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CORRELATION OF UMBILICAL CORD CROSS SECTIONAL AREA WITH BIRTH WEIGHT

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Keywords:
Correlation, Umbilical Cord, Birth Weight etc

Abstract

INTRODUCTION: Over the years, clinical, laboratory and ultrasonographic measurements have been used to estimate foetal weight and studied by various authors.This study is aimed to evaluate the correlation between umbilical cord cross-sectional area with birth weight and to compare large cross-sectional area of umbilical cord with macrosomia.

AIM: To correlate fetal umbilical cord cross-sectional area with birth weight.To compare large cross-sectional area of the umbilical cord with macrosomia.

MATERIALS AND METHODS: Umbilical cord cross-sectional area of 250 antenatal women was evaluated from 34 weeks of gestational age. The outcome measured was birth weight. Weight of newborn was measured immediately after birth. Macrosomia was defined as birth weight >90th percentile in the study group. Descriptive statistical analysis has been carried out in the present study.

RESULTS: Out of 250 antenatal women who were recruited for the study primigravida, multigravidas were 49.6% and 50.40% respectively. there were a total of 25 (10.0%) cases in the lean cord, 13 (5.2%) in the large cord group and 212 (84.8%) in the normal cord group. The study showed that as umbilical cord cross-sectional area increases, mean birth weight also increases.

CONCLUSION: There is a positive correlation between umbilical cord cross-sectional area and birth weight. As umbilical cord cross-sectional area increases, there is increase in mean birth weight.

INTRODUCTION

The umbilical cord is a vital lifeline between the fetus and placenta. It is formed by the fifth week of development and it functions throughout the pregnancy to protect the vessels that travel between the foetus and the placenta. Cord abnormalities can lead to foetal morbidity and mortality. This unique lifeline therefore needs optimal protection which is provided by Wharton's jelly, the coiling of the umbilical vessels and the amniotic fluid.

Any compromise of the foetal blood flow through the umbilical cord vessels can have serious deleterious effects on the health of the foetus and the newborn.

The watchword in obstetrics is agreed to be `watchful expectancy' and `timely intervention.'

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With modern ultrasound techniques it has now become possible to search for abnormalities of the cord before birth. As a consequence there is a renewed interest, and a number of studies done in recent years about abnormalities in umbilical cord.

Neonatal survival depends not only on the gestational age but also on the weight of the infant. Assessment of foetal weight in utero hence, leads to an improved prospective management of high risk pregnancies, thus playing a role in reducing maternal and perinatal morbidity and mortality.

Accurate prediction of foetal weight has been of great interest as it helps the obstetrician to decide whether or not to deliver the foetus and also on the mode of delivery. It has also become increasingly important, especially in preventing mishaps of prematurity, fetopelvic disproportions, induction of labour in high risk pregnancies before term and in detection of intrauterine growth retardation (IUGR).

Hence, a quick, easy and an accurate method of estimating foetal weight in utero would be infact a boon to an obstetrician. Over the years, clinical, laboratory and ultrasonographic measurements have been used to estimate foetal weight and studied by various authors.

This study is aimed to evaluate the correlation between umbilical cord cross-sectional area with birth weight and to compare large cross-sectional area of umbilical cord with macrosomia.

AIMS OF THE STUDY

- To correlate fetal umbilical cord cross-sectional area with birth weight.
- To compare large cross-sectional area of the umbilical cord with macrosomia.

REVIEW OF LITERATURE

The umbilicus, actually a scar, is the only visible memento of our close connection with our mother before birth. This was by means of the umbilical cord, which determined not only our welfare, but our existence. Together with the placenta it is the only organ that dies when life begins. Although the umbilical cord is one of the most intriguing of the human organs, it is one of the least investigated.

However, after the turn of twentieth century the interest in the cord declined. This is mainly because most of the perinatal complications involving the umbilical cord were detected only after birth, since the cord was inaccessible during antenatal period. With modern possible ultrasound techniques it has now become possible to search for abnormalities of the cord before birth.

Embryology of the umbilical cord

Blastocyst develops into embryo. At a very early stage in development, embryo proper acquires the form, a 3 layered disc called embryonic disc.

The three layers that constitute the embryonic disc are:

1. Endoderm
2. Ectoderm
3. Mesoderm

A space appears between the ectoderm (below) and the trophoblast (above) called as amniotic cavity (Fig 1A), filled by amniotic fluid or liquor amni.

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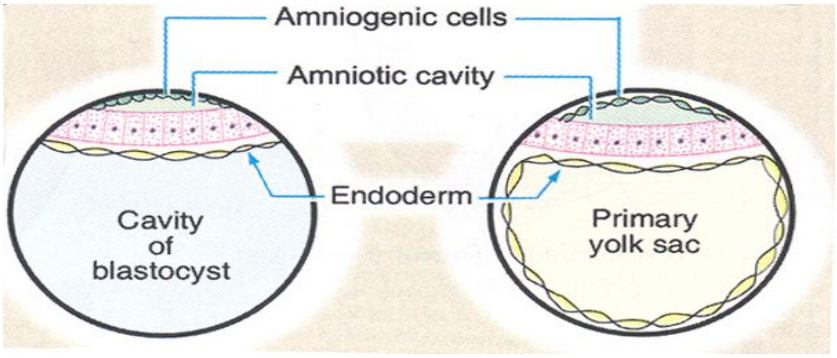

A **B**

Fig. 1. A, B

Flattened cells arising from the endoderm spread and line the inside of the blastocyst cavity. This cavity which is lined on all sides by cells of endodermal origin is called the primary yolk sac (Fig. 1B).

The cells of the trophoblast give origin to a mass of cells called extra-embryonic mesoderm (Fig. 2)

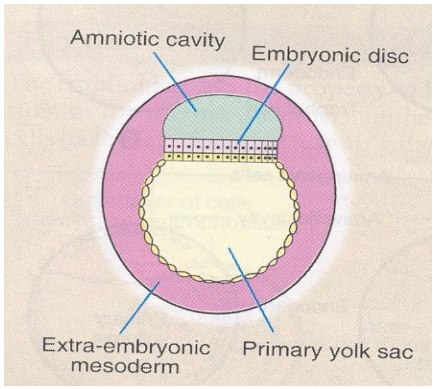

Fig. 2

Small cavities appear in the extra-embryonic mesoderm. Gradually, they join together to form larger spaces and, ultimately one large cavity is formed. This cavity is called the extra embryonic coelome, which is split into two layers called the somatopleuric (parietal) extra-embryonic mesoderm and splanchnopleuric (visceral) extra-embryonic mesoderm.

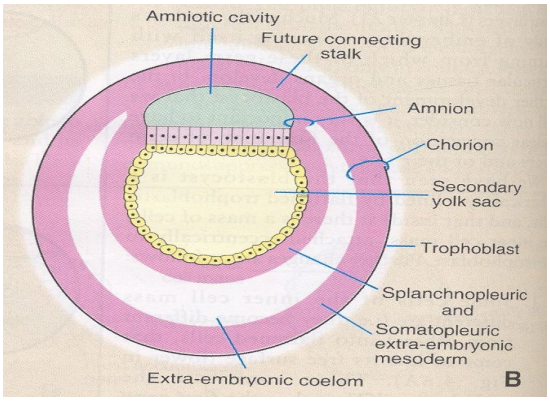

Fig. 3

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From fig. 3, it is clearly seen that the extra-embryonic coelom does not extend into that part of extra-embryonic mesoderm which attaches the wall of the amniotic cavity to the trophoblast.

Connecting stalk (future umbilical cord)

The developing embryo, along with the amniotic cavity and yolk sac, is now suspended in the extra embryonic coelom, and is attached to the wall of the blastocyst only by this unsplit part of extra embryonic mesoderm. This mesoderm form a structure called the connecting stalk.

At 18 days post-conception the connecting stalk develops, which connects the early embryo to the trophoblast. The importance of the connecting stalk is obvious when we see that this is the only connecting link between the embryo and the placenta i.e., the future umbilical cord.[1]

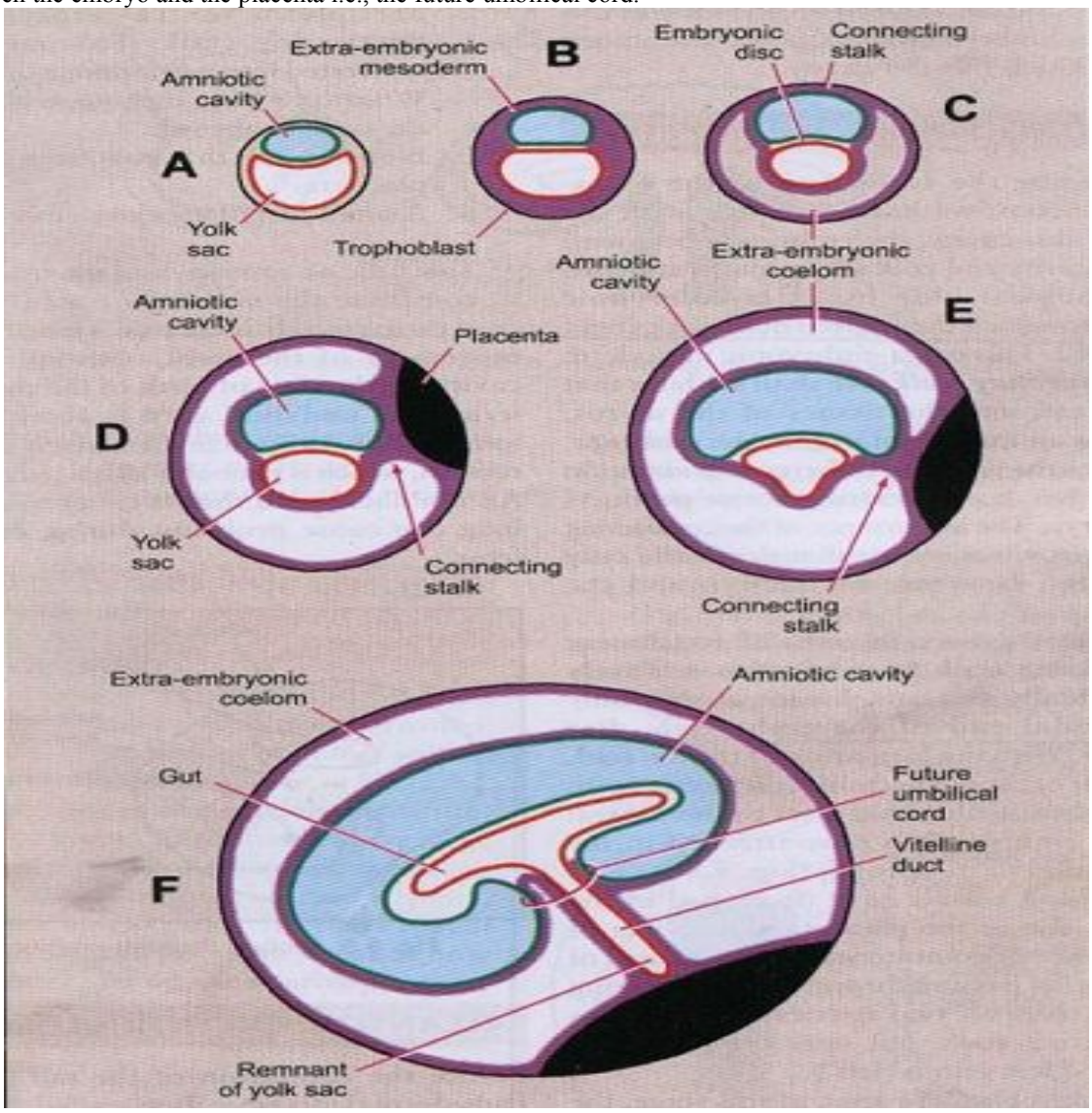

Fig 4 Stages in establishment of umbilical cord

As the embryo grows, the area of attachment of the connecting stalk to it become relatively smaller. Gradually this attachment is seen only near caudal end of embryonic disc (Fig. 4 D, E). With the formation of the tail fold, the attachment of the connecting stalk moves (with the tail end of embryonic disc) to the ventral aspect of embryo. It is now attached in the region of umbilical opening Fig. 4E.

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By now, blood vessels have developed in the embryo, and also in the placenta. These sets of blood vessels are in communication by means of arteries and veins passing through the connecting stalk. In this connecting stalk lies the transitory allantois, the primitive extraembryonic urinary bladder.[2] The primary yolk sac is lined with endoderm and forms the central portion of the embryonic gut.[3] After contributing to the embryonic gut, the remains of the primary yolk sac elongate ventrally, thereby narrowing the connection to the midgut. The connection forms the ductus vitellinus (Fig. 4 F, 5).

In humans, the secondary yolk sac is small and rudimentary. At 4 weeks post-conception the connecting stalk and the yolk sac duct merge, forming the umbilical cord. In humans the yolk sac is a rudimentary organ, that probably has a nutritive function only very early in pregnancy.[1]

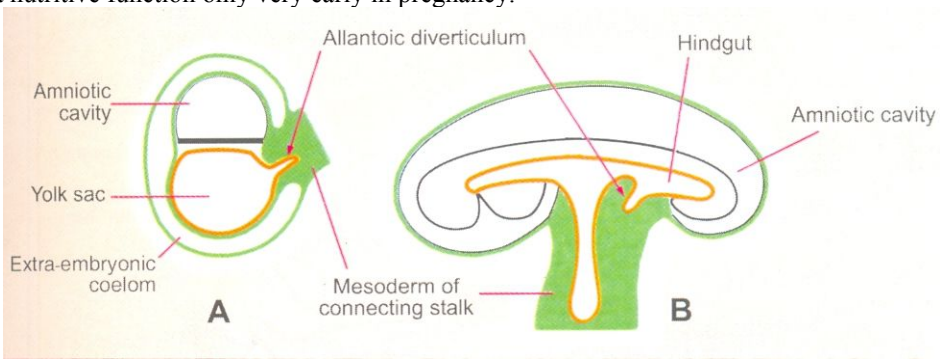

Fig. 5

The oval line of reflection between the amnion and embryonic ectoderm is the primitive umbilical ring. At the fifth week of development, the following structures pass through the ring.

a) The connecting stalk
b) The yolk sac
c) The canal connecting the intra embryonic and extra-embryonic cavities

During further development, the amniotic cavity enlarges rapidly at the expense of the chorionic cavity, and the amnion begins to envelop the connecting and yolk sac stalks, crowding them together and giving rise to the primitive umbilical cord.[2]

The umbilical cord consists of an outer layer of epithelium from the amnion, with an internal mesodermal mass, the Wharton's jelly. In this jelly there are two endodermal ducts: the allantois and the vitelline duct, and the umbilical vessels (Fig 6). The umbilical cord is formed at 4 to 6 weeks post conception.

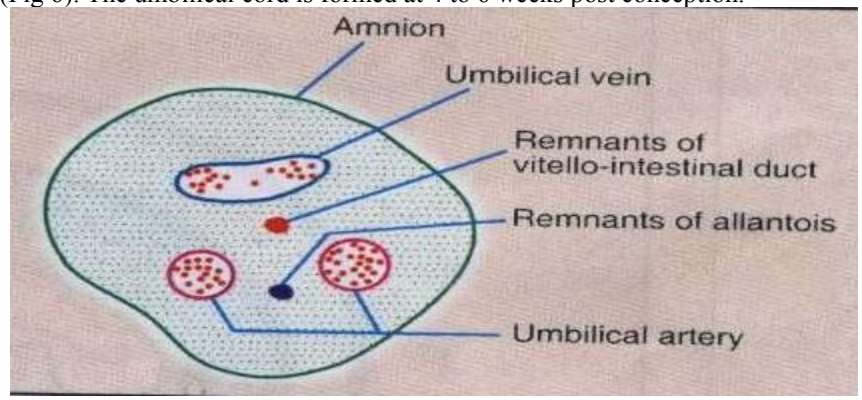

Fig. 6: Section through umbilical cord

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Vascular system in umbilical cord

The development of vascular system starts with the formation of blood islands in the mesoderm of the yolk sac, connecting stalk and chorion at the beginning of 3 weeks post-conception. Two days later, angiogenesis begins in the intra embryonic mesoderm.

The allantois arteries appear 3 weeks post-conception as ventral branches of the paired dorsal aortas. Portion of the allantois will give rise to the urinary bladder, from which the urachus extends as a tiny duct, accompanied by the allantois arteria. They course to umbilical ring and into the umbilical cord.

Umbilical Arteries

Before the fusion of the two dorsal aortae, the umbilical arteries appear as continuations of their distal ends (Fig. 7A). After fusion of the dorsal aortae, they appear as lateral branches of the single dorsal aortae (Fig. 7B). Subsequently, each umbilical artery gets linked up with that part of the fifth lumbar intersegmental artery which forms the internal iliac artery (Fig. 7C).

The part of the umbilical artery, which lies between the aorta and the anastomosis with the internal iliac, disappears so that the umbilical artery is now seen as a branch of the internal iliac (Fig.7D, E).

In postnatal life, the proximal part of the umbilical artery becomes the superior vesical artery, while its distal part is obliterated to form the medial umbilical ligament.

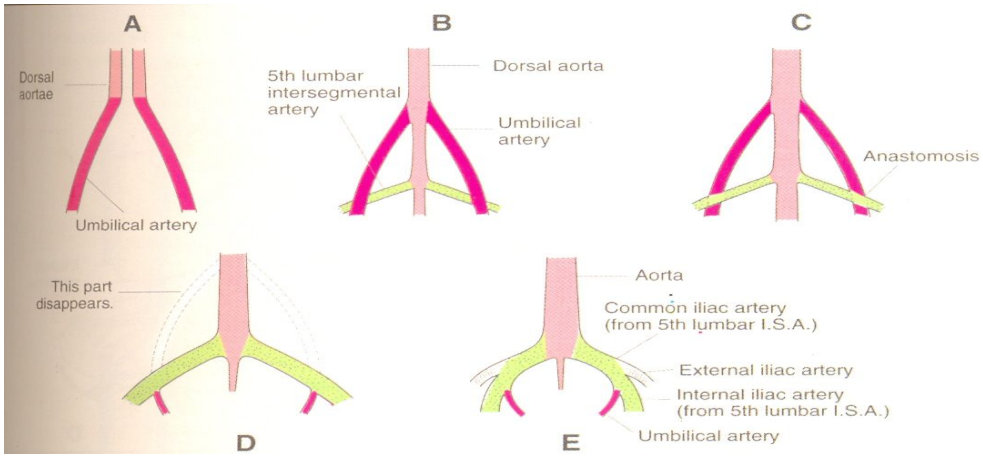

Fig. 7

Umbilical veins

The visceral veins of the embryo are:

- Right and left vitelline veins from the yolk sac.
- Right and left umbilical veins from the placenta.

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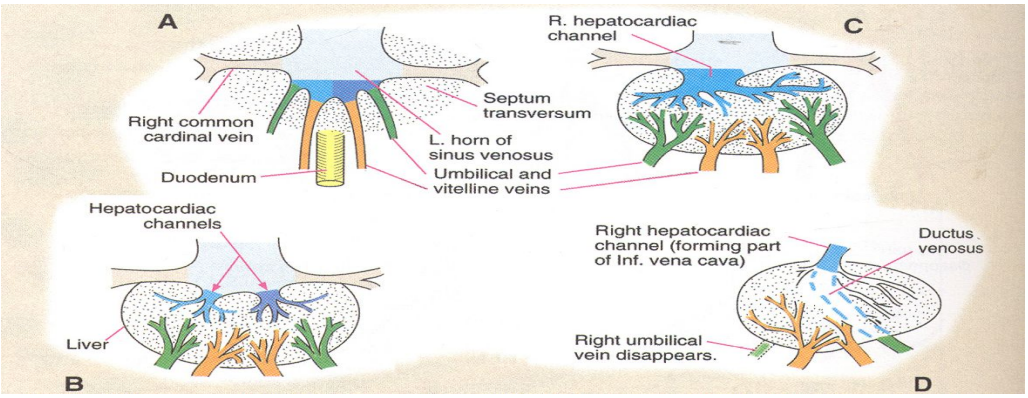

Fig. 8

The umbilical and vitelline veins open into the corresponding horn of the sinus venosus (Fig. 8A). The parts of these veins that are nearest to the heart are embedded in the septum transversum.

These veins undergo considerable changes as follows:

a) With the development of the liver, in the septum transversum, the proximal parts of the vitelline and umbilical veins become broken up into numerous small channels that contribute to sinusoids of liver. These sinusoids drain into the sinus venosus, through the persisting terminal parts of the vitelline veins that are now called the right and left hepatocardiac channels (Fig.8 B). The proximal parts of the umbilical veins lose their communications with the sinus venosus.

b) Mean while, the left horn of the sinus venosus undergoes retrogression and as a result the left hepatocardiac channel disappears. All blood from the umbilical and vitelline veins now enter the sinus venosus through the right hepatocardiac channel (also called common hepatic vein). This vessel later forms cranial most part of the inferior venacava (Fig.8C).

c) The right umbilical vein disappears and all blood from the placenta now reaches the developing liver through the left vein (Fig.8 D).In order to facilitate the passage of this blood through the liver, some of the sinusoids enlarge to create a direct passage connecting the left umbilical vein to the right hepatocardiac channel. This passage is called the ductus venosus.

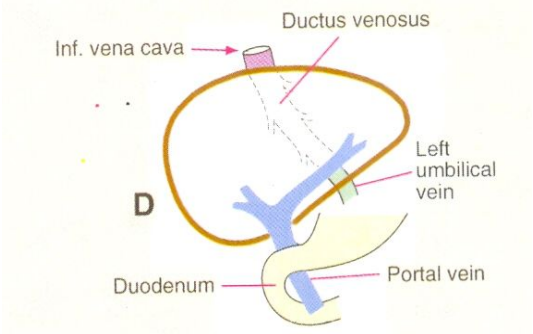

Fig. 9

d) While these changes are occurring within the liver, the parts of the right and left vitelline veins that lie outside the substance of the liver undergo alterations leading to the formation of the portal vein.

The left umbilical vein now ends in the left branch of the portal vein (Fig. 9), while the ductus venosus connects the left branch of the portal vein to the inferior venacava (right hepatocardiac channel). The embryonic circulation is effective at 22-23 days post conception, when the umbilical arteries have fused with the internal iliac arteries, and the umbilical vein with the ductus venosus, which enters the hepatic vein.

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Until 11 weeks post-conception there are intestines in the umbilical cord, giving it a swollen appearance. There after the intestines have retracted into the abdominal cavity.[2] The allantois, ductus vitellinus and vessels of the yolk sac obliterate, and all that remains in the umbilical cord are the umbilical vessels, surrounded by whartons's jelly. In the normal umbilical cord there are two umbilical arteries, and one vein (the right vena umbilical is usually obliterates).[2] The two arteries are smaller in diameter than the vein. In 96% of all umbilical cords there is an anastomosis or, in 3% even fusion of the two umbilical arteries within 1.5 cm of the placental insertion site. This warrants an equalization of flow and pressures between two arteries and a uniform distribution of blood to the different lobes of the placenta.[3]

One of the most common vascular anomalies in humans is the absence of one umbilical artery, occurring in about 1 in 200 newborns.[3] About ¼th of all infants with only one umbilical artery have associated congenital anomalies.[4]

Wharton's jelly

Wharton's jelly, derived from mesenchyme, and formed by fibroblasts, consists of collagen and hyaluronic acid, some muscular fibres and water. This material seems to be responsible for the strength of the umbilical cord. It provides mechanical supports and structural protection for umbilical vessels, and has angiogenic and metabolic role for the umbilical circulation.[4]

The osmotic environment is of utmost importance to the Wharton's jelly. Changes in osmolarity of 5 to 10 milliosmol cause evident swelling or shrinking of the cord.

Wharton's jelly has thyrotrophic properties, i.e., this semisolid gelatinous substance liquefies due to pressure.[5]

The amount of Wharton's jelly is a good predictor of perinatal complications, evidence cumulates that, an umbilical cord with a diameter < 10^{th} centile is an early marker for the delivery of a small for gestational age infant and the occurrence of intrapartum complication.

Coiling of umbilical cord

The helical course of the umbilical vessels can be observed as early as 28 days post-conception, and is clearly visible from 7 weeks post-conception in 95% of all foetuses.[6,7] The origin of the coiling is unknown. The hypothesis includes:

- foetal movements
- active or passive torsion of the embryo
- differential umbilical vascular growth rates
- foetal hemodynamic forces
- the muscular fibers in the arterial wall
- genetic factor

Possibly there is a genetic factor, although in a small series of monozygotic twins no uniform concordance in the umbilical coiling index was found.[7]

According to Roach, the coiling is caused by muscular fibers in the arterial walls.[8]
There are four different muscles in the arterial wall:

- inner circular layer, regulating flow.
- inner longitudinal layer, which closes the artery postpartum.
- large coiling muscle, which has an intrinsic twist that makes the cord coil small coiling muscle, which makes the arteries coil.

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The large helical muscle has a long pitch which is comparable to the pitch of the coils of the cord itself. Attachments of the coiling muscle of the artery to the cord substance are responsible for coiling of the cord itself. When there is enough hydrostatic pressure, the cord coils in the direction opposite to the direction of the fibers in the helical muscle.

The cord is gradually covered by the amniotic membrane from 4 weeks post-conception onwards. The amniotic cavity continues to enlarge and the amnion sheathes the umbilical cord in the direction of the placenta.[8]

Umbilical Cord Complications

Umbilical cord abnormalities are numerous, ranging from false knots, which have no clinical significance, to vasa previa, which often leads to foetal death. As prenatal ultrasound becomes increasingly sophisticated, many of these conditions are being diagnosed in utero. However, many are not apparent before delivery, and the only forewarning is related to their association with certain conditions such as monochorionic twins and placental abruption.

List of complication

- Cord Length related
- Single Umbilical Artery
- Velamentous insertion of umbilical cord
- Vasa previa
- Cord knots
- Nuchal cord
- Cord stricture
- Cord hematoma
- Cord ulceration
- Cord cyst
- Cord varix

Cord Length

The length of the umbilical cord varies from no cord (achordia) to 300 cm, with diameters up to 3 cm. Umbilical cords are helical in nature, with as many as 380 helices. At term the umbilical cord has an average length of 55cm (usual range 30-100 cm).[9] Leonardo da Vinci postulated the rule of thumb that the umbilical cord at any gestational age is on average as long as the foetus itself, with a diameter of 1-2 cm and 11 helices. For unknown reasons, most cords coil to the left. About 5% of cords are shorter than 35 cm, and another 5% are longer than 80 cm.-

Causes of differences in cord length are unknown, however the length of the cord is thought to reflect movement of the foetus in utero. Short cords are associated with foetal movement disorders and intrauterine constraint, as well as placental abruption.

Although short cords have been blamed for the inability of some foetuses to deliver vaginally, available data suggest that vaginal delivery can take place with cords as short as 13 cm, which is much shorter than the normal range. Excessively long cords are associated with cord around the neck, foetal entanglement, true knots, and thrombi. Assessing cord length prenatally is not possible.

Straight cords with few or absent helices have been associated with adverse foetal outcomes. In cases of placental abruption, oligohydramnios, or breech presentation, consideration may be given to measurement and documentation of cord length after birth, because an abnormal cord length argues for a long-term foetal condition.

Single Umbilical Artery

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The umbilical cord normally contains 2 arteries and a single vein. Occasionally, one umbilical artery is absent, with the left artery absent more commonly than the right. Single umbilical arteries are associated more commonly with foetal anomalies than normal cords.

Single umbilical artery occurs in fewer than 1% of cords in singletons and 5% of cords in at least one twin. Single umbilical artery also occurs more often in foetal demise than in live births. The incidence can be overestimated with gross examination of the cord, especially if the portion close to the placenta is examined, because the arteries may fuse close to the placenta. Single umbilical arteries are found twice as often in white women than in African American and Japanese women. Diabetes increases the risk significantly. The male-to-female ratio is 0.85:1.

Single umbilical artery is believed to be caused by atrophy of a previously normal artery, presence of the original artery of the body stalk, or agenesis of one of the umbilical arteries.

The vessels in the cord are clearly identifiable with ultrasonography. The vein is usually larger than the arteries. Single umbilical artery may be diagnosed prenatally with the finding of only 2 vessels on a cross section of the cord, or a vessel seen on only 1 side of the foetal bladder.

Of infants with a single umbilical artery, 20% or more are reported to have associated foetal anomalies, including cardiovascular abnormalities, GI defects, oesophageal atresia, a variety of renal defects, and multiple anomaly syndromes. The association with foetal defects is more striking in series reported from prenatal diagnosis than in newborn studies; this difference may be due to prenatal diagnosis occurring in a selected, high-risk population. In addition, almost 20% of cases of single umbilical artery diagnosed prenatally in a high-risk population were associated with chromosomal anomalies. Trisomy 18 is the chromosomal anomaly most highly associated with single umbilical artery.

Velamentous insertion of umbilical cord

With velamentous insertion, the umbilical cord inserts into the chorion leave at a point away from the placental edge, and the vessels pass to the placenta across the surface of the membranes between the amnion and the chorion.

One percent of singletons have velamentous insertion; however, this condition occurs in almost 15% of monochorionic twins and is common in triplets.

Velamentous insertion occurs when

1. Placental tissue grows laterally, leaving the centrally located umbilical cord in an area that becomes atrophic, or
2. The cord implants in the trophoblast anterior to the decidua capsularis rather than the trophoblast tissue that is destined to become the placental mass.

Velamentous insertion has been diagnosed by ultrasonography with a sensitivity of 67% and specificity of 100% in the second trimester; first trimester diagnosis is also possible. The condition is associated with a lower maternal serum alpha-fetoprotein (AFP) and higher maternal serum human chorionic gonadotropin (hCG).

Velamentous insertion can cause haemorrhage if the vessels are torn when the membranes are ruptured, most often with a vasa previa. Velamentous insertion of the cord is associated with low birth weight, prematurity, and abnormal foetal heart patterns in labor. If detected, foetal growth may be monitored with ultrasonography in the third trimester.

Consider an elective caesarean delivery to avoid a vasa previa rupture or foetal distress if the velamentous insertion is in the lower segment.-

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Vasa previa

Vasa previa occurs when the foetal vessels in the membrane are situated in front of the presenting part of the foetus. This may occur because of a velamentous insertion of the cord or with vessels running between the placenta and a succenturiate lobe. Vasa previa may also exist over the dividing membrane when a second twin has a velamentous insertion of the umbilical cord.

This condition occurs in 1 per 2000-3000 deliveries. The cause of vasa previa is unknown. Vasa previa may be associated with low-lying placenta, placenta with accessory lobes, and with multiple pregnancies.

Vasa previa occasionally may be felt on palpation and ultrasonographic detection has been reported. Color Doppler ultrasonography can be used to visualize the course of the vessels, and pulse Doppler ultrasonography can be used to confirm the foetal origin. A series of gray lines in the vicinity of the internal os may be diagnostic of vasa previa. A sinusoidal foetal heart pattern, foetal bradycardia, or foetal heart rate decelerations during labor may all indicate a ruptured vasa previa. A Kleihauer-Betke or Apt test may detect the presence of foetal cells in the vaginal discharge; however, in the face of a ruptured vasa previa, foetal distress is usually apparent before the test results become available.

The risk of foetal exsanguination is significant if the vessels are torn when the membranes rupture, with an associated 50-75% foetal mortality rate. If compressed during labour, the vessels can cause foetal heart decelerations. Compression of the vessels during labour can also cause the vessels to thrombosed.

Caesarean delivery is the preferred mode of delivery for known vasa previa after confirming foetal lung maturity and is mandatory if significant vaginal bleeding occurs. Prenatal diagnosis of vasa previa can markedly improve outcome. In one report, pregnancies diagnosed prenatally had a 97% foetal survival as compared with 48% in those not diagnosed prenatally. Consider endovaginal colour flow Doppler ultrasonography to rule out vasa previa for patients with a known succenturiate lobe or velamentous insertion of the cord.

Cord knots

True knots and false knots can form in the umbilical cord. True knots occur in approximately 1% of pregnancies, with the highest rate occurring in monoamnionic twins. False knots (kinks in the umbilical cord vessels) are more common.

True knots arise from foetal movements and are more likely to develop during early pregnancy, when relatively more amniotic fluid is present and greater foetal movement occurs.

True knots are also associated with advanced maternal age, multiparity, and long umbilical cords. True knots have been reported to lead to a 4-fold increase in foetal loss, presumably because of compression of the cord vessels when the knot tightens.

False knots have no known clinical significance.

Detection of umbilical knots has been reported with ultrasonographic imaging. Prenatal diagnosis has occurred largely in monoamniotic twins, when the condition was specifically sought.

A caesarean delivery may be considered if a diagnosis of a true cord knot is made. The usefulness of antenatal testing in the follow-up of pregnancies with this condition is uncertain.

Nuchal cord

The cord may become coiled around various parts of the body of the foetus, usually around the neck. Nuchal cord is caused by movement of the foetus through a loop of cord.

One loop around the neck occurs in approximately 20% of cases, and multiple loops occur in up to 5% of pregnancies.

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Nuchal cord has been associated with labour induction and augmentation, prolonged second stage of labour, and foetal heart rate abnormalities. One report has described a decrease in umbilical cord pH at delivery with nuchal cord, but the difference found (7.32 vs. 7.30) does not appear to be clinically significant. Nuchal cord can be detected using colour Doppler ultrasound, with a sensitivity of over 90%.

Nuchal cords rarely cause foetal demise and are not intrinsic reasons for intervention. Given the minor decrease in pH, foetal monitoring in labour would appear to be prudent, but no data are available to address this issue.

Cord stricture

Cord stricture is constriction or occlusion of the cord.

This condition is found in 19% of foetal demises. Familial recurrence of umbilical cord strictures has been described.

The etiology of umbilical cord stricture is unknown. There is a deficiency in Wharton jelly in the umbilical cord in the area of stricture, however this could be a post-morbid change.

This condition cannot be diagnosed prenatally.

Most infants with cord stricture are stillborn.

Cord hematoma

A cord hematoma is extravasation of blood into the Wharton jelly surrounding the umbilical cord vessels.

This condition is rare in live-born infants. Cord hematoma can occur after the rupture of a varix of the umbilical vein, with subsequent effusion of blood into the cord. Invasive prenatal procedures can also cause hematomas.

Finally, cord hematoma can occur spontaneously and in association with cord cysts. The vein-to-artery ratio is 1:9.

Cord hematoma has been described as a cause of acute foetal distress. A more chronic presentation of a cord hematoma may appear as a mass in the umbilical cord. Doppler studies can evaluate a suspected hematoma, which increases vascular resistance.

If the diagnosis of cord hematoma is confirmed with a stable foetus, an amniocentesis may be performed, and delivery can be undertaken when the foetus is documented to be mature.

Cord ulceration

Ulceration of the umbilical cord has been described with perforation of the vessels and intrauterine hemorrhage.

This complication is rare. The cause of cord ulceration is unknown, although it has been described most often in association with foetal upper intestinal atresias.

Umbilical cord ulceration has not been diagnosed prenatally. No evidence suggests appropriate prenatal management.

Cord cysts

Cord cysts can be defined as true or false cysts, and they can occur at any location along the cord. They are irregular in shape and are located between the vessels.

Cysts are found in 0.4% of pregnancies. True cysts are small remnants of the allantois (i.e., allantoid cysts) or the umbilical vesicle.

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Cysts have an epithelial lining, occur at the foetal end of the cord, and usually resolve during the first trimester. True cysts can be associated with hydronephrosis, patent urachus, omphalocele, and Meckel's diverticulum.

False cysts can be as large as 6 cm and represent liquefaction of Wharton jelly. They do not have an epithelial lining and are most commonly found at the foetal end of the cord. Pseudo cysts are associated with chromosomal anomalies, omphalocele, and patent urachus. Of cord cysts of any type, 20% are associated with structural or chromosomal anomalies.

During foetal anatomy scans, the abdominal wall near the cord insertion is the most likely location to detect a cyst. Cysts can be visualized most easily with colour Doppler studies during the first trimester, when the umbilical vessels are small.

Persistent cysts may be observed with foetal karyotyping and level 2 second trimester ultrasonography. In patients with large cysts, caesarean delivery undertaken as soon as foetal lung maturity is achieved may help to avoid foetal damage from cyst rupture during labour.

Cord varix

Cord varix is a cystic dilatation that can occur in any portion of the umbilical vein. Cord varix rarely occurs, and its cause is unknown. Colour Doppler flow studies show very turbulent flow through the cyst, which is continuous with the umbilical vein.

Reports have documented poor foetal outcomes in the presence of varices and an association with foetal anomalies and with emergent caesarean delivery.

Once detected, regular foetal testing, third trimester interval growth studies, and karyotyping may be considered. Some authors have recommended elective delivery when the foetus is mature because of the high risk of foetal distress.

Umbilical cord

The umbilical cord contains two arteries, a vein and Wharton's jelly enveloped in amniotic epithelium or at the foetal end, a Malpighian keratinized epithelium[10]. Wharton's jelly contains myofibroblasts immersed in an extracellular matrix. It consists of a spongy network of interlacing collagen fibres and small woven fiber bundles that encases the umbilical vessels, protecting them from twisting and compression during pregnancy and delivery. The ground substance is composed mainly of hyaluronic acid (70%) and some sulphated glycosaminoglycans and proteoglycans (30%) in an aqueous solution of salts, metabolites and plasma proteins.[11] Hyaluronic acid can bind a substantial amount of water and plays a major role in the interstitial transport dynamics and osmotic pressure. However, the composition of Wharton's jelly may vary.

The umbilical cord is tasked with providing unimpeded blood flow to the developing foetus. The tissues of the umbilical cord must work to maintain blood flow during foetal grasping, normal movements, and forces of labor, and in the presence of cord abnormalities such as knots or loops.

The umbilical vessels differ in structure and function as compared to the major vessels in the body. The two umbilical arteries coil around the vein in a helical fashion. Blood flows in a pulsatile manner from the foetus to the placenta through the arteries. A small pulse remains in the more passive transfer of blood back to the foetus through the umbilical vein.

The umbilical arteries do not possess an internal elastic membrane and contain little elastin, in general, while the vein contains an elastic subintimal layer. Collagen, a mechanically stiff protein, typically serves to limit radial vessel distension at high loads. Elastin, by contrast, is highly extensible at low loads. Within most arterial tissues, the elastic fibers are bound together into fenestrated sheets that exhibit a near perfectly elastic mechanical response.

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Elastin therefore functions to provide the recoverable, elastic extensibility and subsequent contraction in arteries during pulsatile blood flow. Smooth muscle cells lie throughout the arterial media and participate in regulating muscular tone and eliminate the need for substantial elastin content.

The arteries lack an adventitia of the form that is observed in cardiovascular vessels. Instead, the rigid Wharton's jelly performs the function of the adventitia. The Wharton's jelly consists of a porous, extracellular matrix (ECM) based backbone and ground substance. This fibrous, porous scaffold is made of collagen, elastin fibers and likely contributes to the firmness of the intact cord. The pores within the Wharton's jelly form canalicular structures that house the proteoglycans, hyalauronic acid, and other molecules that interact with water to form a highly viscous, mucoid fluid. The thickness and turgidity of the Wharton's jelly varies with the expansion and contraction of the vessels and may structurally support and prevent over distension of the vessels. Myofibroblasts, cells possessing ultra structural characteristics of both fibroblasts and smooth muscle cells, within the Wharton's jelly form collagen and other proteins and may actively contract to assist in regulating umbilical blood flow. The endothelial cells that reside within the arteries and vein are unusually rich in organelles that may play a role in amniotic fluid formation. The amnion is structurally comparable to that found in the foetal membranes and may actively maintain fluid pressure in the Wharton's jelly.

The smaller amount of Wharton's jelly may be the consequence of either an extracellular dehydration or a reduction in extracellular matrix component. It has been proposed that Wharton's jelly cushions umbilical blood vessels, preventing disruption of flow due to compression or bending caused by foetal movements and uterine contraction at delivery. Wharton's jelly appears to serve the function of adventitia, which the umbilical cord lacks, binding and encasing the umbilical vessels. It has been speculated that the cells of Wharton's jelly may participate in the regulation of umbilical blood flow and that, at least in some cases; the reduction in foetal growth could be the consequence of diminution of Wharton's jelly leading to vascular hypoplasia of the umbilical vessels. In fact, a reduction in wall thickness of umbilical cord arteries and vein has been found in IUGR infants with abnormal umbilical artery flow when compared to IUGR infants without increased umbilical artery resistance. Therefore, it can be hypothesized that the greater the reduction in the amount of Wharton's jelly, the greater the damage to the umbilical cord vessels and the greater the compromise to the growth of the foetus.

Macrosomia is defined as birth weight >4000g or birth weight >90 th percentile for a given gestation.

Risk factors for macrosomia are:

- Maternal diabetes
- Parents size especially maternal
- Multiparity
- Prolongrd gestation
- Increased maternal age
- Male fetus
- Race and ethnicity

The delivery of a macrosomic infant has potentially severe consequences for both the newborn and the mother. Increased birth weight heightens the risk in the foetus of shoulder dystocia and permanent brachial plexus injury, and those infants weighing ≥4500 g are at increased risk for neonatal morbidity, including the need for assisted ventilation and meconium aspiration.[12]

Maternal complications result from operative delivery and include postpartum haemorrhage, third or fourth-degree lacerations and postpartum infection. Despite the fact that current evidence does not support intervention for suspected macrosomia, maternity care professionals continue to search for accurate methods of predicting foetal weight in an effort to ameliorate the adverse outcomes that are associated with traumatic delivery.

The clinician's intention in anticipating macrosomia is not only to employ cut-offs of estimated foetal weight (EFW) for selecting cases in which the risk of birth trauma is high enough to warrant delivery by caesarean section, but also to adapt the usual intrapartum management (i.e. cautious approach to operative vaginal delivery, low threshold for emergency caesarean section when second stage descent is slow, on-site presence of experienced staff) and to plan for induction of labour, at least in diabetic pregnancies.

Unfortunately, despite advances in ultrasound technology, our longstanding experience in obtaining foetal biometric measurements and research efforts to date, the diagnosis of macrosomia still remains problematic.

Investigators have attempted to improve ultrasound based prediction of foetal macrosomia by various methods, including assessment of fat deposition at a variety of locations i.e. shoulder soft tissue thickness, humeral soft tissue thickness, foetal fat layer, intraventricular septum and abdominal circumference[13,14,15], three-dimensional ultrasound technology[16] and more sophisticated bioinformatics processing systems that incorporate parental and pregnancy-specific information.[17] None of these has gained wide popularity and ultrasound methods that account for subcutaneous fat thickness have not been shown consistently to improve our ability to estimate foetal weight accurately using formulae derived from conventional biometric parameters.

Raio et al[18] found an association between the presence of a "lean" umbilical cord (cross-sectional area<10thcentile) and the delivery of a small for gestational age infant (SGA). During the study period, 860 patients met the inclusion criteria, 3.6% delivered a SGA infant. The proportion of SGA infants was higher among foetuses that had a lean umbilical cord on ultrasound examination than among those with a normal umbilical cord (11.5% vs. 2.6%, $p < 0.05$). Patients with a "lean" umbilical cord had a 4.4-fold higher risk (95% confidence interval, 2.16–8.85) of having an SGA infant than those with a normal umbilical cord.

Patel D et al[19] heavier neonates have also been reported to have a larger umbilical cord circumference at birth.

Weissman and Jakobi[20] found a significant correlation between Wharton's jelly area and umbilical cord diameter, as assessed by ultrasound and EFW in a cohort of 100 foetuses of mothers with gestational diabetes mellitus.

Cromi A et al[21] in their study group of 1026 patients found that Fifty-three (5.2%) newborns had a birth weight > 4000 g, and 22 (2.1%) weighed > 4500 g. The proportion of cases with a large umbilical cord was significantly higher in the group of macrosomic compared with non-macrosomic infants (54.7% vs. 8.7%). The combination of abdominal circumference > 95(th) centile and large cord predicted 100% of macrosomic infants. The proportion of umbilical cords with a Wharton's jelly area > 95(th) centile for gestation was significantly higher in macrosomic fetuses of diabetic compared with non-diabetic mothers. So they concluded that, a large umbilical cord area assessed by ultrasound is a simple and reliable marker of foetal macrosomia.

Sobolewski K et al showed that pre-eclampsia is associated with reduced gelatinase activity, expression of insulin-like growth factor-I binding protein, and cathepsin D activity[22]. In such pregnancies, the Wharton's jelly and umbilical vein areas are reduced compared with those in normal pregnancies.

Ghezzi F et al[23] in a study of 160 foetuses after the 20 weeks of gestation showed that lean umbilical cords are associated with low birth weight and unfavourable neonatal outcome. The reduced amount of Wharton's jelly in such pregnancies probably reflects a general shift in metabolic and endocrine activity, but also correspondingly altered hemodynamics. Small umbilical vein diameters and low blood velocity and flow in such foetuses further suggest hemodynamics as another determinant for Wharton's jelly development.

Vasques et al[24] in their prospective cross sectional study showed a strong correlation between the umbilical cord cross sectional area and the estimated foetal weight by ultrasound examination and also with the foetal anthropometric parameters (results UCCSA × BPD = 0.622; UCCSA × HC = 0.617; UCCSA × AC = 0.625;

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UCCSA × FL = 0.604, all of them significant at the level of $P < 0.01$) and they concluded that the Umbilical cord cross sectional area is a parameter that can be included as the routine of obstetrical ultrasound examinations.

Hall[25] stated that "the thin cord is a dangerous cord and a fat cord is a safe cord, all other factors being equal." The major limitation of these observations is that the umbilical cord was assessed after delivery.

Raio L et al[18] shown that foetuses with a thin umbilical cord on sonography during the second and third trimester of gestation are at increased risk of adverse perinatal outcome.
It has been demonstrated that a lean umbilical cord is associated with growth developmental disorders, preeclampsia, oligohydramnios, and foetal distress during labour. **Raio et al**[26] in his study of 25 pre eclamptic women admitted after 20 weeks gestation has found that the proportion of patients with a lean umbilical cord was higher among those with early-onset preeclampsia than in those with late-onset preeclampsia (12 of 19 versus 0 of 6, P =.014) and has concluded that early onset preeclampsia is frequently associated with reduced Wharton's jelly area and umbilical vein area compared with normal pregnancy. Sonographic umbilical cord morphometry might have clinical value for prompt identification of women at risk for preeclampsia.

Prabhacharan G, Jarjoura D[27] observed a correlation between measurements of Wharton's jelly area on frozen sections of umbilical cords and birth weight. Little is known about the function, formation and deposition of Wharton's jelly. Pathological studies and case reports have demonstrated that a thin umbilical cord is associated with adverse pregnancy outcome.

Labarrere and colleagues[28] have described an association between a reduced amount of Wharton's jelly and foetal or neonatal death when the length and insertion of the umbilical cord at the placental site were normal in the absence of known risk factors for foetal or neonatal death. The reduced amount of Wharton's jelly may be the result of an inherited disorder in the deposition of Wharton's jelly, making the umbilical circulation vulnerable to insults rather than the consequence of foetal disease per se. Indeed, successive foetal deaths in the same family due to torsion of the umbilical cord as the consequence of primary absence of Wharton's jelly have been described.

A lean umbilical cord at birth has also been associated with oligohydramnios and foetal distress. **Silver and colleagues**[29] have reported that, in post-term pregnancies, the diameter of the umbilical cord is smaller in patients with oligohydramnios than in those with normal amniotic fluid. In addition, these authors found a higher incidence of variable decelerations antepartum in patients with a small umbilical cord diameter than in those with a normal umbilical cord. Moreover, isolated cases of thin umbilical cord associated with small –for gestational age (SGA) infants have been described by several authors.
Bruch et al[30] reported that growth-retarded foetuses with or without Doppler abnormalities of umbilical arteries have a smaller umbilical cord cross-sectional area at delivery than normal healthy foetuses. These authors found that growth-retarded foetuses with normal Doppler waveforms of umbilical arteries have a reduction in the total umbilical cord area when compared to that of healthy infants. However, no modifications were observed in the total lumen area of both arteries, suggesting that the difference in the cross-sectional area of the umbilical cord between IUGR and normal foetuses is mainly due to Wharton's jelly diminution and umbilical vein reduction.

Estimated foetal weight
Recent epidemiological and experimental studies show that abnormal foetal growth can lead to serious complications. These include intrauterine deaths, stillbirth, perinatal morbidity and disorders extending well beyond the neonatal period. Much of the evidence to support Barker Hypothesis[31] of foetal origin of adult disease is based on birth weight, placental weight and their ratio. It is clear that the intrauterine milieu is as important as genetic endowment in shaping the future health of the conceptions.

A microscopic monocellular zygote that is formed by fertilization ovum by a sperm is endowed with tremendous growth potential. From a weight of 0.005 mg at conception, it grows rapidly to achieve an average weight at

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delivery. Initially, growth is due to organogenesis as in first trimester and later, their differentiation and increasing functional maturity.

Estimation of foetal weight in utero is difficult as the foetus cannot be weighed as a separate entity. Total maternal weight gain during pregnancy will include factors such the enormous growth of uterus, the development of placenta, increase in quantity of liquor and physiological changes in physique of the mother.

Foetal weight estimation in utero has become increasingly important in modern obstetrics in for:

- Prevention of prematurity.
- Diagnosis of intrauterine growth restriction.
- Induction of labour before term in case of complicated pregnancies.
- Evaluation of fetopelvic disproportions.
- As a decderatum in the mode of delivery.

Estimating foetal weight in utero is of paramount importance in obstetric decisions. However, an ultimate consensus on its methodology has been lacking. Though the years, various methods for estimating foetal weight have been proposed, tested and also discarded with regular frequency. The more common methods that have persisted include clinical estimation by examination of the patient, estimation by simple maternal abdominal measurements and ultrasonographic measurements of foetal parameters.

Since the introduction of modern ultrasound to obstetrics in mid 1900s, it has become possible to visualize the foetus and to make direct measurements of the foetal weight more effectively. With the use of consistently obtainable ultrasound measurements, it has been possible to reliably predict gestational age using crown rump length in 6-12 wks and bi-parietal diameter from 13-30 wks.

Bi-parietal diameter (BPD) is the most documented obstetric ultrasound measurement taken in the trans-axial plane of the widest portion of the foetal skull with thalamus positioned in the midline. **Donald F et al (1961)[32]** found accuracy in using BPD only was ±485g in 66% cases.

Willocks and associates[33], Koharn[34], Thompson et al[35] and Ianniruberto et al[36] tried with less success to relate foetal BPD to birth weight. They found that although their average error of prediction was approximately 400g, their accuracy was much less with weight <2500 g and >4200g, especially in babies with IUGR.

Foetal abdominal circumference (AC) measured at the level of portal vein, also reflects the size of the liver in the foetus. **Campbell and Wilkin (1975)[37]** reported use of foetal abdomenometry for prediction of foetal weight. They showed 95% predicted values were within 290,450, 590 g for actual weights of 2Kg, 3Kg, and 4Kg respectively.

Kurjak and **Breyer (1975)[38]** estimated birth weight within 250g in 94% cases using foetal abdominal circumference.

Warsof et al (1977)[39], in an effort to better predict foetal weight, performed a direct computer assisted statistical analysis to determine which of the three-BPD, abdominal circumference or intrauterine volume correlated best with birth weight. Results show that birth weight is a logarithmic function of foetal body parameters and that abdominal circumference has the single best correlation with birth weight. Best linear regression with use of two foetal dimensions with birth weight. Best linear regression with use of two foetal dimensions (AC and BPD) had a standard deviation of 106 g/kg foetal weight.

Absolute mean error in Birth weight was 228g (8%). 78% estimates fell within 10% of actual birth weight. Sheppard et al (1982) found that foetal weight was underestimated by approximately 3-4% using warsofs regression model. They developed another regression model with standard equivalent and systematic underestimation of weight.

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Hadlock et al (1985)[40] demonstrated that the most accurate estimates of foetal weight in utero using ultrasound were those based on at least three measurements . BPD/HC as an index of head size, abdominal circumference as an index for body girth and femur length as an index of crown heel length.

Cromi A et al (2007)[21] concluded that, a large umbilical cord area assessed by ultrasound is a simple and reliable marker of foetal macrosomia. Umbilical cord area measurement may be combined with the standard foetal biometric parameters to improve the accuracy of identification of foetal macrosomia, allowing it to be better managed without unnecessary intervention, while possibly avoiding permanent injury.

Thus, estimating foetal weights though, appears simple, is actually a complex problem. Clinical examination appears to be routine procedure but requires expertise. Disadvantages being that maternal abdominal fat, liquor volume, multiple gestation may alter the perception regarding foetal size. Ultrasound allows us to measure the foetal parameters and thus reduce effects of maternal abdominal fat content, uterine wall, and the liquor volume. It also allows for assessing biophysical profile of the foetus. Ultrasound biometry is also subjected for limitation, hence requires additional methods for precision.

The purpose of this study was to determine the correlation between sonographic cross-sectional area of the umbilical cord with birth weight and, if so, to assess whether inclusion of umbilical cord area measurement in conventional biometry may improve prenatal detection of macrosomia.

MATERIALS AND METHODS

This study was conducted between August 2010 and August 2011, in the Department of Obstetrics and Gynaecology, St. Philomena's Hospital, Bangalore.

Umbilical cord cross-sectional area of 250 antenatal women was evaluated from 34 weeks of gestational age.

Inclusion criteria

- Singleton pregnancies.
- Gestational age at and above 34 weeks.
- Presence of three vessel cord.
- Intact membranes. umbilical

Exclusion criteria

1. Multiple pregnancies
2. Intrauterine death
3. Presence of foetal anomalies.
4. PPROM and PROM

Calculation of gestational age was based on reliable recollection of the last menstrual period and confirmed or modified by ultrasound within the first 14 weeks of gestation. All sonographic examinations were performed using Toshiba Nimio Ultrasound Machine with 5 MHz transabdominal transducer. All sonographic measurements were obtained by same experienced operator.

Foetal anthropometric parameters, biparietal diameter (BPD), abdominal circumference (AC) and femur length (FL), were measured in all foetuses.

EFW (Estimated foetal weight) was obtained using the formula proposed by Hadlock et al additionally, the sonographic cross-sectional areas of the umbilical cord was measured in a free loop of the umbilical cord, using the software of the ultrasound machine, as previously described.

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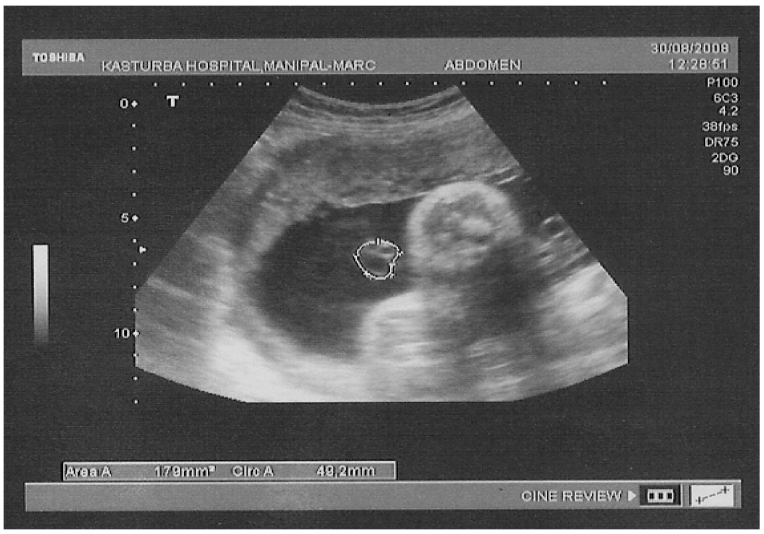

A large umbilical cord was defined when its sonographic cross sectional area was above the 95th percentile.

A lean umbilical cord was defined when its sonographic cross-sectional area was less than 10th percentile.

The outcome measured was birth weight. Weight of newborn was measured immediately after birth. Macrosomia was defined as birth weight >90th percentile in the study group.
Small for gestational age was defined as a birth weight below the 10th percentile in the study group.

The following data were collected from the medical records: parity, maternal age, sex, body mass index (BMI), gestational diabetes mellitus, gestational age at delivery, mode of delivery, birth weight of the neonate.

Statistical Methods: Descriptive statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean(SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5% level of significance. The following assumptions on data is made. Assumptions**: 1.** Dependent variables should be normally distributed, 2. Samples drawn from the population should be random, Cases of the samples should be independent

Analysis of variance (ANOVA) has been used to find the significance of study parameters between three or more groups of patients, Student "t" test (two-tailed, independent) has been used to find the significance of study parameters on continuous scale between two groups Inter group analysis) on metric parameters. Leven1s test for homogeneity of variance has been performed to assess the homogeneity of variance. Chi-square/Fisher Exact test has been used to find the significance of study parameters on categorical scale between two or more groups.

1. **Sample Size estimation**

Proportion Known populations

$n = [(z^2 * p * q) + ME^2] / [ME^2 + z^2 * p * q / N]$

Proportion Unknown population

$n = [(z^2 * p * q) + ME^2] / (ME^2)$

ME: is the margin of error, measure of precision.
and Z is 1.96 as critical value at 95%CI
N: population size
n: Sample size

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σ: Standard deviation
z: Critical value based on Normal distribution at 95% Confidence Interval

Standard deviation: $$SD = \sqrt{\frac{\sum (x - \bar{x})^2}{n-1}}$$

2. **Analysis of Variance**: F test for K Population means

Objective: To test the hypothesis that K samples from K Populations with the same mean.

The mathematical model that describes the relationship between the response and treatment for the one-way ANOVA is given by:

$$Y_{ij} = \mu + \tau_i + \varepsilon_{ij}$$

where Y_{ij} represents the j-th observation (j = 1, 2, ...n_i) on the i-th treatment (i = 1, 2, ..., k levels)

Limitations: It is assumed that populations are normally distributed and have equal variance. It is also assumed that samples are independent of each other.

Method: Let the j^{th} sample contain n_j elements (j=1,2,…K). Then the total number of elements is

$$N = \sum nj \qquad x.j = \sum \frac{xij}{nj}$$

$$S_1^{\,2} = \frac{\sum \sum_{i-1}^{n1} (x1 - \overline{x.j})^2}{N - K} \qquad S_2^{\,2} = \frac{\sum_{i-1}^{n1} nj(\overline{x.j} - \overline{x..})^2}{K - 1}$$

$F=S_2^2/S_1^2$ Which follows F distribution (K-1, N-K)

3. **The Kruskal Wallis Test**

To compare the means of K samples (K>2) using non-parametric methods

a. Pool the observations overall samples, thus constructing a combined sample of size N= $\sum ni$
b. Assign the ranks to the individual observations, using the average rank in the case of tied observations
   Compare the rank sum Ri for each k samples
c. Compute the test statistic

$$H = \frac{12}{N(N-1)} X \sum \frac{Ri^2}{ni} - 3(N+1)$$

Follows Chi-square distribution with k-1 df

4. **Tukey test**

D=Q $\sqrt{\frac{MSE}{N/J}}$ **,** N is the total number of subjects and MSE is the mean square error in ANOVA, J is the number of groups to be compared

5. **Mann Whitney U test**

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$$Z=\frac{Tobs-\mu_T}{\sigma_T}$$

Where T_{Obs} Sum of ranks in n_a Group A and n_b Group B

($_T$ Expected values is equal to $\frac{na(N+1)}{2}$ for TA and $\frac{nb(N+1)}{2}$ for TB

6. **Wilcoxon Signed Rank test**

Procedure:

a. Obtain the differences between two sets of data and rank the differences after arranging the differences in ascending and descending order.
b. Compute the rank sum of R1 of the positive differences
c. Compute

$$T=\frac{\left[\left|R1-(n(n+1)/4\right|-1/2\right]}{\sqrt{(n(n+1)(2n+1)/24}}$$

d. If T>Z1-(/2, then reject the null hypothesis, otherwise accept the null hypothesis

7. **Chi-Square Test**: The chi-square test for independence is used to determine the relationship between two variables of a sample. In this context independence means that the two factors are not related. In the chi-square test for independence the degree of freedom is equal to the number of columns in the table minus one multiplied by the number of rows in the table minus one

$$\chi^2=\frac{\sum(Oi-Ei)^2}{Ei}$$

, Where Oi is Observed frequency and Ei is Expected frequency
With (n-1) df

The Assumptions of Chi-square test

The chi square test, when used with the standard approximation that a chi-square distribution is applicable, has the following assumptions:

- **Random sample**: A random sampling of the data from a fixed distribution or population.
- **Sample size (whole table)**: A sample with a sufficiently large size is assumed. If a chi square test is conducted on a sample with a smaller size, then the chi square test will yield an inaccurate inference. The researcher, by using chi square test on small samples, might end up committing a Type II error.
- **Expected Cell Count**: Adequate expected cell counts. Some require 5 or more, and others require 10 or more. A common rule is 5 or more in all cells of a 2-by-2 table, and 5 or more in 80% of cells in larger tables, but no cells with zero expected count. When this assumption is not met, Fisher Exact test or Yates' correction is applied.

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8. **Fisher Exact Test:** The Fisher Exact Test looks at a contingency table which displays how different treatments have produced different outcomes. Its null hypothesis is that treatments do not affect outcomes-- that the two are independent. Reject the null hypothesis (i.e., conclude treatment affects outcome) if p is "small".

The usual approach to contingency tables is to apply the (^{2} statistic to each cell of the table. One should probably use the (^{2} approach, unless you have a special reason. The most common reason to avoid (^{2} is because you have small expectation values

Fisher Exact test (rxc tables)

Let there exist two such variables X and Y, with m and n observed states, respectively. Now form an $m \times n$ matrix in which the entries a_{ij} represent the number of observations in which $x = i$ and $y = j$. Calculate the row and column sums R_i and C_j, respectively, and the total sum

$$N = \sum_i R_i = \sum_j C_j$$

of the matrix. Then calculate the conditional probability of getting the actual matrix given the particular row and column sums, given by

$$P_{\text{cutoff}} = \frac{(R_1!\, R_2! \cdots R_m!)\,(C_1!\, C_2! \cdots C_n!)}{N! \prod_{i,j} a_{ij}!},$$

which is a multivariate generalization of the hypergeometric probability function.

9. **Student t test (Two tailed, independent)**

Assumptions: Subjects are randomly assigned to one of two groups. The distribution of the means being compared are normal with equal variances.

Test: The hypotheses for the comparison of two independent groups are:

H_o: $u_1 = u_2$ (means of the two groups are equal)

H_a: u_1 u_2 (means of the two group are not equal)

The test statistic for is t, with $n_1 + n_2 - 2$ degrees of freedom, where n_1 and n_2 are the sample sizes for groups 1 and 2. A low p-value for this test (less than 0.05 for example) means that there is evidence to reject the null hypothesis in favour of the alternative hypothesis. Or, there is evidence that the difference in the two means are statistically significant. The test statistic is as follows:

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t-Test: Two-Sample Assuming Equal Variances

$$S_p = \sqrt{\frac{(n_1 - 1)S_1^2 + (n_2 - 1)S_2^2}{n_1 + n_2 - 2}}$$

In all work with two-sample t-test the degrees of freedom or df is:

$$df = n_1 + n_2 - 2$$

The formula for the two sample t-test is:

$$T = \frac{\overline{X} - \overline{Y}}{S_p \sqrt{\frac{1}{n_1} + \frac{1}{n_2}}}$$

Pre-test: Test for variance assumption: A test of the equality of variance is used to test the assumption of equal variances. The test statistic is F with n_1-1 and n_2-1 degrees of freedom.

t-Test: Two-Sample Assuming Unequal Variances

$$T = \frac{\overline{X} - \overline{Y}}{\sqrt{\frac{S_X^2}{n_1} + \frac{S_Y^2}{n_2}}}$$

Note in this case the Degree of Freedom is measured by

$$df' = \frac{\left(\frac{S_1^2}{n_1} + \frac{S_2^2}{n_2}\right)^2}{\frac{\left(\frac{S_1^2}{n_1}\right)^2}{n_1 - 1} + \frac{\left(\frac{S_2^2}{n_2}\right)^2}{n_2 - 1}}$$

and round up to integer.

Results of the t-test: If the p-value associated with the t-test is small (< 0.05), there is evidence to reject the null hypothesis in favour of the alternative. In other words, there is evidence that the means are significantly

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different at the significance level reported by the p-value. If the p-value associated with the t-test is not small (> 0.05), there is not enough evidence to reject the null hypothesis, and you conclude that there is evidence that the means are not different.

10. **Significant figures**

+	Suggestive significance	(p value: 0.05<p<0.10)
*	Moderately significant	(p value: 0.01<p(0.05)
**	Strongly significant	(p value: p(0.01)

Statistical software: The Statistical software namely SAS 9.2, SPSS 15.0, Stata 10.1, MedCalc 9.0.1, Systat 12.0 and R environment ver.2.11.1 were used for the analysis of the data and Microsoft Word and Excel have been used to generate graphs, tables etc**.**

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RESULTS AND ANALYSIS

This was a prospective observational study of 250 pregnant women at or more than 34 weeks of gestation, attending antenatal clinic, in the Department of Obstetrics and Gynaecology, St. Philomena's Hospital, Bangalore between August 2010 and August 2011.

Table 1: Study population (n=250)

Parity	Number of cases n (%)
Primi	124 (49.6)
Multi	126 (50.4)

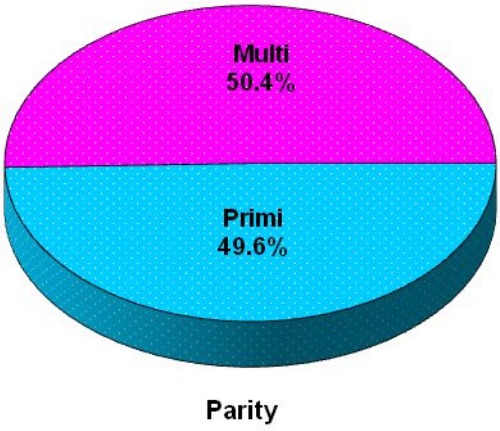

Figure 10

Out of 250 antenatal women who were recruited for the study primigravida, multigravidas were 49.6% and 50.40% respectively (Table 1, Fig 10).

Table 2: Age distribution in Study population (n=250)

Age in years	Number of cases n (%)
18-20	15 (6.0)
21-30	207 (82.8)
>30	28 (11.2)

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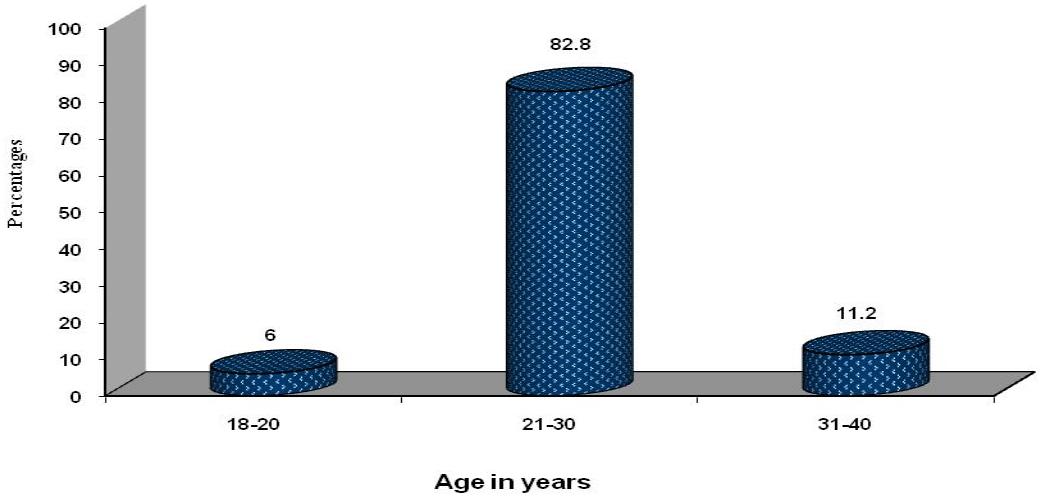

Figure 11

Age distribution among the study population was shown in Table 2 (Fig 11).

6% were in the age group of 18-20 years, 82.8% were in the age group of 21-30 years and 11.2% were in the age group of 31-40 years.

Table 3: Range & Mean of umbilical cord cross-sectional area

n=250	Range	Mean	SD
Umbilical cord cross sectional area (mm^2)	112-320	212.388	49.76

The mean umbilical cord cross-sectional area of all the patients recruited in this study (N=250) was 212.388 millimetre square with a standard deviation of 49.76. The umbilical cord cross-sectional area was in the range of 112-320 mm^2.

Table 4: Categories of umbilical cord cross-sectional area

	Umbilical cord cross-sectional area (mm^2)	n=250	%
Lean cord	<146.2	25	10.0
Large cord	>288	13	5.2
Normal cord	146.2 - 288	212	84.8

Table 4 portrays the division of women into three umbilical cord cross-sectional area categories based on the percentiles. The 10^{th} and the 95^{th} percentile values were 146.2 and 288 mm^2 respectively. Hence, cases with umbilical cord cross-sectional area < 146.2 mm^2 were categorized as lean cord and those with umbilical cord cross-sectional area > 288 mm^2 were categorized as large cord. Those between 146.2 and 288 mm^2 were categorized as normal cord.

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- **Lean cord** < 146.2 mm^2
- **Large cord** > 288 mm^2
- **Normal cord** 146.2 – 288 mm^2

As shown in table 4 there were a total of 25 (10.0%) cases in the lean cord, 13 (5.2%) in the large cord group and 212 (84.8%) in the normal cord group.

Table 5: Range and mean of birth weight

n=250	Range	Mean	Standard deviation
Birth weight (grams)	1900 – 4320	3015.628	480.17

The mean birth weight in the study population (n=250) was 3015.628 grams with a standard deviation of 480.17. The birth weight was in the range of 1900-4320 grams.

Table 6: Categories of birth weight

	Birth weight (grams)	n=250	%
SGA	< 2441	25	10.0
Macrosomia	>3699	25	10.0
Normal	2441 – 3699	200	80.0

Table 6 portrays the division of infants into three birth weight categories based on the percentiles. The 10^{th} and the 90^{th} percentile values were 2441 and 3699 grams respectively. Hence cases with birth weight < 2441 grams were categorized as small for gestational age infants and those with birth weight > 3699 grams were categorised as macrosomic infants.

- **SGA** < 2441 grams
- **Macrosomia** > 3699 grams
- **Normal** 2441 – 3699 grams

As shown in table 6 there were a total of 25 (10.0%) cases in the SGA group, 25 (10.0%) in the macrosomic group and 200 (80.0%) in the normal birth weight group.

Table 7: Correlation between umbilical cord cross-sectional area with birth weight

Umbilical Cord cross-sectional area (mm^2)	Number of babies (n=250)	% of babies	Mean birth weight(gm)	SD
100-125	8	3.2	2136.25	348.83
126-150	21	8.4	2427.62	256.81
151-175	38	15.2	2673.42	231.51
176-200	46	18.4	2845.02	234.54
201-225	44	17.6	3061.84	285.66

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226-250	26	10.4	3160.85	291.04
251-275	22	8.8	3368.36	298.58
276-300	45	18.0	3608.20	340.34

The study showed that as umbilical cord cross-sectional area increases, mean birth weight also increases (Table 7).

Table 8: Correlation between umbilical cord cross-sectional area with birth weight

Pearson correlation	r = 0.819 , p < 0.001 (significant)

In the study population, there was positive correlation between umbilical cord cross-sectional area and birth weight, which was statistically significant (Table 8).

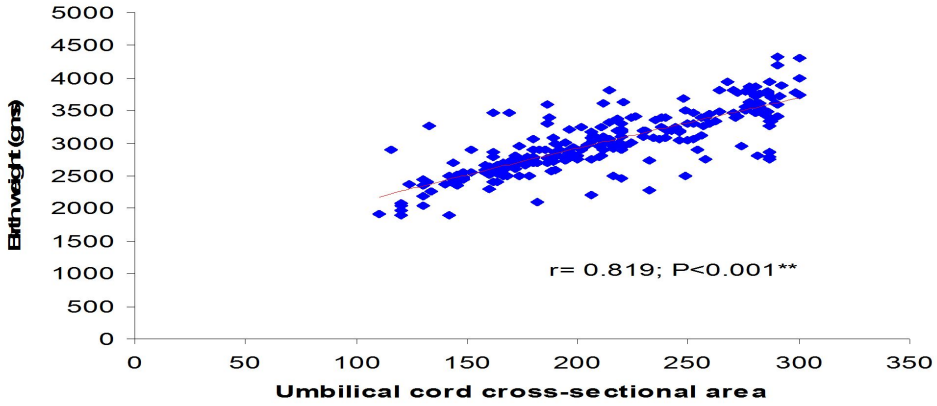

Fig. 12: Correlation between umbilical cord cross-sectional area and birth weight (Scatter plot graph)

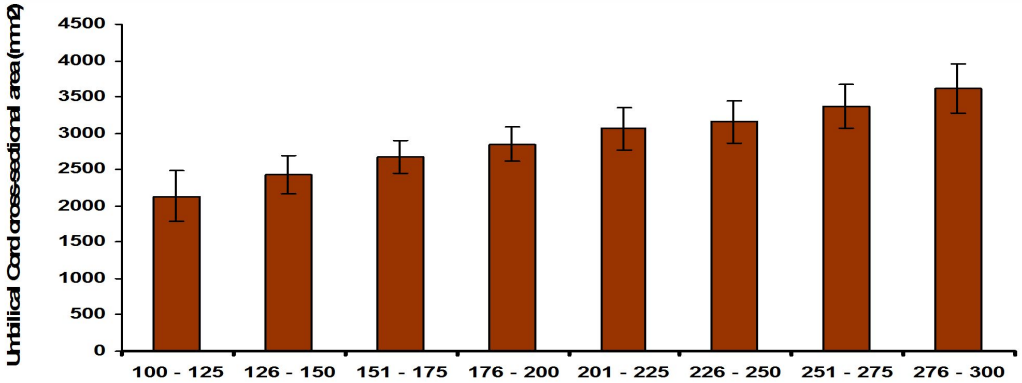

Fig.13.Correlation between umbilical cord cross-sectional area and birth weight

In the study population as umbilical cord cross-sectional area increased, the mean birth weight also increased (Fig.12, 13).

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Table 9: Comparison of large cord (> 95th percentile) with macrosomia

Umbilical cord cross-sectional area(95th percentile)	Birth weight (gm):90th percentile	
	<3699 grams	>3699 grams
<288.0 mm^2	221(98.2%)	16(64.0%)
>288.0 mm^2	4(1.8%)	9(36.0%)
Total	225(100.0%)	25(100.0%)
Inference	Higher umbilical cord cross-sectional area (mm^2) is significantly associated with higher birth weight (>3699 gm) in 90th percentile with (^{2}=53.456; p<0.001**(significant)	

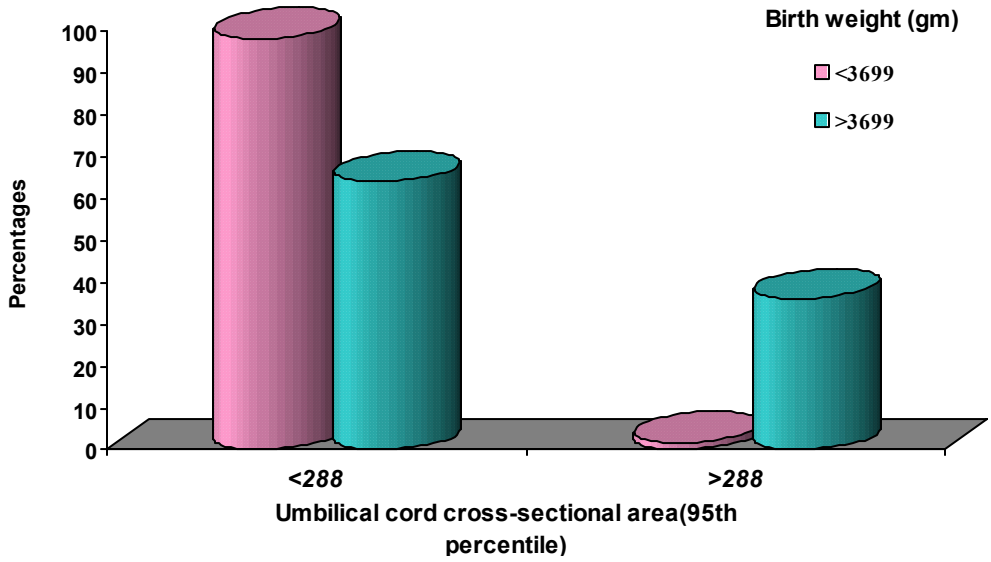

Figure 14

In our study, proportion of cases with a large umbilical cord was significantly higher in the group of macrosomic (36.0%) compared with non macrosomic infants (1.8%). This difference was statistically significant (Table 9, Fig 14).

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Table 10: Comparison of lean umbilical cord with small for gestational age (SGA)

Umbilical cord cross-sectional area(10th percentile)	Birth weight (gm):10th percentile	
	<2441grams	>2441grams
<146.20 mm^2	18(72.0%)	7(3.1%)
>146.20 mm^2	7(28.0%)	218(96.9%)
Total	25(100.0%)	225(100.0%)
Inference	Lower umbilical cord cross-sectional area (mm^2) is significantly associated with lower birth weight <2441 gm) 10th percentile with (^{2}=118.642; p<0.001**	

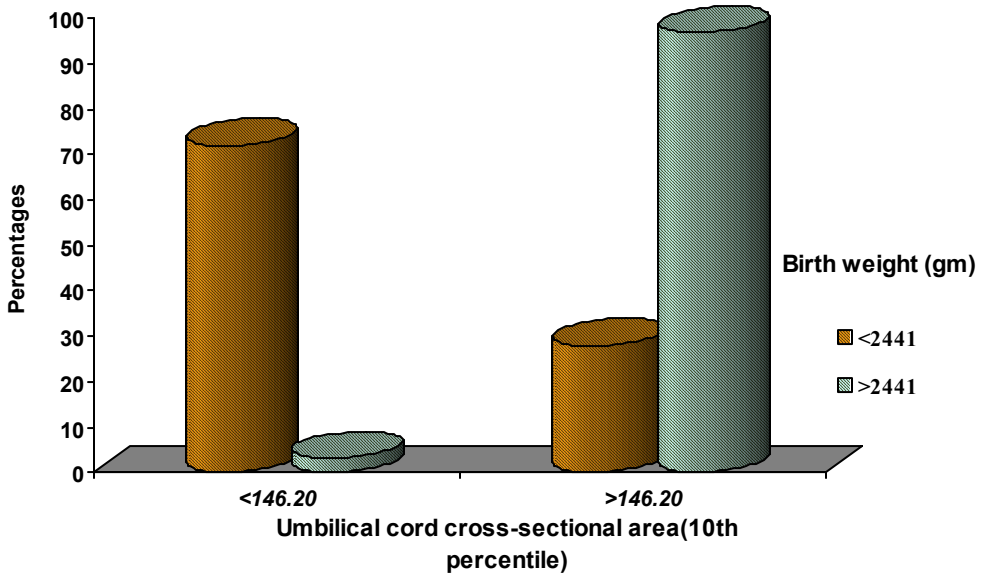

Figure 15

In our study, proportion of cases with a lean umbilical cord was significantly higher in the group of small for gestational group (72.0%) compared with other group (3.1%).This difference was statistically significant (Table 10,Fig 15).

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Table 11: Comparison of maternal BMI with birth weight

BMI (kg/m^2)	Number of neonates	Birth weight (gm)		
		<10th percentile	10-90 percentile	>90 percentile
<25.0	150	23(15.3%)	119(79.4%)	8(5.3%)
25.0-30.0	95	1(1.1%)	77(81.1%)	17(17.9%)
>30.0	5	1(20.0%)	4(80.0%)	0
Total	250	25(10.0%)	200(80.0%)	25(10.0%)
Inference	BMI is significantly associated with birth weight with (^{2}=22.1; p<0.001**			

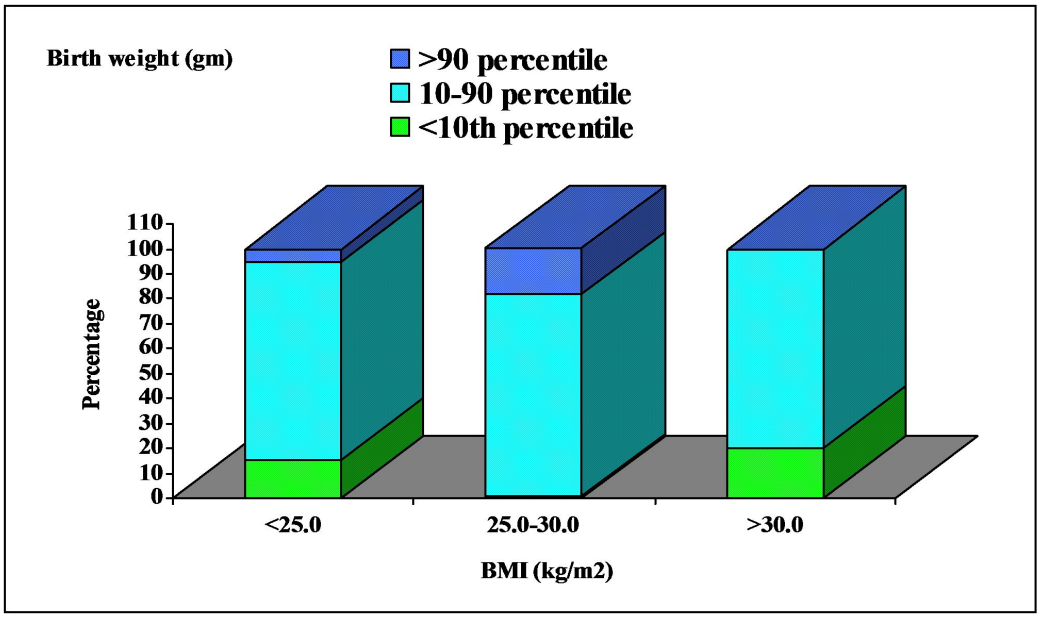

Figure 16

In our study, comparison of maternal BMI with infant birth weight was found to be statistically significant p < 0.001(Table11,Fig 16).

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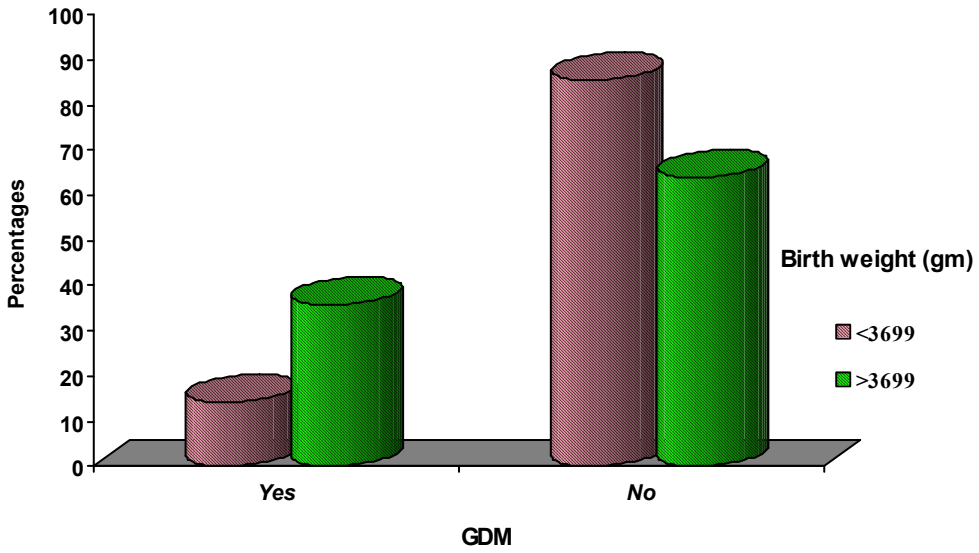
Table 12: Correlation between Incidence of GDM with birth weight (90th percentile)

GDM	Birth weight (gm):90th percentile	
	<3699 grams	>3699 grams
Yes	32(14.2%)	9(36.0%)
No	193(85.8%)	16(64.0%)
Total	225(100.0%)	25(100.0%)
Inference	Incidence of GDM is significantly associated with higher birth weight (>3699 gm) in 90th percentile with (^{2}=7.783; P=0.005**	

Figure 17



In our study incidence of GDM is significantly associated with higher birth weight, which was statistically significant (^{2}=7.783; P=0.005 (Table 12, Fig 17)

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Table 13: Correlation between Incidence of GDM with large umbilical cord cross-sectional area(95th percentile)

GDM	Umbilical cord cross-sectional area(95th percentile)	
	<288 mm²	>288 mm²
Yes	36(15.2%)	5(38.5%)
No	201(84.8%)	8(61.5%)
Total	237(100.0%)	13(100.0%)
Inference	Incidence of GDM is significantly associated with higher UCCA (>288 mm2) in 95th percentile with (^{2}=4.866; p=0.027*	

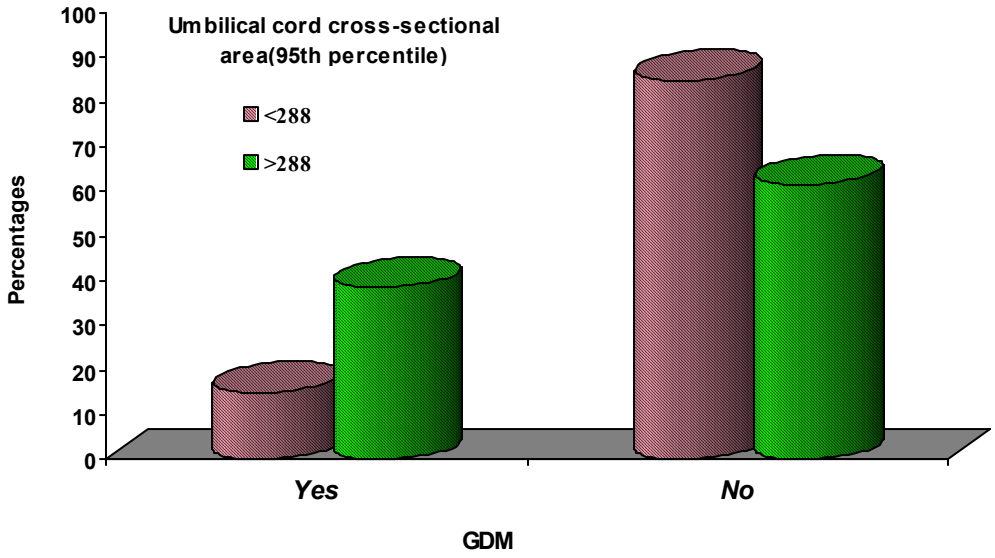

Figure 18

In our study pregnancies complicated by GDM had large umbilical cord compared with non GDM. This difference was statistically significant (^{2}=4.866; p=0.027 (Table 13, Fig 18).

DISCUSSION

Since ages clinical experience had shown association between cord abnormalities and adverse foetal outcomes, in terms of foetal growth restriction and macrosomia which in turn translates into an increased incidence of perinatal morbidity and mortality.

Research has been done to find an ideal sonographic parameter to estimate the foetal weight, accuracy of which will help in appropriate decision making and hence optimize the pregnancy outcome.

In the quest to search for such a parameter my study has attempted to correlate cross sectional area of umbilical cord with birth weight and if so whether it can be used as a parameter to measure foetal birth weight.

This prospective observational study was conducted between August 2010 and August 2011, in the Department of Obstetrics and Gynaecology, St. Philomena's Hospital, Bangalore, to evaluate the correlation between foetal umbilical cord cross-sectional area with birth weight and to compare large cross-sectional area of the umbilical cord with macrosomia.

Umbilical cord cross-sectional area of 250 antenatal women was evaluated from 34 weeks to 40 weeks of gestation, excluding multiple pregnancies and those with presence of foetal anomalies.

All sonographic examinations were performed using Toshiba Nimio Ultrasound Machine with 5 MHz transabdominal transducer. The sonographic cross-sectional area of the umbilical cord was measured in a free loop of the umbilical cord.

There is a progressive increase of the umbilical cord diameter and cross-sectional area up to 32 weeks of gestation.This is in agreement with the study by Weissman et al[41] who reported nomograms of the umbilical cord diameter and vessels. These authors extrapolated the surface area of Wharton's jelly at each gestational age and found that there is a reduction of Wharton's jelly toward the end of the pregnancy. This is in keeping with previous reports in which decreased umbilical cord water content has been noted with increase in gestational age. So we have considered the gestational age group between 34 to 40 weeks in our prospective study.

In our study 124(49.6%) were primigravida and 126(50.6%) were multigravida, so parity distribution was similar.

In the study majority were i.e., 82.8% were between 21-30 years of age, the prime reproductive age group in our population.

To compare the umbilical cross sectional area with birth weight it is important to define what are the normal dimensions of large, lean and normal cord.In our study lean cord was defined as less than 10th percentile which corresponds to less than 146.2 mm^2, large cord more than 95th percentile which corresponds to more than 288 mm^2 and normal cord between 10th and 95th percentile i.e., 146.2 – 288 mm^2 similar approach was done by **Cromi et al**[21].

In our study to define macrosomia, SGA and normal for gestational age, we have considered more than 90th percentile which corresponds to 3699gms as macrosomia, less than 10th percentile which corresponds to 2441gm as SGA and between 10th and 90th percentile i.e., between 2441- 3699gms was taken as normal birth weight.

In our study we found that as the umbilical cord cross-sectional area increased and birth weight also increased (Table 7, 8). This is in accordance with a previous study by **Prabhcharan and Jarjoura**[42] who reported a significant relationship between the umbilical cord cross-sectional area and neonatal birth weight.

In our study, proportion of cases with a large umbilical cord was significantly higher in the group of macrosomic (36.0%) compared with non macrosomic infants (1.8%) (Table 9, Fig 14), this is in accordance with a previous study by **Cromi A et al**[21] and proportion of cases with a lean umbilical cord was significantly higher in the group of small for gestational group (72.0%) compared with other group (3.1%) as also found by **Raio et al**.[18] This difference were statistically significant (Table 10, Fig 15).

Several reports in the literature have described a large umbilical cord associated with other foetal structural anomalies such as umbilical cord tumour, urachal cysts, umbilical cord mucoid degeneration and omphalomesenteric cysts[20] but in our study no such abnormalities noted.

In our study, comparison of maternal BMI with infant birth weight was found to be statistically significant (Table 11). This finding is in accordance with that of **Prabhacharan and Jarjoura**[42] who reported that infants born to women with higher prepregnancy weight are heavier at birth. This is because they have an advantage with regard to the quantity of Wharton's jelly wrapped around their umbilical cord vessels.

In our study, incidence of macrosomia is higher in GDM mothers and also in pregnancies complicated by diabetes mellitus 5 (12.1%) had large umbilical cord, whereas only 8 (3.8%) of non GDM group had large umbilical cord. This difference was statistically significant (p=0.027) (Table 12,13, Fig 17,18). Weissman and Jakobi[20] reported that foetuses of patients with gestational diabetes have larger umbilical cords than foetuses of non-diabetic patients and that this is mainly due to a higher content of Wharton's jelly. These authors found an alteration in the distribution of Wharton's jelly fibers with large empty spaces among them and speculated that this could cause accumulation of fluid and plasma proteins within the Wharton's jelly, resulting in an increased surface area. This

modification was already present at 24 weeks' gestation suggesting that the involvement of the umbilical cord in foetuses of diabetic mothers is a phenomenon that occurs early in pregnancy.

Therefore, it can be speculated that an abnormally large umbilical cord might serve as an additional parameter that can help to identify foetuses of a mother with some kind of glucose intolerance during pregnancy. Further studies are, however, needed to confirm it.

Antenatal measurement of the umbilical cord area is probably a better parameter than determination of the umbilical cord diameter to identify foetuses at risk of being small for gestational age at delivery or macrosomic, because it has been demonstrated that, in the case of segmental thinning of the umbilical cord, the greater reduction of Wharton's jelly occurs especially around the umbilical arteries. Thus, considering that the cross-sectional shape of the umbilical cord may not be perfectly circular, minimal reduction of Wharton's jelly without modification of the arterial lumen could be underestimated with only the evaluation of the umbilical cord diameter. Since the umbilical cord cross-sectional area is easy to measure and nomograms are now available, we suggest that the measurement of umbilical cord cross sectional area could be part of a routine ultrasound evaluation and should prompt the physician to carefully evaluate the case whenever there is a discrepancy between the observed and the normal values. In case of abnormal size of the umbilical cord, a careful monitoring of the pregnancy should be undertaken.

CONCLUSION

There is a positive correlation between umbilical cord cross-sectional area and birth weight. As umbilical cord cross-sectional area increases, there is increase in mean birth weight. Proportion of cases with a large umbilical cord was significantly higher in the group of macrosomic compared with non-macrosomic neonate (36.0% vs. 1.8%).

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