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REVIEW

Imaging assessment and interventional management of paediatric portal hypertension: an update

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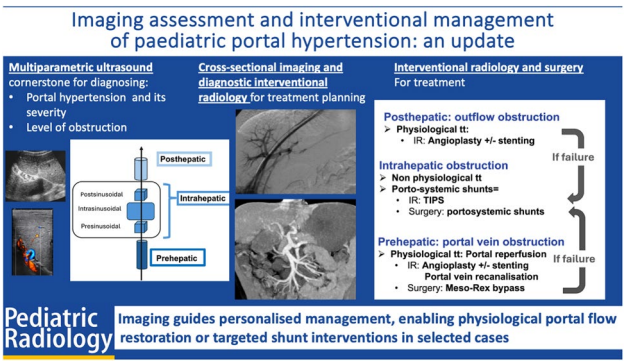
Abstract

Portal hypertension in children presents distinct etiologies, clinical manifestations, and management strategies compared with adults, making accurate imaging evaluation essential across the diagnostic and therapeutic pathway. Ultrasound remains the first-line modality, providing real-time assessment of vascular anatomy, haemodynamics, splenic involvement, and portosystemic collaterals. Colour Doppler ultrasound and pulsed-wave Doppler ultrasound, along with systematic scanning protocols, enable early identification of portal vein obstruction, cavernous transformation, and hepatofugal flow patterns. Elastography techniques, particularly spleen stiffness measurement, provide valuable non-invasive biomarkers of clinically significant portal hypertension and variceal risk and complement conventional imaging. When ultrasound findings are inconclusive, contrast-enhanced ultrasound, computed tomography (CT), and magnetic resonance imaging (MRI) offer detailed vascular mapping, characterisation of parenchymal changes, and preoperative evaluation. MRI—especially dynamic contrast-enhanced sequences, magnetic resonance cholangiopancreatography (MRCP), and magnetic resonance elastography—provides comprehensive anatomical and functional assessment without ionising radiation. Interventional and surgical procedures, including portal vein recanalisation, Meso-Rex bypass, and transjugular intrahepatic portosystemic shunts, require precise imaging both before and after treatment. Long-term surveillance relies on multimodal imaging to detect stenosis, thrombosis, or flow-related complications and to guide timely reintervention. With continuous advances in elastography, high-resolution CT, and MRI-based haemodynamic techniques, imaging plays an expanding role in the tailored management of paediatric portal hypertension, supporting early diagnosis, therapeutic decision-making, and structured follow-up.

Graphical Abstract



Keywords Doppler ultrasound · Elastography · Interventional radiology · Paediatric imaging · Portal hypertension · Portal vein · Cavernous transformation of [supplementary concept]

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Introduction

Portal hypertension is a rare condition in paediatric patients, characterised by distinctive clinical features and potentially different management approaches compared to adults. As in adults, portal hypertension is defined as portal venous pressure exceeding 10 mmHg and a hepatic venous pressure gradient exceeding 5 mmHg, with complications becoming more likely when the hepatic venous pressure gradient exceeds 10 mmHg [1]. Hepatic venous pressure gradient measurement is an invasive, albeit minimally invasive, procedure that generally requires general anaesthesia in paediatric patients, which likely explains its infrequent use in this population. As a result, the concept of clinically evident portal hypertension is often preferred in children and is based on clinical signs such as splenomegaly with hypersplenism, varices, and ascites [2]. The causes of paediatric portal hypertension can be categorised as prehepatic (e.g. extrahepatic portal vein obstruction), intrahepatic (presinusoidal, sinusoidal, or postsinusoidal), or posthepatic [1]. In children, the main causes vary by country, but the most common are extrahepatic portal vein obstruction and biliary cirrhosis, most frequently related to biliary atresia. The causes of paediatric portal hypertension are summarised in Table 1.

Non-invasive imaging of portal hypertension: diagnosis approach and algorithm

Ultrasound (US) represents the cornerstone of the diagnostic evaluation of suspected portal hypertension in children, providing a comprehensive and reliable first-line assessment of vascular anatomy, haemodynamics, and associated abdominal findings [3–5]. In selected clinical scenarios, cross-sectional imaging techniques, including computed tomography angiography (CTA), magnetic resonance angiography (MRA), and quantitative imaging methods for liver and spleen assessment, offer complementary

Table 1 Causes of portal hypertension in paediatric patients and their key clinical and imaging features. Biliary atresia and cystic fibrosis may both cause sinusoidal and presinusoidal portal hypertension. *EHPVO* extrahepatic portal vein obstruction, *FNH* focal nodular hyperplasia, *PSVD* porto-sinusoidal vascular disease

Category	Main causes	Key imaging features
Prehepatic	- Extrahepatic portal vein obstruction (EHPVO): umbilical vein catheter, umbilical infection, rare congenital vascular malformations, inherited thrombophilia (protein C/S deficiency, factor V Leiden, antiphospholipid syndrome) - Arterio-portal fistula, acquired or congenital	- Cavernous transformation of the portal vein +/- portal hypertension - May present with splenomegaly, hypersplenism, or portosystemic varices - Normal liver stiffness and elevated spleen stiffness - Reversed and arterialised portal flow (more or less extended)
Intrahepatic- presinusoidal	- Porto-sinusoidal vascular disease (PSVD) including congenital hepatic fibrosis: cystic fibrosis and biliary atresia	- Narrowing or occlusion of intrahepatic portal veins sometimes involving the main portal vein - Cavernoma with patent main portal vein can indicate PSVD - FNH like lesions - Liver morphology ranging from normal to cirrhosis-like appearance - Normal or mildly increased liver stiffness and increased spleen stiffness
Intrahepatic- sinusoidal	- Biliary cirrhosis: biliary atresia, cystic fibrosis, primary sclerosing cholangitis - Other causes of chronic liver disease: metabolic, viral, autoimmune, genetic	- Signs of chronic liver disease (surface nodularity, parenchymal heterogeneity, dysmorphy) - Increased liver and spleen stiffness
Intrahepatic- postsinusoidal	- Sinusoidal obstruction syndrome (veno-occlusive disease): e.g. secondary to chemotoxicity, toxins…	- Enlarged liver, heterogeneous - Slow, reduced or even reversed portal venous flow - Increased liver and spleen stiffness
Posthepatic	- Budd–Chiari syndrome - Inferior vena cava malformations - Right heart failure or elevated right atrial pressure - Constrictive pericarditis	- Hepatic venous outflow obstruction: hepatic vein enlargement, stenosis, thrombosis - Slow or reversed portal venous flow - Hepatomegaly with heterogeneous parenchyma - Increased liver and spleen stiffness - Associated cardiac abnormalities on further evaluation

information, enhancing diagnostic accuracy and enabling more detailed characterisation of the underlying pathology [6].

Multiparametric ultrasound assessment

A comprehensive abdominal ultrasound examination is strongly recommended in any clinical context suggestive of portal hypertension, particularly in children presenting with gastrointestinal bleeding, unexplained splenomegaly, abnormal hepatic or biliary findings, or known conditions predisposing to chronic liver disease and/or portal hypertension, as it directly influences subsequent diagnostic evaluation and therapeutic decision-making.

Table 2 summarises the main B-mode and colour Doppler ultrasound features used in the evaluation of paediatric portal hypertension.

Table 3 proposes an imaging-based diagnostic approach to paediatric portal hypertension, integrating key findings to guide aetiological orientation.

The ultrasound assessment should be performed systematically and comprehensively to ensure complete evaluation of hepatic morphology, portal venous anatomy, haemodynamics, and potential collateral pathways.

Table 2 Summary of B-mode and colour Doppler ultrasound features used in the evaluation of paediatric portal hypertension. *EHPVO* extrahepatic portal vein obstruction, *IVC* inferior vena cava, *PHT* portal hypertension, *PSVD* porto-sinusoidal vascular disease

Structure/vascular territory	Feature assessed	Key imaging findings	Clinical relevance
Liver	Size and margins	- Hepatomegaly or atrophy - Irregular or nodular surface	Suggests chronic liver disease, cirrhosis
	Echotexture	- Coarse, heterogeneous, or nodular parenchyma	Supports fibrosis, cirrhosis, chronic liver disorder, infiltrative, or neoplastic processes
		- Focal echogenic or hypoechoic lesions or parenchymal heterogeneity	May suggest metabolic, storage infiltrative, or neoplastic disease
	Biliary tree	- Bile duct dilatation, wall thickening, or biliary sludge	Suggests obstruction, malformation, or sclerosing cholangitis
		- Bile lakes: anechoic or echogenic cystic lesions	Suggests biliary atresia in cholestatic patients with acholic stools
Portal vein	Patency, size, wall	- Dilatation in intrahepatic PHT except sometimes in biliary atresia - Reduced calibre or mural irregularity in chronic portal vein obstruction	Helps differentiate intrahepatic vs. extrahepatic disease and assess chronicity
	Flow (direction, velocity, waveform)	- Reduced velocity, to-and-fro or hepatofugal flow	Marker of PHT severity
		- Arterialised flow	Suggests an arterio-portal shunt
Spleen	Size, echostructure	- Splenomegaly	Supports PHT
Splenic vein	Patency, flow	- Reduced velocity, to-and-fro or hepatofugal flow - Thrombosis	Suggests severe PHT
Cavernous transformation	Collateral periportal network	- Serpiginous periportal collaterals replacing portal vein	Indicates chronic EHPVO
		- Cavernoma with patent portal vessel	Suggests PSVD
Hepatic veins/IVC	Patency, waveform	- Loss of triphasic waveform, continuous flow, reduced flow velocity, reversed flow, or absent flow	Suggests hepatic venous outflow obstruction
Collateral circulation	Collateral pathways	- Varices, vascular masses, omental thickening	Confirms PHT and bleeding risk
		- Patent ductus venosus beyond 1 month of age	Suggests PHT with neonatal onset
Kidneys	Size, morphology	- Enlarged or small kidneys - Cysts - Abnormal corticomedullary differentiation	Suggests syndromic disease (e.g. congenital hepatic fibrosis, Alagille syndrome...)
Abdomen/pelvis	Ascites	Anechoic peritoneal fluid	Suggests severe PHT or hepatic decompensation

Table 3 Imaging-based diagnostic approach to paediatric portal hypertension. *EHPVO* extrahepatic portal vein obstruction, *IH* intrahepatic, *IVC* inferior vena cava, *N* normal, *PHT* portal hypertension, *PSVD* porto-sinusoidal vascular disease, *SOS* sinusoidal obstruction syndrome

Liver parenchyma	Main portal vein	Hepatic veins IVC	Spleen size	Splenic vein	Elastography		Most likely aetiology
					Liver	Spleen	
Normal	Patent hepatopetal	Patent	↑	Patent	N	N	Early PHT or mild suspicion – monitor
	Patent	Patent	↑	Thrombosed	N	↑	Splenic vein thrombosis
	Replaced by collaterals	Patent	↑	Patent hepatopetal/ hepatofugal	N	↑	EHPVO
Altered	May be enlarged or thrombosed	Patent	↑	Patent hepatopetal/ hepatofugal	↑	↑	Intrahepatic PHT
	Small	Patent	↑	Patent hepatopetal/ hepatofugal	↑	↑	Biliary atresia
	May be enlarged or thrombosed	Patent	↑	Patent	N or ↑	↑	PSVD
Enlarged Normal or altered	Patent	Thrombosed/stenosed	N or ↑	Patent	↑	↑	Budd–Chiari or cardiac cause or SOS

B-mode, colour Doppler, and pulsed-wave ultrasound

Ultrasonography, including colour Doppler ultrasound and pulsed-wave Doppler, is essential for assessing portal hypertension, as it enables direct visualisation of vascular anatomy and real-time haemodynamic evaluation. Changes in the calibre of the extrahepatic portal vein can indicate underlying pathology: dilation of the main portal vein suggests portal hypertension, whereas a reduced calibre vessel may be observed in chronic extrahepatic portal vein obstruction or portal vein hypoplasia, such as in biliary atresia.

Under normal conditions, portal and splenic vein flow is hepatopetal and continuous, with gentle modulation by respiration. In contrast to other organs, the hepatic artery and the portal veins run in the same direction, producing the same colour encoding on colour Doppler ultrasound. In portal hypertension, portal venous flow velocities and phasicity may be progressively and significantly reduced with the worsening of the disease. A to-and-fro pattern may be observed in advanced disease, whereas sustained complete hepatofugal flow indicates severe portal hypertension. Hepatofugal flow in the splenic vein should raise concern for significant portal hypertension, although it may occasionally reflect segmental portal hypertension confined to the splenic vein. Arterialisation of the portal flow on pulsed-wave Doppler of any segment of the portal system should raise the possibility of arterio-portal communications.

Portal venous flow is influenced by mesenteric inflow related to digestion, leading to significant differences between the fasting and postprandial states. In healthy fasting supine individuals, transient flow reversal may occur in the anterior subsegmental portal veins. The fasting condition should then be considered when interpreting Doppler findings.

A major limitation of colour Doppler ultrasound is patient body habitus or marked hepatic morphological distortion, particularly in cases of atrophy, which may compromise the feasibility of the examination.

Cavernous transformation is a common result of chronic extrahepatic portal vein obstruction, characterised by the replacement of the normal portal vein with a network of tortuous periportal collaterals [7]. A frequent pitfall is misinterpreting a large cavernous vein as a patent main portal vein. The normal main portal vein typically appears as a straight vessel at the liver hilum, whereas cavernous veins always demonstrate tortuosity along their course (Fig. 1). Another potential pitfall is the presence of cavernous veins alongside a patent main portal vein or intrahepatic veins. Conversely, the presence of cavernous collaterals alongside a patent main portal vein or patent intrahepatic portal branches should raise suspicion of porto-sinusoidal vascular disease (PSVD) rather than extrahepatic portal vein obstruction [8, 9] (Fig. 1).

Finally, the ultrasound assessment must include careful evaluation of the hepatic veins and the inferior vena cava (IVC). Although Budd–Chiari syndrome is uncommon in children, it remains an important differential diagnosis, particularly in certain geographic regions such as Asia. Imaging findings suggestive of hepatic venous outflow obstruction include enlarged, narrowed, or occluded hepatic veins or IVC; loss of the normal triphasic hepatic venous waveform; continuous low-velocity flow; focal velocity acceleration at sites of stenosis; and the presence of abnormal venous pathways or intervenous collateral connections. Additional

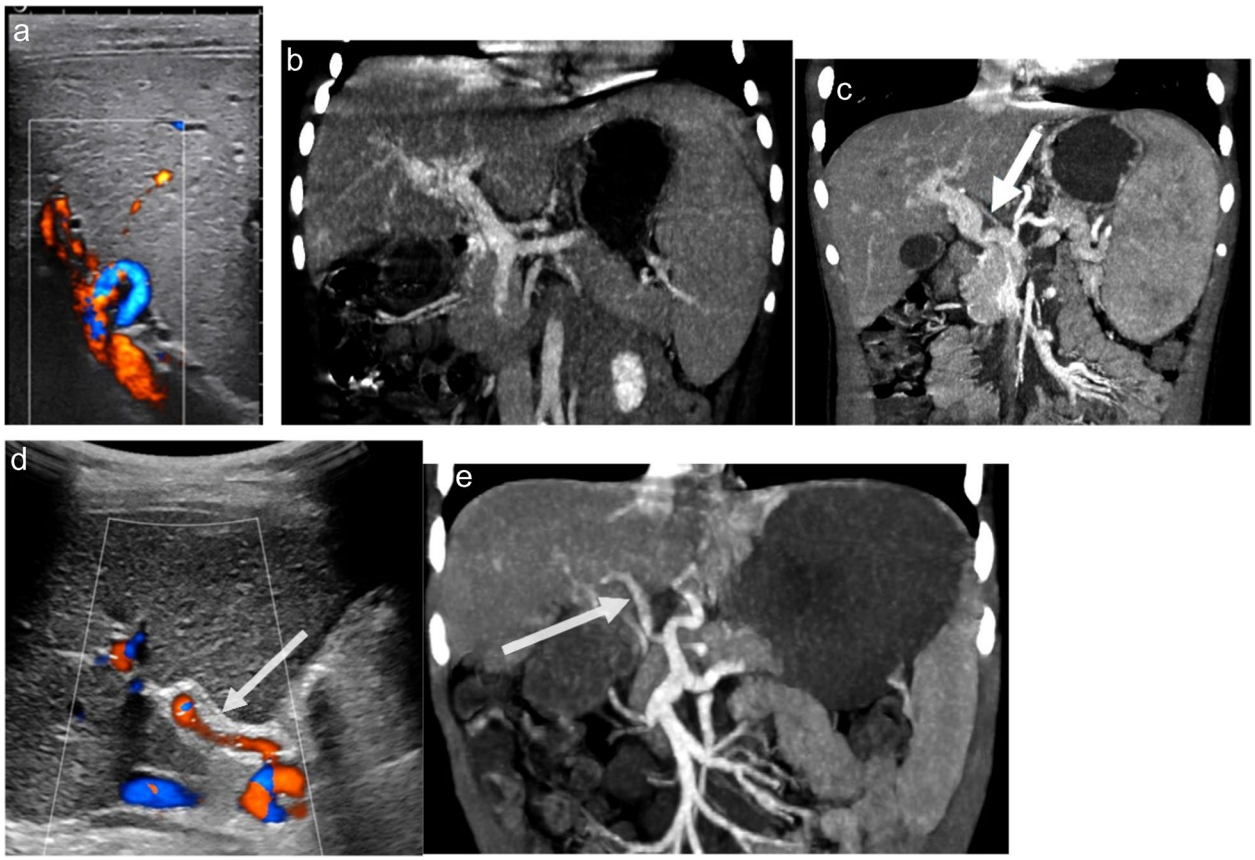

Fig. 1 Pitfalls in the evaluation of extrahepatic portal vein obstruction (EHPVO) in a 5-year-old boy referred for the unconfirmed suspicion of EHPVO with cavernous transformation (**a**, **b**), an 11-year-old boy referred for EHPVO (**c**), and a 1-year-old boy referred for portal hypertension of unknown origin (**d**, **e**). **a** Colour Doppler ultrasound intercostal view shows the hepatic hilum with the simultaneous visibility of a patent portal vein and collateral venous circulation not consistent with EHPVO. **b** Coronal-reformatted maximum intensity projection (MIP) contrast-enhanced CT (CE-CT) confirms the coexistence of a patent main portal vein and parallel periportal collateral veins. This pattern suggests a porto-sinusoidal venous disorder associated with congenital hepatic fibrosis, which was confirmed histologically. **c** Coronal-reformatted MIP CE-CT obtained in the portal venous phase shows a large tortuous collateral vessel consistent with cavernous transformation and a thin straight remnant of the main portal vein (*arrow*). **d** Colour Doppler ultrasound intercostal view shows a tortuous periportal vessel at the hepatic hilum initially misinterpreted as a patent main portal vein (*arrow*). **e** Coronal reformatted MIP CE-CT in the portal venous phase confirms the cavernous transformation of the portal vein (*arrow*) consistent with EHPVO

supportive features include hepatomegaly, slow or reversed portal venous flow, and ascites.

Assessment of portosystemic collaterals is an essential component of the ultrasound examination, as their presence confirms portal hypertension and provides indirect information regarding its severity. Specific scanning approaches may facilitate their detection. In infants, thickening of the lesser omentum may suggest portal hypertension [5], and a persistent patent ductus venosus beyond the first month of life may indicate neonatal portal hypertension. In older children, coronal imaging planes including the spleen and left kidney are particularly useful for identifying direct and indirect spleno-renal collaterals. Patency of the para-umbilical vein can be assessed by tracing its course from the left portal vein along the ligamentum teres. Typical collateral pathways and flow patterns are illustrated in Fig. 2.

The spleen is another key component of the ultrasound evaluation, as splenomegaly represents the most frequent imaging sign of portal hypertension across all paediatric age groups and has been reported in up to 98% of affected children [10]. However, splenomegaly may be less prominent or absent in posthepatic portal hypertension, such as in Budd–Chiari syndrome.

Spleen size increases with age, height, and weight, with the strongest correlations usually seen with height and weight. Established normative reference values are available and should be used for interpretation [11–13]. Measurements should be obtained in a reproducible longitudinal plane and interpreted using age- and size-adjusted reference standards. In addition to splenic size, parenchymal echotexture should be assessed, and focal lesions or infarctions should be documented.

Because splenomegaly may represent the earliest or only imaging manifestation of portal hypertension, colour Doppler

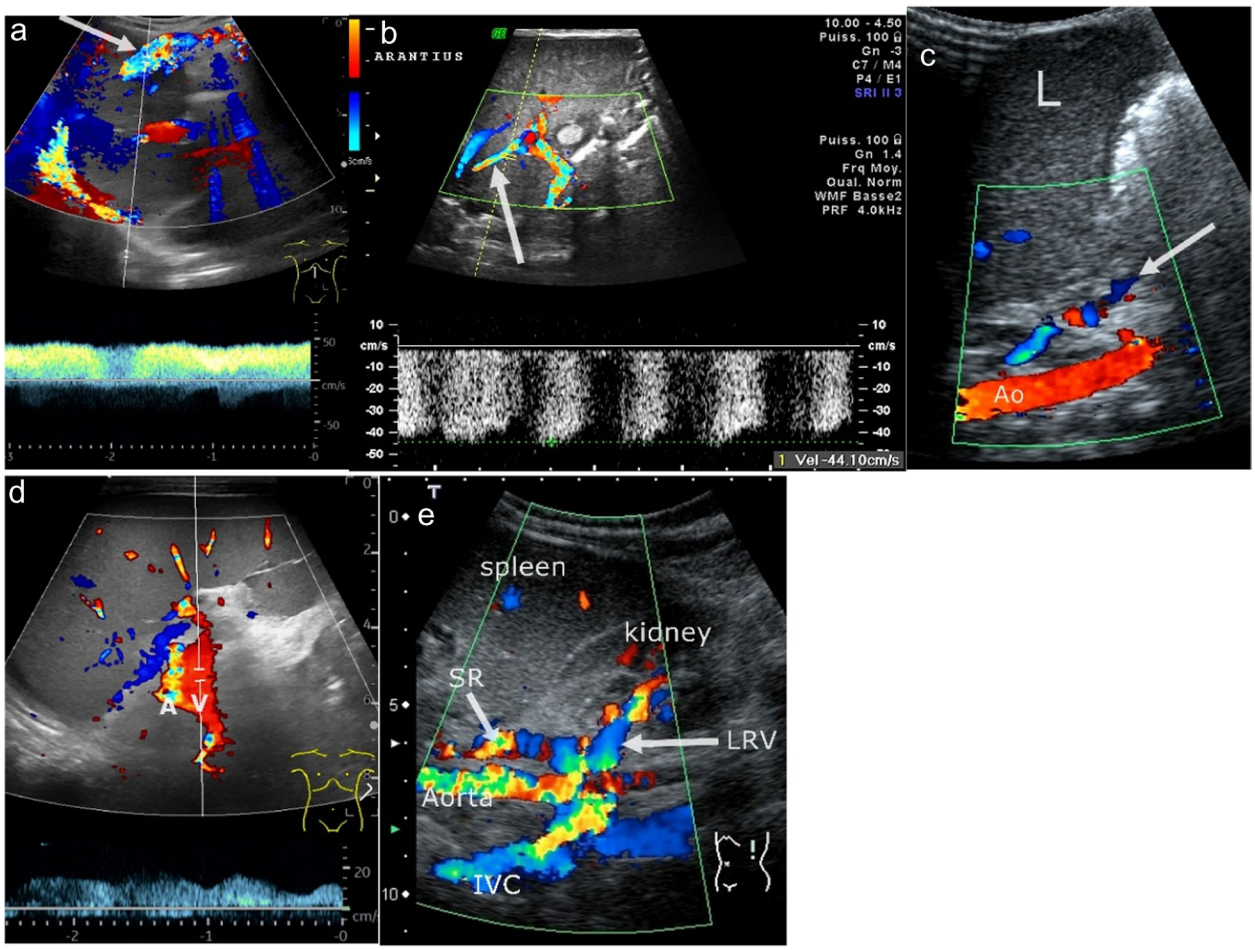

Fig. 2 Detection of flow alterations and portosystemic collaterals using colour Doppler ultrasound in a 16-month-old boy with portal hypertension due to biliary atresia (**a**) and a 2-month-old infant with neonatal cirrhosis (**b**, **c**, **d**, **e**). **a** Sagittal view of the left liver shows hepatofugal flow in para-umbilical veins within the ligamentum teres (*arrow*). **b** Colour Doppler ultrasound shows a persistent patent ductus venosus of normal calibre (*arrow*), which should be differentiated from malformative ductus venosus, which is a type of congenital portosystemic shunt. **c** Sagittal view of the left liver (*L*) and the aorta (*Ao*), shows hepatofugal flow in the left gastric vein (*arrow*). **d** Reversed (hepatofugal) flow in the splenic vein (*v*), indicating severe portal hypertension. **e** Coronal view through the splenic acoustic window shows the spleno-renal collateral (*SR*, *arrow*), running parallel to the aorta on its left side and draining into the left renal vein (*LRV*), which drains into the inferior vena cava (*IVC*)

evaluation of the portal and splenic veins is essential when splenomegaly is detected without an alternative explanation. The presence of accessory spleens or polysplenia may also support the diagnosis of underlying portal hypertension.

Ultrasound elastography

Liver and spleen stiffness measurements (LSM and SSM) are well-established surrogate markers of chronic liver disease and portal hypertension in adults [14]. Their use is feasible in paediatric clinical practice with ultrasound or magnetic resonance elastography. US elastography is more widely available and easier to implement in routine clinical settings, although measured values may vary with the technique used, the manufacturer, and the type of probe used.

LSM primarily reflects liver fibrosis and congestion but may be influenced by confounding factors such as inflammation and cholestasis [15, 16]. In contrast, SSM mainly reflects portal venous congestion and structural changes associated with portal hypertension, although its confounding factors remain less well defined. Increasing paediatric evidence suggests that spleen stiffness measurement (SSM), performed using transient elastography (TE) or shear-wave elastography (SWE), is an effective non-invasive approach for evaluating portal hypertension and identifying oesophageal varices (EV) in children. Several studies have shown that SSM consistently outperforms LSM in predicting clinically significant varices, with diagnostic thresholds typically ranging from 28 kPa to 40 kPa and diagnostic accuracy characterised by area under the receiver operating

characteristic curve (AUROC) values above 0.85 to 0.90 [17–21].

SSM has also been shown to decrease significantly following portosystemic shunt creation in children with extrahepatic portal vein hypertension, supporting its value for monitoring treatment response [22]. By comparison, LSM shows a weaker correlation with variceal status [23]. Despite heterogeneity in study design and patient populations, current evidence supports SSM as a promising biomarker for screening and risk stratification of portal hypertension and varices in paediatric patients [24].

In adult patients, the Baveno VII guidelines recommend the use of LSM and SSM for the non-invasive detection of clinically significant portal hypertension [14]. Although equivalent paediatric recommendations are not yet available, emerging data suggest that elastography provides good diagnostic performance for detecting portal hypertension and assessing its severity in children [19–21].

Combining LSM and SSM appears to improve diagnostic performance for portal hypertension and may help differentiate its underlying mechanisms, monitor disease progression, and assess treatment response [15, 25]. In particular, the SSM-to-LSM ratio has been proposed as a parameter to distinguish presinusoidal from sinusoidal portal hypertension, although its diagnostic performance remains moderate [25].

Paediatric reference ranges remain limited due to variability between platforms and vendors across elastography techniques. Although methodological differences persist, quantitative elastography of the liver and spleen provides valuable information when available, serving as a non-invasive assessment tool. Long-term monitoring of paediatric portal hypertension is enhanced by longitudinal elastography follow-up, which offers valuable insights into the progression of individual portal hypertension. For reliable serial assessment, measurements should be performed using the same ultrasound system and transducer whenever possible.

Contrast-enhanced ultrasound

Although ultrasound contrast agents are not formally approved for intravascular use in children in many countries, several studies have shown that their use can significantly improve the visualisation of vascular anatomy in the upper abdomen [26]. In selected cases, contrast-enhanced ultrasound can help delineate the extrahepatic portal vein and other vascular structures when conventional colour Doppler ultrasound is inconclusive. However, it is not yet considered a routine test for evaluating paediatric portal hypertension.

Contrast-enhanced computed tomography

For vascular mapping in the context of portal hypertension, CE-CT in the portal phase offers excellent spatial resolution and detailed vascular delineation. However, due to exposure to ionising radiation, its use in children should be limited to specific clinical indications, such as preoperative vascular mapping or surgical planning [27]. Multiphasic CE-CT protocols should be reserved for highly selected cases.

With the introduction of dose-optimised, child-specific low-dose protocols and the development of photon-counting CT scanners, the role of CE-CT in paediatric imaging might expand again, balancing diagnostic accuracy with radiation safety.

Magnetic resonance imaging

Magnetic resonance imaging (MRI) is a valuable complementary modality when ultrasound findings are inconclusive or insufficient [18]. Multiphasic dynamic contrast-enhanced MRI (DCE-MRI) enables separate evaluation of arterial, portal venous, and systemic venous phases, thereby improving the visualisation of portosystemic collaterals and varices. MRI also provides accurate evaluation of liver and spleen size and enables detailed characterisation of hepatic parenchymal changes and focal lesions [6].

Magnetic resonance cholangiopancreatography (MRCP) provides additional detail on the biliary tree that may help diagnose portal biliopathy in the context of extrahepatic portal vein obstruction or biliary involvement in other conditions. Diffusion-weighted imaging (DWI) aids in non-contrast assessment of hepatic parenchymal health. The use of hepatocyte-specific contrast agents, although off-label in children, can improve the characterisation of atypical focal lesions, detect parenchymal changes, and provide indirect information about hepatocellular function [6, 28].

Magnetic resonance elastography (MRE) enables non-invasive measurement of liver and spleen stiffness. Although current MRE techniques assume linear elastic tissue properties, ongoing research seeks to integrate viscoelastic modelling to enhance accuracy and specificity [16]. MRE offers high reproducibility across imaging platforms but remains technically more demanding, less widely available, and more challenging to perform in paediatric patients compared with ultrasound elastography.

Management of paediatric portal hypertension: radiological and surgical treatments

Initial management of portal hypertension in children primarily relies on medical and endoscopic therapies aimed at controlling complications, particularly gastrointestinal bleeding [29, 30]. When these conservative measures fail or when portal hypertension remains severe, radiological and surgical interventions become necessary. The choice

of treatment depends on the underlying aetiology, portal venous anatomy, hepatic function, severity of portal hypertension–related complications, and the overall clinical condition of the patient. Management should be guided by multidisciplinary discussion in specialised centres, as local expertise plays a critical role in optimising outcomes. A clear understanding of both radiological and surgical treatment options is therefore essential for appropriate patient selection and management.

Principles of surgical and radiological treatment

The primary objective of surgical and radiological interventions in portal hypertension is to reduce portal venous pressure in order to prevent or treat complications such as variceal bleeding, refractory ascites, and hypersplenism.

In the absence of intrahepatic obstruction, particularly in prehepatic or posthepatic portal hypertension, the therapeutic goal is to restore or improve hepatopetal portal venous flow whilst, whenever possible, reducing portal pressure. Restoration of physiological intrahepatic portal perfusion may be achieved either by endovascular recanalisation of obstructed or stenotic vessels—such as hepatic vein recanalisation in Budd–Chiari syndrome or portal vein recanalisation in extrahepatic portal vein obstruction—or surgically through a Meso-Rex bypass, which connects the extrahepatic portal venous system to the umbilical segment of the left portal vein within the Rex recess [31–33]. These approaches are considered physiological because they restore hepatopetal portal perfusion and preserve the normal first-pass hepatic circulation of mesenteric and splenic venous blood, thereby supporting normal liver development and metabolic function (Fig. 3).

When the obstruction is intrahepatic—due to cirrhosis or presinusoidal or postsinusoidal disorders—or when physiological treatments are unsuccessful in prehepatic or posthepatic portal hypertension, restoration of normal intrahepatic portal flow is not feasible. In such cases, decompression of the portal venous system via a portosystemic shunt is the only therapeutic option before liver transplantation. A portosystemic shunt may be created either by interventional radiology, through the placement of a transjugular intrahepatic portosystemic shunt (TIPS), or surgically, through the creation of portosystemic shunts that do not preserve physiological portal circulation (Fig. 3) [34–36]. However, liver failure or complications of pre-existing acquired portosystemic shunts, such as porto-pulmonary hypertension, hepatopulmonary syndrome, hepatorenal syndrome, or portosystemic encephalopathy, represent contraindications, as these conditions may worsen following further portal diversion. In such situations, liver transplantation remains the definitive treatment option.

Other interventional radiology procedures may be performed, either in a palliative context or as a bridge to liver transplantation, particularly to manage intractable gastrointestinal bleeding or severe hypersplenism. In cases of bleeding from ectopic or gastric varices that are inaccessible via endoscopy, direct embolisation, through transhepatic or transsplenic access, or via a gastrorenal shunt using balloon-occluded retrograde transvenous obliteration (BRTO) or coil/plug-assisted retrograde transvenous obliteration (CARTO/PARTO), may control bleeding, although most published data derive from adult populations [37]. Paediatric data remain limited. In patients with symptomatic hypersplenism and severe cytopenias, partial splenic artery embolisation may be considered, though its efficacy can be variable and often temporary; the procedure may need to be repeated eventually.

Preoperative evaluation

Preoperative imaging is crucial and should be tailored to the aetiology of portal hypertension and the planned intervention.

Regardless of the cause, precise delineation of portal venous anatomy is essential for selecting the appropriate treatment. The role of non-invasive imaging techniques has been described above.

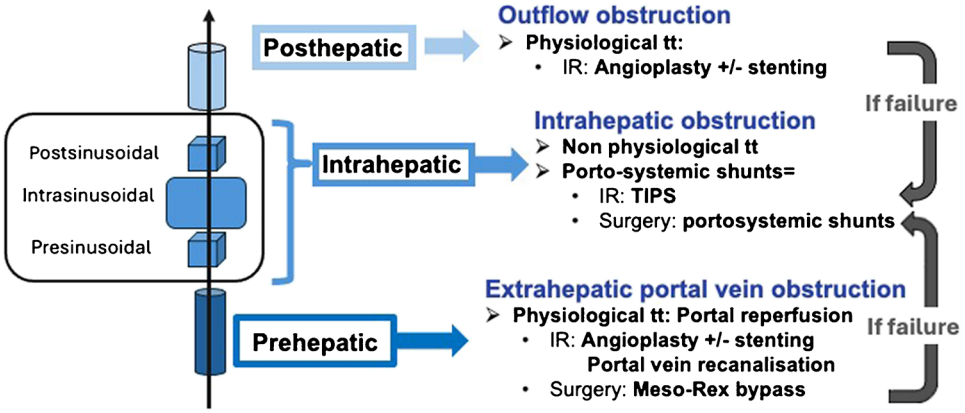

Fig. 3 Schematic overview of the anatomical levels of portal hypertension in children and the corresponding therapeutic approaches. The diagram illustrates the anatomical classification of portal hypertension and the corresponding treatment strategies. *IR*, interventional radiology; *tt*, treatment; *TIPS*, transjugular intrahepatic portosystemic shunt

Invasive imaging methods remain important in the assessment of extrahepatic portal vein obstruction. In this context, evaluation of the patency and calibre of the Rex recess of the left portal vein is essential to determine the feasibility of a Meso-Rex bypass (MRB) [32, 38]. When intrahepatic portal branches cannot be adequately visualised using non-invasive imaging, particularly in cases of extrahepatic portal vein obstruction related to umbilical vein catheterisation, wedged hepatic venous portal venography (WHVP) is considered the gold standard for evaluating the Rex recess of the left portal vein and intrahepatic portal venous communications. This technique also allows indirect measurement of intrahepatic portal pressure, which is typically low in patients with extrahepatic portal vein obstruction. Some centres consider that unexpectedly elevated intrahepatic pressure may reduce the likelihood of successful surgical portal reperfusion and should prompt consideration of alternative strategies. WHVP is performed via a jugular approach, with sequential hepatic venography to obtain wedged venograms. This technique may demonstrate portal veins that are not visible on ultrasound or CT. The Baveno paediatric group recommends a classification of intrahepatic portal vein patterns, first proposed in 2014, to select candidates for MRB [39]. A similar classification has been used in adult patients undergoing portal vein recanalisation [40] (Fig. 4).

In extrahepatic portal vein obstruction, conventional imaging can also identify anatomical features that favour portal vein recanalisation (PVR), such as the presence of a remnant main portal vein (MPV), a straight, although sometimes narrowed vessel within the cavernoma connecting the splenomesenteric confluence to the portal bifurcation, as well as preserved straight native intrahepatic portal branches, in contrast to tortuous cavernomatous vessels (Fig. 1).

With advances in cross-sectional imaging, diagnostic direct portal venography and delayed venous phases of splenic or mesenteric arteriography are no longer recommended for routine diagnostic purposes.

A liver biopsy may be required to exclude fibrosis or porto-sinusoidal vascular disease before portal vein reperfusion. However, differentiating between portal deprivation caused by extrahepatic portal vein obstruction and porto-sinusoidal vascular disease may be challenging histologically. Liver biopsy is typically performed percutaneously under ultrasound guidance using a 16–18-G needle. In cases of severe thrombocytopenia, coagulopathy, or ascites, a transjugular approach should be considered.

Physiological management of extrahepatic portal vein obstruction: portal vein recanalisation versus Meso-Rex bypass

The feasibility of a Meso-Rex bypass (MRB) depends on the patency of the Rex recess of the left portal vein and, by extension, the integrity of the intrahepatic portal venous system, as well as the availability of a suitable extrahepatic vein for anastomosis. PVR may be attempted in most patients, particularly those ineligible for MRB or for whom MRB has failed. PVR offers a potential physiological option; however, paediatric data remain limited. Reported success rates vary and generally decrease with increasing extent of thrombosis involving the intrahepatic or extrahepatic portal venous system [33]. Given its lower invasiveness and promising outcomes, PVR may emerge as

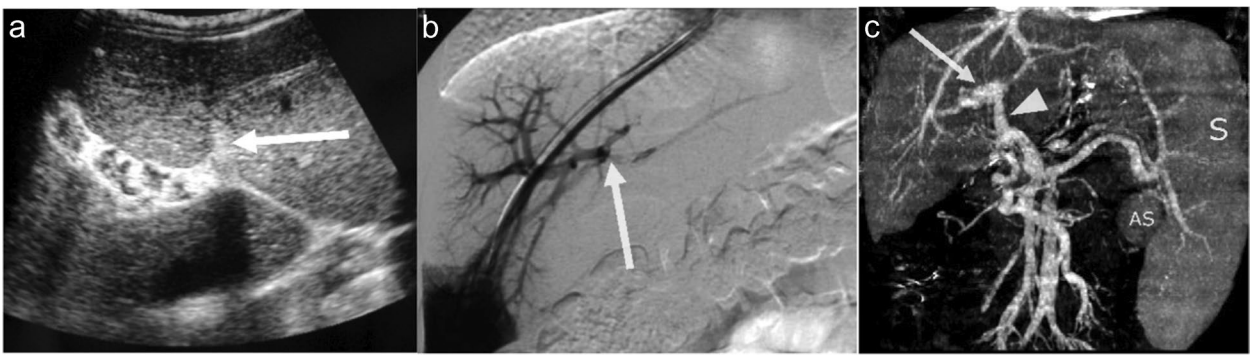

Fig. 4 Pre- and postoperative evaluation in a 3-year-old boy with extrahepatic portal vein obstruction, cavernous transformation, and severe portal hypertension. **a** Ultrasound, epigastric view at the level of the portal bifurcation shows absence of visible intrahepatic portal veins, which are replaced by cavernous vessels. The lumen of the Rex recess of the left portal vein is not visualised (*arrow*). **b** Antero-posterior view of the wedge hepatic venous portal venography via the right hepatic vein demonstrates opacification of the right and left portal veins and the Rex recess of the left portal vein (*arrow*). **c** After Meso-Rex bypass, contrast-enhanced CT coronal image in the portal venous phase shows restoration of portal flow. The *arrow* indicates the patent Rex recess of the left portal vein, and the *arrowhead* indicates the venous graft connecting the cavernomatous vein to the Rex recess of the left portal vein. Note splenomegaly (*S*) and accessory spleen (*AS*), consistent with chronic portal hypertension

a first-line treatment in experienced centres, including for patients who are also eligible for MRB [33]. Access routes for PVR include transhepatic and transsplenic approaches. Transmesenteric access may also be achieved via mini-laparotomy or direct percutaneous puncture. Whilst balloon angioplasty alone may be sufficient in selected cases, most patients require stent placement to ensure durable portal vein patency (Fig. 5).

Non-physiological management: surgical vs transjugular intrahepatic portosystemic shunt

Portosystemic shunting is indicated when physiological portal decompression cannot be achieved, due to intrahepatic block, unfavourable anatomy, or failure of MRB or PVR. Surgical portosystemic shunts may be considered for various causes of portal hypertension, provided contraindications are carefully evaluated. These can be constructed using autologous (e.g. jugular vein) or prosthetic grafts.

Major types include:

- mesocaval shunt: connecting the superior mesenteric vein (SMV) to the IVC
- portocaval shunt: connecting the portal vein to the IVC
- distal spleno-renal (Warren) shunt: diverting splenic venous flow to the left renal vein whilst preserving SMV hepatopetal flow

These portosystemic shunts are effective in reducing portal hypertension (Fig. 6).

Transjugular intrahepatic portosystemic shunt (TIPS) placement offers a less invasive interventional radiology alternative by creating a transhepatic communication between a hepatic vein and a portal vein branch via a transjugular approach (Fig. 7). Initially developed for intrahepatic or posthepatic portal hypertension, TIPS has more recently been used in selected prehepatic cases, particularly when portal vein recanalisation alone is insufficient. However, technical failure rates remain relatively high in children [41, 42]. In paediatric patients, TIPS is most commonly used as a bridge to transplantation or when surgical options are not feasible. Despite these limitations, satisfactory long-term patency and clinical effectiveness have been reported. The small vascular calibre in infants and young children makes the procedure technically challenging and requires careful stent selection. Two recent meta-analyses have shown that TIPS is effective in managing refractory gastrointestinal bleeding and ascites; however, its efficacy in treating hypersplenism, particularly thrombocytopenia, appears more limited [41, 42].

Management and procedure-related complications

Interventional portal venous procedures in children are performed under general anaesthesia. Currently, no standardised anticoagulation protocol exists for either surgical or interventional procedures. Postprocedural anticoagulation is generally required following transsplenic or transhepatic access, although careful monitoring is essential

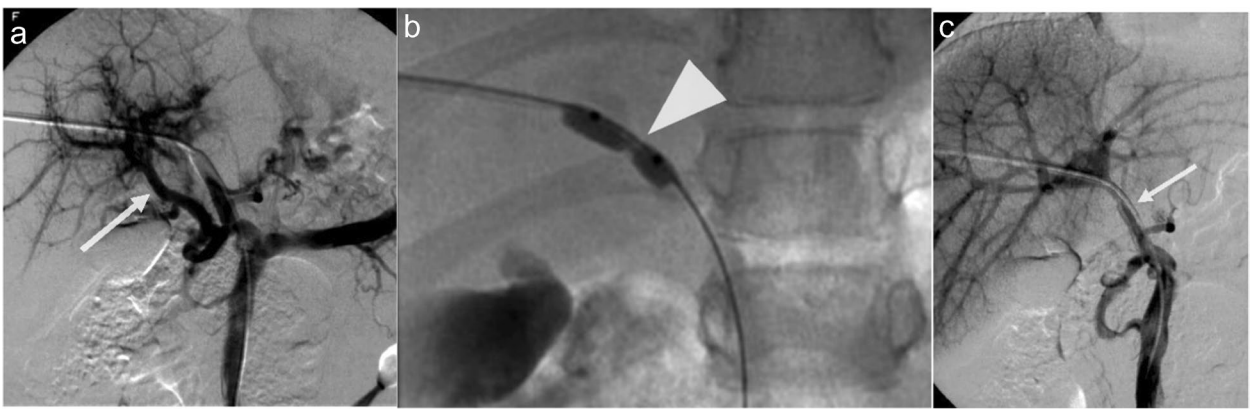

Fig. 5 Portal vein recanalisation in a 9-year-old girl presenting with gastrointestinal bleeding, caused by extrahepatic portal vein obstruction with cavernous transformation. **a** Antero-posterior view of transhepatic portal venography performed after navigating through a very tight stenosis at the distal segment of the main portal vein, which caused obstruction and severe portal hypertension. A large, tortuous cavernous vein feeding the intrahepatic portal veins is visible (*arrow*), along with retrograde opacification of the splenic vein and the left gastric vein, consistent with severe portal hypertension. **b** Balloon angioplasty was performed and shows a characteristic waist (*arrowhead*) that was relieved after complete balloon inflation. **c** Post-procedure venography from the superior mesenteric vein shows restoration of portal vein patency (*arrow*), opacification of intrahepatic portal branches, and disappearance of cavernous transformation, collateral flow, and hepatofugal flow

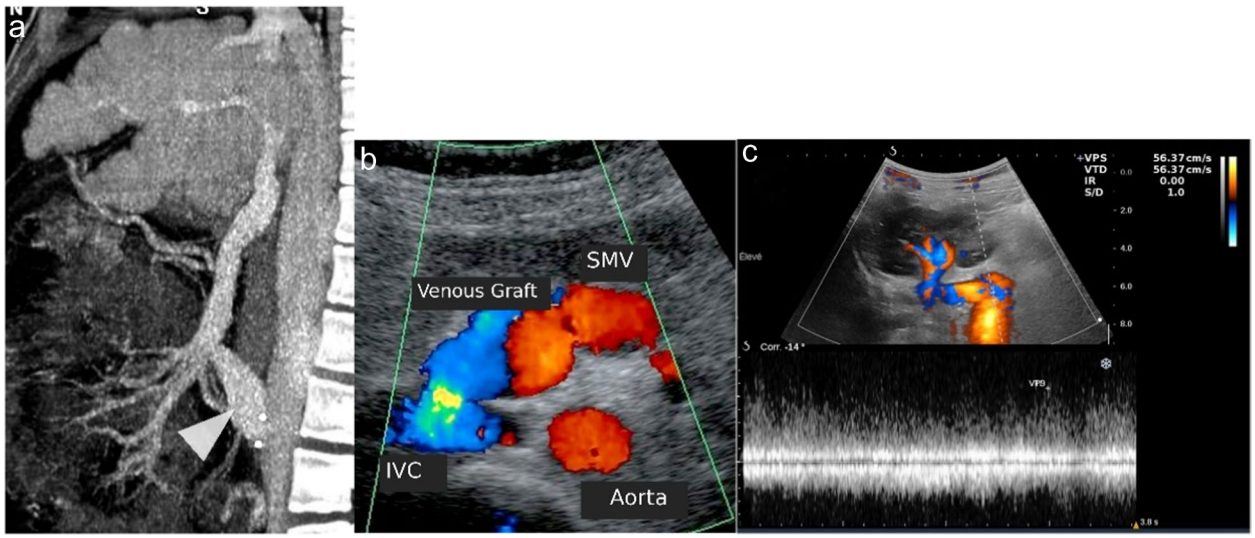

Fig. 6 Surgical mesocaval shunt in a 13-year-old girl with severe portal hypertension secondary to cystic fibrosis-related liver disease. **a** Sagittal-reformatted maximum intensity projection of a contrast-enhanced ct in the portal phase shows the autologous venous graft (internal jugular vein) connecting superior mesenteric vein (*SMV*) and the inferior vena cava (*IVC* and *arrowhead*). Surgical metallic clips are visible at the IVC anastomosis. **b** Colour Doppler ultrasound with an anterior axial view, the venous graft between the SMV and the IVC is fully visualised. **c** Colour Doppler and pulsed-wave Doppler, coronal view through the right kidney and IVC shows the mesocaval graft with hepatofugal flow directed towards the IVC below the level of the right renal vein

because of the associated bleeding risk. Anticoagulation regimens should be tailored to the individual patient and institutional protocols, both intraoperatively and postoperatively.

Follow-up after surgical or radiological treatments for portal hypertension

Potential complications

Although complications following surgical and interventional radiological procedures for portal hypertension are relatively uncommon, their true incidence is likely underestimated in the literature. These complications can be broadly classified as general procedure-related complications and disease-specific complications.

Among surgical complications, postoperative haemorrhage and surgical site infections are the most frequently reported. In interventional radiology, complications include adverse reactions to iodinated contrast agents, ranging from allergic reactions to contrast-induced nephropathy, as well as haemorrhagic complications, which may occur but are not always clinically significant. Particular attention should be paid to radiation exposure [27].

Clinically significant complications include thrombosis and stenosis. Thrombosis typically occurs early after both surgical and interventional procedures, potentially resulting in an acute clinical presentation. Conversely, stenosis—more common in both surgical and interventional settings—typically develops weeks to months after treatment and may lead to recurrence of portal hypertension and even portal thrombosis.

A specific consideration in paediatric patients undergoing TIPS is that somatic growth may result in inadequate stent length to maintain the hepatic vein–inferior vena cava connection. In such cases, elective stent extension may be required to prevent outflow obstruction. Early stenosis may also occur, most often due to technical factors such as stent kinking, malposition, or neointimal hyperplasia, particularly with uncovered stents.

Surgical and radiological procedures may also result in iatrogenic biliary injury, especially in patients with portal biliopathy. Clinical manifestations include haemobilia, biliary laceration with secondary infection and sepsis, or extrinsic biliary compression resulting in segmental cholestasis or obstructive jaundice.

In patients with portal hypertension refractory to physiological treatments, additional therapeutic options include splenic artery embolisation and splenectomy. Post-embolisation syndrome (fever, left upper quadrant pain, leucocytosis) is common but usually self-limited. However, more severe complications, including pleural effusion, portal vein thrombosis, or splenic abscess, require close clinical and imaging follow-up. Splenectomy and partial splenectomy are also associated with potentially significant complications and should be considered cautiously in specialised centres.

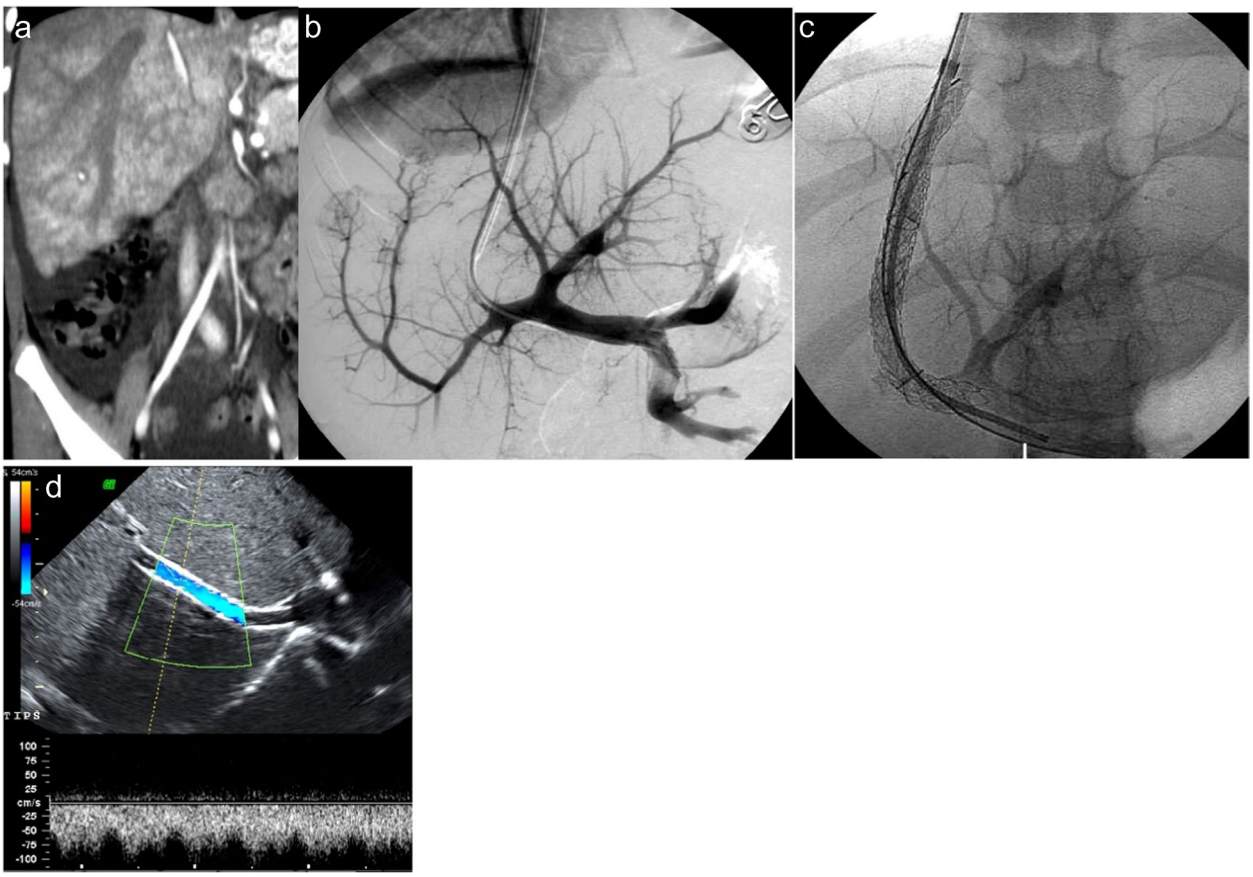

Fig. 7 Budd–Chiari syndrome in a 7-year-old girl presenting with refractory ascites secondary to portal hypertension. **a** Coronal-reformatted maximum intensity projection contrast-enhanced CT in the portal venous phase shows heterogeneous hepatomegaly with absence of enhancement of the enlarged right hepatic vein and partial enhancement with distal narrowing of the middle hepatic vein. After unsuccessful thrombectomy and angioplasty of the hepatic veins, a transjugular intrahepatic portosystemic shunt (TIPS) was created. **b** Antero-posterior view of the portal venography, following puncture of the right portal branch from the middle hepatic vein, demonstrates normal opacification of the intrahepatic portal veins, with retrograde opacification of the main portal vein, superior mesenteric vein, and the splenic vein. **c** A TIPS was placed between the right portal vein and the middle hepatic vein to bypass the hepatic venous obstruction, allowing portal blood to drain directly into the systemic circulation and relieving portal hypertension. **d** Colour Doppler ultrasound image shows normal hepatofugal flow within the TIPS

Role of non-invasive imaging

Long-term non-invasive imaging follow-up is essential for the early detection of complications or treatment failure after interventions for portal hypertension. Imaging assessment includes techniques that directly evaluate shunt patency and haemodynamics—such as colour Doppler ultrasound, pulsed-wave Doppler, CTA, MRA, and 4D-flow MRI—as well as elastography, which provides indirect assessment of portal venous congestion. Accurate interpretation requires detailed knowledge of the surgical or interventional procedure performed and the modified vascular anatomy.

Colour and pulsed-wave Doppler remain the primary imaging modalities, enabling real-time assessment of intrahepatic and extrahepatic haemodynamics, shunt patency, and intrastent flow. These techniques can be performed at the bedside, including perioperatively and in the immediate postoperative period. However, limitations include a restricted acoustic window, particularly in the early postoperative phase, which may hinder visualisation of deep mesenteric or splenic anastomoses, and acoustic shadowing from covered stents (e.g. TIPS stents, usually visible after ≥72 h post-placement), which may also limit visualisation. For example, after mesocaval shunt procedures, the surgical anastomosis may not be visible on ultrasound from an anterior view in the early postoperative period; however, a lateroposterior approach can provide an adequate acoustic window, as shown in Fig. 6.

Ultrasonographic monitoring of surgical bypasses and shunts requires careful evaluation of vascular morphology and accurate interpretation of flow direction. Specifically, large portosystemic shunts typically result in flow reversal and progressive reduction in calibre of intrahepatic portal branches. In contrast, selective shunts may preserve hepatopetal flow. Additionally, in Meso-Rex

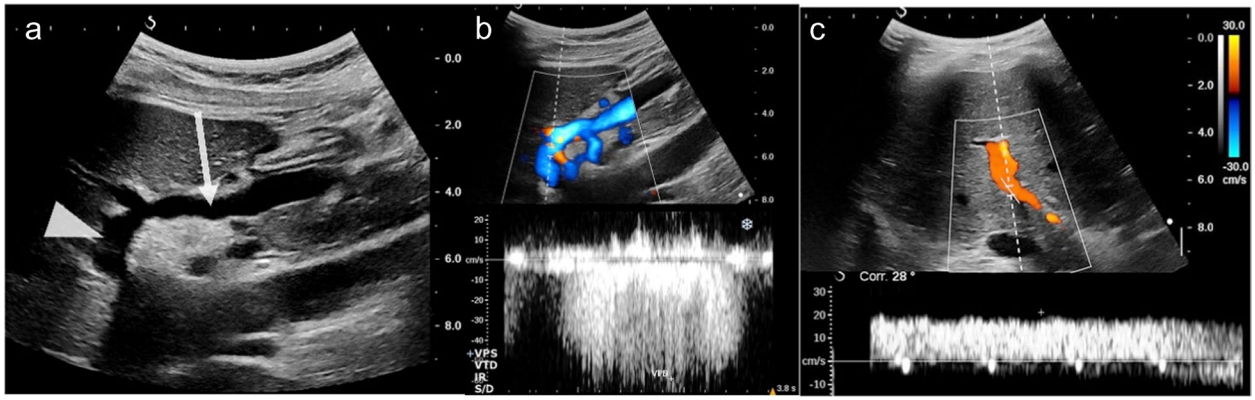

Fig. 8 Meso-Rex bypass morphology and flow on colour Doppler ultrasound in a 9-year-old boy. **a** Anterior sagittal view shows the venous graft (*arrow*) surgically anastomosed to the Rex recess of the left portal vein (*arrowhead*) at the level of the ligamentum teres. **b** Colour and pulsed-wave Doppler demonstrate reversed portal flow in the proximal portion of the left portal vein, and **c** right intercostal view shows normally directed hepatopetal flow in the right portal vein

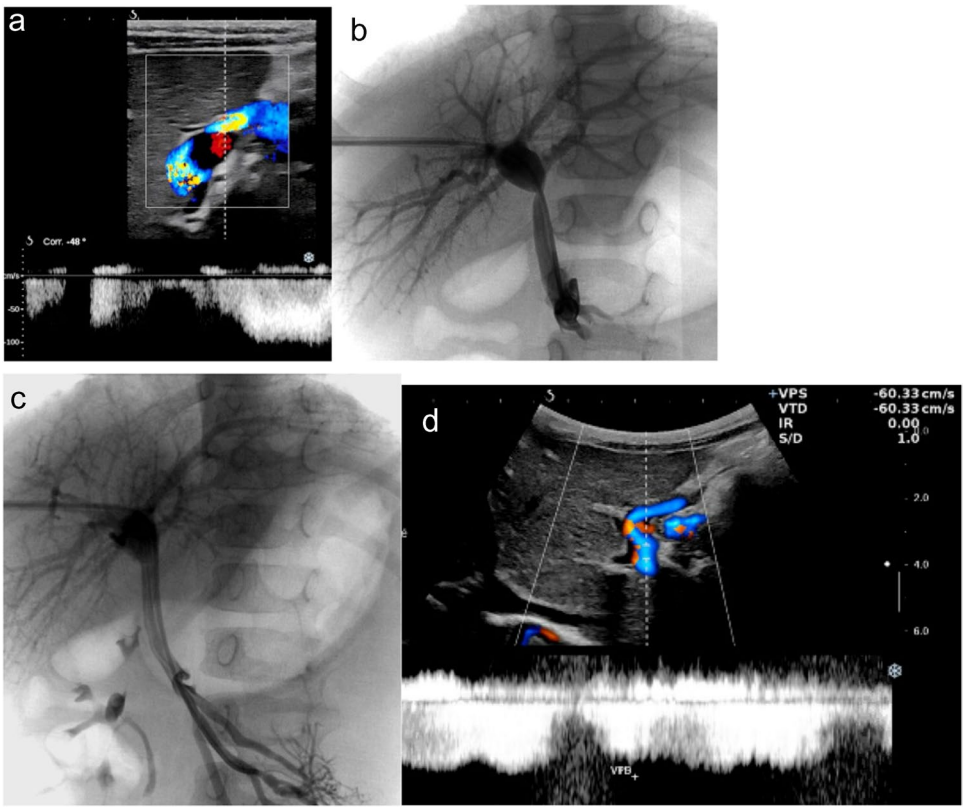

Fig. 9 Recurrent gastrointestinal bleeding after a Meso-Rex bypass surgery in a 2-year-old girl. **a** A routine colour Doppler ultrasound and pulsed-wave Doppler demonstrate severe stenosis of the anastomosis between the venous graft and the Rex recess of the left portal vein with elevated flow velocities (approximately 1 m/s). **b**, **c** Antero-posterior views during transhepatic portal venography show a significant stricture at the site of the Meso-Rex bypass anastomosis (**b**) and resolution of the stenosis following balloon angioplasty (**c**). **d** Post-angioplasty colour Doppler ultrasound and pulsed-wave Doppler shows normal flow within the venous graft, no residual stenosis, and expected reversed flow in the left portal vein

bypass (MRB), the venous graft connects the superior mesenteric vein to the Rex recess of the left portal vein, resulting in expected flow reversal in the left portal vein as shown in Figs. 8 and 9. Following successful portal vein revascularisation, intrahepatic portal branches often enlarge, with an associated increase in liver volume.

Assessment of flow velocities is essential in identifying dysfunction or stenosis. Because of the high variability between patients, absolute velocity thresholds are less reliable than relative changes. A focal threefold increase in velocity associated with downstream turbulence suggests significant stenosis, whereas low velocities (e.g. <15 cm/s) may indicate dysfunction (Fig. 10).

When ultrasound findings are inconclusive, contrast-enhanced ultrasound may provide additional diagnostic information, particularly in suspected thrombosis. Serial spleen stiffness measurements may also provide complementary information.

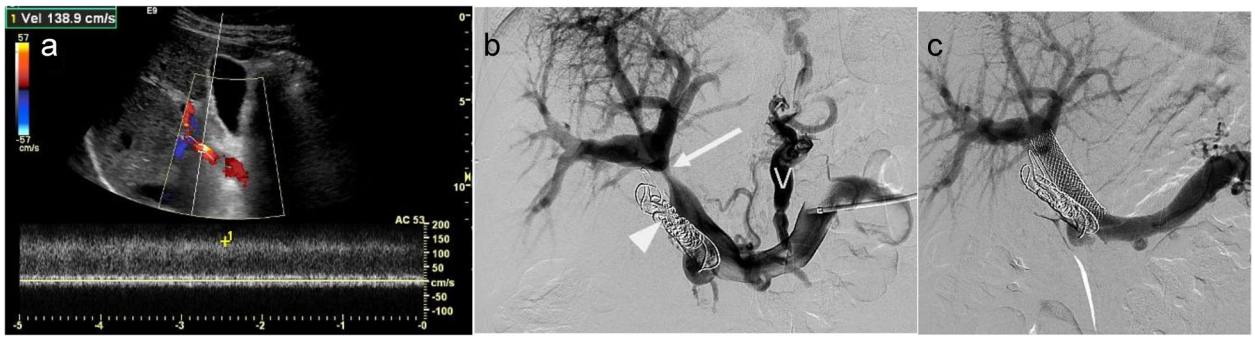

Fig. 10 Recurrence of portal vein stenosis after recanalisation in a 14-year-old boy following Meso-Rex failure. **a** Colour and pulsed-wave Doppler ultrasound on an intercostal view shows abnormally elevated flow velocity at the hepatic hilum, consistent with a haemodynamically significant stenosis of the main portal vein. **b** Antero-posterior transsplenic portal venography confirms severe stenosis of the main portal vein just proximal to the portal bifurcation (*arrow*), with retrograde opacification of gastric varices (*V*). Metallic coils previously deployed to occlude the cavernoma are also visible (*arrowhead*). **c** Antero-posterior transsplenic portal venography performed after angioplasty and stent placement demonstrates resolution of the stenosis and disappearance of retrograde opacification of the gastric varices

If abnormalities are suspected, cross-sectional imaging should be performed. CT is preferred in urgent situations, particularly when thrombosis is suspected, whereas MR venography is preferred in non-urgent cases to avoid radiation exposure. However, MRI availability may be limited and may require general anaesthesia in young children.

Management of procedure-related complications

When non-invasive imaging demonstrates dysfunction, surgical revision or, more commonly, an interventional radiology procedure may be performed to restore patency in a minimally invasive manner. In acute thrombosis, pharmacological thrombolysis or mechanical thrombectomy may be performed via selective catheterisation. For a portosystemic shunt, endovascular treatment is generally considered lower risk because it requires only venous access. In contrast, treatment of a Meso-Rex bypass or portal vein recanalisation typically requires transhepatic or transsplenic portal vein access, which is associated with an increased risk of haemorrhagic complications (Fig. 8). Thrombolysis may be achieved by transcatheter administration of thrombolytic agents or by mechanical thrombectomy using dedicated devices. However, mechanical thrombectomy requires significantly larger vascular access, increasing the risk of bleeding and procedural blood loss.

For chronic thrombosis or stenosis, the preferred treatment is percutaneous angioplasty, with or without stent placement (Figs. 8 and 9). Depending on the vascular anatomy, access may be achieved via transvenous, transhepatic, or transsplenic routes.

Portal hypertension is often associated with the development of secondary portosystemic collaterals, which can cause persistent flow diversion even after portal vein revascularisation. If significant flow steal leads to recurrent thrombosis, embolisation of competing collateral vessels may be required. Similarly, persistent cavernomatous transformation may require embolisation if it compromises portal vein patency.

In conclusion, paediatric portal hypertension is increasingly well understood owing to major advances in non-invasive imaging and refinements in interventional and surgical techniques. Ultrasound, particularly Doppler imaging and elastography, remains the cornerstone of diagnosis and follow-up, whilst CT and MRI provide essential complementary evaluation of vascular anatomy and hepatic parenchyma.

Physiological treatments, including portal vein recanalisation and Meso-Rex bypass, can restore hepatopetal portal flow in selected patients. When physiological restoration is not feasible, TIPS and surgical portosystemic shunts remain important therapeutic options.

Future developments—including the standardisation of paediatric elastography, advances in magnetic resonance elastography, and emerging techniques such as photon-counting CT and 4D-flow MRI—are expected to further improve diagnostic accuracy and haemodynamic assessment. Further prospective paediatric studies are required to validate imaging biomarkers and optimise treatment algorithms. In parallel, continued progress in paediatric interventional devices and imaging guidance will likely expand the role of minimally invasive therapies and enhance their safety.

Together, these advances will contribute to more individualised management strategies and improved long-term outcomes for children with portal hypertension.

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Data availability No datasets were generated or analysed during the current study.

Declarations

Conflicts of interest None

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