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A large-scale investigation by ultrasound of fetal hepatic venous system variants in China

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Abstract

Aim: To investigate the types, associated anomalies and postnatal outcomes of fetal hepatic venous system (HVS) variants by ultrasound in China. **Material and methods:** A large-scale and prospective investigation of HVS variants for low-risk singleton pregnant women was performed in three academic tertiary referral care centers in China. Ultrasound imaging was used for the identification and follow-up of anatomical variants. Follow-up was conducted once every four weeks prenatally and every two months postnatally, mainly concerned on the adverse events that may appear. **Results:** There were 20848 cases with anatomical variants of fetal HVS identified from 46179 candidates during the study period. Following the anatomical position of variants occurring, four main divisions were present: main portal vein variants (17.9%), intrahepatic portal vein variants (21.30%), intrahepatic persistent right umbilical vein (0.27%) and hepatic vein variants (5.67%). In the fetal period, the pregnancy of all cases was normally continued, except that the pregnancy of two cases, which were associated with multiple anomalies and were terminated by their parents. After birth, approximately 99.47% of the cases with isolated variants or being associated no clinic significant anomalies were normally alive. Approximately 0.50% cases were associated with simple ventricular septum defect or tetralogy of Fallot and further treatment was needed. **Conclusion:** The anatomical variants of fetal HVS may appear as numerical, morphological or positional variants of MPV, intrahepatic PV branches, intrahepatic PRUV and HVs. The majority of cases are isolated or their associated anomalies are not clinically significant and have normal life after birth.

Keywords: fetal hepatic venous system; anatomic variants; ultrasound; prenatal diagnosis

Introduction

The fetal hepatic venous system (HVS) is a special part of the fetal cardiovascular system, which consists of an intra-abdominal segment of umbilical vein (UV), intra- and extrahepatic portal veins (PVs), ductus venous (DV), hepatic veins (HVs) and a portion of inferior vena cava (IVC) [1,2]. The highly oxygenated blood from the placenta is redistributed in fetal HVS before it flows into fetal heart, which is considerably vital to the normal development and growth of fetus [3,4].

Early in the embryonic development, two UVs and two vitelline veins (VVs) supply blood to the fetal liver, and the subsequent development of UVs and VVs follow a set of biological procedures [5-7]. The entire right UV and the cranial portion of the left UV (LUV) regress and the caudal portion of LUV contributes to the left PV (LPV). A great portion of VVs incorporate into the liver parenchyma as hepatic sinusoids and develop the intrahepatic PV branches and HVs. Meanwhile, some portions of the right VV form the intrahepatic segment of IVC, DV and the main portal vein (MPV). Unfortunately, some unexplained factors may interfere the embryonic

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procedures and contribute to the anatomic variants and abnormalities of fetal HVS [8], the variants and abnormalities usually having important similarities in structure, difficult to be differentiated. Prenatal misjudgment may result in mistakes in giving prenatal counseling to the families.

To the best of our knowledge, most of the previous studies focused on the fetal HVS shunts [9-14], only a few studies reported their variants [15]. The variants of fetal HVS have not been systemically described before. Moreover, accurate recognizing and understanding of the ultrasonographic characteristics of the variants is considerably important to distinguish them from the abnormalities. In this study, we aimed to detailly describe the types, associated anomalies and postnatal outcomes of the variants of fetal HVS.

Material and methods

Study population

A prospective study was performed on low-risk singleton pregnancy women in three academic tertiary referral care centers in China over three years, from August 2016 to December 2019. The criteria for inclusion were: *i)* the age of the pregnancy women was from 21 to 30 years and their ultrasound (US) images were clear enough to be read, *ii)* cases with structural abnormalities or "soft labels" were definitely identified and included, and *iii)* follow-up data was complete. All the ethical committees of the participating hospitals approved this study and all pregnant subjects signed their written informed consent.

Ultrasound equipment

Conventional ultrasound screening for fetal structural anomalies was performed according to the International Society of Ultrasound in Obstetrics and Gynecology practice guidelines [16], using Voluson E8 ultrasound system (GE Healthcare Ultrasound, Milwaukee, WI, USA) coupled with a C1-5-D transducer (frequency 2-5 MHz) and IE33 ultrasound system (Philips Medical Systems, Andover, MA) coupled with a C5-1 transducer (frequency 1-5 MHz).

Standardized scanning protocol

Besides the classic three planes scanning approach of fetal abdominal precordial veins, proposed by Yagel et al [17-19], other two planes were added into the screening protocol of the present study. The classic three planes consist of: Plane A, a slightly ventral plane from the classic transverse plane of fetal upper abdomen, to show the connection of MPV and the main intrahepatic PV branches; Plane B, a slightly cephalad to the transverse section of fetal upper abdomen, to evaluate the drainages of DV and HVs into IVC; and Plane C, a parasagittal plane, to assess the courses of UV and DV. The additional two planes added into the protocol were: plane D, a coronal to plane A, to confirm the confluence of superior mesenteric vein (SMV) and spleen vein (SV) into MPV and plane E, the classic transverse plane of fetal upper abdomen, to show the connection of UV to intrahepatic PV (fig 1).

Common anatomy of fetal HVS

The most common anatomy of fetal HVS was defined as follows: the intra-abdominal portion of UV goes up in the left side of fetal abdomen to connect with the sagittal portion of LPV. LPV separately gives rise to its superior, inferior and medial branches, to feed the segments II, III and IV of fetal liver. Then, LPV turns almost 90° to the right to join the right PV (RPV) in the shape of letter L, which is known as the portal sinus (PS). DV emerges from PS and ends at IVC. Outside of the fetal liver, SMV and SV joint together to form MPV. Then MPV bifur-

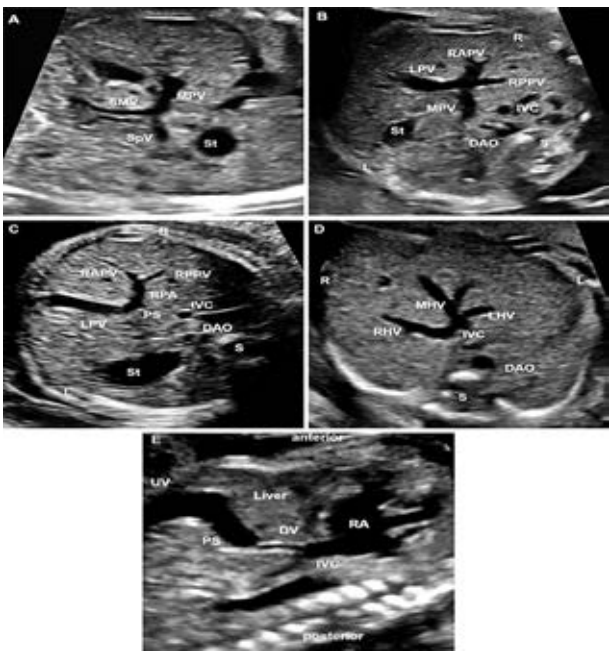

Fig 1. The common anatomy pattern of fetal HVS. Image A) shows that MPV bifurcates into LPV and RPV, and RPV divides into RAPV and PRPV. B) shows that LHV, MHV and RHV collect the venous flow of fetal liver, and usually LHV and MHV share a trunk. C) DV emerges from PS, and ends at IVC with its opening towards FO. D) is the coronal plane shows the confluence of SMV and SV. E) shows that the intra-abdominal segment of UV goes up in the left side of fetal abdomen to connect with the sagittal segment of LPV, then LPV turns almost 90◦ to the right to join the RPV in the shape of letter "L". MPV, main portal vein; LPVi, inferior branch of left portal vein; LPVs, superior branch of left portal vein; LPVm, middle branch of left protal vein; RAPV, right anterior portal vein; RPPV, right posterior portal vein; St: stomach; DAO, descending aorta artery; IVC: inferior vena cava; S: spine; R: right; L: left.

cates into its LPV and RPV. And RPV divides into the anterior (RAPV) and posterior (PRPV) branches, which feed the segment V, VI, VII and VIII of fetal liver. In addition, left, middle and right hepatic veins (LHV, MHV and RHV) drain the hepatic venous flow into IVC. Usually, LHV and MHV share a trunk before they join IVC.

Identification of anatomical variants

Any numerical, morphological or positional variants different from the common anatomy pattern of fetal HVS, but causing no pathophysiological changes in-vivo were considered to be the anatomy variants of fetal HVS. According to this definition, the prenatal judgement of fetal HVS variants and associated anomalies was independently made by two highly-experienced physicians and if a dispute arose, the final decision was made by the third qualified physician.

Non-invasive prenatal testing

Blood samples were collected from the pregnant women to perform non-invasive prenatal testing (Illumina, Inc., San Diego, CA, USA), to test the risks for trisomy 13, 18 and 21.

Follow-up

All the cases with fetal HVS variants were followed-up by US, once every four weeks prenatally and every two months postnatally, from the first time diagnosed with HVS variants to half a year after birth. Follow-up mainly concerned on prenatal adverse event and postnatal treatment.

Statistical analysis

Statistical analysis was performed with SPSS version 19 for Windows (IBM Corp., Armonk, NY, USA). Demographic data are expressed as the mean (range), and the incidence rate results are expressed as number (percentage).

Results

Demographic characteristics

Throughout the study period, there were 20848 (45.15%) cases with the anatomical variants of fetal HVS identified in 46179 candidates who underwent traditional obstetric scanning in the participating hospitals. The main demographic characteristics of all subjects are summarized in Table I.

According to the anatomy positions that the variants occur, all cases were divided into four main types: i) MPV variants, ii) intra-hepatic PV variants, iii) intrahepatic persistent right umbilical vein (PRUV) and iv) HV variants. The definition of these variants and the incidence rates found in our study are detailed in Table II. Figures 2-6 illustrate some of the variants encountered during the study.

Table I. Demographic data for all positive subjects

Parameter	Value
Maternal age (years)	24.48 (21-30)
Gestational age (weeks)	23.71 (21-27)
Fetal biometry (mm)	
BPD	57.71 (51-66)
HC	206.45 (198-236)
AC	179.18 (168-206)
FL	40.31 (35-44)

Data are expressed as mean (range). AC, abdominal circumference; FL, femur length; BPD, biparietal diameter; HC, head circumference.

To be specific, in cases with PRUV, the intra-abdominal portion of UV goes up in the right side of fetal abdomen (being opposite to that of the fetuses with LUV); the distribution of RPV, RAPV and RPPV was in the shape of letter "Π" (being same as that of LPV, LPVi and LPVs in the LUV fetuses); and the distribution of LPV, LPVi and LPVs was in the shape of letter "Y" (being same as the shape of RPV, RAPV and RPPV in LUV fetuses). In brief, the intrahepatic PV distribution of PRUV cases were the mirror image of that of LUV fetuses.

Associated anomalies and NIPT results

The majority of the variants of fetal HVS were isolated (99.06%), only a small proportion of cases being associated with structural anomalies (0.94%) (Table III) and approximately 96.44% of the associated anomalies were related to fetal cardiovascular system. Specifically, two cases in PRUV group were identified to be associated with multiple malformations, and one of them was confirmed with trisomy 18 by NIPT. The rest of the cases were low risk for trisomy 13, 18 and 21.

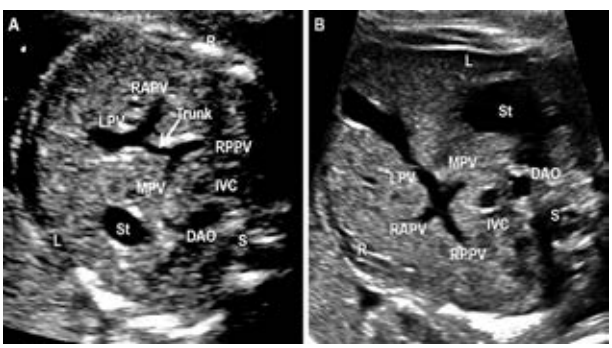

Fig 2. Main portal vein (MPV) variants: A) MPV bifurcates into RPPV and a trunk, then the trunk divides into RAPV and LPV; B) Trifurcation "MPV", MPV directly divides into RAPV, RPPV and LPV. LPV, left portal vein; RAPV, right anterior portal vein; RPPV, right posterior portal vein; St, stomach; DAO, descending aorta artery; IVC, inferior vena cava; S, spine; R, right; L, left.

Follow-up findings

In the fetal period, due to the poor prognosis, the pregnancy of the two cases being associated with multiple malformations in the PRUV group were terminated by their parents. After birth, the cases with isolated variants and those being associated with no clinical significant anomalies were alive, with the incidence rate being approximately 99.47%. There were 105 (0.50%) cases being associated with simple ventricular septum defect or tetralogy of Fallot, which needed surgical repairs or interventional treatment. Additionally, one case in HVV group being associated with right aortic arch had no airway or esophagus obstruction symptoms till six months old.

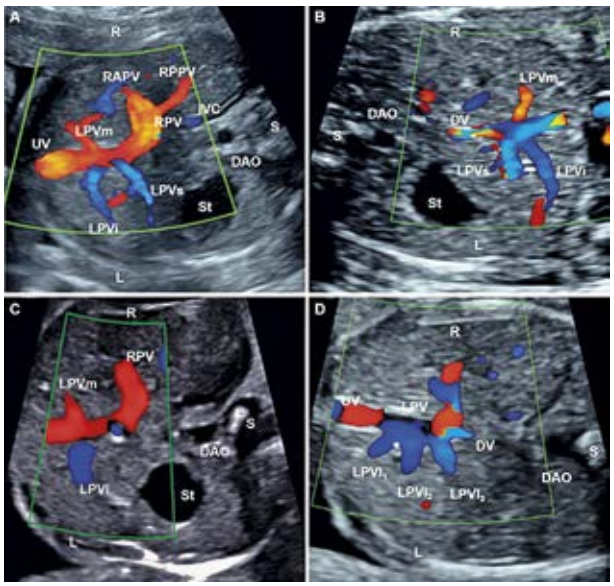

Fig 3. LPV branches variants: A) shows the distribution of the normal intra-hepatic portal veins with color doppler; B) shows the LPVs and LPVi share a trunk. The branches of LPV feeding the lateral lobe of fetal liver could be one (C) or three (D). MPV, main portal vein; LPVi, inferior branch of left portal vein; LPVs, superior branch of left portal vein; LPVm, middle branch of left protal vein; RAPV, right anterior portal vein; RPPV, right posterior portal vein; St: stomach; DAO, descending aorta artery; IVC: inferior vena cava; S: spine; R: right; L: left.

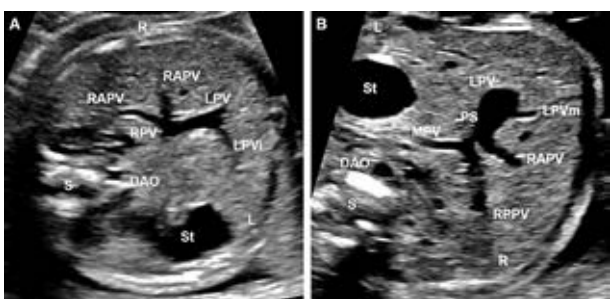

Fig 4. Absence of some segments of intra-hepatic PV. Image A) shows that the LPVh was absent, RPV directly connected to PS, with non-visualization of the typical letter "L" sign. Image B) show that both LPVh and RPV were absent. MPV, main portal vein; PS, portal sinus; LPV, left portal vein; LPVi, inferior branch of left portal vein; LPVh, the horizontal segment of left portal vein; RPV, right portal vein; RAPV, right anterior portal vein; RPPV, right posterior portal vein; St: stomach; DAO, descending aorta artery; S: spine; R: right; L: left.

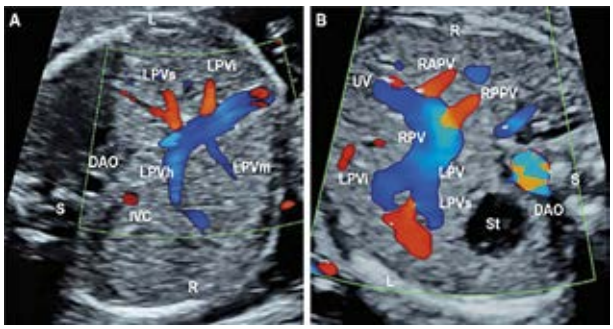

Fig 5. PRUV variants. A 23-week fetus with PRUV, its intrahepatic PV distribution is the mirror image of that of the normal left umbilical vein. LPV, left portal vein; RPV, right portal vein; LPVh, the horizontal segment of left portal vein; LPVi, inferior branch of left portal vein; LPVs, superior branch of left portal vein; LPVm, middle branch of left protal vein; RAPV, right anterior portal vein; RPPV, right posterior portal vein; St: stomach; DAO, descending aorta artery; IVC: inferior vena cava; S: spine; R: right; L: left.

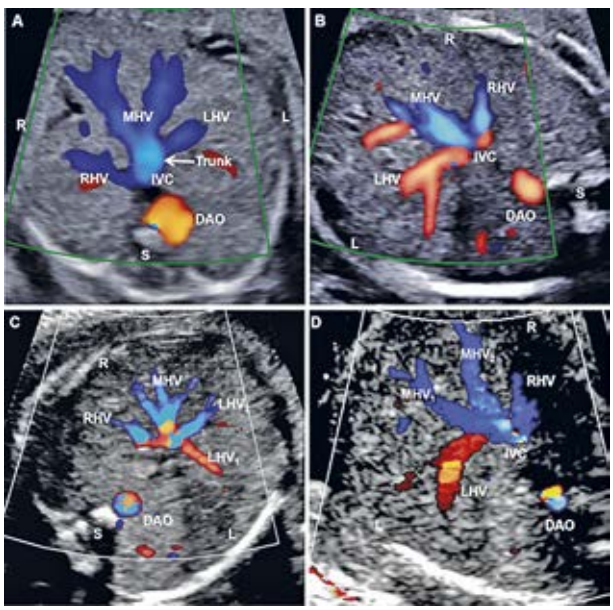

Fig 6. HV variants. Image A) shows that LHV and MHV share a trunk before connecting to IVC. Image B) shows that LHV, MHV and RHV connect to IVC respectively. Sometimes there could be two LHVs (C) or MHVs (D). LHV, left hepatic vein; MHV, middle hepatic vei; RHV, right hepatic vein; DAO, descending aorta artery; IVC: inferior vena cava; S: spine; R: right; L: left.

Table II. Division of fetal HVS variants

Types	Definition	Incidence rates
MPV variants		8266 (17.90)
RAPV and LPV share a trunk	MPV bifurcates into RPPV and a trunk, then the trunk divides into RAPV and LPV	5609 (12.15)
Trifurcated MPV	MPV divides into LPV, RAPV and RPPV	2657 (5.75)
Intrahepatic PV variants		9839 (21.30)
LPVi and LPVs share a trunk	The beginning portion of LPVi and LPVs share a trunk	4917 (10.65)
Numberical variants	The number of LPV branches feeding left lateral lobe of fetal liver could be one or three	3349 (7.25)
Absence of LPVh	LPVh is absent	871 (1.89)
Absence LPVh and RPV	Both LPVh and RPV are absent	702 (1.52)
Intrahepatic PRUV		124 (0.27)
Intrahepatic PRUV	The intrahepatic PV distribution is the mirror image of that of LUV	124 (0.27)
Hepatic veins variants		2619 (5.67)
One LHV, MHV and RHV	LHV, MHV and RHV independently connect to IVC	1450 (3.14)
Two LHVs	One MHV, one RHV, but two LHVs connect to IVC	645 (1.40)
Two MHVs	One LHV, one RHV, but two MHVs connect to IVC	524 (1.13)

The results are expressed as number (%). MPVV: main portal vein variants; IHPVV: intra-hepatic vein variants; PRUV: persistent right umbilical vein; HVV: hepatic vein variants; PLSVC: persistent of superior vena cava; VSD, ventricular septum defect; TOF: tetralogy of Fallot, RAA: right aortic arch; AVSD: atrioventricular septal defect; CPC: choroid plexus cyst; CLP: cleft lip and palate; SUA: single umbilical artery; TOP: termination of pregnancy; PNIPT: positive non-invasive prenatal testing; SNAB: surgery needed after birth; WAAB: well alive after birth.

Table III. Associations and outcomes of fetuses with HVS variants

Items	MPVV	IHPVV	PRUV	HVV
Isolated rate	8211 (99.33)	9713 (98.72)	119 (95.97)	2608 (99.60)
Associated anomalies				
	VSD: 18 (0.22) CPC:7 (0.08) TOF: 4 (0.05) PLSVC: 26 (0.31)	SUA: 46 (0.47) VSD: 80 (0.81)	VSD: 3 (2.42) VSD+CPC+Rocking chair foot 1 (0.81, trisomy 18) AVSD+CLP+Hemivertebrae 1 (0.81)	PLSVC:10 (0.38) RAA:1 (0.04)
Outcomes				
IUI	0	0	0	1 (0.04)
TOP	0	0	2 (1.61)	0
SNAB	22 (0.27)	80 (0.81)	3 (2.42)	0
WAAB	8244 (99.73)	9759 (99.19)	119 (95.97)	2609 (99.96)

The results are expressed as number (%). MPVV: main portal vein variants; IHPVV: intra-hepatic vein variants; PRUV: persistent right umbilical vein; HVV: hepatic vein variants; PLSVC: persistent of superior vena cava; VSD, ventricular septum defect; TOF: tetralogy of Fallot, RAA: right aortic arch; AVSD: atrioventricular septal defect; CPC: choroid plexus cyst; CLP: cleft lip and palate; SUA: single umbilical artery; TOP: termination of pregnancy; PNIPT: positive non-invasive prenatal testing; SNAB: surgery needed after birth; WAAB: well alive after birth.

Discussion

In the present study we found that the anatomical variants of fetal HVS may appear as the numerical, morphological or positional variants of MPV, intrahepatic PV branches, intra-abdomen UV and HVs. Each subtype of fetal HVS variants has its unique features in anatomy, which are the key points of distinguishing them from each other during prenatal examination. This study allowed for a better understanding of the US characteristics of the variants of fetal HVS.

The MPV variants focus on the patterns that MPV giving rise to its branches. The classifications of MPV variants in our study have something in common with that of MPV-PS anastomosis patterns described in previous studies, although it is not exactly the same. The patterns of fetal MPV-PS anastomosis were divided into H-shaped, T-shaped and X-shaped junction [15]. X-shaped anastomosis is similar to the most common bifurcate MPV, H-shaped anastomosis is similar to that RAPV and LPV sharing a trunk in the present study. But T-shaped anastomosis shows a large range of angle of

connection of MPV and PS, could be any type of the MPV variants described in the present study. In the fetal period, an accurate recognition of the variants is essential to avoid the misdiagnosis of HVS abnormalities. In adult patients needing liver surgery, a precise analysis of all hepatic vascular abnormalities before an operation is mandatory, especially in liver transplantation. Trifurcation of the MPV, and RAPV and LPV sharing a trunk has been claimed to increase the complexity of surgical procedures, making the portal vein clamping more difficult [20].

Both the intrahepatic PV variants and the cases with intrahepatic PRUV occur at the branches of intrahepatic PV, including the numerical, morphological or distribution variants of intrahepatic PV branches. Same as the MPV variants, the intrahepatic PV variants cause no negative results to fetal development. In adult cases needing hepatectomy, when being associated with numerical intrahepatic PV variants, the PV branches that will be resected should be completely occluded during hepatic dissection, to decrease the risk of bleeding. The morphological or distribution of the intrahepatic PV variants increase the complexity of the procedure [20-22].

The variants of fetal HVs are commonly numerical and these variants have no negative influences on hepatic venous return. In adult cases, the presence of the HV variants is of prime importance during the insertion of a transjugular intrahepatic portosystemic shunt [21,22].

In recent years, the prenatal diagnosis of the structural abnormalities of fetal HVS have been increasedly causing concern in the clinical practice [22-26]. The variants and the abnormalities of fetal HVS have a lot of similarities in anatomy, but the outcomes of them are greatly different. An accurate differentiation between the two situations during prenatal examination is pretty important to predict the outcomes after birth. The detailed description of the clinic characteristics of the variants of fetal HVS in the present study provided the knowledge of distinguishing the variants from the abnormalities and is potentially valuable in giving a comprehensive and promising maternal-fetal counseling.

To describe the variants of fetal HVS prenatally in-vivo, US is used as a non-invasive and convenient imaging technique. However, compared to fetal magnetic resonance, US imaging is feasible to demonstrate the main branches of fetal HVS, but the small intrahepatic PV branches and the accessory HVs may not be clearly detected.

In **conclusion**, fetal HVS variants may appear as numerical, morphological or positional variants of MPV branches, intrahepatic PV branches, intrahepatic PRUV and HVs. A majority of cases are isolated or their associated anomalies are not clinically significant and have normal life after birth.

Conflict of interest: none

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