Large Placental Chorioangioma as a Cause of Congestive Heart Failure in Newborn Infants

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SUMMARY. Two rare cases of infants born from pregnancies complicated by large placental chorioangiomas are reported. Congestive heart failure occurred early in the neonatal period as the main complication. Chorioangiomas may be diagnosed early in pregnancy by ultrasound examination. Since both maternal and neonatal complications may indicate premature termination of the pregnancy or be conducive to premature birth, repeated ultrasound examinations, including fetal echocardiography and flow measurements, are suggested to determine the optimal time of delivery. Possible pathophysiological mechanisms causing neonatal complications are discussed.

KEY WORDS: Chorioangioma — Congestive heart failure — Fetus — Newborn infant — Polyhydramnion

Placental tumors are quite common, although rarely diagnosed. When small, these tumors rarely cause problems for the clinical outcome of the pregnancy or delivery, or for the infant. The incidence of chorioangioma, the most common placental tumor, has been reported to be about 1 in 100 placentas examined microscopically [2, 12], whereas that of large chorioangiomas causing clinical symptoms is 1:3500 to 1:16000 [3, 12]. These symptoms are reported to be serious both for the mother [2, 4, 10] and newborn infant [2, 10, 11] and can even lead to death of the fetus [9].

Case Reports

Measurements of fetal circulation were carried out by the method described by Eik-Nes et al. [6], involving both arterial and venous blood flow in the umbilical cord and flow in the descending aorta. Neonatal circulation was studied with a HP 77020AC real-time 5 MHz Doppler ultrasound scanner. Neonatal cardiac performance was monitored, including pulmonary and aortic blood flow, valve regurgitation, blood shunting through the foramen ovale and arterial duct, and cerebral circulation. Cardiac output was measured according to Alverson [1].

Case 1

A hydropic female infant was born by cesarean section under epidural anesthesia at a gestational age of 32 weeks, 3 days after spontaneous rupture of the fetal membranes and developing polyhydramnion. She was marked swollen, weighed 2610 g; Apgar scores were 5 and 6 at 1 and 5 min of age, respectively. She was immediately intubated and ventilated and transferred to the newborn intensive care unit. She developed no respiratory problems and was extubated during the first day. A systolic murmur, tachycardia, and signs of cardiac failure were noted. The first x-ray showed an enlarged heart and fluid in both the pleural cavity and lung parenchyma. The ECG had prominent P waves, a sign of atrial hypertrophy. No structural anomalies of the heart were found on echocardiography, but tricuspic regurgitation was observed. After 36 h of digitalis and furosemide treatment she weighed 640 g less than at birth and her cardiac failure had greatly improved. The infant was observed to have hemolytic disease without blood group incompatibility, and this was treated by exchange transfusion. The child was discharged from the hospital at 15 days in good condition, but continued digitalis treatment for 1.5 months. Since then she has managed well and developed normally.

Case 2

The infant was 4300 g girl, born at a gestational age of 36 weeks, 3 days. The pregnancy was terminated by cesarean section be-
cause of rapidly developing fetal tachycardia. She was cyanotic and limp at birth; her Apgar scores were 6 and 7 at 1 and 5 min of age, respectively. She was immediately intubated and ventilated and admitted to the neonatal intensive care unit. A coarse holosystolic murmur was heard from the beginning, and the liver was palpated 4 cm below the coastal margin. Digitalization and diuretics were started. An x-ray showed enlargement of the heart, and ultrasound examination demonstrated a markedly hypertrophic atrium and ventricles with impaired myocardial contractility (Figs. 1 and 2). The ductus arteriosus was found to be open, and the tricuspid valve leaking. Pathological circulation in the brain was noted (Fig. 3). Arterial oxygen saturation remained low even at maximal respiratory settings. The infant developed tachycardia (pulse level 240/min) at the age of 6 h, and cardiac failure ensued. No improvement in cardiac function was observed in response to inotropic treatment (dobutamine and dopamine), and the infant died at the age of 26 h. She appeared at autopsy to have been full-term and mature. There were no dysmorphic features. The skin was conspicuously cyanotic, the heart was hypertrophic, and the right atrium dilated. The tricuspid valve was hyperplastic with knotty leaflets. The lungs were atelectatic. The liver showed chronic congestion, and the spleen was large and congested. Bilateral double ureters were also found.

Prenatal and Postnatal Echocardiographic Findings

Case 1

The first ultrasound examination was performed in the 31st gestational week by a gynecologist because of developing polyhydramnion. It revealed polyhydramnion and a large, coarse, solid hemangioma-like placental tumor, about 10 cm in diameter. The ultrasound examination also aroused suspicions of a fetal cardiac anomaly, which was excluded postnatally.

Case 2

The first ultrasound examination in the 17th gestational week showed the fetus to be normal, but a central hemangioma was observed in the placenta (Fig. 4). By the 22nd week the hemangioma was estimated to be 8 cm in diameter and considerable polyhydramnion was observed, for which the patient was hospitalized. The α-fetoprotein value was clearly pathological (1175 μg/L). The situation remained stable during gestational weeks 24–29 and the polyhydramnion subsided, so that on the 31st week the amniotic fluid volume was normal and fetal growth good. Another ultrasound examination 2 weeks later (33 gestational weeks) revealed a macrosomic fetus with a reasonable good blood flow in the aorta with an identical pulsation rate to that in the fetal arteries was observed inside the hemangioma. No further ultrasound examinations were performed, and delivery occurred 4 weeks later. The postnatal findings in the two cases are summarized in Table 1.

Discussion

The pathogenesis and histology of hemangioma of the placenta has been previously reviewed by Dunn [5]. A majority of opinion would incline toward its being a hamartomatous malformation.

The fetal and maternal complications of a large placental chorioangioma have been explained as developing via a number of possible pathophysiological mechanisms. The large vascular tumor can act as a physiological and functional dead space and thus cause fetal distress [2, 11]. This is supported by the demonstration in our cases of an arterial flow inside the hemangioma at the fetal pulse rate. The hemangiomas probably also caused a purely mechanical obstruction of flow in the umbilical cord and placenta due to their large size and central origin. This obstruction, in addition to the classical