Echogenic and Cystic Fetal Lung Lesions: Evaluation by Prenatal Sonography and Postnatal Imaging

G Ho¹, KW Cheung², HL Lam¹, JK Ip¹, HYM Tang², WWM Lam¹
¹Department of Radiology, and ²Department of Obstetrics and Gynaecology, Queen Mary Hospital, Pokfulam, Hong Kong

ABSTRACT

Objectives: We set out to review our unit’s experience in evaluating fetal lung lesions and the accuracy of prenatal diagnosis, and to illustrate the prenatal sonographic and postnatal radiological features of detected lesions.

Methods: Retrospective review of women whose fetuses were diagnosed with echogenic and/or cystic lung lesions in our institution. Their prenatal ultrasound, postnatal radiological, clinical, and histological data were retrieved for appraisal.

Results: In a 3-year period, 24 patients were referred to our unit with a cystic and/or echogenic fetal lung lesion. Of the 18 cases confirmed surgically and/or histologically, 15 were correctly diagnosed by prenatal ultrasound, with congruent postnatal radiological diagnosis. Of those misdiagnosed on prenatal ultrasound, postnatal imaging provided the correct diagnoses in most cases. Prenatal sonograms and postnatal images were reviewed in parallel for various congenital entities, with special attention to the cases misdiagnosed on imaging grounds. Fetal lung lesions were receded from view on prenatal ultrasound in later gestation in four cases. Their postnatal imaging findings were all abnormal.

Conclusion: Through appreciating the spectrum of imaging appearances of fetal lung lesions, which can be very similar, and acknowledging diagnostic pitfalls, we hope to refine diagnostic imaging accuracy. This could facilitate optimal prenatal counselling and postnatal management planning. Diagnoses made with prenatal ultrasound in our unit were correct in the majority of fetuses in this study. In-utero regression of fetal lung lesions in the course of prenatal ultrasounds may not indicate genuine resolution. Discrepancies in diagnoses gleaned from prenatal ultrasounds and postnatal imaging have been observed. Postnatal imaging should therefore be considered in all cases for confirmation and management planning.

Key Words: Cystic adenomatoid malformation of lung, congenital; Infant, newborn; Lung diseases; Tomography, X-ray computed; Ultrasonography, prenatal

中文摘要

胎兒的肺回聲異常及囊性肺病變：憑產前超聲和產後影像作出評估

何潔明、張嘉宏、林海苓、葉精勤、唐海燕、林慧文

目的：回顧本部門評估胎兒肺病變的經驗，包括產前診斷的準確度；並說明探測到的病變的產前超聲和產後放射學影像特徵。
INTRODUCTION

The more common prenatally diagnosed fetal lung lesions include congenital cystic adenomatoid malformations (CCAMs) / congenital pulmonary airway malformation, bronchopulmonary sequestration (BPS), and congenital diaphragmatic hernia (CDH). They usually manifest cystic, echogenic, or mixed cystic-echogenic lung lesions revealed by prenatal ultrasonography, and often have overlapping sonographic features that pose a diagnostic challenge. The outcome of the fetuses being diagnosed with these congenital lung lesions varies from asymptomatic course, to those requiring urgent postnatal surgery, and dismally intrauterine death. Accurate prenatal diagnosis is thus imperative to permit early and appropriate prenatal management planning and counselling. This review aimed to describe the prenatal sonographic features of the fetal lung lesions detected in our unit, and to evaluate them with their postnatal imaging and fetal neonatal outcome. Most cases have been histologically confirmed, where relevant. The accuracy of prenatal diagnosis in our unit was also evaluated retrospectively.

METHODS

We retrospectively reviewed the prenatal sonographic features, postnatal image findings, and fetal-neonatal outcomes of fetuses with echogenic and cystic lung lesions. We searched the database at the Prenatal Diagnosis Clinic, Tsan Yuk Hospital / Queen Mary Hospital, Hong Kong for the period January 2008 to December 2010. A total of 24 women with fetuses diagnosed to have echogenic and cystic lung lesions were identified. Echogenic lung lesions were defined as abnormally hyperechoic areas in the fetal chest; such lesions being more echogenic than normal lung or liver. Prenatal ultrasounds were performed using a Philips iU22 (Philips, Bothell [WA], USA), General Electric Voluson 730 (GE, GE Medical Systems Kretztechnik GmbH & Co OHG, Austria), and Acuson Sequoia machines (Siemens, Mountain View [CA], USA). Almost all of these newborns received tertiary neonatal care at Queen Mary Hospital. The postnasal imaging studies, age at surgery, final diagnosis, and follow-up records were retrieved and reviewed alongside with corresponding prenatal diagnoses. Two cases were excluded because the newborns were lost to follow-up shortly after birth; their postnatal courses and outcomes were therefore not available. Prenatal sonographic appearances and progress of the lung lesions were also studied through reviewing the serial prenatal ultrasound images and records.

RESULTS

Twenty-two cases with complete data are summarised in Table 1. All fetuses had unilateral echogenic, cystic, or mixed echogenic-cystic lung lesions and no other structural abnormality detected on ultrasound. Their gestational age at diagnosis ranged from 18 to 35 weeks; 18 (82%) had prenatal diagnosis at 18 to 25 weeks of gestation.

All fetuses except one survived to delivery. The demised fetus (case 8) belonged to a twin pregnancy and had a prenatal ultrasound diagnosis showing CCAM, and at 26 weeks of gestation ultrasonography showed hydrops. No autopsy was performed and the demise was believed to be multifactorial (mass effect by lung lesion displacing the oesophagus, twin pregnancy...
Table 1. Twenty-two patients with prenatal sonographic findings of echogenic-cystic fetal lung lesions; comparison with postnatal diagnoses and outcomes.

<table>
<thead>
<tr>
<th>Case No.</th>
<th>GA at Dx (weeks)</th>
<th>Appearance</th>
<th>Location</th>
<th>Medial shift</th>
<th>Dx</th>
<th>Lesion progress</th>
<th>Imaging</th>
<th>Dx (location)</th>
<th>Pathology</th>
<th>Follow-up (age)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>18</td>
<td>Mixed macrocystic and microcystic</td>
<td>Right</td>
<td>Yes, later resolved</td>
<td>CCAM</td>
<td>Static</td>
<td>CECT</td>
<td>CCAM II (RLL)</td>
<td>CCAM I (surgery at 34 days old)</td>
<td>Well (38 months)</td>
</tr>
<tr>
<td>2</td>
<td>35</td>
<td>Stomach and bowel in chest</td>
<td>Left</td>
<td>Yes</td>
<td>CDH</td>
<td>Static</td>
<td>CECT</td>
<td>CDH</td>
<td>CDH (surgery at 33 months)</td>
<td>Surgical complication and repaired. Well after (45 months)</td>
</tr>
<tr>
<td>3</td>
<td>23</td>
<td>Echogenic, no cysts</td>
<td>Left</td>
<td>Yes</td>
<td>CCAM</td>
<td>Smaller</td>
<td>CECT</td>
<td>CECT</td>
<td>BBPS or CCAM focal air-trapping</td>
<td>Well (3 months)</td>
</tr>
<tr>
<td>4</td>
<td>21</td>
<td>Echogenic and multi-cystic</td>
<td>Left</td>
<td>Yes, later resolved</td>
<td>CCAM</td>
<td>Receded</td>
<td>NCCCT</td>
<td>CCAM II (LLL)</td>
<td>CCAM II (surgery at 42 months)</td>
<td>Well (42 months)</td>
</tr>
<tr>
<td>5</td>
<td>22</td>
<td>Echogenic and multi-cystic</td>
<td>Left</td>
<td>Yes, later resolved</td>
<td>CCAM</td>
<td>Receded</td>
<td>OXCT</td>
<td>OXCT</td>
<td>CCAM II (surgery at 4 months)</td>
<td>Well (35 months)</td>
</tr>
<tr>
<td>6</td>
<td>23</td>
<td>Echogenic, no extra vessel or cyst</td>
<td>Right</td>
<td>No</td>
<td>CCAM</td>
<td>Smaller</td>
<td>CECT</td>
<td>CECT</td>
<td>CCAM II (surgery at 5 months)</td>
<td>Well (2 months)</td>
</tr>
<tr>
<td>7</td>
<td>21</td>
<td>Echogenic, vascular supply from aorta</td>
<td>Left</td>
<td>No</td>
<td>BBPS</td>
<td>Receded</td>
<td>OXCT</td>
<td>OXCT</td>
<td>NSCCT</td>
<td>BBPS extralobar (surgery at 2 months)</td>
</tr>
<tr>
<td>8</td>
<td>18</td>
<td>-</td>
<td>Left</td>
<td>Yes</td>
<td>CCAM</td>
<td>Smaller</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>9</td>
<td>25</td>
<td>Echogenic and multi-cystic, with vascular supply from aorta, Stomach bubble beneath diaphragm</td>
<td>Left</td>
<td>Yes</td>
<td>CCAM</td>
<td>Smaller</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>Well (4 months)</td>
</tr>
<tr>
<td>10</td>
<td>22</td>
<td>Echogenic and multi-cystic</td>
<td>Left</td>
<td>Yes, later resolved</td>
<td>CCAM</td>
<td>Smaller</td>
<td>CECT</td>
<td>CECT</td>
<td>CCAM II (surgery at 18 months)</td>
<td>Well (19 months)</td>
</tr>
<tr>
<td>11</td>
<td>27</td>
<td>Macrocystic</td>
<td>Left</td>
<td>Yes, later resolved</td>
<td>CCAM</td>
<td>Smaller</td>
<td>CECT</td>
<td>CECT</td>
<td>CCAM II (surgery at 18 months)</td>
<td>Well (19 months)</td>
</tr>
<tr>
<td>12</td>
<td>21</td>
<td>Echogenic, no cysts</td>
<td>Right</td>
<td>No</td>
<td>CCAM</td>
<td>Smaller</td>
<td>CECT</td>
<td>NCCCT</td>
<td>CCAM or focal air-trapping</td>
<td>Well (3 months)</td>
</tr>
<tr>
<td>13</td>
<td>22</td>
<td>Echogenic, no cysts</td>
<td>Left</td>
<td>No</td>
<td>CCAM</td>
<td>Smaller</td>
<td>CECT</td>
<td>CECT</td>
<td>BBPS extralobar (surgery at 8 months)</td>
<td>Well (17 months)</td>
</tr>
<tr>
<td>14</td>
<td>24</td>
<td>Macrocystic</td>
<td>Right</td>
<td>No</td>
<td>CCAM</td>
<td>N/A</td>
<td>CECT</td>
<td>CECT</td>
<td>CCAM II (surgery at 18 months)</td>
<td>Well (17 months)</td>
</tr>
<tr>
<td>15</td>
<td>23</td>
<td>Echogenic</td>
<td>Right</td>
<td>Yes, later resolved</td>
<td>CCAM</td>
<td>Smaller</td>
<td>CECT</td>
<td>CECT</td>
<td>CCAM III (surgery at 2 months)</td>
<td>Well (22 months)</td>
</tr>
<tr>
<td>16</td>
<td>19</td>
<td>Echogenic and multi-cystic, vascular supply from aorta and pulmonary artery Stomach in chest</td>
<td>Left</td>
<td>Yes, later resolved</td>
<td>Mixed CCAM-BPS</td>
<td>Smaller</td>
<td>CECT</td>
<td>CECT</td>
<td>Refused operation, well (3 months)</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>22</td>
<td>Echogenic</td>
<td>Left</td>
<td>Yes</td>
<td>CDH</td>
<td>Static</td>
<td>CECT</td>
<td>CECT</td>
<td>CCAM II and BPS intra-lobar (LL)</td>
<td>Well (11 months)</td>
</tr>
<tr>
<td>18</td>
<td>20</td>
<td>Mixed macrocystic and microcystic</td>
<td>Left</td>
<td>Yes, later resolved</td>
<td>CCAM</td>
<td>Static</td>
<td>CECT</td>
<td>CECT</td>
<td>CCAM II (surgery at 6 months)</td>
<td>Well (11 months)</td>
</tr>
<tr>
<td>19</td>
<td>28</td>
<td>Mixed cystic with heterogeneous echogenicity</td>
<td>Left</td>
<td>Yes, later resolved</td>
<td>BBPS</td>
<td>Smaller</td>
<td>CECT</td>
<td>CECT</td>
<td>CCAM II and BPS intra-lobar (surgery at 9 months)</td>
<td>Well (11 months)</td>
</tr>
<tr>
<td>20</td>
<td>21</td>
<td>Mixed cystic, vascular supply from aorta</td>
<td>Left</td>
<td>Yes, later resolved</td>
<td>Mixed CCAM-BPS</td>
<td>Static</td>
<td>CECT</td>
<td>CECT</td>
<td>CCAM II and BPS intra-lobar (surgery at 6 months)</td>
<td>Well (11 months)</td>
</tr>
<tr>
<td>21</td>
<td>20</td>
<td>Microcystic</td>
<td>Right</td>
<td>Yes</td>
<td>CCAM</td>
<td>Static</td>
<td>CECT</td>
<td>CECT</td>
<td>CCAM II (surgery at 9 months)</td>
<td>Well (13 months)</td>
</tr>
<tr>
<td>22</td>
<td>33</td>
<td>Mixed echogenicity</td>
<td>Left</td>
<td>Yes</td>
<td>CCAM</td>
<td>N/A</td>
<td>OXCT</td>
<td>CCAM-BPS</td>
<td>CCAM II (surgery at 2 days old)</td>
<td>Discharged at 20 days old, lost to follow-up afterwards</td>
</tr>
</tbody>
</table>

Abbreviations: BBPS = bronchopulmonary sequestration; CCAM = congenital cystic adenomatoid malformation; CDH = congenital diaphragmatic hernia; CECT = contrast-enhanced computed tomography; CLE = congenital lobar emphysema; CXR = chest radiograph; Dx = diagnosis; GA at Dx = gestational age at time of diagnosis; LLL = left lower lobe; LUL = left upper lobe; MRA = magnetic resonance angiography; N/A = not available or not applicable; NCCT = non-contrast computed tomography; RLL = right lower lobe; USG = ultrasound.
and / or maternal gestational diabetes mellitus). All neonates were discharged in good condition, except one case diagnosed with CDH passed away shortly after birth (case 9). Four cases have neither surgical or histopathology correlation, because they were managed conservatively, refused operation, or refused autopsy (cases 3, 8, 12, 16). Seventeen neonates had surgery; all were in good condition till the last follow-up, except for one who was lost to follow-up after discharge (case 22). The prenatal ultrasound diagnosis was correct in 15 (83%) instances out of the 18 who had surgery and / or histopathological confirmation.

Discrepancies between prenatal ultrasound and postnatal imaging diagnoses were observed. One fetus (case 3) was prenatally diagnosed to have CCAM but postnatal computed tomography (CT) showed congenital lobar emphysema (CLE) and was treated conservatively. Another had a prenatal diagnosis of mixed CCAM-BPS but was found to have CDH on chest radiography, and had surgery shortly after birth (case 22). Two fetuses, prenatally misdiagnosed as CCAM, were found to have BPS at surgery (cases 4 and 13).

There were nine confirmed cases of CCAM, four of BPS, one of mixed CCAM and BPS, and four cases of CDH, all surgically or pathologically proven. One case had a postnatal CT diagnosis of CLE. Images of the prenatal ultrasound, postnatal CT and radiography of the different diagnoses were reviewed in parallel (Table 2; Figures 1 to 25).

**DISCUSSION**

Ever-improving ultrasound technology has allowed better depiction and detection of fetal structural abnormalities. Fetal lung lesions are most easily noticeable on ultrasound when they cause a mass effect displacing the mediastinum (Figures 3a, 6a, 14a, 16b), and are therefore the most readily picked up on a four-chamber view of the heart.

Fetal lung lesions have been considered as a continuum of developmental abnormalities involving the pulmonary parenchyma, the pulmonary vessels, or a combination of both. Consequently, there are considerable overlapping features in the sonographic appearance of fetal lung lesions, from echogenic, cystic, to mixed echogenic-cystic. In our study, the lesions

<table>
<thead>
<tr>
<th>Fetal lung lesion</th>
<th>Case No.</th>
<th>Corresponding Figure No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>CCAM</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 1</td>
<td>1</td>
<td>1-2</td>
</tr>
<tr>
<td>Type 2</td>
<td>18</td>
<td>3-5</td>
</tr>
<tr>
<td>Type 3</td>
<td>15</td>
<td>6-8</td>
</tr>
<tr>
<td>BPS</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>9-10</td>
</tr>
<tr>
<td></td>
<td>13</td>
<td>11-13</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>14-15</td>
</tr>
<tr>
<td>Mixed CCAM + BPS</td>
<td>20</td>
<td>16-18</td>
</tr>
<tr>
<td>CDH</td>
<td>17</td>
<td>19-20</td>
</tr>
<tr>
<td></td>
<td>22</td>
<td>21-22</td>
</tr>
<tr>
<td>CLE</td>
<td>3</td>
<td>23-25</td>
</tr>
</tbody>
</table>

Abbreviations: CCAM = congenital cystic adenomatoid malformation; BPS = bronchopulmonary sequestration; CDH = congenital diaphragmatic hernia; CLE = congenital lobar emphysema.

**Table 2. Cases reviewed.**

**CCAM type I (case 1)**

![Figure 1](image1.png)

**Figure 1.** Transverse sonograms of fetal thorax at (a) 23 weeks and (b) 25 weeks of gestation, showing mass with anechoic cysts in the right lower hemithorax. Largest cyst exceeds 2 cm, and mediastinal shift appears to be present.

![Figure 2](image2.png)

**Figure 2.** A transverse computed tomographic lung window image showing heterogeneous cystic lesions in the right lower lobe. Post-lobectomy pathology revealed large and medium-sized cystic spaces, consistent with ultrasound findings compatible with a CCAM type I.
identified were CCAM, BPS, CDH and CLE, with the former two being more common.

CCAM is a bronchopulmonary malformation presenting as an intrapulmonary mass that is usually localised to one lung lobe.\(^2\) It is most often diagnosed and accounts for 30 to 40% of all congenital diseases.\(^3,4\) In 1977, Stocker et al\(^5\) first categorised three subtypes based on histological features and size. Later he revisited this classification and extended it to five types with the preferred new term “congenital pulmonary airway malformation”.\(^6\) In brief, type I refers to one or more cysts larger than 2 cm in diameter, surrounded by smaller cysts and a compressed normal parenchyma;

### CCAM type II (case 18)

**Figure 3.** (a) Transverse and (b) longitudinal sonograms of the fetal thorax at 20 weeks of gestation, showing an echogenic lung lesion with multiple cysts of <2 cm in the left lower hemithorax and an intact diaphragm. (c) Follow-up transverse sonogram at 36 weeks of gestation showing an unchanged left lung lesion.

**Figure 4.** A chest radiograph of a neonate (same fetus in Figure 3) showing multicystic spaces at the retrocardiac part of the left lower lung, compatible with CCAM.

**Figure 5.** Contrast-enhanced computed tomographic lung window in (a) transverse and (b) coronal planes, as well as a (c) 3-dimensional volume-rendered image depict the multicystic lung lesion in left the lower lobe, consistent with the surgically proven type II CCAM.
type II consists of cyst(s) less than 2 cm in diameter lined by bronchiolar epithelial cells; type III is any microcystic lesion. The sonographic appearance of CCAM varies depending on the subtype, and in our study they could be readily correlated with postnatal CT findings. Types I and II lesions manifest as a multi-cystic pulmonary mass (Figures 1-2 and 3-5, respectively), while type III lesions appear to be solidly echogenic due to innumerable interfaces of microcystic lesions that reflect the ultrasonic beam (Figures 6-8). Another classification proposed by Adzick et al. divided CCAM into two groups — macrocystic >5 mm and microcystic <5 mm — based on sonographic and anatomical findings, and has also been used by some authorities.

BPS is the second most common prenatally diagnosed lung lesion after CCAM. It is characterised by a portion of lung that does not connect to the tracheobronchial tree and has a systemic arterial supply, usually from the thoracic or abdominal aorta. BPS is divided into two types: intralobar and extralobar. Extralobar BPS has its own pleural investment and systemic venous drainage, whereas the intralobar variety shares its pleural cover with the normal lung and usually drains via the pulmonary venous system. Extralobar BPS may be associated with other congenital anomalies such as CDH. On sonography, BPS can appear similar to a CCAM (Figures 9-15). Colour Doppler is the key to distinguish BPS from other entities by its systemic arterial supply from the aorta, while a CCAM is supplied via the pulmonary artery. Due to the powerful Doppler signal from the adjacent cardiac pulsation, detecting its systemic supply may not be easy, especially when the lung lesion is close to the heart. Using postnatal contrast-enhanced CT, the feeding vessel can be clearly depicted with a multiplanar reformat (Figures 10 and 18). Detecting the systemic arterial supply is vital; not only does it allow an accurate diagnosis of BPS, it also permits safer dissection of the vessels during lung resection. Of note is that the diagnosis is not excluded if one is unable to see the systemic feeding vessel. This is proven in cases 4 and 13, both of which were thought to be CCAM as no feeding vessel was identified on prenatal ultrasounds, but turned out to be BPS. A non-contrast CT thorax performed in case 4

---

**CCAM type III (case 15)**

**Figure 6.** (a) Transverse and (b) longitudinal sonograms of the fetal thorax at 27 weeks of gestation, show an echogenic lung lesion occupying almost whole of right lung with mediastinal shift. No obvious cyst was detected.

**Figure 7.** A chest radiograph of the same neonate as in Figure 6, showing lesion with multiple tiny cystic changes, in the mid-to-lower zone of the right lung.

**Figure 8.** Contrast-enhanced computed tomographic thorax in the lung window in (a) axial and (b) coronal planes, showing a large microcystic lung lesion in right lower lobe, corresponding to the fetal echogenic lung lesion, which suggests a type III CCAM.
BPS (case 7)

Figure 9. (a) Transverse and (b) longitudinal sonograms of a fetal thorax at 24 weeks of gestation, showing an echogenic lung lesion in the left lung. (c) A colour Doppler sonogram demonstrates an aberrant systemic feeding artery arising from aorta. Also note the intact diaphragm.

Figure 10. (a) A coronal multiplanar reformatted contrast-enhanced CT image of the same neonate as in Figure 9, which demonstrates the aberrant feeding artery from aorta (arrow) to the left lower lobe. (b) Coronal plane in a lung window shows the defined focal area of decreased attenuation in the left lower lung zone. Venous drainage of the lesion is to the left inferior pulmonary vein. These features were consistent with the prenatal ultrasound findings and diagnosis of pulmonary sequestration (intralobar type).

BPS (case 13)

Figure 11. (a) A transverse sonogram of fetal thorax at 22 weeks of gestation, showing an echogenic lung lesion in lower zone of the left lung. No cyst detected. (b) A coronal sonogram of fetal lower thorax with colour Doppler at 37 weeks of gestation failed to detect any aberrant feeding vessel to the echogenic left lung lesion.

Figure 12. A chest radiograph of the neonate in Figure 11, showing focal triangular opacity at left retrocardiac region in the left lower lung zone (arrow).

Figure 13. Transverse contrast-enhanced helical computed tomograms of a neonatal thorax show consolidative lesion in posterior left lung base. A small vascular structure (v) appears to arise from the aorta (a) through the lesion. This patient had a surgically proven pulmonary sequestration of extralobar type. The feeding vessel as diagnostic discrimination was not detected in the prenatal ultrasound, possibly due to its small calibre.
(Figure 15) was unable to establish the correct diagnosis of BPS. Radiation exposure to paediatric patients is of paramount concern, thus we recommend a single contrast-enhanced CT thorax would be sufficient for diagnosis. Hybrid lesions with imaging and pathological features of both BPS and CCAM (particularly type II) have been described in the literature.4,7 This was also seen in one of our patients (case 20; Figures 16 to 18).

In CDH, the pleuroperitoneal canal fails to close completely, resulting in herniation of abdominal viscera into the thoracic cavity.10,12 It is associated with high rates of morbidity and mortality.10 Prognosis can be poor from lung hypoplasia and a high frequency of associated anomalies.13,14 The only neonatal death in our series is one with CDH and hypoplastic lung (on autopsy).

Accurate diagnosis is crucial for parental counselling, as CDH warrants immediate operative repair soon after birth, in contrast to CCAM and BPS where neonates may usually receive operation some time later or be managed conservatively. Three out of four cases of CDH were correctly identified using prenatal ultrasound. A herniated stomach can appear as a cystic lung lesion on the prenatal ultrasound (Figure 19a). Identification of the diaphragmatic defect in the coronal or sagittal plane gives away the correct diagnosis (Figure 19b). One can be misled to think otherwise, when the stomach bubble is seen at the normal left upper abdomen, in a case of left fetal lung lesion (Figure 21c). This was the case for a lesion erroneously diagnosed as a fetal lung entity, eventually it was unravelled by postnatal radiography to be CDH with herniated bowel

---

**BPS (case 4)**

<table>
<thead>
<tr>
<th>(a)</th>
<th>(b)</th>
<th>(c)</th>
</tr>
</thead>
</table>

**Figure 14.** (a) Transverse and (b) longitudinal sonograms of fetal thorax at 21 weeks of gestation showing an echogenic lung lesion, with multiple small cysts in the left lung and mediastinal shift. (c) A colour Doppler sonogram of the lung lesion failed to detect any aberrant feeding vessels.

**Figure 15.** A transverse non-contrast computed tomography (CT) lung window image of the neonate in Figure 14, showing a focal area with small cystic lesions and consolidation in left lower lobe. No contrast-enhanced CT was performed, therefore findings were unable to suggest pulmonary sequestration. Intra-operatively, there was feeding vessel from aorta near the diaphragm, suggesting intralobar pulmonary sequestration.
Mixed CCAM + BPS (case 20)

Figure 16. (a) A transverse sonogram of a fetal thorax at 21 weeks of gestation, and (b) longitudinal and transverse sonograms of the fetal thorax at 36 weeks of gestation, show an echogenic lung lesion, with multiple tiny cysts (arrow), in left lung. Also noted mediastinal shift and intact diaphragm. (c) A colour Doppler sonogram of the lung lesion shows aberrant systemic feeding vessel (arrow) from descending aorta. Ultrasound diagnosis was mixed CCAM and BPS, later proven to be correct.

Figure 17. A chest radiograph of the newborn in Figure 16 showing focal multicystic lesion in left lower lung zone.

Figure 18. (a) Transverse contrast-enhanced computed tomography (CT) lung window showing a cystic lung lesion and consolidative lesion in left lower lobe. (b) A coronal multiplanar reformatted CT image showing systemic arterial feeding vessel arising from aorta (arrow) supplying the consolidative area in the left lower lobe. The CT diagnosis of mixed CCAM type II and intralobar pulmonary sequestration was surgically proven to be correct.
loops, and the stomach bubble with tip of a nasogastric tube was in the left abdomen (Figure 22). Cautious visualisation of the diaphragmatic contour on sagittal and coronal planes facilitates an accurate diagnosis, though the diaphragmatic defect can be small and not easily to detect. Herniated bowel may mimic a cystic lung lesion such as CCAM. Real-time scanning for bowel peristalsis within the thoracic cavity and sliding movements through the defect would aid diagnosis. In cases where differentiating between CDH and CCAM seem difficult, fetal magnetic resonance imaging has proven to be useful.\textsuperscript{14,15}
CLE, also termed congenital lobar overinflation, is characterised by progressive lobar overexpansion, and is rarely detected on prenatal ultrasounds. Fetal ultrasound shows a uniformly echogenic lesion with an associated mass effect (Figure 23). Its appearance can mimic other fetal lung lesions as in case 3, which was thought to be CCAM. Postnatal CT is helpful in differentiation by revealing hyperexpansion and decreased attenuation of the affected lobe and absence of lung cysts (Figure 25).

Diagnoses based on prenatal ultrasounds were accurate in the majority of fetuses in this study. In our experience, when a fetal lung lesion is encountered using prenatal ultrasoundography, application of colour Doppler to look for a systemic arterial feeding vessel and scrutiny for an intact diaphragm on coronal and sagittal planes are of utmost use in differentiating between the more common congenital entities. Serial scanning is crucial to monitor the progress of the lung lesion and its effect on fetal growth, bearing in mind that many of the fetal lung lesions are more noticeable on initial scans due to mass effects causing mediastinal shift. In four cases, the fetal lung lesions were receded from view on ultrasound with resolution of mediastinal shift in later gestation. Yet, postnatal CT findings in all of these cases were abnormal, indicating that apparent in-utero regression may not be genuine; this concurs with previous local and other publications.

Congenital lung abnormalities, other than those described, include abnormal lung with normal vasculature such as bronchogenic cysts, bronchial atresia, congenital high airway obstruction syndrome, and those of normal lung with abnormal vasculature, such as hypogenetic lung syndrome. These were not detected in our series.

CONCLUSION

Prenatal sonography remains the primary modality for fetal structural assessment. Accurately diagnosing fetal lung lesions can be a challenge in prenatal ultrasound due to overlapping sonographic features. Through presenting a series of prenatal sonograms of fetal lung

**CLE (case 3)**

**Figure 23.** (a) Transverse and (b) longitudinal sonograms of the fetal thorax at 23 weeks of gestation show a uniform echogenic left lung lesion with mediastinal shift. No cyst is evident. Left hemidiaphragm is intact. The initial sonographic diagnosis of a CCAM was inaccurate.

**Figure 24.** Chest radiograph of the neonate in Figure 23 shows a hyperlucency and expansion in left upper-mid lung zone with relative paucity of vascular markings.

**Figure 25.** (a) Transverse and (b) coronal contrast-enhanced CT lung window images depicting hyperinflated left upper lobe, consistent with congenital lobar emphysema. No cystic change is seen.
lesions collected in our institution, we were able to correlate findings with postnatal images, and review diagnostic pitfalls. A correct diagnosis is imperative for prenatal counselling, management, as well as anticipation of possible neonatal outcomes. Postnatal imaging studies, contrast-enhanced CT in particular, are necessary in evaluating and confirming the diagnosis of the lung lesions, even in cases of in-utero regression.

ACKNOWLEDGEMENTS
We would like to express much gratitude to Drs Charas Ong, CP Lee, and SF Wong (Department of Obstetrics and Gynaecology, Queen Mary Hospital) for their dedicated work at the Prenatal Diagnostic Clinic, Tsan Yuk Hospital, and their permission to use the data collection in this study. We would also like to thank Ms Lilian Ngan (Radiographer, Department of Radiology, Queen Mary Hospital/Tsan Yuk Hospital) for helping to retrieve the images in the manuscript.

REFERENCES