What is your diagnosis?

A 28-year-old primigravida visited obstetrics outpatient department at 26 weeks of gestation with complaint of loss of perception of fetal movements for six hours. There was no history of abdominal pain, vaginal leakage or bleeding. There was no history of fall or any blunt trauma to the abdomen. There was no history of diabetes, hypertensive disorders, or any other chronic illness in self or family. The present pregnancy was conceived spontaneously, from a non-consanguineous marriage and the patient was on regular follow up, since the sixth week of pregnancy. First trimester screening results with combined test were suggestive of low risk for trisomy 13, 18 or 21. Nuchal translucency was 0.9 mm, normal for gestational age. Targeted ultrasound scanning for detailed fetal anatomical assessment was normal, with growth of fetus appropriate for gestational age. Fetal echocardiography at 24 weeks was also normal.

On examination, general physical examination was unremarkable. On per-abdominal examination, fundal height corresponded to 22 weeks of gestation; with the fetus in breech presentation and fetal heart sounds were not heard. An urgent ultrasound examination was done, which was suggestive of an intra-uterine fetal demise. After taking an informed consent, induction of labour was done with misoprostol, with dosage appropriate, as per gestation. Patient delivered a male fetus weighing 510 grams. The baby had no external malformations. Placenta and membranes were delivered complete and intact. The fetus was sent for autopsy and placenta for histopathological examination. The karyotype of the baby was normal.

The male fetus corresponded to a gestational age of 24 weeks; the weight was 510 grams. The crown to rump length was 19 cm; the crown to heel length was 28.2 cm; the chest and abdominal circumference were 18.6 cm and 15.7 cm respectively. The biparietal diameter was 9 cm. The distance between the inner and outer canthus was 0.52 cm and 2.9 cm respectively. The fetus had brachycephaly. Palpebral fissures were normal with bilaterally normal eyeballs. There were bilaterally low set ears. The external auditory canal was patent on both sides. The nose was funnel shaped with flat nasal bridge and patent choana. Oral cavity was normal. The fetus had barrel shaped chest with rounded abdomen. The external urethral meatus and anal orifice were normal. Scrotum was empty and there was no midline defect. All four limbs were symmetrical. On internal examination of the fetus, it was found that right lobe of the liver was macerated and there were blood clots in the pleural cavity. Except for these, the internal features were normal for the gestational age. The placenta was discoid shaped weighing around 8.8 g. The maternal surface was normal. The umbilical cord was 19 cm long with marginal insertion on the placenta. The cord had two veins and one artery. The umbilical cord presented a typical stricture of length 5.9 mm and diameter 8 mm at the fetal insertion site (Figure 1). The distal segment to it was dilated to 3.1 cm and congested. This constricted part of the umbilical cord was subjected to histopathological examination.

On histopathological examination of placenta, placental development was found to be appropriate, corresponding to second trimester of pregnancy. Focal dystrophic calcification was present with increased syncytial knot formation. There was no evidence of infarct or any other vascular lesion, and no signs of inflammation. Membranes were unremarkable. The umbilical cord showed three vessels, both in the normal and in the stricture region. At the stricture site, the umbilical cord showed complete loss of Wharton's jelly with areas of fibrosis and neo-vascularization at the periphery (Figure 2).

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Figure 1. Umbilical cord showing stricture at the foetal insertion site

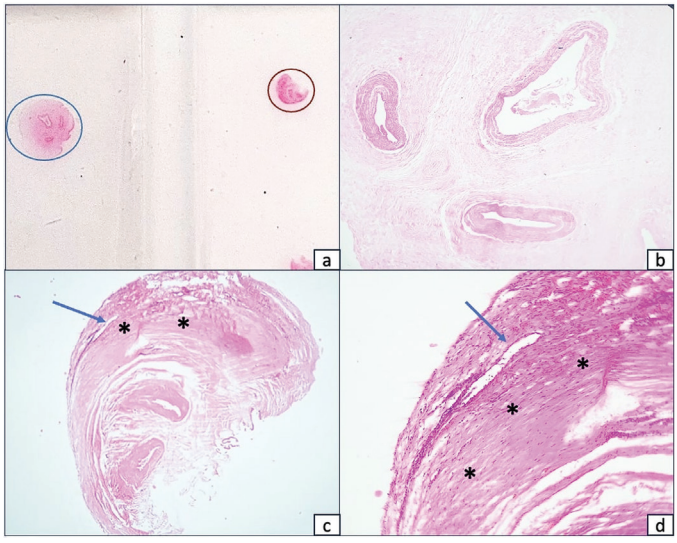

Figure 2. Histopathological findings of the stricture site
(a) Relative comparison of cross-sectional area of the umbilical cord from the normal (circled in blue) and stricture site (circled in red); (b) Microphotograph of the umbilical cord from the normal area showing normal morphology with preserved Wharton's jelly (H&E stain, 40×); (c,d) Microphotograph of the umbilical cord from the stricture site showing loss of Wharton's jelly with areas of fibrosis (asterix) and neovascularisation (arrow) at the periphery (c: H&E stain, 40×; d: H&E stain, 100×)
H&E: Hematoxylin & eosin

Answer

This typical histological finding confirms this is a case of second trimester intrauterine fetal demise as a result of umbilical cord stricture. The histological feature is characteristic, and has been hypothesized due to differentiation of mesenchymal stem cell or as an attempt to form collateral circulation (1).

Stillbirth in the late second trimester remains a significant and distressing event. While many cases can be attributed to well-known factors, such as maternal health issues, placental abnormalities, or fetal genetic conditions, some causes are less apparent and often go undiagnosed. One such under recognized cause is umbilical cord stricture, characterized by a localized narrowing of the umbilical cord, and has been associated with fetal demise by compromising blood flow and nutrient exchange between the mother and fetus (2). In the second or third trimester of pregnancy, clinically, the sole sign is typically a reduction in foetal movements, and foetal death happens shortly after. Until recently, umbilical cord stricture and torsion could only be diagnosed postmortem. This has led to significant discussion over whether or not these findings actually represent causes of death or are simply artefacts from the postmortem process (3). The causes of umbilical cord stricture are the subject of several hypotheses. According to one concept, dubbed the "stretch hypothesis," the length of the cord is determined by tension generated by fetal movements; the greater the length of the umbilical cord, the greater the fetal movements (4). The structure of umbilical cords is helical, with up to 380 turns. An umbilical cord's average length is around 55 cm, and its diameter is 1-2 cm. The first and second trimesters, when the fetus is known to be the most active, are when the majority of the cord's length is reached. The stricture observed in certain umbilical cords has been hypothesized to happen in the second trimester following excessive fetal movements (5). In the present case, the umbilical cord was 19 cm long. The etiopathology of umbilical cord stricture could possibly be a vicious cycle, with narrowing leading to reduced blood flow through the umbilical vessels leading to ischemia and tissue damage, further promoting thrombosis which can further exacerbate narrowing and impede blood flow. Fibrosis and calcification, thereafter contribute to the rigidity and narrowing of the cord. Thrombosis of the chorionic plate vessels often occurs with umbilical cord stricture and over coiling, especially when both are present. This can reduce blood flow, causing hypoxemia and abnormal fetal movements. Fetal death may result if these movements twist the unprotected, stenosed cord section (6). Based on the etiopathology, conditions predisposing are listed in Figure 3.

Another postulated reason for the umbilical cord stricture has been inadequate Wharton's jelly. As chondroitin sulphate and hyaluronic acid are abundant in Wharton jelly, umbilical arteries are shielded from compression. If the Wharton jelly is lost, the fetoplacental circulation may be hampered, which might result in fetal death (7). In the presented case, the umbilical cord from the stricture site showed complete loss of Wharton's jelly

with areas of fibrosis and neovascularisation at the periphery. Blichárová et al. (8), has also reported a similar case where the umbilical cord in stricture area revealed loss of Wharton's jelly, replacement with extensive fibrosis and capillary vessel formation. Peng et al. (6), has documented that Whartons's jelly deficiency was noted in 25% of the cases with umbilical cord stricture.

Previous studies have reported different timings of intrauterine death varying between 21 and 40 weeks of gestation as a consequence of umbilical cord stricture (Table 1) (3,5,9,10). This depends upon when the oxygen need of the fetus is significantly affected. This might differ depending on the number, degree of stricture present and could also change from pregnancy to pregnancy. The growing fetus experiences intrauterine growth limitation, hypoxia, and acidosis as a result of the stricture of the cord and the constriction of the blood vessels inside it, which results in a degree of reduction in oxygen supply and fetal demise.

Though there are several cases of umbilical cord stricture causing intrauterine death, there is a paucity of literature on the possibility of its recurrence. Bakotic et al. (2) reported recurrent umbilical cord over-coiling and stricture resulting in fetal death across three successive pregnancies. In the third pregnancy, the primary stricture site did not exhibit the pronounced coiling seen in the previous two. Instead, the affected cord region showed significant attenuation and fibrosis with only mild coiling. The authors proposed that cord stricture, rather than significant coiling, may have been a more critical factor in the fetus's demise.

Ultrasonographic prediction of umbilical cord stricture is challenging, but certain clues can raise suspicion. Key indicators include localized narrowing of the umbilical cord, often accompanied by reduced or absent Wharton's jelly, which appears as a hypoechoic region surrounding the umbilical vessels. Increased echogenicity in the affected segment may suggest fibrosis. Abnormal cord coiling, particularly hypercoiling or hypocoiling, is also linked to stricture risk. Doppler studies play a crucial role, with elevated resistance in the umbilical artery, intermittent absent or reversed end-diastolic flow, and

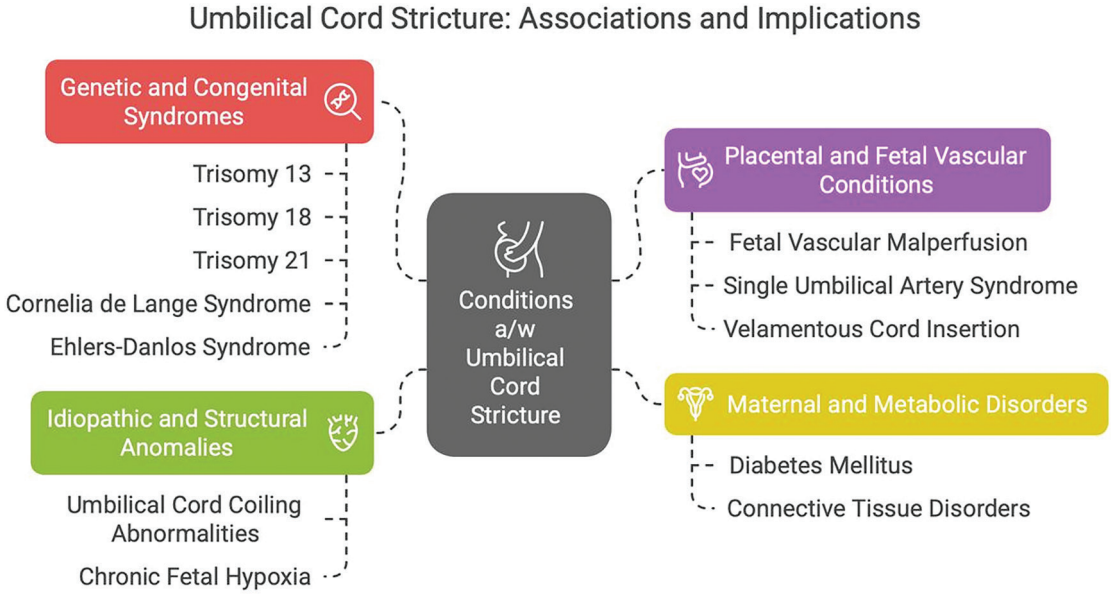

Figure 3. Syndromes and obstetric complications associated with umbilical cord stricture

Table 1. Umbilical cord stricture and intrauterine fetal demis

No	Authors	Timing of IUD	History given by patient	Associated fetal anomaly	Length of umbilical cord
1	Langhe et al. (9)	38 weeks	Absence of fetal movements	No	50.5 cm
2	Ling et al. (5)	22 weeks	Absence of fetal movements	No	Length of the cord not available
3	Chew et al. (10)	33 weeks	Absence of fetal movements	No	37 cm
4	Blichárová et al. (1)	37 weeks	Absence of fetal movements	No	49 cm
5	Present case report	24 weeks		No	19 cm
IUD: Intrauterine device					

Table 2. Guidelines for obstetric ultrasound examination from various medical organizations emphasize different aspects of evaluating the umbilical cord and placental insertion

1	The American Institute of Ultrasound in Medicine (AIUM) (9)	The umbilical cord should be examined to determine the number of vessels and the fetal and placental insertion sites should be assessed during standard 2nd/3rd trimester ultrasound examinations.
2	The Australasian Society for Ultrasound in Medicine (13)	Evaluate carefully, the placental cord insertion, highlighting the identification of marginal and velamentous anomalies. Transvaginal colour or power Doppler scans should be used to rule out vasa praevia.
3	The American College of Radiology (ACR) and the American College of Obstetricians and Gynecologists (ACOG) (14)	Do not advocate routine assessment of placental cord insertion but recommend imaging the umbilical cord and counting the vessels when feasible.
4	The Society of Obstetricians and Gynaecologists of Canada (15)	The placental cord insertion should be evaluated, only in cases of low-lying placenta. It also recommends transvaginal evaluation of the internal cervical os in situations involving placenta praevia, low or velamentous cord insertion, vaginal bleeding, or bilobed/succenturiate placenta.
5	The Royal College of Obstetricians and Gynaecologists (16)	There is insufficient evidence to support routine second-trimester screening for vasa praevia in the general population, despite the high accuracy and low false-positive rate of transvaginal ultrasound scans.
6	The International Society of Ultrasound in Obstetrics and Gynaecology (ISUOG) (17)	Although formal assessment of the umbilical cord insertion is not part of the routine mid-trimester scan, if marginal or velamentous cord insertion is visualized, it should be reported.

turbulent flow patterns being potential warning signs. Additional indicators such as fetal growth restriction, oligohydramnios, or reduced fetal movements may further raise suspicion. While predicting umbilical cord stricture with certainty remains difficult, combining detailed morphological assessment with serial Doppler studies in high-risk pregnancies can improve early detection and enable closer fetal monitoring (11,12).

In subsequent pregnancies following an umbilical cord stricture, careful monitoring and preventive measures are important to reduce risks, as the condition has tendency to recur. Preconception counseling is essential to assess maternal risk factors and review previous pregnancy outcomes. Early and regular prenatal care should include detailed ultrasonographic evaluation, with a focus on umbilical cord morphology, coiling patterns, and Wharton's jelly assessment. Serial growth scans combined with Doppler studies are recommended to monitor umbilical artery blood flow for signs of increased resistance or compromised circulation. Fetal surveillance methods, such as non-stress tests and biophysical profiles, may be initiated in the third trimester to detect fetal distress. Lifestyle modifications, including proper nutrition and smoking cessation, further support maternal and fetal well-being. Delivery timing should be individualized based on fetal growth, Doppler findings, and maternal health, with early delivery considered in high-risk cases. Postpartum evaluation of the placenta and umbilical cord is advised to identify potential risk factors for future pregnancies, and referral to a maternal-fetal medicine specialist may help tailor a comprehensive management plan (11,12).

The guidelines for obstetric ultrasound examination from various medical organizations emphasize different aspects of evaluating the umbilical cord and placental insertion (9,13-17) (Table 2). Other umbilical cord pathologies are generally considered incidental findings and are not specifically screened for. The guidelines do not include the evaluation of free cord loops that might indicate true knots, positional anomalies, structural anomalies, or helical pattern anomalies. These guidelines clearly avoid recommending sonographic measurements of the umbilical cord, Wharton's jelly, or identifying potential abnormal cord morphology (18). They also do not address cord entanglement issues, such as nuchal cords, true knots, or complex entanglements, which are discussed in detail elsewhere. In accordance with a few other authors, we also suggest that the entire length of the umbilical cord available for sonographic assessment should be thoroughly scanned for potential abnormalities in cord morphology. As sonographic resolution continues to improve, diagnostic accuracy will undoubtedly increase (19).

To conclude, the acute and often lethal nature of umbilical cord stricture, along with the rarity of its prenatal sonographic diagnosis, suggests that careful attention should be given to the sonographic appearance of the umbilical cord, particularly the presence of Wharton's jelly near the fetal abdominal wall insertion, during the second and third trimester of pregnancy, when possible. Due to reports of recurrence in subsequent pregnancies, special consideration should be given to detailed sonographic evaluation of Wharton's jelly in this anatomical location for women with previous pregnancy losses, especially in cases attributed to this condition or unexplained.

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