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LETTERS TO THE EDITOR

Hematoma of the umbilical cord secondary to cordocentesis for intrauterine fetal transfusion

We have read with great interest the article by Keckstein *et al.* (1990), which describes three cases of umbilical cord hematoma after cordocentesis for fetal transfusion *in utero*.

We observed recently a similar case. The mother had developed anti-C and anti-E antibodies during her first normal pregnancy, and subsequently had two pregnancies complicated by the intrauterine death of normally formed hydropic fetuses. In the present pregnancy, six intravascular fetal blood transfusions were given. Cordocenteses were uneventful, except at 32 weeks' gestation, when, at the end of the procedure, fetal heart rate decelerations to 80 bpm were observed for 2 min. The umbilical cord at the placental insertion became hyperechogenic. However, the post-transfusion umbilical vein blood gases ($pO_2 = 30$ mmHg, $pCO_2 = 41{\cdot}4$, $pH = 7{\cdot}36$) were within the normal range for gestation.

On account of premature labour and breech position, the patient was delivered by elective cesarean section at 34 weeks of a normal male infant weighing 2200 g. Pathologic examination of the cord revealed a 4 cm organized fusiform hematoma near the placental insertion. Serial histologic sections of

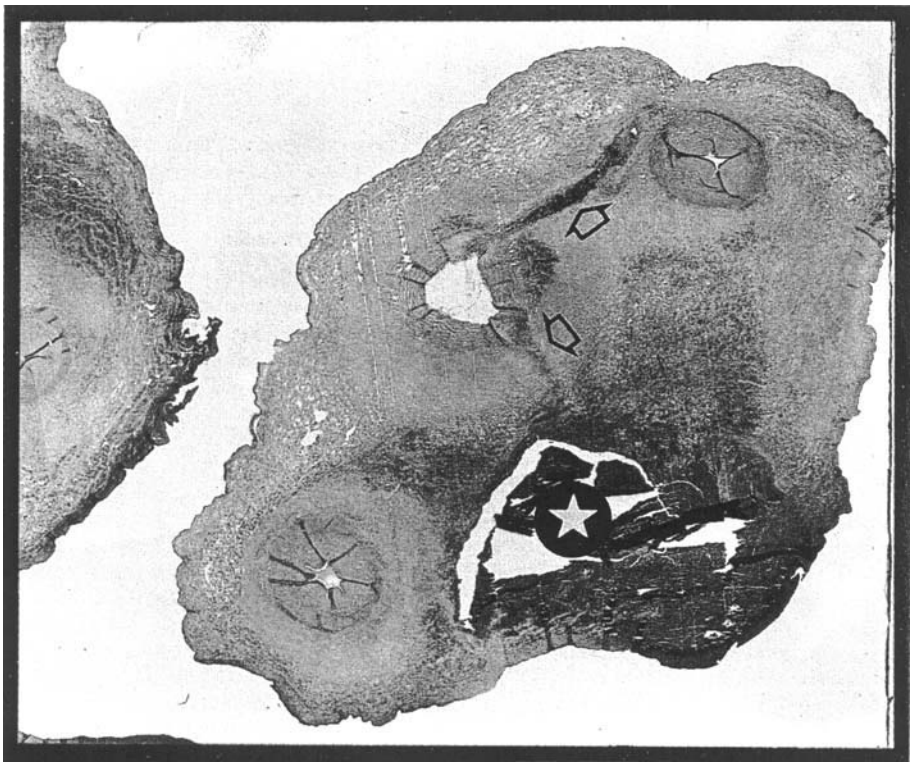

Figure 1. Histologic section of the umbilical cord at the level of the lesion showing two needle tracks (arrows) entering the vein and extended extravasation of blood (★). Serial sections failed to demonstrate vascular compression (H & E × 10)

the cord (every 5 μ) confirmed the presence of blood in the Wharton's jelly, together with traces of the needle entry through the vessel's wall. The puncture sites contained fibrinoid material, but there was no evidence of thrombus formation in the vessel's lumen or of vascular compression by the hematoma (Figure 1).

Theoretically, the potential cord lesions due to the procedure of cordocentesis are vascular thrombosis, hematoma formation and laceration, or rupture, of the cord vessels.

Thrombosis of an umbilical vessel after cordocentesis has never been documented. Pathologic examination of the site of needle insertion in 50 cases of cordocentesis failed to demonstrate intravascular thrombus formation (Jauniaux *et al.*, 1989).

Laceration or rupture of an umbilical vessel may result in intra-amniotic bleeding or in the formation of a hematoma (Jauniaux *et al.*, 1989). Increased intravascular pressure, which may be present in the recipient twin of a twin–twin syndrome (Jauniaux *et al.*, 1989), or direct blood injection in the Wharton's jelly during fetal transfusions, as in the present case, may also result in an hematoma and consequent tamponade of the umbilical cord vessels. The end result of a cord hematoma varies from complete occlusion of the cord vessels, with inevitable fetal death, to varying degrees of fetal distress, which are either acute or chronic. This accident should be suspected when fetal bradycardia and/or an hyperechogenic cord area develop during cordocentesis (Moise *et al.*, 1987; Keckstein *et al.*, 1990).

Pathologic examination of the cord may be suggested in all complicated cases so that the consequences of this new procedure are fully documented. However, compression of an umbilical vessel by a developing hematoma is difficult to demonstrate. Occlusion of the vascular lumen can be related to delivery events rather than to blood extravasation in the Wharton's jelly. In the present case, despite fetal bradycardia and sonographic and macroscopic demonstration of a large hematoma, serial histologic sections of the entire length of the lesion failed to demonstrate vascular occlusion or compression.

Our impression is that other factors, such as a temporary vasospasm suggested by Moise *et al.* (1987), should be considered. The use of Doppler in these cases, together with subsequent histologic correlation, might help to elucidate the physiopathology of fetal distress in complicated cordocentesis.

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In utero resolution of a fetal cystic hygroma in a male with a 46,XY karyotype and normal development at one year

The presence of a fetal cystic hygroma coli diagnosed *in utero* by antenatal ultrasound has been associated with a poor prognosis (Abramowicz *et al.*, 1989), particularly if fetal hydrops is also involved (Chervenak *et al.*, 1983). The differential diagnosis for this malformation is approached first by careful ultrasound evaluation to detect potential associated anomalies (Cowchock *et al.*, 1982; Pearce *et al.*, 1985), and then by fetal chromosome study (through amniocentesis or fetal blood sampling) to rule out possible aneuploidy. The most common chromosome abnormalities seen with this malformation are: monosomy X, trisomy 21, and trisomy 18 (Abramowicz *et al.*, 1989; Pijers *et al.*, 1988). Isolated cystic hygroma not associated with the back of the neck is usually a benign condition with a good prognosis after surgery (Benacerraf *et al.*, 1987). The findings of a cystic hygroma coli diagnosed *in utero* can be associated with Noonan's syndrome and may regress during