Magnetic Resonance Imaging in the Evaluation of Normal Fetal Brain Development and Brain and Spinal Abnormalities

Poster No.: C-0317
Congress: ECR 2011
Type: Educational Exhibit
Authors: M. Recio¹, P. Martínez Ten¹, J. Pérez Pedregosa¹, J. Carrascoso Arranz², V. Martínez de Vega³, D. Martín Fernández Mayoralas¹, R. Cano Alonso⁴, J. J. Gómez Herrera⁵; ¹Madrid/ES, ²PUZUELO DE ALARCON. MADRID/ES, ³Pozuelo de Alarcon Madrid/ES, ⁴POZUELO de ALARCON, MADRID/ES, ⁵ES
Keywords: Congenital, Diagnostic procedure, MR, Neuroradiology spine, Neuroradiology brain, Foetal imaging
DOI: 10.1594/ecr2011/C-0317

Any information contained in this pdf file is automatically generated from digital material submitted to EPOS by third parties in the form of scientific presentations. References to any names, marks, products, or services of third parties or hypertext links to third-party sites or information are provided solely as a convenience to you and do not in any way constitute or imply ECR's endorsement, sponsorship or recommendation of the third party, information, product or service. ECR is not responsible for the content of these pages and does not make any representations regarding the content or accuracy of material in this file.

As per copyright regulations, any unauthorised use of the material or parts thereof as well as commercial reproduction or multiple distribution by any traditional or electronically based reproduction/publication method is strictly prohibited.

You agree to defend, indemnify, and hold ECR harmless from and against any and all claims, damages, costs, and expenses, including attorneys' fees, arising from or related to your use of these pages.

Please note: Links to movies, ppt slideshows and any other multimedia files are not available in the pdf version of presentations.
Learning objectives

Fetal MR imaging (MRI) is an increasingly available technique used to evaluate the fetal brain and spine, as it provides a unique opportunity to evaluate fetal brain development and make an early diagnosis of congenital abnormalities that may be inadequately visualized or even undetectable by means of prenatal sonography.

One hundred nineteen fetuses between the 20\textsuperscript{th} and the 37.5\textsuperscript{th} weeks of gestation were examined employing FIESTA, SSFSE, diffusion and gradient T1 pulse sequences, using a 1.5T MR scanner.

Background

We evaluate the main indications for fetal brain MRI and the findings of normal brain development and brain and spinal abnormalities.

• The use of MRI in pregnancy was first reported in 1983 by Smith et al. in "The Lancet".

• More than 20 years later, we must acknowledge that they were right:

- A PubMed search (in December 2010) on 'fetal MRI' found more than 3200 references, 1320 of which concerning 'fetal brain MRI'.

• Fetal brain MRI is available in many prenatal diagnostic centers and radiologists have developed highly specialized skills to perform and interpret such examinations.

• MRI allows a better differentiation between normal and abnormal signal intensity of the fetal tissues because of its higher contrast resolution, as compared to prenatal sonography (US). Therefore, structural abnormalities such as cerebral malformations and destructive lesions that may be sonographically occult on prenatal ultrasound can be detectable at fetal MRI.

Imaging findings OR Procedure details

SAFETY ISSUES
• Many studies have been performed to assess the safety of MRI in pregnant animals and animal embryos. It is difficult to directly extrapolate these data to clinical human fetal MR examinations. Studies concerning MRI safety in pregnant women are even more limited.

• The American College of Radiology White Paper on MR safety (published in 2002) states that: "Pregnant patients can be accepted to undergo MR images at any stage of pregnancy if, in the determination of a Level Two MR Personnel-designated attending radiologist, the risk-benefit ratio to the patient warrants that the study be performed."

• However, because of the potential risk of MRI to the developing fetus and the current limitations of fetal MRI, it is prudent to wait until after the first trimester before performing a fetal MRI.

- It is preferable to wait until at least gestational week 20 to minimize the difficulties created by the small size and excessive motion of younger fetuses.

• Intravenous contrast material is not recommended in fetal MRI because of the potential risk to the fetus.

ADVANTAGES OF FETAL MR IMAGING OVER PRENATAL SONOGRAPHY.

• Fetal MR imaging has improved contrast resolution compared with prenatal sonography. A phased-array surface coil should be used (Cardiac 8 channels).

• Fetal MR imaging also allows direct visualization of both sides of the fetal brain (additional limitations of sonography, resulting from decreased amniotic fluid volume, fetal positioning, and acoustic shadowing from the ossifying calvaria, can also be overcome by fetal MR imaging).

• Fetal MR imaging allows a more detailed evaluation of the developing brain (including direct visualization and assessment of the developing cortex and sulcation pattern, which is extremely difficult and often impossible with sonography).

LIMITATIONS OF FETAL MR IMAGING.

• Fetal motion because . Fast sequences should be performed, and fasting (# 4 hours) is recommended in order to reduce movement artifacts.

• The small size of the structure being imaged (usually the fetal brain or spine) and the large distance between the fetus (which lies within the uterine cavity) and the receiver coil (which lies on the mother's abdomen and pelvis). These limitations are currently being overcome with advances in coil design, such as parallel imaging with increasing number
of channels, but are still important factors contributing to the inherent limitations of fetal MR imaging with young gestational age fetuses.

• Maternal claustrophobia and discomfort during the scan are other limitations of fetal MR imaging compared with sonography, though these are typically more problematic with advanced gestational age.

**INDICATIONS FOR FETAL BRAIN MRI:**

• A history of severe brain abnormality in a previous pregnancy, but ultrasound examination is considered normal; MRI is performed in order to look for subtle signs of recurrence.

• An abnormality identified on ultrasound examination that appears to be isolated (typically ventriculomegaly or corpus callosum agenesis); MRI is performed to look for potential abnormalities that may have been overlooked by ultrasound.

• An abnormality diagnosed on ultrasound examination, but the examination cannot be completed because of technical problems (e.g. maternal adiposity, fetal position); MRI is performed to supplement ultrasound.

• A high risk of development of brain abnormality, especially in cases of fetal infection (mostly cytomegalovirus, varicella and toxoplasmosis) or ischemic damage (in-utero death of a monochorionic twin, twin-to-twin transfusion syndrome).

**FETAL BRAIN MRI: HOW?**

• Fetal MRI should be performed using a high-field MR scanner (1.5 T) in order to obtain optimal imaging results.

• Coil selection: A phased-array surface coil should be used (Cardiac 8 channels).

• Fast T2 weighted sequences remain the basis of fetal MRI. They are the most frequently used (SSFSE, FIESTA).

• Fasting (# 4 hours) is recommended in order to reduce movement artifacts.

• Fast sequences should be performed during maternal breathholding.

• Sedation is not routinely employed.

• Overall scanning time: 30-40 minutes.
IMAGING PROTOCOL:

- Patients should be scanned in the supine position. If this is not possible, as in advanced stages of pregnancy, the lateral decubitus position is preferred to avoid compression of the inferior vena cava.

- Fetal MR examination should start with a localizer in three planes along maternal axes, in order to further orientate the examination.

- All subsequent scans should be oriented along fetal axes.

(Figure 1). on page 10

HICH SEQUENCES?

- Fast T2-weighted sequences, such as Single Shot Fast Spin Echo T2 (SSFSE T2) and Fast Imaging with Steady-State free precession (FIESTA), are the most frequently used. These sequences provide good resolution of fetal tissues.

- T1 weighed sequences (gradient dual echo, or fast multiplanar spoiled gradient) are primarily used to detect hemorrhage, calcification, lipomas, tubers, small calcified leukomalacia and myelination.

- Diffusion-weighted MRI (DWI) is useful in early detection of lesions leading to cerebral ischemia, and is also employed to assess brain maturation.

(Figure 2). on page 10

sequence parameters for fetal brain imaging:

- **Single Shot Fast Spin Echo T2 (SSFSE T2):** TR =1088ms, TE = minimum (90 ms), matrix = 256 x 256, FOV = 34 cm, Bandwith = 20,83 KHz. NEX =0,5. thickness/gap, mm= 3.0/0.0.

- **Fast Imaging with Steady-State free precession (FIESTA):** TR = 3,9 ms,TE = mínimo (1,7 ms), matrix = 256 x 256, FOV =35 cm, Bandwidth = 125 KHz. NEX = 2, espesor thickness/gap mm= 5.0/0.0. Flip angle (degrees): 45°.

- **3D Gradient-echo:** T1: TR = 8,5 ms, TE = minimum (2,4ms), matrix =336 x 256, FOV = 39 cm, Bandwith = 62,5 KHz, NEX = 0,69, thickness/gap mm = 6.0/0.0. Flip angle (degrees)= 12º.

- **Diffusion-weighted imaging:** TR = 2500 ms, TE = mínimo (69 ms), matrix = 128x128, FOV = 36 cm, Bandwith = 62-250 KHz, NEX = 6, thickness/gap mm= 4.0/0.0.mm, distancia entre cortes = 0 mm y b = 1000 s/mm².
EVALUATION OF NORMAL FETAL BRAIN DEVELOPMENT: WHAT TO LOOK FOR?

1. BIOMETRIC MEASUREMENTS.

2. GYRATION ANALYSIS.

3. MYELINATION ANALYSIS.

4. NEURONAL MIGRATION.

1. BIOMETRIC MEASUREMENTS.

• FETAL BRAIN VOLUME can be quickly estimated employing the following measurements:

  1. Fronto-occipital diameter (FOD).
  2. Cerebral biparietal diameter (BPDc).

• Differences between bone biparietal diameter (BPDb) and BPDc are proportional to the volume of pericerebral spaces.

• CORPUS CALLOSUM: Its length (LCC) can be measured on a sagittal plane, from the genu to the posterior extremity of the splenium.

• CEREBELLAR MEASUREMENTS:

  1. Vermis: anteroposterior diameter (vermis APD), height and surface area.
  2. Transverse cerebellar diameter (TCD). (Figure 3) on page 11, (Figure 4) on page 12, (Figure 5) on page 13, (Figure 6) on page 14, (Figure 7) on page 15, (Figure 8) on page 16

2. GYRATION ANALYSIS.

• At 20 gestational weeks (GW), only the interhemispheric fissure and lateral sulcus should be visible.

• At 25 GW, the interhemispheric fissure, lateral sulcus, internal parieto-occipital, hippocampal, as well as callosal, calcarene and cingulate sulci should already be present.

• At 27 GW, the interhemispheric fissure as well as the lateral sulcus, internal parieto-occipital, hippocampal, callosal, calcarene, cingulate and central sulci should already be present.
• At 29 GW, marginal, pre- and postcentral, intraparietal, collateral, superior temporal and frontal sulci should be visible, while the central sulcus should reach half of the cerebral hemisphere in depth.

• At 31 GW, the inferior frontal sulcus should be visible.

• Finally, at 35 GW, the temporal lobe should present all of its sulci, including superoanterior, inferior and external occipitotemporal sulci. At this gestational age, the gyration should have obtained its definitive pattern. (Figure 9) on page 17, (Figure 10) on page 18, (Figure 11) on page 19, (Figure 12) on page 20, (Figure 13) on page 21, (Figure 14) on page 22, (Figure 15) on page 23, (Figure 16) on page 24, (Figure 17), on page 25 (Figure 18) on page 26, (Figure 19 on page 27), (Figure 20) on page 28, (Figure 21) on page 29, (Figure 22) on page 30, (Figure 23) on page 31, (Figure 24) on page 32, (Figure 25) on page 33

3. MYELINATION ANALYSIS.

Myelination is a good indicator of fetal cerebral maturation. The increase in cholesterol and glycolipids accompanying the formation of myelin results in an increase in bound water, leading to a shortening of both T1 and T2 relaxation times. It manifests at MR as hyperintensity on T1-weighted images and hypointensity on T2-weighted images.

- Such signal changes can be depicted in the white matter, starting at 20 GW in the posterior brainstem.

- At 27 GW, some myelin is visible at the level of the vermis and middle cerebellar peduncles. A moderate signal is also visible at the central basal ganglia.

- Up to 33 GW, myelination of the posterior limbs of the internal capsules occurs, extending progressively to the globus pallidus at 35-36 GW. (Figure 26) on page 34, (Figure 27) on page 35, (Figure 28) on page 36, (Figure 29) on page 37

4. NEURONAL MIGRATION

• Neuronal migration is determined by the multilayered appearance of the cerebral mantle on T2-weighted images.

• Three layers (inner germinal matrix, intermediate migrating wave of neurons, and outer immature cortex) are observed at 16-28 GW.

• Two layers (inner white matter and outer cortex) are depicted at 34 GW. (Figure 30) on page 38, (Figure 31) on page 39
BRAIN AND SPINAL ABNORMALITIES

The main applications for fetal brain and spinal MRI are illustrated:

A) BRAIN:

• **Posterior fossa:** Dandy Walker malformation, vermian hypoplasia, mega cisterna magna, unilateral cerebellar damage, and Chiari II (Figure 32) on page 40, (Figure 33) on page 41, (Figure 34) on page 42, (Figure 35) on page 43, (Figure 36) on page 44, (Figure 37) on page 45, (Figure 38) on page 46, (Figure 39) on page 47, (Figure 40) on page 48

• **Midline:** Callosal agenesis, holoprosencephaly and cavum cyst. (Figure 41) on page 49, (Figure 42) on page 50, (Figure 43) on page 51, (Figure 44) on page 52, (Figure 45) on page 53, (Figure 46) on page 54

• **Ventricules:** ventriculomegaly and hydrocephalus. (Figure 47) on page 55

**Periventricular:** subependymal heterotopias, subependymal nodules in tuberous sclerosis and germinal matrix hemorrhage (Figure 48) on page 56, (Figure 49) on page 57, (Figure 50) on page 58, (Figure 51) on page 59

• **Cerebral parenchyma:** hemorrhagic-ischemic damage, congenital infection and fetal brain tumor. (Figure 52) on page 60, (Figure 53) on page 61, (Figure 54) on page 62

• **Cerebral surface:** lissencephaly, polymicrogyria, schizencephaly, hemimegalencephaly, heterotopias and laminar necrosis (Figure 55) on page 63, (Figure 56) on page 64, (Figure 57) on page 65, (Figure 58) on page 66, (Figure 59) on page 67

• **Pericerebral spaces:** subdural hematoma.

B) SPINE:

• **Sacrococcygeal teratoma.** (Figure 60) on page 68

• **Myelomeningocele.** (Figure 61) on page 69
Images for this section:

**IMAGING PROTOCOL**

- Patients should be scanned in the supine position. If this is not possible, as in advanced stages of pregnancy, the lateral decubitus position is preferred to avoid compression of the inferior vena cava.

- Fetal MR examination should start with a localizer in three planes along maternal axes, in order to further orientate the examination.

- All subsequent scans should be oriented along fetal axes.

**Fig. 1:** Figure 1
WHICH SEQUENCES?

- Fast T2-weighted sequences, such as Single Shot Fast Spin Echo T2 (SSFSE T2) and Fast Imaging with Steady-State free precession (FIESTA), are the most frequently used. These sequences provide good resolution of fetal tissues.

- T1 weighed sequences (gradient dual echo, or fast multiplanar spoiled gradient) are primarily used to detect hemorrhage, calcification, lipomas, tubers, small calcified leukomalacia and myelination.

- Diffusion-weighted MRI (DWI) is useful in early detection of lesions leading to cerebral ischemia, and is also employed to assess brain maturation.

Fig. 2: Figure 2
WHAT TO LOOK FOR?

1. BIOMETRIC MEASUREMENTS

- **FETAL BRAIN VOLUME** can be quickly estimated employing the following measurements:
  1. *Fronto-occipital diameter* (FOD).
  2. *Cerebral biparietal diameter* (BPDc).

  - Differences between *bone biparietal diameter* (BPDb) and BPDc are proportional to the volume of pericerebral spaces.

- **CORPUS CALLOSUM**: Its length (LCC) can be measured on a sagittal plane, from the genu to the posterior extremity of the splenium.

- **CEREBELLAR MEASUREMENTS**:
  1. Vermis: anteroposterior diameter (vermis APD), height and surface area.
  2. Transverse cerebellar diameter (TCD).

**Fig. 3:** Figure 3
Fig. 4: Figure 4

(A) Sagittal SSFSE T2, fronto-occipital diameter (FOD); (B) Sagittal FIESTA, length of corpus callosum (LCC); (C) Coronal FIESTA, cerebral biparietal Diameter (BPDc); (D) Coronal FIESTA, bone biparietal diameter (BPDb).
Fig. 5: Figure 5

(A) Sagittal SSFSE T2: vermis anteroposterior diameter; (B) Sagittal SSFSE T2: vermis height; (C) Sagittal SSFSE: vermis surface area; (D) Axial SSFSE T2: tranverse cerebellar diameter (DTC).
## BIOMETRIC MEASUREMENTS


<table>
<thead>
<tr>
<th>GA (weeks)</th>
<th>n</th>
<th>FOD (mm)</th>
<th>BPDc (mm)</th>
<th>BPDh (mm)</th>
<th>LCC (mm)</th>
<th>Vermis height (mm)</th>
<th>Vermis APD (mm)</th>
<th>Vermis surface area (mm²)</th>
<th>TCD (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>33</td>
<td>20</td>
<td>92–103</td>
<td>70–80</td>
<td>74–87</td>
<td>37–45</td>
<td>17–21</td>
<td>11–16</td>
<td>221–300</td>
<td>40–44</td>
</tr>
</tbody>
</table>

These are crude observed measurements without smoothing. APD, anteroposterior diameter; BPDc, cerebral biparietal diameter; BPDh, bone biparietal diameter; FOD, fronto-occipital diameter; GA, gestational age; LCC, length of corpus callosum; TCD, transverse cerebellar diameter.

**Fig. 6:** Figure 6
Fig. 7: Figure 7
**Vermis: BIOMETRIC MEASUREMENTS**

1. Lingula
2. Central lobule
3. Cuimen
4. Declive
5. Folium
6. Tuber
7. Pyramis
8. Uvula
9. Nodulus

---

**Table 2** Mean ± SD measurements obtained by volume contrast imaging in the coronal plane of the cerebellar vermis in 203 normal fetuses

<table>
<thead>
<tr>
<th>Gestational age (weeks)</th>
<th>Patients (n)</th>
<th>Craniocaudal diameter (mm)</th>
<th>Anteroposterior diameter (mm)</th>
<th>Surface area (cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>18–19</td>
<td>10</td>
<td>10.5 ± 1.3</td>
<td>8.3 ± 0.8</td>
<td>0.6 ± 0.05</td>
</tr>
<tr>
<td>20–21</td>
<td>19</td>
<td>12.7 ± 1.4</td>
<td>9.1 ± 1.6</td>
<td>0.7 ± 0.3</td>
</tr>
<tr>
<td>22–23</td>
<td>46</td>
<td>14.2 ± 1.6</td>
<td>10.5 ± 1.7</td>
<td>1.2 ± 0.2</td>
</tr>
<tr>
<td>24–25</td>
<td>45</td>
<td>15.8 ± 1.6</td>
<td>12 ± 1.4</td>
<td>1.5 ± 0.3</td>
</tr>
<tr>
<td>26–27</td>
<td>28</td>
<td>17.6 ± 1.7</td>
<td>13.5 ± 1.8</td>
<td>1.7 ± 0.3</td>
</tr>
<tr>
<td>28–29</td>
<td>19</td>
<td>19.6 ± 1.7</td>
<td>13.9 ± 1.1</td>
<td>2.1 ± 0.2</td>
</tr>
<tr>
<td>30–31</td>
<td>16</td>
<td>20.9 ± 1.5</td>
<td>15.5 ± 1.6</td>
<td>2.4 ± 0.06</td>
</tr>
<tr>
<td>32–33</td>
<td>20</td>
<td>22.8 ± 1.6</td>
<td>18.2 ± 1.7</td>
<td>3.4 ± 0.2</td>
</tr>
</tbody>
</table>

F. Viñals, 2005


---

**Fig. 8: Figure 8**
2. GIREATION ANALYSIS

- At 20 gestational weeks (GW), only the interhemispheric fissure and lateral sulcus should be visible.
- At 25 GW, the interhemispheric fissure, lateral sulcus, internal parieto-occipital, hippocampal, as well as callosal, calcarine and cingulate sulci should already be present.
- At 27 GW, the interhemispheric fissure as well as the lateral sulcus, internal parieto-occipital, hippocampal, callosal, calcarine, cingulate and central sulci should already be present.
- At 29 GW, marginal, pre- and postcentral, intraparietal, collateral, superior temporal and frontal sulci should be visible, while the central sulcus should reach half of the cerebral hemisphere in depth.
- At 31 GW, the inferior frontal sulcus should be visible.
- Finally, at 35 GW, the temporal lobe should present all of its sulci, including superoanterior, inferior and external occipitotemporal sulci. At this gestational age, the gyration should have obtained its definitive pattern.

Fig. 9: Figure 9
Fig. 10: Figure 10
At 20 GW, only the interhemispheric fissure (1), and lateral sulcus (2) should be visible.
At 20 GW, only the interhemispheric fissure (1) and lateral sulcus (2) should be visible.

**Fig. 12:** Figure 12
At 20 GW, only the interhemispheric fissure (1) and lateral sulcus (2) should be visible.

**Fig. 13:** Figure 13
At 25 GW, only the interhemispheric fissure (1), lateral sulcus (2), parietooccipital (3), calcarine (4), and hippocampic fissures (5) should be visible.
At 25 GW, only the interhemispheric fissure (1), lateral sulcus (2), parietooccipital (3), calcarine (4), and hippocampic fissures (5) should be visible.

Fig. 15: Figure 15
At 25 GW, only the interhemispheric fissure (1), lateral sulcus (2), parietooccipital (3), calcarine (4), and hippocampic fissures (5) should be visible.

**Fig. 16:** Figure 16
At 29 GW, marginal (6), pre (7), central (8) and postcentral (9), intraparietal, collateral (10) and superior temporal (11) and frontal sulci (12) should be visible and the central sulcus should reach half of the cerebral hemisphere in depth (1).

Fig. 17: Figure 17
At 29 GW, marginal (6), pre (7), central (8) and postcentral (9), intraparietal, collateral (10) and superior temporal (11) and frontal sulci (12) should be visible and the central sulcus should reach half of the cerebral hemisphere in depth (1).
At 29 GW, marginal (6), pre (7), central (8) and postcentral (9), intraparietal, collateral (10) and superior temporal (11) and frontal sulci (12) should be visible and the central sulcus should reach half of the cerebral hemisphere in depth (1).
At 31 GW, the inferior frontal sulcus should be visible.

**Fig. 20:** Figure 20
Fig. 21: Figure 21

At 31 GW, the inferior frontal sulcus should be visible.
At 31 GW, the inferior frontal sulcus (13) should be visible. Colateral sulcus (10).

Fig. 22: Figure 22
At 35 GW, the temporal lobe should have all of its sulci, including superoanterior, inferior and external occipitotemporal sulci. At this gestational age, the gyration should have obtained its definitive pattern.

Fig. 23: Figure 23
At 35 GW, the temporal lobe should have all of its sulci, including superoanterior, inferior and external occipitotemporal sulci. At this gestational age, the gyration should have obtained its definitive pattern.
At 35 GW, the temporal lobe should have all of its sulci, including superoanterior, inferior (14) and external occipitotemporal sulci (15). At this gestational age, the gyration should have obtained its definitive pattern.

Fig. 25: Figure 25
WHAT TO LOOK FOR?

3. MYELINATION ANALYSIS

- **Myelination** is a good indicator of fetal cerebral maturation.
- The increase in cholesterol and glycolipids accompanying the formation of myelin results in an increase in bound water, leading to a shortening of both T1 and T2 relaxation times. It manifests at MR as hyperintensity on T1-weighted images and hypointensity on T2-weighted images.
  - Such signal changes can be depicted in the white matter, starting at 20 GW in the posterior brainstem.
  - At 27 GW, some myelin is visible at the level of the vermis and middle cerebellar peduncles. A moderate signal is also visible at the central basal ganglia.
  - Up to 33 GW, myelination of the posterior limbs of the internal capsules occurs, extending progressively to the globus pallidus at 35–36 GW.

Fig. 26: Figure 26
Brainstem myelination in normal fetus at 25 weeks’ gestation. The myelination of the supratentorial brain has not begun. The myelination of pontine tegmentum (arrowheads) is blurred in SSFSE T2. FIESTA shows early brainstem myelination. The signal intensity of the pallidum and the thalamus is isointense to white matter (arrows).

**Fig. 27:** Figure 27
The signal intensity of the pallidum and the thalamus becomes low on T2-weighted images from 27 to 34 weeks (arrows).

**Fig. 28:** Figure 28
At 29 GW, some myelin is visible at the level of the superior cerebellar peduncles (blue arrow) and middle cerebellar peduncles (red arrow). The signal intensity of the pallidum and the thalamus becomes hiperintense on T1-weighted images (arrowheads) at GW 35.

Fig. 29: Figure 29
4. NEURONAL MIGRATION

- Neuronal migration is determined by the multilayered appearance of the cerebral mantle on T2-weighted images.

- Three layers (inner germinal matrix, intermediate migrating wave of neurons, and outer immature cortex) are observed at 16-28 GW.

- Two layers (inner white matter and outer cortex) are depicted at 34 GW.

Fig. 30: Figure 30
Fig. 31: Figure 31

(A, B) Coronal SSFSE T2; (C, D) Coronal FIESTA. Three layers: Inner germinal matrix (red arrows), intermediate migrating wave of neurons (red arrowheads), and outer immature cortex (blue arrows) at 20.4 weeks. Two layers: Inner white matter (white arrow) and outer cortex (white arrowhead) at 35 weeks.
Fig. 32: Figure 32

(A) Sagittal FIESTA; (B) Coronal SSFSE; (C) Axial SSFSE T2; (D) Axial FIESTA.
Megacisterna magna (arrows). GW: 34.
Posterior fossa: DANDY-WALKER

(A) and (B): Echography; (C) Sagittal SSFSE T2; (D) Sagittal FIESTA; (E) Axial SSFSE T2; (F) Axial FIESTA. Dandy-Walker with cerebellar hypoplasia (arrows) and hydrocephalus (arrowheads).

GW: 22 Evolution: Termination of pregnancy

Fig. 33: Figure 33
Fig. 34: Figure 34

(A) Sagittal SSFSE T2; (B) Coronal FIESTA; (C) Sagittal SSFSE T2; (D) Coronal SSFSE T2; (E) and (F) Axial SSFSE T2. Dandy-Walker with vermian hypoplasia and malrotation (red arrow), cerebellar hypoplasia (blue arrows) and hydrocephalus (red arrowheads). Agenesis of the corpus callosum (blue arrowheads). Left parietal cleft with pial-ependymal communication, suggesting open-lip schizencephaly (yellow arrow). Subependymal heterotopia protruding ventricular wall of the right frontal horn (yellow arrowhead) GW: 19.6
Fig. 35: Figure 35

(A) and (B) Axial FIESTA; (C) and (D) Coronal FIESTA. (E) Sagittal SSFSE T2. (F) Sagittal FIESTA. Dandy-Walker with vermian hypoplasia (red arrow) and cerebellar hypoplasia (red arrowhead). Cyst dilatation of 4th ventricle with enlargement of posterior fossa. Parcial agenesis of the corpus callosum (blue arrows). Genu and part of the body of corpus callosum connect the two frontal lobes (blue arrowheads). Right interhemispheric cyst (yellow arrow) communicates with the right lateral ventricle (yellow arrowhead). Brainstem hypoplasia (white arrow). GW: 28
(A) Coronal FIESTA; (B) Sagittal FIESTA, fetus 1; (C) Axial SSFSE T2, fetus 1; (D) Sagittal FIESTA, fetus 2; (E) Sagittal FIESTA, fetus 3; (F) Coronal FIESTA, fetus 3. GW: 25. Multiple pregnancy in a 42 year-old woman with OVODON IVF and transfer of 3 embryos. Two monochorionic diamniotic male fetuses (fetus 1 in cephalic presentation and fetus 3 in breech presentation) with vermian hypoplasia and malrotation (red arrows) and wide communication between the fourth ventricle and cisterna magna (red arrowheads). Female fetus with normal vermis (blue arrow) in transverse position.

**Fig. 36:** Figure 36
(A) and (B) Sagittal SSFSE T2; (C) Sagittal FIESTA; (D) and (E) Axial SSFSE T2; (F) Coronal SSFSE T2. GW: 28, 4. Vermian hypoplasia and malrotation (red arrows) and wide communication between the fourth ventricle and cisterna magna (red arrowheads).

Fig. 37: Figure 37
**Figure 38:** Figure 38

GW:22.2: (A) Sagittal SSFSE T2; (B) Axial SSFSE T2; (C, D) Coronal SSFSET2. Right cerebellar hemisphere hypoplasia (red arrows) and vermian hypoplasia (blue arrows).
Fig. 39: Figure 39
**Posterior fossa: CHIARI II WITH MYELOMENINGOCELE**

(A) Sagittal SSFSE; (B) Coronal SSFSE; (C) Axial SSFSE T2; (D) Axial SSFSE T2. **GW: 20.** Ventriculomegaly. Chiari II malformation (red arrow). Myelomeningocele (blue arrow). **Evolution:** Termination of pregnancy.

**Fig. 40:** Figure 40
Fig. 41: Figure 41

GW: 29. (A) Coronal FIESTA; (B) Axial SSSFSE T2, (C) Sagittal SSSFSE T2. Complete agenesis of the corpus callosum of the twin in cephalic presentation back-left (arrows) with colpocephaly (arrowhead). The other twin presented no alterations.
Fig. 42: Figure 42

Midline: AGENESIS OF THE CORPUS CALLOSUM

(A) Sagittal FIESTA; (B) Sagittal FIESTA; (C) Coronal FIESTA; (D) Coronal FIESTA; (E) Axial FIESTA; (F) Axial FIESTA. GW 25.6. Complete agenesis of the corpus callosum (arrows) with colpocephaly (arrowhead)
Midline: AGENESIS OF THE CORPUS CALLOSUM

GW: 22 weeks. (A) Echography; (B) Sagittal FIESTA; (C) Axial FIESTA; (D) Coronal FIESTA; (E) Sagittal SSFSE T2; (F) Axial SSFSE T2.

Evolution: Termination of pregnancy (TOP).

Fig. 43: Figure 43
Midline: AGENESIS OF CORPUS CALLOSUM IN MONOCHORIONIC DIAMNIOTIC TWINS WITH HISTORY OF CORRECTED TWIN-TWIN TRANSFUSION SYNDROME

(A) Axial SSFSE T2; (B) Axial Fiesta; (C) Sagittal SSFSE T2; (D) Sagittal FIESTA. Agenesis of corpus callosum with interhemispheric cyst (arrows) in one of the monochorionic twins.

GW: 25. Evolution: Vaginal delivery at 34 weeks. The interhemispheric cyst was operated 3 weeks after birth. A neuroenteric cyst was proved at biopsy.

Fig. 44: Figure 44
Case 1. (A) Sagittal SSFSE T2; (B) Axial SSFSE T2; (C) Axial SSFSE T2. **GW:** 27.2. Agenesis of the genu and the anterior 2/3 of the corpus callosum (red arrow in A). Anterior fusion of both frontal lobes (red arrowhead) and absence of frontal horns (blue arrow in B). (C) Normal right kidney. Multicystic dysplastic left kidney (blue arrowhead). **Case 2.** (D) Coronal SSFSE T2; (E) Axial SSFSE T2; (F) Axial SSFSE T2. **GW:** 21.4. Semilobar holoprosencephaly with fusion of thalami (yellow arrowhead) and frontal lobes (yellow arrows). Formation of temporal and occipital horns (white arrow). Rudimentary falx (white arrowhead).

**Fig. 45: Figure 45**
**Fig. 46:** Figure 46

GW: 30.2. (A) Sagittal FIESTA; (B) Sagittal SSFSE T2; (C) Coronal SSFSE T2; (D) Axial SSFSE T2. Cavum veli interpositi (arrows).
Fig. 47: Figure 47

(A) Sagittal SSFSE T2; (B) Axial SSFSE T2; (C) Sagittal FIESTA; (D) Axial FIESTA. GW: 21. Occipital meningocele (red arrows) with cerebellar hypoplasia. Hydrocephalus with a defect of the septum pellucidum (arrowhead). Uterus arcuatus (blue arrow) with uterine leiomyomas (white arrows). FIESTA sequence provides better evaluation of uterine pathology than SSFSE T2 does. Evolution: Termination of pregnancy.
Fig. 48: Figure 48

Periventricular: TUBEROUS SCLEROSIS WITH ASSOCIATED CARDIAC RHABDOMYOMA

(A) Sagittal SSFSE T2; (B) Coronal SSFSE T2; (C) Axial SSFSE T2; (D) Sagittal FIESTA; (E) Echo-doppler; (F) Axial FIESTA. Subependymal node near the right foramen of Monro (white arrows) with associated cardiac rhabdomyomas (white arrowheads).
Fig. 49: Figure 49

Periventricular: FRONTONASAL DYSPLASIA AND SUBEPENDYMAL HETEROTOPIAS

GW: 19.5 (A) Sagittal SSFSE T2. Subependymal heterotopia protruding left ventricular wall of the atrium (white arrow). (B) Sagittal SSFSE T2. Control at GW: 29 shows no changes (white arrow). (C) and (D): 3D SPGR VR reconstruction at postnatal control. The presence of a left perialtrial heterotopia is confirmed, as well as several periventricular heterotopias (arrows in D). (E) Prenatal 3D surface reconstruction ultrasound; (F) Postnatal photograph. Frontonasal dysplasia.
Fig. 50: Figure 50

(A) and (B) Sagittal Gradient Echo T1; (C) and (D) Diffusion weighted images. High signal intensity without acute infarction (arrowhead). (E) Axial SSFSE T2; (F) Coronal SSFSE 2; (G) Axial FIESTA; (H) Coronal FIESTA. Frontal subacute hemorrhagic infarction (red arrows) and intraventricular haemorrhage (blue arrows). GW: 35. Evolution: Term neonate with motor sequelae.
(A) and (B) Sagittal Gradient-Echo T1; (C) Axial Gradient-Echo T1; (D) Sagittal SSFSE T2; (E) Axial SSFSE T2; (F) Coronal SSFSE T2; (G) and (H) Diffusion weighted images. Frontal subacute hemorrhagic infarction (red arrow), germinal matrix haemorrhage (arrowhead) and intraventricular haemorrhage (blue arrows). GW:27. Evolution: Term neonate with motor sequelae.
(A) Sagittal Gradient-Echo T1; (B) and (C) Axial SSFSE T2; (D) Axial FIESTA; (E) Coronal SSFSE T2; (F) Sagittal FIESTA. Left fronto-parieto-occipital subacute infarction (red arrows), and intraventricular haemorrhage (blue arrows). (G) and (H) Diffusion weighted images. Subacute infarction with mild increase in signal intensity (arrowheads)

GW:35,3.

Fig. 52: Figure 52
Cerebral parenchyma: INJURIES PARENCHYMAL. ISCHEMIC LESION OR CONGENITAL INFECTION? (1)

(A) and (B) Axial SSFSE T2; (C) and (D) Coronal SSFSE T2; (E) and (F) Sagittal SSFSE T2. There is a large area of encephalomalacia (red arrow) involving the left parietal lobe. Right parietal lobe presented no alterations (blue arrow). GW:20. Evolution: TOP

Fig. 53: Figure 53
Fig. 54: 

(A) and (B) Coronal FIESTA (C) Coronal SSFSE T2 and (D) Gradient-Echo T1. Varus deformity of both feet with right leg in hyperextension (red arrow) and left leg in flexion (blue arrow). Dilated stomach and duodenum to the ligament of Treitz (yellow arrow). Hepatic calcifications (red arrowhead). GW:20. Evolution: TOP
**GW: 25** (A-B) Coronal SSFSE T2; (C) Axial SSFSE T2; (D) Sagittal SSFSE T2. Right insular cleft (white arrows) with pial-ependymal communication, suggesting open-lip schizencephaly (type II). A thin septum is seen, probably corresponding to ventricular wall (black arrow in B). E) Coronal FIR; (F) Axial 3D FSPGR reconstruction. MRI was performed at the age of 6 because the patient suffered from seizures since the age of 5 (controlled with valproic acid) and a mild left brachial spastic monoparesis. The open-lip schizencephaly has evolved into closed-lip schizencephaly (arrow in D) (opercularization occurs between days 22-38). Polymicrogyria is present in the cleft (blue arrow) with subependymal heterotopias in inferior and posterior locations (yellow arrow).

**Fig. 55:** Figure 55
**Fig. 56**: Figure 56

(A) Sagittal FIESTA; (B) Axial FIESTA; (C) Sagittal SSFSE T2; (D) Axial SSFSE T2. **GW: 23.** Severe hydrocephalus. Calcarine and Silvio fissures and parieto-occipital sulci are not visible. Agenesis of the corpus callosum (arrow). Male fetus.
Cerebral surface: LISSENCEPHALY TYPE 1. Third pregnancy

(A) Sagittal SSFSE T2; (B) Axial SSFSE T2; (C) Coronal SSFSE T2; (D) Coronal FIESTA. GW: 21.1. Severe hydrocephalus. Calcarine and Silvio fissures and parieto-occipital sulci are not visible. Agenesis of the corpus callosum (arrow). Male fetus.

Fig. 57: Figure 57
Fig. 58: Figure 58

(A), (B) and (C) Axial SSFSE T2; (D) and (E) Coronal SSFSE T2. (F) SSFSE T2 GW:21.5. Severe ventriculomegaly. Calcarine and Silvio fissures and parieto-occipital sulci are not visible. Male fetus.
Cerebral surface: POLYMICROGYRIA ASSOCIATED WITH AGENESIS OF THE CORPUS CALLOSUM

(A) and (B) Axial SSFSE T2; (C) and (D) Coronal SSFSE T2; (E) and (F) Sagittal SSFSE T2. Agenesis of the corpus callosum (red arrow) and trigonum (arrowhead). Bilateral generalized polymicrogyria (blue arrow). GW: 37.5

**Fig. 59:** Figure 59
Fig. 60: Figure 60

(A) Sagittal SSFSE T2; (B) Echo Doppler; (C) 3D surface reconstruction ultrasound.; (D) Sagittal Fiesta. GW: 20. Type I sacrococcygeal teratoma (arrow), presenting as a predominantly exophytic lesion covered with skin. Note the presence of a minimal presacral component. The patient underwent surgery 48 hours after birth. (E) Pre-surgery photograph of teratoma. (F) Post-surgery photograph.
Fig. 61: Figure 61
Fig. 62: Figure 62

(A) Coronal SSFSE; (B) Axial SSFSE; (C) Sagittal FIESTA. GW: 26.5
Chiari II malformation (red arrow). Myeloschisis (blue arrow).
Ventriculomegaly (arrowhead).
Conclusion

- Fetal MRI is a powerful technique used to evaluate the fetal brain, and is also a valuable complement to prenatal sonography:
  - Fetal MRI has higher contrast resolution than prenatal sonography and therefore, allows better differentiation between normal and abnormal tissues.
  - Structural abnormalities such as cerebral malformations and destructive lesions can be sonographically occult on prenatal sonography yet detectable by fetal MR imaging.
  - Moreover, fetal MRI is not susceptible to many of the limitations of sonography.
- This imaging modality is becoming an increasingly important tool in the evaluation of fetuses who have abnormalities suspected on the basis of family history or fetal sonography. With continuing improvements in technology, this will continue to be a rapidly growing field in future years.

Personal Information

Dr. Manuel Recio Rodríguez.
Hospital Quirón Madrid.
Mail: mrecio.mad@quiron.es

References