FETOMATERNAL HEMORRHAGE CAUSED BY AN INTRAPLACENTAL CHORIOCARCINOMA

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Intraplacental choriocarcinoma is rare, and occasionally results in massive feto-maternal hemorrhage. We describe a case of an intraplacental choriocarcinoma diagnosed postpartum after a preterm cesarean delivery of a severely anemic newborn. The microscopic examination showed that clusters of malignant trophoblasts arose from residual normal chorionic villi and infiltrated into the intervillous spaces, confirmed as intraplacental choriocarcinoma. Fetomaternal hemorrhage is a rare complication of choriocarcinoma but its presence should always warrant detailed examination of placenta, mother, and infant.

Keywords: Fetomaternal hemorrhage; Fetal distress; Intraplacental choriocarcinoma

CASE REPORT

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Gestational trophoblastic disease encompasses a spectrum of interrelated tumors, including the hydatidiform moles, either partial or complete, to the more malignant forms, the invasive mole, the placental site and epithelioid trophoblastic tumors and choriocarcinoma [1]. Intraplacental choriocarcinoma, defined as choriocarcinoma in the placenta, is a rare variant of gestational choriocarcinoma accounting for no more than approximately 0.04% of gestational trophoblastic disease [2]. Older than 35 years or very young age of mother can be risk factors of gestational trophoblastic disease [3]. As fetomaternal hemorrhage can cause fetal distress and death, the possibility of intraplacental choriocarcinoma should be considered in these cases [4]. In this report, we present a case of an incidental intraplacental choriocarcinoma that was discovered when the placenta was examined in order to identify the cause of a newborn’s anemia.

Case Report

A 27-year-old woman, gravida 1, was referred to our institution for evaluation of fetal cardiomegaly and ascites at 31+4 weeks’ gestation. Her past history was uneventful, and the results of antenatal laboratory tests and examinations were normal. The mother presented with decreased fetal movement from around 31 weeks of gestation. A transabdominal ultrasound scan with 3.5-5 MHz transducers (Accuvix XQ, Medison, Seoul, Korea) revealed a severely dilatated heart, skin edema, pleural effusion and ascites, suggesting fetal hydrops. Cardiotocography was severely pathologic, showing a repetitive late deceleration and decreased variability (Fig. 1). Emergency cesarean delivery was performed because of fetal hydrops and fetal distress. At delivery, the newborn baby weighed 2,020 g and had Apgar scores of 3 at 1 minute and 5 at 5 minutes. The baby was immediately intubated due to weak respiratory movement and was mechanically ventilated in the neonatal intensive care unit. He was markedly anemic and edematous. Initial chest X-ray showed a total white out pattern on the both lung fields. After admission, the baby’s condition deteriorated...
progressively, with decreased arterial saturation. Sufficient oxygenation was not achieved on surfactant therapy and high-frequency oscillatory ventilation. Nitric oxide (NO) inhalation was applied because of continued hypoxemia. On laboratory tests, the hemoglobin concentration in the umbilical artery was 2.9 g/dL. He was treated with blood transfusion. Echocardiography showed severe distention of the right side of the heart, tricuspid valve regurgitation and patent ductus arteriosus with bidirectional shunt in accordance with persistent pulmonary hypertension of the newborn. Ultrasonographic scan of the brain showed cerebral infarction on left parietal lobe. At 4 days of age, he developed increasing respiratory distress and hypoxemia. In spite of the ventilator therapy with NO inhalation, blood transfusion and inotropic agents, he expired. Therefore fetomaternal hemorrhage was suspected. To clarify the mechanism of this fetomaternal hemorrhage, the placenta was examined, the pathological diagnosis of intraplacental choriocarcinoma was made. The mother was promptly examined because of the risk of metastasis. A detailed physical examination was unremarkable. Chest X-ray, computed tomography (CT) scan of brain, chest, abdomen and pelvis revealed no metastatic lesion. Serial serum concentrations of human chorionic gonadotropin (hCG) were measured, for the once a week until normalization of serum hCG level. On the 18th day postpartum the hCG was 195 IU/L dropping to 2.54 IU/L at 58 days postpartum. Since there were no signs of dissemination, the mother was not treated with chemotherapy.

1. Pathological findings

The fresh placenta weighed 691.4 g in weight and 22.6 × 17.2 × 2.8 cm in dimensions. There were several foci of hemorrhage and early thrombus (about 10% of total disk volume) and infarction (about 3%). Adjacent to a hemorrhagic focus that was at the central portion of the disk showed an ill-defined brown to gray colored friable lesion, measuring about 2 × 1 cm in size (Fig. 2). Microscopically, the placental disk was immature; decreased number of terminal villi and increased composition of intermediate villi with increased stroma. There were several histologic findings that suggest feto-maternal hemorrhage; segmental infarct (arrow, A), intervillus hemorrhage (arrowhead, A) and increased number of nucleated red blood cells (RBCs) (white arrow, B) within intervillus he-

**Fig. 1.** Cardiotocographic monitoring before delivery shows repetitive late decelerations and decreased variability.

**Fig. 2.** The cut surface of placental disk is pale but not hydropic. Under the cord insertion site, an ill-defined brown to gray lesion is present (white arrow) and the background area is hemorrhagic.