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# Vasa previa rupture in velamentous insertion of the umbilical cord: an analysis and report of a case

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**Summary**

Ruptured vasa previa in term pregnancy is rare but usually catastrophic if emergency delivery is not achieved. The authors present a case of ruptured vasa previa in velamentous cord insertion placenta. The fetus survived after intensive treatment immediately after delivery by cesarean section, but, unfortunately, died after the family gave him up. Defects in the vessel wall architecture were visualized and confirmed by histopathologic examination and might be responsible for the vessel rupture. Prenatal sonographic identification of cord insertion site into the placenta is encouraged as standard of practice to prevent this accident.

*Key words:* Vasa previa; Velamentous insertion; Umbilical cord.

## Introduction

The present is an analysis and report of a case of ruptured vasa previa in velamentous cord insertion placenta. A 31-year-old woman, gravida 5 and para 1 (G5P1), at a gestational age of 39 weeks and six days. The placenta was observed with velamentous cord insertion and vasa previa. One blood vessel of the vasa previa had ruptured, showing the broken ends after delivery. The newborn appeared to have no breath and no heartbeat, with pale skin, sluggish limbs, no reflection, and no vital signs. He died after the family gave him up. So ruptured vasa previa in term pregnancy is rare but usually catastrophic if emergent delivery is not achieved.

## Case Report

A 31-year-old woman, gravida 5 and para 1 (G5P1), at a gestational age of 39 weeks and six days, was admitted to the present hospital due to vaginal discharge for over one hour. The patient had no vaginal bleeding and no history of abdominal pain during her pregnancy. An ultrasound examination at 24 weeks of gestation indicated placenta previa in a breech presentation, which had not been followed up through re-examination. Reproductive history: 1-0-3-1 (last pregnancy: vaginal birth of a healthy infant at term). A physical examination upon hospitalization showed a body temperature (T) of 36.7°C, a pulse rate (P) of 92 beats/minute, a respiratory rate of 20 breaths/minute, and a blood pressure (BP) of 118/80 mmHg. The patient was conscious and calm and was in normal general condition, without cardiovascular or pulmonary abnormalities. The abdomen was soft and non-tender, without rebound tenderness or organomegaly. No shifting dullness (-) was detected, and no edema (-) was observed in either extremity. The abdomen was distended and consistent with pregnancy. Occasional uterine contractions were observed. One fetus was in the right occiput anterior (ROA) position. Fetal heart rate was 136 bpm. The abdominal circumference was 100 cm, and the symphysio-fundal height was 35 cm. Estimated fetal weight was 3,500 grams. A gentle pelvic examination with genital disinfection indicated a cervical dilation of one-cm, in a -3 position; the fetal membrane ruptured, the amniotic fluid was clear, and no vaginal bleeding was observed. Diagnosis upon admission: G5P1, 39 weeks and six days gestation, ROA position, premature rupture of fetal membrane, and suspected placenta previa.

The patient had abrupt vaginal bleeding during fetal monitoring, amounting to approximately 150 ml (mixed with amniotic fluid) and bright red in color. The fetal monitoring indicated a rapid drop in fetal heart rate to approximately 55 bpm (see fetal heart monitoring trace, Figure 1), and the fetal heartbeat subsequently became undetectable. After continuously repeated examination of the fetal heart rate, it was detected to be 50 bpm at 7:00. The patient immediately underwent a lower uterine segment cesarean section under local and epidural anesthesia due to "antepartum hemorrhage (suspected vasa previa rupture and placenta previa) and fetal distress". Intraoperative observation indicated clear amniotic fluid amounting to approximately 300 ml. At 7:15, a male infant was delivered in an left occiput transverse (LOT) position. The infant weighed 3,325 g with an Apgar score of 0-2-4. The umbilical cord was 50 cm long and pale in color. No nuchal cord was observed. The placenta was located in the posterior uterine wall and naturally peeled off intact. The placenta was observed with velamentous cord insertion and vasa previa. One blood vessel of the vasa previa had ruptured, showing broken ends (Figure 2).

The newborn appeared to not be breathing, with no heartbeat, pale skin, sluggish limbs, no reflection, and no vital signs. Oropharyngeal cleaning was administered immediately, and two ml of mucus was aspirated. An endotracheal tube was inserted, with pressurized oxygen administered through a balloon. Chest compressions were conducted. Following the injection of one ml of 1:10,000 epinephrine and 30 ml of normal saline through the umbilical vein, the heart rate gradually increased to 80 bpm. The skin of the body trunk turned slightly red. Pressurized oxygen was administered con-

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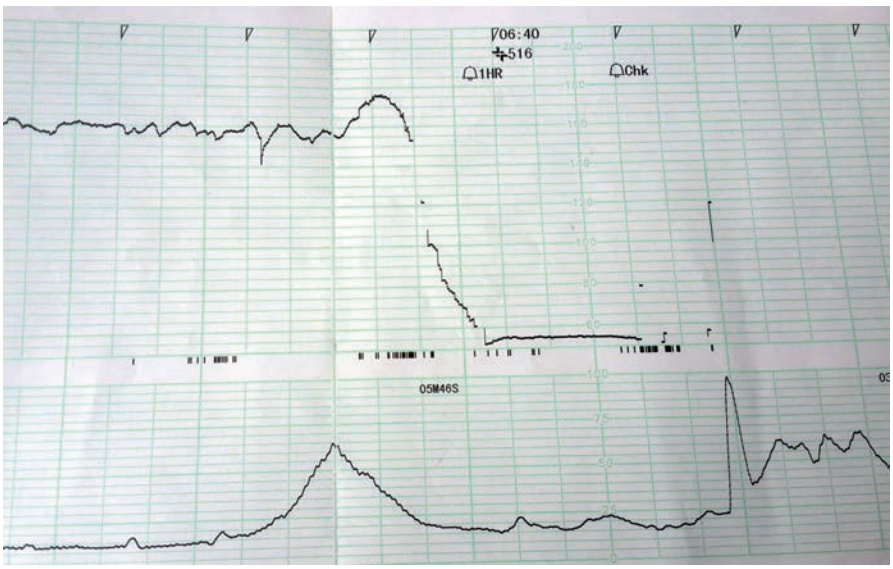


Figure 1. — Fetal monitoring indicated a rapid drop in fetal heart rate to approximately 55 bpm from 155 bpm.

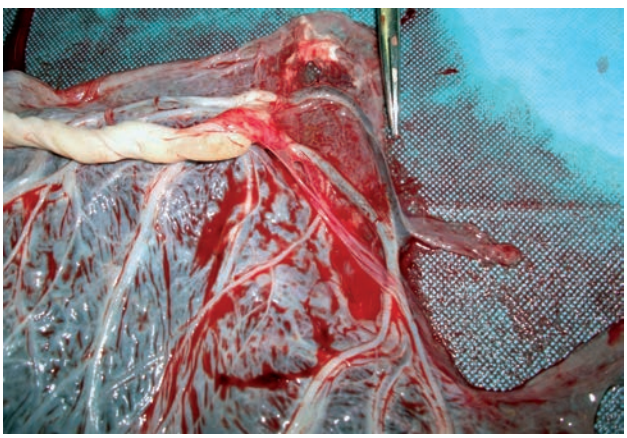

Figure 2. — One blood vessel of the vasa previa has ruptured, showing the broken ends.

tinuously, and the heart rate increased to 120 bpm, with weak heart sounds. Shallow breathing was observed 20 minutes after birth, at a rate of ten bpm. The Apgar score was then 0-2-4-7. After a venous cannulation was performed, an intravenous injection of naloxone hydrochloride, cefotaxime sodium, penicillin, vitamin K1, etamsylate, and normal saline was administered. Pressurized oxygenation was continued through endotracheal intubation to maintain arterial hemoglobin oxygen saturation (SapO2) of 85-90%. Spontaneous breathing reached 30-40 breaths/minute at 45 minutes after birth, along with a heart rate of 120 beats/minute and SapO2 at 93%. One hour later, the patient and her family decided to give up the treatment of the newborn due to financial reasons and the newborn died. Routine blood test results from the newborn showed white blood cell count at $7.83\times10^9$/L, red blood cell count at $2.63\times10^{12}$/L, hemoglobin at 98 g/L, hematocrit at 35.6%, platelet count at $128\times10^9$/L, percentage of neutrophils at 51.9%, percentage of lymphocytes at 41.8%, CRP below 1 mg/L, an O blood type of Rh D+, and blood glucose at 2.6 mmol/L (using a glucose meter).

Placental pathological report (2013-07428): (1) Placenta with velamentous cord insertion in late gestation with one umbilical cord and three blood vessels (two arteries and one vein). The rupture of a vein traveling within the fetal membrane, accompanied by hemorrhage, was observed five cm from the site where the blood vessels in the umbilical cord connected to the placenta. (2) No abnormal development of the placental villi was observed. A small number of villi showed mild aging, with slightly narrowed intervillous spaces, mild increase in fibrinous exudates, and focal hemorrhage in the placental floor. (3) Focal hemorrhage was observed in the fetal membrane, accompanied by mild edema.

## Discussion

### *Cause of vasa previa rupture*

Placenta with velamentous cord insertion refers to the condition in which the umbilical cord attaches to the fetal membrane, and the umbilical vessels insert into the fetal membrane without a protective covering. The umbilical vessels travel between the amniotic and chorionic membranes before reaching the placenta. This condition is also called velamentous umbilical cord insertion. The fetal membrane vessels that lack the support of the umbilical cord or placenta and run through the lower uterine segment or across the internal orifice of the uterus in front of the fetal presentation are called vasa previa [1]. Although the cause of vasa previa remains unclear, most researchers believe the hypothesis proposed by Benirseake to be the most reasonable. In this hypothesis, the umbilical cord is normally attached to the placenta initially; however, during the subsequent placental developmental process, the umbilical cord is left behind due to the lateral growth of the chorionic villi, leading to malnutrition of the attached segment of the umbilical cord. As a result, the atrophic chorionic villus becomes smooth chorion, and velamentous umbilical cord insertion occurs, followed by the secondary formation

of the vasa previa [2, 3]. This patient was a multipara with a history of three instances of induced abortion and thus was very likely to have "endometrial barrenness" caused by endometrial infection and transformation. Consequently, malnutrition at the point where the umbilical cord attached could cause a lack of peripheral protection from the Wharton's jelly of the blood vessels attached to the fetal membrane, leading to intrapartum rupture of the vessels during labor.

*Incidence rate of vasa previa*

Vasa previa rupture is very rare in clinical settings. However, it is life threatening to the fetus once it occurs because the rupture of blood vessels leads to acute blood loss and hypoxia, resulting in fetal suffocation and death within a short period of time [4]. Under normal circumstances, in approximately 90% of pregnancies, the umbilical cord attaches to the middle portion or slightly deviated from the middle on the fetal side of the placenta, while in approximately 10% of cases, the umbilical cord attaches to the edge of the placenta. Velamentous umbilical cord insertion occurs in only 1% of pregnancies [5]. The incidence rate of vasa previa rupture is even lower, only approximately 0.02% according to domestic reports. It has been reported that fetal distress occurred in 46.1% of vasa previa rupture cases, and the mortality rate was 75-100% [6, 7].

*Characteristics of vasa previa rupture*

(A) Vasa previa rupture and the resulting death usually occur in late gestation or in the intrapartum period, related to the changes in intrauterine pressure. (B) The rupture of blood vessels is often accompanied by the rupture of the fetal membrane. (C) The vaginal bleeding is painless, with a continuous fresh blood stream. (D) The rupture of the vasa previa is followed by fetal heart rate changes. Continuous fetal heart rate monitoring can detect fetal abnormalities in a timely manner. (E) The laboratory tests can distinguish between maternal and fetal blood through vaginal blood smears, blood type testing, and the Apt–Downey test (APT). The case in this report was a typical example of natural intrapartum rupture of the fetal membrane along with vaginal bleeding, amniotic fluid flow, and a sudden drop in the fetal heart rate from 155 beats/min to 55 beats/min (Figure 1) in a burst of contractions.

*Impact on perinatal infants*

Once vasa previa rupture is diagnosed, cesarean section should be performed immediately to terminate the gestation if there is no condition for vaginal operative delivery. The rupture of the vasa previa is clinically rare. Once it occurs, it threatens fetal life because the rupture of the blood vessels causes acute fetal ischemia and hypoxia that lead to perinatal sudden death. The amount of vaginal bleeding is a direct factor affecting the prognosis of the fetus because the lost blood comes directly from the fetus. The full-term fetal blood volume is approximately 240-300 ml. When acute blood loss reaches one-third of the fetal blood volume, the body loses its full compensatory capability and regulatory mechanisms, resulting in hemorrhagic shock and death within a short period of time [8]. Therefore, the umbilical cord should be compressed to push as much blood to the fetus as possible before severing the umbilical cord, or 20-30 ml of placental blood should be injected into the newborn through the umbilical vein after severing the umbilical cord to reduce the series of neonatal complications caused by anemia. The newborn in this report did not receive timely blood transfusion to correct anemia after birth, and the family gave up the rescue because of financial reasons, leading to neonatal death, from which lessons should be learned.

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