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TOPIC- COMMON UMBILICAL CORD ACCIDENTS AND THEIR EFFECT ON THE IMMEDIATE POSTNATAL OUTCOME.

# INDEX

## INTRODUCTION

According to Donald “The baby’s life hangs by a cord.”[1] We realize this when we encounter the heavy toll on foetal life because of cord complications. Every obstetrician has this bewildering and frustrating experience of a sudden and unaccountable foetal death before or during labour when the pregnancy had been normal in all respects. Umbilical cord accidents require judgement and experience of the obstetrician at the same moment and place. The issue of umbilical cord related foetal harm and foetal still birth is unaddressed in modern practice. 30% of births have some type of umbilical cord finding.[2] One has to wonder what thoughts prehistoric humans had when confronted with the still birth of a baby entangled in its umbilical cord.



Umbilical Cord Accidents are a type of ‘Sudden Antenatal Death Syndrome’ which needs study and account for 15% of sudden antenatal death cases.[2]

As of the year 2002, given the best evidence published, a rate of 2 deaths/1000 births is due to an umbilical cord accidents.[3, 4]

Medical and lay literature on umbilical cord Accident has accumulated since the Collaborative Perinatal Project (CPP). The CPP data is used by the National Institutes of Health to study the current obstetrical issues.[5, 6, 7]

Practically one out of every 3 to 5 deliveries will have an umbilical cord finding. One out of 100 deliveries will be at risk of umbilical cord accident still birth. Of the 2 umbilical cord accident stillbirths /1000 births, one will be a term stillbirth.[8,9]

Umbilical Cord Accident can also repeat in the same patient. Reported cases have described subsequent pregnancies with similar umbilical cord accident, although not

necessarily stillbirth.[10,11] A mother gave birth four times and in each pregnancy a true knot.[2]

In order to solve the issue of umbilical cord accidents, an Institute called as Pregnancy Institute dedicated to solving the problem of umbilical cord accidents has been set up. It is a 50/©- 3 non profit medical research corporation cofounded by JASON H COLLINS, M.D., an obstetrician interested in improving birth outcomes, Charles L Collins, BSE, M.D., a pathologist interested in placental changes, and Candace C. Collins, M.D., a paediatric ophthalmologist interested in learning disabilities.[2]

Since tragedies such as perinatal death or severe neurological impairment are increasingly being recognized as caused by pathological conditions of umbilical cord that frequently develop long before labour and delivery and cannot be prevented by the best of the obstetrical care.[12,13] That is why nowadays they are increasingly being diagnosed by prenatal Ultrasonography ,thereby warning of potential complications at delivery and providing clues to the general well being of the foetus.[1] This topic needs study as the literature of relevance of these cord lesions has been tarnished by ascertainment of selection bias.



And altogether in any community hospital practising obstetrics, cord complications will be observed more often than congenital defects. For instance, one umbilical review of just hematoma formation of umbilical cord at Johns Hopkins Medical Centre was observed in one out of 5,505 cases. Mongolism, a common genetic alteration known as Trisomy 21 occurs overall 1 in every 660 deliveries.[2]

Ultrasonographic identification of umbilical cord accidents is possible and follow up of these foetuses require current obstetrical practices of Non Stress Test,

Bio Physical Profile and Induction when indicated. Special attention during labour and delivery may help avoid an unexpected adverse event in an otherwise normal pregnancy.[15]

The practical goal of 21st century obstetrics is not only to successively deliver an infant but to do so without a poor outcome. The interval from 36-38 weeks may require more scrutiny of the foetus especially where an umbilical cord accidents is identified.

Management of prenatally diagnosed umbilical cord accidents and determination of foetal intervention will create the ability to avoid stillbirth.[16, 17]

# DEVELOPMENT AND ANATOMY OF UMBILICAL CORD

Synonym- FUNIS

The fully developed umbilical cord is on an average 50 cm long and 1-2 cm wide in diameter.[18]

1. Length : The length varies from 20-120 cm.[18] A short cord is <35 cm in length ( lower-6th percentile ) and a long cord measures >80 cm ( upper 6th percentile )[19]
2. Circumference: The mean umbilical cord circumference at 40 weeks is 3.6 cm. (Range – 2.6 to 6 cm) [20]. The 90th percentile for the area of umbilical cord at term is 1.3 $cm^2$ . . [21]
3. Covering epithelium : The exterior of umbilical cord is dull white, moist and covered by amnion through which umbilical vessels maybe seen.[22]
4. Wharton' jelly: It is a specialized tissue serving many purposes for the developing foetus. Its specialized cells contain gelatine like mucus that encase fibres, these properties give it an elastic and cushion effect, which can tolerate the vibration, bending, stretching and twisting of an active foetus. In addition, it holds the vessels together, may regulate blood flow, plays a role in providing nutrition to foetus, stores chemistry for the onset of labour and protects the supply line[2] .

   Umbilical cords with decreased Wharton's jelly are prone to compression and complete absence is usually associated with foetal death. If an umbilical cord is twisted or knotted, it is more likely to tighten where there is less resistance, such as an area low in Wharton's jelly.[2]

5. Blood vessels: Initially there are 4 vessels – 2 arteries and 2 veins. The arteries are derived from internal iliac arteries of the foetus and carry venous blood from the foetus to the placenta. Of the 2 umbilical veins, the right one disappears by the end of the 2nd month at 6 mm stage leaving behind one vein which carries oxygenated blood from placenta to the foetus. Presence of a single umbilical artery is often associated with congenital anomalies. In 1 in 200 newborns there is 20% chance of having cardiac and other vascular defects with one umbilical artery.[23]
6. Remnant of the umbilical vesicle (yolk sac) and its vitelline duct : Remnant of the yolk sac may be found as a small yellow body near the attachment of the cord to the placenta or rarely the proximal part of the duct persists as Meckel's Diverticulum.[23]
7. Allantois : A blind tubular structure may occasionally be present near the foetal end which is continuous inside the foetus with its urachus and bladder.[23]
8. Obliterated Extra Embryonic Coelom : In the early period, Intra embryonic coelom is continuous with the Extra embryonic coelom along with herniation of coils of intestine (midgut). This connection may persist as congenital umbilical hernia or exomphalos.[23]

   Attachment of the cord: According to Inderbir singh 1991 [24] ,in the early period the cord is attached to central surface of the embryo close to the caudal extremity, but as the coelom closes and the yolk sac atrophies, the point of attachment is moved permanently to the centre of the abdomen by the 4th month. Unlike the foetal attachement, the placental attachment is inconsistent.[24]

Types of attachment: (1) Central

(2) Eccentric – commonest

(3) Marginal

(4) Velamentous

9. Tensile strength: Several reports have measured the breaking point of human umbilical cord. Because of the differences in wharton's jelly, collagen content and muscle layer structure, there is a range of breakage points and sites[25,26(As quoted by Collins)] .The average load required to break the majority of human umbilical cord is 10-14 lbs, the range being 4-24 lbs (at term 1.81-10.89 kg). Cords ruptured 22.5% of the time at the placental end. The human umbilical cord is elastic and can stretch to 12.5% of its original length. The tensile strength may average 2.5% of foetal weight.[2]

## DEVELOPMENT OF UMBILICAL CORD [27]

By day 13 (post conception) the blastocyst contains an embryo surrounded by a loose meshwork of extra embryonic mesoderm. The embryo is composed of 2 cavities – the amniotic cavity and the primary yolk sac. The embryonic disc forms where these 2 cavities meet and consists of 2 epithelial layers- the ectoderm, which is continuous with the amniotic epithelium and the endoderm, which partially surrounds the primary yolk sac cavity.

By day 18, the endoderm completely surrounds the yolk sac and the exocoelom has cavitated within the extra embryonic mesoderm. Part of this mesoderm, the chorionic mesoderm, lines the inner surface of the trophoblastic shell, whereas the remainder covers the surface of the two embryonic cavities. These 2 portions of extraembryonic mesoderm are connected in only one place, basal to the amniotic cavity. This mesenchymal bridge, the connecting stalk,will ultimately form the umbilical cord. The allantois, the primitive extraembryonic urinary bladder, forms as a duct like extension of the primary yolk sac that protrudes into the connecting stalk at the presumptive caudal end of the embryonic disc.



Three developmental processes occur during the subsequent 3 weeks until day 40.

1. The embryo rotates. The yolk sac, which originally faced away from the implantation site, turns towards the implantation site.
2. The amniotic cavity enlarges considerably, extending around the embryo, compressing the exocoelom, which gradually disappears by midgestation.

3. The originally flat embryonic disc bends in the anteroposterior direction and rolls up in the lateral direction, thus herniating into the expanded amniotic cavity. By bending, the embryo subdivides the yolk sac into an intra embryonic part (the primitive intestinal tract) and an extra embryonic part (the omphalomesenteric) or vitelline duct with its accompanying vessels and its dilated peripheral position, the secondary yolk sac. The latter comes to reside within the connecting stalk, as the amniotic cavity expands and compresses the stalk, allantois and yolk sac into a slender cord covered by amniotic epithelium, thus forming the umbilical cord.

   Both the Omphalomesentric duct and the allantois ducts extrude from the future embryonic gut into the connecting stalk. During the 3$^{rd}$ week, they become supplied with foetal vessels.

   The two allantoic arteries originating from the internal iliac arteries, and one (left) allantoic vein, which enters the left hepatic vein, unite with a plexus of sinusoid spaces that develop within the cord mesoderm and ultimately with villous vessels. The right umbilical vein originally present disappears by the 6 mm stage (the end of the 2$^{nd}$ month).



The umbilical cord is analogous to erectile tissue in 2 respects. The loose cushion like jelly with its stellate stromal cells in vitro is confined in vivo, to a thin rim of tissue that creates a semi rigid structure and resistant sheath about the turgid vessels.

Secondly, the umbilical vein pressure is maintained by a sphincter like mechanism innervated by vagus nerve that regulates outflow resistance at the ductus venosus.

There are no lymphatic vessels in the cord and fixed macrophages are essentially absent.

The extra corporeal human umbilical vessels differ from all other foetal vessels of the comparable size. The vein, but not the artery has a well developed lamina elastica interna. The media has no true circular or longitudinal layers, but consists of decussating helicoidal smooth muscle bundles that shorten to become more nearly circular with contraction. Furrows on the outer surface of arteries correspond to inner wall folds of Hoboken that alternate with dilated nodes of Hoboken, structures that are present in vivo, occlude the lumen in spasm, and provide an intrinsic capacity to control bleeding in unassisted labor.

The cords' nutritional requirements are met with transmural diffusion, primarily across the thin walled vein. The placenta has no nerve supply and no nerves traverse the umbilical cord from the foetus to the placenta. Within 2-3 cm of the placental insertion ,in 95% of all cords, arterial fusion or macroscopic anastomosis equalizes the flow and pressure in the two arterial placental territories. This anastomosis is a safety valve, in the event of luminal compression.



The cord vessels are arranged in a spiral or helicoidal fashion along the cord. The average number of cord helices is 11, but as many as 380 have seen described. Approximately 75% of cord helices are left handed (wind counter clockwise) regardless of which direction the cord is viewed[2].

What is remarkable about the umbilical cord is that it is a blood vessel without branches. The umbilical cord has two-way traffic. The arteries carry blood pumped by the heart away from the foetus and this circulation surrounds the vein normally, the umbilical vein returns blood to the foetus from the placenta rejuvenated with oxygen and nutrients and devoid of waste products.[2]

How this happens is still surrounded by mystery. The foetal heart cannot expand or work harder because it is surrounded by a fluid filled lung. Therefore as the foetus grows exponentially, it accommodates the increased blood volume overtime by elongation of cord in length and width.[2]

## AIIMS AND OBJECTIVES

To study five commonly occurring umbilical cord accidents;

1. Nuchal cord - Tight nuchal Cord and Loose Nuchal Cord
2. Body Loop / Extremity Loop
3. True Knots
4. Cord Prolapse
5. Marginal Insertion of Placenta

Their Affect on the Immediate Postnatal Outcome in terms of the following was noted ;

1. Stillbirth
2. 1 Minute Apgar score of < 7
3. Neonates requiring Active Resuscitation measures like Ambubag, Endotracheal Intubation , Chest Compressions or Use of Drugs.
4. Neonates requiring NICU ( Neonatal intensive care unit ) Admissions.

## REVIEW OF LITERATURE

According to Fox (1978)[28] , it is the cliché of obstetric writing that the umbilical cord is the “Lifeline of Fetus”. Cord accidents are still one of the major causes of fetal mortality.

Browne (1925) [29] was the first to publish a detailed study of cord pathology.

The cord serves a vital function, but is unfortunately susceptible to entanglement, compression and occlusion.[30]Collins and Collins (2000)[30] reported a 1.1% incidence of potentially harmful cord complications.[30]

It is not a recent idea that cord accidents may cause fetal distress and intrapartum complications. It has been reported that coiling of the umbilical cord has been described as one of the dangers of the eighth month by Hippocrates or an unknown disciple in an entitled “De Octimestri Partu”. [31]



Early descriptions of fetal loss from cord entanglement date as far as 200 years ago. In olden times umbilical cord was considered as an omen, a sacred talisman, predictor of future fertility. In Europe, Australia, Africa and Hawaii, the umbilical cord was dried and soaked in water for consumption to ensure future fertility. Chinese literature suggests the cord had medicinal properties.[2]

European insights beginning with Galen (129-200) suggested the umbilical cord served to nurture the fetus through arteries and veins.[2]

Leonardo da vinci (1452-1519) observed that the cord was as long as the fetus itself at a given gestational age.[2]

Spigilus (1631)(as quoted by Collins) [2] determined blood flow direction, and Harvey (1657) (as quoted by Collins)[2] suggested that interruption of this blood flow could be the cause of fetal death if the cord was compressed.[2]

In 1741, the British obstetrician Dr. John Burton described several cases of cord accidents whose delivery was impeded by a short cord and an entangled cord.[2]

In 1750, the British obstetrician William Smithe describes a case #172 in treatise on the theory and practice of midwifery as a stillborn with four loops of nuchal cord.[2]

One of the first published drawings of an Umbilical Cord Accident was by Andrew Bell in the 'Encyclopedia Britanica', 1st edition in 1769, Edinburgh, Scotland, depicting a fetal death with a combination of one nuchal cord, a body loop and a true knot.[2]



By the 1800's , many observations were recorded of distressed foetuses born with cord entanglement and cord abnormalities.[2]

The normal umbilical cord may have been best illustrated and described in 1882 by E S Tamter.[2]

There are 8 different types of umbilical cord.[32,33,34,35,36]

Dr. Nelah Sackett in 1933 described a case where he reached inside the uterus, "encountered 2 loops around the baby's neck and extracted the infant in situ (version extraction)" rupturing the cord in the process.[2]

Umbilical cord accidents occur throughout gestation from conception to delivery with predisposition in each trimester. In 60% of umbilical cord accidents still births occur prior to 20 weeks, 5% from 20-36 weeks and at the rate of 15% from 36-40 weeks.[8,37]

Jason H Collins M.D., Charles L Collins M.D., & Candace C Collins M.D. have cofounded a non profit 501(c) -3 ; Medical Research Foundation dedicated to solving the problems of umbilical cord accidents and have also published a book called a “SILENT RISK” dedicated to parents who have experienced the loss of a newborn secondary to an umbilical cord accident..[2] Their address is Pregnancy Institute P.O. Box 806 , New Roads LA 70760 . office – 985-710-0672. Fax – 225-638-8351. Email – haydell @bellsouth.net.

According to Collins[2], list of commonly occurring Umbilical Cord Accidents are [2] :

| Type of Umbilical cord accident | Incidence | mortality |
|---|---|---|
| Short cord <35 cm | 2% | unknown |
| Long cord >70 cm | 3.7% | unkown |
| Abnormal cord form | 10-15% | unknown |
| Hyperhelical (coiled) | 1% | unknown |
| Hypohelical( straight) | 5% | unknown |
| Single umbilical artery | 0.2-3.6% | 7% |
| Nuchal loop | 14-305 | unknown |
| Nuchal cord | 14-30% | unknown |
| Type a | 14-30% | unknown |
| Type b | 0.02-1% | unknown |
| Torsion | 1-10% | 20-40% |
| Body loop | 5-24% | 9-10% |
| True knot | 1-25% | 1.7-10% |
| Marginal insertion | 5-7% | unknown |
| Vellamentous insertion | 0.54%-2.2% | 30% |
| Vasa previa | 1%-11% | 30-100% |
| Cord prolapse | 0.1-1% | 5% |
| Monoamniotic twins | 70% | 50-70% |
| Umbilical constriction | 1% | 100% |

According to Collins[2], umbilical cord accidents cause stillbirth , asphyxia, emergency caesarean sections, fetal distress and neurological damage which may consist of learning disabilities and cerebral palsy.[2]

According to Chauhan SP[38], umbilical cord accidents can repeat in the next pregnancy.[38] There is 25% general risk of ocurrence of Umbilical cord accidents and 50% is its risk of recurrence.[38]

According to Collins, these umbilical cord complications occur atleast once in every 4 deliveries and their problems cannot be avoided.[2]



According to Collins, causes of Intrauterine Anoxia causing stillbirths are : [2]

| | Cause of Intrauterine anoxia | Number of stillbirths | Percentage |
|---|---|---|---|
| 1 | Cord accidents | 17 | 28.8% |
| 2 | Intrauterine asphyxia | 14 | 23.7% |
| 3 | Abruption placenta | 10 | 16.9% |
| 4 | Pre-eclampsia/ eclampsia | 7 | 11.9% |
| 5 | Placenta previa | 6 | 10.2% |
| 6 | Rupture of uterus | 2 | 3.4% |
| 7 | Shoulder dystocia | 2 | 3.4% |
| 8 | Foetal distress | 1 | 1.7% |
| | TOTAL | 59 | 100% |

According to Collins[2], the Worldwide frequency of Umbilical Cord Accidents and Stillbirths due to them are : [2]

| | Umbilical Cord Accidents | Incidence | Mortality |
|---|---|---|---|
| 1 | Single umbilical artery | 0.2-3.6% | 7% |
| 2 | Non coiled cords | 4.3% | unknown |
| 3 | True knots | 1% | 6% |
| 4 | Nuchal cords | 14-30% | unknown |
| 5 | Body loops | 1% | 10% |
| 6 | Short cords | Unknown | unknown |
| 7 | Velamentous cords | 0.54-2.17% | 30% |
| 8 | Torsioned cords | 6% | 20% |



According to Collins[2], there may be increased risk of umbilical cord accident still birth when a combination is present like marginal insertion-nuchal cord and a true knot-all in the same fetus.[2]

According to Collins[2], monoamniotic twins (MAT) have a 70% or more risk of cord entanglement. These cases have been prenatally diagnosed with a variety of umbilical cord complications which can have 10%mortality if managed to a 70% mortality if unmanaged. Several cases of monoamniotic triplets have been reported to have been managed for cord entanglement complications.[2]

According to Collins[2], two types of deaths seem to be related to cord complications: sudden death such as with cord rupture, hematoma, prolapse, and may be entanglement. And slow death- such as with nuchal cord, knots, body loops.[2]

According to Collins[2], in addition to mortality from umbilical cord accidents, there is also an issue of morbidity from umbilical cord accidents. The Collaborative Perinatal Project(CPP) noted umbilical cord accidents associated fetal effects such as meconium, asphyxia, foetal growth disruption and low IQ levels.[2]

According to Collins[2], umbilical cord accidents are related to prolonged second stage of labour, failure of descent of the presenting part, post partum bleeding and foetal distress. A case report on the failure of descent due to nuchal cord and reports of consequences of tight nuchal cord illustrate the contribution of umbilical cord accident to foetal morbidity. In human case reports umbilical cord accident associated injuries documented are : neurological, cardiac, renal, pulmonary, gastrointestinal, hepatic, vestibular and ophthalmic.[2]



According to Collins[2], umbilical cord blood flow can be studied with colour Doppler velocimetry techniques. Doppler velocimetry has demonstrated alterations in blood flow with nuchal cord, true knots, single umbilical arteries and in foetuses with underdevelopment and cardiac failure.[2]

Baden in 1955 wrote so eloquently,"Iintrauterine life sustained only by two small arteries and a tortuous vein coursing through a long flexible cord, hangs by a very delicate thread". To those who practise obstetrics , it seems that this delicate thread too often wrapped around the newborn's neck ,much like a hangman's noose ,the

nuchal cord is often blamed for problems that are encountered during delivery and has often acted as a major cause of foetal distress and perinatal mortality. However the actual significance that a nuchal cord has on the outcome of an infant ,is controversial.[42]

In 1977 Dr. M. Maneshci ( Universitad di Palemo Sicili ) – reported a still born with two nuchal cords and a true knot and recommended that a situation of this kind could be suspected on the basis of certain clinical findings to be elicited by questioning the patient about the intensity of active foetal movements, presence of changes of presentation of the foetus and findings of a high presenting part at term.[2]

In 1981, Dr. N.V.Strizkova, chain of department of obstetric and gynaecology, Moscow Institute and Dr. S.M. Petrikovsky, obstetrical professor, university of New York used endoscopic devices to visualize foetuses in the uterus with the cord entanglement during labour.[2]



Recent articles recommend prenatal review of umbilical cord.[2]

In 1988, a study reviewing 1000 births concluded ," there is no clinical pattern and therefore no place of treatment for the obstetrician to save these babies which are killed by their own umbilical cord." ----- Harold Mc Lellan MD, Australia 1988.[2]

In 1982, P. Jouppila MD (dulu, finland) described the ability to visualize the umbilical cord abound the fetal neck and he wrote, Antepartum diagnosis of cord coilings could be of clinical value for the adequate suppression and management of this complication.[2]

In 1989, In a review by Dr Ingo Clauses Prof of obgy University of Arahus, Denmark, structural anomalies of the umbilical cord were mentioned as being diagnosed with ultrasound.[2]

Weber T[40] studied the influence of cord complications on the fetal ph ,neonatal apgar score ,acid base status and oxygenation of the umbilical artery and vein on 152 deliveries and found that the cord complications in general did not influence the apgar scores ,the acid base status or the oxygenation of the fetus and newborn . In very few cases of tight nuchal cord complications ,ph showed a marked decrease during the last 30 minutes of labor,the umbilical blood was slightly acidotic but the apgar scores were normal (8-10 in one and five minutes) after delivery. No cases of prolapsed of cord were found. Cord complications were very common but in most cases completely harmless.[40]



According to Dursun M et al[31], asphyxia is most frequently occured as a result of reduced placental circulation but in rare occasions, it might be due to umbilical cord complications. Cord accidents comprise 5-18% of all fatal foetal asphyxia cases and 10% of stillbirths were due to umbilical cord complications.[31]

## NUCHAL CORD

As quoted by Spellacy[41], incidence of Nuchal cord is 15.8- 30%.[41]

According to Colllins[2], Nuchal cord is a common Umbilical cord accident which occurs in about 15% of deliveries. It is separate from Nuchal loops (coils) which by definition does not encircle the fetal neck (which also occur at the rate of 15%). Nuchal cord can be one 360$^{o}$ encirclement to more than 8 (reported) and has been reported in conjoined twins.[2] In 1816, Thomas Denman (as quoted by Spellacy)[41] ,said that after the head is delivered there is no need to cut a nuchal coil, for the fundus will descend and give a more effective workable cord length.



According to Collins[2], there are 8 umbilical cord types (anatomies/designs). There are 2 types of nuchal cord – type A and type B.[2] Collins J H[2] describes nuchal cord – type A and type B.[2]

According to Collins[2], Type A encircles the neck in an unlocked pattern. Type B encircles the neck in a locked pattern. In a prospective review of nuchal cords, the type B pattern occurred 1 in 50 births. Caesarean section and still birth were associated with type B nuchal cord.[2]

According to Miser WF[42], Nuchal cord is often blamed for problems that are encountered during delivery and is often citied as a major cause of fetal distress and perinatal mortality. However the actual significance that a Nuchal cord has on the outcome of an infant is controversial.[42]

According to Dursun M et al[31], nuchal cord is defined as the umbilical cord being 360$^{o}$ around foetal neck. Nuchal cord is one of the most common umbilical cord

accident and any pregnancy might be complicated with Nuchal cord. It is estimated that Nuchal cord affects 23%-33% of all pregnancies.[31]

According to Collins[2], Nuchal cords are the centre of the supply line controversy because so much differing information exists. In general,the consensus of opinion has been that a nuchal cord is unlikely to compromise a fetus and is rarely, if even a cause of fetal death.[2]

A similar opinion is shared by Torgini Sornes, M.D., dept of obgy, Akershus Central hospital, Nordbyhagen, Norway, 1995, who has studied this issue for 2 decades.[2]

The pathology of umbilical cord encirclements around fetal body, neck or extremities has not been subjected to thorough study for years and little is known of their etiology, pathogenesis or effects on fetus.[2]



According to Miser WF[42] nuchal cords are rare before 20 weeks of gestation, mostly because the umbilical cord is shorter than the fetal body till then, and so has no opportunity to wrap around the neck or head.[42]

As quoted by Begum AA et al[97], Walker and Pye found male babies had longer cord than female babies and it was assumed to be due to higher level of intrauterine activities of male fetus.[97] Similar findings are shared by Begum AA et al[97] and Rhoades DA et al[88].

According to Collins[2], Larson JD[44], Rayburn WF[19] the pattern of most nuchal cords >80% are wrapped right to left around the fetal neck. Most torsion >70% is counter clockwise (sinister, left handed) away from the fetus. This suggests that the fetus when stimulated manoeuvres in the same direction most of the time. The fetus can

develop entanglement and escape from it. It is possible for a 20 week fetus to free itself from the loops by 28 weeks.[43,44]

According to Collins[2], cords can wrap around the fetal neck as soon as the cord reaches 4/5$^{th}$ of the fetal length. This occurs as early as 10 to 12 weeks of gestational age.[2]

As quoted by Begum AA et al[97], Crawford in 1962 first defined nuchal cord as the cord in which umbilical cord was round atleast once around the neck of the foetus.

There are two patterns for the formation of nuchal cord,either the cord crosses over itself or it crosses under itself—this pattern is needed for knot formation and can cinch a nuchal cord if it rolls back on itself.[2]



A very active fetus, especially one with a high level of amniotic fluid volume and a high level of energy derived from placental design may be predisposed to nuchal cord formation.[2]

According to Larson JD[44], Rayburn WF[19] about 28% of all pregnancies have a nuchal cord and off these, 3.7% have two or more loops. The farther along in a gestation a delivery occurs, the higher the probability of a nuchal cord being present- from around 10% at 24 weeks to around 18% at 32 weeks and around 30% at term following an almost perfectly linear distribution and coils may form shortly before delivery.[44]

Collins[2] says, if the umbilical cord gets overly stretched or compressed during labour, it usually causes the baby's heart rate to slow down temporarily. This is the baby's reflex response to less blood flowing back to its heart. These brief variable heart rate decelerations are not harmful but if it slows to below 100 beats per minute

and does not return to normal within a few minutes, measures should be taken to relieve the presumed cord compression. These include giving oxygen and fluid to the mother and changing her position. If there is still concern about pressure on the umbilical cord, a caesarean delivery may be needed.[2]

According to Collins, one of the concerns that can increase the risk of cord problems is decreased amniotic fluid. This doesn't allow for the free movement of the baby and the cord, on and off each other, raising the possibility of a cord accident.[2]

According to Collins, during labour, the only indication of the umbilical cord being wrapped around the baby may be variable fetal heart decelerations on the fetal monitor. These are generally timed with contractions as at that time the cord is stretched more tightly.[2]



According to De Haan HH[46], nuchal coiling can occur in shorter cords, in which ,it tends to be more tightly wrapped around the infant's neck.Greater than 50% interruption of umbilical blood flow is significant for creating fetal hypoxia. Sustained or repetitive compressions eventually lead tofetal compromise. Occlusion of the uterine artery has similar effects on the fetus with significant differences on the fetal heart and brain. Combined umbilical cord occlusion and uterine artery occlusion has effect on fetal organs and metabolism.Fetal heart rate changes do not reflect cardiovascular deterioration during brief repeated umbilical cord occlusions in near term fetal lambs.[46]

According to Collins[2], interruption of uterine blood flow of more than 50% is significant for creating fetal hypoxia.[2]

Usually a one time, one min 100% compression of the cord takes 5 minutes to completely correct. But within that one minute, oxygen levels have decreased 50% and the fetus must reset the valuable energy and chemistry it has expended. In a recent experiment complete cord compression every 5 minutes required 30 minutes for recovery. Continued 5 minute, compressions every 30 minutes caused fetal decompensation.[2]

According to Weber TF[40], Hankin GD[47], Synder RR[47], Clap JF[48], Nelson KB[49], cord compression, whether chronic, intermittent or acute, ultimately stimulates the fetus to shunt its blood flow, vasoconstrict its extremities and protect itself through a centralized circulation. (heart,adrenal,brain ), baroreceptor and chemoreceptor responses occur with release of catecholamines, cortisol, vasopressin, angiotensin and other biochemical agents to initiate a foetal response to developing hypoxia. Fetal metabolism of glucose and gluconeogenesis are induced by cord compression. Arterial lactate elevations may be a measurable result of umbilical cord compression. These protective steps over time can give way to bradycardia, vasodilatation, foetal hypotension, acidosis, depletion of glycogen stores and blunting of the cortisol response. Eventually foetal compensation fails and peripheral vasodilatation occurs with heart failure, arrhythmias and foetal death.[40, 47, 48, 49]



In an intermittent cord compression (one complete occlusion every 5 minutes), sheep model with term foetuses, foetal collapse occurred in 45 minutes to one hour. In a model ,of 5 minutes complete cord compression repeated every 30 minutes, the foetuses died after 3-4 occlusions. Clinical signs which may be present, given the biochemical status of the foetus at risk of umbilical cord compression are hiccups, hyperactivity, decreased foetal movement and foetal heart rate changes. This acidosis is of a mixed (68%) or a respiratory (23%) type and is corrected quickly

by prompt ventilation of the newborn. Thus, though the nuchal cord group did tend to have a larger percentage of infants born with an apgar score of less than 7, nuchal cords are not major causes of foetal asphyxia.[40, 47, 48, 49]

Both support that, nuchal cord associated with meconium or an abnormal fetal heart rate pattern, and one that has multiple loops or is extremely tight may cause a subclinical deficit in neurodevelopment performance or unexplained spastic quadriplegia.[40, 47, 48, 49]

Spellacy[41] found nuchal cord to be a common cause of foetal distress, as manifested by changes in foetal heart rate pattern. As quoted by Spellacy[41], Browne demonstrated simple in vitro experiments that one simple coil of the cord produces an increase in resistance to blood flow and increased incidence of fetal heart rate abnormalities .[41]



According to Spellacy[41], the presence of meconium staining of liquor increases 2 to 7 times more often with nuchal coils. A low apgar score has been found in those infants who had nuchal coils. The Nuchal coil is reportedly, however, a rare cause of infant mortality.[41]

To assess the significance of nuchal cord, Hankins, Synder et al (1987)[4] analysed 110 affected women- infant pairs at term. They selectively analysed umbilical cord, pH and blood gases to document the metabolic status of foetus at birth. They evaluated the association between nuchal umbilical cord and the incidence of abnormal intrapartum foetal heart rate patterns, abnormal umbilical cord ph blood gas determinants and early neonatal morbidity.

According to Hankins,Synder et al (1987)[47] ,newborns with a nuchal cord had a increased prevalence of umbilical cord acidemia and more variable foetal heart rate deceleration in the first stage of labour. In newborn with a nuchal cord the umbilical cord acidemia was usually mixed 68% case, or respiratory in origin (23%) and pure metabolic acidemia was infrequent (9%). It can therefore, be said that nuchal cord are associated with an increased prevalence of foetal heart rate deceleration in the first and second stage of labour and with an increased incidence of umbilical artery acidemia.[4]

In modern obstetrics, Kan Pan Shui & Eastman[50] published the first well designed study about the nuchal cord and its perinatal effects in 1957. In this study the incidence of one and two nuchal loops around the foetal neck was 20% and 2.5% respectively and the authors could not demonstrate the relationship between the presence of nuchal cord and perinatal mortality. Shui and Eastman[50] support the view that nuchal cord around neck is a cause of foetal death.[50]



Larson et al[44] reported that the presence of multiple nuchal cord entanglement was significantly associated with abnormal foetal heart rate pattern during labor $p < 0.001$ and that infants were more likely to have meconium , $p = 0.013$ with low 1 min Apgar score $p<0.001$ and low umbilical ph ≤7.10.[44]

As quoted by Onderoglu et al [31] ,Martin et al also reported the increased prevalence of acidosis in newborns with nuchal cords [31]

Clapp et al[48] suggested that the presence of tight or multiple nuchal cords might be associated with the development of subclinical deficit in the neurodevelopment performance.

Nelson and Grether[49] also reported that there may be an association between tight nuchal cord and unexplained spastic quadriplegia.[49]

However, (As quoted by Onderoglu) [31] ,Greenwood and Impey could not demonstrate an association between cerebral palsy and pregnancies complicated with multiple nuchal cords.

Hankins et al[47] analyzed 110 affected women – infant pairs at term. They selectively analyzed umbilical cord, ph and blood gases to document the metabolic status of fetus at birth. They evaluated the association between nuchal cord and the incidence of abnormal intrapartum fetal heart rate patterns, abnormal umbilical cord ph blood gas determinants and early neonatal morbidity and he reported that newborns with nuchal cord had an increased prevalence of umbilical artery acidemia and more variable fetal heartrate decelerations in the first and second stages of labour.[47]



Fortunately the coiling of cord around neck is an uncommon cause of antepartum fetal death or neurological damage (Clapp and colleagues, 2003, Nelson and Grether, 1998).[48,49]

As reprted by Hankin GD, Synder RR, Hauth JC, et al [47,73] ,Since entwined cords, however may cause intrapartum complications, As labour progresses and there is fetal descent, compression may compress the cord vessels. This causes fetal heart rate decelerations that persists until the contractions ceases. In labour, 20% of foetuses with nuchal cord have moderate or severe variable heart rate decelerations, and they also are more likely to have a lower umbilical artery pH .

According to Collins[2], nuchal cords have been observed to be predisposed to anemia. Not all nuchal cord births are the same, but four tight nuchal cord coils seem predisposed to produce anemia in the newborn. In one study 18.5% of nuchal cord births were anaemic.[2]

Collins JH, Geddes D, Collins CL, De Angelis L (J la state medical society, 1991 did a study to find the association between placental location and nuchal cord incidence and concluded a significant relationship between the two.[51]

Heifitz SA[27] reports incidence of nuchal loops-20- 25% and reported them to be assosciated with cords of excessive length and male foetuses.[27]



As quoted by Williams[30], tight nuchal cord can lead to fetal demise by cord compression and obstruction of jugular venous return , congestion of cerebral and meningeal vessels and intracranial haemorrhage . A cord tightly entangled about the neck or other body part may leave a recognizable mark on the skin, an increased incidence of neonatal anaemia and instances of hypovolemic shock with nuchal loops may be due to decreased fetoplacental transfusion by relative compression of umbilical vein.[30]

In 1995, Dr Torginn Selnus (Akerhsus central hospital Norway), suggested that, there is an association between cord encirclement and foetal heart rate deviation independent of cord length ,and an intense follow up should be done on these foetuses[2]. In relation to these a study was done in 1978 by Dr John Rolshau Odensa, Denmark, 1978. It has been documented that a thin umbilical cord is

associated with low birth weight . Also the RNA/DNA ratio was raised, indicating tissue stress( in the placenta) in case of battledore insertion of cord.[2]

In a study by Kumari et al[89] who did a prospective study on 12000 singleton pregnancies , of which 258 had cord abnormalities (2.15%) . Nearly 32% of these cases had fetal distress and 20.15% had 1 min apgar score of <6 . Perinatal mortality rate with cord problems was 85.27/1000 births . Neonatal problems noted were septicaemia (4.56%), aspiration syndromes (13.48%) , hypoxic ischemic encephalopathy (7.30%) , neonatal convulsions (2.14%) and concluded that these parameters are significantly affected by tight nuchal cord.[89]

Gregory C Martin ,Robert S Green ,Ian R Holzman[95] did a study to compare Apgar scores and cord blood gases between a group of babies with nuchal cord and without nuchal cord and found that median Apgar scores in the nuchal cord babies were 9 and 9 at 1 minute and 5 minutes respectively, which did not differ from normal infants. [95]



Kitagawa M in 1989 (as quoted by Begum AA et al)[97], used analysis of umbilical cord gases and CTG (Cardiotocograph) and found a positive relation of variable deceleration, and a low apgar score and significant respiratory acidosis in cases with nuchal cord.

William F Miser[42], did a study on 765 infants to note the incidence of nuchal cord and neonatal morbidity and mortality associated with it from June 1986 to December 1986 and found 167 case of nuchal cord while rest 523 served as the control group.

Sample – 765

Incidence – 23.7%

Incidednce of one coil- 21.7 %

Incidence of two coils -1.7 %

Incidence of three coils- 0.3%

Result of his study was that vaginal deliveries were more common in the nuchal cord group with no statistical differences in the frequency of primary caesarean sections and operative deliveries. There was no significant difference between the mean of both the 1 min and 5 min Apgar scores(8, and 9 ) respectively, between the two groups, but infants born with nuchal cords tended to have lower scores at 1 min,and the percentage of infants born with an initial Apgar score of <7 in the nuchal cord group was at most twice that of infants in the control group ( 8.4% and 4.8%) respectively. This trend was was not evident in the 5 min apgar score. There was only 1 stillbirth( incidence- 0.1%) which occurred in the control group.[42]



According to a study done by J.D. Kemfang Ngowa et al[94], who studied 9275 deliveries in a retrospective descriptive study to analyze the perinatal outcome and incidence of nuchal cord at delivery – found nuchal cord in 16.2% of cases, of which 75.81% were loose and 24.18% were tight. He reported that Caesarean section rate was lower in the nuchal cord group when compared to control group. Apgar score <7 at 1 and 5 min was less in the loose nuchal cord group as compared to control group. In the tight nuchal cord group, low Apgar scores at <7 at 1 min was significantly higher when compared to control group. Transfer rate to Neonatal Intensive Care Unit was lower in the nuchal cord group as compared to control group. However, he reported that tight

nuchal cord may be associated with increased risk of low Apgar score <7 at 1 min.[94]

According to a case control study done by KK Dhar et al[87] on 3580 deliveries at PGI Chandigarh – perinatal events of 180 babies with nuchal cord over 1 year were studied. The incidence found was 5.74% with more than 1/3 i.e. 39% having Tight nuchal cord and 61% having Loose nuchal cord. Babies with Tight nuchal cord were more frequently met with fetal distress (51%), prolonged 2nd stage (11%), non toxaemic accidental haemorrhage (7%) and operative delivery (56%) when compared with babies with Loose nuchal cord and controls (without Loose nuchal cord). Nearly 1/3 (24%) of babies with Tight Nuchal Cord were born small for date, had birth asphyxia (61%) and 8.5% died during perinatal period. Perinatal outcome was adversely affected by Tight Nuchal Cord in comparision to Loose Nuchal Cord and controls.
He also quoted in his study that several studies have incriminated nuchal cord as a cause of increased incidence of fetal distress, prolonged labour, operative delivery and perinatal mortality.[87]



However as quoted by Dhar KK et al [87] ,the study of Mc Lenan et al and Morwitz et al did not reveal any adverse effect of nuchal cord on perinatal outcome[87], maybe because these authors did not attempt to assess the effect of tightness of nuchal cord on perinatal outcome.
As quoted by Dhar KK et al[87] Weiss et al found that an acute resistance or mechanical occlusion of the umbilical cord produces an alteration of flow curves, from normal to absent to reverse diastolic flow for about 20-25

seconds with ultrasound Doppler. Similar findings are likely to be observed during intrapartum period in cases of Tight Nuchal Cord. Antenatal Ultrasonography search for umbilical cord around neck followed by Cardiotocograph monitoring during labour can significantly reduce perinatal mortality and morbidity.

According to a study done by Spellacy et al[41] on 15,709 deliveries found nuchal cord to be present in 4237 cases, i.e. 24.6%, true knots in 180 cases i.e. 1% and body loop in 339 cases i.e. 2% and reported that these three complications occur atleast once in every 4 deliveries and their problems cannot be avoided. Out of the various factors he analyzed ,he found that only cord length was statistically significantly associated with higher degree of cord complications. Also he quoted that malpresentations of infants has been attributed to these cord complications. But in his study, he found fewer nuchal coils in the breech presentation group (13.7%) than in the vertex presentation group (25.5%). The incidence of breech presentation was reported to be higher in the group with 2 or more nuchal coils. His study not only disproved the above hypothesis but also suggested that a vertex presentation predisposes to the production of nuchal coils and possibly that the presence of two or more coils could predispose to a breech presentation. He reported that neonatal distress in terms of heart rate <100/min at 1 min of age was higher in all the three cord complication groups. He also reported 1 min Apgar score to be significantly lower in the cord complication group as compared to control and in Tight nuchal cord group as compared to Loose nuchal cord group and no difference between the two at 5 min Apgar score and quoted

Hon's finding that this may be due to more vagotonia produced by tighter coils around neck, and finally reported that results of cord complications are transient.

He also reported a review of 1 year neurological complications in the nuchal coil group to study mental deficiency and cerebral palsy as caused by birth asphyxia due to nuchal coils as reported in literature but did not find this theory correct, but he quoted that further follow up examinations will be needed before any such definitive statement can be made.

In a study by Shui K.P, Eastman MJ et al[50], reported a significant association between number of nuchal loops and cord length, but also stated that this phenomenon can also occur occasionally with short cords. He also stated that still births and neonatal death rates were higher in the coiled cases than in the non coiled and the conclusion is that coiling around foetal neck even in cases of short cords is rarely a cause of perinatal death.



In a cross sectional prospective study done by Begum AA et al[97] on 1646 deliveries, 152 cases of nuchal cord were found during a study period of 2 years in a Military hospital in Bangladesh. They found no increased incidence of operative delivery in nuchal cord group. Tight Nuchal Cord group neonates had significantly low Apgar scores at 1 min as compared to Loose Nuchal Cord group and significantly low Apgar scores at 5 min in babies born with multiple nuchal cords. He also quoted that although the newborns with cord

encirclement required resuscitations, foetal or neonatal death could rarely be attributed to the nuchal cord.[97]

As quoted by E. Assimakopoulos[86] the clinical significance of nuchal cord is controversial. Although in many cases , it does not seem to do any harm but it has been reported to be associated with increased risk of variable decelerations in both first and second stage of labour , acidemia, significantly higher incidence of low 1 min Apgar score , meconium stained amniotic fluid , emergency caesarean section , need for neonatal resuscitation and admission to Neonatal Intensive Care Unit and possibly perinatal death.[86]

## NUCHAL COILS

According to Reiss (1958)[52], the coiling of the umbilical cord around the neck is a common complication of labour and occurs once in about 5 deliveries. Fetal asphyxia, deflexed attitude and malpresentations are sequels of this complication.[52]

Shui and Eastman[50] found the occurrence of one nuchal cord to be 20.6%, two coils to be 2.5% and three coils to be 0.5%.[50]

According to Mc Caffrey (1927)[53], Gray reported a case with 9 coils around the neck and Crede one with 8 coils around the neck. [53]

According to Kashianan M, Akbarian A, Koohpayehzadeh J[55]. did a study on umbilical coiling index and adverse perinatal outcome .A prospective study was performed on 699 pregnant women who were 37-40 wks .Umbilical coiling index was determined by dividing the total number of complete vascular coiling by the total umbilical cord length in centimetre. Then the relationship between umbilical coiling index and neonatal weight, amniotic fluid index , meconium, Apgar score and fetal distress was evaluated. It was found that there was a significant difference between normal and hypocoiled group in relation to to Apgar score <7 in 5 min and AFI(Amniotic fluid index) ≤5 and between normo and hypercoiled group in relation to to Apgar score <7 in 5 min and AFI (Amniotic fluid index) ≤5, meconium and foetal distress . Neonatal weight was higher in the hypocoiled group as compared to the hypercoiled group . Their conclusion was that abnormal Umbilical cord insertion may relate to adverse perinatal outcome.[55]

Ogueh O , AL- Tarkait A, Vallerand D, Rouah F[56] have reported umbilical cord nuchal loops to be associated with induction of labour ,slow progress of labour and shoulder dystocia.[56]

Larson JD, Rayburn WF[44] have reported multiple nuchal cord entanglements to be associated with greater risk of meconium, abnormal foetal heart rate patterns during advanced labour , but no added risk of adverse neonatal outcome.[44]

Onderoglu LS, Dursun P , Durukan T[31] found out that the umbilical cord blood ph-7.32 and oxygen saturation were significantly lower in nuchal cord group compared with controls. The presence of a single nuchal cord may negatively affect the umbilical cord blood gases without significant perinatal complications. However, multiple nuchal cords may increase the development of intrapartum complications and low apgar scores.[31] Schaffer L ,Burkhardt L, Zimmermann R[57] found incidence of nuchal cords in term pregnancies as 33.7%, multiple nuchal cords as 5.8% .He found unfavourable neonatal blood gas values in the nuchal cord group but 5 min Apgar score <7 , and admission to Neonatal Intensive Care Unit was not found more frequently in the nuchal cord group. According to Jauniaux E, Ramsay B, Peellaerts C, Scholler Y[92], multiple looping of the umbilical cord around neck was the main factor accounting for the higher incidence of complications like higher incidence of Apgar score<7 at 1 min , meconium stained amniotic fluid , emergency caesarean section , need for neonatal resuscitation , admissions to Neonatal Intensive Care Unit and perinatal deaths in the nuchal cord group as compared to the control group.[92]

However, the insufficient data as regards the role of nuchal cord in foetal morbidity and mortality is a source of anxiety and frustration to both parturients and health care professionals.[95]

## CORD LENGTH AND UMBILICAL CORD ACCIDENTS

As quoted by Spellacy[41], Napier in 1882 found that cords vary from 5-175 cm with an average length of approx 50 cm. After 28 weeks of gestation the cord does not appear to lengthen significantly. Also Spellacy[41] ,quoted that males reportedly have slightly longer cords than females.Also in his study, on 15,709 deliveries to study 3 main umbilical cord complications i.e. nuchal coils, body loop and true knots analyzed various factors and reported a significant association between cord length and higher degree of cord complications .Spellacy[41] reported a significant correlation between them. He also reported significantly increased proportion of tight coils in the shorter cord length group.



According to Shui KP and Eastman NJ[50] – a certain correlation exists between average cord length and the number of coils around the neck. They stated that as demonstrated by standard errors, the increased cord length in the “one coil” group over the length in the “no coil” group is unquestionably a valid observation, and there are 7 chances in 8 that the next difference shown, between cord length in the “one coil” and “two coil” groups is not a sampling error . He also stated that nuchal coiling can occur occasionally with cords of short length.

According to Sarwano ,Disse et al [98] ,the risk of complications increase with increasing cord length.The umbilical cord length increases progressively from a mean of 32 cm at 20 weeks to 60 cm at term.(Naeye R.L.)[107] . According to Rayburn[66] , Umbilical cord accidents are mostly associated with a long cord . Almost 62% of all cord accidents are associated with a long cord.[66] Due to extreme variation in umbilical cord length it is difficult to define the limit that should

be exceeded for the cord to be considered as unduly long. According to Purola[108] , 100 cm was the maximum normal length and found only 0.5% of cords to exceed this length. But according to a study by Begum A A[97]et al, no statistical difference of mean cord length was found in cases with and without nuchal cord.

## BODY LOOP/ EXTREMITY LOOP

Spellacy, Gravem et al[41] (1966), cord around body occurred in 2% of cases[41], associated with long cords and its significance is unknown.

The potential dangers of coiling around the body are presumed to be the same as in other cord complications.(Kasturi lal 1970)[75]

Avert (1957) [8]discussed the risks of coils around the foetal body leading to its intrauterine death.[8]

But Shun and Eastman (1957)[50] stated that the lethal role assigned to cord coiling has been thought to be the cause of foetal death in umbilical cord coiling.[50]



According to Collins[2], whether such a mechanism of vascular spasm plays any part is doubtful. Probably it is the risk to the foetus due to mechanical interference with the placental circulation as a result of tightening caused by free movements of the limbs.[2]

According to Collins[2], body loops can be single or multiple and can exist with nuchal cords. The number of body loop incidences is unknown. Most of these entanglements are undone at delivery as the infant is being born, therefore, they are never witnessed and never recorded.[2]

The effect of a body loop is cord compression. Tight loops have made impressions on the skin of the foetus and can restrict foetal movements in utero. Loops around the extremity can affect circulation of the extremity and cause damage to foot or

hand. These events can change the oxygen supply to the fetus and cause growth disturbances or death.[2]

The Collaborative Perinatal Project (CPP) reports no ill effects from body loop entanglement.[2]

Still births are observed with multiple loops around ankles, necks and bodies, yet it is difficult to determine which compressed segment causes death.[2]

As quoted by Collins, Chinese and Russian medical literature tends to suggests that loops are benign. Body loops can act like wenches, taking upslack and causing relative shortness of cord. This shortness may lead to placental separation, and in one report amniotic fluid embolism.[2]

## TRUE KNOTS

Knots of the umbilical cord are classified as true or false. The false knots consists of simple dilatations of the umbilical vessels that look like knots but are considered devoid of any clinical significance. True knots are formed when the foetus passes through a loop of umbilical cord.[2]

As quoted by Collins[2], factors predisposing to true knots are commonly believed to include monoamniotic twins, long cords and polyhydramnios. It seems logical that knots are formed in early pregnancy, when it is possible for the foetus to go through a loop of umbilical cord. Tightening of the knot can occur during labour. Knots can be simple or multiple.[59,60,61,62,63,64,65]

According to Rayburn et al[66] and Fox Harold[28], the incidence of umbilical cord accidents, i.e. nuchal cords, cord prolapse, torsion and true knot formation are more frequently associated with excessively long cords.[66,28]



Williams[67] says, several mechanical and vascular abnormalities of umbilical cord are capable of impairing foetal placental blood flow of which important ones are knots.[67]

True knots result from active foetal movement.[67]

In nearly 17,000 deliveries, in the collaborative study on cerebral palsy, Spellacy and co-workers (1966) found an incidence of true knots of 1.1%[41]. The incidence is especially high in monoamniotic twins. Venous stasis may lead to mural thrombosis and foetal hypoxia, causing death or neurological morbidity.Collins and Collins (2000) reported a 6% incidence of still births when true knots are found.[67] (As quoted by Williams)[67]

The exact cause of formation of true knot is not known. It is found in association with long cord, polyhydroamnios, a small infant, monoamniotic twins, overactive foetus or as a result of external version.[28,68]

Chasnoff, 1977[60] says, the pathogenesis of true knots remains obscure since the formation of a true knot could not be observed, studies on true knots were done either retrospectively or done on artificially made knots in intrinsically normal cords.[60]

As quoted by Spellacy[41], though knots usually lack clinical significance, they may if pulled tight, produce foetal asphyxia. The literature contains several case reports of cord knots and associated foetal death in utero.[41]

Spellacy[41] found that one min Apgar scores showed significantly lower scores with all the cord complications like body loops, nuchal cord and true knots at all birth weights, while the 5 min apgar scores did not show any difference between the cord complication group and the control group[41] and so radical obstetric management of these complications seems unjustified. He also stated that there is an appreciable increase in the still birth rate with true knots, and he stated that true knots are indeed related to foetal death.[41]

Browne,1925 [29]showed that in vitro conditions, a progressive tightening of a knot was accompanied by an increasing resistance to infection through the umbilical cord. It seems, therefore, that a tight knot can obstruct the foetal circulation through the cord, and it is not surprising that the presence of such knots is associated with a higher perinatal mortality. But this view of Browne was not agreed upon by all.[29]

Chasnoff [60]challenged Browne's results and found that a loose knot did not affect the venous perfusion contrary to the information available in literature because he found

that a loose true knot did not affect the venous perfusion pressure, and observed that with a tightened knot, the smaller the cord's diameter, the greater was the pressure required to perfuse past the knot. It is important to note that the Chasnoff's[60] elegant study was performed on artificial knots in intrinsically normal cords. The examination of the knot after the delivery of the foetus cannot tell a tight knot from a loose one as traction of the umbilical cord at the actual delivery changes considerably with the intrauterine degree of tightness.[60]

The umbilical vessels, protected by myxomatous structure of wharton's jelly, are rarely completely occluded. These findings correlate clinically with the relatively high incidence yet low mortality rate actually due to a true knot in the umbilical cord.[6]

Spellacy quotes Bandelcorque (1978)(As quoted by Spellacy)[41] as having reported a case of a newborn infants with 3 knots who survived.[41] In 1875, Chatreuil (as quoted by Spellacy) postulated that true knots formed between 9$^{th}$ and 12$^{th}$ weeks of gestation, because foetus was the most active and there was a relatively large amount of amniotic fluid.[41]



According to Fox[28] and Hytrl[69] in 1870 postulated that the time of formation of knot could be determined by the site of the knot on the cord, thus closer the knot to the foetus, the earlier it was formed.

But Klickstein, Schwartz et al[70] did not agree with this theory.[70]

According to Rayburn, Benyen et al[66] the incidence of true knot in a long cord is 3 times as that in a short cord. In their study of 536 cords they found that in cases with short umbilical cord length (<35 cms) the incidence of true knots was nil, whereas in case of long umbilical cord >80 cms, the incidence was 3%.[66]

According to Klickstein, Schwartz et al[70] the sequence of events in true knots formation could be envisioned as-during an uneventful pregnancy (probably in a multiparous gravida with a spacious uterus) ,there is a moment, when cord length is sufficient to form a loop,the diameter of which is large enough to allow the foetus to pass through it.

Heifitz SA[27] found true knots in 0.5% cords but are more common among male foetuses, monoamniotic twins, multiparas. They are associated with overall mortality rate of 8-11%.[27]

Heifitz SA[27] says, excessively long cords are apt to become knotted. True knots must be distinguished from false knots that are not associated with cords of excessive length.[27]False knots are focal nodular congestion of branched ecstatic vessels, varicosities, localized vascular loops (redundancies) or focal accumulations of wharton's jelly. They are usually of no clinical importance, but thrombotic occlusion rarely results in foetal death.[27]



According to Heifitz SA[27], true knots- 0.5% of cords but are more common among male foetuses, monoamniotic twins (intertwining), multiparous women, hydramnios, foetal growth restriction or long umbilical cords. They are formed as a result of the foetus moving through a loop of cord during its activities in the uterus, the vast majority must form early in gestation except with monoamniotic twins, knots rarely tighten before labour such may do so intrapartum, during foetal descent at delivery.[27]

According to Collins[2], even a slack knot maybe sufficient to interfere with, if not completely obstruct the cord circulation but that any pull upon the knot such as might be exerted if the cord was found wound around the child's neck or body as to cause a relative shortening of it would easily cause sufficient tightening to impede

the circulation completely- Francis J Browne MD, Edinburgh Royal Maternity Hospital, Great Britain, 1923.[2]

Reports of infants born with more than one knot and as many as 3 imply that foetal activity and repositioning are key factors. The excessive length increases the probability of knot formation but it is not necessarily deadly. Increased slack decreases the chance of tension, cord architecture is also a factor.[2]

A deadly combination is a cord under torsion with a shoulder loop, nuchal cord, body loop, or extremity loop.[2]

Hershovitz[18] reported an incidence of true knot as 1.2% (841 knots/69,139 births) in a large study.[18] the Collaborative Perinatal Project(CPP)[2] for comparison reported an occurrence of 1% in 55,908 births. The practical meaning of these statistics is that a true knot will be encountered once or more each if by the obstetrical care giver. With a 1.7%-10% mortality risk in singletons, (>50% mortality risk in monoamniotic twins), all true knots should be taken seriously.[2]



True knots are the result of type B nuchal cords, and when considered as such (1type BNC/50 NC births), the risk of foetal compromise is increased.[2]

Cord knotting may occur as a result of delivery (a type B loop being pulled off its neck, waist or extremity position). The hockle of umbilical cord engaged by over the foetal body with time and creates a true knot. Double and complex knots are formed based on the number of twins in hockle and number of hockles. A cord with a double turn hockle can become a double knot. These events probably takes days to weeks. Evidence of Foetal Heart Rate changes, umbilical blood flow restrictions, decreased foetal movement and hiccup has been reported with cord knotting.[2]

True knot has been prenatally identified with ultrasound and managed based on fetal heart rate, tachycardia and decreased foetal movement.[2]

## UMBILICAL CORD PROLAPSE

Umbilical cord prolapse is a rare obstetric complication usually necessitating emergent delivery. Three types of Umbilical cord prolapse are described:[100]

1. Overt umbilical cord prolapse.
2. Funic (cord presentation)
3. Occult cord prolapse. (Overt)

Umbilical cord prolapse occurs after the membranes are ruptured and is defined as the descent of funis through the cervix into the vagina or on to the vulva.[100]



Funic presentation is the presence of one or more loops of umbilical cord between the foetal presenting part and foetal membranes overlying the cervical os[100].

Occult cord prolapse occurs if the cord is alongside the presenting part and is palpable only by passing the examining finger into the cervical canal. [100]

As quoted by Monique G.[100], the incidence of Umbilical cord prolapse reported in literature ranges from 0.1% to 0.6% and has remained constant over the last century.The most serious complication of this condition is prolonged cord compression from either mechanical occlusion (foetal head) or vasospasm (from the comparatively cooler temperature in the vagina) leading to perinatal Hypoxic Ischemic Encephalopathy[100].

As quoted by Monique G, [100]In the early to mid 1900's, the foetal mortality ranged between 32% and 47% Since then mortality has decreased significantly both as a result of more liberal use of caesarean delivery and improved neonatal resuscitation and care. In the last two decades, perinatal mortality rates reported in association with umbilical cord prolapse have decreased to less than 10%.

Critchlow and colleagues using Washington state Birth Record Data, conducted the largest case control study to date (n = 709 cases) designed to explore the association between various antenatal factors and cord prolapse. They found clinical cord prolapse at the time of delivering a low birth weight infant. After controlling for birth weight, breech presentation, second born twin and male gender remained significant risk factors.[100]



These factors have been consistently associated with umbilical cord prolapse in other case control studies and numerous large case series.[100]

The incidence of umbilical cord prolapse according to the type of malpresentation observed has been described in some studies. The incidence of umbilical cord prolapse in breech presentation ranges from 4% -6% and 7%-15% for those with a fetus in transverse lie at the time of delivery. The risk of umbilical cord prolapse is lower with a complete breech than with a footling or compound breech.[100]

**According to Monique G[100], Risk Factors for Umbilical Cord Prolapse are;**

| Risk Factors | Present Incidence in Umbilical cord Prolapse |
|---|---|
| Maternal factors | |
| Grand multiparity | 8-27% |
| Abnormal pelvis | 6-30% |
| Foetal factors | |
| Abnormal presentation | 35-50% |
| Twin gestation | 7-23% |
| Male foetus | 58% |
| Birth weight < 1500 g | 10-13% |
| Prematurity ( birth < 37 wks gestation) | 19-57% |
| Polyhydramnios | 3-5% |
| Obstetric manipulation | 8-15% |
| Amnitomy | 10% |
| Placement of internal monitors | 24% |
| External cephalic version | 2-8% |
| Manual rotation | 1% |
| Other | |
| Abruption | 7-11% |
| Previa | 3-5% |
| Abnormal(> 80cm)long umbilical cord | 23-32% |

As quoted by Monique G[100] , Kahana et al in a large case control study (n =456) reported a higher incidence of polyhydraminos, grand multiparity, labor induction and gestational diabetes in women with umbilical cord prolapse compared with those without it. The association of multiparity was also observed in another case control study. In contrast Critchlow et al did not identify an association between umbilical cord prolapse and either multiparity or polyhydraminos even after controlling for birth weight.[100]

Of the situations that may be avoidable, some have urged that obstetric manipulation such as amniotomy, amnioinfusion, labor induction and external cephalic version may increase the risk of umbilical cord prolapse.[100]



The pathophysiology of umbilical cord prolapse posited by Clark has been that normal feto placental circulation protects the cord from prolapse by maintaining stiffness through turgor pressure. However after repeated or prolonged cord compression it can more easily prolapse[100].

More, recently Mc Daniels et al argued that acidemia is the cause and not the effect of cord prolapse. In contrast to Clark who argued that a stiff cord is less likely to prolapse, Mc Daniels posited that the increased rigidity leads to decreased buoyancy and predisposes the cord to prolapse.[100]

In the early 1900's when caesarean delivery was avoided if at all possible because it was a very morbid procedure, maternal position change (deep Trendelberg or knee chest position) with funic reduction and vaginal delivery was the preferred technique

for management of umbilical cord prolapse.[100] ,if the cervix was incompletely dilated, duhrssen incisions or the use of an inflatable bag to dilate the cervix was advocated to expedite delivery. Funic reduction was accomplished either manually or by use of a bougie.[100]

A more innovative procedure, Braxton Hicks version (internal podalic version) was used as a treatment for recurrent prolapse.[100]

Fortunately these aggressive procedures have been abandoned in modern obstetrics.[100]

Among large case series, cord prolapse occurring outside the hospital is consistently associated with a high perinatal mortality (38-44%).[100]



At present the mainstay of treatment for umbilical cord prolapse with a viable foetus is emergent caesarean delivery or vaginal (if appropriate). The importance of immediate delivery is illustrated by Block et al who reported two case series of umbilical cord prolapse in two different hospitals.[100]

In a retrospective review by Koonings et al[100], 252 cord prolapse cases were identified over a 9 year period at a women's hospital in Los Angeles and identified 89 cases of umbilical cord prolapse who had electronic foetal monitoring before delivery, and concluded that foetuses with a normal foetal heart rate tracing at the time of umbilical cord prolapse diagnosis have a low incidence of adverse neonatal outcome.[100]

Critchlow et al[100] showed that the risks of an Apgar score <3 at 5 minutes and subsequent neonatal mortality are reduced by a factor of 5 and 2.5 respectively,

when the infant is delivered by caesarean compared with nonoperative vaginal delivery.[100]

Lange et al[78] conducted a prospective study demonstrating 9 of 1471 i.e. 0.61% patients with cord presentation. Of the 9 patients-7 were delivered by caesarean section and 2 vaginally. In 4 out of 7 cases delivered by caesarean section , a cord presentation was found. In the other 3, it was suspected. Of the 2 vaginal deliveries, one was a still birth associated with a cord prolapse and the other underwent a spontaneous version with resolution of cord presentation.[78]

According to Mengert and Longwell [79]when umbilical cord length >75 cm in 545 cases, cord prolapse occurred in 20 cases, i.e. 6 times more often than in the entire obstetrics.



Mclaverty[80] quotes the longest cord prolapsed to be 120 cms.[80]

Rhoades[81] reports the same as 135 cm.[81] (as quoted by Munrokerr)

Mclaverty[80] has emphatically stated that “short cords cannot prolapse”. It is virtually impossible for a cord <45 cm in length to prolapse.” This finding was also agreed upon by Rayburn et all[66] and Disse et al[98] who said that the risk of complications increase in parallel with the cord length and that the cord prolapse is associated with adverse postnatal outcome. In their analysis of 536 term deliveries they found no instances of cord prolapse with short cords (umbilical cord <35 cm) while in those with long cords >80 cm, cord prolapsed occurred in 6% of cases.[98]

Dilbaz B, Onderoglu E, Dilbaz s, Oztruk N did a study to utilize infant outcomes and to identify risk factors associated with umbilical cord prolapse and 800 randomly selected controls were reviewed retrospectively.[82]

The results of his study were;

1. Incidence of umbilical cord prolapse = 0.47% (n=80/16,874)
2. Multiparity was more common in patients with umbilical cord prolapse 63.8-49.4% (p=0.014)
3. It occurred in breech presentation in 6 cases. (7.5%) ,and in transverse presentation in 3 cases. (3.8%)
4. Foetuses with umbilical cord prolapse had lower foetal weight<2.5 kg as a significant risk factor.
5. Spontaneous rupture of membranes, bishop's score>8 and polyhydraminos were other risk factors.
6. Newborns born with umbilical cord prolapse had low apgar score <7 at 5 min (6.3-14.1) p=0.0002



The Conclusion of his study was that abnormal fetal presentation, multiparity, low birth weight, prematurity, polyhydraminos, spontaneous rupture of membranes, in particular with high bishop score are risk factors for Umbilical Cord Prolapse.

According to Collins the most unwanted effect of supply line distribution i.e. umbilical cord disruption is still birth and the most dramatic example of this in obstetrics is an emergency called as "prolapsed umbilical cord".[2]

The chance of having a prolapsed cord at birth is 1 out of 239 cases and 1 out of 865 cases. The reported chance of foetal loss from a prolapsed cord is 8.6% to 49% of these prolapsed occurrences .

The Collaborative Perinatal Project (CPP) reports a high frequency of still birth with foetal heart rate ominous recordings in 41% of cases and questionable recordings in 16% of cases. Breech presentation, twins, long umbilical cord and preterm births are common risk factors.[2]

According to Dare FO, Owolbi AT, Fasubea OB, Ezechi OC[76], the incidence of umbilical cord prolapse over a 10 year period was 0.42% i.e. one in 240 deliveries. Incidence is higher among unbooked patients-76.7%. Analysis of 2 out of 60 cases revealed multiparity, unengaged presenting part from Cephalo Pelvic Disproportion, prematurity, prelabor spontaneous rupture of membranes, breech presentation and multiple pregnancy are major contributory factors.[76] Perinatal mortality was – 36.7% in booked cases, as compared to 86.4% in unbooked cases. Caesarean section gave better results except when cervix was full dilated.[76]



According to Levy H, Meier PR[77], the overall incidence of cord prolapse ranges from 0.1%-0.6% and in cases of breech presentation, the incidence is slightly higher than 1.1%[77]

Murphy D Z, Mackenzie I Z[99] reported 130 babies born with identification of cord prolapse from January 1984 to December 1992 and reported 114426 total births. They concluded that cord prolapse occurs with a relatively stable incidence and despite ominous cardiotocograph's, low Apgar scores and acidemia on blood gas analysis, the foetal outcome is not as poor as might be expected and mortality is predominantly attributed to congenital anomalies and prematurity rather than birth asphyxia.[99]

In a large study reported by Collins involving 56,283 births, 132 cases of cord prolapse were documented and outcome included 6 stillbirths, 6 neonatal deaths and one neurological injury.[2]

In a study by Dilbaz B et al[82] on 16,874 deliveries, identified 80 cases of umbilical cord prolapse and studied them retrospectively and found 0.47% as the incidence and that multiparity was more common in patients with umbilical cord prolapse. There were 6 cases of breech presentation and 3 cases of transverse lie. Foetuses with umbilical cord prolapse had lower foetal weight <2.56kg and was found as a significant risk factor ($p<0.001$). Other risk factors found were spontaneous rupture of membranes, bishop score >8, polyhydraminos & low apgar score.

In a study done by Shakeel A Faiz et al[101], 11 cases of cord prolapse were identified in 55,789 deliveries. The incidence found was 1 in 503 cases. 64.9% were in vertex presentation and 35.1% were non vertex presentation including breech and transverse presentation. 6.55 % were vaginal deliveries and 93.5% were caesarean deliveries. Apgar score was < 7 in 39.6% of babies at 1 min and in 4.55 of the babies at 5 min. 36.9% of the babies were admitted in Nicu (Neonatal Intensive Care Unit). There was no perinatal mortality due to shorter duration of diagnosis to delivery interval in their study. Despite emergency delivery, cord prolapse leads to significant perinatal morbidity. They observed a significant association between mode of delivery and diagnosis to delivery interval ($p<0.05$).

In a study of Dufor P[102] et al on 1450 case of cord prolapse ,reported 72% incidence of caesarean deliveries and 28% incidence of operative vaginal deliveries . He reported neonatal mortality in 20/1000 cases and worse neonatal outcome and poor prognosis.[102]

In another study by Traore Y[103] et al in 16,924 deliveries identified 47 cases of cord prolapse in premature deliveries, multiple pregnancies, dystocic presentations and in spontaneous rupture of membranes. 61.7% had caesarean deliveries and 36.3% of neonates died before the 5th min of life concluding umbilical cord prolapse as a grave obstetrical complication that compromises foetal prognosis.[103]

## MARGINAL INSERTION OF PLACENTA

Cord insertion at the placental margin is sometimes referred to as a Battledore placenta. It is found in about 7% of term placentas. (Benirschke and Kaufmann, 2000)[71]. Central and eccentric insertions account for >90%, the rest are marginal and the least frequent is vellamentous.[104]

With the exception o the cord being pulled off during delivery of placenta, it is of little clinical significance.[71]

According to Earn 1951[63] and Fox 1978[28], the incidence varies widely from 1.9% to 7%. It is less common than eccentric cord insertion but is more important.[63,28]

According to Monie (1965)[72] an increased incidence is found in malformed foetuses.[72]



According to Hathout (1964)[73] marginal insertion was more common in abortions.[73]

Fox (1978)[28] quoted Brody and Frenkel 1953 as having found Battledore placenta in cases of premature labour.[28]

Ujavanwah – Akpom and Fox in 1977[74] analyzed 1000 deliveries but did not find an increased incidence of abortion, low birth weight, premature delivery, foetal malformations, foetal hypoxia and intrauterine foetal death in cases of Marginal Insertion of placenta.[74]

Heifitz SA[27] describes umbilical cord insertion at the edge of placenta in 7% of cases. He has described peripheral cord insertion to be associated with discordant growth and growth impairment in twins. The major risk of peripheral cord insertion is rupture of fetal blood vessels at the time of rupture of membranes or during 2$^{nd}$ stage

of labour (ruptured vasa previa). Twisting of vessels at the peripheral insertion site can lead to progressive variable decelerations and foetal acidosis.[27]

According to Collins, there has been considerable debate regarding the pathogenesis of peripheral cord insertions, with numerous investigations with each of the two mutually contradictory theories. Abnormal implantation-“polarity” theory and trophotropism theory i.e.- placental “wandering” towards better perfused deciduas.[2]

The abnormal implantation theory postulates that during nidation of the blastocyst, the embryo rather than facing the endometrium is obliquely oriented towards the chorion leavae.Thus when the vascular stalk develops, it has to seek its connection with the future implantation site by extending its vessels from the embryo to the chorion frondosum, the vessels must therefore become membranous. The trophotropism theory, on the other hand is supported by the higher frequency of peripheral cord insertions in all twin placentas except dichorionic diamniotic twin placentas. When the placenta develops, it sometimes migrates and dissolves from its original site. This sometimes can result in what appears to be a relocation of the placenta. The placental tissue dissolves, leaving a membrane (amnion) remaining which can then be the connection (insertion) site of the umbilical cord. This results in the umbilical cord placental end looking like it is connected to the edge of the placenta (called as marginal or Battledore) insertion.[2]



Also, .Ujvanwah- Akpom & Fox[74] have quoted the factors leading to peripheral cord insertion. According to Von Franque (1900) an Ottow (1922 and 1923),(as quoted by Ujvanwah Akpom and Fox) they have suggested that the abdominal pedicle normally arises from that part of chorion which is richly vascularised; this is usually that in contact with decidua basalis but in some instances, the deciduas capsularis

may, in early stages of pregnancy, be the area of maximum vascularity and hence the pedicle will take origin from the chorion in contact with this. As gestation progresses, the vascularity of the decidua capsularis will dimish whilst that of decidua basalis increases and hence the site of maximum vascularity will shift to that part of the chorion which is in contact with deciduas basalis and from which the definitive placenta will eventually develop; meanwhile however, the abdominal pedicle retains its original position and hence the cord will be inserted peripherally.[74]

As quoted by Ujvanwah- Akpom and Fox[74], Benirschke and Driscoll argue that the cord is originally always inserted centrally but becomes as the placenta expands, peripherally sited as a result of central atrophy and unidirectional lateral growth of the chorion frondosum whilst Monie (1965) and McLennen (1968) have maintained that peripheral insertion is a consequence of an oblique implantation of the blastocyst.[74]



The placental umbilical cord insertion site can be studied with black and white ultrasonography.[2]

To elucidate the effect of position of placental cord insertion on the postnatal outcome, a study was done by S. Pathak et al in 861 unselected women at term with singleton pregnancies. S. Pathak has quoted a term called as “cord centrality Index”(CI). It is a term used to describe how far or close the point of umbilical cord insertion is from the centre of the placenta. The Cord centrality Index is calculated as the ratio of the distance of the umbilical cord insertion from the placental centre to the half of the longest diameter. The greater the Cord centrality Index, the more peripheral the umbilical cord insertion would be with 0 representing absolutely central insertion and 1 representing completely marginal insertion. He quotes

increased incidence of Intrauterine growth retardation, miscarriages, preterm labour, stillbirths and neonatal deaths in cases of Marginal Insertion of Placenta.[104] The distance of the umbilical cord insertion from the centre has been considered as a marker of placental insufficiency.[104]

In a study done by Robinson LK et al[105] to study the structural defects associated with velamentous and marginal insertion of umbilical cord on 4,677 consecutive placentas at the University of California, found 454 marginal insertions and 72 velamentous insertions and reported an increased incidence of structural defects to be associated with velamentous insertion of cord.

As quoted by Junichi Hasegawa et al[106], abnormal cord insertion is associated with IUGR, preterm labour, abnormal intrapartum fetal heart rate pattern, low Apgar scores at 1 and 5 min, neonatal death and Abruptio placenta.[106]



According to J. Hasegawa et al[106], the placental cord insertion site should be evaluated in the mid-trimester as visualization of the placental cord insertion site becomes more difficult with advancing gestation. He also reported that cord insertion site was determined less frequently in the cases of marginal cord insertion

## Material and Methods

A cross sectional study of 300 deliveries irrespective of age, parity and mode of delivery was done from oct 2009- may 2011.

**Exclusion criteria**

- Deliveries with preexisting high risk factors responsible for adverse postnatal outcome such as medical disorder in the mother like Cardiac disorders, Diabetes, Hypertension, Bronchial asthma, and any Renal disorders were excluded from the study.
- Presence of severe IUGR or pregnancy >42 weeks and pregnancy <34 weeks was also excluded from the study.



300 randomly selected deliveries were observed and the presence of various umbilical cord accidents as enlisted in our study and their effect on the immediate postnatal outcome in terms of stillbirths,1 minute apgar score <7, neonates requiring active resuscitation measures like ambubag, endotracheal intubation , chest compressions and use of drugs, NICU (neonatal intensive care unit ) admissions were noted.

The various umbilical cord accidents included in our study were;

1. Presence of nuchal cord.

   The nuchal cord group was further subdivided into 2 groups :

   i. Tight nuchal cord group

   ii. Loose nuchal cord group

Nuchal cord has been defined as one , encircling the neck in a 360 degree fashion.

Tight nuchal cord: was defined as that which was not reducible prior to delivery of neonate's body and cord needed to be clamped and cut before the delivery of baby.

Loose nuchal cord: cord could easily be uncoiled before complete delivery of baby and its clamping and cutting did not differ from those neonates without nuchal cord.

2. Body loop/extremity loop was observed at delivery of baby.
3. Prolapse of cord if any was noted.
4. Then the examination of the umbilical cord was done ;
   i. Length of umbilical cord was noted in centimetres with a doctors measuring tape[standard 1 meter tape]
   ii. Presence of true knots- loose or tight were noted
   iii. Site of insertion of cord on the placenta was noted and presence of marginal insertion if any was noted (that is attachment of cord to the margin of placenta was noted).

   All deliveries were attended by the pediatric residents who recorded apgar scores and provided neonatal resuscitation if any required.

   Foetal distress was recognized from the presence of meconium in the amniotic fluid and/or abnormal foetal heart rate monitored by Doptone.

   We recognized the various enlisted umbilical cord accident group cases and we considered a control group composed of deliveries without the various enlisted umbilical cord accidents.

Statistical analysis was performed using the Microsoft Excel sheet . Statistical significance was ascertained using the chi square test , z test and a p value of < o.o5 was considered to be statistically significant.

## RESULTS

Total sample size = 300

Cases of umbilical cord accidents in our study = 63

1. Nuchal cord – Tight nuchal cord – 14 cases

   Loose nuchal cord – 33 cases

2. Body loop / extremity loop - 7 cases
3. Cord prolapse - 2 cases
4. True knots - 2 cases
5. Marginal insertion of placenta - 5 cases



Mean umbilical cord length in cases of umbilical cord accident group - 61.06 ± 9.52 cm

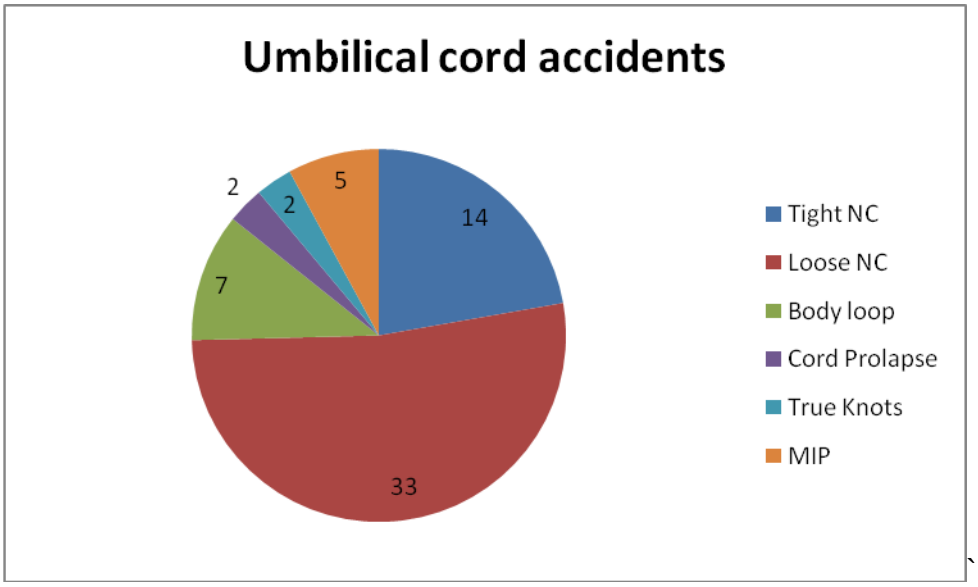
Mean umbilical cord length in cases of control group - 49.66 ± 3.10 cm

The Difference in the mean umbilical cord length was found to be statistically significant in the umbilical cord accident group as compared to the control group.

Z = 9.37

P <0.001 ( SIGNIFICANT)

**Graph 1: Distribution of umbilical cord accidents**

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Tight NC- Tight nuchal cord

Loose NC- Loose nuchal cord

MIP – Marginal insertion of placenta

Table 1 - **Distribution according to age**

| Age in years | Umbilical cord accident group (n=63) | Control group (n=237) |
|---|---|---|
| ≤ 18 years | 4 | 7 |
| 19-24 years | 40 | 166 |
| 25-30 years | 16 | 58 |
| 31-35 years | 3 | 6 |
| >35 years | 0 | 0 |

Chi sq value 2.15

P value 0.54

This is statistically non significant

**Graph 2: Age-wise distribution of study subjects**

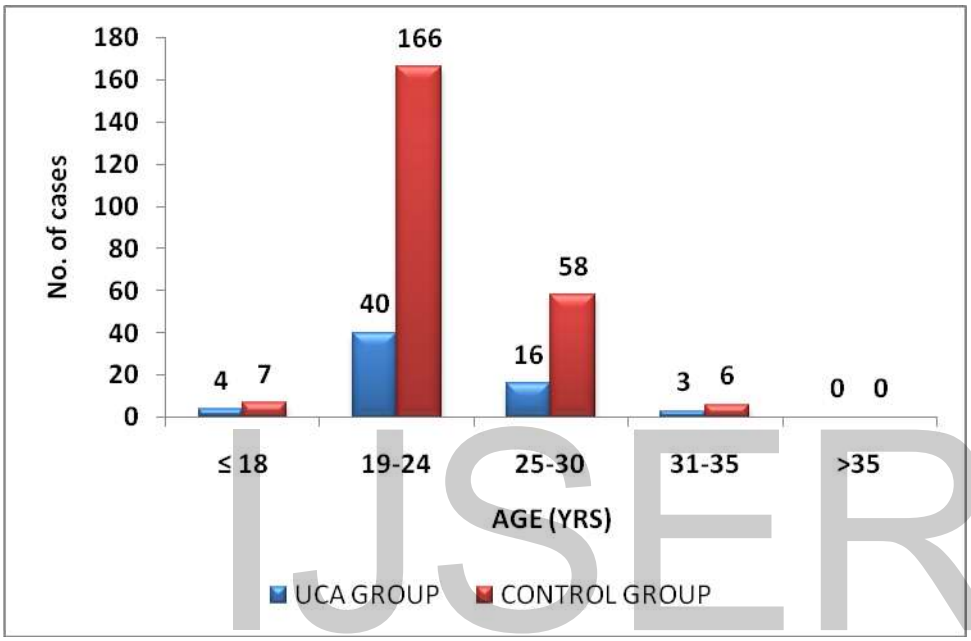


**Table 2 - Distribution according to parity**

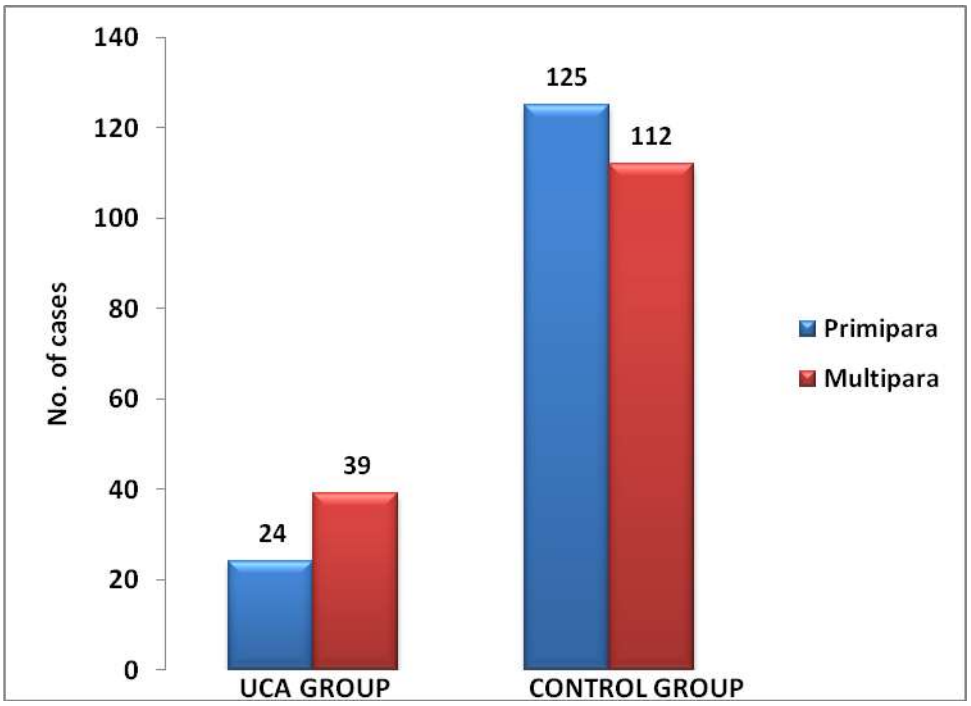
| Parity | Umbilical cord accident group | Control group |
|---|---|---|
| Primiparous | 24 | 125 |
| Multiparous | 39 | 112 |

Chi sq value 3.70

P value 0.054

This is statistically non significant

**Graph 3: Distribution of study subjects according to parity**



Total cases = 300

Nuchal cord incidence= 15.66%= 47 cases/300

Distribution of 47 cases:

LNC(Loose nuchal cord)- 33 cases i.e 70.2%

TNC(Tight nuchal cord)- 14 cases i.e 29.78%

**Table 3-Comparison of mode of delievery between nuchal cord group and control group**

| | Vaginal delievery % | Caesarian section % | Vaccum delivery | Forceps delivery |
|---|---|---|---|---|
| Nuchal cord group | 40 (85.10%) | 5 (10.63%) | 1 (2.12%) | 1 (2.12%) |
| Control group | 177 (63.63%) | 50 (19.76%) | 13 (5.13%) | 13(5.13%) |
| Chi sq | 4.54 | 2.20 | 1.626 With yates correction | 1.626 With yates correction |
| P value | 0.033(S) | 0.1376(NS) | 0.81 (NS) | 0.81 (NS) |



According to this table, vagina delivery in the nuchal cord group is significantly more than in the control group, while the other modes of delivery do not have any statistical difference between the nuchal cord group and the control group.

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**Graph 4: Comparison of mode of delivery between Nuchal cord group and Control group**

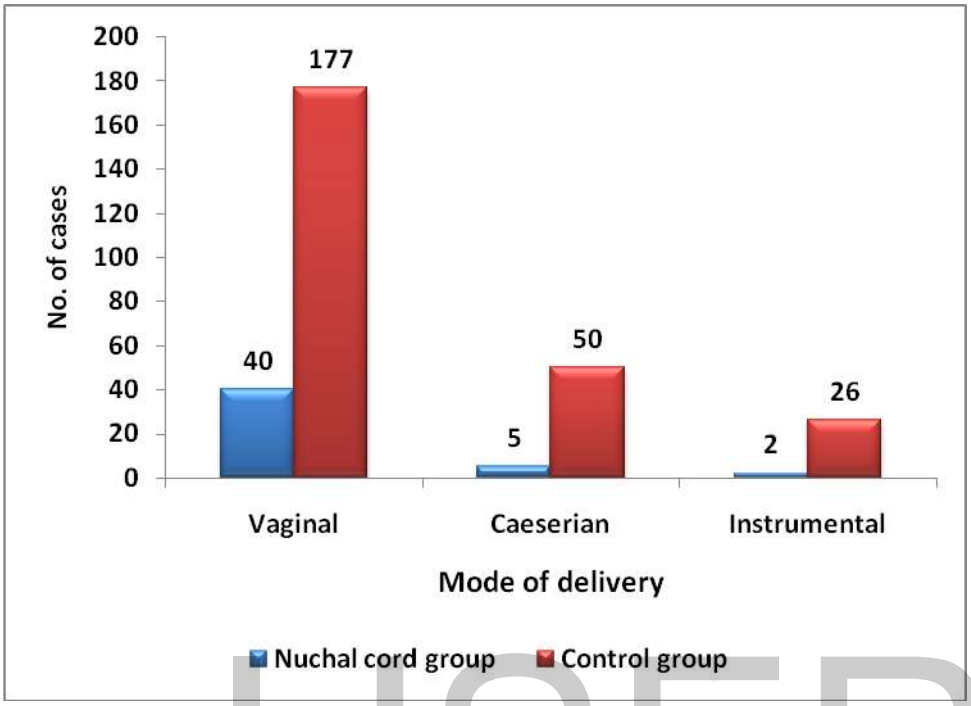
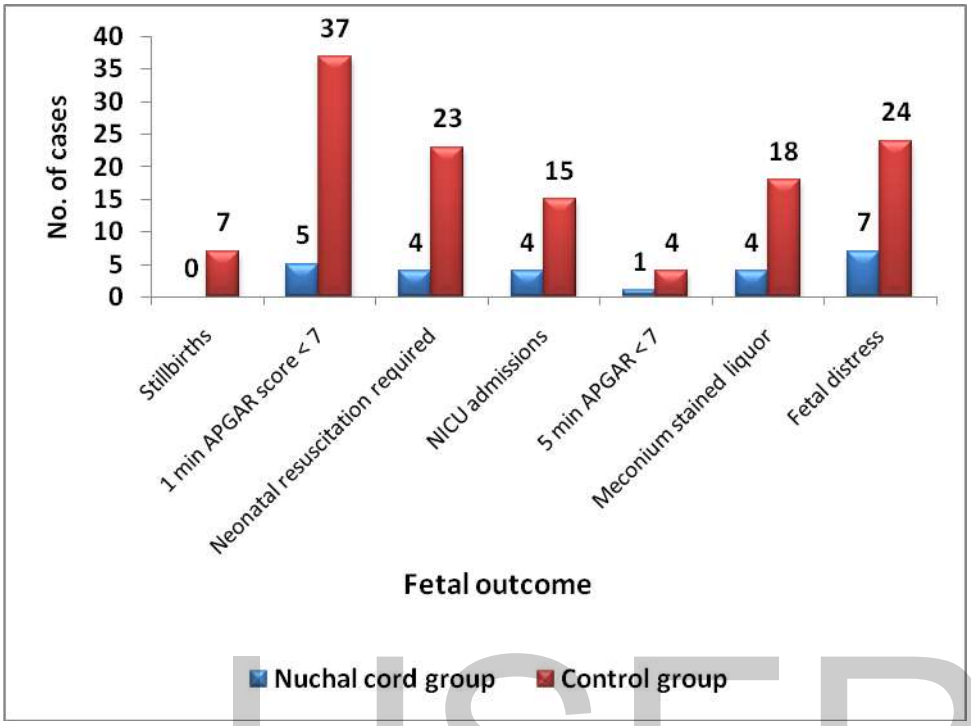


**Table 4- Comparison of Foetal outcome between Nuchal cord group & Control group**

| Foetal outcome | Nuchal cord group (n=47) | Control group (n= 253) | Chi sq value | P value |
|---|---|---|---|---|
| Stillbirths | 0 | 7 (2.76%) | - | >0.05 (ns) |
| 1 min apgar<7 | 5(10.63%) | 37(14.26%) | 0.52 | 0.47 (ns) |
| Active neonatal resuscitation required | 4(8.51%) | 23(9.09%) | 0.01 | 0.89(ns) |
| Nicu admissions | 4(8.51%) | 15(5.92%) | 0.44 | 0.50(ns) |
| 5 min apgar <7 | 1(2.127%) | 4(1.58%) | 0.07 | 0.788(ns) |
| Meconium stained liquor | 4(8.51%) | 18(7.11%) | 0.113 | 0.73 (ns) |
| Fetal distress | 7(14.89%) | 24(9.48%) | 1.25 | 0.26(ns) |

Ns – non significant

**Graph 5: Comparison of foetal outcome between Nuchal cord group and Control group**



We first divided our study population into 2 groups :

-Nuchal cord group

-Control group

Further we divided Nuchal cord into :

-Loose nuchal cord

-Tight nuchal cord

We defined Nuchal cord as Loose nuchal cord if it could be easily uncoiled before complete delivery of baby and Tight if it was not reducible prior to the delivery of neonates body and needed to be clamped and cut before the delivery of the baby.[84]

**Table 5 -Comparison of mode of delivery between loose Nuchal cord group and the Control group**

| | Loose nuchal cord group (n= 33) | Control group (n=253) | CHI SQ | P value |
|---|---|---|---|---|
| Vaginal delivery | 30 (90.90%) | 177 (69.96 %) | 6.41 df=1 | 0.0114 (S) |
| Emergency caesarean section | 2(6.06%) | 44(17.39%) | 3.67 df=1 | 0.09 ( NS) |
| Elective caesarean section | 0 | 6(2.37%) | 2.37 with yates correction | 0.3713 (NS) |
| Instrumental delivery | 1(3.03%) | 26(10.27%) | 2.74 with yates correction | 0.180 (NS) |

S – Significant

NS – Non significant

The vaginal delivery in the loose nuchal cord group is statistically significant as compared to that in the control group.

The emergency caesarean section rate in the control group is more than in the loose nuchal cord group but non significantly.

The Instrumental delivery rate is also higher in the control group but not significant .

**Table 6 -Comparison of Mode of delivery between Tight nuchal cord group and the Control group**

| | Tight nuchal cord group(n=14) | Control group (n=253) | Chi sq with Yates correction | P value |
|---|---|---|---|---|
| Vaginal delivery | 10 (71.42%) | 177(63.63%) | 0.334 | 0.907 ( NS) |
| Emergency caesarian section | 3(21.4%) | 44(17.39%) | 0.00066 | 0.699(NS) |
| Elective caesarean section | 0 | 6(2.37%) | 2.277 | 0.560(NS) |
| Instrumental delivery | 1(7.14%) | 26(10.27%) | 0.6954 | 0.7049(NS) |

S – Significant

NS- Non significant

The various modes of delivery did not have any statistical difference when compared between Tight nuchal cord group and the control group.

**Table 7-Comparison of foetal outcome between Loose nuchal cord group and the Control group**

| | Loose nuchal cord group n = 33 | Control group n = 253 | Chi sq | P value |
|---|---|---|---|---|
| Fetal distress | 2 (6.06%) | 24(9.48%) | 0.9326 | 0.519 (NS) |
| Apgar <7 at 1 minute | 1 (3.03%) | 37(14.26%) | 4.487 | 0.0649 (NS) |
| Apgar <7 at 5 minutes | 0 | 4(1.58%) | 2.297 | 0.467(NS) |
| Nicu admissions | 0 | 15 (5.92%) | 3.43 | 0.151(NS) |



NS- Non significant

Foetal outcome in terms of foetal distress, apgar score at 1 minute and 5 minutes and NICU (Neonatal Intensive Care Unit) admissions were found to be statistically non significant between Loose nuchal cord group and the Control group.

**Table 8 -Comparison of foetal outcome between Tight nuchal cord group and Control group**

| | Tight nuchal cord group | Control group | Chi sq with Yates correction with df = 1 | P value |
|---|---|---|---|---|
| Foetal distress | 5 (35.71%) | 24 (9.48%) | 6.91 | 0.002 (S) |
| Apgar < 7 at 1 minute | 3 (21.42%) | 37 (14.26%) | 0.096 | 0.49(NS) |
| Apgar <7 at 5 minutes | 1 (7.14%) | 4 (1.58%) | 0.232 | 0.14 (NS) |
| Nicu admission | 3 ( 21.42%) | 15(5.92%) | 2.903 | 0.024 (S) |



S – Significant

NS-Non significant

Fetal distress and NICU (Neonatal Intensive Care Unit) admission rate has been found to be statistically significant in the Tight nuchal cord group as compared to the Control group while both the groups do not have any statistical difference as regards apgar score <7 at 1 and 5 minutes.

**Table 9-Comparison of mode of delivery between Loose nuchal cord group and Tight nuchal cord group**

| Type of delivery | Loose nuchal cord group (n= 33) | Tight nuchal cord group(n= 14) | Chi sq value | P value |
|---|---|---|---|---|
| Vaginal delivery | 30(90.90%) | 10(71.42%) | 2.94 | 0.08(NS) |
| Instrumental delivery | 1 (3.03%) | 1(7.14%) | 0.40 | 0.52(NS) |
| Caesarian section | 2(6.06%) | 3(21.4%) | 2.44 | 0.11(NS) |

NS - non significant



The mode of delivery between Loose nuchal cord group and the Tight nuchal cord group was found to be statistically non significant.

In the Tight nuchal cord group;

There were 3 caesarian sections, all three were emergency primary caesarian sections taken for fetal distress.

In the Loose nuchal cord group;

There were two emergency caesarean sections , one was a primary caesarean section and the other was a repeat caesarean section.

The primary caesarean section was taken for face presentation with failure to progress.

The emergency repeat caesarean section was done in a $G_2P_1L_1$ with previous LSCS one year back for foetal distress.

**Graph 6: Comparison of mode of delivery between Loose nuchal cord group and Tight nuchal cord group**

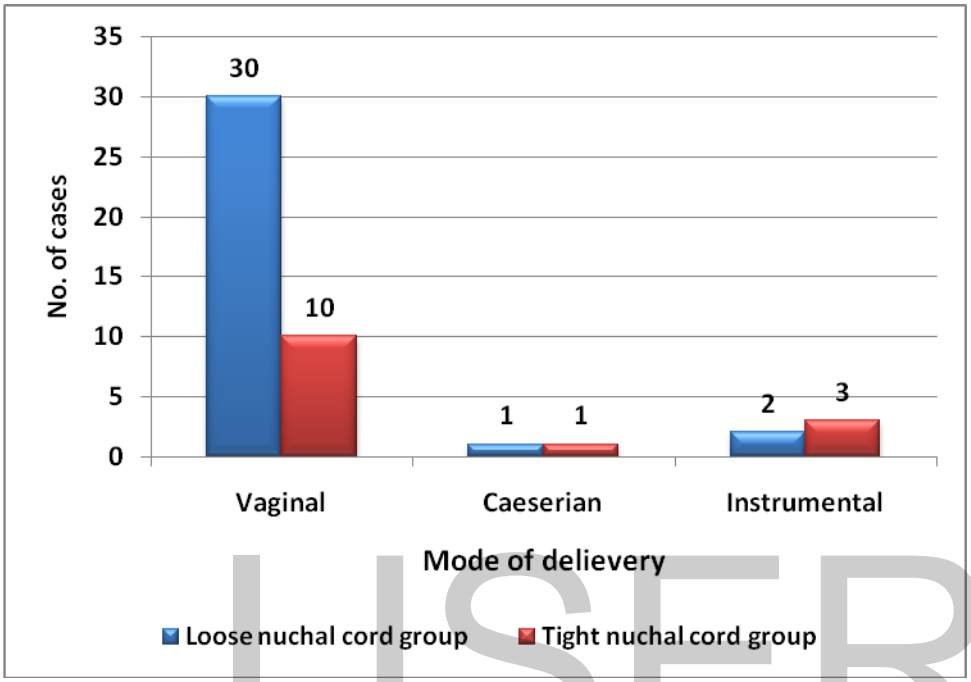


**Table 10- Comparison of Foetal outcome between Loose nuchal cord group and Tight nuchal cord group**

| Fetal outcome | Loose nuchal cord | Tight nuchal cord | Chi sq value | P value |
|---|---|---|---|---|
| Stillbirths | 0 | 0 | - | - |
| Fetal distress | 2(6.06%) | 5(35.71%) | 6.81 | 0.009(significant) |
| Meconium stained liquor | 1(3.03%) | 3(21.42%) | 4.27 | 0.03(significant) |
| 1 min apgar <7 | 1(3.03%) | 3(21.42%) | 4.27 | 0.03(significant) |
| 5 min apgar <7 | 0 | 1(7.14%) | 7.059 with Yates correction | 0.120 ( NS) |
| Neonates requiring resuscitation measures | 0 | 3(21.42%) | 11.57 with Yates correction | 0.0059 (significant) |
| Nicu admissions | 0 | 3(21.42%) | 11.57 with Yates correction | 0.0059 ( significant) |



NS- non significant

Foetal distress , meconium stained liquor , 1 min apgar <7 , neonates requiring resuscitation measures and NICU (Neonatal Intensive Care Unit) admissions were found to be significantly higher in the Tight nuchal cord group as compared to the control group.

In the Tight nuchal cord group – total 14 cases

One had tight nuchal cord with a body loop and an extremity loop taken for emergency caesarean section for foetal distress. It had one 1 min apgar score <7 and was actively resuscitated with endotracheal intubation and chest compressions. The neonate was taken to NICU(Neonatal Intensive Care Unit) for admission. This baby died in NICU (Neonatal Intensive Care Unit) after a few days due to sepsis.



The other also born by Primary emergency caesarean section for foetal distress also had one min apgar score of <7 and had to be resuscitated actively with ambubag and endotracheal intubation and was admitted in NICU(Neonatal Intensive Care Unit)

The third baby of Tight nuchal cord born by Primary emergency caesarean section cried immediately after birth , had one minute apgar score of > 7 and did not require any active resuscitation .

In the Tight nuchal cord group there was only one Instrumental delivery with outlet Wrigleys forceps . This baby had one minute as well as 5 minute apgar score of <7 and was actively resuscitated and required NICU(Neonatal Intensive Care Unit) admission.

Out the other ten babies of Tight nuchal cord group , one had one min apgar score <7 with foetal distress with Meconium stained liquor but had five min apgar score of >7 and was routinely resuscitated and did not require NICU(Neonatal Intensive Care Unit) admission.

The other nine babies born by vaginal delivery had no complications.



In the Loose nuchal cord group- total 33 cases

Two cases of Loose nuchal cord group were taken for caesarean section.

One was primary emergency Lower Segment Caesarean Section for face presentation with failure to progress. It had one minute apgar score of >7 and did not require any active resuscitation or NICU(Neonatal Intensive Care Unit) admission.

The other was an emergency repeat caesarean section for foetal distress . This baby had one minute apgar score of < 7 but it required only routine resuscitation and had 5 min apgar score of > 7 and did not require any NICU(Neonatal Intensive Care Unit) admission.

One case of Loose nuchal cord group was an instrumental delivery done with ventousse .There was light meconium stained liquor with foetal distress and prolonged second stage . This baby had one minute apgar score of >7 and did not require any active resuscitation or NICU(Neonatal Intensive Care Unit) admission.

**Graph 7: Comparison of Foetal outcome between the Loose nuchal cord group and the Tight nuchal cord group**

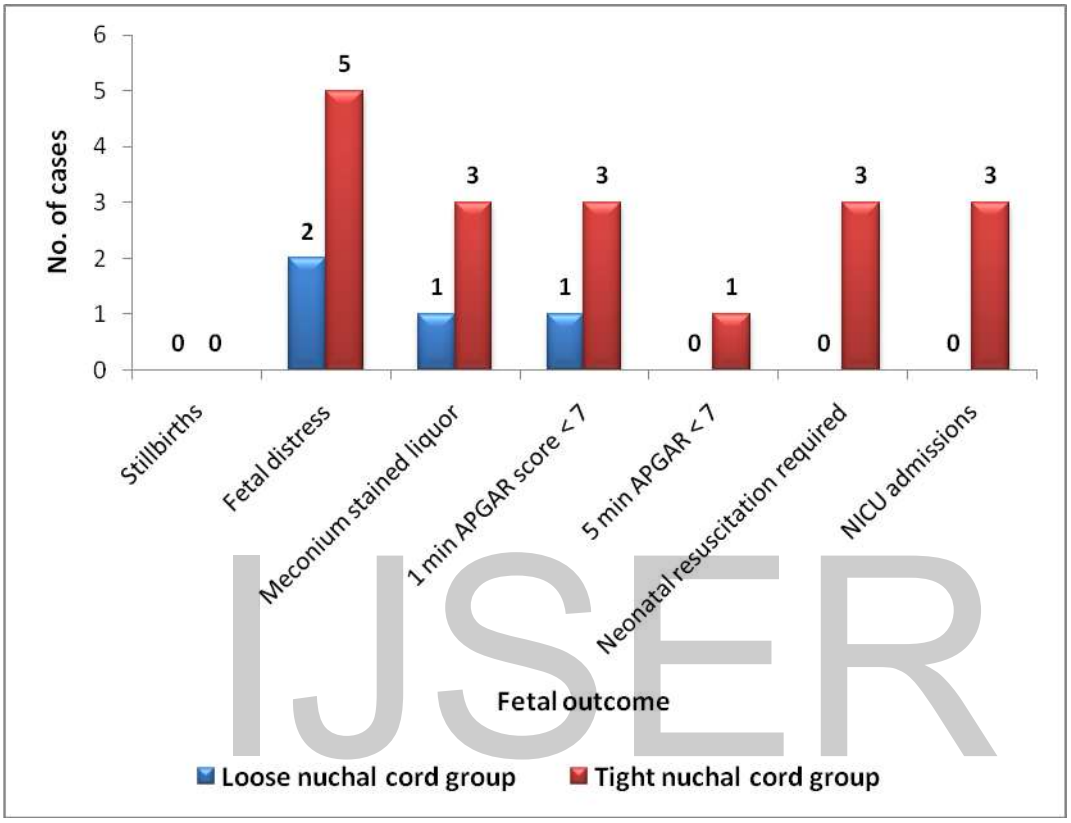


**TABLE 11- Nuchal cord loops**

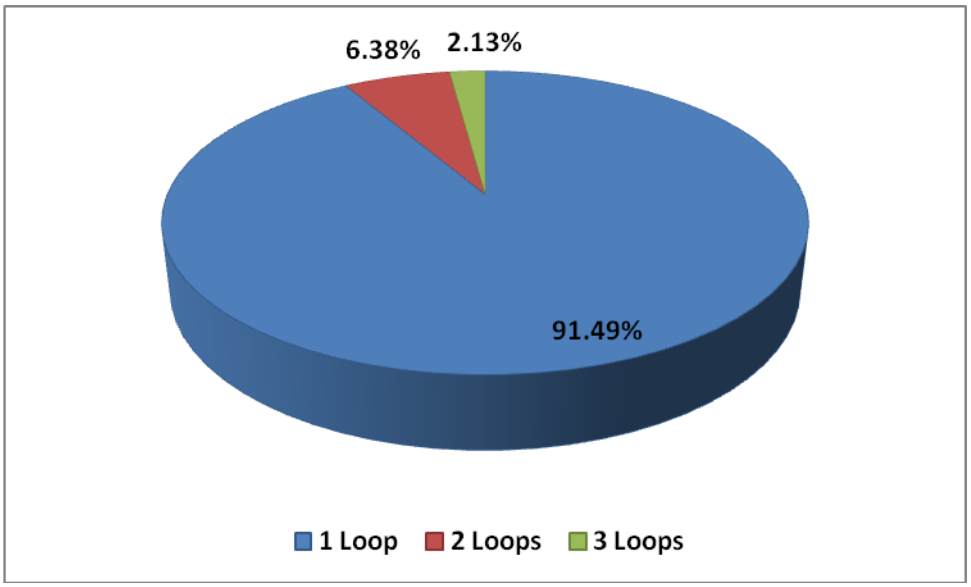
| | Cases | % |
|---|---|---|
| One loop | 43 | 91.48% |
| Two loops | 3 ;<br>1 in tight nuchal cord group<br>2 in loose nuchal cord group | 6.38% |
| Three loops | 1 in loose nuchal cord group | 2.13% |
| | Total=47 cases | 100% |



In the two loops around the neck group , out of three cases ,only one case who had two tight loops of cord around neck had one minute apgar score of < 7 and was actively resuscitated and required NICU(Neonatal Intensive Care Unit) admission while the other two had two loose loops of cord around neck did not have any such problem.

In the three loops around the neck group it was a loose nuchal cord with three loops, this baby had foetal distress and one minute apgar score <7 but did not require active resuscitation or NICU (Neonatal Intensive Care Unit) admission

**Graph 8: Distribution of Nuchal cord group cases according to number of loops**



**BODY LOOP/ EXTREMITY LOOP**

Incidence= 7 cases/300= 2.33%

In our study out of 7 cases only 1 case of body loop that was associated with Tight nuchal cord was found to have foetal distress, with 1 min and 5 min apgar score <7, who needed active resuscitation and was admitted to and NICU(Neonatal Intensive Care Unit) who died in NICU(Neonatal Intensive Care Unit) of sepsis 5 days later.

The other six cases did not have any adverse postnatal outcome.

**TRUE KNOTS**

Incidence – 2 cases out of 300 ie 0.66%

Our study had 2 cases of true knots ;

One was discovered on an emergency caesarean section taken for primipara with breech with premature rupture of membranes. It was a loose true knot. The baby cried immediately ,had one minute apgar score of >7 and did not require any active resuscitation or NICU (Neonatal Intensive Care Unit) admission.

The second case of true knot was seen after the delivery of placenta in a full term normal vaginal delivery as a chance finding. It was also a loose true knot .The baby cried immediately after birth and had one minute apgar score of > 7.

No incidence of birth asphyxia was noted in our study in cases of true knot.

**MARGINAL INSERTION OF PLACENTA**

Incidence – 5 cases out of 300 ie 1.66%

In all the 5 cases of marginal insertion of placenta in our study ,we did not find any adverse postnatal outcome in any of the cases.

## CORD PROLAPSE

Incidence - 2 cases out of 300 ie 0.66%

There were 2 cases of cord prolapsed in our study;

- Both were unregistered

- One was a $G_3P_2L_2$ with full term pregnancy with neglected shoulder with hand prolapse with cord prolapse and the foetus died within 3-4 min of admission in the casualty, it was a fresh stillbirth and was taken for emergency Lower Segment Caesarean Section.

- The other was also an unregistered case $G_3P_2L_2$ with full term pregnancy with polyhyraminos with premature rupture of membranes taken for emergency Lower Segment Caesarean Section, the neonate had 1 min and and 5 min apgar score <7 and was actively resuscitated and was admitted in the NICU (Neonatal Intensive Care Unit)

## DISCUSSION

In our study , a total of 300 deliveries of > 34 weeks gestation and < 42 weeks gestation irrespective of age , parity and mode of delivery were studied.

The main objective of study was to find out the immediate postnatal outcome due to the various enlisted commonly occurring umbilical cord accidents in our study .

The various commonly occurring umbilical cord accidents included in our study were;

- Nuchal cord ( tight and loose nuchal cord)
- Cord prolapse
- True knots
- Body loop / extremity loop
- Marginal insertion of placenta

## NUCHAL CORD

The clinical significance of nuchal cord is controversial. Although in many cases , it does not seem to do any harm but it has been reported to be associated with significantly higher incidence of 1 min apgar score, meconium stained liquor, emergency caesarean section , need for neonatal resuscitation and admission to Nicu (Neonatal Intensive Care Unit) and possibly perinatal death.[3]

Variable tightness of the cord , cords wrapped around the neck in a locked fashion or not , and multiple cord entanglements around foetal neck may be contributors to this controversy, because a moderately tight cord around the neck would impair cephalic venous blood flow only, whereas a very tight cord would compromise the umbilical circulation and produce systemic hypoxia , hypercapnia and acidemia.[3]



In our study no correlation was found to exist between cord complications and maternal age or parity , because they presumably do not serve as etiological factors . This statement is supported by Spellacy et al[41] , Dhar k k et al[87] J.D.. Kemfang Ngowa.[94]

## INCIDENCE OF NUCHAL CORD

| | | Sample Size | Incidence |
|---|---|---|---|
| 1 | Singh Gurmesh, Dasgupta Ellora[84] | 690 | 19.71% |
| 2 | Shreshtha NS, Singh N[85] | 512 | 22.85% |
| 3 | E. Assimakopoulos et al[86] | 352 | 15.8-30% |
| 4 | Dhar KK, Ray SN et al [87] | 3,580 | 5.74% |
| 5 | Rhoades DA[88] | 5426 | 55.28% |
| 6 | Kumari et al[89] | 12,000 | 1.7% |
| 7 | Mastrobattista et al[90] | 4426 | 17.51% |
| 8 | Miser WF[42] | 706 | 23.7% |
| 9 | Collins[2] | - | 20.4% |
| 10 | Sheiner E[95] | 1,66,318 | 14.7% |
| 11 | Spellacy et al 1966[41] | 17,190 | 24.6% |
| 12 | Schaffer L, Burkhardt T[57] | - | 33.7% |
| 13 | Reiss 1958[52] | - | 20% |
| 14 | Begum AA et al[97] | 1646 | 9.23% |
| 15 | J.D. Kemfang Ngowa et al[94] | 9275 | 16.2% |
| 16 | Our study | 300 | 15.66% |

Our incidence of presence of nuchal cord correlates with other studies.These differences in the incidence could be explained by the differing sample sizes and the gestation period of delivery taken into account.[94]

**INCIDENCE OF MULTIPLE NUCHAL LOOPS**

| | | Incidence |
|---|---|---|
| 1 | Onderoglu et al[31] | 10.6% |
| 2 | Larson JD, Rayburn WF et al[44] | 3.8% |
| 3 | Mastrobattista et al[90] | 1.9% |
| 4 | Singh Gurmeesh et al[84] | 2.03% |
| 5 | Miser WF[42] | 2% |
| 6 | Spellacy et al[41] | 3.4% |
| 7 | Begum AA et al[97] | 12.5% |
| 8 | Our study | 1.33% |



Our incidence correlates with other studies.

**INCIDENCE OF NUMBER OF LOOPS OF NUCHAL CORD AROUND NECK**

| | | Singh Gurmeesh, Dasgupta et al[84] | Miser WF[42] | Shui & Eastman[50] | Spellacy et al[41] | J.D. Kemfang Ngowa et al[94] | Dhar KK et al[87] | Our study |
|---|---|---|---|---|---|---|---|---|
| | | n=690 | n =706 | | n =17,190 | n= 9275 | n=3133 | n =300 |
| 1 | 1 loop | 17.68% | 21.7% | 20.6% | 21.27% | 14.18% | 133 (73.8%) | 14.33% |
| 2 | 2 loops | 1.45% | 1.7% | 2.5% | 3.3% | 1.67% | 37 (20.5%) | 1.1% |
| 3 | 3 loops | 0.58% | 0.3% | 0.5% | | 0.32% | 9 (5%) | 0.33% |
| 4 | 4 loops | - | - | 0.1% | | | 1 (0.6%) | |
| 5 | 5 loops | - | - | - | | | | |



Our incidence correlates with other studies.

Shui KP et al[50] have reported that the number of loops around the neck correlates with the cord length as they have reported significantly longer cord lengths in such cases. Our findings correlate as our mean cord length is higher in cases of umbilical cord accident group including nuchal coils as compared to the control group.

We first divided our study population into two groups :

-Nuchal cord group

-Control group

Further we divided the nuchal cord group into :

-Loose nuchal cord group

-Tight nuchal cord group

We defined Nuchal cord as Loose nuchal cord if it could be easily uncoiled before complete delivery of the baby and Tight if it was not reducible prior to the delivery of neonate's body and needed to be clamped and cut before the delivery of the baby.



**Comparison of sample size and the distribution of the number of cases**.

| | Singh Gurmesh[84] | WF Miser[42] | Mastrobattista[90] | Our study |
|---|---|---|---|---|
| Sample size | 690 | 706 | 4426 | 300 |
| Nuchal cord group | n=136 (19.71%) | n=167 (23.7%) | n=691 (15.6%) | n=47 (15.66%) |
| Control group | n=554 (80.28%) | n=523 (74.07%) | n=3735 (84.38%) | n=253 (84.33%) |

Our cases of nuchal cord were less as compared to the other studies maybe because of the smaller sample size so taken.

**DIVISION OF NUCHAL CORD GROUP AS LOOSE NUCHAL CORD AND TIGHT NUCHAL CORD**

| | Singh Gurmesh et al[84] | Dharr KK et al[87] | Miser et al[42] | J.D. Kemfang Ngowa et al[94] | Our study |
|---|---|---|---|---|---|
| Nuchal cord group | n=136 cases | n=180 | n=167 | n= 9275 | n=47 |
| Loose nuchal cord group | 97 cases (71.32%) | 108 (61%) | 43 cases (25.7%) | 7031 cases (75.81%) | 33 cases (70.21%) |
| Tight nuchal cord group | n=39 cases (28.67%) | 70 (39%) | 38 cases (22.8%) | 14 cases (29.78%) | 14 cases (29.78%) |

Our results correlate with the study of Singh Gurmeesh et al[84].

**Comparison of mode of delivery between Nuchal cord group and Control group**

| | Nuchal cord group | Control group | Chi square | P value |
|---|---|---|---|---|
| **Singh et al[84]** | | | | |
| Vaginal delivery | 91.18% | 74% | 18.396 | 0.001 |
| Caesarean section | 8.82% | 25.9% | 2.669+17.047 | <0.2+0.001 |
| Vaccum delivery | 0.74% | 1.08% | 0.131 | 1 |
| Forceps delivery | - | - | - | - |
| | | | | |
| **Mastrobattista etal[90]** | | | | |
| Vaginal delivery | 91.2% | 89.1% | - | 0.12 |
| Caesarean section | 5.2% | 11.4% | - | <0.01 |
| Vaccum delivery | - | - | - | - |
| Forceps delivery | - | - | - | - |
| | | | | |
| **Miser WF[42]** | | | | |
| Vaginal delivery | 83.8% | 73.4% | - | <0.01 |
| Caesarean section | 11.4% | 20.9% | - | NS+<0.01 |
| Vaccum delivery | 1.2% | 1.5% | - | NS |
| Forceps delivery | 3.6% | 4.2% | - | NS |
| | | | | |
| **Begum AA et al[97]** | | | | |
| Vaginal delivery | 84.21% | 68.61% | - | - |
| Caesarean section | 13.8% | 30.72% | - | - |
| Vaccum delivery | 1.97% | 0.67% | - | - |
| Forceps delivery | - | - | - | - |
| | | | | |
| **Our study** | | | | |
| Vaginal delivery | 40(85.1%) | 177(63.6%) | 4.54 | 0.033 (S) |
| Caesarean section | 5(10.6%) | 50(19.76%) | 2.20 | 0.1376 (NS) |
| Vaccum delivery | 1(2.12%) | 13(5.13%) | 1.626 with Yates correction | 0.81(NS) |
| Forceps delivery | 1(2.12%) | 13(5.13%) | 1.626 with Yates correction | 0.81(NS) |

S- Significant, NS- Non significant

According to Singh et al [84] and Miser WF[42] , vaginal delivery was significantly higher in the nuchal cord group as compared to the control group. Our findings correlate as our study also had significantly higher rates of vaginal delivery in the nuchal cord group.

Vaginal delivery in the nuchal cord group was also higher in a study by Mastrobattista et al [90], though non significantly as compared to the control group.

Also Begum A A e al [94] have reported a higher incidence of vaginal delivery in the nuchal cord group but no comment regarding its statistical significance has been made.

In cases of caesarean section as a mode of delivery , it was found to be significantly higher in the control group as compared to the nuchal cord group in a study by Mastrobattista et al [90] and Shreshtha NS et al.[85]

It was also found higher though non significantly in the control group in a study by Singh et al [84] and Miser WF[42]. Our study correlates with them as we had higher caesarean section rates in the control group as compared to the nuchal cord group though non significantly.

Also Begum AA et al [97] found higher rates of caesarean section in the control group but no statistical significance has been attributed to it. But, according to Assimakapoulos et al [86] , caesarean section rate was significantly higher in the nuchal cord group as compared to the control group.

Incidence of Instrumental delivery was found to be non significantly higher in the control group in a study by Singh etal [84] and Miser WF[42]. Our findings correlate with them while Assimakapoulos et al [86] found it significantly higher in the nuchal cord group as compared to the control group. Same findings were reported by Begum AA etal [97] though without any statistical comment.

**Comparision of Mode of Delivery Between Loose Nuchal Cord Group and Control Group.**

| | J.D. Kemfang Ngowa[94] | | | Dhar KK et al[87] | | | Our Study | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | n= 1138 LNC group | n= 7774 control group | P value | n= 108 LNC group | n= 356 control group | P value | n= 33 LNC group | n= 253 control group | Chi sq value | P Value |
| Vaginal del | 887 (78.35%) | 5883 (75.03%) | P= 0.03 (S) | - | - | - | 30 (90.90%) | 177 (63.63%) | 6.41 df =1 | 0.0114 (S) |
| Emerg--ency C.S. | 28 (2.46%) | 608 (7.82%) | P<0.001 (S) | 17 (15.7%) | 43 (12.1%) | P > 0.05 NS | 2 (6.06%) | 44 (17.39%) | 3.67 df =1 | 0.09 NS |
| Instrum-ental del | 7 (0.61%) | 72 (0.92%) | 0.39 NS | 16 (14.8%) | 55 (15.4%) | P >0.05 NS | 1 (3.03%) | 26 (10.27%) | 2.74 with yates correction df=1 | 0.180 NS |



del – Delivery
C.S. – Caesarean section
S- Significant

NS- Non significant

According to JD Kemfang Ngowa[94] the rate of vaginal delivery is significantly higher in the Loose nuchal cord group. Our findings correlate with him as we had significantly higher rate of vaginal delivery in Loose nuchal cord group.

The rates of emergency caesarean section were found significantly higher in the control group according to JDKemfang Ngowa[94]. We also found higher rates in control group , though non significantly.

But, they were found higher in the loose nuchal cord group though non significantly in a study by Dhar KK et al [87].

The rates of instrumental delivery have been found to be non significantly higher in the control group in studies done by Dhar KK et al [87] and J.D.Kemfang ngowa[94]

**Comparision of Mode of Delivery Between Tight Nuchal Cord Group and Control Group**

| | J.D. Kemfang Ngowa[94] | | | Dhar K K et al [87] | | | Our study | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | n= 363 TNC group | n= 7774 control group | P value | n= 70 TNC group | n= 356 control group | P value | n= 14 TNC group | n= 253 control group | CHI SQ with yates correction | P value |
| Vaginal delivery | 288 (77.33%) | 5833 (75.03%) | 0.37 NS | - | - | - | 10 (71.42%) | 177 (63.63%) | 0.0334 | 0.907 (NS) |
| Emergency caesarean section | 13 (3.58%) | 608 (7.82%) | < 0.05 (S) | 19 (27.1%) | 43 (12.1%) | P <0.01 (S) | 3 (21.4%) | 44 (17.39%) | 0.00066 | O.699(NS) |
| Instrumental delivery | 7 (1.92%) | 72 (0.92%) | 0.1 NS | 20 (28.5%) | 55 (15.4%) | P < 0.01 (S) | 1 (7.14%) | 26 (10.27%) | 0.6954 | 0.7049 (NS) |



TNC – Tight Nuchal Cord

S- Significant

NS – Non significant

In this table , the rates of vaginal delivery were non significantly higher in the Tight nuchal cord group according to J.D. Kemfang Ngowa[94] , In our study also they were found non significantly higher in the Tight nuchal cord group.

According to J.D. Kemfang Ngowa [94], the rates of emergency caesarean section were found significantly higher in the control group.Contrary to this, according to Dhar KK et [87]the rates were found significantly higher in the tight nuchal cord group. We also found higher rates in the Tight nuchal cord group though results were statistically non significant.

In Dhar KK et al study[87] , Instrumental delivery rates were significantly higher in the Tight nuchal cord group though non significantly, but our study had non significantly higher rates in the control group. This may be due to the lesser incidence of Tight nuchal cord in our study.

**Comparision of Mode of Delivery Between Loose Nuchal Cord and Tight Nuchal Cord**

| | Singh Gurmesh et al | | | | | Our study | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Vag | Primary c.s. | Repeat c.s. | V | f | vag | Primary c.s. | Repeat c.s. | V | f |
| Loose nuchal cord | 95.87 % | 3.09% | 1.03% | - | - | 30 (90.90%) | 1 (3.03 %) | 1 (3.03 %) | 1 (3.03 %) | - |
| Tight nuchal cord | 79.48 % | 17.95 % | 2.56% | 2.56 % | - | 10(71.42 %) | 3 (21.42 %) | - | - | 1 (7.14 %) |
| Chi square | 9.287 | 9.012 | 0.451 | 2.506 | - | 2.94 | 2.44 | 0.1995 With Yates correction | 0.1995 With Yates correction | 7.059 |
| P value | <0.01 | 0.01 | 1 | 0.2 | - | 0.08 NS | 0.11 NS | 0.510 NS | 0.510 NS | 0.120 NS |

Vag = vaginal delivery

V = vaccum

f = forceps

NS- Non significant



According to Singh et al study[84] & Dhar KK et al[87], there is a higher rate of Caesarean section in cases with Tight nuchal cord due to increased incidence of foetal distress in such cases. In our study also the rate of caesarean section was higher in the Tight nuchal cord group though we found non significant results due to low sample size.

Rates of vaginal delivery were found significantly higher in the Loose nuchal cord group , according to Singh Gurmeesh et al [84] study. We also found higher rates in the Loose nuchal cord group though insignificantly , while According to JD Kemfang Ngowa[94] et al, they are higher in the Tight nuchal cord group because in his study he found that vaginal delivery and instrumental delivery were higher in the tight nuchal cord group and emergency caesarean section rate was lower as compared to the control group.

Regarding Instrumental delivery , Dhar KK et al [87] has reported significantly higher results in cases of Tight nuchal cord group as compared to the Loose nuchal cord group and Singh Gurmeesh et al[84] has reported non significant results.J.D. Kemfang Ngowa[94], J.M. Mastrobattista et al[90] , Jauniaux Eet al[91], Shreshtha NS[85] have reported no significant differences in the rates of instrumental delivery between the loose nuchal cord group and the Tight nuchal cord group .

## Comparision between Nuchal Cord Group and Control Group in terms of Foetal Outcome

| | Singh Gurmesh et al[84] | | | | W.F. Miser[42] | | | | Our study | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | N.cord group | Control group | Chi sq | P value | N.cord group | Control group | Chi sq | P value | N.cord group | Control group | Chi sq | P value |
| Meconium stained | 5.88% | 7.04% | 0.230 | 1 | - | - | - | - | n=47 8.51% | n=253 7.11% | 0.113 | 0.73 NS |
| Foetal distress | 11.11% | 9.02% | 0.514 | 1 | 18.61% | 9.6% | - | < 0.01 | 14.89% | 9.48% | 1.25 | 0.26 NS |
| 1 min apgar <7 | 7.35% | 5.78% | 0.475 | 1 | 8.4% | 4.8% | - | n.s. | 10.63% | 14.62% | 0.52 | 0.47 NS |
| 5 min apgar <7 | 2.94% | 1.44% | 1.432 | 1 | 1.2% | 1.1% | - | n.s. | 2.127% | 1.58% | 0.07 | 0.788 NS |
| Still births | 0 | - | - | - | - | - | - | - | 0 | 7/253 2.76% | - | - |
| Neonatal resuscitation | - | - | - | - | - | - | - | - | 4/47 8.51% | 23/253 9.09% | 0.01 | 0.89 NS |
| NICU admissions | - | - | - | - | - | - | - | - | 4/47 8.51% | 15/253 5.92% | 0.44 | 0.50 NS |
| Perinatal deaths | - | - | - | - | 0 | - | - | - | 1/47 2.127% | 5/253 1.97% | 0.87 | >0.05 NS |

NS – Non Significant

N.cord = nuchal cord

According to Singh Gurmesh Dasgupta Ellora[84], there are no significant difference between the two groups as regards the foetal outcome and that the nuchal cords did not increase the foetal morbidity as compared to the control group.

Our findings correlate with Singh et al[84] study.

Our findings also correlate with Weber T[40], Mastrobattista JM[90], Holler LM[90], Yeomans ER et al[90],Hankins GD[47], Synder RR[47], Hauth JC et al[73], Abramowicz JS[95], Levy H et al[77], Peregrine et al[96], that nuchal cords do not increase the foetal morbidity and mortality.

In a study by WF Miser[42] , he found significant fetal distress in cases of nuchal cord but there were no perinatal deaths associated with nuchal cord.

Our findings do not correlate with Assimakopoulos et al[86] who reported that nuchal cords result in lower apgar scores at 1 & 5 minutes, mainly as a consequence of higher operative deliveries with p = 0.001 at 1 min apgar score <7 and p = 0.027 at 5 min apgar score <7.



Our findings do not correlate with Rhoades DA et al[88] who found an increased risk of foetal distress (OR-2.7, 95% CI- 21.1-3.4), meconium staining (OR-2.1, 95% CI- 1.7-2.6) and 5 min apgar <7 (OR-1.6, 95% CI- 1-2.4), assisted ventilation <30 min (OR 1.9, 95% CI = 1.4-2.6) in the nuchal cord group. According to him, certain adverse perinatal outcomes are increased in neonates with nuchal cord. However he stated that neonates with nuchal cord do not have significantly longer Nicu (Neonatal Intensive Care Unit) stays and thus the adverse effects of nuchal cord may be transient and we agree with this statement. Also Sheiner E [95] has reported significantly lower 1 minute apgar score <7 in the nuchal cord group.

Our findings also correlate with Shrestha NS study[85] in which though FHR( Fetal Heart Rate) irregularities and meconium staining were increased in the nuchal cord

group but were not statistically significant. Also though apgar score at 1 min <7 was significantly higher in nuchal cord group (p=0.010) but apgar score at 5 min <7 and NICU ( Neonatal Intensive Care Unit )admissions were not more common. In their study, nuchal cord was not associated with any adverse perinatal outcome.

Our findings correlate with Schaffer L, Burkardt T [57] that admissions to NICU ( Neonatal Intensive Care Unit ) are not required more frequently in the nuchal cord group.

Our findings also correlate with Sheiner et al[95] who reported significantly lower perinatal mortality in nuchal cord group p=0.001.

**Comparision of Fetal Outcome between Loose Nuchal Cord Group and Control Group**

| | JD Kemfang Ngowa[94] | | | Dhar K K et al[87] | | | Our study | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | LNC group | Control group | P value | LNC group | Control group | P value | LNC group | Control group | CHI SQ | P value |
| | n= 1138 | n= 7774 | | n= 108 | n= 356 | | n= 33 | n= 253 | | |
| Fetal distress | 18 (1.58%) | 227 (2.91 %) | 0.01 Significant | 20 (18.5 %) | 35 (9.8 %) | < 0.05 Significant | 2 (6.06% ) | 24 (9.48 %) | 0.9326 | 0.519 (NS) |
| Apgar score <7 at 1 min | 117 (10.28% ) | 958 (12.32 %) | 0.06 NS | 7 (6.4%) | 30 (8.3 %) | P > 0.05 NS | 1 (3.03% ) | 37 (14.26 %) | 4.487 | 0.0649 (NS) |
| Apgar score <7 at 5 min | 16 (1.40%) | 122 (1.56 %) | 0.7 NS | - | - | - | - | 4 (1.58 %) | 2.297 | 0.467( NS) |
| NICU admissions | 89 (7.82%) | 1135 (14.59 %) | <0.001 Significant | - | - | - | - | 15 (5.92 %) | 3.43 | 0.151( NS) |

NS – Non Significant

According to JD Kemfang Ngowa[94], the rates of low apgar scores at 1 and 5 min is lower in the Loose Nuchal Cord group as compared to control group though non significantly. Our findings correlate with him. He also found significantly lower rates of foetal distress and Nicu (Neonatal Intensive Care Unit) admissions in the Loose nuchal cord group . Our findings were the same but statistically non significant maybe due to low sample size.

According to Dhar KK[87] et al al, fetal distress was found to be significantly high in the Loose nuchal cord group as compared to control group. Apgar score <7 at 1 min was found to be non significantly higher in the control group as compared to Loose nuchal cord group.

**Comparision of fetal outcome between Tight nuchal cord group and Control group**

| | JD Kemfang Ngowa[94] | | | Dhar KK et al[87] | | | Our study | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | TNC group | Control group | P value | TNC group | Control group | P value | TNC group | Control group | Chi sq with yates correction | P value |
| | n= 1138 | n= 7774 | | n=70 | n=356 | | n= 14 | n= 253 | | |
| Fetal distress | 8 (2.20%) | 227 (2.91%) | 0.4 (NS) | 36 (51.4%) | 35 (9.8%) | P < 0.001 (S) | 5 (35.71%) | 24 (9.48%) | 6.91 df = 1 | 0.002 (S) |
| Apgar score <7 at 1 min | 83 (22.86%) | 958 (12.32%) | < 0.001 (S) | 43 (61.3%) | 30 (8.3%) | P < 0.001 (S) | 3 (21.42%) | 37 (14.26%) | 0.096 df = 1 | 0.49 (NS) |
| Apgar score <7 at 5 min | 10 (2.75%) | 122 (1.56%) | 0.14 (NS) | - | - | - | 1 (7.14%) | 4 (1.58%) | 0.232 df = 1 | 0.14 (NS) |
| NICU admission | 28 (7.71%) | 1135 (14.59%) | <0.001 (S) | - | - | - | 3 (21.42%) | 15 (5.92%) | 2.903 df = 1 | 0.024 (S) |

TNC- Tight nuchal cord

S - Significant

NS- Non significant

According to JD Kemfang Ngowa[94] and Dhar KK et al [87], the rate of low agpar score at <7 at 1 min is significantly higher in the Tight nuchal cord group as

compared to control group ,however the rate of low apgar score at 5 min decreased in the Tight nuchal cord group but remained non significantly higher than in the control group. Also in his study , the rate of Nicu ( Neonatal Intensive Care Unit) admissions were significantly higher in the Tight nuchal cord group as compared to control group.

Our findings somewhat correlate with him as we found higher incidence of 1 minute and 5 minute apgar <7 in Tight nuchal cord group as compared to control group but non significantly while our Nicu (Neonatal Intensive Care Unit) admission rate was comparable to his study as we had significant admission rate in the Tight nuchal cord group. Our findings correlate with him.

According to Dhar K K et al[87], the incidence of fetal distress was found significantly higher in the Tight nuchal cord group.

**Outcome Of Infants With Multiple Nuchal Loops**

| | Onderoglu et al[31] | | | Our study | | | |
|---|---|---|---|---|---|---|---|
| | Multiple nuchal cord group | Control group | P value | Multiple nuchal cord group N= 4 | Control group n=253 | Chi sq | P value |
| Apgar score <7 at 1 min | 28.1% | 9.2% | 0.007 | 2 (50%) | 18(7.11%) | 5.0003 | 0.0014(S) |

S - Significant

According to Ongeroglu LS, Dursun P[31], the number of neonates with apgar score <7 at 1 min was significantly higher in neonates with multiple nuchal cords. Intrapartum abnormalities were also more in frequently seen in newborns with multiple nuchal cords, and his study concluded that multiple nuchal cords increase the development of intrapartum complications and lower apgar scores.

Our study also found significantly lower apgar score <7 at 1 minute in neonates with multiple nuchal loops.

Also,Larson JD et al[44] have reported that pregnancies with multiple entanglement were more likely to exhibit an abnormal fetal heart rate pattern and are more likely to have meconium p=0.013 and low 1 min apgar score p<0.001 and an increased need for operative delivery.

Also, according to Jauneaux E et al[91], multiple looping of the umbilical cord around neck was the main factor accounting for the higher incidence of complications like higher incidence of apgar score <7 at 1 min, meconium stained amniotic fluid,

emergency caesarean section, need for neonatal resuscitation, admission to NICU( Neonatal Intensive Care Unit) and perinatal deaths.

## Comparision of Foetal Outcome between Loose Nuchal Cord And Tight Nuchal Cord

| | Singh Gurmesh et al[84] | | | | Dhar KK et al[87] | | | | Our study | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | LNC group | TNC group | Chi sq | P value | LNC group | TNC group | Chi sq | P value | LNC group | TNC group | Chi sq | P value |
| | n=97 | n=39 | | | n= 108 | n= 70 | | | n=33 | n=14 | 4.27 | 0.03 |
| Meconium stained | 4<br>4.12% | 4<br>10.26 % | 1.889 | 0.2 | - | - | - | - | 1<br>3.03% | 3<br>21.42 % | 6.81 | 0.009 |
| Foetal distress | 7<br>7.22% | 8<br>20.51 % | 5.011 | 0.05 | 20 (18.5%) | 36 (51.4% ) | - | P <0.01<br>(S) | 2<br>6.06% | 5<br>35.71 % | 4.27 | 0.03 |
| 1 min apgar <7 | 2<br>2.06% | 8<br>20.51 % | 13.901 1 | 0.001 | 7 (6.4%) | 43 (61.3% ) | - | P <0.001<br>(S) | 1<br>3.03% | 3<br>21.42 % | | |
| 5 min apgar <7 | 1<br>1.03% | 3<br>7.69% | 4.324 | 0.05 | | | | | 0 | 1<br>7.14% | | |
| Still births | 0 | 0 | | | 0 | 5 | - | - | | | | |



LNC= Loose nuchal cord

TNC= Tight nuchal cord

S = Significant

We agree with the findings of Singh Gurmesh et al[84] that the subgroup having tight nuchal cord had significantly higher proportion of fetal distress and low apgar scores at 1 and 5 minutes ,though our 5 minute apgar score <7 was non significantly high. According to Adinma JIB[93] the perinatal mortality is higher in tight nuchal cord group due to progressive diminution of blood supply to foetus leading to a stage of total acute asphyxia.

According to Kumari et al[89], out of 13 still births in her study population of 258, Tight nuchal cord was seen in 144, and the study concluded that tight nuchal cord was

associated with a higher perinatal mortality as compared to loose nuchal cord. Our findings do not correlate with this as we did not have any still birth and also because our sample size was small.

According to Kumari et al[89], the reason for the higher perinatal mortality in the Tight nuchal cord group was that neonates with Tight nuchal cord are deprived of upto 20% of their blood volume by feto-placental transfusion. The cord gets compressed as it encircles the fetal neck.

In our study we found that fetal distress (p=0.009) and 1 min apgar score <7 at 1 min was significantly higher in the Tight nuchal cord group i.e. p= 0.03 as compared to the Loose nuchal cord group but the 5 min apgar score <7 was not significantly higher (with p>0.05), this implies that birth asphyxia is as a result of cord compression during labour in the presence of Tight nuchal cord , but the decrease of low apgar score <7 at 5 min in the Tight nuchal cord group indicates that primary neonatal adaptation is not impaired by Tight nuchal cord. These findings are similar to those of some other authors i.e. JD Kemfang Ngowa et al[94], JD Larson[44], WF Rayburn et al[44], L Schaffer et al[57], NS Shreshtha et al[85], Singh Gurmesh et al.[84]

According to Dhar KK et al[87], the incidence of fetal distress and 1 min apgar <7 were significantly higher in the Tight nuchal cord group as compared to Loose nuchal cord group. Our findings correlate. Also in his study he found 5 still births and one early neonatal death in the Tight nuchal cord group while in our study we found no stillbirth but one early neonatal death in NICU (Neonatal Intensive Care Unit )in the Tight nuchal cord group. In his study he reported that Loose nuchal cord entanglement does not affect the perinatal outcome adversely. Our findings also reveal the same.

Also Spellacy et al[41] reported significantly lower apgar score in the Tight nuchal cord group at 1 min and Hon as quoted by Spellacy[41] has postulated that it is due to more vagal stimulation caused by tighter coils while he reported no difference in the apgar scores at 5 min between Loose nuchal cord group and Tight nuchal cord group. Our findings correlate with him.

Also in a study by Begum AA et al[97] – the results were the same, i.e. neonates with Tight nuchal cord group showed low 1 min apgar score but there was no stiatistical difference of apgar score at 5 min between Loose nuchal cord group and Tight nuchal cord group.



In a study by JD Kemfang Ngowa[94], apgar score <7 at 1 and 5 min and NICU ( Neonatal Intensive Care Unit )admissions were lower in the Loose nuchal cord group as compared to the control group.

In the Tight nuchal cord group, the rate of low apgar score at 1 min was significantly higher than in the control group ($p<0.001$), however, the rate of low apgar score at 5 minutes was decreased and remained non significantly higher than in the control group (i.e.$p= 0.14$). The NICU (Neonatal Intensive Care Unit) admission rate was also lower in the Tight nuchal cord group as compared to the control group.

As similar to other studies, i.e. JD Kemfang Ngowa[94], JM Mastrobattista[90], E Peregrine et al[96], E. Sheiner[95], NS Shreshtha[85], our study did not find an increase in the rate of transfer to NICU ( Neonatal Intensive Care Unit ) in both the nuchal cord

groups. However limits to our study were that the sample size was low and the analysis did not control for some confounders of perinatal outcome such as birth weight and fetal presentation.

**BODY LOOP / EXTREMITY LOOP**

| | Sample size | Incidence in % |
|---|---|---|
| Spellacy et al[41] | 17,190 | 2% |
| W.F. Miser[42] | 706 | 1.84% |
| Kumari et al [89] | 12,000 | 0.008% |
| Collins[2] | - | <0.5% |
| Our study | 300 | 2.33% |

According to Collins[2], body loops can exist with nuchal cords. The effect of a body loop is cord compression.[2]



We also had one case in which there was a body loop along with Tight nuchal cord, and an extremity loop, who was taken for emergency lower segment caesarean section , had one minute and 5 min Apgar score<7 , was actively resuscitated and was admitted in Neonatal Intensive Care Unit and who died of sepsis 5 days later. The possible reason was birth asphyxia due to cord compression of the umbilical cord around the neck as well as extremity and body.

Collins[2] has quoted about 4 published studies which specified on body loop with an incidence of <0.5% and a 10% stillbirth rate.

Our study did not have any stillbirth and incidence was higher – 2.33%

As quoted by Collins[2], a prospective study using Doppler, NST and imaging for body loops reported an occurrence of body loop of 23.23% with a fetal distress rate of 85.29%, with a Caesarean section rate of 88.28%.

Our study findings do not correlate with this statement of Collins.

According to Spellacy,[41] cord around the body are associated with long cords .[41]

Our study correlates as we found longer mean umbilical cord length in cases of Umbilical Cord Accident group as compared to control group.

**TRUE KNOTS**

| | Sample size | Incidence | Still birth rate |
|---|---|---|---|
| Kumari et al[89] | 12,000 | 0.08% | - |
| Spellacy et al[41] | 17,190 | 1% | 6% |
| Chasnoff, Corkill TF, Di TerLIzzi, Earn AA, Schaffel T et al[60,61,62,63,65] | - | 0.04-1% | 8%-11% |
| Klickstein, Schwartz et al[70] | - | 1.22% | |
| Heifitz SA[27] | - | 0.5% | 8-11% |
| Collins[2] | 55,908 | 1% | 6% |
| Our study | 300 | 0.66% | - |



Our incidence correlates with other studies but we did not have any case of birth asphyxia or still birth in our study, maybe because of low sample size as compared to other studies.

According to Rayburn[66] and Klickstein[70], the incidence of a true knot is 3 times more in long cords. We agree because in our study in cases of umbilical cord accidents, the mean umbilical cord length was found to be greater than the mean umbilical cord length in cases without umbilical cord accidents.

This statement is also supported by Heifitz SA[27], Klickstein[70], Schwartz[70] et al.

According to Spellacy[41], Collins[2] and Browne[29], true knots are one of the causes of severe fetal asphyxia, especially when they become tight.

Spellacy[41] has reported an increase in the incidence of stillbirth rate with true cord knots with a significant decrease in the 1 min apgar score<7.

Our findings do not correlate with these authors but our study goes in support of Chasnoff's[60] findings that true knots do not interfere with venous perfusion when they are loose, because in our study we had 2 loose true knots and both did not have any adverse postnatal outcome.

**CORD PROLAPSE**

| | Sample size | Incidence |
|---|---|---|
| W F Miser[42] | 706 | 0.42% |
| Kumari et al[89] | 12,000 | 0.26% |
| Lange et al[78] | 1,471 | 0.6% |
| Collins[2] | - | 0.5% |
| Dilbaz B [82] | 16,874 | 0.47% |
| Dare F O et al[76] | - | 0.42% |
| Traore Y et al[103] | 16,924 | 0.28% |
| Our study | 300 | 0.66% |



Our incidence correlates with other studies.

Our mean umbilical cord length in cases of umbilical cord accidents is more than in the control group including in cases of Cord Prolapse. Our findings correlate with Mc Laverty[80], as he reported that short cords cannot prolapse. He quoted that, it is virtually impossible for a cord < 45 cm in length to prolapse[80]. Also Mengert and Longwell[79] postulated that if the cord length was more than 75 cm, the incidence of Cord prolapse was six times more than in the entire population.[79]

Our two cases of cord prolapse, both underwent caesarian section. One was a fresh stillbirth and the other had to be actively resuscitated and was admitted to NICU(Neonatal Intensive Care Unit). Thus we confirm the findings of Sarwano E, Disse W S[98] et al, that the risk of complications increase in parallel with the cord length and that Cord prolapse is associated with adverse postnatal outcome.

But , Murphy D J, Mackenzi I Z[83] does not agree with this and in his study found 130 cases of Cord prolapse out of which there were 6 still births and 6 neonatal deaths. There were 120 survivors. He reported that despite high incidence of ominous CTG's(Cardiotocographs) and low apgar scores , the fetal outcome is not as poor as might be expected and mortality is predominantly attributed to congenital anomalies and prematurity rather than birth asphyxia.



Dilbaz B [82] et al in his study on 16,874 deliveries retrospectively found 30 cases of Cord prolapse and identified multiparity, malpresentation, spontaneous rupture Of membranes, birth weight < 2.5 kg, polyhydraminos & bishop > 8 as risk factors for umbilical cord prolapse, Also low apgar score < 7 at 5 min has been reported by Dilbaz B et al[82]. Our findings correlate with his finding.

According to a study by Dare F O[76] et al the incidence of umbilical cord prolapse was higher among the unbooked patients. He also revealed that multiparity, CPD(Cephalo Pelvic Disproportion) , prematurity, spontaneous rupture Of membranes , malpresentation and multiple pregnancy are contributing factors and

he reported increased perinatal Mortality in unbooked patients. Our findings correlate as our both patients were unbooked unregistered multiparous patients with malpresentation with spontaneous rupture of membranes, a [101]In a study by Shakeel A Faiz[101] et al in 55,789 deliveries, 111 patients of Cord prolapse were identified. (93.5%) was the caesarian delivery rate and only 6.5% was the Vaginal delivery rate. 39.6% of babies had low Apgar score <7 and 4.5% of babies had low 5 min Apgar <7. 36.9% babies were admitted in NICU (Neonatal Intensive Care Unit) and there was no perinatal mortality in their study. The higher incidence of Caesarian section was justified as an attempt at fetal salvage.

Our findings somewhat correlate as in our both cases of cord prolapse, Emergency Caesarian Delivery was done and the one that survived had low 1 min and 5 min Apgar score and required NICU( Neonatal Intensive Care Unit) admission.



Similarly Dufor P[102] et al in his observation of 50 case of Cord Prolapse reported 72% caesarian deliveries and obstetrical maneuvers in 28% cases, neonatal mortality in 20/1000 and reported bad fetal prognosis.

Also Trore Y[103] in his study on 16,924 deliveries identified 47 cases of cord prolapse in malpresentation, spontaneous rupture of membranes. 61.7% had caesarian delivery and 36.2 % of fetuses died before 5$^{th}$ minute of life concluding umbilical cord prolapse a grave obstetrical complication that compromises fetal prognosis. We agree with his statement.

## CORD PROLAPSE

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[101]In a study by Shakeel A Faiz[101] et al in 55,789 deliveries, 111 patients of Cord prolapse were identified. (93.5%) was the caesarian delivery rate and only 6.5% was the Vaginal delivery rate. 39.6% of babies had low Apgar score <7 and 4.5% of babies had low 5 min Apgar <7. 36.9% babies were admitted in NICU (Neonatal Intensive Care Unit) and there was no perinatal mortality in their study. The higher incidence of Caesarian section was justified as an attempt at fetal salvage.

Our findings somewhat correlate as in our both cases of cord prolapse, Emergency Caesarian Delivery was done and the one that survived had low 1 min and 5 min Apgar score and required NICU( Neonatal Intensive Care Unit) admission.

Similarly Dufor P[102] et al in his observation of 50 case of Cord Prolapse reported 72% caesarian deliveries and obstetrical maneuvers in 28% cases, neonatal mortality in 20/1000 and reported bad fetal prognosis.

Also Trore Y[103] in his study on 16,924 deliveries identified 47 cases of cord prolapse in malpresentation, spontaneous rupture of membranes. 61.7% had caesarian delivery and 36.2 % of fetuses died before 5$^{th}$ minute of life concluding umbilical cord prolapse a grave obstetrical complication that compromises fetal prognosis. We agree with his statement.

**MARGINAL INSERTION OF PLACENTA**

| | |
|---|---|
| Earn 1951[63] | 15% |
| Fox 1978[28] | 5.6% |
| Benirschke (2000) and Kaufmann[71] | 7% |
| Heifitz SA[27] | 7% |
| Krone 1961[104] | 10% |
| Gerlach 1962[104] | 8% |
| Krone 1965[104] | 7.9% |
| Dorste 1971[104] | 14.7% |
| Agboola(1978)[104] | 9.2% |
| S. Patrak[104] | 8% |
| Our study | 1.66% |



Our incidence is less as compared to other studies maybe because of less sample size.

Fox (1978)[28] did not find any correlation between marginal cord insertion and neonatal asphyxia. Our study findings also correlate with his statement.

In a study by S. Pathak et al [104]– it has been quoted that marginal insertion is more susceptible to vessel rupture and associated with IUGR, stillbirths and neonatal deaths.[104]

But in our study we did not find any such adverse outcome.

## Summary

A cross sectional study of 300 deliveries irrespective of age , parity and mode of delivery excluding pregnancies <34 weeks and > 42 weeks or presence of any medical disorders like heart disease , hypertension, or any renal disorders was conducted in a Tertiary care hospital , between oct 2009 and may 2011 and the five commonly occurring umbilical cord accidents were noted ;

Presence of Nuchal cord (Tight or loose)

Presence of Body loop/ Extremity loop

Presence of Cord Prolapse

Presence of True Knots

Presence of Marginal Insertion of Placenta



Their affect on the immediate postnatal outcome in terms of the following was studied;

Stillbirth

1 minute apgar score <7

Neonates if required resuscitation measures like ambubag, endotracheal intubation , chest compressions or use of drugs.

Neonates if required Neonatal Intensive Care Unit (Nicu) admissions.

The results are as follows:

1. The age and the parity were found to be statistically non significant between the umbilical cord accident group and the control group.
2. The mean umbilical cord length found in cases of umbilical cord accident group was found to be statistically significant as compared to the control group , that is cases with umbilical cord accident tend to have longer cords as compared to the control group.
3. Vaginal delivery was found to be statistically significant in the nuchal cord group as compared to the control group while other modes of delivery were non significant.
4. The fetal outcome in terms of stillbirths, meconium stained liquor, fetal distress, 1 minute apgar <7 , 5 min apgar < 7 , neonates requiring active resuscitation like ambubag, endotracheal intubation , chest compressions or use of drugs and Nicu (Neonatal Intensive Care Unit) admissions was found to be statistically non significant between the nuchal cord group and the control group.
5. Vaginal delivery was found to be statistically significant in the loose nuchal cord group as compared to the control group.
6. Emergency caesarian section was found to be higher but statistically non significant in the control group as compared to the loose nuchal cord group.
7. Vaginal delivery was found to be non significantly higher in the tight nuchal cord group as compared to the control group.

8. Emergency caesarian section and Instrumental delivery were found to be statistically non significant in the tight nuchal cord group as compared to the control group.
9. The mode of delivery was found to be statistically non significant between the loose nuchal cord group and the tight nuchal cord group.
10. The loose nuchal cord group and the control group did not have any statistical difference as regards the fetal outcome.
11. While comparing the fetal outcome between the tight nuchal cord group and the control group, the tight nuchal cord group had significant fetal distress and significant number of Nicu (Neonatal Intensive Care Unit) admissions.
12. In terms of fetal outcome between the loose nuchal cord group and the tight nuchal cord group there were no stillbirths in both the groups
    - The fetal distress was found to be statistically more in the tight nuchal cord group as compared to the loose nuchal cord group.
    - Meconium stained liquor was found to be significantly more in the tight nuchal cord group as compared to the loose nuchal cord group.
    - 1 minute apgar score <7 was also found to be statistically more in the tight nuchal cord group as compared to the loose nuchal cord group.
    - 5 minute apgar score < 7 was not statistically significant between the two groups.
    - Neonates requiring active resuscitation measures and Nicu (Neonatal Intensive Care Unit) admissions were also found to be statistically significant in the tight nuchal cord group.

13. We found significantly lower 1 minute apgar score <7 in neonates with multiple nuchal loops as compared to the control group.
14. We found two loops of nuchal cord around the neck in three cases, two cases had tight loops around the neck and one case had loose loops around the neck. The neonates with tight loops had one minute apgar<7 and were actively resuscitated and required Nicu (Neonatal Intensive Care Unit) admission , while the one with two loose loops did not have any adverse postnatal outcome
15. We had one case of three loose loops of nuchal cord around the neck , this neonate had fetal distress and 1 minute apgar score < 7 but did not require resuscitation or Nicu (Neonatal Intensive Care Unit) admission.
16. We had seven cases of body loop/ extremity loop , out of which only the one with an associated nuchal cord had fetal distress , 1 min and 5 min apgar score < 7 , was actively resuscitated ,was admitted to Nicu(Neonatal Intensive Care Unit) and who died in Nicu(Neonatal Intensive Care Unit) 5 days later due to sepsis. The other 6 did not have any adverse postnatal outcome.
17. We had two cases of true knots , both were loose true knots ans no adverse postnatal outcome was noted in any of the cases.
18. We had two cases of cord prolapse . Both the unregistered cases and both were taken for emergency caesarian section, one was a fresh stillbirth, the other had a poor postnatal outcome requiring Nicu (Neonatal Intensive Care Unit)admission.
19. We had five cases of Marginal insertion of placenta an in all five no adverse postnatal outcome was observed.

## CONCLUSION

Umbilical Cord Accidents are usually incidental findings , but they may aid in explaining certain cases of adverse postnatal outcome especially the tight nuchal cord . They can lead to significant fetal distress and low 1 minute apgar of <7 due to vagotonia produced due to tighter coils around the neck. Since the 5 minute apgar score is not significantly affected, this implies that neonatal adaptation is not impaired by the tight nuchal cord . The adverse Immediate postnatal outcome is transient , but cases of tight nuchal cord sometimes do require resuscitation and Nicu (Neonatal Intensive Care Unit) admissions.

Umbilical Cord Prolapse is an infrequent Obstetrical Complication that usually necessitates emergent Delivery to salvage the fetus as it increases the fetal hypoxemia and asphyxia.



In our study it had an adverse postnatal outcome in both the cases.

The loose nuchal cord, loose true knots, presence of body Loop/extremity loop and marginal insertion of placenta do not seem to have any adverse postnatal outcome according to our study.

Thus , overall the Umbilical Cord Accidents do not require any radical obstetric management. They only require close monitoring during labor if detected in the antenatal period on Doppler Sonography.

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ANNEXURE I

**PREFORMA**

- Name of the patient –
- Age -
- Address of the patient-

- IPD no -
- Date of delivery-
- Chief complaints of the patient-

- Menstrual history



- LMP- Gestational Age by Date -
- EDD-

- Past menstrual history-

- Obstetrical history

| Sr no | Duration of pregnancy | Mode of delievery | Age of baby | Birth wt | Complications if any |
|---|---|---|---|---|---|
| | | | | | |
| | | | | | |
| | | | | | |
| | | | | | |
| | | | | | |



- Past history ( any medical disorders)

- Family history(any medical disorders)

- Personal history

- General physical examination;

Height Weight

BP Pulse Rate

Pallor Edema

- Systemic examination

CVS -

CNS-

RS-

PA-

Investigations

- Hb -
- Blood group-
- Urine (routine& microscopy)-
- HIV/ HBSAG/ VDRL status-
- Detailed USG record of ANC period

| USG serial number | Date | Gestational period by LMP | Gestational period by USG | Growth lag if any | Any other significant finding |
|---|---|---|---|---|---|
| | | | | | |
| | | | | | |
| | | | | | |

- Any sign of IUGR from clinical examination/ USG/ colour doppler

- Labor record
- Duration of first stage of labor
- Complications if any
- Prolapse of cord (y /n)
- Duration of second stage of labor
- Complications if any
- Color of liquor
- Prolapse of cord(y/n)
- Presence of nuchal cord (360 degree around the neck)-

  tight/loose-

  no of loops
- Presence of any body/ extremity loop-
- Duration of third stage of labor
- Complications if any
- Condition of placenta a) weight-

  b) retroplacental clot-

  c) infarcts

  d) calcification

- Umbilical cord a)True knot

  b)False knot

- Length of cord in cm-
- Presence of marginal insertion of cord -
- Any other abnormal finding of cord -

"Postnatal outcome"

- Whether baby cried immediately after birth-(y/n)-
- Meconium staining of umbilical cord(y/n)-

- 1 min apgar score-
- 5 min apgar score-

- Neonates who required active resuscitation measures like :
    - Intermittent positive pressure ventilation like ( ambubag or Endotracheal (et) intubation) or chest compressions or use of drugs in resuscitation - (y/n)-
    - If yes which measure was taken-
    - If neonate required NICU admission-(y/n)-
    - If yes then what was the indication of NICU admission -

ANNEXURE II

## MASTER CHART

| Id | Age | Parity | Cord length (cm) | MOD | Nuchal Cord | TNC | LNC | No of Loops | BODY Loop | CORD prolapse | MIP | TRUE knots | Still Births | 1 AI |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 1 | 1 | 48 | 2 | n | n | n | n | n | n | n | n | n | |
| 2 | 3 | 2 | 52 | 2 | y | n | y | 1 | n | n | n | n | n | |
| 3 | 3 | 1 | 49 | 2 | n | n | n | n | n | n | n | n | n | |
| 4 | 3 | 2 | 54 | 2 | n | n | n | n | n | y | n | n | n | |
| 5 | 3 | 2 | 54 | 1 | n | n | n | n | n | n | n | n | n | |
| 6 | 2 | 1 | 46 | 2 | n | n | n | n | n | n | n | n | n | |
| 7 | 1 | 1 | 60 | 2 | y | y | n | n | y | n | n | n | n | |
| 8 | 2 | 2 | 61 | 1 | n | n | n | n | n | n | n | y-loose | n | |
| 9 | 2 | 1 | 50 | 1 | n | n | n | n | n | n | n | n | n | |
| 10 | 3 | 2 | 46 | 3-forcep | n | n | n | n | n | n | n | n | n | |
| 11 | 3 | 2 | 52 | 1 | n | n | n | n | n | n | n | n | n | |
| 12 | 2 | 2 | 58 | 1 | n | n | n | n | n | n | y | n | n | |
| 13 | 3 | 2 | 47 | 1 | n | n | n | n | n | n | n | n | n | |
| 14 | 2 | 2 | 72 | 2 | n | n | n | n | n | y | n | n | y | |
| 15 | 3 | 1 | 45 | 1 | n | n | n | n | n | n | n | n | n | |
| 16 | 3 | 2 | 54 | 1 | n | n | n | n | n | n | n | n | n | |
| 17 | 2 | 1 | 46 | 1 | n | n | n | n | n | n | n | n | n | |
| 18 | 2 | 1 | 56 | 2 | n | n | n | n | n | n | n | y-loose | n | |
| 19 | 3 | 2 | 55 | 1 | n | n | n | n | y | n | y | n | n | |
| 20 | 3 | 2 | 53 | 3-vento | n | n | n | n | n | n | n | n | n | |
| 21 | 3 | 2 | 51 | 2 | n | n | n | n | n | n | n | n | n | |
| 22 | 2 | 2 | 62 | 1 | y | n | y | n | y | n | n | n | n | |
| 23 | 3 | 1 | 62 | 1 | n | n | n | n | y | n | n | n | n | |
| 24 | 3 | 2 | 51 | 1 | n | n | n | n | n | n | n | n | n | |
| 25 | 3 | 1 | 45 | 2 | n | n | n | n | n | n | n | n | n | |
| 26 | 3 | 1 | 71 | 2 | y | n | y | 3 | y | n | n | n | n | |
| 27 | 3 | 2 | 50 | 1 | n | n | n | n | n | n | y | n | n | |
| 28 | 2 | 2 | 69 | 2 | y | y | n | 1 | y | n | n | n | n | |
| 29 | 2 | 1 | 71 | 1 | y | n | y | 2 | y | n | n | n | n | |
| 30 | 3 | 1 | 49 | 2 | n | n | n | n | n | n | n | n | n | |
| 31 | 3 | 2 | 52 | 1 | n | n | n | n | n | n | n | n | n | |
| 32 | 3 | 2 | 52 | 1 | y | y | n | 1 | n | n | n | n | n | |
| 33 | 3 | 1 | 52 | 1 | n | n | n | n | n | n | n | n | n | |
| 34 | 2 | 2 | 62 | 3- | y | y | n | n | n | n | n | n | n | |

| | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | forcep | | | | | | | | | | |
| 35 | 3 | 2 | 50 | 2 | n | n | n | n | n | n | n | n | n | |
| 36 | 3 | 2 | 55 | 3-vento | y | n | y | 1 | n | n | n | n | n | |
| 37 | 2 | 1 | 46 | 1 | n | n | n | n | n | n | n | n | n | |
| | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | |
| Id | Age | Parity | Cord length (cm) | MOD | Nuchal Cord | TNC | LNC | No of Loops | BODY Loop | CORD prolapse | MIP | TRUE knots | Still Births | 1 AI |
| 38 | 2 | 2 | 55 | 1 | n | n | n | n | n | n | n | n | n | |
| 39 | 3 | 2 | 50 | 1 | n | n | n | n | n | n | n | n | n | |
| 40 | 3 | 1 | 71 | 1 | y | n | y | 2 | n | n | n | n | n | |
| 41 | 2 | 2 | 51 | 1 | n | n | n | n | n | n | n | n | n | |
| 42 | 3 | 1 | 50 | 1 | n | n | n | n | n | n | n | n | n | |
| 43 | 3 | 2 | 49 | 3-forcep | n | n | n | n | n | n | n | n | n | |
| 44 | 3 | 2 | 50 | 2 | n | n | n | n | n | n | n | n | n | |
| 45 | 3 | 2 | 55 | 1 | n | n | n | n | n | n | y | n | n | |
| 46 | 2 | 1 | 51 | 3-vento | n | n | n | n | n | n | n | n | n | |
| 47 | 3 | 2 | 57 | 1 | n | n | n | n | n | n | y | n | n | |
| 48 | 2 | 1 | 69 | 2 | y | y | n | 1 | n | n | n | n | n | |
| 49 | 3 | 2 | 49 | 3-vento | n | n | n | n | n | n | n | n | n | |
| 50 | 3 | 2 | 55 | 1 | n | n | n | n | n | n | n | n | n | |
| 51 | 2 | 2 | 48 | 1 | n | n | n | n | n | n | n | n | n | |
| 52 | 3 | 1 | 49 | 1 | n | n | n | n | n | n | n | n | n | |
| 53 | 3 | 2 | 51 | 1 | n | n | n | n | n | n | n | n | n | |
| 54 | 2 | 1 | 51 | 1 | n | n | n | n | n | n | n | n | n | |
| 55 | 3 | 1 | 58 | 2 | y | y | n | 1 | n | n | n | n | n | |
| 56 | 3 | 1 | 49 | 2 | n | n | n | n | n | n | n | n | n | |
| 57 | 2 | 1 | 45 | 1 | n | n | n | n | n | n | n | n | n | |
| 58 | 3 | 1 | 54 | 1 | n | n | n | n | n | n | n | n | n | |
| 59 | 2 | 2 | 50 | 1 | n | n | n | n | n | n | n | n | n | |
| 60 | 3 | 2 | 64 | 1 | y | n | y | n | n | n | n | n | n | |
| 61 | 3 | 1 | 52 | 2 | n | n | n | n | n | n | n | n | n | |
| 62 | 2 | 2 | 53 | 3-vento | n | n | n | n | n | n | n | n | n | |
| 63 | 3 | 2 | 47 | 2 | n | n | n | n | n | n | n | n | n | |
| 64 | 2 | 2 | 48 | 1 | n | n | n | n | n | n | n | n | n | |
| 65 | 2 | 2 | 47 | 1 | n | n | n | n | n | n | n | n | n | |
| 66 | 3 | 1 | 48 | 3-vento | n | n | n | n | n | n | n | n | n | |
| 67 | 2 | 1 | 55 | 2 | n | n | n | n | n | n | n | n | n | |
| 68 | 3 | 1 | 49 | 1 | n | n | n | n | n | n | n | n | n | |

| | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 69 | 2 | 2 | 48 | 1 | n | n | n | n | n | n | n | n | n | |
| 70 | 3 | 1 | 60 | 1 | y | n | y | 1 | n | n | n | n | n | |
| 71 | 1 | 1 | 59 | 1 | y | y | n | 1 | n | n | n | n | n | |
| 72 | 2 | 1 | 63 | 1 | y | n | y | 1 | n | n | n | n | n | |

| Id | Age | Parity | Cord length (cm) | MOD | Nuchal Cord | TNC | LNC | No of Loops | BODY Loop | CORD prolapse | MIP | TRUE knots | Still Births | 1 AI |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 73 | 3 | 1 | 48 | 1 | n | n | n | n | n | n | n | n | n | |
| 74 | 2 | 1 | 49 | 2 | n | n | n | n | n | n | n | n | n | |
| 75 | 3 | 2 | 53 | 1 | n | n | n | n | n | n | n | n | NA | |
| 76 | 3 | 2 | 45 | 1 | n | n | n | n | n | n | n | n | n | |
| 77 | 3 | 2 | 48 | 1 | n | n | n | n | n | n | n | n | n | |
| 78 | 2 | 1 | 47 | 3-vento | n | n | n | n | n | n | n | n | n | |
| 79 | 2 | 1 | 53 | 1 | n | n | n | n | n | n | n | | n | |
| 80 | 3 | 1 | 53 | 1 | n | n | n | n | n | n | n | n | n | |
| 81 | 4 | 2 | 55 | 1 | y | n | y | 1 | n | n | n | n | n | |
| 82 | 3 | 2 | 52 | 2 | n | n | n | n | n | n | n | n | n | |
| 83 | 3 | 1 | 53 | 1 | n | n | n | n | n | n | n | n | n | |
| 84 | 3 | 2 | 46 | 1 | n | n | n | n | n | n | n | n | n | |
| 85 | 2 | 2 | 45 | 1 | n | n | n | n | n | n | n | n | n | |
| 86 | 3 | 2 | 46 | 1 | n | n | n | n | n | n | n | n | n | |
| 87 | 2 | 2 | 53 | 1 | y | n | y | 1 | n | n | n | n | n | |
| 88 | 3 | 1 | 45 | 1 | n | n | n | n | n | n | n | n | n | |
| 89 | 2 | 2 | 49 | 1 | n | n | n | n | n | n | n | n | n | |
| 90 | 3 | 1 | 47 | 1 | n | n | n | n | n | n | n | n | n | |
| 91 | 3 | 2 | 45 | 2 | n | n | n | n | n | n | n | n | n | |
| 92 | 2 | 2 | 56 | 1 | y | n | y | 1 | n | n | n | n | n | |
| 93 | 2 | 1 | 56 | 2 | y | n | y | 1 | n | n | n | n | n | |
| 94 | 3 | 2 | 50 | 1 | n | n | n | n | n | n | n | n | n | |
| 95 | 2 | 1 | 51 | 1 | n | n | n | n | n | n | n | n | n | |
| 96 | 3 | 2 | 52 | 1 | n | n | n | n | n | n | n | n | n | |
| 97 | 2 | 2 | 49 | 3-forcep | n | n | n | n | n | n | n | n | n | |
| 98 | 3 | 2 | 47 | 3-vento | n | n | n | n | n | n | n | n | n | |
| 99 | 3 | 2 | 48 | 1 | n | n | n | n | n | n | n | n | n | |
| 100 | 3 | 1 | 55 | 2 | n | n | n | n | n | n | n | n | n | |
| 101 | 3 | 2 | 51 | 2 | n | n | n | n | n | n | n | n | n | |
| 102 | 3 | 1 | 52 | 1 | n | n | n | n | n | n | n | n | n | |
| 103 | 2 | 2 | 55 | 1 | n | n | n | n | n | n | n | n | n | |
| 104 | 3 | 2 | 47 | 3-vento | n | n | n | n | n | n | n | n | n | |

| | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 105 | 3 | 2 | 49 | 2 | n | n | n | n | n | n | n | n | n | |
| 106 | 2 | 2 | 55 | 1 | y | n | y | 1 | n | n | n | | n | |
| 107 | 3 | 2 | 45 | 1 | n | n | n | n | n | n | n | n | n | |
| 108 | 2 | 2 | 46 | 1 | n | n | n | n | n | n | n | n | n | |
| | | | | | | | | | | | | | | |

| Id | Age | Parity | Cord length (cm) | MOD | Nuchal Cord | TNC | LNC | No of Loops | BODY Loop | CORD prolapse | MIP | TRUE knots | Still Births | 1 AI |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 109 | 3 | 2 | 48 | 3-forcep | n | n | n | n | n | n | n | n | n | |
| 110 | 3 | 2 | 52 | 1 | n | n | n | n | n | n | n | n | n | |
| 111 | 3 | 2 | 48 | 1 | n | n | n | n | n | n | n | n | n | |
| 112 | 3 | 2 | 55 | 3-vento | n | n | n | n | n | n | n | n | n | |
| 113 | 3 | 2 | 51 | 1 | n | n | n | n | n | n | n | n | n | |
| 114 | 3 | 1 | 50 | 1 | n | n | n | n | n | n | n | n | n | |
| 115 | 4 | 2 | 54 | 1 | n | n | n | n | n | n | n | n | n | |
| 116 | 3 | 1 | 62 | 1 | y | y | n | 1 | n | n | n | n | n | |
| 117 | 2 | 2 | 50 | 1 | n | n | n | n | n | n | n | n | n | |
| 118 | 3 | 2 | 45 | 1 | n | n | n | n | n | n | n | n | n | |
| 119 | 2 | 2 | 51 | 1 | n | n | n | n | n | n | n | n | n | |
| 120 | 3 | 1 | 48 | 3-vento | n | n | n | n | n | n | n | n | n | |
| 121 | 3 | 2 | 45 | 1 | n | n | n | n | n | n | n | n | n | |
| 122 | 2 | 1 | 54 | 1 | n | n | n | n | n | n | n | n | n | |
| 123 | 3 | 1 | 47 | 1 | n | n | n | n | n | n | n | n | n | |
| 124 | 2 | 2 | 49 | 1 | n | n | n | n | n | n | n | n | n | |
| 125 | 3 | 1 | 46 | 1 | n | n | n | n | n | n | n | n | n | |
| 126 | 3 | 2 | 54 | 2 | n | n | n | n | n | n | n | n | n | |
| 127 | 3 | 2 | 47 | 2 | n | n | n | n | n | n | n | n | n | |
| 128 | 3 | 2 | 62 | 1 | y | n | y | 1 | n | n | n | n | n | |
| 129 | 3 | 2 | 45 | 1 | n | n | | n | n | n | n | n | n | |
| 130 | 2 | 1 | 47 | 1 | n | n | n | n | n | n | n | n | n | |
| 131 | 2 | 2 | 45 | 1 | n | n | n | n | n | n | n | n | n | |
| 132 | 2 | 2 | 54 | 1 | n | n | n | n | n | n | n | n | n | |
| 133 | 3 | 1 | 54 | 1 | n | n | n | n | n | n | n | n | n | |
| 134 | 2 | 1 | 63 | 1 | y | y | n | 1 | n | n | n | n | n | |
| 135 | 3 | 2 | 52 | 1 | n | n | n | n | n | n | n | n | n | |
| 136 | 3 | 1 | 54 | 1 | n | n | n | n | n | n | n | n | n | |
| 137 | 2 | 2 | 55 | 3-vento | n | n | n | n | n | n | n | n | n | |
| 138 | 3 | 1 | 53 | 1 | n | n | n | n | n | n | n | n | n | |
| 139 | 3 | 1 | 51 | 1 | n | n | n | n | n | | n | n | n | |
| 140 | 2 | 1 | 51 | 1 | n | n | n | n | n | n | n | n | n | |
| 141 | 2 | 1 | 47 | 2 | n | n | n | n | n | n | n | n | n | |

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 142 | 3 | 2 | 50 | 1 | n | n | n | n | n | n | | n | n |
| 143 | 3 | 2 | 51 | 1 | n | n | n | n | n | n | | n | n |

| Id | Age | Parity | Cord length (cm) | MOD | Nuchal Cord | TNC | LNC | No of Loops | BODY Loop | CORD prolapse | MIP | TRUE knots | Still Births | 1 AP |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 144 | 4 | 2 | 49 | 1 | n | n | n | n | n | n | n | n | n | |
| 145 | 2 | 2 | 52 | 2 | n | n | n | n | n | n | n | n | n | |
| 146 | 4 | 2 | 45 | 1 | n | n | n | n | n | n | n | n | n | |
| 147 | 3 | 2 | 52 | 1 | y | n | y | 1 | n | n | n | n | n | |
| 148 | 3 | 2 | 51 | 1 | n | n | n | n | n | n | n | n | n | |
| 149 | 2 | 1 | 49 | 1 | n | n | n | n | n | n | n | n | n | |
| 150 | 3 | 1 | 53 | 1 | n | n | n | n | n | n | n | n | n | |
| 151 | 2 | 2 | 45 | 1 | n | n | n | n | n | n | n | n | n | |
| 152 | 2 | 2 | 52 | 1 | n | n | n | n | n | n | n | n | n | |
| 153 | 4 | 2 | 62 | 2 | y | y | n | 1 | n | n | n | n | n | |
| 154 | 2 | 1 | 46 | 1 | n | n | n | n | n | n | n | n | n | |
| 155 | 2 | 1 | 53 | 1 | n | n | n | n | n | n | n | n | n | |
| 156 | 3 | 2 | 47 | 1 | n | n | n | n | n | n | n | n | n | |
| 157 | 2 | 1 | 55 | 1 | y | n | y | 1 | n | n | n | n | n | |
| 158 | 2 | 1 | 51 | 3-vento | n | n | n | n | n | n | n | n | n | |
| 159 | 2 | 1 | 55 | 1 | n | n | n | n | n | n | n | n | n | |
| 160 | 2 | 1 | 45 | 2 | n | n | n | n | n | n | n | n | n | |
| 161 | 3 | 2 | 55 | 1 | n | n | n | n | n | n | n | n | n | |
| 162 | 2 | 2 | 46 | 1 | n | n | n | n | n | n | n | n | n | |
| 163 | 4 | 2 | 49 | 1 | n | n | n | n | n | n | n | n | n | |
| 164 | 3 | 2 | 55 | 2 | n | n | n | n | n | n | n | n | n | |
| 165 | 2 | 2 | 48 | 1 | n | n | n | n | n | n | n | n | n | |
| 166 | 3 | 2 | 45 | 1 | y | n | y | 1 | n | n | n | n | n | |
| 167 | 2 | 2 | 53 | 1 | n | n | n | n | n | n | n | n | n | |
| 168 | 3 | 2 | 48 | 2 | n | n | n | n | n | n | n | n | n | |
| 169 | 2 | 2 | 54 | 1 | n | n | n | n | n | n | n | n | n | |
| 170 | 3 | 1 | 46 | 1 | n | n | n | n | n | n | n | n | n | |
| 171 | 2 | 2 | 45 | 1 | n | n | n | n | n | n | n | n | n | |
| 172 | 3 | 1 | 54 | 1 | n | n | n | n | n | n | n | n | n | |
| 173 | 2 | 1 | 46 | 1 | n | n | n | n | n | n | n | n | n | |
| 174 | 2 | 2 | 45 | 1 | n | n | n | n | n | n | n | n | n | |
| 175 | 2 | 2 | 52 | 1 | y | n | y | 1 | n | n | n | n | n | |
| 176 | 3 | 1 | 50 | 3-forcep | n | n | n | n | n | n | n | n | n | |
| 177 | 1 | 1 | 46 | 1 | n | n | n | n | n | n | n | n | n | |

| Id | Age | Parity | Cord length (cm) | MOD | Nuchal Cord | TNC | LNC | No of Loops | BODY Loop | CORD prolapse | MIP | TRUE knots | Still Births | 1 AI |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 178 | 2 | 1 | 51 | 1 | n | n | n | n | n | n | n | n | n | |
| 179 | 3 | 2 | 54 | 1 | n | n | n | n | n | n | n | n | n | |
| 180 | 3 | 2 | 48 | 1 | n | n | n | n | n | n | n | n | n | |
| 181 | 3 | 2 | 53 | 1 | n | n | n | n | n | n | n | n | n | |
| 182 | 2 | 1 | 56 | 1 | y | n | y | 1 | n | n | n | n | n | |
| 183 | 3 | 1 | 48 | 1 | n | n | n | n | n | n | n | n | n | |
| 184 | 2 | 2 | 52 | 1 | y | y | n | 1 | n | n | n | n | n | |
| 185 | 3 | 1 | 51 | 2 | n | n | n | n | n | n | n | n | y | |
| 186 | 2 | 2 | 46 | 3-forcep | n | n | n | n | n | n | n | n | n | |
| 187 | 3 | 2 | 55 | 1 | n | n | n | n | n | n | n | n | n | |
| 188 | 2 | 2 | 49 | 2 | n | n | n | n | n | n | n | n | NA | |
| 189 | 2 | 2 | 48 | 3-vento | n | n | n | n | n | n | n | n | n | |
| 190 | 3 | 1 | 51 | 2 | n | n | n | n | n | n | n | n | n | |
| 191 | 2 | 2 | 50 | 1 | n | n | n | n | n | n | n | n | n | |
| 192 | 2 | 2 | 56 | 1 | y | n | y | 1 | n | n | n | n | n | |
| 193 | 3 | 1 | 53 | 1 | y | n | y | 1 | n | n | n | n | n | |
| 194 | 2 | 2 | 46 | 1 | n | n | n | n | n | n | n | n | n | |
| 195 | 2 | 2 | 55 | 2 | n | n | n | n | n | n | n | n | n | |
| 196 | 2 | 1 | 63 | 1 | y | n | y | 1 | n | n | n | n | n | |
| 197 | 2 | 2 | 68 | 1 | y | n | y | 1 | n | n | n | n | n | |
| 198 | 2 | 1 | 68 | 1 | y | y | n | 1 | n | n | n | n | n | |
| 199 | 2 | 1 | 49 | 2 | n | n | n | n | n | n | n | n | n | |
| 200 | 2 | 2 | 61 | 1 | y | n | y | 1 | n | n | n | n | n | |
| 201 | 2 | 2 | 51 | 1 | n | n | n | n | n | n | n | n | n | |
| 202 | 2 | 2 | 51 | 1 | n | n | n | n | n | n | n | n | n | |
| 203 | 2 | 1 | 45 | | n | n | n | n | n | n | n | n | n | |
| 204 | 2 | 1 | 47 | 1 | n | n | n | n | n | n | n | n | n | |
| 205 | 2 | 2 | 52 | 1 | n | n | n | n | n | n | n | n | n | |
| 206 | 2 | 2 | 54 | 1 | n | n | n | n | n | n | n | n | n | |
| 207 | 2 | 1 | 61 | 1 | y | n | y | 1 | n | n | n | n | n | |
| 208 | 2 | 2 | 52 | 1 | n | n | n | n | n | n | n | n | n | |
| 209 | 2 | 2 | 68 | 1 | y | y | n | 1 | n | n | n | n | n | |
| 210 | 2 | 2 | 49 | 3-forcep | n | n | n | n | n | n | n | n | n | |
| 211 | 2 | 2 | 53 | 1 | n | n | n | n | n | n | n | n | n | |
| 212 | 2 | 1 | 46 | 1 | n | n | n | n | n | n | n | n | n | |

| Id | Age | Parity | Cord length (cm) | MOD | Nuchal Cord | TNC | LNC | No of Loops | BODY Loop | CORD prolapse | MIP | TRUE knots | Still Births | 1 AI |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 213 | 2 | 1 | 49 | 2 | n | n | n | n | n | n | n | n | n | y- |
| 214 | 2 | 2 | 54 | 1 | n | n | n | n | n | n | n | n | n | n |
| 215 | 2 | 1 | 45 | 1 | n | n | n | n | n | n | n | n | n | n |
| 216 | 2 | 2 | 60 | 1 | y | n | y | 1 | n | n | n | n | n | n |
| 217 | 2 | 2 | 67 | 1 | y | n | y | 1 | n | n | n | n | n | n |
| 218 | 2 | 1 | 47 | 1 | n | n | n | n | n | n | n | n | n | n |
| 219 | 2 | 2 | 51 | 1 | n | n | n | n | n | n | n | n | n | n |
| 220 | 2 | 1 | 68 | 1 | y | y | n | 1 | n | n | n | n | n | n |
| 221 | 2 | 1 | 49 | 3-forcep | n | n | n | n | n | n | n | n | n | n |
| 222 | 2 | 2 | 60 | 1 | y | y | n | 1 | n | n | n | n | n | n |
| 223 | 2 | 1 | 61 | 1 | y | n | y | 1 | n | n | n | n | n | n |
| 224 | 2 | 2 | 52 | 2 | n | n | n | n | n | n | n | n | n | n |
| 225 | 2 | 2 | 52 | 3-forcep | n | n | n | n | n | n | n | n | n | n |
| 226 | 2 | 2 | 52 | 1 | y | n | y | 1 | n | n | n | n | n | n |
| 227 | 2 | 1 | 52 | 1 | n | n | n | n | n | n | n | n | n | n |
| 228 | 2 | 2 | 54 | 1 | n | n | n | n | n | n | n | n | n | n |
| 229 | 2 | 1 | 49 | 3-vento | n | n | n | n | n | n | n | n | n | n |
| 230 | 2 | 2 | 49 | 1 | n | n | n | n | n | n | n | n | n | n |
| 231 | 2 | 2 | 52 | 2 | n | n | n | n | n | n | n | n | n | n |
| 232 | 2 | 1 | 49 | 2 | n | n | n | n | n | n | n | n | n | n |
| 233 | 2 | 1 | 49 | 1 | n | n | n | n | n | n | n | n | n | n |
| 234 | 2 | 2 | 46 | 1 | n | n | n | n | n | n | n | n | n | n |
| 235 | 2 | 1 | 45 | 1 | n | n | n | n | n | n | n | n | n | n |
| 236 | 2 | 1 | 60 | 1 | y | n | y | 1 | n | n | n | n | n | n |
| 237 | 2 | 2 | 47 | 1 | n | n | n | n | n | n | n | n | n | n |
| 238 | 2 | 1 | 50 | 1 | n | n | n | n | n | n | n | n | n | n |
| 239 | 2 | 2 | 52 | 1 | n | n | n | n | n | n | n | n | n | n |
| 240 | 2 | 1 | 50 | 1 | n | n | n | n | n | n | n | n | n | n |
| 241 | 2 | 2 | 46 | 2 | n | n | n | n | n | n | n | n | n | n |
| 242 | 2 | 2 | 51 | 1 | n | n | n | n | n | n | n | n | n | n |
| 243 | 2 | 1 | 51 | 1 | n | n | n | n | n | n | n | n | n | n |
| 244 | 2 | 2 | 48 | 1 | n | n | n | n | n | n | n | n | n | n |
| 245 | 2 | 1 | 51 | 1 | n | n | n | n | n | n | n | n | n | n |
| 246 | 2 | 2 | 46 | 1 | n | n | n | n | n | n | n | n | n | n |

| Id | Age | Parity | Cord length (cm) | MOD | Nuchal Cord | TNC | LNC | No of Loops | BODY Loop | CORD prolapse | MIP | TRUE knots | Still Births | 1 AI |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 247 | 2 | 2 | 63 | 1 | y | n | y | 1 | n | n | n | n | n | n |
| 248 | 2 | 2 | 51 | 1 | n | n | n | n | n | n | n | n | n | n |
| 249 | 2 | 1 | 48 | 1 | n | n | n | n | n | n | n | n | n | y- |
| 250 | 3 | 2 | 52 | 2 | n | n | n | n | n | n | n | n | n | y- |
| 251 | 2 | 2 | 49 | 1 | n | n | n | n | n | n | n | n | n | n |
| 252 | 2 | 2 | 45 | 1 | n | n | n | n | n | n | n | n | n | n |
| 253 | 2 | 2 | 53 | 1 | n | n | n | n | n | n | n | n | y | NA |
| 254 | 3 | 1 | 53 | 1 | n | n | n | n | n | n | n | n | y | NA |
| 255 | 2 | 2 | 52 | 2 | n | n | n | n | n | n | n | n | n | n |
| 256 | 2 | 1 | 47 | 1 | n | n | n | n | n | n | n | n | n | n |
| 257 | 2 | 1 | 50 | 2 | n | n | n | n | n | n | n | n | n | n |
| 258 | 3 | 2 | 53 | 1 | n | n | n | n | n | n | n | n | n | n |
| 259 | 2 | 2 | 53 | 1 | n | n | n | n | n | n | n | n | n | n |
| 260 | 2 | 1 | 53 | 1 | n | n | n | n | n | n | n | n | n | n |
| 261 | 3 | 2 | 46 | 1 | n | n | n | n | n | n | n | n | n | y- |
| 262 | 2 | 1 | 49 | 1 | n | n | n | n | n | n | n | n | n | n |
| 263 | 2 | 1 | 50 | 2 | n | n | n | n | n | n | n | n | n | n |
| 264 | 3 | 2 | 53 | 2 | n | n | n | n | n | n | n | n | n | n |
| 265 | 2 | 1 | 53 | 1 | n | n | n | n | n | n | n | n | n | n |
| 266 | 2 | 2 | 49 | 1 | n | n | n | n | n | n | n | n | n | n |
| 267 | 2 | 1 | 46 | 1 | n | n | n | n | n | n | n | n | n | n |
| 268 | 2 | 2 | 45 | 2 | n | n | n | n | n | n | n | n | n | n |
| 269 | 1 | 1 | 48 | 2 | n | n | n | n | n | n | n | n | n | y- |
| 270 | 2 | 2 | 46 | 1 | n | n | n | n | n | n | n | n | n | n |
| 271 | 3 | 2 | 53 | 1 | n | n | n | n | n | n | n | n | n | n |
| 272 | 2 | 1 | 48 | 1 | n | n | n | n | n | n | n | n | n | n |
| 273 | 1 | 1 | 55 | 1 | n | n | n | n | n | n | n | n | n | n |
| 274 | 2 | 1 | 46 | 2 | n | n | n | n | n | n | n | n | n | y- |
| 275 | 2 | 2 | 46 | 1 | n | n | n | n | n | n | n | n | n | y- |
| 276 | 2 | 2 | 50 | 1 | n | n | n | n | n | n | n | n | NA | NA |
| 177 | 2 | 1 | 54 | 1 | n | n | n | n | n | n | n | n | n | n |
| 178 | 2 | 2 | 51 | 1 | n | n | n | n | n | n | n | n | n | n |
| 179 | 3 | 1 | 52 | 1 | n | n | n | n | n | n | n | n | n | n |
| 280 | 3 | 2 | 46 | 1 | n | n | n | n | n | n | n | n | n | n |

| Id | Age | Parity | Cord length (cm) | MOD | Nuchal Cord | TNC | LNC | No of Loops | BODY Loop | CORD prolapse | MIP | TRUE knots | Still Births | 1 AI |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 281 | 2 | 1 | 52 | 1 | n | n | n | n | n | n | n | n | n | n |
| 282 | 3 | 2 | 52 | 1 | n | n | n | n | n | n | n | n | n | n |

| | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 283 | 2 | 2 | 52 | 1 | n | n | n | n | n | n | n | n | n | n |
| 284 | 3 | 1 | 47 | 1 | n | n | n | n | n | n | n | n | n | y- |
| 285 | 1 | 1 | 50 | 1 | n | n | n | n | n | n | n | n | n | n |
| 286 | 2 | 2 | 54 | 3-forcep | n | n | n | n | n | n | n | n | NA | N |
| 287 | 2 | 1 | 54 | 1 | n | n | n | n | n | n | n | n | n | n |
| 288 | 2 | 2 | 48 | 1 | n | n | n | n | n | n | n | n | n | n |
| 289 | 3 | 1 | 55 | 1 | n | n | n | n | n | n | n | n | n | n |
| 290 | 2 | 2 | 55 | 1 | n | n | n | n | n | n | n | n | n | y- |
| 291 | 2 | 1 | 55 | 1 | n | n | n | n | n | n | n | n | n | n |
| 292 | 2 | 1 | 55 | 2 | n | n | n | n | n | n | n | n | n | n |
| 293 | 2 | 1 | 45 | 1 | n | n | n | n | n | n | n | n | n | n |
| 294 | 2 | 1 | 50 | 1 | n | n | n | n | n | n | n | n | n | n |
| 295 | 2 | 2 | 49 | 1 | n | n | n | n | n | n | n | n | n | y- |
| 296 | 4 | 2 | 51 | 1 | n | n | n | n | n | n | n | n | y | N |
| 297 | 2 | 1 | 46 | 1 | n | n | n | n | n | n | n | n | n | n |
| 298 | 2 | 1 | 46 | 1 | n | n | n | n | n | n | n | n | n | n |
| 299 | 2 | 1 | 52 | 2 | n | n | n | n | n | n | n | n | n | n |
| 300 | 2 | 1 | 50 | 1 | n | n | n | n | n | n | n | n | n | n |
| | | | | | | | | | | | | | | |

**Key for the master chart**

1. Age - ≤18 years - 1

   19-24 years - 2

   25-30 years – 3

   31-35 years- 4

   >35 years - 5

2. Parity - Primiparous - 1

   Multiparous - 2

3.MOD - Mode of Delievery

4.Nuchal cord - n- No

   y- Yes

5. TNC - Tight Nuchal Cord

6. LNC - Loose Nuchal Cord

7.No of Loops – n - Number of loops

8.Body Loop - n - No

   y- Yes

9.Cord Prolapse - n – No

   y- Yes

10. MIP - Marginal Insertion of Placenta

11.True Knots- n - No

y - Yes

12.Stillbirths- n - No

y - Yes

13. 1 min Apgar <7 - 1 minute apgar sore of less than 7

14. Neonatal Resuscitation- Active neonatal resuscitation required;

am- Ambubag

et- Endotracheal Intubation

cc- Chest Compressions

dr- drugs used

15. NICU Admissions- n – No

y- Yes