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9

Management of fetal tumors

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A B S T R A C T

In this review article, we discuss the most common fetal tumors, their prenatal management, and outcomes. Overall, the most important outcome predictors are tumor histology, size, vascularity, and location. Very large lesions, lesions causing cardiac failure, and hydrops and lesions obstructing the fetal airway have the poorest outcome, as they may cause fetal death or complications at the time of delivery. Fetal therapy has been developed to improve outcomes for the most severe cases and can consist of transplacental therapy (sirolimus for rhabdomyomas or steroids for hemangiomas and microcystic lung lesions) or surgical intervention (shunting of cystic masses, tumor ablation, occlusion of blood flow or airway exploration, and protection). Given the rarity of fetal tumors, patients should be referred to expert centers where care can be optimized and individualized to allow the best possible outcomes.

Introduction

Neonatal tumors account for 2% of all pediatric tumors [1] and affect approximately 1 in 12–27000 live births [2]. The prevalence of fetal tumors is most likely higher but is underestimated because of the fact that many result in stillbirth. Indeed, fetal tumors were found in 0.5% of all

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stillbirths [3] and in 1.2% of fetuses with birth defects [4]. Prenatal diagnosis of congenital tumors is essential, as in many cases, it will influence pregnancy management or the location or mode of delivery. The aim of this review is to offer an overview of the most common fetal tumors and their current management options.

Brain tumors

Congenital brain tumors account for 5–12% of all congenital tumors. The most common fetal brain tumors are teratomas, followed by glial tumors (astrocytoma), choroid plexus tumors, and primary neuroepithelial tumors [5–12]. Craniopharyngioma [7,10,11] (Fig. 1), ependymoma [7], hemangiomas [8,11], hamartomas [10,11], schwannoma, and endodermal sinus carcinoma [11] are more rare.

Fetal brain tumors typically present in the third trimester of gestation, although hamartomas have been reported as early as 21 weeks [7,13] [12]. In addition to the finding of an (often large) intracranial mass, indirect signs at presentation include macrocephaly, hydrocephalus [12], and asymmetric skull growth [7]. The majority of tumors are supratentorial. It is critical to differentiate tumors from more benign arachnoid cysts, intracranial bleeds, vascular malformations, such as vein of Galen aneurysms, and hemimegalencephaly. Doppler ultrasound and magnetic resonance imaging (MRI) are often helpful.

The prognosis of cerebral tumors is poor and many result in stillbirths [7,14]. Given that the fetal cranial sutures are not fused, two thirds of cases managed expectantly will present with significant macrocephaly at the time of birth, thereby complicating delivery and causing prolonged or obstructed labor [6]. Other common complications include fetal intracranial hemorrhage and fetal distress [6,12]. In some cases, where the mass is predominantly cystic, cephalocentesis can be done to facilitate delivery. The latter procedure is, however, associated with a high risk of fetal demise [15] because of the rapid intracranial decompression.

For most fetal brain tumors, postnatal treatment consists of surgery, eventually combined with chemotherapy. However, 25–50% are inoperable [6,9,12]. Ventricular drainage can be done for associated hydrocephalus.

The general prognosis of fetal brain tumors is poor, with survival rates ranging between 10% and 73% at 1 year of age [5,11]. This wide range can be mainly explained by the variable rate of operable tumors in the series. Mean postnatal survival for teratomas is 21 days, 5 months for primary neuroectodermal tumors, and 26 months for astrocytomas [6].

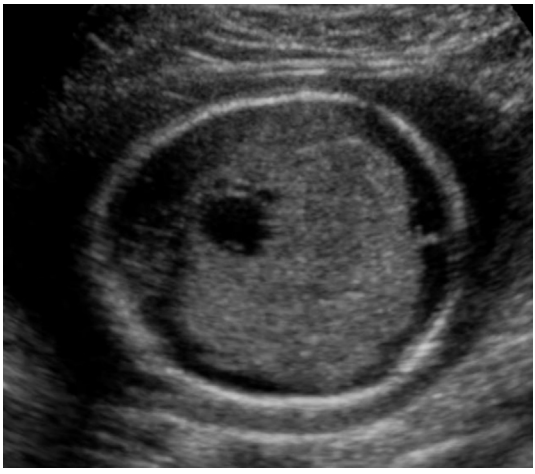

Fig. 1. Axial ultrasound image of a fetal cerebral craniopharyngioma at 22 weeks. Note the large echogenic intracranial mass.

Neck and face tumors

The most common head and neck fetal tumors are hemangiomas and lymphangiomas (Fig. 2), accounting for 42% of all head and neck fetal neoplasms [16]. Teratomas follow with almost 30% [17,18]. More rarely, tumors of the mouth and the soft tissues can occur, including ranula, epignathus, epulis, and nasal glioma (Fig. 3) [19].

Fetal head-and-neck tumors are usually incidental findings at the time of routine fetal anatomy screening, and most lesions will present in the second trimester of pregnancy [17,20,21]. Indirect signs include fetal head hyperextension in the case of large neck tumors and polyhydramnios if the mass impairs normal fetal swallowing [17,20]. Prenatal diagnosis of neck and face masses is essential, as all can lead to airway obstruction at birth [17,18]. Detailed ultrasound is the first step in the assessment of the airway, and this will often be complemented with MRI, which, in some cases, may give a better topographic overview of the location of the mass in relation to the trachea and the head and neck structures [22]. Fetuses with an obstructed airway should be delivered in tertiary centers that have expertise in managing these. The ex utero intrapartum treatment (EXIT)-procedure is an established mode of delivery for fetuses with obstructed airway. This procedure is a modified cesarean section under maternal general anesthesia during which only the head and the neck of the fetus are delivered. The umbilical cord is not clamped, and tocolysis is used to prevent placental separation. By doing so, the fetus remains oxygenated through the placental circulation. This allows time for the neonatal team to establish an airway (either by endotracheal intubation or by tracheostomy). In rare cases, a tumor excision on placental support is required to access the airway. Once the airway is secured, the umbilical cord is clamped and the baby is fully delivered for further management [20]. Where untreated, large cervical teratomas with airway obstruction result in near universal mortality [23], large EXIT series have shown survival rates as high as 75–100% [20,24].

EXIT nevertheless carries significant risks for mother; while uterine relaxation is warranted to ensure fetal blood supply, it can lead to uterine atony and excessive blood loss [20,24,25]. Moreover, because of the sometimes prolonged surgeries in less optimal sterile circumstances because of the involvement of multiple teams (neonatology, ENT, and obstetrics), maternal surgical site infections are more common and complicate up to 20% of cases [20,24,25].

These maternal risks have led to the development of alternative strategies for the management of fetal airways compromise: in cases where imaging studies do not provide certainty about airway patency, direct inspection of the fetal airway through fetoscopic laryngo-tracheoscopy may provide further information [26] and in some cases avoid an EXIT procedure. A fetoscopic airway inspection can also be complemented by a fetoscopic tracheal intubation, which consists in the insertion of an endotracheal tube into the fetal airway, through the cannula used for fetoscopy, followed by a routine cesarean section [27,28]. The most optimal treatment strategy needs to be individualized for each patient.

Postnatal outcomes are tumor dependent. Whereas teratomas and epignathus generally tend to be large and require postnatal debulking [18,29,30] with ensuing complications (severe defiguration, cranial nerve dysfunction, vocal cord dysfunction, and eating limitations), lymphovascular lesions and epulis are often less problematic and result in better functional and cosmetic outcomes.

Chest tumors

Lung tumors and masses

True fetal lung tumors are rare and need to be differentiated from the extremely common benign lung malformations, which include congenital cystic airway malformations (CCAM) and broncho-pulmonary sequestrations (BPS).

The latter 2 lesions consist of abnormally formed lung parenchyma and can be diagnosed in the second trimester of pregnancy as either solid or cystic lung masses on ultrasound [13]. These lung lesions will typically grow until ~26–28 weeks but then regress. Weekly to biweekly surveillance of

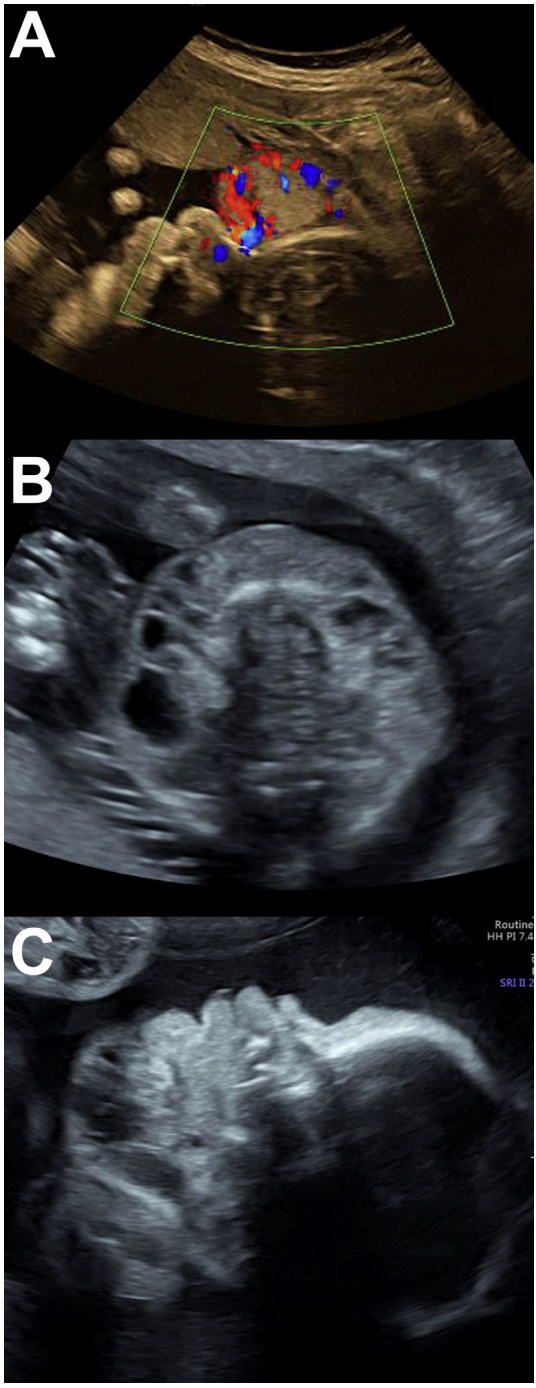

Fig. 2. Fetal head and neck tumors. (A) Sagittal view of a facial hemangioma at 35 weeks. (B) Axial view of a lymphangioma of the mandible at 21 weeks of gestation. (C) Sagittal view of the same lymphangioma at 28 weeks.

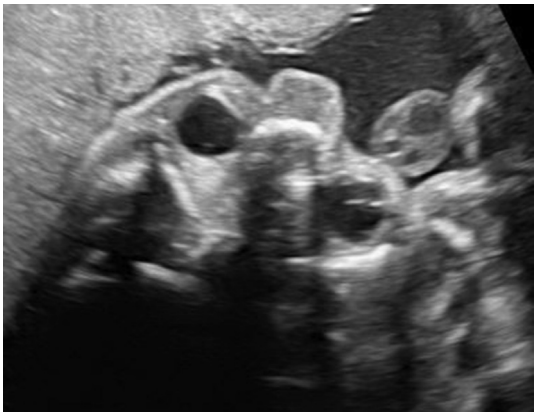

Fig. 3. Axial view of a nasal glioma at 27 weeks of gestation. Note the homogenous echogenic mass at the base of the nose.

mass growth by ultrasound is appropriate [31]. In case of rapid growth or associated pleural effusions, hydrops can develop as a consequence of mediastinal shift and increased intrathoracic pressure. The latter is associated with a poor prognosis and the risk of fetal or perinatal death then approaching 95% [32,33].

For very large lesions, in utero therapy has been described. In BPS, this typically involves ablation of the vascular supply to the lesion from the aorta, which can be achieved by intrafetal laser coagulation [34], coiling, or radiofrequency ablation [35,36]. Thoraco-amniotic shunting can be done for associated pleural effusions [35]. Case reports and small series show good outcomes with these strategies, but potential complications include damage to adjacent tissues, hyperkalemia, gas embolization, hyperthermia-induced hemolysis, PPROM, and preterm labor [36]. Intrafetal laser coagulation can require more than one intervention because of re-appearance of blood supply after the first intervention [37].

Large macrocystic CCAMs causing hydrops can be treated with thoraco-amniotic shunting [32]. Typically, a single shunt will be sufficient, as all cysts intercommunicate. Microcystic CCAMs are not eligible for shunting, but mass proliferation can be halted by administration of transplacental steroids, with resolution of hydrops in up to 80% of cases and decrease of the mass in 60% [38,39]. This has made treatment by open fetal surgery almost obsolete [32,33]. Results of recent studies of fetal therapy for lung lesions are summarized in Table 1.

Cardiac tumors

Fetal cardiac tumors generally only appear from 20 weeks of gestation onward and have an incidence of 11 in 10,000 pregnancies [40]. The most frequent fetal cardiac tumor is rhabdomyoma (Fig. 4), followed by fibroma, teratoma, myxoma, and hemangioma [13]. Rhabdomyomas tend to be multiple, whereas teratomas, fibromas, and hemangiomas are mostly unique [41]. Approximately 80% of fetuses with cardiac rhabdomyomas have tuberous sclerosis [42]. As such, the presence of multiple cardiac tumors should trigger genetic investigations and further neuroimaging looking for cerebral tubers. Small rhabdomyomas and fibromas are usually benign and regress over time [43], but large lesions (>2 cm) can cause ventricular dysfunction, outflow tract obstruction, and arrhythmia [40,41,43–50] [44,48,49]. Niewiadomicz et al. reported a 9% rate of fetal death [44]. Postnatally, mTOR inhibitors have been used as effective treatment for symptomatic rhabdomyomas, and this has recently also been reported in the fetal setting, with excellent results [51].

Teratomas, mostly arising from the pericardium, represent a challenge when detected before birth. They are often associated with large pericardial effusions, thereby exerting significant mass

Table 1
Outcomes reported in recent series on fetal therapy for lung lesions.

Author	Year	Diagnosis	N	Hydrops	Therapy	Outcome/survival
Mallmann [35]	2014	BPS	12	5	7 pleuro-amniotic shunting 5 laser	6/7 survival at birth, 2/7 partial regression, no complete regression. 5/5 survival, 5/5 partial or complete regression. No postnatal surgery.
Cruz-Martinez [96]	2015	BPS	8		8 laser	8/8 partial regression, 8/8 survival. No postnatal surgery.
Cruz- Martinez [97]	2017	BPS	15	4	15 laser	15/15 survival at birth, partial regression, and disappearance of fetal effusions. No postnatal surgery
Wu [98]	2017	Macrocystic BPS	1	1	Cyst aspiration, OK-432 injection, thoraco-amniotic shunting	1/1 Resolution of hydrops, live birth. Postnatal surgery at 5 months.
Gottschalk [37]	2017	BPS	12	12	12 laser	11/12 partial or complete regression. 1/12 failure of treatment. 2/11 postnatal surgery.
Perrenteau [99]	2017	Macrocystic CCAM	38	28	38 thoraco-amniotic shunting	36/38 survival at birth, 25/28 hydrops resolution. All postnatal surgery.
Litwinska [100]	2017	Macrocystic CCAM	12	8	12 thoraco-amniotic shunting	10/12 survival at birth, 9/12 postnatal surgery.
Cruz-Martinez	2015	CCAM	1	1	1 fetal bronchoscopy and laser permeabilization of bronchial atresia	1/1 survival, resolution of hydrops and contralateral hypoplastic lung.
Cruz-Martinez [101]	2017	CCAM	5	1	1 laser	3/5 progressive regression, 2/5 postnatal surgery
Min [102]	2014	CCAM	6	1	6 cystic shunting 1 OK-432 injection 1 cyst aspiration	6/6 survival at birth, 6/6 postnatal surgery
Walker [103]	2017	CCAM	7	3	7 thoracoamniotic shunting	5/7 survival at birth

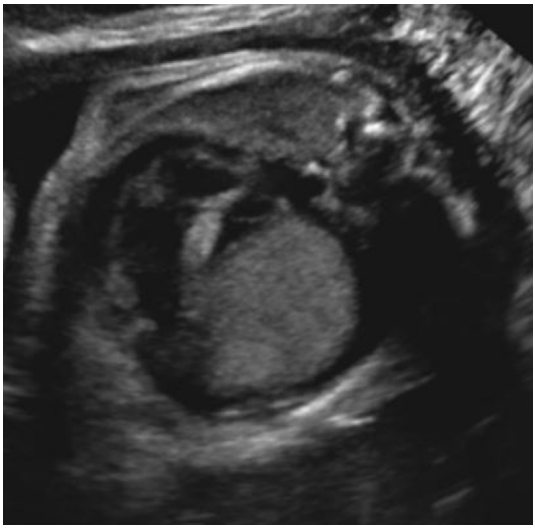

Fig. 4. Large cardiac rhabdomyoma in the left ventricular wall. Note the echogenicity of the interventricular septum, which holds a second lesion.

effect on the heart and lungs. Prenatal treatment options include expectant management, pericardiocentesis or shunting [52], open fetal resection, or delivery and postnatal surgery if the pregnancy is sufficiently advanced [48]. Given the rarity of these tumors, only small case series are available documenting outcomes. Nassr et al. systematically reviewed the literature, gathering 67 cases of fetal intrapericardial teratomas [53]. Of these cases, 69% presented with hydrops and 25.4% with polyhydramnios. In the series, 21 fetuses underwent pericardiocentesis or shunting, two underwent laser ablation of the vessels in the teratoma (both alive at 10 months), one had EXIT to resection (alive at age of 5 years), and 3 had open fetal resection (2 survived until birth). Of 40 singleton fetuses with hydrops, survival to birth was 72% among those who underwent an in utero intervention (pericardiocentesis/shunting) compared to only 36% in those who did not. Overall, 72% of the fetuses were alive at birth (58% in the hydropic group and 95% in the nonhydropic group), and postnatal survival was 90% [53].

Abdominal tumors

Hepatic tumors

The most frequent fetal and neonatal hepatic tumors are hemangiomas (60%) (Fig. 5), mesenchymal hamartoma (23%), and hepatoblastoma (17%) [54].

Hepatic hemangiomas can be unique or multiple. Small hemangiomas typically present as an echogenic focus in the liver [13], but larger lesions show as a well-circumscribed heterogeneous mass on ultrasound [13]. Larger lesions may also cause hepatomegaly, and some will induce a consumptive coagulopathy and microangiopathic hemolysis (Kasabach-Merritt syndrome) leading to thrombocytopenia and anemia [55]. This may present as high middle cerebral artery peak systolic velocity, elevated cardiac output, heart failure, and – in advanced stages – hydrops [55,56]. In rare cases, hepatic hemangioendothelioma can be associated with disseminated hemangiomatosis and Beckwith-Wiedemann syndrome [55]. Postnatal treatment of small liver hemangiomas is usually conservative, and some will regress spontaneously. In the presence of large lesions, prednisolone and beta-blockers are the first choice postnatally [55,56]. Surgery is typically reserved for lesions resistant to medical therapy [55–57]. In the presence of large lesions causing Kasabach-Merritt syndrome or heart failure antenatally, corticosteroids (betamethasone or dexamethasone) have been tried, success is variable [57–60].

Hepatic mesenchymal hamartoma is a benign proliferation of mesenchymal hepatic tissue with large cysts [13,57]. The echographic appearance of this tumor is mainly mixed and cystic and is to be differentiated from hemangiomas or liver cysts [13]. It has been associated with placental mesenchymal dysplasia [57]. When associated with placental mesenchymal dysplasia, poor outcome with 3 out of 8 cases who did not survive has been reported in a review by Harris et al. The deaths were caused by umbilical vein compression and heart failure [61]. The natural evolution of hepatic hamartoma is variable. Rapid growth, as well as spontaneous regression, have been reported [62]. Antenatal treatment options are limited [57]. Postnatal surgical resection is the treatment of choice, if the tumor is symptomatic and does not spontaneously regress [57,62].

Fetal hepatoblastoma is very rare but is the most common liver malignancy in childhood [63]. It is an epithelial, rapidly growing, malignant tumor [57,63] usually diagnosed late in pregnancy [63–66]. On ultrasound, it appears as an echogenic well-defined lesion, possibly with calcifications [13]. Hepatoblastoma carries a poor prognosis, with fetal survival estimated at approximately 22% [54]. Related complications, include compression of the inferior vena cava, fetal hydrops, respiratory distress, metastases, anemia due to tumor bleeding, consumptive coagulopathy, and thrombocytopenia [54,64,65]. Hepatoblastomas are associated with preterm birth and low birth weight [67]. Oue et al. reported an incidence of hepatoblastoma of 0.5% among extremely low birth weight children [68]. The treatment of choice is postnatal surgery, eventually after neoadjuvant chemotherapy [63,64,69].

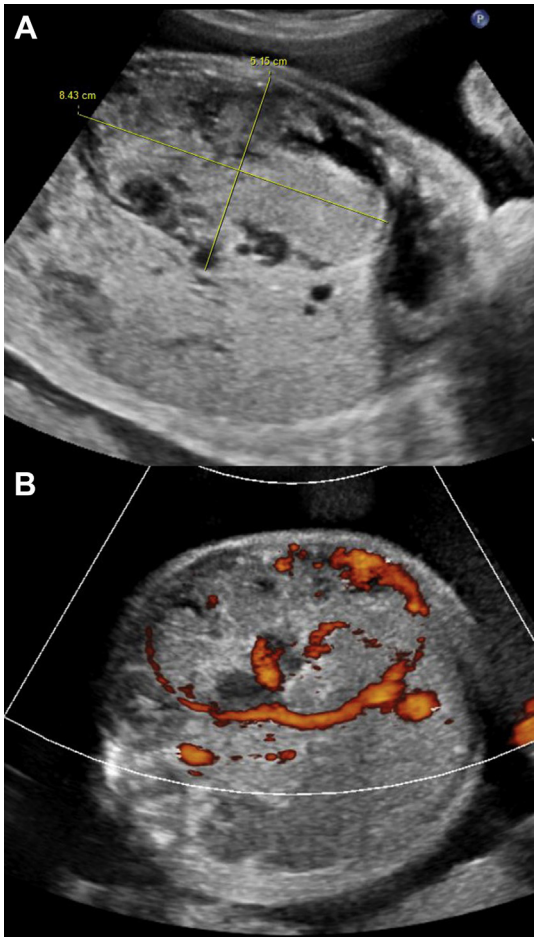

Fig. 5. (A) Sagittal and (B) axial view of a large liver mass, compatible with a hemangioendothelioma. Note the vascularity, which, in this case, induced a Kasabach-Merritt syndrome.

Renal and adrenal tumors

Congenital renal tumors are rare and account for less than 10% of the neonatal tumors. The most common solid congenital kidney tumor is congenital mesoblastic nephroma [70]. Congenital mesoblastic nephroma is a benign, large, well-circumscribed tumor. When it is prenatally diagnosed, its echographic aspect is an enlarging, homogeneous mass arising from the kidney usually in the third trimester [70–72]. Congenital mesoblastic nephroma is associated with polyhydramnios [71,72] in up to 40% of cases and therefore preterm labor [70]. Other complications include the development of hemorrhagic and necrotic cysts [72], hydrops, postnatal hemodynamic instability, and hypertension [70]. Being a benign tumor, congenital mesoblastic nephroma should be surgically resected. Leclair et al. report excellent oncological outcomes after postnatal surgical treatment with only 1 postoperative death out of 27 cases [70].

Nephroblastoma (Wilms Tumor) is extremely rare in the neonate, and thus even more prenatally. Ultrasound finding is generally a heterogeneous mass arising from the kidney. Ultrasound is nevertheless insufficient to distinguish nephroblastoma from congenital mesoblastic nephroma [70]. Ritchey et al. retrospectively studied 15 cases of nephroblastoma, of which only 3 were diagnosed antenatally. Obstetrical complications related to Wilms tumor include polyhydramnios because of decreased fetal

swallowing caused by abdominal compression, hydrops, fetal distress, anemia, ascites, and stillbirths have been described [73,74]. Postnatal treatment consists of surgery and eventual chemotherapy [73].

Neuroblastoma

Neuroblastoma has an incidence of 38.5 per million in children under 1 year of age [75]. Almost all of the prenatally diagnosed neuroblastomas are adrenal in origin [76,77]. The echographic aspect of neuroblastoma is a suprarenal, usually hyperechoic, solid or cystic mass, typically occurring in the third trimester of pregnancy [13,62,64]. The differential diagnosis of neuroblastoma includes adrenal hemorrhage, bronchogenic cysts, and infradiaphragmatic pulmonary sequestration [13,78]. Neuroblastomas are not associated with specific obstetric complications and treatment can be postponed until after birth. Postnatal treatment consists of chemotherapy and surgery, but conservative management can be chosen in selected cases, as spontaneous regression has been documented in up to 60% [79–81]. [82]. Neuroblastoma in fetuses carries a better prognosis than in children because it is more rarely associated with MYCN-oncogene or diploidy, which is associated to unfavorable outcomes. Also, cystic neuroblastoma, a localized form of neuroblastoma associated with favorable histology and biology, occurs mainly perinatally and carries the highest survival rate [83]. Postnatal survival rate ranges from 75% [83] to 100% [78,81].

Pelvic cavity tumors

Sacrococcygeal teratoma (SCT) is the most common congenital tumor and has a prevalence of 1 in 27,000 live births, with a female predominance [84]. It has been classified into 4 types according to the extent of the intrapelvic extension of the tumor. Type 1 is mostly external (Fig. 6) and type 4 is located in the pelvis with intra-abdominal extension. This classification is mainly relevant regarding the ease of postnatal surgical resection [85]. While the outcome of SCT is considered good in children [86], with survival rates of approximately 95%, prenatally diagnosed SCTs carry a high rate of obstetrical complications [87,88].

Indeed, large cystic lesions can cause prematurity because of their mass effect [89]. Very large cystic lesions can be drained or shunted prenatally, either to prevent preterm birth or to facilitate (cesarean) delivery. Overall, however, the outcome of cystic SCTs is excellent, with postnatal survival rates close to 100% in different series [88,89].

Large solid SCTs, on the other hand, are often highly vascularized and can cause high-output cardiac failure, as the tumor acts as a large arteriovenous shunt [90]. Moreover, fetal anemia can be observed because of hemolysis in the tumor bed. Clinically, heart failure and anemia will present as polyhydramnios, hydrops, and ultimately preterm birth or fetal death. Tumor size is an indicator of these complication risks and can be used in combination with cardiac output measurement to identify cases that would benefit from an intervention [91]. A solid tumor volume-to-head volume ratio >0.12, tumor volume-to-fetal weight ratio >0.12 prior to 24 weeks, rapid tumor growth, and high cardiac output have all been identified as predictors of cardiac failure and hydrops [87] and also of more difficult delivery and maternal complications [92].

The different prenatal treatment options explored for these solid lesions can be subdivided into minimally invasive options aimed at reducing the vascular supply (coiling, embolization, sclerotherapy, laser, and radiofrequency ablation) and open fetal resection. These interventions are typically reserved for hydropic fetuses in the previable period. A recent systematic review of the literature on fetal interventions for solid SCTs shows 44% survival after minimally invasive procedures [93]. Survival was lower in cases with hydrops than in nonhydropic cases (67% versus 45%). The complications associated with minimally invasive treatments are prematurity (mean gestational age at intervention was 23.2 weeks, and mean gestational age at birth was 29.7 weeks) and collateral damage to the skin, pelvic bones, musculature, and nerves. Survival after open fetal surgery was 55% [93], with a mean gestational age at delivery of 29.8 weeks (mean gestational age at intervention was 24.6 weeks). Complications of open fetal surgery included rectal fistulas and wound dehiscence [89], in addition to the usual fetal and maternal complications of open fetal surgery (preterm birth, PPROM, and uterine scarring) [94].

Fig. 6. Three-dimensional rendering of a fetal sacrococcygeal teratoma at 23 weeks.

Given the suboptimal success rates and significant complications seen with fetal therapy, early delivery and postnatal care should certainly be considered after 27 weeks [95].

Summary

We here reviewed the most common fetal tumors, their prenatal management, and their outcomes. Overall, the most important outcome predictors are tumor histology, tumor size, and location. Very large lesions, lesions causing cardiac failure, and hydrops and lesions obstructing the fetal airway have the poorest outcomes. Given the rarity of fetal tumors, patients are best referred to expert centers where care can be optimized and individualized to allow for the best possible outcomes.

Practice points

- Fetal tumors are often diagnosed late in pregnancy, and final histologic diagnosis and prognosis can often not be determined until after birth.
- Prenatal diagnosis is important to plan timing, location, and mode of birth.
- The prenatal management of fetal tumors depends on tumor size, vascularity, location, and signs of fetal decompensation.
- Small tumors are usually managed conservatively, but large lesions or lesions causing fetal decompensation may benefit from prenatal intervention.
- Given the rarity of fetal tumors, referral to expert centers is required to optimize and individualize care.

Research agenda

- Development of new methods to treat fetal tumors minimally invasively (including high-intensity focused ultrasound or transplacental medical therapy)
- Transplacental medical treatment.
- Imaging techniques to confirm diagnosis noninvasively

Conflicts of interest

The authors have no conflicts of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bpobgyn.2019.01.006.

References

[1] Halperin EC. Neonatal neoplasms. Int J Radiat Oncol Biol Phys 2000;47:171–8.
[2] Moore SW, Satgé D, Sasco AJ, Zimmermann A, Plaschkes J. The epidemiology of neonatal tumours. Report of an international working group. Pediatr Surg Int 2003;19:509–19. https://doi.org/10.1007/s00383-003-1048-8.
[3] McPherson E, Cold C, Johnson P, Schema L, Zaleski C. Neuroblastoma in a 17-week fetus: a stimulus for investigation of tumors in a series of 2786 stillbirth and late miscarriages. Am J Med Genet A 2015;167A:246–9. https://doi.org/10.1002/ajmg.a.36829.
[4] Sbragia L, Paek BW, Feldstein VA, Farrell JA, Harrison MR, Albanese CT, et al. Outcome of prenatally diagnosed solid fetal tumors. J Pediatr Surg 2001;36:1244–7. https://doi.org/10.1053/jpsu.2001.25785.
[5] Rickert CH. Neuropathology and prognosis of foetal brain tumours. Acta Neuropathol 1999;98:567–76.
[6] Buetow PC, Smirniotopoulos JG, Done S. Congenital brain tumors: a review of 45 cases. AJR Am J Roentgenol 1990;155:587–93. https://doi.org/10.2214/ajr.155.3.2167004.
[7] Isaacs H. Fetal brain tumors: a review of 154 cases. Am J Perinatol 2009;26:453–66. https://doi.org/10.1055/s-0029-1214245.
[8] Carstensen H, Juhler M, Bøgeskov L, Laursen H. A report of nine newborns with congenital brain tumours. Childs Nerv Syst ChNS Off J Int Soc Pediatr Neurosurg 2006;22:1427–31. https://doi.org/10.1007/s00381-006-0115-6.
[9] Jurkiewicz E, Brożyna A, Grajkowska W, Bekiesińska-Figatowska M, Daszkiewicz P, Nowak K, et al. Congenital brain tumors in a series of 56 patients. Childs Nerv Syst ChNS Off J Int Soc Pediatr Neurosurg 2012;28:1193–201. https://doi.org/10.1007/s00381-012-1798-5.
[10] Cassart M, Bosson N, Garel C, Eurin D, Avni F. Fetal intracranial tumors: a review of 27 cases. Eur Radiol 2008;18:2060–6. https://doi.org/10.1007/s00330-008-0999-5.
[11] Cavalheiro S, Moron AF, Hisaba W, Dastoli P, Silva NS. Fetal brain tumors. Childs Nerv Syst ChNS Off J Int Soc Pediatr Neurosurg 2003;19:529–36. https://doi.org/10.1007/s00381-003-0770-9.
[12] Manoranjan B, Provias JP. Congenital brain tumors: diagnostic pitfalls and therapeutic interventions. J Child Neurol 2011;26:599–614. https://doi.org/10.1177/0883073810394848.
[13] Cho JY, Lee YH. Fetal tumors: prenatal ultrasonographic findings and clinical characteristics. Ultrason Seoul Korea 2014;33:240–51. https://doi.org/10.14366/usg.14019.
[14] Isaacs H. II. Perinatal brain tumors: a review of 250 cases. Pediatr Neurol 2002;27:333–42.
[15] den Hollander NS, Vinkesteijn A, Schmitz-van Splunder P, Catsman-Berrevoets CE, Wladimiroff JW. Prenatally diagnosed fetal ventriculomegaly; prognosis and outcome. Prenat Diagn 1998;18:557–66.
[16] Woodward PJ, Sohaey R, Kennedy A, Koeller KK. From the archives of the AFIP: a comprehensive review of fetal tumors with pathologic correlation. Radiogr Rev Publ Radiol Soc N Am Inc 2005;25:215–42. https://doi.org/10.1148/rg.251045156.
[17] De Backer A, Madern GC, van de Ven CP, Tibboel D, Hazebroek FWJ. Strategy for management of newborns with cervical teratoma. J Perinat Med 2004;32:500–8. https://doi.org/10.1515/JPM.2004.122.
[18] Azizkhan RG, Haase GM, Applebaum H, Dillon PW, Coran AG, King PA, et al. Diagnosis, management, and outcome of cervicofacial teratomas in neonates: a Childrens Cancer Group study. J Pediatr Surg 1995;30:312–6.

[19] Tonni G, Granese R, Martins Santana EF, Parise Filho JP, Bottura I, Borges Peixoto A, et al. Prenatally diagnosed fetal tumors of the head and neck: a systematic review with antenatal and postnatal outcomes over the past 20 years. J Perinat Med 2017;45:149–65. https://doi.org/10.1515/jpm-2016-0074.
[20] Laje P, Johnson MP, Howell LJ, Bebbington MW, Hedrick HL, Flake AW, et al. Ex utero intrapartum treatment in the management of giant cervical teratomas. J Pediatr Surg 2012;47:1208–16. https://doi.org/10.1016/j.jpedsurg.2012.03.027.
[21] Hirose S, Sydorak RM, Tsao K, Cauldwell CB, Newman KD, Mychaliska GB, et al. Spectrum of intrapartum management strategies for giant fetal cervical teratoma. J Pediatr Surg 2003;38:446–50. https://doi.org/10.1053/jpsu.2003.50077. Discussion 446-450.
[22] Ryan G, Somme S, Crombleholme TM. Airway compromise in the fetus and neonate: prenatal assessment and perinatal management. Semin Fetal Neonatal Med 2016;21:230–9. https://doi.org/10.1016/j.siny.2016.03.002.
[23] Martino F, Avila LF, Encinas JL, Luis AL, Olivares P, Lassaletta L, et al. Teratomas of the neck and mediastinum in children. Pediatr Surg Int 2006;22:627–34. https://doi.org/10.1007/s00383-006-1724-6.
[24] Laje P, Peranteau WH, Hedrick HL, Flake AW, Johnson MP, Moldenhauer JS, et al. Ex utero intrapartum treatment (EXIT) in the management of cervical lymphatic malformation. J Pediatr Surg 2015;50:311–4. https://doi.org/10.1016/j.jpedsurg.2014.11.024.
[25] Laje P, Howell LJ, Johnson MP, Hedrick HL, Flake AW, Adzick NS. Perinatal management of congenital oropharyngeal tumors: the ex utero intrapartum treatment (EXIT) approach. J Pediatr Surg 2013;48:2005–10. https://doi.org/10.1016/j.jpedsurg.2013.02.031.
[26] Shamshirsaz AA, Nassr AA, Erfani H, Olutoye OA, Espinoza J, Olutoye OO, et al. Fetoscopic laryngo-tracheoscopy: a novel diagnostic modality to avoid unnecessary ex-utero intrapartum treatment (EXIT) in cases with suspected fetal airway compromise. Ultrasound Obstet Gynecol Off J Int Soc Ultrasound Obstet Gynecol 2018. https://doi.org/10.1002/uog.19033.
[27] Chmait RH, Chon AH, Anselmo D, Vanderbilt DL, Townsend J, Julian-Wang B, et al. In utero fetal intubation for a large neck mass: a minimally invasive EXIT option. Fetal Diagn Ther 2018. https://doi.org/10.1159/000487394.
*[28] Cruz-Martinez R, Moreno-Alvarez O, Garcia M, Méndez A, Pineda H, Cruz-Martinez MA, et al. Fetal endoscopic tracheal intubation: a new fetoscopic procedure to ensure extrauterine tracheal permeability in a case with congenital cervical teratoma. Fetal Diagn Ther 2015;38:154–8. https://doi.org/10.1159/000362387.
[29] Steigman SA, Nemes L, Barnewolt CE, Estroff JA, Valim C, Jennings RW, et al. Differential risk for neonatal surgical airway intervention in prenatally diagnosed neck masses. J Pediatr Surg 2009;44:76–9. https://doi.org/10.1016/j.jpedsurg.2008.10.014.
[30] Sheikh F, Akinkuotu A, Olutoye OO, Pimpalwar S, Cassady CI, Fernandes CJ, et al. Prenatally diagnosed neck masses: long-term outcomes and quality of life. J Pediatr Surg 2015;50:1210–3. https://doi.org/10.1016/j.jpedsurg.2015.02.035.
[31] Macardle CA, Ehrenberg-Buchner S, Smith EA, Dillman JR, Mychaliska GB, Treadwell MC, et al. Surveillance of fetal lung lesions using the congenital pulmonary airway malformation volume ratio: natural history and outcomes. Prenat Diagn 2016;36:282–9. https://doi.org/10.1002/pd.4761.
[32] Cavoretto P, Molina F, Poggi S, Davenport M, Nicolaides KH. Prenatal diagnosis and outcome of echogenic fetal lung lesions. Ultrasound Obstet Gynecol Off J Int Soc Ultrasound Obstet Gynecol 2008;32:769–83. https://doi.org/10.1002/uog.6218.
[33] Grethel EJ, Wagner AJ, Clifton MS, Cortes RA, Farmer DL, Harrison MR, et al. Fetal intervention for mass lesions and hydrops improves outcome: a 15-year experience. J Pediatr Surg 2007;42:117–23. https://doi.org/10.1016/j.jpedsurg.2006.09.060.
[34] Oepkes D, Devlieger R, Lopriore E, Klumper FJCM. Successful ultrasound-guided laser treatment of fetal hydrops caused by pulmonary sequestration. Ultrasound Obstet Gynecol Off J Int Soc Ultrasound Obstet Gynecol 2007;29:457–9. https://doi.org/10.1002/uog.3984.
[35] Mallmann MR, Geipel A, Bludau M, Matil K, Gottschalk I, Hoopmann M, et al. Bronchopulmonary sequestration with massive pleural effusion: pleuroamniotic shunting vs intrafetal vascular laser ablation. Ultrasound Obstet Gynecol Off J Int Soc Ultrasound Obstet Gynecol 2014;44:441–6. https://doi.org/10.1002/uog.13304.
*[36] Baud D, Windrim R, Kachura JR, Jefferies A, Pantazi S, Shah P, et al. Minimally invasive fetal therapy for hydropic lung masses: three different approaches and review of the literature. Ultrasound Obstet Gynecol Off J Int Soc Ultrasound Obstet Gynecol 2013;42:440–8. https://doi.org/10.1002/uog.12515.
[37] Gottschalk I, Strizek B, Mallmann MR, Müller A, Geipel A, Gembruch U, et al. Outcome of bronchopulmonary sequestration with massive pleural effusion after intrafetal vascular laser ablation. Fetal Diagn Ther 2017. https://doi.org/10.1159/000479793.
[38] Curran PF, Jelin EB, Rand L, Hirose S, Feldstein VA, Goldstein RB, et al. Prenatal steroids for microcystic congenital cystic adenomatoid malformations. J Pediatr Surg 2010;45:145–50. https://doi.org/10.1016/j.jpedsurg.2009.10.025.
[39] Vu L, Tsao K, Lee H, Nobuhara K, Farmer D, Harrison M, et al. Characteristics of congenital cystic adenomatoid malformations associated with nonimmune hydrops and outcome. J Pediatr Surg 2007;42:1351–6. https://doi.org/10.1016/j.jpedsurg.2007.03.039.
[40] Groves AM, Fagg NL, Cook AC, Allan LD. Cardiac tumours in intrauterine life. Arch Dis Child 1992;67:1189–92.
[41] D'Addario V, Pinto V, Di Naro E, Del Bianco A, Di Cagno L, Volpe P. Prenatal diagnosis and postnatal outcome of cardiac rhabdomyomas. J Perinat Med 2002;30:170–5. https://doi.org/10.1515/JPM.2002.022.
[42] Yinon Y, Chitayat D, Blaser S, Seed M, Amsalem H, Yoo S-J, et al. Fetal cardiac tumors: a single-center experience of 40 cases. Prenat Diagn 2010;30:941–9. https://doi.org/10.1002/pd.2590.
[43] Holley DG, Martin GR, Brenner JI, Fyfe DA, Huhta JC, Kleinman CS, et al. Diagnosis and management of fetal cardiac tumors: a multicenter experience and review of published reports. J Am Coll Cardiol 1995;26:516–20.
[44] Niewiadomska-Jarosik K, Stańczyk J, Janiak K, Jarosik P, Moll JJ, Zamojska J, et al. Prenatal diagnosis and follow-up of 23 cases of cardiac tumors. Prenat Diagn 2010;30:882–7. https://doi.org/10.1002/pd.2586.
[45] Geipel A, Krapp M, Germer U, Becker R, Gembruch U. Perinatal diagnosis of cardiac tumors. Ultrasound Obstet Gynecol Off J Int Soc Ultrasound Obstet Gynecol 2001;17:17–21. https://doi.org/10.1046/j.1469-0705.2001.00314.x.
[46] Pucci A, Botta G, Sina N, Tibaldi M, Valori A, Grosso E, et al. Life-threatening tumors of the heart in fetal and postnatal age. J Pediatr 2013;162:964–9. https://doi.org/10.1016/j.jpeds.2012.10.055. e1.

[47] Zhou QC, Fan P, Peng QH, Zhang M, Fu Z, Wang CH. Prenatal echocardiographic differential diagnosis of fetal cardiac tumors. Ultrasound Obstet Gynecol Off J Int Soc Ultrasound Obstet Gynecol 2004;23:165–71. https://doi.org/10.1002/uog.979.
[48] Rychik J, Khalek N, Gaynor JW, Johnson MP, Adzick NS, Flake AW, et al. Fetal intrapericardial teratoma: natural history and management including successful in utero surgery. Am J Obstet Gynecol 2016;215:780.e1–7. https://doi.org/10.1016/j.ajog.2016.08.010.
[49] Chao AS, Chao A, Wang TH, Chang YC, Chang YL, Hsieh CC, et al. Outcome of antenatally diagnosed cardiac rhabdomyoma: case series and a meta-analysis. Ultrasound Obstet Gynecol Off J Int Soc Ultrasound Obstet Gynecol 2008;31:289–95. https://doi.org/10.1002/uog.5264.
[50] Bruch SW, Adzick NS, Reiss R, Harrison MR. Prenatal therapy for pericardial teratomas. J Pediatr Surg 1997;32:1113–5.
[51] Barnes BT, Procaccini D, Crino J, Blakemore K, Sekar P, Sagaser KG, et al. Maternal sirolimus therapy for fetal cardiac rhabdomyomas. N Engl J Med 2018;378:1844–5. https://doi.org/10.1056/NEJMc1800352.
[52] Devlieger R, Hindryckx A, Van Mieghem T, Debeer A, De Catte L, Gewillig M, et al. Therapy for foetal pericardial tumours: survival following in utero shunting, and literature review. Fetal Diagn Ther 2009;25:407–12. https://doi.org/10.1159/000236156.
[53] Nassr AA, Shazly SA, Morris SA, Ayres N, Espinoza J, Erfani H, et al. Prenatal management of fetal intrapericardial teratoma: a systematic review. Prenat Diagn 2017;37:849–63. https://doi.org/10.1002/pd.5113.
[54] Isaacs H. Fetal and neonatal hepatic tumors. J Pediatr Surg 2007;42:1797–803. https://doi.org/10.1016/j.jpedsurg.2007.07.047.
[55] Samuel M, Spitz L. Infantile hepatic hemangioendothelioma: the role of surgery. J Pediatr Surg 1995;30:1425–9.
[56] Alamo L, Perrin L, Vial Y, Anooshiravani M, Meuli R. Prenatal imaging of congenital hepatic tumors: a report of three cases. Clin Imaging 2017;41:112–7. https://doi.org/10.1016/j.clinimag.2016.10.003.
[57] Makin E, Davenport M. Fetal and neonatal liver tumours. Early Hum Dev 2010;86:637–42. https://doi.org/10.1016/j.earlhumdev.2010.08.023.
[58] Morris J, Abbott J, Burrows P, Levine D. Antenatal diagnosis of fetal hepatic hemangioma treated with maternal corticosteroids. Obstet Gynecol 1999;94:813–5.
[59] Sheu BC, Shyu MK, Lin YF, Lee CN, Hsieh FJ, Chou YH, et al. Prenatal diagnosis and corticosteroid treatment of diffuse neonatal hemangiomatosis: case report. J Ultrasound Med Off J Am Inst Ultrasound Med 1994;13:495–9.
[60] Mejides AA, Adra AM, O'Sullivan MJ, Nicholas MC. Prenatal diagnosis and therapy for a fetal hepatic vascular malformation. Obstet Gynecol 1995;85:850–3.
[61] Harris K, Carreon CK, Vohra N, Williamson A, Dolgin S, Rochelson B. Placental mesenchymal dysplasia with hepatic mesenchymal hamartoma: a case report and literature review. Fetal Pediatr Pathol 2013;32:448–53. https://doi.org/10.3109/15513815.2013.835893.
[62] Kamata S, Nose K, Sawai T, Hasegawa T, Kuroda S, Sasaki T, et al. Fetal mesenchymal hamartoma of the liver: report of a case. J Pediatr Surg 2003;38:639–41. https://doi.org/10.1053/jpsu.2003.50142.
[63] Huang L-C, Ho M, Chang W-C, Chen H-Y, Hung Y-C, Chiu T-H. Prenatal diagnosis of fetal hepatoblastoma with a good neonatal outcome: case report and narrative literature review. Pediatr Hematol Oncol 2011;28:150–4. https://doi.org/10.3109/08880018.2010.536299.
[64] Catanzarite V, Hilfiker M, Daneshmand S, Willert J. Prenatal diagnosis of fetal hepatoblastoma: case report and review of the literature. J Ultrasound Med Off J Am Inst Ultrasound Med 2008;27:1095–8.
[65] Ergin H, Yildirim B, Dagdeviren E, Yagci B, Ozen F, Sen N, et al. A prenatally detected case of congenital hepatoblastoma. Pathol Oncol Res 2008;14:97–100. https://doi.org/10.1007/s12253-008-9001-8.
[66] Shih JC, Tsao PN, Huang SF, Yen BL, Lin JH, Lee CN, et al. Antenatal diagnosis of congenital hepatoblastoma in utero. Ultrasound Obstet Gynecol Off J Int Soc Ultrasound Obstet Gynecol 2000;16:94–7. https://doi.org/10.1046/j.1469-0705.2000.00168.x.
[67] Feusner J, Plaschkes J. Hepatoblastoma and low birth weight: a trend or chance observation? Med Pediatr Oncol 2002;39:508–9. https://doi.org/10.1002/mpo.10176.
[68] Oue T, Kubota A, Okuyama H, Kawahara H, Nara K, Kawa K, et al. Hepatoblastoma in children of extremely low birth weight: a report from a single perinatal center. J Pediatr Surg 2003;38:134–7. https://doi.org/10.1053/jpsu.2003.50027. Discussion 134-137.
[69] Tiao GM, Bobey N, Allen S, Nieves N, Alonso M, Bucuvalas J, et al. The current management of hepatoblastoma: a combination of chemotherapy, conventional resection, and liver transplantation. J Pediatr 2005;146:204–11. https://doi.org/10.1016/j.jpeds.2004.09.011.
[70] Leclair M-D, El-Ghoneimi A, Audry G, Ravasse P, Moscovici J, Heloury Y, et al. The outcome of prenatally diagnosed renal tumors. J Urol 2005;173:186–9.
[71] Do AY, Kim J-S, Choi S-J, Oh S-Y, Roh C-R, Kim J-H. Prenatal diagnosis of congenital mesoblastic nephroma. Obstet Gynecol Sci 2015;58:405–8. https://doi.org/10.5468/ogs.2015.58.5.405.
[72] Jones VS, Cohen RC. Atypical congenital mesoblastic nephroma presenting in the perinatal period. Pediatr Surg Int 2007;23:205–9. https://doi.org/10.1007/s00383-006-1831-4.
[73] Sarin YK, Rahul SK, Sinha S, Khurana N, Ramji S. Antenatally diagnosed wilms' tumour. J Neonatal Surg 2014;3:8.
[74] Suresh I, Suresh S, Arumugam R, Govindarajan M, Reddy MP, Sulochana NV. Antenatal diagnosis of Wilms tumor. J Ultrasound Med Off J Am Inst Ultrasound Med 1997;16:69–72.
[75] Nakata K, Ito Y, Magadi W, Bonaventure A, Stiller CA, Katanoda K, et al. Childhood cancer incidence and survival in Japan and England: a population-based study (1993-2010). Cancer Sci 2018;109:422–34. https://doi.org/10.1111/cas.13457.
[76] Acharya S, Jayabose S, Kogan SJ, Tugal O, Beneck D, Leslie D, et al. Prenatally diagnosed neuroblastoma. Cancer 1997;80:304–10.
[77] Granata C, Fagnani AM, Gambini C, Boglino C, Bagnulo S, Cecchetto G, et al. Features and outcome of neuroblastoma detected before birth. J Pediatr Surg 2000;35:88–91.
[78] Sauvat F, Sarnacki S, Brisse H, Medioni J, Rubie H, Aigrain Y, et al. Outcome of suprarenal localized masses diagnosed during the perinatal period: a retrospective multicenter study. Cancer 2002;94:2474–80. https://doi.org/10.1002/cncr.10502.

[79] Ikeda H, Suzuki N, Takahashi A, Kuroiwa M, Nagashima K, Tsuchida Y, et al. Surgical treatment of neuroblastomas in infants under 12 months of age. J Pediatr Surg 1998;33:1246–50.
[80] Kaneko M, Iwakawa M, Ikebukuro K, Ohkawa H. Complete resection is not required in patients with neuroblastoma under 1 year of age. J Pediatr Surg 1998;33:1690–4.
*[81] Cozzi DA, Mele E, Ceccanti S, Natale F, Clerico A, Schiavetti A, et al. Long-term follow-up of the "wait and see" approach to localized perinatal adrenal neuroblastoma. World J Surg 2013;37:459–65. https://doi.org/10.1007/s00268-012-1837-0.
[82] Hero B, Simon T, Spitz R, Ernestus K, Gnekow AK, Scheel-Walter H-G, et al. Localized infant neuroblastomas often show spontaneous regression: results of the prospective trials NB95-S and NB97. J Clin Oncol Off J Am Soc Clin Oncol 2008;26:1504–10. https://doi.org/10.1200/JCO.2007.12.3349.
[83] Isaacs H. Fetal and neonatal neuroblastoma: retrospective review of 271 cases. Fetal Pediatr Pathol 2007;26:177–84. https://doi.org/10.1080/15513810701696890.
[84] Swamy R, Embleton N, Hale J. Sacrococcygeal teratoma over two decades: birth prevalence, prenatal diagnosis and clinical outcomes. Prenat Diagn 2008;28:1048–51. https://doi.org/10.1002/pd.2122.
*[85] Altman RP, Randolph JG, Lilly JR. Sacrococcygeal teratoma: American academy of pediatrics surgical section survey-1973. J Pediatr Surg 1974;9:389–98.
[86] Rescorla FJ, Sawin RS, Coran AG, Dillon PW, Azizkhan RG. Long-term outcome for infants and children with sacrococcygeal teratoma: a report from the Childrens Cancer Group. J Pediatr Surg 1998;33:171–6.
[87] Wilson RD, Hedrick H, Flake AW, Johnson MP, Bebbington MW, Mann S, et al. Sacrococcygeal teratomas: prenatal surveillance, growth and pregnancy outcome. Fetal Diagn Ther 2009;25:15–20. https://doi.org/10.1159/000188056.
[88] Hedrick HL, Flake AW, Crombleholme TM, Howell LJ, Johnson MP, Wilson RD, et al. Sacrococcygeal teratoma: prenatal assessment, fetal intervention, and outcome. J Pediatr Surg 2004;39:430–8. Discussion 430-438.
*[89] Benachi A, Durin L, Vasseur Maurer S, Aubry M-C, Parat S, Herlicoviez M, et al. Prenatally diagnosed sacrococcygeal teratoma: a prognostic classification. J Pediatr Surg 2006;41:1517–21. https://doi.org/10.1016/j.jpedsurg.2006.05.009.
[90] Lee M-Y, Won H-S, Hyun M-K, Lee H-Y, Shim J-Y, Lee P-R, et al. Perinatal outcome of sacrococcygeal teratoma. Prenat Diagn 2011;31:1217–21. https://doi.org/10.1002/pd.2865.
[91] Westerburg B, Feldstein VA, Sandberg PL, Lopoo JB, Harrison MR, Albanese CT. Sonographic prognostic factors in fetuses with sacrococcygeal teratoma. J Pediatr Surg 2000;35:322–5. Discussion 325-326.
[92] Gebb JS, Khalek N, Qamar H, Johnson MP, Oliver ER, Coleman BG, et al. High tumor volume to fetal weight ratio is associated with worse fetal outcomes and increased maternal risk in fetuses with sacrococcygeal teratoma. Fetal Diagn Ther 2018. https://doi.org/10.1159/000486782.
*[93] Van Mieghem T, Al-Ibrahim A, Deprest J, Lewi L, Langer JC, Baud D, et al. Minimally invasive therapy for fetal sacrococcygeal teratoma: case series and systematic review of the literature. Ultrasound Obstet Gynecol Off J Int Soc Ultrasound Obstet Gynecol 2014;43:611–9. https://doi.org/10.1002/uog.13315.
[94] Al-Refai A, Ryan G, Van Mieghem T. Maternal risks of fetal therapy. Curr Opin Obstet Gynecol 2017;29:80–4. https://doi.org/10.1097/GCO.0000000000000346.
[95] Baumgarten HD, Gebb JS, Khalek N, Moldenhauer JS, Johnson MP, Peranteau WH, et al. Preemptive delivery and immediate resection for fetuses with high-risk sacrococcygeal teratomas. Fetal Diagn Ther 2018:1–8. https://doi.org/10.1159/000487542.
[96] Cruz-Martinez R, Méndez A, Dueñas-Riaño J, Ordorica-Flores R, Nieto-Zermeño J, Malagón-Salazar P, et al. Fetal laser surgery prevents fetal death and avoids the need for neonatal sequestrectomy in cases with bronchopulmonary sequestration. Ultrasound Obstet Gynecol Off J Int Soc Ultrasound Obstet Gynecol 2015;46:627–8. https://doi.org/10.1002/uog.14921.
[97] Cruz-Martínez R, Nieto-Castro B, Martínez-Rodríguez M, Gámez-Varela A, Ahumada-Angulo E, Luna-García J, et al. Thoracic changes after full laser ablation of the feeding artery in fetuses with bronchopulmonary sequestration. Fetal Diagn Ther 2017. https://doi.org/10.1159/000481170.
[98] Wu W-J, Shih J-C, Sago H, Chen M. Complete resolution of hydrops by placement of double basket catheter in a case of macrocystic type multilocular pulmonary sequestration. Taiwan J Obstet Gynecol 2017;56:402–5. https://doi.org/10.1016/j.tjog.2017.04.027.
[99] Peranteau WH, Adzick NS, Boelig MM, Flake AW, Hedrick HL, Howell LJ, et al. Thoracoamniotic shunts for the management of fetal lung lesions and pleural effusions: a single-institution review and predictors of survival in 75 cases. J Pediatr Surg 2015;50:301–5. https://doi.org/10.1016/j.jpedsurg.2014.11.019.
[100] Litwińska M, Litwińska E, Janiak K, Piaseczna-Piotrowska A, Gulczyńska E, Szaflik K. Thoracoamniotic shunts in macrocystic lung lesions: case series and review of the literature. Fetal Diagn Ther 2017;41:179–83. https://doi.org/10.1159/000448095.
[101] Cruz-Martinez R, Martínez-Rodríguez M, Bermúdez-Rojas M, Magaña-Abarca C, Narvaez-Dominguez V, Rojas-Macedo A, et al. Fetal laser ablation of feeding artery of cystic lung lesions with systemic arterial blood supply. Ultrasound Obstet Gynecol Off J Int Soc Ultrasound Obstet Gynecol 2017;49:744–50. https://doi.org/10.1002/uog.16011.
[102] Min J-Y, Won H-S, Lee M-Y, Suk H-J, Shim J-Y, Lee P-R, et al. Intrauterine therapy for macrocystic congenital cystic adenomatoid malformation of the lung. Obstet Gynecol Sci 2014;57:102–8. https://doi.org/10.5468/ogs.2014.57.2.102.
[103] Walker L, Cohen K, Rankin J, Crabbe D. Outcome of prenatally diagnosed congenital lung anomalies in the North of England: a review of 228 cases to aid in prenatal counselling. Prenat Diagn 2017;37:1001–7. https://doi.org/10.1002/pd.5134.