MRI 'Promising' in Evaluating Fetal, Intracranial Lesions, But Challenges Remain

The technology is costly and its availability is limited; false-positive and false-negative diagnoses also at issue.

BY JERRY INGRAM
Contributing Writer

ORLANDO — MRI for evaluating fetal and intracranial lesions shows promise, but challenges remain, Asad U. Sheikh, M.D., said at the annual meeting of the American Institute of Ultrasound in Medicine.

"MRI has become more useful for evaluating fetal and intracranial lesions. We're starting to see application as the technology advances for abdominal lesions as well. But still, we have considerable difficulty with interpretation," explained Dr. Sheikh, director of the division of maternal-fetal medicine at the University of South Alabama.

MRI should be considered to further delineate intracranial anomalies to better prepare families and care providers for postnatal expectations.

Dr. Sheikh reported on two specific cases of schizencephaly, a brain lesion characterized by abnormal choronal migration.

The first case was of a 20-year-old woman at 19 weeks' gestation who presented with maternal serum alpha fetoprotein (AFP) elevated by 4.4 multiples of the median (MOM). Initial ultrasound evaluation indicated a left-sided intracranial cyst, Dr. Sheikh said. Investigators found that tests for karyotype and infection proved negative.

When they performed another sonogram, they found a large cystic structure replacing the left frontal temporal region. After the 35th week of gestation, they performed MRI, which showed bilateral schizencephaly. The patient delivered vaginally at term.

The second case was a 27-year-old woman at 21 weeks' gestation with an elevated AFP of 3.6 MOM; she was in her fourth pregnancy. Ultrasound revealed bilateral ventriculomegaly.

Amniocentesis was declined, and infection studies were negative. The physicians performed fetal MRI at 24 weeks' gestation and found left-sided schizencephaly and agenesis of the corpus callosum. The image quality was poor, due to fetal movement, however. The patient delivered vaginally at term.

Dr. Sheikh and his team performed MRIs to confirm their earlier diagnoses.

In the first case, they confirmed the findings of bilateral schizencephaly.

For the second patient, they performed cranial computed tomography, which illustrated bilateral schizencephaly with left more involved than right. Additionally, they observed agenesis of the corpus callosum.

Dr. Sheikh notes that these cases are unique in terms of the elevation in maternal serum AFP, which led to further evaluation. However, he points out that ventricular anomalies detected by ultrasound studies were more "precisely defined by prenatal MRI."

He believes MRI should be considered to further delineate intracranial anomalies to better prepare families and care providers for postnatal expectations; however, there are limitations, he noted.

"One of the current limitations is that MRI is much more costly than ultrasound is, and there is limited availability. In addition, we don't know what the false-positive and false-negative diagnoses will be with fetal application of MRI. Nevertheless, it seems to be a promising new tool," he concluded. ■

Placental Compensation May Affect Fetal Growth

BY KAREN RICHARDSON
Contributing Writer

QUEBEC CITY — Placental compensation may influence fetal growth in women with gestational hypertension, according to research presented at the annual meeting of the Society of Obstetricians and Gynaecologists of Canada.

"Pregnancies complicated by gestational hypertension and to a greater extent preeclampsia had significantly lower birth weight/placental weight ratios, compared with the controls at 38 and 39 weeks," said Rebecca Cash, M.D., a resident in obstetrics and gynecology at the University of Toronto.

Larger placenta size suggests there is a compensatory increase in placental weight for decreased function in gestational hypertension.

In the retrospective cohort study, Dr. Cash and her associates analyzed information on 12,422 term pregnancies (37-41 weeks) using data from the St. Joseph's Health Care, London perinatal database on births from Nov. 1, 1995 to November 1999. Singleton pregnancies complicated by gestational hypertension (1,084 cases), preeclampsia (144), or chronic hypertension (129) were compared with pregnancies in normotensive controls (11,065).

At 38 weeks, women with preeclampsia had significantly smaller babies than did controls (3,350 g vs. 3,520 g), whereas there was no significant difference in birth weight in infants born to women with gestational hypertension and controls.

"In preeclampsia, the reduction in the ratio indicates that the fetus is undergrown in relation to placental size, suggesting functional placental impairment," Dr. Cash said.

Pregnancies complicated by gestational hypertension showed statistically significantly larger placenta weights vs. pregnancies in the control group at 38 and 39 weeks (692 g vs. 682 g, respectively), but not at 40 and 41 weeks.

Larger placenta size suggests there is a compensatory increase in placental weight for decreased function in gestational hypertension, which may influence fetal growth. "Abnormal placentation is thought to play a central role in the pathophysiology of preeclampsia," said Dr. Cash. She added that this may have an effect on long-term outcomes, as findings of low birth weight and large placenta are independent risk factors for cardiovascular disease in adulthood.

In the study, gestational hypertension was defined as maternal blood pressure greater than 140/90 after 20 weeks' gestation without proteinuria. Preeclampsia was defined as maternal blood pressure greater than 140/90 after 20 weeks' gestation, accompanied by proteinuria or other end organ abnormalities. Chronic hypertension was defined as maternal blood pressure greater than 140/90 before 20 weeks' gestation.

Pregnancies complicated by diabetes, stillbirth, and congenital or chromosomal abnormalities were excluded from the analysis. Placental weights were routinely determined without trimming membranes or draining blood.

Dr. Cash's associate in the study was Rob Gratton, M.D., of the University of Western Ontario, London. ■

T1-Weighted MRI Confirms Postdural Puncture Headache

BY KERRI WACHTER
Senior Writer

ASHEVILLE, N.C. — If you suspect a postdural puncture headache but aren't sure, order a T1-weighted MRI with gadolinium contrast for the patient, David C. Mayer, M.D., advised at the Southern Obstetric and Gynecologic Seminar.

"It used to be that there were no imaging studies available to make the diagnosis of postdural puncture headache. That has now changed," said Dr. Mayer, a professor of obstetrics and gynecology and of anesthesiology at the University of North Carolina at Chapel Hill.

Signs of postdural puncture headaches (PDPH) cannot be seen on CT scans (with and without contrast) or noncontrast MRI.

MRI (T1 weighted) with gadolinium contrast, however, reveals changes that can make a difference in the diagnosis of PDPH. This particular type of MRI rules out more serious conditions, such as subdural hematoma and intracranial masses. The two key findings using T1-weighted contrast MRI are meningeal enhancement and descent or sagging of the brain. Diffuse meningeal enhancement is seen on the MRI. "The meninges . . . light up with gadolinium," Dr. Mayer explained.

'It used to be that there were no imaging studies available to make the diagnosis of postdural puncture headache. That has now changed.'

Less frequently, the pituitary may appear large—though this can be seen with CT as well—and engorged cerebral venous sinuses may also be seen.

Downward displacement of the brain can also be seen (similar to a Chiari malformation) with this type of imaging. There may also be descent of the cerebellar tonsils, obliteration of prepontine, perichiasmatic cisterns, flattening of the optic chiasm, crowding of the posterior fossa, as well as decreased ventricular size, according to Dr. Mayer.

PDPH onset commonly occurs while the patient is in the hospital.The headache usually has a postural component—worsening on standing and decreasing in a prone position. Other common symptoms include neck pain, nausea and vomiting, changes in hearing, and visual blurring or field cuts. However, atypical symptoms include interscapular pain, low-back pain, face numbness or weakness, galactorrhea, and radicular upper-limb symptoms.

"What people are now learning is that it is not just a pressure problem, it's a volume problem," Dr. Mayer said.

CSF volume is a very well-regulated system. When volume changes occur, the system compensates. Intracranial veins dilate to maintain intracranial volume. Extensive venodilation may exert pressure on pain-sensitive structures (such as the meninges). The pituitary may enlarge. Brain sag—possibly as a result of reduced CSF pressure/volume—can compress and stretch structures and veins in the brain, leading to an increased risk of subdural hematoma. ■