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)
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.
| 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) | 29 wks          | lagging fundal growth    | Hepatic vein-hepatic artery AVM | 104 cm/s                        | cardiomegaly, cardiac failure | left hepatic lobe                                                                  | 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 |
| Jouanpic (1998) | 30 weeks        | Vascular hypoechoic image with Doppler signal | 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 | • Cardiac failure • Embolization • Died (32th days of life) |
| Tseng (2000)  | 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)  | 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, progression of heart failure, labor Induction | 34 weeks Emergency CS | 26-45 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)   | 25 weeks        | Unclear supra renal aortic dilatation | Hepatic vein-hepatic artery AVM | Mild cardiomegaly, no hydrops | Left hepatic lobe | Monitoring, progression of heart failure, labor Induction | 37 weeks Emergency CS | 26-45 g, APGAR 1/7 | • Cardiac failure • Coagulopathy/Kasabach–Merritt sequence • diuretic, cardiokinetic treatment • Embolization (4th 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, cardionkinetic \* Left heptectomy (2nd day of life) \* Alive |
| Gedikbasi (2008) | 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 heptectomy with cholecystektomy (19th day of life) \* Alive |
| Douhni (2019) | 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) | 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 3070g, APGAR 9/9/10 | \* No cardiac failure/no coagulopathy \* Propranolol and steroid treatment for prophylaxis \* Right heptectomy (2nd month) due to growth of AVM \* Alive |
| 2 Case | 24 weeks | Agensis 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 2730g, APGAR 9/9/10 | \* No cardiac failure/no coagulopathy \* Propranolol treatment continued \* Alive |
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.\(^2^6\) 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, hepatohepatic 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.\(^1^0\)

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.\(^1^1^,^1^2\) 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.\(^1^3^,^1^4\) Feeding vessels may also be enlarged, and visualization of the ductus venosus can be difficult.\(^1^5\)

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 hepatopatic and hepatoporal shunts).\(^4^)

If a hepatopatic 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.\(^1^4^,^1^6\) 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.\(^7^,^1^7^,^1^8\)

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. 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. 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.

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. 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.

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. 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.

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. Delivery should be induced, 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.

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.

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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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