ASSOCIATION OF UMBILICAL CORD ABNORMALITIES AND NON REASSURING FETAL HEART RATE AND ITS PERINATAL OUTCOME

By

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Dr. NEHA B.S

LIST OF ABBREVIATIONS USED

ACOG	American College of Obstetricians and Gynaecologists
AFI	Amniotic Fluid Index
APGAR	Appearance, Pulse ,Grimace, Activity and Respiration
BPP	Biophysical Profile
BPM	Beats Per Minute
CTG	Cardiotocography
FHR	Fetal Heart Rate
LSCS	Lower Segment Caesarean Section
NICE	National Institute for Health and Care Excellence
NICU	Neonatal Intensive Care Unit
NRFHR	Non Reassuring Fetal Heart Rate
IUGR	Intra Uterine Growth Restriction
pH	Power of Hydrogen
RBC	Red Blood Cell
REM	Rapid Eye Movement
UCA	Umbilical Cord Abnormalities
UCI	Umbilical Coiling Index

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ABSTRACT

INTRODUCTION:

Constant observation of fetal heart rate during labour has been used over recent 50 years for antenatal assessment of fetus in view of hypoxemia and acidemia .[1]Abnormality found in fetal heart rate during labour is one of the major indications for immediate delivery or emergent caesarean section worldwide.The mechanisms leading to fetal distress are complex and broad ,involving pregnancy complications, maternal and fetal diseases and clinical events which may compromise oxygen supply to the fetus.Among the various complications ,several umbilical cord abnormalities are related to fetal distress and adverse perinatal outcome.Such umbilical cord abnormalities include cord entanglements ,hypercoiling, true knots ,strictures and short cords .Intraoperative findings such as tight cord entanglements, uterine rupture or placental abruption may also cause fetal hypoxia leading to non reassuring fetal heart rate. Umbilical cord which acts as a lifeline between mother and fetus is an easily accessible and assessable structure and there is some evidence that adverse antenatal and perinatal events could be predicted by examination of umbilical cord abnormalities intraoperatively or postnatally and thus the perinatal outcome can be detected.

OBJECTIVES OF THE STUDY

- To study the correlation between umbilical cord abnormalities and non reassuring fetal heart rate.
- Neonatal outcome in patients with umbilical cord abnormalities.

MATERIALS AND METHODS

Source of data: A total of 146 women with a live singleton fetus in the cephalic presentation with term gestation (37^{+6} to 41^{+6} weeks) getting admitted to labour ward of RLJH hospital during the period of study.

Study design: A prospective observational study .

Study period: October 2018-June 2020**.**

METHODOLOGY

All those women whose Cardiotocography showing non reassuring fetal heart rate(According to NICE guidelines 2019 – FHR 100-109 or 160-180 with baseline changes under 5 for 30-50 minutes or above 25 for 15 to 25 minutes with variable decelerations with no concerning characteristics for 90 minutes or with any concerning characteristics in upto 50% of contractions for 30 minutes or more or less or late decelerations in over 50% of contractions for less than 30 minutes ,with no maternal or fetal clinical risk factor will be considered for the study.

Patients will be followed up till the surgery and intraoperative findings will be noted or will be followed up till vaginal delivery. APGAR scores at 1 minute and 5 minutes seen and cord blood pH (7.36-7.45 as normal) is taken into account to study perinatal outcome. The purpose of the study will be detecting the prevalence and types of umbilical cord variabilities and their correlation with non reassuring heart rate of the fetus and its perinatal outcome.Short cord of less than 30 cms and long cord of more than 70 cms is considered for the study.

In our tertiary care hospital in Kolar district of Karnataka, we decided to conduct study with a sample size of 146 patients.

Baseline demographic details along with pregnancy risk factors were taken into account .Non stress test of each patient is taken into consideration for the study.

RESULTS:

A total of 150 women were taken into the study who belonged within the inclusion criteria .Non reassuring and abnormal CTG were followed up till delivery and umbilical cord abnormality was noted. All types of cord abnormalities were documented .Further perinatal outcome was noted using Apgar scores, cord blood Ph and NICU admission.Correlation between CTG and cord abnormalities showed that 73.3% were associated with non reassuring CTG and 26.7% were associated with abnormal CTG which was similar to the study conducted by Weiner et al, which showed 93.2 % of them had non reassuring CTG and 36% of them had abnormal CTG. Among the umbilical cord abnormalities noted, 44% were cord entanglements, 25% were long cord and 15% were short cord. In present study, correlation between cord abnormalities and CTG has been established which showed that 30% of long cord showed non reassuring CTG and 80% of cord entanglement showed abnormal CTG .P value being <0.001 was found to be statistically significant on Chi square test. Thus cord abnormalities were associated with abnormal CTG. The present study shows correlation between cord abnormality and umbilical cord pH which is considered as parameter for assessment of fetal outcome.Mean values of pH with long cord is 7.21, short cord is 7.25, knot of the cord is 7.21 and cord entanglement is 7.29 which indicates acidic cord blood pH .P value on anova test is <0.05 which appears to be statistically significant. Hence cord abnormality has been associated with acidic cord blood pH which indicates adverse perinatal outcome.

CONCLUSION:

Umbilical cord abnormalities is being commonly noted during the deliveries but the significance had been ignored. This study shows that there is correlation between incidence of umbilical cord abnormalities and fetal distress identified prior to delivery which is proved statistically significant.

Further study establishes correlation between umbilical cord abnormalities and adverse perinatal outcome depicted as cord blood pH acidosis and increased NICU admission.

INTRODUCTION

ASSOCIATION OF UMBILICAL CORD ABNORMALITIES AND NONREASSURING FETAL HEART RATE AND ITS PERINATAL OUTCOME

INTRODUCTION:

Constant observation of fetal heart rate during labour has been used over recent 50 yrs for antenatal assessment of fetus in view of hypoxemia and acidemia[4]. Abnormality found in fetal heart rate during labour is one of the important indications for immediate delivery or emergent cesarean section worldwide.

The mechanisms leading to non-reassuring fetal heart rate tracings are complex and broad, involving pregnancy complications, maternal and fetal diseases, and clinical events which may compromise oxygen supply to the fetus[4].

Umbilical cord abnormalities (UCA) usually describe situations where fetal blood flow is decreased or interrupted because of altered structure or function. UCA is correlated with adverse pregnancy outcomes including birth asphyxia and emergency Caesarean birth[1]. Of the reported UCA, the nuchal cord, where there is coiling of umbilical cord at least once around the fetal neck, has increased incidence peaking at birth[2,3].

Among the various complications, several umbilical cord abnormalities have been correlated with the abnormality of fetal heart rate and adverse perinatal outcome. Such umbilical cord abnormalities include cord entanglements, hypercoiling, true knots, strictures, and short cords.

Intraoperative findings such as tight cord entanglements, uterine rupture, or placental abruption may also cause fetal hypoxia leading to non-reassuring heart rate of the fetus [6].

The cord around the umbilicus which acts as a major connection between mother and fetus is an easily accessible and assessable structure and there is some affirmation that adverse antenatal and perinatal events could be predicted by examination of umbilical cord abnormalities intraoperatively or postnatally and thus the perinatal outcome can be detected.

The relation of cord around the neonatal neck and pregnancy outcome has been studied extensively suggesting an expanded risk for induction of labour, slow progress of labour, foetal distress, shoulder dystocia, meconium, low APGAR scores, and a higher rate of instrumental and caesarean deliveries.Non availability of source about cord entanglement, sites of entanglement, and other cord abnormalities induces the need for this study[5].

Gross cord abnormalities make the fetus liable to stasis induced vascular ectasia and thrombosis thus leading to vascular obstruction and adverse neonatal outcomes, including IUGR and stillbirth[7]. Careful interpretation of FHR patterns help to detect fetal asphyxia. Further supplementary examination of the umbilical cord for abnormalities will assess their correlation with non-reassuring fetal heart rate and thus perinatal outcome could be detected.

AIMS & OBJECTIVES

OBJECTIVES OF THE STUDY:

- To study the correlation between umbilical cord abnormalities and non-reassuring fetal heart rate.
- Neonatal outcome in patients with umbilical cord abnormalities.

REVIEW OF LITERATURE

REVIEW OF LITERATURE:

Fetal compromise,acts as a major contributor to perinatal morbidity which is of great concern for both obstetrician and a pediatrician. The umbilical cord is the lifeline of the fetus as it supplies water, nutrients and oxygen to the growing parasite. Helically arranged blood vessels which are three in number are present along the length of the cord.

Fatal compromise of umbilical circulation is suspected in at least 20% of stillbirths at autopsy [4]. Any kind of force that compresses umbilical cords may lead to decreased blood flow in umbilical vessels and further fetal hypoxia or circulatory compromise. Mechanical cord compression or ‘‘cord accident’’ may be caused by cord entanglements (nuchal/body cords) and cord prolapse; or it should arise from an abnormal configuration of the cord such as true knots, increased coiling/twisting, abnormally long cords, abnormal cord insertions, or strictures [7].

Many intrapartum hurdles are more commonly correlated with umbilical cord abnormalities, including stillbirth, intrauterine growth restriction (IUGR), non-reassuring fetal heart tracing (NRFHT), low APGAR scores, and meconium staining, and certainly depend on duration and degree of occlusion. Besides, late neonatal obstacles have also been associated with obvious cord abnormalities, including pulmonary hypertension and neurologic impairment. However, these same cord abnormalities can also be found in unremarkable live births, and as such remains controversial [7].

Cardiotocography (CTG) is one form of fetal assessment that simultaneously records fetal heart rate (FHR), fetal movements and uterine contraction patterns to analyse hypoxia. CTG is demanding in high-risk cases where variable decelerations are depicted to have great influence on perinatal outcome, in the mode of delivery and NICU admission[1.]

Fetal heart rate traces are categorized according to NICE guidelines in 2019.

The study carried out to evaluate perinatal outcome in newborns with hypercoiled and hypocoiled cords concluded that hypercoiled and hypocoiled were associated with low birth weight, low APGAR score, meconium stained liquor and intrauterine growth restriction[17].

The study conducted on cases with variable length of cords showed an increased incidence of cord complications, increased incidence of operative interference, intrapartum complications, increased abnormalities in fetal heart rate and more chances of birth asphyxia[13].

A Study on singleton pregnancies showed that 1-minute APGAR scores <7 and umbilical artery pH <7.1 were significantly more usual in umbilical cord entanglement groups than with other cord groups.

Georgiadis et al. noted a possible correlation of a cord of short length leading to abruption of the placenta[18.]

Vasa et al. studied about the correlation between nuchal cord and fetal acid-base equilibrium and showed that 23.5% of pregnancies were disturbed by the presence of

nuchal cord at delivery and incidence increases with age of gestation . It was revealed that the danger of the nuchal cord is greater if NRFHT is noted during labor. There was also higher umbilical artery acidosis as noted within the study[19]

Joshi et al. studied about the incidence of nuchal cord and its impact on labour and perinatal outcome and showed that 15%–30% of pregnant women had the cord entanglement of the foetus and only some of them had tight or multiple nuchal cords. They demonstrated that tightness of loop or multiple loops adversely affected perinatal outcomes such as intrapartum FHR deceleration, meconium staining of liquor, decreased Apgar score at 1 min and higher incidence of operative delivery[20].

Njoku et al. studied about cord length and abnormalities in singleton pregnancies and showed that abruptio placentae and breech presentation were greater among fetuses with a short umbilical cord than normal and cord round neck was greater among long umbilical cord than the normal cord length[21].

Ramaprabha established a study on the correlation between non-reassuring fetal heart rate and adverse perinatal outcome and finalised that umbilical cord blood pH values immediately following child birth were associated with abnormal FHR patterns and there was a significant association ($P < 0.01$) between low CTG scores and acidosis[22].

Gurusamy U et al. presented a study on abnormalities of the umbilical cord and its association with placental histology and perinatal outcome and depicted that gross UCAs, usually when multiple ,were associated with clinically significant placental findings and adverse perinatal outcome. Hence the study strengthens that all placentas

with gross UCAs should be subjected for examination with complete umbilical cord and coiling index with patterns of coiling must be a part of routine examination[23].

Balkawade et al. studies showed the significance of understanding the length of the cord. Cases which had short and long cords were shown as abnormal cord length and these had a increased incidence of cord complications, higher incidence of operative interference, intrapartum complications, comparatively more variations in the fetal heart rate and more chances of birth asphyxia[24].

Algreisi F et al. showed that most of the umbilical cords are within normal length range and if the umbilical cord is abnormally long or short then it might be a reasonable explanation of abnormal outcome in absence of an obvious cause of the unexpected outcome[25].

UMBILICAL CORD

The umbilical cord or funis forms the connection between the growing fetus and the placenta through which the fetal blood flows to and from the placenta. It is extending from the umbilicus of the fetus to the fetal surface of the placenta.

DEVELOPMENT OF UMBILICAL CORD

As the blastocyst develops, embryo changes itself into a three-layered disc called an embryonic disc. These 3 layers consists of endoderm, ectoderm and mesoderm.

Blastocyst are spherical cysts lined by flattened trophoblastic cells.

The umbilical cord comes from and contains remnants of the allantois and yolk sac. It is created by 5^{th} week of development, replacing yolk sac which acts as the source of nutrients for the embryo.

By day 13, blastocystic cavity contains an embryo covered by a loose meshwork of extraembryonic mesoderm. The embryo constitutes two cavities, the amniotic cavity and primary yolk sac. The embryonic disc is formed and contains two epithelial layers –ectoderm which is in line with the amniotic epithelium and endoderm which partly surrounds the primary yolk sac cavity.

By day 18, endoderm surrounds yolk sac in a complete manner in which exocoelom would have cavitated within the extraembryonic mesoderm. Part of this mesoderm, the chorionic mesoderm, lines the internal region of the trophoblastic shell whereas the rest conceals the two embryonic cavities. These two parts of extraembryonic mesoderm are connected in only one place, basal to the amniotic cavity. This mesenchymal bridge, the connecting stalk, will finally form the umbilical cord.

This extraembryonic mesoderm gives rise to connecting stalk. This connecting stalk is the only association between embryo and placenta. Caudal end of the embryonic disc and the ventral aspect of the fetus are being attached by the growing embryo.

By now the blood vessels have developed within the embryo. These set of blood vessels are in connection through arteries and veins passing through the connecting stalk. Initially, there are double vessels carrying arterial blood and two veins but later right vein disappears.

Contents of connecting stalk include:

- Vitello-intestinal duct and remnants of yolk sac
- Mesoderm –which later gets transformed into Wharton's jelly which takes care of blood vessels in the cord
- Blood vessels that pass from embryo to placenta
- A short portion of extraembryonic coelom.

This tube of amnion, and the contents within it, make the cord.This cord gradually increases in length and hence permits free movement of the embryo within the amniotic cavity. At the time of birth of the child, the umbilical cord is about half a metre in length and 2 cms in diameter.

STRUCTURES OF UMBILICAL CORD:

- **COVERING EPITHELIUM**: It's lined by one layer of amniotic epithelium but depicts stratification like that of fetal epidermis at term.

- **WHARTON'S JELLY**: It contains elongated cells in an exceedingly gelatinous fluid formed by mucoid degeneration of the extraembryonic mesodermal cells. It is rich in mucopolysaccharides and possesses a protective function to the umbilical vessels.

- **BLOOD VESSELS**: 2 vessels carrying oxygenated blood and 2 vessels carrying non-oxygenated blood are present . The arteries are derived from the internal iliac arteries of the fetus and carry the venous blood towards the placenta from the embryo. Of the 2 umbilical veins, the right one obseletes by the 4th month,

leaving behind one vein which continues to carry oxygenated blood from the placenta to the fetal veins.

- **REMNANT OF THE UMBILICAL VESICLE (YOLK SAC) AND ITS VITELLINE DUCT**: Remnant of the yolk sac has been found as a small body close to the association of the cord or rarely, the initial part of the duct persists as Meckel's diverticulum.

- **ALLANTOIS:** A blind tubular structure may be sometimes present near the fetal end which is in line with the inside of the fetus with its urachus and bladder.

CHARACTERISTICS:

- The umbilical cord is about 50-60 cm in length with a normal variation of 40–70 cm.
- Its diameter is of average 1.5 cm with a variability of 1–2.5 cm.
- Its thickness is not same but presents nodes or swelling at few regions. Local collection of Wharton jelly can be a cause for these swellings .
- Long cord may even form a cord entanglement (20–30%).
- It shows a spiral turn from left to right from as early as 12th week since spiral turn was taken by the vessels—vein around the arteries.
- The umbilical arteries don't contain an internal elastic lamina but have got well formed muscular coat. These help in effective closure of the arteries as spasmodic reflex occurs soon after the birth of the fetus.

- The umbilical vein provides the fetus with oxygenated, nutrient-rich blood from the placenta. Conversely, the fetal heart pushes low oxygen-containing blood, nutrient-depleted blood all along the umbilical arteries back to the placenta.
- The two main layers of the umbilical cord are outer layer containing smooth muscle cells arranged circularly and an inner layer which clearly shows almost irregularly and loosely arranged cells have been embedded in abundant ground substance staining metachrome.
- The smooth muscle cells of the layer are more often poorly differentiated and consist of few tiny myofilaments and hence won't contribute actively to the process of post-natal closure.
- The lining of the umbilical cord is an effective good provenance of mesenchymal and epithelial stem cells.
- Approximately 35 ml/min of blood flows through the umbilical cord at around 20 weeks of gestation, and 240 ml/min at 40 weeks of gestation.
- The proximal part of an umbilical cord refers to the segment closest to the fetus, whereas the distal part refers to the segment closest to the placenta.

ATTACHMENT OF UMBILICAL CORD

In the beginning of gestational period, there is attachment of cord to the front surface of the embryo nearer to the caudal extremity but as yolk sac atrophies and by the end of 4^{th} month the point of attachment is moved permanently to the middle of the abdomen .

- Unlike fetal attachment, the placental attachment is not consistent.
- Insertion of the umbilical cord eccentrically has been defined as the association of the surface of the fetus of the placenta in the middle and to the margin of placenta.
- Central or marginal attachment of placenta can be present
- Velamentous insertion of placenta is defined as attachment to chorion leave which is at a distance from margin of placenta.

FUNCTIONS OF UMBILICAL CORD

- The umbilical cord is considered both the physical and emotional attachment between mother and fetus.
- It allows movement of oxygen and nutrients from the maternal circulation into fetal circulation while simultaneously removing waste products from fetal circulation to be eliminated maternally.

ABNORMALITIES OF LENGTH:

Cord length is influenced positively by both amnionic fluid volume and fetal mobility

SHORT CORD:

Umbilical cord less than 30cm is defined as short cord.

It may cause: fetal-growth restriction, congenital malformations, failure of external version, prevent the descent of the presenting part especially during labor, separation of a normally situated placenta, favour malpresentation, Fetal distress in labor and prolonged labour.

LONG CORD:

Umbilical cord more than 70 cm is defined as long cord. It may cause cord prolapse, cord present around the neck or the body, sufficient compression on the cord vessels so as to produce fetal distress or rarely death.

False knots are the result of accumulation of Wharton's jelly or due to varices higher occurrence of cord around neck.

UMBILICAL CORD DIAMETER:

- Lean cords are associated with IUGR
- Large diameter cords are associated with macrosomia

UMBILICAL CORD COILING

- Cord vessels spiral through the cord.
- UCI (Umbilical Coiling Index) - is the number of complete coils divided by the cord length in cm.
- A normal antepartum index derived sonographically is 0.4, and this contrasts with a normal value of 0.2 derived postpartum by actual measurement.
- They grouped the UCI as follows:
  - < 10th percentile — hypocoiled;
  - 10th – 90th percentile — normocoiled;

- ❖ >90th percentile — hypercoiled
- ➢ Antenatal UCI has a lower sensitivity than when measured postpartum.
- ➢ Hypocoiling is linked with fetal demise and hypercoiling is associated with IUGR &intrapartum hypoxia.
- ➢ Abnormal UCI has been related to trisomies & single umbilical artery.

CORD ATTACHMENT ABNORMALITIES

- ➢ Usually the cord is inserted at or near the center of the fetal surface of placenta.
- ➢ Various cord insertion variations are:
- ➢ **Furcate insertion:** Umbilical vessels separate from the cord substance before their insertion into the placenta which is very rare.
- ➢ **Marginal Insertion**: cord insertion at the placental margin. (BATTLEDORE PLACENTA). If associated with low implantation of the placenta, there's chance of cord compression in vaginal delivery resulting in fetal anoxia may be even death. Its more frequently associated with multifetal pregnancy, especially those conceived using assisted reproductive technology, and they may be associated with weight discordance.
- ➢ **Velamentous Insertion:** Cord is attached to the membranes**.** Umbilical vessels separate in the membranes at a distance from the placental margin. More frequently seen with twins.
- ➢ **Vasa Previa:** This is associated with velamentous insertion when some of the vessels of the fetus in the membranes cross the region of os of the cervix below the presenting fetal part.There is interposition of vessel between the cervix and presenting fetal part. Hence, they are vulnerable to compression and also to

laceration or avulsion with rapid fetal deterioration. It's also increased in pregnancies conceived by artificial reproduction.

ABNORMALITIES OF VESSELS NUMBER:

- **Single umbilical artery:** The foremost common aberration is that of a single umbilical artery, with a cited incidence of 0.63 percent in liveborn neonates, 1.92 percent with perinatal deaths, and 3% in twins. Atrophy of the previously existing umbilical artery might happen. Cause might be due to inability of development of one artery. Wastage of artery can also occur in later months .Fetuses with major malformation frequently have a single umbilical artery. And when seen in an anomalous fetus, the aneuploidy risk is greatly increased, and amniocentesis is usually recommended. Cardiac, genital and urinary anomalies are more common. A single artery has also been related to fetal-growth restriction. It is more common in twins and in babies born for mother with diabetes, epilepsy, oligohydramnios, hydramnios, pre-eclampsia and antepartum hemorrhage. There is frequent correlation with congenital anamoly of the fetus (20–25%). Renal and genital anomalies, Trisomy 18 are common. There is increased chance of abortion, fetal aneuploidy, prematurity, and increased perinatal mortality.

- **Fused umbilical artery** with a shared lumen has been a common malformation. It arises from failure of the two arteries to split during embryological development. The common lumen may extend through the whole cord, but if partial, is usually found near the placental insertion site. Associated congenital

malformations include aneuploidies, tracheooesophageal fistula, renal agenesis, imperforate anus, vertebral defects.

REMNANTS AND CYSTS

Remnants: A number of structures are housed in the umbilical cord during fetal development, and their remnants may be seen when the mature cord is viewed transversely. Remnants of vitelline duct, allantoic duct, and embryonic vessels were seen. There was very rare correlation with congenital anamolies or near birth complications.

Cysts: Cysts occasionally are found along the course of the cord. They are designated according to their origin. True cysts are epithelium-lined remnants of the allantoic or vitelline ducts and tend to be located closer to the fetal insertion site. In contrast, the more common pseudocysts form from local degeneration of Wharton jelly and occur anywhere along the cord. Both have a similar sonographic appearance.

Single umbilical cord cysts detected in the early pregnancy to resolve completely, however, multiple cysts may lead to miscarriage or aneuploidy. Structural and chromosomal anomalies were correlated with cysts after first trimester of pregnancy.

KNOTS:

True knots: These are caused by active fetal movements and are seen in approximately 1 percent of births. Monoamnionic twins are commonly associated with true knots.Four- to ten fold high risk of still birth has been associated with true knots in singleton pregnancy . FHR abnormalities are common during labor.

False knots -Result from kinking of the vessels to accommodate length of cord and are of no clinical significance and appear as knobs protruding from the cord surface. These are focal redundancies of a vessel or Wharton jelly.

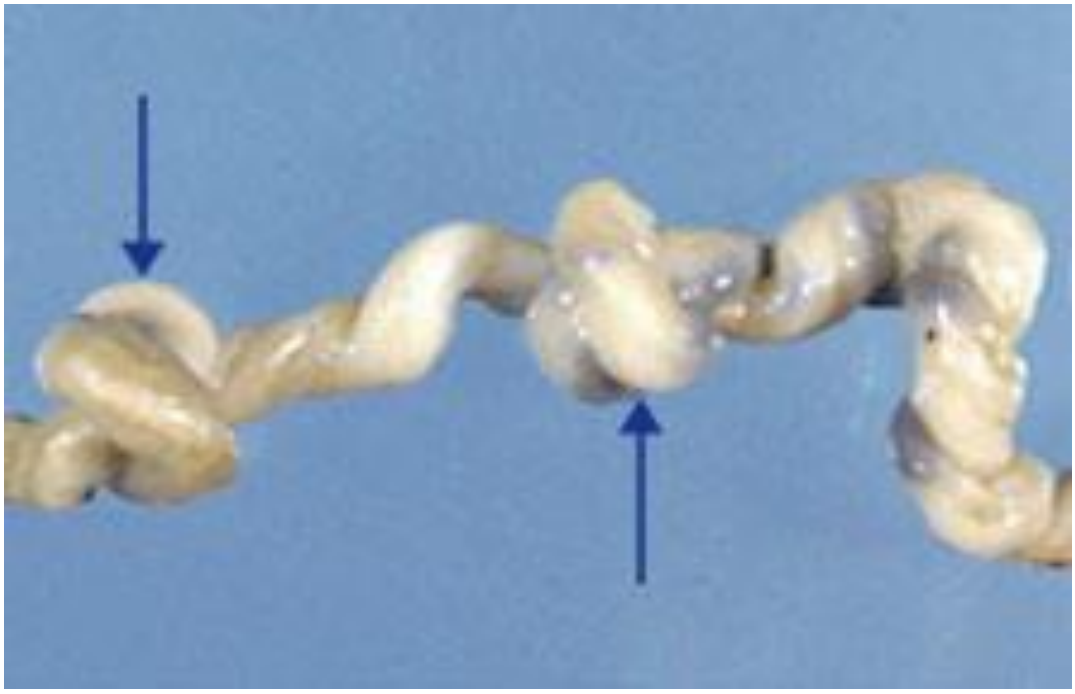


Figure 1. True knots depicted in the picture

CORD STRICTURE: Cord stricture is a focal narrowing of its diameter that usually develops near the fetal cord insertion. Characteristic pathological features of strictures are absence of Wharton jelly and stenosis. In most of the cases it will be stillborn. Even less commonly stricture of the cord is caused by an amnionic band.

CORD LOOPS: Cord loops are frequently encountered and are caused by coiling around various fetal parts during movement. Longer cords are frequently found when compared to others. A cord around the neck also called as nuchal cord has been frequently noted abnormality. The cord is frequently coiled around the fetus. During labor these loops can result in fetal heart rate decelerations that persist during a

contraction. Studies have shown 20 percent of fetuses with a nuchal cord are associated with decreased umbilical cord pH indicating acidosis with moderate to severe variable fetal heart rate deceleration. Its been considered that single is protective than multiple cord entanglements around the fetal neck.

Type A: nuchal cord in a sliding manner (less dangerous)

Type B: Twisting and locking manner of nuchal cord (very dangerous).

At the time of birth, if it is loose enough ,cord will slip over the head of the fetus .If the cord is wrapped multiple times it may take a while. At this time, if the cord is too tight, it has to be cut before the baby is born. This necessitates rapid birth, since it is no longer getting nutrients from the mother via placenta.

CORD HEMATOMAS:

Cord hematomas are uncommon and have been associated with abnormal cord length, umbilical vessel aneurysm, trauma, entanglement, umbilical vessel venipuncture, and funisitis. They can follow varix rupture, which is usually of the umbilical vein. They are recognized sonographically as hypoechoic masses that lack blood flow. Umbilical cord vessel thromboses are in utero events. Approximately 70 percent are venous, 20 percent are venous and arterial, and 10 percent are arterial thromboses. Compared with venous thromboses, those in the artery have higher fetal death rates and are associated with fetal-growth restriction, fetal acidosis, and stillbirths. Complications may include rupture or thrombosis, compression of the umbilical artery, and fetal cardiac failure due to increased preload. They may be visualized during sonography as a cystic dilatation of the umbilical vein.

The rare umbilical artery aneurysm is caused by congenital thinning of the vessel wall with diminished support from Wharton jelly. Indeed, most form at or near the cord's placental insertion, where support is absent. These are associated with single umbilical artery, trisomy 18, amniotic fluid volume abnormalities, fetal-growth restriction, and stillbirth. At least theoretically, these aneurysms could cause fetal compromise and death by compression of the umbilical vein. These aneurysms may appear sonographically as a cyst with a hyperechoic rim. Within the aneurysm, Doppler flow studies demonstrate either low velocity or turbulent nonpulsatile flow.

FETAL HEART RATE MONITORING

INTRAPARTUM FETAL MONITORING (IFM)

It implies watching of fetal behaviour during labor. Goal of IFM is to detect hypoxia in labor and to initiate management depending upon the severity of hypoxia. Continuous electronic fetal monitoring (EFM) was introduced into obstetrical practice in the late 1960s. It provided accurate information and information was of value in diagnosing fetal distress. Fetal death can be prevented . When first introduced, electronic monitoring of fetal heart rate was used primarily in complicated pregnancies, but gradually became used in most pregnancies. Electronic monitoring of fetus can be done by direct or indirect methods.

Fetus has been attached with bipolar electrode for direct fetal heart measurement. Vaginal body fluids create a the circuit and permits measurement differences of voltage in between electrodes. Reference electrode has been attached to the thigh of the mother so that electrical interference can be vanished. The electrical fetal cardiac

signals are increased and put in a cardiac monitor for heart rate calculation. The trustworthy parameter is peak R – wave.

EXTERNAL FETAL HEART RATE MONITORING

External monitoring of fetal heart rate can be used to prevent membrane rupture. Internal monitoring is more accurate when compared to external. Heart rate of the fetus can be identified by colour doppler in blood vessels.

Fetal heart action can be best detected by keeping the transducer on the abdomen of the mother. Changes in the ultrasound signals will be made before fetal heart rate is printed onto monitor paper. Reflected ultrasound signals from moving fetal heart valves are analysed through a microprocessor that compares incoming signals with the most recent previous signal. This process is helped by the regularity of fetal heart rate.

The fetal heart rates are added for their mean values and is considered as increase of five beats per minute during a 10-minutes segment. The definition of base value is defined as minimum 2 minutes window or prior 10-min window. Normal FHR baseline is 110–160 bpm and tachycardia refers to FHR > 160 bpm and bradycardia is FHR < 110 bpm. Variability is visually quantified as the amplitude of peak-to-trough in bpm .It is considered absent if amplitude range is undetectable, minimal if amplitude range detectable but ≤ 5 bpm or fewer, moderate (normal) if amplitude ranges between 6–25 bpm and marked if amplitude is > 25 bpm.

Acceleration is defined as sudden rise in the FHR. At and more than 32 weeks , an acceleration is called as increase in 15 bpm or further, period of 15 sec or further but within 2 min from onset to return. Prolonged acceleration lasts ≥ 2 min, but < 10 min and if an acceleration lasts 10 min then it is change in reference line.

Deceleration is transient fall in heart rate of fetus by 15 beats per minute or more and lasting > 15 seconds.

Early deceleration is a slow fall and rebound of the FHR which is correlated with a uterine contraction. Peak of the contraction correlates with nadir of deceleration.

Late deceleration is visually apparent similar gradual fall and coming back of the heart rate of fetus correlated with uterine contraction.

Variable deceleration is sudden fall in FHR .A sudden fall in FHR is explained as from the beginning to the FHR nadir of < 30 sec. The fall in heart rate of fetus is ≥ 15 bpm, lasting ≥ 15 seconds, and < 2 min in duration.

Prolonged deceleration is fall in fetal heart rate under the baseline .If a deceleration lasts ≥ 10 min, it is a baseline change.

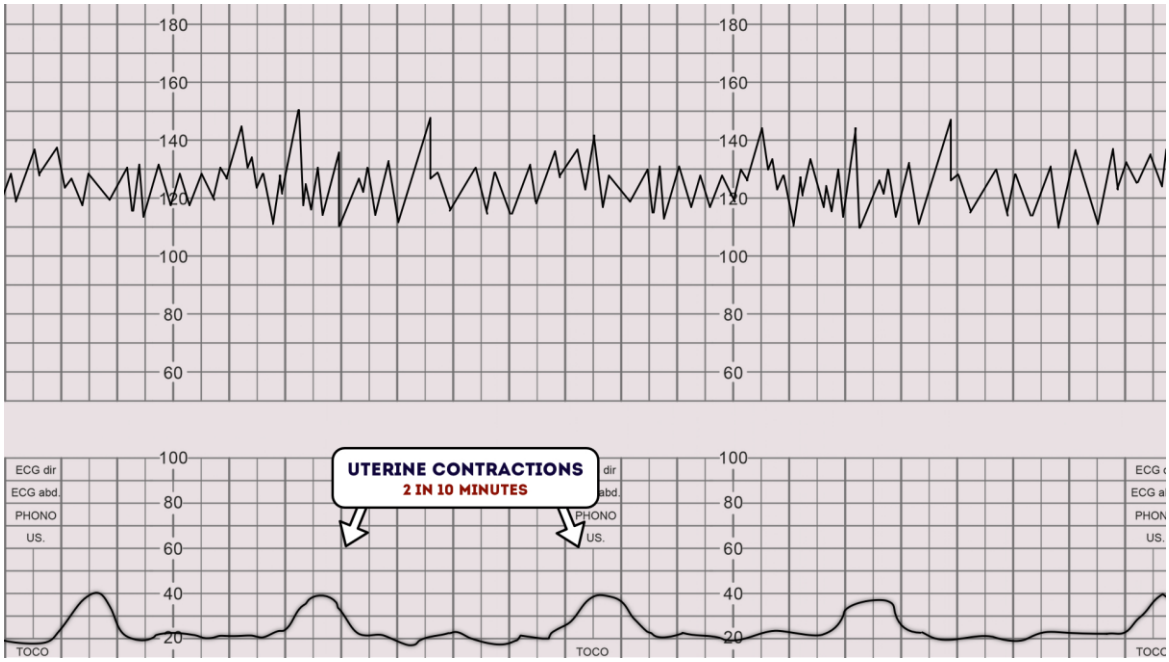


FIGURE 2. NORMAL CARDIOTOCOGRAPHY

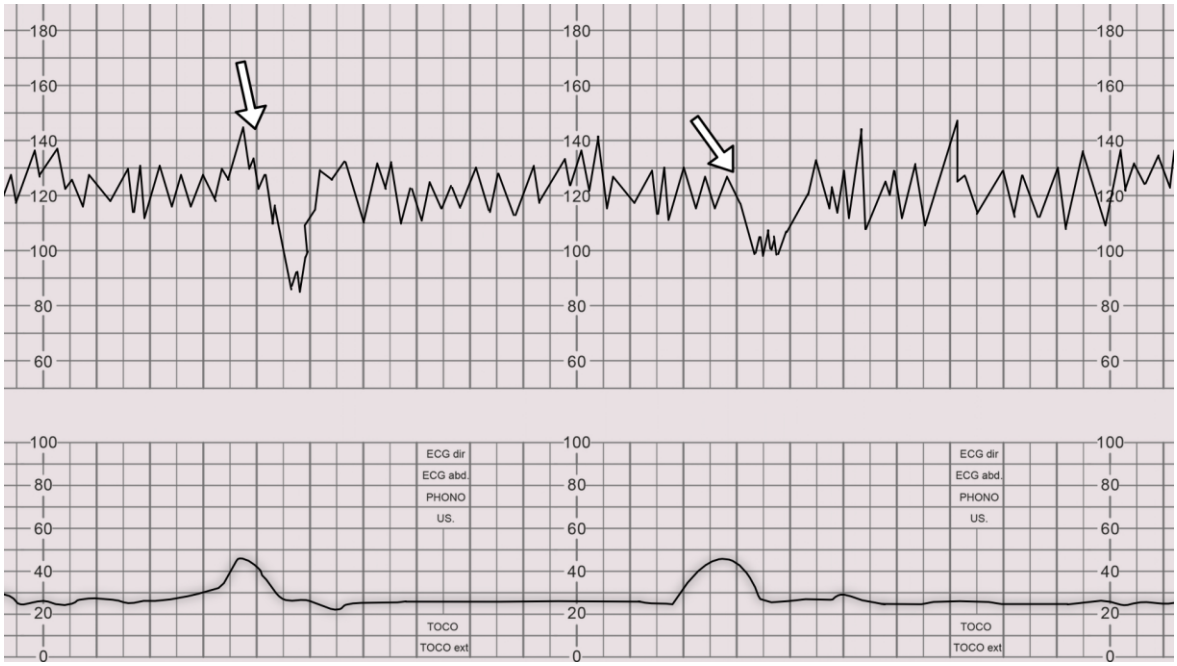


FIGURE 3. NON REASSURING CTG SHOWING VARIABLE DECELERATIONS

NICE GUIDELINES 2019

Feature	*Baseline (bpm)*	*Variability (bpm)*	*Decelerations*	*Accelerations*
Reassuring	110-160	≥ 5	None	Present
Non-reassuring	100-109 161-180	< 5 for 40-90 minutes	Typical variable decelerations with over 50% of contractions, occurring for over 90 minutes Single prolonged deceleration for up to 3 minutes	The absence of accelerations with otherwise normal trace is of uncertain significance
Abnormal	< 100 > 180 Sinusoidal pattern ≥ 10 minutes	< 5 for 90 minutes	Either atypical variable decelerations with over 50% of contractions or late decelerations, both for over 30 minutes Single prolonged deceleration for more than 3 minutes	

CLASSIFICATION ACCORDING TO CATEGORY OF CTG

Category	**FHR tracing**
Category I	Baseline rate: 110–160 beats per minute
	Baseline variability: moderate
	Late or variable decelerations: absent
	Early decelerations: present or absent
	Accelerations: present or absent
Category II	Includes all tracings not categorized as Category I or III
Category III	Absent baseline FHR variability and any of the following • Recurrent late decelerations • Recurrent variable decelerations • Bradycardia
	Sinusoidal pattern

INTRAPARTUM FETAL ASSESSMENT:

Fetal heart rate, movement, breathing, and amniotic fluid production are helpful in assessing fetal wellbeing.

FETAL MOVEMENTS:

By 7 weeks' gestation activity of fetus starts and becomes prominent later. After 20 weeks and before 30 weeks, movements of the body will be organized, and the fetus starts to show rest-activity cycles. Fetal movement maturation continues until approximately 36 weeks, when behavioural states are established in most normal fetuses.

Nijhuis and colleagues described four fetal behavioural states:

State 1F is a quiescent state that is small nap with a narrow oscillatory bandwidth of heart rate of the fetus .

2F state involves entire body movements, continuous eye movements, and variability in the heart rate of fetus.

3F state involves progressive moving in the eye with no movements of the body and no variability in heart rate of the fetus. The existence of this state is disputed.

4F state involves robust movements of the body along with movements of eye and heart rate variability.

1F and 2F are the common places for fetus to stay.

At 38 weeks, 75 percent will be in duration of these two states. These behavioural states particularly 1F and 2F, which correspond to quiet sleep and active sleep have been helpful in assessing fetal behaviour. Tests for monitoring fetal heart rate were employed if sonographic pictures were unusual. Pregnancy outcomes were not much

affected by decreased fetal movement until and unless maternal comorbidities were associated.

FETAL BREATHING:

Movements of chest wall were 2 in number including gasps or deep breaths occurring once in one to four in sixty seconds and secondly are irregular bursts of breathing happening till two hundred forty cycles in one minute. These fast respiratory movements were associated with rapid eye movements–REM. Among them were decreased sugar levels , smoking, invasive first trimester procedures , expected preterm labor, age of gestation and labor and is usual for respiration to cease. Thus it is now one among the contents of biophysical profile.

CONTRACTION STRESS TESTING:

Increase in the amniotic fluid pressure associated with uterine contractions results in increase in the myometrial pressure leading to collapse of vessels passing through uterine muscle. Further causes decreased blood flow to the intervillous space. Brief periods of impaired oxygen exchange result, and if uteroplacental pathology is present, these elicit late fetal heart rate decelerations. Contractions also may produce a pattern of variable decelerations as a result of cord compression. Oxytocin challenge test was later called the contraction stress test. Intravenous oxytocin was used to stimulate contractions, and heart beat of fetus response was recorded.

The criterion for a positive test result, that is, an abnormal result, was uniform repetitive late fetal heart rate decelerations. To perform the test, heart rate of the fetus and contractions of the uterus are recorded simultaneously with an external monitor.

If at least three spontaneous contractions of 40 seconds or longer are present in 10 minutes, no uterine stimulation is necessary (American College of Obstetricians and Gynecologists, 2012a). Contractions are induced with either oxytocin or nipple stimulation if less than 3 in 10 minutes present . For oxytocin use, a dilute intravenous infusion is started at 0.5 milliunits/minute and doubled every 20 minutes until a satisfactory contraction pattern is established. The results are interpreted as negative if no late or significant variable decelerations and positive if late decelerations following 50% or more of contractions .

NONSTRESS TESTS:

This test involved the use of Doppler-detected fetal heart rate acceleration coincident with fetal movements perceived by the mother. Around 1970s, the nonstress test was considered the first way for testing fetal health.

Simplistically, the nonstress test is done for fetal condition, and it is different when compared to other tests , and is done for uteroplacental function. Currently, nonstress testing is the most widely used primary testing way and has also been incorporated into the biophysical profile.

The definition currently recommended by the American College of Obstetricians and Gynecologists and the American Academy of Pediatrics (2012) is two or more accelerations that peak at fifteen beats per minute or more above baseline, each lasting 15 seconds or more, and all occurring within 20 minutes of beginning the test . Abnormal non stress tests include pattern consisting of a fetal heart rate baseline that oscillates less than 5 bpm and presumably indicated absent acceleration and beat-to-

beat variability. Non stress tests which are nonreactive for 90 minutes were almost invariably associated with significant perinatal pathology. Tests with baseline oscillation of less than 5 bpm, absent accelerations, and late decelerations with spontaneous uterine contractions were also considered abnormal. 7 days interval between tests appears to have been recommended with nonstress testing. According to the American College of Obstetricians and Gynecologists (2012a), more frequent testing is advocated by for women with postterm pregnancy, multifetal gestation, type 1 diabetes mellitus, fetal-growth restriction, or gestational hypertension. In these circumstances, performing twice-weekly tests, with additional testing for maternal or fetal deterioration regardless of the time elapsed since the last test. The American College of Obstetricians and Gynecologists (2012a) has concluded that variable decelerations, if nonrepetitive and brief less than 30 seconds have no need for obstetrical intervention. In contrast, repetitive variable decelerations at least three in 20 minutes even if mild, have been associated with higher danger of caesarean delivery for fetal distress. Decelerations lasting 1 minute or longer have been reported to have an even worst prognosis.

BIOPHYSICAL PROFILE:

Manning and colleagues (1980) showed usage of 5 fetal biophysical variables as a more accurate means of assessing fetal health than a single element. Typically, these tests require 30 to 60 minutes of examiner time. There are the five fetal biophysical components assessed:

(1) heart rate acceleration, (2) breathing,(3) movements, (4) tone, and (5) amniotic fluid volume. Normal variables were assigned a score of 2 each, and abnormal variables were given a score of 0. Thus, the highest score possible for a normal fetus

is 10. Biophysical score of 0 was almost invariably associated with significant fetal acidosis, whereas a normal score of 8 or 10 was associated with normal ph. An equivocal test result a score of 6 was a poor predictor of abnormal outcome. As the abnormal score decreased from 2 or 4 down to a very abnormal score of zero, this was a progressively more accurate predictor of abnormal fetal outcome. Because the biophysical profile in labor is intensive and requires a person trained in sonography, a vibroacoustic nonstress test was performed twice weekly and combined with amnionic fluid index determination for which < 5 cm was considered abnormal. This abbreviated biophysical profile required approximately 10 minutes to perform, and it was finalised as a better antepartum surveillance method because there were no unexpected fetal deaths.

CORD BLOOD PH:

Sample of blood in the cord is to be taken from the placental end of cord. About 5 mL of blood (2 mL oxalated and 3 mL clotted) should be collected for the following tests:

Clotted blood - ABO and Rh grouping, direct Coombs' test and serum bilirubin. Oxalated blood - Hemoglobin estimation and blood smear for presence of immature RBC.

Cord blood gas analysis has become widely performed to objectively determine the fetal metabolic condition when umbilical circulation stops[26].Multiple studies showed that this analysis when combined with other neonatal factors, can help identify infants at risk for neonatal encephalopathy, which is vital for early initiation of

neuroprotective therapeutic strategies[27].Sample has to be collected from umbilical vein as it has large diameter.But pH analysis in blood of the cord is more reliable for better neonatal outcomes[28].

The American College of Obstetricians and Gynecologists and the American Academy of Pediatrics now recommend cord blood ph analysis to be performed in all high-risk deliveries in which there is a suspicion of a defect in the fetal metabolism. The mean cord arterial pH is 7.24 to 7.27, and the mean cord venous pH 7.32 to 7.34[30].Preterm newborns had increased pH, and observations noted a gradual reduction with increasing gestational age. A cord blood pH less than 7, when combined with other abnormal clinical findings, strongly correlates with adverse neonatal outcomes.

MATERIALS AND METHODS

MATERIALS AND METHODS

- A total of 146 pregnant women having live singleton fetus in the cephalic presentation with term gestation (37 to 42 weeks) getting admitted to labour ward of RLJH hospital during the period of study.
- Study design: A prospective observational study.
- Study period: October 2018-June 2020.

Inclusion criteria:

- Age between 18 and 35 years
- Period of gestation 37-42weeks
- Single live fetus in cephalic presentation

Exclusion Criteria:

- Malpresentation
- Previous 2 caesarean section
- Abnormal progress in labour
- Multiple pregnancies
- Preterm labour (<37 weeks of gestation)
- Fetal or neonatal malformations
- IUGR

Study population and sample size:

The sample size is calculated based on the difference between 2 groups with umbilical cord abnormalities that is between emergency caesarean section group and vaginal

delivery group –Association between umbilical cord abnormalities and development of fetal distress leading to emergency caesarean deliveries done in the year 2015 .Observed variance estimate is of 40% difference, 80%power, 5% alpha and with 95% confidence interval, This being prospective observational study ,total of 146 women are taken in to the study after taking consent form, irrespective of mode of delivery.

Sample size 146.

Formula

$$n = \frac{2s_p^2\left[Z_{1-\alpha/2} + Z_{1-\beta}\right]^2}{\mu_d^2}$$

$$s_p^2 = \frac{s_1^2 + s_2^2}{2}$$

Where,

s_1^2 : Standard deviation in the first group

s_2^2 : Standard deviation in the second group

μ_d^2 : Mean difference between the samples

α : Significance level

1- β : Power

METHODOLOGY

All pregnant women whose Cardiotocography is showing non-reassuring fetal heart rate (According to NICE guidelines 2019 – FHR 100-109 or 160-180 bpm with baseline variability less than 5bpm for 30-50 minutes or more than 25bpm for 15 to 25 minutes with variable decelerations with no concerning characteristics for 90 minutes or with any concerning characteristics in upto 50% of contractions for 30 minutes or more or less or late decelerations in over 50% of contractions for less than 30 min, with no maternal or fetal clinical risk factor are considered for the study.

Patients are followed up till the surgery and intraoperative findings are noted or are followed up till vaginal delivery and umbilical cord length, morphology and abnormalities are noted .

Short cord is considered below 30 cms and long cord is considered more than 70 cms in our study. Apgar score at 1 minute and 5 minutes is documented and cord blood pH is taken into account and perinatal and neonatal outcome assessed.

Cord blood collection: Double clamping of an umbilical cord is done as early as possible after delivery. Blood is drawn into a pre-heparinized syringe from this isolated segment. Blood is collected from umbilical artery and vein (from placental side of the clamped umbilical cord) for blood gas analysis and is processed in an arterial blood gas analyser for cord blood ph. Cord blood pH is documented.

STATISTICAL ANALYSIS:

Categorical data was represented in the form of number and percentage. Association between variables were assessed with Chi Square Test.

Quantitative data was represented as Mean & Sd. Comparison of variables has been done with Unpaired t test.

ANOVA was applied to comparison of more than two groups.

A P value of <0.05 was considered statistically significant.

Analysis of data was done with IBM SPSS Version 25 for windows.

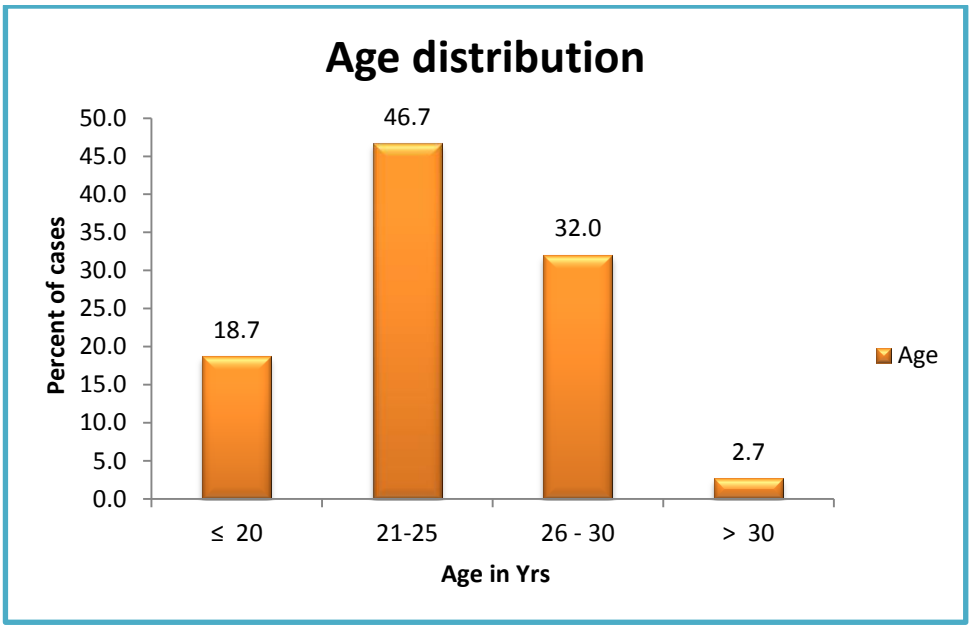
RESULTS

RESULTS :

1.DISTRIBUTION OF UMBILICAL CORD ABNORMALITIES IN VARIOUS AGE GROUPS

Age (years)	No of Cases	Percent
≤ 20	28	18.7
21-25	70	46.7
26 - 30	48	32.0
> 30	4	2.7
Total	150	100.0

Figure 4:Graphical representation of age distribution among cord abnormalities



Incidence of cord abnormalities is seen more in 21-25 yrs of age representing 46% of the total study group.

2. MEAN DISTRIBUTION OF AGE IN THE STUDY

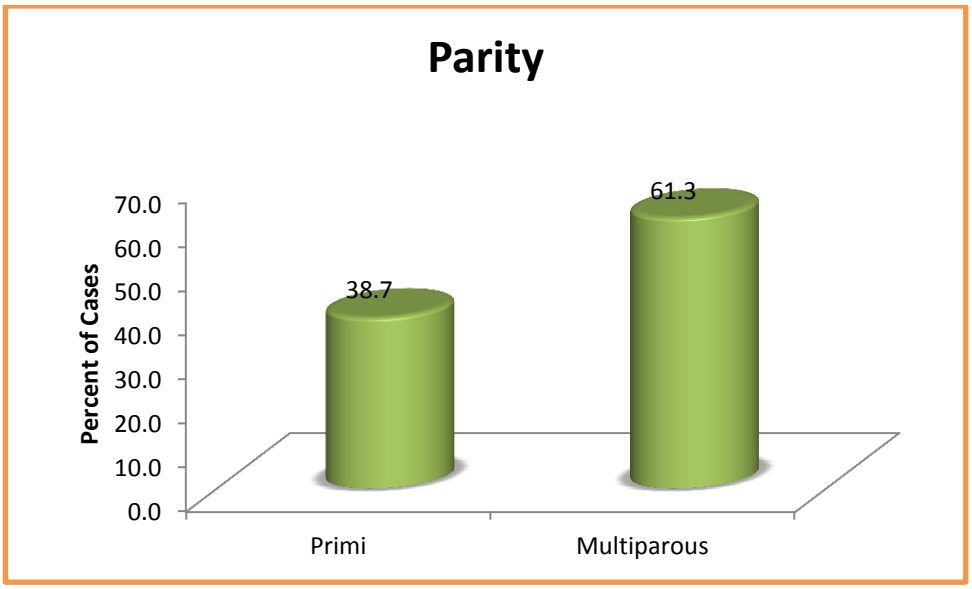
Age	
Mean	24.49
Std. Deviation	3.55
Minimum	18.00
Maximum	35.00

Mean age group included in the study is 24 years of age

3. DISTRIBUTION OF UMBILICAL CORD ABNOMALITIES ACCORDING TO PARITY.

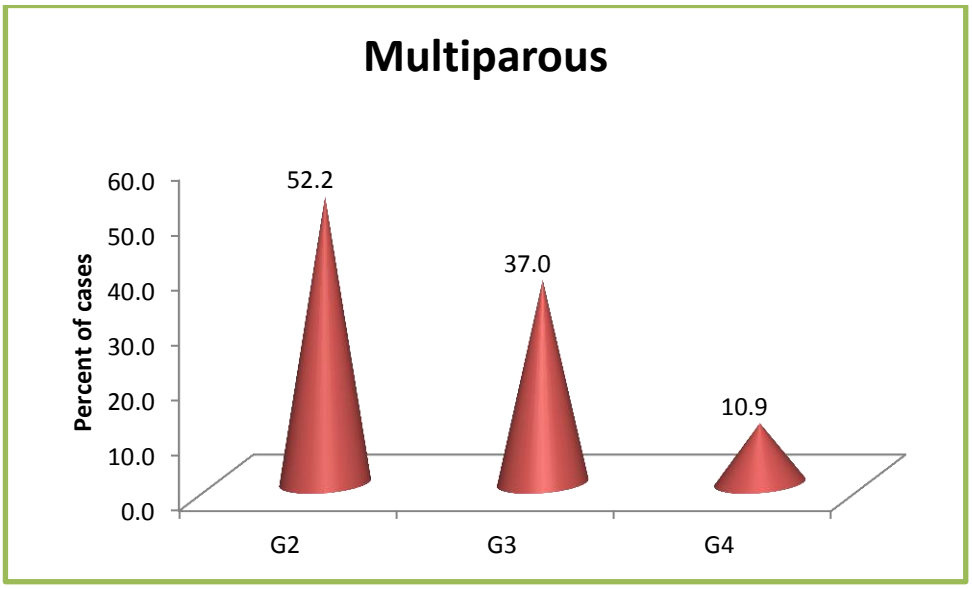
Parity	No of Cases	Percent
Primi	58	38.7
Multiparous	92	61.3
Total	150	100.0
Multiparous		
G2	48	52.2
G3	34	37.0
G4	10	10.9
Total	92	100.0

Figure 5: Graphical representation of parity among different groups of cord abnormalities



Incidence of cord abnormalities is seen more in multiparous women when compared to primipara where multiparaous women constitute 61% of the study group .

Figure 6: Graphical distribution among multiparous groups

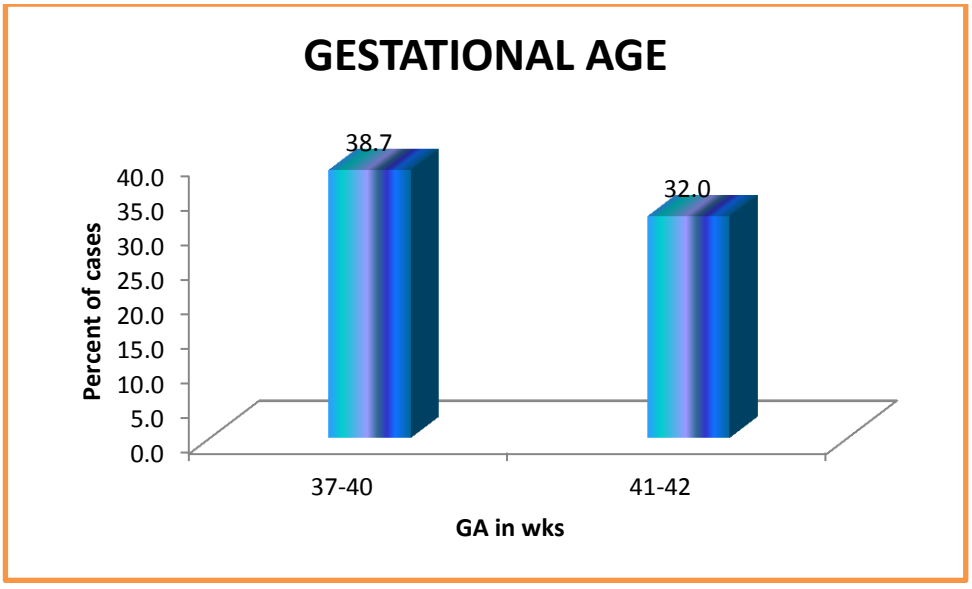


Among the multiparous women second gravida have higher incidence constituting 52 % of the total study group

4. DISTRIBUTION OF UMBILICAL CORD ABNORMALITIES ACCORDING TO GESTATIONAL AGE

GESTATIONAL AGE (IN WKS)	No of Cases	Percent
37-40	58	38.7
41-42	48	32.0
Total	150	100.0

Figure 7: Graphical distribution of gestational age among cord abnormalities

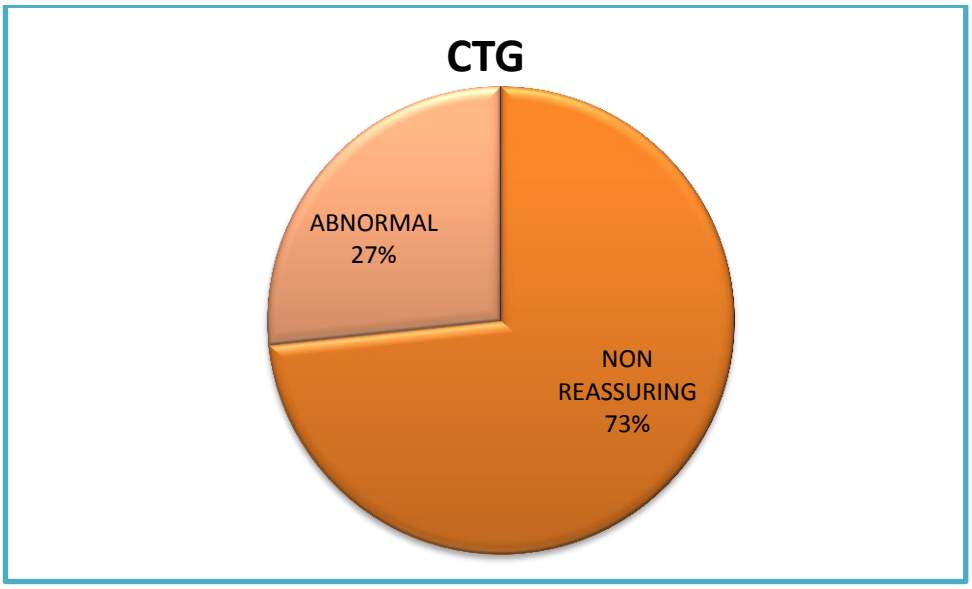


Incidence of cord abnormalities was not correlating with gestational age as there was near equal distribution of gestational ages among the study groups.

5. CORRELATION BETWEEN UMBILICAL CORD ABNORMALITIES AND CTG

CTG	No of Cases	Percent
NON REASSURING CTG	110	73.3
ABNORMAL CTG	40	26.7
Total	150	100.0

Figure 8: Graphical distribution of CTG among cord abnormalities

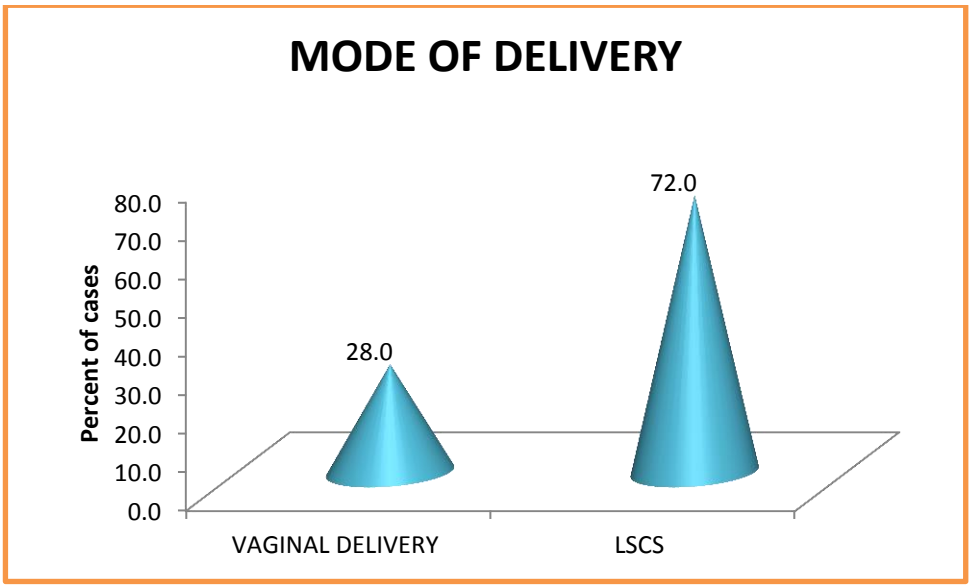


Incidence of cord abnormalities was associated with non reassuring fetal heart rate where 73 % presented with non reassuring fetal heart rate CTG and 27% presented with abnormal fetal heart rate CTG.

6. CORRELATION OF UMBILICAL CORD ABNORMALITIES WITH MODE OF DELIVERY:

MODE OF DELIVERY	No of Cases	Percent
VAGINAL DELIVERY	42	28.0
LSCS	108	72.0
Total	150	100.0

Figure 9: Graphical distribution of mode of delivery among cord abnormalities

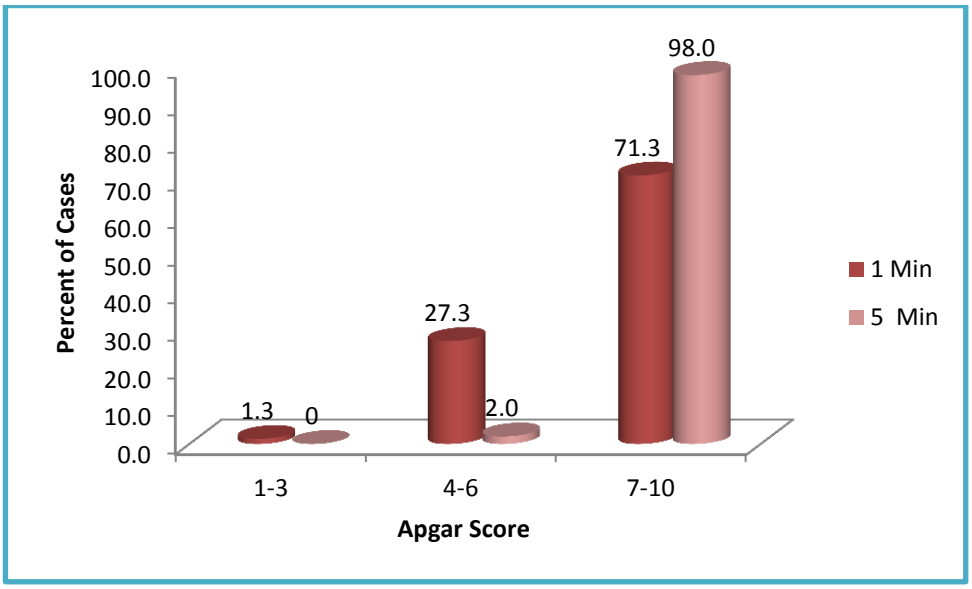


Incidence of cord abnormalities was associated with increased incidence of caesarean section constituting 72% of total study group and only 28% of the study population underwent vaginal delivery.

7. CORRELATION BETWEEN UMBILICAL CORD ABNORMALITIES AND APGAR SCORE :

APGAR SCORE	1 MIN		5 MIN	
	No of Cases	Percent	No of Cases	Percent
1-3	2	1.3	0	0
4-6	41	27.3	3	2.0
7-10	107	71.3	147	98.0
Total	150	100.0	150	100.0

Figure 10: Graphical distribution of APGAR score among cord abnormalities



Incidence of cord abnormalities has no much significance on apgar score as 98% of the study group depicted normal apgar score at 5^{th} minute .

8. MEAN DISTRIBUTION OF APGAR SCORE AT 1ST MINUTE AND 5TH MINUTE

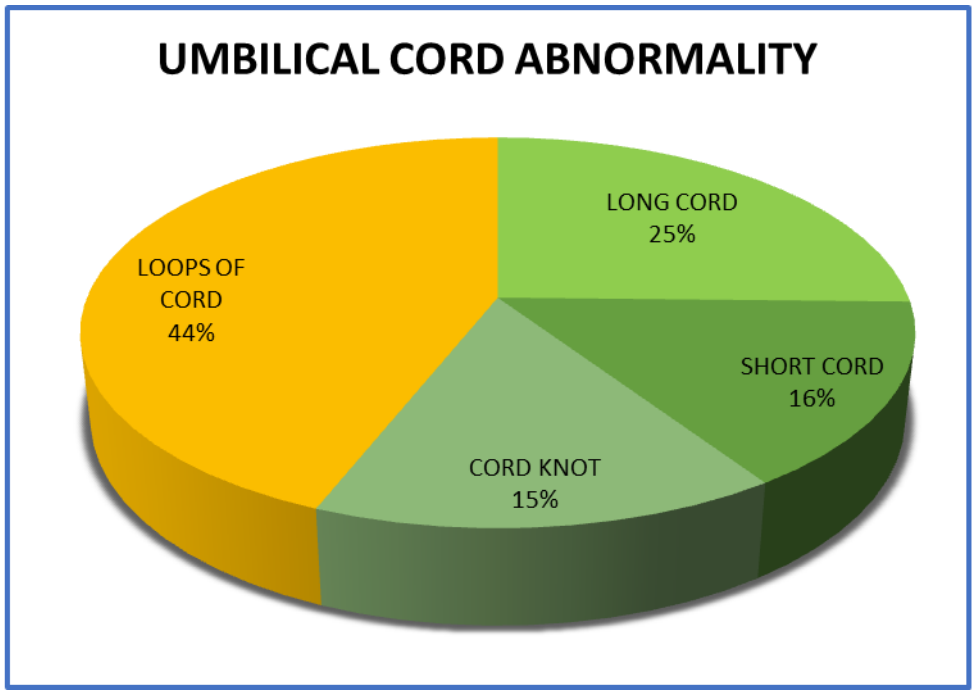
Measures	APGAR SCORE	
	1 MIN	5 MIN
Mean	6.61	8.66
Std. Deviation	0.74	0.68
Minimum	3.00	5.00
Maximum	7.00	9.00

Mean distribution of APGAR score showed 6 at 1st minute and 8 at 5 th minute.

9. DISTRIBUTION OF VARIOUS UMBILICAL CORD ABNOMALITIES.

UMBILICAL CORD ABNORMALITY	No of Cases	Percent
LONG CORD	38	25.3
SHORT CORD	23	15.3
CORD KNOT	23	15.3
LOOPS OF CORD	66	44.0
Total	150	100.0

Figure 11: Graphical distribution of umbilical cord abnormalities

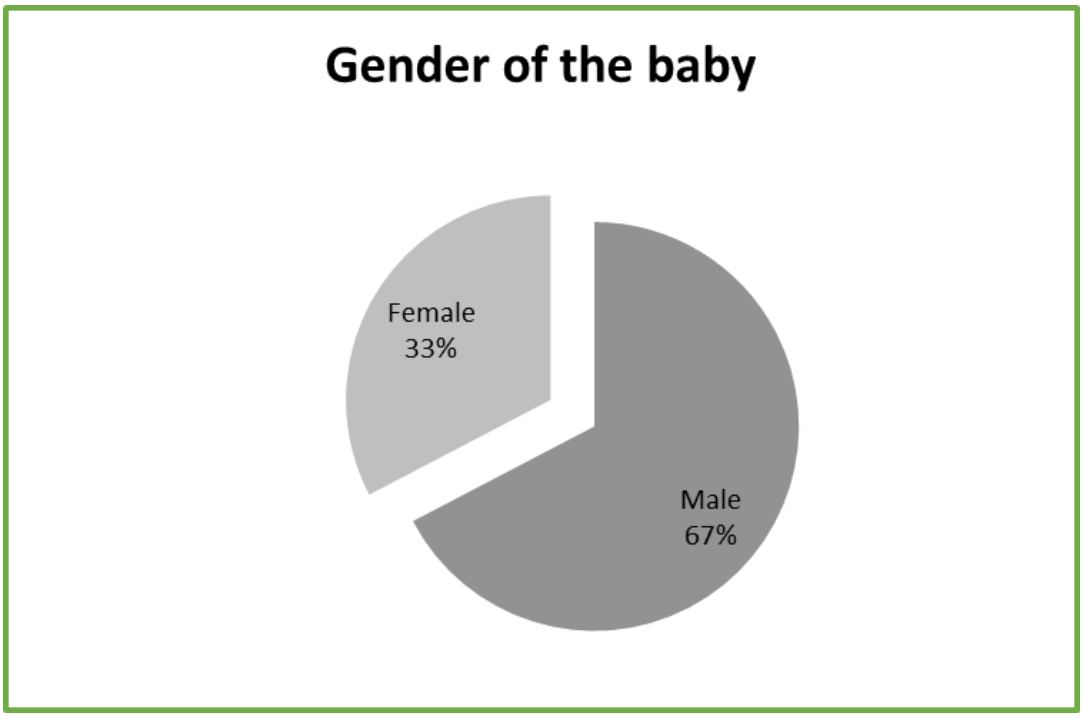


Incidence of loop of cord around the neck was the most common abnormality found in the study constituting 44%of the study group ,cord knot occupying 15%, short cord occupying 16% and long cord constituting 25%.

10. DISTRIBUTION OF UMBILICAL CORD ABNORMALITIES AND SEX OF THE BABY

Gender of the baby	No of Cases	Percent
Male	101	67.3
Female	49	32.7
Total	150	100.0

Figure 12: Graphical distribution of sex of the baby among cord abnormalities



Incidence of Cord abnormalities had no significance on sex of the baby.

11. DISTRIBUTION OF UMBILICAL CORD ABNORMALITIES AND THE WEIGHT OF THE BABY

BABY WEIGHT (in kgs)	No of Cases	Percent
< 2.5	42	28.0
≥ 2.5	108	72.0
Total	150	100.0

Figure 13: Graphical distribution of weight of the baby among cord abnormalities

Incidence of cord abnormalities was not associated with birth weight of the baby.

12. MEAN DISTRIBUTION OF WEIGHT OF BABY.

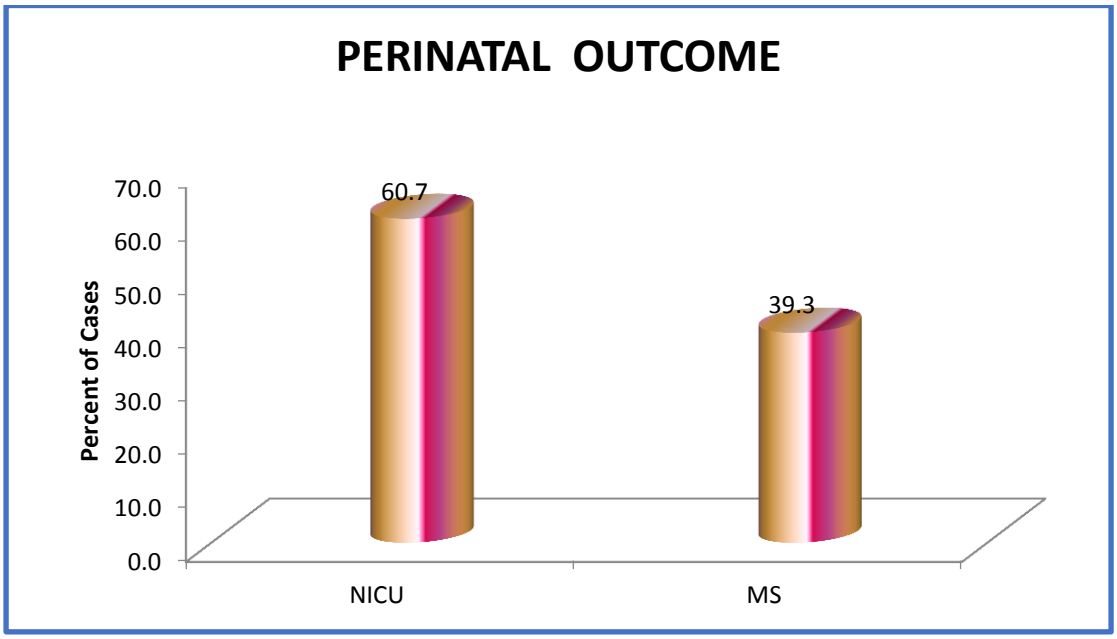
BABY WEIGHT	
Mean	2.83
Std. Deviation	0.44
Minimum	1.72
Maximum	4.24

Mean baby weight is 2.83 kgs in the study.

13. DISTRIBUTION OF UMBILICAL CORD ABNORMALITIES AND PERINATAL OUTCOME

PERINATAL OUTCOME	No of Cases	Percent
NICU	91	60.7
MS	59	39.3
Total	150	100.0

Figure 14: Graphical distribution of perinatal outcome among cord abnormalities

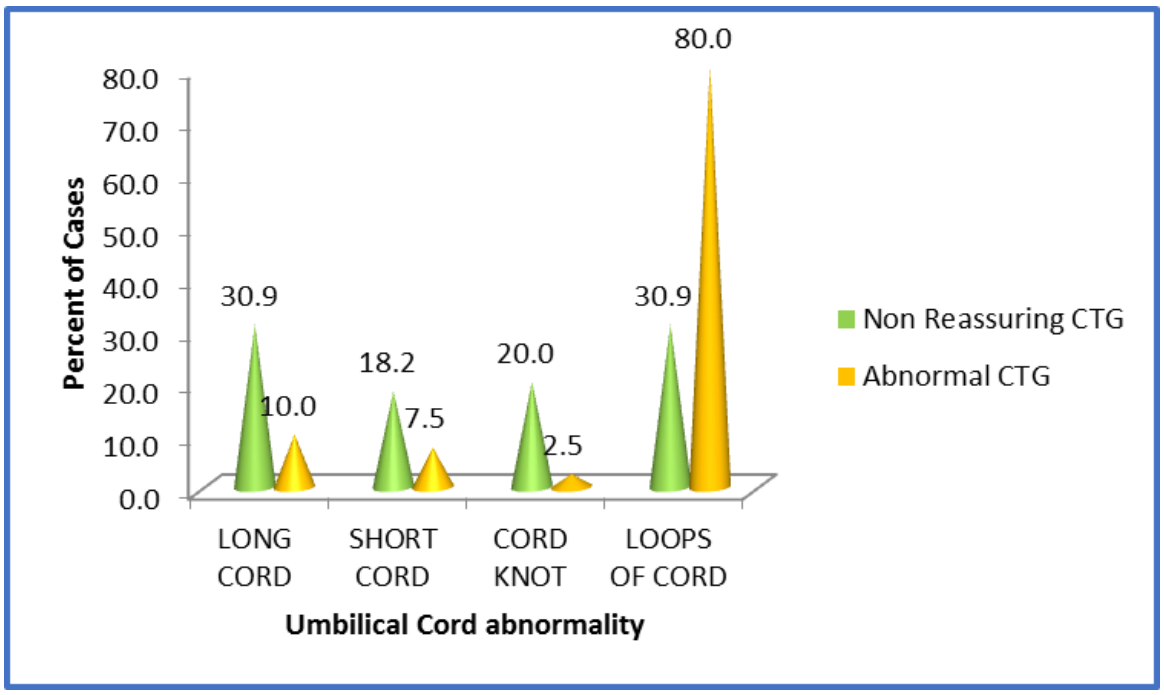


Incidence of cord abnormalities was associated with increased NICU admission of the baby as 60% of the study group showed NICU admission.

14. CORRELATION BETWEEN UMBILICAL CORD ABNORMALITIES AND CTG

UMBILICAL CORD ABNORMALITY	CTG				Chi Square test	
	NON REASSURING CTG (n=110)		ABNORMAL CTG (n=40)		P Value	Sig
	N	%	N	%		
LONG CORD	34	30.9	4	10.0	P<0.001	Highly Sig
SHORT CORD	20	18.2	3	7.5		
CORD KNOT	22	20.0	1	2.5		
LOOPS OF CORD	34	30.9	32	80.0		

Figure 15 : Graphical distribution of correlation between umbilical cord abnormalities and CTG

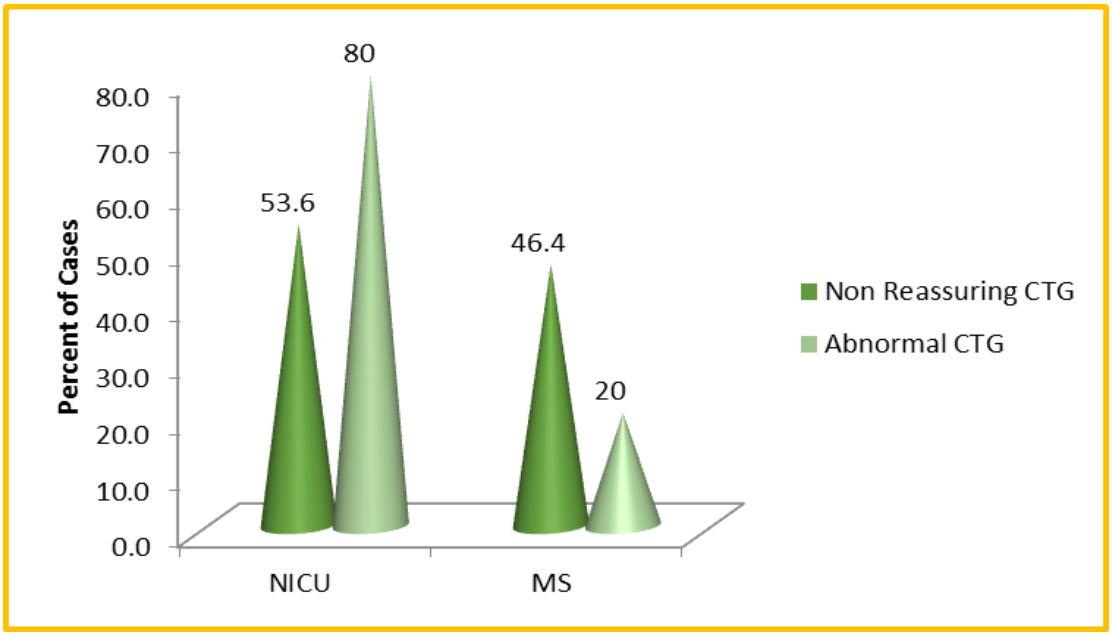


Incidence of cord abnormalities was associated with non reassuring and abnormal CTG where 80% of cord entanglement showed non reassuring fetal heart rate which was found to be statistically significant with P value of less than 0.001.

15.CORRELATION BETWEEN CTG AND PERINATAL OUTCOME

PERINATAL OUTCOME	CTG				Chi Square test	
	NON REASSURING CTG (n=110)		ABNORMAL CTG (n=40)		P Value	Sig
	N	%	N	%		
NICU	59	53.6	32	80	P<0.005	Highly Sig
MS	51	46.4	8	20		

Figure 16: Graphical distribution of correlation between CTG and perinatal outcome



Non reassuring CTG and abnormal CTG was associated with adverse perinatal outcome depicting 80% cases having NICU admission which was found to be statistically significant with P value of less than 0.005.

16. CORRELATION BETWEEN UMBILICAL CORD ABNORMALITIES AND CORD BLOOD PH

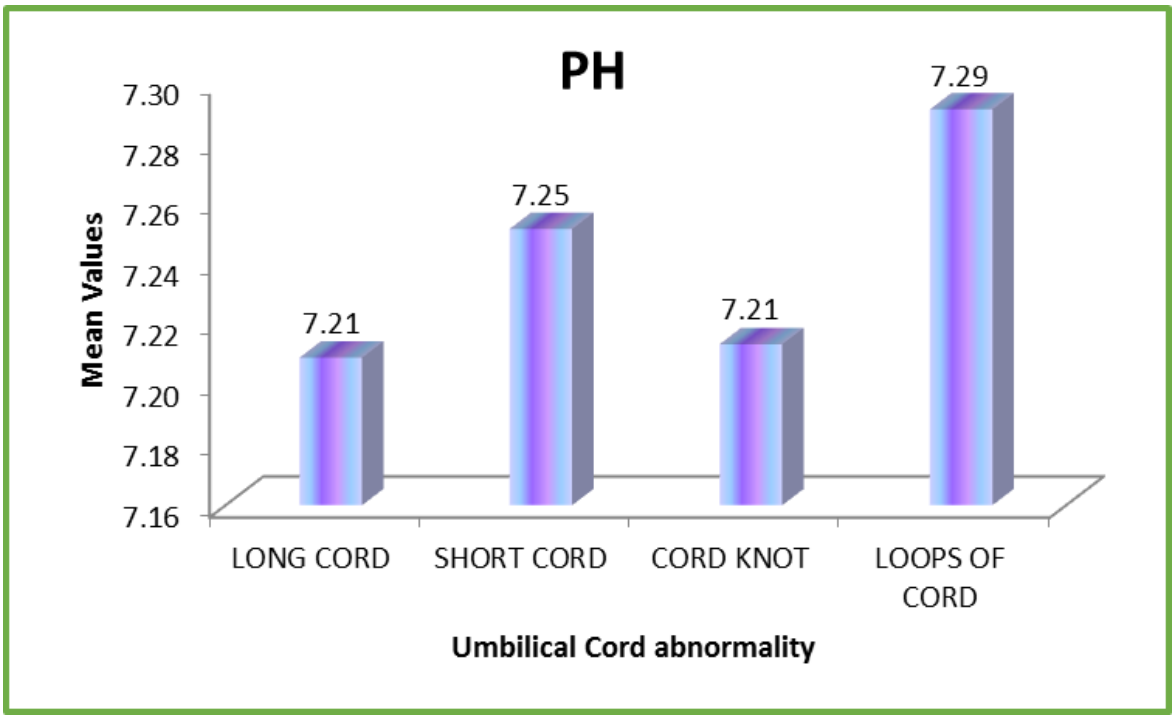
UMBILICAL CORD ABNORMALITY	PH		ANOVA	
	Mean	Std. Deviation	P Value	Sig
LONG CORD	7.21	0.12	P<0.05	Sig
SHORT CORD	7.25	0.17		
CORD KNOT	7.21	0.16		
LOOPS OF CORD	7.29	0.12		

17. MEAN DISTRIBUTION OF UMBILICAL CORD BLOOD PH

CORD BLOOD PH	
Mean	7.28
Std. Deviation	0.13
Minimum	7.04
Maximum	7.70

Mean cord blood ph is 7.28 in the study.

Figure 17: Graphical distribution of correlation between umbilical cord abnormalities and cord blood pH



Incidence of cord abnormalities has correlation with ph of cord blood and adverse perinatal outcome as cord blood ph among all the abnormalities is depicting acidosis and has been proved to be statistically significant with P value of less than 0.05.

DISCUSSION

DISCUSSION :

This study is a correlation between fetal heart rate which is non reassuring or abnormal with umbilical cord abnormalities and associated perinatal outcome .

Study is prospective observational and is conducted in the Department of Obstetrics and Gynaecology from October 2018 to June 2020 at Sri Devraj Urs Medical College, Tamaka, Kolar.

A total of 150 pregnant ladies were involved.Non reassuring and abnormal CTG were followed up till delivery and umbilical cord abnormality was noted. All types of cord abnormalities were documented .Further perinatal outcome was assessed with the help of Apgar scores, cord blood pH and NICU admission.

The women included in the study were among the age group of 18 to 35 years . The mean age of distribution is 24.4 years. Majority of women were in 21 to 25 years. There was no statistical significance in age distribution.Joshi et al also showed similar incidence regarding age with no statistical significance.[20]

Distribution of gravidity shows majority of women were multiparous constituting 61.3% among which 52.2% were gravida 2 , 37% were gravida 3 and 10% were gravida 4 .There was no statistical significance between incidence of cord abnormalities and gravidity.Distribution of gestational age showed no much variability where 38.7% were between 37 to 40 weeks gestation and 32% were between 41 to 42 weeks of gestation with no statistical significance.

Correlation between CTG and cord abnormalities showed that 73.3 % were associated with non reassuring CTG and 26.7% were associated with abnormal CTG

which was similar to the study conducted by Weiner et al, which showed 93.2 % of them had non reassuring CTG and 36% of them had abnormal CTG .

In the present study 72 % underwent emergency LSCS whereas 28% had vaginal delivery. In Joshi et al, similar study percentage of caesarean delivery was noted although difference was not statistically significant[20]. Weiner et al, reported that the rate of LSCS was greater with short cords[4]. Balkawade et al, showed that short-cord was associated with higher incidence of LSCS rates which was statistically significant[13].

In present study 71 % had APGAR of 7 at first minute and 98% had APGAR of 9 at fifth minute. Algriesi et al, showed that there was increased incidence of APGAR less than 7 at first minute with short cords when compared to long cords[25].Linde et al, showed there was risk of low APGAR at 5 minutes with cord entanglements and short cords[67].Vasa et al, study showed correlation between cord abnormality and apgar at 5th minute which showed P value of 0.01 which was statistically significant[19].

Among the umbilical cord abnormalities noted, 44% were cord entanglement, 25% were long cord and 15% were short cord. Linde et al, also showed similar incidence with 20% of cord entanglements and 9% of short cords[67].

In the present study 67% were males and 32% were female babies, 72% had birth weight more than 2.5 kg which showed no significance.

Perinatal outcome was also assessed with NICU admission of the baby and in the present study 60% of babies had NICU admission .Balkawade et al, proved the

increased incidence of NICU admission in babies associated with long and short cords and low APGAR scores[24]

CORRELATION OF PARAMETERS WITH VARIOUS STUDIES

PARAMETERS	**LINDE ET AL STUDY(2018)**	**VASA ET AL STUDY (2018)**	**WEINER ET AL STUDY(2019)**	**PRESENT STUDY**
MATERNAL AGE	<24 years of age (22%)	20-34 years of age(69%)	-	21-25years of age(78%)
PARITY	GRAVIDA 2 (22%)	Multiparity (73%)	Multiparity(64%)	GRAVIDA 3(84%)
GESTATIONAL AGE	37-41 weeks (23%)	>37weeks (89%)	-	38-40weeks (93%)
MODE OF DELIVERY	Caesarean(21%)	Vaginal (71%)	Caesarean(40%)	Caesarean(72%)
CTG ABNORMALITY	-	NRFHR(50%)	NRFHR (93%)	NRFHR(73%)
APGAR SCORES	Low 5 min APGAR(23%)	Normal(95%)	Low at 5 minutes(40%)	NORMAL(71%)
CORD PH	-	Acidosis (76%)	Acidosis(68%)	Acidosis(68%)
NICU ADMISSION	More(22%)	-	More(40%)	More (65%)

In present study , correlation between cord abnormalities and CTG has been established which showed that 30% of long cord showed non reassuring CTG and 80% of cord entanglement showed abnormal CTG .P value being <0.001 was found to statistically significant on Chi square test. Thus cord abnormalities were associated with abnormal CTG.Vasa et al, study showed significance between cord abnormality and non reassuring fetal heart rate with significant p value of <0.001.Weiner et al, showed similar significance where cord entanglements ,especially multiple loops were associated with non reassuring fetal heart rate and adverse perinatal outcome[4].

In present study ,strong correlation was found between CTG and NICU admission .53 % of non reassuring CTG was associated with NICU admission and 80% of abnormal CTG were admitted to NICU .P value was found to be <0.005 which was statistically highly significant.Cord abnormalities associated with abnormal CTG had increased admission to NICU which depicted adverse perinatal results. Ramaprabha et al, conducted a study which showed similar results where abnormal CTG was associated with adverse perinatal outcomes[22]. Vasa et al, also showed correlation between NICU admission and CTG abnormality with p value of 0.03 which was statistically significant[19].

The present study shows correlation between cord abnormality and umbilical cord pH.Long cord is associated with mean pH of 7.21,short cord with pH of 7.25 , knot of the cord with pH of 7.21 and cord entanglement with pH of 7.29 which indicates acidic cord blood pH .P value on anova test is <0.05 which appears to be statistically significant. Hence cord abnormality has been associated with acidic cord blood pH which indicates adverse perinatal outcome. Vasa et al, showed similar significance in their study where cord abnormalities were associated with cord blood pH acidosis indictaing adverse perinatal outcome[19].

LIMITATION:

The limitation of the study is mainly the sample size and lack of more studies to support the evidence.

RECOMMENDATION:

This study helps in early detection of abnormal fetal heart rate and aims towards better perinatal outcome.

CONCLUSION

CONCLUSION:

Umbilical cord abnormalities is being commonly noted during the deliveries but the significance had been ignored. This study shows that there is correlation between incidence of umbilical cord abnormalities and non reassuring fetal heart rate identified before child birth which is proved statistically significant.

Correlation between umbilical cord abnormalities and abnormal perinatal outcome can be assessed through cord blood pH acidosis and increased incidence of NICU admission in this study.

Prediction of adverse perinatal outcome associated with cord abnormalities can be detected early with the help of non reassuring CTG and this acts as prognostic tool in preventing the same.

SUMMARY

SUMMARY:

- This is a prospective observational study conducted in the Department of Obstetrics and Gynecology from October 2018 to June 2020 at Sri Devraj Urs Medical College, Tamaka, Kolar.
- The women included in the study were among 18 to 35 years. The mean age of distribution is 24.4 years. Majority of women were in the age group of 21 to 25 years.
- Distribution of gravidity shows majority of incidence among third and fourth gravida compared to primigravida.
- Distribution of gestational age showed no much variability with similar distribution in term gestation.
- Correlation between CTG and cord abnormalities were associated with majority of non reassuring CTG compared with abnormal CTG.
- In the present study, incidence of emergency LSCS was more compared to vaginal delivery.In present study, APGAR score was found to be within normal limits.
- Among the umbilical cord abnormalities noted, highest incidence of cord entanglement present and least incidence of cord knot.
- Significant correlation has been established between cord abnormalities and CTG which showed that cord abnormalities have increased incidence of non reassuring and abnormal CTG.
- In this study, association of non reassuring CTG with NICU admission has been found to be statistically significant.

- The present study also shows that cord abnormalities have increased incidence of cord blood pH acidosis depicting adverse perinatal outcome which was proved to be statistically significant .

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ANNEXURES

ANNEXURES

PROFORMA

- **Name :**
- **I.P.No:**
- **Age:**
- **Occupation:**
- **Address:**
- **Husband's Occupation:**
- **Socio-economic Status:**
- **History of presenting illness:**
- **Menstrual history:**
- **Obstetric history: ML-**
- **Past Medical history**
- **Family History:**
- **Personal History:**

Sleep:

Appetite:

Diet:

Bowel & Bladder:

- **G.P.E:**

Build: **Nourishment:**

- **Pallor:** **Icterus:** **Cyanosis:** **Clubbing:**
- **Lymphadenopathy:** **Pedal edema:**
- **Pulse:** **B.P.:** **Temp:**
- **Breast:** **Thyroid:**

Systemic examination:

- **CVS:**
- **RS:**
- **CNS:**
- **Abdominal Examination:**
- **Per speculum examination:**
- **Per vagina –**

- **Total Bishop Score**

<u>Investigations:</u>

- **Complete blood picture**
- **BT, CT.**
- **Serology**
- **Random Blood sugar**
- **NST/CTG**

Mode of delivery:

Umbilical cord abnormality :

Baby details-

- **Sex of the baby**
- **Birth weight**
- **APGAR 1' 5'**

  Cord blood ph

PATIENT CONSENT FORM

ASSOCIATION OF UMBILICAL CORD ABNORMALITIES AND NON REASSURING FETAL HEART RATE AND ITS PERINATAL OUTCOME

I have read the foregoing information, or it has been read to me. I have had the opportunity to ask questions about it and any questions that I have asked have been answered to my satisfaction. I have understood that I have the right to refuse consent or withdraw it at any time during the study and this will not affect my treatment in any way. I consent voluntarily to participate in this study

Name of Participant_____

Signature/ thumb print of Participant _____

Date _____

Statement by the researcher/person taking consent:

I have accurately read out the information sheet to the potential participant and to the best of my ability made sure that the participant has understood the procedure.

I confirm that the participant was given an opportunity to ask questions about the study and all the questions asked by the participant have been answered correctly and to the best of my ability. I confirm that the individual has not been coerced into giving consent, and the consent has been given freely and voluntarily.

Name of Researcher/person taking the consent: Dr. Neha B S

Signature of Researcher /person taking the consent_____

Date _____

Name and Address of Principal Investigator:

Dr.NEHA B. S

R.L Jalappa Hospital

Tamaka, Kolar.

PATIENT INFORMATION SHEET

Study title: ASSOCIATION OF UMBILICAL CORD ABNORMALITIES AND NON REASSURING FETAL HEART RATE AND ITS PERINATAL OUTCOME

Study location: R L Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar.

Patients who are of clinically indicated for induction admitted to OBG department of R L Jalappa hospital attached to Sri Devaraj Urs medical college are recruited in the study after obtaining patient information consent.

Details-

Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study we will collect information (as per proforma) from you or from a person responsible for you or both. Relevant history will be taken. This information collected will be used only for dissertation and publication.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the Institutional Ethics Committee. There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

For further information contact

Dr.NEHA B S

Post graduate, Department of obstetrics and Gynaecology

R L Jalappa hospital, Kolar.

KEY TO MASTER CHART

- **PARITY**

  PRIMIGRAVIDA -1

  G2-2

  G3-3

  G4-4

- **GESTATIONAL AGE**

  37-40 WEEKS - 1

  41-42 WEEKS -2

- **CTG**

  NON REASSURING CTG-2

  ABNORMAL CTG- 3

- **MODE OF DELIVERY**

  VAGINAL DELIVERY -1

  LSCS- 2

- **CORD ABNORMALITIES**

  LONG CORD-1

  SHORT CORD -2

  CORD KNOT-3

  LOOPS OF CORD- 4

- **SEX OF THE BABY**

  MALE-1

  FEMALE-2

- **PERINTAL OUTCOME**

  NICU-1

  MS - 2

MASTER CHART

Sl No.	OP No.	IP No.	NAME	AGE	PARITY	GESTATIONAL AGE	CTG	MODE OF DELIVERY	APGAR SCORE		CORD BLOOD PH	UMBILICAL CORD ABNORMALITY	BABY SEX	BABY WEIGHT	PERINATAL OUTCOME
				YEARS					1 MIN	5 MIN				KILO GRAM	
1	682088	189332/2019	YASMEEN	29	2	1	2	2	7	9	7.36	2	1	2.7	2
2	654033	181095/2018	ARPITHA P	20	1	1	2	2	7	9	7.12	1	1	3.34	1
3	713697	198191/2019	ROOPA M A	26	1	1	2	2	6	8	7.22	2	2	2.4	1
4	756092	228117/2020	LEKHA C P	29	2	1	2	1	7	9	7.14	2	2	2.72	1
5	695236	193201/2019	NAZIMA BEGUM	25	1	1	2	2	7	9	7.37	2	1	3.32	2
6	825902	226781/2020	ASHWINI	25	2	2	2	1	6	8	7.42	2	1	3.28	2
7	825005	226536/2020	SHILPA T	25	3	1	2	1	7	9	7.24	1	1	3.18	1
8	810398	233530/2020	SUMALATHA	27	3	1	2	2	7	9	7.04	3	1	3.52	1
9	805868	221982/2019	PALLAVI	27	4	1	2	1	7	9	7.14	2	1	3.1	1
10	810383	226134/2020	SUDHA N	30	2	1	2	2	7	9	7.38	4	1	3.2	1
11	846869	232750/2020	KAVITHA N	24	3	2	2	2	7	9	7.42	2	2	2.96	2
12	733835	203223/2019	SNEHA	20	1	1	2	2	7	9	7.16	2	1	2.13	1
13	800156	220696/2019	ANJUM TAJ	30	1	1	2	2	6	8	7.18	1	1	2.34	1
14	799214	220420/2019	ZAIBA BANU	22	2	1	2	1	6	8	7.19	2	2	2.51	1
15	720078	219730/2019	PRIYANKA C	22	1	1	2	2	7	9	7.39	2	1	2.54	2
16	821814	225763/2020	NANDINI G	19	3	1	3	2	7	9	7.41	4	2	2.98	2
17	835080	229137/2020	NAHEEDA SULTHANA	20	1	1	3	1	7	9	7.22	4	1	2.82	1
18	732873	221832/2019	VANI	24	2	1	2	2	6	8	7.21	1	2	3.29	1
19	672697	186586/2019	BHAGYA	25	1	1	2	2	7	9	7.22	1	1	2.42	1
20	794676	219305/2019	NETHRAVATHI	28	2	1	2	2	7	9	7.42	1	1	2.6	2
21	741250	208454/2019	NANDINI B N	29	4	1	2	2	7	9	7.38	1	1	3.02	2
22	410679	228978/2020	LAKSHMI BANU	18	2	1	3	2	7	9	7.13	4	1	3.48	1
23	796384	219729/2019	FRIDOSE	20	1	1	3	1	7	9	7.22	4	2	2.77	1
24	783276	219131/2019	SHAILJA T N	25	1	1	3	2	6	8	7.23	4	1	3.6	1
25	852208	224479/2020	PUSPHA	28	1	2	2	2	7	9	7.22	3	1	2.9	2
26	849034	233475/2020	PUSPHA K N	23	1	1	2	1	7	9	7.41	3	1	2.52	2
27	855502	235985/2020	ANITHA K	22	1	1	2	2	7	9	7.24	3	1	3.65	1
28	799221	220427/2019	PADMA SUDHA	35	3	1	2	2	7	9	7.32	3	2	2.7	2
29	679465	188529/2019	HAZEERA	28	1	1	2	2	5	7	7.66	2	2	2.8	1
30	683368	195074/2019	NAGAMMA	20	2	1	2	2	7	9	7.12	1	1	3.23	1
31	808932	231801/2020	CHANDANA	20	1	1	2	1	7	9	7.42	3	1	2.78	2
32	846005	232449/2020	SUMA	22	1	1	2	2	7	9	7.22	3	2	2.45	2
33	671881	186123/2019	BHAGYAMMA	24	3	1	2	2	6	8	7.57	3	1	2.6	1
34	732866	222128/2019	LAVANYA	26	1	1	3	1	7	9	7.55	4	2	3.01	1
35	815478	224183/2020	GOWTHAMI Y N	24	1	1	2	2	7	9	7.24	4	1	2.7	1
36	842293	232033/2020	VEENA	29	2	1	2	2	7	9	7.43	3	1	2.96	2

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37	714775	220047/2019	TRIVENI	22	1	1	2	2	7	9	7.12	2	2	2.56	1
38	803468	228650/2020	KAVITHA G	29	2	1	2	2	7	9	7.22	3	1	2.7	1
39	795110	219402/2019	MANJULA	24	4	1	2	2	7	9	7.43	3	1	3	2
40	843257	233171/2020	RASIKA HARISH	21	1	1	2	2	6	8	7.23	3	2	3	1
41	827564	227201/2020	VEENA	32	2	1	2	2	7	9	7.44	3	1	2.9	2
42	793598	218988/2019	PALLAVI	28	2	1	2	2	6	8	7.19	4	2	2.7	1
43	767515	213357/2019	MEGHA K	24	1	1	3	2	7	9	7.24	4	1	2.8	1
44	853696	234866/2020	SUMA	26	4	1	2	2	6	8	7.38	1	2	3.66	2
45	857446	236025/2020	NAZMA	30	2	1	2	1	7	9	7.25	3	1	2.1	2
46	824551	226426/2020	PAVITHRA	24	1	1	2	1	7	9	7.26	3	1	2.7	1
47	844301	231884/2020	NETHRAVATHI	31	1	1	2	2	7	9	7.12	3	1	3.2	1
48	789012	217873/2019	VIJAYAKSHMI N V	28	2	1	2	2	7	9	7.56	2	2	2.17	1
49	813825	223806/2020	ASMA BANU	30	1	1	2	2	7	9	7.37	3	2	3	2
50	856946	235860/2020	NAZIYA BANU	20	2	1	2	2	7	9	7.42	1	1	2.74	2
51	697557	193682/2019	SHANTHAKUMARI	27	1	1	2	2	6	8	7.23	3	2	1.98	1
52	797455	220622/2019	SUMITHRA	25	1	1	2	2	7	9	7.16	4	1	3.4	1
53	835931	229360/2020	RADHIK	24	3	1	2	2	7	9	7.14	1	1	3.78	1
54	838084	229938/2020	VARALAKSHMI	22	1	1	2	1	7	9	7.38	4	1	2.92	2
55	703614	195598/2019	SANDHYA	24	3	1	2	2	7	9	7.37	1	2	3.4	2
56	815531	224198/2019	MONISHA	20	1	1	3	2	7	9	7.18	4	1	2.86	1
57	702489	195209/2019	AFRINA BEGUM	19	1	1	2	2	5	7	7.56	4	1	2.62	1
58	583048	163420/2018	NAVYA	20	3	1	2	2	7	9	7.14	3	2	2.96	2
59	663888	183779/2018	KAVITHA	20	2	1	2	2	7	9	7.38	3	2	2.88	1
60	777659	222052/2019	MANJULA DEVI	32	3	1	2	2	7	9	7.36	2	1	2.8	1
61	821382	225657/2020	NETHRAVATHI	23	1	1	2	2	7	9	7.13	2	2	2.5	1
62	841169	232028/2020	KAVITHA	29	2	1	2	1	7	9	7.42	4	1	3.59	2
63	779935	220094/2019	SHIREESHA	24	2	1	2	2	7	9	7.11	1	1	3.06	1
64	797867	220099/2019	DURGA	23	2	2	2	1	7	9	7.44	4	1	2.82	1
65	820080	225371/2020	MUBEENA	20	1	1	2	2	7	9	7.22	4	2	2.48	1
66	793822	219085/2018	NAGMA SILTANA	26	2	1	2	2	6	8	7.32	4	1	2.82	2
67	766708	211944/2019	ARCHANA G N	25	1	1	3	2	6	8	7.14	4	1	2.98	1
68	857206	235972/2020	JAYASHREE	24	2	1	3	1	7	9	7.36	2	1	2.8	2
69	801886	221010/2019	SIRISHA M	20	1	1	2	1	7	9	7.22	4	1	1.72	1

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70	764255	211294/2019	SHIREEN TAJ	23	3	1	2	2	7	9	7.61	4	2	2.7	1
71	765655	221608/2019	SUMIYA KOUSER	19	2	1	2	2	5	7	7.12	3	1	2.3	1
72	791862	218587/2019	CHANDHINI	22	1	1	3	1	7	9	7.22	4	2	1.9	1
73	721312	219326/2019	ASHA M	29	2	1	2	2	7	9	7.32	4	1	2.44	2
74	819617	227483/2020	ROOPA K	28	3	1	2	2	7	9	7.42	4	1	2.5	2
75	792195	218627/2019	FIZA SAMARIN	19	3	1	2	2	6	9	7.11	1	1	2.97	1
76	800535	220712/2019	LALITHA	22	1	1	3	1	7	9	7.2	4	2	2.88	1
77	783526	223911/2020	MEENA	23	2	1	3	2	3	5	7.18	4	1	2.9	1
78	817355	224676/2020	MEENAKSHI	26	1	1	2	2	7	9	7.29	4	2	2.58	2
79	779325	215318/2019	PAVITHRA.V	27	1	1	2	1	7	9	7.33	4	1	2.18	1
80	786486	217211/2019	SHRAVANI	22	2	1	2	1	7	9	7.7	4	2	2.27	1
81	647710	178972/2018	HEENA SURAYA	22	1	1	2	1	6	8	7.32	4	1	2.64	1
82	849107	233510/2020	YASMIN	20	2	1	2	2	5	7	7.24	2	2	2.54	1
83	654770	181065/2018	NASEEMA	26	2	1	2	2	6	9	7.23	1	1	3.38	2
84	775871	214369/2019	HEENA KOUSAR	25	2	1	2	2	7	9	7.36	1	1	3.1	2
85	680305	188796/2019	ANJUM	25	3	1	2	1	6	8	7.22	1	2	2.52	1
86	777492	218976/2019	PADMINI M	29	1	1	3	2	7	9	7.17	1	1	2.14	1
87	664182	185223/2018	MANUJA MUNIRAJ	24	1	1	2	2	7	9	7.29	1	1	3.64	2
88	792640	218722/2019	AMREEN TAJ	30	4	1	2	1	6	8	7.32	1	1	2.7	1
89	664707	183995/2018	REKHA	24	2	1	2	2	7	9	7.22	1	1	3.64	2
90	705581	196076/2019	AMALA	25	2	1	2	2	6	9	7.14	2	2	3.06	1
91	742177	205476/2019	SHAZEEYA	28	4	1	2	2	7	9	7.21	1	2	2.8	1
92	823549	226158/2020	PRIYANKA M B	20	1	1	2	1	7	9	7.13	1	1	3	1
93	787339	217403/2019	NIRMALA N	23	3	1	2	2	6	9	7.37	1	1	3.47	2
94	846401	232594/2020	SUPRIYA	20	1	1	2	2	7	9	7.18	1	1	2.1	1
95	726246	222213/2019	MANJULA	25	2	1	2	2	6	9	7.29	1	1	2.7	1
96	841452	230764/2020	VEENA A	27	1	1	2	1	7	9	7.34	1	1	2.51	2
97	762682	210856/2019	RADHIKA M	25	3	1	2	2	7	9	7.38	1	1	3.7	2
98	605224	188427/2019	DEEPIKA	20	1	1	2	1	6	8	7.28	1	2	2.52	1
99	674018	186831/2018	ANUREKHA	30	1	1	2	2	7	9	7.21	1	2	2.42	2
100	747331	206872/2019	SUSHMITHA	24	1	1	2	2	7	9	7.32	1	1	2.06	1
101	677174	188258/2019	NETHRAVATHI N R	24	1	1	2	1	5	7	7.12	1	2	3.02	1
102	787510	222680/2019	CHAITHRA	27	3	1	2	2	6	8	7.14	1	1	3	1
103	812703	223588/2019	CHANDINI	21	1	1	2	1	7	9	7.33	4	1	2.74	2
104	787953	217606/2019	CHANDIDNI	27	1	1	2	1	3	6	7.13	2	1	2.4	1
105	689713	216345/2019	SARASWATHI	30	2	1	2	1	7	9	7.66	4	2	3.88	1
106	796981	219897/2019	SALMA TAJ	18	1	1	2	1	7	9	7.32	4	1	2.6	2

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107	835558	230788/2020	MONICA V	24	1	1	2	2	7	9	7.4	3	1	2.86	1
108	844846	232097/2020	AMBIKA	27	1	1	2	1	7	9	7.14	1	1	2.57	2
109	793815	219080/2019	NAGGINA TAJ	24	2	1	3	1	6	8	7.17	4	1	2.6	2
110	851981	234429/2020	GOUSIYA	28	3	1	2	2	7	9	7.42	4	1	4.24	2
111	796011	219639/2019	SUPRIYA	22	2	1	2	2	7	9	7.32	4	1	3.4	2
112	794231	219173/2019	SUMITHRA	19	2	1	3	1	6	8	7.22	4	2	2.58	1
113	724989	220341/2019	SOUNDRYA	21	1	1	2	2	7	9	7.31	4	1	2.92	2
114	816810	224505/2019	ARCHANA	20	1	1	2	2	7	9	7.34	4	1	2.8	2
115	702736	195751/2019	ARUNA	29	2	1	2	2	7	9	7.21	4	2	3.08	2
116	793813	219076/2019	HEMAVATHI	23	2	2	2	2	6	8	7.22	4	1	3.54	2
117	797853	220094/2019	SHIEESHA	24	2	1	2	1	7	9	7.21	1	1	3.06	2
118	835521	229241/2020	MAMATHA	25	1	1	3	2	7	9	7.43	4	2	2.7	2
119	800516	220696/2019	ANJUM TAJ	30	1	1	3	2	7	9	7.32	4	1	2.34	1
120	787849	217539/2019	ARUNA C V	25	3	1	3	2	7	9	7.21	4	2	2.74	1
121	857451	236027/2020	ASMA BANU	21	2	1	2	2	7	9	7.19	4	1	2.96	1
122	863172	237816/2020	BHAVANI	22	1	1	3	2	7	9	7.14	4	1	2.66	1
123	858814	236457/2020	FARNAZ BEGUM	25	3	1	3	1	7	9	7.24	2	2	3.86	2
124	779245	228750/2020	LOKESHWARI	30	3	1	3	2	7	9	7.22	4	1	2.8	1
125	857157	235934/2020	MUSKAN	23	3	1	3	2	7	9	7.32	4	1	3.2	2
126	834541	236058/2020	NANDINISHREE	26	2	1	3	2	7	9	7.13	1	2	2.88	1
127	795133	219416/2019	NETHRAVATHI	28	4	1	2	2	6	8	7.15	4	2	2.56	2
128	802754	221211/2019	ASMA M	19	1	1	3	2	7	9	7.28	4	1	2.2	1
129	782646	216190/2019	LAKSHMIDEVI	30	2	1	3	2	6	8	7.26	4	1	2.8	1
130	848723	236103/2020	SANDHYA	27	2	1	3	2	7	9	7.42	4	2	3.2	2
131	697551	212146/2019	SHANTHAKUMARI	27	3	1	3	2	6	8	7.13	4	2	1.98	1
132	861795	237385/2020	SHILPA	25	3	1	3	1	4	6	7.14	4	1	3.08	1
133	497258	163229/2018	SUMA	25	3	1	3	2	7	9	7.12	3	2	2.56	2
134	777405	228835/2019	NUSHRATH	23	2	2	3	2	7	9	7.22	4	1	2.8	1
135	841233	236027/2020	LAKSHMI	24	3	1	3	1	7	9	7.32	4	1	2.96	1
136	800534	221673/2019	MATHEENA	22	2	2	2	2	6	8	7.24	4	1	3.1	1
137	780398	221673/2019	MANJULA	23	3	1	3	1	7	9	7.21	2	1	2.5	1
138	868215	239302/2020	FARZANA	24	3	1	2	2	7	9	7.13	4	1	3.2	1
139	860943	237731/2020	GOWTHAMI	25	2	1	2	2	6	8	7.14	4	1	2.34	2
140	850422	233967/2020	SHANBANA	25	3	2	3	2	7	9	7.17	1	2	2.42	2
141	540092	167049/2018	ANITHAKUMARI	22	3	1	3	2	7	9	7.28	4	1	2.9	2
142	675962	233215/2019	PUSHPA H D	22	3	1	3	2	7	9	7.22	4	1	2	1
143	577791	165538/2018	LALITHA	24	2	1	2	2	4	7	7.23	4	2	2.9	1

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144	792057	218832/2019	ASHWINI	25	3	1	2	2	7	9	7.18	2	1	2.7	1
145	793261	187656/2019	BHARGAVI	27	4	2	3	1	7	9	7.29	4	1	2.6	2
146	829429	223421/2019	ASWANI	28	4	1	3	2	6	8	7.22	4	1	3.1	1
147	841987	214345/2019	NANDINI	18	2	2	2	2	7	9	7.6	4	1	3	1
148	841911	221243/2019	SALMA	19	4	1	3	2	7	9	7.15	4	2	2.8	2
149	876420	198734/2020	SUMA	19	3	2	2	1	6	8	7.16	2	1	3	1
150	857594	223432/2020	SWAPNA	30	3	1	3	2	6	8	7.5	1	1	2.7	1
					PRIMIGRAVIDA -1	37-40 WEEKS - 1	NON REASSURING CTG-2	VAGINAL DELIVERY -1				LONG CORD-1			
					G2-2	41-42 WEEKS -2	ABNORMAL CTG- 3	LSCS- 2				SHORT CORD -2			
					G3-3							CORD KNOT-3	MALE-1		NICU-1
					G4-4							LOOPS OF CORD- 4	FEMALE-2		MS - 2