Vasa Previa: An Unusual Cause Of Fetal Distress

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Vasa previa is an unusual but potentially treatable cause of fetal distress that can cause extreme fetal morbidity and mortality. An illustrative case of vasa previa is presented, followed by discussions of approaches to incidence, diagnosis, treatment, and prognosis.

Case Report
A 24-year-old primigravida was first seen for obstetric care at 13 weeks' gestation. Initially the pregnancy progressed without apparent difficulty, but the fundal height was found to be somewhat small for dates at 38 weeks' gestation. A sonogram obtained at that time showed a 33-week-sized fetus, with all measurements being concordant and no sonographic evidence of intrauterine growth retardation. The biophysical profile was 8/8 (normal movement, tone, respirations, and qualitative amniotic fluid volume), and the placenta was grade I. A repeat ultrasonic scan 3 weeks later, on 12 June 1989, showed a 35.7-week-sized fetus, with measurements again concordant; the placenta was now grade IV and mature, and the biophysical profile was still 8/8.

The patient was hospitalized for induction on 18 June 1989, at 41 weeks' gestation by dates. Examination showed a vertex presentation with the cervix at fingertip dilatation, anterior, soft, and long, and the fetal head was ballotable. Using a catheter, 0.5 mg of prostaglandin E2 gel was instilled in the cervix. Shortly thereafter, deep variable fetal heart decelerations were noted over a period of 2 to 3 minutes with minimal contractions (Figure 1). Because of the decelerations, the patient was monitored overnight, but no further difficulties were noted. The following day she received oxytocin. There was minimal cervical change over 8 hours, but the fetal heart tracing had been stable. The patient slept through the night.

On 20 June 1989 oxytocin was restarted. The cervix was unchanged. At 1400 hours there was spontaneous rupture of the membranes with clear fluid. An acceptable labor pattern developed. At 1700 hours, however, episodes of deep fetal bradycardia down to 90 beats per minute developed (Figure 2). At this time dilatation was 2 cm. The oxytocin was discontinued, terbutaline was given subcutaneously, and an immediate Cesarean section was performed. A full-term female infant, with Apgars of 8 and 9 and weighing 2315 g (5 lb, 6 oz) was delivered. The placenta and vessels showed membranous insertion and vasa previa (Figure 3). Both mother and baby have done well.

Discussion
For vasa previa to occur, there first must be a velamentous insertion of the umbilical cord, in which there is attachment of the vessels into the fetal membranes between the amnion and the chorion.1 The incidence of velamentous insertion is 0.24 to 1.8 percent in singletons, and it is six times higher in multiple gestations.1 In vasa previa these unsupported vessels then cross below the presenting fetal part in the lower uterine segment.

In the current literature, the incidence of vasa previa is variously reported as between 1 in 2000 and 1 in 5000 deliveries,1-6 but this condition is also thought to be underreported,7-9 perhaps so much so that its incidence is unknown.2,10

From a historical perspective, in 1801, Lobstein recognized the relation of velamentous cord insertion to hemorrhage and fetal death, but the
The striking aspect of vasa previa is its associated fetal mortality, even with current obstetric advances. When bleeding is present, mortality ranges from 60 to 100 percent. With cord compression or distress, fetal mortality ranges from 50 to 72 percent. In 1987 Gianopoulos, et al. compared fetal wastage with intact versus ruptured membranes and found a 50 to 60 percent mortality associated with the intact membranes and 75 to 100 percent mortality associated with ruptured membranes.

Prospective diagnosis of this uncommon problem is difficult, partly because it occurs so rarely. In addition, the rapidity of fetal compromise and subsequent death, once the signs of vasa previa become manifest, makes prenatal diagnosis unlikely before intervention is necessary.

Occasionally the vessels can be palpated or seen through the cervix. Real-time ultrasonic scanning can be useful, but only if the vessels are large enough to be seen. Magnetic resonance imaging (MRI) has the potential to become increasingly valuable, as it allows direct visualization of even small vessels, but it is expensive and not yet cost effective. If there is bleeding, determining that the blood is of fetal origin will confirm the diagnosis, but fetal blood identification is often done too late or is not done routinely at most centers.

Management, once the diagnosis is made, is directed toward fetal survival, and the most expeditious method of delivery is used, usually Cesarean section.

Our case is somewhat atypical in several respects. Repeated level II sonograms did not show the vasa previa, the manifestations of the problem were somewhat slow in developing, and in spite of spontaneous rupture of the membranes, the outcome was good.

In 1980 Kouyoumdjian wrote, “one must therefore conclude that in spite of the advances in perinatal care, the prognosis for the fetus in vasa previa will remain very poor for the foreseeable future.” There is hope for improvement with the development of higher resolution sonograms and by using selected MRI studies. A higher priority in differential diagnosis for this complication also should improve outcome. Vasa previa should be considered in the differential diagnosis of fetal distress, especially when the distress is associated with vaginal bleeding or if vaginal bleeding occurs during the third trimester or during labor. Detection of vasa previa should then be “an indication for immediate Cesarean section with the same priority as cord prolapse.” Such aggressive intervention should bring a decline in the appalling fetal wastage.

References


