



# Complete regression of a huge fetal hepatic hemangioma with cardiac complication after atrioventricular septal repair: a case description and literature analysis

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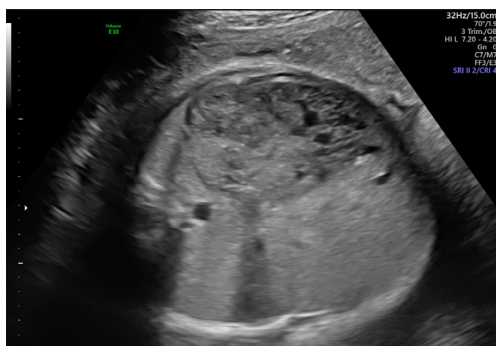
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## Introduction

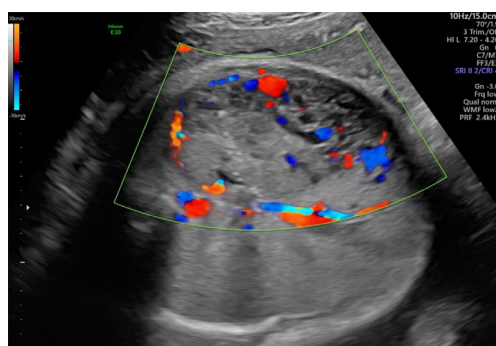
Hepatic hemangioma (HH) is the most common benign mesenchymal tumor of the liver in fetus and infants (1,2). According to International Society for the Study of Vascular Anomalies (ISSVA), HH was classified as congenital hepatic hemangioma (CHH) and infantile hepatic hemangioma (IHH) (3,4). Most CHH could regress partially or completely without therapies (5), but non-involuting CHH or progressive IHH may lead to serious complications, such as high-output congestive heart failure, cardiomegaly, non-immune hydrops, intratumoral bleeding, thrombocytopenia, and so on (6-8). Thus, positive therapies for non-involuting CHH and progressive IHH are needed. The most common therapy is oral propranolol with or without dexamethasone or rapamycin (9-13). Selective catheterization and coil embolization of the right and left hepatic and right phrenic arteries could be performed when the drug resistance appeared (13). If the patients have liver failure, surgery or liver transplantation needs to be considered (10,11,13-15). However, medical therapy has side effects, resistance, and no response of tumor (16). Embolization, surgery or liver transplantation is invasive. So we performed a literature analysis on the basis of the case, a rapidly regressed HH with cardiac surgery, to explore new strategies for treating similar cases in the future.

## Case presentation

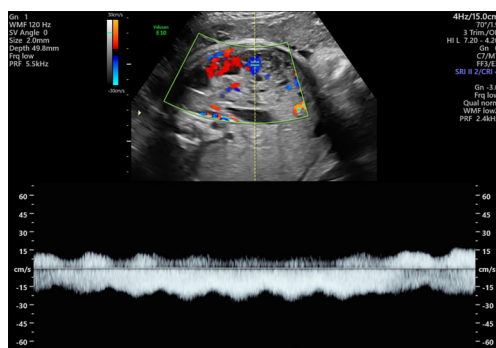
A 30-year-old pregnant woman was referred to West China Second Hospital for further diagnosis at 31 weeks of gestation because of a large fetal abdominal mass detected during a routine third-trimester scan. The first detection of the neoplasm was 29 weeks and 3 days of gestation by ultrasound examination in other hospital. In our hospital, ultrasound examination of the fetus revealed a well-delineated, hypoechoic, heterogeneous solid mass measuring 72 mm × 35 mm × 66 mm in close relation to the left hepatic lobe at 35 gestational weeks. The internal echo was cellular and there were irregular, echoless areas in center (*Figure 1*). The color Doppler showed that there exists abundant blood flow in and surrounding the mass with arteriovenous shunts (*Figures 2,3*). The drained veins were enlarged. Fetal MRI showed an extensive heterogeneous upper abdominal mass that compromised left hepatic lobe with flow-voids within it (*Figure 4*). According to the imaging features, the diagnosis of fetal HH was firstly considered. During the follow-up, fetal echocardiography at 34 weeks of gestation showed the possibility of ventricular septal defect (measuring 3 mm) without other complications. At 37 weeks of gestation, ultrasound showed that the fetal abdominal circumference exceeded 41 gestational week and the mass enlarged to 84 mm × 41 mm × 82 mm. However, no signs of hydrops,



**Figure 1** The prenatal two-dimensional ultrasound features of hepatic hemangioma in this case. Well-circumscribed, hypoechoic, and predominantly solid mass with echoless areas in center.

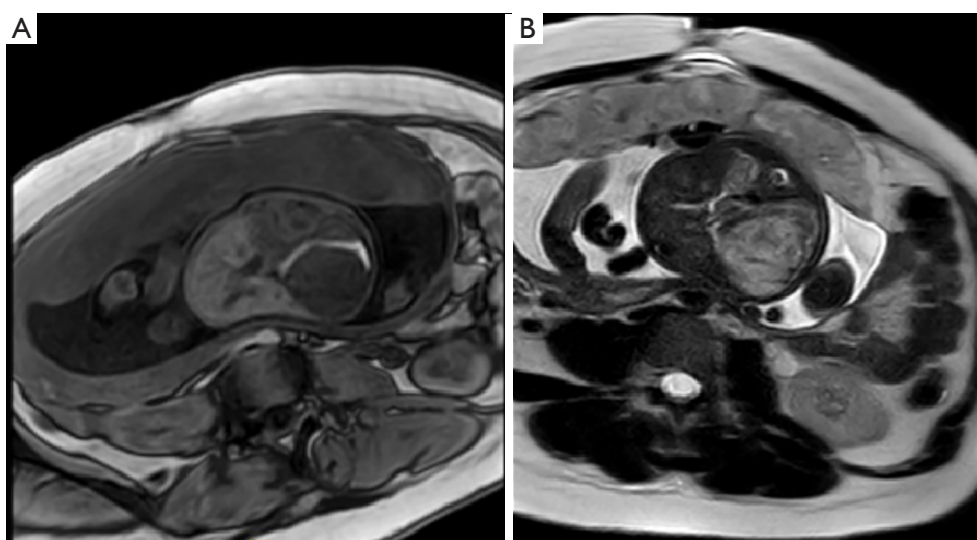


**Figure 2** The prenatal color-Doppler ultrasound of the tumor. The blood flow was abundant in and surrounding the mass and drained into dilated veins.

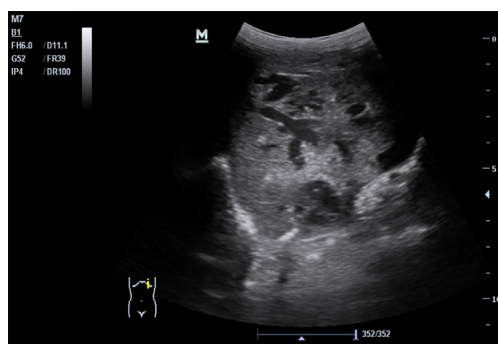


**Figure 3** There exist arteriovenous shunts in the mass.

cardiac insufficiency, or fetal anemia were noted. At 38 weeks of gestation, non-stress test (NST) demonstrated an unresponsive pattern and polyhydramnios, an elective cesarean section was performed delivering a female infant in breech presentation weighing 3,680 g. Apgar score was 10-10-10 at 1, 5 and 10 minutes, respectively. The infant was referred to division of neonatology because of abdominal distention and respiratory embarrassment. Her respiratory rate was 62–65 times per minute, and saturation of peripheral oxygen ( $\text{SpO}_2$ ) was 88–90%. The bedside ultrasound revealed a 71 mm × 65 mm × 85 mm heterogeneous left hepatic mass with tubular or reticular hypoechoic areas (*Figure 5*), supplied by affluent blood flow and drained by left hepatic vein. A postnatal computed tomography (CT) scan with contrast enhancement demonstrated a heterogeneous hepatic mass, behaving centripetal enhancement with central necrosis. There exists many vessels in the mass and drained into the inferior vein. Echocardiography showed atrioventricular septal defect, mitral, tricuspid regurgitation and pulmonary arterial hypertension. The blood test of white blood cell (WBC), hemoglobin (HGB), hematocrit (HCT), platelet (PLT) was normal, but alpha-fetoprotein (AFP) was 64,271.7 ng/mL and NSE was 33.00 ng/mL. The diagnosis of HH was strongly considered, but hemangioendothelioma, hepatoblastoma, germ cell tumour, neuroblastoma and mesenchymal hamartoma should be differentiated. Then the neonate received oral propranolol because of the cardiac complication and was monitored by ultrasound examination routinely. As the infant grew, the hepatic mass gradually shrank, however, the pulmonary artery pressure gradually increased and the left ventricular ejection fraction gradually decreased. When the infant was 4 months old, the size of the hepatic mass reduced to 37 mm × 41 mm × 50 mm. At the same time, echocardiography revealed severe pulmonary arterial hypertension and reduced left ventricular systolic function. Therefore, the infant received atrioventricular septal repair surgery. During the hospitalization following cardiac surgery, the HH was almost undetectable by bedside ultrasound (*Figure 6* was the postoperative imaging of 1 month). The interval since the last ultrasound examination was only 1 week. The baby was followed up by ultrasound until the age of 2 years and 4 months. The mass regressed



**Figure 4** The fetal magnetic resonance imaging characteristics of hepatic hemangioma. (A) Low, nonuniform signal intensity on T1-weighted images. (B) High signal intensity on T2-weighted images, with flow voids.



**Figure 5** The vascular malformation of hepatic hemangioma can present as tubular or reticular hypoechoic areas within the mass (neonate period).



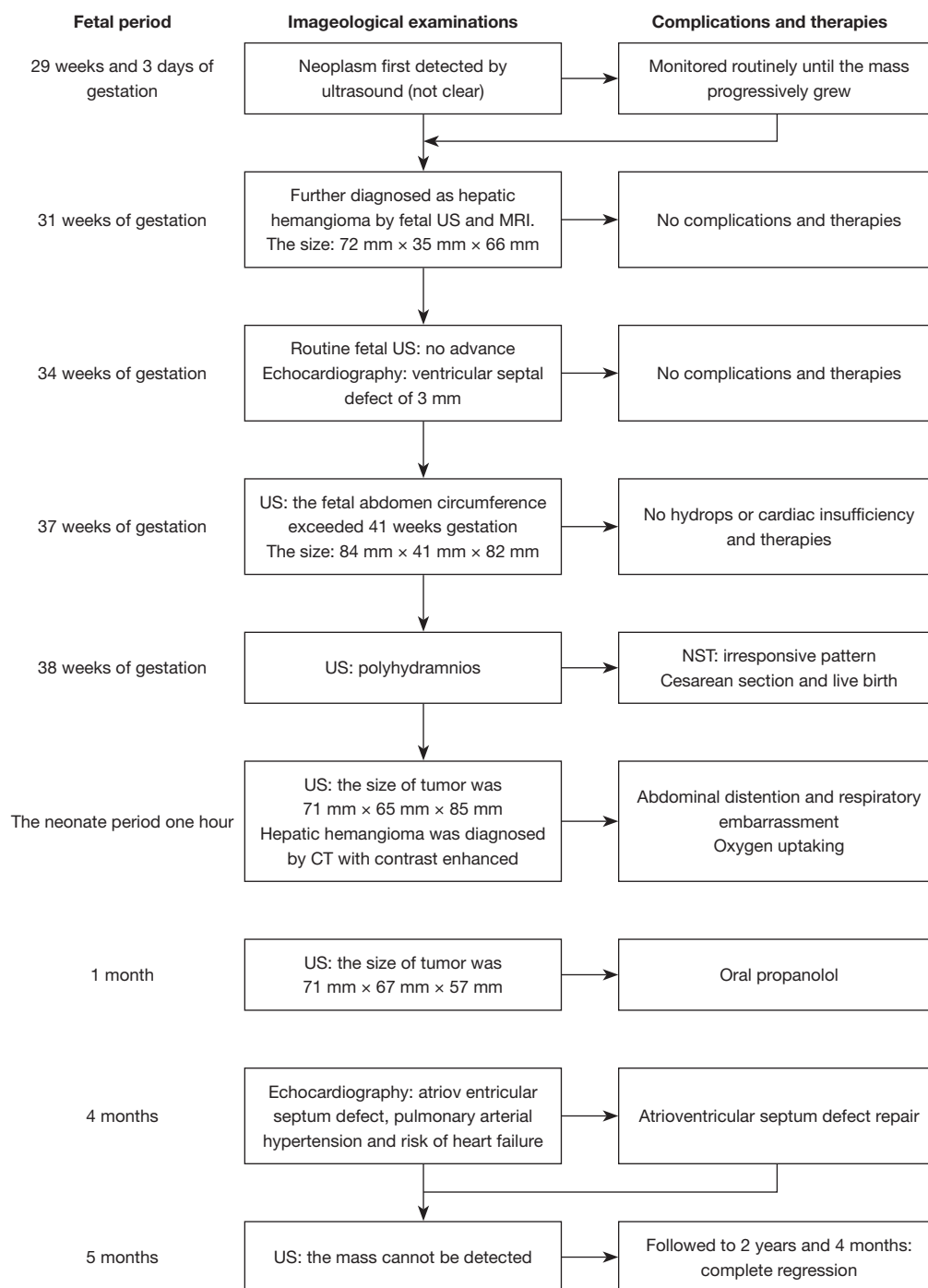
**Figure 6** The ultrasound imaging of the infant at 5 months.

completely and all the examinations were normal (the course was list in *Figure 7*).

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this article and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

## Discussion

There exists some research about the clinical manifestation, diagnosis, therapy and prognosis of fetal HH (9,17-19). Based on the guidance of the ISSVA, HHs are classified into CHH and IHH (3,4). CHH was common in fetus, as IHH in infancy. CHH was fully formed and present at birth, while IHH was detected incidentally (5). According to the “Guidance Document for Hepatic Hemangioma (Infantile and Congenital) Evaluation and Monitoring”, the diagnosis of hemangioma is often clear after a review of patient’s history, physical examination, and imaging (20-23), histologic confirmation is rarely required (19). If HH occurred after infancy, the imaging features were not classic or if the clinical history did not support a



**Figure 7** Course of disease (including diagnosis by imaging, complications and therapies during prenatal and neonate). CT, computed tomography; MRI, magnetic resonance imaging; NST, non-stress test; US, ultrasound.

hemangioma diagnosis, magnetic resonance imaging (MRI) of the liver with dynamic acquisition pattern for clarification was recommended and biopsy should be considered (19). Most majority of CHH usually regress spontaneously after delivery without severe complications (5). However, some huge congenital hemangiomas or progressive IHH may have poor prognosis if they appear serious complications (6-8). The common therapy methods include intrauterine and neonate treatments (continue monitoring, medical, interventional treatments and surgery) (10,11,14,16). After systematic monitoring and therapies, many hemangiomas could gradually reduce in size, even regress completely or partially, but some diffuse IHH without therapies may lead to death (24).

In this report, we presented a case of a giant fetal HH diagnosed prenatally using two-dimensional, color Doppler ultrasound and fetal MRI between 29 and 34 weeks' gestation. Although biopsy and immunohistochemical staining for glucose transporter protein-1 (GLUT1) was not performed to confirm the pathological type (25,26), according to the progress of genesis and development of lesion and typical features of ultrasound and MRI (27-30), the diagnosis of HH was not complex (RICH most possibly). Meanwhile, ventricular septal defect was also diagnosed by fetal echocardiography. The fetus was monitored closely without any complication. After the live birth of the fetus, the mass gradually shrank as the infant grew and the size of tumor was 37 mm × 41 mm × 50 mm. However, the pulmonary artery pressure gradually increased and the left ventricular systolic function decreased with atrioventricular septal defect, mitral and tricuspid regurgitation. To avoid the complication of cardiac failure, at 4 months, atrioventricular septal repair was selected and awesomely, the HH regressed completely during the hospitalization following cardiac surgery. The interval between the last ultrasound examination and the involution of the lesion was only 1 week.

Is it an occasional case or there exists correlation between atrioventricular septal defect and HH? If there were possible positive impacts on HH with heart complication through cardiac surgery? In order to explore probable mechanisms and potential added therapies for clinical experience, we need to compare the diagnosis, clinical symptoms, unique therapeutic method and prognosis of this case with other studies. For these purposes, we conducted an updated review of the literature for cases of HH. According to the ISSVA's classification of vascular tumors (International Society of Study of Vascular

Anomalies), a computerized review of the MEDLINE-PubMed database and China National Knowledge Infrastructure was performed using the terms "vascular anomalies", "hemangioma", "hemangioendothelioma" (as the classification may not be normative before 2014, this item was included for studies before that year), "hepatic and/or liver", "tumor and/or neoplasms", and "congenital and/or infantile hepatic hemangioma" in combination with, but not limited to "fetal", "fetus", "prenatal diagnosis", "in utero", "infant", "infancy" and "neonate". Table 1 presents the cases with diagnosis of HH (including CHH, IHH and infantile hepatic hemangioendotheliomas). Cases that were not followed from the fetal to neonatal period, those with a final diagnosis of hamartoma, hepatoblastoma, or vascular malformations, cases with unclear imaging manifestations or without therapies during the prenatal and neonatal periods, lesions without follow-up or endpoint, or cases involving induction of the fetus, as well as review studies, were not included in our review.

Twenty-four studies of 168 cases were included in the comparative summary (Tables S1,S2), and we found that the most common fetal complications were as previously mentioned (6-8). Other rare complications in utero were polyhydramnios (35), NST demonstrating a reassuring or non-reassuring pattern (14) and gradually slowing down of fetal heart rate (9). Aiming to ease the risk of fetal complications, some studies introduced treatments in utero, including injection of corticosteroids and beta blockers through placenta or oral dexamethasone by pregnant woman (11,35,36).

The complications of neonates and infancy included anemia, thrombocytopenia, high-output congestive heart failure, pulmonary arterial hypertension, patent ductus arteriosus (PDA), atrial septal defect (ASD), respiratory failure (11,13,14,31,32,34,39,40) and so on. Monitoring, medical treatment, occlusion of the supplied arteries, and excision of neoplasm were the therapeutic methods (31,34,37-41). Schmitz *et al.* (11) selected the occlusion of parts of the hepatic arteries and the infant survived to 2 years old (tumor regressing or not was unclear). Sepulveda *et al.* (17) and one case of Ling (9), the tumor completely regressed using transcatheter embolization or interventional sclerotherapy with pingyangmycin. Three neonates with cardiac complications received hepatic artery embolization and the size of mass reduced without complete regression (39). Although, for most rapidly involuting congenital hemangioma (RICH), there was no need for medical or surgery therapies (41). Selective catheterization



**Table 1** The clinical characteristics, echocardiographic manifestations, complications and therapies of hepatic hemangioma from fetus to endpoint of follow-up of the included studies in the literature review

Study	No. of cases	First detection	Largest axis (cm)	Echocardiography in utero	Complications in utero	Treatments in utero	Echocardiography after birth	Medication after birth	Interventional therapy after birth	Endpoint
Zenzen 2009 (13)	1	3 days neonate	5.2	NC	NC	NC	3 days: patent ductus arteriosus, biventricular hypertrophy; 20 days: right ventricular dilatation and atrial septal defect	Conservative treatment with prednisolone	Selective catheterization and coil embolization of the right and left hepatic and right phrenic arteries	Liver transplantation, RICH was confirmed
Liu 2023 (31)	1	32gw	5.8	NC	NO	NO	NC	NO	NO	Resection with laparotomy
Prokurat 2002 (32)	17	Neonate to 16 yr	4 to 20	NC	NC	NC	NC	Steroids	Embolization	7 were arteriovenous malformations or hamartomatoid component; of the HH, 4 lobectomy, 6 segmental hepatectomy; all survival
Boon 1996 (33)	39	Neonate to 6 months	NC	NC	NC	NC	NC	Corticosteroid, interferon alfa-2a	Embolization	7 died, 23 alive and well, 9 without follow-up or with NC outcome
Zhang 2010 (34)	12	Fetus to 5 yr	5 to 13.8	NC	NC	NC	NC	NO	NO	10 well alive and 2 fetal demise
Schmitz 2009 (11)	1	28+1 gw	6.3	Cardiomegaly	NC	Oral dexamethasone	Cardiomegaly	Platelets transfusion; corticosteroids	Partly hepatic arterials occlusion	Survival (with tumor or not is unclear)
Cabrita 2009 (7)	1	26 gw	7.3	NO	None	None	NO	Prednisolone; $\alpha$ -2a-interferon	NO	Survival (with asymptomatic tumor)
Morris 1999 (35)	1	17 gw	4.5	Dilated right ventricle and depressed function	Polyhydramnios	Oral dexamethasone	Dilated right ventricle, slightly depressed function, low-normal left ventricular function	Prednisone	NO	Undetectable on sonography
Chou 2005 (30)	1	33 gw	3.72	NO	Normal	None	NO	Interferon	NO	NC
Morimura 2003 (14)	1	36+5 gw	9.6	NO	Reassuring pattern (NST)	None	NO	$\alpha$ -interferon, prednisolone	NO	Died for DIC
Sepulveda 2021 (17)	3	33+3 gw	8.9	NO	Normal	None	NO	NA	NA	Fetal demise
		30 gw	8.1	NO	Normal	None	NO	Vasoactive drugs, corticosteroids, blood transfusions	Transcatheter embolization	Complete regression
		34 gw	6.0	NO	Normal	None	NO	NO	NO	Normal
		29+3 gw	4.5	NO	NC	NC	NO	NC	NC	Spontaneous regression
Ling 2018 (9)	6	36 gw	8.6	NC	Slowing down fetal heart rate	None	NC	Oral propranolol, dexamethasone	NO	Tumor shrinkage and calcification plaques
		31+6 gw	9.2	NC	Cardiothoracic ratio (<0.33)	None	NC	Oral propranolol, dexamethasone	NO	Tumor shrinkage and calcification plaques
		36 gw	4.5	NC	None	None	NC	Oral propranolol, dexamethasone	NO	Tumor shrinkage and calcification plaques
		34+2 gw	5.6	NC	None	None	NC	Oral propranolol, dexamethasone	NO	Tumor shrinkage and calcification plaques
		36+3 gw	5.0	NC	None	None	NC	Oral propranolol, dexamethasone	NO	Tumor shrinkage and calcification plaques
		37+1 gw	5.5	NC	None	None	NC	NO	Interventional sclerotherapy	Tumor shrinkage and calcification plaques
		38 gw	4.3	NC	None	None	NC	NO	NO	Survival with tumor
Zhang 2019 (10)	6	37 gw	4.3	NC	None	None	NC	Oral propranolol	NO	Tumor shrinkage
		31 gw	3.8	NC	None	None	NC	NO	NO	No growth of tumor
		34 gw	5.7	NC	None	None	NC	Oral propranolol	NO	Complete regression
		35 gw	0.9	NC	None	None	NC	NO	NO	Survival with tumor
		38 gw	2.4	NC	None	None	NC	NO	NO	Little reduction in size

**Table 1** (continued)

Table 1 (continued)

Study	No. of cases	First detection	Largest axis (cm)	Echocardiography in utero	Complications in utero	Treatments in utero	Echocardiography after birth	Medication after birth	Interventional therapy after birth	Endpoint
Chuileannain 1999 (18)	1	33 gw	12	NC	Abnormal fetal heart rate	None	NC	NO	Intravenous alpha-interferon	Died for hemorrhage
Shen 2018 (27)	1	33 gw	4.4	NC	NC	NC	NC	NO	NO	Surgery and diagnosed as IHH
Chen 2021 (29)	15	29.5±3.6 gw	8.4	NC	None	None	NC	1 glucocorticoid	3 intravenous alpha-interferon	1 surgery, 3 regression, 11 tumor shrinkage
Yang 2022 (12)	14	28–39 gw	3.2–5.8	2 has cardiac insufficiency	NC	None	2 cardiac insufficiencies	Propranolol, glucocorticoid	NO	4 complete regression, 2 unchanged, 8 tumor shrinkage
Tang 2023 (36)	1	23 gw	6.4	NC	Cardiothoracic ratio 0.5, pericardial and pleural effusion	Corticosteroids and β-blockers through placenta	NC	NO	NO	Normal (with tumor or not is unclear)
Xie 2022 (8)	22	30–40 gw	4.2–9.9	NC	7 cases of intrauterine distress	None	NC	Propranolol, dexamethasone or rapamycin	Interventional therapy of tumor	3 complete regression, 19 partial regression
Long 2020 (23)	4	33 gw to 2 days	1.9–7.8	NC	None	None	NC	NO	NO	Reductions in size with calcification
Li 2023 (37)	12	22–39 gw	2.9–6.9	NC	2 cardiomegaly and 1 pleural effusion	None	NC	Oral medicine	NO	10 complete Regression,1 partial regression, 1 stable
Wang 2020 (38)	2	27 gw	NC	ASD, patent ductus arteriosus, pulmonary arterial hypertension	High cardiothoracic ratio	None	ASD, patent ductus arteriosus and pulmonary arterial hypertension	Methylprednisolone, hydrochlorothiazide, milrinone	NO	Surgery
		15 days neonate	NC	ASD, pulmonary arterial hypertension	NC	NC	ASD, pulmonary arterial hypertension	Milrinone, hydragogue	Hepatic artery embolization	Died for respiratory and cardiac failure
Shen 2017 (39)	3	7–34 days	NC	PDA, ASD, pulmonary arterial hypertension	NC	NC	PDA, ASD, pulmonary arterial hypertension	Propranolol, milrinone and sildenafil	Hepatic artery embolization	1 died for cardiac failure and 2 tumors with size decreasing

ASD, atrial septal defect; DIC, disseminated intravascular coagulation; gw, gestation week; HH, hepatic hemangioma; IHH, infant hepatic hemangioma; NA, not applicable; NC, not clear; NO, did not receive this examination or therapy; NST, non-stress test; PDA, patent ductus arteriosus; PE, pleural effusion; RICH, rapidly involuting congenital hemangioma; yr, year.

and coil embolization of the right and left hepatic and right phrenic arteries was performed by Zenzen *et al.* (13), but finally liver transplantation was chosen even for RICH because of liver failure. Other studies presented the medical therapy for inhibiting the growth of neonates and infancy liver tumors, including propranolol with or without dexamethasone or rapamycin (9-13), interferon (7,14,25,30), prednisolone (7,13), methylprednisolone (38), hydrochlorothiazide, milrinone, vasoactive drugs, and corticosteroids (11,17,25). However, only 22 cases (22/168) underwent the complete regression of mass with medical treatments (10,12,35,37), while 24 cases received surgery of liver (13,27,29,31,32,38) and 11 cases died for DIC, hemorrhage, respiratory and cardiac failure (14,33,35,37-39,41). Partial regression or survived with tumor was the most common endpoint. Among these studies included, there were no reports about the correlation between cardiac surgery and complete regression of HH. Our case report presented an unexpected disappearance of a HH after cardiac atrioventricular septal defect repair surgery. This may provide new ideas for treating similar cases in the future. However, the correlation between atrioventricular septal defect and hemangioma, as well as the mechanism underlying haemodynamic changes after atrioventricular septal repair, remain unclear and require further research.

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**Ethical Statement:** The authors are accountable for all aspects of the work in ensuring that questions related

to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this article and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

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