"CLINICAL PROFILE OF CANDIDIASIS IN NEONATES"

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In

PEDIATRICS

Under the guidance of

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LIST OF ABBREVIATION

NICU- neonatal intensive care unit

PNC- Post Natal Cases

CoNS- Coagulase Negative Staphylococci

VLBW- Very Low Birth Weight

Efg1- Transcriptional Regulator For Adhesion

SAP- Specific Secreted Aspartyl Transaminase

UTI- Urinary Tract Infection

CSF- Cerebro Spinal Fluid

TRM- Tetrazolium Reduction Medium

GTT- Germ Tube Test

RSA- Rice Starch Agar

CMA- Corn Meal Agar

AG- Agglutination

RIA – Radio Immune Assay

KOH- Potassium Hydroxide

GS- Gram Stain

BA- Birth Asphyxia

MV- Mechanical Ventilation

POS- Positive

ABSTRACT

INTRODUCTION

Opportunistic infections are increasing in Neonatal Intensive Care Unit (NICU). Neonates often have compromised skin integrity, gastrointestinal tract disease, chronic malnutrition, central venous catheters, long term endotracheal intubation and others factors that lead to increased risk of acquiring such infections[1]. Infections with fungi (candida) and with coagulase-negative staphylococci (CoNS) are especially prevalent

Preterm infants are predisposed to candida infections because of immaturity of their immune system. Transmission of candida may be vertical (from maternal vaginal infection) or nosocomial. Invasive fungal infection occurs in approximately 6% to 7% of all infants admitted to the neonatal intensive care unit (NICU) , but the incidence is inversely correlated with birth weight: the lower the birth weight, the greater the risk of invasive fungal infection[4]

METHODS AND MATERIALS

296 babies admitted in NICU (96 babies) and PNC (200 babies) of shri B M Patil medical college, hospital. Bijapur to study clinical profile of candidiasis in neonates and risk factors associated with them .Clinical examination was done and investigations included KOH examination of oral swab, Gram stain of the swab and blood culture of suspected sepsis babies.

RESULTS

In the present study, incidence of candidiasis in neonates revealed 13.5% of babies admitted in NICU. Male babies outnumbered the female babies in incidence of candidiasis in neonates. Males formed 69% and females 31% of positive cases.Most of neonates admitted in NICU (96) were of low birth weight between 1500gm to 2500gm. Out of which most of cases positive for candidiasis/candidemia were belonged to 1000gm-1500gm. In present study, 13 babies were positive for candidiasis,of which 5 babies(38.3%) were of birth weight between 1000gm-1500gm.

Birth asphyxia is an important risk factor in development of candidiasis in neonates. In present study, birth asphyxia has played a significant role in development of candidiasis in neonates admitted in NICU. Mechanical ventilation is also a important risk factor in development of candidiasis in neonates. In present study mechanical ventilation had played a significant role in development of candidiasis in neonates admitted in NICU.

CONCLUSION

The present study revealed the clinical profile of candidiasis in neonates associated with various risk factors. Study shows that low birth weight, birth asphyxia and mechanical ventilation were significant risk factors for candidiasis in neonates. Blood cultures were positive in babies without mucosal lesions suggesting the importance of diagnosing fungal sepsis.

KEY WORDS

Oral candidiasis, birth asphyxia, candidiameia.

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INTRODUCTION

Opportunistic infections are increasing in Neonatal Intensive Care Unit (NICU). Neonates often have compromised skin integrity, gastrointestinal tract disease, chronic malnutrition, central venous catheters, long term endotracheal intubation and others factors that lead to increased risk of acquiring such infections[1]. Infections with fungi (candida) and with coagulase-negative staphylococci (CoNS) are especially prevalent [1].

Candidiasis refers to fungal infections with fungi of genus *candida*. Candidemia is presence of *candida* fungi in the blood. Most of neonatal infections are caused by candida albicans or candida parapsilosis. Preterm infants are predisposed to candida infections because of immaturity of their immune system. Transmission of candida may be vertical (from maternal vaginal infection) or nosocomial. Approximately 10% full term infants become colonised in gastrointestinal tract and respiratory tracts in first 5 days of life[3]. Colonization of health worker is as high as 30%. Initial site of colonization is gastrointestinal tract[2]. Skin colonisation is common after 2 weeks of age[3].

Invasive fungal infection occurs in approximately 6% to 7% of all infants admitted to the neonatal intensive care unit (NICU) , but the incidence is inversely correlated with birth weight: the lower the birth weight, the greater the risk of invasive fungal infection[4].

Host factors that contribute to the susceptibility of the NICU infant to fungal infection include birth weight of less than 1500 g, 5-minute Apgar scores of less than 5, disruption of cutaneous barriers by percutaneous catheters and relative immunocompromise ascribable to reduced numbers of T cells, impaired neutrophil number and function, and reduced levels of complement. Concomitants of nursery care that are thought to increase the risk of fungal infections include prolonged use of antimicrobials (especially third-generation cephalosporins), indwelling central venous catheters, abdominal surgery; parenteral nutrition, parenteral lipid formulations, histamine H_2 receptor antagonists, endotracheal intubation and length of stay more than 1 week.[4]

Risk factors include: a) very low birth weight (<1500gms); b) use of broad spectrum and or multiple antibiotics; c) use of central venous catheters; d) parenteral alimentation and intravenous fat emulsion; e) colonization of candida and or previous episode of mucocutaneous candidiasis; f) prolonged urinary catheterization[2].

Common presenting symptoms of systemic candidiasis are worsening respiratory function, apnea, thrombocytopenia. Localized signs of infections at one or more of following site:

- Skin and mucous membrane:(thrush, diaper rash and other areas)[2]
- Central nervous system: meningitis is present in up to 64% of fatal cases, and survivors have a high incidence of sequelae including hydrocephalus, psychomotor and mental retardation[2].
- Eyes: fundoscopic examination is essential for early diagnosis of candidiasis. Endophthalmitis has been noted in as many as 45% cases. Cotton ball exudates are typical of candidal retinal pathological conditions[3].

- Heart: candidia endocarditis is the second most common form of endocarditis in Very Low Birth Weight (VLBW) infants. Clinical features include cardiac murmurs, petechiae, skin abscess, arthritis, hepatomegaly and splenomegaly[2].
- Kidneys: candida is the most frequent cause of Urinary Tract Infection in intensive care nurseries. Up to 50%of these babies have candidemia and predisposed to renal candidiasis, with development of fungus ball or abscess. Renal manifestation may be the first clinical manifestation of invasive candidiasis[2].
- Arthritis is a complication of 20% of cases[3].

The need of study is to know the clinical profile of candidiasis in neonates in our setup and to determine associated risk factors of candidiasis.

AIMS AND OBJECTIVES

To study the clinical profile & assessment of risk factors of candidiasis in neonates admitted in the neonatal intensive care unit and Post natal wards of Shri B M Patil Medical college, hospital & research center; Bijapur.

REVIEW OF LITERATURE

HISTORY:

Histroy of Oral candidiasis has been recognized since 4th century, the time of Hippocrates in his book "Epidemics" who described oral thrush in debilitated patients[5,6].

The word "thrush" is derived from ancient Scandinavian or Anglo-Saxon words for the disease[6,7]. The French word for the condition is 'le Muguet', meaning 'lily of the valley'. "torsk" is the Swedish synonym for oral thrush[5].

In 1890, Zopf suggested the name of the fungi as Monilia albicans which derived Moniliasis (clinical entity), the early name of Candidiasis[8]. Berkhout in 1923, after recognizing the difference between Monilia species isolated from rotting plants and those isolated from medical cases established the genus Candida to accommodate the later[5].

This was accepted as the official name of the genus by the Eighth botanical congres in Paris in 1954[9]. In 1945, Conant et al described identification of Candida species based on fermentation of glucose, maltose, lactose, sucrose.

In 1948, Wickerham et al described the assimilation method[7].

In 1956, Reynolds and Braude described the germ tube test for identification of C.albicans[7].

In 1959, Vishwanathan and Randhwana isolated C. vishwanthii from India[5].

In 1960, Taschdjian et al, described chalmydospore formation by C. albicans in cornmeal agar[5].

In 1968 Brown Thompson in Denmark observed that different strains of C. albicans produced varying morphologies when streaking on Malt agar[10].

In 1971 Dolan C T gave the identification scheme for yeasts[10].

In 1975 Holt R J gave details of methods for evaluation of sensitivity of the pathogenic fungi to therapeutic agents, media, incubation temperature, drug solution and time[10].

TAXONOMY

The Genus Candida belongs to the phylum Deuteromycota, in class Blastomycetes, in order Moniliasis and family Cryptococcaceae[11].

Genus *Candida* includes more than 163 anamorphic species[5].

Frequent human pathogens are[9]-

1. Candida albicans (Robin, Berkhout) 1923

   Synonym : *Oidium albicans / Monilia albicans/ C.intestinalis*

2. Candida guilliermondii (Castellani)1938

   Synonym : *Endomyces guilliermondii/ Monilia guilliermondii*

3. Candida glabrata (Anderson, Meyer & Yarrow)1978

   Synonym: *Torulopsis glabrata*

4. Candida krusei (Castellani, Berkhout) 1923

   Synonym : Saccharomyces Krusei / Endomyces Krusei/ Monilia parakrusei, Candida lobata

5. Candida parapsilosis (Ashford) 1959

   Synonym: *Monilia parapsilosis*

ECOLOGY

Candida species are recognized to be commensal or normal flora of alimentary tract, upper respiratory tract, female genital tract especially vagina and on the skin. It is known that these species serve to cause endogenous infection due to its commensal nature[12].

According to many literatures the source of candidiasis in humans is mostly endogenous; studies have largely focused on the distribution of yeast flora in patients and in healthy persons[13]. The prevalence of Candida species reported from different anatomic sites varies greatly depending on the subjects sampled and the isolation method used, although C. albicans is most common[6].

In healthy individuals the commensal strain and the infecting strain are same, usually single species. Even recurrent candidiasis is caused by single persistent strain unique to particular patient[14].

VIRULENCE FACTORS

The state of the host is of primary importance in determining Candida pathogenicity. There must be a breakdown of mucosal surfaces or in the host defense for diseases to occur. However, there are factors associated with the organism rather than the host that contribute to its ability to cause disease and explained the differences among species in their pathogenicity.

1 Adherence of Candida species to host cell

Adherence of Candida species to a wide range of tissue types and inanimate surfaces is essential and important in the early stages of colonization and tissue invasion. Germinated C. albicans cells adhere to host tissue more readily than do yeast phase cells[15].

Hyphal dimorphism status of Candida species is still inconclusive, dimorphism may have role as a virulence factor. Hyphal wall protein coded by HWP1 gene and other hyphal growth factors interacts with host receptors (Flucosyl glucosamine, Fibronectin, Arginine-glycine-aspargine) with specific ligand receptor interaction and non specific electrostatic forces, Vanderwaal's forces[16,17].

Hyphae of C. albicans have a sense of touch so that they grow along grooves and through pores (thigmotropism). This may aid infiltration of epithelial surfaces during tissue invasion. This is controlled by Efg1 – transcriptional regulator for adhesion[16].

I. Enzymes

Production of hydrolytic enzymes is important determinant for tissue invasion and Candida species are able to produce 14 different hydrolytic enzymes[6].

a) Specific secreted aspartyl proteinase (SAP)

SAP is an extracellular enzyme. This enzyme coded by SAP gene is important component of pathogenecity and also correlates with the active disease process. It helps in tissue invasion by degradation of keratin, collagen, mucin[18].

SAP2, it degrades immunoglobulins, complements and cytokines. Produced by C. albicans, C.dubliniensis, C. tropicalis and C. parapsilosis[19].

a. Phospholipase production

   Coded by PLB1 gene expression. This enzyme concentrated at hyphal tips have greater potential for invasion by hydrolyzing phospholipids in host cell membrane[20].

b. Alkaline phosphatases

c. Peptidases

d. β- glucosidase

e. plasma coagulases

f. Leucine amino peptidases

g. Metallopeptidases

h. Haemolytic factors

i. Siderophores

II. Yeast hyphae- Dimorphism

Hyphae transformation occurs in active disease and facilitate penetration. These are regulated by regulators *Cph1, Efg, CaTec* which regulate the expression of Hyphal dependent gene - SAP4 and SAP6 and Hyphal independent gene - SAP1 and SAP3 respectively[21,21].

III. Phenotype switching

Soll et al described this phenomenon, which is the reversible morphological variation among the strains of organisms due to various contributing factors like synthetic media, repeated cultivation, prolonged incubation. Also there is change in epitopes expressed on their surfa g fhis contributes to virulence of C.albicans by facilitating its ability to survive, invade tissues and escape from host defenses[23].

IV. Others

Mannan

Thrombin induced platelet induced microbicidal protein

Temperature

Azole resistance

Biofilms

Pathogenesis and Pathology

The most studied candida species C.albicans has several known virulence factor contributing to its pathogenecity that includes adherence to epithelial and endothelial cells, proteinase production, phenotype switching, phospholipase production and antigenic modulation as a result of pseudohyphae formation. The transformation into the hyphal form is observed during an active infection[8].

Most of the manifestations are associated with biofilm formation with the cells in candidosis. Biofilms formation in candida dubilinensis may represent key factor for survival of these species, which seems to be particularly well adapted to colonization of the oral cavity.

NEONATAL CANDIDIASIS:

Candida is a common cause of oral mucous membrane infection and perineal skin infections in newborn infants. Disseminated candidiasis and candidemia have become a frequent problem in NICU[24].

Over the last 2 decades, yeasts have become important nosocomial pathogen, *Candida*species being the most frequent isolate. This rise is largely attributed to extensive use of broad-spectrum antibiotics and advances in medical

field, which contri-bute towards the large pool of susceptible population available for these opportunistic pathogen[25].

Importance of Candida species in nursery and intensive care units (ICUs) is increasingly being recognized. Candida species account for 9-13% of all blood isolates in neonatal intensive care units (NICUs) Although C. albicans has historically been the most frequently isolated species, infections caused by the nonalbicans Candida have been diagnosed with increasing frequency in recent years, notably C. tropicalis, C. glabrata, and C. parapsilosis. Common use of broad-spectrum antibiotics, low birth weight (LBW), prematurity, and intravenous catheter, etc., makes neonates prone to candidemia[25].

The incidence and associated mortality due to candidemia can be influenced by several factors including characteristic of the population at risk, standard of the health care facilities available, distribution of *Candida* species, and prevalence of antifungal resistance. These factors may vary from one geographical region to other. The increased isolation rates of nonalbicans *Candida* species and a gradual shift in the antifungal susceptibility profile have underlined the need to monitor laboratory data for possible emergence of resistance and to select most appropriate antifungal agent for therapy.[26]

Recently, non-albicans *Candida* have emerged as important opportunistic pathogen, notably *C. tropicalis, C. glabrata* and *C. parapsilosis*. This could be because of selection of lesser susceptible non-albicans species due to frequent use of fluconazole.

Incidence:

Approximately 7% of infants weighing less than 1500gm will develop evidence of invasive candidal infection such as candidemia or disseminates candidiasis. This relatively high incidence contrasts with logarthimically smaller incidence (0.6%) of candidemia in infants weighing more than 2500gm[4.]

According to Warris and associates,[27] candidal bloodstream infections can be strongly expected after the third week of admittance in very premature neonates who are mechanically ventilated and treated with multiple classes of antibiotics for a prolonged period. The presence of these risk factors in a "septic" premature infant receiving antibiotic treatment justifies the empiric use of antifungal drugs.

In a study made by Gupta P, Faridi MM, Rawat S, Sharma P[28] would provide the clinical profile and assess the significance of various risk factors contributing to the occurrence of oral candidosis in newborns : Oral candidosis was documented in 3.2% (20/650) cases in the NICU. Acute pseudomembranous candidosis was the most common presentation. The mean age of onset was 10.5 days[28]. Candida albicans was isolated in 50% cases in addition to C. tropicalis, C. paratropicalis, C. krusei, C. glabrata and C. parapsilosis. On univariate analysis, male sex, birth asphyxia and prolonged antibiotic therapy had a significant correlation with occurence of oral candidosis in neonates. Out of these, birth asphyxia was the only factor significantly associated with oral candidosis (OR 8.09, 95% CI 1.34-48.8, p = 0.0226) on multivariate analysis. The multiple logistic regression revealed that birth asphyxia was the only significant factor responsible for oral thrush in newborns[28].

In a study made by NICHD Neonatal Research Network centers Premature neonates and particularly low birth weight infants require invasive diagnostic and aggressive therapeutic interventions, many of which increase the risk factors for developing *Candida* infections . In addition the immaturity of the immune system specially among preterm neonates, which mainly involves T-cells and neutrophils, further predisposes this population to infections [29]. Indeed, the anti-*Candida* activity of lung macrophages in neonates has been shown to be reduced. Initial reports on neonatal candidiasis consistently found *Candida albicans* to be responsible for the majority of cases [30-31]. However, similar to change of the epidemiologic patterns that has been observed in adults, a changing spectrum of species is being noted among neonates. This change is characterized by a progressive decrease in the rate of isolation of *Candida albicans* and an emergence of non-*albicans* species [32-33]. Unlike the situation with adults however, it is *Candida parapsilosis* that is becoming the most prevalent species. In some centers it has replaced *C. albicans* as the most frequent species.

Study from National Nosocomial Infections Surveillance (NNIS) system the incidence of Candida infections is greatest in extremely low birth weight (ELBW) infants with birth weights below 1000 g[33.]

In the Agarwal study *Candida* was the commonest isolate from neonates clinically suspected to have septicemia. Majority (76/90) of the isolates were non-albicans *Candida*. Blood stream infection cases due to non-albicans *Candida* have been reported to range from 14-100%. In a retrospective analysis in an NICU, authors found >11 fold increase in rate of candidemia over a fifteen year period (2.5/1000 discharges in 1981 to 28.5/1000 discharges in 1995(15). A shift from *C.albicans* to

non-albicans was noted by this group,*C. parapsisosis* being most prevalent isolate in latter years. Similar trend was also observed by an Indian group in their study done over a period of ten years. There is marked increase in rate of blood stream infection caused by *Candida*, over last two years at there center[25].

FACTORS DETERMINING NEONATAL CANDIDIASIS:

There are several factors associated with development of neonatal candidiosis. Of them, prematurity, LBW, perinatal birth asphyxia, long term antibiotics, central venous catheters, mechanical ventilation, septicemia, played a major roles in development of candidosis[10].

PREMATURITY: Preterm babies are more prone for neonatal candidosis due to immaturity of the immune system[29]. These babies are more vulnerable to develop neonatal septicemia. Unlike term babies, lack of mature oral microflora these babies develop oral candidosis[29].

LOW BIRTH WEIGHT: Babies weighing less than 2.5 kg prone to develop neonatal candidosis. Significant risk factor to develop candidosis was noticed when babies BW < 1.5 kg. Due to lack of maturity of immune system, integrity of cellular and defensive mechanism of host, these babies develop candidemia and other candidal infections[29.]

CLINICAL MANIFESTATIONS:

1) Skin and mucous membranes

Mucocutaneous candidiasis in neonates may present with the classic thrush, diaper rash and/or any variety of this erythematous rash with papules and/or pustules affecting usually wet cutaneous surfaces[34]. The most common presentation is perineal candidiasis.

Two unique varieties of neonatal candidal skin diseases are:

1. Cutaneous Congenital Candidiasis
2. Invasive Fungal Dermatitis. It is recently described skin disorder[35] affects extremely low birth weight neonates. Characteristically ulcerative and erosive lesions with extensive crusting are seen. More than half of these Candida-related skin infections were associated with the occurrence of invasive candidiasis. Most of theories have postulated that immature skin becomes in these cases the portal of entry for Candida[35-36.]

Oral candidiasis:

Oral candidiasis: oral thrush or oral pseudo membranous candidiasis is a superficial mucous membrane infection that affects approximately 2-5% of normal newborns. Infants acquire candida from their mothers and remain colonized. Thrush may developed as early as 7-10 days of age. The plaques of thrush invade mucosa superficially and may be found on the lips, buccal mucosa, tongue and palate. Removal of plaques from these surfaces may cause mild punctuate areas of bleeding which helps to confirm the diagnosis[37].

Oral candidiasis in the breast fed infant is often associated with the superficial or ductal candidiasis in the mother's breast. Concurrent treatment

of both the mother and infant is necessary to eliminate the continual cross infection.

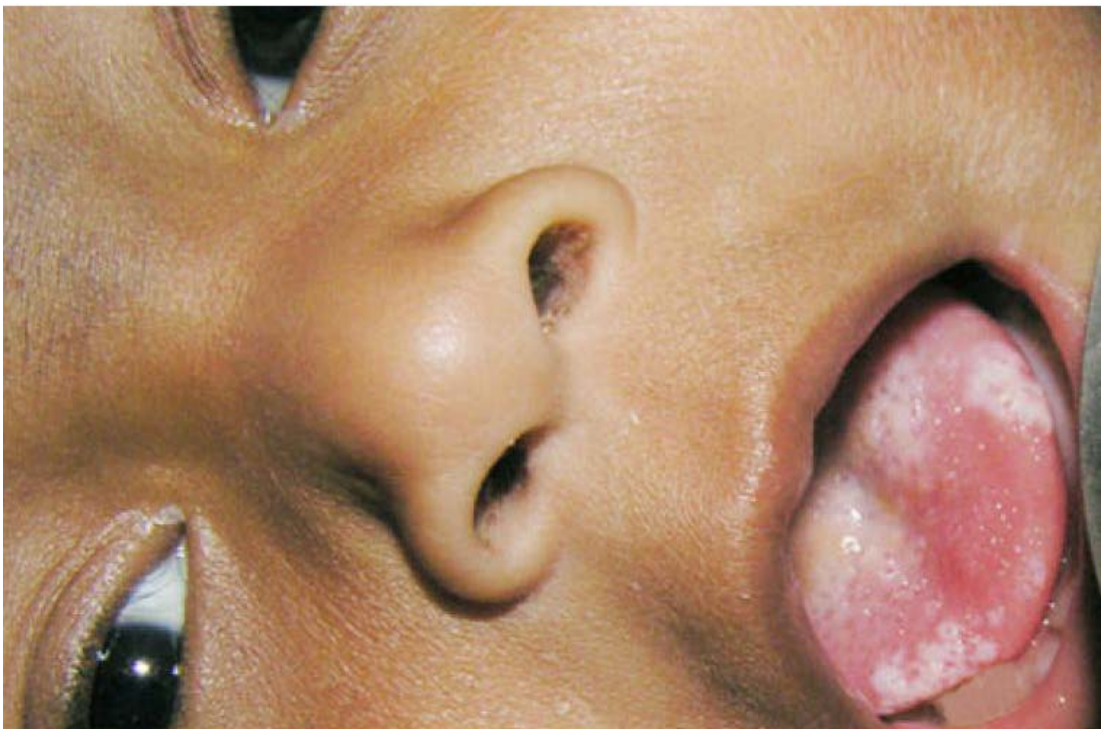


Figure:1 ORAL CANDIDIASIS IN NEONATE

2) Kidneys:

Candidal UTI is the most frequent cause of urinary tract infection in the NICU[38]. About half of these babies are found to have concomitant candidemia[38]. Most of babies suffering from renal candidiasis, refers to renal fungus balls or renal fungal abscesses. Renal insufficiency could be the first clinical manifestation of invasive candidiasis[39].

Isolation of Candida species from a catheterized specimen or suprapubic aspiration is a reliable indicator of infection, although asymptomatic colonization of urinary catheters, stents, or nephrostomy tubes can be difficult to distinguish from true infection[40].

The presence of candiduria in the NICU infant is associated with renal candidiasis - the latter manifested by cortical abscesses or fungal mycelia in the collecting system ("fungus balls")-nearly half the time. Thus, in contrast to older children or adults, the finding of candiduria in the NICU infant should prompt blood cultures and renal imaging at the very least. If blood cultures prove to be positive, a full evaluation for disseminated candidiasis should be undertaken[41].

Diagnosis requires the isolation of Candida species from suprapubic aspiration or catheterized urine specimens. Because of the high prevalence of associated upper tract disease, imaging of the kidneys by ultrasonography should be performed on isolation of the organism from a sterile urine specimen[41].

3) Candidemia / Line Infections:

The median time of onset is at approximately 30 days of age . In a large multicenter study, colonization of the gastrointestinal tract preceded candidemia in 43% of case, a frequency suggesting that other sites of colonization, such as intravascular catheters or endotracheal tubes, may contribute to the risk of candidemia[42].

A variety of nonspecific clinical symptoms may be associated with this presentation of candidal disease, including respiratory decompensation, feeding intolerance, temperature instability, and mild thrombocytopeuia.

Although C. albicans and C. parapsilosis account for almost 90% of candidemias, other species such as C. krusei are increasing in prevalence and may be resistant to azoles.[43,44]

Candida species isolated from a blood culture should never be regarded as a contaminant but should prompt an immediate search for evidence of disseminated disease, which occurs in approximately 10% of premature newborns[45,46].

- A thorough evaluation includes ophthalmologic examination and ultrasonography of the heart, venous system, and abdomen. When lumbar puncture is performed in the evaluation 'for disseminated candidiasis, as many as 50% of candidmic infants may be found to have associated meningitis.

Numerous studies have shown that central venous catheters should be removed within 24 hours after the diagnosis of candidemia in particular, removal of the central venous catheter within 3 days is associated with a significantly shorter median duration of candidemia (3 versus 6 days) and a reduced mortality rate[47].

4) Central nervous system:

Among neonates, Candida meningitis is one of the most common manifestations of invasive candidiasis[48,49,50]. Up to 64 percent of neonates dying with invasive candidiasis have CNS involvement and more than 2/3 of these babies have positive CSF cultures at some point in their disease[50].

Neurological clinical manifestations in this particular population are few and related to increased intracranial pressure (bulging fontanelle and splitting sutures). Instead, general signs of sepsis and progressive clinical deterioration are commonly found. In other words, Candida meningitis usually presents as part of the syndrome of invasive or disseminated candidiasis. Therefore, a physician dealing with sepsis in a high risk neonate, should suspect Candida meningitis if

Candida spp. is recovered from the blood, urine or other site suggestive of heavy colonization[50].

Candida meningitis carries a high rate mortality and for survivors a high incidence of severe sequelae (hydrocephalus, psychomotor retardation, and aqueductal stenosis)[51].

5) Eyes :

The use of fundoscopic exam has been recommended as a tool for early diagnosis of invasive disease. The only prospective study evaluating neonates with either candidemia or CSF positive for Candida found an incidence of Candida endophthalmitis of 50%[52].

6) Heart:

Candidal endocarditis has been found to be the second most common form of endocarditis in this age group[53,54]. Clinically, classic findings are expected, including cardiac murmurs, petechiae, skin abscesses, arthritis, hepatomegaly and splenomegaly. Right-sided intracardiac fungal masses can manifest with heart failure or even with pulmonary fungal embolism[55,56,57].

7) Congenital Candidiasis

Congenital candidiasis is a rare clinical entity in which intrauterine candida infection becomes manifest at birth. Congenital candidiasis is not related to vaginal delivery, premature rupture of membranes, prematurity, maternal age, duration of labor or parity[58,59]. Intrauterine contraceptive devices have been frequently associated with this condition[60,61,62]. Two forms of disease have been described.

1. Congenital cutaneous candidiasis

   In this case an extensive skin rash becomes manifest within the first 12 hours of life[63]. A macular erythema that may evolve from a pustular, papular or vesicular phase finally results in extensive desquamation Paronychia and dystrophy of the nail plates have been also described[63,64]. The most commonly affected areas include the trunk, neck, face and extremities[63]. These cutaneous lesions usually resolve spontaneously or after short courses of oral nystatin[63.]

2. Congenital systemic candidiasis

   In certain cases, the picture may evolve to an invasive infection and death, particularly in very low birth weight infants[65]. This form of the disease has a high mortality rate. Importantly, at least half of the cases do not develop the cutaneous phase previously described.[66] Pneumonia with respiratory distress is the most common presentation of systemic or invasive candidiasis. Other presentations include candidal meningitis, candiduria and/or candidemia[64,66,67,68].

8) Bones and Joints

   Candida spp. have been repeatedly listed among the three most common agents causing neonatal arthritis[69,70,71]. Warmth and fusiform swelling of the lower extremities in combination with radiographic evidence of osteolysis and cortical bone erosion are the expected findings in cases of candidal osteomyelitis and/or arthritis in the neonate[72,73].

Laboratory Diagnosis:

An early diagnosis of candidiasis should be established as these infections have high mortality. The difficulty in diagnosis lies due to the absence of specific symptoms and signs as well as opportunistic nature of the yeasts. The following protocol is instituted to validate the diagnosis of suspected cases of candidiasis in the laboratory.

Direct Examination

The clinical specimens are collected depending upon the site of involvement i.e. from superficial lesions or deep-seated infections[5]. White patches from the mucous membrane of the mouth are collected with the help of sterile swabs. These are examined in KOH wet mount or normal saline preparation. Gram staining is performed to see the presence of yeast and pseudohyphae of Candida species. The yeast cells are approximately 4-8 μm with budding and pseudohyphae. The presence of pseudohyphae shows colonization and tissue invasion hence their demonstration in the direct smear of tissue is highly significant. Calcofluor white stain may also be used to highlight the fungal elements[5].

The biopsy specimens are kept in tube containing KOH for an overnight period at 37^0 C and after mincing these are examined under the microscope for yeast cells and pseudohyphae. Other stains like H&E and Gomori's methenamine silver stain are also done for the demonstration of fungal elements in tissues[5].

Fungal Culture

The clinical specimens can be cultured on Sabouraud dextrose agar with antibacterial antibiotics and incubated at 25° C and 37° C. The colonies appear in 3-4 days as cream colored, smooth and pasty. Sometimes growth may be observed after an overnight incubation as seen in bacteria. The bacterial culture media like blood agar may also be used for growing the Candida species. The LCB mount is prepared from the colonies to examine for the presence of yeast cells and pseudohyphae[5].

For systemic infection like candidemia, blood culture is done in biphasic medium like brain- heart infusion agar-broth and incubated at both the temperatures. The detection of Candida species in blood culture is one of the most important advances in the recent diagnostic procedures because in earlier times, blood culture techniques often did not recover Candida species[5].

The growth of Candida species is also seen on Tetrazolium Reduction Medium (TRM) and compared to the standard colors to identify various species. The Candida isolates are identified by standard protocols that include germ tube formation, chlamydospore production on cornmeal agar and sugar fermentation and assimilation tests. The salient cultural and morphological features pertaining to individual species of Candida are given below[5]:

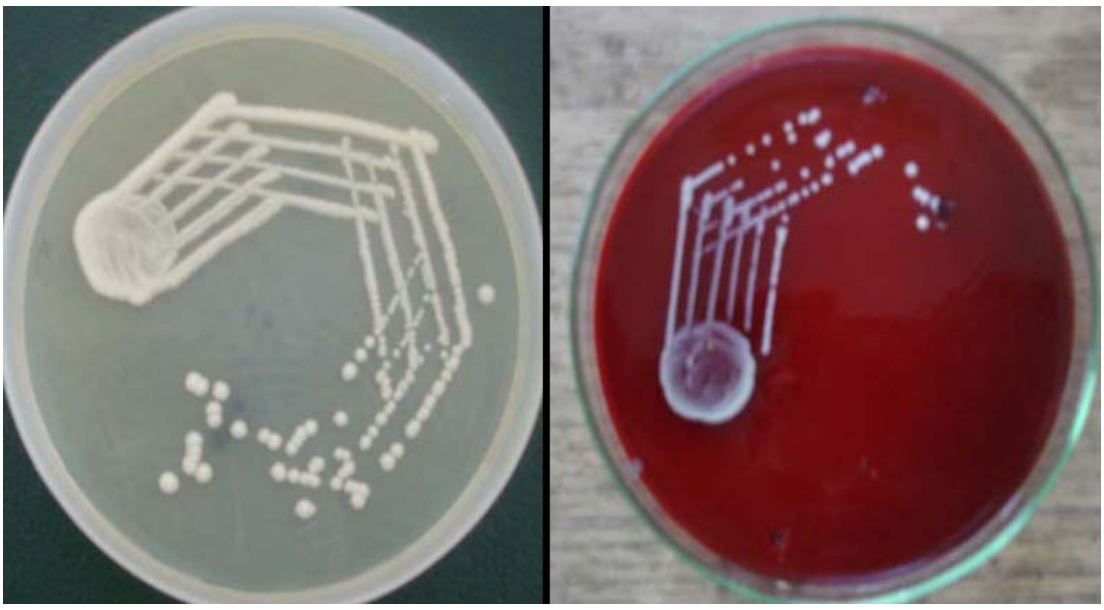
Candida albicans – (Latin - candidus – glowing white) Candida albicans is an opportunistic fungal pathogen with host interaction abilities that range from commensal through life-threatening disseminated diseases[5].

The interplay between Candida and host defense is essential in determining outcome of the infection. The colonies are cream- colored, pasty and smooth. On

cornmeal agar at 25° C, there are large thick-walled, terminal chiamydospores, characteristic of this species[5].

On CHRO Magar Candida, the colonies of C.albicans appear as light green to bluish-green in colour. Due to the relatively high DNA homology between C.albicans and C.stellatoidea, the latter has been re-classified as sucrose negative variant of F.albicans[5.]

Candida tropicalis - C.tropicalis cannot be differentiated from C.albicans on colony morphology or growth rate[5]. The colonies are cream-colored to off white, glistening to dull, soft, smooth or wrinkled with mycelial fringe. On cornmeal agar it forms blastospores singly or in small groups it has gained more importance in patients with neutropenia and hematologic malignancies[5]. There is thin pellicle on primary cultures requires application of newer strategies.



(A) (B)

FIG:2 : GROWTH ON (A) SEBOURAUD DEXTROSE AGAR AND (B) BLOOD AGAR

Germ Tube Test

This procedure is used for presumptive identification of Candida species and is also called as germ tube test (GTT)[5]. The culture of Candida species is treated with sheep or normal human serum and incubated at 37°C for 2-4 hours. A drop of suspension is examined on the slide under the microscope. The germ tubes are seen as long tube- like projections extending from the yeast cells. There is no constriction at the point of attachment to the yeast cell as seen in case of pseudohyphae[5].

FIG:3: GROWTH ON SEBOURAUD DEXTROSE AGAR

Chlamydospore Formation

The suspected strain of the Candida isolates grown on cornmeal agar (CMA) or rice starch agar (RSA) and incubated at 25°C. It shows the formation of large, highly refractile, thick-walled, terminal chlamydospores after 2 to 3 days of incubation . These are seen in the clinical isolates of C.dubliniensis[5].

Chlamydospores are refractile, thick-walled cells that are produced under nutrient poor; oxygen limited conditions at low temperatures. There are several media, such as corn meal agar (CMA) and cream rice agar, which have been used for the production chlamydospore Recently, media such as Staib's agar, Pal's agar, casein agar and tobacco agar have been used for the production of chlamydospores[5].

CHRO Magar Candida

CHROMagar Candida is rapid, plate-based test for the simultaneous isolation and identification of various Candida species[5]. This is relatively new medium that distinguishes different Candida species by color as a result of biochemical reactions. This can be used for simultaneous isolation and presumptive identification of various Candida species like C.albicans, C.krusei, C.tropicalis, C.gtabrata[5].

Candida species	Glu	Mal	Suc	Lac
C.albicans	AG	AG	-	-
C.tropicalis	AG	AG	AG	-
C.kefyer	AG	AG	AG	-
C.guilliermondii	AG	-	AG	-
C.parapsilosis	AG	-	-	-
C.krusei	AG	-	-	-
C.glabrata	AG	-	-	-

Fermentation Reactions of candida species

Assimilation reactions of different candida species and their growth on Tetrazolium Reduction Medium.

Candida Species	Glu	Mal	Suc	Lac	Cel	Gal	Tre	Raff	Mel	Xyl	Ino	Dul	TRM
Candida albicans	+	-	-	-	-	-	+	-	-	+	-	-	PP
C.tropicalis	+	-	-	-	-	-	+	-	-	+	-	-	M
C.kefyer	+	-	-	-	-	-	-	-	-	+	-	-	SP
C.parapsilosis	+	-	-	-	-	-	+	-	-	+	-	-	RP
C.guilliermondii	+	-	-	-	-	-	+	-	-	+	-	+	PsP
C.krusei	+	-	-	-	-	-	-	-	-	+	-	-	DP
C.glabrata	+	-	-	-	-	-	+	-	-	+	-	+	PP

Note : Glu = Glucose, Mal Maltose, Suc = Sucrose, Lac = Lactose, Cel = Cellobiose, Gal Galactose, Tre = Trehalose, Raf Raffinose, Mel = Melibiose, Xyl = Xylose, mo Inositol, Dul = Dulcitol; + = Positive Reaction, — Negative Reaction, V=Variation.

Tetrazolium Reduction Medium (TRM) : PP = Pale Pink, OP = Orange Pink, M = Maroon, SP = Salmon Pink, RP = Rose Pink, PsP Pink and Pasty, DP = Pink and Dry.

Immunodiagnosis

Numerous serological and molecular techniques have been developed for the diagnosis of Candida species[5]. Efforts have been made to find either antibody against Candida albicans molecules or Candida-derived molecules whose presence in patients' sera could indicate tissue invasion. Tests have been developed to detect Candida albicans proteins, metabolites, DNA and polysaccharides[5]. Various approaches for the serological diagnosis of candidiasis have been concentrated on the detection of Candida albicans-derived molecules[5]. These molecules have been

detected either on the basis of their antigenicity or through biochemical-enzymatic procedures. More recent progress has been made on the latter methods and kits are commercially available for the detection of arabinitol and glucans whereas PCR-based tests for Candida-DNA detection routinely performed in some of the laboratories[5].

Mannari is major structural component of the cell wall of yeasts and the principal surface antigen that is available for immune interaction with colonized or infected hosts. It is large molecular weight protein polysaccharide whose carbohydrate portion contains backbone chains of repeating mannose units in a (1,6) - linkage and numerous a (1,2) - and a (1,3) - oligomannoside side chains. The whole cell agglutination by specific antisera identifies two major serotypes of C.albicans, serotype A an4, B and mannan comprises the type specific antigen[5].

A positive test does not necessarily indicate infection ‘since the antigens used are unable to differentiate antibodies formed during mucosal colonization and from those produced during deep infection[5].

Antigen tests based on ELISA, RIA, CIE. PHA and LPA to detect either cell wall mannan or cytoplasmic components are being developed and antigen detection is likely to become the main method for serodiagrosis of systemic candidiasis[5].

Detection of Metabolites

There are certain species of Candida produce the metabolite D-arabinitol including C.albicans, C. tropicalis, C.parapsilosis, C.guilliermondii and C.pseudotropicalis but not C.krusei or C.glabrata. Serum arabinitol can be measured by gas liquid chromatography[5].

Serological Tests for Invasive Candidisis

A) Detection of Antibodies

Slide agglutination

Immunodiffusion

Phytohaemagglutination

Coelectosynersis

Immunoprecipitation

A and B immunofluorescence

B) Nonspecific Candida Antigens

Latex agglutination

Immunobloting

C) Cell Wall Components

Cell Wall Mannoprotein (CWMP)

β-(l,3)-D-glucan

D) Candida Enolase Antigen Testing

D-mannose and D-arabinitol are metabolites of Candida species that can be detected in sera by gas liquid chromatography. By using suitable standard, serum concentrations – of the metabolites can be found out. The detection of circulating candidal antigens as diagnostic marker of disseminated candidiasis has been the subject of intensive research for more than two decade[5].

1) Temperature instability
2) Hypotension
3) Respiratory deterioration and apnea
4) Abdominal distension

5) Guaiac positive stools

6) Carbohydrate intolerance

TREATMENT:

The ways in which neonates differ in terms of relevant strategies are increasingly appreciated. Important differences on the pharmacokinetics but particularly on the toxicity profile of available antifungal agents have been demonstrated.

1 Amphohotericin B

Initial reports on the use of amphotericin B in neonates were somewhat alarming. In particular, the report by . implicated this agent in a high mortality rate in 10 infants with invasive candidiasis and caused skepticism among neonatologist[74,75].

In a study by Johnson et al. did not find a single case of significant renal toxicity with amphotericin B in a group of 21 infants (birth weight < 1,500 grams) with neonatal candidiasis treated with this agent. In addition, the classic infusion-related side effects, fever, chills, nausea and vomiting are especially seen in this population.. And even when amphotericin B is known to inhibit erythropoietin production, anemia has not been described as a significant finding among babies treated with this drug[76,77]..

The lipid preparations of amphotericin B are represented by 3 different commercially available products composed of different phospholipids and sterols,each with different physiochemical properties: amphotericin B lipid complex, amphotericin B colloidal dispersion, and liposomal amphotericin B.

Indeed, the use of amphotericin B alone for the treatment of neonatal candidiasis has been advocated by some references in view of the lack of an intravenous formulation of 5-fluorocytosine and the immaturity of the GI tract in neonates[76]. A retrospective analysis of such approach revealed that transient azotemia, elevations in serum creatinine and hypoalkemia occurred in about half of cases, but all these complications were satisfactorily managed with short interruptions of therapy or adjustment in dosing intervals. In addition, a comparison of the mortality rate of these infants treated without 5-fluorocytosine with the one reported by authors using the classic combination revealed they were similar or even lower[76].

2 5-fluorocytosine

Study by Johnson et al. also emphasized the lack of cases of either bone marrow or liver toxicity when using 5-fluorocytosine for the treatment of neonatal candidiasis. They used doses of between 20 to 200 mg/kg/day[78]..

3 Fluconazole

More recently a single randomized study compared fluconazole with amphotericin B for the treatment of 23 infants with neonatal candidiasis [79]. However, a heterogeneous group of babies was included. Single stool culture positive for Candida was accounted as, a criteria for invasive candidiasis in one case. Therefore, no major conclusions can be made from this study. There is no doubt that fluconazole deserves further evaluation[79].

In conclusion, amphotericin B alone or in combination with 5-fluorocytosine remains the standard of care for neonatal candidiasis. The

optimal duration of therapy is unknown. However, it is recommended to complete a minimun of 10 to 15 mg/kg of amphotericin B in cases of uncomplicated catheter-related candidemia and between 25 to 30 mg/kg total for patients with invasive disease. When using 5-fluorocytosine, 100 mg/kg/day given in four equal doses is recommended [76].

METHODOLOGY

1. Materials and Methods:

It is a prospective study to ascertain clinical profile of fungal infections & its incidence in neonates admitted in Shri B M Patil Medical College, Hospital & Research Center; Bijapur.

Source of data:

All the neonates admitted in the neonatal intensive care unit and post natal ward of Shri B M Patil Medical College, Hospital & Research Center; Bijapur.

Duration of study- 1st November 2010- 31st march 2012

2. Methods of collection of Data:

After taking written informed consent from the parents and fulfilling inclusion and exclusion criteria of neonates will be included in the study.

Method of study:

1) Neonates hospitalized for more than three days will be serially studied until discharge from the neonatal intensive care unit,
2) Detailed physical examination will be undertaken to look for mucocutaneous candidiasis in newborns.
3) Scrapings from oral thrush, diaper rash, & skin rash will be examined for presence of candida species by Gram Stain and KOH preparation.
4) Gram staining will be done to identify fungi.
5) KOH mounting will be done to see the fungus clearly.
6) Blood culture to diagnose systemic candidiasis will be done in babies positive for mucocutaneous candidiasis.

Blood culture for candidiasis will also be done in babies presenting with septicemia.

7) Species identification will be done using biochemical tests.
8) Newborns with mucocutaneous candidiasis will be started with local antifungal application and will be monitored for response.
9) Babies showing positive blood culture will be started on systemic antifungal drugs and will be followed by blood culture after one week.
10) Babies showing positive blood culture will be followed in OPD once in a week or 15 days.

FIG : 4: LABORATORY CHEMICALS USED TO DEMONSTRATE CANDIDA ORGANISMS

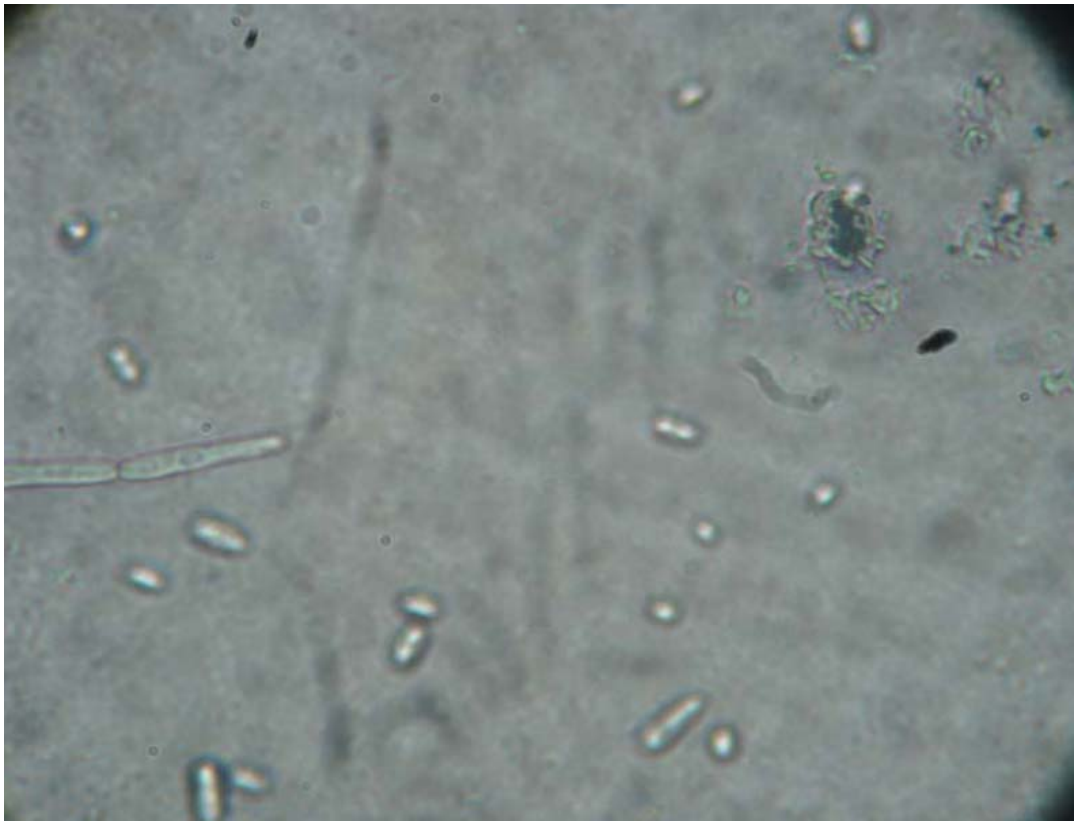


FIG: 5: KOH MOUNT TO DEMONSTRATE CANDIDA HYPHAE WITH BUDDING OF YEAST CELLS.

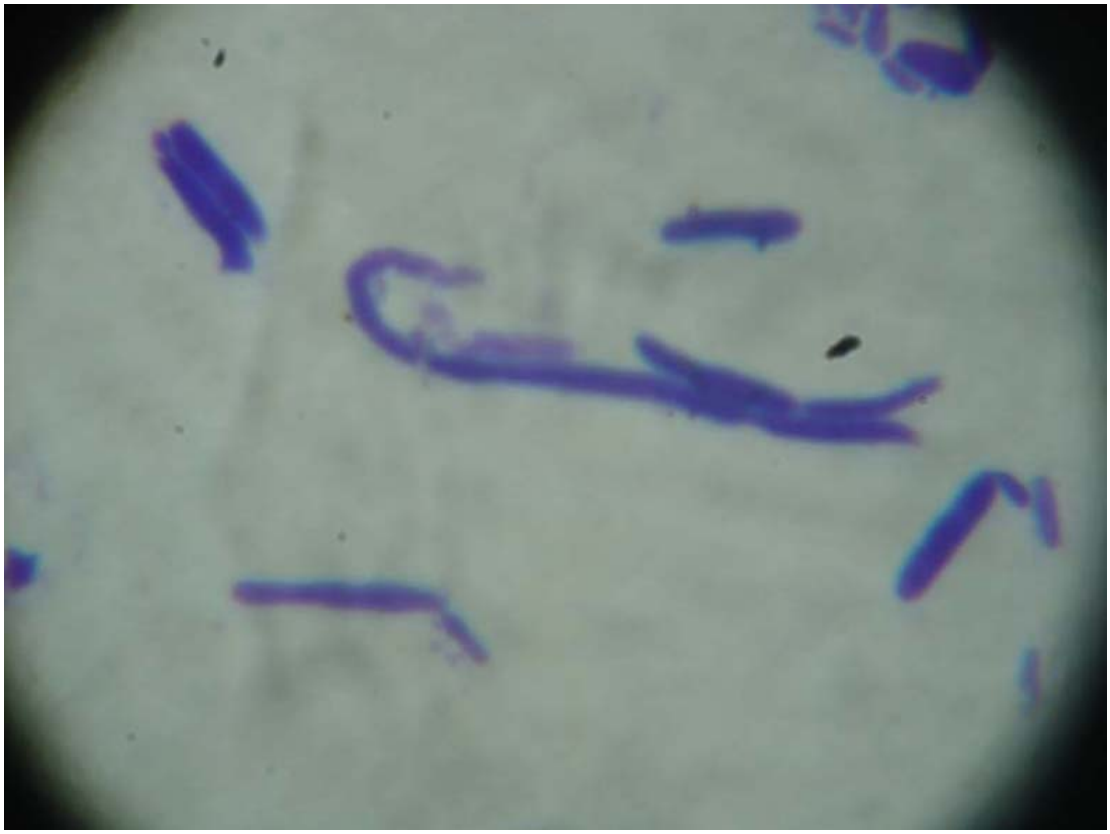


FIG:6 : GRAM STAIN TO DEMONSTRATE CANDIDA HYPHAE

Sample size;

Determination of sample size (n)

With 10% proportion of candidiasis in neonates[3] and considering 90% confidence limit, +/-3 margin of error, required sample size is 384 by using formula

$$n = \frac{Z^2 pq}{d^2}$$

$$= \frac{(1.96)^2 x\ p\ x\ q}{(d)^2}$$

= 384 sample size

i.e n = sample size

Z = Table value of the standard normal variant (SNV)

p= proportion of neonates having the Disease

q= proportion of neonates not having the Disease

d= margin of error for this formula d value specified as ± 3

Statistical Data

For prospective study the parameters may be

1) Expressed in terms of percentage & represent there by suitable diagram like bar/pie diagram
2) Calculate mean / Standard deviation
3) If any situation of comparison between the parameters over the different age or birth weight there we may use t/z/f test (statistical test) for testing significant variation between the parameters under the study.

Laboratory evaluation:

1) Scrapings from oral thrush, diaper rash, & skin rash was examined for presence of candida species by Gram Stain and KOH preparation.
2) Blood culture to diagnose systemic candidiasis was done in babies positive for mucocutaneous candidiasis.
3) Blood culture for candidiasis was done in babies presenting with septicemia.
4) Species identification was done using biochemical tests.

Selection criteria

Inclusion criteria

All neonates admitted in the neonatal intensive care unit of Shri B M Patil Medical college, hospital & research center; Bijapur.

All Neonates admitted in Post natal wards of Shri B M Patil Medical College, Hospital & Research Center; Bijapur.

Exclusion Criteria

i. Neonates already on antifungal drugs for suspected candidiasis.
ii. Neonates which died within 48hrs of admission because of obvious causes like severe birth asphyxia and fatal congenital anomalies

OBSERVATION

INCIDENCE

296 babies admitted in NICU and PNC ward of shri B M Patil medical college hospital, had been evaluated for candidiasis in neonates. Out of 296 babies, 96 babies were admitted in NICU, rest of them were admitted in PNC. Out of 96 babies admitted in NICU, 13 babies(13.5%) were positive for candidiasis including both gram stain and culture for candidiasis . None of babies admitted in PNC were positive for candidasis.

CASES	POSITIVE	PERCENTAGE	NEGATIVE	PERCENTAGE	TOTAL
NICU	**13**	**13.5%**	**83**	**86.5%**	**96**
PNC	**0**	**0**	**200**	**100**	**200**
	13		**283**		**296**

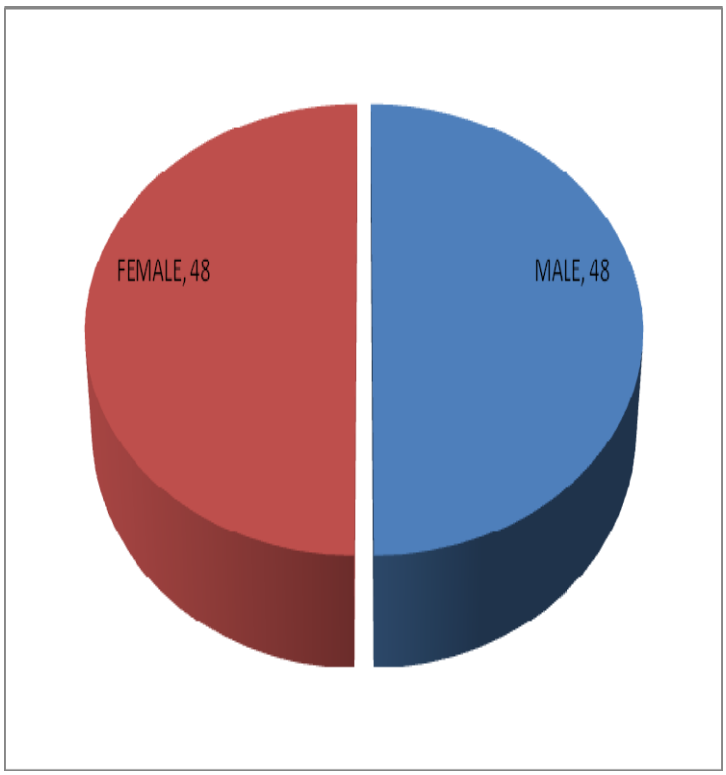
TABLE-1: INCIDENCE OF NEONATAL CANDIDIASIS IN NICU AND PNC

SEX DISTRIBUTION:

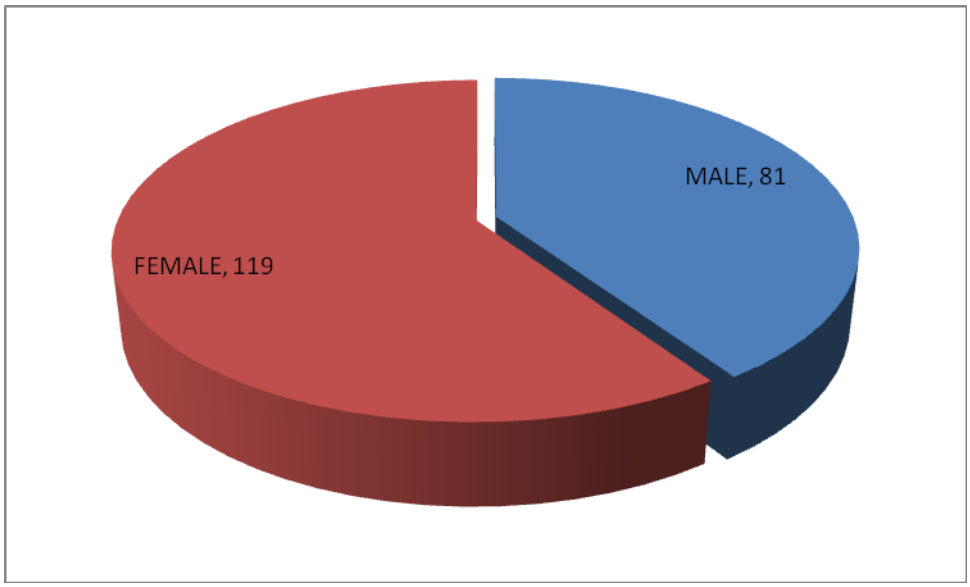
Out of 296 babies, 96 were admitted in NICU and evaluated for candidiasis. Remaining 200 babies were screened for candidiasis admitted in PNC. Among babies admitted in NICU have equal sex distribution, compare to babies screened for candidiasis in PNC. Out of 13 positive cases, male babies had more incidence of candidiasis 9(69%) as compared to females 4(31%). However the results were not significant($p > 0.3$)

SEX	NICU	POSITIVE CASES	PNC	POSITIVE CASES
MALE	48	9 (69%)	119	0
FEMALE	48	4 (31%)	81	0
TOTAL	96	13	200	0

TABLE-2: SEX DISTRIBUTION IN NICU AND PNC.



GRAPH 1: SEX DISTRIBUTION IN NICU



GRAPH 2: SEX DISTRIBUTION OF BABIES ADMITTED IN PNC

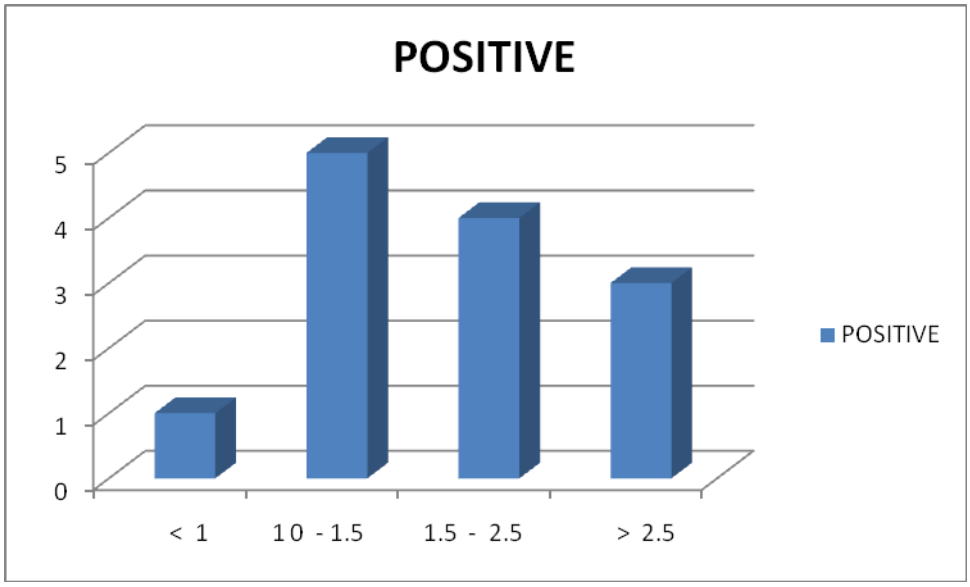
BIRTH WEIGHT AND POSITIVITY OF GRAM STAIN AND CULTURE:

Among 296 babies evaluated for candidiasis, 96 babies were admitted in NICU. Maximum number of babies were between 1500 gm to 2500gm(n=47). Babies more than 2500gm were 38 .

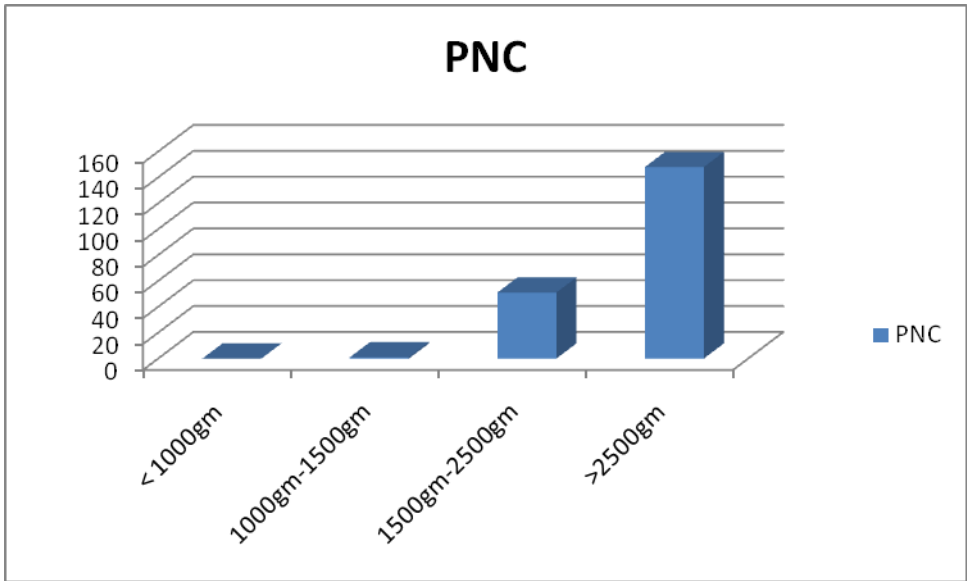
Birth weight(BW)	NICU	Positive gram stain and culture for candidosis.	Percentage	P value	PNC	Positive gram stain and culture for candidosis.	Percentage
< 1000gm	1	1	100%		0	0	0%
1000gm-1500gm	10	5	50%	P<0.0313	1	0	0%
1500gm-2500gm	47	4	8.5%		51	0	0%
>2500gm	38	3	7.8%		148	0	0%
Total	96	13	13.5%		200	0	0%

TABLE:2 DISTRIBUTION OF GRAM STAIN AND BLOOD CULTURE POSITIVE CASES FOR CANDIDOSIS IN RELATION TO BIRTH WEIGHT IN NICU.

Out of 13 babies positive for both gram and culture, maximum number of babies were between 1000gm to 1500gm (5out of 13) 38.3%, p<0.0313 in NICU.



GRAPH:3 DISTRIBUTION OF GRAM STAIN AND CULTURE POSITIVE CASES FOR CANDIDOSIS IN RELATION TO BIRTH WEIGHT IN NICU.



GRAPH: 4 DISTRIBUTION OF CASES FOR CANDIDOSIS IN RELATION TO BIRTH WEIGHT IN PNC.

RISK FACTORS OF CANDIDIASIS IN NEONATES:

BIRTH ASPHYXIA:

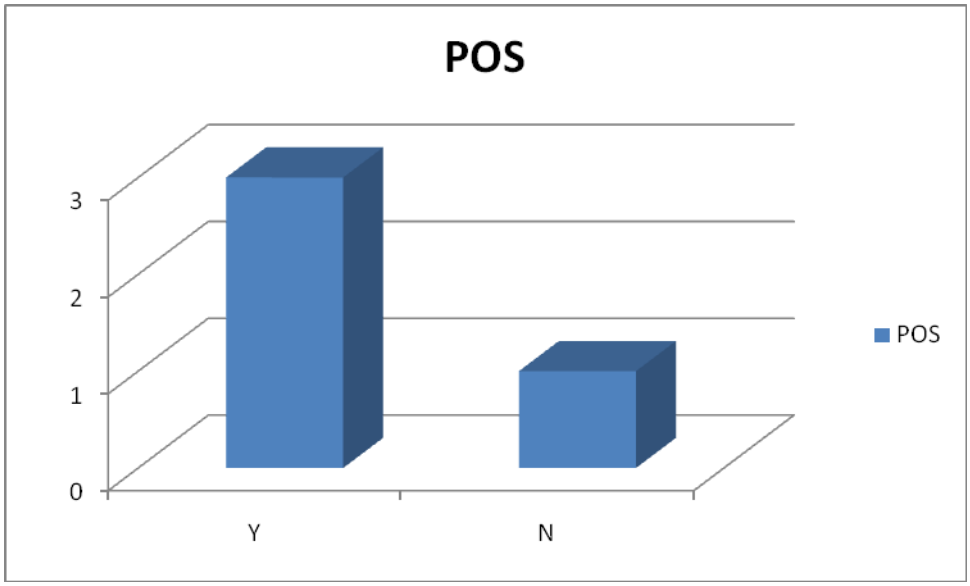
Among 96 neonates admitted in NICU, 19 had birth asphyxia. 5 neonates of which developed oral candidiasis (P<0.004, CI-0.079) and were positive with gram stain and 3 babies had developed candidemia(P<0.004) . Babies born in PNC had no significant relation to birth asphyxia.

BA	GS		TOTAL	PNC		TOTAL	P value
	POSITIVE	NEGATIVE		POSITIVE	NEGATIVE		
Y	5(55%)	14	19	NIL	NIL		0.004
N	4(44%)	73	77	NIL	200		
	9	87	96		200	296	

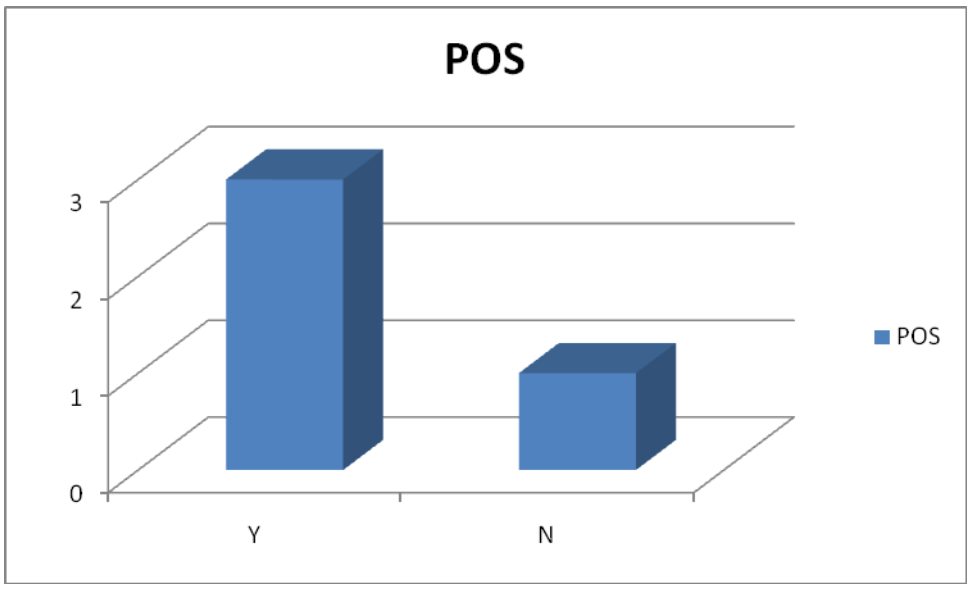
TABLE 4: DISTRIBUTION OF GRAM STAIN POSITIVE CASES IN RELATION TO BIRTH ASPHYXIA IN NICU AND PNC.

BA	BLOOD CULTURE		Total	P value
	POSITIVE	NEGATIVE		
Y	3(75%)	16	19	0.004
N	1(25%)	76	77	
	4	92	96	

TABLE:5 DISTRIBUTION OF BLOOD CULTURE POSITIVE CASES IN RELATION TO BIRTH ASPHYXIA IN NICU



GRAPH:5 : DISTRIBUTION OF GRAM STAIN POSITIVE CASES IN RELATION TO BIRTH ASPHYXIA IN NICU



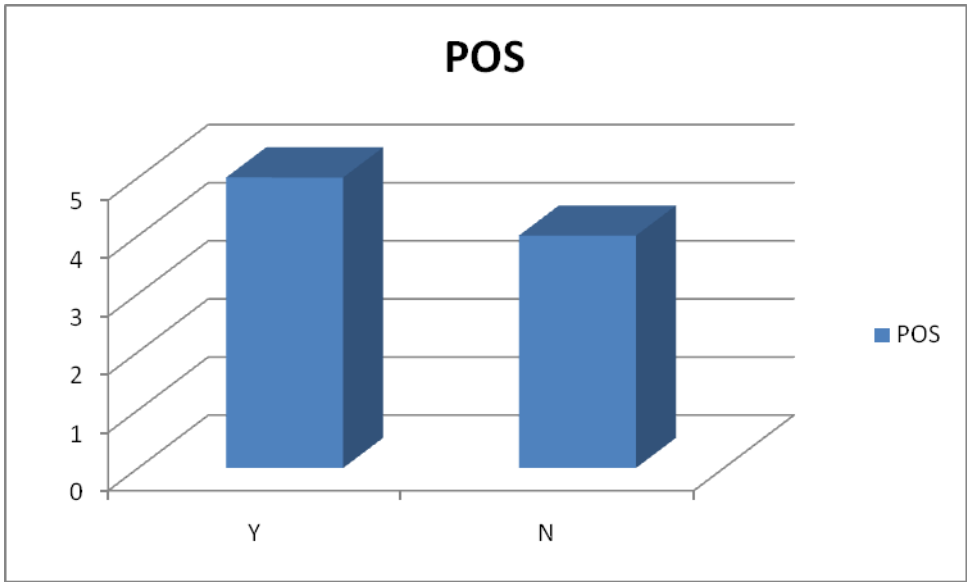
GRAPH:6 DISTRIBUTION OF BLOOD CULTURE POSITIVE CASES IN RELATION TO BIRTH ASPHYXIA IN NICU.

MECHANICAL VENTILATION:

96 neonates were admitted in NICU, of which 13 babies needed mechanical ventilation support, of which 5 neonates developed oral candidiasis and were positive with gram stain (p<0.0001,CI-0.067).

MV	GS		Total	P value
	POSITIVE	NEGATIVE		
Y	5(55%)	8	13	0.0001
N	4(44%)	79	83	
	9	87	96	

TABLE:6 DISTRIBUTION OF GRAM STAIN POSITIVE CASES IN RELATION TO MECHANICAL VENTILATION IN NICU



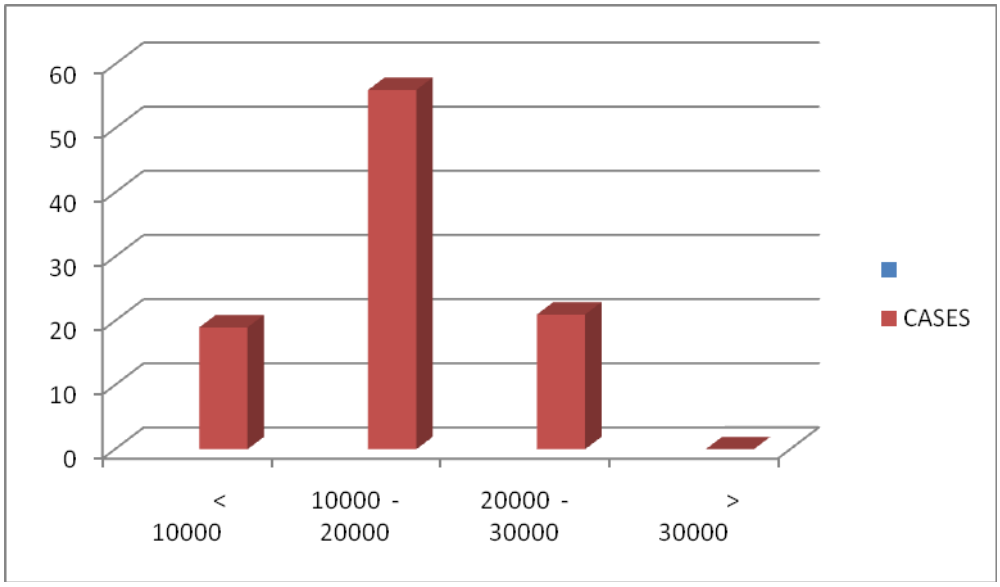
GRAPH:7: DISTRIBUTION OF GRAM STAIN POSITIVE CASES IN RELATION TO MECHANICAL VENTILATION IN NICU

HEMATOLOGICAL PROFILE:

96 babies admitted in NICU underwent septic screening like total leucocyte count, CRP, blood culture. Out of 96 babies, 56 cases has increased total counts compare to other groups($p<0.8$). Among 13 positive cases, most of neonates had total counts between 10000 to 30000.

TC	CASES	POSITIVE CASES	PERCENTAGE	P value
< 10000	19	3	15%	
10000 - 20000	56	5	8.9%	0.8
20000 - 30000	21	5	23%	
> 30000	0	0		

TABLE:7 DISTRIBUTION OF GRAM STAIN AND BLOOD CULTURE POSITIVE CASES IN RELATION TO TOTAL LEUCOCYTE COUNT IN NICU



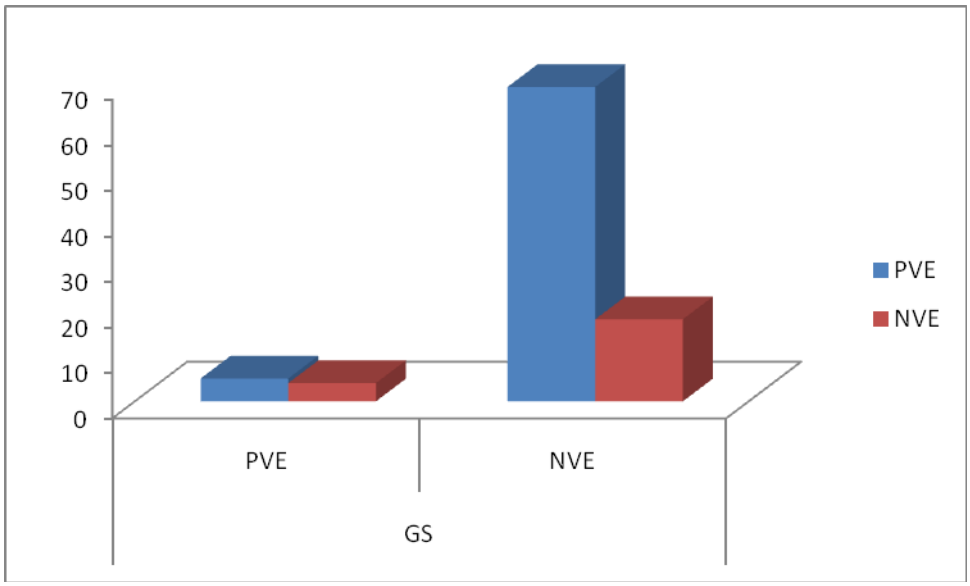
GRAPH:8 DISTRIBUTION OF CASES IN RELATION TO TOTAL LEUCOCUTE COUNT IN NICU

C-REACTIVE PROTEIN:

Among 96 babies admitted in NICU, 74 babies had positive CRP . Out of 74 babies, 5 cases were positive for gram stain (p<0.9) and 4 babies with negative CRP were also positive for gram stain. Interestingly 3 cases which were blood culture positive for candidiasis were negative for CRP(p<0.3)

CRP	GS		TOTAL	P value
	POSITIVE	NEGATIVE		
POSITIVE	5(55%)	69	74	0.3
NEGATIVE	4(44%)	18	22	
	9	87	96	

TABLE:8 DISTRIBUTION OF GRAM STAIN POSITIVE CASES IN RELATION TO CRP IN NICU.



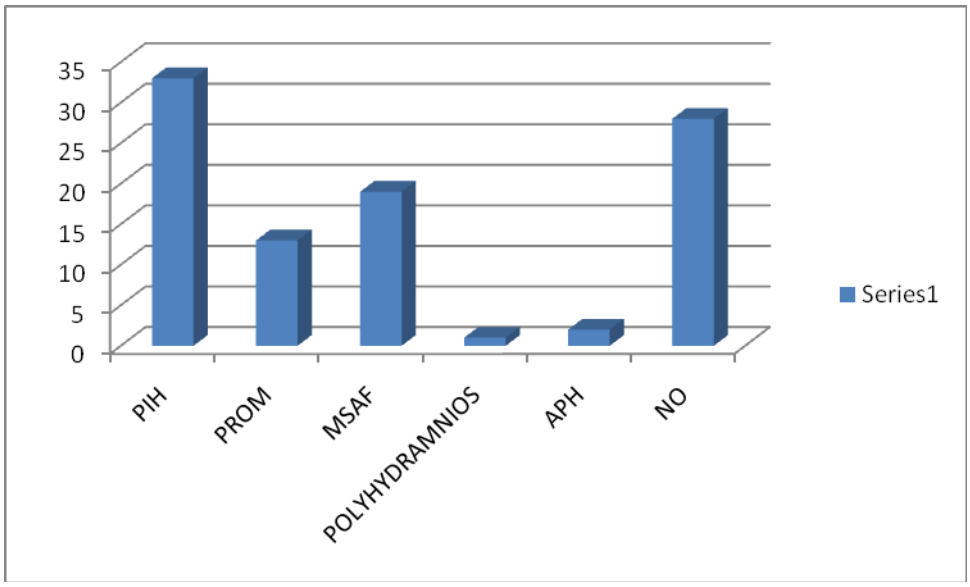
GRAPH:9 DISTRIBUTION OF GRAM STAIN POSITIVE CASES IN RELATION TO CRP IN NICU.

MATERNAL RISK FACTORS:

The various maternal risk factors like, premature rupture of membrane(PROM), meconium stain amnioitic fluid (MSAF), fetal distress, preeclampsia, pregnancy induced hypertension(PIH) clinically associated with candidiasis of neonates.

Risk factors	No of cases in NICU	POSITIVE CASES
PIH	33	5
PROM	13	3
MSAF	19	4
POLYHYDRAMNIOS	1	1
APH	2	0
NO risk factors	28	0

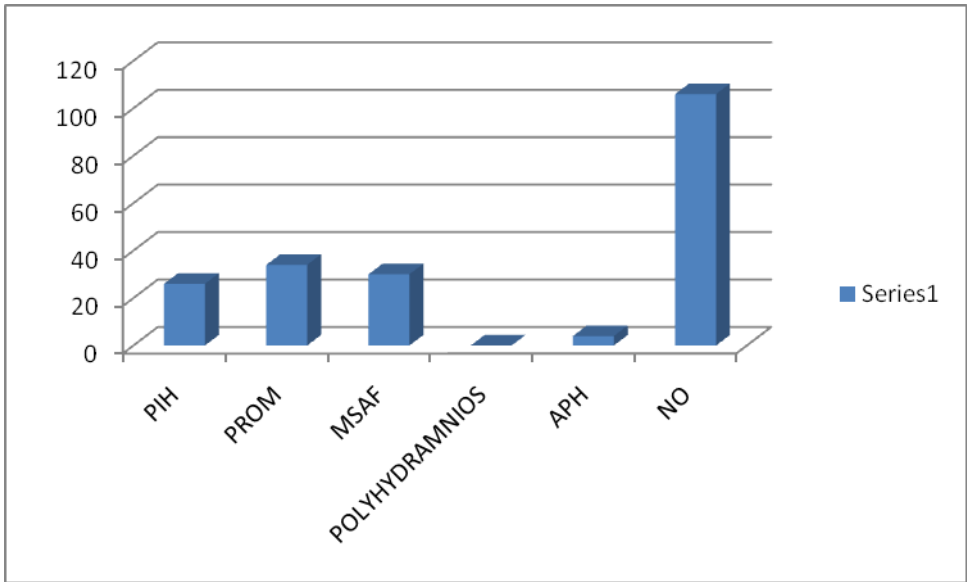
TABLE:9 DISTRIBUTION OF CASES IN RELATION TO MATERNAL RISK FACTORS IN NICU



GRAPH:10 DISTRIBUTION OF CASES IN RELATION TO MATERNAL RISK FACTORS IN NICU

Risk factors	No of cases in PNC
PIH	26
PROM	34
MSAF	30
POLYHYDRAMNIOS	0
APH	4
NO risk factors	106

TABLE:10 DISTRIBUTION OF CASES IN RELATION TO MATERNAL RISK FACTORS IN PNC



GRAPH:11 DISTRIBUTION OF CASES IN RELATION TO MATERNAL RISK FACTORS IN PNC

DISCUSSION

Neonatal candidosis had a various presentations and this study reveals clinical profile of these babies admitted in NICU. In order to decrease the morbidity of these neonates suffering from candidosis, early detection and management of candidosis is required. Hence the present study has made an attempt to evaluate the various risk factors and their significance in the development of candidosis. In the present study babies admitted in PNC wards had not developed candidosis since babies got discharged within 3-5 days and for colonization a minimum period of 10 days are required.

My observations are compared with those of others who have undertaken similar studies.

INCIDENCE:

296 babies were evaluated in study and revealed the incidence of candidiasis is 13.5% among babies admitted in NICU of 96 babies. The present study had more prevalence among babies admitted in NICU compared to PNC and these results are comparable to study conducted by Martin and Stephen[84] and Gupta study[26].

STUDY	INCIDENCE IN NICU
Gupta et al[26]	3.2%
Martin and Stephen[84]	10%
Present study	13.5%

TABLE:11 COMPARISON OF INCIDENCE OF CANDIDIASIS IN VARIOUS STUDIES ADMITTED IN NICU

In present study, cases suspected of sepsis babies were investigated for fungal culture and it was positive in 4 cases(30%). Interestingly cases without oral or cutaneous candidiasis were also blood culture positive.

SEX INCIDENCE:

Among babies admitted in NICU, male sex has a predominant role in development of candidiasis in neonates. Among 96 babies, 13 neonates had candidiasis, of which 9 are male babies (69%) compare to females. In present study, male sex has more predominance over females to develop candidiasis and these results are comparable to study conducted by the Gupta[26].

STUDY	**MALES(%)**	**FEMALES(%)**
Gupta etal[26]	80	20
Present study	69	31

TABLE:12 COMPARISON OF SEX DISTRIBUTION IN VARIOUS STUDY ADMITTED IN NICU

BIRTH WEIGHT:

96 neonates admitted in NICU were of birth weight between 1000gm to 2500gm. Out of which most of cases positive for candidiasis/candidemia were belonging to 1000gm-1500gm. In present study, 13 babies were positive for candidiasis, of which 5 babies(38.3%) of birth weight between 1000gm-1500gm (P=0.0313) has developed candidiasis compared to 19% of cases as studied by el-mohandes & coworkers[80] and 40% of cases by Ritu agarwal study[81]. As the birth weight decreases, the incidence of candidiasis in neonates increases as revealed by the present study.

Study	**Percentage of LBW babies**	**P VALUE**
Ritu agarwal study[81]	40%	0.03
El-Mohandes and coworkers[80]	19%	0.045
Present study	38.3%	0.0313

TABLE:13 COMPARISON OF SIGNIFICANCE OF LBW IN VARIOUS STUDIES IN RELATION TO CANDIDOSIS IN NICU

BIRTH ASPHYXIA:

Birth asphyxia is an important risk factor for the development of candidiasis in neonates. In the present study, birth asphyxia has a significant association in development of candidiasis in neonates admitted in NICU as compared to other studies by Jyostna[82] and Gupta et al study[26].

Type of study	**Confidence interval(95%)**	**P value**
Jyostna study[82]	1.43-46.6	0.03
Gupta etal study[26]	1.34-48.8	0.0226
Present study	0.118-0.277	0.004

TABLE:14 COMPARISON OF SIGNIFICANCE OF BIRTH ASPHYXIA IN VARIOUS STUDIES IN RELATION TO CANDIDOSIS IN NICU

MECHANICAL VENTILATION:

Mechanical ventilation is an important risk factor for development of candidiasis in neonates. In the present study,mechanical ventilation has a significant association in development of candidiasis in neonates admitted in NICU as compared to other studies by Anil kumar[83].

Study	**Percentage**	**P value**
Anil kumar study[83]	35%	
Present study	30%	0.0001

TABLE:15 COMPARISON OF SIGNIFICANCE OF MECHANICAL VENTILATION IN VARIOUS STUDIES IN RELATION TO CANDIDOSIS IN NICU

Others risk factors like maternal risk factors were studied in relation to candidiasis in neonates. But study revealed no significant association to develop neonatal candidiasis to support the association.

Laboratory parameters like CRP and total leucocyte count were studied in babies with candidiasis. But study revealed that no significant association exists between CRP posititivity ($p<0.9$) and total leucocyte count ($p<0.8$)

SUMMARY

296 babies were evaluated of which 96 babies(32%) admitted in NICU and remaining 200 babies (68%) admitted in PNC of shri B M Patil medical college, hospital and research centre, Bijapur for candidiasis in neonates and risk factors associated with them.

In the present study, candidiasis in neonates revealed 13.5%(13 out of 96) of babies admitted in NICU.

Male babies out numbered the female babies in incidence of candidiasis in neonates. Male babies were 9 (69%) and females 4 (31%) of positive cases.

Most of neonates admitted in NICU (96) were of low birth weight between 1500gm to 2500gm. Out of which most of cases positive for candidiasis/candidemia were belonged to 1000-1500gm. In present study, 13 babies were positive for candidiasis,of which 5 babies(38.3%) were of birth weight between 1000-1500gm.

Birth asphyxia is an important risk factor in development of candidiasis in neonates. In present study, birth asphyxia had played a significant role in development of candidiasis in neonates admitted in NICU.

Mechanical ventilation is also a important risk factor in development of candidiasis in neonates. In present study mechanical ventilation had played a significant role in development of candidiasis in neonates admitted in NICU.

CONCLUSION

- The observations made both in clinical and laboratory parameters in present study was compared with the other studies.
- Incidence of candidiasis in present study reveals 13.5%.
- Male babies outnumbered the females in positive cases of neonatal candidiasis.
- Low birth weight babies of 1000gm to 1500gm had more positive cases of candidiasis compared to other group of babies.
- Birth asphyxia and mechanical ventilation are significant risk factor in development of neonatal candidiasis as revealed in the present study.
- C reactive protein is one of screening profile in neonatal septicemia. Interestingly 4 positive cases had negative CRP and 3 cases with positive blood culture had a negative CRP. So CRP does not play a significant role in diagnosis of neonatal candidiasis.

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ANNEXURES - I

PROFORMA

SCHEME OF CASE TAKING:

Name	**:**	**CASE NO**	**:**
Age	**:**	**IP NO**	**:**
Sex	**:**	**DOA**	**:**
Religion	**:**	**DOD**	**:**
Residence	**:**		
Occupation & Income Of parents	**:**		

MATERNAL MEDICAL History-

1. History suggestive of diabetes mellitus **yes/no**

2. History suggestive of cardiac diseases **yes/no**

3. History suggestive of renal diseases **yes/no**

4. History suggestive of hypertension /PIH/eclampsia **yes/no**

5. History suggestive of chronic diseases **yes/no**

6. History suggestive of chronic drug intake **yes/no**

7. History suggestive of anaemia **yes/no**

8. History suggestive of intercurrent infections **yes/no**

9. Past History **:**

10. Family history:

11. ANC **:** **ANTENATAL:**

NATAL:

POSTNATAL:

13) General Physical Examination

SINGLE/TWIN…….

EGA:by dates…….weeks :byexam…….weeks;

LENGTH……..cms; MAC……..cms

BIRTH WEIGHT……..gms; HC…….cms

NEWBORN MATURITY RATING AND CLASSIFICATION

ESTIMATION OF GESTATIONAL AGE BY MATURITY RATING

Symbols : X - 1st Exam O- 2nd Exam

NEUROMUSCULAR MATURITY

	0	1	2	3	4	5
Posture						
Square Window (Wrist)	 90°	 60°	 45°	 30°	 0°	
Arm Recoil	 180°		 100°-180°	 90°-100°	 <90°	
Popliteal Angle	 180°	 160°	 130°	 110°	 90°	 <90°
Scarf Sign						
Heal to Ear						

Gestation by Dates _____wks

Birth Date _____Hour _____ am pm

APGAR _____1 min _____ 5 min

SCORING SECTION

1st Exam =X 2nd Exam=O

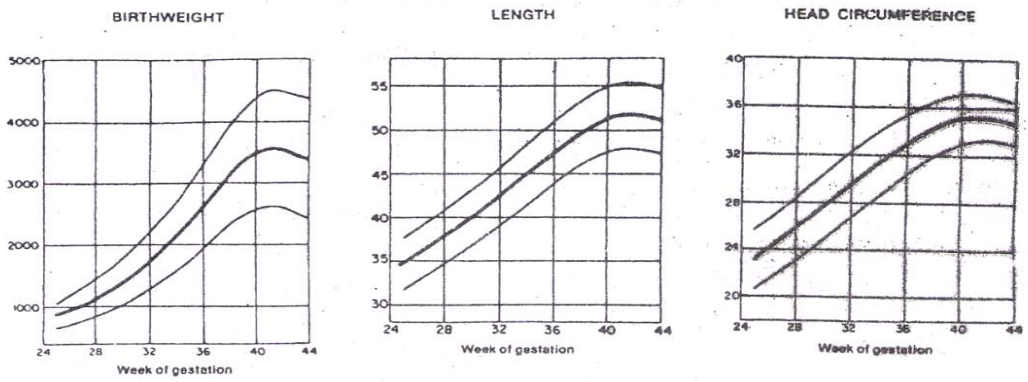
Estimating Gest Age by Maturity Rating	_____Weeks	____ Weeks
Time of Exam	Date_____ Hour____ am pm	Date_____ Hour____ am pm
Age at Exam	_____Hours	_____Hours
Signature of Examiner	_____ _____M.D.	_____ _____M.D.

PHYSICAL MATURITY

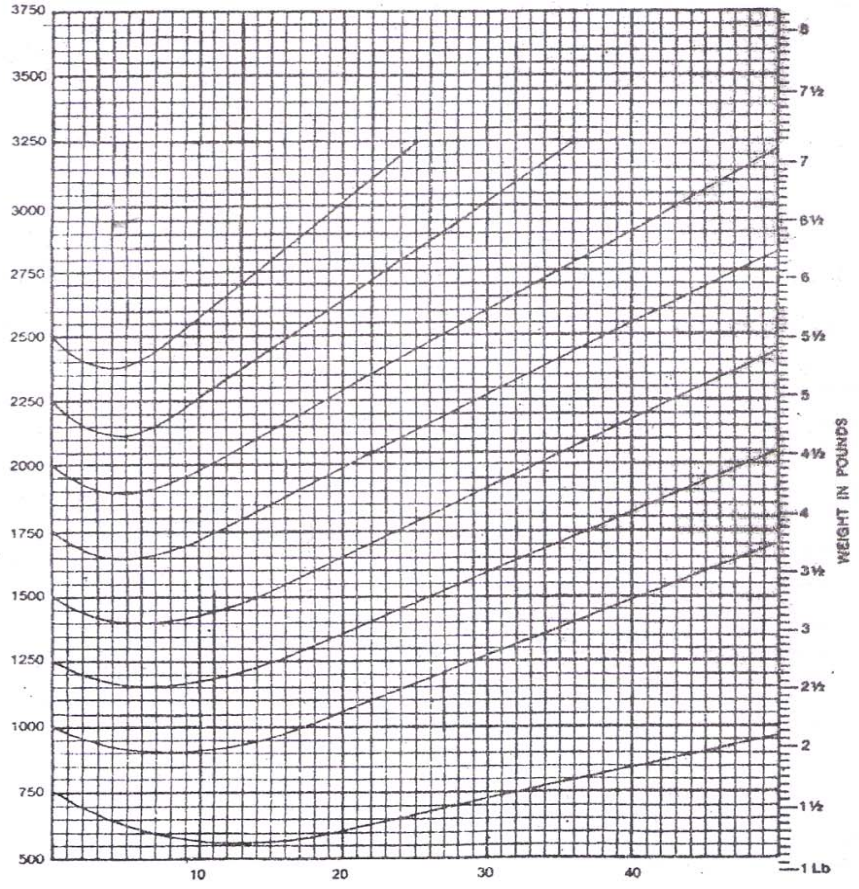
	0	1	2	3	4	5
Skin	gelatinous red, transpare-nt	Smooth pink, visiblle veins	superfici-al peeling & /or rash few veins	cracking pale area rare veins	parchment deep cracking no vessels	leathery cracked wrinkled
Lanugo	none	abundant	thinning	bald areas	mostly bald	
plantar Creases	no crease	faint red marks	anterior transverse crease only	creases ant. 2/3	creases cover entire sole	
Breast	barely percept.	flat areola no bud	stippled areola 1-2 mm bud	raised areola 3-4 mm bud	full areola 5-10 mm bud	
Ear	pinna flat, stays folded	sl. curved pinna; soft with slow recoil	well-curv. pinna; soft but ready recoil	formed & firm with instant recoil	thick cartilage ear stiff	
Genitals	scrotum empty no rugae		testes descendi-ng, few rugae	testes down, good rugae	testes pendulous deep rugae	
Genitals	prominent clitoris & labia minora		majora & minora equally prominent	majora large, minora small	clitoris & minora complete-ly covered	

MATURITY RATING

Score	Wks
5	26
10	28
15	30
20	32
25	34
30	36
35	38
40	40
45	42
50	44



GROWTH RECORD



14) Systemic examination:

- **Vitals:**
- **Skin:**
  - **Anterior aspect of forearm**
  - **Behind the ear.**
- **Craniofacial:**
- **Oral cavity**
- **Chest:**
- **Cardiovascular system:**
- **Respiratory system:**
- **Abdomen:**
- **Genitalia:**
  - **Perianal region**
  - **Nappy Rash**
- **Extremities:**
- **Back:**
- **Spine:**
- **Central nervous system:**

Impression

Provisional Diagnosis:

Investigation:

Complete Hemogram:

Special investigations:

1) Gram stain
2) KOH study
3) Fungal culture
4) Blood culture

Final Diagnosis:

Follow Up

1. **First Week**

2. **Second Week**

3. **Third Week**

4. **Fourth Week**

ANNEXURE – II

BLDEA's Shri B.M.PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, BIJAPUR-586103.

RESEARCH INFORMED CONSENT FORM

TITLE OF THE PROJECT	:	CLINICAL PROFILE OF CANDIDIASIS IN NEONATES
GUIDE	:	Dr. S. S.KALYANSHETTAR ASSOCIATE PROFESSOR
P G STUDENT	:	Dr. VEERESH BABU D V

PURPOSE OF RESEARCH:

I have been informed that the present study will help in assessing the clinical profile of fungal infections in neonates and improve the quality of life in these neonates

PROCEDURE:

I understand that after having obtained a detailed clinical history, thorough clinical examination and relevant investigations, a final work up for the etiological identification and appropriate management is planned.

RISK AND DISCOMFORTS:

I understand that I may experience some pain and discomforts during the examination or during my treatment. This is mainly the result of my condition and the procedures of this study are not expected to exaggerate these feelings which are associated with the usual course of treatment.

BENEFITS:

I understand that my participation in the study will have no direct benefit to me other than the potential benefit of the treatment.

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of hospital records and will be subject to the confidentiality. Information of sensitive personal nature will not be part of the medical record, but will be stored in the investigations research file.

If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs will be used only with special written permission. I understand that I may see the photograph before giving the permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at anytime; Dr. Veeresh babu D V at the department of pediatrics is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL FOR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice. I also understand that Dr. Veeresh babu D V may terminate my participation in the study after he has explained the reasons for doing so.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, the appropriate treatment would be available to me. But, no further compensation would be provided by the hospital. I understand that by my agreements to participate in this study and not waiving any of my legal rights.

I have explained to _____the purpose of the research, the procedures required and the possible risks to the best of my ability.

_____ _____

Dr. Veeresh babu D V Date

(Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that Dr. Veeresh babu D V has explained to me the purpose of research, the study procedure, that I am willing to allow my baby to undergo the investigation and the possible discomforts as well as benefits. I have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give consent to participate as a subject in this research project.

_____ _____

(Participant) Date

_____ _____

(Witness to signature) Date

ANNEXURE – III

KEY TO MASTER CHART

BW: BIRTH WEIGHT

BA : BIRTH ASPHYXIA

MV: MECHANICAL VENTILATION

MRF: MATERNAL RISK FACTORS

TLC: TOTAL LEUCOCYTE COUNT

CRP: C REACTIVE PROTEIN

B/C: BLOOD CULTURE

GS: GRAM STAIN

PROM: PREMATURE RUPTURE OF MEMBRANE

PIH: PREGNANCY INDUCED HYPERTENSION

MSAF: MECONIUM STAINED AMNIOTIC FLUID

ANNEXURE – IV
MASTER CHART

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
1	**B/O SUNITA**	**5D**	**M**	**25827**	**760GMS**	**YES**	**YES**	**PIH**	**5000**	**POSITIVE**	**NEG**	**POSITIVE**	**NEG**
2	B/O SHEETA BAI	3D	M	5171	2.96	YES	NO	NO	9300	POSITIVE	NEG	NEG	NEG
3	B/O BHUVENESWAR	3D	F	□7078	2.5	NO	NO	PROM	7500	POSITIVE	NEG	NEG	NEG
4	B/O DEEPA	3D	M	5138	2.65	YES	NO	PIH	10,800	NEG	NEG	NEG	NEG
5	B/O DHANAMMA	3D	M	4912	3.2	NO	NO	MSAF	26200	NEG	NEG	NEG	NEG
6	B/OSHABINA	4D	M	4099	1.7	NO	NO	PIH	2900	POSITIVE	POSITIVE	NEG	NEG
7	B/O SUPRABHAT	4D	F	13444	3.69	NO	NO	PIH	17000	POSITIVE	NEG	NEG	NEG
8	B/O YASMIN	3D	F	16900	2.9	YES	NO	NO	3800	POSITIVE	NEG	NEG	NEG
9	**B/O KALAVATI**	**5D**	**F**	**16089**	**2.3**	**YES**	**NO**	**PIH**	**7400**	**POSITIVE**	**NEG**	**POSITIVE**	**POSITIVE**
10	B/O SARASVATI	4D	M	3828	2.62	NO	NO	MSAF	22,000	POSITIVE	NEG	NEG	NEG
11	B/O SHRUTI	3D	M	10249	3.2	NO	NO	NO	22100	POSITIVE	NEG	NEG	NEG
12	B/O SUBANGI	3D	F	21103	1.84	NO	NO	PROM	19150	NEG	NEG	NEG	NEG
13	B/O SAVITRI	4D	F	15686	2.5	NO	NO	MSAF	18500	NEG	NEG	NEG	NEG
14	B/O VIJAY LAXMI	3D	M	11098	3.5	NO	NO	PIH	16500	NEG	NEG	NEG	NEG
15	B/O GOURAMMA	4D	M	11101	1.41	NO	NO	PROM	7500	POSITIVE	NEG	NEG	NEG

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
16	B/O RESHMA	3D	F	27609	2.75	NO	NO	NO	22500	POSITIVE	NEG	NEG	NEG
17	**B/O ASHWINI**	**3D**	**M**	**24804**	**1.65**	**YES**	**YES**	**PIH**	**25200**	**POSITIVE**	**NEG**	**POSITIVE**	**NEG**
18	B/O CHINAMMA	4D	M	11264	3	YES	YES	NO	24900	NEG	NEG	NEG	NEG
19	B/O MADURI	4D	F	8821	3.5	NO	NO	MSAF	16100	POSITIVE	NEG	NEG	NEG
20	B/O MAHADEVI	5D	M	10879	1.8	NO	NO	NO	14900	NEG	NEG	NEG	NEG
21	B/O SUNANDA	5D	M	11031	1.74	NO	NO	NO	16700	POSITIVE	NEG	NEG	NEG
22	B/O PAVITRA	7D	F	11647	3.45	NO	YES	YES	18900	POSITIVE	NEG	NEG	NEG
23	B/O SAVITRI	5D	F	10251	2	NO	NO	NO	5900	POSITIVE	NEG	NEG	NEG
24	B/O SUNANDA	3D	M	8710	1.74	NO	NO	NO	3200	POSITIVE	NEG	NEG	NEG
25	B/O VIDYA	3D	F	11408	3.15	YES	YES	NO	5500	POSITIVE	POSITIVE	NEG	NEG
26	B/O ASHWINI	3D	M	8663	1.5	NO	NO	PIH	17100	POSITIVE	NEG	NEG	NEG
27	B/O NAZIA	3D	F	3864	2.23	NO	NO	NO	14400	POSITIVE	NEG	NEG	NEG
28	**B/O ROOPA**	**4D**	**M**	**2464**	**2.75**	**YES**	**NO**	**POLYHYD-RAMNIOS**	**7400**	**POSITIVE**	**NEG**	**NEG**	**POSITIVE**
29	B/O BISMILLA	7D	M	8955	1.8	NO	NO	PIH	17400	POSITIVE	NEG	NEG	NEG
30	B/O SUDHA	4D	F	21094	2.15	NO	NO	MSAF	26600	NEG	NEG	NEG	NEG

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
31	B/O SMITHA	3D	F	7476	2.34	NO	NO	PROM	28200	POSITIVE	POSITIVE	NEG	NEG
32	B/O LAXMIBAI	3D	M	10811	1.8	NO	NO	PIH	14200	POSITIVE	NEG	NEG	NEG
33	B/O NANDA	3D	F	5389	2	YES	YES	APH	29200	POSITIVE	NEG	NEG	NEG
34	B/O RUXANA	3D	F	10839	2.5	NO	NO	PIH	17500	POSITIVE	NEG	NEG	NEG
35	B/O LUCHHABAI	3D	M	17591	2.66	NO	NO	NO	13900	NEG	NEG	NEG	NEG
36	**B/O DEVIKA**	**3D**	**M**	**28078**	**2.5**	**NO**	**NO**	**PIH**	**15500**	**POSITIVE**	**POSITIVE**	**NEG**	**POSITIVE**
37	B/O JAYASHRI	3D	M	17022	1.8	NO	NO	PIH	9700	POSITIVE	NEG	NEG	NEG
38	B/ORAJASHREE	3D	F	235	2.5	NO	NO	PROM	7500	POSITIVE	NEG	NEG	NEG
39	B/OTANUJA	3D	F	5259	1.35	NO	NO	PIH	21000	POSITIVE	POSITIVE	NEG	NEG
40	B/O MEGHA	5D	F	5690	1.8	NO	NO	PIH	14500	POSITIVE	NEG	NEG	NEG
41	B/OPINKY	3D	M	16340	2.6	NO	NO	PIH	20200	NEG	NEG	NEG	NEG
42	B/O RENUKA	4D	M	5400	2.6	NO	NO	NO	5100	POSITIVE	NEG	NEG	NEG
43	**B/O RENUKA**	**5D**	**M**	**17484**	**1.92**	**YES**	**YES**	**PIH**	**8200**	**POSITIVE**	**NEG**	**POSITIVE**	**NEG**
44	B/O SUJATHA	5D	M	21306	2.25	YES	NO	NO	21600	POSITIVE	NEG	NEG	NEG
45	B/OLAXMI	3D	F	4986	2	NO	NO	PIH	9300	POSITIVE	NEG	NEG	NEG

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
46	B/O SUJATHA	5D	M	19059	1.44	NO	NO	PROM	16600	POSITIVE	NEG	NEG	NEG
47	B/O KAMLABAI	3D	F	7324	2.2	NO	NO	PROM	17500	POSITIVE	NEG	NEG	NEG
48	B/O KAVITHA	3D	M	27720	1.25	NO	NO	PIH	15200	POSITIVE	NEG	NEG	NEG
49	B/O RAJANI	3D	M	5125	1.9	NO	NO	NO	5600	POSITIVE	NEG	NEG	NEG
50	B/O SUJATHA	3D	M	11224	1.5	NO	NO	PIH	15600	POSITIVE	NEG	NEG	NEG
51	B/O SHAHNAZ	5D	M	11092	2.75	YES	YES	NO	25600	POSITIVE	NEG	NEG	NEG
52	B/O KASTURIBAI	5D	M	18850	2.93	NO	NO	MSAF	10200	POSITIVE	NEG	NEG	NEG
53	**B/O SHOBA**	**5D**	**F**	**2403**	**2.7**	**YES**	**YES**	**PIH**	**17200**	**POSITIVE**	**POSITIVE**	**NEG**	**POSITIVE**
54	B/O NETRAVATI	3D	M	27887	2.5	NO	NO	MSAF	17100	POSITIVE	NEG	NEG	NEG
55	B/O RENUKHA	3D	M	27676	1.8	NO	NO	PIH	14500	POSITIVE	NEG	NEG	NEG
56	B/O POOJA	5D	F	92235	1.7	NO	NO	NO	18500	POSITIVE	NEG	NEG	NEG
57	B/O RUKMABAI	3D	F	147976	2.7	NO	NO	MSAF	21500	POSITIVE	NEG	NEG	NEG
58	B/O BHAGYASHREE	3D	F	139034	2.8	NO	NO	PIH	17600	POSITIVE	NEG	NEG	NEG
59	B/O ANITA	3ED	M	278	1.9	NO	NO	NO	17200	POSITIVE	NEG	NEG	NEG
60	B/O SUJATHA	5D	M	16338	2.8	NO	NO	NO	18200	NEG	NEG	NEG	NEG

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
61	B/O MAHALAKSHMI	5D	F	134667	1.7	NO	NO	PIH	18600	POSITIVE	NEG	NEG	NEG
62	B/O KALLUBAI	4D	M	21782	2.7	YES	YES	NO	22500	POSITIVE	POSITIVE	NEG	NEG
63	B/O SAVITA	5D	F	136546	2.7	NO	NO	NO	15200	NEG	NEG	NEG	NEG
64	B/OLAXMI	3D	M	21667	2	NO	NO	PIH	17200	POSITIVE	NEG	NEG	NEG
65	B/O SAVITA	3D	M	56413	1.65	NO	NO	PROM	18500	POSITIVE	NEG	NEG	NEG
66	**B/O JAYASHREE**	**3D**	**M**	**28495**	**1.4**	**NO**	**NO**	**PIH**	**15500**	**NEG**	**POSITIVE**	**POSITIVE**	**NEG**
67	B/O LAXMIBAI	3D	F	131717	2.5	NO	NO	MSAF	14500	POSITIVE	NEG	NEG	NEG
68	**B/O SUREKHA**	**3D**	**M**	**10790**	**1.3**	**YES**	**YES**	**NO**	**10800**	**NEG**	**NEG**	**POSITIVE**	**NEG**
69	B/O BABYKALA	3D	F	130663	1.8	NO	NO	NO	18500	POSITIVE	NEG	NEG	NEG
70	B/O BHARATI	4D	F	122907	2.7	NO	NO	MSAF	21500	POSITIVE	NEG	NEG	NEG
71	B/O NINGAMMA	3D	M	1928	1.8	NO	NO	PIH	17500	POSITIVE	NEG	NEG	NEG
72	B/O ROOPA	5D	M	12893	2.89	YES	NO	MSAF	23000	POSITIVE	NEG	NEG	NEG
73	B/O SREEDEVI	3D	M	9208	1.75	NO	NO	PROM	9200	POSITIVE	NEG	NEG	NEG
74	B/O SHREEDEVI	3D	F	18391	1.5	NO	NO	NO	12000	NEG	NEG	NEG	NEG
75	**B/O SIDDARAM**	**3D**	**M**	**28287**	**1.2**	**NO**	**NO**	**MSAF**	**21500**	**POSITIVE**	**NEG**	**POSITIVE**	**NEG**

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
76	B/O RENUKA	4D	M	140557	2.8	NO	NO	MSAF	22500	POSITIVE	NEG	NEG	NEG
77	B/O GEETA	3D	F	145937	1.75	NO	NO	PROM	14500	POSITIVE	NEG	NEG	NEG
78	**B/O RUKMINI**	**4D**	**M**	**1435**	**1.36**	**NO**	**NO**	**PIH**	**18500**	**POSITIVE**	**NEG**	**POSITIVE**	**NEG**
79	B/OMUNNII	3D	F	97673	2.2	NO	NO	NO	17500	POSITIVE	NEG	NEG	NEG
80	B/O KAVERI	4D	F	97750	1.8	NO	NO	PIH	14500	POSITIVE	NEG	NEG	NEG
81	B/O AMIRBEE	6D	F	99853	1.2	NO	NO	PIH	19200	POSITIVE	NEG	NEG	NEG
82	B/O SUJATHA	2D	F	101233	2.7	YES	YES	MSAF	21500	POSITIVE	NEG	NEG	NEG
83	**B/O BORAWWA**	**3D**	**M**	**28044**	**2.7**	**NO**	**NO**	**NO**	**8800**	**POSITIVE**	**NEG**	**POSITIVE**	**NEG**
84	B/O SUJATHA	3D	F	101233	2.8	NO	NO	MSAF	14500	POSITIVE	NEG	NEG	NEG
85	B/O SRUTI	4D	F	96316	2.7	NO	NO	MSAF	12500	POSITIVE	NEG	NEG	NEG
86	B/O BHARATI	2D	F	93205	2.2	NO	NO	MSAF	17500	POSITIVE	NEG	NEG	NEG
87	B/O POOJA	3D	F	92235	1.2	NO	NO	PROM	14500	NEG	NEG	NEG	NEG
88	B/O SHIVALEELA	3D	F	27094	2.8	NO	NO	NO	15500	NEG	NEG	NEG	NEG
89	B/O KAMALABAI	3D	F	89353	2.6	NO	NO	PIH	19500	NEG	NEG	NEG	NEG
90	B/O MAITRABAI	4D	F	88803	2.4	YES	YES	PROM	14500	POSITIVE	NEG	NEG	NEG

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
91	B/O KAVERI	5D	F	29007	2.8	NO	NO	PIH	15500	NEG	NEG	NEG	NEG
92	B/O LAILABANU	3D	F	86596	2.6	NO	NO	MSAF	12800	NEG	NEG	NEG	NEG
93	B/O BHAGYASHREE	6D	F	81592	2.5	NO	NO	PROM	12000	NEG	NEG	NEG	NEG
94	B/O NAKUSA	6D	F	95601	2.65	YES	YES	NO	21500	POSITIVE	NEG	NEG	NEG
95	B/O GEETA	1D	F	68261	2.5	NO	NO	MSAF	18900	NEG	NEG	NEG	NEG
96	**B/O HIRABHAI**	**4D**	**F**	**9167**	**1.25**	**YES**	**YES**	**PIH**	**19400**	**POSITIVE**	**NEG**	**POSITIVE**	**NEG**
97	B/O ANITA	3D	F	6898	2.8	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
98	B/OSUSALA	5D	F	6874	2.95	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
99	B/OLAXMIBAI	4D	F	6895	2.46	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
100	B/OMALAMMA	3D	F	6966	2.4	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
101	B/ODEVAMMA	2D	F	10091	2.8	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
102	B/OGOURABAI	5D	F	18606	2.8	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
103	B/OSASHIKALA	4D	F	6245	1.56	NO	NO	PROM	5650	POSITIVE	NEG	NEG	NEG
104	B/OLAXMI	3D	F	4986	2	NO	NO	NO	9300	POSITIVE	NEG	NEG	NEG
105	B/OLAXMI	2D	F	5448	1.12	**NO**	**NO**	PROM	7300	POSITIVE	**NEG**	**NEG**	**NEG**

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
106	B/OSAVITA	5D	F	4946	2.9	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
107	B/OASHWINI	4D	F	10112	3.2	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
108	B/OINDRABAI	3D	F	11050	2.67	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
109	B/OMEENAXI	2D	F	9217	2.82	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
110	B/OSWETA	5D	F	9243	3.25	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
111	B/OSUJATA	4D	F	16300	3.2	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
112	B/OSAVITA	3D	F	16211	2.7	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
113	B/OSHOBA	2D	F	18388	2.8	**NO**	**NO**	PROM	NORMAL	NILL	**NEG**	**NEG**	**NEG**
114	B/OASHWINI	5D	F	18142	2.6	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
115	B/OLALITA	4D	F	18092	2.45	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
116	B/OREKHA	3D	F	21263	2.3	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
117	B/OSREEDEVI	2D	F	6085	2.8	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
118	B/OPARIDA	5C	F	6095	2.75	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
119	B/OASMA	4C	F	27778	2.65	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
120	B/OMAHANANDA	3D	F	233	2.6	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
121	B/OSAVITA	2D	F	27743	2.55	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
122	B/OMASABEE	5D	F	5625	2.45	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
123	B/OYELLAWWA	4D	F	23404	2.75	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
124	B/OSHOBA	3D	F	23531	2.9	**NO**	**NO**	NO	NORMAL	NILL	**NEG**	**NEG**	**NEG**
125	B/OLAXMI	2D	F	24746	2.8	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
126	B/OASWINI	5D	F	7632	2.5	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
127	B/OSHREEDEVI	2D	F	23498	2.45	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
128	B/OINDUBAI	3D	F	21102	2.48	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
129	B/OSHILPA	4D	F	18943	2.68	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
130	B/O SHREEDEVI	3D	F	10973	2.7	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
131	B/OITTABAI	3D	F	8204	2.68	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
132	B/OLAXMI	3D	F	7488	2.65	**NO**	**NO**	NO	NORMAL	NILL	**NEG**	**NEG**	**NEG**
133	B/OPARWATI	5D	F	3335	2.72	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
134	B/ORATNABAI	4D	F	3269	2.8	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
135	B/OROOPA	3D	F	8010	2.78	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
136	B/OAMBIKA	4D	F	2156	2.65	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
137	B/OAMBIKA	4D	F	2953	2.68	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
138	B/OLAXMI	3D	F	2934	2.9	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
139	B/OASHA	4D	F	2849	2.68	**NO**	**NO**	NO	NORMAL	NILL	**NEG**	**NEG**	**NEG**
140	B/OPARVATI	4D	F	3169	2.88	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
141	B/OSAVITA	3D	F	11149	2.98	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
142	B/OANUSABAI	5D	F	21875	2.96	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
143	B/OGEETA	2D	F	13388	2.76	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
144	B/OPOOJA	5D	F	17021	2.66	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
145	B/OKALPANA	5D	F	17010	2.56	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
146	B/OARATI	3D	F	16947	2.86	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
147	B/OBASAMMA	4D	F	10931	2.84	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
148	B/OBORAMMA	5D	M	309	2.76	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
149	B/OSUJATHA	4D	M	10023	2.55	**NO**	**NO**	PROM	NORMAL	NILL	**NEG**	**NEG**	**NEG**
150	B/OSHOBHA	3D	M	18682	2.68	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
151	B/OJAYASHREE	2D	M	11497	2.1	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
152	B/ONEELAKKA	5D	M	11519	2.75	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
153	B/OJAYASHREE	4D	M	5655	1.75	NO	NO	NO	NORMAL	POSITIVE	NEG	NEG	NEG
154	B/ODHANESHWARI	3D	M	6869	3.2	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
155	B/OGURUSANGAM	2D	M	4681	3.45	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
156	B/ONINGAMMA	5D	M	11134	1.6	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
157	B/OGEETA	4D	M	11052	2.34	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
158	B/OGEETA	3D	M	11153	3.4	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
159	B/OPREMA	5D	M	10767	2.9	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
160	B/OANUSABAI	4D	M	11185	2.53	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
161	B/OKASTURABAI	3D	M	11156	2.75	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
162	B/OPUSPA	3D	M	9298	2.8	**NO**	**NO**	NO	NORMAL	NILL	**NEG**	**NEG**	**NEG**
163	B/ONETRAVATI	4D	M	23415	2.65	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
164	B/O SAVITHA	5D	M	5879	2.65	**NO**	**NO**	NO	NORMAL	NILL	**NEG**	**NEG**	**NEG**
165	B/O AWAKKA	4D	M	5926	2.78	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
166	B/OBHARATI	3D	M	8140	2.66	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
167	B/OGEETA	2D	M	7789	2.44	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
168	B/OMUDEMMA	3D	M	7820	2.56	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
169	B/OGEETA	5D	M	7570	2.72	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
170	B/OYJAYASHREE	4D	M	7492	2.82	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
171	B/ORENUKA	5D	M	23244	2.46	**NO**	**NO**	PROM	NORMAL	NILL	**NEG**	**NEG**	**NEG**
172	B/OPARAVEEN	4D	M	7881	2.78	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
173	B/OLAXMIBAI	2D	M	4232	2.59	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
174	B/OANITA	3D	M	4231	2.46	**NO**	**NO**	NO	NORMAL	NILL	**NEG**	**NEG**	**NEG**
175	B/OPRABHAVATI	4D	M	23486	2.76	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
176	B/OLAXMIBAI	3D	M	27886	2.86	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
177	B/ODRAKSHAYANI	5D	M	6047	2.89	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
178	B/OSATEWWA	4D	M	20936	2.76	NO	NO	APH	NORMAL	NILL	NEG	NEG	NEG
179	B/OASHWINI	3D	M	22916	3.2	**NO**	**NO**	APH	NORMAL	NILL	**NEG**	**NEG**	**NEG**
180	B/OSAVITA	2D	M	23103	3.16	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
181	B/OVIJAYALAXMI	5D	M	23193	3.15	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
182	B/OPUSHPA	4D	M	22915	3.16	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
183	B/OGAYATRI	3D	M	5971	2.75	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
184	B/O SUJATHA	2D	M	6075	2.76	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
185	B/OMEENAKSHI	5D	M	17807	2.48	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
186	B/OSAVITRI	4D	M	18960	2.46	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
187	B/OREKHA	3D	M	15392	2.82	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
188	B/OKHAJBEE	2D	M	18322	2.72	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
189	B/ORUKMABAI	5D	M	16795	2.86	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
190	B/ORENUKA	4D	M	16728	2.76	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
191	B/O SUNANDA	3D	M	16623	2.82	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
192	B/OPRIYANKA	2D	M	16438	2.76	**NO**	**NO**	MSAF	NORMAL	NILL	**NEG**	**NEG**	**NEG**
193	B/OKOMAL	5D	M	21880	2.46	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
194	B/OHASINA	4D	M	21881	2.52	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
195	B/O SOMAWWA	3D	M	21665	2.89	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
196	B/OMAHANANDA	2D	M	3144	2.66	**NO**	**NO**	MSAF	NORMAL	NILL	**NEG**	**NEG**	**NEG**
197	B/OMOHINI	3D	M	3263	2.76	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
198	B/OSAVITA	5D	M	21934	2.48	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
199	B/OJAYASHREE	4D	M	21936	2.48	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
200	B/OSHANTABAI	3D	M	21938	2.46	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
201	B/OSANTOSHAMMA	2D	M	2343	2.56	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
202	B/OMANJULA	5D	M	2650	2.74	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
203	B/OMAHANADA	4D	M	3060	2.86	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
204	B/OKAVITA	3D	M	3080	2.54	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
205	B/OYOGITA	2D	M	3071	3.2	**NO**	**NO**	NO	NORMAL	NILL	**NEG**	**NEG**	**NEG**
206	B/OSMITA	5D	M	10850	2.56	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
207	B/OROOPA	4D	M	97691	2.5	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
208	B/ONEELAKKA	3D	M	97865	2.5	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
209	B/ONEELAMMA	2D	M	95047	2.42	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
210	B/OSWATI	5D	M	97338	2.36	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
211	B/OLALITA	4D	M	92241	2.56	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
212	B/OFATIMA	3D	M	608	2.42	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
213	B/ONAGAMMA	2D	M	776	2.56	**NO**	**NO**	MSAF	NORMAL	NILL	**NEG**	**NEG**	**NEG**
214	B/OMALAKAMMA	5D	M	797	2.42	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
215	B/ONASHIN	4D	M	1562	2.86	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
216	B/ODEEPA	3D	M	569	2.92	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
217	B/OSHILPA	2D	M	27970	2.76	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
218	B/ORAJASHREE	5C	M	235	2.52	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
219	B/OBORAMMA	4C	M	309	2.42	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
220	B/ODEEPA	3D	M	307	2.46	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
221	B/OAPSARA	2D	M	324	2.42	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
222	B/OSHAHMA	5D	M	495	2.46	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
223	B/O KALASUMBI	4D	M	10061	2.52	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
224	B/OROOPA	3D	M	81953	2.56	**NO**	**NO**	NO	NORMAL	NILL	**NEG**	**NEG**	**NEG**
225	B/OGANGABAI	2D	M	88750	2.65	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
226	B/OASHA	5D	M	88829	2.66	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
227	B/OAKKAMMA	2D	M	89356	2.67	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
228	B/OJYOTI	3D	M	91484	2.78	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
229	B/OJAYASHREE	4D	M	96630	2.69	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
230	B/OITTABAI	3D	M	10030	2.7	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
231	B/OAMBIKA	3D	F	24895	1.84	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
232	B/OVIMALA	3D	M	68282	2.48	**NO**	**NO**	NO	NORMAL	NILL	**NEG**	**NEG**	**NEG**
233	B/OSIDDAMMA	5D	M	68293	2.76	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
234	B/IJAYASHREE	4D	F	70988	2.82	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
235	B/OSUNANDA	3D	M	73200	1.98	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
236	B/ORAJESHWARI	4D	M	73208	2.56	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
237	B/OSREEDVI	4D	F	73247	2.92	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
238	B/OMALLAMMA	3D	F	73985	2.2	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
239	B/ONAGAWWA	4D	M	73256	1.56	**NO**	**NO**	NO	NORMAL	NILL	**NEG**	**NEG**	**NEG**
240	B/OPAVITRA	4D	M	74018	2.48	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
241	B/OSUMITRA	3D	M	74277	2.76	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
242	B/OFARJANA	5D	M	74267	2.65	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
243	B/OGEETA	2D	F	68261	2.56	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
244	B/ORESHMA	5D	M	30975	2.24	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
245	B/ONINGAMMA	5D	F	33075	2.32	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
246	B/ONEELAKKA	3D	M	32104	1.56	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
247	B/OREVAMMA	4D	F	45673	2.52	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
248	B/O CHINAMMA	5D	F	47865	2.96	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
249	B/O MADURI	4D	F	5674	2.76	**NO**	**NO**	MSAF	NORMAL	NILL	**NEG**	**NEG**	**NEG**
250	B/O MAHADEVI	3D	F	4567	2.66	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
251	B/O SUNANDA	2D	F	3456	2.56	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
252	B/O PAVITRA	5D	F	2345	2.86	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
253	B/O SAVITRI	4D	F	2346	2.84	NO	NO	MSAF	NORMAL	POSITIVE	NEG	NEG	NEG
254	B/O SUNANDA	3D	F	3245	2.76	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
255	B/O VIDYA	2D	F	2456	2.55	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
256	B/O ASHWINI	5D	F	2567	2.68	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
257	B/O NAZIA	4D	M	2678	2.1	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
258	B/O BABYKALA	3D	M	4563	2.75	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
259	B/O BHARATI	5D	M	4567	1.75	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
260	B/O NINGAMMA	4D	M	2456	3.2	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
261	B/O ROOPA	3D	M	3456	3.45	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
262	B/O SREEDEVI	3D	M	4563	1.6	**NO**	**NO**	NO	NORMAL	NILL	**NEG**	**NEG**	**NEG**
263	B/O SHREEDEVI	4D	M	6785	2.34	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
264	B/O SUJATHA	5D	M	6759	3.4	**NO**	**NO**	NO	NORMAL	NILL	**NEG**	**NEG**	**NEG**
265	B/O SRUTI	4D	M	7869	2.9	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
266	B/O BHARATI	3D	M	7685	2.53	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
267	B/O POOJA	2D	M	7890	2.75	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
268	B/O SHIVALEELA	3D	M	8907	2.8	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
269	B/O KAMALABAI	5D	M	9078	2.65	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
270	B/O MAITRABAI	4D	M	9870	2.65	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
271	B/O KAVERI	5D	F	8594	2.78	**NO**	**NO**	PROM	NORMAL	NILL	**NEG**	**NEG**	**NEG**
272	B/O LAILABANU	4D	F	5678	2.66	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
273	B/O BHAGYASHREE	2D	F	4563	2.44	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
274	B/O NAKUSA	3D	F	5674	2.56	**NO**	**NO**	NO	NORMAL	NILL	**NEG**	**NEG**	**NEG**
275	B/O GEETA	4D	F	9087	2.72	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
276	B/O SARASVATI	3D	M	9870	2.82	NO	NO	PIH	NORMAL	NILL	NEG	NEG	NEG
277	B/O SHRUTI	5D	F	9654	2.46	NO	NO	APH	NORMAL	NILL	NEG	NEG	NEG
278	B/O SUBANGI	4D	F	9453	2.78	NO	NO	APH	NORMAL	NILL	NEG	NEG	NEG
279	B/O SAVITRI	3D	M	9342	2.59	**NO**	**NO**	NO	NORMAL	NILL	**NEG**	**NEG**	**NEG**
280	B/O VIJAY LAXMI	2D	F	9900	2.46	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
281	B/O GOURAMMA	5D	F	10123	2.76	NO	NO	PROM	NORMAL	NILL	NEG	NEG	NEG
282	B/O RESHMA	4D	F	10234	2.86	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
283	B/OANITA	3D	M	13452	2.89	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
284	B/OPRABHAVATI	2D	M	14562	2.76	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
285	B/OLAXMIBAI	5D	M	16754	3.2	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG

Sl.No	NAME	AGE	SEX	IP NO	BW (KG)	BA	MV	MRF	TC	CRP	B/C	GS	BLOOD CULTURE FOR CANDIDEMIA
286	B/ODRAKSHAYANI	4D	M	16547	3.16	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
287	B/OSATEWWA	3D	F	16897	3.15	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
288	B/OASHWINI	2D	F	16908	3.16	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
289	B/OSAVITA	5D	F	12347	2.75	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
290	B/OVIJAYALAXMI	4D	F	17234	2.76	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
291	B/OPUSHPA	3D	M	17345	2.48	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
292	B/OANUSABAI	2D	M	17456	2.46	**NO**	**NO**	NO	NORMAL	NILL	**NEG**	**NEG**	**NEG**
293	B/OGEETA	5D	M	17658	2.82	NO	NO	MSAF	NORMAL	NILL	NEG	NEG	NEG
294	B/OPOOJA	4D	M	16785	2.72	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
295	B/OKALPANA	3D	M	14563	2.86	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG
296	B/OARATI	2D	M	13522	2.76	NO	NO	NO	NORMAL	NILL	NEG	NEG	NEG