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## Ultrasound guided drainage of an esophageal duplication cyst in a newborn in respiratory distress



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#### ABSTRACT

Newborns with chest masses may present with respiratory distress in the perinatal period. We present the case of a term male infant with a cystic right upper chest mass born in respiratory distress at 39 4/7 weeks gestation who underwent ultrasound guided percutaneous drainage of the mass. The patient responded well to the percutaneous drainage and two days later the mass was surgically excised. Final pathology revealed an esophageal duplication cyst. This report describes a unique management of a patient with a congenital cystic chest mass presenting in respiratory distress.

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Fetal chest masses may be identified and characterized during fetal ultrasound and MRI [1,2]. Bronchopulmonary malformations result from a spectrum of foregut, airway, lung and vascular malformations, and major categories include congenital lobar emphysema, bronchogenic cysts, congenital pulmonary airway malformation (CPAM), bronchopulmonary sequestration, hypogenetic lung syndrome and pulmonary ateriovenous malformation [3]. Congenital cystic lesions including bronchogenic, esophageal duplication, neurenteric, pericardial, and thymic cysts are epithelium lined lesions that contain fluid [2,4,5]. Prenatal imaging can help distinguish amongst these possibilities and provide prognosis and aid in counseling parents about expected fetal outcome and postnatal course. Although some congenital masses of the chest, most notably CPAMs, may involute or reduce in size during gestation, many lesions persist into the newborn period [6].

A case series reviewing more than 175 prenatally diagnosed fetal chest masses, which included CPAMs and bronchopulmonary sequestrations revealed that overall prognosis depends on the size of

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the thoracic mass and secondary physiologic derangement [6]. Large fetal chest masses, due to their compression of mediastinal vascular structures and the esophagus, can cause hydrops and polyhydramnios in utero and can lead to in utero death. Postnatal presentation is quite variable ranging from asymptomatic to severe respiratory distress requiring mechanical ventilation. Tachypnea, cyanosis with oxygen requirement, and carbon dioxide retention are common clinical features. Fetal chest masses can also impede normal growth and development of lung tissues, which can subsequently lead to persistent pulmonary hypertension of the newborn (PPHN). Masses that contain air or cause air trapping due to compression of the tracheobronchial tree can lead to pneumothoraces. Smaller thoracic masses are typically undiagnosed in the neonatal period and can present in older children or adults with pneumonia, hemoptysis, pneumothorax, dysphagia, or signs of caval obstruction.

We present a case of a newborn with a large congenital chest mass born in respiratory distress. Ultrasound guided percutaneous drainage of the cyst resulted in stabilization of the patient and a successful and full recovery.

#### 1. Case report

The infant was a 39 4/7 week male with a prenatal diagnosis by ultrasound and MRI of a bilobed cystic right chest mass that did not regress in size over the course of fetal gestation (Fig. 1a and b). There was no defined feeding vessel or communication to the esophagus or trachea. Right lung volume was estimated at 40% of predicted. The smaller of the cystic components crossed midline

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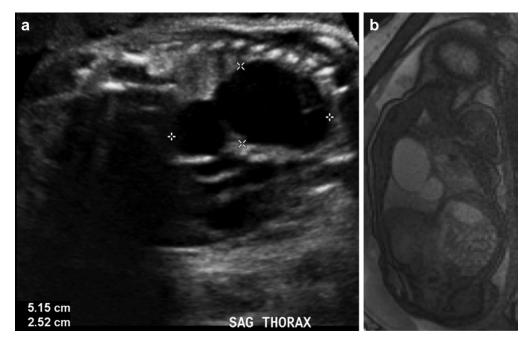


Fig. 1. (a) Prenatal ultrasound demonstrates a bilobed anachoic  $5.2 \times 2.5$  cm mass in the right hemithorax. (b) Coronal fetal MRI demonstrates a bilobed mass in the right hemithorax similar to that seen on the fetal ultrasound.

posterior to the left atrium and the descending thoracic aorta. There was no midline mediastinal shift. A fetal ECHO at 36 weeks gestation revealed no structural defects and qualitatively good biventricular function.

Apart from maternal hypertension, maternal history and pregnancy was unremarkable. Prior to delivery, which was by elective cesarean section for macrosomia, there was no fetal distress. Rupture of the membranes with clear fluid occurred at delivery. The infant emerged without spontaneous cry and bradycardia  $\sim 70$  beats per minute (bpm). Positive pressure ventilation was initiated at  $\sim 30$  s of life, but at 1 min the infant had no spontaneous respirations and heart rate remained  $\sim 70$  bpm, so the infant was

intubated, which did not improve respiratory function or heart rate. Chest compressions were initiated and Epinephrine was given. Suctioning through the endotracheal tube yielded copious viscous clear fluid. Heart rate improved to >100 bpm and oxygen saturation was  $\sim\!90\%$  on 100% FiO $_2$  with improved chest rise. At 30 min cord pH was 7.09.

In the NICU the infant remained difficult to ventilate despite transitioning from conventional ventilation to high frequency oscillatory ventilation. Umbilical arterial gas was: pH = 6.85,  $pCO_2 = 106$ , and  $pO_2 = 74$  on 100% FiO<sub>2</sub>.

A postnatal echocardiogram showed right ventricular hypertension with near systemic right ventricular pressures and a large

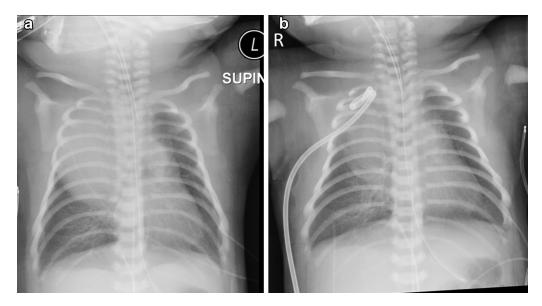


Fig. 2. (a) Radiodense opacity occupying the right upper hemithorax with resulting right lower lobe atlectesis. A nasogastric tube and an endotracheal tube are in place. (b) Post procedural chest radiograph demonstrates a pigtail catheter in the right upper chest mass, which has decreased in size compared to the pre-procedure study. A nasogastric tube and an endotracheal tube are in place.

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