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Cardiovascular profile score (CVPS) and selected cardiac parameters in fetuses with Vein of Galen Malformation

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ABSTRACT

Objectives: Vein of Galen Malformation (VGAM) is a rare congenital cerebrovascular anomaly. Early detection and monitoring of concurrent fetal heart failure in VGAM are crucial for improving outcomes.

The study aims to evaluate heart anatomy, systolic and diastolic heart function, and indicators of heart failure in fetuses referred to a tertiary center due to VGAM detected in the second or third trimester.

Material and methods: This single-center retrospective study of echocardiographic data from five fetuses with VGAM examined between 2008 and 2023. Parameters analyzed included gestational age, reason for referral, cardiovascular profile score (CVPS), systolic and diastolic heart function and selected cardiac parameters

Results: Cardiomegaly as a sign of congestive heart failure was the main reason for referral most of the fetuses (4/5 — 80%) diagnosed later as VGAM in our center. Abnormal cerebral vessel flow was visualized in all cases, with normal umbilical arterial flow. Three fetuses were

treated prenatally with digoxin due to congestive heart failure. Monophasic tricuspid valve inflow and decreased shortening fraction (SF) were observed in three fetuses (60%) at the time of diagnosis. CVPS scores ranged from 2 to 10, correlating with the severity of cardiac compromise. All five patients demised, three died in utero, and two shortly after birth.

Conclusions: This study emphasizes the severe cardiac implications of VGAM detected in utero and the need for early and comprehensive fetal assessment. Despite early diagnosis, outcomes remain poor, necessitating further research into effective prenatal treatments and management protocols to enhance survival for affected fetuses.

Keywords: Vein of Galen Malformation; fetal echocardiography; cardiovascular profile score

INTRODUCTION

Vein of Galen Malformation (VGAM) is a rare congenital cerebrovascular anomaly, with an incidence estimated between 1/25 000 to 1/58 000 live births [1, 2].

The prognosis for VGAM may vary, with a diverse clinical presentation that depends on the angioarchitecture of the VGAM, the presence of cardiac compromise, and neurological complications [3–5]. According to a meta-analysis published by D'Amico in 2022, prenatal diagnosis is linked with cardiac morbidities, brain damage, and poor neurodevelopmental outcomes [6].

Clinical presentation of VGAM is heterogenous, with brain and cardiac manifestations. Neurological abnormalities associated with VGAM are present in half of the cases and include ventriculomegaly, hydrocephaly, cortical thickening, polymicrogyria, or porencephaly [4, 7]. Cardiac signs may present as cardiomegaly, tricuspid valve insufficiency, and right-heart failure with hydrops [7, 8].

Understanding the progression and implications of fetal heart failure in VGAM is essential for developing targeted therapeutic strategies. Early detection and monitoring can significantly influence clinical decisions, potentially improving outcomes for affected patients.

Objective

This study aimed to evaluate heart anatomy, cardiovascular profile score, systolic, and diastolic heart function and selected cardiac parameters in fetuses examined in the tertiary center due to Vein of Galen Malformation (VGAM).

MATERIAL AND METHODS

This is a single-center retrospective study of clinical and echocardiographic data obtained from fetuses with Vein of Galen Malformation, examined in our hospital between 2008 and 2023. The study included five patients referred to the tertiary center due to abnormal findings on ultrasound examination. Fetal echocardiography was performed by an experienced sonographer using Voluson 730 and Expert (between the years 2008 and 2015) and Voluson E8 and Samsung Hera (since 2015).

Fetal echocardiography was performed to evaluate cardiac abnormalities in both structure and function. In particular, pulsed Doppler examination was used to assess fetal hemodynamics [9].

Analyzed clinical data included (Tab. 1):

- gestational age at the time of examination,

- reason for referral,
- assessment of CVPS at the time of diagnosis,
- assessment of systolic and diastolic right and left ventricle (SF LV%, SF RV%, Tei index of RV and LV),
- assessment of Z-scores (AoV, PAV, TV, MV),
- assessment of peripheral flows (umbilical vessels, ductus venosus, and middle cerebral artery),
- treatment and outcome.

Echocardiographic findings analyzed in the study are presented in Table 2.

RESULTS

Mean gestational age at the time of the examination was 27 + 0/7 weeks (minimum 22 + 4/7, maximum 33 + 0/7). Gestational age was determined based on crown rump length (CRL) examined in the first trimester (data from the patient's history).

In all cases, the reason for referral for fetal echocardiography was abnormal results of previous obstetric ultrasound examinations. Four fetuses presented cardiomegaly, and additionally, in one of them, ascites was observed. Out of those fetuses, only one fetus had a suspected Vein of Galen Malformation before the referral. In one case, due to fetal growth restriction, middle cerebral arterial (MCA) Doppler was conducted at 21 weeks gestational age, revealing abnormal flow in cerebral vessels, which subsequently resulted in the diagnosis of VGAM (Fig. 1).

Abnormal flow in cerebral vessels, typical for arteriovenous malformation, with a high systolic and diastolic velocity of the blood flow and low pulsatility index (PI), was visualized in all cases. Umbilical arterial flow was normal in all cases. Normal flow in ductus venosus (DV) was observed in four cases, while reversal flow in the DV was noted in one fetus with congestive heart failure (CHF). This finding was indicative of advanced circulatory insufficiency.

On transversal 3-vessel-view scan of the upper mediastinum, enlargement of the superior vena cava (Fig. 2) was visualized in patients with cardiomegaly. The brachiocephalic vein was dilated in all cases where the scan showing its course was assessed (Tab. 2).

In all fetuses diagnosed with heart failure due to vascular malformation, both systolic and diastolic function of the right ventricle were impaired, the inflow through the tricuspid valve

was monophasic, and the shortening fraction (SF) measured by M-mode echocardiography was decreased (according to Cardiovascular Profile Score [CVPS] scale, reduced SF was interpreted as below 28%) [10].

Holosystolic tricuspid valve regurgitation was present in all cases. Additionally, insufficiency of other valves was present in all fetuses with cardiomegaly (Tab. 2).

The assessment of fetal heart function was analyzed based on the Huhta scale (CVPS) [10]. Three fetuses without ascites and with normal peripheral flows scored 7 points. One fetus with ascites and cardiomegaly, with HA/CA > 0.5, and reversal flow in DV scored 2 points. The only hemodynamically stable (10 points in CVPS) fetus was the one referred due to abnormal cerebral vessel imaging and examined in the second trimester.

Three patients were treated prenatally with digoxin, maternal serum concertation was controlled during the therapy until desirable levels were reached. Indication for digoxin therapy was the presence of congestive heart failure and abnormal heart systolic function. Two patients were referred to a tertiary center in Warsaw, with planned interventional treatment after birth. Due to progressive signs of congestive heart failure, patients MV and KJ were delivered via caesarean section at respectively 32 and 33 weeks of gestation. Because of critical condition after birth, none of them underwent interventional treatment of the malformation. Patient MV died within the first 24 hours, patient KJ died on the second day of life.

Three fetuses demised in utero. Patient SW demised one week after the diagnosis due to severe cardiac compromise. At the time of diagnosis, SW had already presented with ascites and reversal DV flow. Based on the initial assessment, the prognosis was defined as poor.

Patient KP demised in utero at 28 weeks of gestation due to congestive heart failure. Patient SJ demised in utero at 24 weeks as a complication of maternal urinary tract infection.

Four fetuses (80%) had cardiomegaly — Table 2. One fetus (SJ) did not present cardiomegaly or brachiocephalic vein enlargement, contrary to the other fetuses. However, it is worth noting that diagnosis of VGAM in this case was made early, in the second trimester, prior to the possible onset of cardiac decompensation.

DISCUSSION

VGAM is a rare congenital vascular malformation. During the normal embryological period, the median prosencephalic vein (MVP, also known as the vein of Markowski) develops between the 6^{th} and 11^{th} weeks of gestation and becomes a precursor to the great vein of Galen. In the malformation, arteriovenous shunts drain into the MVP, leading to the formation

of dilated vessel [11]. The pathogenesis of developing abnormal arteriovenous lesions remains unclear. Genetic studies in the previous years identified certain mutations in gene coding proteins involved in the process of pathologic angiogenesis, such as mutations of RASp21 Protein Activator 1 (RASA1) and Ephrin type-B receptor 4 (EPHB4) [12, 13].

Nowadays, diagnosis of VGAM occurs prenatally, mainly in the third-trimester scan, although some authors report diagnosis made in the second trimester. [3, 5, 14]. State-of-the-art prenatal assessment involves a comprehensive evaluation, including cerebral ultrasound scanning, fetal magnetic resonance imagining (MRI), and echocardiography [7].

In our study, referral to the tertiary center was made both in the second (n = 2) and third (n = 3) trimester. Only one fetus had suspected VGAM prior to hospitalization, and referral was made based on clinical symptoms in the fetuses. However, it is worth noting that study data were collected over a 15-year period, and advancement of assessment methods by obstetricians may have varied.

Neurosonography commonly reveals a hypoechoic cystic mass within the subarachnoid space, situated in the midline above the third ventricle. Color Doppler allows to differentiate vascular nature of the cyst from other anomalies by visualization of turbulent blood flow [4] (Fig. 3–5).

In routine obstetric ultrasound scan in normal fetuses, Doppler assessment of cerebral vessels is not mandatory. In our study, most of the fetuses were referred to our center due to cardiac symptoms and heart failure. Referral based on fetal findings revealing signs of cardiac overload is a phenomenon commonly described in the literature [7, 14]. Therefore, we emphasize the importance of a thorough examination of brain structures during the second trimester.

According to Gillet de Thorey (2022), signs of cardiac compromise are present in an average of 60% of cases, with cardiomegaly being the most frequently occurring condition among them [7]. Determination of prognosis based on echocardiographic signs remains a challenge because of the discrepancy among authors. Some authors describe tricuspid insufficiency in correlation with both major brain damage and poor outcome, with cardiomegaly being a predictive factor of heart failure at birth but not of mortality [5, 7]. In contrast, in the study by Buratti et al. [15] aimed at identifying risk factors of adverse outcomes, an increased cardiothoracic index was the sole cardiac finding significantly associated with severe high-output heart failure at birth and mortality. Identification of cardiac components related to poor prognosis remains a part of further research among VGAM patients.

Additional echocardiographic parameters may also play a role in diagnosing VGAM. In the 2012 study on measurements of the fetal left brachiocephalic vein, out of 11 fetuses with dilatation of this vessel, four were diagnosed with VGAM. The presence of an enlarged superior vena cava and brachiocephalic vein was notable and may be considered in the future in the differential diagnosis of circulatory failure [16, 17].

For a comprehensive assessment, in addition to neurosonography and echocardiography, performing a fetal MRI is advisable.

The objective of MRI is to visualize the malformation and to identify factors that may be related to poor prognosis, including perinatal death or severe cardiac or neurological compromise. Additionally, MRI scans have been effective in visualizing signs of irreversible brain damage [7].

Over the years, many components were analyzed to help predict the clinical outcome and to estimate the required treatment after birth. Major brain lesions and volume of the VGAM >/ 20 000 mm^3 have been identified as correlating with poor prognosis [5].

In a recent study on MRI predictive values, L. Arko et al. [19] found that maximal mediolateral diameter measured at the narrowest point of the straight of the falcine sinus (SS-MD) has the strongest predictive value. The larger the diameter, the greater the likelihood of being at risk of neonatal cardiopulmonary decompensation and urgent embolization treatment. Moreover, in the Buratti et al. study, out of neuroradiological factors, SS-MD was statistically significantly ($p = 0.01$) associated with high-output heart failure at birth [15].

Prenatal treatment options for VGAM in the past decades have been limited. Pharmacological treatment option involves maternal supplementation with digoxin.

Berenstein et al. described a protocol for antenatal digoxin treatment initiated when the cardio-thoracic ratio (CTR) is above 0.5. As a result, in this study, 44% of the newborns with heart failure were managed pharmacologically and did not require postnatal early embolization [19].

In recent years the possibility of interventional treatment has emerged with the development of fetal interventions. To date, there have been two publications on prenatal treatment of vein of Galen malformation [20, 21]. The first clinical trial on the safety and efficacy of prenatal embolization of VGAM was initiated in 2022 by Darren B. Orbach et al. in Boston, USA.

The fetal intervention was a maternal percutaneous transuterine ultrasound-guided coil embolization. The authors described a reduction of blood flow through the falcine sinus seen

on ultrasound during the procedure. Post the procedure, fetal echocardiography revealed a 43% reduction in cardiac output. Fetal magnetic resonance revealed the shrinkage of both the prosencephalic vein and the falcine sinus. In the following weeks, the newborn required no further embolization treatment, and on clinical assessment, there was no neurological or cardiovascular compromise [19]. The second published case of prenatal VGAM embolization was conducted in France in September 2022. An 18-gauge needle was inserted through the posterior fontanelle under the ultrasound guidance, and platinum coils were subsequently inserted through a microcatheter. Real-time ultrasound revealed blood flow reduction at the time of the procedure. Post-procedure follow-up consisted of ultrasound and MRI imaging, which revealed a gradual reduction in the diameter of the falcine sinus and a decrease in the cardiothoracic index. Delivery was inducted at term. The newborn required endovascular embolization (EVT) on day 3 of life due to signs of right heart failure, resulting in an improvement of general condition. The boy was discharged home after one month of hospitalization. Additional EVT on the 67th day of life led to complete occlusion of the malformation. Neurological assessment at 11 months of age revealed normal neurological development [20].

Postnatal treatment often involves intensive care measures, including cardiopulmonary support, as well as embolization treatment.

The main limitations of our study were the small study cohort and a long data collection period. To conduct a reliable statistical assessment, it is necessary to analyze a larger number of subjects, which can be achieved through a multi-center study.

Although in our study, diagnosis was made early, in the second or early third trimester, it did not have an impact on the favorable outcome. It is important to highlight that among the five fetuses examined, four had already shown echocardiographic signs of cardiac compromise at the time of diagnosis, and one of these cases resulted in fetal demise due to maternal complications (infection), not because of the malformation. Our study reported a 100% mortality, which is not consistent with other publications. However, it is common for authors to exclude cases where the pregnancy was terminated (TOP) from their analysis. Moreover, given our small sample size, this mortality rate cannot be considered statistically significant.

In a study by Deloison et al. [4], which analyzed 21 cases of VGAM, more than 40% of pregnancies underwent termination of pregnancy. The remaining fetuses (n = 12) were delivered at term, and among these newborns, nine patients (75%) had a poor outcome.

It is also crucial to highlight the advanced stage of progression of heart failure among our patients at the time of referral. During the observation period, cardiac compromise was

progressively worsening, leading to intrauterine and neonatal death. Palladini [5] described a progression of the brain lesion, which occurred in 10% of the cases. The progression of heart failure was not analyzed in their study.

CONCLUSIONS

Our study emphasizes the necessity for early and accurate diagnosis, along with continuous echocardiographic monitoring, to manage the rapid progression of heart failure in affected fetuses. Establishing a diagnostic protocol with regular check-ups is crucial to identify fetuses at risk of rapid deterioration and to facilitate timely interventions.

Article information and declarations

Data availability statement

Original contributions presented in the study are included in the article, further inquiries can be directed to the corresponding author.

Ethics statement

Not applicable — retrospective study.

Author contributions

AJ — concept, study design, analysis and interpretation of data, article draft. TW — investigation, data collection, follow-up, writing–editing. WC — concept, study design, revised article critically. AW — concept, study design, aquisiton of data, revised article critically.

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Conflict of interest

The authors declare no conflict of interest.

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Table 1. Clinical data of fetuses with VGAM

Patient (Initials)	**GA dgn wks**	**Reason of referral to tertiary center for fetal echo**	**Heart size (HA/CA)**	**Presence of hydrops (at time of diagnosis)**	**Abnormal flow in cerebral vessels**	**Umb artery flow**	**DV, umb vein**	**Outcome**
MV	28 + 2	Cardiomegaly	0.48	No	Yes	n	n	Demise after birth Digoxin no postnatal neurological treatment CC 32 wks
SW	33 + 0	Cardiomegaly, ascites	0.52	Yes	Yes	n	r	Demise in utero — 34 wks
KJ	27 + 6	Cardiomegaly	0.44	No	Yes	n	n	Demise after birth Digoxin CC 33 wks

								No postnatal neurological treatment
SJ	22 + 4	Abnormal flow cerebral vessels	0.28	No	Yes	n	n	Demise in 24 wks in utero (infection)
KP	23 + 3	Cardiomegaly VGM susp	0.40	No	Yes	n	n	Demise in utero 28 wks, Digoxin, no treatment

n — normal; r — reverse; wks — weeks

Table 2. Echocardiographic findings in fetuses with VGM

	2D				PW				M-mode	
Patient	Heart size (HA/CA)	3VV (enlargement of SVC)	Brachiocephalic vein enlargement	> Z-score Ao, PAV, TV, MV	Inflow TV	TR holo	MR/ AoI, PAI	Tei index	SF LV % SF RV %	CVPS
MV	0.48	Yes	Not assessed	Yes	M	+	+	LV — 0.58 RV — 0.68	LV — 27% RV — 25%	7
SW	0.52	Yes	Yes	Yes	M	+	+	LV — 0.62 RV — 0.70	LV — 26% RV — 20%	2
KJ	0.44	Yes	Yes	Yes	M	+	+	LV — 0.60 RV—	LV — 25% RV—	7

								0.69	22%	
SJ	0.28	No	No	No	B	+	–	LV — 0.35 RV — 0.39	LV — 32% RV — 30%	10
KP	0.40	Yes	Yes	No	B	+	+	LV — 0.48 RV— 0.56	LV — 28% RV — 30%	7

AoI — aortic valve insufficiency; B — biphasic flow; CVPS — cardiovascular profile scor; M — monophasic flow; MR — mitral regurgitation; PAI — pulmonary valve regurgitation; SF LV — shortening fraction of left ventricle; SF RV — shortening fraction of right ventricle; SVC — superior vena cava; TR holo — tricuspid regurgitation holosystolic; TV — tricuspid valve

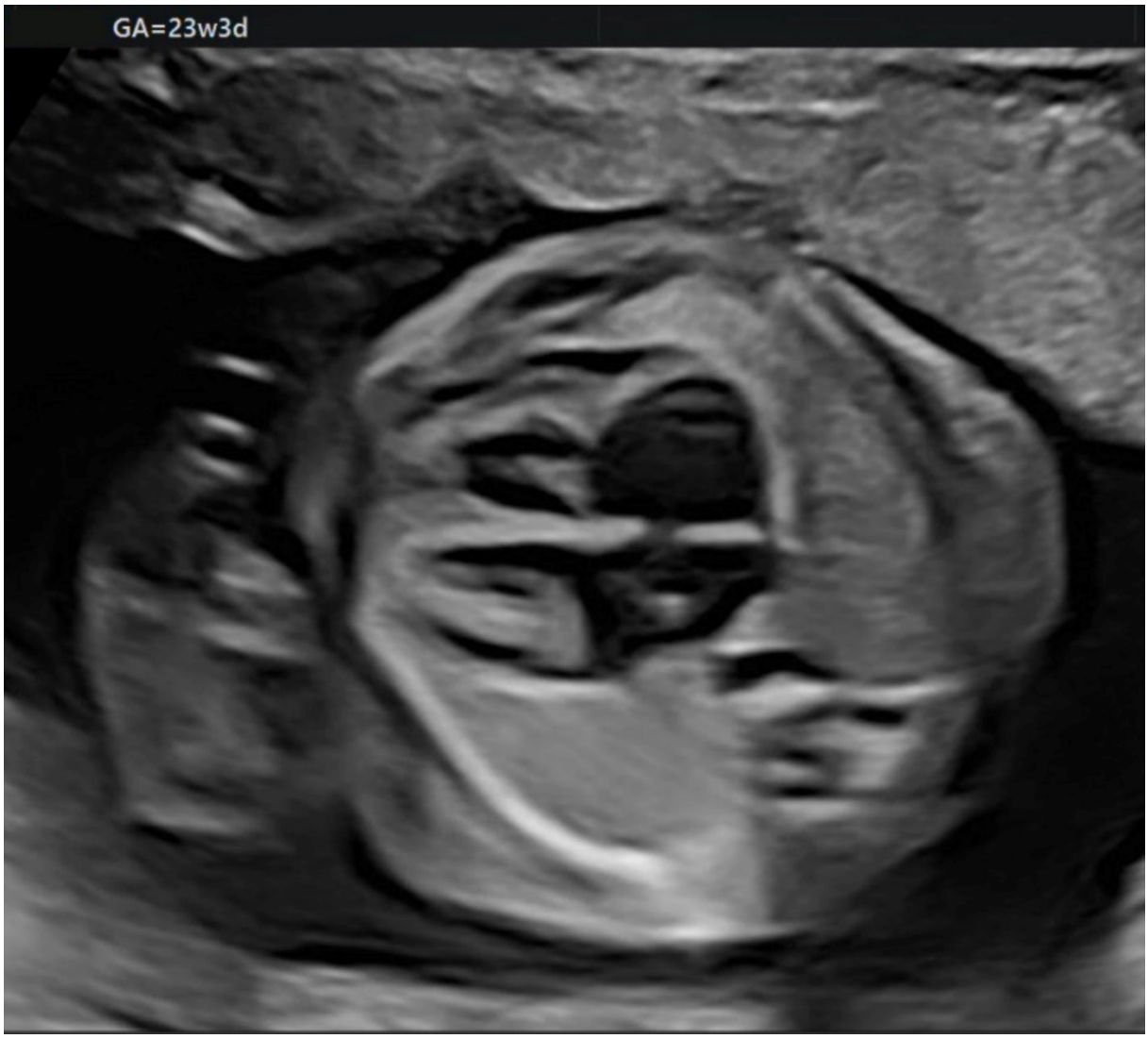

Figure 1. Four chamber view scan — cardiomegaly diagnosed at 23 + 3 weeks of GA (HA/CA–0.40)

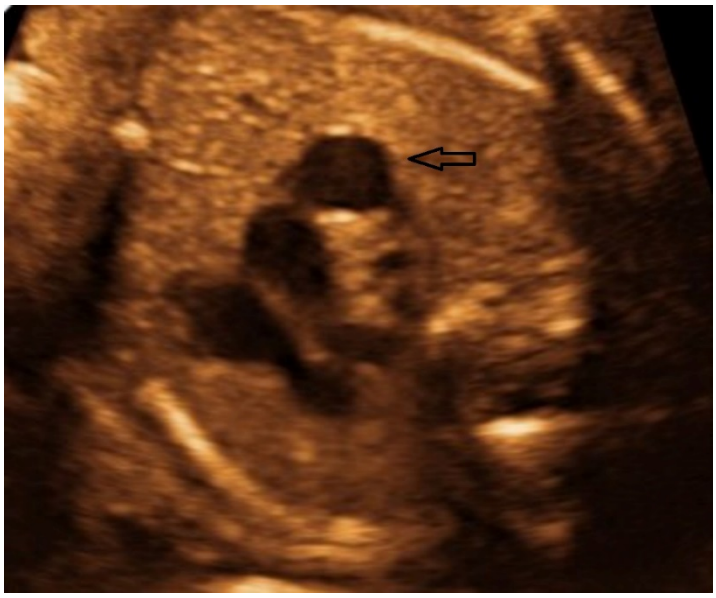

Figure 2. Three-vessel view (3VV) scan with dilated superior vena cava (black arrow)

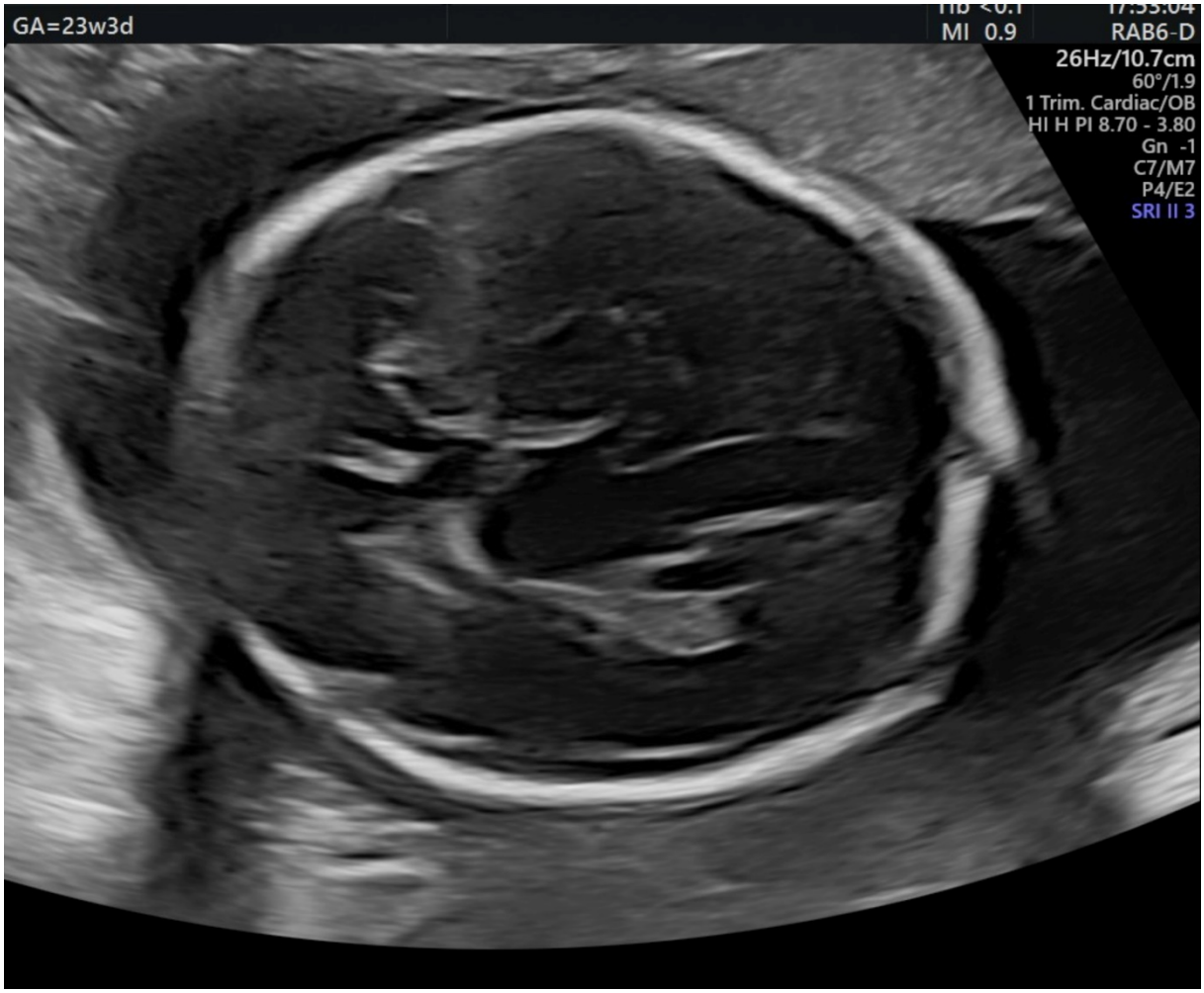

Figure 3. Hypoechogenic cystic mass in the fetal brain in 2D (23+3/7 weeks of GA)

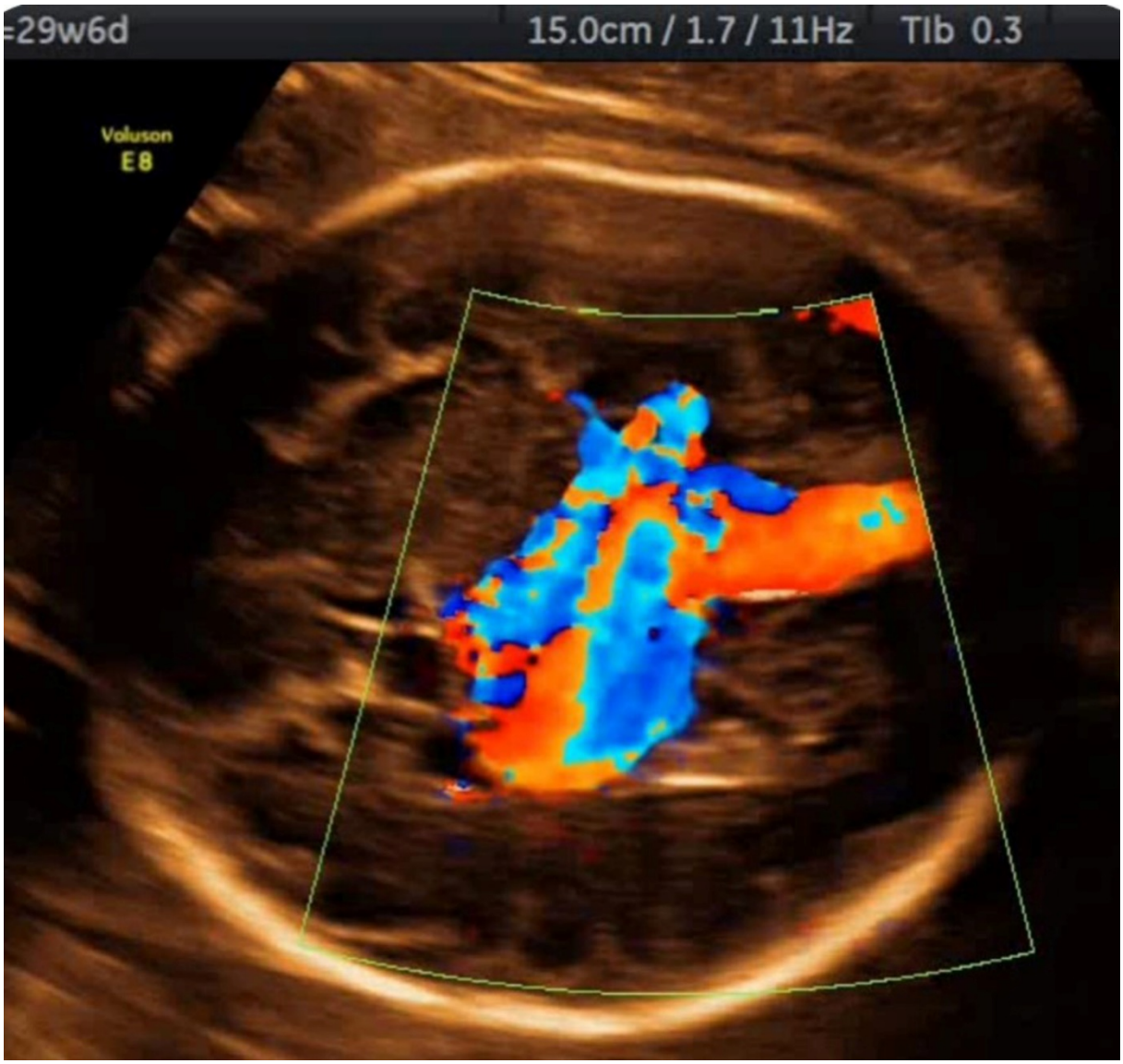

Figure 4. Abnormal arteriovenous malformation coded with color Doppler in fetal brain

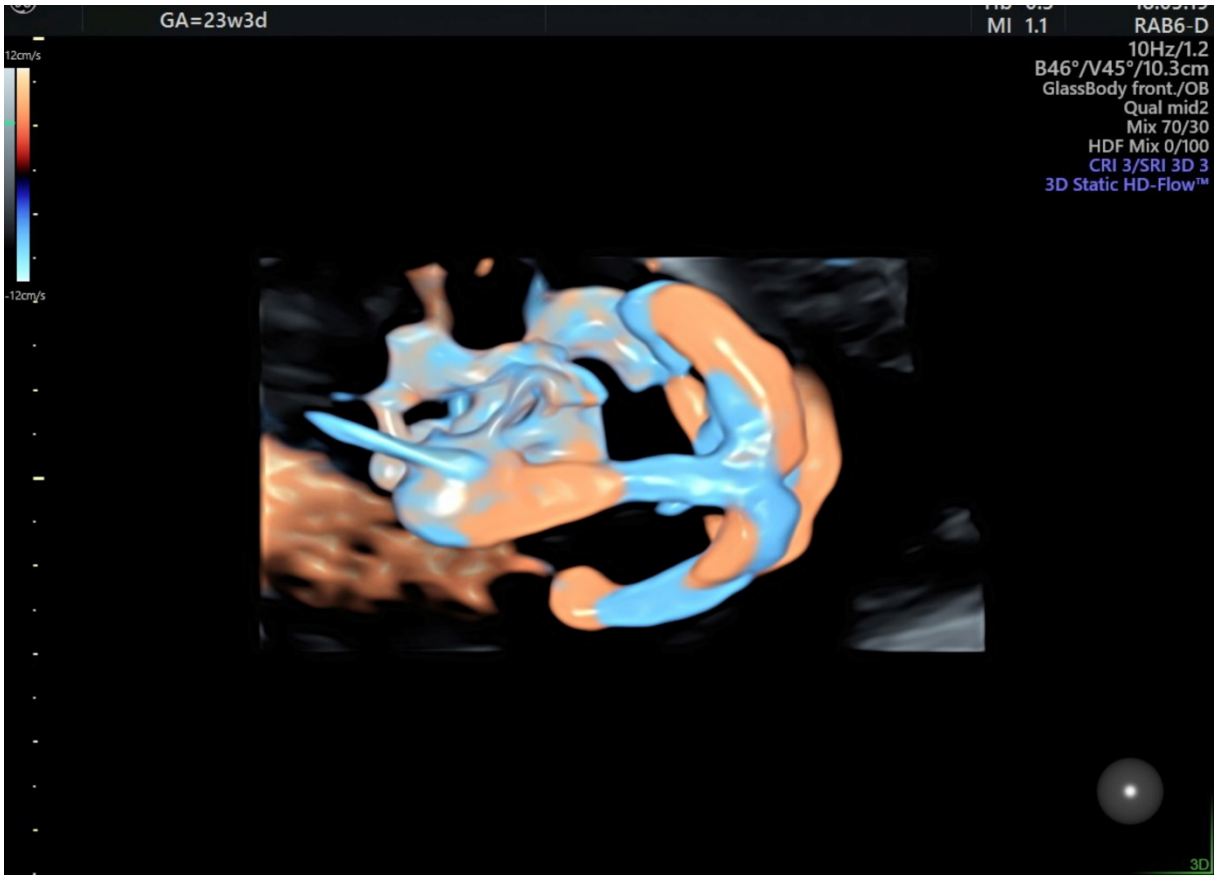

Figure 5. A 3D view of the Vein of Galen malformation