Case report

A prenatal diagnosis of umbilical cord oedema made by ultrasound; a case report


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Introduction

Marked oedema of the umbilical cord is an uncommon finding, generally diagnosed only at delivery.

In our review of the literature we did not find any cases in which a diagnosis was made by ultrasound. Oedema of the cord has been described associated with congenital abnormalities. However, we report a case of marked oedema of the umbilical cord in which no other congenital abnormalities were found. The diagnosis was made by ultrasound.

Case report

A 27-yr-old woman, gravida II, bloodgroup A Rh pos, was admitted to our hospital at 33 wk gestational age. At 12 wk amenorrhoea a cervical encerclage using mersilene tape was performed because of cervical incompetence during the first pregnancy. She presented with premature uterine contractions and vaginal blood loss.

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A vaginal examination revealed that the cervix was 2 cm dilated and the mersilene tape had cut through the cervix. Tocolysis was established with fenoterol infusions. Ultrasound examination showed a normal fetus; the biparietal diameter and fetal trunk area were in accordance with gestational age. The amount of amniotic fluid was normal and the placenta was situated in the upper anterior segment, showing no abnormalities. A large transsonic structure was seen with its origin at the fetal abdomen (Fig. 1). It contained three vessels and did not contain any fetal abdominal organs. A defect of the fetal abdominal wall could not be detected. Therefore a diagnosis of umbilical cord oedema was made.

Amniocentesis was performed, which showed an L/S ratio of more than 3/1, lecithin and total phospholipids measuring 63 and 176 µmol/l, respectively. The cervical suture was removed, fenoterol infusions were stopped and a spontaneous delivery took place the same day.

A male infant weighing 2760 g, with Apgar scores of 4 at 1 min, 6 at 3 min and 9 at 5 min, was delivered.

The placenta appeared slightly oedematous with no apparent infarctions and weighed 960 g. Histological examination of the placenta, however, showed no signs of oedema. There was a well-vascularized stroma which contained some intra-inter-
villous fibrin deposits at several points. A few signs of recent infarction and bleeding were seen. Over a distance of 30 cm the umbilical cord showed a marked oedema starting about 16 cm from the placental insertion, regaining its normal appearance 3 cm from the fetal abdomen (Fig. 2), with at cross-section a measurement of 6 cm. It contained two arteries and one vein. Histological examination showed a highly oedematous Wharton jelly. There was no thrombosis of the umbilical arteries or vein.

Physical examination of the baby revealed no congenital malformations. The fetal abdomen showed no herniation or exomphalos, and no oedema of the skin was apparent. Cardiac, renal and respiratory systems were normal.

Discussion

Bender et al. (1978) in their review of the literature found umbilical cord oedema in 10% of all deliveries. It was associated with placental abruption, diabetes mellitus, intra-uterine fetal death, Rhesus incompatibility, premature delivery, respiratory distress syndrome and Caesarean section. They did not find a significant relationship with fetal distress, neonatal asphyxia or maternal hypertension or oedema.

Konstantinova (1977) described an association with anencephaly as well as hydrocephaly, while Howorka and Kapczyński (1971) reported an association with polyhydramnios. Chantler et al. (1969) reported a case associated with a patent urachus and fused umbilical arteries.

Coulter et al. (1975) in their study explained the production of oedema by a low oncotic pressure with a raised hydrostatic pressure in the placenta and the umbilical cord, and an increase in total fluid content of the feto-placental unit. If the cord
oedema reflects similar changes in the lungs, it may predispose the infant to develop respiratory distress syndrome. However, this study seems like an explanation for general fetal hydrops, which Coulter does not report. Perhaps the cord is more sensitive to oncotic or hydrostatic changes.

The possibility of excessive Wharton jelly without evidence of oedema has also been observed (Scott and Wilkinson, 1978).

An ultrasound diagnosis of umbilical cord oedema without evidence of gross fetal abnormalities has to our knowledge not been discussed before, perhaps because the ultrasound features of the cord are not considered to be of importance in routine gestational ultrasound examination (Yiu Chiu and Chiu, 1981). It has been suggested that more information about the cord can be obtained by using a contact sector scanner in addition to the usual linear array scanner (Morin and Winsberg, 1978). By using pulsed Doppler ultrasound it is possible to measure the umbilical flow (Stuart et al, 1980).

It is not known to what extent the cord oedema acts as a causal agent in premature delivery or is the result of it. In our patient the oedema was clearly preexistent, but it cannot be assumed to have had a proven labour-inducing effect because of the coexistence of cervical incompetence.

This case shows that a highly oedematous umbilical cord may exist without additional fetal pathology.

References


