THE CLINICAL PROFILE OF NEONATAL LATE ONSET SEPSIS IN AN INTRAMURAL SETUP

Submitted in partial fulfillment for

M.D. DEGREE EXAMINATION

MD BRANCH VII

PAEDIATRIC MEDICINE

Stanley Medical College, Chennai

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU.

APRIL 2012

CERTIFICATE

Certified that this dissertation entitled **'THE CLINICAL PROFILE OF NEONATAL LATE ONSET SEPSIS IN AN INTRAMURAL SETUP'** is the bonafide work done by **DR.P.HAVINRAJA,** Post graduate student of Paediatric Medicine, Stanley Medical College Hospital, Chennai -1, during the academic year 2010 – 2012.

Prof.Dr.T.RAVICHANDRAN, M.D.,D.C.H.,
Professor of Paediatrics,
GOVT RSRM Hospital, Chennai.

Dr. AMBIGAPATHI, M.D,D.C.H.,
Professor and The Director I/C,
Institute of Social Pediatrics,
Stanley Medical College,
Chennai – 1.

Prof. Dr. R.SELVI, M.D.,
Dean I/C,
Stanley Medical College,
Chennai – 1.

DECLARATION

I Declare that this dissertation entitled **"THE CLINICAL PROFILE OF NEONATAL LATE ONSET SEPSIS IN AN INTRAMURAL SETUP"** has been conducted by me at Government RSRM hospital functioning under the Stanley Medical College. It is submitted in part of fulfillment of the award of the degree of M.D (pediatrics) for the April 2012 examination to be held under the Tamilnadu DR.M.G.R Medical University, Chennai. This has not been submitted previously by me for the award of any degree or diploma from any other university.

Place : Chennai

Date : **(Dr.P.HAVINRAJA)**

ACKNOWLEDGEMENT

I express my sincere thanks to **Prof. Dr.R.SELVI, Dean I/C,** Stanley Medical College for allowing me to conduct this study using the available facilities.

I am greatly indebted to Neonatology chief **Dr.T.RAVICHANDRAN, M.D.,D.C.H.,** Professor of Paediatrics, GOVT RSRM Hospital, for his unstinted guidance, encouragement, support given to me throughout the study.

I wholeheartedly thank **Dr.P. AMBIGAPATHY., M.D. D.C.H.,** Professor and The Director I/C, Institute of Social Paediatrics, Stanley Medical College for his valuable guidance and suggestions.

I am grateful to **Dr.G. KARUNAKARAN., M.D.D.C.H.,** Professor of The Institute of social Pediatrics, Stanley Medical College for his guidance.

I am grateful to **Dr. AMUTHA RAJESWARI., M.D.D.C.H.,** former Director, ISP, Stanley Medical College for allotting the study and for her invaluable guidance.

I am Grateful to **Dr .R .SELVI, M.D.,** former Head of the Department of Microbiology for helping me in my study and the support given by her department.

I thank the Assistant Professors of the Department of Pediatrics at Stanley Medical College Hospital, **DR.ANBU.M.D. D.C.H., Dr.A.ARAVIND., MD, D.C.H., DR. GANESH. MD., D.C.H., RATHNAVEL. MD, D.C.H, DR. ILANGO MD, D.C.H, DR. RATHIKA MD, and DR. KUMAR. D.C.H** for the help rendered.

I wish to express my sincere thanks to my postgraduate colleagues and ward staff nurses for their help and cooperation.

INDEX

INTRODUCTION

I. INTRODUCTION

Sepsis continues to be an important cause of neonatal morbidity and mortality. It is a common problem in the newborn intensive care unit (NICU) population particularly in premature neonates. National Neonatal Perinatal Database (NNPD)[1] 2003 has reported the incidence of neonatal mortality as 38 per 1000 intramural live births in tertiary care institutions and the incidence of sepsis was 3%. Invasive bacterial infections primarily involve the blood stream during the first month of life. That leads to meningitis, pneumonia and multiorgan dysfunction Septicemia was the commonest clinical category with an incidence of 2.1%. Meningitis was diagnosed in 0.3 per 1000 live births[1].

Neonatal sepsis is the common cause of neonatal mortality contributing to 23%[1] of all neonatal deaths. Neonates come from clean uncontaminated inutero environment to the environment contaminated with harmful micro organism. Newborn babies especially with low birth weight and very low birth weight are immunologically immature and vulnerable to acquiring infections.

Late onset sepsis comprises the majority in this population with high rate of morbidity, mortality, longer hospital stay and increased cost. Traditionally neonatologists attribute the prevalence of late onset sepsis in the NICU population to a combination of environment and host factors including immature neonatal immune system, a compromised skin barrier, the need for invasive procedures, the prolonged use of invasive life support apparatus such as endotracheal tubes and central venous catheters and prolonged hospital stay[2-6]. Irrational usage of antibiotics and prolonged

use of empirical antibiotics make the baby prone for Neonatal necrotizing enterocolitis (NNEC) and Late onset sepsis(LOS)[7]. Since in early stages of LOS the signs and symptoms are subtle or absent it is mandatory to profile the LOS.

Since the gold standard blood culture and sensitivity test takes 2 to 3 days to obtain the result, it is very essential to have a knowledge about the prevalence of organism in the NICU and the antibiotic sensitivity pattern to form an empirical antibiotic protocol. Thereby we can reduce the morbidity mortality and infant mortality rate. In addition, having knowledge about the maternal, neonatal and environmental risk factor will be useful to prevent LOS.

Government RSRM Hospital, functioning under Stanley medical college is a tertiary care centre and caters to a large population from North Chennai. Nearly 15000 deliveries are conducted per year with an average of 1200 per month. We admit about 300 neonates per month in the newborn unit with the major cause of admissions being respiratory distress, low birth weight, prematurity, birth asphyxia, jaundice and sepsis etc.

ETIOPATHOGENESIS

II. EPIDEMIOLOGY AND PATHOGENESIS

Infections with varying outcomes may be caused by pathogens acquired at different times during intrauterine and neonatal life. No agent and no matter how obscure should be ignored as a possibility, when initial probabilities do not seem to be likely. Infections can occur inutero (congenital infection), at the time of delivery (natal infection) and after birth but within the neonatal period (postnatal infection).

Newborn infections are unique for several reasons. Infectious agent can be transferred from mother to fetus or newborn by diverse modes. Newborn infants are less capable of responding to Infection because of one or more immunologic deficiencies, more so, when the infant is preterm and low birth weight. Coexisting conditions often complicate the diagnosis and management of neonatal infections.

The clinical manifestations of newborn infections vary and include subclinical infections, mild to severe manifestations of focal or systemic infections, and rarely congenital malformations resulting from infection in the first trimester. The timing of exposure, inoculums size, immune status and virulence of etiologic agent influence the expression of disease in a fetus or newborn infant. Wide variety of etiologic agents infect the newborn including bacteria, viruses, fungi, protozoa, mycoplasma and ureaplasma urealyticum

The maternal socioeconomic status, race and ethnic background may affect the propensity for infection for reasons not entirely clear. Prematurity is a major risk

factor for neonatal sepsis. A mother from poor socioeconomic status may have poor nutrition and housing and live in more crowded poor hygienic conditions.

NEONATAL FACTORS:

1. Prematurity[3, 4, 5] -Prematurity is the foremost risk factor for neonatal sepsis for both early onset and nosocomial infection. Preterm infants have even more significant deficiencies in immune defense than term neonates when compared to adults.
2. Gender -Male gender[2, 3] is an important risk factor for development of neonatal sepsis. Males are 2 to 6 times more likely than females to develop sepsis.
3. Exposure to certain medications such as steroids may cause risk to the neonate beyond the risk of broad – spectrum antibiotic use resulting in not only colonization but also with multiresistant organisms[7]. Immune deficiency syndromes can also pose great likelihood of causing neonatal infection.

ENVIRONMENT FACTORS:

Ordinarily the umbilical stump skin and the nasopharynx become colonized within a few days with the organisms in the nursery. Within first week of life, the gastrointestinal tract is also colonized. Among breast-fed infants, lactobacillus species combined with E.coli are found in the stool, whereas formula fed infants have a predominance of E.coli alone. In a nursery environment where antibiotic usage is common, the individual neonate is more likely to be colonized with

antibiotic-resistant micro-organisms, even if that infant has not been given broad-spectrum antibiotics.

Newborns are so fragile and tender. Their skin is easily broken and allows organism to enter the blood stream very easily. Intra venous cannulation without aseptic precaution or infusion of contaminated intravenous fluids, drugs and prolonged IV catheter in situ will allow the normal skin commensals to enter in to the blood stream producing sepsis.

Any procedure like exchange transfusion without aseptic precaution leads to entry of organism in the blood stream leading to late onset sepsis. In NICU, incubator, overhead radiant warmer, ventilator, and accessories, if not sterilized periodically, multidrug resistant organism will grow and chances of introduction of organisms in to the healthy newborn will be unavoidable.

Classification of the neonatal sepsis 1. Early onset sepsis -Onset of illness within 72 hours from birth. 2. Late onset sepsis -Onset of illness after 72 hours from birth and may extend to about 8-12 weeks.

EARLY ONSET AND LATE ONSET NEONATAL INFECTIONS:

The terms early onset and late onset infection refer to the age of onset of infection in the neonatal period and was originally derived arbitrarily as infections occurring before and after 72 hours of life respectively but it is more useful to separate early and late onset infections according to peripartum pathogenesis. Early onset infections are acquired before or during delivery. Late onset infections are

acquired after delivery in the post natal ward, normal newborn nursery, neonatal intensive care unit or the community. Now many books define early onset sepsis as that occurring from birth to 7 days and late onset sepsis with onset of 7 days to 8 to 12 weeks of life.

PATHOGENESIS OF LATE ONSET SEPSIS:

After birth, neonates are exposed to infectious agents in the nursery or in the community. Postnatal infections may be transmitted by direct contact with hospital personnel, the mothers or other family members. The most common source of postnatal infections in hospitalized newborns is contaminated hands of health care personnel.

CLINICAL FEATURES OF NEONATAL SEPSIS:

A neonate with sepsis may be asymptomatic or have signs and symptoms which may be subtle. The clinical manifestations may depend on whether the infant has Early or Late onset infection. Late onset disease is not usually associated with the risk factors noted with early onset disease and usually has an insidious onset with non-specific signs and may cause meningitis,osteomyelitis with or without bacteremia, septic arthritis,lymphadenitis,soft tissue infection, breast abscess, otitis media, endocarditis, pericarditis, purulent conjunctivitis,periumbilical erythema and induration[2,3,4]. Meningitis is most commonly present in late neonatal sepsis. So babies with late onset sepsis, the diagnostic lumbar puncture is beneficial in 95% of cases.

The septic infant may have temperature instability including hypothermia, hyperthermia or both. Ten percent of full term newborn with temperature more than 37.8^0 C has bacterial sepsis. Among afebrile newborns, the incidence of bacterial disease is 1:10000. Fever in the first hours of life is usually related to maternal fever. Newborns with fever and bacterial disease usually, but not always, have other symptoms suggestive of infection and they have to be evaluated. The infant may not look well, may feed poorly and may be lethargic and irritable. Refusal of feed is the most common complaint in late neonatal sepsis. Hypothermia, abdominal distention, tachypnea may be present. If a baby develops apnea after one week of life will raise the suspicion of sepsis. Infant may develop cardiovascular problems with tachycardia, bradycardia, supra ventricular tachycardia(SVT) and congestive cardiac failure.

Poor perfusion and peripheral cyanosis may be the signs of shock. Nonspecific generalized abdominal distension, vomiting, diarrhea, or ileus may be present, regardless of direct involvement of the gastrointestinal tract. Jaundice is present in approximately one third of septic infants and has been attributed to direct hyperbilirubinemia. Infant with direct hyperbilirubinemia should not be considered as an indicator of incipient sepsis, if it is the only manifestation of the problem.

Late onset jaundice with conjugated bilirubin more than 20% of total serum bilirubin may be seen in sepsis. Urosepsis has notably been associated with jaundice. Neurologically, septic infants may be lethargic or irritable. They may also develop jitteriness, hypotonia and seizure even in the absence of meningitis. A bulging

fontanels, sutural diastasis are important signs of sepsis. Typical meningeal sign like nuchal rigidity is rare in the neonates.

Petechiae may be seen in early stages of sepsis, whereas purpura, thrombocytopenia and disseminated intravascular coagulation(DIC) are more likely to occur late in the illness. Neonatal sepsis may localize in any organ system. Meningitis is common presentation in LOS with pneumonia, urinary tract infection, otitis, and peritonitis also do occur. An infant with decreased mobility of an extremity or swelling and erythema over a bone or joint may have osteomyelitis.

APPROACH TO DIAGNOSIS OF NEONATAL SEPSIS:

Since the clinical signs are subtle high index of suspicion is essential. The spectrum of severity of symptoms required to prompt a sepsis work up is a matter of clinical judgment and cannot be dictated by protocol alone. The practitioner must rely on careful maternal and newborn history, physical examinations and laboratory findings.

The goal is to identify and treat all infected neonates but to avoid unnecessary investigation and antibiotics in non infected neonates. The following are the common scenarios with which a newborn may presents with sepsis. Neonate may come from the nursery with clinical feature suggestive of sepsis. Neonate usually of low birth weight admitted in hospital and who develops a nonspecific signs (eg: apnea, feed intolerance, lethargy).

Prevalence of organism and their sensitivity vary from country to country and from hospital to hospital. In western country group B streptococcus(GBS) and E.coli are common. But the above is not true in India. Hence, their data may not be applicable to our country.

Since definitive data in India is lacking, an empirical approach has been recommended. So it is mandatory to have the knowledge about the organism in the nursery and their antibiotic sensitivity to start empirical antibiotic therapy. Investigations may be divided into specific diagnostic tests, nonspecific diagnostic tests and miscellaneous tests.

SPECIFIC DIAGNOSTIC TESTS:

1. Isolation of bacteria from blood or cerebro spinal fluid(CSF) is the standard method for diagnosis of neonatal sepsis. However, rate of proven plus highly suspected infection is undoubtedly greater than culture proven rate. Among the neonates with bacteremia and without antibiotics, vast majority of cultures are positive by 48hrs.
2. CSF culture
3. Urine culture
4. Detection of bacterial antigens
5. Non-specific diagnostic tests.

WBC and DC:

Of the nonspecific tests, neutropenia is the best predictor of sepsis. Neutropenia in a newborn with fever is highly suggestive of bacterial disease. Neutrophilia does not correlate well with neonatal sepsis. Ratio of immature to total neutrophil (I: T ratio) more than 0.2 is predictive of neonatal bacterial disease. It has a sensitivity of 82 to 90%. Neutrophil vacuolation and toxic granulation are also suggestive of bacterial infection. Presence of Dohle bodies[2] (aggregates of rough endoplasmic reticulum) which stains light blue with giemsa stain.

C- Reactive protein: A positive test will suggest the presence of sepsis has more negative predictive value. Value more that 1mg/dl is considered positive. It has a sensitivity and specificity of 87% and 83%. respectively.

Micro ESR: More than 15 mm at one hour is considered positive and would suggest sepsis.

MISCELLANEOUS TESTS :

1. Procalcitonin(PCT)[2] is a precursor protein of the hormone calcitonin which is produced in the plasma of the patients suffering from bacterial and fungal infections.. It is physiologically elevated during first 3 days of life. But it has been found to be reliable marker of late onset sepsis with the sensitivity and specificity of 100%. Quantitative measurement of PCT is performed, using an immunoluminometric assay with two monoclonal antibodies. After the initial surge of PCT during first 3 days of life, the mean normal serum PCT level is around 2ng/ml.

2. Other markers are serum IgM, Leucocyte alkaline phosphatatase, Fibronectin, Haptoglobin, Elastase, Alphaproteinase inhibitor levels, Limulus lysate[2] test for endotoxin detection and cytokinin like IL-1,IL-6[2]. They are unreliable when used individually, but may be helpful when used in combinations as part of septic screening.

ROLE OF BLOOD CULTURE :

It is the gold standard for the diagnosis of septicemia and should be done in all cases of suspected sepsis prior to starting antibiotics. Sterile acquisition by venipuncture of 0.5 ml or 1 ml of blood, plated in a standard soy broth or brain heart infusion agar and incubated for 5 days. A positive blood culture with a pathogenic organism is not only diagnostic of neonatal sepsis but also provides valuable information regarding prevailing organisms in a particular setting. Multiple samples taken from various sites can avoid false positive results due to contaminants.

Role of septic screen :

All newborns suspected to have neonatal sepsis should have a septic screen to corroborate the diagnosis of sepsis. A positive sepsis screen does not confirm sepsis but a negative screen strongly rules out sepsis. The various components of the septic screen include total leukocyte count, absolute neutrophil count, immature to total neutrophil count, micro-ESR and C- reactive protein. The above tests have been recommended as sepsis screen panel by National Neonatal Perinatal Database 2003.

1.SEPSIS SCREEN PANEL:

Components: Total leukocyte count <5000/ cubic mm of blood 2. Absolute neutrophil count <1800 3. Band forms >0.2 4. Micro ESR of > 15 mm in 1st hour 5. C-reactive protein of > 1 mg/dl. Presence of any abnormal two parameters in a screen have the sensitivity of 93%-100%, specificity of 83%, positive and negative predictive values of 27% and 100% in detecting sepsis.

2. The culture studies are time consuming. But the bacterial antigen can be identified by counter immune electrophoresis.

3. Buffy coat smear examination[2] involves staining of Buffy layer of the blood obtained after centrifugation and separation of plasma. If carefully done, pathogens can be identified in 57 to 70 percent cases of neonatal sepsis.

4. Suprapubic puncture to be done to rule out urinary tract infection. More than 10 leucocytes per mm in uncentrifuged urine and colony count of more than 10^4 is suggestive of pyelonephritis.

5. Chest x -ray to be taken to document pneumonia, while abdominal film will show ileus and features of necrotizing enterocolitis. Stool should be examined for occult blood. Urine metabolic screening to be done to rule out inborn errors of metabolism (IEM) since clinical features of IEM simulates late neonatal sepsis.

MANAGEMENT:

In any infant, suspected to have sepsis, antimicrobial therapy should be initiated immediately after completion of the diagnostic evaluation as progression of the disease is rapid and one cannot await confirmation from blood or other cultures. If the infant is unstable, antibiotic therapy should be initiated after obtaining a blood culture and the lumbar puncture should be done at a more appropriate time and place.

The duration of therapy is 10-14 days for gram positive organism and for gram negative infection the total duration of therapy is 21 days[3]. For meningitis the total duration of therapy is 21 days[3]. In late onset sepsis the baby will acquire the organism prevailing in the community or in the intensive care unit or in the nursery. Common organisms are gram negative organism like Klebsiella pneumonia, Enterobacteria, E.coli, Proteus, Citrobacter and Serratia, gram positive organisms like coagulase negative staphylococci (CONS) and staphylococcus aureus. Since most of the organisms are gram negative, staphylococcus aureus and albus, the initial antibiotic must cover this pathogen. Combination of amoxicillin-clavulanate with aminoglycosides or ceftizoxime and cloxacillin are effective against these organisms.

The aminoglycosides, including kanamycin, tobromycin, gentamicin and amikacin, and cephalosporin, including cefatoxime, caftazidime and ceftriaxone should be kept in reserve to treat the meningitis and life threatening illness.

Ciprofloxacin should be used as a last resort in critically sick neonates when bacterial isolates are resistant to all other antibiotics.

In nosocomial sepsis, antibiotics must cover staphylococci and the gram-negative bacilli, including pseudomonas. Most often naficillin or methicillin can be used to cover S.aureus. In nurseries where methicillin-resistant strains have emerged or where coagulase –negative staphylococci are frequent pathogens, vancomycin coverage is necessary. Ceftazidime and piperacillin provide adequate coverage for Pseudomonas.

- Granulocyte transfusions[2] and IV immunoglobulin have been used in neonatal sepsis but are not recommended for routine use. Exchange blood transfusion may help to improve the peripheral as well as pulmonary circulation. The procedure is recommended in critically ill neonate with sclerema, disseminated intravascular coagulation (DIC) and hyperbilirubinemia.
- Granulocyte infusion is suggested as an adjunct to immunotherapy for septic newborn with Neutropenia. Immunoglobulin preparation containing type specific antibodies to GBS may be beneficial.
- Fibronectin[2] in a septicemic neonate influences the neutrophil and macrophage response to infection.
- Colony stimulating factor can enhance the production and functional capabilities of granulocytes

REVIEW OF LITERATURE

III. REVIEW OF LITERATURE

Literature review is done to understand the current status of the clinical profile, causative organisms and their sensitivity pattern, laboratory findings, neonatal and maternal risk factors of neonatal sepsis and neonatal outcome in similar hospitals in India and abroad.

Bizzarro et al[6] found that shared environment like inutero, intrapartum and maternal factors influence late onset sepsis. Unshared environment like hospital stay, duration of IV catheter in situ, duration of antibiotics and mechanical ventilation also influence.

Genetic factors also contribute late onset sepsis by means of preterm low birth weight etc. Joseph.B. Catney et al[8] found that prolonged use of antibiotics or unnecessary empirical antibiotics leads to NNEC and LOS.

Up to 60% of hospital staff's uniforms are colonized with potentially pathogenic bacteria, including drug- resistant organisms. It remains to be determined whether these bacteria can be transferred to patients and cause clinically relevant infection.

According to NNPD 2003, neonatal mortality was 38 per 1000 live births. They also found that incidence of sepsis was 3% and incidence of pneumonia was 1.1% followed by meningitis with the incidence of 0.3%. They has shown that the incidence of culture proven sepsis reduced from 21% in 1995 to 15.8% in 2000-

2003. Male babies were more affected than female babies. Low birth weight babies and preterm babies were more prone for sepsis. Congenital pneumonia is more common in EOS which leads to respiratory distress and simulating respiratory distress syndrome. According to their study respiratory distress was the commonest presentation in EOS. They demonstrated the blood culture sensitivity of 55%.

The author found that Klepsiella pneumonia (32.5%) followed by Staphylococcus (13.6) as the common organism. As far as antibiotic sensitivity is concerned, NNDP found that Klepsiella was sensitive to amikacin, gentamycin and resistant to ampicillin.

Shasikala A Tallur et al[9] stated prematurity and LBW as the risk factors and she has shown that respiratory distress, jaundice, apnea as the common clinical features. In her study she has used total count, differential count, band form percentage, ESR, CRP, CSF examination and blood cultures for diagnosis for sepsis. She had 64.87 % cases of culture proven sepsis and she has shown Klebsiella pneumonia (49%) and pseudomonas (17.20%) as the most common organisms.

The author has shown that K.pneumonia showing high sensitivity to gentamicin (78%) amikacin (87%) and mild sensitivity to ampicillin (26%). She has shown an overall mortality rate of 47.5%.

Upadyay A et al[10] has found that 21.9 % had culture proven sepsis. He found Staph.aureus as the commonest organism followed by Klebsiella and Coagulase negative Staph.aureus (34%). He has shown that the overall mortality in the study group was about 34%.

Pneumonia was the most common presentation (66.7%) in a study conducted by Choko Betty et al[6]. The author has found 43% of the cases they studied had culture proven sepsis. She isolated Pseudomonas as the commonest organism followed by E.Coli and Klebsiella pneumonia. His study has shown a case fatality rate of 19.4 %. The author has concluded in her study that screening for sepsis in an asymptomatic neonate is warranted only in the presence of maternal risk factors even if the neonate is at high risk of developing sepsis due to associated problems of prematurity.

Kurien Anil Kuruvilla et al[12] has shown that E.coli was Sensitive to cefotaxime, gentamicin and amikacin. The author has shown 9.8% culture proven neonatal sepsis. He has found E.coli and Klebsiella as the commonest organism and he has shown in his study that the case fatality rate for sepsis was 14.4%.

Richard A.Polin et al[13] found that blood culture was positive in 40% and CONS was the commonest organism in late onset sepsis. Poor feeding and hypothermia were the common presentation in the study.

Bizzarro et al stated that CONS was the commonest organism followed by S.aureus, enterococcus and klebsiella pneumonia. He found low birth weight and prematurity as risk factor of septicemia. In their study the common presentation were poor feeding lethargy, poor reflexes, hypo or hyperthermia and abdominal distension.

Stuart E.Starr et al[7] stated that Imipenam with broadest spectrum of activity has great potential for the treatment of neonatal sepsis. It is active against all the gram negative, gram positive organisms, listerio monocytogens and anaerobes including

Bacterioid fragilis. Stuart E Starr et al found that culture proven sepsis was 41% and stated that CONS was the commonest organism in his study.

Anita Sharma et al[14] in their study found blood culture positivity in 20% cases. Bhutta ZA et al[15] ,Daoud AS et al[16], Chugh K,Aggarwal BB et al[17], Singel JD, McCraken GH et al[18] and Monga K Fernandez A et al[19] found Klebsiella as the commonest organism in their respective studies.

Ashok deori[20] in his article stated that Group B Streptococcus (GBS) contributed to only a small number of cases in our Indian population. Harton.JA, Hemming et al[21] have shown 12.4% of culture proven neonatal sepsis. Patrick S. Safford et al[22] found that CONS was positive in 15%.of sepsis

Avery[4] - Text book of newborn have given GBS as the commonest organism, Nelson[3] – Text book of pediatrics' has given respiratory distress as common presentation and Klebsiella pneumonia as the commonest organism.

Roberton[5]- Textbook of Neonatology has given K.Pneumoniae as the commonest organism. The mortality rate is 15% for Early onset sepsis and 9% for late onset sepsis

Baker et [23]al found feed intolerance and apnea are the commonest presentation.

Ashok Deora et al have shown that Klebsiella over years has reduced trend of antibiotic sensitivity to gentamicin, amikacin and cefotaxime. Staph.aureus has reducing trend of antibiotic sensitivity to amikacin, gentamicin, and cefotaxime.

Deepak chawla et al[24] in his article- 'Rational approach for diagnosis and management for sepsis' has highlighted factors like Low birth weight, prematurity for developing sepsis.

Piyush gupta et al[25] has found obstetric risk factors to be significantly high among affected neonates.

In NNF Journal, Vol 20 No 1 Jan-Mar 2006, it was mentioned that the blood culture as the gold standard for diagnosing sepsis. Recommendations were given for diagnostic lumbar puncture in the cases of sepsis. Lumbar puncture should be done in all the neonates with symptoms of meningitis, in symptomatic babies in whom sepsis is the probable diagnosis and in babies with positive blood cultures.

Urine culture becomes most appropriate while investigating for LOS. Suprapubic aspiration is the preferred route.

Total leucocyte count has low predictive value because of wide range of normal counts from 8000-20000/cu.mm. Leucopenia <5000 or lecocytosis > 15000 are usually associated with neonatal sepsis although many septic infants have higher counts giving this test a low sensitivity of 29% but reasonable specificity of 91%. CRP has shown to have higher sensitivity and Negative predictive value.

The sensitivity increases from 48-63% when a single test is performed to 84% -90% when multiple CRP screen are performed over 24-48 hrs following onset of symptoms. They also stated that CRP is a better indicator of infection than the WBC indices.

William E Benitz et al[27] has shown that serial serum CRP levels are useful in the diagnostic evaluation of neonates with suspected infection.

Robyn L.Rodwel et al[28] stated about early diagnosis of neonatal sepsis using haematological scoring system. It was formulated that assigns a score of 1 for each of seven findings:

i. Abnormal total leukocyte count
ii. Abnormal total neutrophil count
iii. Elevated immature PMN count
iv. Elevated total to immature ratio
v. Immature to total mature PMN ratio > 0.3
vi. Platelet count <150000
vii. Pronounced degenerative changes in PMN.

Top feeding is associated with risk of contamination and infant is denied humoral, cellular and other anti-infective factors available in the breast milk.

Excessive administration of vitamin E and early supplementation of iron to preterm babies is also associated with increased risk of bacterial infection.

TYPES OF INFECTION

The infection may remain superficial and localized or the baby may develop fulminant and disseminated systemic infection. Majority of the superficial infections are caused by gram positive organisms, Staphylococcus aureus (SA) and albus. The infection is conveyed to the baby through contaminated hands of nurses, doctors and relatives who may be nasal carriers of SA. Due to inability on the part of the neonate to develop sufficient inflammatory response to localize the infection, leads to sepsis.

Superficial infection may be pyoderma, conjunctivitis, umbilical sepsis and oral thrush.

OBJECTIVES OF THE STUDY

IV. OBJECTIVES OF THE STUDY

1. To study the babies present with strong clinical evidence of sepsis with positive rapid septic screen test and positive blood culture.
2. To study the babies with strong clinical evidence of sepsis and positive rapid septic screen test.
3. To study the babies with strong clinical evidence of sepsis with negative rapid screen test and negative blood culture
4. To study the correlation between maternal factor and late onset sepsis.

MATERIALS AND METHODOLOGY

V. MATERIALS AND METHODOLOGY

This study was undertaken in newborn unit Govt.RSRM Hospital functioning under Stanley Medical College Hospital during the period of October 2010 to September 2011. This is a tertiary care unit which caters to a large area of population from north Chennai and peoples from borders of Andhra pradesh.

The unit functions as an intramural unit round the clock. On an average of about 300 neonates are admitted every month in the newborn unit for various reasons. The study was commenced after getting the formal approval of the ethical committee of our hospital. The study population includes all the babies admitted in the neonatal ward with the history and clinical features suggestive of sepsis.

INCLUSION CRITERIA:

Babies between the age group 72 hours to 28 days of life who were born to booked and immunized mother with the gestational age of more than 28 weeks and birth weight of more than 1 kg with strong clinical evidence of sepsis like lethargy, refusal of feeds, hypothermia, seizures, respiratory distress and abdominal distention.

EXCLUSION CRITERIA:

1. Age less than 72 hours

2. Severe birth asphyxia with (Hypoxic ischemic encephalopathy(HIE) stage 2 and 3
3. Major intra cranial injury
4. Major congenital anomalies
5. Babies born with meconium stained liquor / foul smelling amniotic fluid
6. Newborn with the history of prolonged labour
7. Prolonged premature rupture of membrane(PPROM) more than 18 hours
8. Intrapartum fever
9. Mother with bad obstetric history
10. Mother with acute active infection

METHODS

1. All the newborns who were apparently normal since 72 hours of life admitted with the clinical features of sepsis and the new born admitted in the NICU for various causes apart from exclusion criteria developed signs of sepsis were enrolled. After getting informed consent from the parents, detailed clinical history, r feeding history, any BCRP (bad child rearing practices), general condition of the mother and the attendees were documented. A detailed history about the complaints were sought and documented in a clinical proforma. Thorough clinical examination of the baby were made and documented.

2. Blood was collected from the babies and sent for complete blood count, rapid septic screen test, liver function test, renal function test and blood culture and sensitivity.

3. All the babies with sepsis who were stable, a diagnostic lumbar puncture were done and the same were sent for biochemistry, gram staining, cell count, culture and sensitivity.

4. Urine routine examination and culture.- Urine were collected by suprapubic puncture and the same sent for culture and sensitivity

5. X-ray chest and abdomen

6. As blood culture would be reported only after 48-72 hours the following investigations were considered important. Total WBC count <5000/cmm of blood, Band Form ratio >0.2, Absolute neutrophil count <1800/cmm of blood, CRP>1mg/dl, Micro ESR>15mm/hr.

7. Other supportive investigations like blood sugar, calcium, serum electrolyte profile were routinely done.

8. The babies were then initiated on empirical antibiotics which would be later modified based on blood culture and antibiogram report. All the babies were carefully followed for the appearance of any new signs.

9. Babies less than 1 week of life were renamed as extended early onset sepsis. Because of the concept of maternal risk factors may influence sepsis even after 72 hours of life.

10. In above cases, all the mothers of these particular babies were examined and they were classified as symptomatic and asymptomatic.

11. A high vaginal swab done with the help of obstetrician in all the symptomatic as well as asymptomatic mother and the same sent for culture and sensitivity to look for any correlation between the maternal culture and baby's blood culture.

12. Similarly blood was collected by venipuncture from symptomatic mother sent for culture and sensitivity.

13. urine was collected by mid stream clean catch method and sent for culture and sensitivity. It was performed for all the mothers. Immunological status was documented.

Based on the clinical features and results obtained from the investigations babies were classified for analytical purpose into three groups as those with

1. Proven sepsis 2. Probable sepsis 3. Clinical sepsis

DEFINITIONS:

Proven sepsis - They are neonates where bacteria were isolated from blood, cerebrospinal fluid, or urine.

Probable sepsis - They are neonates where clinical and laboratory findings are consistent with bacterial infection but without a positive culture.

Clinical sepsis - They are neonates where diagnosis is made only on clinical profile of sepsis.

After discharge the babies were followed up in our high risk clinic. The statistical analysis in this study was performed by using SPSS software version 16. Package. Statistical tools such as chi-square test and independent t-test were used in the analysis. P-value of <0.05 is considered statistically significant.

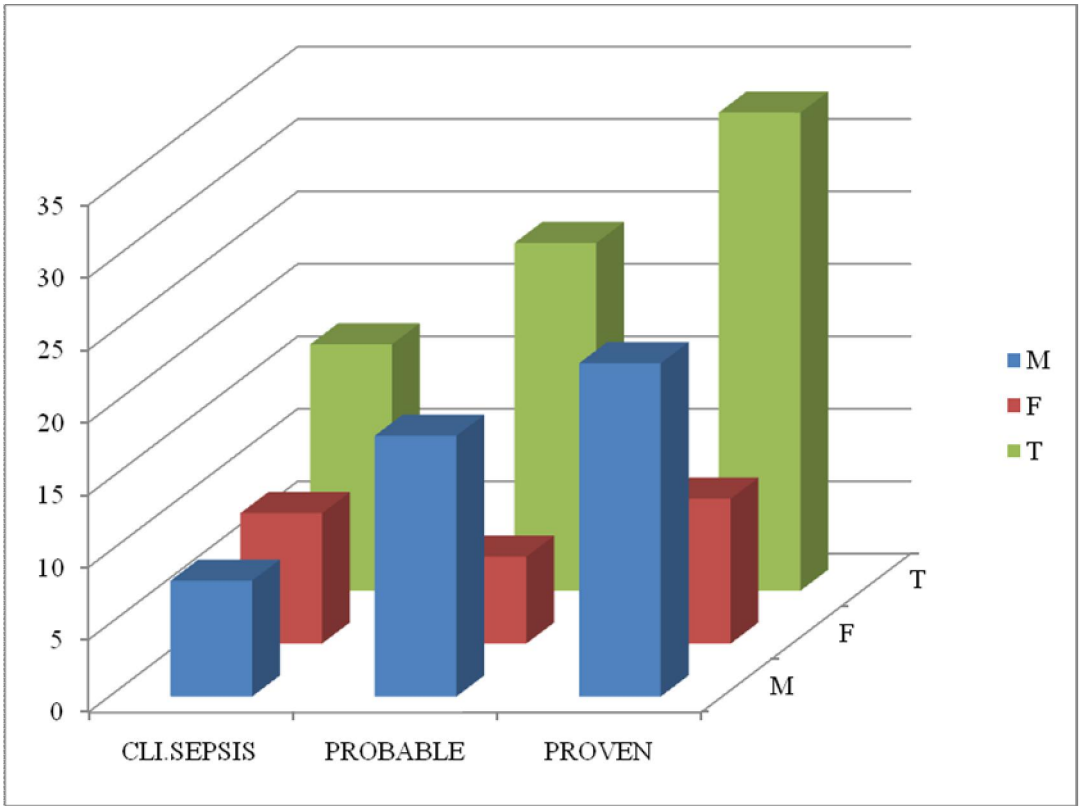
OBSERVATION AND RESULTS

VI. OBSERVATION AND RESULTS

Seventy four neonates with suspected sepsis admitted in the new born ward were enrolled in the present study. Based on the sepsis screen results, the babies were categorized into 3 groups.

Figure-1

CLASSIFICATION OF NEONATAL SEPSIS



For study purposes all the babies with sepsis were classified in to clinical sepsis, probable sepsis and culture proven sepsis. The above bar chart shows that

among the 74 neonates in our study 33 neonates had proven sepsis, i.e. culture positive. 17 cases had clinical evidence of sepsis, 24 had probable sepsis.

TABLE-1

EXTENDED EARLY ONSET SEPSIS VS LATE ONSET SEPSIS

S.No.	TYPE OF SEPSIS	MALE	FEMALE	TOTAL
1.	EEOS	33	18	51
2.	LOS	17	6	23
	TOTAL	50	24	74

Based on the age of onset of symptoms the neonates were classified as early extended onset of sepsis and late onset sepsis *(*72 hours of life to 7 days of life) and late onset sepsis (LOS) when the age at presentation was >7days of life. Table 1 shows the proportions of Extended early onset sepsis and Late onset sepsis in the present study. Table -1 shows that of the total number of cases is 74.Of which extended early onset sepsis is 51 and 23 cases is Late onset sepsis.

TABLE-II

NICU Vs POSTNATAL WARD

S.No	NICU	POSTNATAL WARD
1.	**38**	**36**

AGE DISTRIBUTION

In the present study; out of 74 babies, 12 babies with EEOS and 5 babies with LOS were found to have clinical sepsis. 18 babies with EEOS and 6 babies with LOS were found to have probable sepsis. 21 babies with EEOS and 12 babies with LOS were found to have proven sepsis. It is elaborated in figure-2

Figure-2

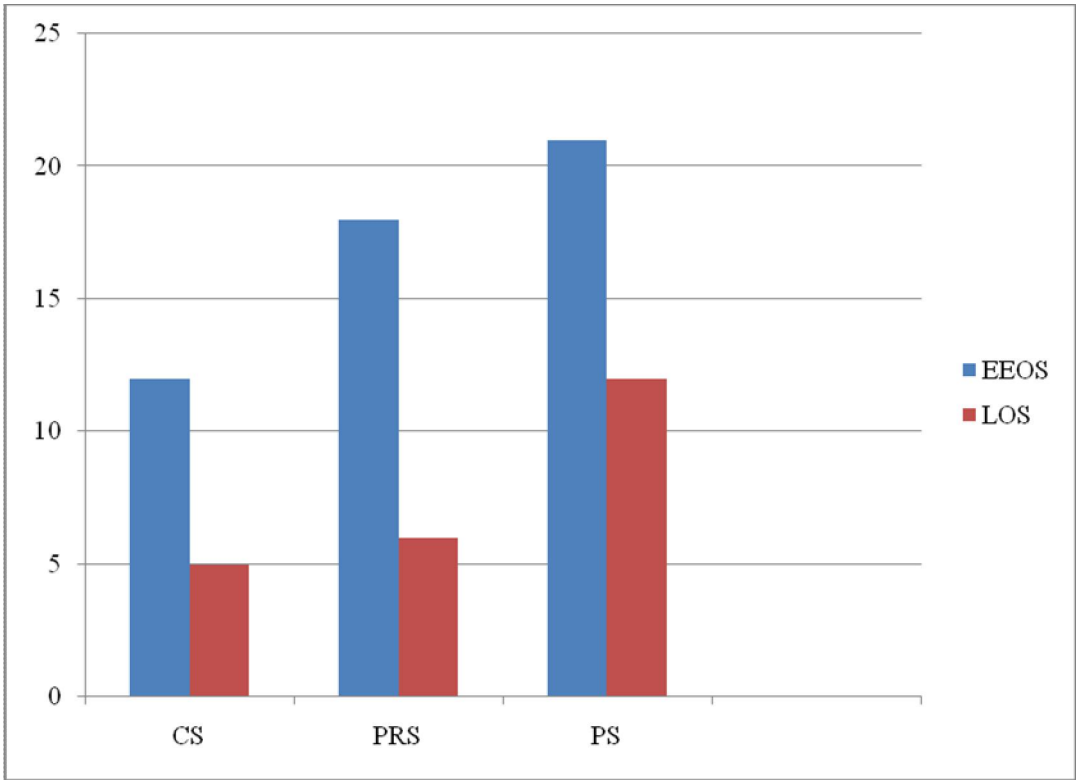
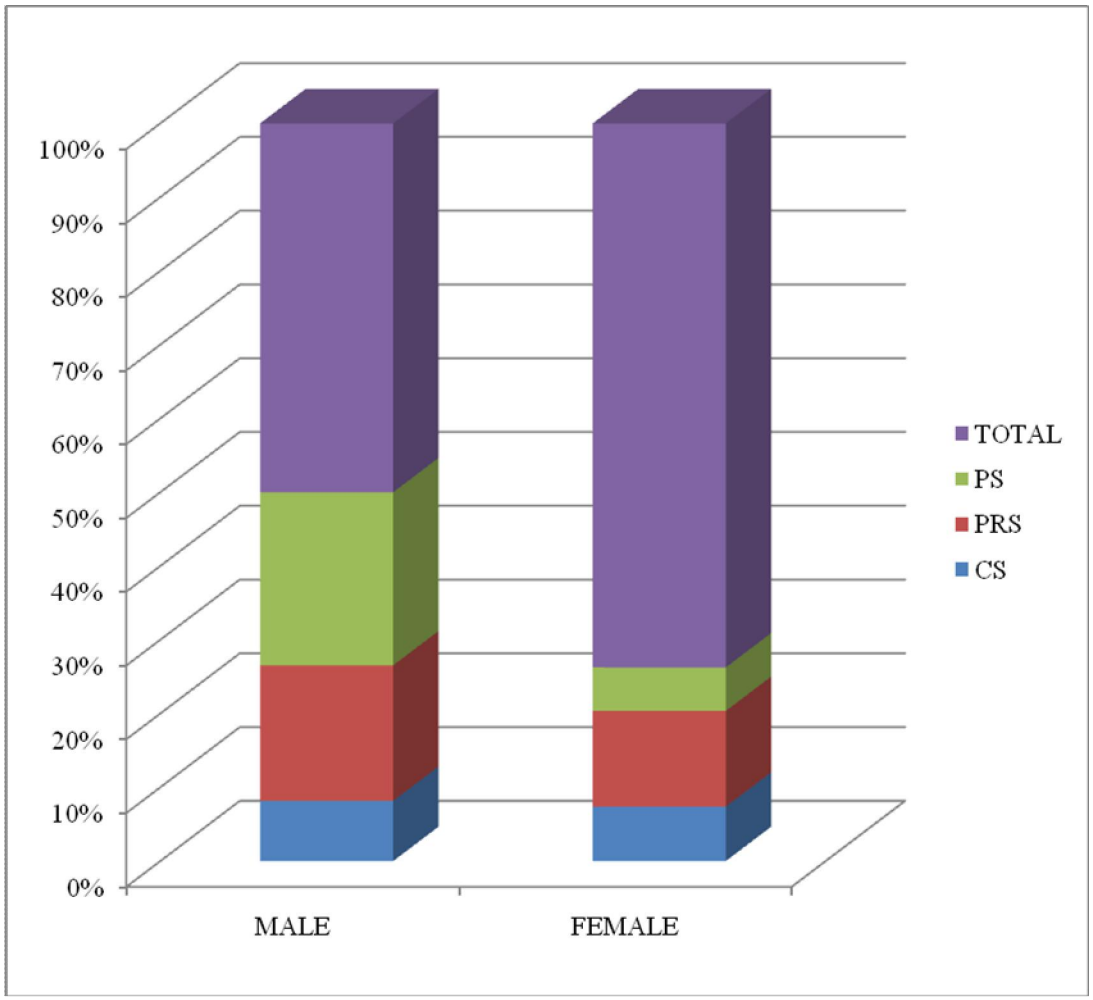


Figure-3

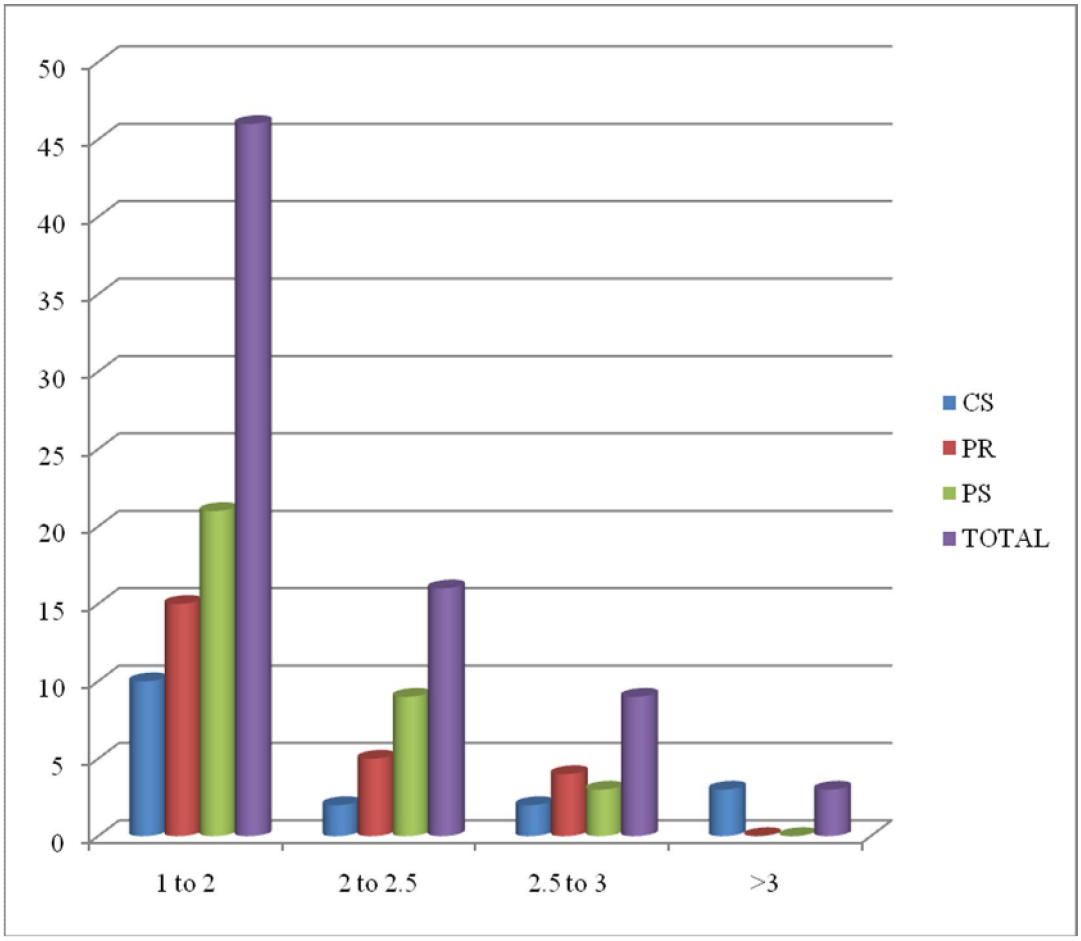
SEX DISTRIBUTION



Regarding prevalence of sepsis in sexes, male neonates found to have been affected more than female neonates. In our study 8 male babies and 9 female babies were found to have clinical sepsis. 18 male babies and 6 female babies had probable sepsis. 23 male babies and 10 female babies had culture proven sepsis.

Figure-4

BIRTH WT VS SEPSIS

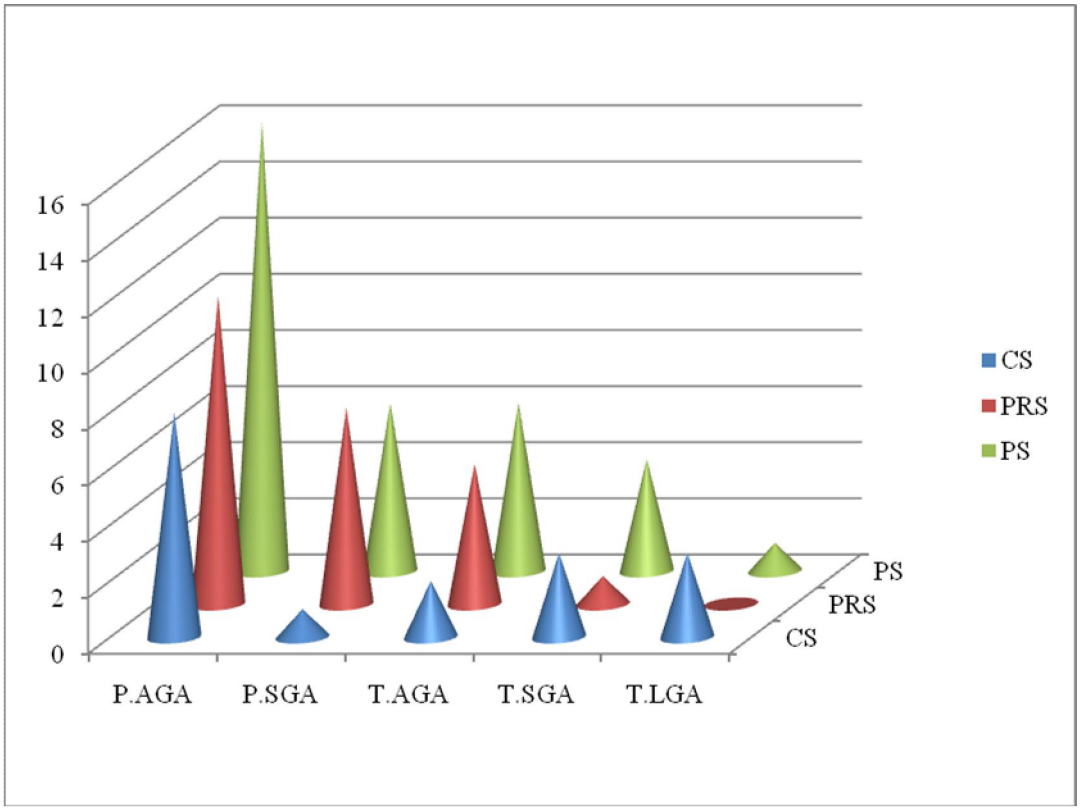


Birth weight is an important risk factor for the sepsis. Birth weight is inversely proportional to sepsis, especially late onset sepsis. In the study 46 babies with the birth weight of 1 to 2 kg found to have sepsis, 16 babies with the birth weight of 2 kg to 2.5 kg were found to have sepsis, 9 babies with the birth weight of 2.5 kg to 3 kg were found to have sepsis and 3 babies with birth weight of more than 3 kg were found to have sepsis. Figure -4 shows the distribution of various birth weights with various groups of sepsis

Figure-5

GESTATIONAL AGE VS

SEPSIS



Preterm babies are more prone for sepsis. In our studies 35 babies were preterm AGA, 14 babies were preterm SGA, 13 babies were term AGA, 8 babies were term SGA and 4 babies were term LGA. The data are given in the above chart.

Babies were brought to NICU with various symptoms and signs suggestive of sepsis. Symptoms like poor feeding, irritability, abdominal distention, convulsion and respiratory distress. Poor feeding were found in 53 (71.6%) babies. Respiratory distress was observed in 33(44.5%) babies. The percentage of various signs and symptoms are given in the following tables.

TABLE:III

CLINICAL FEATURES

S.NO	VARIABLES	n	PERCENTAGE
1.	Poor Feeding	53	71.6
2.	Respiratory Distress	33	44.5
3.	Lethargy	27	36.4
4.	Cold To Touch	26	35.0
5.	Abdominal Distention	18	24.3
6.	Vomiting	17	22.9
7.	Loose Stool	13	17.5
8.	Irritability	8	10.9
9.	Fever	8	10.8
10.	Shrill Cry	6	8.1

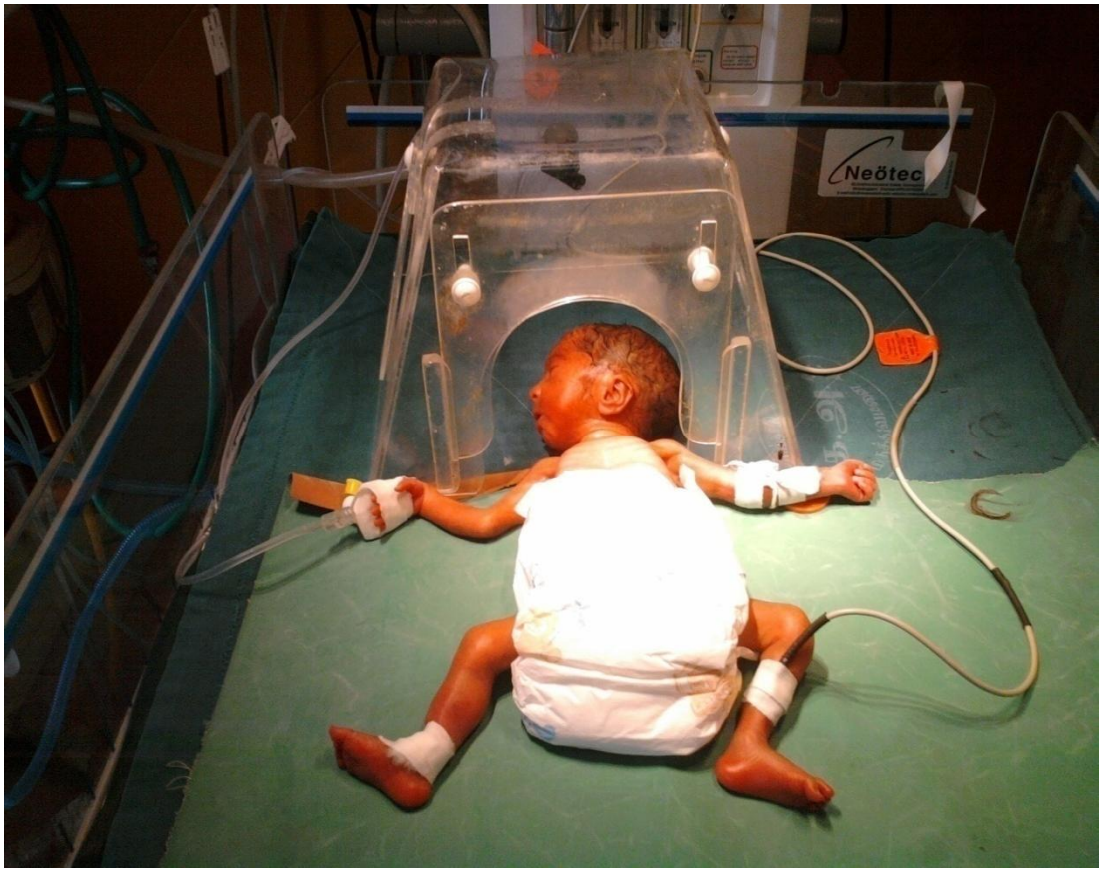
TABLE-IV

CLINICAL SIGNS

S.NO	Variables	n	Percentage
1.	Jaundice	37	50.0
2.	Respiratory distress	33	44.5
3.	Pallor	32	43.2
4.	Pulse	29	39.1
5.	Thrombocytopenia	28	37.8
6.	Cyanosis	25	33.7
7.	Apnea	23	31.0
8.	Prolonged CRT	23	31.0
9.	Abdominal distention	22	24.3
10.	Convulsion	17	22.9
11.	Pustules	9	12.1
12.	Indurarations	9	12.1
13.	Abscess	8	10.8
14.	Arthritis	6	8.1
15.	Bleeding tendency	4	5.4
16.	SVT	1	1.3

Figure-6

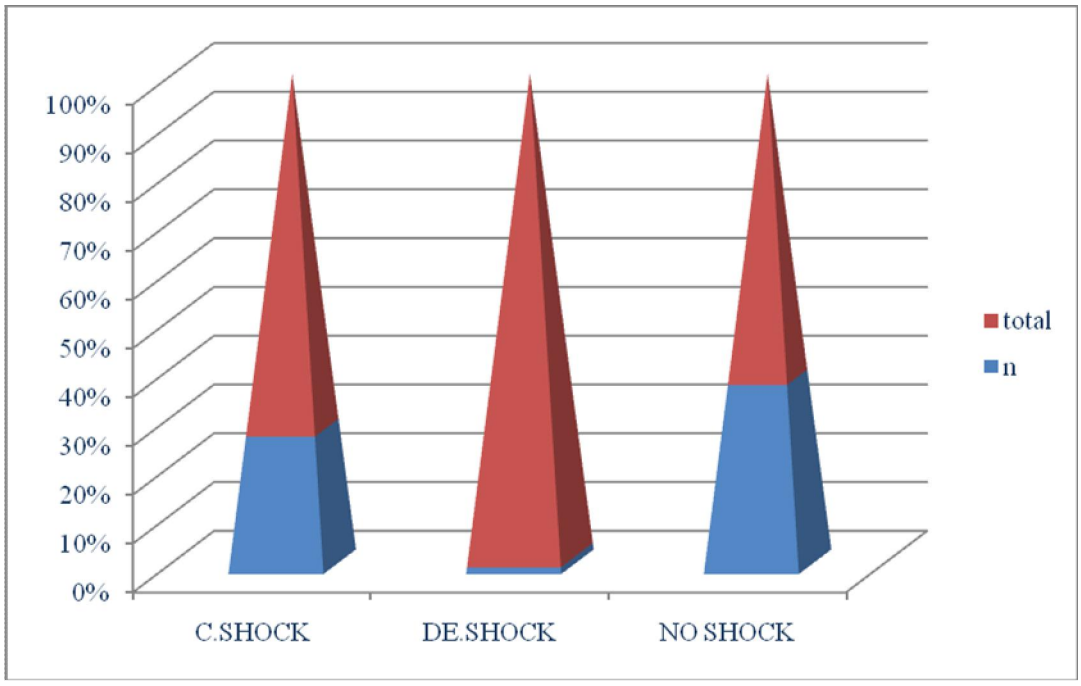
PRETERM BABY WITH SEPSIS



BABIES WITH SHOCK

Babies with septic shock carries worst prognosis. In our studies 29 babies found to have compensated shock, 1 baby found to have decompensated shock and 44 babies were not having any evidence of sepsis. The details are given below in the pyramid chart.

Figure-7



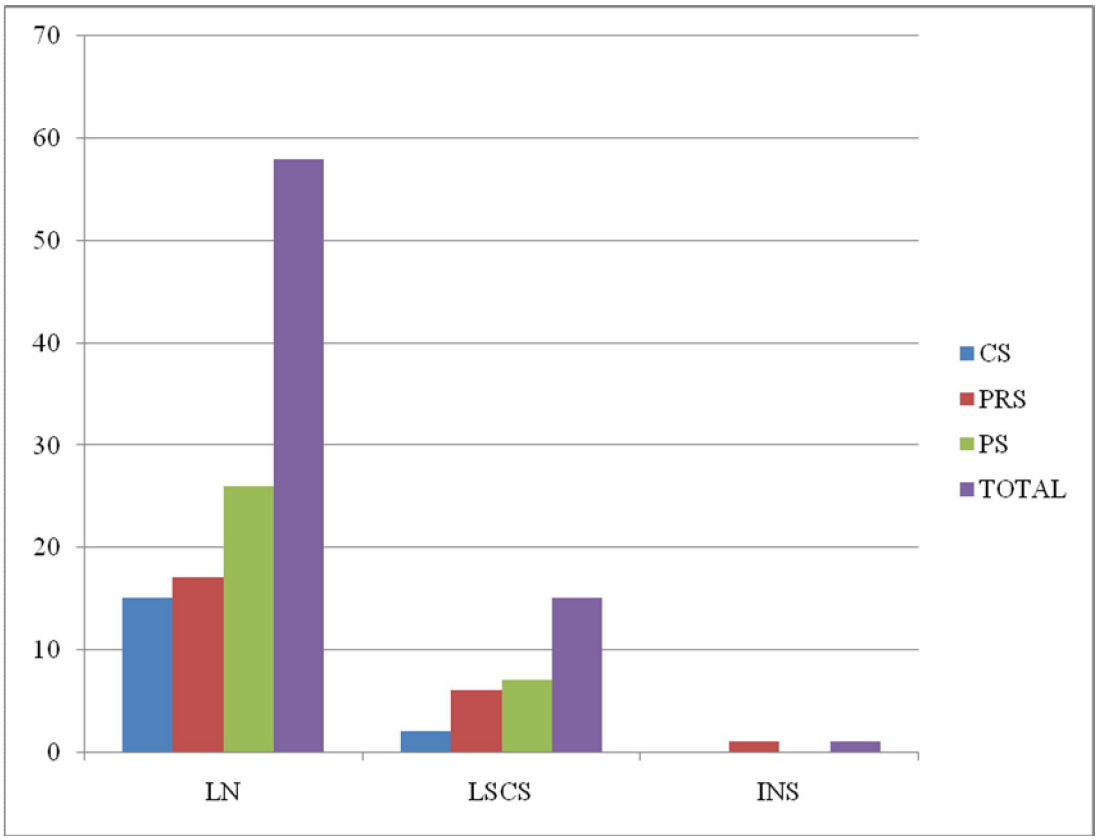
It has been noted that many babies initially presented with tachypnea tachycardia, and abdominal distention progressed on to have respiratory failure, shock, and finally succumbed.

MATERNAL RISK FACTOR:

In the present study group, 73 out of 74 mothers were booked and were having regular antenatal check up in the nearby primary health centre or urban health post or by GOVT RSRM hospital itself. In our study all the babies were born at GRSRM hospital. Out of 74 babies 58 babies were delivered by labour natural, 15 babies were delivered by LSCS due to various reason and one baby was delivered by outlet forceps. The details are given in figure-8

Figure-8

NATURE OF DELIVERY VS SEPSIS



Maternal factors like PPROM, prolonged labour, chorioamnionitis, and maternal fever are major risk factor for early onset sepsis. Unlike EOS maternal risk factor will not contribute much for late onset sepsis. But in this study, I looked for maternal contributions. Since any sepsis less than 7 days of life is called extended early onset sepsis and mother also present in the environment and handling the baby, demonstration of maternal risk factor can be beneficial. In this study 51 babies developed sepsis between 72 hours of life to 7 days of life. So their mother were examined thoroughly and looked for any symptoms of UTI, respiratory tract infection fever, wound infection and foul smelling lochia. They were classified into symptomatic and asymptomatic accordingly.

MATERNAL CONDITIONS

Mothers of extended early onset sepsis : 51

1. Mothers with symptoms : 19
2. Mothers without symptoms : 32

Blood was collected from the symptomatic mother and urine sample from both symptomatic and asymptomatic mother and high vaginal swab were sent for culture and sensitivity from both symptomatic and asymptomatic mother. I looked for any correlation between maternal organism and neonatal organism clinically and statistically. The results are given in the table-V

TABLE-V

MATERNAL INVESTIGATIONS

S.no	Investigation	Symptomatic	Asymptomatic
1.	Blood c/s	0/19	NIL
2.	Urine c/s	6/19	9/32
3.	High vaginal swab	5/19	5/32

Group B streptococci were detected in 2 symptomatic and in one asymptomatic mothers. E.coli was detected in 1 symptomatic and in 3 asymptomatic mothers. Enterobacter were detected in 2 symptomatic and 1 asymptomatic mothers. The details are given in the table-VI

TABLE-VI

HIGH VAGINAL SWAB STUDY

S.no	Organism	Symptomatic	Asymptomatic
1.	E.coli	1/19	3/32
2.	GBS	2/19	1/32
3.	Enterobacter	2/19	1/3

Urine was collected from both symptomatic and asymptomatic mothers. 6 symptomatic mothers and 9 asymptomatic mothers showed positive urine culture.

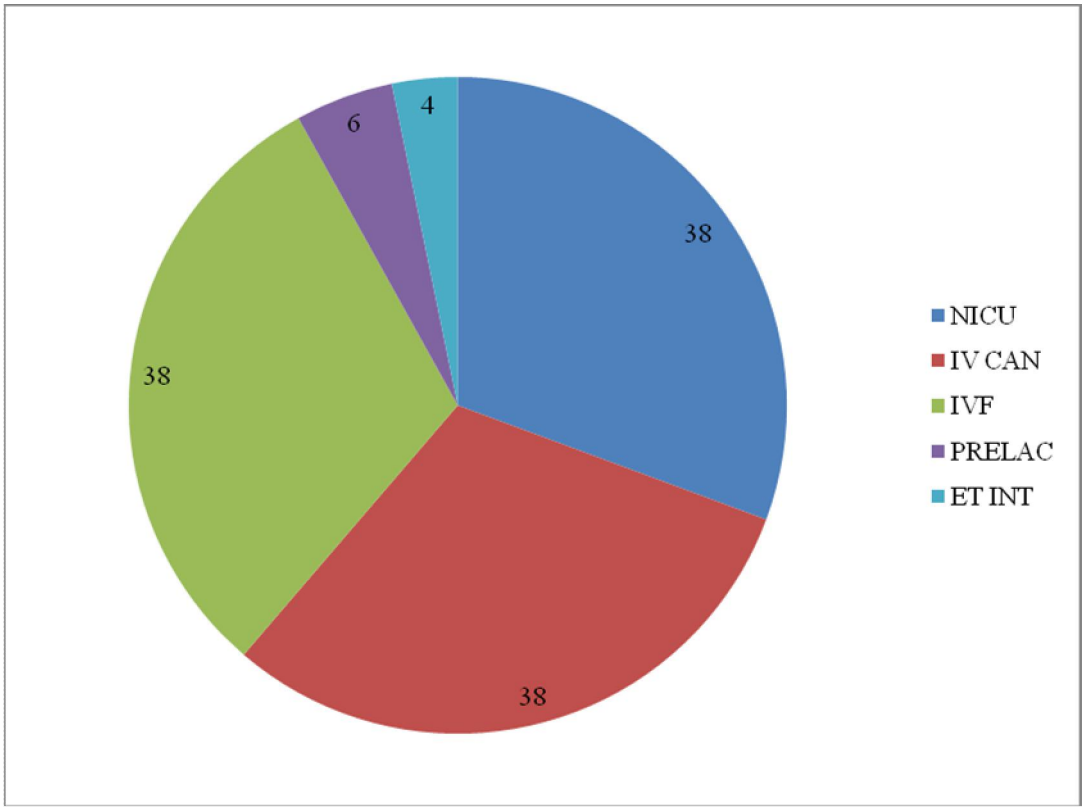
The commonest organism were Kleb.Pneumonia followed by Proteus, Pseudomonas and Enterobacter.

ENVIRONMENTAL FACTORS:

Environment is very important factor in causing neonatal sepsis. If the environment (nursery, NICU) is unhygienic or contaminated with organism, there are more chances of acquiring organism and sepsis. In this study I studied about some environmental factors and looked for any relation between risk factor and sepsis.

Figure-9

NICU STATUS



Out of 74 babies, 38 babies were hospitalized in our NICU for preterm and low birth weight management. 12 babies developed sepsis on the 4th day of life, 15 babies from 4 to 7 days of life and the remaining 11 were developed sepsis after 7 days of life. Babies with the weight of 1 to 1.5 and maturity of 28 to 30 weeks developed sepsis earlier than the remaining babies. One baby with prelacteal feed was reported.

MARKERS OF SEPSIS:

Babies admitted with symptoms and signs suggestive of sepsis were subjected to rapid septic screening test(RSS). The details of RSS in our studies were explained in the following table -VII

TABLE-VII

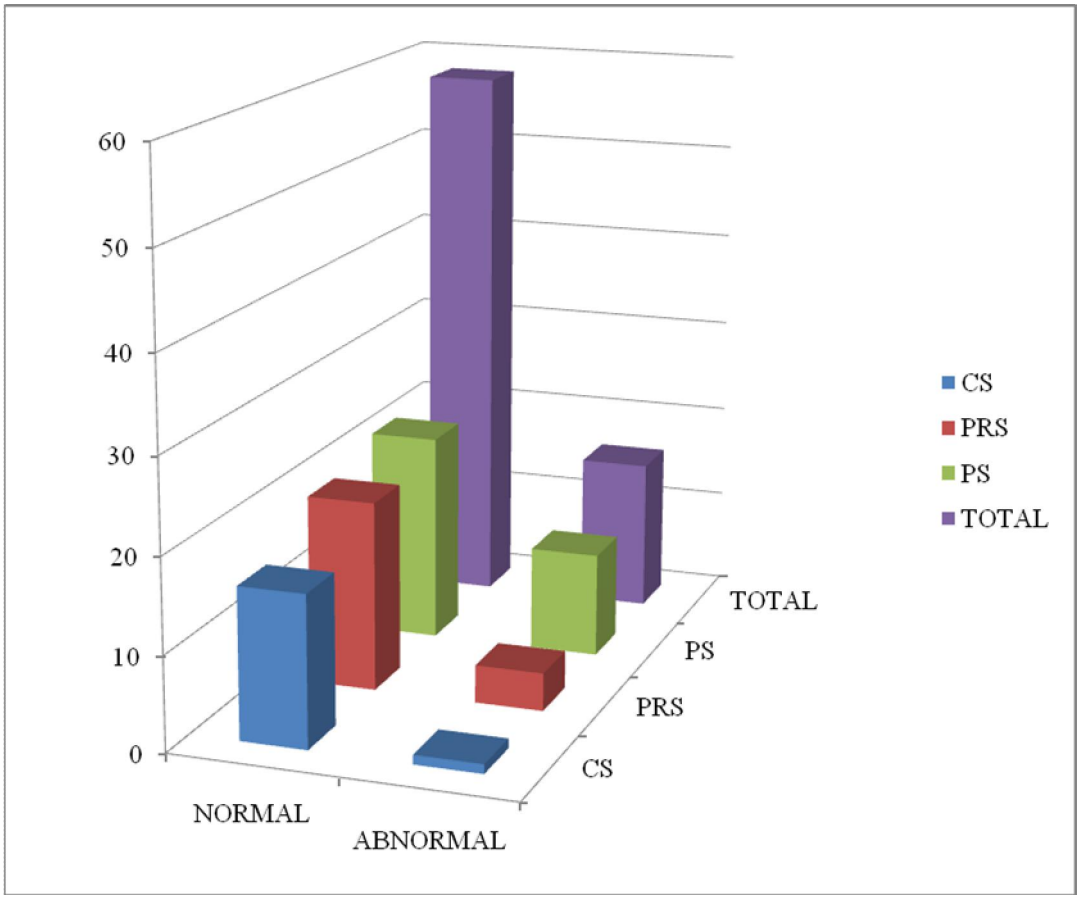
RAPID SEPTIC SCREENING TEST

S.NO	TEST	VALUE	N	TOTAL	%
1.	TC	<5000	29	74	39.1
2.	ANC	<1800	24	74	32.4
3.	I:T	>0.2	24	74	32.4
4.	MICRO ESR	>15mm/hr	56	74	75.6
5.	CRP	>1mg/dl	57	74	77

CSF analysis was done for all the 74 babies. Out of 74 babies 16 babies had abnormal biochemical findings (increased protein, decreased CSF sugar) and increased cell count favour of meningitis. One baby with clinical sepsis, 4 babies with probable sepsis and 11 babies with culture proven sepsis had abnormal CSF biochemistry and cell count.

The distribution is given in figure-10

Figure-10



CSF culture was positive in 2 babies with proven sepsis. CONS were reported in all the 2 cases. Distribution is given in table-VIII.

TABLE-VIII

MICROBIOLOGY OF CSF

S.NO	ORGANISM	N	%
1.	CONS	2	2.7

Urine analysis and urine culture were also performed in all the neonates. E.Coli and Kleb.pneumonia were the most common organism reported. The details are given in the following table-IX

TABLE-IX

URINE CULTURE n-5

S.NO	ORGANISM	3 TO 7 DAYS	>7 DAYS
1.	KLE.PNEUMONIAE	0	2
2.	E.COLI	0	1
3.	PROTEUS MIRABILIS	0	1
5.	CONS	0	1

Figure-11

BLOOD SAMPLE FOR CULTURE AND SENSITIVITY

TABLE-X

BLOOD MICROBIOLOGICAL PATTERN

S.NO	ORGANISM	3TO7 DAYS	>7DAYS	TOTAL
1.	CONS	9/51	5/23	14/74
2.	KLEP.PNEUMONIAE	6/51	2/23	8/74
3.	S.AUREUS	2/51	1/23	3/74
4.	PROTEUS	1/51	1/23	2/74
5.	PSEUDOMONAS	2/51	0/23	2/74
6.	ACINITEOBACTER	2/51	1/23	3/74
7.	E.COLI	0	1/23	1/74

Blood culture was done for all the 74 babies in this study. Blood cultures were positive in only 33 cases (44%). In our study, the most common organisms were CONS in 14 babies, Kleb.pneumoniae in 8 babies, Staphylococcus aureus in 3 babies, E.coli in 1 baby, pseudomonas in 2 cases, acinitobacter in 3 babies and proteus mirabilis 2 babies.

Figure-12

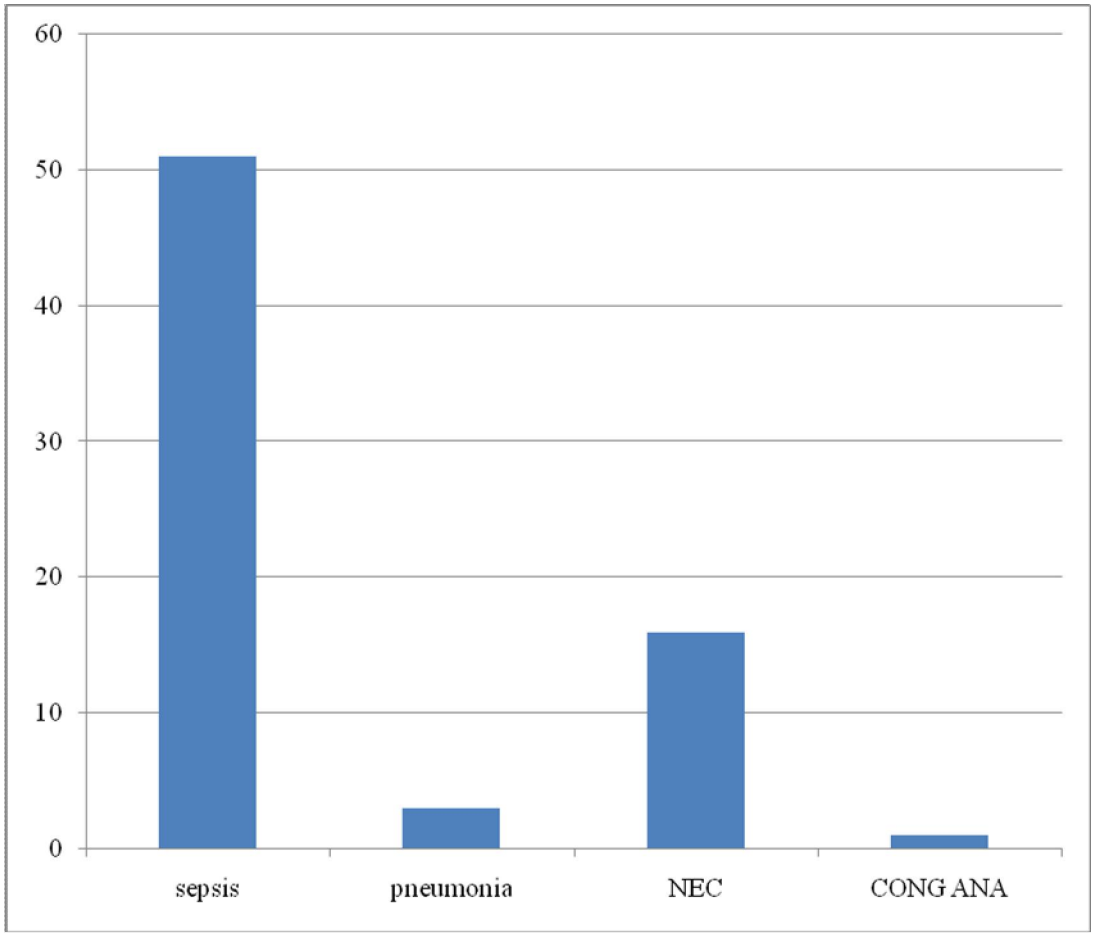
PETRI DISH FOR CULTURE

Figure-13

BLOOD CULTURE SHOWS GROWTH

Figure-14

X-RAY CHEST AND ABDOMEN



X-ray chest and abdomen have been taken to all the babies with the history suggestive of sepsis. Out of 74 babies, 3 babies showed pneumonia one baby showed right sided aortic aortic arch and 51 babies showed radiographic evidence of sepsis like hyper inflated lung field, narrow pedicle, tenting of diaphragm and dilated bowel loops.

TABLE-XI

Orga	Ampi	Amox	Cipro	Cefota	ceftaz	Vanco	Pipear	Genta	Amika	Erythro
Cons	H -0 M-0 R-14	-0 0 14	12(85 2 14	0 0 14	0 0 14	12(851 2 0	2(14) 0 12	0 0 14	0 2(16.6) 12(85.7	7(50) 5(35.7%) 2(14%)
K.pn	H-0 M-2(25) R-6	0 0 8	8(100 0 0	6(75) 2 0	8(100 0 0	0 0 8	0 0 8	0 0 8	0 0 8	0 0 8
Prot	H-0 M-0 R-8	0 0 2	2(100 0 0	2(100 0 0	2(100 0 0	0 0 2	0 0 2	0 0 2	0 2(100) 0	0 0 2(14%)
E.col	H-0 M-0 R-1	0 0 1	1(100 0 0	1(100) 0 0	1(100 0 0	0 0 1	0 0 1	0 0 1	1(100) 0 0	0 0 1
S,au	H-0 M-0 R-3	0 0 3	0 0 3	0 0 3	1(33) 1 1	3(100) 0	O 0 3	0 0 3	0 0 3	2(66.6%) 1(33.3%) 0
Pseu	H-0 M-0 R-2	0 0 2	2(100 0 0	0 1 1	2(100 0 0	0 0 2	2(100) 0 0	0 0 2	0 0 2	0 0 0
Acin	H-0 M-0 R-3	0 0 3	3(100 0 0	2(66.6 1(33.3 0	2(66) 1 0	0 0 3	0 0 3	1(33.3 2(66.6 0	2(66.6) (33.3) 0	0 0 3

MARKERS OF PROBABLE SEPSIS:

For early diagnosis of sepsis the following parameters were considered as the indicators for possibility of sepsis. They are C-reactive protein, Band form percentage more than 0.2 and total count of WBC less than 5000 /cmm. The P-value of individual test was found to be significant for all i.e. less than 0.05. The frequency distribution of all above test and their Chi -square value and p value are depicted below.

TABLE-XII

TOTAL COUNT: <5000/CMM, N-75

	Clinical	**Probable**	**Proven**	**Total**
Positive	0	8	21	29
Negative	17	16	12	45
Total	17	24	33	74

Chi square- 19.5 p-value: <0.05

TABLE-XIII

ABSOLUTE NEUTROPHIL COUNT<1800/CMM, N-74

	Clinical	**Probable**	**Proven**	**Total**
Positive	0	6	18	24
Negative	17	18	15	50
Total	17	24	33	74

Chi- square: 16.12, P –value :<0.05

TABLE-XIV

I:T RATIO>2, n-74

	Clinical	Probable	Proven	Total
Positive	0	8	16	24
Negative	17	16	17	50

chi- square: 12.04, p- value :< 0.05

TABLE-XV

CRP, n-57

	Clinical	Probable	Proven	Total
Positive	2	23	32	57
Negative	15	1	1	17

Chi- square: 53.1, p value:< 0.05

Table-XVI

Micro-ESR, n-74

	Clinical	Probable	Proven	Total
Positive	1	23	32	56
Negative	16	1	1	18

chi-square: 58.4 p-value :<0.00

Validity of individual tests can be tested when each of these tests are compared with that of the gold standard test. For neonatal sepsis, blood culture is considered as the gold standard test. Hence we have made an attempt to validate the individual tests against the blood culture, the gold standard test.

ANC test has the highest sensitivity of 75%. I:T has the highest specificity of 68%. The highest positive predictive value is for CRP and micro –ESR with 76.9% and highest negative predictive value is for ANC with 85.3%. The Sensitivity, Specificity, Positive Predictive Value and Negative Predictive Value of all the individual is tabulated in Table XVII.

Table-XVII

S.No	Test	Sensitivity	Specificity	PPV	NPV
1.	TC	72.4	62	72.4	80.4
2.	ANC	75	60	54.5	85.3
3.	I:T	66.6	68	48.4	80
4.	CRP	56.17	58	76.9	58
5.	M-ESR	57.1	55	76.9	41.4

OUTCOME:

Of the 74 babies in our study group 24 babies died and 50 babies were discharged. Those babies discharged from the newborn unit were advised to attend

high risk follow up clinic every week. Out of 24, 14 babies had culture proven sepsis, 7 babies had probable sepsis and 3 babies had clinical sepsis.

Table-XVIII

OUTCOME

S.No	Alive	Death	Total
Clinical	14	3	17
Probable	17	7	24
Proven	19	14	33

Out of 33 death, 3 babies had clinical sepsis, 7 had probable sepsis and 14 had culture proven sepsis.

TABLE-XIX

MORTALITY VS ORGANISM

ORGANISM	DEATH(N-24)
CONS	8 (33.3%)
K.pneumoniae	4 (16.6)
Staph.aureus	1 (4.1%)
Proteus mirabilis	1 (4.1%)

Out of 24 deaths, 14 were culture positive. Out of 14, CONS was demonstrated in 8 babies, K.pneumoniae in 4 babies, S.Aureus in 1 baby and Proteus in one baby.

An attempt was taken to correlate the major clinical findings and the investigations with poor outcome to identify the poor prognostic factors. Abnormal CSF findings were found to strongly correlate with mortality with a P-value of 0.01 (<0.05). Among the laboratory finding, the platelet count less than 1 lakh was well correlated with death with a P-value of <0.05. The data have been given in table-XX

TABLE-XX

S.**No**	**Variables**	**Death**	**P-value**
1.	Respiratory distress (n-33)	n-15	0.03
2.	Shock (n-23)	n-11	0.1
3.	Cyanosis (n-25)	n-12	0.04
4.	CRT>2sec (n-23)	n-11	0.05
5.	Pallor (n-32)	n-15	0.02
6.	Per umbilical erythema (n-10)	n-8	0.01
7.	Induration (n-9)	n-8	0.01
8.	Blood c/s (n-33)	n-16	0.008
10.	CSF findings (n-16)	n-12	0.01
11.	Culture positive CSF (n-2)	n-2	0.01

DISCUSSION

VII. DISCUSSION

Neonatal sepsis is the major contributor of the neonatal mortality and there by infant mortality. The aim of our study was to know about the profile of late neonatal sepsis and assess the environmental and maternal risk factors influencing late onset sepsis.

Our study was done over a period of one year from October 2010 to September 2011. Seventy four babies were included in our study. Out of 74 babies 38 babies had been admitted in the NICU since birth for preterm and low birth weight management. The remaining 35 babies were healthy at the time of delivery without any maternal risk factor, kept along the mother side, developed sepsis after 72 hours of life. In the present study, out of 74 babies 24 babies died and 50 babies were discharged and followed up in the high risk clinic.

National Neonatal Perinatal Database coined the norms of early onset sepsis and late onset sepsis. But recent Journals and some text book of neonatology state that EOS may present as late as 7 days of life and LOS from 7 days of life to as late as 8-12 weeks of life. So in our study we classified the late onset sepsis into extended early onset sepsis (3 days to 7 days) and late onset sepsis (7 days to 28 days of life). Some journals have also included another category namely Late late onset sepsis(LLOS) which would be sepsis beyond the neonatal period until 12 weeks of age.

In my study we have further divided the group into proven sepsis, probable sepsis and clinical sepsis for analytical purpose. Similar classification was first tried by William E Benitz et al in USA and later by Anita Sharma et al in her study.

In my study population, the commonest clinical symptom were refusal of feeds (71.6%) and respiratory distress(44.4%). Richard. A.Polin et al also found that feed intolerance and apnea were the common presentation.

Both Shashikala S Tallur et al and Chacko Betty et al have found respiratory distress as the commonest presenting complaint. The same has been corroborated by National neonatal Perinatal Database 2003 and Roberton Text Book Neonatology. It may be due to inclusion of EOS in their studies where respiratory distress is the most common presentation.

In the present study I have found that the Preterm babies were more prone to neonatal sepsis because the immunological system is not mature enough to combat bacterial infection more effectively. Prematurity has been noted as very important neonatal risk factor for sepsis. In my study group of 74 babies, 49 babies were preterm and 25 babies were term. In addition, 62 out of 74 babies were Low birth weight, of whom 12 were term and 50 were preterm.

Kurein Anil Kuruvilla , Deepak Chawla et al and Shashikala S Tallur et al stated that the preterm babies have inherent immature complement system, macrophage and neutrophil which are not very effective in mounting a full resistance to infection thus making the preterm more prone for infection. The

compromised skin barrier and need for invasive procedure, prolonged use of life supporting apparatus make them more prone for late onset sepsis.

In the present study, the male babies were more prone for sepsis than female babies. The same has been documented by Upadhyay et al and Nelson Text Book of Pediatrics 24 page 628. Our study revealed that out of 74 septic babies 50 were male and 24 were females. Though it has been documented in several standard books, the reason for male predominance is unknown. The possible locus of gene for synthesis of immunoglobulins at X chromosome probably accounts for relative resistance of the female infants to infections.

Obstetric risk factors are mainly responsible for Early onset sepsis. In our analysis the environmental risk factors were divided in to shared and unshared environment. Shared environmental effects (in utero, maternal and intrapartum variables) did not have any significant influences on late onset sepsis. Following are shared environmental influences

- Positive vaginal swab
- Foul smelling lochia
- Wound infection
- UTI (urinary tract infection)
- RTI(Respiratory tract infection) with fever
- Mastitis.

We have done all the investigations in symptomatic mothers. We have looked for correlation between maternal risk factors and late neonatal sepsis. But

there wasn't any correlation clinically as well as statistically. Many text books of neonatology and journals confirmed the same.

Unshared environmental influences (eg: co morbidities and other variables related to hospital course) were determined to account for a significant component of the variance in liability to late onset sepsis. These findings support the late neonatal sepsis as a hospital acquired disease process in a highly vulnerable patient population. In the present study 35 babies were delivered without any complication and allowed to stay along with the mother in the postnatal ward developed sepsis after 72 hours of life. These babies might have been handled by the mother, attendees, and hospital personnel. **Out of 35 babies 9 babies had significant pustules, 8 babies had multiple abscesses and 6 babies had septic arthritis of knee joint**. It may be due to poor hygiene of the babies or the attendees.

Usually vernix caseosa and the maternal blood are the good culture media for multidrug resistant nosocomial micro organisms. If it is not removed properly, it facilitates the colonization of bacteria which produces pustules, abscess and septic arthritis. Those lesions lead to sepsis. In the present study it was clearly proved.

The major independent risk factors previously identified for LOS include

- Duration of hospital stay
- Duration of use of central venous catheters
- Duration of empirical antibiotics
- ET intubation
- Duration of mechanical ventilation
- Duration of TPN(Total parenteral nutrition)

- Contaminated procedures
- Handling the babies with unclean hands
- Longer the stay in NICU and prolonged IV cannula in situ increased the chance of sepsis.

Joseph B. cantey et al, Bizzarro et al has found the same in their studies. He also found that prolonged empirical antibiotics leads to NNEC, LOS and multidrug resistant organism colonization. Patrikck S spafford et al found that colonization rate of central IV catheter is 40%. Another study stated that the rate of 10%.

Recent studies states[29] that apron of the hospital personnel harboring micro organism may spread to babies whether it cause sepsis or not. In the present study, about 38 babies out of 74 babies were admitted in the NICU for preterm and low birth weight care developed sepsis. 2 babies were intubated for surfactant therapy developed sepsis.

There are specific and nonspecific tests[30] for diagnosis of neonatal sepsis. Of the specific tests, blood culture is considered as the single most important test. Though this test is considered to be the gold standard test for neonatal sepsis, it has a sensitivity of 55%. This was given by Sangamithra datta et al[32] in her article about laboratory diagnosis of neonatal sepsis in NNF journal.

The commonest organism identified in the present study is CONS followed by Klebsiella and Staphylococcus aureus. According to NNPD 2002-2003 report, the commonest organism isolated from all the 18 centres in India has been Klebsiella

followed by Staphylococcus aureus. The data included early onset sepsis also. It could be the reason for Klebsiella as the commonest organism.

Recent articles and studies in various Indian NICU set up showed CONS as the commonest organism. Initially it was thought as harmless contaminant. But now it was found to produce serious life threatening illness like sepsis and meningitis. In our study it was isolated from the CSF of two babies.

CONS emerged as the most common causative organism for late neonatal sepsis in India and it has developed resistance to conventionally advocated antibiotics like ampicillin and gentamicin. The sensitivity to conventional antibiotics is as low as 14% in our study group.

For Staphylococcus aureus vancomycin, cefotaxime and ciprofloxacin are the most sensitive. First line antibiotics recommended by WHO like Ampicillin and Gentamycin are almost resistant to hospital acquired infection. Most of the organism showed resistant to cefatoxime and amikacin also.

Coagulase negative Staphylococcus aureus (CONS) has become almost resistant to conventionally used antibiotics like ampicillin and gentamicin. They are sensitive to vancomycin, ciprofloxacin and erythromycin in our study. Klebsiella has remained sensitive to amikacin in 31.2 % of cases in NNPD. But in our study it is resistant to Amikacin. It is sensitive to ciprofloxacin, cefotaxime and ceftazidime. Narrow spectrum antibiotics must be used wherever possible. The broad spectrum antibiotics should be restricted and used for specific situations when warranted.

As a part of sepsis work up it is imperative to do urine examination, blood culture and CSF analysis. We have done CSF analysis in all cases with bactremia and in those babies with signs and symptoms of late onset sepsis. We have done CSF analysis in 74 babies. 16 babies had abnormal CSF findings. CONS was isolated from CSF of two babies.

Urine culture was done for all the 74 babies. Organism was positive only in 5 babies. Of which, K.pneumoniae was positive in 2 babies followed by E.Coli(1), proteus(1) and CONS.(1).

The incidence of meningitis in neonatal sepsis has varied from 0.3 to 3% in various studies. In the present study, blood culture was done for all the 74 babies. Blood cultures were positive in 33 cases(44%). In our study, CONS is the commonest organism(14) followed by K.pneumonia(8) and S.Aureus(3).

The sensitivity and specificity of early diagnostic markers were obtained by validating it against the blood culture. Anita Sharma et al have also done the validation of the entire test as against the blood culture. Since positive blood culture is the investigation to confirm bactremia, all the other early markers of sepsis can only be validated when compared to blood culture even though it has got a sensitivity to a maximum of 50-60%.

In the present study ANC has got a high sensitivity of 75% and specificity of 60%. Sensitivity and specificity of CRP were 56.1% and 58% respectively. In the study of Anita Sharma et al Sensitivity for CRP was 80%. Similar results were obtained by other workers also. Anita Sharma et al have obtained moderate

sensitivity for band forms with sensitivity of 60% and specificity of 56.2% In our study, for Band form percentage the sensitivity and specificity obtained were 66.6% and 68% respectively. Leukopenia <5000 is considered an important marker of neonatal sepsis. Sensitivity and Specificity for this study being 72.4% and 62% respectively.

MORTALITY:

In the present study 24 babies were died due to sepsis. Out of 24, 14 babies were culture positive. Mortality was higher among the babies infected by CONS followed by K.pneumoniae.

In the present study,the case fatality rate is 32.4%. The rate is comparable with many studies done in India. In Chako Betty et al study the case fatality rate was 19.4 %. A higher case fatality rate of 47.52% was observed by Shasikala S Tallur et al in her study in Hubli. Roberton text book of neonatology gives a case fatality rate of 15% for LOS. The prognosis of the baby depends on etiologic agent, timing of diagnosis, therapy and complications in surviving infant.

Therapy is initiated with conventional antibiotics for commonly prevailing organism in the nursery pending blood culture reports. Guided by culture reports antibiotics are changed. The prevailing pattern of antibiotic therapy may not be sustainable in the long run due to the emergence of resistant strains and new organism. Antibiotic class cycling may be advocated as a potential strategy for reducing antimicrobial resistance. A class of antibiotic is withdrawn from use for a defined period and reintroduced at a later time in an attempt to limit bacterial

resistance. Further withdrawing an antibiotic from use can potentially restore its potency. Though blood culture is considered as the gold standard test it has a sensitivity only to 50-60%.

Very often other problems may be mistaken for sepsis and therefore misdiagnosed. A high index of suspicion will prevent overdiagnosis of the same.

The differential diagnosis of neonatal sepsis

1. Metabolic abnormalities like inborn error of metabolism hypoglycemia, electrolyte imbalance
2. Hemolytic disease of newborn
3. Congenital heart disease
4. Congenital and nataly acquired infections eg syphilis, HSV, CMV, and Rubella.

From this study we can understand that apart from poor socioeconomic status and bad child rearing practices, nosocomial infection is the major contributor of late onset sepsis. Nowadays survival of preterm infant and low birth weight neonate is increasing because of the advancement in the medical field like surfactant therapy and ventilator support.

Figure-15

HAND WASHING TECHNIQUE

RULES OF TEN

1. By practicing a simple manoeuvre like proper hand washing before and after touching each baby will change the entire scenario.
2. Injudicious use of antibiotics and prolonged antibiotics can be avoided.
3. Usage of sterile disposable instruements may helpful in the prevention of hospital acquired infection.
4. Ventilator and incubator to be sterilized periodically.
5. NICU and POSTNATAL ward to be fumigated periodically.
6. Babies to be treated in an open care system.
7. Apron of the hospital personnel to be changed every day and to be autoclaved properly.
8. Restriction of attender and formation of specific protocol for them will helpful to prevent hospital acquired infection.
9. Following strict aseptic precaution while doing procedure is life saving
10. Finally information education and communication to the hospital personnel as well as mother and attender will be very helpful, because "**PREVENTION IS BETTER THAN CURE"**

Figure-16

OPEN CARE SYSTEM

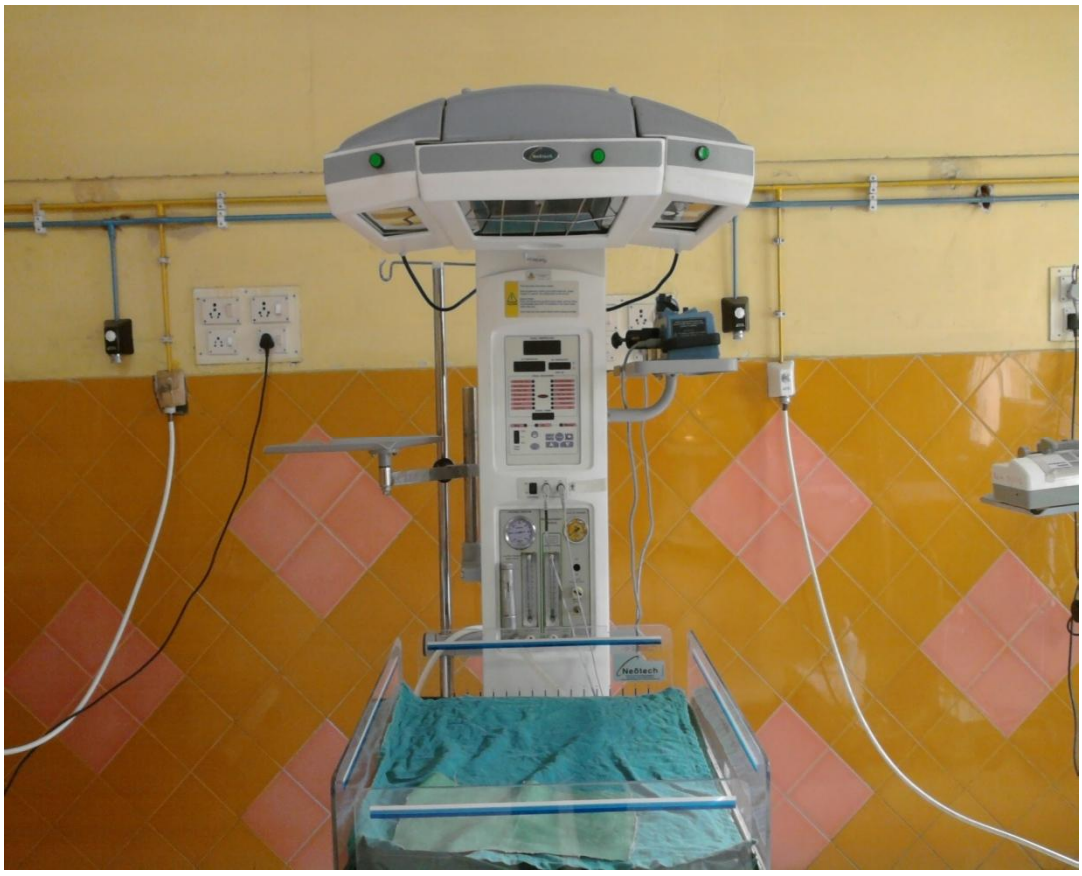


Figure-17

UV. STERLISATION OF THE WARD

CONCLUSION

VIII. CONCLUSION

1. Neonatal sepsis can be classified in to extended early onset sepsis and late onset sepsis. It is further classified in to clinical, probable and proven sepsis.

2. Refusal of feed is identified as the commonest presentation of neonatal sepsis.

3. Coagulase negative staphylococcus aureus followed by klebsiella and staphylococcus aureus are the most common organisms isolated in blood culture of the septic neonates.

4. Absolute neutrophil count <1800/cmm of blood is the most sensitive and I:T ratio >2 is the most specific laboratory markers for neonatal sepsis.

5. Culture positive CSF is the best predictor for the outcome of the disease**.**

6. Klebsiella Pneumoniae is resistant to conventional antibiotics and highly sensitive to cefotaxime, ciprofloxacin and imipenam.

7. CONS are resistant to all the first line antibiotics and highly sensitive to vancomycin. Ciprofloxacin and erythromycin.

8. Unshared environment plays an important risk factor for late neonatal sepsis.

9. Information education and communication to hospital personnel and the mother and attender regarding neonatal sepsis is beneficial.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. National Neonatal Perinatal Database – report 2002-2003, AIIMS, NEW DELHI.

2. CARE of the NEWBORN 6th Edition by Meharban singh page 209-215

3. Nelson textbook of paediatrics (17th edition) Eds Behrman RE, Klegman RM, Jenson HB, Philadelphis, WB Saunders Company, 2004; page 623-630.

4. Avery text book of neonatology - chapter Bacterial sepsis and meningitis. 8th edition. Richar A polin.page 557-580.

5. Roberton textbook on neonatology - Peter Dear - Infection in newborn

6. Bizzarro et al the impact of environmental and genetic factors on late –onset sepsis vol158. No2 feb 2011,journal of pediatrics

7. Stuart E. Starr et al-Antimicrobial therapy of bacterial sepsis in the newborn infant-The journal of pediatrics,vol-106,pages 1043-1046

8. Joseph B. Cantey et al : proonged antibiotic therapy for culture sepsis in preterm infants

9. Shashikala S Thallur *et al* : Clinico-Bacteriological Study of Neonatal Septicemia in Hubli. Indian journal of pediatrics 2000;67(3):169-174

10. Upadyay A,Aggarwal R *et al.*, Profile of Neonatal Sepsis in a Tertiary Care Neonatal Unit from India : A Retrospective Study Journal of Neonatology.NNF vol 20.no.1.jan-mar 2006.page 50-57.

11. Chacko Betty *et al*: Early onset neonatal sepsis. Indian journal of pediatrics 2006 Nov; 72:23-26.

12. Anil Kuruvilla *et al*: Bacterial Profile of Sepsis in a Neonatal unit in South India. Indian pediatrics 1998 volume 35 851-858.

13. Richard A. Polin- The ins and outs of neonatal sepsis. The journal of pediatrics.-vol 143 issue1,july 2003.

14. Anita Sharma *et al*: Evaluation of Sepsis Screen for Diagnosis of Neonatal Septicemia. Indian J pediatric; 60:559-563

15. Bhutta ZA *et al.*Neonatal Sepsis in Pakistan.Acta paediatric scand 1991 80:596-601.

16. Daoud AS *et al*: The Changing faces of Neonatal Septicemia. Ann Trop Pediatr 1995; 15:93-96

17. Chugh K *et al*: Neonatal Septicemia. Arch Dis Child 1985; 60:542-546.it's time to stop,the journal of paediatrics

18. Singel JD mc craken GH et al- journal of paediatrics. Management of sepsis. NNF Journal Vol 20 No 1 Jan-Mar 2006

19. Mongo K,Fernandez *et al*.Changing Bacterial patterns in neonatal septicemia.

20. Ashok K Deori *et al* Changing Pattern of Bacterilogic Profile in Neonatal Sepsisamong Intramural babies. Journal of Neonatology.NNF vol20 no 1 2006 page 8-15.

21. Horton JA ,Hemming VG et al: A survey of infants with neonatal sepsis in USA.

22. P.S Spafford et al prvention of central venous catheter CONS sepsis in neonatal sepsis in newborns,The journal of paediatrics volume 125, no2.

23. Baker et al-neonatal bacterial infection-the journal of pediatrics, sep 1981,page-452.

24. Deepak Chawla *et al*: Rational Approach to Diagnosis of Neonatal Sepsis. Journal of Neonatology vol 20 No 1 2006 page 4 -7.

25. Piyush Gupta *et al*: Clinical Profile of Klebsiella Septicemia in Neonates.indian j pediatr 1993; 60:565-572.

26. Cotten CM et al. prolonged duration of initial empirical antibiotics increased with incidence of NEC and death in VLBW babies

27. William E benitz *et al*: Serial serum C reactive Protein Levels in the Diagnosis of Neonatal Infection. Pediatrics vol 102 No. 4 1998 p 41.

28. Robyn L Rodwel et al- Early diagnosis of neonatal sepsis using a hematologic scoring system-The journal of pediatrics may-1988, page 761-765

29. Alan H. Jobe et al –finally a diagnostic marker for neonatal sepsis-Journal of paediatrics April 2009, page-620

30. Squire En *et al:* Criteria for the discontinuation of antibiotic therapy during presumptive treatment of suspected neonatal infection. Pediatric infect dis1982; 1:85-90.

31. Rajesh Agarwal *et al*: Sepsis in newborn Indian journal of pediatrics vol 68 20011143-1147.diagnosis of neonatalsepsis. Indian pediatr 1987 24:39-43.

32. Sanghamitra Datta *et al*: Laboratory Diagnosis of Neonatal Sepsis. Journal of neonatology NNF 2006 vol 20 no.1 16-23.

33. Mandira banergee *et al*: Outbreak of Neonatal Septicemia with Multidrug Resistant Klebsiella pneumoniae. Indian j of pediatric 1993, 60 25-27.

.

ANNEXURES

CLINICAL PROFORMA

S.No **IP.No**

NAME :

AGE : SEX :

ADDRESS :

WEIGHT :

LENGTH : GEST.AGE :

PI : OFC :

HISTORY OF PRESENT ILLNESS:

H/O irritability Y/N

H/O shrill cry Y/N

H/O lethargy Y/N

H/O poor feeding Y/N

H/O vomiting Y/N

H/O loose stool Y/N

H/O cold to touch Y/N

H/O fever Y/N

H/O pustules	Y/N
H/O abscess	Y/N
H/O difficulty in breathing	Y/N
H/O abdominal distention	Y/N
H/O bleeding from umbilicus	Y/N
H/O convulsions	Y/N
H/O joint swelling	Y/N

MATERNAL HISTORY:

Age of the mother	
Booked and immunized	Y/N
Any medical illness during pregnancy	Y/N
Maternal weight gain?	
Viral exanthematous fever?	Y/N
Drug intake	Y/N

INTRAPARTUM

PPROM more than 18 hours?	Y/N
Meconium stained amniotic fluid?	Y/N

Maternal fever?	Y/N
Foul smelling amniotic fluid?	Y/N
Prolonged labour?	Y/N
Nature of delivery	Y/N
What is the APGAR score?	
Was oroparyngeal suction applied?	Y/N

POSTNATAL

H/O prelacteal feed?	Y/N
H/O bottle feed	Y/N
H/O bad child rearing practices?	Y/N
NICU admission	Y/N
IV and cannulation	Y/N
IV fluid	Y/N

CLINICAL EXAMINATION:

Pallor, anemia/,cyanosis/, jaundice

Petechiae /purpura /eccymoses

Periumbilical erythema/ induration

Umbilical discharge	Y/N
Pustules	Y/N
Abscess	Y/N
Sclerema	Y/N

VITAL SIGNS:

Heart rate

Respiratory rate

Temperature

Pulse

CRT

Blood pressure

CNS:

Sensorium

Tone

Reflexes

Anterior fontanels

CVS:

Respiratory system:

Abdomen**:**

Distention:

Tenderness:

Bowel sound

Bone and Joint

Spine and Cranium

INVESTIGATIONS:

Complete blood count

I:T

Renal function Test

Liver function test:

C reactive protein

Micro ESR

Blood culture/S

Diagnostic Lumbar puncture

Chest X ray

Urine culture C/S

Blood culture

Maternal symptom

Mat urine c/s

Mat blood c/s

High vaginal swab

MASTER CHART

NAME	AGE	SEX	WT	GES	IR	SC	LE	PF	VOM	LS	HY.T	APN	PU	AD	RD	BT	CON	ART	NICU	IVF	JA	ET	BOOK	MI	MW	DRUG	DEL	PRE	PAL	CRT	CYA
B/O SAROJA	1	1	4	5	1	2	2	2	2	2	2	2	2	2	2	2	2	2	1	1	2	2	2	1	2	2	1	1	2	1	1
B/O KALAIVANI	2	1	1	2	2	2	2	1	1	2	2	2	2	1	2	2	2	2	1	1	1	2	2	3	1	1	1	2	2	1	1
B/O MANI	1	2	1	2	2	2	1	2	2	2	2	2	2	2	1	2	1	2	1	1	1	2	2	1	1	2	1	2	2	1	1
B/O DHANAM	1	1	1	1	1	1	2	1	2	2	2	2	2	2	1	2	1	2	1	1	1	2	2	1	1	2	1	2	1	1	1
B/O ANUHA	1	1	1	1	2	2	1	1	2	1	2	2	2	2	2	2	2	2	1	1	1	2	2	1	1	2	2	2	2	1	1
B/O KARUNYA	1	1	3	1	1	2	2	1	2	2	2	2	2	1	1	2	2	2	2	2	2	2	1	1	1	2	2	1	2	1	1
B/O SUREKA	1	1	2	1	2	2	2	1	1	2	1	1	2	2	1	2	2	2	2	2	1	2	2	1	1	2	1	2	1	2	2
B/O LOGA	1	1	1	2	2	2	1	1	2	2	1	2	2	2	2	2	1	2	1	2	2	2	2	1	1	2	1	2	1	2	2
B/O HEPSIBA	1	2	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2	1	2	1	2	2	1	2	2	1	2	2	1	1
B/O VANI	1	2	1	1	2	2	1	1	1	2	2	2	2	2	2	2	2	1	2	1	2	1	2	2	2	2	1	2	1	1	1
BIO KUPPAMAL	2	1	1	2	2	2	2	1	2	2	2	2	2	1	1	2	2	1	2	2	2	2	2	1	1	2	1	2	2	1	1
B/O SUMA	2	2	1	2	2	2	1	1	2	1	1	2	2	2	2	2	2	2	1	1	2	2	2	1	1	2	1	2	1	2	2
B/O KANNAGI	2	2	1	1	2	2	2	1	2	2	2	2	1	2	1	2	2	2	1	1	2	2	2	1	1	2	1	2	2	1	1
B/O AFREEN	1	2	1	1	2	2	1	2	2	1	1	2	2	2	1	2	2	2	1	1	1	2	2	1	2	2	2	2	1	1	1
B/O AYISA	2	1	1	2	2	2	2	1	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	1	1	2	2	2	2	1	1
B/O UNA SUND	1	2	2	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	1	1	2	2	2	2	1	1
B/O AMALA	1	2	1	1	2	2	2	1	2	2	1	2	2	2	2	2	2	2	2	2	1	2	2	1	1	2	1	2	1	2	2
B/O SUMA	1	1	3	3	1	1	2	1	2	1	2	1	1	2	1	2	1	2	2	2	1	2	2	1	2	2	2	1	2	1	1
B/O SAROJA	1	1	1	1	2	2	2	2	2	2	2	2	1	1	2	2	2	2	1	1	2	2	2	2	1	1	2	2	2	2	2
B/O MERCY	1	1	1	1	2	2	1	1	2	1	2	2	2	2	1	2	2	2	1	1	2	2	2	3	2	1	2	2	1	1	1
B/O MONISHA	1	1	1	1	2	2	2	1	2	2	1	2	2	2	2	2	2	2	1	1	1	2	2	1	1	2	1	2	1	2	2
B/O LATH	1	1	1	1	2	2	1	1	2	2	1	1	2	2	1	2	2	2	1	1	2	2	2	1	1	2	1	2	1	2	2
B/O MALA	1	1	3	3	2	2	2	1	2	1	1	2	2	2	2	2	2	2	2	2	2	2	2	1	1	2	3	2	1	2	2
B/O SWATHY	2	1	1	1	1	1	2	1	2	2	2	1	2	2	1	2	1	2	1	1	2	2	2	1	1	2	1	2	2	1	1
B/O BIOVAAR	2	1	1	1	2	2	2	1	2	1	2	1	2	2	2	2	1	2	1	1	1	2	2	2	1	1	1	2	2	1	1
B/O PRIYA	1	1	2	4	2	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	1	1	2	1	2	1	1	1
B/O SARANYA	1	2	1	1	2	2	2	1	2	2	1	1	2	1	2	2	1	2	1	1	2	2	2	1	1	2	1	2	1	1	1
B/O SELVI	1	1	1	2	2	2	2	1	2	2	1	2	2	2	1	2	2	2	1	1	1	2	2	1	1	2	1	2	1	2	2
B/O SARITHA	2	1	1	1	2	2	1	2	2	1	2	2	1	2	2	2	2	2	1	1	2	2	2	1	1	2	2	2	2	1	1
B/O BEEVI	1	2	1	2	2	2	2	1	1	2	1	1	2	2	1	2	2	2	1	1	1	1	2	4	2	2	1	2	1	2	2
B/O USHA	2	1	1	1	2	2	1	2	2	2	1	1	2	2	2	2	1	1	1	1	1	2	2	1	1	2	1	2	1	1	1
B/O DURGA	1	1	1	1	2	2	2	1	1	2	1	2	1	2	2	2	2	2	2	2	2	2	2	1	1	2	1	2	1	1	2
B/O ARIFA	1	1	2	5	2	2	2	1	2	2	1	1	2	1	1	2	2	2	2	2	1	2	2	1	1	2	1	2	1	2	2
B/O MANOHARI	1	1	2	1	2	2	2	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	1	2	1	2	2
B/O SUMATHI	1	2	2	4	1	1	2	1	1	2	2	2	1	1	2	1	1	1	2	2	2	2	2	1	1	2	1	2	2	1	1
B/O LAKSHMI	2	2	1	1	2	2	2	2	2	2	2	2	1	2	2	2	2	2	1	1	1	2	2	1	1	2	1	2	2	1	1
B/O PRIYA	1	2	1	4	2	2	1	1	2	2	2	2	1	2	1	2	2	2	2	2	1	2	2	1	1	2	1	2	2	1	1
B/O JEEVA	1	1	1	2	2	2	2	1	2	1	2	2	2	2	2	2	2	2	1	2	2	2	2	2	1	1	1	2	2	1	1

NAME	AGE	SEX	WT	GES	IR	SC	LE	PF	VOM	LS	HY.T	APN	PU	AD	RD	BT	CON	ART	NICU	IVF	JA	ET	BOOK	MI	MW	DRUG	DEL	PRE	PAL	CRT	CYA
B/O BHAVANI	2	2	1	2	2	2	2	1	2	2	2	1	2	2	2	2	1	1	1	1	1	1	2	1	1	2	1	2	2	1	1
B/O V.LAKSHMI	1	1	1	1	2	2	1	2	2	2	1	1	2	2	1	2	2	2	1	1	1	2	2	1	1	2	1	2	1	2	2
B/O YALINI	2	1	2	3	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	1	2	1	2	2	1	1
B/O ANITHA	1	2	1	1	2	2	1	1	1	2	2	2	2	2	1	2	2	2	1	1	1	2	2	1	1	2	1	2	2	1	1
B/O SUGANYA	1	1	1	1	2	2	2	1	2	2	1	2	2	2	2	1	2	2	2	2	1	2	2	1	2	2	1	2	1	2	2
B/O AMMU	1	2	2	3	2	2	1	1	2	2	2	2	2	2	1	2	1	2	2	2	2	2	2	2	1	1	2	2	1	1	2
B/O ALAMELU	1	2	2	3	2	2	2	1	1	2	1	2	2	2	1	2	1	2	2	2	1	2	2	1	2	1	2	2	1	2	2
B/O ASWINI	2	1	2	3	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	1	1	2	2	2	1	2	2
B/O SHANTHI	2	1	1	1	2	2	1	2	2	1	2	2	2	2	1	2	2	1	1	1	2	2	2	1	2	2	1	2	2	1	1
B/O VIJAYA	2	2	1	1	2	2	2	1	2	2	1	1	2	2	2	2	1	2	1	1	1	2	2	2	1	1	1	2	1	2	2
B/O DHANALA	1	2	1	1	2	2	2	1	1	2	2	1	2	2	2	2	2	2	1	1	1	2	2	1	2	2	1	2	2	1	1
B/O MAHALAK	1	2	2	4	2	2	2	1	2	2	2	2	2	2	1	2	1	2	2	2	2	2	2	1	1	2	1	2	2	1	1
B/O BHAVANI	1	1	3	3	2	2	1	1	1	2	2	2	2	2	2	2	1	2	2	2	1	2	2	1	1	2	1	2	2	1	1
B/O SELVI	1	1	1	1	2	2	2	1	2	1	2	1	1	2	2	2	2	2	2	2	2	2	2	1	2	2	1	2	2	1	1
B/O AMSAVALLI	2	2	1	1	2	2	2	1	2	2	2	2	2	2	1	2	2	2	1	1	1	2	2	1	1	2	1	2	2	1	1
B/O JAYANTHI	2	2	1	1	2	2	1	1	1	2	2	1	2	2	2	2	2	2	1	1	1	2	2	1	2	2	1	2	2	1	1
B/O SEETHA	1	1	2	4	2	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	1	1	2	1	2	1	1	1
BI/O INDIRA	1	1	4	5	2	2	1	2	2	2	2	2	2	2	2	2	1	2	1	1	2	2	2	3	2	1	2	2	2	1	1
B/O GAYATHRI	2	1	3	3	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	2	1	2	2	1	2	2	1	2	2	1	1
B/O AMSAVENI	2	1	2	4	2	2	1	1	1	2	2	2	2	2	1	2	2	2	2	2	1	2	2	1	2	2	1	2	2	1	1
B/O ELISA	1	2	1	1	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	1	2	2	1	1	2	1	2	1	2	2
B/O BEULA	1	1	1	2	2	2	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	1	2	1	2	2	1	1
B/O AMALA	1	2	2	3	2	2	2	1	2	2	1	2	2	2	1	2	2	2	2	2	1	2	2	1	1	2	1	1	1	2	2
B/O KUMUTHA	1	1	1	1	2	2	2	2	1	2	2	1	2	2	2	1	2	2	1	1	1	1	2	2	2	1	1	2	2	1	1
B/O KAMATCHI	1	1	1	2	2	2	2	1	1	2	2	1	2	2	1	2	2	2	1	1	2	2	2	2	1	1	1	2	2	1	1
B/O JOSHWIN	1	1	1	1	2	2	1	1	2	1	1	1	2	2	1	2	2	2	1	1	2	2	2	1	2	2	1	2	1	2	2
B/O KALPANA	1	2	1	1	2	2	2	2	1	2	2	2	2	2	2	2	2	2	1	1	1	2	2	1	1	2	1	2	2	1	1
B/O SUMATHI	2	1	1	2	2	2	1	1	2	2	1	1	2	2	1	2	2	2	1	1	2	2	2	1	2	2	1	2	2	1	1
B/O LAVANYA	2	1	3	3	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	1	1	2	2	2	1	2	2
B/O MEGALA	1	1	3	3	2	2	1	1	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	1	2	2	1	2	2	1	1
B/O VALLI	1	1	3	3	1	1	2	1	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	1	1	2	1	1	2	1	1
B/O REKHA	1	1	2	4	2	2	1	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	1	1	1	2	2	1	1
B/O DEVI	1	1	2	4	2	2	2	2	2	2	1	1	2	2	1	2	1	2	2	2	2	2	2	1	1	2	2	2	1	2	2
B/O PUSHPA	2	1	4	3	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	1	2	1	2	2	1	1
B/O SHAMILA	1	1	1	1	2	2	2	2	2	2	1	1	2	2	1	2	2	2	1	1	2	2	2	1	2	2	1	2	1	2	2
B/O BANU	2	1	3	5	1	1	2	2	2	2	2	2	2	1	2	1	2	2	2	2	2	2	2	1	1	2	1	1	2	1	1

BT	AD	IND	ERY	PUL	HR	RR	REFL	CVS	RS	TEN	BS	BONE	HB	TC	ANC	IT	PLT	GLUC	UREA	CREA	BILI	CRP	ESR	BCS	UCS	CSF	XRAY	MS	MUCS	MSYM	CSFS	SEP	OC
2	2	2	2	1	2	2	1	1	1	2	1	1	2	2	2	2	1	2	1	1	2	2	2	1	1	1	1	1	1	2	1	1	1
2	2	2	2	1	2	2	1	1	1	2	1	1	3	2	2	2	2	2	2	2	2	1	1	1	1	1	1	1	1	1	1	2	1
2	2	2	2	1	2	1	1	1	1	2	1	1	2	2	2	1	1	2	1	1	3	2	2	1	1	2	1	1	1	1	1	2	2
2	1	2	2	2	2	2	2	1	1	2	1	1	3	2	2	2	2	2	1	1	2	1	1	2	1	2	1	1	1	1	1	2	2
2	2	2	2	1	2	2	1	1	2	2	1	1	2	2	2	2	2	2	1	1	2	2	2	1	1	1	2	1	1	1	1	1	1
2	2	2	2	1	2	2	1	1	1	2	1	1	3	1	1	1	1	2	1	1	2	1	1	1	1	1	2	1	1	2	1	2	1
1	1	2	2	2	2	2	2	1	2	1	1	1	2	1	1	2	2	1	1	1	2	1	1	2	1	1	2	1	2	2	1	2	1
2	2	2	2	2	2	2	2	1	1	2	1	1	3	1	2	2	2	1	2	2	2	1	1	2	1	1	2	1	1	1	1	3	1
2	2	2	2	1	2	2	1	1	2	2	1	1	2	2	2	2	2	1	2	2	3	1	1	1	1	1	3	1	1	1	1	2	1
2	1	2	2	1	2	2	2	1	1	2	1	1	3	2	2	2	2	2	2	2	3	2	2	1	1	2	2	1	2	1	1	1	2
2	2	2	2	1	1	1	1	1	1	2	2	1	3	2	2	2	2	2	2	2	2	1	1	1	1	1	1	1	1	1	1	3	1
2	2	1	1	2	2	2	2	1	1	2	1	1	2	1	2	1	1	2	1	1	2	1	1	2	1	2	1	1	1	1	1	2	2
2	2	2	2	1	2	2	1	1	1	2	1	1	2	2	2	2	2	1	2	2	3	2	2	1	1	1	2	1	1	2	1	1	1
2	1	2	2	2	2	1	2	1	1	2	2	1	2	1	1	1	2	1	2	2	2	1	1	1	1	1	2	2	2	1	1	3	1
2	2	2	2	1	2	1	1	1	1	2	1	1	2	2	2	1	2	2	2	2	2	1	1	1	1	1	2	2	1	2	1	3	1
2	2	2	2	1	2	2	1	1	2	2	1	1	2	2	2	2	2	2	1	1	2	1	1	1	1	1	2	1	1	1	1	2	1
2	1	2	2	2	2	2	2	1	1	1	2	1	2	2	2	2	1	1	1	1	2	2	2	1	1	1	2	2	1	1	1	1	1
2	1	2	2	1	2	2	2	1	2	2	1	1	2	1	2	2	1	2	2	2	3	1	1	2	1	1	2	1	1	1	1	3	1
2	2	2	2	1	1	2	2	1	1	2	1	1	2	1	1	2	1	1	1	1	3	1	1	1	1	1	1	1	1	1	1	2	2
2	2	2	2	1	2	2	2	1	1	2	1	1	3	2	2	1	1	1	1	1	2	1	1	1	1	1	2	1	2	1	1	2	2
2	2	2	2	2	2	2	2	1	1	2	1	1	3	1	1	2	1	2	1	1	3	1	1	1	1	1	1	1	1	1	1	2	1
2	1	1	1	2	2	2	2	1	1	1	3	1	3	1	1	1	1	1	1	1	2	1	1	2	2	2	2	1	1	1	2	3	2
2	2	2	2	2	2	1	2	1	1	2	1	1	3	1	2	2	1	2	1	1	2	1	1	1	1	1	2	1	1	1	1	2	1
2	2	2	2	1	2	1	1	1	2	2	1	1	2	1	1	1	1	2	1	1	4	1	1	2	1	1	3	1	1	1	1	3	2
2	2	2	2	1	2	2	2	1	2	2	1	1	2	2	2	2	2	1	1	1	3	1	1	1	1	1	2	1	1	1	1	2	1
2	2	1	1	2	2	1	2	1	1	2	1	1	2	2	2	2	2	2	1	1	3	2	2	1	1	1	2	1	2	1	1	1	2
2	2	2	2	3	2	2	2	1	1	2	1	1	2	2	2	2	2	1	1	1	4	1	1	2	1	1	2	1	2	2	1	3	1
1	1	1	1	3	2	1	2	1	1	1	2	1	2	2	2	2	1	1	1	1	3	1	1	1	1	1	2	1	1	2	1	2	2
2	2	2	2	1	2	2	1	1	2	2	1	1	2	2	2	1	2	1	1	1	4	2	2	2	1	2	2	1	1	1	1	3	2
1	2	2	2	3	2	2	2	1	1	2	1	1	2	1	1	2	1	1	1	1	3	1	1	2	2	1	2	1	2	1	1	3	2
2	2	2	2	3	2	2	2	1	1	2	1	1	3	2	2	2	2	1	2	2	4	1	1	1	1	1	2	1	1	2	1	2	1
2	2	2	2	2	2	2	2	2	1	2	1	1	2	1	1	2	1	1	1	1	3	1	1	2	1	1	3	2	1	1	2	3	2
2	2	2	2	3	2	2	2	2	1	2	1	1	3	1	1	1	1	2	1	1	4	1	1	2	1	2	2	2	1	1	1	3	1
2	2	1	1	3	2	2	2	1	1	2	1	1	1	1	1	1	1	1	1	1	2	1	1	2	1	2	2	1	2	1	1	3	2
2	1	2	2	1	2	1	2	1	2	1	3	1	2	2	2	2	2	2	1	1	3	1	1	1	1	1	2	1	1	1	1	2	1
2	2	2	2	1	2	2	2	1	1	2	1	1	2	2	2	2	2	1	1	1	4	1	1	2	1	1	2	1	1	1	1	3	1
2	2	2	2	1	2	1	1	1	1	2	1	1	2	2	2	2	1	2	1	1	3	2	2	1	1	1	2	2	1	2	1	1	1
2	2	2	2	1	2	2	2	1	1	2	1	1	2	1	1	2	2	1	1	1	4	1	1	2	1	1	2	1	1	1	1	3	1

BT	AD	IND	ERY	PUL	HR	RR	REFL	CVS	RS	TEN	BS	BONE	HB	TC	ANC	IT	PLT	GLUC	UREA	CREA	BILI	CRP	ESR	BCS	UCS	CSF	XRAY	MS	MUCS	MSYM	CSFS	SEP	OC
2	2	2	2	1	2	2	1	1	1	2	1	1	3	2	2	2	2	1	1	1	4	2	2	1	1	1	2	1	1	1	1	1	1
2	2	2	2	3	2	1	2	1	1	2	1	1	2	1	1	2	1	2	2	2	4	1	1	2	1	1	2	1	1	1	1	3	1
1	2	2	2	1	2	1	1	1	1	2	1	1	2	2	2	1	2	1	1	1	4	1	1	1	1	1	2	1	2	2	1	2	1
2	1	2	2	1	2	2	1	1	2	1	2	1	3	2	2	2	2	1	1	1	4	1	1	2	1	1	2	1	1	1	1	3	1
2	2	1	1	3	2	1	2	1	1	2	1	1	2	1	1	1	1	1	1	1	4	1	1	1	2	2	1	1	1	1	1	2	1
2	2	2	2	3	2	1	2	1	1	2	1	1	2	2	2	2	2	1	1	1	4	1	1	1	1	1	1	2	1	2	1	2	1
2	2	1	1	2	2	1	2	3	1	2	1	1	2	1	1	1	2	2	1	1	4	1	1	2	1	1	2	1	1	1	1	3	2
2	2	2	2	3	2	1	2	1	1	2	1	1	2	1	1	2	1	1	1	1	4	1	1	2	1	2	2	1	1	1	1	3	1
2	2	2	2	1	2	2	1	1	2	2	1	1	2	2	2	2	1	1	2	2	4	1	1	2	1	1	2	1	1	1	1	3	1
2	2	2	2	3	2	1	2	1	1	2	1	1	2	1	1	1	2	2	1	1	4	1	1	2	1	2	2	1	1	1	1	3	2
2	1	2	2	1	1	1	1	1	1	1	3	1	2	2	2	2	2	2	1	1	4	2	2	1	1	1	2	1	1	2	1	1	1
2	2	2	2	1	2	2	2	1	2	2	1	1	3	2	2	2	2	2	1	1	3	2	2	1	1	1	1	1	2	1	1	1	1
2	1	2	1	1	1	2	1	1	2	1	3	1	2	2	2	2	2	2	1	1	4	1	1	1	2	1	2	1	1	1	1	2	1
2	2	2	2	1	2	1	1	1	1	2	1	1	2	2	2	1	2	2	1	1	3	1	1	1	1	1	1	1	2	2	1	2	1
2	2	2	2	1	2	1	2	1	1	2	1	1	2	2	2	2	2	1	1	1	4	2	2	1	1	1	2	1	1	1	1	1	1
2	2	2	2	1	2	2	1	1	2	2	1	1	2	2	1	2	2	1	1	1	4	1	1	2	1	1	2	1	1	1	1	3	1
2	1	2	2	1	2	1	1	1	1	2	1	1	1	1	2	2	2	1	2	2	3	1	1	2	1	1	2	1	1	2	1	3	1
2	2	2	2	1	2	1	2	1	1	2	1	1	2	2	2	2	1	1	2	2	4	2	2	1	1	1	1	1	1	1	1	1	1
2	1	2	2	1	1	1	1	1	1	1	2	1	2	2	2	1	2	1	2	2	4	1	1	2	1	1	2	1	1	1	1	3	1
2	2	2	2	1	2	2	1	1	1	2	3	1	2	2	2	2	1	2	2	2	4	1	1	2	1	1	1	1	1	2	1	3	2
2	2	2	2	2	2	1	2	2	1	2	1	2	2	2	2	2	2	1	2	2	4	1	2	1	1	1	2	1	1	1	1	1	1
2	2	2	2	1	2	2	1	1	1	2	1	1	2	2	2	2	2	1	1	1	3	1	1	1	1	1	2	1	1	2	1	2	1
2	1	1	1	3	1	2	2	1	1	1	3	1	3	1	1	1	2	2	1	1	4	1	1	2	1	2	2	1	1	1	1	3	2
2	2	2	2	1	1	2	1	1	2	2	1	1	2	2	2	2	2	1	1	1	4	2	2	1	1	1	2	1	2	1	1	1	1
2	2	2	2	1	2	1	1	1	1	2	1	1	3	2	2	2	2	1	2	2	4	1	1	1	1	1	1	1	1	2	1	2	2
2	2	2	2	3	2	1	2	1	1	2	1	1	2	1	1	1	1	2	1	1	4	1	1	2	1	2	1	1	1	1	1	3	1
2	1	2	2	1	2	2	2	1	1	1	3	1	2	2	2	1	1	1	1	1	4	1	1	2	1	1	2	1	1	1	1	3	2
2	2	2	2	1	2	2	1	1	1	2	1	1	2	1	2	2	2	2	2	2	4	1	1	2	1	2	2	2	1	1	1	3	2
2	2	2	2	3	2	1	2	1	1	2	1	1	2	1	1	1	1	2	2	2	4	1	1	1	1	1	1	1	1	1	1	2	1
2	2	1	1	1	2	2	1	1	2	2	1	2	3	2	2	2	2	2	2	2	4	2	2	1	1	1	2	1	2	1	1	1	2
2	2	2	2	1	1	2	2	1	1	2	1	1	2	2	2	2	2	2	2	2	4	1	1	2	2	1	2	1	2	2	1	3	1
2	1	2	2	1	2	1	1	1	1	1	3	1	2	1	1	2	2	1	1	1	4	1	1	2	1	1	2	2	1	1	1	3	1
2	2	2	2	2	1	2	2	1	2	2	1	1	2	1	2	2	1	2	2	2	4	1	1	2	1	2	2	1	1	2	1	3	2
2	2	2	2	1	1	1	1	1	1	2	1	1	3	2	2	2	2	1	1	1	2	1	1	1	1	1	1	2	1	1	1	1	1

KEY WORDS

AGE

1. <7 days of life
2. >7DAYS OF LIFE

SEX

1- male

2-female

MATURITY

1- term

2- preterm

BIRTH WEIGHT

1. 1- 2kg
2. 2 to 2.5 kg
3. 2 to 3 kg
4. > 3kg

CLINICAL FEATURES

1- yes

2- no

MATERNAL ILLNESS DURING PREGNANCY

1- No

2- PIH

3- GDM

4- Anemia cmplicting pregnancy

PERIPHERAL PULSE

1 -normal

2- week pulse

3- absent pulse

BOWEL SOUND

1- normally heard

2- sluggish

3- absent

NATURE OF DELIVERY

1- Labour natural

2- LSCS

3- Instrumental delivery

INVESTIGATIONS

HAEMOGLOBIN

1. <10gm/dl
2. 2-10 to 14 gm/dl
3. >14gm/dl

TOTAL COUNT

1. <5000/cmm
2. >5000/cmm

IMMATURE TO TOTAL RATIO

1. >2
2. <2

PLATELET COUNT

1. <1.5 lakhs/cmm of blood
2. >1.5 lakhs/cmm of blood

BLOOD GLUCOSE

1. <40mg/dl
2. >40mg/dl

BLOOD UREA

1. <25 mg/dl
2. >25 mg/dl

SERUM CREATININE

1. <0.5mg/dl
2. >0.5mg/dl

SERUM BILIRUBIN

1. <5mg/dl
2. 5 to 10 gm/dl
3. 10 to 15 mg/dl
4. >15mg/dl

C-REACTIVE PROTEIN

1. Positive
2. Negative

Micro-ESR

1. >10mm/hr
2. <10mm/hr

ABSOLUTE NEUTROPHIL COUNT

1. <1800/cmm of blood
2. >1800/cmm of blood

CULTURE

1. No growth
2. Positive growth

SEPSIS

1. Clinical sepsis
2. Probable sepsis
3. Proven sepsis

OUTCOME

1. Alive
2. Dead

ABBREVIATIONS

AGE	-	Age
SE	-	Sex
WT	-	Weight
GA	-	Gestational Age
IR	-	Irritability
SC	-	Shrill cry
LE	-	Lethargy
PF	-	Poor feeding
VOM	-	Vomiting
LS	-	Loose Stool
HYT	-	hypothermia
APN	-	Apnea
PU	-	Pustules
AD	-	Abdominal Distention
BT	-	Bleeding Tendencies
CON	-	Convulsion
ART	-	Arthritis
NIC	-	Neonatal intensive care unit
IVF	-	IV fluids
JA	-	Jaundice
ET	-	Endotacheal Intubation
BOOK	-	Booked and Immunised
MI	-	Maternal Illness
MW	-	Maternal weight Gain
Drug	-	Maternal drug intake

DEL	-	Nature of delivery
PRE	-	Prelacteal feed
PAL	-	Pallor
CRT	-	Capillary refilling time
CYN	-	Peripheral cyanosis
ERY	-	Periumbilical erythema
IND	-	Indurations
PUL	-	Peripheral pulse
HR	-	Heart rate
RR	-	Respiratory rate
REFL	-	Reflex
CVS	-	Cardio vascular system
RS	-	Respiratory system
TEN	-	Abdominal Tenderness
BS	-	Bowel Sound
BONE	-	Bone
HB	-	Haemoglobin
TC	-	Total WBC count
ANC	-	Absolute neutrophil count
IT	-	Immature to total count ratio
PLT	-	Platelet
GLU	-	Glucose
UREA	-	Urea
CREA	-	Creatinine
BILI	-	Bilirubin
CRP	-	C-reactive protein

ESR	-	Micro-ESR
BCS	-	Blood culture and sensitivity
USC	-	Urine culture and sensitivity
XRAY	-	X-ray chest and abdomen
CSF	-	Cerebro spinal fluid analysis
MS	-	Maternal high vaginal swab
MUCS	-	Maternal urine culture and sensitivity
MSYM	-	Maternal symptom
CSFS	-	CSF culture
SEP	-	Types of sepsis
OC	-	Outcome

INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : A Study the clinical profile on neonatal late onset Sepsis in a intramural setup

Principal Investigator : Dr.P.Havin Raja

Designation : PG in MD (Paed)

Department : Department of Paediatrics
Government Stanley Medical College,
Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 01.02.2011 at the Modernized Seminar Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate form the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

28/2/11

தகவல் படிவம்

பிறந்த குழந்தைக்கு தாமதமாக ஏற்படும் விஷக்காய்ச்சலின் காரணங்களையும், அதன் தன்மைகளையும் கண்டறிவதற்கான ஓர் ஆய்வு.

தங்களின் குழந்தைக்கு உயிர்க்கொல்லி நோயான விஷக்காய்ச்சலின் தீவிர அறிகுறி உள்ளது. நோயிற்கான காரணத்தையும், அதன் தன்மைகளையும் கண்டறிவதற்காக ஓர் ஆய்வு மேற்கொள்ளபட உள்ளது. தங்கள் குழந்தையின் நோய் குறித்த விபரங்கள் மற்றும் இரத்தம், சிறுநீர் மற்றும் ஸ்கேன் பரிசோதனைகளின் முடிவுகளை தங்கள் சம்மதத்துடன் இவ்வாய்வில் பயன்படுத்த விரும்புகிறோம்.

இதன் மூலம் நோயின் காரணங்களையும் தன்மையும் அறிந்து கொண்டு மருந்துகவ சிகிச்சையை மேலும் மேம்படுத்துவதற்காக மேற்கொள்ளப்பட உள்ள ஆய்வு.

தாங்கள் விரும்பினால் மருத்துவ ஆய்விலிருந்து எப்பொழுது வேண்டுமானாலும். எந்த சட்ட சிக்கலுக்கும் உட்படாமல் தாங்கள் தன் குழந்தையை ஆய்விலிருந்து விலகிக் கொள்ளலாம்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களும், பரிசோதனை முடிவுகளும் தங்களின் ஒப்புதலின் மூலம் மட்டுமே மருத்துவ ஆய்வில் பயன்படுத்தப்படும்.

ஆய்வாளர் கையொப்பம் :

ஆய்வாளரின் பெயர் :

இடம் :

நாள் :

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு

பிறந்த குழந்தைக்கு தாமதமாக ஏற்படும் விஷக்காய்ச்சலின் காரணங்களையும், அதன் தன்மைகளையும் கண்டறிவதற்கான ஓர் ஆய்வு.

ஆராய்ச்சி நிலையம் : அரசு R.S.R.M. மருத்துவமனை, சென்னை-600 001.

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின் எண் :

பங்கு பெறுபவர் இதனை (✓) குறிக்கவும்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது. ☐

எனது குழந்தையை நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்க வைக்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் என் குழந்தையை இவ்வாய்வில் இருந்து விலகிக் கொள்ளலாம் என அறிந்துக் கொண்டேன். ☐

இந்த ஆய்வின் சம்மந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும். இந்த ஆய்வில் பங்கு பெறும் மருத்துவர் என்னுடைய குழந்தையின் மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் என் குழந்தையை ஆய்விலிருந்து விலக்கிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும் பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதை பிரசுரிக்கவும் என முழு மனதுடன் சம்மதிக்கிறேன். ☐

இந்த ஆய்வில் எனது குழந்தை பங்குக் கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி, நடந்துக் கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் குழந்தையின் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராதா வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ அதை உடனே மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதியளிக்கிறேன். ☐

இந்த ஆய்வில் எனது குழந்தையின் இரத்தம், சிறுநீர் எக்ஸ்ரே, இ.இ.ஜி. ஸ்கேன் பரிசோதனை செய்துக்கொள்ள நான் முழுமனதுடன் சம்மதிக்கிறேன். ☐

பங்கேற்பவரின் கையொப்பம்..... இடம்..... தேதி

கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம்.....

ஆய்வாளரின் கையொப்பம்.....இடம்.....தேதி

ஆய்வாளரின் பெயர்.....