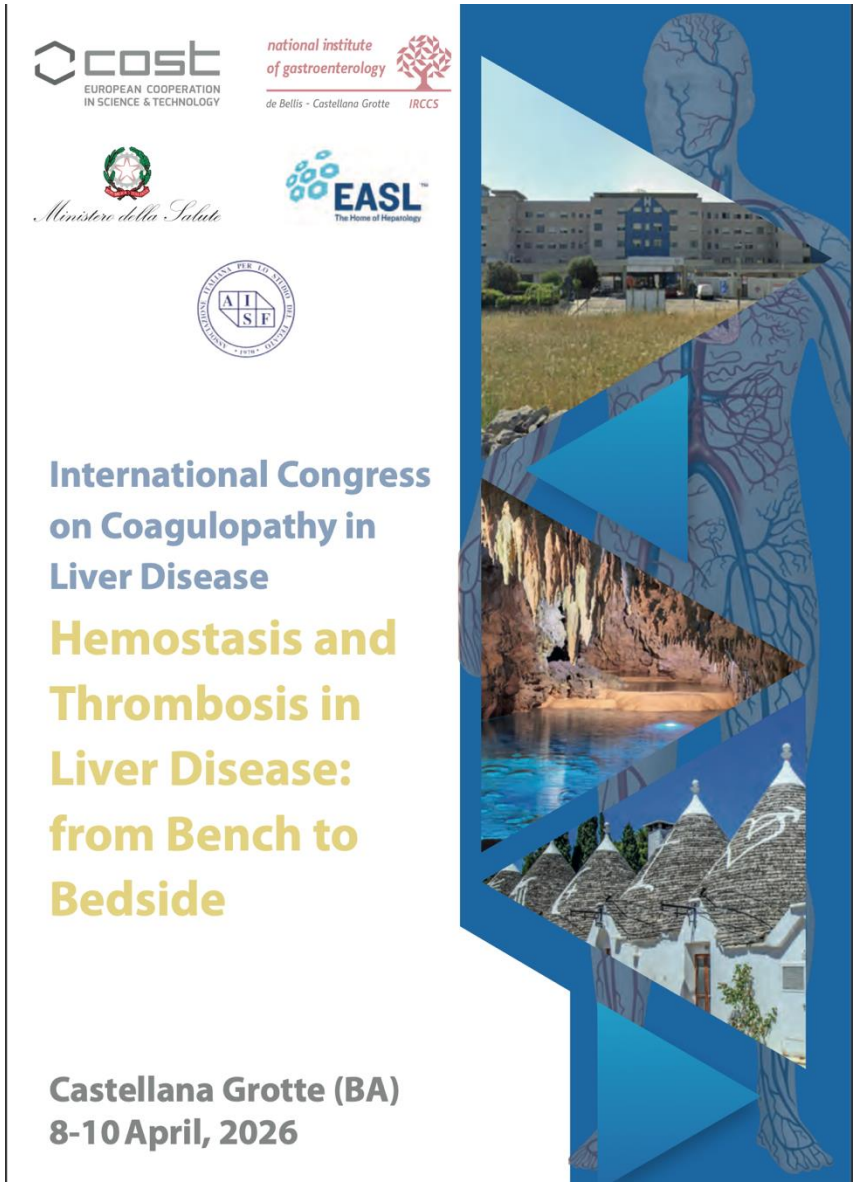
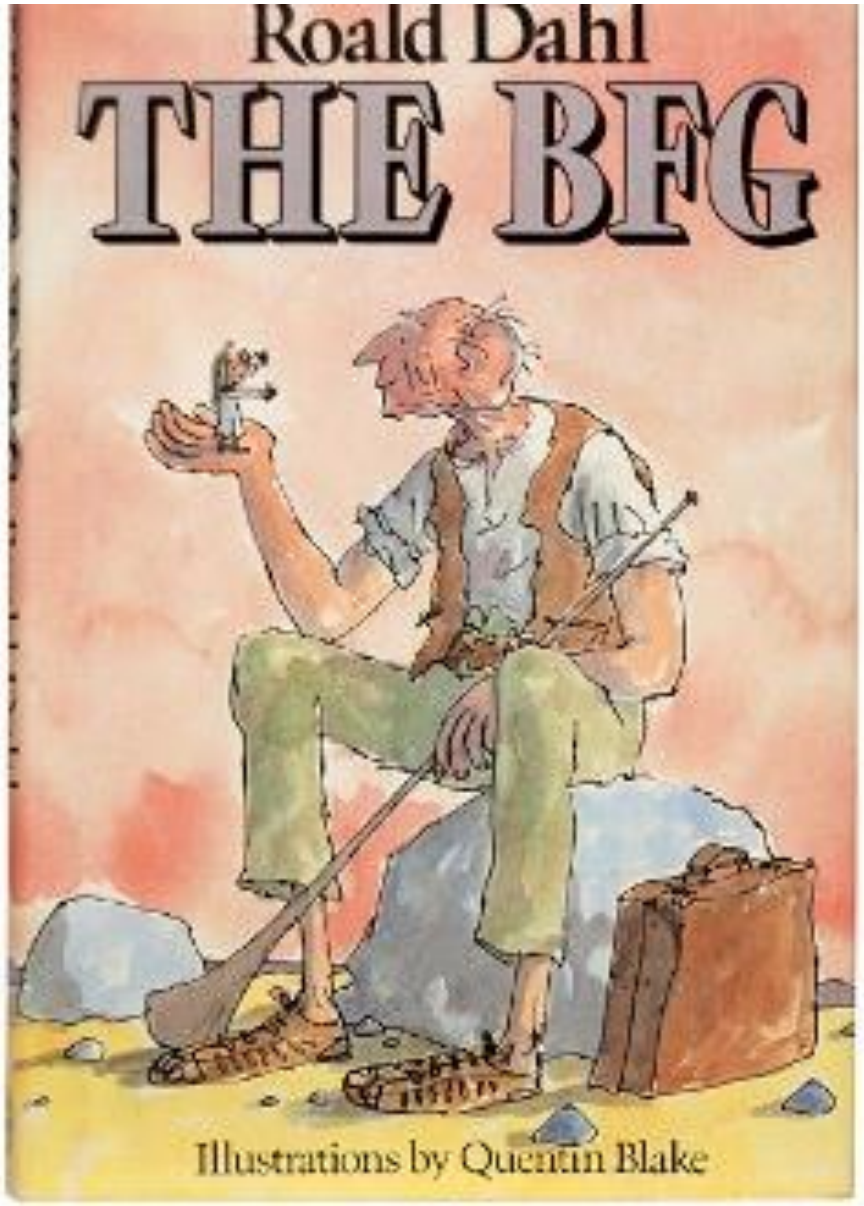


Coagulopathy and thrombosis in children with liver disease

Marianne Samyn

London, UK


Roald Dahl
THE BFG
Illustrations by Quentin Blake

Coagulopathy in Children With Liver Disease

*Patricia S. Kawada, †Aisha Bruce, †Patti Massicotte, †Mary Bauman, and *Jason Yap

‘Rebalanced homeostasis’

	Neonate	Childhood
FII, FVII, FIX, FX	↓ (50%)	↑ 6 mths
FVIII	↑ day 1 ↓ newborn	↑
VWF	=	
Protein C and S	↓	↑
INR	↑ but normal range	
Thrombin generation	↓ (50%)	↑ after 1 year
TEG	↑ 3-12 mths	

JPGN 2017;65: 603–607

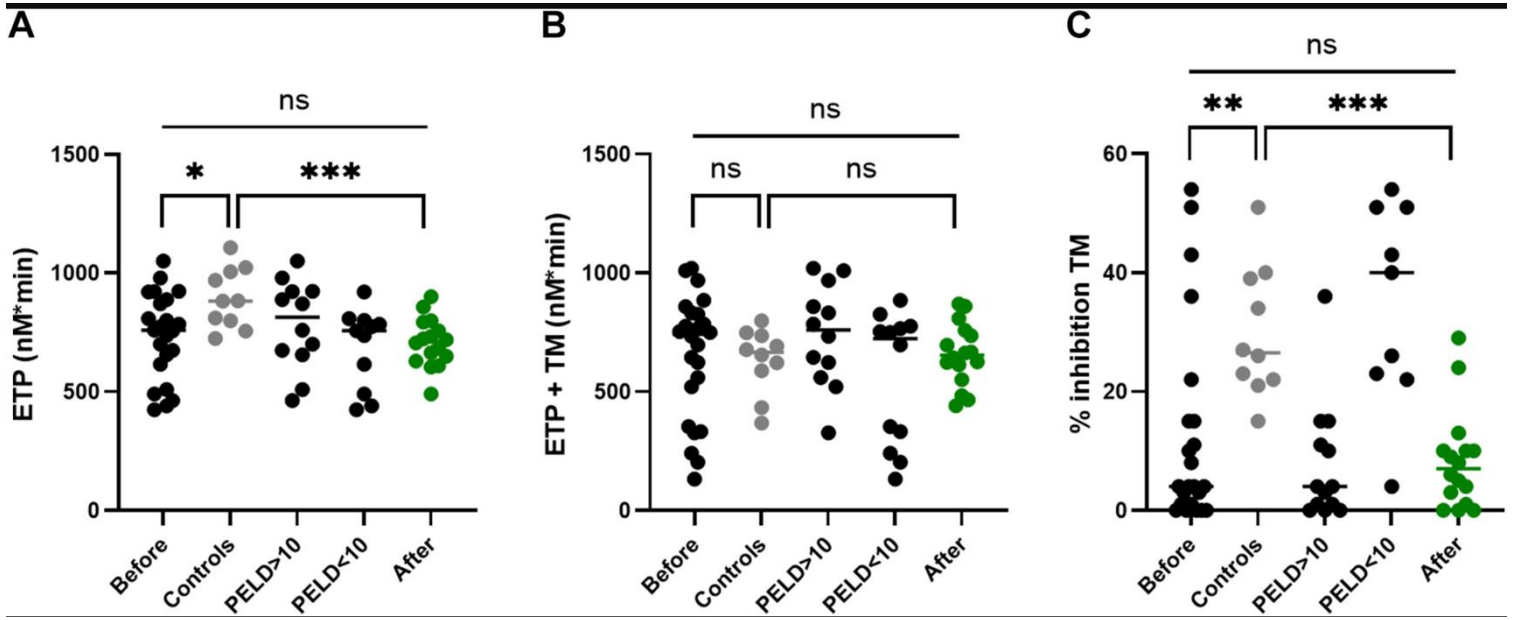
Primary hemostasis in children with cirrhosis prior to liver transplantation: key roles of liver disease severity, von Willebrand factor, and platelet count

N=27
HC n=15
post LT n=9

Haemostatic system generally well preserved
2 groups

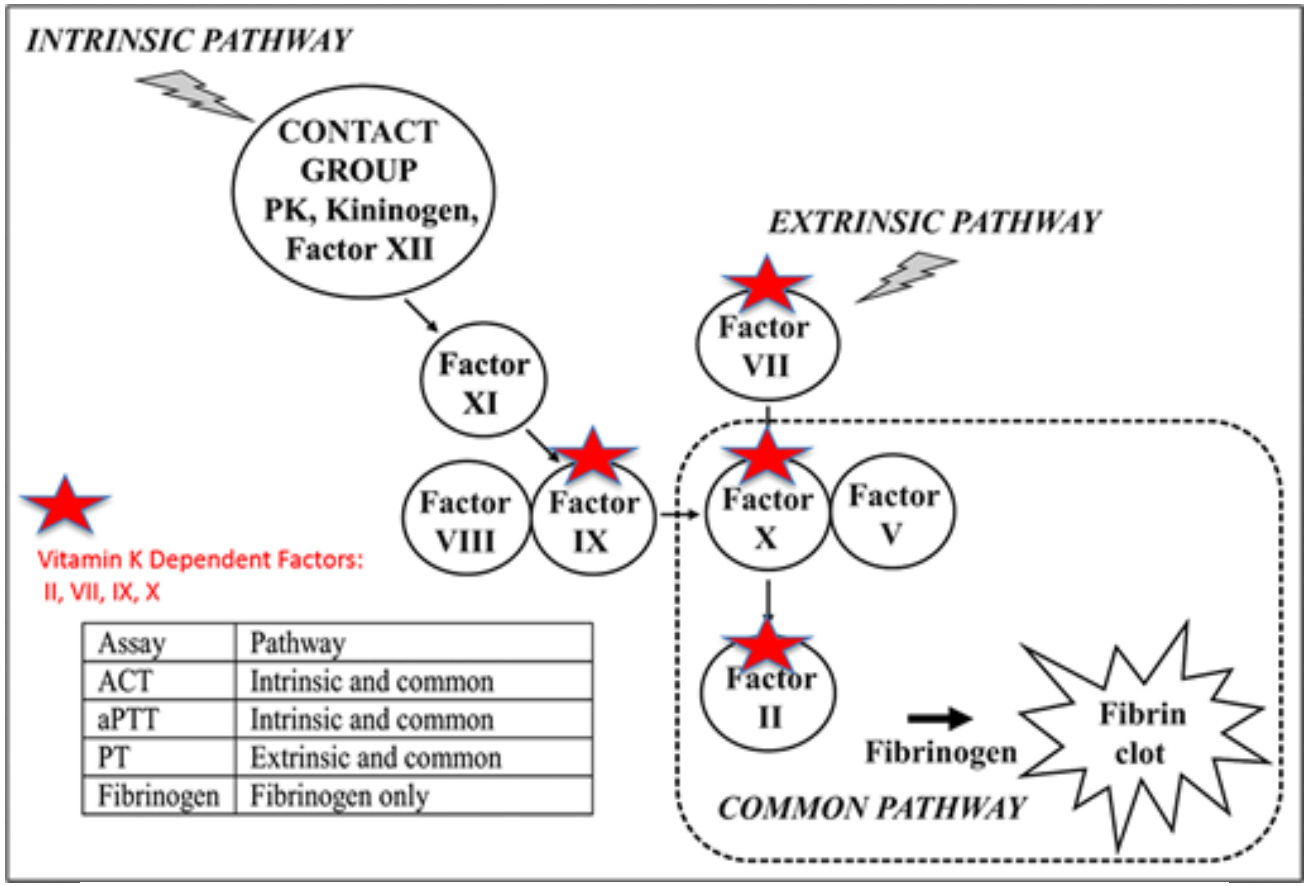
1. Preserved primary haemostasis with higher PELD and higher platelet count
2. Moderate haemostatic impairment with lower platelet count

Not fully reverted 3 months after liver transplantation



Persisting thrombomodulin resistance after transplant

van Dievoet et al. J Thromb Haemost. 2025;23:2297–2313. Res Pract Thromb Haemost. 2025;9:e102709



Assay	Pathway
ACT	Intrinsic and common
aPTT	Intrinsic and common
PT	Extrinsic and common
Fibrinogen	Fibrinogen only

Mnemonic for Vitamin K Dependent Clotting Factors

"Two plus seven is nine NOT ten!"

2 7 9 10

Vitamin K deficiency bleeding

Mortality 14-50%

- Early onset (24hrs):
  - anticonvulsants, barbiturates
  - anti-TB
- 2 weeks - 6 months
  - Breastfeeding
  - Vitamin K after birth

Refusal of Vitamin K by Parents of Newborns

Meta-analysis

Global refusal	1.1%
USA	0.9-1.6%
Scotland	0.9%
Australia	2%

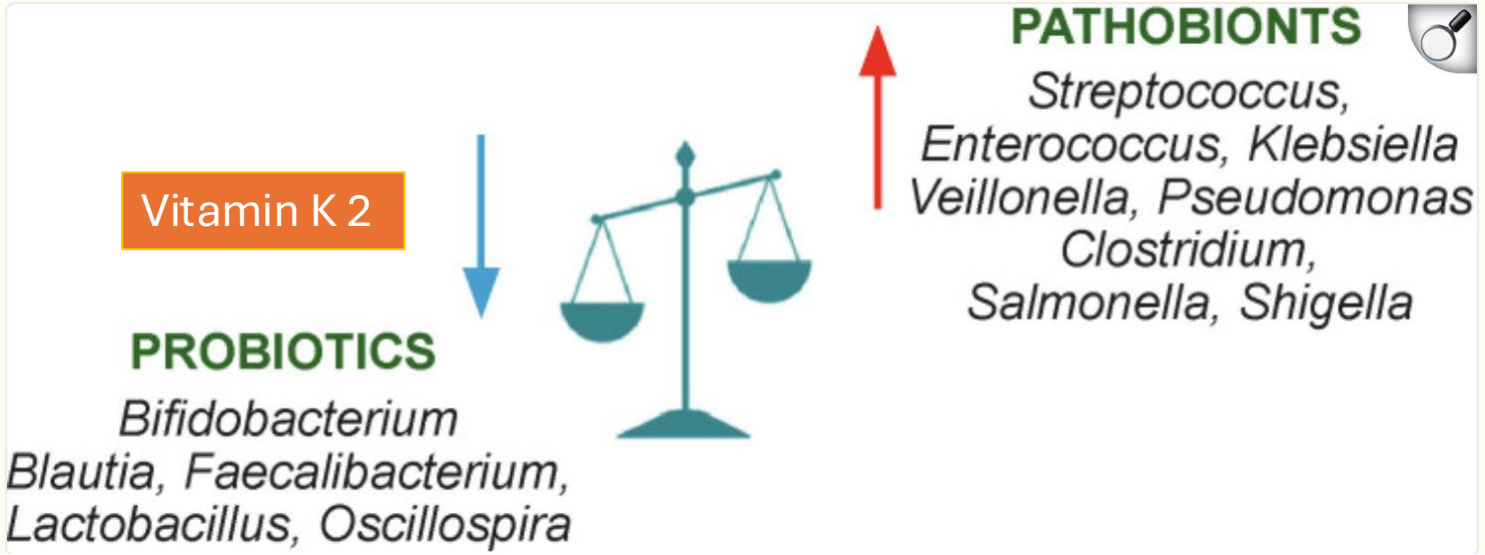
RISK FACTORS
Poor intake
Malabsorption
Gut flora

Loyal et al. (2019). Academic Pediatrics, 19(7), 793-800. Pubmed ID: 30981025.

Neonatal cholestasis

RISK FACTORS
Poor intake
Malabsorption
Gut flora

Biliary atresia



Feng S, et al(2024) Front. Cell. Infect. Microbiol. 14:1411843. Jain. World J Pediatr Surg. 2026 Jan 7;9(1):e001068.

Prevention of vitamin K deficiency bleeding in breastfed infants: lessons from the Dutch and Danish biliary atresia registries

	Feed	Birth	After	Vit K Deficiency	RR of bleeding vs formula
N '91-03	BF	1 mg oral	25 mcg D	25/30 (83%)	77.5
D '94-00	BF	2 mg oral	1 mg W	1/13 (2%)	7.2
D '00-05	BF	2 mg IM		1/10 (1%)	9.3
	Formula			1/98	

Prophylactic Dosing of Vitamin K to Prevent Bleeding

	Birth	After	VKDB	IC bleeding
N n=55	1 mg oral	25 mcg D	45/55 (82%)	22/45 (49%)
N n=11	1 mg oral	150 mcg D	9/11 (82%)	3/9 (33%)
D n=25	2 mg IM		1/25 (4%)	0/25 (0%)

van Hasselt et al. Pediatrics. 2008 Apr;121(4):e857-63; Witt et al. PEDIATRICS Volume 137 , number 5 , May 2016 :e 20154222

Prevalence of portal vein thrombosis in pediatric patients with cirrhosis and intrahepatic non-cirrhotic portal hypertension

Hakan Ozturk*, Sinan Sari, Odul Egritas Gurkan, Buket Dalgic

Cirrhotic n=142 PVT 8.4%
Non-cirrhotic n=41 PVT 9.7%

Table 1
Clinical characteristics of cirrhotic patients.

	Patients with PVT (n = 12)	Patients without PVT (n = 130)	P-values
Age (months, mean±SD)	64.7 ± 74.5	64.6 ± 66	0.995
Male gender, n (%)	4 (33.3)	79 (60.8)	0.065
Etiology of cirrhosis			0.189
PFIC	2 (16.6)	41 (31.5)	
Cryptogenic	1 (8.3)	26 (20)	
Biliary atresia	5 (41.6)	20 (15.3)	
Wilson disease	1 (8.3)	22 (16.9)	
Autoimmune hepatitis	1 (8.3)	11 (8.4)	
Others	2 (16.6)	10 (7.7)	

Table 2
Clinical characteristics of intrahepatic non-cirrhotic portal hypertension patients.

	Patients with PVT (n = 4)	Patients without PVT (n = 37)	P-values
Age (months, mean ± SD)	119.2 ± 84	126.7 ± 63.1	0.828
Male gender, n (%)	3 (75)	22 (59.5)	0.545
Etiology of intrahepatic non-cirrhotic PHT	3 (75)	13 (35.1)	0.120
Congenital hepatic fibrosis	1 (25)	24 (64.9)	
Idiopatic non-cirrhotic portal hypertension			

Digestive and Liver Disease 57 (2025) 1451–1454

Liver and Systemic Hemodynamics in Children With Cirrhosis: Impact on the Surgical Management in Pediatric Living Donor Liver Transplantation

Catherine de Magnée,[1] Francis Veyckemans,[2] Thierry Pirotte,[2] Renaud Menten,[3] Dana Dumitriu,[3] Philippe Clapuyt,[3] Karlien Carbonez,[4] Catherine Barrea,[4] Thierry Sluysmans,[4] Christine Sempoux,[5] Isabelle Leclercq,[6] Francis Zech,[7] Xavier Stephenne,[8] and Raymond Reding[1]

51 children
LT 1 (0.5-14) years

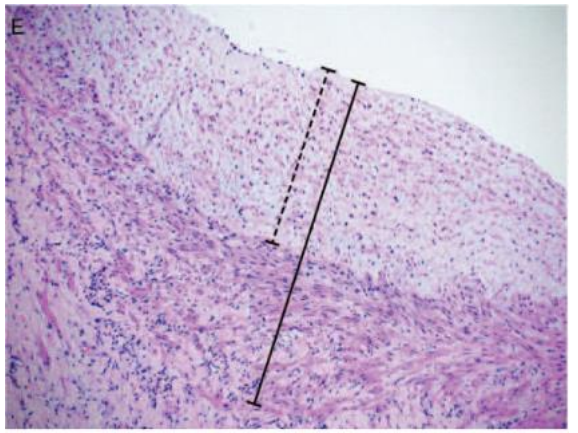
BA n=33
Non-BA cirrhotic n =13
Non-cirrhotic n=6

Native Liver Doppler Ultrasound Hemodynamic Parameter	Patients With Cirrhosis Without BA (n = 13)	Patients With Cirrhosis With BA (n = 33)	*P* Value[a]	Children Without Cirrhosis (n = 6)
Internal PV diameter, mm	6.4 (4.6-8.7)	3.8 (3.4-4.2)	0.001	5.9 (4.0-6.7)
PV hypoplasia, %	4/13 (31%)	21/33 (64%)	0.02	0/6 (0%)
Pathologic PV flow, %	4/13 (31%)	19/33 (58%)	0.25 (NS)	0/6 (0%)
PV velocity, cm/seconds	23.5 (16.0-30.5)	12.5 (3-16)	0.005	28.5 (18-51)
PV flow, mL/minute	261 (122-382)	78 (51-105)	0.002	230 (91-321)
Mean HA velocity, cm/seconds	46 (36-63)	55 (47.5-63.0)	0.27 (NS)	41 (21-79)
Hepatic ARI	0.8 (0.7-0.9)	0.9 (0.9-1.0)	0.004	0.7 (0.6-0.8)

LIVER TRANSPLANTATION, Vol. 23, No. 11, 2017

PV intima/total wall thickness ratio

	Brussels	PV intima/wall thickness
BA successful Kasai		
Non cirrhotic LD	6	Median 0.06 (CI 0.0-0.1)
Cirrhotic LD		
BA failed Kasai	**33**	**Median 0.5 (CI 0.4-0.5)**
non-BA	13	Median 0.07 (CI 0.0-0.2)
BA primary LT		



Biliary Atresia Patients With Successful Kasai Portoenterostomy Can Present With Features of Obliterative Portal Venopathy

Kalyani R. Patel, Sanjiv Harpavat, Zahida Khan, Sadhna Dhingra, Norma Quintanilla, Mihail Firan, and John Goss

Biliary atresia n=68
14 successful Kasai
LT for portal hypertension (13/14)
Median age 10 (2.5-16.2) years
PV patent 13/14
Hepatic artery index elevated 7/14

Explant 13/14
PV intima thickness median 2.89 (1.64-6.12) mm

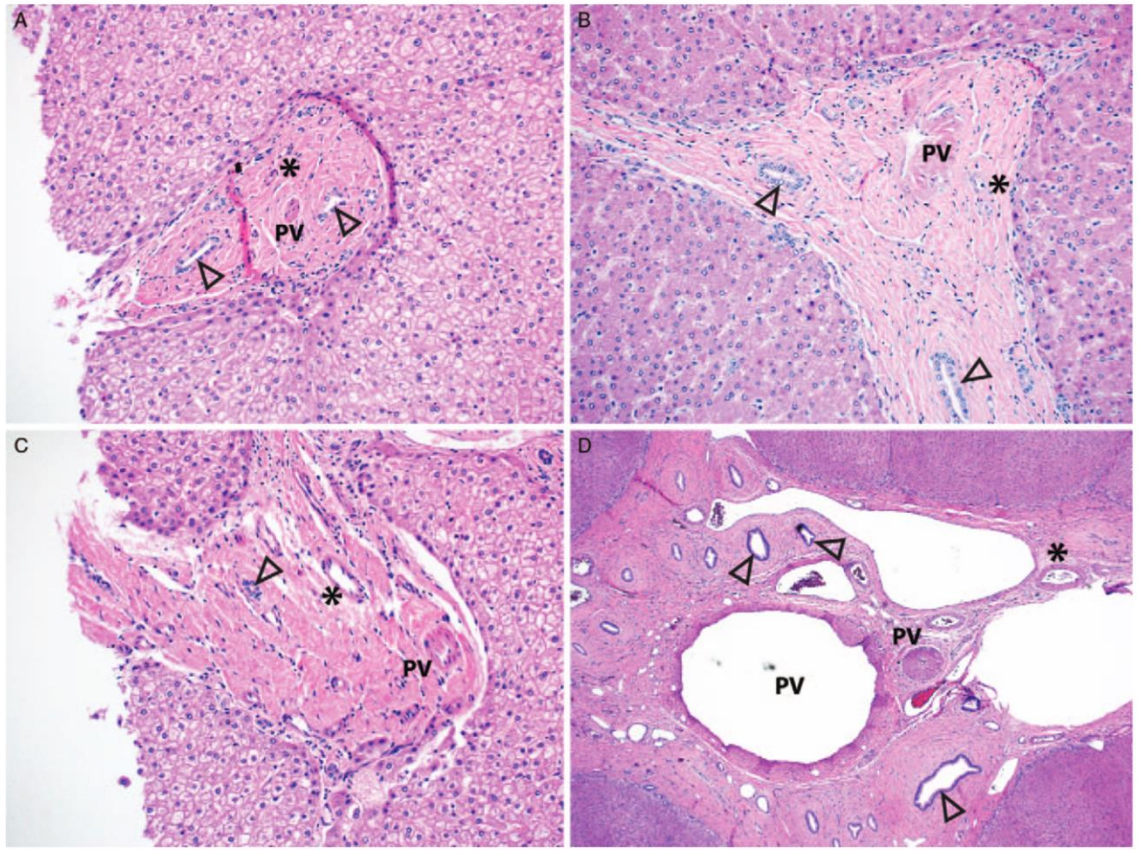
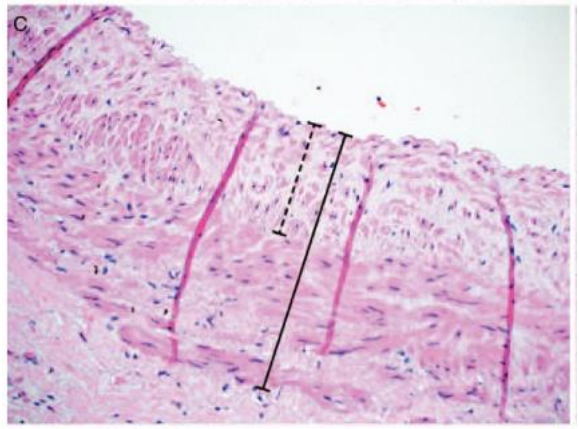


FIGURE 3. (A–C) Sclerotic portal tracts show multiple bile ducts (Δ), hepatic artery branches (*), and portal veins with complete (A) and incomplete occlusion (B and C) (H&E, 200×). (D) Large portal tract is hypervascular and shows an occluded portal vein with multiple thin walled vascular channels (H&E, 20×). H&E = hematoxylin-eosin.

Patel et al. JPGN 2020; 71 (1)

PV intima/ total wall thickness ratio



	Brussels	PV intima/wall thickness	Texas	PV intima/wall thickness
BA successful Kasai			**14**	**0.6 +/- 0.11**
Non cirrhotic LD	6	Median 0.06 (CI 0.0-0.1)	27	0.08 +/- 0.09
Cirrhotic LD			19	0.34 +/- 0.2
BA failed Kasai	**33**	**Median 0.5 (CI 0.4-0.5)**	**6**	**0.59 +/- 0.17**
non-BA	13	Median 0.07 (CI 0.0-0.2)	13	0.2 +/- 0.1
BA primary LT			**9**	**0.69 +/- 0.08**

Journal of Pediatric Gastroenterology and Nutrition
47:630–634 © 2008 by European Society for Pediatric Gastroenterology, Hepatology, and Nutrition and
North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition

Aetiology and Management of Extrahepatic Portal Vein Obstruction in Children: King's College Hospital Experience

Nehal Abd El-hamid, Rachel M. Taylor, Daniela Marinello, G.J. Mufti, Raj Patel,
Giorgina Mieli-Vergani, Mark Davenport, and Anil Dhawan

Liver, Pancreas and Biliary Tract

Etiology, presenting features and outcome of children with non-cirrhotic portal vein thrombosis: A multicentre national study

Angelo Di Giorgio[a], Paola De Angelis[b], Maurizio Cheli[c], Pietro Vajro[d], Raffaele Iorio[e], Mara Cananzi[f], Silvia Riva[g], Giuseppe Maggiore[h], Giuseppe Indolfi[i], Pier Luigi Calvo[j], Emanuele Nicastro[a], Lorenzo D'Antiga[a,*]

Digestive and Liver Disease 51 (2019) 1179–1184

1979-2005
N=108 (62% male)

Median age 4.75 (range 1day-16.3) y
42% < 5years

Risk factors
Umbilical vein catheterisation 12%
Abdominal sepsis 8%
Neonatal sepsis 8%
Associated congenital anomalies 2

Single vs multi-centre	108 vs 187
Prematurity	Italy 61%
UV catheter	12% vs 65%
Infection/sepsis	16% vs 11%
Associated anomalies	24% vs 62%
Splenomegaly	63% vs 40%
GI bleeding	44% vs 36%

TABLE 1. *Characteristics at presentation at King's C Hospital*

	No. of patients	
Splenomegaly	68	63
Bleeding	48	44
Haematemesis	47	—
Melaena	33	—
Abnormal LFTs	10	9
Hepatomegaly	5	8
Ascites	3	4
Jaundice*	1	1

LFT = liver function test.
* Portal pyemia, liver abscess.

Table 1
Risk factors and clinical features at diagnosis of 187 Italian children with portal vein thrombosis

Risk factors	
	86/187 (46%)
	101/187 (54%)
	70/172 (41%)
	105/172 (61%)
al illnesses	
prematurity	123/156 (79%)
ations	70 (53%)
formations	12 (9%)
s or omphalitis)	13 (11%)
disorders	11 (9%)
	8 (6%) 9 (7%)
ization	107/165 (65%)
nosis	
rs)	4.0 y (SD 3.7)
on	
nomegaly	68/172 (40%)
Gastrointestinal bleeding	63/172 (36%)
Detection of hypersplenism	10/172 (6%)
Incidental	31/172 (18%)
Laboratory features (mean values)	
WBC (mm/3)	6.074 (SD 3.342)
Haemoglobin (gr/dl)	10.4 (SD 2.4)
PLT (mm/3)	141.978 (SD 103.583)
INR	1.17 (SD 0.16)
Total bilirubin (mg/dl)	0.9 (SD 0.3)
Ascites	3/172 (2%)
Upper varices at first endoscopy	62/71(87%)

Coagulation factors (n=30)
Low protein C n=6
Protein C deficiency n=1
Low protein S n=2
LAC positive n=1
AT, FV Leiden, JAK2 V617F negative

4 died of associated cardiac anomalies

Shunt operation 16% (n=17)
Failure of endoscopic treatment n=13
Emergency shunt n=4
Lienorenal n=9
Mesocaval n=5
Meso-Rex n=2
Porto azygos n=1

Functioning shunt 53%
12.8 years follow up

Table 2
Inherited prothrombotic disorders in 76 patients witl

Parameters	All patients N = 76 (%)	G1 N = 53 (%)	G2 N = 23 (%)	P value
Protein C deficiency[a]	6 (20%)	5 (83%)	1 (17%)	0.04
Protein S deficiency[a]	2 (7%)	1 (50%)	1 (50%)	1.00
Protein C and S deficiency[a]	7 (23%)	5 (71%)	2 (28%)	0.14
Prothrombin (G20210A)	2 (7%)	2 (100%)	0 (0%)	0.16
Factor V leiden-M	2 (7%)	2 (100%)	0 (0%)	0.16
MTHFR (C677T and A1298C)	11 (37%)	8 (73%)	3 (27%)	0.04
Positive Pro-thrombotic screening	30 (39%)	23 (77%)	7 (23%)	0.21

Table 3
Data at last follow up in 187 children with EHPVO.

Mean follow up	11.2 years (SD 4.8)
Survived	185/187 (99%)
Died	2/187 (1%)
Conservative treatment	124/187 (66%)
Surgery or TIPS	63/187 (34%)
Meso-Rex bypass[a]	30 (48%)
Distal spleno-renal shunt	16 (25%)
Proximal spleno-renal shunt[b]	7 (11%)
Meso-caval shunt[c]	4 (6%)
Isolated splenectomy	2 (3%)
Porto-caval shunt	1 (2%)
Liver Tx	1 (2%)
TIPS	2 (3%)
Patent Shunts at last FU	54/60 (90%)

Digestive and Liver Disease 51 (2019) 1179–1184

Portal-vein Obstruction in Children Leads to Growth Retardation

SHIV K. SARIN, ANUPAM BANSAL, SHAILJA SASAN AND ARUNA NIGAM
Department of Gastroenterology, G. B. Pant Hospital, New Delhi, 110 002, India

Hepatology, 1992

Moreover, several children who have been under follow-up since adolescence have shown endocrinal anomalies along with delayed growth. In three young female patients, menarche has not been achieved even at 17,18 and 20 yr. In one male patient, secondary sexual characters have still not developed at 20 yr.

61 children (male 46)
8.4 years

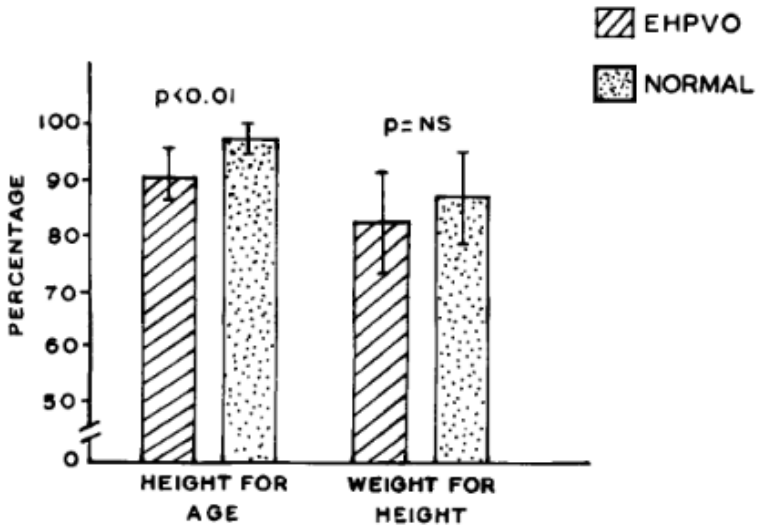


FIG. 1. Anthropometric indices in EPVO patients and healthy controls.

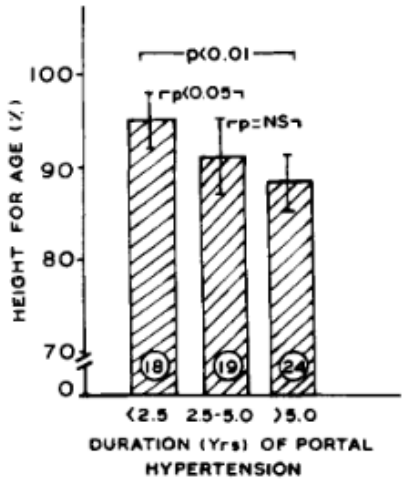


FIG. 2. HA in EPVO patients with clinical portal hypertension of different durations.

Abdominal Radiology (2019) 44:1379–1394
https://doi.org/10.1007/s00261-018-1836-1

REVIEW

Multimodality imaging of the Meso-Rex bypass

Vincenzo Carollo[1] · Gianluca Marrone[1] · Kelvin Cortis[2] · Giuseppe Mamone[1] · Settimo Caruso[1] · Mariapina Milazzo[1] · Luigi Maruzzelli[1] · Fabrizio di Francesco[1] · Martin Delle[3] · Roberto Miraglia[1] · Jean de Ville de Goyet[1]

Abdominal Radiology (2019) 44:1379–1394

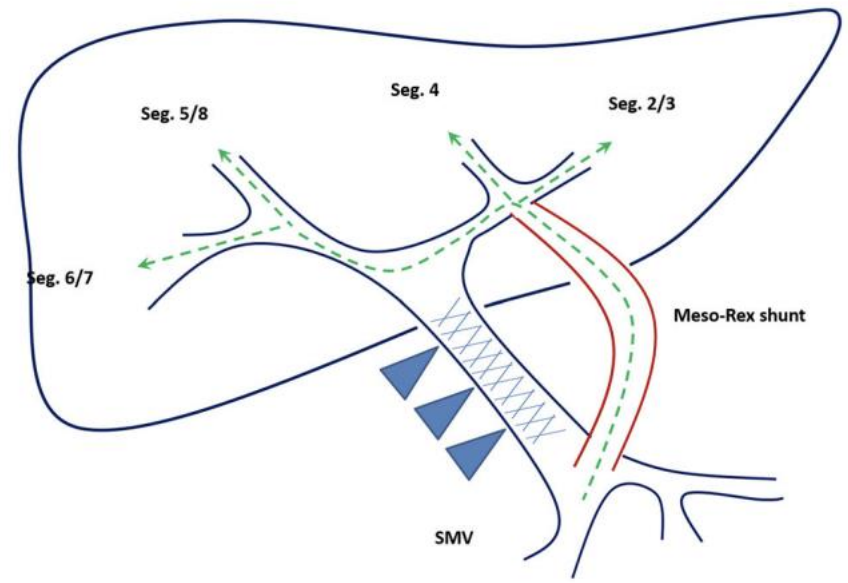


Fig. 14 Schematic illustration of the Meso-Rex shunt. Arrows denote appropriate direction of flow. Arrowheads indicate occluded extra-hepatic portal vein.

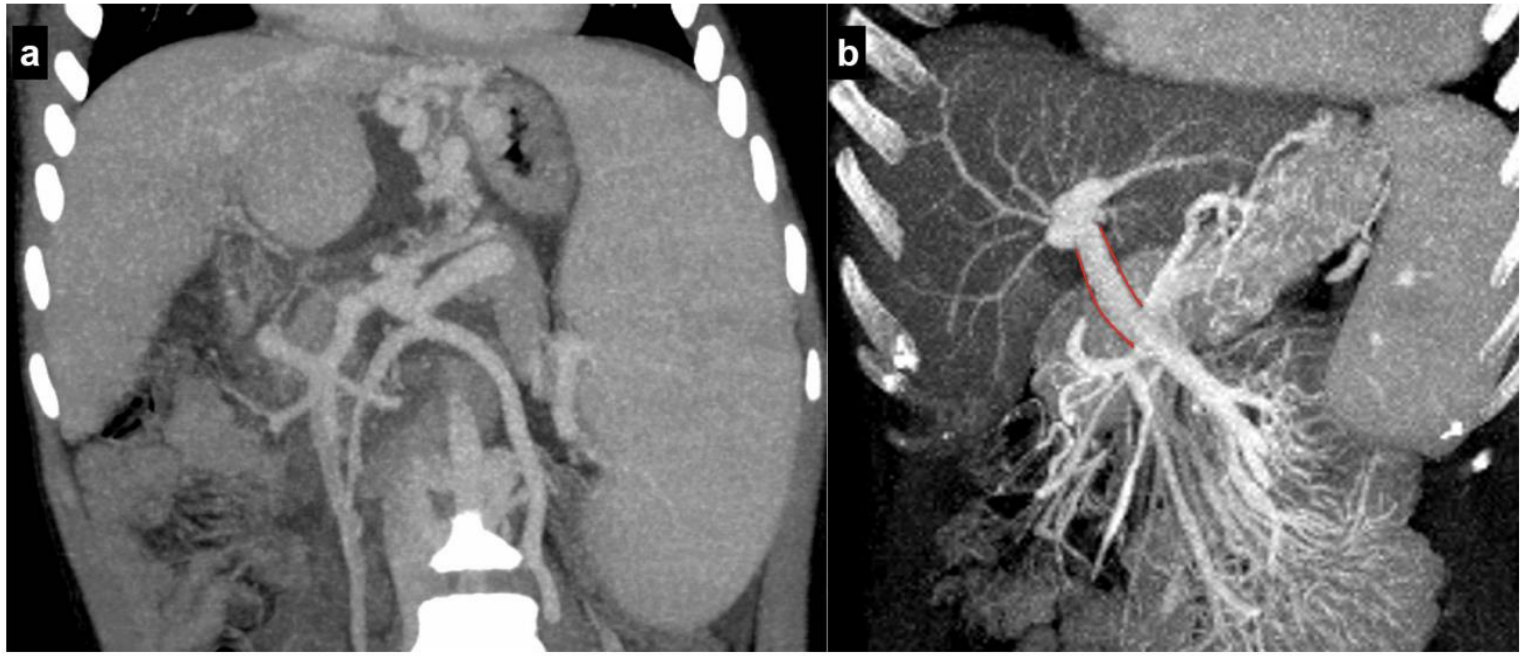


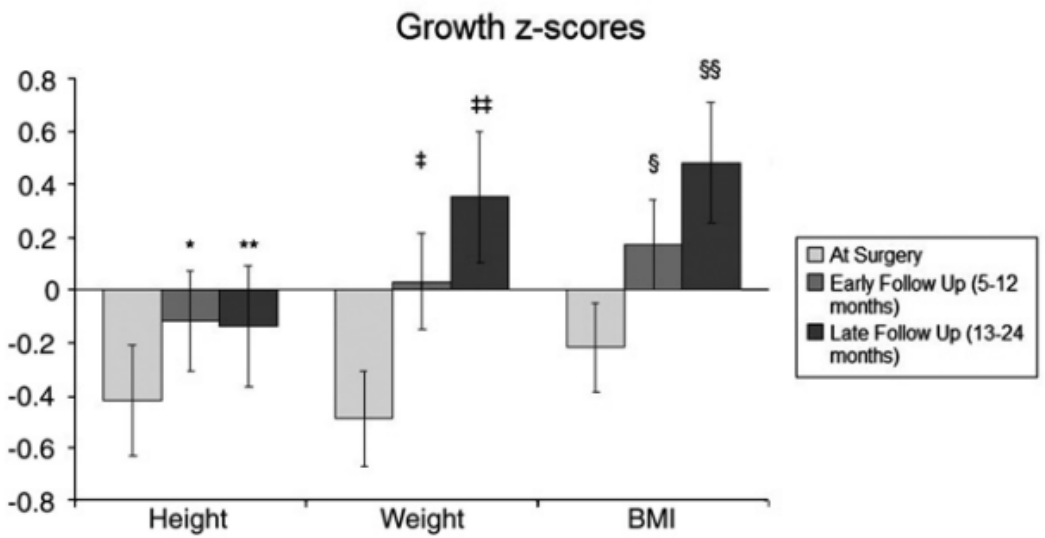
Fig. 17 Coronal CT images prior to, and two years following, Meso-Rex shunt surgery (**a** and **b**, respectively). The portosystemic collaterals visible in (**a**) have resolved post-surgery, given restoration of physiological hepatopetal flow. Liver morphology has normalized and the spleen size has also reduced.

Original articles

Growth impairment in children with extrahepatic portal vein obstruction is improved by mesenterico-left portal vein bypass

Timothy B. Lautz[a,*], Shikha S. Sundaram[b], Peter F. Whitington[b], Lisa Keys[a], Riccardo A. Superina[a]

N=45 (17 male)
Age 8.4 years



J of Pediatric Surgery, 2009

Percutaneous recanalization of non-cirrhotic extrahepatic portal vein obstruction in children: technical considerations in a preliminary cohort

Paolo Marra[1*], Stephanie Franchi-Abella[2,3], José A. Hernandez[4,5], Maxime Ronot[6], Riccardo Muglia[1], Lorenzo D'Antiga[7,8] and Sandro Sironi[1,8]

Table 1 Demographics and summarized baseline clinical data of the study population

Age	Median (range), 7 (1–14), years
Sex	*N* = 6 (55%) males; *N* = 5 (45%) females
Putative etiology of EHPVO	*N* = 8 (73%) UVC *N* = 1 (9%) perinatal sickness without UVC *N* = 2 (18%) unknown
Clinical manifestations of portal hypertension	*N* = 7 (64%) history of gastrointestinal bleeding *N* = 4 (36%) high-risk varices *N* = 11 (100%) hypersplenism
Rex recessus patency	*N* = 4 (36%) yes *N* = 7 (64%) no
Spleno-mesenteric obstruction	*N* = 2 (18%) yes *N* = 9 (82%) no

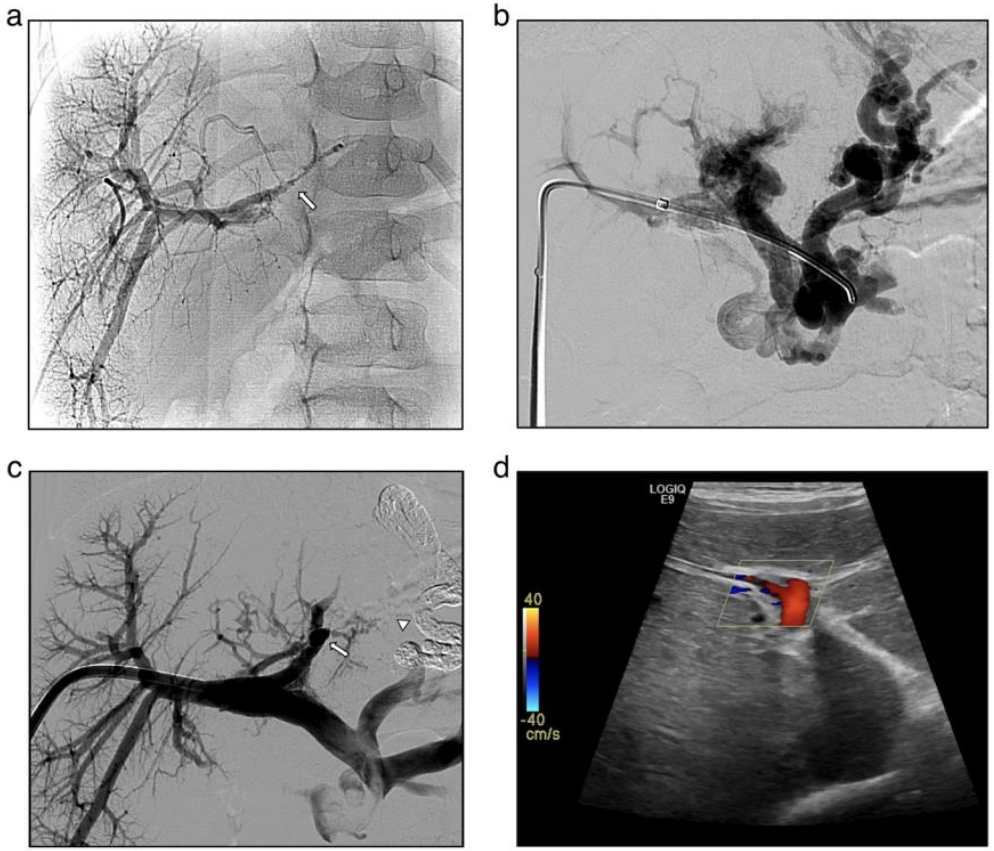


Table 3 Outcome data of successful PVR procedures

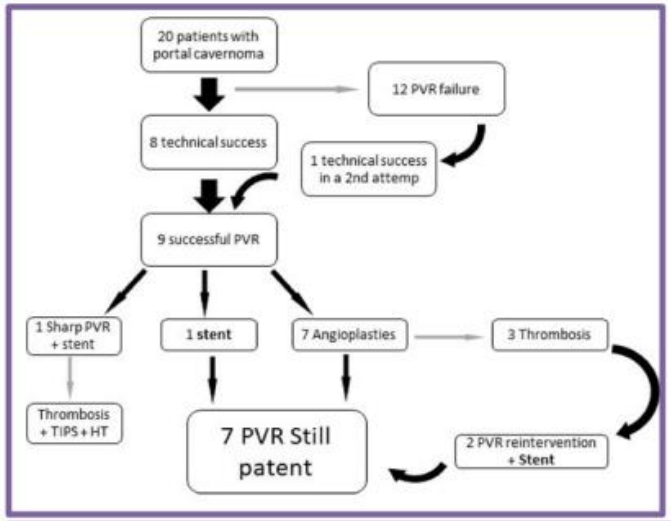
	***N* = 5 successful PVRs**
Rex recessus patency at baseline	*N* = 2
Portal vein patency at last follow-up	*N* = 5
Rex recessus patency at last follow-up	*N* = 3
Follow up time	Median (range), 6 (6–14) months
Clinical manifestations of portal hypertension	*N* = 5 none
Platelet count at baseline	Median (range), 89 (72–174) × 10^9/L
Platelet count at last follow-up	Median (range), 170 (148–266) × 10^9/L

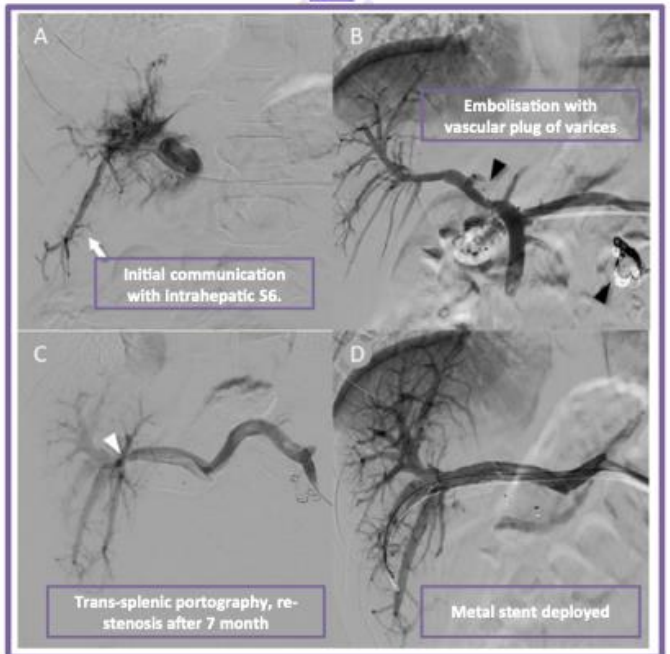
Marra et al. European Radiology (2025) 35:3262–3269

The official journal of the Cardiovascular and Interventional Radiological Society of Europe
CVIR CardioVascular and Interventional Radiology
Springer
CIRSE

PERCUTANEOUS RECANALIZATION OF THE PORTAL VEIN IN CHILDREN WITH EXTRAHEPATIC PORTAL VEIN OBSTRUCTION

Evaluate feasibility, technical success, clinical outcomes and safety of percutaneous portal vein recanalization (PVR) in children with extrahepatic portal vein obstruction.

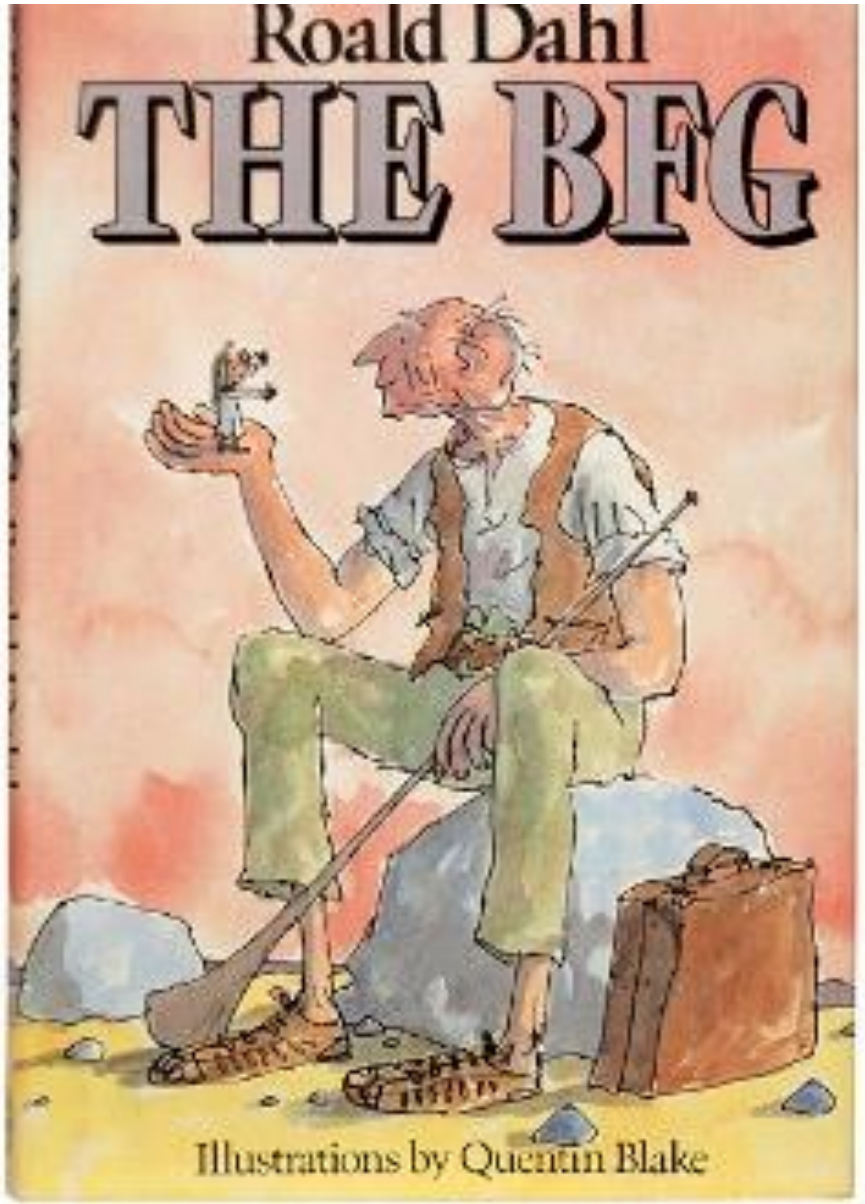




Outcome data of successful PVR N=9	
Portal vein patency at last follow up	N=7
Median Follow-up time	17,3 (5-30) months
Median primary patency	10,5 (1,8-18.3) months
Median assisted patency	16 (1,8 – 26) months
Stent deployed at first attemp	1
Stents in secondary procedures	2
Spleen size at baseline (median z-score)	6,30cm [SD = 5,00]
Spleen size at lastfollow -up(median z-score)	4,67 [SD = 2,5]
Platelet count at baseline	164.11 × 10^9/L (SD = 126.24)
Platelet count at lastfollow up	213.75 × 10^9/L (SD= 99,95)

PVR is a feasible and minimally invasive option. When successful, it may restore the physiological intrahepatic portal inflow and improve portal hypertension-related complications in children. Despite its complexity and not being without complications, PVR may serve as an alternative to surgical approaches. ✓

Daniel Barnes-Navarro et al. 2026



‘Rebalanced homeostasis’ relates to children with liver disease but does not recover fully within 3 months after liver transplantation.

Vitamin K deficiency bleeding is a concern in infants with liver disease with long-term consequences.

How do the findings of PV intima thickness in biliary atresia relate to adult data?

EHPVO is prevalent in children with premature infants with umbilical catheters being more at risk.

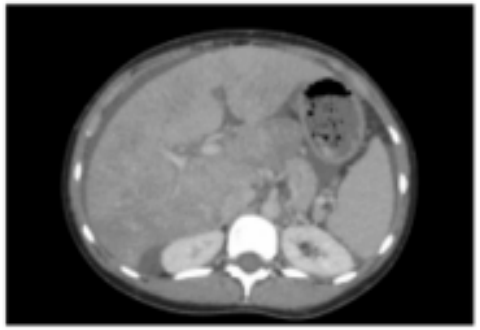
Restoration of intrahepatic portal vein flow should be favoured to avoid long-term consequences including growth, hormones and cognitive function.

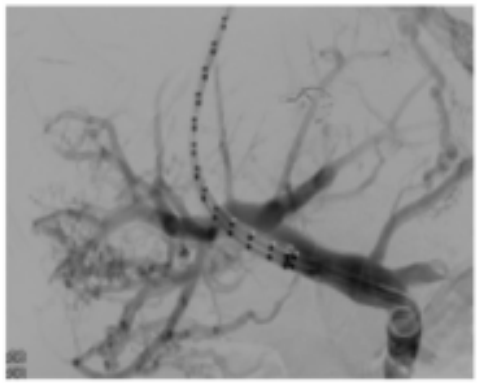
The official journal of the Cardiovascular and Interventional Radiological Society of Europe

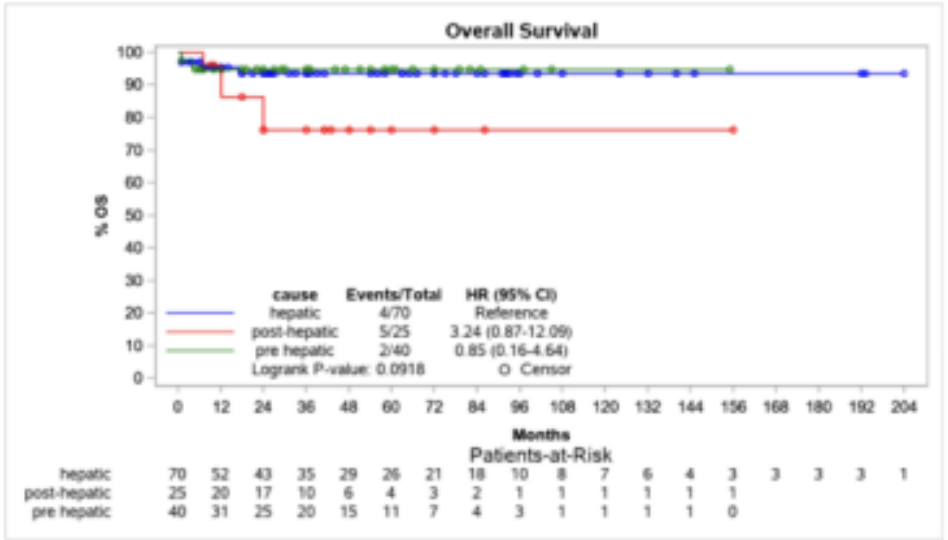
CardioVascular and Interventional Radiology

Sinan Deniz, Regina Schinner, Eric J. Monroe, Simon Horslen, Ravi N. Srinivasa, Yong Lv, Daiming Fan, Guohong Han, Moinak Sen Sarma, Anshu Srivastava, Ujjal Poddar, Rajanikant Yadav, Thi Phuong Thao Hoang, Christian M. Lange, Osman Öcal, Jens Ricke, Max Seidensticker, Eberhard Lurz, Angelo Di Giorgio, Lorenzo D'Antiga, Moritz Wildgruber

Outcome of Children and adolecesents with Transjugular Intrahepatic Portosystemic Shunt – a meta-analysis of individual patient data







- TIPS in children is safe and associated with good patency
- Survival after TIPS is impaired in case of ascites and in cases of post-hepatic obstruction
- TIPS in children should be performed in dedicated centers with the option for pediatric liver transplantation

TABLE 1. Demographic details, radiological features, management, and outcome of children with Budd-Chiari syndrome

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7
Age at diagnosis, y	2	15	10	3	11	14	17
Gender	F	F	M	M	M	M	F
Duration of symptoms, days	150	14	60	14	75	21	180
Type of presentation	II	III	II	I	II	III	II
Variceal bleed	−	−	+	−	−	−	+
Hypercoagulable state	PRV (JAK2 +) ACLA IgG + Protein-C def (51%) Anti-thrombin-III def (74%)	PRV (JAK2 +)	ACLA IgG +	Protein C deficiency (29%) Anti-thrombin-III def (66%)	Protein-C def (63%)	PNH LA +	FVL
Associated disease (s)	Dural sinus thrombosis Nephrotic syndrome	−	−	−	IBD	Sagittal sinus thrombosis	−
Radiographic features							
HV (s) occlusion	RHV not visualised LHV and MHV attenuated	All 3 HVs blocked	LHV not visualised RHV and MHV attenuated	All 3 HVs blocked	All 3 HVs blocked	All 3 HVs blocked + IVC	All 3 HVs blocked
IVC occlusion	Compressed	Compressed	−	−	Compressed	Partially thrombosed	Compressed
Focal lesion	−	+ (NRH)	−	−	+ (multiple NRH)	−	+ (multiple NRH)
Management	Anticoagulation PEG-IFN alpha Hydroxyurea	Anticoagulation Venesection PEG-IFN alpha TIPSS	Anticoagulation HV stenting	Liver transplantation Anticoagulation	Meso-caval shunt Anticoagulation	Anticoagulation TIPSS Partial ET BMT	Liver transplantation Anti-coagulation
Complications	Nil	Nil	Stent block Infection of stent	Nil	Hepatic encephalopathy	TIPSS occlusion	Nil
Follow-up, y	4	6	4	3	13	9	12
Follow-up progress	JAK2 clones reduced from 27% to 3% Patent vessels on Uss Recurrence at 3 years with nephrotic syndrome; treated with steroids; PEG-IFN alpha changed to hydroxyurea and ruxolitinib	JAK2 clones reduced from 14 to 3.4% Patent TIPSS with NRH on Uss Normal LFT and AFP	Stent blocked (balloon dilated) Stent infected (prolonged IV antibiotics). Subsequently asymptomatic Patent HVs and stent on Uss	Triple IMS Continued on anticoagulation Died at 3 years of follow-up due to subarachnoid haemorrhage	Anti-ammonia measures for HE Asymptomatic since Patent shunt with multiple regenerative nodules on Uss Normal LFT and AFP	Recanalization of TIPSS with balloon dilatation No evidence of any PNH clone in the recent bone marrow	Dual IMS. Continued on anti-coagulation Patent vessels in liver graft on Uss

Hepatocyte Transplantation for Inherited Factor VII Deficiency

Anil Dhawan, Ragai R. Mitry, Robin D. Hughes, Sharon Lehec, Claire Terry, Sanjay Bansal, Rupen Arya, Jim J. Wade, Anita Verma, Nigel D. Heaton, Mohamed Rela, Giorgina Mieli-Vergani

TABLE 1. Details of hepatocyte infusions in children with FVII deficiency

	Patient 1	Patient 2
Age at time of first infusion	3m	2y 11m
Weight (kg)	6.1	15.2
No. of infusions	3 cryo	3 fresh + 2 cryo
Total no. of cells	1.09×10^9	2.18×10^9
Cell viability (%)[a]	90 (80–90)	80 (50–90)
Portal pressure (mm Hg)[a]		
Pre	7 (7–8)	7 (5–11)
Post	10 (8–17)	14 (10–26)
Dose of rFVIIa (μg/kg/day)		
Pretransplant	118	58
3 months	28	13
6 months	35	17

[a] Median (range).

cryo, cryopreserved hepatocytes; FVII, factor VII; rFVIIa, recombinant factor VIIa.

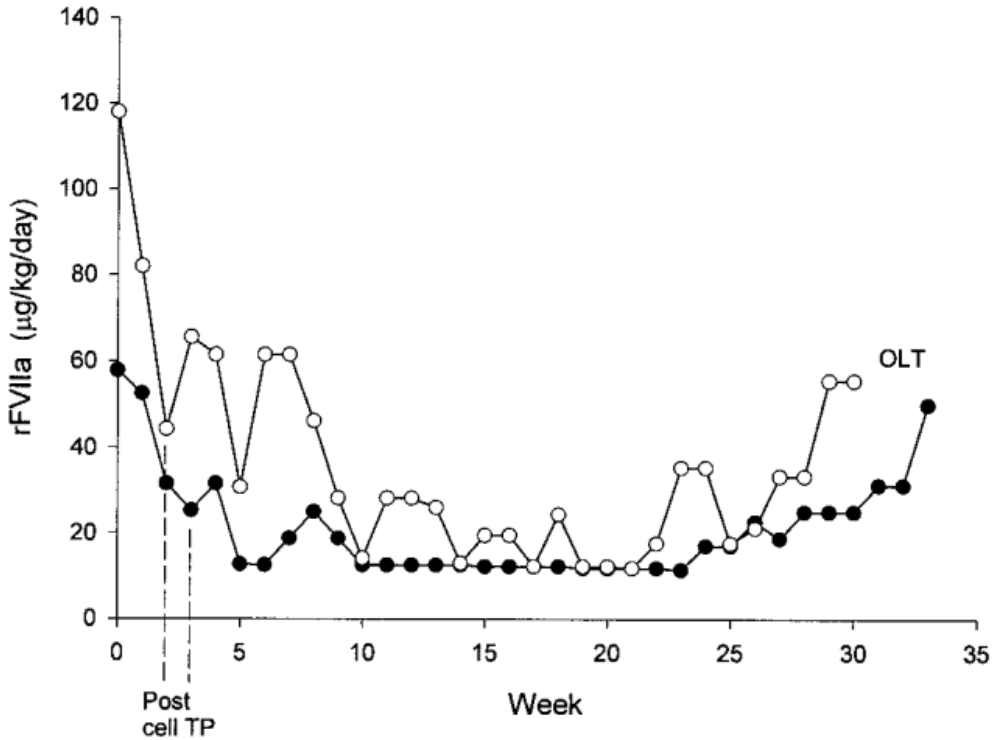


FIGURE 1. Daily dose of recombinant factor VIIa (rFVIIa) in patients receiving hepatocyte transplantation. Patient 1 (*open circle*) and patient 2 (*filled circle*). OLT, orthotopic liver transplantation; TP, transplantation.