Prenatal Sonographic Appearance of Hemorrhagic Cerebellar Infarction

Angela C. Ranzini, MD, Susan Shen-Schwarz, MD, Edwin R. Guzman, MD, Allan J. Fisher, MD, Mary White, RDMS, Anthony M. Vintzileos, MD

To date, the prenatal diagnosis of cerebellar hemorrhage has been limited to isolated case reports, which have demonstrated either a hyperechoic cerebellar hemisphere or a hyperechoic mass within the cerebellum in near-term fetuses. We demonstrate the ultrasonographic findings of intracerebellar hemorrhagic infarction in a fetus at approximately 21 weeks' gestation. In contrast to previous case reports, the hemorrhagic infarcts seen in our case were hypoechoic.

CASE REPORT

An obese 23 year old gravida 4 para 3003 woman with an unknown last menstrual period was referred for routine sonographic evaluation of fetal anatomy and consultation for hypertension, which had been diagnosed on her initial prenatal visit 1 week previously. On ultrasonography, the fetus was found to be symmetrically grown. Biparietal diameter was 5.0 cm, consistent with a 21 week 1 day gestation; head circumference was 19.7 cm, consistent with a 21 week 6 day gestation; femur length was 3.7 cm, consistent with a 21 week 5 day gestation; and abdominal circumference was 16.6 cm, consistent with a 21 week 4 day gestation. The posterior fossa of the fetal brain appeared abnormal, and the cisterna magna was absent. In addition to an absent cerebellar vermis, the cerebellar hemispheres were unequal in size and unusually hypoechoic, with poorly defined borders. The right cerebellar hemisphere also appeared to contain an area with internal echoes that was relatively hyperechoic in comparison to those of the left side (Fig. 1). The remainder of the fetal survey and the other brain structures, including the lateral, third, and fourth ventricles, anterior portion of the frontal lobes, and head size, were unremarkable.

One week after ultrasonographic evaluation (22 weeks’ gestation by ultrasonographic dates), the patient complained of right upper quadrant pain and headaches. She was diagnosed as having severe preeclampsia complicated by hemolysis, elevated liver function tests, low platelet counts (HELLP syndrome). Prostaglandin induction of labor for maternal indications was performed, and she was delivered of a stillborn infant weighing 507 g. The patient’s postpartum recovery was uneventful.
Fetal autopsy revealed ischemic encephalopathy with findings including a subacute hemorrhagic infarct with hemosiderin-laden macrophages and gliosis replacing the left cerebellum, cerebellar vermis; and a portion of the right cerebellum; old bilateral posterior fossa hematomas adherent to the cerebellar hemispheres; subependymal germinal matrix hemorrhages in the right cerebral hemisphere, which were both recent and old; and ischemic neuronal necrosis of the pontine nuclei and hippocampus. External features suggested the fetus was of 24 to 26 weeks’ gestation. The placenta showed decidual vasculopathy, segmental villous fibrosis, and marginal hemorrhages, both recent and old. The findings were suggestive of severe chronic uteroplacental fetal insufficiency, most likely related to maternal preeclampsia. The brain findings were indicative of a process that occurred 1 to 3 weeks prior to the fetal death.

DISCUSSION

Four major types of intracerebral hemorrhages have been described in the neonate: subdural, primary subarachnoid, intracerebellar, and intraventricular. Each type has a different pathophysiology. Subdural hemorrhages occur more frequently in term infants, whereas the other types occur more frequently in preterm infants. Intracerebellar hemorrhage is uncommon in the neonate, being observed more commonly in the preterm period. When it occurs, the neurologic outcome is usually poor. The pathogenesis of intracerebellar hemorrhage is unclear; however, the pediatric literature implicates both trauma and hypoxic events.

To date, we have located only three reported cases of antenatally diagnosed intracerebellar hemorrhages. The earliest case was diagnosed at 31 weeks’ gestation and the other two at 35 weeks and 36 weeks of gestation. The latter two cases were diagnosed after complaints of decreased fetal movement led to ultrasonographic evaluation. Sonographic findings in these three previously reported cases consisted of hyperechoic cerebellar hemispheres in two fetuses and a hyperechoic mass in the cerebellum in one fetus. Ventriculomegaly was found in all cases. Two of the three cases resulted in neonatal death, and in the third neurodevelopmental delay was evident at 6 months of age. The differential diagnosis of abnormal posterior fossa structures includes Dandy-Walker malformation, cerebellar tumors, and intracranial hemorrhage.

Our case differs from the previously reported cases in that the cerebellar hemispheres appeared hypoechoic, irregular, and unequal in size and echogenicity; in addition, a vermis could not be identified. Hemorrhagic sonographic features have previously been shown to change over time, initially appearing hyperechoic and later becoming hypoechoic. It is likely that the hypoechoicinity seen in our case is due to the long duration of the hemorrhage, which was confirmed at necropsy.

Only rarely is the cause of intracranial hemorrhage identified. Antenatal causes that have been implicated include preeclampsia, alloimmune thrombocytopenia, pancreatitis, maternal seizures, and blood clotting abnormalities, including factor V and factor X deficiencies. In previous reports preeclampsia has been associated with intracranial (intraparenchymal, subdural, and intraventricular) hemorrhage in four cases, which resulted in fetal death at 26 and 27 weeks’ gestation and termination of pregnancy in two cases.

To date, no cause has been proposed for any prenatally diagnosed case of intracerebellar hemorrhage. In our case, it is possible that the early-onset preeclampsia which caused intrauterine growth delay and placental pathology also was responsible for the intracerebellar, posterior fossa, and germinal matrix hemorrhages; however, the etiologic mechanism remains unknown. As the fetus was symmetrically grown and the patient’s LMP was uncertain, the fetal growth disturbance was not identified by a single ultrasonographic examination.
In summary, intracerebellar hemorrhage can be identified in some fetuses in the second trimester. Although previously reported cases of cerebellar hemorrhage have had a hyperechoic appearance on sonograms, cerebellar hemorrhage also may appear hypoechoic if it has been of long duration. Unequal, hypoechoic or irregular cerebellar hemispheres with an absent cerebellar vermis and an obliterated cisterna magna may suggest intracerebellar and posterior fossa hemorrhage.

REFERENCES