

Doppler studies in preterm prelabor amniorrhexis

Based on Doppler in Obstetrics: by K Nicolaides, G Rizzo, K Hecher

PATHOPHYSIOLOGY

Preterm birth is the leading cause of perinatal death and handicap. About half of preterm births are associated with preterm prelabor amniorrhexis (rupture of membranes; PPRM) and, in a high proportion of such cases, the underlying cause may be ascending infection from the lower genital tract. Thus, positive amniotic fluid cultures, with organisms commonly found in the vagina, are present in about one-third of cases with PPRMs and in one-third of these there is fetal bacteremia.

In a study of pregnancies with PPRM, the diagnosis of intrauterine infection was based on the results of culture of amniotic fluid and fetal blood obtained by amniocentesis and cordocentesis, respectively.¹ In patients with fetal bacteremia, there was spontaneous delivery within 5 days of amniorrhexis, whereas, in those with negative fetal blood and amniotic fluid cultures, the interval between amniorrhexis and delivery was prolonged by up to 5 months and subsequent cultures of blood obtained from the umbilical cord at delivery or from the neonates were negative. These findings suggest that: first, infection is one of the causes rather than the consequence of amniorrhexis and second, in PPRM, infection may be the cause of subsequent preterm labor and delivery. The likely mechanism for the link between infection and labor is infection-mediated release of cytokines which stimulate the production of prostaglandins that induce uterine contractions.

In pregnancies complicated by PPRM there are essentially two causes of perinatal death: prematurity and pulmonary hypoplasia. In cases with intrauterine infection, delivery occurs within a few days and therefore survival depends on the gestation at amniorrhexis. In patients with no infection, pregnancy may be prolonged by several weeks and, in these cases, there is a risk of postnatal death due to pulmonary hypoplasia.² The risk of death is inversely related to the gestation at amniorrhexis and decreases from approximately 50% for amniorrhexis at <20 weeks, to 20% for amniorrhexis at 20-24 weeks and to <5% for amniorrhexis >24 weeks. Consequently, in the management of pregnancies complicated by amniorrhexis, the major issue is prediction of intrauterine infection and pulmonary hypoplasia.

Fetal blood gases

Cordocentesis in pregnancies with preterm prelabor amniorrhexis has demonstrated that the mean umbilical venous blood pO₂ and pH are not significantly different from the appropriate normal mean for gestation, and there are no significant differences between those with positive or negative fetal blood and amniotic fluid cultures.³ These findings suggest that, in the presence of intrauterine infection, fetal oxygenation is not impaired.

DOPPLER STUDIES

Prediction of intrauterine infection

The rationale for the use of Doppler in pregnancies with PPRM is that infection of the amniotic

fluid and choriodecidua causes constriction of the umbilical cord and chorionic vessels and may consequently impair fetal perfusion. However, Doppler studies immediately before cordocentesis and amniocentesis for bacteriological studies in pregnancies with PPROM demonstrated that the mean pulsatility indices in the uterine arteries, umbilical arteries and fetal middle cerebral arteries were not significantly different from the appropriate normal mean for gestation and there were no significant differences in these values between those with and without intrauterine infection.³

These findings suggest that chorioamnionitis is not associated with a major degree of vasoconstriction in the uteroplacental or fetoplacental circulation. Consequently, Doppler does not provide a clinically useful distinction between infected and non-infected cases. However, Doppler studies in pregnancies with suspected amniorrhexis may be useful in the differential diagnosis from oligohydramnios due to uteroplacental insufficiency and fetal growth restriction. In the latter, there is an increase in PT in the uterine and / or umbilical arteries with decreased PI in the fetal cerebral vessels.

Prediction of pulmonary hypoplasia

Attempts at antenatal prediction of pulmonary hypoplasia in pregnancies with PPROM have focused on ultrasonographic assessment of lung size, amniotic fluid volume and fetal breathing movements. Studies examining fetal thoracic circumference and lung size have reported favorable results in the prediction of pulmonary hypoplasia, whereas studies that attempted to quantify the degree of oligohydramnios or fetal breathing movements have generally reported poor prediction.⁴⁻⁷ Prediction of pulmonary hypoplasia has also been attempted by antenatal Doppler studies. Blood flow in the ductus arteriosus, as assessed by Doppler ultrasound, is altered by breathing movements; in a study of 12 cases of PPROM and severe oligohydramnios, the alteration in ductal blood flow by breathing movements was normal in seven cases with normal lungs, and reduced in all five cases with pulmonary hypoplasia.⁸ Rizzo et al. measured the PI in the peripheral pulmonary arteries (Figure 1) in 20 pregnancies complicated by amniorrhexis at <24 weeks' gestation and reported that in fetuses that subsequently developed pulmonary hypoplasia, the PI was increased from as early as 2 weeks after amniorrhexis.⁹

CONCLUSIONS

In preterm prelabor amniorrhexis:

- There is a high risk of preterm birth, due to intrauterine infection, as well as neonatal death, due to pulmonary hypoplasia.
- Intrauterine infection is not associated with altered fetal oxygenation or a major degree of vasoconstriction in the uteroplacental or fetoplacental circulation.
- Doppler assessment does not provide a clinically useful distinction between infected and non-infected cases.
- Doppler assessment may be useful in the prediction of pulmonary hypoplasia.

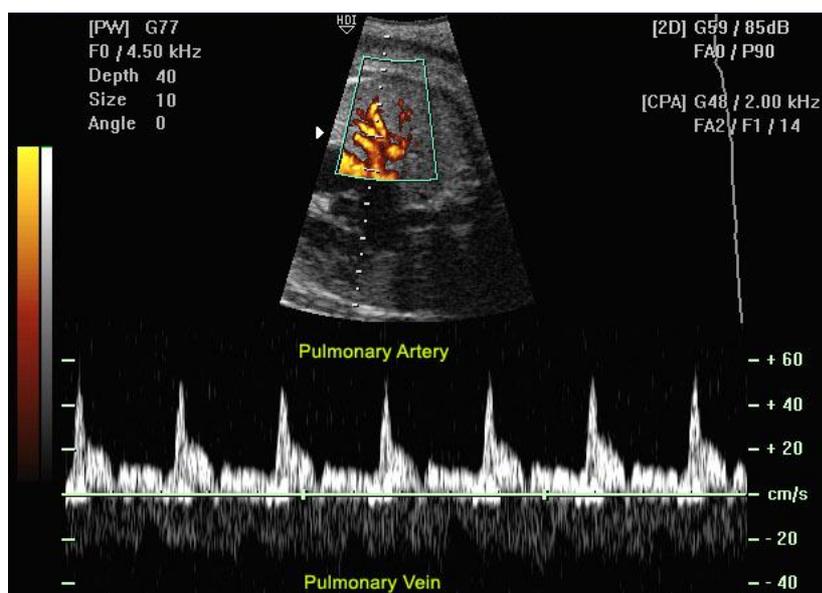


Figure 1: Flow velocity waveforms (left) from the fetal peripheral pulmonary artery at 28 weeks' gestation in a normal pregnancy.

REFERENCES

1. Carroll SG, Ville Y, Greenough A, Gamsu H, Patel B, Philpott-Howard J, Nicolaides KH. Preterm prelabour amniorrhesis: intrauterine infection and interval between membrane rupture and delivery. *Arch Dis Child* 1995;72:F43-6.
2. Carroll SG, Blott M, Nicolaides KH. Preterm prelabor amniorrhesis: outcome of livebirths. *Obstet Gynecol* 1995;86:18-25.
3. Carroll SG, Papaioannou S, Nicolaides KH. Doppler studies of the placental and fetal circulation in pregnancies with preterm prelabour amniorrhesis. *Ultrasound Obstet Gynecol* 1995;5:184-8.
4. Carroll SG, Papaioannou S, Nicolaides KH. Assessment of fetal activity and amniotic fluid volume in the prediction of intrauterine infection in preterm prelabor amniorrhesis. *Am J Obstet Gynecol* 1995;172:1427-35.
5. Blott M, Greenough A, Nicolaides KH, Campbell S. The ultrasonographic assessment of the fetal thorax and fetal breathing movements in the prediction of pulmonary hypoplasia. *Early Hum Dev* 1990;21:143-51.
6. Roberts AB, Mitchell JM. Direct ultrasonographic measurement of fetal lung length in normal pregnancies and pregnancies complicated by prolonged rupture of membranes. *Am J Obstet Gynecol* 1990;163:1560-6.
7. Rotschild A, Ling EW, Puterman ML, Farquharson D. Neonatal outcome after prolonged preterm rupture of the membranes. *Am J Obstet Gynecol* 1990;162:46-52.
8. Van Eyck J, van der Mooren K, Wladimiroff JW. Ductus arteriosus flow velocity modulation by fetal breathing movements as a measure of fetal lung development. *Am J Obstet Gynecol* 1990;163: 558-66.
9. Rizzo G, Capponi A., Angelini E, Mazzoleni A, Romanini C. Blood flow velocity waveforms from fetal peripheral pulmonary arteries in pregnancies with preterm premature rupture of membranes: relationship with pulmonary hypoplasia. *Ultrasound Obstet Gynecol* 2000;15:98-103.