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**Original Research Article**

# Abnormalities of the umbilical cord: correlation with placental histology and perinatal outcome

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## ABSTRACT

**Background:** The purpose of this study was to evaluate gross umbilical cord abnormalities and to examine its relationship with placental pathological features and perinatal outcome.
**Methods:** We retrospectively collected 101 singleton placentas with gross umbilical cord abnormalities over 3-year period (delivered on or after 20 weeks of gestational age). Association between gross umbilical cord abnormalities such as hypercoiled cords, hypocoiled cords, thin cords, true knot, strictures, excessively long cord, and abnormal cord insertions were reviewed and evaluated for placental histology, clinical parameters and perinatal outcome.
**Results:** The most common umbilical cord abnormality (UCA) observed was coiling abnormality (hypercoiling, hypocoiling and strictures). Gross UCAs were associated with maternal factors (preeclampsia, oligohydramnios and gestational diabetes), foetal factors (intrauterine foetal death, SGA and intrapartum complications such as NRFHR tracing and low Apgar scores) and histologic placental features (maternal vascular malperfusion, foetal vascular malperfusion, villous capillary lesions and inflammation). Cords with multiple abnormalities were significantly correlated with histologic evidence of foetal vascular obstruction and intrauterine foetal death.
**Conclusions:** Gross UCAs, especially when multiple were associated with clinically significant placental findings and adverse perinatal outcome. Therefore, our study reinforces that all placentas with gross UCAs should be submitted for examination with complete (full length of the) umbilical cord.

**Keywords:** Coiling abnormality perinatal outcome, Foetal vascular malperfusion, Maternal vascular malperfusion, Placental histology, Umbilical cord abnormalities

## INTRODUCTION

Umbilical cord (UC) is a vital structure which acts as a conduit between the developing foetus and placenta. It carries nutrients, oxygen and fluids necessary for the intrauterine life. This unique lifeline needs optimal protection which is provided by Wharton's jelly, amniotic fluid and coiling of the umbilical vessels.[1] The fully developed umbilical cord is pearly white, 50-60cms long with an average diameter of 1-2cm. It contains 2 arteries and a vein which course through the Wharton's jelly in a helical fashion with one coil/twist every 5 cms of cord. The cord normally inserts on the placental surface, more often near or at the center.[2]

Conditions such as abnormal cord length, abnormal coiling, knots, entanglements, constrictions, velamentous vessels, single umbilical artery (SUA) and thrombosis

may lead to cord compression and consequent reduction of blood flow in the umbilical vessels and have been associated with adverse foetal outcome and intrauterine foetal demise (IUFD).[3]

The purpose of this study was to evaluate gross patterns of umbilical cord abnormalities and determine correlations with placental histology and perinatal outcome.

## METHODS

This retrospective study was carried out from January 2014 to December 2016 in the Departments of Obstetrics and Gynaecology and Department of Pathology.

101 Placentas with gross umbilical cord abnormalities delivered on or after 20 weeks of gestational age were taken for study consideration. Clinical indications for histological examination of the placenta were stillbirth, foetal growth restriction, preterm premature rupture of membranes, severe pre- eclampsia, diabetes, gross abnormalities of the placenta, clinical suspicion of infection or abruption and compromised neonatal outcome.

Umbilical cord abnormalities (UCA) included in the study were hypercoiled cord (>3 coils/10cm segment of umbilical cord), hypocoiled cord (< 1 coil per 10 cm), thin /narrow cord due to diminished Wharton's jelly (maximum cord diameter <0.8 cm), strictures (focal narrowing/thinning in relation to the remaining UC), true knot(TK), SUA, abnormal cord insertion-marginal (< 1cm from the true disc margin-MUC) / velamentous ( membranous insertion-VEL) / furcate (umbilical vessels splitting and leaving Wharton's jelly prior to reaching the chorionic plate surface).[4] Excessively long umbilical cord (ELUC) was defined as those UCs measuring approximately 2 standard deviations (SD) above the mean or ≥ 70cms in length.[4,5]

Clinical details obtained along with the placental specimens were maternal age, gravidity and parity, gestational age, medical conditions complicating pregnancies, presence of IUFD, foetal growth restriction (FGR), non-reassuring foetal heart rate (NRFHR), meconium stained liquor, Apgar scores at 1 and 5 minutes and birth weight as recorded by the obstetricians. Infants with birth weight <10$^{th}$ percentile and >90th percentile of the gestational age were termed as small for gestational age (SGA) and large for gestational age (LGA) respectively. The rest were considered as appropriate for gestational age (AGA).[6]

Umbilical cord and placental assessment were based on gross and microscopic examinations. An entire placenta was available from all 101 patients and were fixed in 10% formalin. Placentas were weighed after the membranes and umbilical cord were trimmed off and compared with the reference weights for trimmed singleton placentas.[7]

Umbilical cord was examined for length, diameter, colour, insertion site, number of twists and vessels. Photography of the placentas were routinely taken after examining foetal, maternal surfaces and parenchyma. Relevant lesional sections were taken along with 2 sections of membrane roll, 2 or 3 sections from the UC at the stricture and non-strictured area and 3 full thickness sections of the normal parenchyma. All the histologic sections were stained with haematoxylin and eosin.

Umbilical cord abnormalities were correlated with gross, histologic placental characteristics and foetal outcome.

## RESULTS

Hundred and one cases were identified with gross UCAs. There were 48 cases of hypercoiling (Figure 1), 31 cases of hypocoiling (Figure 2) and 11 cases each of thin cords and strictures (Figure 3). Abnormal cord insertions were seen in 7 cases each of MUC, VEL (Figure 4) and 2 cases of furcate. Other less commonly seen abnormalities were 2 cases each of TK (Figure 5), ELUC and SUA. Of the 101 cases of analysed placentas 20 had more than one umbilical cord abnormality.

Isolated UCAs were each analyzed for association with clinical and histologic findings (Tables 1 and Table 2).

### *Clinical characteristics*

The most common UCA identified was hypercoiling, followed by hypocoiling. Stillbirth was observed in 42 cases across all cord abnormalities but was by far most frequently associated with abnormal coiling.

Preeclampsia, oligohydramnios and gestational diabetes were the commonest maternal characteristics observed in UCAs. Infections, PPROM, polyhydramnios and autoimmune diseases were other maternal features identified in altered structure of UCs.

NRFHR, low Apgar and Meconium stained liquor were common in coiling abnormalities, more so in the group with hypercoiling. Fifty percent of hypercoiled and one third of hypocoiled cords had birth weights of < 10th percentile.

### *Placental findings*

More than 50% of the placentas with UCAs were small for gestational age. Significant histologic features identified were maternal vascular malperfusion (MVM-35/101), foetal vascular malperfusion (FVM-26/101) and villous capillary lesions (19/101). The other microscopic features noted were distal villous immaturity (DVI), massive perivillous fibrin deposition (MPVFD), and inflammatory lesions.

**Table 1: Gross umbilical cord abnormalities and associated placental histology.**

| Features | Hyper Coiled (n= 48) | Hypocoiled (n= 31) | Thin Cord (n=11) | TK (n=2) | Strictures (n=11) | ELUC (n=2) | SUA (n=2) | VEL (n=7) | MUC (n=7) | Furcate cord(n=2) |
|---|---|---|---|---|---|---|---|---|---|---|
| Placental weight | | | | | | | | | | |
| SGA | 31 | 19 | 10 | 0 | 4 | 1 | 2 | 4 | 5 | 1 |
| MVM | 21 | 8 | 4 | 0 | 1 | 0 | 1 | 3 | 2 | 1 |
| Infarcts | 13 | 10 | 4 | 0 | 1 | 0 | 0 | 1 | 2 | 0 |
| Retroplacental haematoma | 3 | 5 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| Intervillous thrombus | 9 | 8 | 2 | 0 | 0 | 0 | 0 | 1 | 1 | 1 |
| MPVFD | 1 | 1 | 0 | 0 | 2 | 1 | 0 | 0 | 0 | 0 |
| FVM | 8 | 10 | 5 | 0 | 6 | 0 | 2 | 3 | 1 | 0 |
| Chorionic plate/stem villous vessel thrombus | 5 | 7 | 2 | - | 6 | 0 | 2 | 3 | 1 | 0 |
| Avascular villi | 8 | 8 | 5 | - | 6 | 0 | 2 | 3 | 0 | 0 |
| Villous Stromal Karyorrhexis | 4 | 5 | 4 | - | 2 | 0 | 1 | 0 | 0 | 0 |
| Nucleated RBCs | 2 | 3 | - | - | 1 | 0 | 1 | 1 | 0 | 0 |
| Inflammation | 8 | 9 | 3 | 1 | 2 | 0 | 1 | 1 | 4 | 1 |
| Acute chorioamnionitis | 2 | 5 | 0 | 1 | 1 | 0 | 0 | 0 | 2 | 0 |
| VUE | 6 | 4 | 3 | 0 | 1 | 0 | 1 | 1 | 2 | 1 |
| Villous capillary/vascular lesions | 14 | 3 | 3 | 2 | 2 | 1 | 0 | 0 | 0 | 0 |
| Chorangiosis | 9 | 1 | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 0 |
| Chorangiomatosis | 3 | 2 | 2 | 0 | 1 | 1 | 0 | 0 | 0 | 0 |
| Villous congestion | 2 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| DVI | 5 | 4 | 1 | 0 | 0 | 0 | 0 | 2 | 1 | 0 |

Infarcts, intervillous thrombus, retroplacental haematoma and MPVFD (histologic markers of MVM) were observed in cases with gross UCAs. The other features noted were decidual vasculopathy, distal villous hypoplasia, increased syncytial knotting, cell islands, villous agglutination, and multinucleate trophoblastic giant cells.

The histologic features of chronic FVM such as chorionic plate /stem villous vessel thrombus, avascular villi, villous stromal karyorrhexis and nucleated red blood cells were significantly more common in cords with hyper, hypo coiling and strictures. Ectatic vessels were also noted in many of the chorionic plate /stem villous vessels of FVM.

Villous vascular lesions such as chorangiosis (increased number of capillaries - more than 10 capillaries/terminal villi, in 10 villi) in many areas of the placental parenchyma, patchy and occasional multifocal chorangiomatosis (excessive capillaries with surrounding pericytes in immature intermediate / stem villi) and villous congestion were found to be associated with hypercoiled cords.

***Multiple UCAs***

Placentas with multiple UCAs and their associations with clinical and histologic findings were also analysed. IUFD was significantly associated with the presence of multiple UCAs (13/20). In the remaining live born cases the clinical outcome was SGA (6/20) and AGA (1/20). The most common maternal obstetric complication was hypertension and the commonest placental histology was foetal vascular malperfusion (FVM) (Figure 6). These findings are summarized in (Table 3).

## DISCUSSION

The coiling pattern of the UC largely affects umbilical blood flow that is vital for sustaining foetal growth and development and its pathophysiological significance has been studied by many authors. UCAs often result in mechanical compression leading to venous stasis, endothelial damage and loss of perfusion to a large area of placental gas exchanging villi and ultimately resulting in foetal distress and hypoxia.[8-10] This study analyzed the UCAs such as hypercoiling, hypocoiling, strictures, thin cord, true knot, SUA, ELUC and abnormal insertions in

relation to gross, histologic placental changes as well as clinical parameters and foetal outcome.

**Table 2: Gross umbilical cord abnormalities with clinical characteristics and perinatal outcome.**

| Features | Hyper Coiled (n= 48) | Hypo Coiled (n= 31) | Thin Cord (n=11) | TK (n=2) | Strictures (n=11) | ELUC (n=2) | SUA (n=2) | VEL (n=7) | MUC (n=7) | Furcate cord (n=2) |
|---|---|---|---|---|---|---|---|---|---|---|
| **Gestational age** | | | | | | | | | | |
| <28 weeks | 8 | 6 | 5 | 0 | 4 | 0 | 0 | 1 | 3 | 1 |
| >28 weeks | 40 | 25 | 6 | 2 | 7 | 2 | 2 | 6 | 4 | 1 |
| **Maternal factors** | | | | | | | | | | |
| HT | 15 | 6 | 2 | 0 | 1 | 0 | 0 | 2 | 3 | 0 |
| GDM | 8 | 0 | 0 | 1 | 1 | 0 | 0 | 1 | 0 | 1 |
| Rh Negative | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| Oligo | 10 | 6 | 2 | 0 | 0 | 0 | 0 | 1 | 3 | 0 |
| PPROM | 3 | 6 | 2 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| Polyhydramnios | 1 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| Auto immune | 1 | 1 | 2 | 0 | 1 | 0 | 0 | 1 | 1 | 0 |
| Infections | 4 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 |
| **Foetal factors** | | | | | | | | | | |
| SGA | 21 | 9 | 5 | 0 | 1 | 0 | 0 | 5 | 2 | 1 |
| AGA | 10 | 8 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 |
| LGA | 3 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| IUFD | 14 | 14 | 5 | 1 | 9 | 2 | 2 | 2 | 5 | 1 |
| NRFHR | 15 | 2 | 1 | 0 | 1 | 0 | 0 | 2 | 1 | 1 |
| **APGAR** | | | | | | | | | | |
| <4 @1 mt | 6 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| <7 @ 1 mt | 7 | 2 | 1 | 0 | 1 | 0 | 0 | 1 | 0 | 0 |
| Thick meconium stained liquor | 4 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

Consistent with prior studies, our investigation showed that UCA has significant association with IUFD, SGA and intrapartum complications such as NRFHR tracing and low Apgar scores.[8-10] Meconium stained liquor was not a consistent finding in our study.

***Umbilical cord coiling abnormality***

Little is known about factors determining cord coiling. It is generally believed that coiling is established very early in gestation and is thought to be the result of foetal activity. Lack of coiling may then reflect foetal inactivity, which is supported by the fact that coiling is reduced in cases of restricted foetal movements due to various causes.1 The prenatal ultrasonographic evaluation of umbilical cord is usually limited to vessel number and umbilical artery Doppler. Predanic et al demonstrated a strong correlation between mid-trimester umbilical coiling index (UCI) and post-natal coiling index.[11]

Among 101 studied placentas, abnormal coiling was the commonest UCA, which accounted for 80%. The frequency and clinical correlations of hypercoiled cords were FGR (43%), IUFD (29%), NRFHR (31%). For hypocoiled cords, the frequencies of adverse outcomes were IUFD (45%) and FGR (29%). Our results were in contrast to the study by Machin et al who observed more IUFDs among hypercoiled cords. Our data support the finding that abnormal coiling may cause chronic umbilical cord obstruction and unfavourable foetal outcome.[12]

Among hypercoiled umbilical cords, specific gross patterns of coiling were recognized with histologic evidence of foetal vascular malperfusion and stillbirth.[8]

The placental weight was small for gestational age in more than two thirds of the studied cases. Placental histologic findings associated with UC coiling abnormalities include villous capillary congestion, chorangiosis, chorionic plate / stem villous vessel thrombi, avascular villi, MVM, inflammatory lesions and DVI. Placental infarcts and intervillous thrombus are the conspicuous lesions observed among coiling abnormality cases in our series.

They represent deficient maternal circulation (maternal vascular malperfusion) leading to parts of placental atrophy or infarction in areas of poor nutrition. However,

the association between MVM and cord coiling abnormalities appear speculative.

**Table 3: Clinical, placental features and foetal outcome among different categories of multiple cord abnormalities.**

| >1UCC | Number of cases | Placental weight | Placental gross | Microscopy | Maternal complications | Foetal outcome |
|---|---|---|---|---|---|---|
| Hypercoiling+ Stricture | 4 | SGA | A) congestion around cord insertion | A) FVM | A) NIL | IUFD |
| | | SGA | B) NIL | B) vascular involution, patchy mild villitis | B) post varicella | IUFD |
| | | SGA | C) marginal atrophy and patchy | C) FVM | C)NIL | IUFD |
| | | SGA | firm pale brown areas D) NIL | D) FVM | D)NIL | IUFD |
| Hypocoiling+ Thin cord | 4 | SGA | NIL | DVI, decidual arteriopathy, laminar necrosis | A) PPROM | AGA |
| | | SGA | Uneven thickness of | FVM | B) Oligo,FGR | SGA |
| | | SGA | parenchyma Intervillous thrombus, infarcts | MVM, FVM | C)preeclampsia | IUFD |
| | | SGA | NIL | Villous hydrops,FVM,mild intervillositis,viral/thrombophilia possibilities | D)Fever | IUFD |
| Hypercoiling+ Thin cord | 3 | A)SGA | Infarcts | FVM, MVM, mild chronic villitis, obliterative vasculopathy | a)Systemic hypertenion, GDM | SGA |
| | | B)SGA | Marginal thinning,uneven thick, yellow brown lesion(infarcts) | extensive multifocal Chorangiomatosis, MVM | b)FGR | SGA |
| | | C)SGA | C subchorionic dark brown congested area | C MVM, Multifocal chorangiomatosis-treated SLE | c)autoimmune disease, PE, FGR, PPROM | SGA |
| Hypercoiling + Marginal Insertion | 2 | A)SGA | A) infarcts, intervillous thrombus | MVM | A)PE | IUFD |
| | | B)SGA | B) Basal yellow brown nodule | Infarcts, laminar necrosis, DVI | B) Autoimmune disease, PPROM | SGA |
| Hypocoiling+ Marginal | 1 | SGA | Focal Atrophy, Subchorionic Hematoma | Large transmural hematoma occupying >50% parenchyma, recanalizing thrombi (stem vill)-FVM, MVM | Systemic hypertension | IUFD |
| Hypocoiling+ Stricture | 1 | SGA | Wedge, pale yellow INFARCT | SMALL, hypocoiled cord with a stricture, MVM | FGR | SGA |
| Hypocoiling+ SUA | 1 | SGA | Edematous, congested cord, parenchymal marginal atrophy | FVM | Polyhydramnios | IUFD |
| VEL + stricture | 1 | SGA | Uneven thickness of parenchyma | Small placenta with umbilical cord stricture and a velamentous vessel with thrombus FVM vascular involution following foetal death with ? Temporal heterogeneity | Autoimmune disease | IUFD |
| VEL+Furcate | 1 | SGA | succenturiate, furcate cord | Chronic histiocytic intervillositis | Fever with rash | IUFD |
| ELUC+Nuchal+Stricture | 1 | SGA | maternal surface-plaque like thickening >25%,6 firm pale-yellow areas in parenchyma | MPVFD, focal chorangiomatosis | NIL | IUFD |
| ELUC+Nuchal+True knot | 1 | AGA | NIL | DVI, FVM | NIL | Term IUFD |

We wondered whether the less placental reserve due to deficient intervillous circulation can affect foetal outcome if cord coiling abnormalities arise additionally.

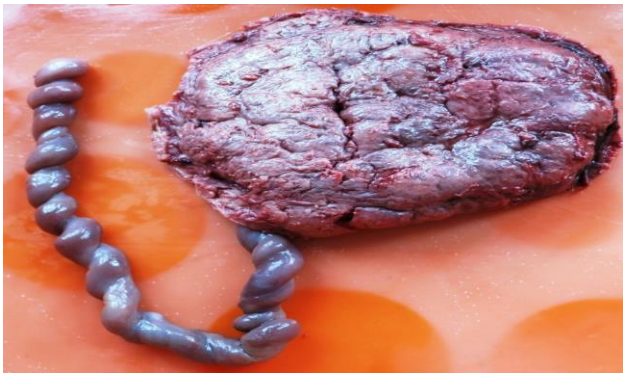

**Figure 1: Hypercoiled umbilical cord.**

Presence of avascular villi, thrombosis of foetal vasculature, VSK and NRBCs reflect foetal blood flow obstruction and hypoxia. In our study 25% of the abnormal coiling cases tended to have FVM suggesting that these lesions would have caused chronic vascular obstruction.[10]

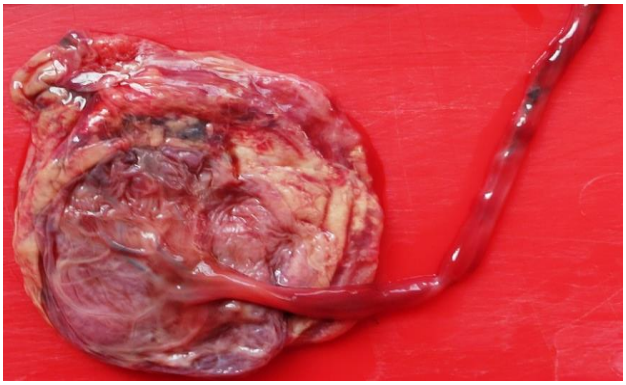

**Figure 2: Hypocoiled and thin umbilical cord.**

**Figure 3: Foetal demise due to excessively long, hypercoiled umbilical cord with multiple strictures (arrow).**

Chorangiosis, multifocal chorangiomatosis are overlapping villous capillary lesions believed to be related to hypoxia. Benirschke observed an association between chorangiosis and cord problems.[13] It is an adaptive mechanism to overcome hypoxia occurring in coiling abnormalities. Our observations were also similar.[14] Some of the cases also showed distal villous immaturity, one other lesion known to be associated with coiling abnormalities.

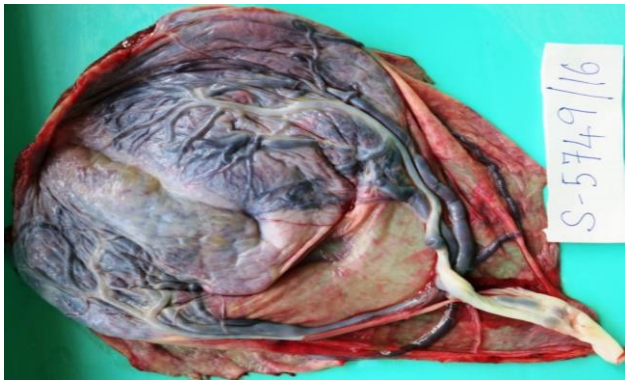


**Figure 4: Velamentous insertion of umbilical cord.**

There was some correlation between inflammatory lesions such as VUE and, acute chorioamnionitis with individual UCA.

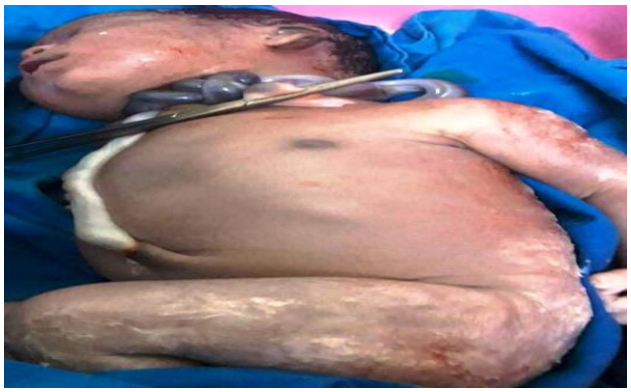

**Figure 5: Nuchal cord with tight true knot encircling the neck causing foetal demise.**

***Cord strictures***

Most investigators considered strictures and hypercoiling to be similar or related entities. In our study we observed a total of 11 cases with cord strictures. Four among the cases were associated with hypercoiling; one each with hypocoiling, velamentous cord insertion and ELUC. 4 cases had isolated stricture. This data suggested that cord stricture is a distinctive entity as agreed upon by Hong Qi Peng et al in their study.[15] We have also noted a high incidence (more than 80%) of foetal demise associated with cord stricture, coiling abnormalities or a

combination of both. Foetal vascular malperfusion was common when strictures and hypercoiling were coexistent. This implies that UC strictures and hypercoiling could decrease the blood flow with possible predisposition to development of thrombosis.

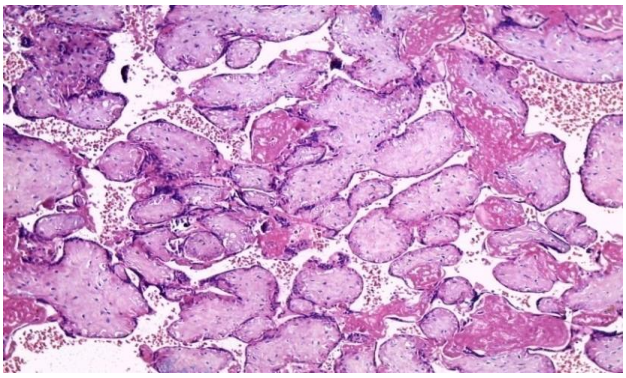

**Figure 6: Foetal vascular malperfusion - groups of avascular hyalinised villi in a case of umbilical cord stricture. (H and E, 10X).**

***Thin cord, SUA***

It has been suggested that diameter of the umbilical cord is determined by the water content of Wharton's jelly. Previous studies have found an association of thin cord with FGR and other poor foetal outcome.[16,17]

A Wharton's jelly reductions have also been recognized as a possible cause of foetal death in the presence of single umbilical artery, which is the most common abnormality of the umbilical cord. These associations were confirmed in our study. According to Heifetz SA, non-malformed SUA infants can have an increased mortality rate, although SUA with associated malformations are the common cause of the high perinatal mortality.[18]

***True knot, ELUC***

Interestingly, both cases of true knot were associated with LGA. The case with a tight TK ended up in IUFD. Common associations like multi gravida and ELUC were also observed in our study. Like earlier studies we also found an association between ELUC and other multiple UCAs.[5] Both cases of ELUC were associated with multiple UCAs such as nuchal cord, TK and stricture and expectantly both resulted in IUFD.

***Abnormal cord insertions***

Marginal and velamentous insertions are suggested to result from disturbances of implantation and considered a marker of placental insufficiency.[19] We found that non-reassuring foetal status, SGA and IUFD were significantly associated with abnormal insertion which may reflect intrauterine hypoxia.

***Multiple UCAs***

Any UCA can sufficiently reduce placental function, when multiple predispose the foetus to placental circulatory stasis lesions and thrombosis, thus leading to vascular obstruction and unfavourable clinical sequelae.[3]

In our study the majority of multiple UCA cases tended to have extensive rather than focal FVM with admixture of avascular hyalinised villi, villous stromal karyorrhexis and nucleated RBCs suggesting that these lesions would have caused prolonged obstruction. Infants with multiple UCAs where FVM is extensive were found to have brain imaging abnormalities and/or abnormal neurological follow-up.

There were some limitations in our data. First, as a retrospective study, the clinical information was available only for the immediate neonatal period and further follow up could not be ascertained. Second, not all placentas and complete length of the UC (that remained attached to the neonate) were available for examination. Finally, different patterns of hypercoiling were not studied.

## CONCLUSION

Gross UCAs, especially when multiple was associated with clinically significant placental findings and adverse perinatal outcome. Therefore, our study reinforces that all placentas with gross UCAs should be submitted for examination with complete (full length of the) umbilical cord. Coiling index with patterns of coiling should be a part of routine examination. A prospective study that also includes the follow up of these neonates could address the clinical effects of UCAs.

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