SASLT
الجمعية السعودية لأمراض وزراعة الكبد
Saudi Society for the Study of Liver Disease and Transplantion

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SASLT NEWSLETTER

16TH ISSUE 2026

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WELCOME TO THE 16TH EDITION OF THE SASLT NEWSLETTER!

Dear SASLT Community,
Welcome to the 16th issue of the SASLT Newsletter. We are pleased to share another edition dedicated to strengthening the knowledge and collaboration of our hepatology community. Each issue reflects our shared commitment to advancing liver health through education, research, and clinical excellence.

In this edition, we focus on portal vein thrombosis (PVT), an increasingly important condition that continues to influence patient outcomes across multiple areas of hepatology. PVT presents significant diagnostic and therapeutic challenges and often requires a multidisciplinary approach involving hepatologists, radiologists, transplant specialists, and other healthcare professionals. Its impact on disease progression, portal hypertension, and transplantation highlights the importance of timely recognition and appropriate management.

This issue explores PVT in non-cirrhotic patients, including underlying risk factors, diagnostic considerations, and the role of early intervention. We also examine PVT in cirrhosis and its relationship with hepatic decompensation and liver transplantation. In addition, we discuss the unique presentation of PVT in pediatric patients and the importance of early diagnosis in supporting long-term liver health and development.

We sincerely thank the SASLT community for your continued dedication, collaboration, and contributions to advancing hepatology care across the Kingdom.

Best regards,

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- Consultant, Adult Transplant Hepatology.
- Liver & Small Bowel Health Centre Department.
- Organ Transplant Center of Excellence KFSHRC, Riyadh.

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ARTICLES

PORTAL VEIN THROMBOSIS IN NON-CIRRHOTIC LIVER PATIENTS: A HEMATOLOGIST PERSPECTIVE

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Portal vein thrombosis (PVT) refers to thrombotic occlusion of the portal vein and/or its tributaries, including super mesenteric and splenic veins. In the absence of cirrhosis or hepatocellular carcinoma, non-cirrhotic PVT (NC-PVT) represents a distinct clinical entity with unique pathophysiologic mechanisms, diagnostic considerations, and therapeutic challenges. Although the overall incidence of PVT is estimated to be <1%, the incidence of NC-PVT is substantially lower (~0.7 per 100,000 person-years), likely reflecting under-recognition due to silent or nonspecific presentations.1,2 Importantly, an underlying prothrombotic disease, either inherited or acquired, is identified in approximately 60 – 70% of patients with NC-PVT.3–5 Additional contributing factors include hormonal and hypercoagulable states, such as pregnancy and oral contraceptive use.

As illustrated in Figure 1, the major predisposing factors for NC-PVT can broadly be categorized into local/anatomical factors and systemic prothrombotic conditions. Local factors include intra-abdominal inflammatory processes, abdominal malignancies, surgery, trauma, and vascular abnormalities. Among systemic causes, myeloproliferative neoplasms (MPNs) remain the most commonly identified prothrombotic condition, accounting for approximately 20 – 30% of NC-PVT cases, particularly JAK2 V617F-positive polycythemia vera and essential thrombocythemia.6–9 Importantly, overt hematologic features may be absent at presentation. Patients may initially demonstrate normal blood counts due to splenic sequestration, plasma volume expansion, iron deficiency, or occult/latent MPN biology at the time of thrombosis. Accordingly, JAK2 V617F mutation testing should be considered mandatory in all cases of NC-PVT, regardless of blood count parameters. Furthermore, bone marrow biopsy should be considered in patients with persistent clinical suspicion for MPN despite a negative molecular testing.7,9,10 Clinically, NC-PVT may present acutely or chronically. Acute PVT commonly manifests with abdominal pain, nausea, or fever, while extension into the mesenteric circulation may result in intestinal ischemia or infarction. Chronic PVT often evolves insidiously into cavernomatous transformation and complications of portal hypertension. Notably, approximately 20% of cases are identified incidentally on imaging.2,6 A proposed diagnostic workup is summarized in Figure 2. Beyond radiologic confirmation, evaluation should focus on identifying both local

precipitating factors and systemic thrombophilic disorders. Assessment for underlying liver disease remains important, and liver biopsy may be considered.10

From a hematological standpoint, unexplained NC-PVT should prompt comprehensive evaluation for occult clonal disorders, particularly MPNs and paroxysmal nocturnal hematuria (PNH). In patients progressing to liver failure requiring transplantation, establishing an underlying clonal hematologic diagnosis may significantly influence post-transplant monitoring, thrombotic risk assessment, and long-term management strategies.[8]

Anticoagulation remains the cornerstone of therapy; however, management requires careful assessment of both thrombotic and bleeding risks. In acute symptomatic or extensive PVT, early initiation of anticoagulation is recommended to prevent thrombus propagation, bowel ischemia, and progressive portal hypertension, while also improving the likelihood of portal vein recanalization, particularly when initiated early after symptom onset.10 In chronic PVT, long-term anticoagulation is generally recommended in the presence of a persistent major prothrombotic condition. In patients with transient provoking factors, anticoagulation decisions should be individualized based on recurrence risk and bleeding profile.10 Evaluation for portal hypertension and esophageal varices is particularly important in chronic PVT before initiation of anticoagulation.

Low-molecular-weight heparin (LMWH) is commonly preferred as the initial anticoagulant, particularly during the acute phase. Long-term therapy may subsequently transition to vitamin K antagonists (VKAs) or direct oral anticoagulants (DOACs), with agent selection guided by the underlying etiology and patient-specific factors.10 For instance, VKAs remain preferred in antiphospholipid antibody syndrome (APLS). In parallel, treatment of the underlying prothrombotic disorder, including cytoreductive therapy in MPNs or complement inhibition in PNH, may further reduce the risk of recurrent thrombosis.

Thrombocytopenia related to hypersplenism and portal hypertension may further complicate anticoagulation decisions. Platelet transfusion thresholds should be individualized according to bleeding risk, thrombotic burden, and procedural needs. In many cases, anticoagulation can still be administered safely in patients with platelet counts of >50 x 109/L in the absence of active bleeding, although management should remain highly individualized.

In conclusion, NC-PVT requires a structured and multidisplinary approach involving hepatologists, hematologists, interventional radiologists, and when indicated transplant specialists. Given the high prevalence of underlying systemic prothrombotic disorders, particularly occult MPNs, hematologic evaluation plays a central role in both diagnosis and long-term management. Early recognition and targeted evaluation remain essential to optimize patient outcomes.

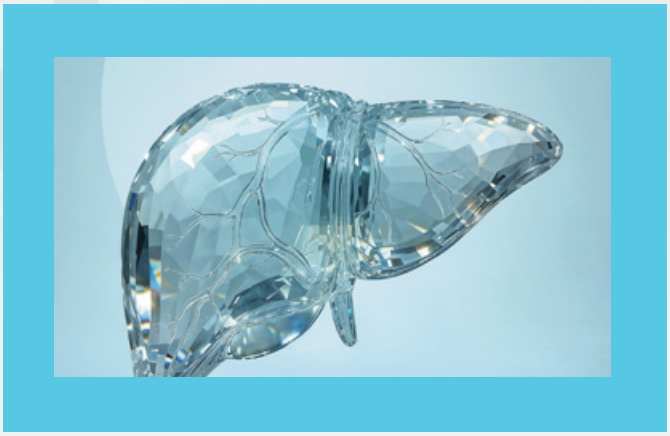

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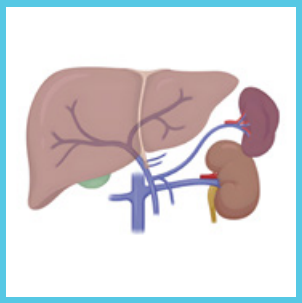
Figure 1: Predisposing factors for PVT in non-cirrhotic patients



Risk Factors for PVT in Non-cirrhotic Patients

Anatomical/Local Factors

- Central obesity
- Intra-abdominal inflammation (e.g. appendicitis, pancreatitis, cholecystitis, IBD)
- Abdominal malignancies
- Abdominal surgery/trauma
- Vascular anomaly/malformation

Prothrombotic States

- MPNS (JAK2 V617F + PV and ET)20-30%
- Inherited/Acquired thrombophilias (AT deficiency, Protein C & S deficiencies, FVL mutation, Prothrombin G20210A mutation) 10-25%
- APLS-5-15%
- PNH (rare)

Figure 2: Diagnostic algorithm for PVT in non-cirrhotic patients

- Anatomical delineation
- Extent of thrombosis
- Bowel viability
- Underlying pathology

Suspicion for PVT

↓

Doppler ultrasound

↓

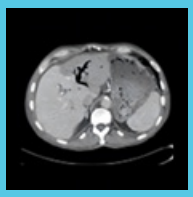
Features of PVT present

↓

Contrast-enhanced CT or MRI

↓

to assess for



Lab Workup

CBC with differential	Flowcytometry for PNH clone	Prothrombin G20210A mutation
Peripheral blood smear	Antiphospholipid workup	NGS Myeloid Panel
JAK2 V617F mutation	AT, Proteins C & S activity*	FVIII levels
CALR/MPL mutations (if JAK2 negative)	FV Leiden mutation	Homocysteine level

*AT activity, Protein C & S levels can be falsely low in the setting of acute thrombosis; repeat testing if often required

Indications for bone marrow biopsy:

- JAK2 V617F negative + persistent MPN suspicion
- JAK2 V617F negative + CALRIMPL negative
- Differentiating MPN subtypes and/or morphology assessment in MF
- Suspected other hematologic malignancies

ARTICLES

PORTAL VEIN THROMBOSIS: NAVIGATING THE COMPLEXITIES OF MANAGEMENT

A Clinical Review for the SASLT Community By Dr. Adel Al Qutub and Dr. Bashayer Alrufayi

Introduction Portal vein thrombosis (PVT) presents a significant clinical challenge in hepatology and gastroenterology, with a prevalence reaching up to 26% in patients awaiting liver transplantation. Historically viewed primarily as a severe complication of end-stage liver disease, the advancement of cross-sectional imaging has revealed PVT to be a relatively common entity with a diverse clinical spectrum. The management of PVT is dictated largely by two intersecting paradigms: the presence or absence of underlying cirrhosis, and the chronicity of the thrombus. Because PVT lies at the intersection of portal hypertension, systemic coagulation, and potentially life-threatening complications such as intestinal ischemia, clinicians constantly face the challenge of balancing the competing risks of thrombosis progression against the risk of hemorrhage.

The Temporal Challenge: Acute vs. Chronic PVT

The terminology regarding the chronicity of PVT has shifted in recent years. Current guidelines recommend moving away from purely symptom-based definitions of acute thrombosis and instead categorizing PVT based on the duration and morphological changes of the affected vessels. The term "recent PVT" is now preferred for thrombi presumed to have been present for less than six months. These recent thrombi are highly amenable to anticoagulation, with spontaneous recanalization occurring in up to 40% of patients with cirrhosis. Conversely, chronic PVT is defined as an obstruction persisting beyond six months. This condition is frequently characterized by cavernous transformation, which involves the development of a network of tortuous collateral vessels bypassing the occluded segment. Recognizing this chronicity is crucial because it dictates the primary therapeutic objective. In recent PVT, the goal is recanalization to restore physiologic blood flow and prevent bowel ischemia. In chronic PVT with cavernous transformation, the probability of recanalization with anticoagulation is exceedingly low, shifting the therapeutic focus toward managing the sequelae of portal hypertension rather than clot dissolution.

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Management in the Cirrhotic Patient: A Delicate Balance

In patients with cirrhosis, the prevalence of PVT ranges from 1% in compensated individuals to over 20% in those awaiting liver transplantation. The pathophysiology is primarily driven by reduced portal flow velocity typically dropping below 15 centimeters per second secondary to advanced portal hypertension. This is combined with the rebalanced hemostatic state inherent to cirrhosis, where decreases in procoagulant factors are mirrored by concurrent decreases in endogenous anticoagulants, such as Protein C and S. Routine screening for thrombophilia in patients with cirrhosis and PVT is not recommended, as local hemodynamic stasis is the primary driver of thrombosis. A comprehensive hypercoagulable workup is only warranted if there is a strong personal or family history of atypical thrombotic events. When managing these patients, the immediate priority is to rule out hepatocellular carcinoma with macrovascular invasion, as the management of a malignant thrombus differs entirely from that of a bland thrombosis. This exclusion requires multiphasic contrast-enhanced computed tomography (CT) or magnetic resonance imaging (MRI). Clinicians must also assess for any signs of intestinal ischemia, such as pain out of proportion to examination, lactic acidosis, or bowel wall thickening. The presence of ischemia mandates urgent, multidisciplinary intervention, which may include anticoagulation or surgical and interventional radiology thrombectomy Treatment strategy further relies on risk stratification based on clot burden. For minimal or partial occlusions involving less than 50% of the vessel lumen, or those confined to intrahepatic branches, observation with serial imaging every three months is a reasonable approach due to the high rate of spontaneous resolution. However, for extensive thrombi occluding more than 50% of the lumen, or those involving the main trunk and mesenteric veins, anticoagulation is strongly advised to prevent progression. This is particularly critical for patients awaiting liver transplantation, where a patent portal vein is essential for a successful surgical anastomosis. In chronic PVT with cavernoma formation, anticoagulation is generally considered futile for clot resolution and is not recommended. Regarding anticoagulant selection, direct oral anticoagulants (DOACs) are increasingly preferred over low-molecular-weight heparin and vitamin K antagonists.

Treatment strategy further relies on risk stratification based on clot burden. For minimal or partial occlusions involving less than 50% of the vessel lumen, or those confined to intrahepatic branches, observation with serial imaging every three months is a reasonable approach due to the high rate of spontaneous resolution. However, for extensive thrombi occluding more than 50% of the lumen, or those involving the main trunk and mesenteric veins, anticoagulation is strongly advised to prevent progression. This is particularly critical for patients awaiting liver transplantation, where a patent portal vein is essential for a successful surgical anastomosis. In chronic PVT with cavernoma formation, anticoagulation is generally considered futile for clot resolution and is not recommended. Regarding anticoagulant selection, direct oral anticoagulants (DOACs) are increasingly preferred over low-molecular-weight heparin and vitamin K antagonists.

DOACs eliminate the need for international normalized ratio (INR) monitoring, which is notoriously unreliable in cirrhosis. They can be used safely in Child-Turcotte-Pugh (CTP) Class A patients and with caution in CTP Class B patients, though they should be avoided in CTP Class C. While endoscopic variceal screening is essential, the initiation of anticoagulation should not be delayed solely to perform prophylactic banding, as early initiation significantly improves recanalization rates, potentially reaching up to 75%.

Management in the Non-Cirrhotic Patient: Hunting the Underlying Cause

PVT occurring in the absence of cirrhosis is a rare entity, and its management challenges differ fundamentally from those seen in the cirrhotic population. In a non-cirrhotic patient presenting with PVT, a systemic or local prothrombotic factor is identified in up to 75% of cases, making a comprehensive thrombophilia workup mandatory. The most common systemic etiologies are myeloproliferative neoplasms, which are often occult and necessitate testing for specific genetic markers such as JAK2 V617F and CALR mutations. Local inflammatory factors, including pancreatitis, diverticulitis, or recent abdominal surgeries like sleeve gastrectomy, must also be thoroughly investigated.

In stark contrast to the observational strategies sometimes employed in cirrhosis, non-cirrhotic patients presenting with recent PVT require prompt and aggressive anticoagulation. The primary objective is to prevent the extension of the thrombus into the mesenteric arcades, which carries a severe risk of fatal bowel infarction. Additionally, early anticoagulation aims to achieve complete recanalization to prevent the development of chronic extrahepatic portal vein obstruction and subsequent portal hypertension. Anticoagulation is typically maintained for at least six months. If an underlying unprovoked prothrombotic state, such as a myeloproliferative neoplasm, is identified, lifelong anticoagulation is generally indicated.

The Role of Interventional Radiology

Interventional radiology is playing an increasingly critical role as both a primary adjunctive and salvage therapy for patients with and without cirrhosis. In patients experiencing progressive PVT despite adequate anticoagulation, or those presenting with refractory ascites and variceal bleeding, portal vein recanalization combined with transjugular intrahepatic portosystemic shunting (PVR-TIPS) can mechanically disrupt the clot and restore physiological flow. This intervention is particularly vital for liver transplant candidates, as maintaining vascular patency prior to surgery is crucial for optimizing post-transplant outcomes.

Conclusion The management of portal vein thrombosis demands a nuanced and highly individualized approach. In the non-cirrhotic patient, clinical efforts must focus on rapid anticoagulation and a comprehensive investigation to identify any underlying prothrombotic defects. In the cirrhotic population, clinicians must carefully weigh the necessity of maintaining portal patency especially for transplant candidates against the complex bleeding risks associated with portal hypertension and rebalanced hemostasis. As the pharmacological armamentarium expands, highlighted by the increasing integration of DOACs, and as interventional radiology techniques advance, the multidisciplinary management of PVT will continue to progress, offering improved therapeutic outcomes for this complex patient population.

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ARTICLES

PORTAL VEIN THROMBOSIS IN CHILDREN

By Dr. Ashraf Alsahafi

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Background

Portal vein Thrombosis (PVT) is defined as thrombosis that develops in the trunk of the portal vein, including its right and/or left intrahepatic branches. It may extend into the splenic or superior mesenteric veins (1). It is the most common cause of portal hypertension in the pediatric age group (2). Estimates of PVT range from 1 in 100,000 births to 36 per 1,000 admissions to the neonatal intensive care unit (3).

Etiology and Risk Factors of Portal Vein Thrombosis in Children

Several studies have investigated the risk factors for PVT in the pediatric age group. A large European cohort study identified several risk factors. Eight percent of patients had positive pro-thrombotic screening and low serum anticoagulant protein C and S. Other rare etiologies include mutations in the prothrombin gene and factor V Leiden. No patient was found to have acquired prothrombotic disorders (4).

Prematurity is an important clinical factor involved in the pathogenesis of PVT, and a history of umbilical catheterization has been reported in 65% of neonates who developed thrombosis (4). Historical aspects may reveal an episode of profound dehydration, exchange transfusion, or sepsis (5).

Chronic PVT is the predominant presentation in the pediatric age group compared to acute recent PVT (5). Portal vein occlusion is usually followed by compensatory vasodilation of the hepatic artery and formation of collaterals that bypass the thrombosed segment. This process results in cavernous transformation of the portal vein or portal cavernoma (6).

Clinical Presentation

The mean age at clinical presentation of portal vein thrombosis is 4 years, and it should be suspected in children presenting with unexplained gastrointestinal hemorrhage and splenomegaly. Other important clinical features include hypersplenism and unexplained pancytopenia (7). Approximately 72% of children with PVT are expected to have one episode of upper gastrointestinal bleeding in their lifetime (8). Growth impairment is a common finding in children with PVT and usually improves after shunt surgery (9, 10).

Management of Portal vein thrombosis

The management of PVT in children depends on the age of presentation and whether it is acute or chronic. Anticoagulation with low-molecular weight heparin may be considered for acute PVT in neonates with careful evaluation of the potential risks and benefits. Catheter-directed thrombolysis for recent PVT has been successful in older children (11). A stepwise approach was suggested by Alberti et al. (12) to manage chronic PVT in the pediatric age group (Fig 1).

Few studies in children have proven that beta-blockers are effective in reducing the risk of variceal bleeding. However, owing to limited evidence, its use cannot be universally recommended. Endoscopic intervention in pediatrics has special considerations compared to adults. Endoscopic variceal ligation is considered a safe intervention and has largely replaced sclerotherapy, with favorable outcomes. Sclerotherapy should be reserved for cases in which EVL is technically challenging. Surgical intervention is warranted when the conservative approach is deemed ineffective. The indications for surgical intervention are listed in (Table 1).

Table 1. Stepwise approach for management of chronic PVT in the pediatric age group – as suggested by Alberti et al (12).

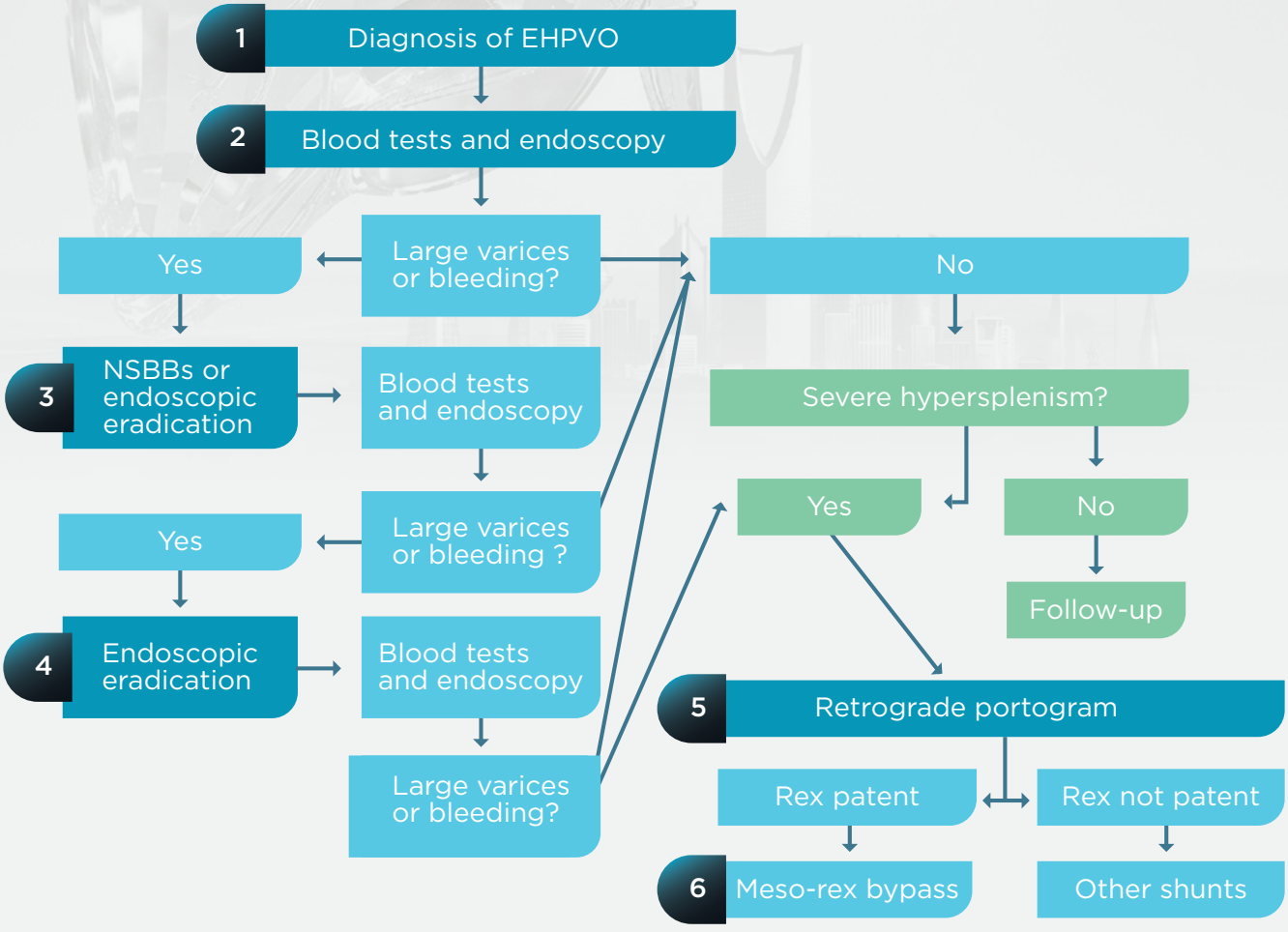

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Table 1. Indications for surgical intervention in Pediatric PVT (7)

• Persistent bleeding after medical and endoscopic treatments
• Large fundal varices
• Massive splenomegaly with hypersplenism.
• Splenomegaly with infarction
• Portal biliopathy
• Colonic varices
• Massive bleeding

Outcome of Pediatric Portal Vein Thrombosis

In the largest reported pediatric cohort which comprised 187 patients, the mortality rate associated with PVT-related complications was 1%, and two-thirds of the patients were managed with conservative treatment. The remaining 30% who failed conservative measures were managed by surgery or Transjugular Intrahepatic Portosystemic Shunt (TIPS). Meso-Rex bypass is the commonest surgical intervention approach, followed by Distal spleno-renal shunt. Shunt patency was observed in 90% of patients. Liver transplantation serves as a last resort is usually performed in 2% of patients (4).

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ARTICLES

FROM SCIENTIFIC EXCHANGE TO PUBLIC AWARENESS: SASLT'S WORLD LIVER DAY 2026 ACTIVITIES

By Dr. Ahmed T. Alhawamdeh

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World Liver Day 2026 gave the Saudi Society for the Study of Liver Diseases and Transplantation (SASLT) an opportunity to engage liver health on two complementary fronts: professional education and public awareness. Rather than marking the day through a single activity, SASLT approached it through a linked program that began with a physician-focused scientific meeting on 10 April and continued with a bilingual public awareness campaign on 19 April. Together, these efforts reflected SASLT's broader mission of advancing hepatology education while strengthening community awareness and prevention.

The first activity took place on 10 April 2026 in Riyadh through the Riyadh Liver Club Cholestatic Liver Disease: Adult and Pediatric Perspectives, sponsored by Ipsen. Structured as a hybrid event for physicians and healthcare professionals, the meeting brought together adult and pediatric perspectives in cholestatic liver disease and created a valuable forum for multidisciplinary discussion.

The scientific program covered a range of clinically relevant topics, including transforming adult cholestatic liver disease through emerging therapies, advancing toward optimal disease control in primary biliary cholangitis, PBC case discussions,

genetic testing in late-onset PFIC, and therapeutic approaches targeting bile acid pathways across PFIC, ALGS, and biliary atresia. By combining expert lectures with case-based learning and panel discussion, the meeting offered both depth and practical relevance for attendees.

The event achieved strong engagement, On 19 April 2026, SASLT extended its

with 62 in-person attendees and 242 virtual attendees. Participant feedback was highly positive: based on 199 responses, the meeting received an average score of 4.79 for overall quality and relevance of content and 4.78 for organization. These outcomes underscored the value of focused, physician-directed educational activities that are scientifically current,

well-structured, and responsive to practice needs. World Liver Day activities through a bilingual awareness campaign published on its official X and LinkedIn platforms. A total of 18 posts in English and Arabic were shared for the general public, using simple and practical messages to highlight prevention, early detection, and the importance of healthy daily habits.
The campaign addressed balanced nutrition, physical activity, regular liver check-ups, and awareness of common liver diseases and risk factors, including MASLD, viral hepatitis, cirrhosis, liver cancer, diabetes, obesity, and dyslipidemia. Importantly, the messaging emphasized that many liver diseases remain silent in their early stages, yet many are preventable, detectable, or easier to manage when identified early.
Taken together, the 10 April scientific meeting and the 19 April public campaign demonstrated a balanced and meaningful observance of World Liver Day 2026. One activity strengthened professional exchange among healthcare providers; the other translated key liver health messages into accessible public communication. Through both initiatives, SASLT contributed to World Liver Day in a way that was educational, practical, and relevant to the needs of both clinicians and the wider community.

GLOBAL FATTY LIVER DAY MEETING

SASLT
الجمعية السعودية لأمراض وزراعة الكبد
Saudi Society for the Study of Liver Disease and Transplantation

GLOBAL FATTY LIVER DAY MEETING

Metabolic Liver Disease - A New Era of Treatment

DISTINGUISHED SPEAKERS

Dr. Mohammed Alghamdi
Consultant Gastroenterologist and Transplant Hepatologist

Dr. Saad Alghamdi
Consultant Gastroenterologist and Transplant Hepatologist

DATE & VENUE

10 June 2026, 09:00 PM KSA

Live Webinar

SCIENTIFIC PROGRAM

Time	Topics	Speaker
09:00 09:10 PM	Welcoming Words & Introduction	Dr. Mohammed Alghamdi
09:10 09:40 PM	The Evolving Treatment Landscape of MASH	Dr. Saad Alghamdi
09:40 10:00 PM	Panel Discussion & Q&A	

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MODERATOR

Dr. Noora H. Alfaraj
Consultant Gastroenterologist, Hepatologist & Liver Transplantation Specialist

SPEAKERS

Dr. Sarah Althonayan
GASTROENTEROLOGY FELLOW

Dr. Mohammed Aljawad
Consultant Gastroenterologist and Transplant Hepatologist

Dr. Mohammed Alghamdi
Consultant Gastroenterologist and Transplant Hepatologist

SCIENTIFIC PROGRAM

08:00 - 08:15 PM
Welcoming Remarks
Principles of Early Referral to a Transplant Center
Dr. Noora Alfaraj

8:15–8:25 PM
Case Presentation
Dr. Sara Althunian

08:25- 08:45PM
HBV: From Infection to Management and Immunoprophylaxis
Dr. Mohammed Aljawad

08:45 - 09:00 PM
Under the Radar: Unseen Challenges in Viral Hepatitis
Dr. Mohammed Alghamdi

Closing remarks & Dinner

Tuesday, June 16, 2026

Kempinski Al Othman Hotel, Dammam

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Click here to register

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WORKSHOP

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MASLD NON-INVASIVE TESTS WORKSHOP

22 June Monday | **08:30 - 10:45** PM PM | **Voco Hotel Riyadh**

Dr. Faisal Abbkhail
Consultant
Gastroenterology and
Transplant Hepatology

Dr. Saad Alghamdi
Consultant, Adult Transplant
Hepatology Liver

Dr. Adel AlQutub
Consultant
Gastroenterology and
Transplant Hepatology

Fees | **200 SAR** Members | **350 SAR** Non-Members

SCIENTIFIC PROGRAM

TIME	TITLE	SPEAKER
8:30- 8:40	Registration & Welcome Coffee	
8:40- 8:50	Opening Remarks & Objectives	Dr. Faisal Abbkhail
8:50- 9:10	MASLD: Disease Overview and Unmet Needs	Dr. Saad Alghamdi
9:10- 9:25	Non Invasive Tests in MASLD: Blood Based and Imaging Tools	Dr. Adel Alqutb
9:25- 10:40	Interactive Case Based Workshop	Dr. Saad AlGhamdi & Dr. Adel Alqutb
10:40- 11:00	Q&A	
	Dinner	

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JOURNAL CLUB IN JEDDAH

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Saudi Society for the Study of Liver Disease and Transplantation

WHEN HEPATITIS C GETS COMPLICATED: CHALLENGING CASES AND CLINICAL PEARLS

MODERATOR

Dr. May Alzahrani

Consultant Gastroenterologist and Transplant Hepatologist

SPEAKERS

Dr. Saad Aldosari

Gastroenterolgist and Transplant Hepatologist

Dr. Waleed Alghamdi

Gastroenterologist and Transplant Hepatlogist

SCIENTIFIC PROGRAM

09:10 – 09:15 PM

Welcoming Remarks introduction

Dr. May Alzahrani

09:15–09:45 PM

1st session case

Dr. Saad Aldosari

09:45– 10:15 PM

2nd session case

Dr. Waleed Alghamdi

10:15– 10:30 PM

Q&A

Closing remarks & Dinner

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Monday, June 29, 2026

Jeddah Hilton Hotel, Jeddah

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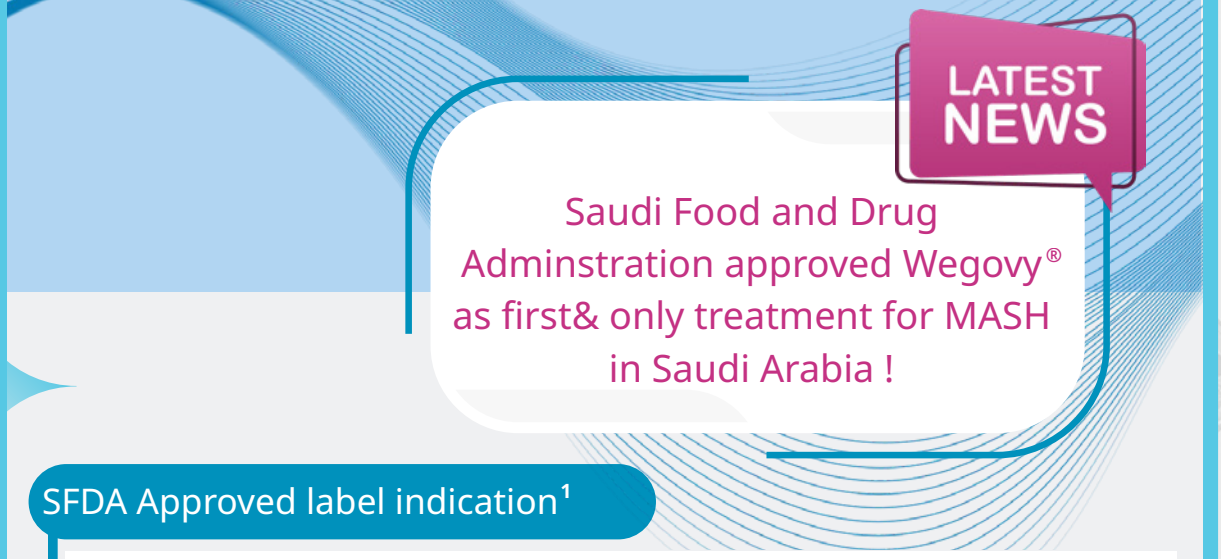
SAUDI LIVER MEETING

SASLT ABSTRACT

SASLT HEPATOLOGY JEOPARDY

LATEST NEWS



Saudi Food and Drug Adminstration approved Wegovy® as first& only treatment for MASH in Saudi Arabia !

SFDA Approved label indication[1]

Adult

Wegovy is indicated as an adjunct to a reduced-calorie diet and increased physical activity:

- For weight management, including weight loss and weight maintenance, in adults with an initial Body Mass Index (BMI) of

⩾30 kg/m2 (obesity), or
⩾27 kg/m2 to <30 kg/m2 (overweight) in the presence of at least one weight-related comorbidity e.g.dysglycaemia (prediabetes or type 2 diabetes mellitus), hypertension, dyslipidaemia, obstructive sleep apnoea or cardiovascular disease.

- For the treatment of noncirrhotic **metabolic dysfunction-associated steatohepatitis (MASH)** in adult with moderate to advanced liver fibrosis (consistent with stages F2 to F3 fibrosis).
- To reduce the risk of major adverse cardiovascular events (cardiovascular death, non-fatal myocardial infarction, or non-fatal stroke) in adults with established cardiovascular disease and either obesity or overweight.

Adolescents (⩾12 years)

Wegovy is indicated as an adjunct to a reduced-calorie diet and increased physical activity for weight management in adolescents ages 12 years and above with

- obesity* and
- body weight above 60 kg.

Treatment with Wegovy should be discontinued and re-evaluated if adolescent patients have notreduced their BMI by at least 5% after 12 weeks on the 2.4 mg or maximum tolerated dose.

Interested to know more about Wegovy® MASH results?
Please Visit **ESSENCE Trial** webpage

SA26SEMO00104

References :
1.Wegovy® summary of product characteristics. Latest SFDA label.

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SASLT NEWSLETTER

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