*** **(n=1)%**	***Entero. aerogens*** **(n=1)%**	**Total Enterobacteriaceae isolates (n=23)%**
Ampicillin	0	0	0	0	0
Amoxyclav	0	1(20)	0	0	1(4.34)
Cefazolin	0	1(20)	0	0	1(4.34)
Cephalothin	0	1(20)	0	0	1(4.34)
Cefuroxime	0	3(60)	0	0	3(13.04)
Cefoperazone	0	3(60)	0	0	3(13.04)
Cefotaxime	4(25)	3(60)	0	0	7(30.43)
Piperacillin	4(25)	4(80)	0	0	8(34.78)
Piperacillin+Tazobactum	16(100)	5(100)	1(100)	1(100)	23(100)
Imipenem	16(100)	5(100)	1(100)	1(100)	23(100)
Aztreonam	4(25)	2(40)	0	0	6(26.08)
Gentamicin	3(18.75)	2(40)	0	0	5(21.73)
Amikacin	9(56.25)	3(60)	0	0	12(52.17)
Tobramycin	7(43.75)	3(60)	0	0	10(43.47)
Kanamycin	9(56.25)	3(60)	0	0	12(52.17)
Ciprofloxacin	9(56.25)	3(60)	1(100)	1(100)	14(60.86)

Table.3 Antimicrobial sensitivity of *Acinetobacter spp.* and *Ps. aeruginosa*

Drugs	*Acinetobacter baumannii* n=10(%)	*Acinetobacter lwoffii* n=1(%)	*Pseudomonas aeruginosa* n=15(%)
Ceftazidime	2(20)	0	4(26.67)
Cefotaxime	2(20)	0	2(13.33)
Cefepime	2(20)	0	4(26.67)
Piperacillin	3(30)	0	5(33.33)
Piperacillin+Tazobactam	8(80)	1(100)	12(80)
Imipenem	9(90)	1(100)	13(86.67)
Gentamicin	4(40)	0	5(33.33)
Amikacin	6(60)	1(100)	12(80)
Tobramycin	6(60)	1(100)	6(40)
Ciproflaxacin	7(70)	1(100)	12(80)

Table.4 Antimicrobial sensitivity of Gram positive cocci

Drugs	*Staph. aureus* n=5(%)	*Coagulase negative Staph. spp.*, n=12(%)	*E.fecalis* n=4(%)	*S. Pneumoniae* n=1(%)	Total gram positive cocci, n=22(%)
Penicillin G	0	0	0	1(100)	1(4.45)
Cefoxitin	0	4(33.33)	-	-	4(18.18)
Erythromycin	0	3(25)	-	1(100)	4(18.18)
Gentamicin	3(60)	4(33.33)	1(25)	-	9(40.9)
Streptomycin	-	-	3(75)	-	3(13.63)
Amikacin	5(100)	8(66.66)	-	-	13(59.09)
Tobramycin	2(40)	8(66.66)	-	-	10(45.45)
Rifampicin	3(60)	5(41.66)	2(50)	1(100)	11(50)
Ciprofloxacin	2(40)	10(83.33)	2(50)	-	14(63.63)
Vancomycin	5(100)	12(100)	4(100)	1(100)	22(100)
Pristinomycin	5(100)	12(100)	4(100)	1(100)	22(100)
Linezolid	5(100)	12(100)	4(100)	1(100)	22(100)

Table.5 Correlation of CRP with blood culture positivity

Blood culture	CRP test		Total
	Positive (%)	Negative(%)	
Blood culture Positive	66(92.95)	5(7.05)	71(35.85)
Blood culture Negative	63(49.6)	64(50.4)	127(64.15)
Total	129(65.15)	69(34.85)	198

Probability of chance (p) = 0.000 (< 0.05)
Positive and negative predictive value of CRP test was 51.16% and 92.75% respectively
Sensitivity and Specificity of CRP test was 92.96% and 50.39% respectively

Table.6 Correlation of mortality with blood culture positivity

Blood culture	Mortality(%)	Survival	Total
Positive Blood Culture	33(46.47)	38(53.53)	71(35.85)
Negative Blood Culture	15(11.81)	112(88.19)	127(64.15)
Total	48(24.24)	150(75.76)	198

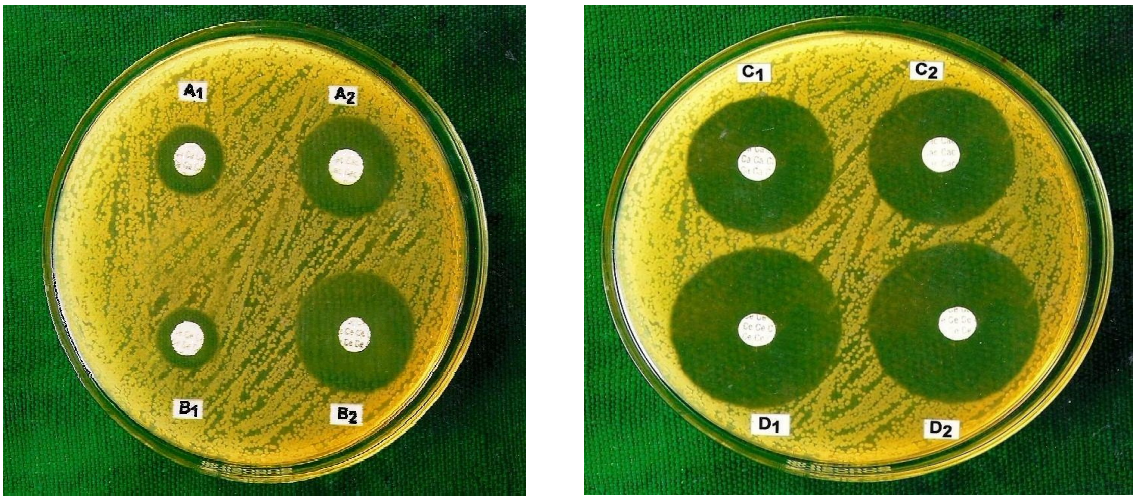
Table.7 Mortality due to the bacterial isolates

Organisms	Total (%)	Mortality (%)
Gram positive organisms	**22(30.99)**	**10(30.3)**
Methicillin sensitive Staphylococcus aureus	0	0
Methicillin resistant Staphylococcus aureus	5(7.04)	3(9.09)
Coagulase negative Staphylococcus spp	12(16.9)	5(15.15)
Enterococcus fecalis	4(5.63)	2(6.06)
Streptococcus pneumoniae	1(1.4)	0
Gram negative organisms	**49(69.01)**	**23(69.70)**
Klebsiella pneumoniae	16(22.53)	12(36.33)
Escherichia. coli	5(7.04)	3(9.09)
Acinetobacter baumannii	10(14.08)	5(15.15)
Acinetobacter lwoffii	1(1.4)	1(3.03)
Pseudomonas aeruginosa	15(21.12)	2(6.06)
Citrobacter freundii	1(1.4)	0
Enterobacter aerogenes	1(1.4)	0
Total	**71**	**33**

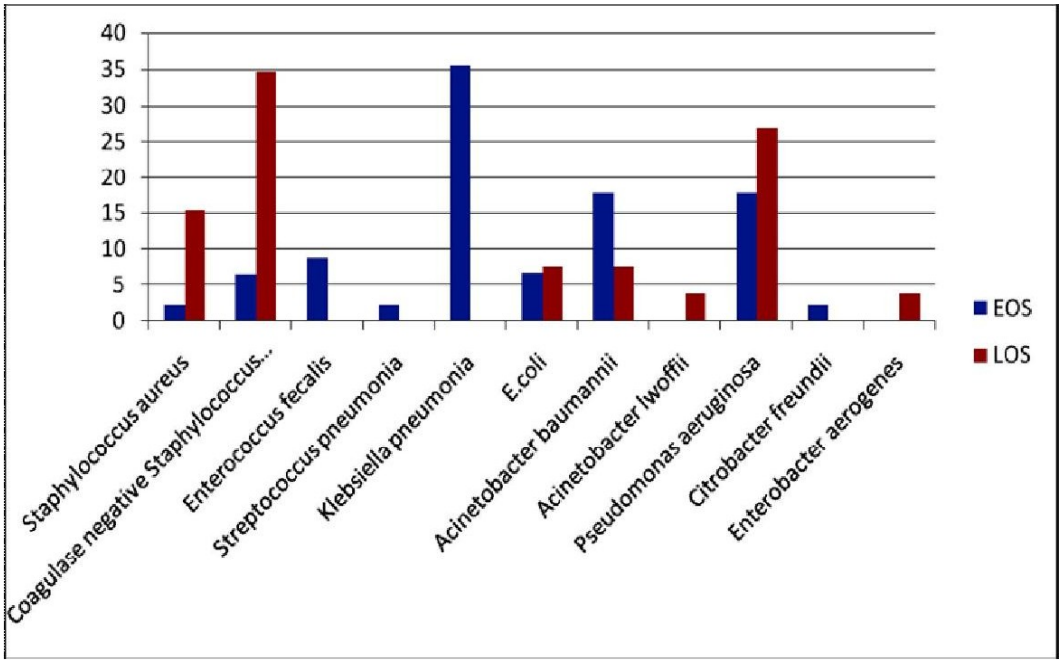
Figure.1 ESBL detection by CLSI method

ESBL Positive –A_2 and B_2 showing wider zone than A_1 and B_1.

ESBL Negative –C_1, C_2 and D_1, D_2 showing no significant difference in zone size.



Graph.1 Distribution of organisms with respect to early and late onset sepsis



One major factor in the high mortality rates in Gram-negative septicemia is probably the emergence of drug resistant strains of these bacteria against the commonly used antibiotics.

Male, preterm and low birth weight neonates were more prone for septicemia. Blood culture should be done in all cases of suspected septicemia prior to starting antibiotics. A good correlation between blood culture and CRP levels was found in the present study. Overall gram negative organisms were the predominant causative agents for neonatal septicemia. Mortality was more due to gram negative bacteria as compared to gram positive bacteria. There was maximum resistance to the commonly used cephalosporins and aminoglycosides among gram negative organisms. Routine ESBL detection should be made imperative and empirical use of third generation cephalosporins must be discouraged. Every hospital should monitor its antibiotic sensitivity pattern against the common isolates that can serve as a basis for empirical therapy in emergency conditions.

Acknowledgment

Author acknowledges the immense help received from the scholars whose articles are cited and included in references of this manuscript.

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