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Retrospective study of prenatal diagnosed pulmonary sequestration

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Abstract

Purpose—To describe the prenatal findings, treatments and outcomes of fetuses with pulmonary sequestrations (PS), which were retrospectively studied.

Methods—From May 2010 to January 2013, 292 women were referred to the Guangdong Women and Children Hospital, Guangzhou because obstetric ultrasound had demonstrated fetal lung lesions. In 68 fetuses, the echogenic lung masses were pulmonary sequestrations deriving arterial blood supply from clearly identifiable systemic arteries rather than the pulmonary artery. We examined records of the 68 fetuses and measured the lesions and congenital cystic adenomatoid malformation volume ratio (CVR), provided prenatal counseling and treatment, documented the CVR, location of lesion, gestational age at diagnosis, need for fetal intervention, perinatal clinical course (including the development of hydrops, effusions, and neonatal

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respiratory distress), gestational age at delivery, postnatal ultrasound and CT, operation treatment, survival and pathology.

Results—There were 68 PS in our study. All of 56 cases with CVR ≤ 1.6 , survived without prenatal hydrops or postnatal respiratory symptoms. In 12 cases with CVR >1.6 , 7 cases (58.3 %, 7/12) had hydrops, 10 cases (83.3 %, 10/12) survived, 2 cases (16.7 %, 2/12) of induced abortion, 8 cases (66 %, 8/12) were postnatal symptomatic. There was statistical significant difference in the incidence rate of hydrops and postnatal respiratory symptoms between the PS with CVR ≤ 1.6 and that with CVR >1.6 . 66 cases postnatal survived, two cases of induced abortion. 64 cases had no prenatal treatment, two cases underwent thoracoamniotic shunt. The mean gestational age at birth was 38 weeks (range 34–40 weeks). 21 asymptomatic cases had no surgery. 45 cases (8 symptomatic and 37 asymptomatic) underwent surgical resections, 43 cases without postoperative complications, two cases of postoperative pneumothorax, no wound infection. Postoperative follow-up showed good growth in all surgery cases.

Conclusion—PS specific absence of hydrop was a congenital disorder with an excellent prognosis. CVR >1.6 was associated with fetal hydrops and postnatal symptoms. However, absolute CVR value cannot be used to select fetuses for fetal treatment before the development of hydrops. Surgery of PS in neonates or infants presented a good outcome.

Keywords

Pulmonary sequestration; Congenital cystic adenomatoid malformation volume ratio; Fetal surgery; Prenatal diagnosis; Hydrops; Ultrasound

Introduction

Pulmonary sequestration (PS) can be described as a segment of lung that had no identifiable communication with the normal bronchial tree and receives its blood supply from one or more anomalous systemic arteries [1]. First described by Huber in 1777 as an accessory pulmonary lobe, it was not until 1964 that Pryce introduced the term sequestration derived from the Latin verb sequestare meaning “to separate” [2]. The incidence of PS was 0.15–1.8 % [3, 4], making PS the second most common congenital lung anomaly. The combination of an aberrant systemic blood supply (identified by color-flow Doppler sonography) and an echogenic lung mass was pathognomonic for the prenatal diagnosis of pulmonary sequestration [5, 6].

Pulmonary sequestrations and congenital cystic adenomatoid malformations (CCAM) had been categorized as congenital cystic lung lesions [7]. It was known that CCAM can regress prenatally, stay the same size, or grow and produce pulmonary hypoplasia, hydrops, or both [8, 9]. The CCAM volume ratio (CVR) was developed for CCAMs without a dominant cyst. The CCAM volume was obtained from sonographic images by obtaining the height, length, and width of the CAM. To eliminate the influence of fetal gestational age by dividing by the head circumference [10, 11]. The CVR allowed direct comparison of fetuses of different gestational age and serial measurements of the CCAM in the same fetus normalized to head circumference, to chart progression, or regression of the CCAM. It was reported that the CVR was a useful sonographic indicator of fetuses at risk for hydrops who require close

ultrasound observation and possible fetal intervention [11]. A CVR of greater than 1.6 at presentation accurately predicted increased risk of hydrops developing in CCAM. We hypothesized that fetuses with PS can have the same natural history of CCAM and thus CVR is also an indicator of fetuses at risk for hydrops as those fetuses with CCAM. The purpose of this study was to examine the perinatal course of PS diagnosed by prenatal ultrasound to better understand the natural history and clinical treatment of this anomaly as a potentially distinct congenital lung lesion.

Materials and methods

A total of 292 women whose fetuses were diagnosed with a congenital lung lesion between May 2010 and January 2013 were evaluated at the Guangdong Women and Children Hospital, Guangzhou. Records were reviewed retrospectively to find patients diagnosed sonographically with a PS. The ultrasonographic criteria used for diagnosis of a PS were the presence of an echogenic fetal thoracic mass and an aberrant systemic arterial supply by color-flow Doppler (Figs. 1a, b, 2a, 3a) [12]. The sonograms of all patients diagnosed with a PS were examined for measuring intrathoracic mass volume and CVR. The maximal length of the PS in sagittal view was used to measure the length. Perpendicular to this axis at the maximal width of the lesion, the width and height of the PS were measured (Fig. 1b). CVR was calculated by $\text{length} \times \text{width} \times \text{height} \times 0.52 / \text{head circumference}$ [11]. The following data were collected: location of lesion, gestational age at diagnosis, need for fetal intervention, perinatal clinical course (including the development of hydrops, effusions, and neonatal respiratory distress), gestational age at delivery, postnatal X-ray, ultrasound (Fig. 3c) and CT (Figs. 2d, 3d), operation treatment and survival. Hydrops was defined as an accumulation of fluid in two or more body spaces, edema of the soft tissues that was not caused by fetomaternal blood group incompatibility or both [13, 14].

Results

Of the 272 fetal lung lesions identified, there were 68 PS (Table 1), 132 CCAM and 72 congenital diaphragmatic hernia (CDH). The mean gestational age at diagnosis was 24 weeks (range 18–30 weeks). There were 39 boys (57.4 %, 39/68) and 29 girls (42.6 %, 29/68), 47 cases in left (69.1 %, 47/68) and 21 cases in right (30.9 %, 21/68), 66 cases (97.1 %, 66/68) continued the pregnancy until the birth and survived, 2 cases (2.9 %, 2/68) of induced abortion in 23 and 26 weeks because the fetal parent's worried about the adverse outcomes of fetal hydrops. The mean gestational age at birth was 38 weeks (range 34–40 weeks). The mean birth weight was 3,316 g (range 2,041–4,856 g). 18 cases (26.5 %, 18/68) disappeared in the following ultrasound but 14 cases (78 %, 14/18) still confirmed by postnatal CT (Fig. 2), 4 cases (22 %, 4/18) did not have postnatal CT, so we cannot conclude that they have resolved.

All of 56 cases (82.4 %, 56/68) with CVR ≤ 1.6 survived without prenatal hydrops or postnatal respiratory symptoms. But in 12 cases with CVR >1.6 , 7 cases (58.3 %, 7/12) had hydrops, 10 cases (83.3 %, 10/12) survived (two cases of induced abortion because of fetal parents initiative request), 8 cases (66.7 %, 8/12) were postnatal symptomatic. There was statistical significant difference between the PS with CVR ≤ 1.6 and that with CVR >1.6 .

The incidence rate of hydrops and postnatal respiratory symptoms in PS with CVR >1.6 were higher than that with CVR ≤1.6. (Table 2).

In prenatal treatment, 64 cases (including 56 with CVR ≤1.6 and 8 with CVR >1.6) had no prenatal treatment, two cases (hydrops and with CVR >1.6) underwent thoracoamniotic shunt to reverse the hydrops, and two cases (hydrops and with CVR >1.6) of induced abortion as described above. No open fetal surgery or ex utero intrapartum therapy (EXIT) were taken in our study.

In postnatal treatment, 21 asymptomatic cases had no surgery, 45 cases (8 symptomatic and 37 asymptomatic) underwent surgical resections. The mean surgery age was 2 months (range 2 h to 3 years). 40 cases had selective surgery, 5 cases immediate surgery. 42 cases formal thoracotomy, and 3 cases thoroscopic surgery (2 cases were interrupted and changed to formal thoracotomy because of the lacking appropriate sizes instruments for infant). In 45 surgery cases, 43 cases were without postoperative complications, 2 cases of postoperative pneumothorax, which cured after treatment of drainage, had no wound infection. The duration of hospital stay in surgery cases ranged from 5 to 21 days. Postoperative follow-up showed good growth and lung function in all surgery cases, although there was no objective lung function measurement. Pathology were taken after surgery in all 45 cases, 39 cases (86.7 %, 39/45) were PS (Fig. 2). 6 cases (13.3 %, 6/45) were PS co-existing with CCAM (Fig. 3). 12 cases (26.7 %, 12/45) were intralobar, and 33 cases (73.3 %, 33/45) were extralobar (27 intrathoracic and 6 infradiaphragmatic).

Discussion

The data from our study indicated that PS specific absence of hydrop was a congenital disorder with an excellent prognosis. CVR >1.6 was associated with fetal hydrops and postnatal symptoms. Surgery of PS in the neonatal period presented a good outcome.

Excessively large fetal chest mass led to fetal hydrops, maldevelopment of lung and so on. In CCAM, it was reported that the CVR was a useful sonographic indicator of fetuses at risk for hydrops who require close ultrasound observation and possible fetal intervention [10, 11]. PS and CCAM have been categorized as congenital cystic lung lesions; we assume that CVR was equally applicable for PS. Our study verified that the incidence rate of hydrops and postnatal respiratory symptoms in PS with CVR >1.6 were higher than in that with CVR ≤1.6. But CVR >1.6 did not uniformly result in hydrops, which occurred in 58.3 % of the fetuses in this category. These limitations of the CVR indicate that in the high-risk PS, an absolute CVR value cannot be used to select fetuses for fetal treatment before the development of hydrops. The CVR maybe, however, a very useful way to stratify patients into categories of high or low risk of hydrops and postnatal respiratory symptoms. This information was invaluable in counseling parents and identifies fetuses in need of close serial sonographic surveillance for early signs of hydrops.

In prenatal diagnosis and differential diagnosis, PS can be seen on fetal ultrasound as a well-defined triangular echogenic mass in the lower chest or the suprarenal region of the abdomen [15, 16]. The differential diagnosis includes CCAM, bronchogenic cysts, enteric

duplication cysts and neuroblastoma [17]. Precise prenatal diagnosis requires visualisation of a systemic feeding artery. In a few cases, there was histological evidence of a mixed PS–CCAM lesion [18]. We also observed six cases with the visualisation of a systemic feeding artery pathologically diagnosed as PS co-existing with CCAM (Fig. 3), suggesting that these conditions may have a common embryological origin [19]. One CDH was misdiagnosed as PS because of the spleen hernia to the left chest. The antenatal ultrasound mistook the spleen as PS lesion and mistook the splenic artery as a systemic feeding artery. CCAM and CDH should be considered as part of the differential diagnosis of antenatal diagnosed PS.

Several studies have reported substantial or complete regression of the lesion on sequential scanning through pregnancy. However, postnatal, cross-sectional imaging almost invariably demonstrates persistence of the PS [17, 20]. In our study, we also found that 18 cases of lesions “disappear” on sequential scanning through pregnancy. However, postnatal CT demonstrated persistence of 14 cases, another four cases did not undergo postnatal CT, so we cannot conclude that they had resolved. It indicated that the lesions had not really “disappeared”. These lesions “disappear” sonographically because the PS tissue becomes isoechogenic with adjacent normal lung parenchyma. In these fetuses, the lesion was still present by postnatal CT. In practical terms, investigation begins with a chest X-ray after birth. But many PS were small and were not visible on a chest X-ray [21]. Enhanced computed tomography (CT) best demonstrated the parenchymal abnormalities associated with PS [22]. In our study, 55 cases (83.3 %, 55/66) can be evaluated by postnatal ultrasound. Although the ultrasound detection rate was not as high as CT, ultrasound was simple, non-invasive, nonradioactive and can be administered besides the bed. Therefore, ultrasound was recommended as the first diagnostic modality for evaluating a suspected supraphrenic chest mass in childhood [23, 24].

In light of PS prenatal treatment, most cases waited for postnatal surgery [25], also known as the common expectation treatment. Fetal treatment for PS was rare [26]. If patients had a large pleural effusion with hydrops, effective antenatal intervention was provided either by placement of thoracoamniotic shunts and consequent resolution of the effusions or by occlusion of the feeding vessel at the hilus of the tumor by ultrasound-guided laser coagulation or injection of a sclerosant agent [25]. A thoracoamniotic shunt may reverse the hydrops and ameliorate pulmonary hypoplasia. Treatment was aimed essentially at drainage of the effusions rather than surgery of the tumor [27]. Open fetal surgery and ex utero intrapartum therapy (EXIT) were rarely indicated for PS [18, 25]. In our study, two cases of PS hydrops and with CVR >1.6 had thoracoamniotic shunts prenatally, and other 64 cases of PS had no fetal treatment.

In the postnatal treatment, surgery for symptomatic PS after birth was not debated, but there was controversy about operating on asymptomatic PS. Many felt they can be left along and the risk of infection was very small, and the risks of surgical morbidity may be weighed against the risks of complications of the PS itself. So they would not recommend surgical resection for the asymptomatic surgical neonate [17]. But others believed that surgery was needed as recurrent infection was the classical complication of PS [28, 29]. In our study, 45 cases underwent surgery and had good recovery. It presented that early surgery for neonates or infants could be safe and had good outcome. Therefore, our study suggested that surgery

should be taken even in cases of asymptomatic PS. Formal thoracotomy remains the standard method for resection. Thorascopic surgery was being advocated by some [30–32]. Advocates of this approach argued that the small incisions may reduce the risk of musculoskeletal deformity, including chest wall asymmetry, scoliosis, rib fusion and winging of the scapula reported after posterolateral thoracotomy. However, there were no long-term follow-up studies supporting this argument. In our study, three cases of newborns with intrathoracic extralobar PS were treated with thorascopic surgery and one case had a successful treatment, but other two were interrupted and changed to formal thoracotomy because of the surgical Instruments. This required more experience and surgical skills.

In conclusion, our retrospective study would contribute to accurate prenatal counseling, surveillance, and formulation of a treatment plan of PS. However, prospective studies should be carried out to evaluate the CVR; additionally more long-term follow-up should be carried out in PS.

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Fig. 1.

Prenatal ultrasound of 25 weeks fetus showed echogenic mass of left chest. **a** Color Doppler shows a systemic feeding artery (*white arrow*). **b** Maximal length of the PS in sagittal view was used to measure the length (*left panel*). Perpendicular to this axis at the maximal width of the lesion, the width and height of the PS were measured (*right panel*). **c** Prenatal MRI showed mass in the left chest (*white arrow*)

Fig. 2.

Intralobar PS. Echogenic mass and a systemic feeding artery are shown in the left chest by prenatal ultrasound at 26 weeks (**a** *white arrow*), the lesion “disappeared” (**b**) but a systemic feeding artery (**c** *white arrow*) can still be observed at 33 weeks. The lesion was still present by postnatal CT (**d** *white arrow*), surgery confirmed as PS pathologically (**e**, **f**)

Fig. 3.

Extrathoracic PS co-existing with CCAM showing an echogenic lung mass with systemic blood supply detected by ultrasound (**a** *white arrow*) at 32 weeks and MRI (**b** *white arrow*) at 35 weeks. Ultrasound (**c** *white arrow*) and CT (**d** *white arrow*) confirmed the diagnosis postnatally. Postoperative pathology confirmed it as PS co-existing with CCAM (**e**, **f**)

Table 1**Antenatal and Postnatal Findings of 68 cases PS**

	Number	Percentage (%)
Sex		
Male	39	57.4 (39/68)
Female	29	42.6 (29/68)
Location		
Left	47	69.1 (47/68)
Right	21	30.9 (21/68)
Clinical outcomes		
Survival after birth	66	97.1 (66/68)
Postnatal surgery	45	68.2 (45/66)
Formal thoracotomy	42	93.3 (42/45)
Thorascopic surgery	3	6.7 (3/45)
Selective surgery	40	88.9 (40/45)
Immediate surgery	5	11.1 (5/45)
No surgery	21	31.8 (21/66)
Active induction of labor	2	2.9 (66/68)
Death after birth	0	0.0 (0/68)
PS lesion follow-up		
Persist during pregnancy	48	72.7 (48/66)
“Disappeared” by prenatal ultrasound	18	27.3 (18/66)
Confirmed by postnatal CT	14	21.2 (14/66)
No postnatal CT	4	6.1 (4/66)
Postnatal symptom		
Asymptomatic	58	87.9 (58/66)
Symptomatic with respiratory system	8	18.2 (8/66)
Pathological findings		
Intralobar PS	12	26.7 (12/45)
Extralobar PS	33	73.3 (33/45)
Intrathoracic extralobar PS	27	60.0 (27/45)
Infradiaphragmatic extralobar PS	6	13.3 (6/45)
Co-existing with CCAM	6	13.3 (6/45)

Table 2

The incidence rate of hydrops and postnatal respiratory symptoms in PS

	Number	Percentage (%)
CVR 1.6	56	82.4 (56/68)
No fetal hydrops	56	100.0 (56/56)
Fetal hydrops	0	0.0 (0/56)
Asymptomatic	56	100.0 (56/56)
Symptomatic with respiratory	0	0.0 (0/56)
CVR >1.6	12	17.6 (12/68)
No fetal hydrops	5	41.7 (5/12)
Fetal hydrops	7	58.3 (7/12) *
Asymptomatic	2	16.6 (2/12)
Symptomatic with respiratory	8	66.7 (8/12) **
Active induction of labor	2	16.6 (2/12)

* Compare with CVR 1.6, $\chi^2 = 30.37$, $P < 0.01$ ** Compare with CVR 1.6, $\chi^2 = 36.13$, $P < 0.01$