MRI in the evaluation of fetal thoracic pathology

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Aims and objectives

Prenatal diagnosis aims to obtain genetic, anatomic, biochemical, and physiological information about the fetus to detect any abnormalities that could have repercussions during gestation or after birth so that families can be provided with information about the suspected abnormality that will enable them to evaluate the therapeutic alternatives during and/or after birth and the risk of recurrence of the abnormality detected.

Advances in both fetal ultrasonography (US) and magnetic resonance imaging (MRI) enable fetal anomalies to be increasingly detected before birth. This is important because it makes it possible to evaluate the viability of the fetus, foresee possible problems in the delivery, and plan treatment after birth.

Postnatal survival is highly dependent on adequate prenatal lung development. Thoracic anomalies comprise a large and varied group of fetal abnormalities that make it necessary for radiologists to recognize the characteristics of the different intrathoracic processes in fetal development.

Apart from showing the MRI appearance of fetal thoracic anomalies, this poster aims to assess the usefulness of MRI in these anomalies. To this end, we compare the MRI and US findings to determine the impact of MRI in the diagnosis and management of each case. Whenever possible, patients with anomalies suspected prenatally were followed up clinically and radiologically. When abortions were performed, all suspected anomalies were contrasted with the pathology findings.
Methods and materials

Our hospital's department of obstetrics and gynecology is the reference center for a population of about 300,000 inhabitants, so it receives patients with known or suspected fetal anomalies from other centers. The department does about 3500 US studies every year. In the early 1990s, a prenatal diagnosis workgroup was created, involving gynecologists, neonatologists, pediatricians, geneticists, biochemists, psychologists, pediatric surgeons, pathologists, and radiologists. This workgroup analyzes the anomalies that are detected and decides on the best way to study them and manage them.

Since 1997, a total of 989 pregnant women with 1030 fetuses have undergone MRI at our center. All MRI studies were done because anomalies had been suspected on the basis of clinical and/or US findings or because of the risk of hereditary anomalies. In these fetal MRI studies, 126 thoracic anomalies were detected in 99 fetuses.

Most fetal MRI studies were done at about 22 weeks' gestation as this is the point in the pregnancy when US studies are done to detect morphological anomalies.

MRI TECHNIQUE:

None of the patients or fetuses underwent any special preparations. All MRI studies were done using multiple-element body coils on a 1.5T scanner. We acquired images using SS-HF-RARE or HASTE sequences; occasionally, we also obtained single colometric images with SS-RARE sequences and T1-weighted fast gradient-echo images. The entire MRI examination took about 20 minutes.

NORMAL FETAL CHEST ANATOMY ON MRI:

On MRI the lungs are depicted superbly: they are hyperintense on T2-weighted sequences, more so than the surrounding fetal organs and somewhat less so than amniotic fluid (Fig. 1 on page 10). The trachea can be identified, even early in gestation, as a hyperintense tubular structure (Fig. 2 on page 10).

The heart is generally identified as a rounded, low-signal-intensity structure in T2-weighted sequences. The chambers and the septum of the fetal heart cannot be seen on MRI. The vascular structures, when they are seen, are hypointense. It is nearly always possible to see part of aorta. The pulmonary arteries, the vena cava, and the other vascular structures can be identified later in gestation, but little information is obtained about these structures. The esophagus is not usually seen unless it contains fluid.
The chest wall is clearly identified in any spatial plane. The information about osseous structures is poor.

1. BRONCHOPULMONARY ANOMALIES:

PULMONARY UNDERDEVELOPMENT:

Pulmonary underdevelopment is classified into three categories (Table 1 on page 11). Unilateral pulmonary agenesis is a rare anomaly that consists of the absence of lung parenchyma, bronchi, and the pulmonary artery on the affected side. More than 50% of the fetuses with this anomaly have associated anomalies in the cardiovascular, gastrointestinal, genitourinary, and/or musculoskeletal system.

The imaging findings in lung aplasia and lung agenesis are similar, except for the presence of a blind-ended bronchus in aplasia. The affected hemithorax is smaller, the mediastinum is displaced, and the ipsilateral diaphragm is elevated.

In pulmonary hypoplasia, the pulmonary artery and the bronchi are present but underdeveloped. Pulmonary hypoplasia can be primary (very uncommon) or secondary, usually to a space-occupying process either inside or outside the chest. The most common space-occupying process inside the chest is a diaphragmatic hernia, although there are other causes, such as congenital pulmonary airway malformations (CPAM), bronchopulmonary sequestration, mediastinal mass, cardiomegaly, or lymphatic or diaphragmatic malformations. These anomalies are discussed individually below.

The most common extrathoracic cause of pulmonary hypoplasia is severe oligohydramnios secondary to congenital anomalies of the genitourinary tract (renal agenesis, cystic renal dysplasia, urinary tract obstruction) (Fig. 3 on page 12, Fig. 4 on page 13 and Fig. 5 on page 14). Other causes of pulmonary hypoplasia include skeletal dysplasia, osteogenesis imperfecta (Fig. 6 on page 15), and severe scoliosis (Fig. 7 on page 16 and Fig. 8 on page 17).

CONGENITAL DIAPHRAGMATIC HERNIA (CDH):

This condition affects 1/2500 to 1/5000 newborn babies. Most CDH are located in the left hemithorax (Fig. 9 on page 18). In 3% of cases, the lesion is bilateral. About 40% of patients with CDH have other congenital malformations. CDH results from the failure of the pleuroperitoneal canals to close during embryological development. The size of the diaphragmatic defect varies and determines the size of the hernia. Sometimes, one
of the two diaphragms is absent. The contents of the hernia can include a combination of intestine, stomach, liver, or other abdominal viscera (Fig. 10 on page 19). When the viscera herniate during early lung development, it results in bilateral pulmonary hypoplasia.

Large hernias early in gestation, right-sided hernias (Fig. 11 on page 20 and Fig. 12 on page 21), and small hernias can be difficult to diagnose by US because the echogenicity of the lungs, diaphragms, liver, spleen, and bowel loops is similar and only the stomach and chambers of the heart have different echogenicity. The diagnosis of CDH is often reached through indirect signs, such as displacement of the heart or the presence of stomach in the thoracic cavity.

When the heart is displaced toward the left (right-sided hernias) or the stomach does not participate in the hernia (Fig. 13 on page 22), it can be difficult to diagnose CDH by US. By contrast, even early in gestation, MRI can diagnose CDH because the different thoracoabdominal organs have very different signal intensities and it is easy to distinguish between them; thus, the diagnosis is easy.

The prognosis of CDH depends on the degree of pulmonary hypoplasia. Delays in the histologic development of the lung and abnormalities in the pulmonary arteries (smooth muscle hypertrophy of the medial wall) also cause pulmonary hypertension of the newborn (persistent fetal circulation). This is a problem, because the hernia can be repaired but pulmonary hypoplasia and vascular abnormalities cannot. The prognosis is worse when the liver or its left lobe is herniated because it results in mechanical problems and comprised fetal circulation through the ductus venosus.

Treatment options include prenatal intervention, surgery after birth, and abortion.

**CONGENITAL PULMONARY AIRWAY MALFORMATION (CPAM):**

CPAM is the pulmonary malformation that is most frequently diagnosed prenatally, accounting for 30% to 40% of all congenital diseases. It is characterized by abnormal branching of the immature bronchi and a lack of normal alveolar development. CPAM is caused by an unknown alteration in embryonic development during the first 10 weeks of gestation. Some theories relate CPAM with bronchopulmonary sequestration, lobar emphysema, and bronchial atresia; in fact, all or some of these conditions are sometimes seen in combination in a single fetus.

Because CPAM generally communicates with the normal tracheobronchial tree, the malformation can contain both cystic and solid components, giving rise to diverse
radiologic manifestations. Although CPAM usually affects a single lobe of the lung, it can affect an entire lung, or even, more rarely, both lungs. Five main subtypes of CPAM have been established in function of their histologic characteristics and size, although only three types (I, II, and III) can be distinguished by imaging techniques (Table 2 on page 23).

On US CPAM is seen as a solid, cystic, or mixed solid-cystic mass, depending on the type of CPAM.

Fetal MRI can help in both the evaluation of the lesion and in the evaluation of the development of the rest of the lung. CPAM is seen as a hyperintense lesion on T2-weighted MRI sequences (Fig. 14 on page 24 and Fig. 15 on page 25).

It can be difficult to distinguish CPAM from pulmonary sequestration, and it is important to remember that there are hybrid lesions combining the two anomalies. The differential diagnosis must also include congenital diaphragmatic hernia, neurenteric cyst, lobar emphysema, and bronchial atresia.

The natural history and prognosis of prenatally diagnosed CPAM vary, but 97% of affected fetuses survive. The most important prognostic factor is the size of the mass itself and the size of the cysts inside the mass: the larger the mass, the greater the resulting pulmonary hypoplasia. Other prognostic factors include mediastinal displacement and the presence of other fetal malformations. The presence of hydrops fetalis is an indicator of poor prognosis, and abortion is usually recommended in these cases. In babies with compromised lungs, the mass should be surgically excised as soon as the patient is stable. In many cases of CPAM, however, the malformation progressively shrinks during the third trimester and the mediastinal displacement resolves, becoming invisible in postnatal chest X-rays (Fig. 16 on page 26). These patients will be asymptomatic at birth; nevertheless, CT studies are essential to confirm the presence of the lesion and plan treatment.

BRONCHIAL ATRESIA:

This is an uncommon anomaly in which a segment of the bronchus does not communicate with the central airway, resulting in secondary dysplastic changes in the distal lung parenchyma (similar to those seen in CPAM). Although the upper lobes are the most commonly affected, bronchial atresia can occur in other locations. The peripheral bronchi are not seen on MRI, but we can suspect this anomaly when we observe hyperintense areas in the lungs in T2-weighted images (Fig. 17 on page 27 and Fig. 18 on page 28). The differential diagnosis includes other lung anomalies like CPAM and pulmonary sequestration.
After birth, patients are usually asymptomatic. Surgical treatment during childhood leads to an excellent prognosis.

**BRONCHOGENIC CYST:**

These are congenital lesions caused by abnormal budding in the ventral foregut from which the tracheobronchial tree develops, probably between the 26th and 40th days of embryogenesis. Most are located in the mediastinum, generally near the carina; less frequently they are located in the lung parenchyma, pleura, or diaphragm. Bronchogenic cysts have thin walls, covered with respiratory epithelium, and can contain mucin.

Prenatal US shows fluid-filled unilocular cysts.

On T2-weighted MRI sequences, the cysts are hyperintense.

The differential diagnosis includes esophageal duplication cysts and neurenteric cysts.

Most bronchogenic cysts are asymptomatic; when they do cause symptoms (wheezing, stridor, dyspnea, or dysphagia), it is due to compression of neighboring structures. The treatment is usually surgical.

**PULMONARY SEQUESTRATION.**

This anomaly consists of normal lung tissue separated from the tracheobronchial tree that receives its blood supply from the systemic circulation. Sequestration can be intralobar or extralobar. Intralobar sequestrations are located within the visceral pleura of the normal adjacent lung parenchyma, whereas extralobar sequestrations are located outside the normal lung and lined with their own pleural membrane. Intralobar and extralobar sequestration also differ in their venous drainage: intralobar sequestrations nearly always drain into the pulmonary veins, whereas extralobar sequestrations drain into systemic veins. However, this difference rarely helps differentiate between them in prenatal imaging (Fig. 19 on page 29 and Fig. 20 on page 30). In contrast to the findings reported in adults, most sequestrations detected in fetuses are extralobar. Most are situated in the base of the left lung. Sequestrations can be any size, but they are usually not very large (Fig. 21 on page 31).

On US they are seen as homogeneous or heterogeneous hyperechogenic masses, usually triangular. On MRI and in T2-weighted sequences, they are hyperintense. They are rarely accompanied by hydrothorax or hydrops. They can be associated with other anomalies such as diaphragmatic hernia or intestinal duplications (Fig. 19).
They can become smaller or even disappear during gestation and after birth (Fig. 22 on page 32 and Fig. 23 on page 33). The prognosis is usually favorable. About 10% of extralobar sequestrations are situated below the diaphragm, and in these cases it is important to differentiate them from neuroblastomas (Fig. 24 on page 34).

2. MEDIASTINAL ANOMALIES:

ESOPHAGEAL ATRESIA:

Esophageal atresia arises from the incomplete division of the foregut into the ventral respiratory portion and the dorsal digestive portion by the tracheoesophageal septum. It is the most common congenital malformation of the esophagus; its estimated incidence is 2 to 12 cases in every 10,000 live births.

The most common findings on MRI are polyhydramnios and an absent or very small stomach. Sometimes dilation of the proximal segment of the esophagus can be seen as a hyperintense structure in T2-weighted sequences (Fig. 25 on page 35), especially in sagittal images acquired in the midline of the fetal chest, but not always.

CERVICOTHORACIC CYSTIC HYGROMA:

This is a congenital malformation of the lymph vessels. One of the most common locations is the neck, and the lesion can extend to the thoracic cavity (Fig. 26 on page 36).

On MRI it is seen as a cystic lesion, hyperintense in T2-weighted sequences, and it is often multiloculated. MRI is useful for evaluating both the extension of the lesion toward the chest and the degree of compromise of the upper airway.

3. OTHER PULMONARY ANOMALIES:

HYDROTHORAX:

The accumulation of fluid in the pleural space is always pathological. The most important thing is to determine whether the hydrothorax is isolated (usually primary chylothorax (Fig. 27 on page 37) or forms part of hydrops fetalis (Fig. 28 on page 38), as the treatment and prognosis differ. Generally, unilateral cases are chylothorax, whereas bilateral cases form part of hydrops. Occasionally, chylothorax can be the cause of the hydrops.
Chylothorax is normally associated with an alteration in the thoracic duct. It can be isolated or associated with Down syndrome, Turner syndrome, pulmonary sequestration, diaphragmatic hernia, or pulmonary lymphangiectasia.

Hydrops fetalis can be have an immune or non-immune cause. Today, the most common type is non-immune, with a wide variety of causes (heart disease, infection, genetic and metabolic diseases, gastrointestinal tract or genitourinary anomalies, and skeletal dysplasias, among others).

On US, fluid, generally anechoic, is detected in the pleural space.

On MRI, hyperintense fluid surrounds the lungs.

The degree of pulmonary hypoplasia will depend on the point in gestation when the hydrothorax develops, on its volume, and on its duration. The prognosis depends on the cause of the hydrothorax; cases with isolated hydrothorax (chylothorax) have a good prognosis; those in which hydrothorax forms part of hydrops fetalis have a poor prognosis.

AXILLARY AND CHEST WALL CYSTIC HYGROMA: already discussed in "Cervicothoracic cystic hygroma" (Fig. 29 on page 39 and Fig. 30 on page 40).
**Fig. 1:** Normal fetal anatomy. (a) Axial, (b) sagittal, and (c) coronal images of the fetal chest. The lungs are hyperintense on T2-weighted sequences (asterisk); the heart is identified as a hypointense unit (H). The arrow head in (a) shows the pulmonary veins, and the arrow in (c) the pulmonary arteries. The arrow in (b) points to the thymus and the arrow head the left diaphragm.

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**Fig. 2:** Normal fetal anatomy. (a) Axial image of the lower part of the thorax. Note the quality of tissue discrimination among the different organs of the thorax and abdomen: liver (L) and stomach (S). The inferior vena cava (white arrow), the esophagus (orange arrow), and the thoracic aorta (red arrow). (b) Coronal image shows the trachea (arrow), always identifiable as a hyperintense tubular structure. (c) Sagittal image of the thorax. The esophagus (arrow) is not usually seen unless image acquisition coincides with fetal deglution or the esophagus is pathologically dilated. The thymus (t) is easily identified occupying the upper anterior portion of the mediastinum.

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### Classification of Pulmonary Underdevelopment

<table>
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<tr>
<th>Category</th>
<th>Description</th>
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<tr>
<td><strong>Pulmonary agenesis</strong></td>
<td>Complete absence of the lung parenchyma, bronchus, and pulmonary vasculature.</td>
</tr>
<tr>
<td><strong>Pulmonary aplasia</strong></td>
<td>Blind-ending rudimentary bronchus is present, without lung parenchyma or pulmonary vasculature.</td>
</tr>
<tr>
<td><strong>Pulmonary hypoplasia</strong></td>
<td>Bronchus and rudimentary lung are present; however, the airways, alveoli, and pulmonary vessels are decreased in size and number.</td>
</tr>
</tbody>
</table>

**Table 1**: Classification of pulmonary underdevelopment.

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Fig. 3: Bilateral renal agenesis. Fetus at 22 weeks’ gestation. (a) Axial, (b) coronal and (c) sagittal images of the fetal abdomen. The kidneys are absent (white arrows); there is no amniotic fluid, and the lungs are hypoplastic (black arrows).

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Fig. 4: Autosomal recessive polycystic kidney disease. Fetus at 24 weeks’ gestation. (a) Fetal coronal image showing the renal anomaly, severe oligohydramnios, and pulmonary hypoplasia (arrows). (b) Anatomic specimen shows these anomalies.

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Fig. 5: Obstruction of the lower urinary tract. Fetus at 17 weeks' gestation. (a) Fetal coronal image shows the pulmonary hypoplasia (arrow) resulting from oligohydramnios caused by obstruction of the lower urinary tract due to urethral atresia; the bladder is enlarged (asterisk).

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Fig. 6: Imperfect osteogenesis type II A. Fetus at 19 weeks' gestation. (a and b) Fetal coronal images. There may be moderate pulmonary hypoplasia. In (a) a small amount of hydrothorax is seen at the right base (arrow) and ascites is present. Fractured ribs can be inferred in (b). Agenesis of the corpus callosum, unsuspected prior to MRI, was also detected (circle in b). (c and d) radiographs and anatomic specimen of this fetus showing the multiple rib fractures.

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**Fig. 7:** Amniotic band syndrome. Fetus at 21 weeks’ gestation. (a and b) Fetal coronal view. Observe the severe thoracic deformity and marked scoliosis, as well as pulmonary hypoplasia. The head is hyperextended and the limbs are flexed. (c) Fetal radiograph showing these anomalies.

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Fig. 8: Pulmonary hypoplasia due to thoracic deformity and diaphragmatic eventration. Fetus at 21 weeks' gestation. (a, b, and c) Fetal axial, coronal, and sagittal images. The lungs are very small and the chest is deformed. Note the position of the spinal canal (red arrow in a), which is not centered in the circumference of the thoracic curve. The left hemithorax is very small. The liver (white arrows in a, b, and c) is very highly located. The thoracic deformity, the diaphragmatic eventration and the pulmonary hypoplasia are shown in the radiograph (d) and anatomic specimen (e).

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**Fig. 9:** Left diaphragmatic hernia. Fetus at 22 weeks’ gestation. This fetus was easily diagnosed at US because the stomach is located within the thoracic cavity. (a, b, and c) Fetal axial, sagittal, and coronal images show rightward displacement of the heart and the stomach within the thoracic cavity (s); bowel loops are also depicted (black arrow in c). The left lung cannot be seen and the right lung is very hypoplastic (white arrow in a). (d) Anatomic specimen of this fetus shows all of the findings described at MRI.

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**Fig. 10:** Left diaphragmatic hernia. Fetus at 22 weeks’ gestation. (a and b) Fetal coronal images show the left liver lobe located within the thoracic cavity as a hypointense structure on T2-weighted sequences. This sign indicates poor prognosis due to possible "distortion" with compression of the ductus venous and vascular compromise (black arrows). Bowel loops are also depicted (white arrow). (c) Axial image shows marked pulmonary hypoplasia and important bilateral hydrothorax (asterisk) are shown. (d and e) Anatomic specimen of this fetus shows all of the findings described at MRI.

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**Fig. 11:** Right diaphragmatic hernia. Fetus at 28 weeks' gestation. Although the entire right hemithorax is filled with the liver and bowel loops, US was unable to detect indirect signs of this hernia until 28 weeks. Leftward heart displacement is more difficult to assess, and the stomach was in its correct position. (a and b) Sagittal images show the bowel loops (white arrow) and the liver (L) in the thoracic cavity. The stomach (s) is in its correct position. (c and d) Coronal images show bowel loops (white arrow), the liver (L), and the heart displaced to the left (H). Note the pulmonary hypoplasia (red arrow). (e) T1-weighted coronal image shows that the large bowel (black arrow) is also herniated.

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Fig. 12: Right diaphragmatic hernia. Fetus at 22 weeks’ gestation. (a) Coronal images: although the entire right hemithorax is filled with the liver (L) and also a small amount of hydrothorax is seen (white arrow). There was leftward heart displacement, and the stomach was in its correct position (s). Note the pulmonary hypoplasia (black arrow). (b) Anatomic specimen of this fetus shows all of the findings described at MRI.

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**Fig. 13:** Left diaphragmatic hernia. Fetus at 21 weeks' gestation, US can only detect rightward mediastinal displacement but it cannot determine its cause. (a, b and c) Coronal and sagittal MRI images of the fetus clearly showing the left diaphragmatic hernia: the stomach (s) seemed to be placed in the correct position. Bowel loops (arrow) are displaced within the thoracic cavity. (d) Anatomic specimen of this fetus shows all of the findings described at MRI, finally stomach was also displaced within thoracic cavity.

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<tr>
<th>Types of CPAM</th>
<th>Characteristics</th>
<th>MRI appearance</th>
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<tbody>
<tr>
<td>Type 0</td>
<td>Severe acinar dysgenesis and severe airway dysplasia.</td>
<td>None; the condition causes death early in gestation.</td>
</tr>
<tr>
<td>Type I</td>
<td>Single or multiple large (3 cm – 10 cm) cysts surrounded by thin walls; hyperintense in T2-weighted small cysts and collapsed parenchyma.</td>
<td>Uni- or multilocular lesions with thin walls; hyperintense in T2-weighted sequences.</td>
</tr>
<tr>
<td>Type II</td>
<td>Multiple small (0.5 cm – 2 cm) cysts.</td>
<td>Appearance varies with composition.</td>
</tr>
<tr>
<td>Type III (adenomatoide)</td>
<td>Microcysts—rarely larger than 0.2 cm.</td>
<td>Homogeneously hyperintense solid masses surrounded by normal parenchyma.</td>
</tr>
<tr>
<td>Type IV</td>
<td>Disorders in acinar development at the distal level.</td>
<td>Large cysts full of air or fluid—indistinguishable from type I pleuropulmonary blastoma.</td>
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**Table 2:** CPAM classification.

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Fig. 14: CPAM type II. Fetus at 20 weeks’ gestation. (a, b, c and d) Fetal axial, sagittal and coronal images show an hyperintense septated, polylobulated structure occupies a large part of the right hemithorax. A small amount of pericardial effusion (white arrow) and left hydrothorax (black arrow) are seen. (e) Anatomic piece shows the lesion described at MRI.

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Fig. 15: CPAM type I. Fetus at 22 weeks’ gestation. (a, b, and c) Fetal coronal, sagittal and axial images: various hyperintense lobulated lesions on the lower right hemithorax (arrows). (d) CT after birth shows a big cystic lesion on the lower right lung. (e) Anatomic piece shows the lesion described at MRI.

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Fig. 16: CPAM type II. Fetus at 24 weeks' gestation. (a, b, and c) Fetal coronal, sagittal, and axial images: an hyperintense septated, polylobulated structure occupies a large part of the right hemithorax (arrows). (d) Normal plain film chest radiography after birth. (e) CT after birth shows this anomaly; the lesion appears to have decreased in size.

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**Fig. 17:** Bronchial atresia. Fetus at 22’ weeks gestation. (a,b and c) Fetal axial, coronal and sagittal images: an hyperintense triangular lesion (arrow) can be seen in the middle and upper right lung lobes. (d) Plain-film chest radiograph after birth shows air trapping in right upper lobe (circle). (e, f and g) CT axial, coronal and sagittal images after birth show an emphysematous area (black arrows) in the upper right lung lobe.

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**Fig. 18**: Bronchial atresia. Fetus at 22' and 30' weeks gestation. (a and b) Fetal coronal images at 22' and 30' weeks gestation show an hyperintense triangular lesion (white arrows) in the upper left lobe. There is also rightward mediastinal displacement. (d) Plain-film chest radiograph after birth shows air trapping in left upper lobe and rightward mediastinal displacement (black arrow).

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Fig. 19: Extralobar sequestration and intestinal duplication. Fetus at 23 weeks’ gestation. (a) Fetal sagittal image. The black arrows point to gastric duplication, and the white arrows show the pulmonary sequestration. The first US examination only detected intestinal duplication. (b, c and d) After birth CT axial images show the pulmonary sequestration in the lower left lobe (white arrow), the gastric duplication (black arrow) and the vein drainage into the suprahepatic veins (arrowhead).

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**Fig. 20:** Extrapulmonary sequestration. Fetus at 23 weeks' gestation. (a, b and c) Fetal coronal, sagittal and axial images. A triangular-shaped structure (white arrows), of greater intensity than the rest of the lung parenchyma is seen in the base of the left lung. A hypointense linear structure (arrowhead) arises from the aorta and courses toward the lesion. (d) CT performed after birth the lesion and the vessel that feeds the lesion arising from the aorta (arrowhead).

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Fig. 21: Extrapulmonary sequestration. Fetus at 23 weeks’ gestation. (a and b) Fetal sagittal and coronal images. A triangular-shaped structure (white arrows), of greater intensity than the rest of the lung parenchyma is seen in the base of the right lung. (c and d) Plain film chest x-ray after birth, lateral view shows pulmonary condensation in the right base (black arrow).

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**Fig. 22:** Extralobar sequestration. Fetus at 21 weeks’ gestation. (a, b, and c) Fetal sagittal, axial and coronal images show an hyperintense lesion (arrows) in the base of the left lung. (d and e) Plain film chest x-ray after birth shows the lesion (arrow). (f) CT performed after birth shows a significant decrease in the size of the (arrow). Although asymptomatic, the patient underwent surgery and the diagnosis was confirmed.
Fig. 23: Extralobar sequestration. Fetus at 21 and 38 weeks’ gestation. (a and b) Fetal coronal and axial images at 21 weeks' gestation show a large hyperintense lesion (arrows) in the left lung, associated to rightward heart displacement. (c an d) Fetal coronal and axial images at 38 weeks' gestation show a significant decrease in the size of the hyperintense left lung lesion (arrows). (e) CT performed after birth shows shows the lesion described at MRI (arrow).

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**Fig. 24:** Infradiaphragmatic extralobar pulmonary sequestration. Fetus at 23 weeks' gestation. (a, b, and c) Fetal axial, coronal and sagittal images. A heterogeneous mass (black arrows) is identified in the left abdomen between the diaphragm and the stomach (s) above the kidney. This mass is shown on all imaging studies after birth (d). The adrenal gland is normal (arrow in US) and the metaiodobenzylguanidine study was negative. The infant is asymptomatic and laboratory test values were normal. Although an anomalous feeding vessel was not found, we believe this to be infradiaphragmatic extralobar pulmonary sequestration and no surgery has been performed.
Fig. 25: Esophageal atresia. Fetus at 33 weeks’ gestation. (a and b) Sagittal images. This fetus was studied because of renal malpositioning (white arrow) and esophageal atresia (black arrow), unsuspected at US, was discovered.

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Fig. 26: Cervicothoracic cystic hygroma. Fetus at 32 weeks' gestation. (a and b) Fetal sagittal and coronal images show the lesion extending toward the mediastinum (arrows). Polyhydramnios due to esophageal compression was present.

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**Fig. 27:** Chylothorax. Fetus at 34 weeks’ gestation. (a, b, and c) Fetal coronal, sagittal, and axial images show right hydrothorax (asterisk); the right lung is compressed and the mediastinum is slightly displaced to the left. The hydrothorax was drained by intrauterine puncture before delivery. (d) Plain-film radiograph after birth shows right pneumothorax, possibly related to intrauterine puncture. This case has evolved satisfactorily.

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Fig. 28: Hydrothorax forming part of hydrops fetalis. Fetus at 21 weeks' gestation. (a, b, and c) Fetal coronal and axial MR images. There is a large amount of pleural fluid (asterisks), the lungs are hypoplastic (white arrows), and significant ascites and edema of the soft tissues (black arrows) are present. (d) Fetal radiograph shows significant edema of the soft tissues.

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**Fig. 29:** Thoracic lymphangioma. Fetus at 36 weeks’ gestation. (a, b, and c) Fetal coronal, left sagittal, and axial images. A hyperintense polylobulated structure in the chest wall and extending toward the axilla can be identified (arrows); it does not appear to affect the intrathoracic structures. (d) Photography of this fetus after birth.

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Fig. 30: Thoracic abdominal lymphangioma. Fetus at 26 weeks' gestation. (a and b) Fetal coronal, and axial images. Hyperintense polylobulated structure in the chest wall and extending toward the abdomen can be identified (arrows); it does not appear to affect the intrathoracic structures. (c) MR sagittal volumetric image shows the lesion. (d) US image shows an anechoic polylobulated lesion. (e) Photography of this fetus after birth.

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Results

<table>
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<tr>
<th>OUR RESULTS</th>
<th>Number of fetuses</th>
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<tr>
<td>Total pregnant women undergo MRI</td>
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<td>Total fetuses undergo MRI</td>
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<td>Esophageal atresia</td>
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<td>Pulmonary underdevelopment</td>
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<td>Hirotorax</td>
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<td>Diaphragmatic hernia</td>
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<td>Malformation Congenital</td>
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<td>Pulmonary sequestration</td>
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<td>Others</td>
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<td>126</td>
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Table 3

References: RADIOLOGY, UDIAT CD, Corporació Sanitària Parc taulí, CSPT - Sabadell/ES
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<th>Associated anomalies</th>
<th>Number of fetuses affected</th>
<th>Anomalies not diagnosed by either US or MRI</th>
<th>Number of fetuses</th>
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<td>Central nervous sistem</td>
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<td>Epiarterial bronchus</td>
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<td>Pulmonar artery stenosis</td>
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<td></td>
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<td>Pulmonar underdevelopment</td>
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<th>Image study</th>
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<td>MRI</td>
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<td>Ultrasounds</td>
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<th>MR influence in case diagnosis</th>
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<tr>
<td>MR added data to US in</td>
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</table>

Table 4

*References*: RADIOLOGY, UDIAT CD, Corporació Sanitària Parc taulí, CSPT - Sabadell/ES
Conclusion

Thoracic anomalies are varied and common. Their diagnosis can predict fetal viability, orient the prognosis and management of the newborn, and have implications for genetic counseling. While US remains the cornerstone of routine intrauterine study, it is sometimes necessary to resort to other diagnostic techniques when the information obtained by US is incomplete or uncertain. In our series, more anomalies were detected with MRI than US. MRI alone could not visualize all of the fetal anomalies, but the percentage of anomalies detected increased considerably when the findings of both techniques were evaluated together. MRI provided additional information in 30% of the cases.

We recommend performing MRI whenever a thoracic anomaly is suspected.
References


