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COARCTATION OF THE UMBILICAL CORD—A CAUSE OF FOETAL DEATH

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Unexplained intra-uterine foetal death ending in abortion is not uncommon. In the literature very little mention is made of umbilical cord abnormalities as a cause of early foetal death. A reason for this may be that inspection of the cord is neglected or the foetus is too macerated for these conditions to be easily recognized.

A report of a case of coarctation of the umbilical cord is therefore considered to be of value.

CASE REPORT

History

Previous pregnancies. Mrs. I.F., a 32-year-old White, had had 3 previous normal pregnancies. She had no abortions. Her blood was group O, Rh positive, Wassermann-negative.

Present pregnancy. Last menstrual period was in May 1963. Expected date of delivery was March 1964. The general medical history was non-contributory. She was 12-weeks pregnant when first seen.

On examination the patient was obese, weighing 170 lb. She was not anaemic—Hb 90%. General physical examination showed no abnormality. Blood pressure was 150/80 mm.Hg. On abdominal palpation the uterus corresponded to 12 weeks' pregnancy.

Vaginal examination. A degree of genital prolapse was present. The cervix was healthy. Papanicolaou smear was negative for malignancy. The size of the uterus was confirmed. The adenexae were normal.

The patient was seen at 3-weekly intervals. At 16 weeks she was perfectly well. At 19 weeks the size of the uterus corresponded to the period of amenorrhoea. No foetal movement had been felt. Two weeks after the last examination, at 21 weeks, the patient reported that there had been vaginal bleeding and associated painful uterine contractions. She was admitted to hospital. Examination showed that the uterus had not increased in size, remaining at the size of 19 weeks' gestation. Strong uterine contractions were present. On examination with a speculum dark blood was seen issuing from the external cervical os. She aborted shortly afterwards.

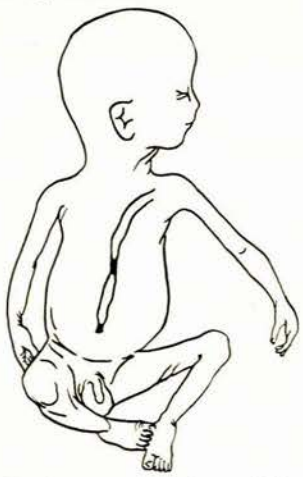

Fig. 1. A macerated foetus of 18 weeks, showing constriction of the cord at its umbilical insertion and a further narrowed area with normal intervening cord.

Examination of Foetus and Placenta

The foetus was macerated and corresponded to the size of 18 weeks' pregnancy, measuring 125 mm. (crown-rump) in length. The placenta appeared to be normal. The umbilical cord was normal in length, measuring 120 mm. On closer inspection 2 constricted areas were noted at the foetal end of the cord (Fig. 1). The first area was at the insertion of the cord into the umbilicus and measured 5 mm. in length. There was 30 mm. of normal cord in between this and the second constricted area which measured 7 mm. The cord appeared to have undergone torsion through 1½ turns at the constricted area. Closer inspection showed that this was not torsion but that the constricted areas appeared to be the result of coarctation of the cord.

Histopathology of the Umbilical Cord

Sections were taken through the normal cord as well as through the constricted areas. The histopathological features showed no abnormality (Fig. 2). Wharton's jelly was normal and the umbilical vein and arteries were of average diameter. The vessel walls were of normal thickness.

Area of cord adjacent to constricted portion. A decreased diameter of the umbilical vein and arteries was noted (Fig. 3). There was commencing thickening of all the vascular walls.

Area of cord at commencement of constricted area. A further decrease in the diameter of the vessels was seen (Fig. 4). There was increasing thickening of the vascular walls.

The constricted area of cord. An area of marked narrowing was noted (Fig. 5). No torsion was seen. There was marked reduction in the diameter of the umbilical vein and arteries, but complete occlusion was absent. There was marked thickening of all the vascular walls and fibrosis in Wharton's jelly.

DISCUSSION

On inspection the pathology appears to be true torsion of the cord. On closer examination, it will be noted that this is not correct and that the narrowing is due to coarctation. It will be clearly seen that there is atresia of the cord in the early weeks of pregnancy and that torsion in these areas is a secondary effect. This is illustrated in the case described above.

It is probable that torsion *per se* is very uncommon during the first trimester, but is secondary to atresia due to a developmental abnormality. In a mobile 18 weeks' foetus with a relatively large volume of liquor amnii, it is unlikely that torsion could have persisted long enough to have given secondary atresia and also to have occurred in two places—the commonest site for this is at the umbilical attachment.

Cord lesions, congenital or acquired, are seldom mentioned as a cause of foetal death and abortion, especially in the pre-viable foetus. Javert and Barton[1] made a study of pregnancies ending in abortion before 22 weeks' gestation with the foetus weighing 500 G or less. They studied 500 cases of abortion and found 133 fit for examination. Thirty-one or 23% were abnormal and in these groups they found 17 cases of torsion *per se*, 7 strictures and 7 true knots.

Physical torsion results from spiralling of the umbilical vessel and the spinning of the foetus may produce additional twisting involving the whole length of the cord, or may be limited to certain segments. Novak[2] has stated that undue twisting of the cord, especially in that portion near the foetus, where Wharton's jelly is not abundant, is a probable cause of foetal death—this being due to asphyxia. Serial sectioning of the cord in its twisted portion has shown obliteration of the vessels. However, he feels that it can never be definitely proved that twisting of the cord does not take place after the death of the foetus. Piraux[3] believes that a developmental narrowing exists and this kills the foetus as its needs increase and that torsion is a secondary effect. He quotes a case of stricture of the cord at the umbilicus without torsion.

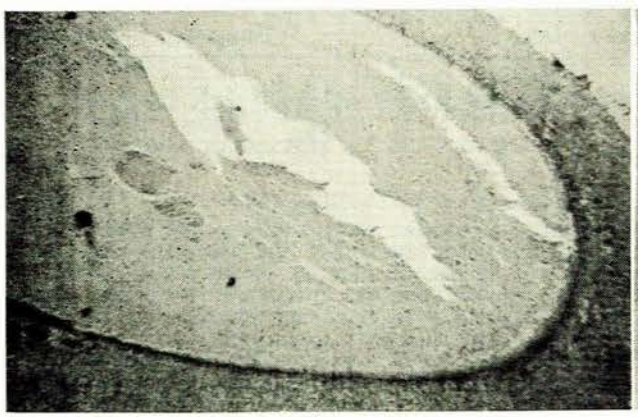

Fig. 2

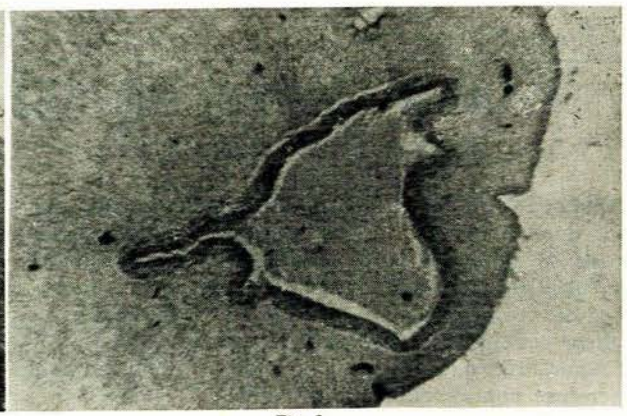

Fig. 3

Fig. 2. Section of normal cord proximal to the constricted area showing the umbilical vein of wide diameter. The vessel wall is not fibrosed or thickened. (H. & E. x 80.)
Fig. 3. Section of cord adjacent to the area of constriction showing decrease in the diameter of the umbilical vein with a definite thickening of the vessel wall. (H. & E. x 80.)

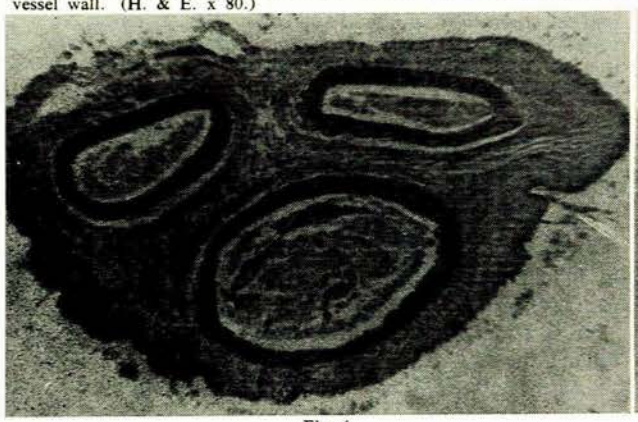

Fig. 4

Fig. 5

Fig. 4. Section of the cord taken at commencement of the constricted area, showing further narrowing of umbilical vein with increasing thickness of vessel walls. There is little difference in the thickening of the walls of the arteries and vein. (H. & E. x 80.)
Fig. 5. Section of cord through constricted area, showing a marked reduction in the diameter of the arteries, without complete occlusion: there is a gross thickening of the vessel walls and fibrosis in Wharton's jelly. (H. & E. x 80.)

Baden[4] disagrees and believes that torsion is the cause of death of the foetus and additional twisting occurs after death. Brunes[5] says there is fibrosis of Wharton's jelly with obliteration of the vessels of the cord and that this is the actual cause of death. Therefore, obliteration of the cord occurs first for some obscure reason and the fibrosis of Wharton's jelly and subsequent torsion is secondary. Wirtinger[6] states that the underlying cause is torsion and this is due to the longitudinal growth of the umbilical arteries along with the increased intravascular pressure caused by the pumping action of the embryonic heart, giving rise to the screw-like contorsions of the cord. Twisting of the cord is often seen by those who milk the cord, and twisting is a normal phenomenon.

CONCLUSIONS

It is true that initial torsion can occur, but the case discussed shows equally that it can follow initial narrowing of the cord—and torsion follows in these narrowed areas after death. This is borne out by the histological findings in the areas of the cord described. The narrowed areas show marked atresia of the vein and arteries with fibrosis of Wharton's jelly, while the intervening cord is perfectly normal. It is unlikely that torsion could have persisted in the mobile foetus of 18 weeks' gestation for long enough for fibrosis and atresia to have occurred.

It is probable that coarctation commences at an early stage of cord development and that the blood flow to and from the foetus will be sufficient for its needs up to a certain stage of development, but when this becomes deficient the foetus dies. A possible explanation of coarctation may be developmental in origin or inflammatory, indicated by fibrosis in Wharton's jelly.

Closer observation of the umbilical cord in cases of abortion should be made to avoid cases of this nature remaining undiagnosed.

SUMMARY

A case of coarctation of the umbilical cord, causing early foetal death, is described. The histopathology in the areas of coarctation and normal umbilical cord is illustrated. The argument as to whether narrowing of the umbilical cord is due to torsion or primarily due to coarctation is discussed. Attention is drawn to the importance of inspection of the umbilical cord in aborted foetuses to avoid abnormalities remaining undiagnosed.

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