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Intrauterine Fetal Demise Associated with Velamentous Cord Insertion: A Case Report and Review of the Importance of Prenatal Diagnosis

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Authors

Sima Giti[1], Fatemeh Jalali Zand[1], Mohammad Reza Nateghi[1,2*]

1- Sarem Gynecology, Obstetrics and Infertility Research Center, Sarem Women's Hospital, Iran University of Medical Science (IUMS), Tehran, Iran.
2- Sarem Cell Research Center (SCRC), Sarem Women's Hospital, Tehran, Iran.

Corresponding Authors:
Mohammad Reza Nateghi; Sarem Gynecology, Obstetrics and Infertility Research Center, Sarem Women's Hospital, Iran University of Medical Sciences, Tehran, Iran.
Address: Sarem Women Hospital, Basij Square, Phase 3, Ekbatan Town, Tehran, Iran. Postal code: 1396956111, Phone: +98 (21) 44670888, Fax: +98 (21) 44670432.

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ABSTRACT

Introduction: Velamentous cord insertion is an important abnormality of the umbilical cord insertion into the placenta, in which the umbilical vessels, before reaching the placenta, traverse between the fetal membranes without the protection of Wharton's jelly. This condition may be associated with multiple complications, including intrauterine growth restriction (IUGR), preterm delivery, fetomaternal hemorrhage, placental abruption, low Apgar score, hypoxic-ischemic brain injury, and intrauterine fetal demise. Despite the clinical importance of this abnormality, its diagnosis in many cases is delayed until delivery or pathological examination of the placenta.

Case Presentation: A 36-year-old woman, G2P1L1, with a history of previous cesarean section due to fetal macrosomia, gestational diabetes, polycystic ovary syndrome, controlled hypothyroidism, and obesity, was followed from 26 weeks of gestation. Throughout pregnancy, serial ultrasound assessments of fetal growth, amniotic fluid volume evaluation, Doppler studies, and fetal well-being tests including biophysical profile and non-stress test (NST) were performed regularly and reported as normal. At 34 weeks of gestation, twice-weekly fetal surveillance was initiated. Despite normal findings in the latest evaluations, the patient experienced decreased fetal movements three days later but presented to the medical center with a two-day delay. Evaluations revealed intrauterine fetal demise. During cesarean section, completely bloody amniotic fluid and evidence of placental hematoma were observed. Pathological examination of the placenta confirmed velamentous cord insertion, a finding that had not been detected in prenatal ultrasounds.

Conclusion: This case report demonstrates that velamentous cord insertion can be associated with catastrophic outcomes such as intrauterine fetal demise, even in the presence of regular prenatal care and apparently normal fetal assessments. Careful evaluation of the umbilical cord insertion site during the second-trimester anomaly scan and documentation of it in the ultrasound report can play an important role in identifying high-risk cases and planning closer maternal-fetal surveillance. Increasing awareness among obstetricians, perinatologists, and sonographers regarding this abnormality may contribute to reducing severe maternal and fetal complications.

Keywords: Velamentous Cord Insertion; Intrauterine Fetal Demise; Stillbirth; Umbilical Cord Abnormalities; Placental Pathology; Placental Abruption; Case Report.

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Introduction

The umbilical cord, as the only connection between the mother and the fetus, plays a vital role in transferring oxygen, nutrients, hormones, and removing fetal waste products. Any abnormality in the structure, insertion site, or function of the umbilical cord can have significant effects on fetal growth and development [1, 2]. Umbilical cord abnormalities, including true knots, excessive coiling, single umbilical artery, marginal cord insertion, and velamentous cord insertion, are among the disorders known to be associated with increased risks of pregnancy complications and adverse perinatal outcomes. In recent years, special attention has been given to the role of these abnormalities in fetal and neonatal mortality, as a significant proportion of unexplained stillbirths may be related to umbilical cord and placental vascular disorders [3, 4].

Velamentous cord insertion (VCI) is one of the most important abnormalities of cord insertion, in which the umbilical cord, instead of inserting directly into the placental disc, attaches to the fetal membranes. In this condition, the umbilical vessels travel some distance between the amnion and chorion before reaching the placenta without the protection of Wharton's jelly [5]. Wharton's jelly is a protective structure that normally shields the umbilical vessels from pressure, traction, and mechanical injury. The absence of this protective layer in VCI exposes fetal vessels to compression, rupture, thrombosis, and impaired blood flow, thereby seriously threatening fetal health and survival [6].

The prevalence of VCI in singleton pregnancies is reported to be approximately 0.5% to 2%, but it is higher in multiple pregnancies, pregnancies achieved through assisted reproductive techniques, abnormal placentation, placenta previa, and some maternal underlying conditions. Although the exact cause of this abnormality is not yet fully understood, the theory of placental migration or trophotropism is considered one of the most accepted mechanisms [7, 8]. According to this theory, changes in placental implantation site during early pregnancy may lead to relative displacement of the cord insertion site and positioning of vessels within the fetal membranes.

The clinical importance of VCI lies in its association with a wide range of obstetric and perinatal complications. Numerous studies have shown that this abnormality is associated with increased risks of intrauterine growth restriction (IUGR), preterm birth, preeclampsia, placental abruption, fetal distress, low Apgar score, neonatal intensive care unit (NICU) admission, and intrauterine fetal demise. In addition, when velamentous vessels pass near the internal cervical os, vasa previa may occur, which is an obstetric emergency with high fetal mortality. Even in the absence of vasa previa, the vulnerability of unprotected vessels may lead to chronic or acute reduction in fetal blood supply [9-11].

One of the most important consequences of VCI is the increased risk of stillbirth and intrauterine fetal demise (IUFD). Several mechanisms have been proposed for this association, including intermittent compression of umbilical vessels, chronic reduction in placental perfusion, fetal vessel thrombosis, fetomaternal hemorrhage, rupture of velamentous vessels, and increased susceptibility to placental abruption. These mechanisms may lead either gradually or suddenly to severe fetal hypoxia and ultimately intrauterine death. Notably, IUFD may occur in many cases without clear warning signs and even after apparently normal fetal surveillance test results [12-14].

Despite its clinical importance, prenatal diagnosis of this abnormality remains challenging. Currently, second-trimester ultrasound, particularly during the anomaly scan between 18 and 24 weeks of gestation, is considered the most effective method for identifying the umbilical cord insertion site. The use of color Doppler can significantly increase diagnostic sensitivity. However, in many centers, systematic evaluation of cord insertion is not part of the routine anomaly scan protocol, and therefore some cases of VCI remain undiagnosed until delivery or placental pathological examination. This results in the loss of an important opportunity to classify the pregnancy as high risk and to plan closer maternal-fetal monitoring. The present report introduces a case of intrauterine fetal demise in the late third trimester of pregnancy that occurred despite regular antenatal care, serial fetal well-being assessments, and normal results of fetal surveillance tests. Ultimately, it manifested as IUFD, and postpartum placental pathology revealed velamentous cord insertion. This case highlights the importance of careful evaluation of the umbilical cord insertion site during prenatal ultrasound, greater awareness among obstetricians and sonographers regarding the potential complications of VCI, and the role of early diagnosis in preventing adverse fetal outcomes.

Case Presentation

The patient was a 36-year-old woman with a history of one live birth and one intrauterine fetal demise (G2P2L1IUFD1), who presented at 26 weeks of gestation to Sarem Specialized Hospital in Tehran. The reason for referral was gestational diabetes and short cervix. The current pregnancy had occurred spontaneously, and the patient had been under routine obstetric care since early pregnancy. In her obstetric history, she had a previous successful pregnancy in 2017, which was terminated by cesarean section due to fetal macrosomia with a birth weight of 4875 grams. In that pregnancy, the glucose challenge test (GCT) was abnormal; however, the glucose tolerance test (GTT) was within normal range, and no further metabolic follow-up was performed.

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In her medical history, the patient reported polycystic ovary syndrome (PCOS), obesity, and medically controlled hypothyroidism. Surgical history included hysteroscopy prior to her first pregnancy due to an endometrial polyp. The patient had no consanguinity with her spouse. In family history, systemic lupus erythematosus in her sister, breast cancer in her mother, and hypertension in first-degree relatives were reported; however, there was no family history of diabetes.

In the first trimester of the current pregnancy, the patient experienced spotting and threatened miscarriage, which resolved with bed rest and progesterone suppositories. First-trimester screening ultrasound had shown placenta previa. Due to advanced maternal age, non-invasive prenatal testing (NIPT) was performed, which reported a low-risk result. In an external anomaly scan, no structural fetal abnormalities were reported; however, the umbilical cord insertion site was not evaluated or documented.

During pregnancy, the patient was followed by an endocrinologist, and regular monitoring of thyroid function and blood glucose was performed. Due to hyperglycemia, metformin 500 mg twice daily was initiated, and the patient simultaneously received aspirin 160 mg daily. At 24 weeks of gestation, ultrasound performed outside the center reported a cervical length of 27 mm. In addition, due to increased uterine artery resistance, enoxaparin therapy was started. With persistent hyperglycemia, metformin was increased to 500 mg three times daily, and long-acting insulin was added to the treatment regimen.

The first ultrasound evaluation at Sarem Specialized Hospital was performed at 26 weeks of gestation. In this assessment, the only notable finding was cervical shortening, while fetal growth, amniotic fluid volume, and other fetal well-being parameters were reported as normal. Recommendations included increased rest, continuation of vaginal progesterone, strict glycemic control, and fetal movement monitoring. Enoxaparin 4000 IU daily was also initiated.

In a follow-up evaluation one week later, cervical length showed slight improvement, and fetal growth and amniotic fluid volume remained normal; however, uterine artery resistance was still elevated. Therefore, the enoxaparin dose was increased to 6000 IU. At 33 weeks of gestation, cervical length remained unchanged, while Doppler indices of uterine arteries showed improvement compared to previous studies. Serial thyroid and glycemic profile tests continued to show postprandial hyperglycemia (2-hour postprandial glucose). Accordingly, the patient was referred again to the endocrinologist for treatment adjustment.

Given the existing risk factors, from 34 weeks of gestation, a close fetal surveillance program including biophysical profile and non-stress test (NST) twice weekly was initiated. All evaluations up to the last visit were normal, with no evidence of fetal distress, intrauterine growth restriction, or oligohydramnios. The patient was advised to continue strict monitoring of fetal movements, blood glucose, and weight.

Three days after the last normal biophysical profile and NST, the patient noticed decreased fetal movements; however, she presented to the hospital with approximately a two-day delay. At presentation, clinical and ultrasound evaluation revealed absence of fetal cardiac activity, and intrauterine fetal demise (IUFD) was diagnosed.

Given the previous cesarean section, unfavorable cervical status for induction, and the patient's psychological condition, termination of pregnancy via cesarean section was decided. During surgery, the demised fetus was delivered without evidence of meconium staining. Intraoperatively, completely bloody amniotic fluid was observed. In addition, after placental delivery, a retroplacental hematoma involving approximately 10% of the placental surface was noted, suggestive of partial placental abruption.

Due to the inability to perform the Kleihauer–Betke test for fetomaternal hemorrhage evaluation, the placenta was sent for pathological examination. Histopathological evaluation revealed velamentous cord insertion (VCI). This finding had not been reported in any prenatal ultrasound during pregnancy. Considering the presence of VCI, intra-amniotic hemorrhage, and placental abruption, the most likely mechanism of IUFD in this patient was acute fetal circulatory compromise due to vascular complications related to velamentous cord insertion.

Discussion

VCI is a relatively rare umbilical cord abnormality in which the umbilical vessels, before reaching the placental disc, traverse between the fetal membranes without the protection of Wharton's jelly. This anatomical feature renders fetal vessels more vulnerable to compression, traction, rupture, and thrombosis, potentially leading to adverse maternal and fetal outcomes.

Multiple studies have demonstrated that VCI is associated with increased risks of intrauterine growth restriction, preterm birth, abnormal placental blood flow patterns, placental abruption, fetomaternal hemorrhage, low Apgar scores, neonatal intensive care unit admission, and intrauterine fetal demise. Despite these potential consequences, many cases of VCI remain undiagnosed during pregnancy and are only identified after delivery and placental pathological examination.

In the present report, a 36-year-old woman with obesity, PCOS, hypothyroidism, and gestational diabetes was followed from 26 weeks of gestation at Sarem Specialized Hospital. During pregnancy, repeated evaluations including fetal growth ultrasound, amniotic fluid assessment, Doppler studies, biophysical profile, and NST were performed regularly, all yielding reassuring results. However, in

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late third trimester and only a few days after the last normal fetal assessment, decreased fetal movements occurred, eventually leading to IUFD. At cesarean section, completely bloody amniotic fluid and evidence of partial placental abruption were observed, and placental pathology revealed velamentous cord insertion, which had not been detected during antenatal care. These findings suggest a possible role of VCI in acute fetal circulatory compromise and IUFD.

Koorn et al. in 2025 investigated the association between VCI and IUFD in a retrospective case-control study and meta-analysis [1]. They found no significant association between VCI and IUFD in their case-control analysis; however, their meta-analysis demonstrated an increased risk of IUFD associated with VCI. They attributed this discrepancy to diagnostic bias, differences in surveillance methods, and confounding factors. The findings of the present case are consistent with the meta-analysis by Koorn et al. [1], as IUFD occurred in the setting of VCI. However, it contrasts with their case-control results showing no increased risk. This discrepancy may be explained by differences in study design, sample size, the single-case nature of the present report, and lack of prenatal diagnosis of VCI. Moreover, in the present case, intra-amniotic hemorrhage and placental abruption were observed, which may represent a direct mechanism of acute fetal circulatory failure leading to IUFD, whereas such associated factors were not specifically assessed in the study by Koorn et al.

In 2024, a case of term IUFD due to umbilical vessel rupture in the setting of furcate cord insertion was reported by Xu et al. [2]. In this rare anomaly, umbilical vessels separate before reaching the placenta and, due to lack of Wharton's jelly protection, are prone to rupture, thrombosis, and hemorrhage. In that report, rupture of unprotected vessels led to bloody amniotic fluid and subsequent IUFD. The authors emphasized the importance of prenatal diagnosis using ultrasound. The findings of the present study are consistent with the report by Xu et al. [2], as both cases suggest acute vascular events related to abnormal cord insertion as the mechanism of IUFD. However, the anomaly in Xu et al. was furcate cord insertion, whereas in our patient it was VCI. Despite anatomical differences, both conditions share the common feature of unprotected fetal vessels with increased risk of acute circulatory compromise and fetal death.

Li et al. (2023) reported two cases of IUFD in pregnancies complicated by type I vasa previa associated with velamentous cord insertion [3]. In both cases, fetal death occurred after 35 weeks of gestation without prior bleeding. The authors suggested that compression of unprotected fetal vessels by the presenting fetal part was the most likely mechanism of IUFD and emphasized the importance of prenatal diagnosis and consideration of earlier delivery. The present case is consistent with Li et al. [3] in that abnormal cord insertion and unprotected vessels were associated with IUFD, and sudden fetal demise occurred despite apparently normal surveillance. However, in their study vasa previa and vascular compression were the main mechanisms, whereas in our case, bloody amniotic fluid, partial placental abruption, and histopathologically confirmed VCI suggest acute vascular injury and hemorrhage as the dominant mechanism.

Conclusion

VCI is an important abnormality of umbilical cord insertion that may be associated with a wide range of obstetric and perinatal complications, including fetal growth restriction, fetomaternal hemorrhage, placental abruption, and intrauterine fetal demise. The present case demonstrated that even with regular prenatal care, serial ultrasound examinations, fetal growth assessment, Doppler studies, and routine fetal surveillance tests showing apparently normal results, sudden IUFD may still occur.

In this patient, VCI was only diagnosed postnatally through placental histopathology. The presence of completely bloody amniotic fluid and partial placental abruption strengthened the hypothesis of an acute vascular event leading to fetal demise. Review of the literature also indicates that fetal vessels lacking Wharton's jelly protection in conditions such as VCI, vasa previa, and furcate cord insertion are susceptible to compression, rupture, thrombosis, and sudden circulatory failure, which may lead to IUFD even in the absence of warning signs.

Although current evidence does not allow precise quantification of IUFD risk in all VCI cases, there is general agreement on the importance of prenatal detection and closer surveillance of affected pregnancies. Accordingly, evaluation and documentation of the umbilical cord insertion site during the second-trimester anomaly scan, particularly with color Doppler, should be considered part of routine obstetric ultrasound assessment.

Increased awareness among obstetricians, perinatologists, and sonographers regarding the clinical significance of VCI may facilitate early identification of high-risk cases, appropriate fetal monitoring strategies, and ultimately reduction of severe perinatal complications and potentially preventable cases of intrauterine fetal demise.

Ethical Issue

In conducting this research, all ethical principles in medical and biological research were observed in accordance with the Declaration of Helsinki, and the rights, dignity, and confidentiality of the participants were protected.

Conflict of Interests

There was no conflict of interest in this study.

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