LONG CORD WITH COILS AND TRUE KNOT

(Case Report with Review of Literature)

by

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Introduction

Cord anomalies (except cord prolapse), as a cause of foetal distress and foetal loss have always had a controversial position in the minds of the obstetricians. Review of the published material in books and journals projects widely ranging opinions from liberally attributing these anomalies as a cause of foetal damage to complete disregard of their role in such mishaps.

Shui and Eastman (1957) reported cases of healthy unaffected new borns with true knots in their cords. Hennesy (1944) in his discussion on management of cord complications, quoted his experiment, where he tried to force fluid with a syringe through the cords of still-born babies having true knots. He was successful in forcing the fluid through all such cords and concluded that knots are an exaggerated cause of foetal distress. Spellacy *et al* (1966) found raised still birth rate in their cases with true knots but found no correlation with neonatal deaths. Peterson (1952), Baden (1955) and Dippel (1964) have reported cases of cord knots with associated death in utero.

Shui and Eastman (1957) found that still-birth and neonatal death rates are not higher in cases with nuchal coils when compared to those without them. They conclude that nuchal coils even when associated with a short cord are rarely a cause of prenatal death. On the other hand, Dippel (1964) and Spellacy (1966) have quoted a higher incidence of meconium staining of liquor, varying between 1.7% to 6% in their cases, which is 2 to 7 times more than in cases without coils. Spellacy (1966) has also reported an increased incidence of second stage distress in such cases.

CASE REPORT

The patient was a booked primipara, aged 27 years with E.D.D. calculated as 11th of September 1978. She was admitted on 8-9-78 at 10.15 PM with history of labour pains for last 12 hours and leaking since 3 PM. The patient's past history had nothing significant to quote and her antenatal booking card showed record of regular antenatal check up, with systemic examination and other parameters within normal limits during each visit. The B.P. was normal throughout without any recorded evidence of oedema or albuminuria.

Pathological investigations recorded a Hb of 9 gm%, blood group 'O', Rh Positive, a non-reactor S.T.S. test and normal routine urine report. The presenting vertex remained free

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Accepted for publication on 16-2-79.

throughout. The pelvis assessed at 36 weeks was recorded as adequate.

On admission the pulse was 90 per minute, B.P. 120/90mm Hg and no oedema. Abdominal examination revealed a full term uterus having mild contractions. Vertex was presenting and was free. F.H.S. was 150 per minute and regular. On vaginal examination cervix was one finger long with intact bag of membranes. There was no leaking and the pelvis was adequate.

After 24 hours on 9-9-78 at 8.30 A.M. the foetal heart was noticed to drop to 100 or below with slight irregularity. Uterine contractions were every 3 to 5 minutes lasting for 40-60 seconds. Cervix was taken up dilated 3 cms with intact bag of membranes. The vertex was still above the brim. An artificial rupture of membranes revealed, liquor laden with thick meconium. Patient was taken up for emergency L.S.C.S. for foetal distress.

Findings on operation: After delivery of the foetal head through the lower segment uterine incision there was some difficulty in delivering the shoulders, when it was noticed that the cord was tightly wound round the chest of the foetus below the arm pits three times and was getting very taught on further pulling the head. Noticing this, one loop of the cord was clamped and cut. A male child weighing 3,600 gms was delivered. The child was asphyxiated with one minute Apgar score of 4/10, 5 minute score of 8/10 and 10 minute score of 10/10. No detectable congenital abnormality was seen. The placenta had central cord attachment and there was a retroplacental haematoma of 3"/2" at one morgin. No abnormal calcification was noticed.

The umbilical cord was 127 cms long, including 25 cm left with the baby. It had 2 umbilical arteries and 1 vein. One true knot of simple variety was seen 37 cm from the placental end.

The postoperative period was uneventful, baby progressed well and both mother and baby were discharged home on 10th day.

Discussion

The interest in the present case was aroused because of the excessively long cord—the longest seen by the authors—associated with tight body coils, a true knot and a retroplacental clot. The patient in question was a booked primipara with normal parameters, developing foetal distress in first stage, which could not be explained except if attributed to the associated tell-tale cord complication.

Cord Length: Napier (1882) reported that cords may vary from 5 cms to 175 cms with an average length of 55 cms and classifies any cord above 70 cms or below 30 cms as abnormal for purposes of description. In the series quoted by Shui and Eastman, cords above 70 cms in length were only 3.8% of the total.

The length of the cord in the present case, including the part left with the baby was 127 cms, while largest cord in the series by Dipple was 120 cms. Shortest cord in his series was 25 cms and was associated with an anencephalic foetus. Hennesy in his discussions quoted one case of still birth due to primary short cord. Developmentally the main gain in cord length is upto 28 weeks of gestation after which it does not appear to lengthen significantly. Why the cord becomes excessively long is not clear, neither has any corelation been established between the placental anomalies and the cord anomalies. Dipple (1964) in his series did not find any corelation between the cord length and maternal age, parity, infant size and foetal position or presentation. Malpas (1964) and Walker (1960) found slightly longer cords in males than in females.

Most of the authors have reported a rise in cord anomalies with increasing cord length. Dippel found the incidence of long cord six times more in cases with cord anomalies than in those without any anomalies. Shui and Eastman have corelated the nuchal coils with the length of the cord; the more the number of coils the higher the average length.

True Knots: These knots can be of simple or figure of eight variety as quoted by Dippel (1964). Wentworth (1965) has quoted an incidence of 0.46% while Spellacy *et al* (1966) have given an incidence of 1.1%.

Number of factors like long cord, excess of liguor, small infants and monoamniotic twins have been held as predisposing to true knot formation by authors like Peterson (1952), Menakar (1956), Walker (1960), Malpas (1964) and Fisher (1964).

In 1875 Chantrenil postulated that true knots were formed between 9th and 12th weeks of gestation because the foetus then is most active and the amount of liguor is relatively large. Atwood (1932) postulates that these are formed during labour. A higher incidence of 0.9% of true knots in aborted foetuses (quoted by Javert 1952), however, supports the supposition that these knots may be formed during early pregnancy. Hyrtl (1970) presumed that the time of formation of knots could be determined by the site of the knot on the cord; thus the closer the knot to the foetus the earlier is it formed. Cord knots 'per se' as a cause of foetal distress have always had a very controversial place.

Coils of the cord—The cord coils are:

(a) *Nuchal coils*—Coils round the neck of the foetus.

(b) *Body coils*—around different parts of the foetus like trunk, leg, arm, foot etc. Both these types of coils can be 'tight coils'—requiring division before delivery of the foetal body and 'loose coils' where there is no need of dividing the cord before delivery.

The quoted incidence of nuchal coils varies between 15.8% to 30%. Shui and Eastman have corelated the incidence with the number of nuchal loops:

One loop round the neck—20.6%	Shu and Eastman (1957) Dippel (1964)
Two loop round the neck— 2.5%	
Three loop round the neck— 0.5%	
Four loop round the neck— 0.1%	

Maximum number of 9 coils has been reported in a case by Mc Caffrey (1927). Increased cord length has been found to be the only causative factor by Spellacy *et al* (1966), while others have associated it with increased liguor, small baby, and multiparity. Differenciating between tight and loose coils Spellacy *et al* found that there was increased proportion of tight coils with shorter cord length and in such cases the cord is likely to be pulled taught round the neck of the foetus.

Harrar and Buchman (1951) found that there was a high incidence of nuchal coils associated with the foetal deaths occurring in 2nd stage of labour.

The incidence of body coils is quoted as 0.5%. Spellacy *et al* report that in this group the effective cord length is severely shortened and rarely may lead to placental separation. In our case the cord was tightly wound thrice round the chest and led to formation of a retroplacental clot.

Foetal distress and cord anomalies

These anomalies of looping, knotting and tortion are probably varying grades of the same general process differing only in degrees from foetal anoxia of different grades to no anoxia at all. Browne (1925) in his 'in vitro' experiments showed that even slackest knot would cause some obstruction to cord blood flow. Clemetson (1953) reported altered oxygen saturation in samples of blood taken from cords forming nuchal coils. Hon has postulated that a slowing of foetal heart rate in these cases is produced by vagotonia and

not by anoxia. Perhaps the tight coils produce more vagotonia.

Spellacy *et al* feel that relative shortening of the cord because of looping could lead to premature separation of placenta due to pulling on the uterine wall. In their cases with cord loops, this complication was thrice more frequent as compared to cases without loops. They found a raised percentage of infants with foetal heart rate less than 100 and significant decrease in one minute Apgar with all the three cord complications. Dippel found 65% greater need for resuscitation in cases with cord complications, and of his sixteen cases developing foetal heart bradycardia, 50 per cent had cord anomalies. The most complex anomalous cord in his series had two true knots and three coils around the neck and was 95 cms long. This foetus died in utero 10 days before the expected date of delivery.

Summay

A case is presented where the tight body coils, leading to effective shortening of the cord and partial premature separation of the placenta, seem to be the most likely cause of foetal distress.

Whether the actual aetiological factor leading to asphyxia is strangulation, decreased blood flow due to taughtness of the cord), vagotonia or abruptio placentae is yet to be decided. It is possible that different factors are responsible in different cases. The possibility of more than one factor acting at one time can not be ruled out.

Clinical stages, from normal unaffected foetal heart to foetal bradycardia, meconium staining or intra uterine death are different grades of the same condition. Their timely detection will affect the management of the case and ultimate foetal outcome. In our case caesarean section had to be performed because distress appeared early in first stage. In the series by Dippel distress developed in 2nd stage and therefore vaginal delivery could be ensured.

Acknowledgement

The Authors are thankful to Mr. B. S. Mohan, Chief Administrator, Narinder Mohan Hospital, GHAZIABAD, for his kind permission to publish this paper.

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Errata

Article 'Pregnancy with Thalassaemia' pages 688-691 Vol. No. XXIX June 1979, inadvertently omitted from contents as well as Index.

Error is regretted.