Published in final edited form as:
J Ultrasound Med. 2007 November ; 26(11): 1513–1522.

What Does Magnetic Resonance Imaging Add to the Prenatal Sonographic Diagnosis of Ventriculomegaly?

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Abstract

Objective—The purpose of this study was to determine the contribution of magnetic resonance imaging (MRI) in evaluating fetuses with the sonographic diagnosis of ventriculomegaly (VM).

Methods—Over 4 years, consecutive fetuses with the sonographic diagnosis of VM at 1 facility who underwent prenatal MRI at a second facility were included. The roles of MRI and follow-up sonography were tabulated. The patients were analyzed in 2 groups based on the presence or absence of other central nervous system (CNS) abnormalities.

Results—Twenty-six fetuses with a gestational age range of 17 to 37 weeks had sonographically detected VM (atria ≥10–29 mm), including 19 with mild VM (atria 10–12 mm). In group 1, 14 had isolated VM, 6 of which reverted to normal by the third trimester. Magnetic resonance imaging showed cerebellar hypoplasia not shown by sonography in 1 fetus and an additional finding of a mega cisterna magna in a second fetus. In group 2, 12 fetuses had VM and other CNS anomalies on sonography. Additional findings were seen with MRI in 10 of these fetuses, including migrational abnormalities (n = 4), porencephaly (n = 4), and 1 diagnosis each of abnormal myelination, hypoplasia of the corpus callosum, microcephaly, a kinked brain stem, cerebellar hypoplasia, and congenital infarction. There were significantly more fetuses with additional CNS anomalies found by MRI among those in group 2 compared with those in group 1 (Fisher exact test, $P = .001$).

Conclusions—Although sonography is an accurate diagnostic modality for the evaluation of fetuses with VM, MRI adds important additional information, particularly in fetuses in whom additional findings other than an enlarged ventricle are seen sonographically.

Keywords

comparison; fetal central nervous system; prenatal magnetic resonance imaging; prenatal sonography; ventriculomegaly

Ventriculomegaly (VM) is a nonspecific dilatation of the lateral ventricles in second- and third-trimester fetuses, which can result from many different types of brain abnormalities or insults. There is a wide range of prognoses and outcomes for fetuses with the in utero diagnosis of VM. Fetuses with isolated mild VM have the most favorable outcomes compared with those who have severe ventricular dilatation or additional central nervous system (CNS) anomalies.

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Guest Editor: Alfred Z. Abuhamad, MD.

[1,2] Therefore, it is crucial for the appropriate counseling of affected patients that the diagnosis of all the CNS abnormalities be accurate and complete.

Although sonography is the mainstay of the evaluation and diagnosis of fetuses with VM, magnetic resonance imaging (MRI) is particularly helpful in fetuses with CNS abnormalities, reportedly improving the precision and accuracy of these diagnoses.[3-6] Agenesis of the corpus callosum (ACC) is an example of such a brain malformation, which can be a subtle diagnosis and is frequently missed during second-trimester sonography.[6,7] Magnetic resonance imaging also improves the detection of brain parenchymal disorders, migrational abnormalities, and irregularities of the ventricular walls, such as heterotopias.[8-10] We undertook this study to evaluate the additional information provided by adding MRI to the imaging protocol for all patients with the sonographic diagnosis of VM and to determine which patients benefited the most from MRI after the sonographic diagnosis of VM.

Materials and Methods

Over the course of 4 years, consecutive second- and third-trimester fetuses with the sonographic diagnosis of VM at Diagnostic Ultrasound Associates (Boston, MA) who underwent subsequent MRI at the Beth Israel Deaconess Medical Center were included in the study. This study was approved by the Institutional Review Board at Beth Israel Deaconess Medical Center, and written informed consent was obtained.

Sonography was performed with a Voluson 730 Expert system and a 4- to 7-MHz transabdominal transducer (GE Healthcare, Milwaukee, WI). A transvaginal examination was performed on all fetuses in the cephalic presentation. The sonograms consisted of a complete evaluation of the brain, with an attempt to visualize the corpus callosum, measure the lateral ventricles, and view the walls of the lateral ventricles, parenchyma of the brain, and posterior fossa. The lateral ventricular atria were measured, and those with a measurement of 10 mm or larger were offered MR examinations. Mild VM was described when the atrial measurement was 10 to 12 mm. Data from the sonographic study included the indication for the examination and fetal gestational age by last menstrual period or by early sonography if the patient had been redated. Although 3-dimensional (3D) sonography was done on a few of our patients, the value of 3D reconstruction was not evaluated and therefore is not included.

After screening for contraindications to MRI, patients underwent MRI on a 1.5-T superconductive system (Symphony, Siemens AG, Erlangen, Germany; or Signa, GE Healthcare) using a 4- or 8-element body phased array coil, a torso coil, or both. The minimum rise time was 600 microseconds (for a 25-mT peak gradient amplitude). The whole-body specific absorption rate was kept at less than 3.0 W/kg. A scout view was obtained, and fetal images were obtained with half-Fourier single-shot fast spin echo imaging in the fetal sagittal, coronal, and axial planes (Siemens protocol: echo spacing, 4.2 milliseconds; repetition time [TR]/echo time [TE], infinite/60 milliseconds; 0.5 excitations; echo train length, 72; 1 acquisition, section thickness, 3–4 mm; field of view tailored to the individual patient; and minimum 192 × 256 acquisition matrix; GE protocol: TR/TE, infinite/120 milliseconds; 0.5 excitations; 1 acquisition; section thickness, 3–4 mm; field of view tailored to the individual patient, minimum 192 × 256 acquisition matrix; and bandwidth, 31.5–62.5 kHz). A refocusing flip angle of 130° to 150° was used to minimize the amount of radio frequency power deposition. T1-weighted images (breath hold; TR/TE, 126/4 milliseconds; flip angle, 80°; 1 acquisition; section thickness, 5 mm; field of view individually tailored; and 128 × 256 matrix) were obtained in at least 1 plane. Sequences were repeated as needed when motion occurred.

Amniocentesis and follow-up scans during pregnancy were performed at the discretion of the referring obstetrician, and data from these studies were obtained. Fetal and neonatal outcomes

were obtained whenever possible, based on autopsy, postnatal imaging studies, and clinical evaluation of the neonate.

Patients were grouped according to their sonographic findings: group 1, sonographically isolated VM (ie, the only sonographic finding in the CNS); and group 2, sonographic VM with other CNS anomalies. The Fisher exact test was used to compare the number of fetuses in each group with respect to whether additional findings were seen on prenatal MRI. The Student *t* test was used to compare the gestational ages of the groups with and without additional findings on prenatal MRI.

Results

Twenty-five women with 26 affected fetuses (3 sets of twins, 1 with both twins having mild VM) at 17 to 37 weeks' gestation had sonographically detected VM (atrial measurement of 10–29 mm). Indications for the scans included a routine sonographic survey (n = 3), a query for a brain abnormality detected on outside sonography (n = 10, 1 with twins, with each having mild VM), twins (n = 1), advanced maternal age (n = 4), previous pregnancy or family history of an abnormality (n = 2), exposure to parvovirus (n = 1), and follow-up of a prior sonographic abnormality in this pregnancy including 1 case each of a question of a clubfoot, a dilated bowel and pericardial effusion, an echogenic bowel, and an echogenic intracardiac focus.

Tables 1 and 2 show the gestational ages at the time of the imaging studies, imaging findings, and outcomes. Nineteen of these 26 fetuses had mild VM; in 13 of these mild cases, the VM was isolated.

Group 1

Fourteen fetuses had VM as the only sonographic finding in the CNS. Eleven of these cases were diagnosed at 24 weeks or earlier, and 3 were diagnosed after 24 weeks. Of these fetuses, 13 had mild VM, which reverted to normal by follow-up second- or third-trimester scans in 6 fetuses. Seven fetuses had sonograms earlier in pregnancy that showed a normal appearance of the CNS at 17 to 20 weeks. One fetus had an MRI diagnosis of cerebelluar hypoplasia, which was not shown on sonography; 1 fetus had an enlarged cisterna magna that was not diagnosed by sonography; and the other 12 fetuses had congruent sonographic and MRI findings. Neonatal outcomes were good in this group, with normal newborn examination findings in 10 of 14, Fanconi anemia in 1, mild proximal weakness in 1, and no follow-up in 2 (1 due to termination without available pathologic evaluation and 1 lost to follow-up).

Group 2

Twelve fetuses had VM with associated CNS abnormalities suspected sonographically, including 4 with a nonvisualized corpus callosum, 2 with a nonvisualized cavum septi pellucidi (1 also with a nonvisualized corpus callosum), 2 with suspected intracranial hemorrhage, and 1 case each of germinal matrix cysts (Figure 1), microcephaly, lissencephaly, cerebellar hypoplasia, and heterotopia (Figure 2). Of the 12 fetuses in this group, 10 had additional findings on MRI. Three of the 4 fetuses with sonographically suspected ACC had ACC confirmed on MRI, and 1 had septo-optic dysplasia. Additional MRI findings were migrational abnormality (n = 4; Figure 3), porencephaly (n = 4), and 1 diagnosis each of abnormal myelination, hypoplasia of the corpus callosum, microcephaly, a kinked brain stem, cerebellar hypoplasia, and congenital infarction. There were 8 terminations of pregnancies or stillborn fetuses, 3 with abnormal neurologic follow-up results and 1 lost to follow-up.

Table 3 compares the 14 fetuses with sonographically isolated VM and the 12 fetuses in group 2 who had additional CNS anomalies seen sonographically in terms of the additional anomalies

identified with MRI. There were significantly more fetuses who had additional CNS anomalies found by MRI among those in group 2 compared with those in group 1 (Fisher exact test, *P* = .001). There was no statistically significant difference in the gestational ages of the 2 groups.

Discussion

Sonography has been the well-accepted mainstay of the prenatal diagnosis of brain abnormalities for many decades. More recently, MRI has provided an opportunity for further evaluation of these abnormalities.[3-6] Our study shows that although sonography was an accurate diagnostic modality for most fetuses with VM, MRI did add important information, particularly for fetuses with other CNS anomalies detected sonographically.

In our study, 10 of 12 fetuses with VM and other sonographic abnormalities had additional findings on MRI compared with sonography. Clearly this is the group of fetuses in which MRI has the largest potential impact. There was a significant difference in the additional information provided by MRI for those fetuses with additional CNS anomalies compared with fetuses with isolated VM on sonography. Only 2 of our 14 cases of isolated VM had additional findings on MRI, including 1 with cerebellar hypoplasia and 1 with a mega cisterna magna. The importance of the cerebellar hypoplasia remained undetermined in this fetus because of termination of the pregnancy. The other infant with the mega cisterna magna had mild proximal weakness but had no other abnormalities as of 6 months of age.

Unlike in other studies, there were no fetuses with ACC that remained unidentified by sonography.[7,8] This is likely due to our increased understanding of the appearance of ACC in the second trimester and attention to views of the frontal horns and cavum septi pellucidi on fetal surveys. Two fetuses sonographically identified as having ACC were additionally found to have migrational abnormalities on MRI. This indicates that in fetuses with ACC, MRI is particularly important in evaluating cortical malformations.

There were 12 fetuses who had a normal second-trimester scan between 16 and 21 weeks but in whom VM developed later in pregnancy, 2 of whom had intracranial bleeding that likely occurred later in gestation as the cause of the VM, 2 of whom had lissencephaly, 1 of whom had a dysplastic brain with a kinked brain stem, and 7 whose causes of VM were unclear.

Importantly, 1 case of heterotopia was correctly identified sonographically. Previously, this was exclusively an MRI diagnosis, but with increased understanding of the appearance of heterotopia as an irregularity projecting into the ventricle, we can now make this diagnosis with sonography.[9,10]

It is difficult to truly compare the benefits of sonography and MRI because of differences in the gestational age when these studies are done.

Sonography is the general screening modality by which fetal anomalies are first discovered; therefore, there is often a lag time between the abnormal sonogram and the MR image. Most studies have not been able to address the affect of the later gestational age at MRI on the additional diagnoses made. It is clear that development of the brain is a continuum, and abnormalities can evolve over time, as shown by the 4 patients in group 2 who initially had normal sonograms and in whom CNS anomalies developed later in gestation.

A limitation of this study was the lack of complete follow-up because not all fetuses had standardized assessments after delivery, and the follow-up interval was limited. We are currently following a larger cohort of fetuses with VM with standardized neurologic assessments. The purpose of this article is to elucidate the different presentations fetuses can have with VM and to illustrate in which cases MRI can add additional information.

Prenatal detection of cortical maldevelopment is also difficult sonographically but has been more successful recently, as reported by Malinger et al.[11] Our study had 2 fetuses with polymicrogyria seen with MRI only and 2 fetuses with lissencephaly, only 1 of which was detected sonographically. Valsky et al[5] also evaluated the role of MRI in 36 fetuses with isolated mild VM. They found that MRI showed additional findings in 3 fetuses: 1 with periventricular cystic lesions and abnormal sulcation and 2 with small germinal matrix hemorrhages. Glenn et al[8] also reported 5 of 8 fetuses with suspected callosal abnormalities on sonography who had additional CNS anomalies detected with MRI. These additional anomalies consisted of an abnormal appearance of brain parenchyma with a shallow sylvian fissure, delayed or abnormal sulcation, a neural migrational disorder with an absent sylvian fissure, schizencephaly, cerebellar dysgenesis, and microphthalmia.

Although most authors agree that MRI does add important information to some cases of VM detected with sonography, other investigators report that sonography and MRI are comparable in accuracy, although they occasionally differ slightly in the interpretation of CNS anomalies.[11,12] Malinger et al[11] suggested that detailed neurosonography is equal to MRI in the diagnosis of fetal brain abnormalities. Monteagudo et al[12] also suggested that transvaginal neurosonography done with 3D imaging is an effective method of evaluating the fetal brain; however, these 3D techniques have not been compared with MRI in a systematic manner.

Nevertheless, sonography is likely to remain at the front line of prenatal diagnosis of CNS anomalies. Our study supports the belief that sonography and MRI are complementary in the delineation of CNS abnormalities of the fetus. In fetuses with sonographically detected VM, MRI can play an important role in detecting additional findings, which may help focus the patient's counseling and management.

Acknowledgements

This study was supported by National Institute of Biomedical Imaging and Bioengineering grant 01998 from the National Institutes of Health.

Abbreviations

ACC, agenesis of the corpus callosum; CNS, central nervous system; MRI, magnetic resonance imaging; TE, echo time; 3D, 3-dimensional; TR, repetition time; VM, ventriculomegaly.

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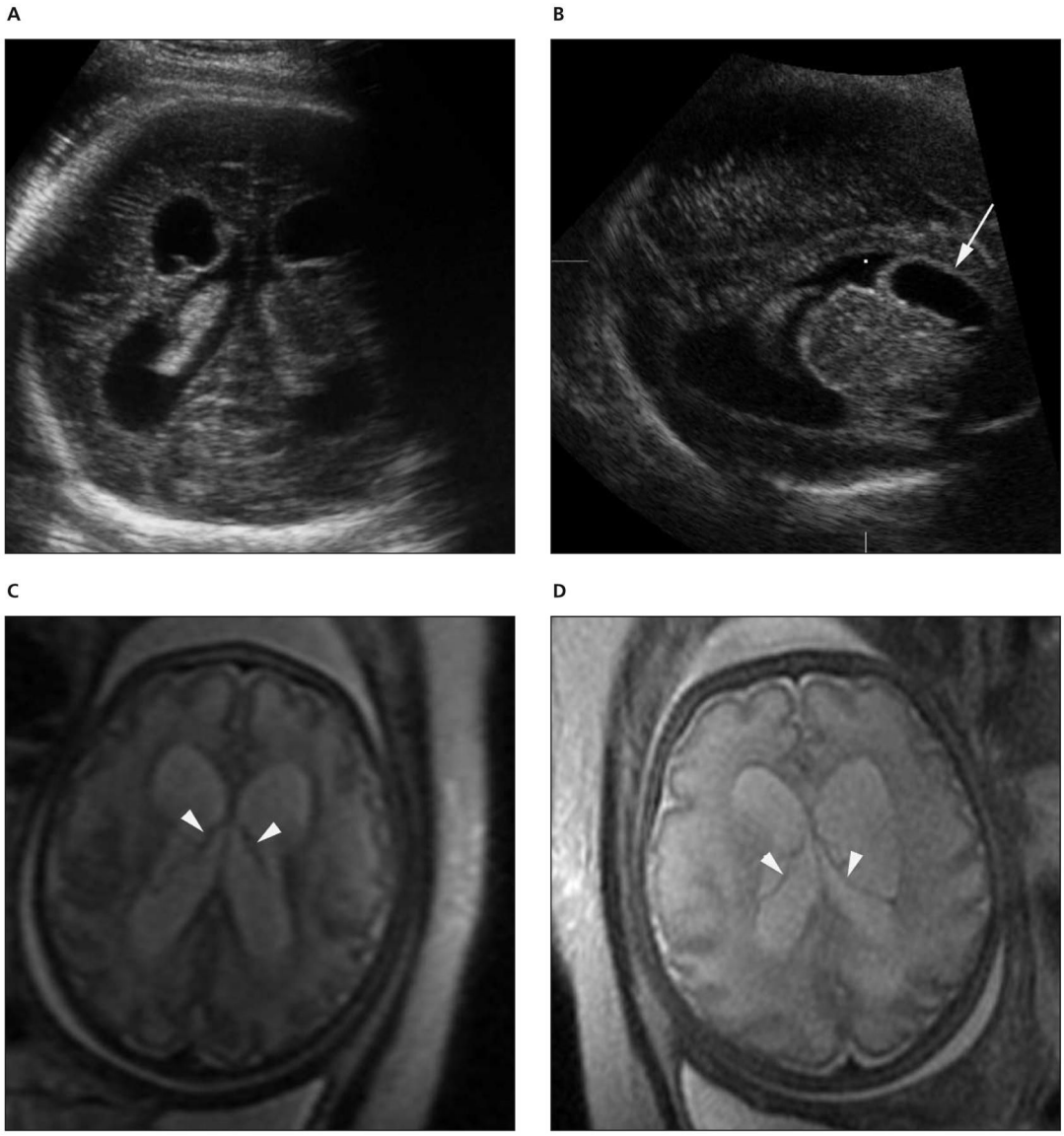

Figure 1.
Fetus at 37 weeks with large germinal matrix cysts. **A** and **B**, Axial (**A**) and sagittal (**B**) sonograms show enlarged ventricles with cysts (arrow) impinging on the frontal horns. **C** and **D**, Axial MR images show the cysts with the cyst walls (arrowheads) shown impinging on the frontal horns. In addition, there is a diffuse abnormal signal in the white matter, suggesting abnormal myelination.

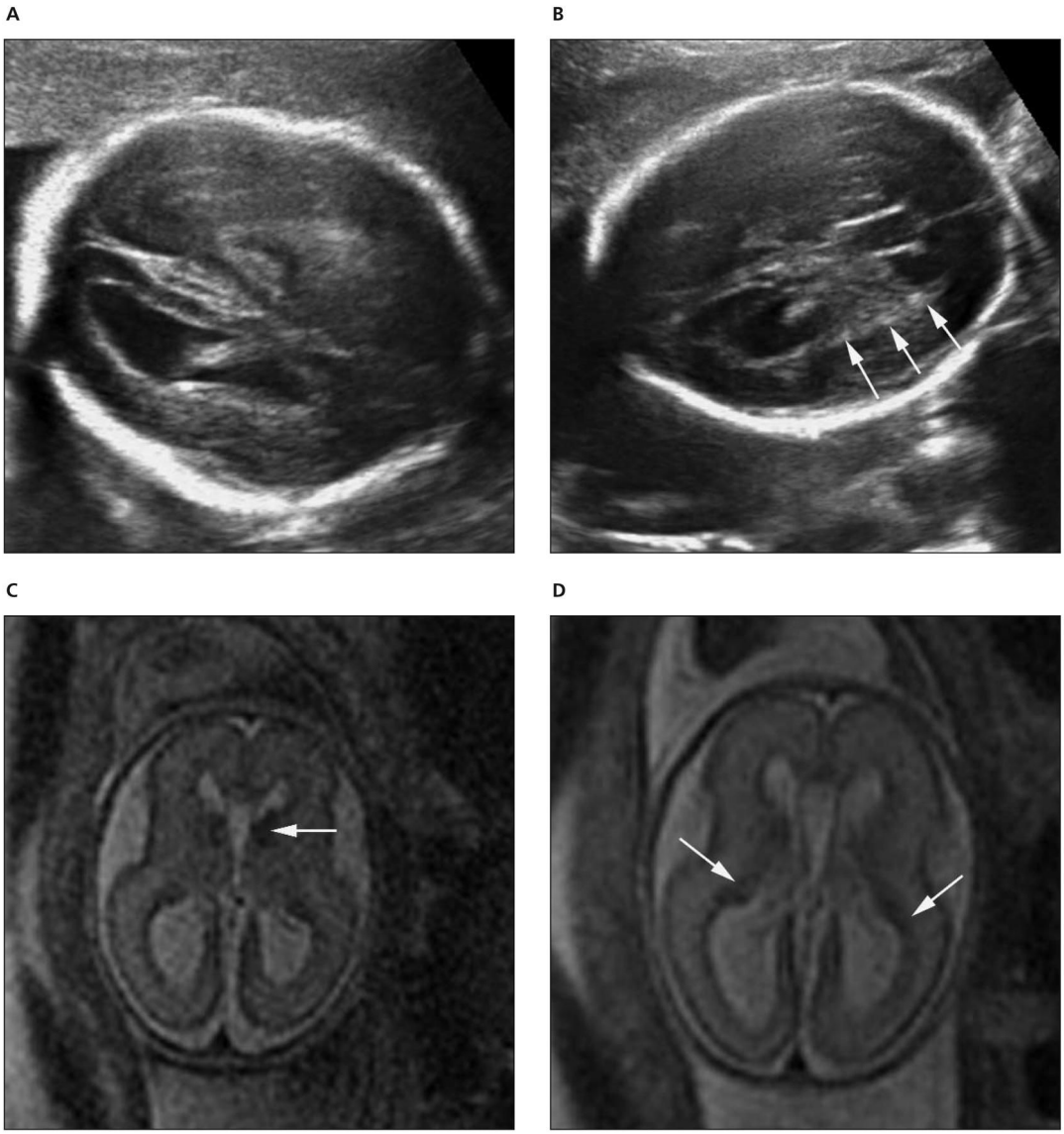

Figure 2.
Fetus at 23 weeks with borderline VM and a question of heterotopia. **A** and **B**, Axial sonograms show nodular irregularities (arrows) of the ventricular lining. **C** and **D**, Axial MR images show nodular elongated areas with a dark signal (arrows) lining the ventricles. This may represent early areas of heterotopia. Other findings (not shown) were a 2-vessel cord and an echogenic bowel. The fetus died in utero within 1 week after the imaging.

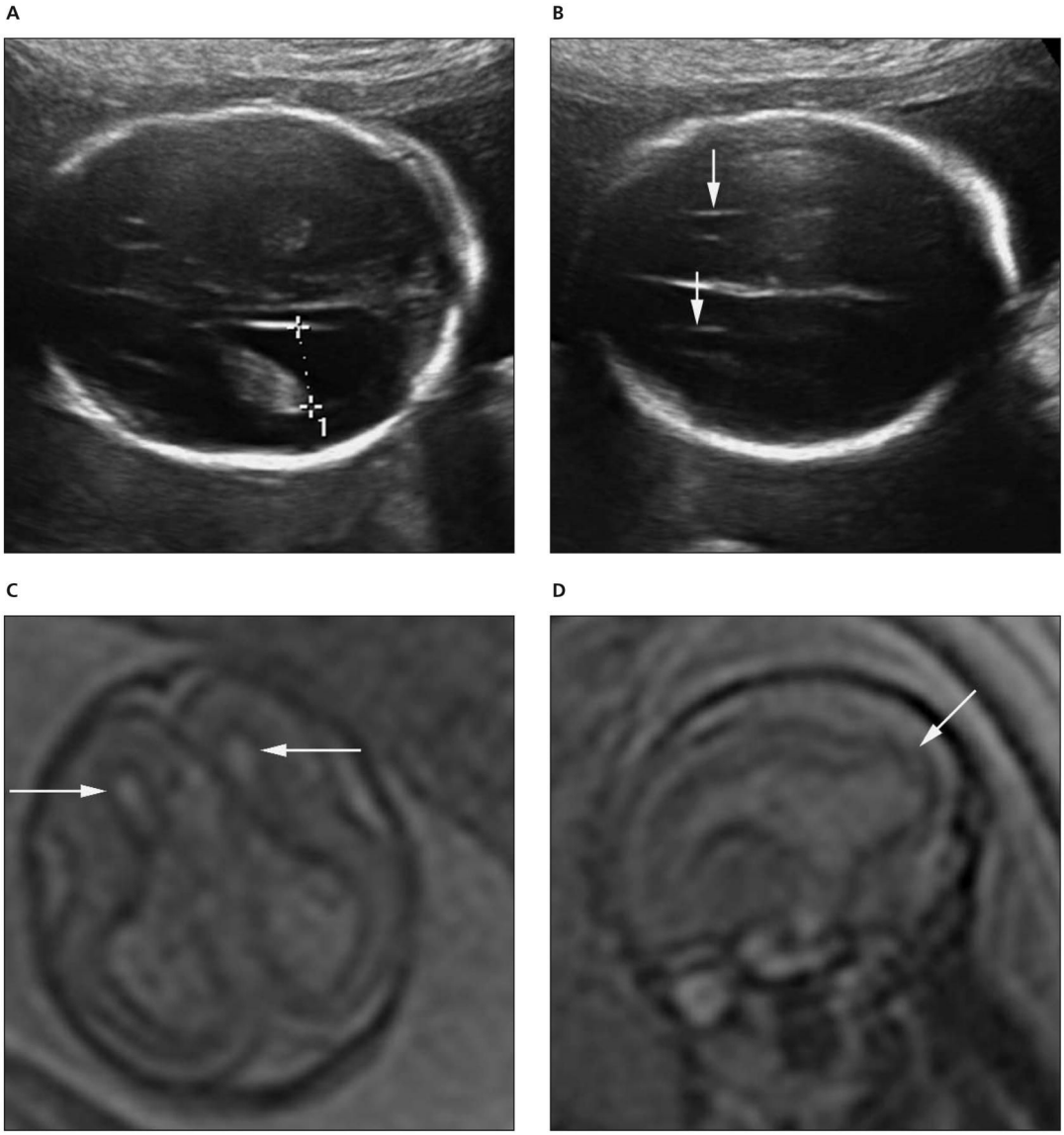

Figure 3.
Fetus at 22 weeks with ACC. **A** and **B**, Axial sonograms show borderline VM (calipers) with a parallel orientation of the frontal horns (arrows), consistent with ACC. **C**, Axial MR image shows an appearance similar to that of the ventricles (arrows). **D**, Sagittal MR image shows an irregular contour of the ventricle and occipital cortex (arrow), suggesting an early appearance of a migrational abnormality that was not visualized sonographically.

Table 1

Fetuses With Sonographically Isolated VM

GA at VM Diagnosis, wk	GA at MRI, wk	Larger Ventricle Diameter at Atrium, mm	MRI Findings Other Than VM	Other Findings on Most Recent or Other Sonographic Studies	Outcome of Birth	Postnatal Imaging, Pathology, Karyotype	Postnatal Clinical Outcome
18	18	10			Live birth	Head sonography: mild VM and CPC	Normal at 5 mo
18	18	10		21 wk mild VM	Live birth		Normal newborn examination
19	20	11			Live birth		Normal newborn examination
19	20	10		21 wk normal	Live birth		Fanconi anemia
20	20	11	Cerebellar hypoplasia		TAB	Pathology: brain fragmented, normal karyotype	NA
21	22	10		19 and 23 wk normal	Live birth	Head sonography: normal	Normal at 6 mo
22	22	13		Progressive ventricular dilatation measuring 14 mm at 24 wk, 20 mm at 28 wk, 33 mm at 36 wk	Live birth	MRI: severe VM, absent septum pellucidum, ? migrational abnormality	Large head size with frontal bossing, otherwise normal at 2 y
22 and 29	29	10		18 wk normal but limited	Live birth		Normal newborn examination
23	23	10		28 and 34 wk mild VM	Live birth	Head sonography: normal	Normal at 6 mo
24	26	12		18 wk normal, 28 wk mild VM, 32 wk normal	Live birth	Head sonography: small left choroid plexus cyst, otherwise normal	Normal newborn examination
24	26	10		18 wk normal, 28 wk mild VM, 32 wk normal	Live birth	Head sonography: normal	Normal newborn examination
25 and 33	33	10	Mega cisterna magna	18 wk survey with dilated bowel and pericardial effusion, 20 wk no change, 37 wk mild VM	Live birth	Sonography/MRI: mild VM	Mild proximal weakness, otherwise normal at 6 mo
30	30	11		19 wk normal with foot slightly turned inward but no clubfoot, 31 wk mild VM, 33 and 35 wk normal	Live birth	Sonography: slight asymmetry of ventricles, most likely a normal variant, otherwise normal	Normal newborn examination
30	31	11		17 wk normal with echogenic intracardiac focus, 34 and 36 wk mild VM	Live birth		Normal newborn examination

CPC indicates choroid plexus cyst; GA, gestational age; NA, not applicable; and TAB, therapeutic abortion.

J Ultrasound Med. Author manuscript; available in PMC 2008 March 4.

Table 2

Fetuses With VM and Other CNS Sonographic Findings

GA at VM Diagnosis, wk	GA at MRI	Larger Ventricle Diameter at Atrium, mm	Other CNS Sonographic Findings at Time of VM Diagnosis	MRI Findings Other Than VM	Other Findings on Most Recent or Other Sonographic Studies	Outcome of Birth	Postnatal Imaging, Pathology, Karyotype	Postnatal Clinical Outcome
17	18	12	Cerebellar hypoplasia	Cerebellar hypoplasia	Hemivertebra, short long bones	TAB	Normal karyotype	NA
18	18	10	ACC	ACC, hypertelorism	16 wk with 9-mm ventricles	Live birth	Sonography/MRI: ACC, fused coronal sutures	Mitten hands, Apert syndrome
18	20	10	ACC, inter-hemispheric cyst	ACC cortical gyral abnormality, inter-hemispheric cyst		TAB	Pathology: ACC, inter-hemispheric cyst; normal karyotype	NA
19	19	14	Cavum septi pellucidi not visualized	Cerebellar hypoplasia, dysplastic brain with kinked brain stem	16 wk normal	TAB	Normal karyotype	NA
20	20	22	Cavum septi pellucidi and corpus callosum not visualized	Septo-optic dysplasia, cerebellar hypoplasia, porencephaly		TAB	Normal karyotype	NA
21	21	11	ACC	ACC, migrational abnormality		TAB		NA
23	23	10	? Heterotopia	Polymicrogyria, heterotopia	Echogenic bowel, 2-vessel cord	IUFD 23 wk		NA
29	29	12	Lissencephaly	Microcephaly, lissencephaly	18, 20, 22, and 28 wk normal	TAB	Karyotype: 46,XX.ish del(17)(p13.3) (LIS1–)	NA
29	29	19	Grade 4 IVH	Grade 4 IVH, congenital infarction, porencephaly	18 and 22 wk with abdominal calcifications	TAB		NA
35	35	29	Bleeding suspected	Grade 4 IVH, porencephaly	22 wk normal	Live birth	MRI: asymmetric VM, left grade 4 IVH	Lost to follow-up
36	36	20	Microcephaly	Microcephaly, hypoplasia corpus callosum, migrational abnormality with primitive gyral pattern	21 wk normal	Live birth	Sonography/ MRI: lissencephaly	At 2 y global weakness and spasticity with developmental delay
37	37	16	Large germinal matrix cysts	Subependymal cysts, abnormal myelination, porencephaly	Non-CNS finding of umbilical vein varix	Live birth	MRI: dysmorphic VM, septations along frontal horns, enlarged 3rd ventricle	At 1 y mild hypotonia, otherwise normal

IUFD indicates in utero fetal death; IVH, interventricular hemorrhage; NA, not applicable; and TAB, therapeutic abortion.

Table 3

Comparisons of Fetuses With and Without Additional Findings on MRI

Parameter	No Additional Findings on MRI	Additional Findings on MRI
Isolated VM on sonography*	12	2
VM and other findings on sonography*	2	10
GA at VM diagnosis, wk	21.8 ± 4.1 (17–30)	26.0 ± 7.0 (18–37)†
GA at time of MRI, wk	22.9 ± 4.7 (18–31)	26.8 ± 7.1 (19–37)†

GA indicates gestational age; values are mean ± SD (range).

*Fisher exact test shows $P = .001$.

†Gestational ages not significantly different.