Received: 4 April 2022 | Revised: 17 July 2022 | Accepted: 18 July 2022

DOI: 10.1002/ccr3.6175

CASE REPORT

Clinical Case Reports Open Access WILEY

Prenatal diagnosis and management of a giant intrahepatic arteriovenous malformation—Sonographic findings, clinical implications, and treatment

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Abstract

Prenatal detection of complex giant hepatic arteriovenous malformation requires an examination of the affected fetal hemodynamic situation with emphasis on the affected arterial supply pattern. Early pediatric surgeon presentation is needed, as timely surgical intervention appears to be essential.

KEYWORDS

hepatic arteriovenous malformation, hepatic tumor, Kasabach–Merritt sequence, prenatal diagnosis, treatment options

1 | INTRODUCTION

A congenital hepatic arteriovenous malformation (AVM) is a rare disorder of vascular morphogenesis occurring in less than 1:100,000 live births.[1,2] Histological examinations demonstrate dysplastic vessels lined with resting endothelium, which form direct arterial connections to a fistula-like venous drainage system bypassing the normal capillary bed.[3,4]

Although these vascular malformations are developmental anomalies, they are rarely diagnosed prenatally and often misdiagnosed.[2,5] Profound knowledge of prenatal findings and prognostic parameters are essential for prenatal consultation. Prenatally as well as postnatally, fetuses might be at risk, as the high-flow, low-resistance shunt can cause acute hemodynamic failure including progressive congestive heart failure, portal hypertension, progressive pulmonary hypertension (PPH), and consumptive coagulopathy with thrombocytopenia and anemia.[6,7]

We describe the largest intrahepatic aneurysmatic AVM nidus diagnosed prenatally, which was successfully treated with serial embolizations following surgical extirpation.

2 | CASE

A 34-year-old woman, gravida 5 para 3, was referred to our department at 33+ 0 weeks of gestation because of suspected fetal liver anomaly. Ultrasound examination confirmed an isolated giant (67.9 × 61.6 × 53.3 mm) pseudoaneurysmatic fluid-filled area affecting almost the entire left hepatic lobe without soft-tissue components (Figure 1). Color and pulsed Doppler imaging demonstrated massive blood flow within the mass. Left hepatic artery was determined to be the main arterial feeding vessel with high velocity and low impedance blood flow (systolic 159.6 m/s, and end-diastolic 90.2 m/s, and the pulsatility index [PI] 0.69) (Figures 2 and 3). Resistance index in the hepatic artery was decreased (PI 0.49), and the peak systolic velocity was 90 cm/s. Left and middle hepatic veins were identified as draining vessels. The clinical features led to the diagnosis of an intrahepatic AVM with extreme pseudoaneurysmatic dilatation.

Biometry revealed fetal macrosomia with an estimated fetal weight of 2976 g (>97th percentile at 33 weeks of gestation), mainly due to the increased abdominal circumference. In addition, polyhydramnios with an amniotic fluid index (AFI) of 26 cm, placenta- and cardiomegaly with a cardiothoracic area ratio (CTAR) of 0.569 and a bilateral atrioventricular valvular regurgitation, were detected, but no hydrops fetalis was seen. Umbilical blood flow was normal. Further arterial Doppler indices showed an unremarkable peak systolic velocity of the middle cerebral artery (MCA-PSV) of 68.1 cm/s (MoM: 1.46) with normal pulsatility (PI 2.81). As a rapid progression of high cardiac output failure could not be excluded, there was a high risk for preterm delivery and antenatal corticosteroid treatment was initiated. Follow-up examinations remained

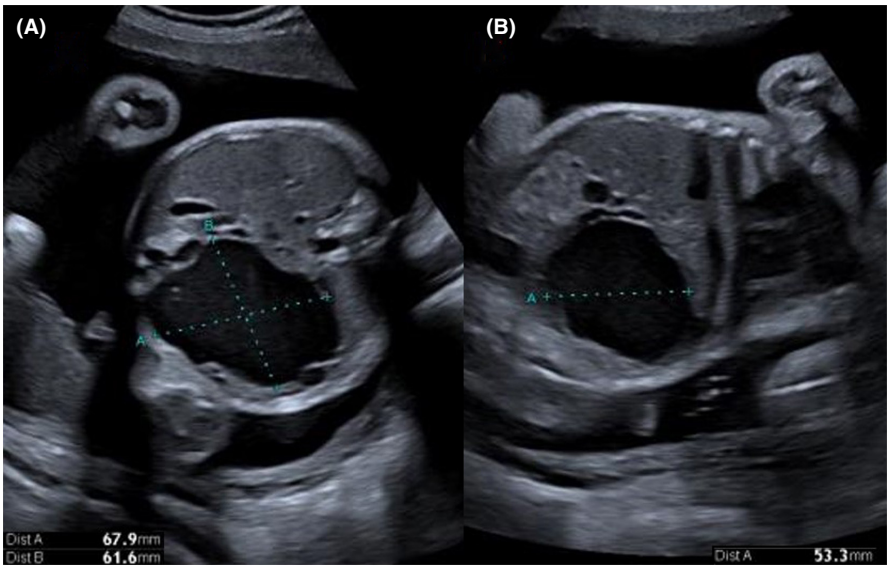

FIGURE 1 Ultrasound examination at 33+ 0 weeks of gestation showing an unclear cystic lesion measuring a total size of 67.9 × 61.9 × 53.3 mm (A + B)

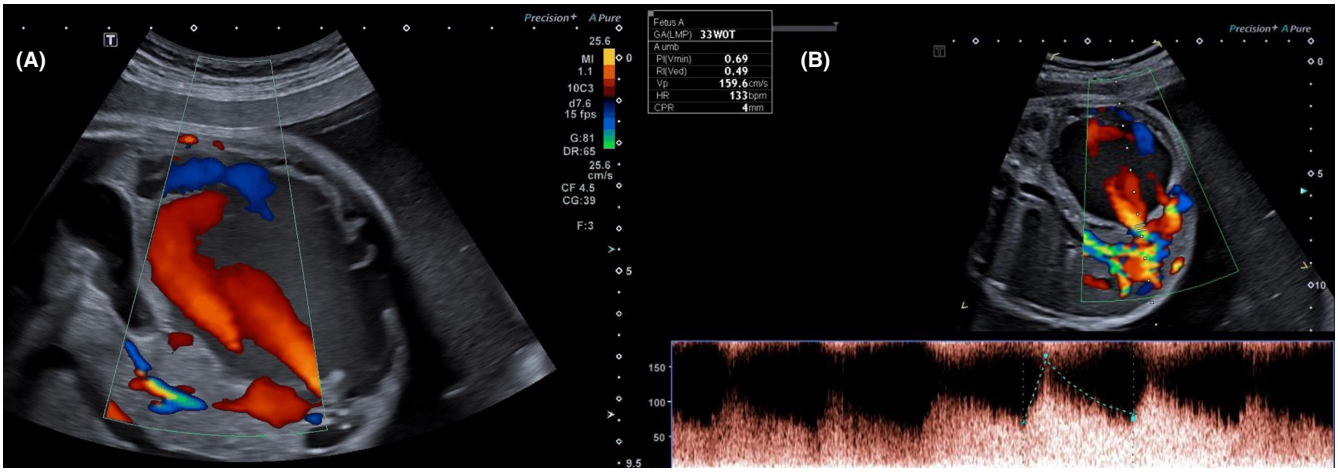

FIGURE 2 Color Doppler examination at 33+ 0 weeks of gestation demonstrating enlarged, abnormal tangle of vessels in the left liver with color Doppler flow (A). Continuous wave Doppler of the left hepatic artery showing an increase of the maximal velocity (peak systolic velocity = 160 cm/s) and a low impedance blood flow (pulsatility index (PI) = 0.69; resistance index (RI) = 0.49) (B)

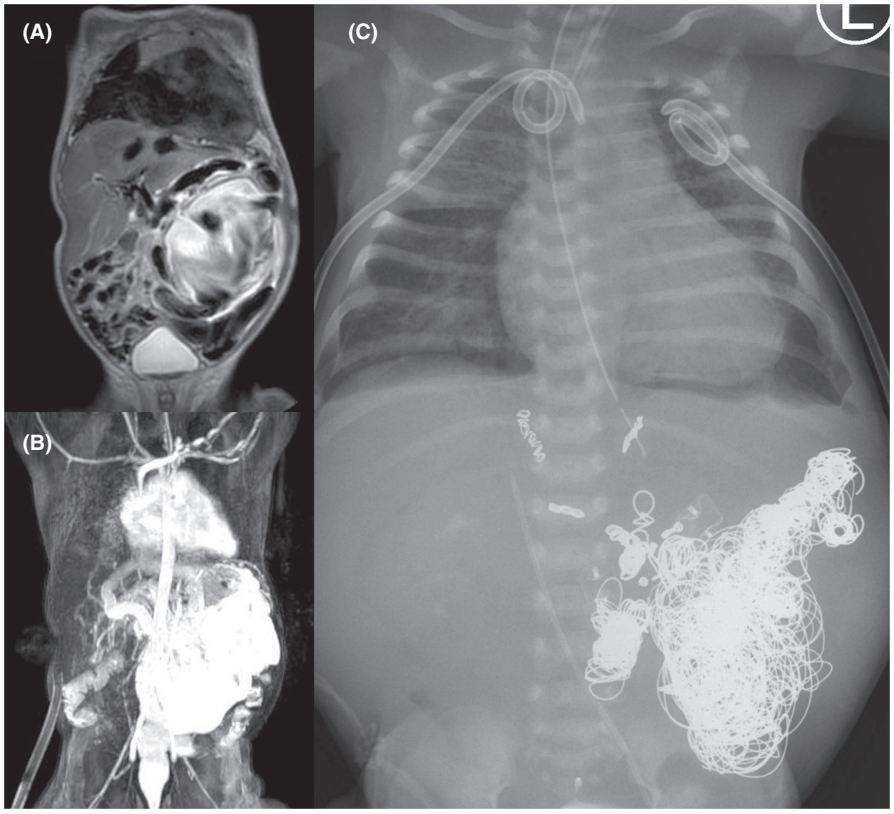

FIGURE 3 Magnet resonance imaging (MRI) (A) and computer tomography (CT) (B) on the first day of life, confirming prenatal findings and demonstrating the intrahepatic AVM fulfilling the entire left hepatic lobe (A + B). On CT multiple arterial branches are detected (B). Angiography on Day 28 of life showing the various coils placed (C)

stable except for a slight increase in MCA-PSV (MCV-PSV 81 cm/s [MOM 1.59] at 35 + 0 weeks of gestation); primary cesarean section was performed at 37 + 0 weeks of gestation. A 3330 g-male infant with Apgar scores of 7, 8, and 9 at 1, 5, and 10 min, respectively, was delivered. Due to persistent pulmonary hypertension, he required respiratory support by CPAP, oxygen supplementation and inhaled nitric oxide as well as a medical treatment with sildenafil and bosentan. Congestive heart failure was treated with dobutamine and milrinone. In the further course, propranolol was administered when the patient developed progressive hypertrophic cardiomyopathy. Apart from this well-established indication for use, propranolol was also reported as effective therapy in a patient with hepatic AVM and thus given for this indication in our case.

Postnatal abdominal ultrasonography, and magnetic resonance imaging confirmed the prenatal diagnosis of a complex giant intrahepatic (*hepatohepatic*) AVM with a total size of 45.2 × 51.4 × 73.5 mm with multiple arterial branches, mainly from the left hepatic artery, truncus coeliacus, phrenic arteries, left internal mammary artery, and left intercostal arteries, drained by middle and left hepatic vein. The patient developed microangiopathic hemolytic anemia (6.9 g/dl) and thrombocytopenia (55 G/L) with consumptive coagulopathy (fibrinogen 68 mg/dl) (Kasabach–Merritt sequence) and subsequently required transfusion of two red cell, three platelet, and six fresh-frozen plasma.

In view of rapid development of cardiac failure and persistent pulmonary hypertension (PPH), embolization had been considered the most appropriate treatment in order to improve clinical condition prior to surgery.

On the 5th, 21st, and 28th day of life, an arteriography was performed and a total of 154 vessel (21 Hilal® coils, 89 Target® coils, 2 Amplatzer™ duct occluder [8 and 10 mm], 25 Nester® embolization coils, 19 Interlock® coils) were placed in the feeding and draining vessels. When he was extubated after the first intervention, the patient had to be resuscitated due to airway obstruction with mucus pluge. During the second intervention, a rapid pulmonary and cardiac deterioration following bilateral tension pneumothorax again required resuscitation in addition to chest drainage. Unfortunately, an angiogram 23 days after the last intervention demonstrated that the large AVM had recanalyzed. Surgical partial left hepatectomy measuring 80.1 × 80.3 × 45.4 mm with complete removal of the AVM was successfully performed on the 61th day of life. Histological examination confirmed benign character of the giant vascular aneurysmatic AVM nidus with multiple thromboses. The patient's clinical condition improved rapidly after surgery. On the 134th day of life, he was discharged in good clinical condition, without any respiratory support and with markedly improved cardiac function. Neurological reassessment did not reveal any abnormalities.

TABLE 1 Prenatal diagnosed published cases with hepatic arteriovenous malformation—management and outcome

Case	GA at diagnosis	Referral reason	AVM size and flow	USG findings	Localization	Prenatal management	GA at delivery, delivery mode	Outcome	Neonatal management and outcome
Mejides (1995) [28]	29 wks	lagging fundal growth	Hepatic vein-hepatic artery AVM 104 cm/s	cardiomegaly, cardiac failure	left hepatic lobe	intrauterine treatment application of hydrocortisone to umbilical vein and amniotic fluid, restart treatment after 1 week	31 weeks CS	Female, 1498 g, APGAR 8/9	• No cardiac failure at birth, PPH • No treatment after birth, • 18-day fetal tachycardia, tachypnea, increase in hepatic vascularity-start steroid and diuretic • Dramatic improvement in a week with steroid and diuretic • **Alive**
Jouannic (1998) [29]	30 weeks	Vascular hypoechoic image with Doppler signal							• Cardiac failure • **Embolization** • **Died** (32th days of life)
Tseng (2000)[11]	35 weeks	Fetal cardiomegaly	Hepatic vein-hepatic artery 32 cm/s	Cardiomegaly, oligohydramnios, no atrioventricular regurgitation, and pericardial effusion	Left hepatic lobe	Monitoring, progression of heart failure, labor Induction	37 wks Vaginal delivery	Female, APGAR 8/9	• Ligature of the left hepatic vein (at 6 months of life) because of the development of shortness of breath, malaise, poor appetite and water diarrhea • **Alive**
Botha (2004)[13]	34 weeks	Abnormal prenatal sonographic findings	Hepatic vein-hepatic artery + right and left internal mammarian artery AVM	Cardiomegaly, progressive cardiac failure	Left hepatic lobe	Monitoring	34 weeks Emergency CS	2648 g, APGAR 1/7	• Cardiomyopathy with cardiac failure • Coagulopathy / Kasabach–Merritt sequence • **Embolization (3rd day of life)** • Recanalization in the follow-up • **Died** (2 weeks of life)
Lima (2005)[8]									
1 Case	25 weeks	Unclear supra renal aortic dilatation	Hepatic vein-hepatic artery AVM	Mild cardiomegaly, no hydrops	Left hepatic lobe		37 weeks		• Cardiac failure • Coagulopathy/Kasabach–Merritt sequence • diuretic, cardiokinetic treatment • **Embolization** (1th day of life) • **Died** (on 3rd day of life) • Autopsy revealed congenital heart and lung malformation

TABLE 1 (Continued)

Case	GA at diagnosis	Referral reason	AVM size and flow	USG findings	Localization	Prenatal management	GA at delivery, delivery mode	Outcome	Neonatal management and outcome
2 Case	27 weeks	AV-Fistula in the liver	Hepatic vein-hepatic artery AVM	Cardiomegaly, cardiac failure, DV not visualized	Right and left hepatic lobe		35 weeks		• No cardiac failure • No coagulopathy • Diuretic, cardiokinetic • Left hepatectomy (2nd day of life) • **Alive**
Gedikbasi (2008)[30]	36 weeks	Dilated gallbladder	Complex hepatic vein-umbilical vein – portal vein+ hepatic artery AVM 22 × 15	No cardiomegaly, no hydrops	Left hepatic lobe	None Prenatal course remained stable	38 weeks Vaginal delivery	Male, 3030 g APGAR 7/9/10	• No cardiac failure/no coagulopathy • Extended right hepatectomy with cholecystektomy (19th day of life) • **Alive**
Douhnai (2019)[15]	22 weeks	suspected polyhydramnios	Hepatic vein-hepatic artery AVM 37 × 68 mm; 33 cm/s	No cardiomegaly, no hydrops	Left hepatic lobe	None Prenatal course remained stable	41 weeks Vaginal delivery	Female, 3470 g	• No cardiac failure/no coagulopathy • No postpartale treatment • **Alive**, 2 years old now stable
Demirci (2020)[24]									
1 Case	32 weeks	Suspected right renal pelvietasis	Hepatic vein- umbilical vein - hepatic artery-AVM 65 × 35 mm; 100 cm/s	No cardiomegaly, no hydrops, DV not visualized	Right hepatic lobe	Prenatal course remained stable	39 weeks CS	Male 3070 g, APGAR 9/9/10	• No cardiac failure/no coagulopathy • Propranolol and steroid treatment for ptophylaxis • Right hepatectomy (2nd month) due to growth of AVM • Alive
2 Case	24 weeks	Agenesis of DV and aorto-portal fistula	Hepatic vein-hepatic artery-AVM 100 cm/s	Initially no cardiomegaly; Follow-up hydrops at 29 weeks	Left and right hepatic lobe	intrauterine treatment-dexamethasone+ propranolol; 2 weeks later heart failure disappeared with progressively shrinking AVM	38 wks CS	Male 2730 g, APGAR 9/9/10	• No cardiac failure/no coagulopathy • Propranolol treatment continued • **Alive**

TABLE 1 (Continued)

Case	GA at diagnosis	Referral reason	AVM size and flow	USG findings	Localization	Prenatal management	GA at delivery, delivery mode	Outcome	Neonatal management and outcome
Our case	33 weeks	Suspected liver malformation	Complex hepatic vein-hepatic artery-AVM with multiple arterial branches with pseudoaneurysmatic appearance 68 × 62 × 53 mm; 160 cm/s	Macrosomia, polyhydramnion, placenta, and cardiomegaly, cardiac failure av-regurgitation	Left hepatic lobe	Betamethasone 12 mg 2× for RDS prophylaxis in risk of progress of high cardiac output failure Prenatal course remained stable	37 weeks Primary CS	Male, 3330 g APGAR 7/8/9	• Cardiac failure, PPH, reanimation • Propanolol for hypertrophic cardiomyopathy and as potential treatment option against progression of AVM • Coagulopathy/Kasabach–Merritt sequence • **Embolization** (5th, 21st, and 28th day of life) • Partial left hepatectomy (61th day of life) • **Alive**

Note: Abbreviations: AVM, arteriovenous malformation, CS, cesarean section, GA, gestational age, USG, ultrasonographic; wks, weeks.

3 | DISCUSSION

Fetal intrahepatic arteriovenous malformations (AVMs) are infrequently diagnosed prenatally and there have been only eight cases published so far, focusing on the postnatal course (Table 1). Due to the rarity and the high mortality rate of fetal intrahepatic AVM, data on long-term outcome are scarce. Survival over up to 9 years after definitive treatment and recurrence of a high-flow vascular anomaly are reported.[26] The appearance in utero can be variable, as in our case, a giant pseudoaneurysmatic appearance was described for the first time (Table 1).

AVMs can be classified as fast-flow conduits.[2,7] Depending on their size and the complexity of involved feeder vessels, they can lead to significant hemodynamic changes already during fetal life. In particular, hepato-hepatic AV shunts connecting hepatic arteries to hepatic veins are crucial, as high pressure to low pressure system is communicating, resulting in a low-resistance arteriovenous shunt.[8,9] Considering that systemic vascular resistance increases at birth and blood flow through the AVM rises, an altered cardiac workload with a risk of developing heart failure soon after birth, explaining a high mortality rate of 50–90%, should be taken into account.[10]

If a relevant shunt is present prenatally, an area of abnormal vascularization without soft-tissue components can be recognized by gray-scale and color Doppler imaging in the fetal liver.[11,12] As systemic blood pressure is higher on the arterial side a progressive distension on the venous drainage, resulting in characteristic sonographic findings of echopenic dilated and tortuous or aneurysmal vascular channels, can be seen.[13,14] Feeding vessels may also be enlarged, and visualization of the ductus venosus can be difficult.[15]

Pulsed wave Doppler should be used to characterize vascular connections in order to distinguish the different types of congenital hepatic vascular malformations (slow-flow: capillary, lymphatic, venous malformations vs. fast-flow: arteriovenous malformations including hepato-hepatic and hepatoportal shunts).[4]

If a hepatohepatic shunt is suspected prenatally, typical features are, demodulation of the arterial flow with low impedance blood flow, diagnosed by pulsed wave Doppler, and high peak systolic and diastolic velocities in both arteries and veins.[14,16] Thus, differential diagnoses such as hemangiomas, dilated gall bladder, cystic lesions, hepatoblastoma, hepatic metastasis of neuroblastoma, or other congenital hepatic vascular malformations can easily be excluded.[7,17,18]

Prenatal assessment should determine the number of feeding arterial branches as they correlate with shunt blood volume and postnatal outcome, considering that an AVM of the central vascular tree in a fetus is entirely

different than an infant.[8,13] Depending on the amount of blood volume shunted through this low-resistance, high-flow outlet, fetal cardiac output must increase to meet the competing demands of fetal growth and the AVM "steal." Therefore, signs of high cardiac-output failure, including cardiomegaly, tricuspid valve regurgitation, polyhydramnios, and fetal hydrops, should be monitored, as well as fetal growth bearing in mind that hepatomegaly may lead to overestimation of fetal weight.[19] It is important to keep all these aspects in mind to time delivery as postnatal catheter embolization or surgical resection should not be performed until weight of >2000 g.[20,21]

Further, detailed fetal ultrasound including Doppler examination of MCA-PSV and DV is essential as other complications such as microangiopathic hemolytic anemia, thrombocytopenia, and consumptive coagulopathy, known as the Kasabach–Merritt sequence, may be detected and require delivery in dependence on cardiac function.[7,22] In these cases, MCA-PSV of ≥1.5 multiples of the median (MoM) should be considered as an indicator of moderate–severe fetal anemia, which can in addition be associated with thrombocytopenia.[23]

4 | CONCLUSION

Early prenatal diagnosis of intrahepatic AVM is important as it might change management and outcome of affected fetuses. Prenatal treatment including propranolol or corticosteroids may be helpful, as described in one case report.[24] Follow-up examinations should be carried out depending on size of the vascular malformation, extent of the perfusion, and signs of high cardiac output failure (severe cardiomegaly, AV valve insufficiency, and hydrops fetalis, respectively) in order to identify progression and to time delivery and therapeutic intervention. Acute prenatal deterioration from time of diagnosis is not generally expected and should be considered when initiating corticosteroid prophylaxis or timing delivery, as these lesions are non-proliferating vascular anomalies that grow proportionally to fetal weight.[2]

Examiner should pay particular attention to signs of high cardiac output failure, underlying syndromic disorders (as Klippel–Trenaunay–Weber syndrome) and to the malformations volume, as tumor volumes above 50 ml in series of hepatic hemangiomas seem to be associated with risk of compartment syndrome and respiratory distress soon after birth.[25] Delivery should be inducted, if deterioration of cardiac function or a centralization of fetal blood flow is prenatally observed. Due to life-threatening complications of AVM such as PPHN and cardiac failure, pregnancies with prenatal diagnosis of intrahepatic AVM should be referred to perinatal centers with level III NICU.

Definitive treatment options include embolization and surgery, which are mandatory as these vascular malformations do not regress spontaneously. Embolization has been performed successfully as definitive treatment in infants with hepatic AVM and is most effective in AVM with a single arteriovenous fistula. In patients with multiple feeding vessels, embolization can help to control congestive heart failure and pulmonary hypertension temporarily prior to definitive treatment.[26,27]

To our knowledge, the present case describes the largest prenatally detected AVM with a giant pseudoaneurysmatic appearance measuring the highest Doppler velocity of the feeding vessels reported so far (Table 1).

AUTHOR CONTRIBUTIONS

AW, EC, AM, JCK, CM, AG, UG, and CS managed the patient. AW, CS, AG, AM, and UG performed the analysis. AW and EC created the figures. All the authors contributed in writing and editing of the manuscript.

ACKNOWLEDGMENT

No acknowledgements to be mentioned. Open Access funding enabled and organized by Projekt DEAL.

FUNDING INFORMATION

This case report was conducted without any financing. Therefore, there are no further financing details to be declared.

CONFLICT OF INTEREST

There are no conflicts of interest to be declared.

DATA AVAILABILITY STATEMENT

The data that support the findings of this case are available from the corresponding author upon reasonable request.

ETHICAL APPROVAL

Written informed consent was obtained from the patient for the publication.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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How to cite this article: Walter A, Calite E, Müller A, et al. Prenatal diagnosis and management of a giant intrahepatic arteriovenous malformation—Sonographic findings, clinical implications, and treatment. *Clin Case Rep*. 2022;10:e06175. doi: 10.1002/ccr3.6175