"INFECTION AS A CAUSE OF STILLBIRTH: ONE YEAR HOSPITAL BASED OBSERVATIONAL STUDY"

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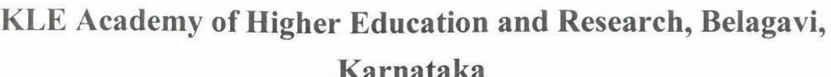

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Dr. TANMAYA METGUD M.D.
Professor & Head,
Department of Paediatrics,
J. N. Medical College,
Nehru Nagar,
Belagavi-590010

Date: 2/1/2023
Place: Belagavi.

Dr. N.S. MAHANTASHETTI M.D.
Principal
J.N. Medical College,
Nehru Nagar,
Belagavi-590010.

PRINCIPAL
J.N. Medical College,
BELAGAVI- 590 010

KAHER
J.N. MEDICAL COLLEGE
BELAGAVI

Date: 2/1/2023
Place: Belagavi.

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Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 - 2471350 0831 - 2470759 www.jnmc.edu principal@jnmc.edu

Ref No: MDC/PG/ Date: 22-12-2022.

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Principal,
J. N. Medical College, Belagavi.

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Reg. No. BM0120015,
Postgraduate Student,
2020-21 Batch,
Department of Paediatrics,
J. N. Medical College, Belagavi.

ETHICAL CLEARANCE CERTIFICATE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)

JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)

Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 157 **Date: 25/01/2021**

REG NO. BM0120015

To,
Dr. Priyam Rastogi
PG student in Paediatrics,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"INFECTION AS A CAUSE OF STILLBIRTH- ONE YEAR HOSPITAL BASED OBSERVATIONAL STUDY",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

11

ABSTRACT

Background and aims

Stillbirth is a common adverse pregnancy outcome, with nearly two million stillbirths occurring worldwide each year[1]. Stillbirth is a sensitive indicator of public health and is often not documented or underreported. Infection related stillbirths are preventable. This study aims to evaluate for infection as a cause of stillbirth using algorithm, organism involved and risk factors for infectious stillbirths.

Material and Methods

This observational study was conducted in a tertiary care centre and 92 stillbirths were evaluated focussing on the objectives of the study. Maternal and stillbirth data was collected by using a structured proforma. An algorithm[2] was used to identify the causes of stillbirths. Biospecimens such as Heart blood, Skin surface swab for culture and placenta for histopathology were collected to aid in the evaluation.

Results

During the study period there were 3207 deliveries and stillbirth rate was 35/1000 births. Infection related stillbirths were 12%, while no cause was found for 31% stillbirths. Ninety percent of the stillbirths occurred in women in the age group of 18-30 years. Asphyxia resulted in 34.7% stillbirths. Preterm stillbirths accounted for 72.8%, and 79.3% were antepartum stillbirths, while 51.1% were macerated. Statistically significant association was found between type of stillbirths and infectious stillbirths as compared to non-infectious stillbirths. Positive heart blood culture, skin surface culture and evidence of infection on placental histopathology was reported in 63.6%, 54.5% and 45.5% of the infectious stillbirths respectively.

Conclusion

Assigning a cause of stillbirth is important for planning interventions to reduce the stillbirth burden. Infection as a cause of stillbirth if detected can make a difference in improving pregnancy outcomes. Since significant number of infectious stillbirths assigned by using algorithm had positive blood culture, skin surface culture and histopathological evidence of infection indicating usefulness of algorithm as reliable method to assign a cause of stillbirth in low resource setting.

Keywords

Stillbirths, Infection, Algorithm, Culture, Histopathology

LIST OF ABBREVIATIONS

AIDS	-	Acquired immunodeficiency syndrome
BMI	-	Body mass index
CONS	-	Coagulase negative Staphylococcus
ENAP	-	Every New-born Action Plan
GBS	-	Group B Streptococcus
HIC	-	High income countries
HIV	-	Human immunodeficiency virus
ICD	-	International classification of diseases
INAP	-	India New-born Action Plan
IUD	-	Intrauterine death
LMIC	-	Low and middle-income countries
PCR	-	Polymerase chain reaction
SBR	-	Stillbirth rate
UN-IGME	-	United Nations Inter-Agency Group for Child Mortality Estimation
WHO	-	World health organization

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INTRODUCTION

"Stillbirth is defined as death that occurs prior to the complete expulsion or extraction from the mother of a fetus of equal to or more than 20 weeks of gestation or weighing equal to or more than 500 grams".[1] Stillbirths taking place in the intrapartum period usually have a normal appearance and are called fresh stillbirth. The skin not being intact, states that death has occurred more than 24 hours before delivery in the antepartum period and is called macerated stillbirth.

In the last 2 decades, India has recorded the highest number of stillbirths in the world with a decline in stillbirth rate by 53% from the years 2000-2019 and annual reduction rate of 4% as per the data collected by United nations Inter-Agency Group for Child Mortality Estimation (UN-IGME), 2020.[2] This data may be an overestimate, as stillbirths are often a neglected issue, especially in lower and low middle income countries.

All stillbirths are a tragedy in itself and a potential life loss. There are, various psycho-social consequences for parents such as anxiety, long-term depression, post-traumatic stress disorder and stigmatization.[3] Women who had a stillbirth are more likely to experience this again in following pregnancies than those who have not. An approximate of 2 million stillbirths occur worldwide every year[2], making stillbirth the fifth leading global cause of death across all age groups and outranking diseases like Diarrhea, HIV/AIDS, Tuberculosis, Road traffic accidents and Cancer.[3]

About 84% of stillbirths take place in low- and middle-income countries (LMIC). Stillbirth rates are maximum in South Asia and sub-Saharan Africa, with rates as high as 30-35/1000 births as compared to 1-2/1000 births in high income countries[2] (HIC). The causes of stillbirth and preterm birth in second trimester overlap

and also preterm delivery rate would be higher in populations with increased stillbirth rate, if preterm stillbirths are counted during estimation of preterm birth rates.[4]

According to the data it was seen that most of these deaths could be prevented. A perinatal audit in LMIC was done as a part of systematic review at a health facility by health care workers aiming at collecting information of timing, clinical demarcation of causes and associated conditions of death, to advance the quality of care.[5] When analyzed, the studies done before and after introduction of perinatal audit, a reduction in perinatal mortality of 30% was observed.

It is vital that we recognize the reasons for stillbirth and develop interventions with a focus on high-risk groups. In developing countries, the causes of stillbirth, generally similar across regions, include maternal infection, fetal asphyxia, trauma, congenital abnormalities, fetal-maternal hemorrhage, and a variety of maternal medical conditions. Yet, in a large number of cases the cause of stillbirth is currently not recognized and accounts for 25-60% of all fetal deaths.[6]

New global health figures show India to be among the six countries which have the highest rates of stillbirth in the world. In India, the rate was 13.9/1000 pregnancies in 2019. It accounted for 340,622 stillbirths out of a total 2 million of such births globally.[2] To reduce stillbirth rates, the prevalence, risk factors and causes must be known.

There are various causes for stillbirth, most common being fetal asphyxia followed by maternal or fetal conditions including obstructed labor, preeclampsia, abruption and placenta previa, umbilical cord complications, and placental malfunction due to thrombosis, necrosis, and fibrosis. Infection is likely the next most common cause of stillbirth.[7] In developed countries, about 15% of stillbirths appear to

be caused by infection. However, in many developing countries, estimates suggest that syphilis, malaria and various other intra uterine infections may contribute to half or more of the stillbirths.[8] Identification of such causes, improving detection by screening, appropriate and timely intervention may help to reduce stillbirths.

Stillbirths are a sensitive indicator of public health. Evaluating the stillbirth with surface swab and blood culture along with placental pathology may help to detect the infective cause of stillbirth. The causes and risks factors are to be studied in detail to plan interventions for preventing stillbirth. Therefore, we plan to evaluate for infection as a cause of stillbirth, risk factors and organisms involved.

OBJECTIVES

PRIMARY:

1) To know the incidence of infection as a cause of stillbirth*.

SECONDARY:

To know the following in stillbirths due to infection*:

1) Maternal and fetal risk factors.
2) Common organism causing infection.
3) Histopathological changes in the placenta.

* As per algorithm, McClure EM, Bose CL, Garces A, Esamai F, Goudar SS, Patel A, et al. Global network for women's and children's health research: a system for low-resource areas to determine probable causes of stillbirth, neonatal, and maternal death. Maternal Health Neonatology Perinatology. 2015;1(1).

REVIEW OF LITERATURE

Stillbirth is a common adverse pregnancy outcome, with nearly two million stillbirths occurring worldwide each year.[2] Eighty-four percent stillbirths occur in low- and middle-income countries (LMIC), and more than one million in the intrapartum period, despite many being preventable. In developing countries, methods of data collection are often inaccurate which results in underreporting of cases leading to low prevalence of stillbirth making it practically difficult to be recognized as a public health issue. Perinatal mortality reflects one of the important health indices and also a sensitive indicator of maternal and child health. In India nearly 60% of perinatal deaths are due to stillbirths, which can be prevented.[9]

Definition

World Health Organization (WHO) in its International Classification of Diseases Revision (ICD-11) defines fetal death as: "A sudden intrauterine death of a fetus at any point in time during the pregnancy and if the fetal death has occurred in the last half of the pregnancy, it can also be referred to as a stillbirth". Stillbirth is indicated after birth where the baby does not show any evidence of life, such as beating of the heart or a cry or movement of the limbs.

The WHO further describes early fetal deaths/stillbirths (death of a fetus weighing at least 500 grams or, if birth weight is unavailable, ≥ 22 weeks gestation, or with a crown-heel length of 25 centimeters or more) and late fetal deaths/stillbirths (fetal death weighing at least 1000 grams or a gestational age of ≥ 28 completed weeks or a crown-heel length of 35 centimeters or more)[10] (Figure 1)[11].

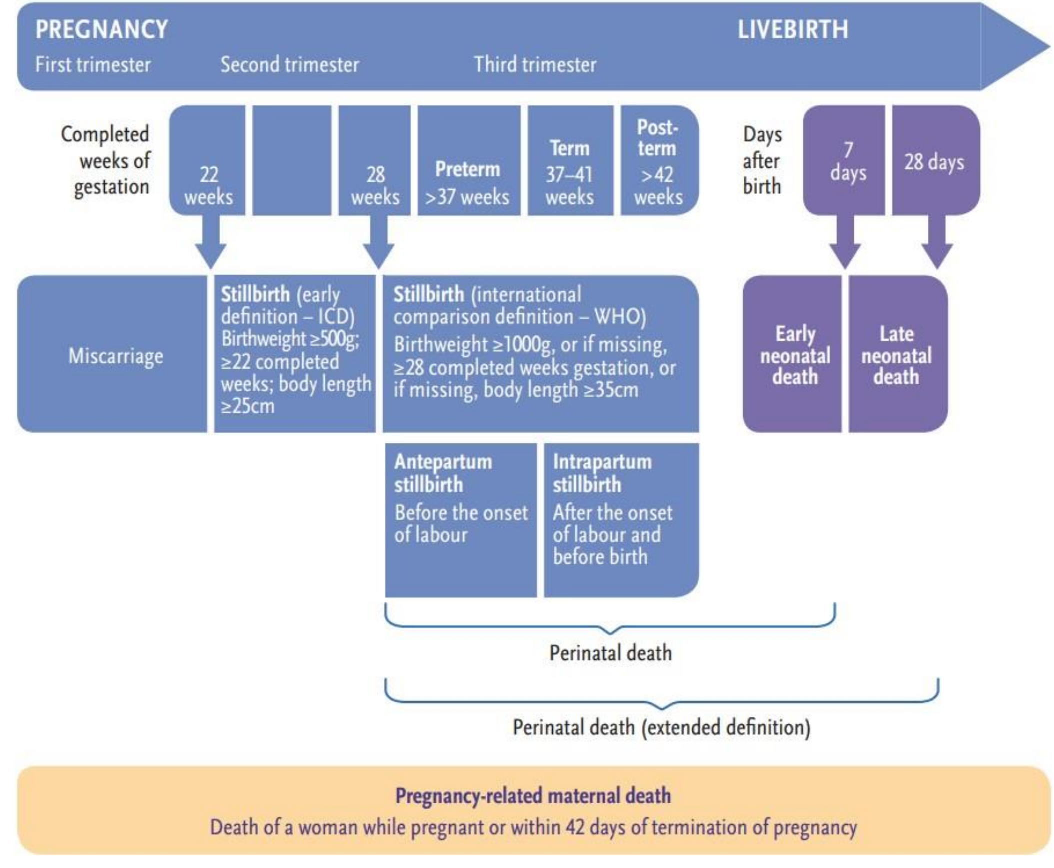


Figure1. Defining stillbirths, neonatal deaths and associated pregnancy outcomes

However, the definition of stillbirth varies from nation to nation and within the same nation. In high income countries, the definition tends to be at lower level of baby's maturity. In the UK, the definition of stillbirth is from 24 weeks of gestation[12] while in Canada and some states in the USA, it is as low as 20 weeks.[13] In LMIC, definitions of stillbirth are typically at a higher level of maturity, for example it is from 28 weeks in Nigeria, South Africa and Nepal.[14] Definitions also vary within countries. In India, Bhattacharya et al.[15] used 28 weeks of gestation as a benchmark for stillbirth, where as another study used 24 weeks.[16]

Stillbirth is categorized based upon the timing of death in relation to labor, intrapartum stillbirths are also known as fresh stillbirths as they occur after the onset

of labour in less than 12 hours before delivery with no skin changes. Antepartum stillbirth or macerated stillbirth, in which a baby is born with all the changes which occur in a fetus retained in utero after death and before the initiation of labour, showing skin and soft-tissue changes (skin discoloration or darkening, redness, peeling, and breakdown).[10]

Antepartum stillbirths occur due to combination of maternal, placental and fetal conditions. There are various risk factors for antepartum stillbirth like advanced maternal age, high parity, maternal smoking, obesity, antenatal infection etc. Intrapartum fetal death usually results from fetal distress and /or obstructed labor and often reflects poor quality of essential obstetric care.[17] Many factors are unidentified in the developing countries, where significant deliveries occur usually at home, although scenarios are changing with promotion of institutional deliveries by virtue of numerous government schemes.

Overview of Global stillbirth rate [3]

The Stillbirth rate is defined as the number of stillbirths per thousand births during the year. It is a sensitive indicator of quality of care in pregnancy, childbirth and a marker of a health system's strength.

One stillbirth happens every 16 seconds equating to about 2 million babies being stillborn at 28 weeks or more of gestation every year. The burden of stillbirths is huge but is often overlooked. Globally, in the past two decades 48 million babies were stillborn. Due to data limitations and the conventional approach taken, the real numbers might be more than reported. Most of them are preventable when life-saving interventions are applied with high quality health care.

According to United nations Inter-agency group for child mortality estimation (UN-IGME), 2020, 75% stillbirths occur in sub-Saharan Africa or Southern Asia. Low- and middle-income countries account for 84% of all stillbirths and 62% of all live births. About half of all the stillbirths occur in 6 countries, with the greatest number in India, followed by Pakistan, Nigeria, Democratic Republic of the Congo, China and Ethiopia.

Trends

Current trends if continued, an additional 20 million stillbirths will take place before 2030. With the current scenarios, the global stillbirth rate in 2019 was about 13.9 per 1000 births and only thirty five percent decline in stillbirth rate is seen since 2000. Every Newborn Action Plan (ENAP) of stillbirth, project a global target of 12 or less per 1000 births by 2030. In LMIC, 57% of the global stillborn babies were recorded, as compared to High-income and upper middle-income countries which had 16% of the global burden and have already achieved the ENAP target of 12 stillbirths or less per 1000 births. In the last two decades, the total number of stillbirths declined by 32 percent, from 2.9 million in 2000 to 2 million in 2019. There are large variations across countries with highest rate of 32.2 compared to lowest rate of 1.4 per 1000 births. The long-term trend indicates that the most significant reduction in stillbirth occurred between the years 1950 and 1975 when stillbirths were reduced by two-thirds. This is mainly attributed to improvement in health facilities and early identification and appropriate treatment of infection and better-quality of obstetric care. Therefore, the poor progress in reducing stillbirth and indeed maternal and neonatal deaths in low- and middle-income countries has been attributed to lack of action rather than knowledge.[18]

Stillbirth rate in India

Perinatal deaths are responsible for about 7% of the total global burden of disease. In a developing country like India there has been enormous improvement in health care system reflecting in the reduction of maternal and perinatal deaths when compared to the previous four decades.[19] The perinatal mortality rate is the index of current obstetrical and neonatal facilities especially with the waning of infant mortality rate to low level. In recent years, perinatal mortality rate is a better and reliable measure of maternal and child health care.

India has the highest number of stillbirths globally with an estimated 340,622 deaths per year with a decline of 60 percent since the last two decades and a WHO estimated rate of 13.9 per 1000 births with a variation of 11.4 to 17 per 1000 births. The government of India has also developed an India newborn action plan (INAP) which aims to reduce stillbirths to <10 per 1000 births by 2030.[20]

Etiology and risk factors of fetal death [21]

For better understanding and evaluation there is an undeniable need to enumerate the multifactorial risk factors and etiology associated with fetal death. The following factors contribute to fetal death:

Fetal:

- Chromosomal anomalies
- Non-chromosomal birth defects
- Nonimmune hydrops

• Infections-viruses, bacteria, protozoa

***Placental*:**

• Abruptio placenta

• Fetal-maternal hemorrhage

• Cord accidents

• Placental insufficiency

• Intrapartum asphyxia

• Placenta previa

• Twin to twin transfusion

• Chorioamnionitis

***Maternal*:**

• Antiphospholipid antibodies

• Diabetes Mellitus

• Hypertensive disorders

• Trauma

• Abnormal labour

• Anemias

• Infection.

Maternal risk factors associated with stillbirth

1. Age

The rate of pregnancy loss was higher among the advanced maternal age (beyond 35 years) as demonstrated by Fretts and colleagues. In many other studies the results have been evaluated, and it states that there is association with age despite correcting for important confounding factors such as inherited problems, birth defects, health problems and maternal weight.[22]

The main cause is the uteroplacental under perfusion. Collagen progressively replaces normal muscle in the walls of myometrial arteries in the older age group there by restricting luminal expansion leading to uteroplacental under perfusion which reduces birth weights more than birth lengths or head circumferences.

Lee et al.[23] evaluating risk factors for stillbirths during the antepartum period in rural Nepal, reported a proportional risk of stillbirth of 2.0 among mothers aged 35 or older (95% CI: 1.51 – 2.63). Mothers who were 30 years or older were observed to have an increased risk of stillbirth in a study in Zambia (OR: 1.79; 95% CI: 1.46 – 2.20) by Stringer et al.[24]. On the other hand, Engmann et al.[25], suggests that teenage mothers are at increased risk of stillbirth than older mothers [OR :1.49 (CI: 1.12–1.99)].

2. Demography

Certain demographic factors for fetal death include race, low socioeconomic status, inadequate prenatal care and less education. Taking into consideration the overall incidence in India, 70% of these pregnancy losses are in the low socio-economic group. Mother’s socioeconomic background are in turn related to the use of

alcohol, tobacco, and medical care. Alcohol consumption increases the risk of early fetal death, due to direct toxic effect, and also appears to cause anomalies.[26]

3. Parity

Engmann et al.[25] and McClure et al.[27], reported that nulliparity and multi-parity are associated with higher rates of stillbirth. A study in England during 2009-2011 to evaluate the key risk factors related with stillbirth in a multiethnic English maternity population recognized a noteworthy risk of stillbirth for parity 0 and ≥ 3 [17].

4. Obesity

Obesity in mothers is increasing gradually and is linked with an amplified risk of fetal macrosomia and perinatal mortality. The explanations for this association are thought to be due to socio-economical, cultural as well as antenatal factors. These women are more likely to have complications like gestational diabetes and hypertension leading to stillbirth.

Even after controlling these issues, a high BMI remains an important risk factor for stillbirth and its association seems to upsurge as the gestation increases. Obesity also led to hyperlipidemia, which may play a role in endothelial dysfunction, platelet aggregation, as well as to clinically significant atherosclerosis. Undeniably, in addition to advanced maternal age and low socio-economic status, the most important risk factor for stillbirth is pre-pregnancy obesity.[28] English maternity population identified a significant risk of stillbirth in women with a BMI ≥ 30, smoking and overt diabetes.[29]

5. Smoking

Smoking is associated with fetal growth restriction and probably also with placental abruption, which are two main causes of stillbirth.[30] Gardosi J et al.[17] showed an average risk of 1.36 for stillbirth in mothers found smoking in early pregnancy. The risk increased (RR 1.8, 95% CI:1.4-1.9), probably as a result of social deprivation, which is strongly related to smoking. Passive or environmental smoking was linked with increased risk of stillbirth by 30%.

6. Access to care

Many studies reported an association between poor antenatal attendance and stillbirth. In a multi-national study, McClure et al.[31] reported that mothers who did not attend antenatal care were almost twice the risk of experiencing a stillbirth than mothers who attended (RR:1.6; 95% CI:1.4–1.9). Bhattacharyya et al.[15] reported mothers who live in rural areas not attending obstetric care had an augmented risk of stillbirth.

7. Socioeconomic factors and education

Low socioeconomic status has been reported to increase the risk of stillbirth in multiple studies. In a study assessing risk factors for stillbirth in rural Nepal, higher socioeconomic status, measured by proxies such as land ownership, lowered the risk of stillbirth (RR: 0.85; 95% CI: 0.74–0.98).[32] Di Mario et al.[33] in a systematic review, showed low socioeconomic status has been reported to have an attributable factor of more than 50%.

Poor maternal education is another demographic factor frequently reported to increase the risk of stillbirth. In a multi-country study of 4,301 births, McClure et al.[31]

reported that women with no education were at higher risk of stillbirth (RR: 1.4, CI: 1.2, 1.5).

8. Emerging factor

Some factors that were rarely reported before are beginning to emerge from various studies. A secondary analysis of data from India by Sehgal et al.[34] has shown a gradual rise of stillbirth among women when used biomass for cooking (OR: 1.26; 95% CI: 1.12, 1.43). This strengthens the earlier report by Pope et al.[35] that indoor air pollution increases the risk of stillbirth (OR: 1.51; 95% CI: 1.23–1.85).

Causes of stillbirth

In order to understand and evaluate stillbirth, analysis and enumeration of its causes is necessary. The causes can be effectively understood when they are classified on the basis of maternal and fetal factors.

a. Hypertensive disorders complicating pregnancy

Hypertensive disorders complicating pregnancy are common and form one of the deadly triads, along with hemorrhage and infection, resulting in a large number of maternal and fetal deaths. The major cause of fetal compromise occurs as a consequence of reduced uteroplacental perfusion.

Decreased uteroplacental perfusion from constriction of the artery is definitely the main culprit contributing to perinatal morbidity and mortality. The average width of the myometrial spiral arterioles of normal prenatal women is 500 µm. The same measurement in women with preeclampsia is 200 µm. Due to chronic placental insufficiency, the fetuses are likely to be growth retarded. In the milder form when the

Blood Pressure is <160/100 mmHg, perinatal loss is about 10%. When blood pressure exceeds 160/110 mmHg, perinatal loss doubles and when complicated by preeclampsia it is three times more.[36]

For maternal conditions causing stillbirth, hypertensive disorders were frequently reported by researchers like McClure et al.[6], Awoleke & Adanikin et al.[37]. In a clinical trial involving 6,285 mothers in Bangladesh which reported patterns of antepartum complications and pregnancy-induced hypertension were found to be significant causes of stillbirth.

b. Infection

McClure et al.[6] have reported infections to be the maternal cause of stillbirth and it is a major contributor in developing countries. Infectious processes contribute to 10-25% of stillbirths in developed countries and even more in developing countries.[38]

The mechanism of stillbirth due to infection are firstly due to mothers' infection leading to severe systemic disease, in which the fetus may die, though the pathogens are not spread to the placenta or fetus. Next, the placenta may be infected without transmission of the pathogens to the fetus but the placenta has a compromised blood flow leading to a stillbirth. Finally, contamination of the fetus due to organisms itself may injure vital organs resulting in stillbirth or an anomaly may occur that subsequently kills the fetus.

A considerable amount of the infection related stillbirths in developing countries are due to fetal infection with microorganisms that also cause

chorioamnionitis. In developing countries, infections with Gram negative organisms such as Klebsiella pneumoniae and E. coli are common.[39]

F. Monari et al.[40] emphasized about the need for elaborative microbiological investigation to identify fetal infection associated with death. Evaluating each stillbirth for evidence of infection is important for making strategies to reduce stillbirth associated with infection.

Strategies include preventive measures focusing on reducing contact with infectious agents, hygienic practices and vaccination of mothers. Treatment comprising of starting empirical antibiotics in mothers with premature rupture of membranes and early interventions in cases of proven sepsis.

c. Diabetes mellitus

To elucidate the magnitude of fetal death, an understanding of the relationship of maternal glycemic levels and its contribution as a factor in causing fetal death is essential. Placental insufficiency, association with preeclampsia, associated fetal anomalies and unexplained are some mechanisms by which Diabetes mellitus contributes to fetal death.

Unexplained fetal deaths are exclusive to pregnancies complicated by overt diabetes. They are acknowledged as unknown as no clear reasons such as placental insufficiency, abruption, fetal growth restriction or oligohydramnios are present. Hyperglycemia mediate chronic aberrations in transport of oxygen and the fetal metabolites may account for the unexplained fetal death.[41]

d. Anemia

Iron deficiency anemia affects the fetoplacental unit which is clearly shown by the fact that in anemic women there is an increase in placental weight, an increase not related to fetal size which suggests inadequate oxygenation of the unit with a hypertrophic trophoblastic tissue. The importance of folate deficiency causes much argument about its effect on fetal wellbeing. At first, considered to be a cause of abruptio placenta and fetal loss, now there is a swing of opinion towards the view that it is important in the invasive stage of trophoblastic activity. The effect on fetus may be abortion, IUD or congenital anomaly.[41]

There is evidence indicating that pre-eclampsia and eclampsia occur more frequently in patient with iron deficiency or megaloblastic anemias than in non-anemic gravidas.

e. Genetic conditions

Chromosomal abnormalities are the best-known genetic cause of fetal demise seen in 6-12 % of stillbirths. Monosomy X (23%), trisomy 18 (21%), trisomy 21 (23%), and trisomy 13 (8%) are the common abnormalities noted. Autosomal recessive conditions as a result of single gene defect include conditions like glycogen storage diseases, metabolic disorders, and hemoglobinopathies, may lead to intrauterine fetal demise. X-linked conditions may cause death in male fetuses.

Few other causes of genetic abnormalities are also linked to stillbirth such as confined placental mosaicism tells us the presence of atypical chromosomes in some placental tissue with a normal fetal karyotype.[42]

f. Post-term pregnancy

Two large Swedish studies[43] analyzed that perinatal mortality amplified when pregnancy surpassed 41 weeks of gestation. Another study evaluated the perinatal outcome in 6624 post term pregnancies and reported that the complications associated with antepartum, intrapartum and neonatal death were higher at 42 weeks of gestation and beyond. The most noteworthy rise occurred during the intrapartum period. The main root cause of death was due to prolonged labor with cephalopelvic disproportion, "unexplained anoxia," and malformations.[44]

g. Antepartum hemorrhage

Placental causes of stillbirth, mainly abruptio placenta and placenta previa, continue to be some of the most frequently investigated causes of stillbirth. In the clinical trial by Khanam et al.[45], antepartum hemorrhage was found to increase the risk of stillbirth almost four-fold (IRR:3.7; 95% CI: 2.3–5). The percentage of stillbirths attributable to placental causes ranges between 8.0% and 17.7% shown by Lori et al.[46].

h. Malpresentation

Breech presentation is the commonest malpresentation. The fetal risk in terms of perinatal mortality is considerable in vaginal breech delivery. It is difficult to assess the magnitude of risk, because the complicating factors such as prematurity, twins, placenta previa, congenital malformations of the fetus etc., which are responsible or associated with breech, might contribute significantly to the fetal hazards. The overall perinatal mortality, ranges from 5-30% in hospital statistics of the developing countries.

Following are fetal damages in vaginal breech delivery.[41]

1. Intracranial hemorrhage, compression followed by decompression during delivery of the unmolded after coming head results in tear of the tentorium cerebelli and hemorrhage into the subarachnoid space. The risk is more in premature when the head is small and fragile. Baby can withstand anoxia following cord compression with the delivery of the trunk for about 5-7mins. A period of more than l0 mins will produce asphyxia of varying degrees.

2. Asphyxia, the prominent cause of fresh stillbirth because the head is delivered too slowly and other causes include:

a. Cord compression,

b. Retraction of the placental site

c. Premature attempt at breathing,

d. Cord prolapses.

i. Cord accidents

Cord accidents include occult prolapse, cord presentation and cord prolapse. It is common in malpresentations, polyhydramnios, twins and prematurity. Prolapse of the cord is also favoured by an unduly long cord (normal length 45-55cms). It is suggested that spasm of the cord vessels may be as important a cause of fetal death as actual mechanical blockage, as in cases when cord is actually allowed to prolapse outside the vagina and suffer a loss of temperature. Furthermore, handling of the cord may also cause spasm in which efforts to replace it can do little good. Prolapse of the cord at full dilatation followed by immediate delivery with forceps may save the baby

from an asphyxial death. The longer the interval between the prolapse of the cord and the delivery of the baby, greater is the fetal mortality. If delivery can be completed within half an hour, fetal mortality can be reduced to 10% or less. If it is more than half an hour it rises to nearly 40%.[47]

MATERIALS AND METHODS

The study was conducted in the department of pediatrics, KLES Dr. Prabhakar Kore Charitable Hospital and Medical Research Centre, Belagavi affiliated to JN Medical College, Belagavi from January 2021 to March 2022.

Study design

Observational study.

Study duration and period

Fifteen months, January 2021 to March 2022.

Place

KLES Dr. Prabhakar Kore Charitable Hospital and Medical Research Centre, Belagavi, a teaching hospital affiliated to Jawaharlal Nehru Medical College, Belagavi.

Source of data

Stillbirths diagnosed at admission or later, in mothers admitted to free labor room of KLES Dr. Prabhakar Kore charitable Hospital, Belagavi.

Sample size

$n = (Z)^2 * p(1-p) / E^2$

Z = 1.96 for 95 % confidence & 10% error

p = 0.40

1-p = 0.60

E = 10% error

$n = (1.96)^2 * 0.40 * 0.60 / (0.1)^2$

$n = 0.9219/0.01 = 92.19 \approx 92$

Total no. of sample size = **92**

Sample size for the study was ninety-two.

Ethical clearance

Prior to the commencement, study was approved by the Ethical and Research Committee, Jawaharlal Nehru Medical College, Belagavi.

Eligibility

Inclusion Criteria

1) Stillborn babies with gestational age ≥ 20 weeks.

Informed Consent

Stillbirths meeting the eligibility criteria were enrolled into the study after obtaining written informed consent from parents. Maternal and stillbirth data were recorded in a structured proforma.

Methodology

Maternal data:

Demographic details (Name, age) along with past and present obstetric history and any associated complication like pregnancy induced hypertension, eclampsia, anemia, gestational diabetes, previous miscarriage and stillbirths, history of drug usage, alcohol intake, tobacco chewing or smoking habits. Evidence of infection during pregnancy like fever, vaginal discharge, genital pruritus, foul smelling liquor, urinary tract infections, genital lesions or rash.

Stillbirth data:

Detailed data including sex, date and time of stillbirth, gestational age (weeks), weight (gm) and type (fresh/macerated) was recorded. Complete clinical examination to rule out gross congenital anomalies, chromosomal abnormalities, maceration, umbilical cord for true knots was done.

Biospecimens including heart blood (1-3 ml) from the right ventricle[48] and surface swab from the external ear canal of stillbirth were collected under all aseptic precautions, within 30 mins of delivery. Samples were sent for culture and sensitivity in blood culture bottle and culture tube (swab stick) to the microbiology department.

For histopathology, three blocks of full thickness of placental tissue, derived from central two third of the disc and one separate block to include the cross section of umbilical cord within 5 cm from the placental insertion[49] were sent in 10% neutral buffered formalin to pathology department.

All the data was recorded in a structured proforma.

Maternal risk factors for etiology of stillbirth comprised of maternal age, education status, parity, history of previous stillbirth, complications like pregnancy induced hypertension, gestational diabetes mellitus, anemia and antepartum hemorrhage. Fetal factors included gestational age, gender, birth weight, type of stillbirth, timing of stillbirth and mode of delivery.

To assign cause of stillbirth an algorithm* was used (annexure II).

***** McClure EM, Bose CL, Garces A, Esamai F, Goudar SS, Patel A, et al. Global network for women's and children's health research: a system for low-resource areas to determine probable causes of stillbirth, neonatal, and maternal death. Maternal Health Neonatology Perinatology. 2015;1(1).

Statistical analysis

The data was coded and tabulated on excel spreadsheet and master chart was prepared. The data was analyzed using IBM SPSS version 22 statistical software. Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency, and proportion for categorical variables. Categorical outcomes were compared between study groups using Chi square test /Fisher's Exact test. P value < 0.05 was considered statistically significant.

RESULTS

Table 1: Stillbirth rate

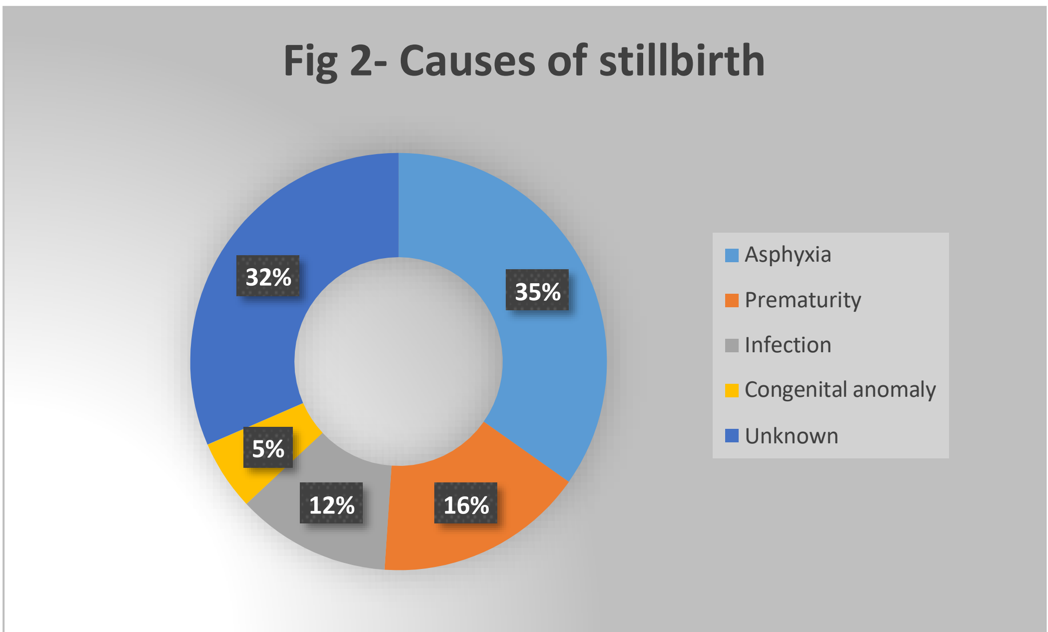
Total No. of Deliveries (≥20 weeks)	3207
Number of Stillbirths	112
Stillbirth Rate	35/1000

In our study, stillbirth rate was 35 per 1000 births.

Table 2: Causes of Stillbirth*

Causes of stillbirth*	No. Of stillbirths	Percentages
Asphyxia	32	34.78%
Prematurity	15	16.30%
Infection	11	11.96%
Congenital anomaly	5	5.43%
Unknown	29	31.52%
Total	92	100%

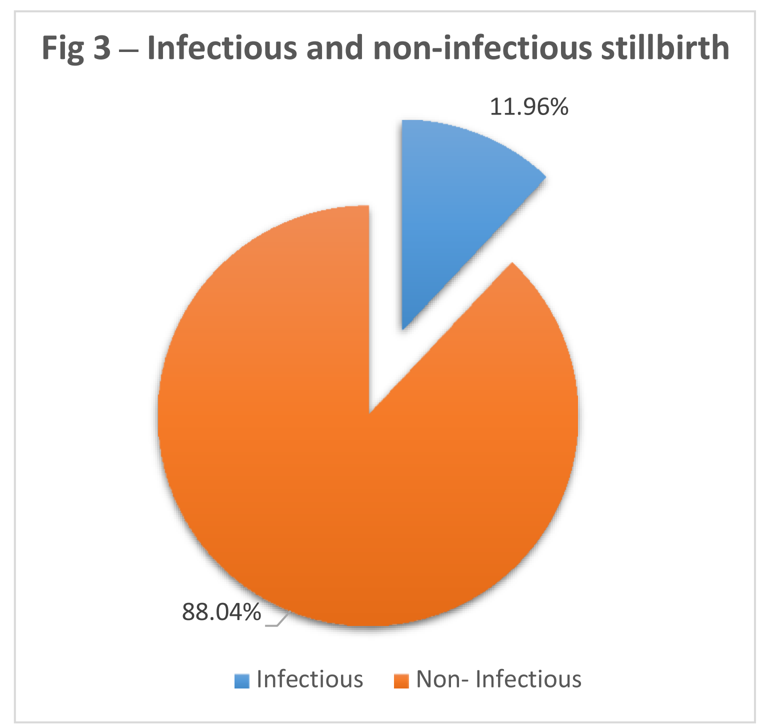
* As per algorithm, McClure EM, Bose CL, Garces A, Esamai F, Goudar SS, Patel A, et al. Global network for women's and children's health research: a system for low-resource areas to determine probable causes of stillbirth, neonatal, and maternal death. Maternal Health Neonatology Perinatology. 2015;1(1).



In more than one-third of the cases, asphyxia was cause of stillbirth followed by prematurity (16.3%) and infection (12%). No cause in 31.52 % of stillbirths.

Table 3: Infectious and non-infectious stillbirth

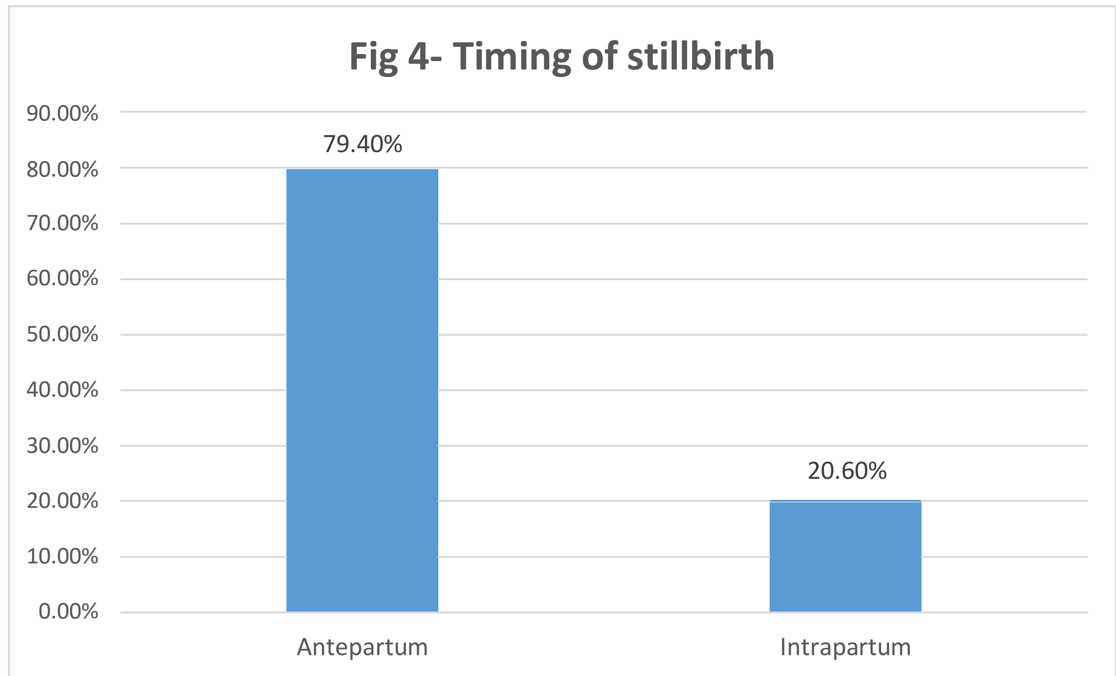
Cause	n (%)
Infectious	11 (11.96%)
Non-Infectious	81 (88.04%)
Total	92 (100%)



In our study, 11.96 % of stillbirths were due to infectious etiology.

Table 4: Timing of stillbirth

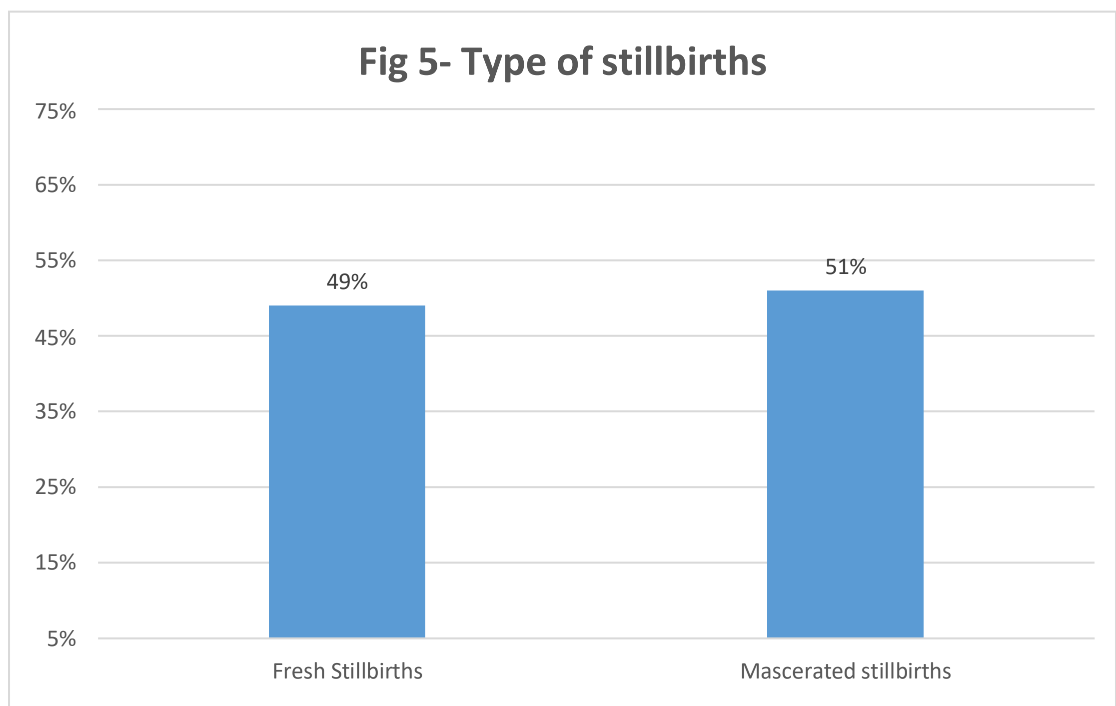
Timing	n (%)
Antepartum	73 (79.3%)
Intrapartum	19 (20.6%)
Total	92 (100%)



Little more than three-fourth (79.3%) of stillbirths occurred in antepartum period and rest were intrapartum.

Table 5: Type of stillbirth

Type	n (%)
Fresh	45 (48.92%)
Macerated	47 (51.08%)
Total	92 (100%)



In our study, little more than half of the stillbirths were macerated (51.08%) and rest were fresh stillbirths.

Table 6: Maternal risk factors

Risk Factors		n (%)		Chi square	P value
		Infectious Stillbirths (n=11)	Non-Infectious Stillbirths (n=81)		
Age (yrs)	18-25	4 (36.36%)	44 (54.32%)	3.972	0.137
	>25-30	7 (63.64%)	28 (34.57%)		
	>30	0 (0%)	9 (11.11%)		
Education	≤ 7 years	0 (0%)	6 (7.4%)	0.9	0.638
	8-12 years	10 (90.9%)	67 (82.7%)		
	> 12 years	1 (9.09%)	8 (9.8%)		
Parity	Primi	4 (36.36%)	41 (50.6%)	2.82	0.420
	Para 1	7 (63.64%)	32 (39.5%)		
	Para 2	0 (0%)	7 (8.6%)		
	Para 3	0 (0%)	1 (1.2%)		
Evidence of infection	Yes	3 (27.27%)	6 (7.41%)	4.330	0.072
	No	8 (72.73%)	75 (92.59%)		
Previous Stillbirth	Yes	1 (9.09%)	2 (2.47%)	1.346	0.321
	No	10 (90.91%)	79 (97.53%)		
Hypertensive disorders of pregnancy	Yes	2 (18.18%)	24 (29.63%)	0.626	0.722
	No	9 (81.82%)	57 (70.37%)		
Gestational Diabetes Mellitus	Yes	2 (18.18%)	5 (6.17%)	1.987	0.196
	No	9 (81.82%)	76 (93.83%)		
Anemia	Yes	2 (18.18%)	6 (7.41%)	1.416	0.244
	No	9 (81.82%)	75 (92.59%)		

In our study 90.2% of stillbirths occurred in women in the age group of 18-30 years with mean age of 25.14 ± 3.82 years. Women with no formal education accounted for 90.2% and 48.9 % were nulliparous. Evidence of infection was seen in 9.7% of the stillbirths and hypertensive disorders of pregnancy in 28.2%.

There was no statistically significant difference ($p > 0.05$) between infectious and non-infectious stillbirths with respect to maternal risk factors like age, education status, parity, evidence of infection, previous stillbirth and co morbidities like hypertensive disorders of pregnancy, gestational diabetes mellitus and anemia.

Table 7: Fetal risk Factors

Risk Factors		n (%)		Chi square	P value
		Infectious Stillbirths (n=11)	Non-Infectious Stillbirths (n=81)		
Gender	Male	4 (36.36%)	43 (53.09%)	1.084	0.298
	Female	7 (63.64%)	38 (46.91%)		
Gestational Age (weeks)	>20-28	3 (27.27%)	21 (25.93%)	2.984	0.394
	>28-32	1 (9.09%)	18 (22.22%)		
	>32-37	5 (45.45%)	19 (23.46%)		
	>37	2 (18.18%)	23 (28.4%)		
Birth weight	501-1000	2 (18.18%)	33 (40.74%)	3.140	0.371
	1001-1500	4 (36.36%)	18 (22.22%)		
	1501-2000	1 (9.09%)	12 (14.81%)		
	>2000	4 (36.36%)	18 (22.22%)		
Timing of stillbirth	Antepartum	11 (100%)	62 (76.54%)	3.25	0.071
	Intrapartum	0 (0%)	19 (23.46%)		
Type of stillbirth	Fresh	1 (9.09%)	44 (54.32%)	7.929	0.005
	Macerated	10 (90.91%)	37 (45.68%)		
Mode of delivery	LSCS	3 (27.27%)	14 (17.28%)	0.641	0.420
	Vaginal	8 (72.73%)	67 (82.72%)		

In our study, 26% of stillbirths were below 28 weeks, 46.7% were between

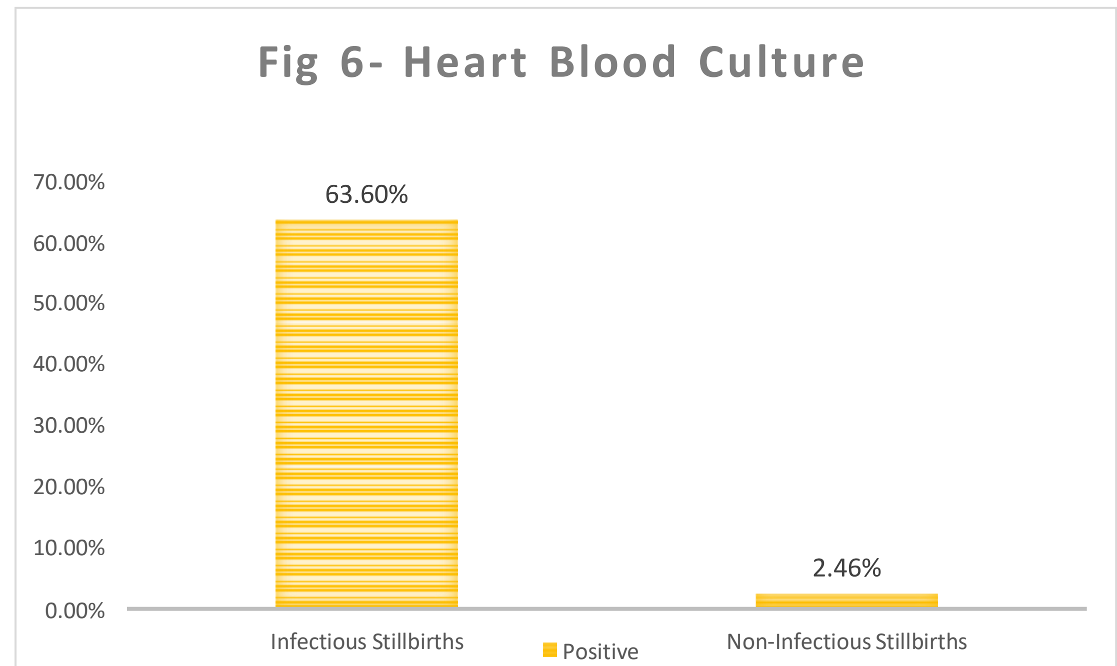
28-37 weeks, thus 72.8% stillbirths were preterm and 27.1% above 37 weeks of gestation with a mean gestational age of 33.14 ± 4.60 weeks.

In our study, 61.9 % weighed < 1500 grams, 14.1 % weighed 1501-2000 grams. Antepartum stillbirths were 79.3% and 51.1% macerated stillbirths. Fifty-two percent were male. Route of delivery in 81.5% of the stillbirths was vaginal.

Type of stillbirth was statistically significant ($p < 0.05$) with respect to infectious and non-infectious stillbirths while other risk factors like gender, gestational age, birth weight, timing, and the mode of delivery were not statistically significant ($p > 0.05$).

Table 8: Heart Blood culture

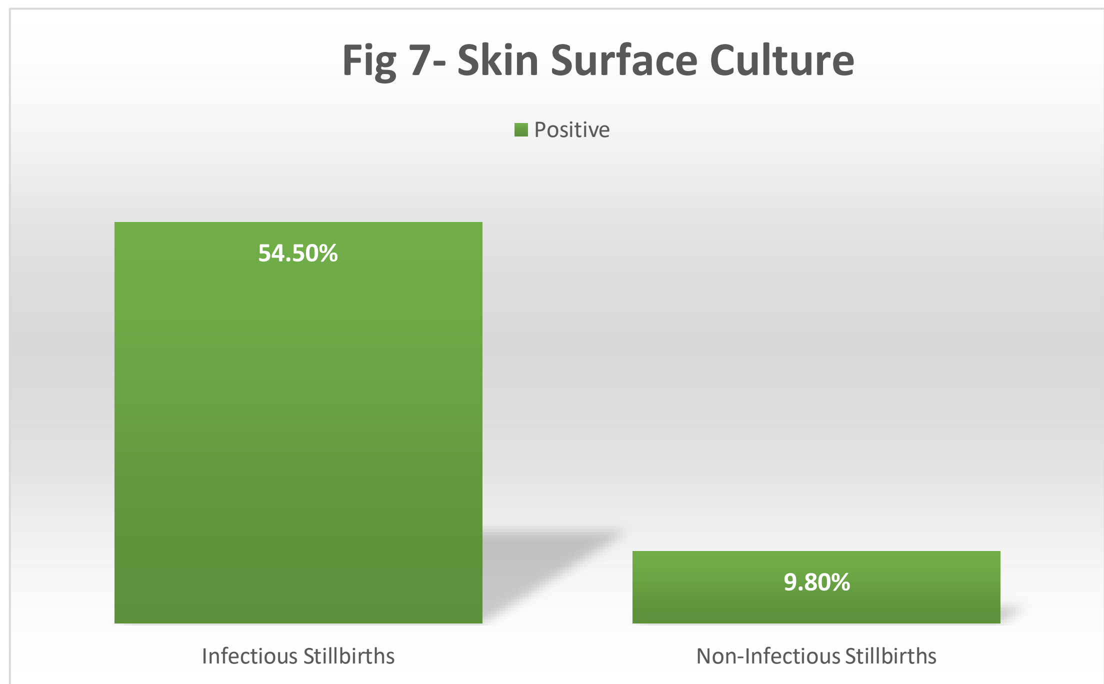
Blood culture	Infectious stillbirths (n=11)	Non-Infectious Stillbirths (n=81)	Chi square	P value
Positive	7 (63.6%)	2 (2.46%)	41.06	<0.001



Almost two-third of the infectious as compared to 2.46% of non-infectious stillbirths had shown growth of an organism in their heart blood which was statistically significant ($p < 0.001$).

Table 9: Skin Surface culture

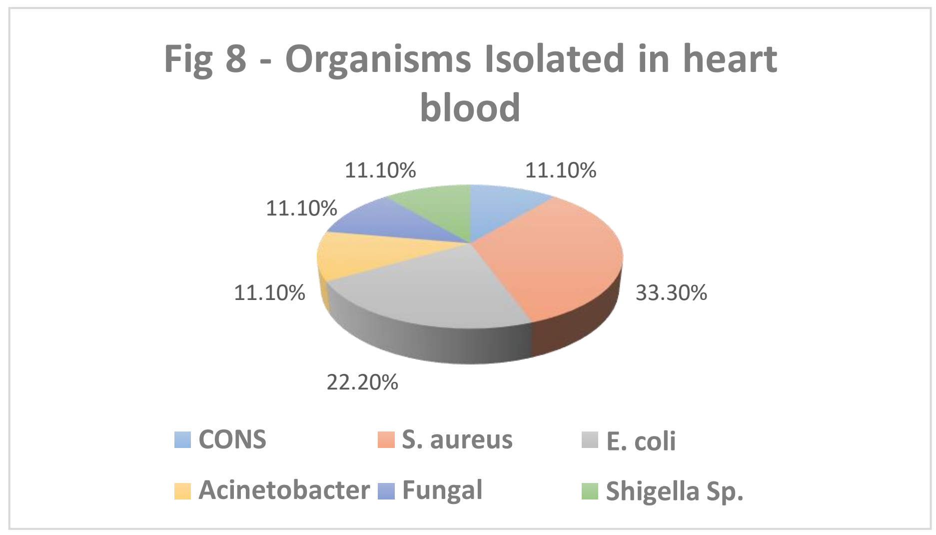
Surface culture	Infectious stillbirths (n=11)	Non-Infectious Stillbirths (n=81)	Chi square	P value
Positive	6 (54.5%)	8 (9.8%)	14.98	0.001



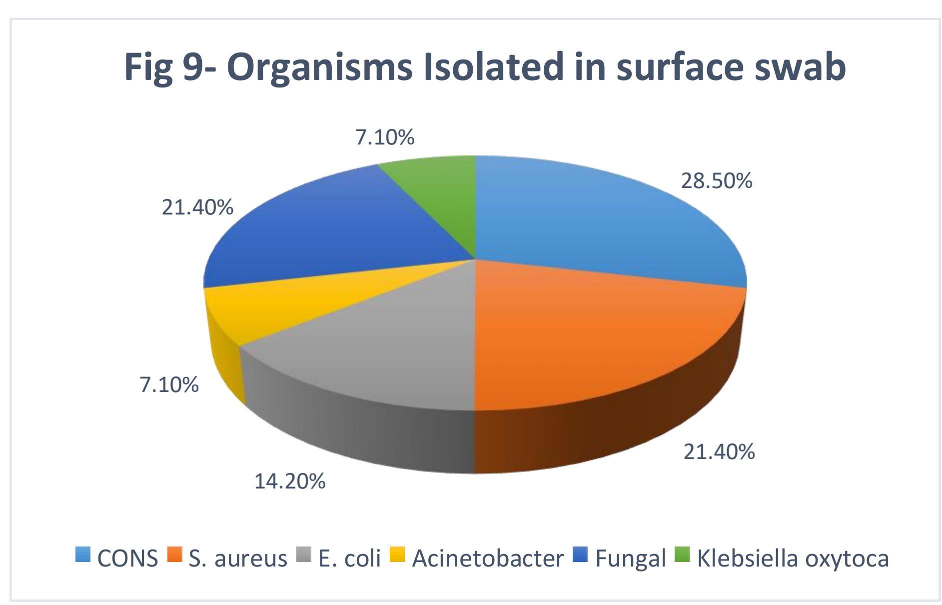
In our study, statistically significant (p = 0.001) association was found between infectious stillbirths and positive skin surface culture compared to non-infectious stillbirths.

Table 10: Organisms Isolated in stillbirths

Organisms Isolated	Heart Blood (n=9)	Surface Swab (n=14)
CONS	1 (11.1%)	4 (28.5%)
S. aureus	3 (33.3%)	3 (21.4%)
E. coli	2 (22.2%)	2 (14.2%)
Acinetobacter	1 (11.1%)	1 (7.1%)
Fungal	1 (11.1%)	3 (21.4%)
Shigella Sp.	1 (11.1%)	0 (0%)
Klebsiella oxytoca	0 (0%)	1 (7.1%)



Heart blood showed growth of S. aureus in 33.3%, E. coli in 22.2% followed by CONS, Acinetobactor, Fungus and Shigella sp. in 11.1% each.

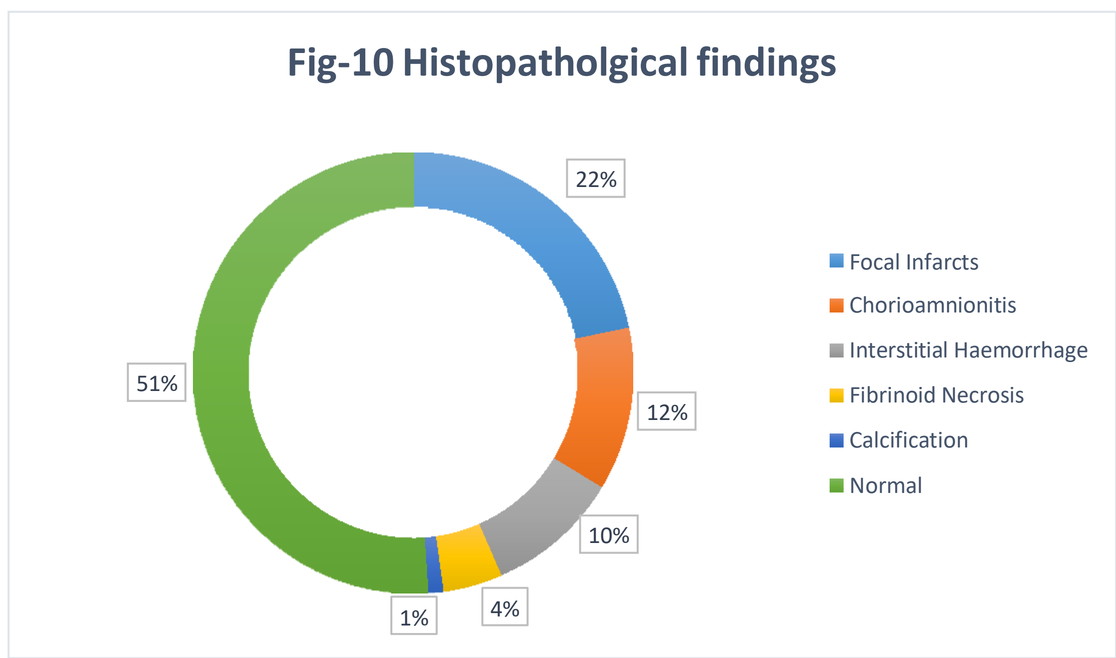


Most common organism isolated from skin surface culture was CONS (28.5%) followed by S. aureus, Fungus each (21.4%), and E. coli (14.2%).

In our study, no growth of organism was seen in 90.2 % of heart blood and 79.3% of surface swab culture.

Table 11: Placental histopathology in stillbirths.

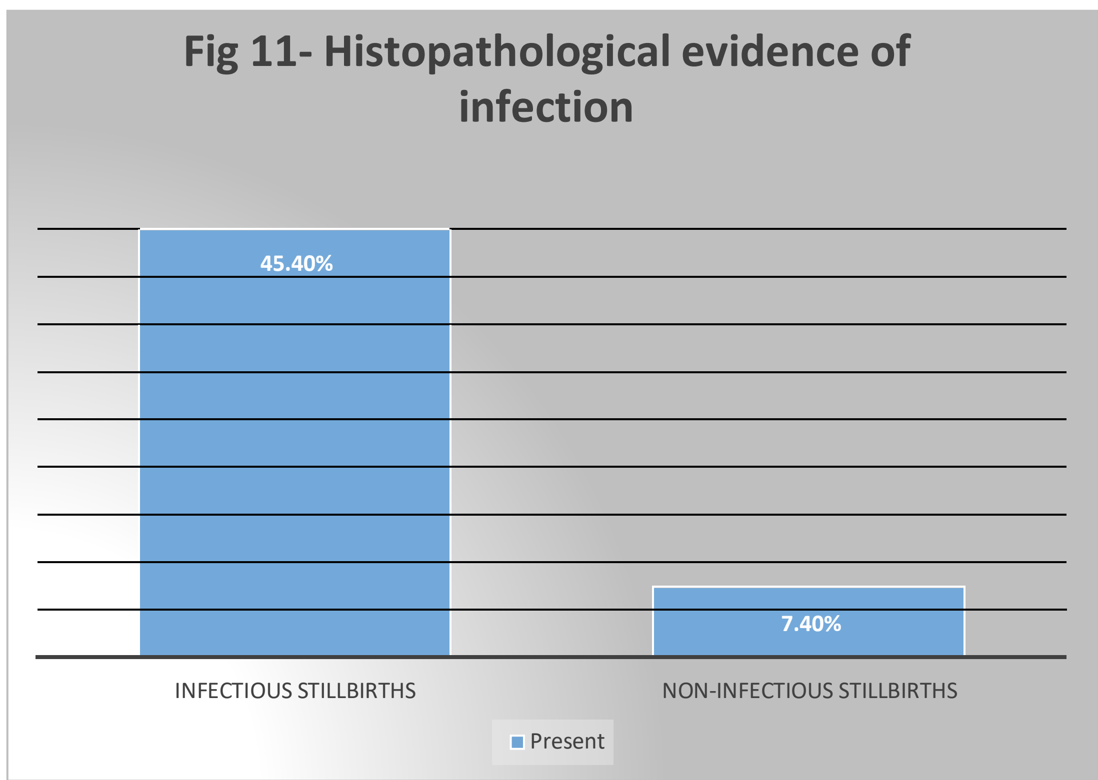
Findings	n (%)
Focal Infarcts	20 (21.74%)
Chorioamnionitis	11 (11.96%)
Interstitial Haemorrhage	9 (9.78%)
Fibrinoid Necrosis	4 (4.35%)
Calcification	1 (1.09%)
Normal	47 (51.09%)
Total	92 (100%)



Placental histopathology findings included focal infarcts (22%), chorioamnionitis (12%), interstitial haemorrhage (10%), other findings were fibrinoid necrosis and calcification. Normal findings were noted in 51% of stillbirths.

Table 12: Evidence of infection in placental histopathology

Histopathological evidence of infection	Infectious stillbirths (n=11)	Non-Infectious Stillbirths (n=81)	Chi square	P value
Present	5 (45.4%)	6 (7.4%)	13.32	0.003



In 45.4% of infectious stillbirths, placental histopathology showed evidence of infection, in contrast to 7.4% of non-infectious stillbirths which was statistically significant (p = 0.003).

DISCUSSION

Low and middle-income countries (LMIC) account for 84% of two million estimated stillbirths annually. A large number of factors like genetic, maternal, systemic infections, placental and fetal pathology have been associated with the risk of fetal death. Defining a cause of stillbirth is a difficult task as the process of death is frequently ambiguous, and thus the reduction in the global burden of stillbirths is dependent on planned actions that require a clear understanding of the stillbirth which is an important step towards the goal of reducing prevalence of stillbirth. In developing countries where hygiene maintenance is a challenge, the rate of infection related fetal deaths is high compared to high-income countries (HIC). As infection is a preventable cause, it needs to be looked upon carefully.

The present study was undertaken to assess the incidence and risk factors related with infectious stillbirths which will aid in initiating appropriate interventions to lessen the burden. In this study, 92 stillbirths were enrolled and analyzed.

The stillbirth rate varies worldwide among different countries and in various regions of the same country. The current stillbirth rate in LMIC is 22.7 per 1000 births and rate in India is 13.9 per 1000 births. In the present study, stillbirth rate is 35/1000 births (Table 1), which is comparable to the reports by Vaishali et al.[50] (35.2), Das et al.[51] (35.6) and Bhattacharya et al.[15] (33.6). Our study stillbirth rate is lower than reports by Kothiyal et al.[52] (78.3), Prasanna N et al.[53] (57.9) and Bellad et al.[54] (43) which may be due to many pregnant women presenting late to the labour room. Nayak et al.[55] reported 23.4 per 1000 births which is low compared to our report. Such major difference may be due to under reporting of stillbirths and our being a

tertiary care hospital, majority of cases referred are with one or more maternal complications.

Infection as cause of stillbirth was 12% which is concordant with reports by Fouks et al.[56] (15%), Page et al.[57] (12.9%) and other previous studies.[38,58] have reported rates of 10-25%. In our study, other causes included asphyxia (34.7%) followed by prematurity (16.3%), and no causes were defined in 31.5% stillbirths as per the algorithm which needs to be investigated further. Using the same algorithm, healthcare workers in a study by McClure et al.[39] assigned the causes of death ,46.6% were attributed to asphyxia, 21.3% to infection, 8.4% to congenital anomalies, 6.6% to prematurity and no cause was assigned in 17.1% of stillbirths. In Asphyxia-related stillbirths, obstructed labour, antepartum hemorrhage and preeclampsia or eclampsia were seen. Limitation in the study by McClure et al.[39] is that no investigations performed for confirming infection as a cause of stillbirth.

Stillbirth is an unpleasant event that is influenced by several factors, which can be maternal, fetal or combination. Maternal factors include mother's age, history of previous stillbirth, comorbidities like hypertensive disorders of pregnancy, gestational diabetes mellitus and anemia. In our study, 90.2% of stillbirths occurred in women within age group of 18-30 years and similar observations were made by Balu et al.[59] (80%), Rajagopal VM et al.[60] (71.4%) and Avachat S et al.[61] (75.8%). Hypertensive disorders of pregnancy were reported in 28.2% of the stillbirths in our study which is comparable to report by Nayak et al.[55] (28.5%) making them the leading cause of antepartum asphyxia. McClure et al.[27] reported that women with no formal education and nulliparous, were found to have an associated high risk for

stillbirths, which was not observed in our study. We did not find statistically significant association between maternal risk factors and infectious stillbirth.

We also evaluated for relation between stillbirths and fetal risks factors which included gender, gestational age, birth weight, timing of stillbirth, mode of delivery and type of stillbirth. The relation between infectious stillbirth and type of stillbirth was statistically significant ($p = 0.005$). In our study, almost 50% of the stillbirths were fresh. This probably may be related to delay in referring women with obstetric emergencies for which we do not have evidence. The other associated fetal causes were congenital anomalies (5.43%) which is similar (4%) to report by Jehan et al.[62]

In our study, heart blood culture grew organisms in 9.7% of the stillbirths, which is similar (7.8%) to the reports by E. Tolockiene et al.[48] and is low in comparison to F. Monari et al.[40] (21%). We observed positive heart blood culture in 63% of infection related stillbirths compared to non-infectious stillbirths which is statistically significant ($p < 0.001$). Literature search did not yield studies looking at positive blood culture in infectious and non-infectious stillbirths. Assigning the cause of stillbirth by using algorithm can identify two-thirds of the infectious stillbirths having a positive blood culture.

In about half of the infectious stillbirths, skin surface culture yielded organisms compared to non-infectious stillbirths which is statistically significant ($p = 0.001$). Study by E. Tolockiene et al.[48] reported positive skin surface culture in 32% of stillbirths. In our study, 54.5% of the infectious stillbirths diagnosed by using algorithm had a positive skin surface culture.

Most common organisms isolated were S. aureus (33.3%) and CONS (28.5%) in the heart blood culture and skin surface culture respectively. Other organisms

isolated were E. coli, Acinetobacter, Klebsiella and Shigella sp. Previous studies [59] have noted E. coli to be the leading pathogen. We did not isolate Group B streptococcus (GBS) in stillbirths enrolled in our study. Isolation of diverse group of organisms may be related to prevalence of organisms in different health care settings.

Placental histopathology findings in our study included focal infarcts (21.7%), chorioamnionitis (12%) and others like interstitial hemorrhage, fibrinoid necrosis, calcification together accounted for 15.2%. Similar observations were made by Page et al.[57] and E. Tolockiene et al.[48] with Focal infarcts 25% and 28%, Chorioamnionitis 9% and 10.6% respectively.

Evidence of chorioamnionitis in placental histopathology in infectious stillbirths is statistically significant ($p = 0.003$) as compared to non-infectious. Fouks et al.[56] reported, 80.5% of infection related stillbirths had evidence of chorioamnionitis on placental pathology which is higher in comparison to our study.

Infectious stillbirths compared to non-infectious had a statistically significant positive heart blood culture, surface swab culture and evidence of infection on placental histopathology indicating that the algorithm is useful in assigning infection as a cause of stillbirth in resource limited setting where laboratory support may not be available.

STRENGTHS AND LIMITATIONS

- Assigning the cause using the algorithm provides advantages of comparability, consistency and transparency.
- Statistically significant number of infectious stillbirths assigned by using algorithm had positive heart blood culture, skin surface culture and histopathological evidence of infection in the placenta. Thus, demonstrating that algorithm can be used to identify the cause of stillbirth in resource limited setting.
- As the study included limited population from single centre, results cannot be extrapolated to the whole population. Therefore, a study with large sample size from different geographical areas should be conducted.
- Another limitation of the study is that in almost one-third of the cases, cause of stillbirth could not be assigned.
- As this study was conducted in a tertiary care center, most enrolled mothers were high risk cases and thus results may not be applicable to the general population.
- There is a scope for using PCR technique which is accurate and reliable in evaluating infectious stillbirths diagnosed by algorithm, increasing the chances of isolating pathogens.

CONCLUSION

Assigning the cause of stillbirth is crucial to decrease stillbirth rate by implementing appropriate interventions. Since significant number of infectious stillbirths assigned by using algorithm had positive blood culture, skin surface culture and histopathological evidence of infection, thus indicating usefulness of algorithm in identifying the cause. The algorithm used in the study is simple and appropriate for use in low resource settings.

SUMMARY

Observational study was conducted from January 2021 to March 2022 in the Department of Pediatrics, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi affiliated to JN Medical College, Belagavi. A total of 92 stillbirths with gestational age equal to or more than 20 weeks were included in the study.

Following is the summary of study findings.

- During the study period there were 3207 deliveries and stillbirth incidence was 3.4%, with stillbirth rate of 35 /1000 births.

- Forty-six percent of stillbirths were in gestation of 28-37 weeks and 26% in 20-28 weeks. Stillbirth incidence in less than 1500 grams was 61.9% and 23.9% in more than 2000 grams.

- More than three fourth (79.3%) of stillbirths occurred in the antepartum period and 51.1% were macerated. There was no gender difference in the incidence of stillbirths.

- No statistically significant relation between maternal risk factors like age, education status, parity, history of previous stillbirth, evidence of infection, and comorbidities with infectious stillbirths.

- With respect to fetal risk factors, statistical significance was found with the type of stillbirth.

- As per the algorithm, 34.78% stillbirths were due to asphyxia, 31.52% classified as unknown, 16.3% due to complications of prematurity, 11.96% caused by infection and congenital anomalies in 5.43%.

- Infectious stillbirths had positive heart blood culture in 63.6%, skin surface culture in 54.5% and evidence of infection on placental histopathology in 45.4%.

- Among the stillbirths with positive blood culture, organisms isolated were S. aureus (33.3%), E. coli (22.2%) and CONS (11.1%).

- In stillbirths with positive skin surface culture, organisms isolated were CONS (28.5%) and S. aureus (21.4%).

- Focal infarcts (21.74%), chorioamnionitis (12%) and interstitial hemorrhage (9.78%) were the findings on histopathological evaluation of placenta.

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ANNEXURE I – CONSENT FORM

CONSENT FOR PARTICIPATION IN RESEARCH

"INFECTION AS A CAUSE OF STILLBIRTH - ONE YEAR HOSPITAL BASED OBSERVATIONAL STUDY"

Principal Investigator: REG NO. BM0120015
Guide: Dr. ______________
Co-Guide: Dr. _________________

Name Of the Participant:
You are hereby requested to involve your stillborn baby in the above said research to be conducted at KLES Dr. Prabhakar Kore Charitable Hospital and Medical Research Centre, Belgaum from January 2021 to December 2021 by me.

Introduction

Stillbirth is defined as death at the time of delivery at or after 20 weeks of gestation. Understanding the infectious cause contributing to stillbirth is needed to identify interventions that will reduce the incidence of stillbirths. Infections are a preventable cause of stillbirth. This needs to be addressed cautiously as stillbirth is a sensitive indicator of public health issues. Samples such as Heart blood and Surface swab will be taken under all aseptic precautions within 30 mins of stillbirth and will be sent to microbiology department in transport media for culture of microorganisms. Blocks of placenta will be sent in 10% neutral buffered formalin in sterile jars to the pathology department where tissue will be studied for histopathological changes.

Voluntary participation

Your stillborn baby's participation in this study is your voluntary decision. Whether to participate or not to participate will not affect your current or future relationship with the KLES Dr. Prabhakar Kore Charitable Hospital and Medical Research Centre, Belgaum.

You are free to discontinue the participation in the study at any time for any reasons and you will not be paid any reimbursement for participation in the research.

Risk and benefits

There are no major risks involved, other than discomfort and pain caused during the interview.

Privacy and Confidentiality

The only people who will know that you are a research participant are member of the research team. No information about you or provided by you, during research will be disclosed to others without your written consent. When the results of the research are published or discussed in the conferences, no information will be disclosed that would reveal your identity. Any information obtained in connections with this study and that can be identified with you remain confidential and will be disclosed only with your permission.

Queries

If you have any queries, you may contact

REG NO. BM0120015

Post Graduate Student

Department of Pediatrics

KLES academy of higher education & research.

Jawaharlal Nehru Medical College

Dr. ______________

Professor, Department of Pediatrics

KLES academy of higher education & research.

Jawaharlal Nehru Medical College, Belagavi-590010

If you have any questions about your rights or research participation you may contact

Dr. Harsha Hegde,
Chairperson, JNMC, IEC
& Scientist D,
ICMR, National Institute of Traditional Medicine
Belgaum -948022500.

You will be given a copy of this form for your information and to keep for your records.

STATEMENT OF CONSENT

I hereby voluntarily agree for my stillborn baby participation in this study. I understand that even if I choose to allow my stillborn baby to take part in this study, I have the liberty to withdraw at any time. My signature below indicates that I have read or have been told about this entire consent form including the risks and benefits and have had all my questions answered. I will be given a copy of this consent form.

Signature of the authorized representative/ parent: _______________

Date: _____________

Name:____

Relation to the Subject: __________________________________

Signature of the witness: ___________________________

Date: _____________

Name: ____

Signature of investigator: ___________________________

Date: _____________

Name: ____

ANNEXURE II – PROFORMA

"INFECTION AS A CAUSE OF STILLBIRTH - ONE YEAR HOSPITAL BASED OBSERVATIONAL STUDY"

Principal Investigator: REG NO. BM0120015

Guide: Dr.________________

Co-Guide: Dr._________________

IP NO. [ ]

SUBJECT NO. [ ]

MATERNAL INFORMATION

1. Name:
2. Age:
3. Education:
4. Occupation:
5. Address:
6. Telephone no.:

OBSTETRIC HISTORY

1. Obstetric score: Gestational age :____wks_____days
2. LMP: _ _/_ _/_ _ EDD: _ _/_ _/_ _
3. HDP / DM / UTI / Anemia/Thyroid disorder:
4. Previous stillbirth:Yes / No _____________________
5. Antenatal diagnosis of anomalies: Yes / No ________________
6. Evidence of infection:

a) Fever: Yes / No

b) Vaginal discharge/pruritis/foul smelling liquor: Yes / No

c) Urinary tract infections (UTI): Yes / No

d) Genital lesions or rash: Yes / No

DETAILS OF LABOUR

1. Date of delivery: _ _/_ _/_ _
2. Time of delivery: ______ AM/PM
3. Duration of labour: ____hrs_____mins
4. Duration of rupture of membranes:_____hrs____mins
5. Fetal heart rate (present/absent) If present,____/min Date: _ _/_ _/_ _
   Time: ______ am/pm
6. Fetal movements last appreciated Date: _ _/_ _/_ _
   Time: ______ am/pm
7. Mode of delivery: _______________
8. Placenta: Wt:______gms Umbilical cord:

STILLBIRTH INFORMATION:

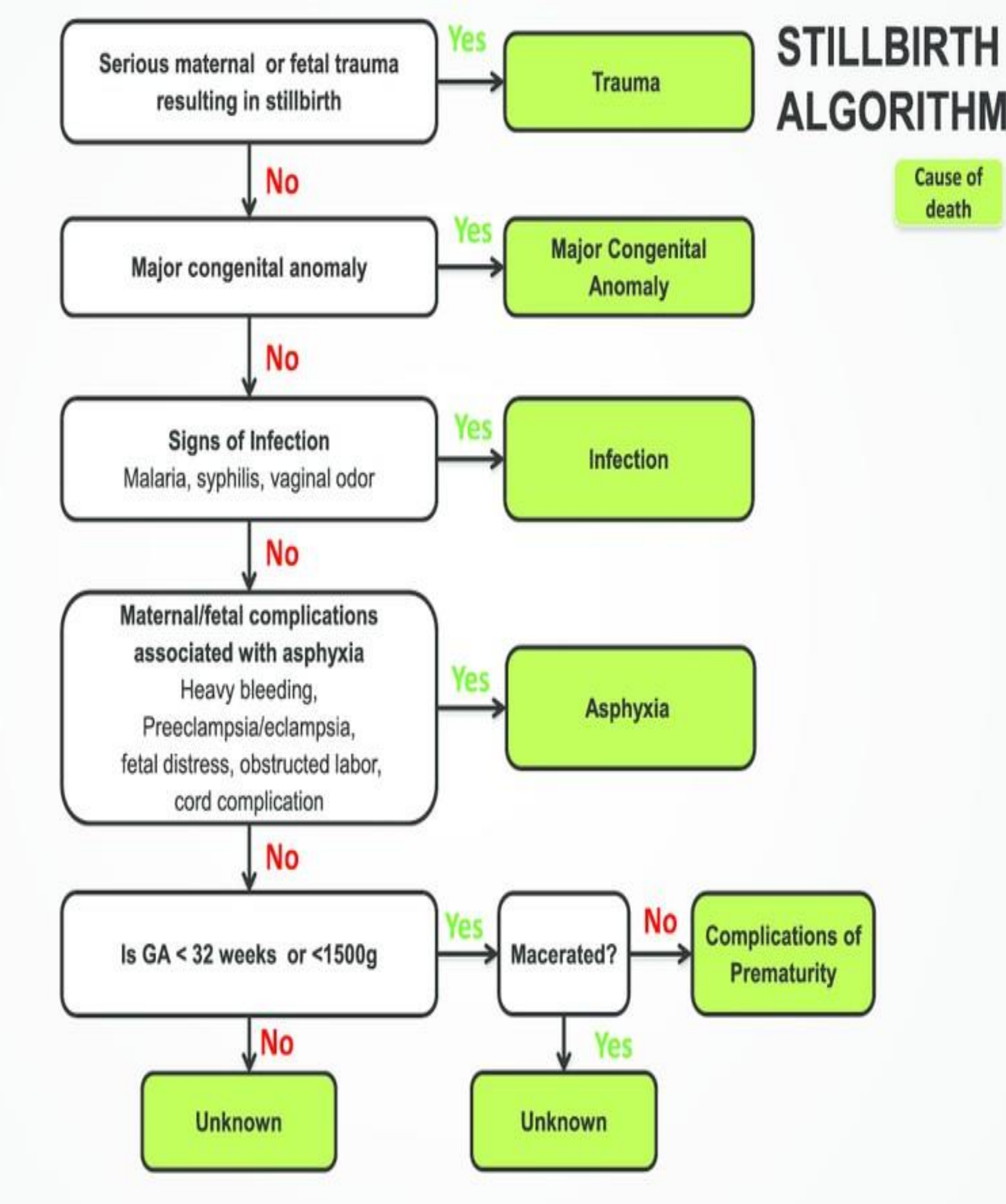
1. Sex: Male / Female Timing: Intrapartum / Antepartum
2. Birth weight:________gms
3. Gross congenital anomaly: Yes / No If yes, specify_______________
4. Chromosomal Abnormalities: Yes / No If yes, specify _______________
5. Stillbirth: FRESH / MASCERATED

INVESTIGATIONS

1) Stillborn baby
   a. Heart blood: _______________
   b. Surface swab: _______________
2) Placenta for histopathology: _________________________________

DIAGNOSIS

Assinging the cause of death:


STILLBIRTH ALGORITHM
Cause of death
Serious maternal or fetal trauma resulting in stillbirth
Yes
Trauma
No
Major congenital anomaly
Yes
Major Congenital Anomaly
No
Signs of Infection
Malaria, syphilis, vaginal odor
Yes
Infection
No
Maternal/fetal complications associated with asphyxia
Heavy bleeding, Preeclampsia/eclampsia, fetal distress, obstructed labor, cord complication
Yes
Asphyxia
No
Is GA < 32 weeks or <1500g
Yes
Macerated?
No
Complications of Prematurity
No
Unknown
Yes
Unknown

ANNEXURE III

KEY TO MASTERCHART

MATERNAL INFORMATION

IP No.

T1- Twin 1

T2- Twin 2

PARITY

Primigravida- 0

Para 1- 1

Para 2- 2

Para 3-3

EVIDENCE OF INFECTION

UTI- Urinary Tract Infection

MODE OF DELIVERY

LSCS- Lower segment caesarean section

GENDER

M- Male

F-Female

TYPE OF STILLBIRTH

FSB- Fresh stillbirth

MSB – Macerated stillbirth

INVESTIATIONS

NOGC- No growth of organism

WNL- Within normal limits

	MATERNAL INFORMATION				OBSTETRIC HISTORY						EVIDENCE OF INFECTION IN MOTHER	LABOR DETAILS				STILLBIRTH INFORMATION						INVESTIGATIONS			DIAGNOSIS
S.NO	IP. NO	AGE (years)	EDUCATION (years)	PARITY	PREVIOUS STILLBIRTH	ANTENATAL FETAL ANOMALY	THYROID DISORDER	HYPERTENSIVE DISEASE OF PREGNANCY	DIABETES MELLITUS	ANAEMIA	FEVER/VAGINAL DISCHARGE/RASH/UTI	DURATION OF RUPTURE OF MEMBRANE (DORM)	DURATION OF LABOUR (DOL)	FETAL HEART SOUND	MODE OF DELIVERY	GESTATION (WKS)	TYPE OF STILLBIRTH	TIMING OF STILLBIRTH	WEIGHT(Gms)	GENDER	GROSS ANOMALIES	HEART BLOOD CULTURE	SURFACE SWAB	PLACENTAL HISTOPATHOLOGY	CAUSE OF DEATH (as per stillbirth algorithm)
1	1027286	26	6	0	NO	NO	NO	NO	NO	NO	NO	2H 15M	8H 40M	PRESENT	VAGINAL	30W	MSB	ANTEPARTUM	1280	F	NO	NOGC	NOGC	WNL	UNKNOWN
2	1028135	26	10	1	NO	NO	NO	NO	NO	NO	NO	10H 30M	4H	ABSENT	VAGINAL	37W 1D	FSB	INTRAPARTUM	2600	M	NO	NOGC	NOGC	WNL	UNKNOWN
3	1028258	24	10	0	NO	NO	NO	YES	YES	NO	NO	7H	10H	ABSENT	VAGINAL	40W 3 D	MSB	ANTEPARTUM	3800	M	NO	NOGC	E.coli	WNL	APSHYXIA
4	1028396	25	12	1	NO	NO	NO	NO	NO	NO	NO	4H 30M	8H	ABSENT	VAGINAL	37W 1 D	MSB	ANTEPARTUM	1200	F	NO	NOGC	NOGC	WNL	UNKNOWN
5	1028593 (T1)	32	8	0	NO	NO	YES	NO	NO	NO	YES	90H	7H	PRESENT	VAGINAL	25W 3D	FSB	INTRAPARTUM	620	M	NO	NOGC	SKIN COMMENSALS	WNL	PREMATURITY
6	1028593 (T2)	32	8	0	NO	NO	YES	NO	NO	NO	YES	72h	7H	PRESENT	VAGINAL	25W 3D	FSB	INTRAPARTUM	580	M	NO	NOGC	NOGC	WNL	PREMATURITY
7	1029264	25	12	0	NO	NO	NO	NO	YES	NO	NO	00H 30M	8H 34M	PRESENT	VAGINAL	35W 5D	FSB	INTRAPARTUM	1000	F	NO	NOGC	NOGC	FOCAL INFARCTS	ASPHYXIA
8	1029528	35	B.com	1	NO	NO	NO	YES	NO	NO	NO	3H	14H	ABSENT	VAGINAL	40W 4D	MSB	ANTEPARTUM	720	M	NO	NOGC	NOGC	FOCAL INFARCTS	ASPHYXIA
9	1030048	22	12	0	NO	YES	NO	NO	NO	NO	NO	4H	12H	ABSENT	VAGINAL	36W 1D	MSB	ANTEPARTUM	2200	M	NO	NOGC	NOGC	WNL	UNKNOWN
10	1031453	25	B.sc	1	NO	NO	NO	NO	NO	NO	NO	00H 30M	9H	ABSENT	VAGINAL	25w 4d	MSB	ANTEPARTUM	785	M	NO	NOGC	NOGC	WNL	UNKNOWN
11	1031741	24	6	1	NO	NO	NO	NO	NO	NO	NO	61H	10H	PRESENT	VAGINAL	22W 1D	FSB	INTRAPARTUM	520	F	NO	NOGC	NOGC	WNL	PREMATURITY
12	1034537	30	B.com	1	NO	YES	NO	NO	YES	NO	NO	1H	12H	ABSENT	LSCS	35W 3D	MSB	ANTEPARTUM	2900	M	NO	STAPH. AUREUS	NOGC	WNL	INFECTION
13	1037071	28	12	1	NO	NO	NO	YES	NO	NO	NO	20H	12H	ABSENT	LSCS	30W 1D	FSB	ANTEPARTUM	1300	M	NO	NOGC	ACINETOBACTOR	WNL	ASPHYXIA
14	1038402	24	10	1	NO	NO	NO	NO	NO	NO	NO	1H 30M	8H 20M	ABSENT	VAGINAL	37W 3D	FSB	ANTEPARTUM	2000	F	NO	NOGC	NOGC	WNL	UNKNOWN
15	1038924	22	12	2	NO	NO	NO	NO	NO	NO	NO	9H	18H 30M	ABSENT	VAGINAL	30W 6D	MSB	ANTEPARTUM	1300	F	NO	NOGC	SKIN COMMENSALS	WNL	UNKNOWN
16	1040176	22	10	0	NO	NO	YES	YES	NO	NO	NO	4H 20M	8H	PRESENT	VAGINAL	26W 4D	FSB	INTRAPARTUM	750	F	NO	NOGC	NOGC	WNL	ASPHYXIA
17	1039657	22	12	1	YES	NO	NO	NO	NO	NO	NO	3H	16H 30M	ABSENT	VAGINAL	35W 1D	MSB	ANTEPARTUM	2200	F	NO	NOGC	NOGC	CHORIOAMNIONITIS	INFECTION
18	1043221	25	12	0	NO	NO	NO	NO	NO	NO	NO	4H 30M	8H	ABSENT	VAGINAL	24W 6D	FSB	ANTEPARTUM	600	F	NO	NOGC	NOGC	FOCAL INFARCTS	PREMATURITY
19	1043188	23	8	0	NO	NO	NO	NO	NO	NO	NO	4H	6H 20M	ABSENT	VAGINAL	37W 2D	MSB	ANTEPARTUM	1900	M	NO	NOGC	NOGC	WNL	UNKNOWN
20	1045959	29	8	1	NO	NO	NO	NO	NO	NO	NO	6H 30M	8H 20M	PRESENT	VAGINAL	27W 4D	FSB	ANTEPARTUM	1100	M	NO	NOGC	NOGC	WNL	PREMATURITY
21	1048018	23	12	0	NO	YES	NO	NO	NO	NO	YES	3H 30M	22H	PRESENT	VAGINAL	24W 1D	FSB	INTRAPARTUM	710	M	YES	NOGC	NOGC	WNL	ANOMALY
22	1048683	24	12	0	NO	NO	NO	NO	NO	NO	NO	2H	14H	ABSENT	LSCS	31W 1D	FSB	ANTEPARTUM	1500	F	NO	NOGC	NOGC	WNL	PREMATURITY
23	1049641	26	12	1	NO	YES	YES	NO	NO	NO	NO	6H	12H	ABSENT	VAGINAL	39W 4D	MSB	ANTEPARTUM	2800	M	NO	NOGC	NOGC	WNL	UNKNOWN
24	1052307	25	10	0	NO	NO	NO	YES	NO	NO	NO	2H 30M	9H 30M	PRESENT	LSCS	40W 1D	FSB	INTRAPARTUM	1800	F	NO	NOGC	NOGC	FOCAL INFARCTS	ASPHYXIA
25	1051390	28	12	1	NO	NO	YES	YES	NO	NO	NO	4H 20M	10H 20M	ABSENT	VAGINAL	31W 1D	MSB	ANTEPARTUM	900	F	NO	NOGC	NOGC	WNL	ASPHYXIA
26	1053140	23	10	0	NO	NO	NO	NO	NO	NO	NO	00H 30M	6H	ABSENT	VAGINAL	27W 6D	MSB	ANTEPARTUM	870	F	NO	E.coli	NOGC	WNL	INFECTION
27	1053679	23	10	1	NO	NO	NO	NO	NO	YES	NO	2H	8H	ABSENT	VAGINAL	28W	MSB	ANTEPARTUM	1200	M	NO	E.coli	E.coli	WNL	INFECTION
28	1051828	22	B.sc	0	NO	NO	NO	NO	NO	NO	NO	2H	12H 30M	ABSENT	VAGINAL	35W 6D	FSB	ANTEPARTUM	2100	F	NO	NOGC	NOGC	FOCAL INFARCTS	ASPHYXIA
29	1054600	24	12	1	NO	NO	YES	NO	NO	NO	NO	8H	12H	ABSENT	VAGINAL	24W 2D	MSB	ANTEPARTUM	720	M	NO	NOGC	NOGC	WNL	UNKNOWN
30	1054807	22	12	1	NO	NO	NO	YES	NO	NO	NO	4H	16H	ABSENT	VAGINAL	38W	MSB	ANTEPARTUM	1800	F	NO	NOGC	NOGC	WNL	ASPHYXIA
31	1059824	24	12	1	NO	NO	NO	NO	NO	NO	NO	4H 40M	4H 40M	ABSENT	LSCS	40W 1D	FSB	ANTEPARTUM	2600	F	NO	NOGC	NOGC	WNL	UNKNOWN
32	1060009	29	12	2	NO	NO	NO	NO	YES	NO	YES	00H 40M	6H	ABSENT	LSCS	31W	FSB	ANTEPARTUM	1500	F	NO	NOGC	NOGC	INTERSTITIAL HMG	PREMATURITY
33	1060018	20	10	0	NO	NO	NO	NO	NO	NO	NO	24H	18H 30M	ABSENT	VAGINAL	35W 5D	FSB	ANTEPARTUM	2500	F	NO	NOGC	NOGC	FOCAL INFARCTS	ASPHYXIA
34	1061778	21	10	0	NO	NO	NO	NO	NO	NO	NO	72H	19H	PRESENT	VAGINAL	29W 2D	FSB	INTRAPARTUM	1000	M	NO	NOGC	CONS;E.coli	WNL	PREMATURITY
35	1062237	32	12	1	NO	NO	NO	NO	NO	YES	NO	9H	8H	ABSENT	LSCS	34W 1D	FSB	ANTEPARTUM	1900	M	NO	NOGC	NOGC	FOCAL INFARCTS	UNKNOWN
36	1062450	30	B.A	0	NO	NO	YES	YES	NO	NO	NO	00H 30M	9H 30M	PRESENT	VAGINAL	32W 2D	FSB	INTRAPARTUM	1000	F	NO	NOGC	NOGC	INTERSTITIAL HMG	PREMATURITY
37	1063829	24	12	2	NO	NO	NO	YES	NO	NO	NO	2H 30M	6H 20M	ABSENT	VAGINAL	38W 1D	MSB	ANTEPARTUM	3300	M	NO	NOGC	NOGC	FOCAL INFARCTS	ASPHYXIA
38	1064072	26	10	0	NO	YES	NO	NO	NO	NO	NO	01H	8H	PRESENT	VAGINAL	24W 6D	FSB	INTRAPARTUM	640	M	NO	NOGC	NOGC	WNL	PREMATURITY
39	1067328	33	12	0	NO	NO	NO	NO	NO	NO	NO	20m	6H	ABSENT	VAGINAL	26W 4D	MSB	ANTEPARTUM	630	M	NO	NOGC	NOGC	WNL	UNKNOWN
40	1067596	24	12	2	NO	NO	NO	YES	NO	NO	NO	10H 30M	20H	ABSENT	LSCS	34W 6D	MSB	ANTEPARTUM	1600	F	NO	NOGC	NOGC	FOCAL INFARCTS	ASPHYXIA
41	1068252	31	12	1	YES	NO	YES	NO	NO	NO	NO	00H 20M	52H	ABSENT	VAGINAL	33W 2D	MSB	ANTEPARTUM	1800	M	NO	NOGC	NOGC	WNL	UNKNOWN
42	1068727	27	12	0	NO	NO	NO	NO	NO	NO	NO	16H	16H	ABSENT	VAGINAL	37W 6D	MSB	ANTEPARTUM	750	M	NO	NOGC	NOGC	CHORIOAMNIONITIS	UNKNOWN
43	1068755	32	5	0	NO	NO	YES	YES	NO	NO	NO	7H 20M	12H	ABSENT	VAGINAL	29W	FSB	ANTEPARTUM	770	M	NO	NOGC	CONS	INTERSTITIAL HMG	ASPHYXIA
44	1068868	22	12	0	NO	YES	NO	NO	NO	NO	NO	38H	8H 30M	ABSENT	VAGINAL	23W 2D	FSB	ANTEPARTUM	520	M	NO	NOGC	NOGC	FIBRINOID NECROSIS	ANOMALY
45	1071163	20	10	0	NO	NO	NO	NO	NO	NO	NO	16H 30M	18H	ABSENT	VAGINAL	40W 3D	MSB	ANTEPARTUM	3100	M	NO	NOGC	NOGC	WNL	UNKNOWN
46	1071160	30	8	1	NO	NO	NO	YES	NO	NO	NO	3H 20M	8H	ABSENT	VAGINAL	33W 4D	MSB	ANTEPARTUM	1300	F	NO	NOGC	CANDIDA ALBICANS	CHORIOAMNIONITIS	INFECTION
47	1071163	20	5	0	NO	NO	NO	NO	NO	NO	NO	00H 40M	12H 30M	ABSENT	VAGINAL	40W 3D	MSB	ANTEPARTUM	3100	F	NO	NOGC	NOGC	WNL	UNKNOWN
48	1072031	26	12	0	NO	NO	NO	YES	NO	NO	NO	00H 30M	8H	ABSENT	VAGINAL	33W 4D	MSB	ANTEPARTUM	1200	F	NO	NOGC	NOGC	CHORIOAMNIONITIS	ASPHYXIA
49	1072459	19	12	0	NO	NO	YES	NO	NO	NO	YES	04H 30M	12H	ABSENT	VAGINAL	28W 2D	MSB	ANTEPARTUM	2200	M	NO	NOGC	MRSA	FOCAL INFARCTS	ASPHYXIA
50	1074525	22	5	1	NO	NO	NO	NO	NO	NO	NO	00H	4H	ABSENT	LSCS	39W 6D	MSB	ANTEPARTUM	1700	M	NO	NOGC	ASPERGILLUS SP.	WNL	UNKNOWN
51	1077082	20	10	1	NO	NO	NO	NO	NO	NO	YES	4H 20M	6H 20M	ABSENT	VAGINAL	33W 2D	MSB	ANTEPARTUM	1300	F	NO	NOGC	NOGC	FOCAL INFARCTS	ASPHYXIA
52	1079619	25	10	0	NO	NO	NO	NO	NO	NO	NO	2H 30M	6H 30M	PRESENT	VAGINAL	30W 4D	FSB	INTRAPARTUM	1200	F	NO	NOGC	MRSA	FOCAL INFARCTS	ASPHYXIA
53	1080518	19	10	0	NO	YES	NO	NO	NO	NO	NO	5H	10H	ABSENT	VAGINAL	36W 6D	MSB	ANTEPARTUM	1300	M	YES	STAPH. AUREUS	SKIN COMMENSALS	FOCAL INFARCTS	ANOMALY
54	1080739	21	10	0	NO	NO	NO	YES	NO	NO	NO	00H	00H	ABSENT	LSCS	32W 4D	FSB	ANTEPARTUM	1200	M	NO	NOGC	NOGC	WNL	ASPHYXIA
55	1082626	27	10	0	NO	NO	NO	NO	NO	NO	YES	2H 30M	8H	ABSENT	VAGINAL	29W 5D	MSB	ANTEPARTUM	1100	F	NO	NOGC	NOGC	CHORIOAMNIONITIS	INFECTION
56	1082768	23	10	0	NO	NO	YES	YES	NO	NO	NO	4H 30M	10H	ABSENT	VAGINAL	41W 5D	FSB	ANTEPARTUM	3000	F	NO	NOGC	NOGC	INTERSTITIAL HMG	ASPHYXIA
57	1084108	32	B.com	1	NO	NO	NO	YES	NO	NO	NO	2H	6H	ABSENT	VAGINAL	32W 1D	MSB	ANTEPARTUM	900	M	NO	NOGC	NOGC	FOCAL INFARCTS	ASPHYXIA
58	1085005	24	10	0	NO	NO	NO	NO	NO	NO	NO	00H 30M	6H 30M	ABSENT	VAGINAL	36W 1D	MSB	ANTEPARTUM	1000	M	NO	NOGC	NOGC	WNL	UNKNOWN
59	1085884	27	10	1	NO	NO	NO	YES	NO	NO	NO	2H	8H	ABSENT	VAGINAL	34W 3D	MSB	ANTEPARTUM	1500	F	NO	NOGC	CONS	CHORIOAMNIONITIS	INFECTION
60	1086429	30	8	3	NO	NO	YES	YES	NO	NO	NO	1H	4H	ABSENT	VAGINAL	24W 3D	MSB	ANTEPARTUM	550	M	NO	Acinetobactor	NOGC	FOCAL INFARCTS	ASPHYXIA
61	1087229	24	10	2	NO	NO	NO	NO	NO	NO	NO	9H	8H 30M	PRESENT	VAGINAL	38W 3D	FSB	INTRAPARTUM	2500	M	NO	NOGC	NOGC	FOCAL INFARCTS	UNKNOWN
62	1086765	30	10	1	NO	YES	NO	NO	NO	NO	NO	3H	4H 30M	PRESENT	VAGINAL	20W 2D	FSB	INTRAPARTUM	520	M	YES	NOGC	NOGC	WNL	ANOMALY
63	1088499	23	12	1	NO	NO	NO	NO	NO	NO	NO	8H	6H 30M	ABSENT	VAGINAL	32W 2D	MSB	ANTEPARTUM	600	F	NO	NOGC	NOGC	INTERSTITIAL HMG	UNKNOWN
64	1090183	26	12	1	NO	NO	YES	NO	YES	NO	NO	8H 30M	10H	ABSENT	VAGINAL	33W	FSB	ANTEPARTUM	1550	F	NO	NOGC	NOGC	FIBRINOID NECROSIS	ASPHYXIA
65	1090205	24	B.sc	0	NO	NO	NO	NO	NO	NO	NO	8H	10H	ABSENT	VAGINAL	40W 4D	MSB	ANTEPARTUM	1600	F	NO	NOGC	NOGC	INTERSTITIAL HMG	UNKNOWN
66	1090429	33	12	1	NO	NO	NO	NO	NO	NO	NO	18H	16H	PRESENT	VAGINAL	28W	MSB	ANTEPARTUM	900	F	NO	NOGC	NOGC	INTERSTITIAL HMG	UNKNOWN
67	1090408	30	10	1	NO	NO	NO	NO	NO	NO	YES	12H 30M	4H 30M	PRESENT	VAGINAL	26W 6D	MSB	ANTEPARTUM	1000	M	NO	MSSA	Ear commensals	WNL	INFECTION
68	1091805	30	12	0	NO	NO	NO	NO	YES	NO	NO	9H 30M	8H	ABSENT	VAGINAL	37W 2D	MSB	ANTEPARTUM	2600	F	NO	NOGC	NOGC	WNL	UNKNOWN
69	1093246	26	10	1	NO	NO	NO	YES	NO	NO	NO	00H 30M	6H	ABSENT	VAGINAL	27W 5D	FSB	ANTEPARTUM	530	F	NO	NOGC	SKIN COMMENSALS	FIBRINOID NECROSIS	ASPHYXIA
70	1093389	19	12	0	NO	NO	NO	YES	NO	YES	NO	00H 40M	3H	ABSENT	VAGINAL	22W 4D	FSB	ANTEPARTUM	520	F	NO	NOGC	NOGC	WNL	ASPHYXIA
71	1093943	28	B.sc	1	NO	NO	NO	NO	NO	NO	NO	2H 30M	8H	ABSENT	VAGINAL	40W 1D	MSB	ANTEPARTUM	2800	F	NO	NOGC	NOGC	CHORIOAMNIONITIS	UNKNOWN
72	1096044	24	12	1	NO	NO	NO	NO	NO	NO	NO	00H 30M	8H	ABSENT	VAGINAL	31W 5D	MSB	ANTEPARTUM	1600	F	NO	NOGC	NOGC	CHORIOAMNIONITIS	ASPHYXIA
73	1097208	22	10	1	NO	NO	NO	NO	NO	YES	NO	2H 30M	8H	ABSENT	VAGINAL	30W 6D	FSB	ANTEPARTUM	1600	F	NO	NOGC	NOGC	FOCAL INFARCTS	ASPHYXIA

74	1096640	19	12	1	NO	NO	NO	NO	NO	NO	NO	40H 30M	6H 20M	PRESENT	VAGINAL	26W 2D	FSB	INTRAPARTUM	850	M	NO	NOGC	NOGC	CHORIOAMNIONITIS	PREMATURITY
75	1098397	24	B.sc	0	NO	NO	NO	NO	NO	NO	NO	00H	00H	PRESENT	LSCS	37W 4D	FSB	ANTEPARTUM	2100	M	NO	NOGC	NOGC	WNL	UNKNOWN
76	1098888	22	12	0	NO	NO	NO	YES	NO	NO	NO	00H 30M	4H	PRESENT	VAGINAL	28W 4D	FSB	INTRAPARTUM	575	M	NO	NOGC	NOGC	FIBRINOID NECROSIS	ASPHYXIA
77	1101524	28	10	1	NO	NO	YES	YES	NO	NO	NO	00H	00H	PRESENT	LSCS	28W 3D	FSB	INTRAPARTUM	520	F	NO	NOGC	NOGC	INTERSTITIAL HMG	ASPHYXIA
78	1101372	19	10	0	NO	NO	NO	NO	NO	NO	NO	00H 30M	4H	PRESENT	VAGINAL	24W 1D	MSB	ANTEPARTUM	600	M	NO	NOGC	Kleibsiella oxytoca	WNL	UNKNOWN
79	1102184	29	12	0	NO	NO	NO	NO	YES	NO	NO	00H	00H	ABSENT	LSCS	38W 4D	MSB	ANTEPARTUM	4300	M	NO	Shigella Sp.	MSSA	INTERSTITIAL HMG	INFECTION
80	1102574	21	6	0	NO	NO	NO	NO	NO	NO	NO	6H	6H	PRESENT	VAGINAL	27W	FSB	INTRAPARTUM	800	F	NO	NOGC	NOGC	WNL	PREMATURITY
81	1102780	23	12	1	NO	NO	NO	NO	NO	YES	NO	10H	9H 30M	ABSENT	LSCS	39W 5D	FSB	ANTEPARTUM	3600	F	NO	CONS	CONS	WNL	INFECTION
82	1102436	29	10	2	NO	YES	NO	NO	NO	NO	NO	8H 30M	4H	PRESENT	VAGINAL	25W 6D	FSB	INTRAPARTUM	600	M	YES	NOGC	NOGC	CHORIOAMNIONITIS	ANOMALY
83	1103168	25	12	1	NO	NO	NO	YES	NO	YES	NO	5H 30M	8H 30M	ABSENT	LSCS	37W 5D	MSB	ANTEPARTUM	2500	F	NO	NOGC	NOGC	WNL	ASPHYXIA
84	1104065	22	12	0	NO	YES	NO	NO	NO	NO	NO	10H 30M	8H	ABSENT	VAGINAL	31W 3D	FSB	ANTEPARTUM	1300	M	NO	NOGC	NOGC	FOCAL INFARCTS	PREMATURITY
85	1103899	26	10	0	NO	NO	NO	NO	NO	NO	YES	12H	8H	ABSENT	VAGINAL	35W	MSB	ANTEPARTUM	1600	F	NO	Candida Sp.	NOGC	CHORIOAMNIONITIS	INFECTION
86	1103876	28	10	0	NO	NO	NO	YES	NO	NO	NO	8H	3H	ABSENT	LSCS	38W 5W	MSB	ANTEPARTUM	4200	M	NO	NOGC	NOGC	WNL	ASPHYXIA
87	1105573	20	12	0	NO	NO	NO	YES	NO	YES	NO	12H	00H	ABSENT	LSCS	32W 4D	FSB	ANTEPARTUM	1200	M	NO	NOGC	NOGC	FOCAL INFARCTS	ASPHYXIA
88	1106017	22	12	1	NO	NO	NO	NO	NO	NO	NO	8H 30M	6H	ABSENT	VAGINAL	31W 5D	MSB	ANTEPARTUM	1100	M	NO	NOGC	NOGC	CALCIFICATION	UNKNOWN
89	1106875	24	8	0	NO	NO	NO	NO	NO	NO	NO	13H	8H	ABSENT	VAGINAL	30W 1D	FSB	ANTEPARTUM	1400	M	NO	NOGC	NOGC	WNL	PREMATURITY
90	1107385	26	8	1	NO	NO	NO	NO	NO	NO	NO	9H	6H	ABSENT	VAGINAL	29W	FSB	ANTEPARTUM	580	M	NO	NOGC	NOGC	WNL	PREMATURITY
91	1111144	22	12	1	NO	NO	NO	YES	NO	YES	NO	10H 30M	8H	ABSENT	VAGINAL	33W 3D	FSB	ANTEPARTUM	1200	F	NO	NOGC	NOGC	FOCAL INFARCTS	ASPHYXIA
92	1111602	24	10	2	YES	NO	NO	NO	NO	NO	NO	12H	9H	ABSENT	VAGINAL	34W 1D	FSB	ANTEPARTUM	1100	F	NO	NOGC	NOGC	WNL	UNKNOWN