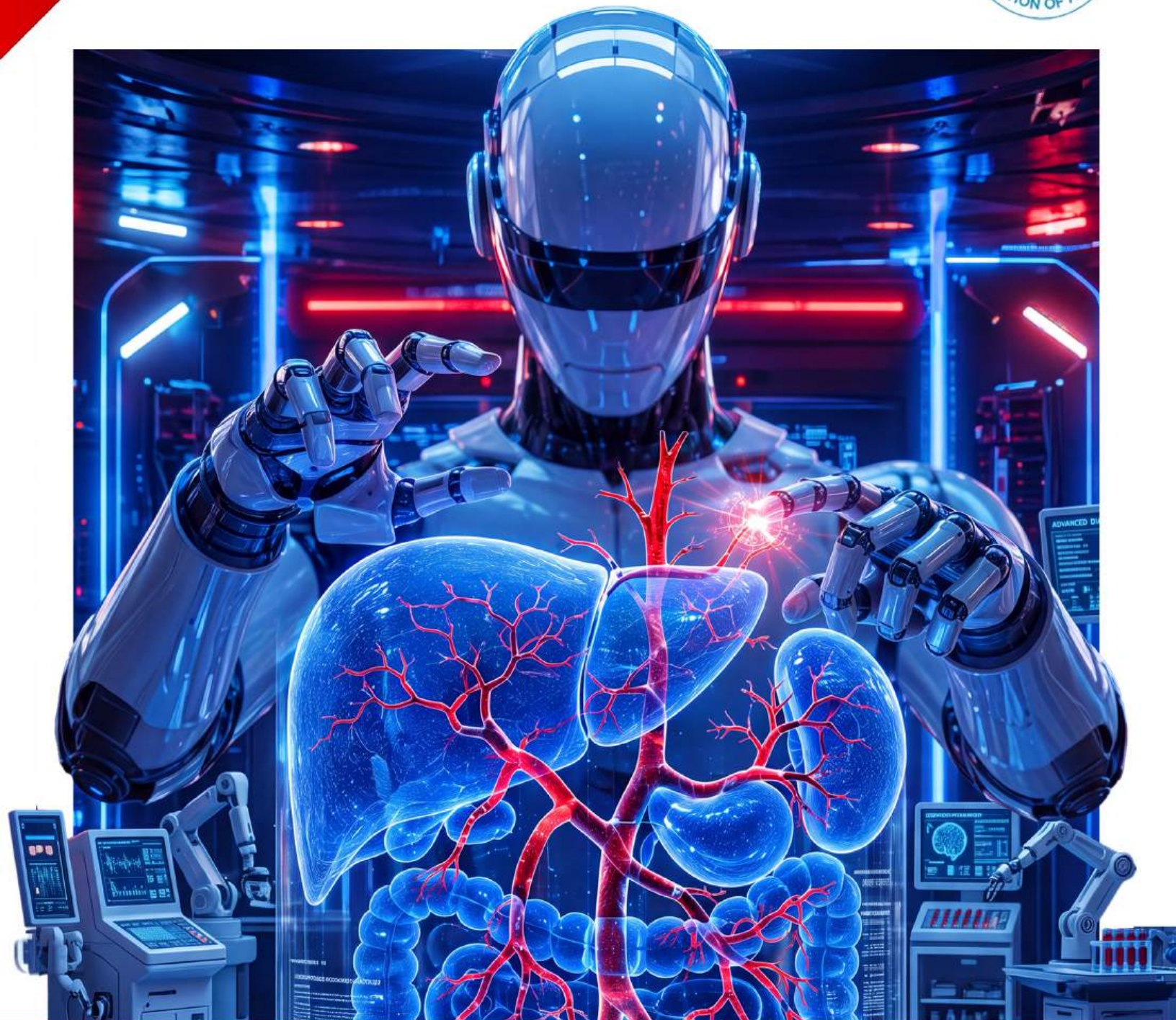


OCTOBER
ISSUE

SUTRA

OCTOBER 2026 | VOL - 04



THE NEWSLETTER OF THE INDIAN ASSOCIATION
OF SURGICAL GASTROENTEROLOGY

SPECIAL ISSUE ON PORTAL HYPERTENSION



NAMAKARANA

WHY SUTRA?

The Many Threads of “Sutra”: A Sanskrit Word’s Rich Tapestry

The Sanskrit word “Sutra” literally means “thread”, “cord” or “string”, forms a foundational concept in Indian intellectual and religious traditions, weaving altogether meanings far beyond its simple material origin. Its significance unfolds across multiple dimensions:

Literal and Practical

At its most basic, a sutra is a physical thread used for sewing, binding, or measuring – the essential tool holding disparate elements together. This tangible sense underpins its profound metaphorical extensions.

Philosophical and Scientific Code

Most famously “Sutra” refers to an extremely concise aphorism or rule. These are dense, formulaic statements designed to encapsulate vast areas of knowledge – philosophy, grammar (like Panini’s Ashtadhyayi), law, ritual, Mathematics, or statecraft (e.g., Kautilya’s Arthashastra). Their brevity was mnemonic, aiding oral transmission. Think of them as “threads” upon which the pearls of complex knowledge were strung, requiring later commentary (bhashya) to unravel their full meaning.

Sacred Scripture

In religious contexts, particularly Buddhism and Hinduism, “Sutra” denotes a category of canonical scripture. Buddhist Sutra (Like the Lotus Sutra and the Heart Sutra) typically record discourses attributed to the Buddha, expounding doctrine and practice. Hindu Sutras (like Brahma Sutras, Yoga Sutras of Patanjali, or various Dharma Sutras) are often collections of those concise aphorisms systematizing theological, ritual, ethical, or meditative knowledge within specific schools.

Structural Principle

The concept of a “thread” implies connection and structure. Sutras serve as the core framework, the essential connecting threads, upon which larger bodies of commentary, explanation, and elaboration are built. They provide the skeletal structure holding the flesh of the tradition together.

Characteristic Brevity

The defining feature of a sutra text is the extreme conciseness. Authors aimed to express the maximum meaning in the minimum words, often omitting connectives and grammatical particles. This compression made the potent but also necessitated expert interpretation.

In essence, “Sutra” embodies the act of binding knowledge. From the literal thread, it evolved into the metaphorical thread connecting ideas, the compressed code preserving wisdom, the foundational text structuring religious paths, and the principle of brevity demanding deep engagement.

It represents the elegant, efficient “threading together” of complex thought into a portable, enduring form – a testament to the power of language to weave intricate tapestries of meaning from the simplest of beginnings.

Suggested by **Dr. Gayatri Balachandran**

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Send their best wishes to this special issue of

SUTRA

on PORTAL HYPERTENSION



Dr. Patta Radhakrishna

Executive Editor, 'SUTRA'
Indian Association of Surgical Gastroenterology.

From the Executive Editor's Desk

Dear Friends,

First and foremost, my sincere apologies for the long gap between the previous issue of Sutra and this one. Rather than dwell on the reasons for the delay, I would prefer to celebrate what has finally emerged—a special issue devoted to Portal Hypertension, with particular emphasis on Non-Cirrhotic Portal Hypertension (NCPH) in the Indian context.

Creating this issue has been a labour of commitment and passion. Our Chief Editor, Dr. Magnus, together with Dr. Shraddha Patkar, Dr. Gayathri Balachandran, Dr. Nisha Jain, Dr. Deeksha Kapoor, Dr. Shravan Nadkarni and Dr. Abhishek Agrawal, has worked tirelessly to bring together material that we hope will be informative, relevant and enjoyable to read.

We are privileged to feature contemporary stalwarts of portal hypertension surgery in India, including Prof. Samiran Nundy and Prof. Surendra K. Mathur. Equally important, we revisit the remarkable contributions of pioneers such as Dr. Bhalerao, Dr. Aikat, Dr. Ramalingaswami, Dr. Ajit Kumar Basu and Prof. Solomon Victor—men whose work helped shape our understanding and management of portal hypertension in India.

A special word of appreciation goes to our graphic artist and visual editor, Mr. Subash Ramalingam, whose creativity, dedication and several sleepless nights have given this issue its final form.

Behind every issue of Sutra are many minds, many hands and countless hours of unseen work. The greatest reward for all of us would be for you to read this issue from cover to cover.

Do write back with your thoughts, criticism and suggestions. They will help us make Sutra better with every issue.

Happy reading!

Dr. Patta Radhakrishna

Executive Editor



Dr. Magnus Jayaraj Mansard

Editor-in-Chief, 'SUTRA'

Indian Association of Surgical Gastroenterology.

From the Editor's Desk

Portal hypertension in India is not a rare disease managed occasionally — it is a large, young, and often socioeconomically disadvantaged patient population, presenting with a disease pattern that the West rarely encounters at this scale. Over five decades, its management has moved from emergency shunt surgery to a multidisciplinary model built on endotherapy, TIPS, and transplantation. What has not changed is the need for surgical expertise when these modalities reach their limits.

Each contribution in this issue takes up a different piece of that argument. Dr. Richa Lal's two-decade pediatric series examines what remains — splenomegaly, growth retardation, portal cavernoma cholangiopathy — after variceal eradication is achieved endoscopically, and why portosystemic shunt surgery still has a defined role beyond it. Dr. Ajit Mishra's account of the proximal splenorenal shunt sets out, step by step, the technical pearls ordinarily passed down only at the operating table. Dr. Samiran Nundy traces fifty years of practice in India, from shunt-first management to the current transplant era, without losing sight of where surgery remains indicated.

Prof. S. K. Mathur's interview closes the issue with a firsthand account of this evolution — from a single Sengstaken-Blakemore tube and a primitive operating room in Surat to the surgical culture he went on to build. It's a timely reminder: the expertise this issue documents took many decades to build and under difficult conditions — it has to be taught, deliberately, to the next generation. The issue also carries several further case reports and technical notes that broaden this theme, touching on portal biliopathy, non-cirrhotic portal hypertension, and the Meso-Rex bypass in children.

I am grateful to every contributor for the time and clinical experience they brought to this issue. My hope is that it functions both as a technical reference and as a case for sustaining this unique surgical skill set.

With regards,

Dr. Magnus Jayaraj Mansard

Editor-in-Chief, Sutra — IASG Newsletter

Consultant, Liver Transplant & HPB Surgery

GEM Hospital, Chennai

TABLE OF CONTENTS

SPECIAL ISSUE ON PORTAL HYPERTENSION

Portosystemic Shunt Surgery for Extrahepatic Portal Venous Obstruction beyond Endoscopic Variceal Eradication	9	Dr. M. M. Zameer - Giving Tiny Lives a Bigger Chance	62
Dr. Richa Lal			
The Recognition of Non cirrhotic Portal Fibrosis	15	Dr Solomon Victor and Surgery for Budd-Chiari Syndrome	63
Proximal Splenorenal Shunt in Extrahepatic Portal Vein Obstruction: Operative Technique and Technical Pearls	18	Interventional Radiology for Variceal Bleeding: A Modern Approach to Portal Hypertension	70
Dr. Ajit Kumar Mishra		Dr. Ranjan K Patel • Dr. B D Pattnaik	
Dr. Rajaram Bal Bhalerao: A Life in Surgery and Theatre	28	Mine: Work Life Balance across a Lens	76
		Dr. Puneet Dhar	
Upfront Hepaticojejunostomy in Portal Biliopathy - Our experience at SGRH	31		
Amitabh Yadav • Samiran Nundy			
Decoding the Deceptive Spectrum of Non-cirrhotic Portal Hypertension (NCPH)	35		
Dr. Vulimiri Ramalingaswami	40		
Prof. Samiran Nundy: A Pioneer of Portal Hypertension Surgery in India	41		
Fifty Years of Experience in Portal Hypertension and Its Surgical Management in India	42		
Amitabh Yadav • Samiran Nundy			
Dr. Gayatri Balachandran in conversation with Prof. S. K Mathur	49		
Professor Ajit Kumar Basu: A Life in Surgery and Service	56		
Meso-Rex Bypass in Children			

Portosystemic Shunt Surgery for Extra-hepatic Portal Venous Obstruction beyond Endoscopic Variceal Eradication

- Author: Dr. Richa Lal

The three key approaches to managing extrahepatic portal venous obstruction (EHPVO) have been as follows: a) Total porto-systemic shunt (PSS) for every case following endoscopic control of first major bleed episode ^{1,2} b) Mesorex bypass (MRB) for primary and secondary prophylaxis should the anatomy and indications be favourable for MRB ³ c) Endotherapy for secondary prophylaxis till endoscopic eradication of esophageal varices (EEEV), regular endoscopic surveillance and surgical intervention for select indications post EEEV ^{4, 5, 6, 7}. The third management protocol has been followed at the reporting center since the year 1992. Further, this management algorithm has provided a unique opportunity to witness the natural evolution of the disease following EEEV.

We have published 2 decades of our pediatric surgical experience with PSS beyond EEEV for EHPVO ⁸. This article aims to share insights from our published experience.

During the study period, 869 new cases were initiated on endotherapy. Of these, only 178 (20%) cases were advised surgery for adverse sequelae of EHPVO not amenable to endotherapy either at initial presentation or post EEEV. At the time of data analysis, 139 cases had undergone surgery ⁸.

The study results documented that endotherapy for secondary prophylaxis till EEEV has resulted in a shift in indications for PSS. Beyond EEEV, surgery was indicated predominantly for non-variceal sequelae of EHPVO. Varices warranted surgery infrequently but, more often from sites less amenable to endotherapy namely isolated gastric varices (IGV) and ectopic varices (EV). The non-variceal sequelae warranting surgery were massive splenomegaly and severe hypersplenism (n=132, 95%), growth retardation (GR) (n=95, 68%) and complications of portal cavernoma cholangiopathy (PCC) (n=07, 5%) (Figs. 1a, 1b & 2). Each of these continued to significantly impact quality of life (QOL) adversely despite variceal eradication ⁹. Further, in this pediatric surgical series, the

median age of presentation of PCC with cholestasis was 14 years (range: 11-21 years) and liver biopsy documented cirrhosis in 1 case as early as 11 years of age ⁸. Our institutional protocol, therefore, comprises of screening every pediatric EHPVO case for PCC through a complete liver function test (LFT) which must include a Gamma glutamyl transferase (GGT). Early portal decompression surgery may halt progression to fixed ischemic biliary strictures ¹⁰. On the other hand, PCC could progress to chronic liver disease necessitating a liver transplant, if not intervened in the appropriate “window of opportunity”. Liver transplant in the setting of EHPVO with PCC presents tremendous technical challenges.

In our study, varices necessitated surgery infrequently either for failed endotherapy and pharmacotherapy or for bleeding/high-risk varices at sites wherein endotherapeutic bleed control and variceal eradication is difficult, namely large IGV (n=32, 23%) and ectopic varices (n=17, 12%) (Figs.3 &4). Ectopic varices were either duodenal (n=8),



Fig. 1a



Fig 1b: Massive splenomegaly with a splenic infarct

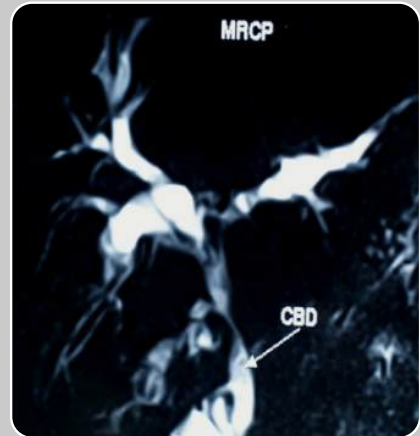


Fig. 2: Portal cavernoma cholangiopathy

small bowel (n=2) or anorectal (n=7). A significant proportion, ie 84 % of IGV and 88% of ectopic varices were secondary⁸. In addition to bleeding varices, we also offered pre-emptive early elective PSS surgery for non-bleeding but high risk IGV and ectopic varices because should these bleed, the bleed is more torrential and less amenable to

endotherapeutic control. Uncontrolled gastric variceal bleed may warrant an emergency bleed control salvage surgery wherein the surgeon may be forced to take recourse to a devascularization procedure instead of a PSS often with a higher mortality as compared with an elective procedure^{4,5,6,7}. Moreover, many of these patients came from geographically remote areas with poor access to emergency medical services.



Fig.3: Isolated gastric varices on CT Enterography angiography



Fig.4: Jejunal ectopic varices on CT Enterography angiography

What is the pathogenesis of these late onset sequelae post EEEV? While endotherapy obliterates natural porto-systemic collaterals in the gastro-esophageal region, persistently elevated portal pressures manifest as secondary IGV, ectopic varices, massive splenomegaly & PCC with cholestasis^{7,9,10..}

The choice of portal decompression procedures was individualized to the anatomy of the porto-spleno-mesenteric venous system and surgical indications.

Central splenorenal shunt

(CSRS) with splenectomy was performed most frequently (n=104) because massive splenomegaly with severe hypersplenism was one of the predominant indications for surgery in 95% patients. Splenectomy alone without concomitant splenorenal shunt results in SV thrombosis and hence loss of a potentially shuntable vein should the case present with other sequelae of EHPVO later. Hence, our surgical practice entails combining splenectomy with a concomitant CSRS whenever SV is shuntable, though varices may have been eradicated endoscopically.

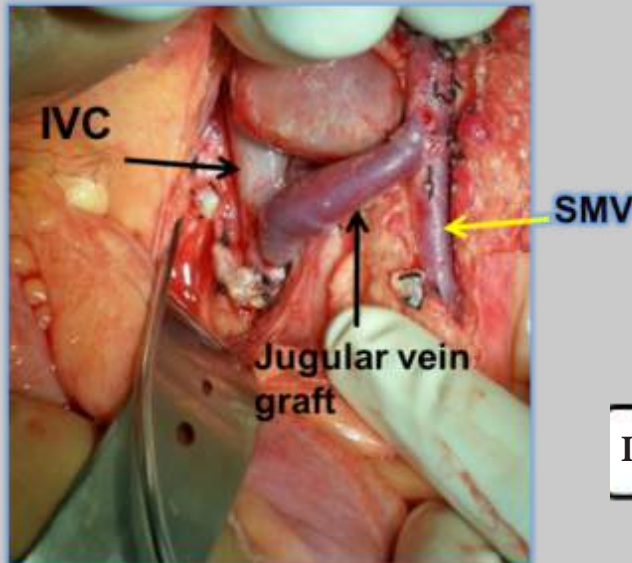


Fig.5: H-graft interposition mesocaval shunt

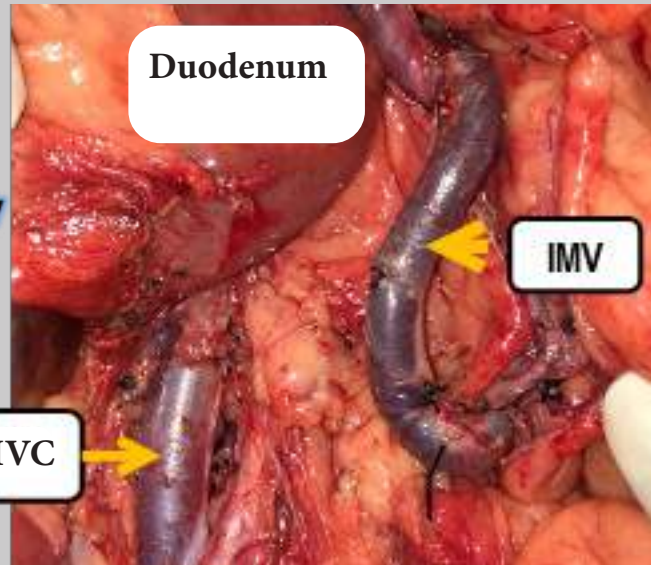


Fig.6: IMV & IVC dissected for a IMV to IVC shunt

A spleen preserving side to side splenorenal shunt (SSRS, n=4) and mesocaval shunt (n=1) (Fig.5) were opted for when issues of massive splenomegaly were absent. For difficult portal or systemic venous anatomy, various “unconventional shunts” were performed. When both splenic vein (SV) and superior mesenteric vein were non-shuntable, the inferior mesenteric vein offered a useful alternative because of easy localization to the left of duodenojejunal flexure, ease of mobilization because of few tributaries and ease of approximation to the left renal vein (LRV) or Inferior vena cava (n=5) 11 (Fig.6). Spleno-adrenal

(n=03) shunts came to rescue for an inadequate SV length or difficult LRV anatomy wherein the adrenal vein served as a robust conduit between the LRV and SV 12. Hence, a surgeon managing EHPVO needs to be equipped with a wide spectrum of procedures in his/her repertoire such that a given case receives the most appropriate procedure individualized to anatomy and surgical indication.

With a well-established high volume endotherapy program at our centre, an emergency surgery was necessitated infrequently (n=12, 9%) following failure of pharmacotherapy and endotherapy and/or variceal bleed from sites not amenable to

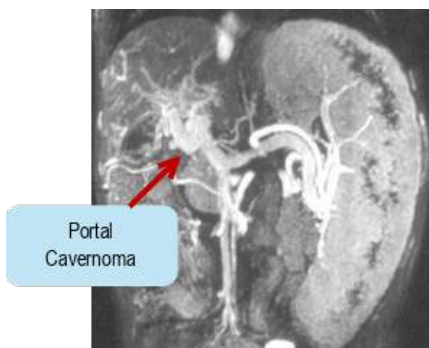


Fig.7a : EHPVO: Preoperative CT portovenogram



Fig. 7b: CT portovenogram showing a patent central splenorenal shunt

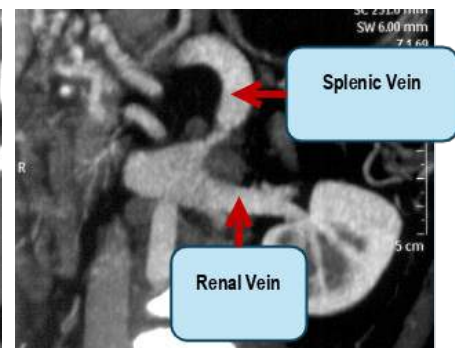


Fig. 7c: CT portovenogram showing a stenosed central splenorenal shunt

endotherapy namely large IGV (n= 9), duodenal varices (n= 1) and jejunal varices (n= 2).

Massive splenomegaly with severe hypersplenism and/or an unfavorable anatomy precluded a MRB for cases in this study. While MRB is theoretically the ideal physiology restoring curative procedure, it has limitations related to unfavorable extrahepatic and/or intrahepatic portal venous anatomy and need for repeated interventional radiological procedures to sustain patency in a large proportion of cases ^{13,14,15}. Further, MRB would not be the preferred procedure of choice when massive splenomegaly with severe hypersplenism is the predominant indication for surgery. Moreover, the left branch of portal vein becomes progressively hypoplastic with increasing age thereby lowering the technical feasibility and success rate of an MRB in a country like ours wherein cases often present late to a tertiary care centre.

The operative mortality in our series was zero. One out-of-hospital mortality was reported at 22 months post SSRS, consequent to jejunal variceal bleed ⁸.

Median follow up (FU) duration was 41 (range 6-228) months. Nineteen (14%) cases had a FU of ≥ 10 years, 59 (42%) cases had a FU ≥ 5 years and 91 (65%) cases had a FU of ≥ 2 years ⁸.

The protocol for post operative shunt patency assessment was a periodic doppler ultrasonography (USG) performed at following time intervals post PSS: day 7; 6 weeks; 6 monthly for the first 2 years and yearly doppler USG thereafter. Failure to visualize PSS on doppler USG indicated a CT portovenogram (Fig.7a, 7b & 7c). An upper GI endoscopy (UGIE) was performed at 6 months post-surgery to document variceal regression. No further UGIE was required for persistently patent PSS.

The other parameters recorded at each FU visit

were: rebleed, post splenectomy sepsis, height and weight z scores, clinical features of encephalopathy, hemogram and LFT. Hemogram and splenic size were surrogate indicators of shunt patency for spleen preserving shunts.

PSS block were detected in 13 cases (11%) on doppler USG and substantiated on CT portovenogram at following time intervals post PSS: day 7 (n=1), week 6 (n=2), 6 months (n=3), 12 months (n=2), 2 years (n=1), 4.5 years (n=1), 09 years (n=2) and 12 years (n=1) respectively. Delayed shunt blocks, therefore, emphasize upon the need for life long FU. Post shunt block sequelae have been recurrent GE variceal bleed warranting endotherapy (n=3); symptomatic portal colopathy (n=1) and progression of PCC related cholestasis (n=2) ⁸.

Significant downgrading of GE varices & ectopic varices was observed in all cases with persistently patent shunts.

For cases presenting with complications of PCC (n=7), persistently patent shunts (n=5) resulted in resolution of symptoms of cholestasis with normalization (n=2) or reduction (n=3) in GGT. However, in cases with early PSS thrombosis, symptomatic PCC related cholestasis persisted (n=2) thereby, necessitating continued endoscopic biliary drainage ⁸.

Issues of splenomegaly resolved in all with significantly improved growth Z scores.

A question in the minds of the readers could be : Should portal decompression surgery be advised to every case as a one-time definitive procedure following endoscopic control of first major bleed episode?

For evidence supported answer to the above question, there is a need for further studies with FU extending into adulthood following EEEV, wherein QOL implications of massive splenomegaly, GR,

PCC, “non- esophageal” varices and the cost of repeated endotherapy are taken into cognizance. Data on proportion of cases who develop adequate spontaneous porto-systemic collaterals for effective portal decompression without surgery, is warranted.

This study is especially relevant to the Indian context wherein overall volume of EHPVO cases is large but, appropriate expertise and infrastructure for surgical management of EHPVO is restricted to select tertiary care centres translating

into prolonged waiting lists for surgery. The management algorithm needs to be tailored to the case load vs the health infrastructural capacity.

Surgery and endotherapy will continue to be complementary in the management of EHPVO and surgical techniques for management of this specialized realm should not be relegated to a dying skill & science. It is imperative that the future generations of surgeons are mentored to hone their skills in this specialized area of surgery.

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Some surgeons build careers. A few build institutions, nurture generations, and leave behind a legacy that continues long after they have stepped away from the operating table. Dr. Richa Lal belongs to that rare group.

Trained entirely at the prestigious AIIMS, New Delhi, in MBBS, General Surgery and Pediatric Surgery, Dr. Lal arrived at SGPGIMS, Lucknow, in 2000 as its first pediatric surgeon. What awaited her was not merely a position—it was an opportunity to build something from the ground up.

For the next nineteen years, she devoted herself to creating and nurturing Pediatric Surgical Superspecialties at SGPGIMS, eventually serving as its Professor and Founder Head. Her particular contributions to complex pediatric gastrointestinal and hepatobiliary surgery—including choledochal cysts, biliary atresia and portal hypertension—brought hope to children and families confronting daunting illnesses.

Yet her legacy extends beyond surgical procedures and publications. It lives in the students she taught, the department she built, and the lives she touched.

Now also trained in Lifestyle Medicine, Dr. Lal's journey beautifully reminds us that medicine is ultimately not about treating disease alone—it is about nurturing life, creating institutions, and leaving the world healthier than we found it.



Dr. B K Aikat

Prof. B.K. Aikat (1916 - 1981)

MD, DCP, Ph.D (London)

Founder - Director, Department of Pathology, PGIMER, Chandigarh (1964 - 1978) Dean, PGIMER (1969 - 1977)

The Recognition of Non Cirrhotic Portal Fibrosis

Dr. B. K. Aikat's contribution to medicine grew from a deceptively simple question: what if severe portal hypertension did not always mean cirrhosis? By examining the liver as a pathologist and relating its structure to the patient's illness, he helped define noncirrhotic portal fibrosis as a distinct vascular disorder. The name has changed across regions and decades, but the central distinction he clarified remains part of international diagnostic practice.

The Clinical Puzzle

For many patients, the signs pointed in one direction. Pressure had risen within the portal venous system. The spleen was greatly enlarged, fragile veins could bleed into the digestive tract, and the danger was immediate. Cirrhosis seemed the obvious explanation because it commonly distorts the liver and obstructs portal blood flow.

Yet a substantial group of patients, particularly in India and other developing regions, did not fit that pattern. They had marked portal hypertension and serious bleeding, but their liver function was often preserved. Their hepatocytes and lobular architecture remained largely intact. The contradiction called for a diagnosis that separated

vascular injury from the diffuse scarring of cirrhosis.

The Foundations of a Research Career

Aikat's interest in this territory was already visible by 1963. That year, he and surgeon A. K. Basu published *Tropical Splenomegaly*, a substantial illustrated monograph that brought clinical and pathological observation together. Its subject placed him close to the unresolved relationship among enlarged spleens, portal pressure, blood flow and liver disease.

At the Postgraduate Institute of Medical Education and Research in Chandigarh, Aikat worked in an environment where clinical care, autopsy pathology and research could illuminate one another. This combination mattered. A liver biopsy offered only a narrow sample, while a carefully studied autopsy could reveal the distribution of vascular damage throughout the organ and along the larger portal branches.

The 1979 Autopsy Study

The decisive account appeared in 1979 as *The Pathology of Noncirrhotic Portal Fibrosis*, a review of thirty-two autopsy cases by Aikat and

his associates. Instead of treating the absence of cirrhosis as an odd exception, the investigators studied the vascular changes on their own terms. The series gave pathological form to a clinical syndrome that had too often been placed under the broad label of cirrhosis.

The main injury lay in the portal venous system. Small and medium intrahepatic portal vein branches showed narrowing, thickening, and fibrous obliteration, a pattern often described as obliterative portal venopathy or phlebosclerosis. In sixty-six percent of the autopsy cases, sclerosis and thrombosis were evident and extended into major portal vein branches. These changes offered a structural explanation for the increased resistance to portal blood flow.

Just as important was what the investigators did not find. The lobular framework and liver cells were comparatively well preserved rather than reorganized into cirrhotic nodules. This helped explain why patients with the disorder could retain useful liver function and tolerate major bleeding better than many patients with advanced cirrhosis. Aikat's work therefore changed the diagnostic question from whether portal hypertension was present to where the obstruction arose and what tissue pattern produced it.

A Disease with Several Names

As reports accumulated internationally, similar conditions acquired different names. Indian clinicians commonly used noncirrhotic portal fibrosis. Japanese literature favored idiopathic portal hypertension, while other accounts referred to hepatoportal sclerosis, obliterative portal venopathy, or nodular regenerative hyperplasia. Later, idiopathic noncirrhotic portal hypertension became a broad clinical term, and porto-sinusoidal vascular disease or disorder was proposed to gather related vascular and architectural abnormalities under a common framework.

The variety of names reflected genuine differences in case definitions as well as regional medical traditions. A 2018 review of idiopathic noncirrhotic portal hypertension continued to emphasize the pathological features that distinguish the condition from cirrhosis. The field had become international, but it still depended on the relationship between clinical evidence and the tissue changes that Aikat and other early investigators had described.

From Early Observation to Modern Consensus

In May 2026, specialists from major liver, pathology and pediatric hepatology societies published the first internationally endorsed, unified diagnostic framework for the disorder. The consensus permits the terms porto-sinusoidal vascular disorder and noncirrhotic portal fibrosis to be used interchangeably as PSVD or NCPF when the same criteria are applied.

The modern definition is broader than the cases available to earlier pathologists. It recognizes that PSVD or NCPF may be diagnosed even before clinical portal hypertension appears and may coexist with other liver diseases, provided cirrhosis is excluded. Diagnosis remains fundamentally clinicopathological. A good-quality liver biopsy is mandatory, and its findings must be integrated with clinical features and the exclusion of alternative causes.

This consensus does not freeze the field in its 1979 form. It confirms the durability of the distinction at the heart of Aikat's work: vascular disease within the liver can produce portal hypertension without cirrhosis. Modern terminology, imaging and scoring systems refine that insight, but they do not erase its origin in close observation of patients and tissue.

A Lasting Place in Pathology

Aikat's influence reaches beyond one paper. His work helped pathologists recognize a pattern that could otherwise be missed or mistaken for cirrhosis, and it helped clinicians understand why severe portal hypertension could coexist with preserved liver architecture and function. The distinction affected diagnosis, prognosis, and how such patients were discussed across specialties.

The Indian Association of Pathologists and Microbiologists preserves his name through the Dr. B. K. Aikat Memorial Oration. The honor is fitting because his achievement was both descriptive and interpretive. He did more than catalog lesions. He showed how the distribution of sclerosis, thrombosis and portal venous obliteration could explain a clinical syndrome. Nearly half a century

after the autopsy series, an international consensus still rests on the boundary he helped make visible.

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Proximal Splenorenal Shunt in Extra-hepatic Portal Vein Obstruction: Operative Technique and Technical Pearls

- Dr. Ajit Kumar Mishra

Introduction

Extra-hepatic portal vein obstruction (EHPVO) is an important cause of non-cirrhotic portal hypertension, particularly in children and young adults. Clinical manifestations are predominantly related to portal hypertension and include splenomegaly, hypersplenism, gastroesophageal variceal bleeding and, in a subset of patients, portal cavernoma cholangiopathy (portal biliopathy).

The proximal splenorenal shunt (PSRS) is a non-selective portosystemic shunt in which the spleen is removed and the splenic vein is anastomosed end-to-side to the left renal vein. The procedure

simultaneously addresses several major consequences of EHPVO: it decompresses the portal venous system, reduces the risk of recurrent variceal bleeding, and removes the enlarged spleen responsible for hypersplenism and mechanical symptoms.

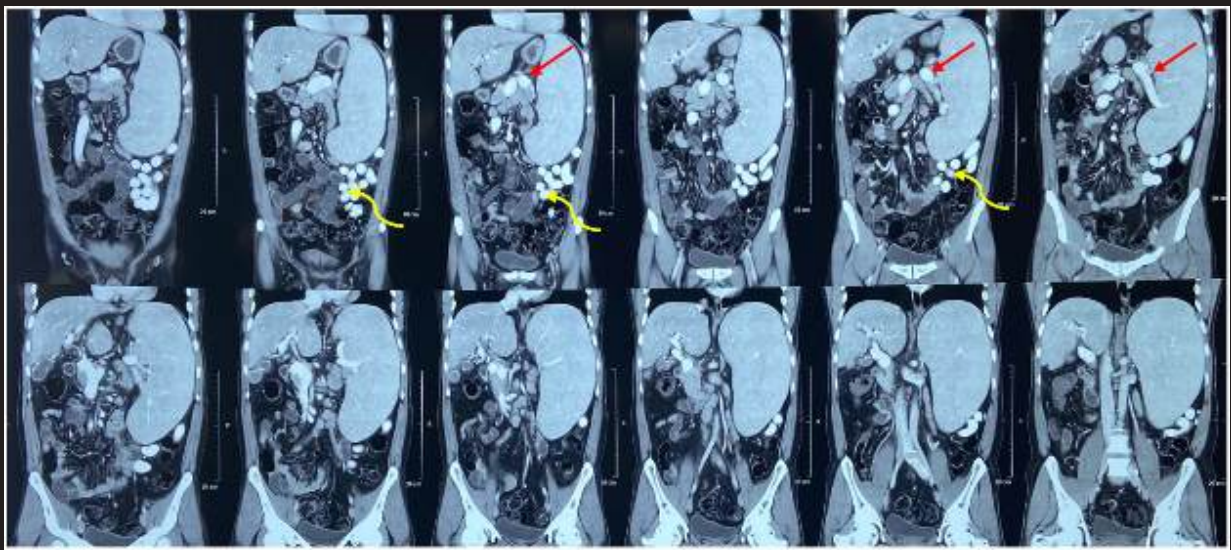
In patients with portal cavernoma cholangiopathy, portal decompression is generally the first therapeutic step when intervention is required. Persistent symptomatic biliary obstruction after adequate portal decompression may subsequently require endoscopic or surgical biliary intervention.

Patient Selection and Preoperative Evaluation

The fundamental anatomical prerequisite for PSRS is a patent splenic vein of adequate calibre and

length to reach the left renal vein without tension or significant angulation.

Although the splenic vein can often be assessed using ultrasonography with colour Doppler, high-quality contrast-enhanced CT is strongly



a. Red arrow is showing splenic vein
b. Yellow curved arrow is showing retroperitoneal collaterals

recommended for operative planning. The CT should be interpreted as a vascular roadmap.

- Patency, calibre and course of the splenic vein.
- Superior mesenteric vein patency and anatomy.
- Splenic vein–portal vein anatomy.
- Perisplenic and perigastric collaterals.
- Retroperitoneal collateral pathways.
- Splenic infarction or previous splenic injury.
- Splenic hilar anatomy.
- Pancreatic anatomy and relationship to the splenic vein.
- Left renal vein anatomy and venous tributaries.
- Splenic artery aneurysm or other vascular abnormalities.

Preoperative CT interpretation is not merely diagnostic; it provides a roadmap for safe dissection. In EHPVO, large spontaneous portosystemic collaterals may be encountered in unexpected locations, and anticipating their location can significantly reduce intraoperative blood loss.

Preoperative Vaccination

- Because PSRS involves splenectomy, appropriate vaccination against encapsulated organisms is an essential component of preoperative preparation.
- For elective splenectomy, vaccination should ideally be completed at least 14 days before surgery, when feasible. Vaccination requirements should be individualized according to the patient's age, previous vaccination history and current national immunization recommendations. Important vaccines include those against:
 - *Streptococcus pneumoniae*
 - *Neisseria meningitidis*
 - *Haemophilus influenzae* Type B

Operative Technique

Position

The patient is placed supine with both arms tucked alongside the body. A small pillow beneath the left flank or kidney region can facilitate exposure of the left upper quadrant.

Incision

An L-shaped incision can provide excellent exposure. The vertical component begins near the xiphisternum and continues inferiorly before curving laterally approximately 4–5 cm cranial to the umbilicus toward the left anterior axillary line. A thoracoabdominal incision has traditionally been used by some centres for extensive exposure. However, an L-shaped abdominal incision can provide adequate exposure in most patients while avoiding a thoracic incision and potentially reducing postoperative pain and pulmonary morbidity.

Initial Exploration and Retractor Placement

After entering the peritoneal cavity, the abdomen is systematically explored.

A subcostal retractor is then positioned to provide adequate exposure of the spleen and upper abdomen.

Technical Pearl—Before applying significant traction with the subcostal retractor, carefully inspect the area between the spleen and the abdominal wall.

In patients with EHPVO, large collateral veins may run between the splenic capsule and parietal peritoneum. Blind placement or aggressive opening of the retractor can avulse these vessels and result in significant bleeding at the very



L Shaped Incision

beginning of the operation. The degree of splenic and perisplenic adhesion should also be assessed at this stage.

Entering the Lesser Sac

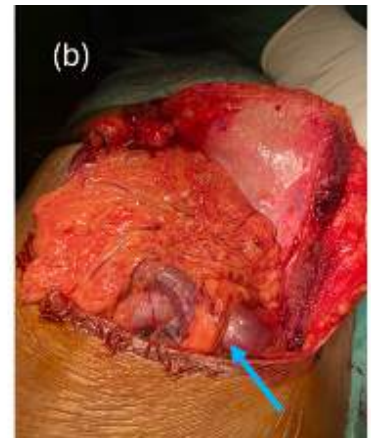
The lesser sac is entered by opening the gastrocolic ligament.

In patients with EHPVO, the omental vessels may be markedly dilated because of portal hypertension. The dissection is continued toward the gastrosplenic region. The short gastric vessels are sequentially identified, ligated and divided, allowing the stomach to be mobilized and subsequently reflected toward the liver. Care should be taken to avoid unnecessary traction on the stomach and spleen during this stage.

Technical pearl-Dilated omental vessels should preferably be individually ligated rather than divided blindly with an energy device



(a) Assessment of Peri- splenic adhesion



(b) Blue arrow indicating large venous collaterals

Portal Pressure Measurement

An accessible omental or mesenteric vein can be cannulated and connected to a pressure transducer referenced to the appropriate zero level. The same technique can subsequently be used to measure portal pressure after completion of the shunt, allowing objective assessment of portal decompression.

Dense adhesions may occasionally be encountered between the superior pole of the spleen, stomach and diaphragm. This area deserves particular caution. Aggressive traction on dense adhesions at the superior pole may result in avulsion of large collateral vessels and torrential bleeding.

Technical pearl - If the superior pole is difficult to dissect safely, do not force the dissection at this

stage. It is often safer to proceed with mobilization of the spleen from the inferior and lateral aspects first. Once the spleen has been mobilized from below and laterally, the superior pole can usually be approached under much better control.



Control of the Splenic Artery

The splenic artery is usually identified along the superior border of the pancreas by its characteristic pulsation. A lymph node may overlie the artery. Careful dissection and removal of this node can expose the artery. The splenic artery is dissected sufficiently to allow secure control and is then doubly ligated and divided. Following ligation, the spleen gradually becomes smaller and areas of ischemia become apparent on its surface. Early control of the splenic artery reduces splenic blood flow and facilitates subsequent mobilization

Important technical point-

At this stage, there is usually no need to dissect or isolate the splenic vein.

Splenic Mobilization

Splenic mobilization is then performed from the abdominal wall and retroperitoneum. The degree of adhesion varies considerably among patients with EHPVO. Some patients have surprisingly minimal adhesions, whereas others have dense vascular adhesions related to previous splenic infarction, inflammation or previous interventions

for variceal bleeding.

A useful starting point is the inferior pole of the spleen. The inferior polar vessels are individually identified, ligated and divided. The dissection is then continued progressively into the splenorenal space.

Fundamental principle- Stay as close to the splenic capsule as possible.

Most small vessels immediately adjacent to the splenic capsule are relatively thin and can be safely controlled by energy devices. In contrast, entering



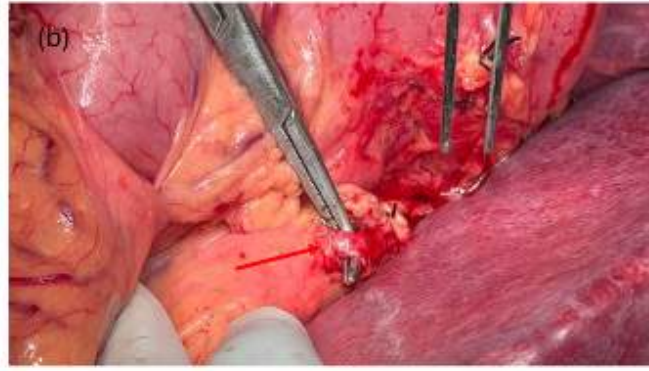
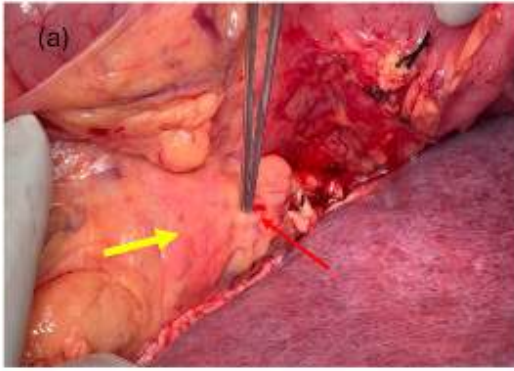
Opening Of Lesser Sac

deeper retroperitoneal planes may expose large spontaneous collaterals. This is where preoperative CT interpretation becomes particularly valuable.

Large retroperitoneal collaterals identified on CT should be anticipated and avoided whenever possible.

If a major collateral is inadvertently entered, immediate proximal and distal control followed by fine vascular suturing may be required.

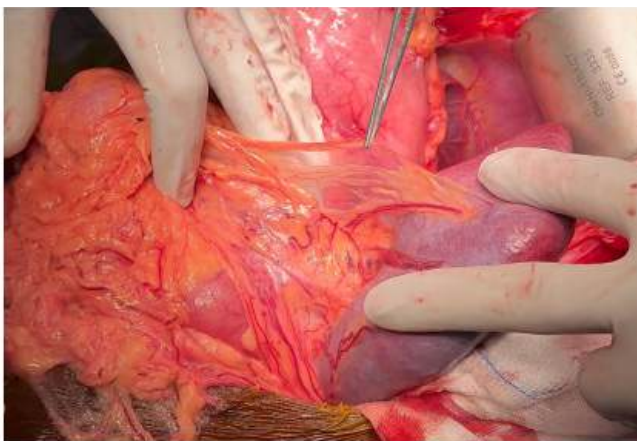
Technical pearl- Do not chase large retroperitoneal collaterals simply because they are visible. The objective is splenic mobilization, not complete skeletonization of the retroperitoneum.



a. Identification of splenic artery at superior border of pancreas
(Yellow arrow showing pancreas ,red arrow indicating splenic artery)
b. Looping of Splenic artery

Completion of Splenic Mobilization

Mobilization is continued circumferentially until the superior pole is reached. The splenic hilum is intentionally left for the final stage. If dense adhesions are present between the stomach and superior splenic pole—particularly in patients who have previously undergone endoscopic glue treatment for variceal bleeding—these adhesions can now be safely addressed after the spleen has been mobilized from the remaining surfaces. In extremely dense adhesions where safe separation is not possible, a small adherent portion of stomach may occasionally be resected en bloc with the spleen using a linear stapler, provided this is technically appropriate.



Splenic Mobilization

Splenic Hilar Dissection

Once fully mobilized, the spleen is elevated. Hilar dissection proceeds preferably from inferior to superior, remaining close to the splenic capsule. Pancreatic attachments are carefully separated. Once the splenic vein is identified, additional length can be obtained by dividing suitable splenic venous branches (Polar branches) rather than dividing the main splenic vein. After the vascular attachments have been controlled, the spleen is removed.

Assessment of the Splenic Bed

The splenic bed is carefully inspected. Bleeding from retroperitoneal, diaphragmatic or pancreatic surfaces is controlled.

Splenic Vein Mobilization

Adequate mobilization of the splenic vein is critical for a tension-free splenorenal anastomosis.

The pancreas is mobilized sufficiently from its retroperitoneal and mesocolic attachments to expose the posterior surface of the pancreatic body and tail.

The splenic vein is then progressively mobilized from the posterior pancreatic surface. Multiple

small pancreatic tributaries enter the splenic vein. These vessels can be fragile, and even minor traction on the splenic vein may avulse a tributary and rapidly obscure the operative field.

Technical pearl—The splenic vein should be handled gently, with controlled division of individual pancreatic tributaries. Fine clips may be used for small tributaries where appropriate.

The objective is to obtain sufficient length of splenic vein so that the final anastomosis reaches the left renal vein without tension, excessive curvature or kinking.

Left Renal Vein Exposure

Attention is then directed toward the left renal vein.

The transverse colon is retracted inferiorly and the small bowel is displaced toward the right side. The left renal vein is located at the level of the duodenojejunal flexure and can usually be identified by first palpating the left renal artery and using its position as an anatomical landmark. The retroperitoneal tissue overlying the anterior surface of the renal vein is carefully dissected. The gonadal vein is usually encountered inferiorly and the left adrenal vein superiorly as they join the renal vein. Only the amount of renal vein required for a safe anastomosis should be exposed.

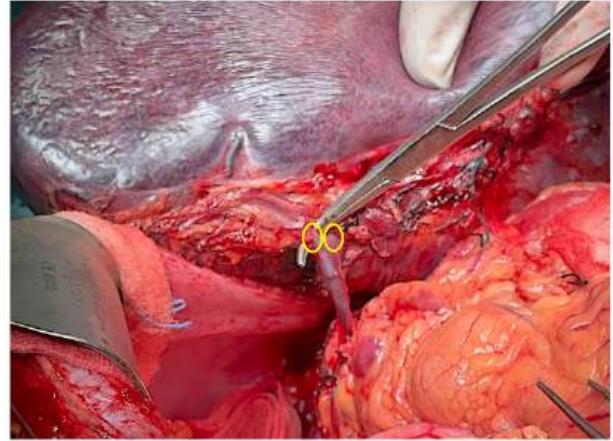
Technical pearl: preserve spontaneous collaterals

The retroperitoneum in EHPVO contains an extensive network of spontaneous portosystemic collaterals. These veins are part of the patient's decompressive circulation. Avoid unnecessary ligation of these vessels. Only collaterals that directly interfere with exposure or the planned anastomosis should be controlled.

The left retroperitoneum contains a rich lymphatic

network. Extensive dissection and indiscriminate use of energy devices may result in postoperative lymphatic leakage or chylous ascites.

Meticulous tissue handling, selective ligation of larger lymphatic channels and judicious use of energy devices can reduce this risk.



Division of splenic vein at the level of its branching

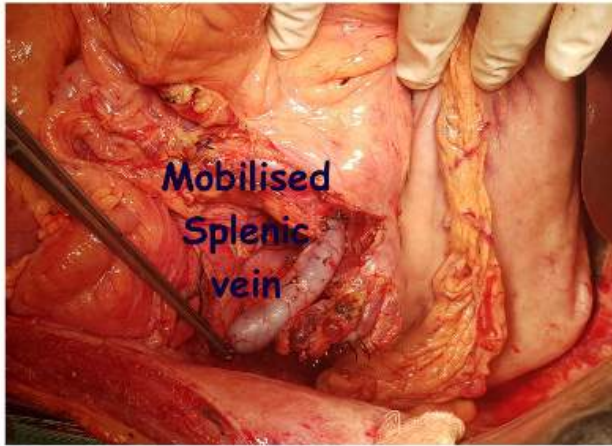
Preparation before Splenorenal Anastomosis

Once an adequate segment of the left renal vein has been exposed, the proposed site of anastomosis is assessed.

A Satinsky side-biting vascular clamp can be temporarily applied to ensure that adequate renal venous lumen remains available for the planned venotomy.

The splenic vein is then brought inferiorly below the pancreatic tail toward the renal vein. Before proceeding, the surgeon should confirm:

- Adequate splenic vein length
- No tension
- No twisting
- No sharp angulation
- A smooth, gentle curvature between the splenic vein and renal



vein The ideal configuration is a **gentle curve rather than a sharp right-angle turn**. Excess splenic vein length can be trimmed after appropriate vascular control.

Systemic heparinization may be performed according to institutional vascular-surgery protocol. After allowing adequate circulation time, the left renal vein is controlled with a vascular clamp. A bulldog clamp is applied to the proximal splenic vein.



Left Renal Vein Exposed

The splenic vein should be protected from stasis and thrombosis during this period, and the lumen

may be flushed with heparinized saline before completing the anastomosis.

An appropriately sized longitudinal venotomy is made on the anterior surface of the left renal vein. The size of the venotomy should be tailored to the calibre of the splenic vein.

A practical approach is to make the venotomy slightly longer than the diameter of the splenic vein, often approximately 1.5 times its diameter, while avoiding an unnecessarily large venotomy.

Both venous lumens are flushed with heparinized saline.

Splenorenal Anastomosis

A continuous end-to-side venous anastomosis is performed using fine monofilament polypropylene, commonly 6-0 Prolene.

Double-armed sutures are placed at both corners using an inside-to-outside technique and left on traction.

Technical pearl -An additional traction suture may be placed at the anterior wall of the venotomy to improve visualization of the posterior wall.

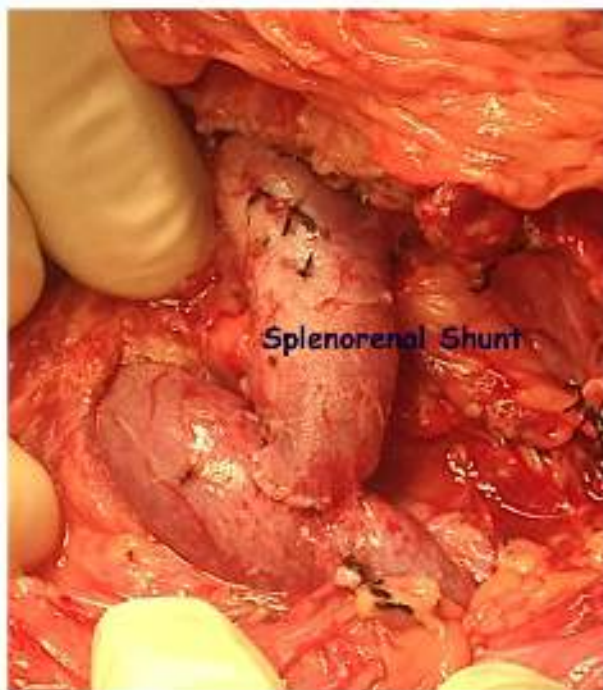
One corner suture is then used to complete the posterior wall with a continuous technique.

After reaching the opposite corner, the same suture is continued along the anterior wall toward the midpoint.

The second suture completes the remaining portion of the anterior wall.

Before the final knots are secured, the venous lumens are flushed with heparinized saline to remove thrombus and air.

The two sutures are tied at the midpoint leaving a growth factor



The left renal vein clamp is then released, followed by release of the splenic vein clamp. The anastomosis is inspected carefully for:

- Immediate flow
- Bleeding
- Kinking
- Tension
- Narrowing at the anastomotic site

A small amount of oozing from the needle holes is common immediately after unclamping.

Technical pearl- Avoid placing multiple unnecessary haemostatic sutures into the anastomosis. A small piece of gauze can be gently placed over the anastomotic line for several minutes to allow needle-hole bleeding to settle. Persistent focal bleeding can then be controlled with a fine interrupted suture.

Post-Shunt Portal Pressure

If baseline portal pressure was measured, the

portal pressure can be measured again after completion of the shunt.

The fall in portal pressure provides an objective assessment of decompression.

Final Haemostasis and Drain Placement

The entire operative field should be re-examined before closure. Particular attention should be paid to:

- Splenic bed
- Pancreatic surface
- Retroperitoneum
- Subdiaphragmatic region
- Splenorenal anastomosis
- Sites of previous collateral bleeding

A drain may be placed in the splenic bed according to the surgeon's preference and intraoperative findings.

The abdominal wall is then closed in layers.

Key Technical Pearls

1. Study the preoperative CECT personally.

The CT should be regarded as an operative vascular roadmap rather than merely a diagnostic study.

2. Anticipate collaterals before opening the upper abdomen.

Avulsion of a large perisplenic collateral during retractor placement can result in significant bleeding.

3. Control the splenic artery early.

Arterial ligation reduces splenic congestion and facilitates subsequent mobilization.

4. Stay close to the splenic capsule during splenic mobilization.

5. Do not chase deep retroperitoneal collaterals.

They are often part of the patient's decompressive portal circulation.

6. Leave difficult superior-pole adhesions until later.

Mobilization from the inferior and lateral aspects usually makes the final dissection safer.

7. Keep hilar dissection for the end.**8. Preserve splenic vein length.**

Divide suitable branches rather than sacrificing the main splenic vein.

9. Handle the splenic vein gently.

Pancreatic tributaries can be fragile and difficult to control once avulsed.

10. Expose only the amount of left renal vein required for the anastomosis.**11. Preserve unnecessary retroperitoneal collaterals.****12. Avoid excessive energy use in the left retroperitoneum.**

Lymphatic injury can result in postoperative chyle leak.

13. A tension-free, non-kinked anastomosis is the goal.

The splenic vein should reach the renal vein in a gentle curve rather than an acute angle.

14. Do not oversize the venotomy.

Tailor it to the calibre of the splenic vein.

15. Do not over-suture the anastomosis.

Minor needle-hole bleeding generally settles after reperfusion and gentle compression.

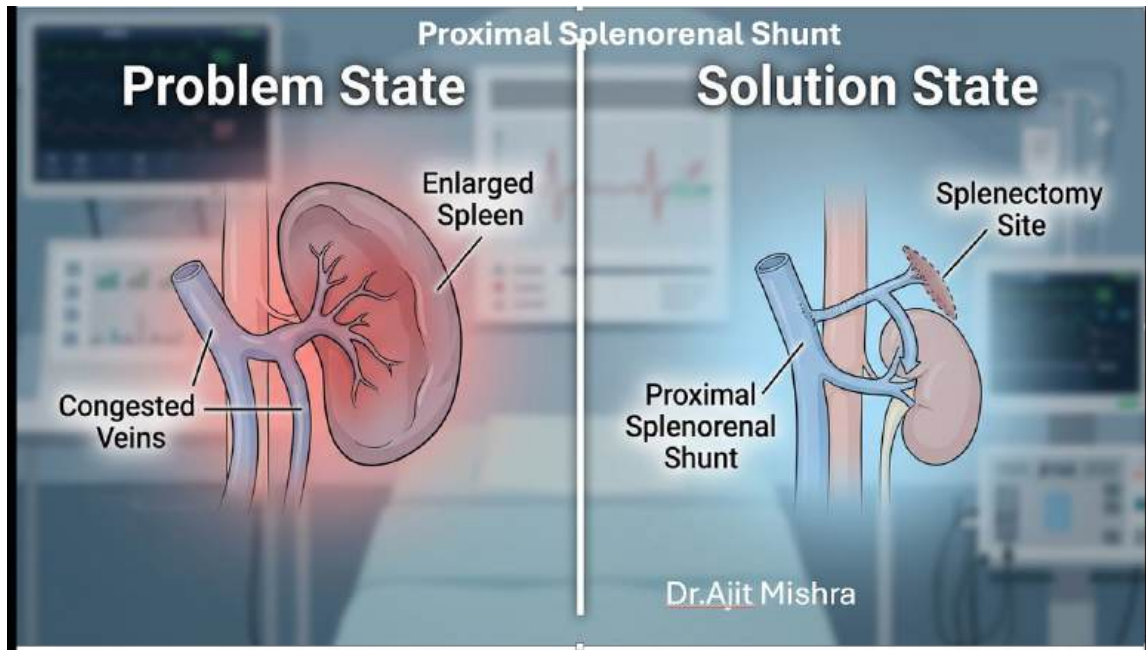
| Conclusion

Proximal splenorenal shunt remains an important surgical procedure for selected patients with EHPVO and clinically significant portal hypertension. Its major advantages are simultaneous portal decompression and splenectomy, thereby addressing recurrent variceal bleeding, hypersplenism and symptomatic splenomegaly.

The success of PSRS depends not only on constructing a technically sound venous anastomosis but also on careful preoperative vascular mapping, anticipation of spontaneous collaterals, meticulous splenic mobilization, preservation of splenic vein length and creation of a tension-free, non-kinked splenorenal anastomosis.

In patients with portal cavernoma cholangiopathy, adequate portal decompression should generally precede definitive biliary bypass or reconstruction when both are required. A substantial proportion of patients experience improvement after shunt surgery alone, while persistent biliary obstruction after adequate decompression may require subsequent biliary intervention.

The operation is therefore best viewed not simply as a splenectomy combined with a venous anastomosis, but as a planned decompressive reconstruction of the portal venous circulation, where knowledge of the patient's collateral anatomy is as important as the technical execution of the shunt itself.



Scan to watch operative video



Dr. Ajit Kumar Mishra
 MBBS, MS, M.Ch (Surgical
 Gastroenterology and Liver Transplant)
 Gastro Intestinal Surgeon
 Artemis Hospital Raipur



Great surgeons do more than master difficult operations—they create possibilities where few existed before. Dr. Ajit Kumar Mishra's journey reflects that spirit.

As Chhattisgarh's first M.Ch. Gastro and Liver Surgeon, Dr. Mishra has played an important role in bringing advanced gastrointestinal, hepatobiliary, pancreatic and liver transplant surgery to Central India. Over the years, he has managed more than 6,000 complex surgical cases, while contributing to the development of liver transplantation services within the state.

Trained at the renowned SGPGIMS, Lucknow, his surgical interests span complex liver and pancreatic surgery, GI oncology, minimally invasive surgery and transplantation.

Today, as Head of GI, HPB and Liver Transplant Surgery at Artemis Hospitals, Raipur, Dr. Mishra represents something larger than individual surgical excellence—the decentralisation of sophisticated healthcare.

His journey reminds us that progress in medicine truly matters when world-class expertise reaches patients closer to their own homes.



Dr. Rajaram Bal Bhalerao

A Life in Surgery and Theatre

Dr. Rajaram Amrut Bhalerao, known to generations of colleagues and theatre lovers as Bal Bhalerao, lived at the meeting point of two demanding worlds. In medicine, he became a distinguished surgeon, teacher, hospital leader, and researcher. In Marathi theatre, he sustained an institution founded by his father, brought classical and modern work to new audiences, and opened doors for younger artists. His life was not divided between these callings. The same discipline shaped both: careful work, faith in institutions, and a willingness to serve beyond the spotlight.

A Childhood Among the Stage

Rajaram was born in 1933, two years before his father, Dr. A. N. Bhalerao, founded the Mumbai Marathi Sahitya Sangh in Girgaum. The new organization answered an urgent cultural need. Marathi theatre was struggling after the arrival of talking pictures, and the Sangh set out to restore audiences' confidence in live performance. From his earliest years, Bal grew up close to that effort and to the artists, writers and organizers who kept it alive.

He first acted on stage in 1942. Although medicine would become his profession, theatre remained a constant presence. He learned early that a stage survives through more than performers: it needs rehearsal rooms, administrators, audiences, teachers and people prepared to do the patient work of building an institution.

The Making of a Surgeon

His medical education took him to Seth G. S. Medical College and King Edward Memorial Hospital in Mumbai. The University of Bombay awarded him a

distinction and gold medal in Anatomy in 1952 and another in Surgery in 1956. He completed his MS in General Surgery and later traveled to Great Britain on a Commonwealth Fellowship. In October 1963, he received the Fellowship of the Royal College of Surgeons of Edinburgh.

Those years also marked an important moment for the Sangh. In 1964, it opened its own auditorium at Kelewadi in Girgaum, designed on the model of London's Old Vic Theatre. The building gave Marathi theatre a permanent home just as Bal was preparing to establish his surgical career in India.

In 1965, he began practicing surgery at KEM Hospital. He remained there for two decades, rising to become Professor and Head of the Department of Surgery. His clinical interests included liver trauma and resection, venous shunts for portal hypertension, and esophageal replacement. He published more than sixty papers in national and international journals and became known for his work on wound infection and operating theatre sterilization.

A Steward of Marathi Theatre

While his medical responsibilities grew, so did his role in the Mumbai Marathi Sahitya Sangh. He eventually became its Chief Secretary and guided it for more than half a century. His contribution was chiefly that of a steward: he helped the organization choose ambitious work, assemble the people needed to stage it, and create the conditions for Marathi theatre to endure.

The Sangh revived popular Sangeet Nataks and returned older Marathi plays to the stage, but it also looked outward. Its productions included *Ammaldar*, associated with P. L. Deshpande and based on Nikolai Gogol's *The Inspector General*; *Rajmukut*, Kusumagraj's Marathi adaptation of Shakespeare's *Macbeth*; works connected with Honaji Bala; and a stage adaptation of Sant Gora Kumbhar.

A defining achievement came in 1974. Bal Bhalerao stewarded the troupe that presented C. T. Khanolkar's *Ajab Nyay Vartulacha*, the Marathi adaptation of Bertolt Brecht's *The Caucasian Chalk Circle*. Directed by Vijaya Mehta in collaboration with Fritz Bennewitz, it was performed at festivals in East Berlin and Zurich and was described as the first play in an Indian language to be staged outside India. The production showed how a firmly rooted Marathi institution could participate in an international theatrical conversation.

Leadership in Medicine and Culture

In 1983, he received the Dr. R. K. Menda Oration. Two years later, after completing his long tenure at KEM Hospital, he joined P. D. Hinduja Hospital and Medical Research Center as Chief of Surgery, specializing in gastroenterology and liver disease. He later served as Director of Professional Services until 2001 and subsequently advised the hospital's trustees on strategy and medical planning.

Recognition followed his medical service. In

2001, he received the Dr. B. C. Roy Award, one of the country's notable honors for physicians. His cultural standing was equally unusual. He served on literature and theatre committees appointed by the Government of Maharashtra and was chosen to preside over the All India Marathi Natya Sammelan. For someone known primarily as a physician and organizer rather than a playwright or star performer, that honor reflected the theatre community's respect for his behind-the-scenes work.

Under his guidance, the Mumbai Marathi Sahitya Sangh continued to connect preservation with education. It organized seminars and workshops, including a ten-day workshop led by Pandit Satyadev Dubey in June 2006. Through its wider associations, it also helped Marathi literature and drama reach audiences in the United States, Dubai and Singapore.

A Home for the Next Generation

In 2013, Bal Bhalerao made one of his clearest investments in the future. He provided space in the Sangh's building to Drama School Mumbai, rent-free for its first year. The gesture was practical rather than ceremonial. A new generation needed sustained training, and an established cultural institution had room to help. By placing education inside the home his father's organization had built, he joined the Sangh's history to the future of the stage.

The Legacy Remembered

Dr. Rajaram Bal Bhalerao died on 21 February 2020. He left behind an uncommon record: a surgeon who advanced clinical practice and medical education, and a theatre patron who gave Marathi performance an institutional home, international reach and a path to renewal.

On 11 April 2026, writers and theatre lovers gathered

before his bust to remember more than eighty-five years of the Bhalerao family's contribution. Madhu Mangesh Karnik and Vishwas Patil were among those present. Rare photographs carried the gathering back through the Sangh's history, including an image of Durga Khote as Anandibai in Bhaubandaki. The tribute made visible what Bal Bhalerao had spent his life protecting: a living

memory passed from one generation to the next.

His achievements in surgery and theatre took different forms, but they rested on the same conviction. Knowledge must be taught, institutions must be tended, and a tradition survives only when someone prepares a place for those who come after.



Honouring Dr. Bal Bhalerao – the good doctor who straddled the world of medicine and theatre.

HOT OFF THE PRESS

Upfront Hepaticojejunostomy in Portal Biliopathy - Our experience at SGRH

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Conflict of Interest - None to Declare; Source of
Funding – None

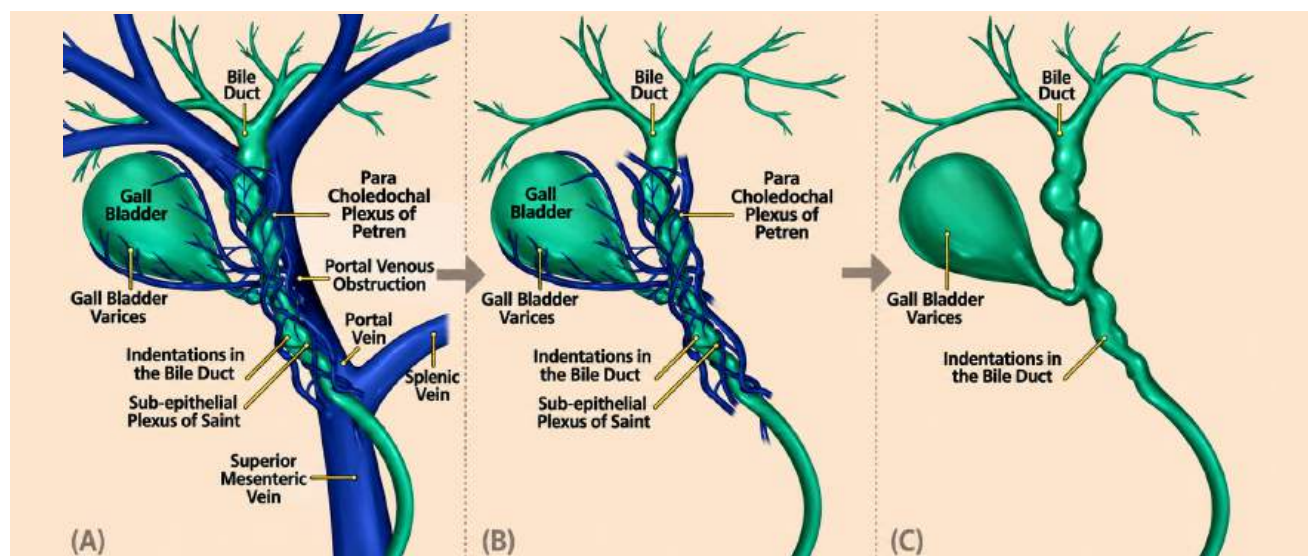
Article information

Upfront hepaticojejunostomy in portal biliopathy:
is a decompressive shunt always necessary?

Annals of Medicine & Surgery 87(12):p
7975-7983, December 2025. | DOI: 10.1097/
MS9.0000000000003923

What is known about the portal biliopathy surgeries

Portal Biliopathy (PB) is the result of biliary
obstruction by the dilated collaterals and is seen
in patients with Extra Hepatic Portal Venous
Obstruction (EHPVO). The thrombosed/blocked
portal vein in EHPVO is replaced by a portal
'cavernoma'. **(Figure 1)**



Biliary changes in portal biliopathy. (A) all three components of biliopathy: portal venous obstruction, cavernoma and resultant changes in the biliary tree. (B) cavernoma with subsequent changes in the biliary tree. (C) only biliary changes – indentations in the bile duct.[1]

These changes are also found in other conditions also, like Non Cirrhotic Portal Hypertension (NCPF) and cirrhosis but are more frequent and pronounced in EHPVO. It may be secondary to the 1) Cirrhosis: resistance to the flow, stasis and subsequent thrombosis in portal vein, 2) Severe local inflammatory conditions like omphalitis, pancreatitis and appendicitis, 3) Trauma due to operative injury to portal vein or physical trauma, 4) Malignant lesions of bile ducts, liver and pancreas and 5) Idiopathic.

The incidence of symptomatic PB is 5-33% in patients of EHPVO.^[2] The luminal changes in the biliary system lead to stasis, which in the long term become converted into ischaemic strictures which are often multifocal. The approximate duration from the diagnosis of EHPVO to PB is reported to be from 8 to 14.5 years.^[3] The complications in PB are related to stones in the biliary system at different locations, strictures: both intra and extra hepatic, and in untreated cases, slowly leading to hepatic parenchymal fibrosis and cirrhosis.^[4] Ascites, low albumin levels and high prothrombin times may indicate the development of advanced liver disease.

It is difficult to achieve resolution from biliary obstruction in complicated cases especially in patients with refractory strictures, intrahepatic lithiasis and ruptured intra hepatic dilated biliary ducts leading to collections. The treatment of portal biliopathy is according to the stage of presentation ranging from UDCA (ursodeoxycholic acid) to biliary stenting and rarely surgery in refractory cases.

The surgical treatment of PB include portal decompression procedures especially splenectomy with lienorenal shunts, devascularization and biliary bypass (intra or extrahepatic). Intrahepatic bypass has a very high incidence of re-stricture rate and the experience of this procedure is also limited.

^[5] Shunts provide greater portal decompression

than devascularization. The biliary bypass is usually considered to be a secondary procedure after a prior portocaval shunt to achieve the lowering of the raised pressure in the periportal veins. An upfront hepaticojejunostomy(HJ) without a prior decompressive shunt is associated with high chances of massive bleeding and hypoperfusion of the liver . In some patients with EHPVO however, who present with dominant biliary obstruction and its complications like ruptured intra hepatic biliary radicles, an upfront HJ is an alternative but difficult surgical option.

Methodology

Study design – Retrospective cohort study with prospectively maintained data

Study period – October 2008 to March 2022

Study group – 20 Adult patients requiring hepaticojejunostomy for portal biliopathy with biliary obstruction and one or more of its complications, like biliary strictures with stones, repeated attacks of cholangitis refractory to endoscopic management, cholangiolar abscesses, and/or ruptured intrahepatic biliary radicles (IHBR). Over the same period, 99 patients of EHPVO were operated for splenectomy with lienorenal shunt. Of these, 22 had biliopathy at the time of primary presentation, and in 11 patients, the biliopathy reversed with shunt only and the rest 11 persisted with biliopathy and formed the group A. These patients were compared in two groups -

Group A – HJ after a prior decompressive shunt (11 patients)

Group B – HJ upfront (9 patients)

They were followed up at 3-monthly intervals for 1 year and then every 6 months with LFTs and USG abdomen.

Primary endpoints

- **Feasibility of surgery - successful completion of the biliary procedure**
- **Long-term outcomes in the form of relief of biliary obstruction**

No attack of cholangitis in the follow-up period

No anastomotic narrowing at the HJ site

Secondary endpoints

- *Blood transfusion requirement*
- *Duration of surgery*
- *Complications - Clavien–Dindo grade III and above*

Result

The incidence of symptomatic PB was 22%. The causes were idiopathic in 75%, previous biliary surgery in 20% and pancreatitis in 5% cases. The prior portal decompression surgery was splenectomy with lienorenal shunt in 91% cases and devascularization in 9% cases. The surgery was successful in 9 out of 11 cases in prior decompression group and 8 out of 9 in upfront group. Out of these three unsuccessful cases, 2 died in each group in the post-operative period (one in group A died due to excessive blood loss and liver ischemia and other in group B due to leak and persistent sepsis) and the third one in prior decompression group was successfully redone over a PTBD stents after 3 months. Two patients in prior decompression group and one in upfront HJ group had partial relief from obstruction and needed repeated course of antibiotics in follow up. One patient from each group had intrahepatic stricture proximal to HJ however, no anastomotic stricture was noted.

Duration and blood loss was less in the upfront group due to virgin abdomen and fewer vascular adhesions. Four patients out of 11 in prior

decompression group (36.4%) and 3 out of 9 patients in upfront group (33.3%) noticed complications of Clavien–Dindo grade III and above.

Commentary

Surgery, in the form of portal decompression, devascularization and biliary bypass, is the mainstay of treatment in cases of portal biliopathy who have complications like stones, strictures, cholangiolar abscesses and ruptured intra hepatic biliary radicles, and are refractory to endoscopic treatment. Repeated stenting and recurrent cholangitis are the primary factors responsible for refractory ischemic biliary strictures. These patients should be free from cholangitis before any surgical intervention and preferably be stented, because during surgery, these stents helps localizing the bile duct in the portal area full of collaterals. In cases where ERCP stenting is not feasible, PTBD and antegrade stenting should be considered. Portal decompression by splenectomy with lienorenal shunt has long been advocated and considered as a preferred surgical procedure for PB as it relieves biliary obstruction in about 64% patients.[6] Proponents of portosystemic shunting prefer it over biliary bypass because of its dual benefit: reduction in the risk of variceal bleeding and relief from biliary obstruction. The interesting observation in the study was that the patient in prior portal decompression group progressed to further advancement of portal biliopathy despite the patent lienorenal shunt. This was probably due to development of ischemic strictures.

Devascularization reduces porto-systemic flow but the degree of reduction in portal pressure is much low in comparison to portocaval shunts. However, in specific situations with diffuse porto mesenteric thrombosis, shunts cannot be done and upfront HJ has a role. Upfront biliary bypass with hepaticojejunostomy is a difficult and challenging procedure due to the risk of massive bleeding from engorged collaterals and the subsequent ischemia

to the liver. Previous literature had quoted a success rate of upto 85% with upfront biliary bypass.^[4,7] The present study also had a feasibility of upfront HJ in 89% cases. The immediate post-operative complications and long term biliary stricture rates were comparable.

Another observation was combining surgeries in these patients where portal and biliary decompression were done together. Four patients in upfront group had portal and biliary decompression together (three had devascularization with HJ and one had lienorenal shunt with HJ) and all the surgical attempts were successful.

Implications for Practice

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Take home message

In select patients of EHPVO with PB, Upfront HJ is a feasible surgery with acceptable post-operative complication risk and long term stricture rate.

shunt before hepaticojejunostomy necessary? *Ann R Coll Surg Engl*. 2003 Sep;85(5):317–20. doi:10.1308/003588403769162422 PubMed PMID: 14594535; PubMed Central PMCID: PMC1964328.

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Decoding the Deceptive Spectrum of Non-cirrhotic Portal Hypertension (NCPH)

Non-cirrhotic portal hypertension (NCPH) includes non-cirrhotic portal fibrosis (NCPF) and extrahepatic portal venous obstruction (EHPVO). Though considered benign, recent observations suggest that a subgroup of patients may eventually develop chronic liver disease (CLD). Besides, differentiating non-cirrhotic portal fibrosis (NCPF) from extrahepatic portal venous obstruction (EHPVO) at times can be challenging particularly in the presence of portal vein thrombosis and portal cavernoma. We present a challenging case to underline the significance of a good clinical acumen.

Case Profile 1: EHPVO or NCPF

A 28-year-old male was referred to us from another institute for management of portal biliopathy. He had H/O hematemesis at 11 years of age was diagnosed as EHPVO and treated with endoscopic sclerotherapy. No other details available. His presenting complaint to us was progressive jaundice without cholangitis with two episodes of hematemesis in recent past managed with EVL.

His total bilirubin was 4.9 mg/dl with direct bilirubin of 3.4 mg/dl.

CECT Scan done revealed moderate splenomegaly with dilated CBD (15 mm), multiple CBD calculi, but normal portal vein with Portal vein diameter: 10 mm, Splenic vein diameter: 16 mm and no ascites.

Upper GI endoscopy showed presence GOV2 and also Portal hypertensive gastropathy (PHG).

ERCP was also done which revealed the findings of dilated CBD with multiple stones for which Endoscopic sphincterotomy was done with an unsuccessful attempt to achieve a complete ductal clearance. A Plastic biliary stent was placed.

MRI with MRCP (Fig 1) showed moderately enlarged liver with irregular liver margins, normal main portal vein and right portal vein. However, the left portal vein was not visualized. SMV too was not visualized. The Splenic vein was dilated measuring 12mm. Multiple perisplenic, peripancreatic and pericholecystic collaterals were seen. Mean liver stiffness on MR elastography was 4.6 Kpa (Fibrosis stage 3/4).

Diagnostic Dilemma:

The investigations could not conclusively confirm whether we were dealing with EHPVO or NCPF. While Clinical presentation at age of 10 years and presence of Portal biliopathy corroborated with the diagnosis of EHPVO, the dilemma was why EHPVO patients should have PHG on endoscopy and why there should be an increased liver stiffness with fibrosis. Thus, this case represents an overlap of EHPVO to NCPF.

Since the patient had symptomatic Portal biliopathy and was a bleeder and had a Splenic vein with diameter of 12 mm, we did a Proximal splenorenal shunt followed by Hepatico-Jejunostomy after 6 months.

Discussion

The differentiating features of EHPVO and NCPF² are well described (Table 1) however at times there exists overlap between the two. EHPVO refers to a more homogenous entity of a vascular disorder characterized by obstruction of the extrahepatic PV with or without the involvement of intrahepatic PV, splenic, or superior mesenteric veins. The

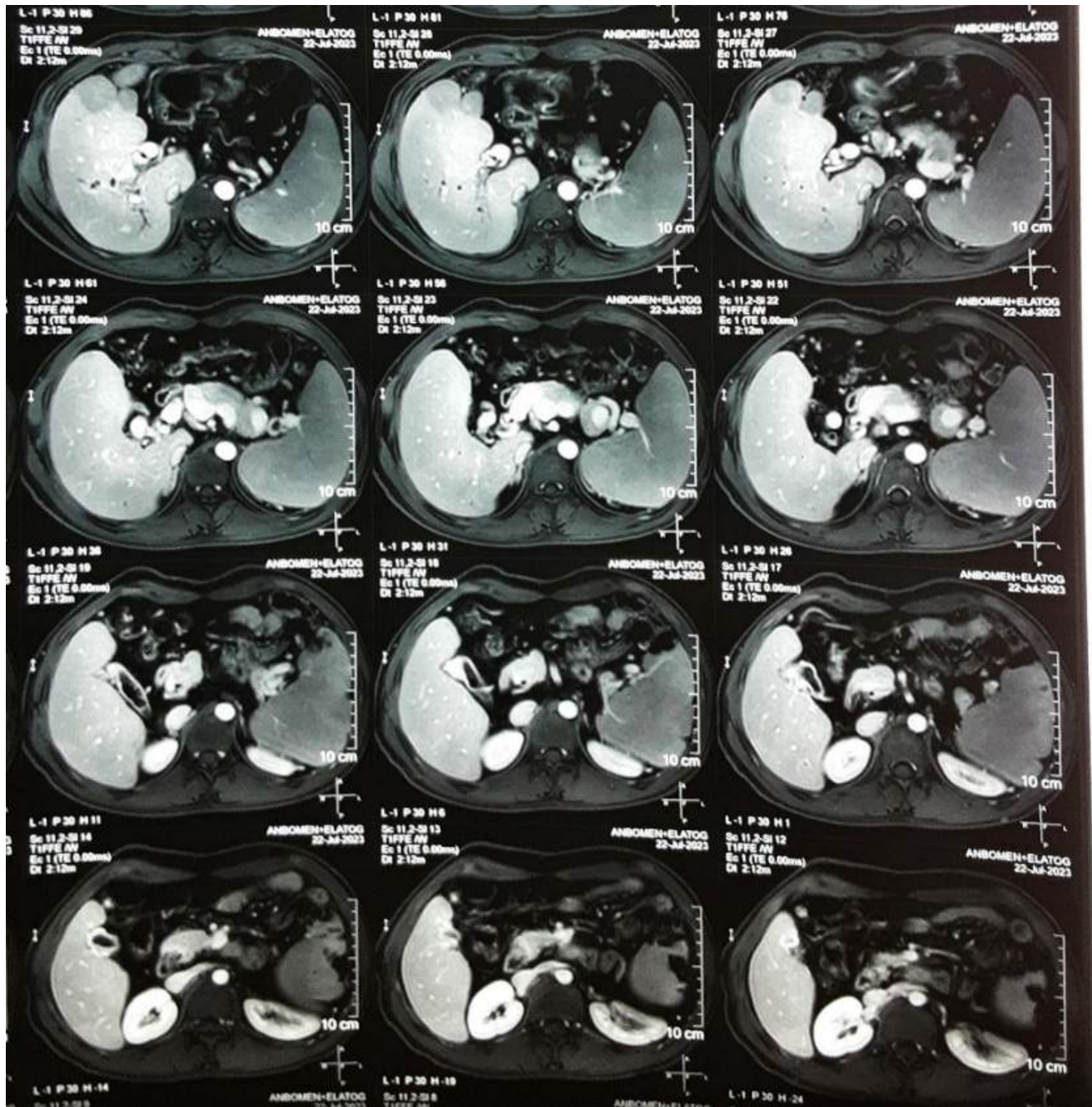


Fig 1: Atrophy of left lobe of liver with irregular liver margins, Normal MPV and RPV. However, LPV & SMV not visualized. The Splenic vein 12mm. Multiple perisplenic, peripancreatic and pericholecystic collaterals were seen.

cavernoma formation is a hallmark of EHPVO.

NCPF/IPH encompasses a group of hepatic vascular diseases with varied etiologies and histopathologic features and characterized by intrahepatic vascular

lesions and evidence of portal hypertension in the absence of histological evidence of cirrhosis³.

Table 1

	EHPVO	NCPF
Age	Childhood /Early age	Middle age / F>M
Pathogenesis	Acute thrombotic event/ predisposition	Recurrent infection / Microthrombi Endotoxins and autoimmune
Features	Growth retardation, PCC	Liver dysfunction rarely
Splenomegaly	Moderate	Massive
	Main PV	Peripheral Br PV
PV	Portal cavernoma	May be normal
Variceal bleed	More common	
Doppler USG	cavernomatous transformation of PV	splenoportal axis is dilated and patent. PV is thickened (>3 mm) There is sudden narrowing of intrahepatic 2nd/ 3rd PV br - “withered tree” appearance
		hepatoportal sclerosis, periportal fibrosis, perisinusoidal fibrosis and nodular regenerative hyperplasia

Case Profile 2: NCPF or CLD

A 34-year-old female was referred to us for management of symptomatic hypersplenism. She had episodes of fever with neutropenia off and on. On investigations UGI endoscopy revealed one small oesophageal varix. Patient was diagnosed as NCPF for 8 years.

USG of the abdomen done revealed ascites with left pleural effusion along with splenomegaly and other features of PHT.

Blood investigations revealed pancytopenia.

However, what was intriguing that the LFT done over a period of about 2 months showed variable range of serum albumin (1.8-2.9) and raised INR (max upto 1.8) and Bilirubin (2.6/1.5-1/0.8) s/o episodic decompensation. Ascitic tap showed high SAAG (1.6) suggestive of Chronic Liver disease.

CECT scan (Fig 2) showed atrophy of the right lobe of liver with caudate lobe hypertrophy and portosystemic collaterals with mild ascites. Fibro scan done revealed Grade II fibrosis. Liver biopsy revealed no cirrhosis.

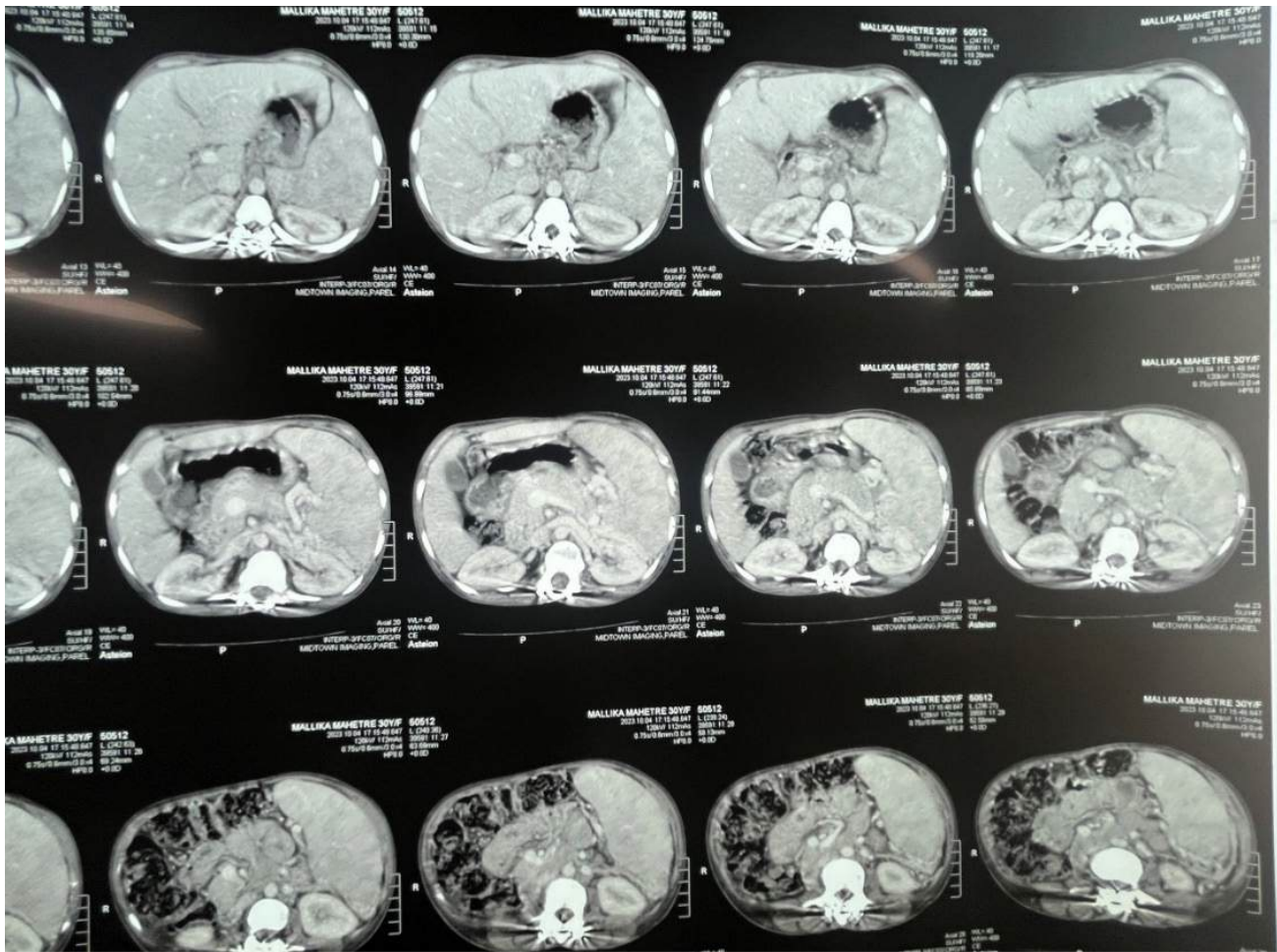


Fig 2: Atrophy of right lobe of liver, Prominent left and caudate lobe, Fissural widening, Surface nodularity. Widespread portosplenic collaterals with massive splenomegaly

Dilemma:

This was clearly a case of NCPF progressing to CLD with symptomatic hypersplenism. The dilemma was what therapeutic option should be offered to the patient for her hypersplenism.

A shunt surgery is less than ideal surgery because of risk of MHE, ascites and decompensation³.

Splenic embolization is another option; however, it is associated with its own set of complications in form of pleural effusion, ascites, splenic abscess, splenic rupture and sepsis⁴.

Another option is devascularization surgery with splenectomy.

At times Splenectomy alone would address the issue of hypersplenism, but then it does not take of other porto-systemic collaterals. Besides it is associated with risk of postoperative infections, particularly overwhelming infections which occur in 3–5%, characterized by bacteraemia and sepsis, with mortality reaching up to 50%⁵.

Discussion

In the context of Non-Cirrhotic Portal Hypertension (NCPH), parenchymal extinction is the localized, irreversible loss of functional liver cells (hepatocytes) and microvascular structures, which are progressively replaced by fibrous tissue⁶.

In both EHPVO and NCPH the primary issue is vascular - specifically the narrowing, thrombosis,

or disappearance of small portal veins. Chronic Ischemia and Hypoxia and Atrophy eventually lead to “extinction” of the affected liver parenchyma.

In view of this, it is prudent that patients with NCPF are carefully evaluated for the indication of surgery.

Patients with failed endotherapy who are at high risk of re-bleeding should undergo a portosystemic shunt such as a proximal LRS.

However, those who have not had variceal bleeding usually have low grade varices and require surgery for hypersplenism or pain related to massive splenomegaly. These patients should be managed with splenectomy and devascularization.

Key Message:

- a)NCPH is a common cause of PHT
- b)NCPF and EHPVO are two distinct diseases, each with its own characteristic features.
- c)Presence of Portal Cavernoma is characteristic diagnostic feature of EHPVO, whereas exclusion of Cirrhosis is a must for confirmation of NCPF.
- d)Hepatic dysfunction can occur late in the natural history of EHPVO, due to hepatic parenchymal extinction.
- e)There may be an overlap between Cirrhosis in NCPF late in its natural history.

f)Surgical management in NCPF is individualized depending on the presentation and Liver function

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Dr. Vulimiri Ramalingaswami

Dr. Vulimiri Ramalingaswami significantly shaped global hepatology by defining and coining Noncirrhotic Portal Fibrosis (NCPF) in 1969. Working alongside fellow pathologist Dr. N.C. Nayak, his landmark histopathological research isolated this unique tropical liver disease from traditional liver cirrhosis. [1, 2]

The Discovery of NCPF

Before Dr. Ramalingaswami's breakthroughs, patients in the Indian subcontinent presenting with severe portal hypertension, massive spleen enlargement (splenomegaly), and life-threatening gastrointestinal bleeding were routinely misdiagnosed with liver cirrhosis. Through extensive liver biopsies and post-mortem studies, his team proved that these patients had completely non-cirrhotic liver tissue. [1, 2, 3]

Instead of the diffuse scarring that characterizes cirrhosis, they observed a specific narrowing, thickening, and destruction of the intrahepatic portal vein branches. Dr. Ramalingaswami initially described this distinct structural change as "obliterative portal venopathy". During a historic 1969 workshop organized by the Indian Council of Medical Research (ICMR), the expert consensus officially adopted the medical term Noncirrhotic Portal Fibrosis (NCPF) to standardize the diagnosis across the subcontinent. [1, 2]

Key Clinical & Pathological Characteristics

Dr. Ramalingaswami's research mapped out the exact clinical presentation of NCPF, distinguishing it by several specific parameters: [1, 2]

- **Preserved Liver Function:** Unlike cirrhosis, the liver cells (hepatocytes) remain functional,

meaning patients rarely suffer from liver failure or jaundice until advanced stages. [1]

- **Massive Splenomegaly:** Patients present with severely enlarged spleens due to intense backward blood pressure. [1, 2]
- **Recurrent Variceal Bleeding:** High pressure causes veins in the esophagus to rupture, leading to repeated episodes of vomiting blood. [1, 2]
- **Phlebosclerosis:** Microscopic viewing reveals heavy scarring and blockages localized inside the small and medium-sized portal vein tracts. [1, 2]

Global Medical Context

NCPF is primarily found in tropical, developing nations. Dr. Ramalingaswami's characterisation of the disease directly aligns with identical global conditions known by different regional names. [1, 2, 3]

Broader Contributions to Liver Disease

Beyond defining NCPF, Dr. Ramalingaswami leveraged his position as the Director of the All India Institute of Medical Sciences (AIIMS) and later Director-General of the ICMR to advance liver science. He debunked existing medical myths by proving that nutritional cirrhosis was not caused by

Region	Regional Nomenclature
Indian Subcontinent	Noncirrhotic Portal Fibrosis (NCPF)
Japan & East Asia	Idiopathic Portal Hypertension (IPH)
Western Countries	Hepatoportal Sclerosis or Portal Sinusoidal Vascular Disease (PSVD)

a virus. He also conducted extensive research into the pathogenetic link between chronic Hepatitis B infection and primary liver cancer (hepatocellular carcinoma). His lifelong work earned him world-

renowned status as a fellow of the Royal Society of London and India's prestigious Padma Vibhushan award. [1, 2, 3]



Prof. Samiran Nundy

A Pioneer of Portal Hypertension Surgery in India

Few surgeons have influenced the evolution of portal hypertension surgery in India as profoundly as Prof. Samiran Nundy. Returning to India after training at Cambridge, London and Harvard, he devoted a substantial part of his academic career at AIIMS, New Delhi, to diseases particularly relevant to Indian patients. His own published account documents this remarkable journey and the development of portal-hypertension surgery in the country. At a time when non-

cirrhotic portal hypertension—especially extrahepatic portal venous obstruction (EHPVO) and non-cirrhotic portal fibrosis (NCPF)—formed an important part of Indian surgical practice, Nundy and his colleagues systematically studied its natural history, diagnosis and treatment. His work helped define the place of portosystemic shunt surgery, while also examining complications such as post-shunt encephalopathy and recurrent variceal bleeding.

His contribution extended beyond the operating theatre. Through decades of research, teaching and publications, he helped establish portal hypertension as a major academic focus of GI surgery at AIIMS; the department continues to identify portal hypertension among its principal specialties.

Later, at Sir Ganga Ram Hospital, his group contributed substantially to understanding portal biliopathy and demonstrated the important role of portal decompression in appropriately selected patients.

Prof. Nundy's enduring achievement was not merely performing shunts—it was helping transform a distinctly Indian clinical problem into a disciplined field of surgical enquiry, treatment and teaching, while training generations of surgeons to carry that work forward.

Fifty Years of Experience in Portal Hypertension and it's Surgical Management in India



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Conflict of Interest - None to Declare; Source of Funding – None

Abstract

The problem of portal hypertension in India is unique with a large number of young patients with extra-hepatic portal venous obstruction and other non-cirrhotic causes. Over the past fifty years, the management of portal hypertension in India has undergone major changes from a primarily surgery based treatment to a multidisciplinary approach with a combination of non-surgical interventions including pharmacological control, endoscopic and interventional guided procedures. The primary goal of the treatment has changed from surgical portal decompression to treatment aimed at preserving liver function and finally achieving liver transplants in refractory cases. This article presents a long-term experience of managing portal hypertension in India, highlighting the unique disease pattern, the role of shunt surgery and the transition to modern therapies. It also includes the growth of liver transplantation in India, which has placed the country among global leaders, especially in living donor liver transplantation. The experience emphasizes the need to adapt treatment to local conditions while preserving surgical expertise

Introduction

The term portal hypertension was first introduced by Augustin Gilbert in 1902.^[1] The common causes of portal hypertension in Western countries are intra-hepatic obstruction of the portal flow i.e. cirrhosis (85.6%), prehepatic portal flow obstruction/ extra hepatic portal venous obstruction (EHPVO) due to portal thrombosis or cavernoma (7.8%), 6.4% had both the elements and 0.2% had segmental splenic vein thrombosis.^[2] Portal hypertension has always been a difficult condition to manage. Over the last fifty years, its treatment in India has changed significantly from surgery based treatment to a non-surgical management involving multiple specialties working together. This experience,

beginning in late seventies reflects not only advances in medicine but also the unique disease patterns seen in India. It highlights how treatment must be adapted to local needs rather than simply following Western models.

Global perspective: Endoscopic treatment to surgery

Gastrointestinal bleeding from the oesophageal varices is the major cause of mortality in portal hypertension apart from liver failure. The chances of variceal bleeding are more with EHPVO than cirrhosis as the mean portal pressure is greater in pre-hepatic obstruction (28.7 mm Hg) than in cirrhosis (21.6 mm Hg). Gastro-intestinal bleeding usually happens with portal pressures above 22 mm Hg.[2] Initially the direct methods with balloon to compress the bleeding oesophageal varices were attempted by Westfal in 1930[3] and Rowntree in 1947[4] but they were sometimes associated with serious complications including oesophageal rupture, so the technique was later improved by Sengstaken and Blakemore[5] in 1950. Crafoord and Fenckner[6] in 1939, used oesophageal injections of a sclerosing agent, a quinine solution around the varices which was also used by Terblanche in 1970s.[7] A few prospective trials in those days showed that the endoscopic sclerotherapy was better than medical treatment.[7,8] In the 1980s, further prospective studies showed similar or better results with endotherapy than shunts for recurrent bleeding varices.[9] Various prospective trials showed better results with injection sclerotherapy than shunts.[10] Since sclerotherapy was associated with frequent oesophageal erosion and ulceration, the elastic band ligation surpassed it and later became the standard of care for bleeding oesophageal varices.[11,12]

The higher chances of recurrence of bleeding after these local measures led to simultaneous development of surgical procedures aimed at

resolving the problem i.e. to lower the portal pressure. These operations were technically demanding and often lengthy procedures. Emergency mortality was initially close to 70%. These surgeries were aimed towards either reducing the inflow or increasing the outflow. [3] The diversion of the flow was achieved by a portocaval anastomosis. Vidal in 1903 performed the first successful portocaval shunt[3], but the patient died of sepsis and liver failure few months later. These procedure were abandoned for nearly 40 years but later Child[13] and Allan Whipple[14] tried a variety of shunts. Later Blalock performed multiple successful porto-caval shunts at the John Hopkins institute. These procedures were reported to regress the varices drastically with almost no risk of bleeding.[15]

Portocaval shunts were effective in controlling the variceal bleeding but had a very high incidence of encephalopathy and subsequent liver failure. Due to these limitations the selective shunts, like Warren's distal spleno-renal shunt[16], gained popularity where the flow to the liver was maintained and the chances of encephalopathy were reduced. These shunts achieved a lower decompression of the portal area but were not associated with hypo perfused liver leading to its failure and hepatic encephalopathy. Splenectomy, the other surgical treatment in portal hypertension, gave only minor reduction to the portal inflow as it decompresses the portal system only by 2.2 mmHg.[2]

The advent of liver transplantation for patients with advanced liver failure, took away the indications for shunts in patients with cirrhosis. The introduction of the Transjugular Intrahepatic Portosystemic shunt (TIPS) further pushed the open procedure to only sporadic indications. The first successful TIPS was done by Rossle, Ritcher, Noldge, and Palmaz. [3,17] It reduced re-bleeding without giving overall survival benefits and increased the risk of hepatic encephalopathy. The shunt thrombosis (about

50% in first two years) was another problem and it required multiple interventions[10], escalating the cost. It is therefore reserved for patients with failed medical/endotherapy.

The non shunt operation in the form of oesophageal transaction had a higher rate of re-bleeding and difficult ascites to treat. The devascularization operation, also called the Sugiura operation was also suggested from Japan, allows disconnection between the portal and systemic circulation.[18] This was helpful in refractory cases of emergency haemorrhage when all other measures to control variceal bleeding have failed.

Potency of surgical procedures

The normal portal pressure is 5-12 mm Hg, 4mm more than the free pressure in the hepatic veins. Significant reduction in portal pressure is recorded across various variety of shunts ranging from 11.7 mm Hg (after termino – lateral troncular portocaval anastomosis), 15.8 mmHg after Latero-lateral PCA, and 13 mmHg after splenorenal or mesocaval anastomosis. Splenectomy reduces pressure by only 2.2 mm Hg. So shunts provide greater protection against bleeding than any other modality of treatment. Previous studies compared re-bleeding (80% vs 1%) and survival rates at 10 years (21% vs 60%) between TIPS with porto caval shunts surgery.[2] TIPS helps in controlling variceal bleed upto 85% while it has 31% higher risk of

encephalopathy than splenorenal or mesocaval shunts.[2,19,20]

The Indian Disease Pattern and Evolution of Surgery

Portal hypertension in India differs from that in Western countries. In the West, most patients have cirrhosis and their liver functions were severely compromised. While in India, many patients are young, have pre-hepatic portal flow obstruction and non-cirrhotic causes like Non-Cirrhotic Portal Fibrosis (NCPF) and Extra Hepatic Portal Venous Obstruction (EHPVO), who had massive upper gastro-intestinal bleeding but had preserved liver functions. The common causes were EHPVO (58%), NCPF (30%), HVOO (3.8%) and others (0.8%).[21] The surgery for portal hypertension started alongside the development of surgical gastroenterology in India. The surgery for portal hypertension, in particular portocaval decompression: lienorenal shunt (Image 1) was started in late 70s in AIIMS and were done in elective situations.

Since the endoscopic sclerotherapy and banding were not available for emergency conditions of variceal bleeding in portal hypertension, Crile's procedure was used as a lifesaving procedure during those days, which had a very high mortality. Lienorenal shunt, on the other hand, was being used in elective situation and showed satisfactory results, so the shunts replaced the Crile's procedure done for emergency situations of variceal bleeding.

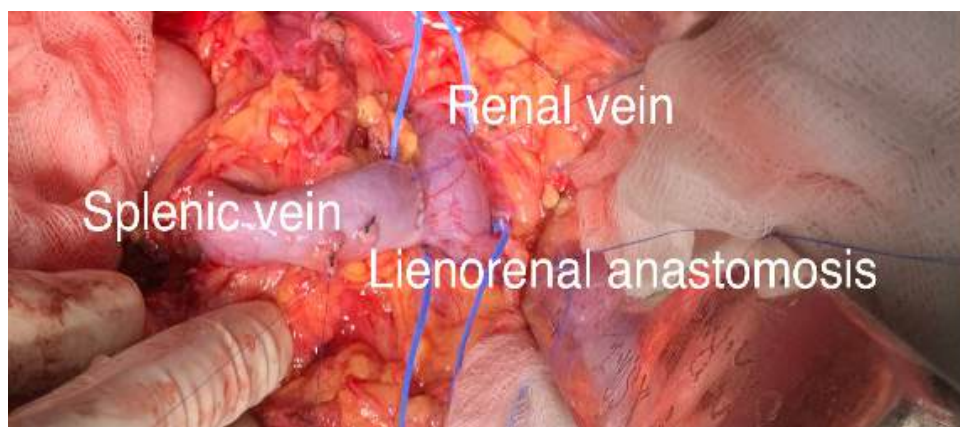


Image 1 – Selective shunt - End to side distal Lienorenal shunt



Image 2 – Combining Surgery (Devascularization and Shunt together) : A – Varices in and around, B – Devascularization, C – Lienorenal Shunt

The elective mortality of first 100 operations for portal decompression in AIIMS was 1% at that time and emergency mortality reduced from 70 to 18%. [21] Portal hypertension with oesophago-gastric varices was the most common cause (55%) of upper gastro-intestinal bleeding in India[22] against the west, so these shunt surgeries revolutionised the management of portal hypertension and later even the prophylactic operations for high risk patients of EHPVO were suggested.[23] For many patients, shunt surgery provided a one-time solution with good long-term results. The risk of

complications such as encephalopathy was low as compared to cirrhotic patients.[24–26] There are reports of combining surgeries where shunt and devascularization clubbed together. (Image 2) The shunt surgeries gave a newer perspective to the patients of EHPVO but a substantial patients of portal hypertension belonged to cirrhosis with poor liver functions who were doomed to die from complications of end stage liver disease and liver failure. Liver transplant gave the another lease of life to these patients, which later became the standard of care and it has been the most important

Table 1. Growth of Liver Transplantation in India: Then and Now

Parameter	2002	2012	2022–2024
Number of transplant centres	Fewer than 10	30–40	More than 150
Annual liver transplants	Fewer than 200	800–1000	3500–4000
Type of transplant	Limited LDLT	LDLT dominant	LDLT dominant + increasing DDLT
Legal framework	Early phase	Established	Mature system
Expertise	Limited	Expanding	Well-established
Global ranking	Not significant	Emerging	3
LDLT ranking	Not defined	Leading	1
Outcomes	Variable	Improving	Comparable to global standards

Source : Tiwari & Nundy, 2024[27]

Changing Trends: Decline of Surgery

With the development of endoscopic treatment and medicines, the need for surgery gradually decreased. Endoscopic band ligation and beta-blockers helped control bleeding without the need for major operations and emergency operations for variceal bleeding squeezed further. Later, radiological procedures such as TIPS provided additional options. Portal hypertension became a condition managed by a multidisciplinary team.

Evolution of Management of portal hypertension in India

- 1975–1985: Surgery as main treatment; high emergency mortality
- 1985–1995: Improved outcomes and expansion of portal decompression surgeries
- 1995–2005: Addition of endotherapy and emergency indications of surgery declined
- 2005–2015: Development of interventional radiology (TIPS)
- 2015–Present: Expansion of liver transplantation and shunts are only done for EHPVO

Current Challenges and Lessons from 50 Years

Despite progress, challenges remain. Bleeding continues in non-cirrhotic patients, while ascites and liver failure remain difficult problems in cirrhotic patients. Access to advanced care is still limited for many patients due to cost and availability [6]. The main lessons are clear. Treatment must be adapted to local disease patterns. Portal decompression surgery still has a role in selected patients of EHPVO. Progress requires persistence and learning from experience. The growth of liver transplantation shows what can be achieved with proper planning and teamwork.

Conclusion

Over the past fifty years, the management of portal hypertension in India has improved significantly. From a time when surgery was the only option, we now have multiple effective treatments. Although surgery is used less often today, it remains important in selected cases. The future lies in combining surgical expertise with modern treatments and ensuring that all patients benefit from these advances.

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Some surgeons are remembered for the operations they perform; others for the people, institutions and generations they help shape. Dr. Amitabh Yadav's career brings these worlds together.

With more than three decades in medicine, Dr. Yadav is today Co-Chairperson of the Institute of Surgical Gastroenterology, GI & HPB Onco-Surgery and Liver Transplantation at Sir Ganga Ram Hospital, New Delhi. His journey began at BRD Medical College, Gorakhpur, where he completed his MBBS and MS, followed by specialised fellowship training in hepatobiliary surgery and liver transplantation at Sir Ganga Ram Hospital in 2005. The hospital records him as both a senior gastrointestinal surgeon and a teaching coordinator—roles that reveal the two enduring strands of his career: healing and teaching.

His clinical interests embrace some of surgery's most demanding territories—pancreatic disease and cancer, HPB malignancies, liver transplantation and advanced minimally invasive gastrointestinal surgery. Yet his contribution extends beyond the operating theatre. His publications, including work with Professor Samiran Nundy, and his contribution to an Oxford University Press surgical text reflect a continuing commitment to scholarship and surgical education.

Dr. Amitabh Yadav's story is ultimately one of quiet, sustained purpose: operating on the difficult, teaching the young, advancing the science—and ensuring that experience becomes a legacy shared with the surgeons who follow.



Dr. Gayatri Balachandran in conversation with Prof. S. K Mathur

Few surgeons can say they helped shape the evolution of an entire specialty. Over four decades, Prof. S. K. Mathur has witnessed portal hypertension (PH) surgery evolve from an era dominated by emergency shunts to one where endoscopic therapy, TIPS and transplantation have transformed patient care. He helped develop surgical solutions for a uniquely Indian disease burden, while simultaneously pioneering indigenous innovations. His work has influenced generations of GI surgeons and transformed the management of PH in resource-limited settings

In this conversation, he reflects on his journey – lessons learnt, lives changed and the culture of excellence he has established.

Could you tell us about your early journey and how GI Surgery eventually became your calling? What first attracted you to PH as an area of interest?

After finishing my MS in 1974, I actually joined Neurosurgery. At that time, I was convinced that I would become a neurosurgeon as my mentor Late Prof. Dharkar was a Neurosurgeon. However, as they say, Destiny often pulls you in different directions. I left that post and joined General Surgery at Maulana Azad Medical College as a Senior Resident. By the end of three and a half years, I was certain that I wanted to stay in an

academic institution rather than venture into private practice.

In Nov of 1978 an opportunity came in an unexpected way. I joined Government Medical College, Surat, as an Assistant Professor. Looking back, that move probably shaped everything that followed. In Delhi I had all the advantages of working in a major teaching institution: senior mentors, good facilities and experienced colleagues. There was a lot of cover for learning and working. Surat was a major transition. I found myself in a government medical college where I had to make independent decisions, work with

limited resources, and where the system was not geared for advanced surgery. Even the diathermy was primitive!

But the patient load was abundant, and one of the most common emergencies was portal hypertensive bleed especially due to extrahepatic portal venous obstruction (EHPVO). These were young patients arriving with massive hematemesis, and this was before the era of endoscopic therapy. If you couldn't control the bleeding with a Sengstaken-Blakemore tube, then emergency surgery was the only option.

That was really where my tryst with PH began. Prior to this, the only real exposure I had to PH was in 1975 when I, as a Senior resident, had assisted my chief at MAMC, Prof. Dr. S N Buddhiraja, for a proximal leinorenal shunt. When I reached Surat, I realized very quickly that performing emergency shunt surgery wasn't always practical. We didn't have specialised vascular instruments. I had read about the Sugiura procedure and thought, "Perhaps this is something we can adapt." The first patient came in the middle of the night with uncontrolled bleeding. I did the classical Sugiura procedure as a single-stage surgery and used a different 2-layer suture technique to strangulate the esophageal varices without esophageal transection. The patient survived. Soon afterwards, together with a cardiac surgery colleague, I performed our first elective proximal leinorenal shunt. From there, the work simply grew.

Knowing the limitations of my progress in such an institution, I was on the lookout for opportunities and came across an advertisement calling for a faculty position in Bombay Municipal Corporation Medical Colleges. I appeared for the interview, despite prevailing rumours that outsiders were never selected. For me, if anything, it was an excuse to visit Bombay to do some sightseeing!!! Fortunately, the interview went well and I was selected. I joined the GS Medical College as Reader

(Associate Professor) and that's where I continued to practice.

Well, they say everything happens for a reason, and its fascinating to see how that rings true in your story. We are very happy you didn't take Neurosurgery !

India has always had a unique experience with PH. Why were EHPVO and non-cirrhotic portal fibrosis (NCPF) so common here?

Let's put it this way. Most of those patients came from poor socioeconomic backgrounds. Neonatal sepsis, diarrheal illnesses, dehydration, recurrent infections : all these probably contributed to portal vein thrombosis early in life and predisposed to EHPVO development.

Do you think we are seeing fewer patients with these conditions in the present day?

No, I wouldn't say that. I'm not convinced the incidence has fallen very much. The difference is that patients no longer come to a handful of tertiary centres. In those days, endoscopic therapy wasn't available everywhere. Naturally, every patient was referred to institutions like KEM Mumbai, AIIMS New Delhi or PGI Chandigarh . Today, almost every city has gastroenterologists who perform endoscopy and variceal band ligation. So, patients are spread across many hospitals. That creates the impression that the disease itself has become uncommon.

You have co-authored a chapter in which you describe the evolution of PH management in India as passing through three distinct eras: the pre-sclerotherapy surgical era, the sclerotherapy era, and the resurgence of surgery during endotherapy.

How did you personally experience these transitions?

When I joined KEM in 1981 Dr. Bhalerao, one of the

veterans of PH surgery, used to performed a couple of shunt procedures every week. Emergency surgery for bleeding varices was routine, with high mortality. It was around this time that the reports of endoscopic sclerotherapy started appearing in literature for control of acute esophageal variceal bleeding.

Dr. Bhalerao asked me, “Can we start this here?” At that time, I was doing diagnostic upper and lower GI endoscopies, polypectomy and even ERCP. He made arrangements for all the equipment and procured an imported Olympus injection needle as well as 30ml of sodium tetradecyl sulphate from the United States. Our first patient had active variceal bleeding. He simply said, “Go ahead. If anything goes wrong, I’ll operate.” Fortunately, nothing went wrong. I was able to perform the sclerotherapy and the bleeding stopped. We did the same procedure for a few more patients and once we saw that it worked, everything changed.

I remember, after those first few sclerotherapy cases, I had to do an emergency case in the middle of one night, when Dr. Bhalerao was away. Due to some unfortunate circumstances, the injector was damaged and our imported supplies of STD ran out. We had to look for local alternatives to the sclerosant and sclerotherapy needle to continue to offer these patient endoscopic sclerotherapy. I reviewed the literature and came across a German publication using phenol-in-oil through a rigid endoscope. We designed animal experiments and eventually developed a phenol-in-glycerin based sclerosant that was inexpensive, effective and locally prepared in the hospital pharmacy. We compared the effect of STD, 3% phenol-in-glycerin and absolute alcohol on veins of dogs, and found that phenol performed favourably. I even improvised the teflon tubing of the old D3 model Olympus fibre-optic endoscope to be used for injection of varices by fixing a 18 G needle to its distal end. The experiment worked, and we published our series of 50 cases of endoscopic

variceal sclerotherapy using 3% Phenol-in-glycerin.

The problem was the solution was too viscous and made the whole process of injecting laboursome. Also, the use of the bare needle through the scope was liable to damage the endoscope. So, I went back to the Goodman Gillman pharmacology textbook for a solution and discovered (in an obscure footnote) that phenol was miscible with water. To establish the efficacy of aqueous solution of phenol, I did another set of animal experiments and proved on histology that it was an effective sclerosant. This 3% phenol-in-water became our standard endoscopic sclerotherapy agent, and that solidified the success of endotherapy; we published our short and long term experience in 300 patients. Gradually we realized that many patients could avoid surgery altogether. Mortality fell, emergency operations became less common, and surgery was reserved for those who failed endoscopic treatment. That’s how the second era of regression of surgery began.

Even today I believe endotherapy should be the first line of treatment in most patients and in almost 80 percent of cases varices can be obliterated completely. If a young patient can live without a laparotomy, that’s always preferable. Surgery should remain available: but for the right indication.

When it came to operating on patients with failed endotherapy or massive rebleed, we wanted to reduce morbidity as much as possible. I revisited the Sugiura procedure, but modified it to avoid the thoracic incision and performed transhiatal devascularization. I also avoided splenectomy and tackled hypersplenism with proximal splenic artery ligation. Having preserved the spleen, I felt that the option of a future shunt was preserved since the splenic vein was intact. Overall, my results were comparable to classic Sugiura surgery, and there was good resolution of hypersplenism

with no increased incidence of splenic abscess. My philosophy was to simplify the whole procedure and make it accessible for even a general surgeon to perform.

It seems to me that your work consistently reflects practical and resource-conscious innovation, and not just for research or technology. Was this a conscious philosophy or just a means to deal with realities of your practice at the time?

People sometimes think innovation means inventing something completely new. I don't see it that way. Innovation is solving a problem safely and making it simpler and cost-effective.

There was recently a report of indigenous modification of machine perfusion for a liver graft using a heart lung bypass machine. I remember doing similar experiments in machine perfusion of liver grafts in dogs in 1990's. The time was not mature for me to publish then... but decades later, it is being studied and applied. I recognized the need then, but people were not convinced as the technology hadn't been honed and developed in the West first.

It is not necessary to ape the West for everything. We must recognize what suits us best, what fits our patients' scenarios. Even today, whenever new technology comes from abroad, my first question is whether it can be adapted to our patients and our healthcare system without compromising results.

Can you tell us a bit more about how your programme in PH evolved at KEM?

As I said earlier, I was developing endotherapy as a therapeutic intervention in KEM, and later a colleague from Medical Gastroenterology, the late Dr. SR Nayak, joined me. We used to work in tandem, handling alternate day calls for emergency upper GI bleed. Once our results began to speak for themselves, our referral base grew and so did

our department. Several prominent GI surgeons of today such as Dr. Avinash Supe, Dr. Abhay Dalvi, Dr. Darius Mirza, Dr. Chetan Kantharia and Dr. Sudeep Shah, trained under me and were able to gain experience in surgical and endoscopic management of PH.

As we all know, there are a myriad of surgical options for PH, and often different centres have their own philosophy and preferred operation of choice. Why do think there is such variety for the same underlying pathology. And, did you have a favourite surgery, one you consider the most elegant?

(Laughs.) Surgeons always have favourites. I don't mean to be harsh, but one's choice must always be guided by what is scientifically most appropriate for the given patient and situation. So, my personal philosophy is that we shouldn't become attached to a particular operation, rather tailor the surgery according to the requirement of the patient.

When the late Dr. Bhalerao and late Dr. A K Basu (both doyens of PH Surgery) were initially operating, the most common operation performed for PH was side-to-side portocaval shunt, with all its complications. Once reports of the Warren shunt emerged in 1967 for cirrhotic patients, both adopted the new procedure in their practice, and demonstrated superior rates of hepatic encephalopathy. When the results were analyzed in patients with NCPF, who essentially have normal livers, it was seen that complete portal diversion resulted in increased incidence of hepatic encephalopathy by depriving the liver of portal flow. Similar results were also seen in data reported from AIIMS with PSRS for NCPF patients. By doing a distal splenorenal shunt (DSRS), this problem was solved. That is why I strongly feel that selective shunts have advantages in these patients. Somehow the Warren shunt was considered technically challenging and was not widely performed or taught to trainees, especially in large-

volume centers in Delhi. I was fortunate enough to learn it from an American surgeon named Davidson, when he demonstrated the procedure in a workshop at Chennai. I was able to refine my own technique, overcome the complexity of the procedure, and adopt it on a larger scale. I found it enjoyable, and in turn, I taught the procedure to my students as well.

Coming back to your question, I think the reason there are so many different approaches and operations to PH is because alternatives were not adequately demonstrated. Take for example, the case of devascularization. Western literature reported a rebleed rate of as high as 35%. Everybody thought that this was, therefore, a bad procedure. I think this was more due to a technical compromise rather than an inadequacy of the procedure itself. I remember demonstrating my modified Sugiura surgery in Lucknow at SGPGI once; I met criticism from certain senior surgeons in the audience who were skeptical. “The esophagus will look blue and this is a dangerous and useless operation!”, they said. I just quietly showed them how it was done, how “blue or pink” the esophagus was at the end of the procedure, and then all critics were silenced... (laughs). The trick is to ligate all the perforators and preserve the arteries. Somehow, they had never seen or performed the operation the way it ought to be done, and hence they had reservations.

As for my favorite operation, I prefer surgeries where the spleen can be preserved. The spleen enlarges because of PH; it isn't the main cause of PH. If you can preserve it safely, why remove it? Rather, adequately decongesting it serves the same purpose, and safeguards the functions of the spleen. So, in cases of EHPVO I prefer a side-to-side lienorenal shunt and in NCPF, a Warren shunt.

Another procedure that I prefer is using the adrenal vein as a conduit, a modification I devised. Interestingly, that idea came during a planned PSRS when the splenic vein was inadvertently injured at

the hand of one of my junior colleagues. When we dissected the renal vein, we encountered a hugely dilated adrenal vein and I used that to complete the shunt. So, innovation often can be the result of something unexpected forcing you to think differently. After this experience, this has been a go-to procedure for me if anatomy is favourable.

What was your preoperative planning process, especially in an era preceding CT and MR venograms?

Those days, we used to perform preoperative splenoportogram via percutaneous splenic puncture using a Chiva needle, and with fluoroscopy. One needs highly skilled radiologists to do this, and I was fortunate that way.

In fact, we looked at all our data and correlated the radiological and endoscopic findings. We mapped the major feeding vessels, especially the left gastric vein. We published our findings in the British Journal of Surgery and this became the basis of our classification of Gastric Varices published in 1989, ahead of Dr. Sarin's eponymous classification. This anatomical planning helped tremendously in achieving complete devascularisation, by being able to tackle all the feeding vessels.

What is your opinion on prophylactic surgery for non-cirrhotic PH?

On principle, I am not a proponent of prophylactic shunt surgery in this group of patients, since it is a supra-major surgery, not without its own morbidity. Even operating for soft indications is also questionable. Hypersplenism is often over-diagnosed based on laboratory reports. Growth retardation is another over-used indication for doing a splenectomy. I have so many children on follow-up, for whom I had performed spleen-preserving surgeries; they are all strapping young adults now, with absolutely no signs of growth impediment!!!!

Often these so-called prophylactic shunts are not actually prophylactic. In a patient with symptomatic hypersplenism, a mere splenectomy for symptom control would hardly suffice as it would aggravate the PH and lead to life-threatening bleed. A shunt is therefore indicated in this situation.

Is PH surgery becoming a lost art?

To some extent, yes. Fewer patients require surgery today, but there will always be patients who fail endotherapy, patients with ectopic varices, biliopathy or other situations where surgery remains the best treatment. The problem is that the centers that do still perform these surgeries in large numbers only do certain procedures. The danger is that expertise and versatility will disappear. If we stop teaching these operations, who will perform them ten years from now?

Endotherapy is very obviously the first line management for UGI bleed in these patients, and all evidence supports this. Evidence-based medicine is important, but surgeons also need to remain part of developing that evidence. Too many guidelines are written without adequate surgical representation. Take for example the Baveno guidelines on management of GI bleed in PH; written entirely by medical gastroenterologists! Surgery is relegated to a mere foot note in the treatment algorithm! This should not be the case. PH should remain a multidisciplinary field and surgeons must remain proactive in its management.

What are your thoughts on academic surgery and inculcating it into your practice?

First and foremost, I always insisted on documentation and data collection, and expected the same from my residents. There is this misconception that basic research at a molecular level is more valuable. But we have a wealth of clinical material to analyze and learn from in

our country, and I firmly believe in good clinical research. Most of my work came from asking simple questions while looking after patients. Why did this happen? Why did this patient bleed again? Why did one operation work better than another?

This kind of research begins with maintenance of careful records. If you don't document, you never ask those questions.

Do you think modern training sometimes over-emphasizes technology and under-emphasizes clinical reasoning?

Technology can never be a substitute for clinical acumen, rather it should be used to strengthen it. Good history taking and thorough examination are the fundamentals that must remain strong; Hutchinson-Hunter and K Das should never be forgotten. Imaging should confirm your diagnosis, not make it for you.

As for surgical technology, one must be willing to adapt. As technology evolves, we must change with it. I trained in open surgery at KEM Hospital, but then learnt operative laparoscopy. I was ready to embrace newer techniques whenever they benefited patients.

What advice would you give young GI surgeons today?

First of all - Master your speciality rather than competing to do routine operations. As GI surgeons you should be performing operations that your general surgery colleagues find difficult to manage.

Second, always remain curious and analytical. Everyone talks about Artificial Intelligence today. I tell my students that Natural Intelligence is still superior. Artificial Intelligence only analyses what human intelligence has first understood. So think, Observe, Question, That's how surgery moves forward.

If you were beginning your career again in 2026, would you still choose GI surgery?

Yes, I think I would. As you know after GI, I branched

my focus into HPB and later liver transplant. But you know what, I still remember my Neurosurgery roots!! (Laughs). In fact there are a lot of similarities between the surgical techniques used in both these fields i.e. Liver and brain surgery!!!



Rapid Fire



One paper or book every trainee should read?

My chapter on portal hypertension (smiles). And Dr. Ananthakrishnan's work on corrosive strictures.

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One innovation that changed surgery forever?

Laparoscopy.

|

One quality more important than technical skill?

An analytical mind.

|

Some interests outside of work and surgery

Playing badminton, reading newspapers and Indian history.

|

One lesson you've learnt over four decades?

Never accept the default superiority of the West. Self-respect and self-pride must never be surrendered to anyone. Indian surgeons have contributed enormously under difficult circumstances, and we should meet the world with confidence, not imitation.

Meso-Rex Bypass in Children.



Professor Ajit Kumar Basu

A Life in Surgery and Service

Professor Ajit Kumar Basu 1912 to 1986

Ajit Kumar Basu helped establish thoracic and cardiovascular surgery as a field of specialized teaching and practice in India. He was also a general surgeon, researcher and exacting teacher whose work ranged from heart surgery to liver disease and tropical splenomegaly. Across more than four decades, he built departments, trained surgeons, strengthened professional bodies and remained committed to public service.

Education and Early Surgical Training

Born in 1912, Ajit Kumar Basu received his early education at Hare School and Presidency College in Calcutta. He then studied at Calcutta Medical College, where he began his surgical training under Colonel John Anderson, remembered as a formidable surgeon and teacher.

After the Second World War, he traveled to England and began a long association with the Royal College of Surgeons. He qualified for the Fellowship of the Royal College of Surgeons in 1946. He later became the first Indian appointed as an examiner of the College and also served as a Hunterian Professor.

A New Direction in Cardiovascular Surgery

In 1950, Basu became Professor and Head of Surgery at Nilratan Sircar Medical College in Calcutta. His work there encompassed general and orthopedic surgery, but a fellowship abroad shifted his career. As a Rockefeller Foundation Fellow in Boston in 1952, he encountered the developing possibilities of cardiac and vascular surgery.

He returned to India when only a small number

of surgeons were developing thoracic and cardiovascular practice. In 1954, he helped form the Thoracic Chapter of the Association of Surgeons of India and became its first Secretary.

Building a Major Surgical Center

Basu completed his tenure at Nilratan Sircar Medical College in 1955. Three years later, when a postgraduate medical institute was established at Presidency General Hospital in Calcutta, he was invited to lead its Department of Thoracic and Cardiovascular Surgery. Under his direction, the department developed into an independent sixty-bed service with facilities considered excellent for the period.

International colleagues supported some of its early experimental work. Dr. C. Walton Lillehei and his associates offered encouragement in the first years; later, Dr. Rygg and colleagues from Copenhagen collaborated with the centre. The department tested the early Polystan machine and the Rygg bag as it developed the equipment and methods required for modern cardiovascular surgery.

Basu's commitment to cardiovascular surgery never displaced his original specialty. As Director

of the Postgraduate Institute of Medical Education and Research, he maintained a general surgical unit and continued to teach and investigate liver surgery and portal hypertension until his retirement.

Research, Teaching and Clinical Principles

His research extended well beyond operative technique. He studied liver disease and tropical splenomegaly with Dr. Amalananda Das, Dr. N. R. Konar and Dr. B. N. Aikat, and later worked with the research wing of Johns Hopkins Hospital and the Indian Council of Medical Research. His monograph on tropical splenomegaly gathered the results of a lifelong inquiry. In all, he produced three research monographs, contributed chapters to eight books and authored nearly two hundred scientific papers.

Patients travelled long distances to seek his care, yet he refused to let private practice weaken his professional principles. As a teacher, he demanded the highest standards of conduct and preparation. He expressed strong views directly, but colleagues also remembered his readiness to respect reasoned disagreement.

Leadership at Home and Abroad

Basu's concern for the surgical profession led to a succession of leadership roles. He served as President of the Association of Surgeons of India from 1970 to 1972 and also presided over the Indian Academy of Medical Sciences. He became Dean of the Medical Faculty of Calcutta University and Chairman of the Governing Committee of the All India Institute of Medical Sciences.

His responsibilities also crossed national borders. He served on the Scientific Advisory Board of the Indian Council of Medical Research, the Council of the International Federation of Surgical Colleges

and the Editorial Board of the British Journal of Surgery. From 1966 to 1976, he was a Governor of the American College of Surgeons. He also held visiting professorships at Johns Hopkins University and at a university in Edmonton.

Honours and Public Service

The Shanti Swarup Bhatnagar Prize recognized his scientific work in 1967, and the Government of India awarded him the Padma Shri in 1970. His other honours included the Dr. B. C. Roy Memorial Award, the Coats Medal of Calcutta University, the Barclay Medal of the Asiatic Society and the Vishnevsky Medal of the Soviet Union. In 1981, Banaras Hindu University conferred on him an honorary Doctor of Science degree.

He retired from active institutional service in 1971, but retirement did not end his public work. The Government of West Bengal recalled him to serve for three years as Vice Chairman of the State Planning Board. He later led the Basu Committee, which examined teaching problems in the state's undergraduate colleges and completed its report in unusually short order.

He continued to serve as an honorary consultant to South Eastern Railways and the Indian Army and as honorary surgeon to the President of India.

The Measure of His Life

Professor Ajit Kumar Basu died in December 1986, leaving his wife, two sons and a large community of students, colleagues and admirers. His achievements can be counted in departments built, operations performed, research published and honours received. The fuller measure lies in the professional culture he helped create: surgeons trained to think rigorously, institutions prepared to support new specialties and public responsibilities accepted without abandoning clinical principle.

Meso-Rex Bypass in Children.

Introduction:

Meso-Rex bypass (MRB) is a portal venous reconstruction procedure that restores physiological hepatopetal portal blood flow to the liver. It is an established surgical option for children with extrahepatic portal vein obstruction (EHPVO). Unlike conventional portosystemic shunts, which decompress the portal venous system by diverting blood into the systemic circulation, MRB re-establishes portal venous inflow to the liver, thereby preserving normal hepatic perfusion and portal pressure. However, the feasibility of MRB is often limited by anatomical considerations, and experience with the procedure remains relatively limited in many centres.

MRB also has a role in selected children following liver transplantation, particularly when the native portal vein is inadequate because of thrombosis, short length or small calibre. In this setting, it can serve as a rescue portal reconstruction when conventional portal venous anastomosis fails or thromboses.

The present study reviews our experience with Meso-Rex bypass in children undergoing portal venous reconstruction for EHPVO and post- liver transplant portal vein thrombosis.

MRB Surgical technique :⁽¹⁾

The child is positioned supine with the neck extended and turned toward the right side. The abdomen is entered through a Mercedes Benz incision. The liver edge is suspended with the help of prolene stitches on either side of the umbilical fissure to expose the inferior surface. The umbilical ligament is then followed into the rex recessus, between segments 3 and 4 of the liver, where it ends in the left branch of the portal vein. The parenchymal bridge between segments III and IV is resected to improve access to the rex recess as well as to prevent compression of the graft vein. The left branch of portal vein is dissected and free major tributaries draining from segments 3 and 4 are slooped but not ligated (Fig 1). It is vital to preserve these as they provide the runoff of portal flow coming into the liver through the graft.

The SMV was accessed in infra-colic compartment by following the middle colic vein and is dissected

free for at least 3–4 cm. The colonic mesentery was divided after dividing the middle colic vein close to its insertion into the SMV. A prepancreatic, retrogastric tunnel of adequate size was prepared for the bypass graft vein to sit. The entire length of left internal jugular vein was harvested through two transverse neck incisions (Fig 2). After applying vascular clamps the left branch of the portal vein is laid open (Fig 3). The graft end to the side of the left portal vein anastomosis is performed first, with 6/0 or 7/0 prolene. A good retrograde filling of the graft after all tributaries are released confirms good drainage. The graft is positioned within the previously created tunnel to bring it into the infracolic compartment. Special care is taken to ensure that the graft does not spiral as it is pulled down through the tunnel. The end of the graft is then anastomosed to the side of the SMV, as close to the inferior edge of the pancreas as possible. The clamps are then released and good craniocaudal flow from SMV toward the left portal vein seen (Fig 4,5) and is confirmed with intraoperative Doppler

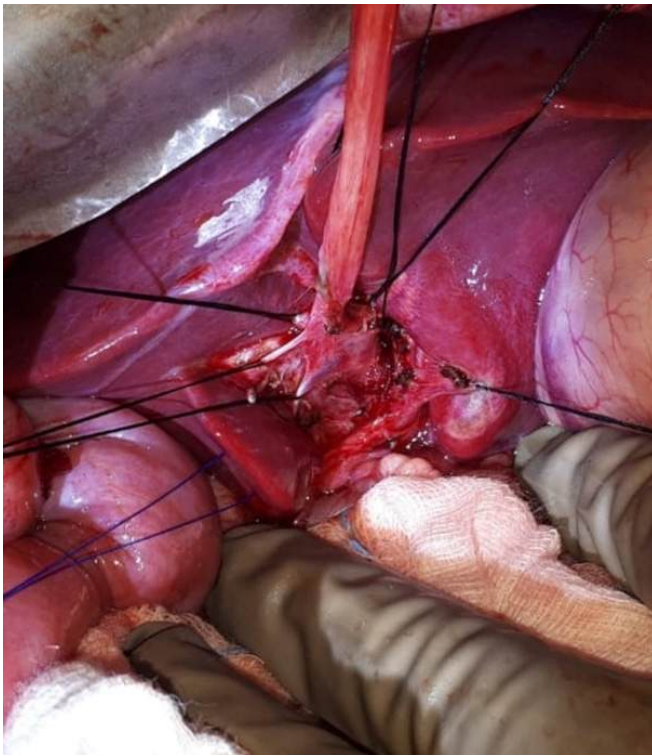


Fig 1. Rex Recessus dissection

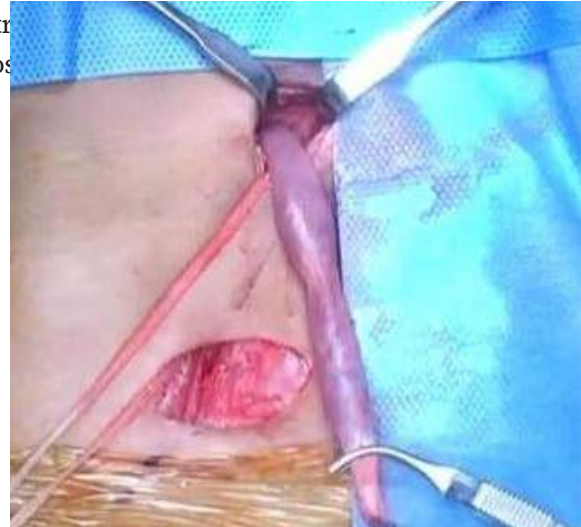


Fig 2. Harvest of IJV

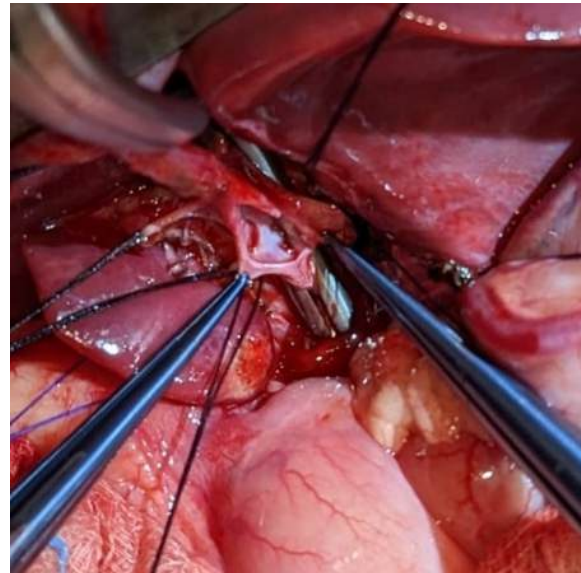


Fig 3. Left portal vein laid open



Fig 4. Final anastomosis (distal end).

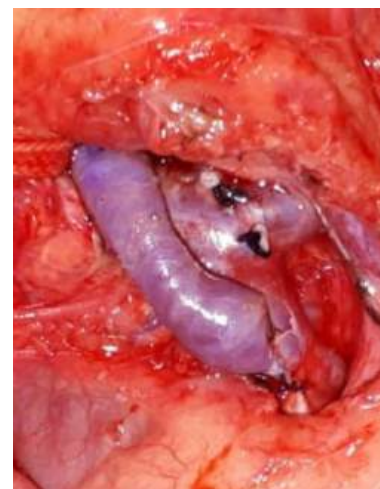


Fig 5. Final anastomosis (proximal end)

Our experience:

A retrospective review was performed of all children who underwent MRB between January 2013 and May 2024 in our center. Children with EHPVO and post pediatric liver transplantation with portal vein thrombosis were considered for Meso Rex bypass. In EHPVO, children were considered for MRB if they had preserved liver function, asymptomatic or minimally symptomatic splenomegaly, mild-to-moderate hypersplenism, a patent superior mesenteric vein, and radiological or evidence suggestive of a patent left portal vein within the Rex recessus. Patients with imaging findings suggestive of a patent Rex recessus underwent exploration with the intention of performing MRB. When intraoperative findings demonstrated unsuitable anatomy, alternative shunt procedures were performed.

In pediatric liver transplant cases, children with portal vein thrombosis (post transplant) or very narrow / short patient portal vein were considered for a meso rex bypass.

A total of 13 Meso-Rex bypass procedures were performed, 10 in children with EHPVO and 3 in liver transplant babies. During the study period, 83 shunt procedures were performed for EHPVO. These included 30 central shunts, 43 spleen-preserving shunts, and 10 Meso-Rex bypass procedures. Seventeen children fulfilled the preoperative criteria for MRB. However, the procedure was feasible in only 10 patients. Thus, although approximately one-fifth of patients appeared suitable on preoperative evaluation, only 12% of all children undergoing shunt surgery ultimately received a Rex bypass(1) .

The cohort comprised eight boys and two girls. The mean age at surgery was 4.5 years (range 1–11 years), and seven children were younger than five years. Children younger than five years were

significantly more likely to undergo successful portal reconstruction than older children. In contrast, older children more commonly required conventional shunt procedures because of unsuitable portal venous anatomy or significant hypersplenism/ massive splenomegaly mandating splenectomy.

In liver transplant patients, the portal vein anastomosis was done with native portal vein in all cases. In the immediate post-operative period some babies developed portal vein thrombosis primarily due to unhealthy portal vein, short & small calibre portal vein. The portal vein anastomosis was redone with the Meso-Rex bypass using the IJV. The proximal anastomosis was done at the confluence of splenic and SMV and the distal anastomosis was done to the graft portal vein.

Early postoperative outcomes were favorable. Two children developed transient ascites that resolved spontaneously. No patient experienced graft thrombosis or other major perioperative complications.

The mean follow-up duration was 67 months. One child developed recurrent hematemesis two years after surgery due to stenosis at the portal venous end of the graft. This was successfully managed by endovascular balloon dilatation. At the latest follow-up, all patients had patent bypasses and no patient required shunt revision or conversion to another procedure.

Importantly, hypersplenism resolved in all children with EHPVO. No recurrent portal hypertensive complications were observed in the remaining patients.

Discussion:

The principal advantage of MRB is that it restores physiological portal venous flow to the liver(2). Unlike conventional portosystemic shunts, which

divert portal blood into the systemic circulation, MRB re-establishes normal hepatopetal flow while simultaneously relieving portal hypertension. This unique feature explains the increasing acceptance of MRB as the preferred surgical strategy for EHPVO.

Our experience also demonstrates that radiological suitability does not always translate into operative feasibility. Although 17 children appeared suitable based on preoperative imaging, only 10 ultimately underwent successful MRB. Determining the patency and adequacy of the intrahepatic portal venous system remains challenging despite advances in imaging. Consequently, the final decision regarding feasibility is often made intraoperatively.

One of the most important findings of this series is the strong association between younger age and successful portal reconstruction. The majority of children who underwent MRB were younger than five years. This observation supports growing evidence that prolonged portal vein obstruction may lead to progressive obliteration of the intrahepatic portal venous system, reducing the likelihood of successful reconstruction as children grow older. This finding has important implications for countries such as India, where delayed referral remains common. Early identification and referral of children with EHPVO may increase the proportion of patients eligible for MRB(3).

The transplant group represents a different but important application of MRB. In these children, MRB was not used as the primary portal reconstruction but as a salvage procedure after failure of the native portal venous anastomosis. A small, short or unhealthy portal vein resulted in early portal vein thrombosis, and the mesenteric

venous system provided an alternative source of portal inflow.

Our results also demonstrate that MRB effectively addresses the clinical manifestations of portal hypertension. Resolution of hypersplenism was observed in all patients, and recurrent bleeding was uncommon. These findings are consistent with published reports showing improvement in hematological parameters, reduction in splenic size, and restoration of normal portal hemodynamics following successful MRB(4,5).

Rather than focusing solely on technical aspects, our experience emphasizes the importance of patient selection and timely referral. In our practice, the greatest determinant of success was not operative technique but the availability of suitable portal venous anatomy at the time of evaluation.

Conclusion:

Meso-Rex bypass remains the only physiological and potentially curative surgical treatment for children with EHPVO. However, its applicability is limited by strict anatomical requirements, making careful patient selection essential. Only a minority of children with EHPVO are ultimately suitable candidates, despite favorable preoperative imaging. Meso-Rex bypass is a good option in liver transplant babies with poor portal vein.

Younger children appear significantly more likely to undergo successful portal reconstruction, highlighting the importance of early diagnosis and referral. When feasible, MRB provides durable long-term patency, resolution of hypersplenism, effective control of portal hypertension, and restoration of physiological portal venous flow.

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Senior Consultant
Paediatric Surgery



Dr. M. M. Zameer — Giving Tiny Lives a Bigger Chance

Dr. M. M. Zameer is an accomplished paediatric surgeon whose career combines technical excellence with compassionate care for some of medicine's smallest and most vulnerable patients.

After completing MS General Surgery at Government Medical College, Nagpur, he secured first rank in the All-India entrance examination for M.Ch. Paediatric Surgery at PGIMER, Chandigarh, where he was also honoured as the **Best Outgoing Resident**.

Today, as a Senior Consultant at **Narayana Health, Bengaluru**, his expertise encompasses neonatal surgery, minimally invasive and robotic surgery, paediatric urology, airway reconstruction, and organ transplantation.

Beyond surgical skill, Dr. Zameer represents the very spirit of paediatric surgery—precision tempered by compassion and courage strengthened by hope.

His greatest achievements are ultimately not operations or accolades, but children given another chance to grow, laugh, dream and live their lives to the fullest.



Dr. Solomon Victor and Surgery for Budd Chiari Syndrome

How a cardiac surgeon in Madras helped turn hepatic venous outflow obstruction into a problem of anatomy, pressure and reconstructive possibility

A historical and clinical article

Prepared from the supplied briefing document and published medical sources

A Surgeon at the Border of the Heart and Liver

Budd-Chiari syndrome presented Solomon Victor with the kind of problem that defined his surgical imagination. The disease begins in the venous drainage of the liver, but its operative solution can require command of the inferior vena cava, the diaphragm, the right atrium and the portal circulation. In Madras during the late 1970s and 1980s, Victor worked with gastroenterologists, radiologists and fellow surgeons to study patients whose hepatic venous outflow was blocked at, or near, the hepatic segment of the inferior vena cava. Their work included mesoatrial decompression, direct repair of caval narrowing, and cavoatrial bypass. It belongs to an era when cross-sectional imaging was limited, interventional radiology was still developing, and liver transplantation was not a realistic option for most Indian patients.

Victor did not invent the mesoatrial shunt. John L. Cameron and William C. Maddrey had reported the operation in 1978, connecting the superior mesenteric vein to the right atrium with a Dacron graft when thrombosis of the inferior vena cava made conventional portosystemic shunts unusable. What Victor and his Madras colleagues

contributed was an Indian program of anatomical diagnosis and tailored reconstruction. They treated Budd-Chiari syndrome not as one uniform lesion, but as a family of obstructions that demanded different operations. Their published record shows a surgeon moving between the portal system and the great veins, choosing a bypass when the obstruction could not be opened and a direct repair when it could.

That distinction matters. A mesoatrial shunt diverts portal blood around the congested liver and obstructed cava to the right atrium. A cavoatrial bypass restores a route for caval blood around an obstructed hepatic segment of the inferior vena cava. Venous isthmusplasty enlarges a short, shelf-like narrowing by excision and patch reconstruction. Victor explored all three strategies because the anatomy of hepatic venous outflow obstruction in South Asia often differed from the thrombotic pattern then emphasized in Western literature.

The Disease Behind the Operation

Budd-Chiari syndrome is defined by obstruction of hepatic venous outflow anywhere from the small hepatic veins to the junction of the inferior

vena cava and right atrium. Blood can enter the liver through the portal vein and hepatic artery but cannot leave normally. The result is sinusoidal congestion, rising portal pressure, enlargement and tenderness of the liver, ascites, and, if the process continues, fibrosis, cirrhosis or liver failure. The clinical course ranges from an acute catastrophic illness to a chronic disorder that develops over months or years.

The anatomy determines the operation. When the hepatic veins are occluded but the inferior vena cava remains usable, a conventional portocaval or mesocaval shunt may decompress the liver. When the hepatic segment of the cava is also severely narrowed or blocked, those operations may drain into a high-pressure or inaccessible venous channel and can thrombose. The right atrium then becomes



the nearest dependable low-pressure destination. This is the logic of both the mesoatrial shunt and the cavoatrial bypass, though the source vessel and physiological purpose are different.

Modern accounts describe thrombotic disorders as major causes of Budd-Chiari syndrome, particularly in Western populations. Historical Asian and African series contained a larger proportion of webs, stenoses and obliterative lesions of the suprahepatic inferior vena cava. Victor and his colleagues used the term coarctation of the inferior vena cava for the severe hourglass constrictions they saw directly at operation. Their emphasis on operative morphology was important: a lesion thought to be a thin membrane might

actually be a longer constricted segment requiring patch enlargement or bypass.

A Collaborative Madras Program

Victor's work grew out of collaboration at the Government General Hospital and Madras Medical College. The published papers repeatedly pair his name with the gastroenterologist N. Madanagopalan and colleagues including V. Jayanthi, I. Kandasamy and others. A later Madanagopalan oration describes the program as a coordinated effort among gastroenterology, cardiac surgery and radiology. Bidirectional cavography, hepatic venography, functional hepatography and pressure measurements were used to locate the obstruction and decide whether the patient needed decompression, direct reconstruction or bypass.

This multidisciplinary structure was not incidental. The surgeon needed to know whether the portal vein was patent, whether the hepatic veins could be entered, how long the caval obstruction extended, and whether the liver had already sustained irreversible damage. Radiology mapped the venous channels; gastroenterology defined the clinical syndrome and liver reserve; cardiac surgery provided safe access to the right atrium and the thoracic segment of the cava. Victor's unusual breadth - he had trained in both cardiovascular surgery and cardiology - suited a disease in which diagnosis, hemodynamics and reconstruction were inseparable.

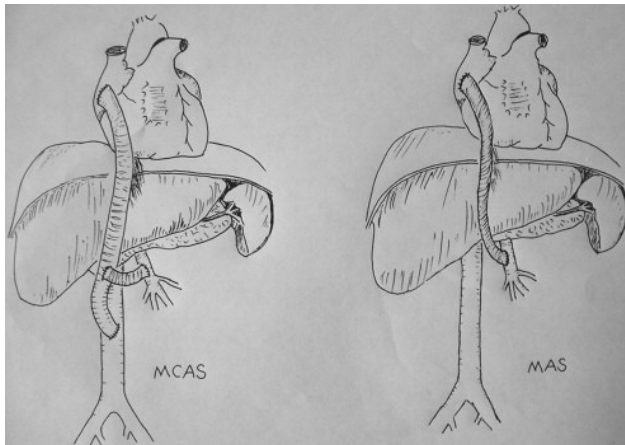
The Mesoatrial Shunt

Why the operation was necessary

The original mesoatrial concept was elegantly direct. If the cava below the right atrium could not accept the flow from a conventional shunt, blood from the portal system could be carried past the obstruction in a long prosthetic conduit. The graft began at the superior mesenteric vein and ended in

the right atrium. By lowering portal and sinusoidal pressure, it aimed to relieve ascites, reduce hepatic congestion and preserve functioning liver tissue.

A 1983 Indian series by P. Khanduri and colleagues described three patients with hepatic vein occlusion and significant obstruction of the hepatic inferior vena cava. Wide-lumen woven Dacron grafts were used. All three patients had a striking reduction in liver size and resolution of ascites. Patency was confirmed by angiography in two and Doppler examination in one. Two patients later died, at five and sixteen months, but both grafts were widely patent at autopsy and lined by neointima; the third patient remained asymptomatic and free of ascites at twenty months. This paper is the source



of several outcome statements in the supplied briefing document. It documents the wider Indian experience with the operation, but its indexed author list does not include Victor.

How the graft worked

The conduit had to bridge two body compartments and two very different vessels. At its abdominal end, it received blood from the superior mesenteric vein, a major contributor to portal flow. It then travelled upward, typically behind the xiphoid and sternum, entered the chest and joined the right atrium. A generous diameter was essential because a long prosthetic graft in a low-pressure venous system is vulnerable to thrombosis. The pressure difference between the portal system and the right

atrium supplied the driving force.

The substernal passage created a mechanical weak point. Cameron, Kadir and Pierce reported in 1984 that the graft could be compressed where it passed behind the xiphoid and sternum. Their modification bonded an external silicone cuff to a Dacron or expanded polytetrafluoroethylene graft at that point. Later series used externally supported grafts and emphasized careful routing, avoidance of kinking and postoperative surveillance. These refinements show why the mesoatrial shunt was more than a pair of vascular anastomoses: its success depended on the entire three-dimensional course of the conduit.

Victor's Reconstructive Path

The most clearly documented part of Victor's Budd-Chiari work is his reconstruction of the obstructed inferior vena cava. In 1983, Victor and colleagues published a case of venous isthmusplasty in a forty-year-old man with an hourglass obstruction of the suprahepatic cava. Through a posterolateral thoracotomy they exposed the lesion, opened the vessel under proximal and distal control, excised a shelf of fibrous tissue and closed the venotomy with a Gore-Tex patch. The cava was patent in the immediate postoperative period. This was anatomy-specific surgery: instead of bypassing a short focal obstruction, the team enlarged the native channel.

Their larger 1986 report described eight patients with chronic Budd-Chiari syndrome caused by caval coarctation. Transatrial dilatation failed in the first patient. In the next seven, a transthoracic, transdiaphragmatic and retroperitoneal approach allowed direct visualization of severe hourglass narrowing just above the right hepatic vein. The surgeons found no inflammation, external compression or thrombosis. Early attempts with antibiotic-sterilized aortic homografts failed in three patients. Victor's team then used

polytetrafluoroethylene grafts for retrohepatic cavoatrial bypass. Five patients, including one whose homograft had failed, had successful prosthetic bypasses and remained free of recurrent symptoms during follow-up ranging from twenty-one months to six years.

The paper is valuable not only for its results but for its surgical reasoning. The failures of homograft material led to a more durable prosthetic solution. Direct observation led the authors to challenge the term membranous obstruction and prefer coarctation, a word that better described a constricted segment. In 1991 Victor and colleagues reported a dorsal cavoatrial bypass using an aortic-occlusion technique, continuing their effort to simplify control of the cava and make the bypass safer.

Imaging Before and After Surgery

Victor's group also helped move follow-up away from symptoms alone. In 1988, Jayanthi, Victor and

colleagues reported real-time ultrasound in nine patients with Budd-Chiari syndrome caused by caval coarctation. Ultrasound aided preoperative diagnosis and confirmed postoperative patency in five dorsal cavoatrial bypass grafts and one isthmusplasty. This joined anatomical imaging to the operative plan and gave clinicians a non-invasive way to monitor reconstruction.

The point was practical. A patient could lose ascites and feel better while a long venous graft was narrowing; conversely, persistent postoperative symptoms might have causes other than graft failure. Doppler and ultrasound could assess flow directly. Later investigators used cavography, mesenteric arterial portography, catheter pressure measurements, computed tomography and Doppler surveillance to examine mesoatrial shunts. The Madras experience anticipated the modern expectation that the success of a vascular reconstruction must be demonstrated, not assumed.

Choosing Among Operations

Operation	Anatomical problem	Purpose and route
Mesoatrial shunt	Hepatic venous obstruction with an inferior vena cava that cannot receive a conventional portosystemic shunt	Decompresses the portal and hepatic sinusoids by carrying blood from the superior mesenteric vein to the right atrium
Cavoatrial bypass	A long or inaccessible obstruction of the hepatic or suprahepatic inferior vena cava	Restores systemic venous return around the blocked caval segment by connecting patent cava to the right atrium
Venous isthmusplasty	A short focal shelf or coarctation that can be exposed and opened safely	Removes or divides the obstruction and enlarges the native cava with a patch

Angioplasty and stenting	A short web or stenosis suitable for catheter treatment	Restores native hepatic or caval outflow without an open bypass; now preferred when technically feasible
TIPS or DIPS	Persistent portal hypertension when hepatic venous outflow cannot be adequately restored	Creates an intrahepatic portal to systemic channel; DIPS connects the portal vein directly to the cava when hepatic veins cannot be used
Liver transplantation	Fulminant failure, decompensated cirrhosis or failure of decompressive treatment	Replaces the diseased liver and its obstructed outflow pathway

No one operation is universally correct. The value of Victor's work lies in the discipline of matching the reconstruction to the obstruction. A patent portal vein, recoverable liver function and an adequate pressure gradient were prerequisites for decompression. Extensive fibrosis, fulminant failure or thrombosis of critical inflow vessels could make a shunt ineffective or dangerous. These judgments remain recognizable today even though the available technology has changed.

Results and Limits of the Historical Evidence

The early literature consists mainly of case reports and small series. It documents physiological success - lower pressure, reduced liver size, resolution of ascites and demonstrable graft flow - but it cannot provide the certainty of a randomized trial. Patients were highly selected, techniques changed during a series, anticoagulation practices varied, and long-term graft patency was difficult to measure uniformly. Death after a technically patent shunt also showed that restoring flow could not reverse advanced hepatic injury or every systemic consequence of the disease.

Even so, later reports support the biological premise. A British series of five patients published in 1989 found resolution of ascites, diminished liver size and patent externally supported polytetrafluoroethylene mesoatrial grafts during nine to sixteen months of follow-up. An Indian series of ten patients treated between 1994 and 2000 reported one death; all grafts were patent over a mean follow-up of forty months, and the survivors were symptom-free with normal liver function at publication. These results do not make the operation routine, but they show that long prosthetic venous conduits could work when diameter, pressure gradient, routing and surveillance were favorable.

From Open Shunts to Endovascular Therapy

The treatment pathway for Budd-Chiari syndrome has since shifted. Anticoagulation and treatment of the underlying prothrombotic disorder form the medical foundation. Short hepatic-vein or caval stenoses may be treated with angioplasty and stenting. When direct recanalization is impossible, transjugular intrahepatic

portosystemic shunts and direct intrahepatic portocaval shunts can decompress the portal system without a thoracoabdominal operation. Liver transplantation is reserved for fulminant disease, advanced decompensation or failure of other measures.

This change does not make Victor's surgery irrelevant. Contemporary catheter interventions follow the same hemodynamic logic: locate the obstruction, restore or bypass the blocked route, lower hepatic congestion and preserve the liver before irreversible damage occurs. Modern imaging provides a map that Victor's team had to assemble from venography, ultrasound, pressure tracings and direct surgical inspection. The long graft from abdomen to atrium has become uncommon, but the questions that justified it remain central.

The Surgeon Beyond the Shunt

Published tributes describe Victor as a leading Indian cardiac surgeon whose interests extended well beyond the operating theatre. He trained at Stanley Medical College and Madras Medical College, pursued advanced cardiovascular training abroad, and returned to academic surgery in Madras. Colleagues remembered his meticulous clinical work, his command of cardiac catheterization and morphology, and his willingness to design affordable solutions for patients who could not reach expensive private care. He later established a heart institute with a strong charitable purpose.

His Budd-Chiari work expresses those traits in concentrated form. It was anatomically exact, collaborative and resource-conscious. The patients were often young and severely disabled

by ascites and venous congestion. Transplantation was remote; endovascular treatment was not yet mature. Victor's answer was neither a single celebrated operation nor a claim of solitary invention. It was a sequence of observations, technical adaptations and published follow-up: expose the lesion, measure the pressure, preserve the native channel when possible, bypass it when necessary, and verify the result.

A Careful Historical Judgment

Solomon Victor deserves recognition as an important Indian surgeon-investigator in Budd-Chiari syndrome, particularly for the operative treatment of inferior vena caval coarctation and for developing a practical repertoire that included atrial bypass procedures. The evidence supports describing him as a pioneer of surgical management in India, not as the inventor of the mesoatrial shunt. His achievement was to make an unusual operation part of a broader, anatomy-led program and to connect the skills of hepatology, radiology and cardiac surgery around patients who otherwise had few options.

That is a more durable legacy than priority alone. The Madras program helped show that hepatic venous outflow obstruction could be dissected into treatable anatomical patterns. Its reports also recorded the limits of materials, the importance of graft geometry, the value of non-invasive surveillance and the need to operate before chronic congestion destroyed the liver. Today's stents, TIPS and DIPS procedures are technologically different, but they pursue the same objective that guided Victor: give the trapped blood a safe route back to the heart while there is still a liver worth saving.

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Interventional Radiology for Variceal Bleeding: A Modern Approach to Portal Hypertension

Authors: Dr. Ranjan K Patel, Dr. B D Pattnaik.

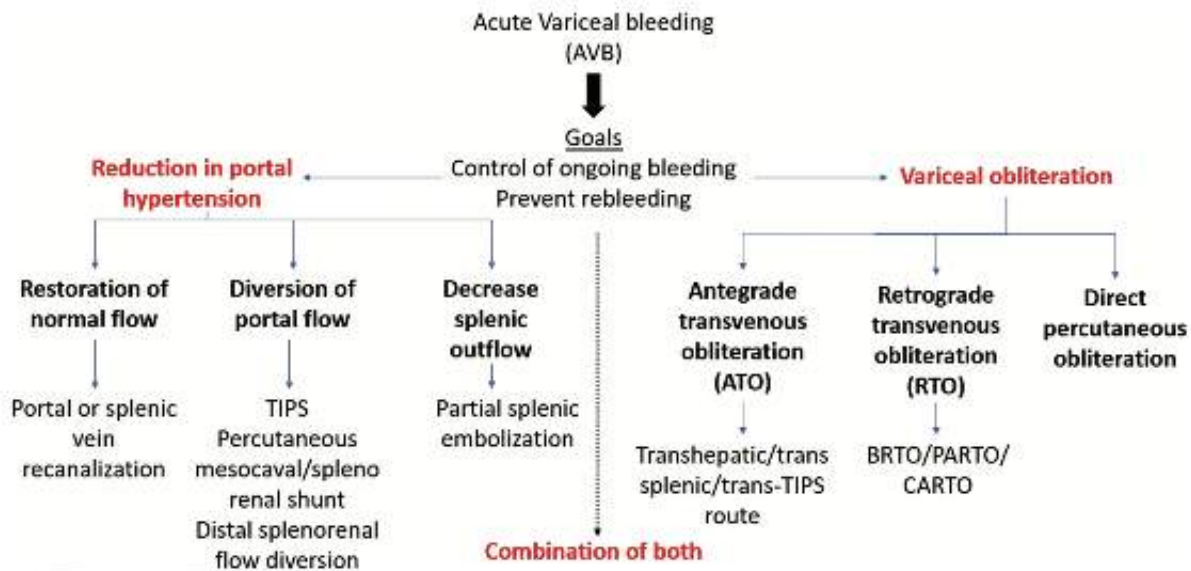
Anatomy, haemodynamics and precision endovascular therapy are transforming the management of complex variceal haemorrhage

Acute variceal bleeding is one of the most dramatic and potentially fatal complications of portal hypertension. Despite advances in pharmacological and endoscopic therapy, mortality remains substantial, particularly in patients with advanced liver dysfunction. Initial management requires rapid resuscitation, vasoactive drugs, antibiotics and urgent endoscopic haemostasis.

the culprit varix.

Cross-sectional imaging is therefore the first step in planning. Contrast-enhanced CT or MRI maps portal and splenic vein patency, portosystemic collaterals, variceal inflow and outflow, and spontaneous shunts. The anatomy often determines the intervention before the catheter ever enters the vessel.

TIPS: Decompression as Definitive Therapy

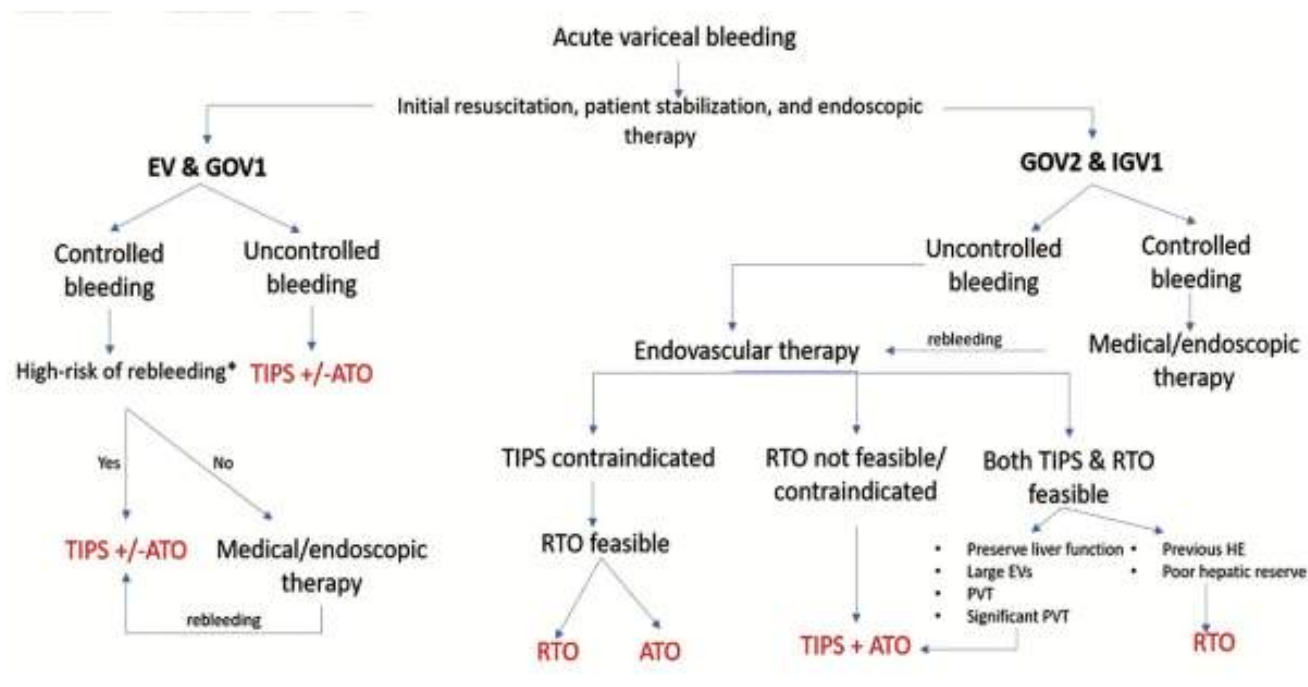


However, when bleeding persists, recurs early, or conventional therapy is anatomically unsuitable, interventional radiology (IR) becomes central to definitive haemostasis.

The contemporary IR approach is no longer synonymous with simply performing a TIPS. It is based on two fundamental principles (Fig 1 & 2): Decompress the portal system or directly obliterate

Transjugular intrahepatic portosystemic shunt (TIPS) creates a low-resistance communication between the portal and systemic venous circulation, reducing the pressure driving variceal haemorrhage. Its role has expanded from a rescue procedure to three distinct clinical scenarios:

Salvage TIPS – uncontrolled bleeding despite medical and endoscopic therapy.



Rescue TIPS – early rebleeding following initial haemostasis.

Preemptive TIPS – early intervention in patients at particularly high risk of treatment failure or recurrent bleeding.

In refractory bleeding, salvage TIPS achieves bleeding control in approximately 80–100% of patients. However, mortality remains high when severe hepatic dysfunction, renal failure, infection or haemodynamic instability coexist. Thus, **technical feasibility must always be separated from clinical suitability**.

Timing also matters. Data from a recent series demonstrated substantially better outcomes when rescue TIPS was performed within the first 8 hours of refractory bleeding compared with later intervention.

How much decompression is enough?

The objective is not simply to reduce portal pressure as much as possible. A post-TIPS portosystemic pressure gradient (PSPG) around **12 mm Hg** is an important haemodynamic reference point.

Further reduction decreases rebleeding risk but may increase hepatic encephalopathy and liver failure. Shunt diameter—8 or 10 mm—should therefore be individualized according to hepatic reserve, bleeding control and encephalopathy risk.

Preemptive TIPS has demonstrated a reduction in early rebleeding in selected high-risk patients, although evidence regarding an overall survival advantage remains inconsistent. The decision should therefore remain **patient-specific and multidisciplinary** rather than algorithmic.

When Obliteration Is Better Than Decompression

Not every bleeding varix requires a TIPS. For gastric and selected ectopic varices, particularly when a suitable systemic drainage pathway is present, retrograde transvenous obliteration (RTO) can provide direct and definitive variceal control without creating an additional portosystemic shunt.

The RTO family includes:

- BRTO – balloon-occluded retrograde transvenous obliteration

- PARTO – plug-assisted retrograde transvenous obliteration (Fig 3)
- CARTO – coil-assisted retrograde transvenous obliteration

The evolution from BRTO to PARTO and CARTO has made variceal obliteration faster and more adaptable. PARTO replaces prolonged balloon occlusion with a vascular plug, while CARTO uses

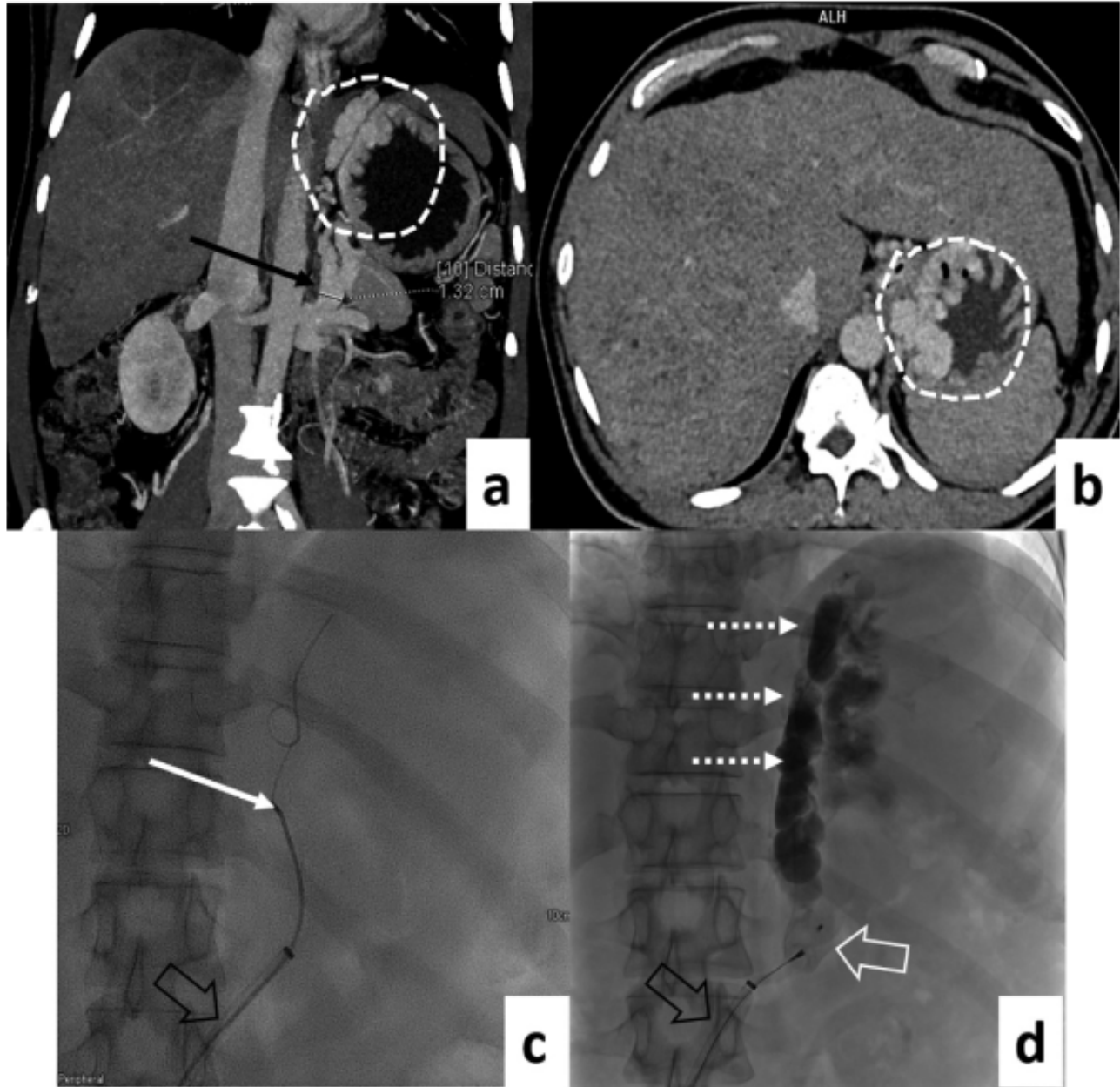


Figure 3: PARTO for bleeding gastric varices. CT portography (a, b) shows large fundal varices with a 13-mm gastrosplenic shunt. (c) Right femoral access was used to catheterize the shunt and variceal complex. (d) The shunt was occluded with a 16-mm vascular plug, followed by injection of 10 mL lipiodol-sclerosant-air mixture (1:2:3), achieving complete hemostasis.

These techniques are particularly useful for GOV2 and IGV1 cardiofundal varices associated with a gastrosplenic or gastrocaval shunt.

coils and embolic material and can accommodate larger or tortuous shunts. IR in variceal bleeding

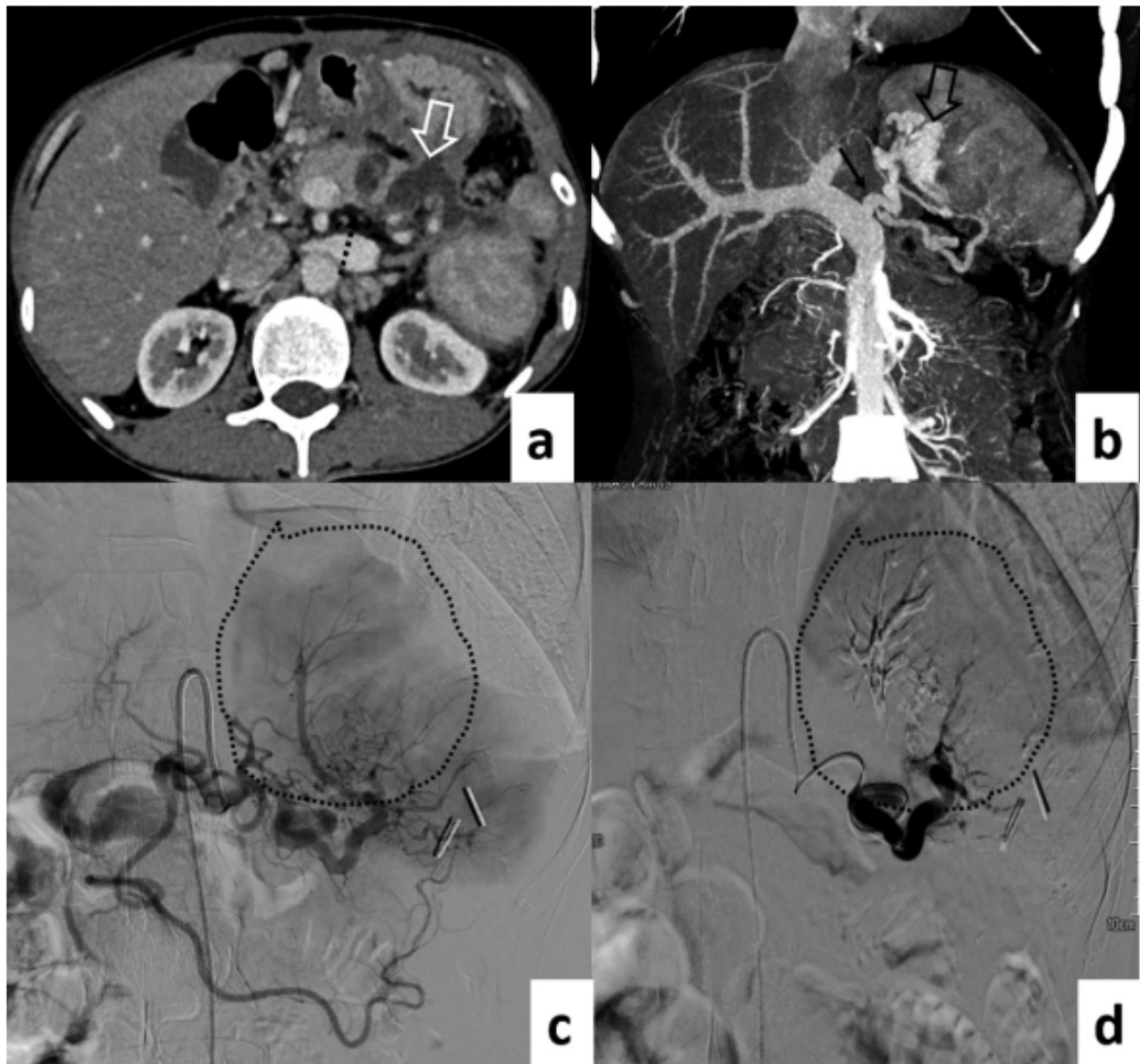


Figure 4: Partial splenic embolization for recurrent gastric variceal bleeding. CT shows splenic vein occlusion with fundal varices (a, b). Splenic angiography demonstrates a dilated splenic artery (c), with selective embolization of upper and midpole branches using 25% glue–lipiodol (d). No further bleeding occurred.

Importantly, RTO does not create additional portosystemic shunting and therefore avoids the encephalopathy risk associated with TIPS. However, occluding a spontaneous shunt may increase portal pressure and subsequently worsen oesophageal varices, ascites or hydrothorax.

When RTO Is Not Possible: Antegrade Obliteration

A suitable systemic outflow shunt is essential for RTO. When such a pathway is absent or inaccessible, antegrade transvenous obliteration (ATO) provides an alternative.

Varices can be approached through the transhepatic, transsplenic or trans-TIPS route, followed by embolization of the varix and its feeding veins using coils, glue, gelfoam, sclerosants

or vascular plugs. ATO is particularly useful for gastric, oesophageal and ectopic varices when RTO is anatomically impossible and TIPS is unsuitable. For ectopic varices, treatment must be tailored to the inflow and outflow anatomy. Duodenal, jejunal, colorectal and stomal varices may cause massive bleeding while remaining inaccessible or unsuitable for conventional endoscopic therapy. TIPS with adjunctive embolization, ATO, RTO, direct embolization or combinations thereof may be required IR in variceal bleeding

Extrahepatic portal vein obstruction: When feasible-Restore, Don't Bypass

One of the most important conceptual shifts in IR is recognizing that **variceal bleeding does not necessarily imply cirrhosis**.

In **extrahepatic portal vein obstruction (EHPVO), chronic portal vein thrombosis and portosinusoidal vascular disease**, the therapeutic objective may be restoration of physiological portal flow rather than creation of an additional portosystemic shunt.

Portal Vein Recanalization (PVR)

PVR can restore portal venous flow and decompress collaterals without the long-term consequences of a portosystemic shunt. In appropriately selected patients with non-cirrhotic portal vein obstruction, PVR—with or without TIPS—can therefore be an attractive strategy.

During acute bleeding, **PVR or PVR-TIPS may be combined with simultaneous variceal embolization**, providing both restoration of portal flow and immediate control of the bleeding collateral.

The Special Case of Left-Sided Portal Hypertension

Isolated fundal varices in a patient without cirrhosis particularly in the setting of pancreatitis or pancreatic disease should raise suspicion for **left-sided (sinistral) portal hypertension** due to splenic vein obstruction.

Here, conventional TIPS does not address the underlying haemodynamic abnormality. Treatment may require **splenic vein recanalization/stenting, variceal embolization or partial splenic embolization (Fig 4)**, depending on the anatomy.

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Mine

Work Life Balance across a Lens

In the demanding world of medicine and even more so in high stakes Surgical gastroenterology, burnout rates are high and over time emotional reserves deplete! We all need to discover our own antidotes - intentionally or by accident. Some of us find solace only in work-others have too many activities. I veer towards the latter but clearly the winner is the unexpected sanctuary through my camera lens—not in the operating theatre, but in the quiet pursuit of wildlife photography especially Birds.

Bird photography chose me, perhaps because it satisfies something that surgery cannot. In the operating room, we control outcomes through skill and judgment. With nature, we surrender control entirely. The fauna appear on their own terms, behave unpredictably, and remind us that not everything can be planned or perfected. This humility is therapeutic.

The passion began serendipitously - I normally love cityscapes, architecture and classical art especially medieval, so I chose my first serious foray into

wilderness on a whim! An email itinerary of wildlife photographers showed a hot air Balloon ride over the great crossing at Masai Mara, Kenya on my fiftieth birthday and seduced me into signing up! Mission was accomplished to get the 'herd' mentality of millions of wildebeest crossing the Mara river on video replete with the crocodile attack. But what really took my breath away was the vivacity and variety of colours on the birds that were so easy to spot. Rest as they say is History. And Geography too - my likely subject if I hadn't taken up medicine - the passion took me to all the remote jungles of the world. From Sub Himalayan Terai undulant to Central African Savanna. Former headhunters paradise in Papua (island of New Guinea) to get birds of paradise displaying in monsoon to aboriginal Andamans. Andes in Columbia for rare hummingbirds to unusual primates and hornbills in Borneo. Western Ghats to highlands and islands in Thailand! Even every of India I may not have bothered to visit despite a transferable army childhood and continued adult passion for travel.

Most Sundays and holidays in Cochin - I woke up much earlier than weekdays with Unni, Prasin and Sabarish - all ex students as UG and PG but



taught me in reverse much more in the backwaters tracking migratories and endemics. Going to Lucknow to conduct exams would catch me before the ward round - doing a perimeter of the SGPGI campus for Sarus cranes or rare eagles with Able there enabling it! It brought me closer to so many people as Prof. SK Mathur will call up asking was that your photo I saw on Crorepati? Prof. Vinay Kapoor will remember me in the midst of nature and send a photo of a bird he had spotted to identify it. Prof. Adarsh Chaudhary may never forgive or forget a trip to Dandeli forests in wild Karnataka but that is a story for another newsletter!



There is a peculiar parallel between what we do in surgery and what wildlife photography demands. Both require patience, observation, and an almost meditative focus. In the operating room, we dissect tissue planes with meticulous care, identifying structures that distinguish the safe from the dangerous. In the field, we wait—sometimes for hours—for that perfect moment when a kingfisher dives or a hornbill takes flight, our finger poised on the shutter just as our scalpel hovers before the critical incision.

Bird photography has taught me many lessons that have potential in my surgical practice:

Patience over haste. Waiting for the right

moment yields better results than forcing action. In surgery, this translates to knowing when to proceed and when to pause, when to dissect and when to reassess or reoperate.

Preparation meets opportunity. The best photographs happen when preparation intersects with chance. You cannot control when a bird appears, but you can ensure your camera is ready, your settings are correct, and your position is optimal without backlight etc. The same principle applies to managing complications—prepare for what you cannot predict.

Appreciation of the ordinary. Some of my most satisfying photographs are of common birds—mynas, sparrows, bee eaters—captured in extraordinary light or behavior. This has reminded me to find meaning in routine cases, not just the complex oncological resections that dominate academic discourse.

The value of failure. For every successful photograph, there are hundreds of missed opportunities—blurred wings, poor light, the bird flying away before the shot. Failure is not a verdict; it is data. Each missed shot teaches you something about timing, positioning, or settings. Surgical complications, similarly, are not merely adverse events but opportunities for learning and improvement.



Conservation and Responsibility

My passion for bird photography has also awakened a deeper environmental consciousness. Through my lens, I have witnessed habitat loss, declining populations, and the fragility of ecosystems. This mirrors what we see in healthcare—the consequences of environmental degradation on human health, from air pollution to water contamination. Many physician-photographers

improve mood. A 2024 study found that just thirty minutes of birdwatching could make people happier, healthier, and foster a deeper connection with nature—effects more pronounced than a simple nature walk. My wife joined for the only long trip abroad and said she finally understood what the Japanese call forest bathing!

For physicians specifically, the benefits are profound. A pilot study of mental health professionals found that ornithological walks



have channeled their hobby into advocacy. Dr. Ramakanta Panda, the renowned cardiac surgeon and Padma Bhushan awardee, established the Asian Wildlife Trust to support conservation efforts, driven by his wildlife photography. While my contributions are more modest, I have begun incorporating environmental health discussions and sustainability into my teaching, reminding residents that patient care extends beyond the hospital walls and wanting to reduce waste from disposables.

Nature's Prescription

The science behind what I experience intuitively is compelling. Birdwatching and nature photography have been shown to reduce anxiety, lower cortisol levels, decrease blood pressure, and

significantly improved mood and reduced subjective stress levels, even in unfavourable weather conditions. “Therapeutic ornithology” would be a better detox than the usual ethanolic binges in conferences we are more used to! Drs Sanjiv and Neebha Haribhakti were visiting me in Rishikesh and while taking them to the various holy places half apologising for my lack of religiousness - but she chided me and said clearly you are not aware of your own inner spirituality - when you are watching birds, you are meditating close to nature! You don't need religion - act on your instincts and I've lived upto that advice for sure!



And surprisingly instead of diverting me from surgery every foray into the forest makes me get back from the field with renewed vigour. So when colleagues sometimes ask whether

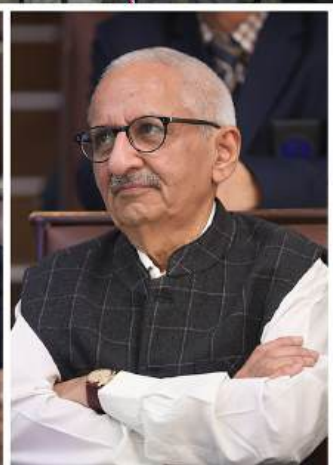
I am “wasting time” on a hobby when clinical demands are so high, I reply - I worry more about what happens when I don’t make this time. The irritability, the mental fog, the emotional distance from patients and family—all of these recede when I return from the field with a memory card full of images and a mind full of stillness. Medicine needs whole humans, not exhausted functionaries. Our patients deserve doctors who are mentally present,

emotionally available, and genuinely engaged with life beyond the hospital walls. Hobbies are not distractions from our work—they are investments in our capacity to do that work well, sustainably, and with joy.

In the operating room, we save lives. In the field, we reclaim ours. In the clinic, we heal others. In nature, we heal ourselves.



- Dr. Puneet Dhar





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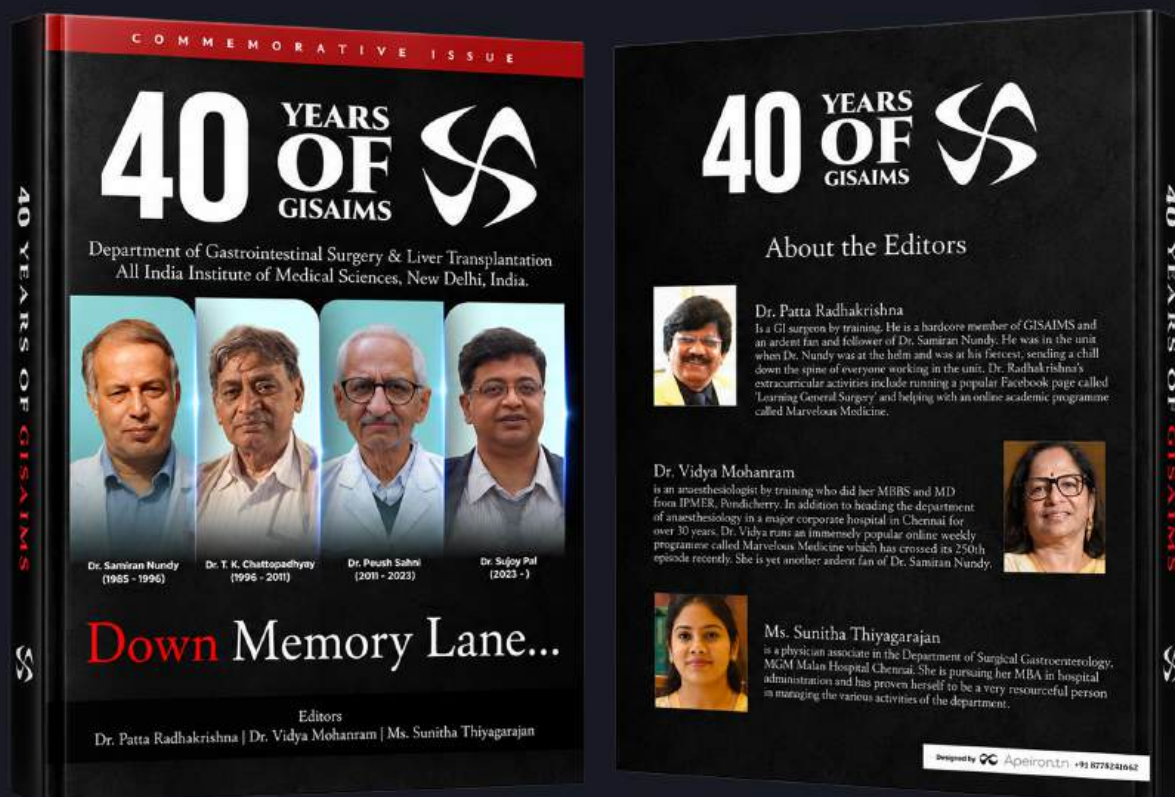


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40 Years of GISAIIMS

A Journey Through Four Decades of Gastrointestinal Surgery



40 Years of GISAIIMS is a commemorative journey documenting four decades of the Department of Gastrointestinal Surgery at AIIMS, New Delhi. The book brings together the department's history, its pioneering surgeons, generations of trainees, landmark clinical work, research, teaching, and the evolution of gastrointestinal surgery in India.

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