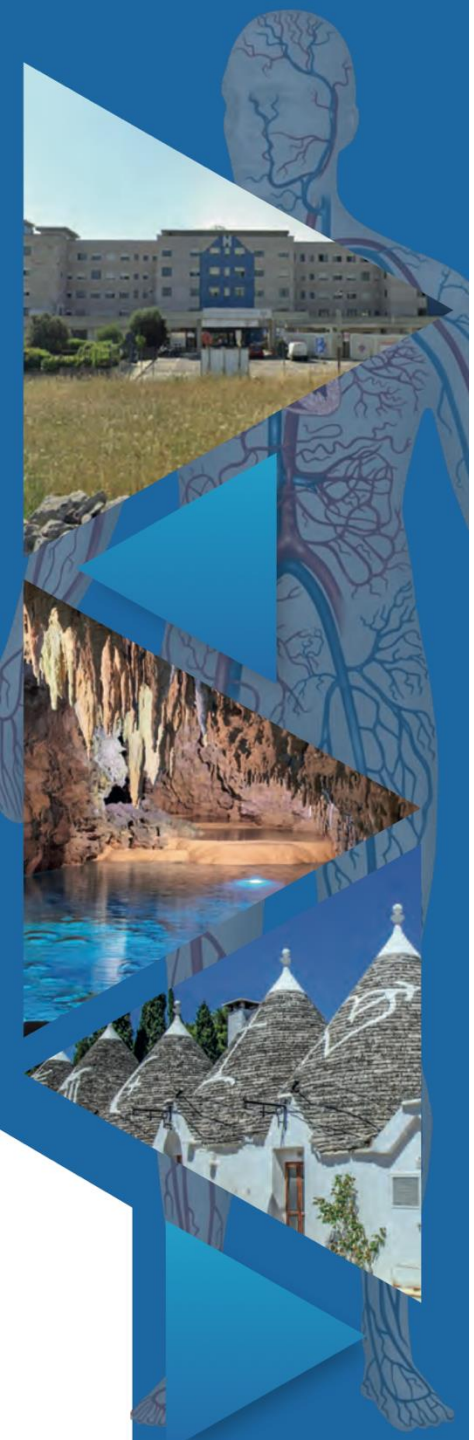

International Congress on Coagulopathy in Liver Disease

Hemostasis and Thrombosis in Liver Disease: from Bench to Bedside

Natural History and Pathophysiology of PVT in Cirrhosis

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9th April 2026

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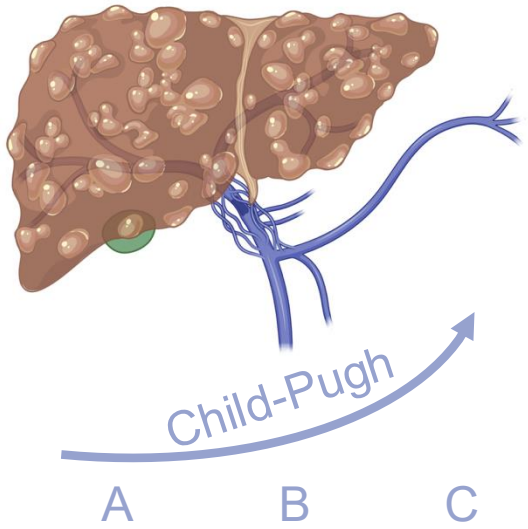
SCHOOL OF MEDICINE AND BIOMEDICAL SCIENCES

Natural History and Pathophysiology of PVT in Cirrhosis - Epidemiology

Epidemiology of portal vein thrombosis in liver cirrhosis: A systematic review and meta-analysis

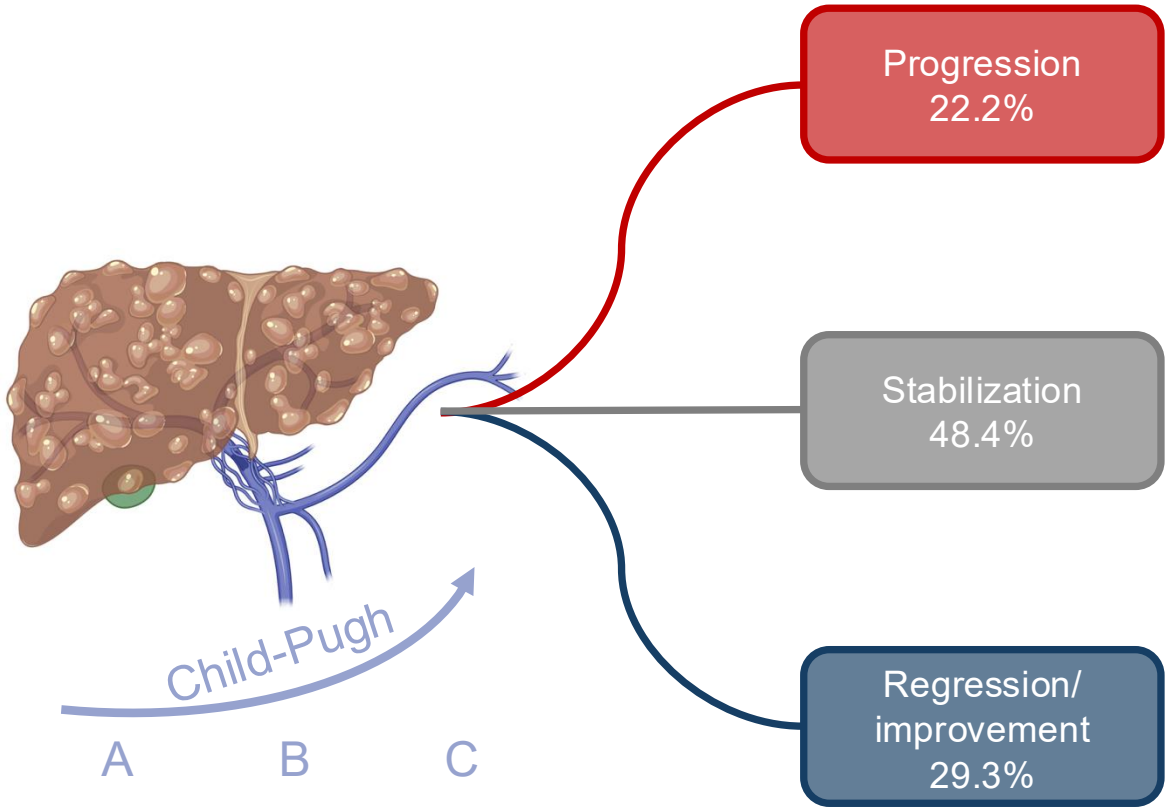
European Journal of Internal Medicine 104 (2022) 21–32

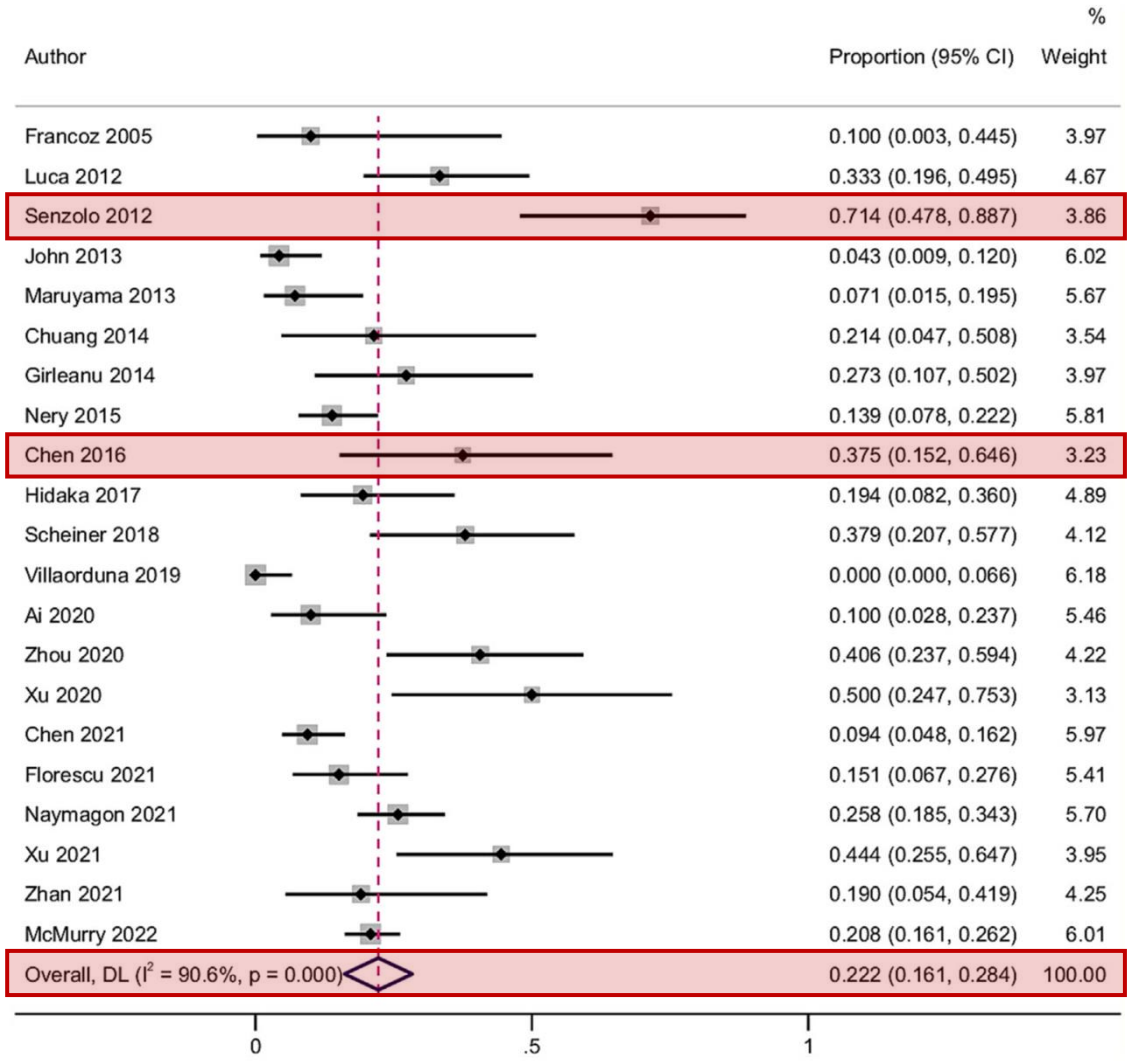
Jiahui Pan [a,b,#], Le Wang [a,c,#], Fangbo Gao [a,b,#], Yang An [a,b], Yue Yin [a], Xiaozhong Guo [a], Filipe Gaio Nery [d,e], Eric M. Yoshida [f], Xingshun Qi [a,b,c,*]



Incidence/ Prevalence	Subgroup/ Outcome	Pooled estimate	95% CI
Incidence	Child-Pugh A	9.89%	4.95% - 16.30%
	Child-Pugh B/C	18.34%	10.79% - 27.35%
Incidence	1-year cumulative	4.78%	2.09% - 8.49%
	3-year cumulative	9.34%	5.89% - 14.48%
Incidence	Partial PVT	9.18%	6.62% - 12.11%
	Complete PVT	1.99%	0.73% - 3.85%
Prevalence	Child-Pugh A	13.54%	9.49% - 18.17%
	Child-Pugh B/C	23.72%	16.75% - 31.48%

Natural History

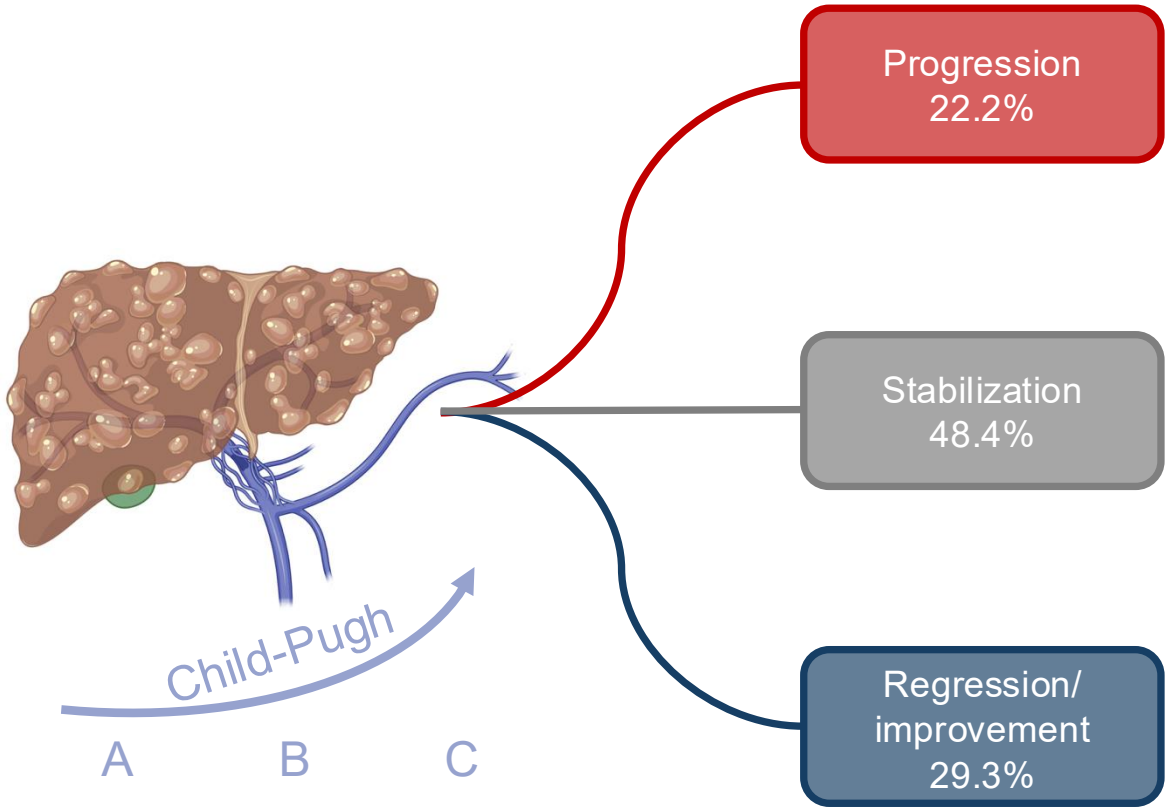


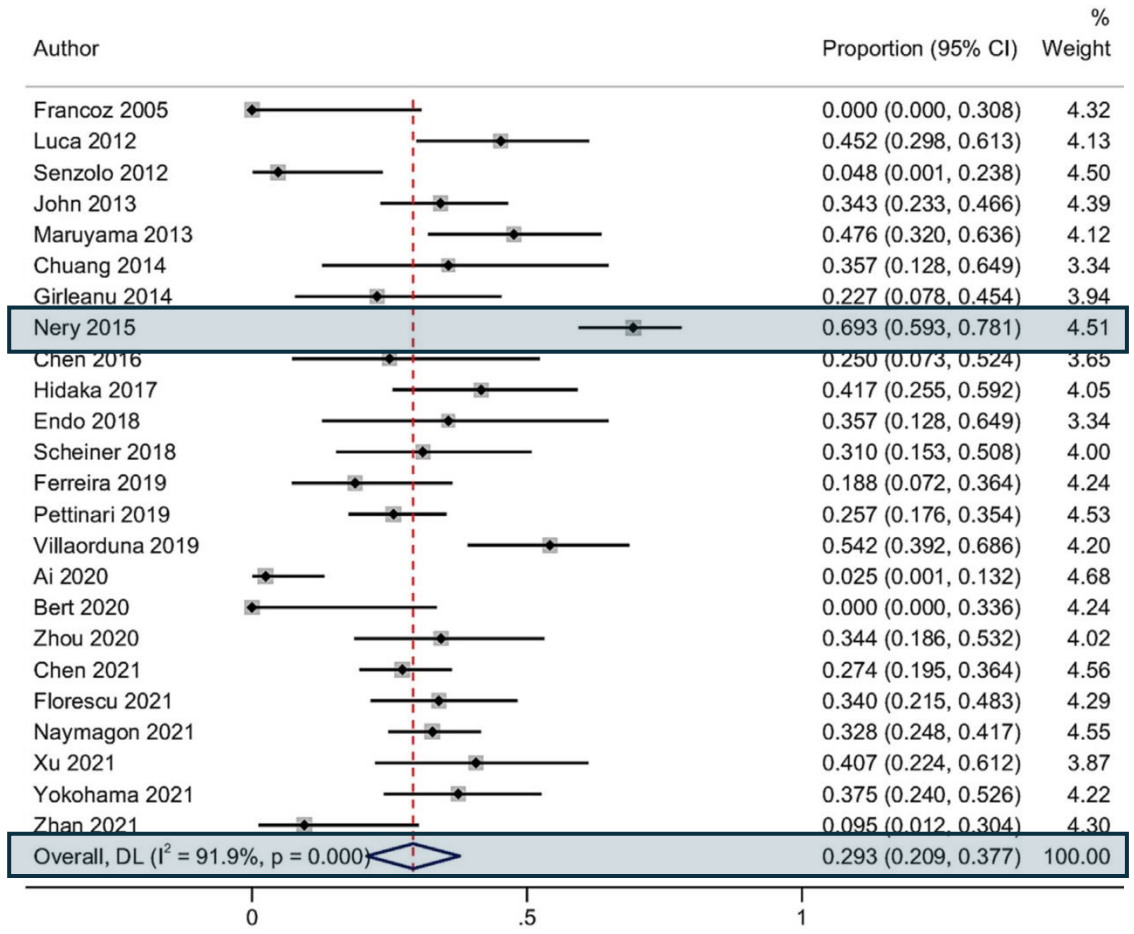


Senzolo M. et al 2012: 76.2% Child-Pugh B/C

Chen et al 2016: 74.2% Child-Pugh B/C

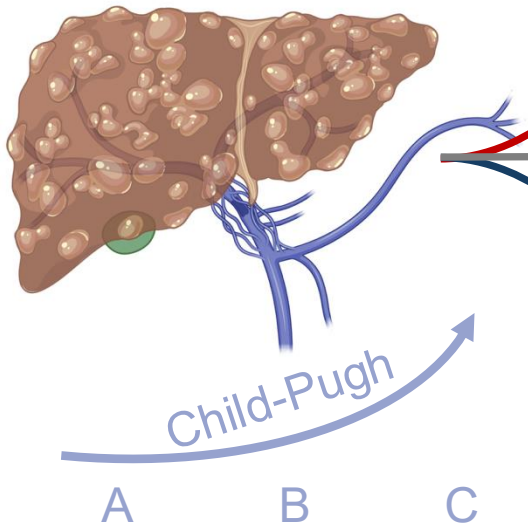
Natural History





Nery F. et al 2015: 863 CP A ; 380 CP B (Total 1243); 101/118 partial PVT

Natural History



Progression
22.2%

Albumin <= 3.3g/dL
Pancreatitis/ Abdominal infections
Large collaterals (high flow)

Maruyama H. et al, Am J Gastroenterol 2013;
Xu S. et al, Eur J Gastroenterol Hepatol 2021;
McMurry H. et al, Thromb Res 2022

Stabilization
48.4%

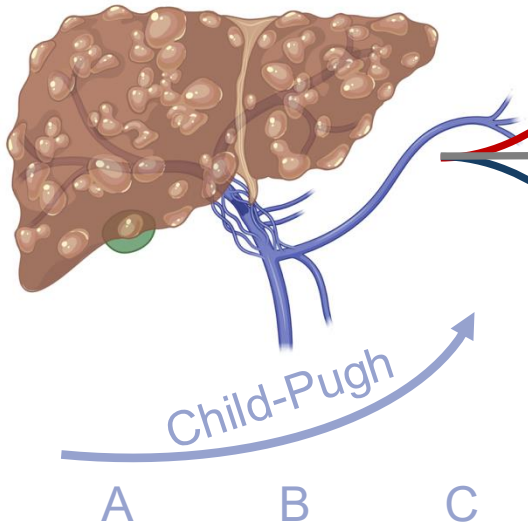
Regression/
improvement
29.3%

ΔMELD score <= -2.185
Absence of large collaterals
Branched PVT

Naymagon L. et al, Dig Dis Sci 2021;
Xu S. et al, Eur J Gastroenterol Hepatol 2021;
Yokoyama S. et al, Hepatol Res 2022

Giri S. et al., J Gastroenterol Hepatol 2023

Natural History



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Xu S. et al, Eur J Gastroenterol Hepatol 2021;
Yokoyama S. et al, Hepatol Res 2022

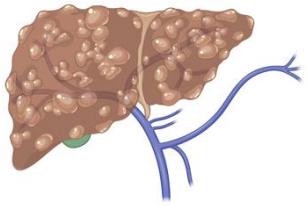
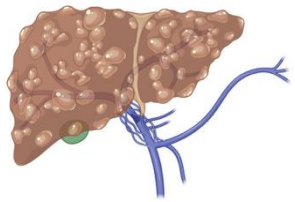
Recurrence/
rethrombosis
24%

Stable thrombi

Complete
recanalization
10.4%

Giri S. et al., J Gastroenterol Hepatol 2023

Natural History

Baseline	Disease course	Pre-LT survival	Post-LT survival
Cirrhosis without PVT 	Clinical evolution depends mainly on hepatic reserve, portal hypertension severity, underlying etiology, …	**Reference pre-LT mortality** Comparator arm for natural history	**Liver transplantation without prior PVT** Lower technical complexity Lower recurrent PVT risk Favourable post-LT survival
Cirrhosis with PVT 	Decompensation vs no decompensation	**Pre-LT mortality often similar** to non-PVT cohorts	**Liver transplant with prior PVT** Higher operative complexity Higher recurrence PTV risk Increased post-LT morbidity/mortality

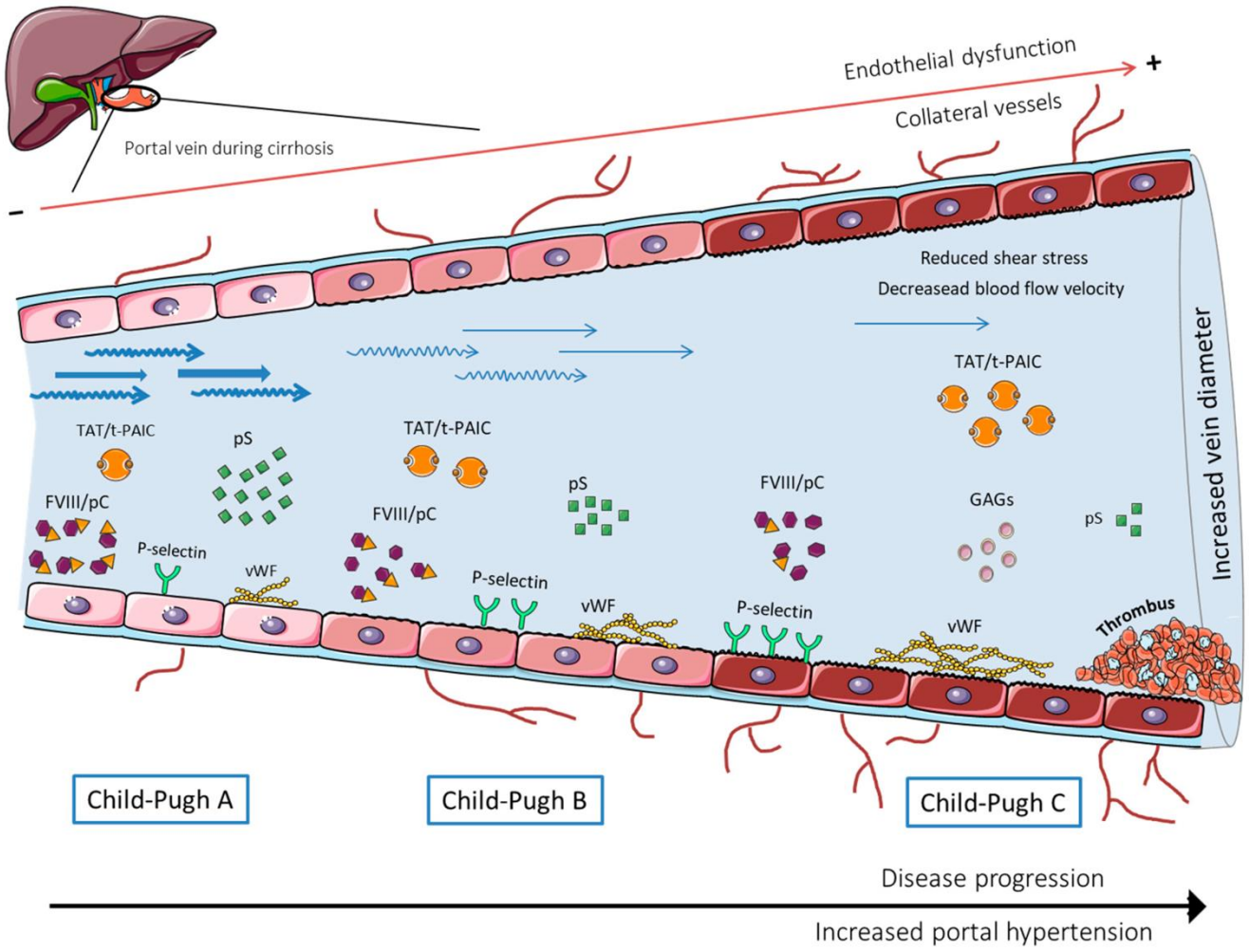
Pre-transplant: similar overall mortality in many cohorts

Chen H et al, Liver Transpl. 2016;
Ghabril M et al, Transplantation 2016

Zanetto A et al, Transpl Int 2018;
Yeo JW et al, Clin Transplant 2022;
Barrera-Lozano LM et al, J Clin Med 2023

In cirrhosis, PVT is best viewed as an epiphenomenon of advanced portal hypertension and hepatic dysfunction before LT; however, once transplantation is undertaken, pre-existing PVT clearly worsens surgical and post-transplant outcomes

Pathophysiology of PVT in Cirrhosis



"Classical view"

when risk factors are conflated with pathophysiology

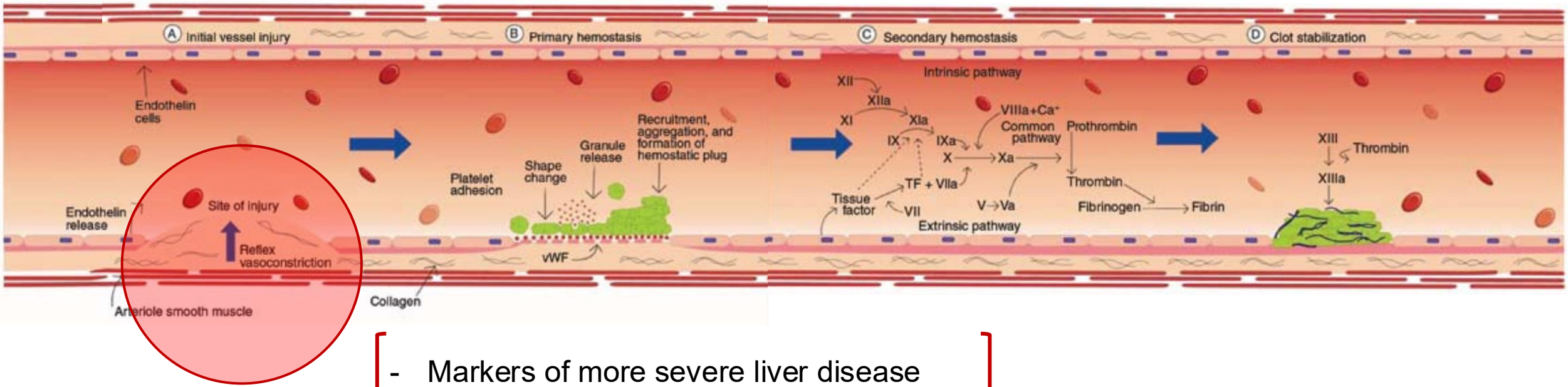
Risk factor: who is more likely to get the disease
Pathophysiology: how the disease actually happens

EXAMPLE (smoking and lung cancer):
- Smoking is a risk factor
- DNA damage, mutational burden, immune evasion, tumor growth are the pathophysiology

Anton A. et al, J Clin Med 2022

Pathophysiology of PVT in Cirrhosis

The physiology behind clot formation – from general concept to new knowledge in PVT

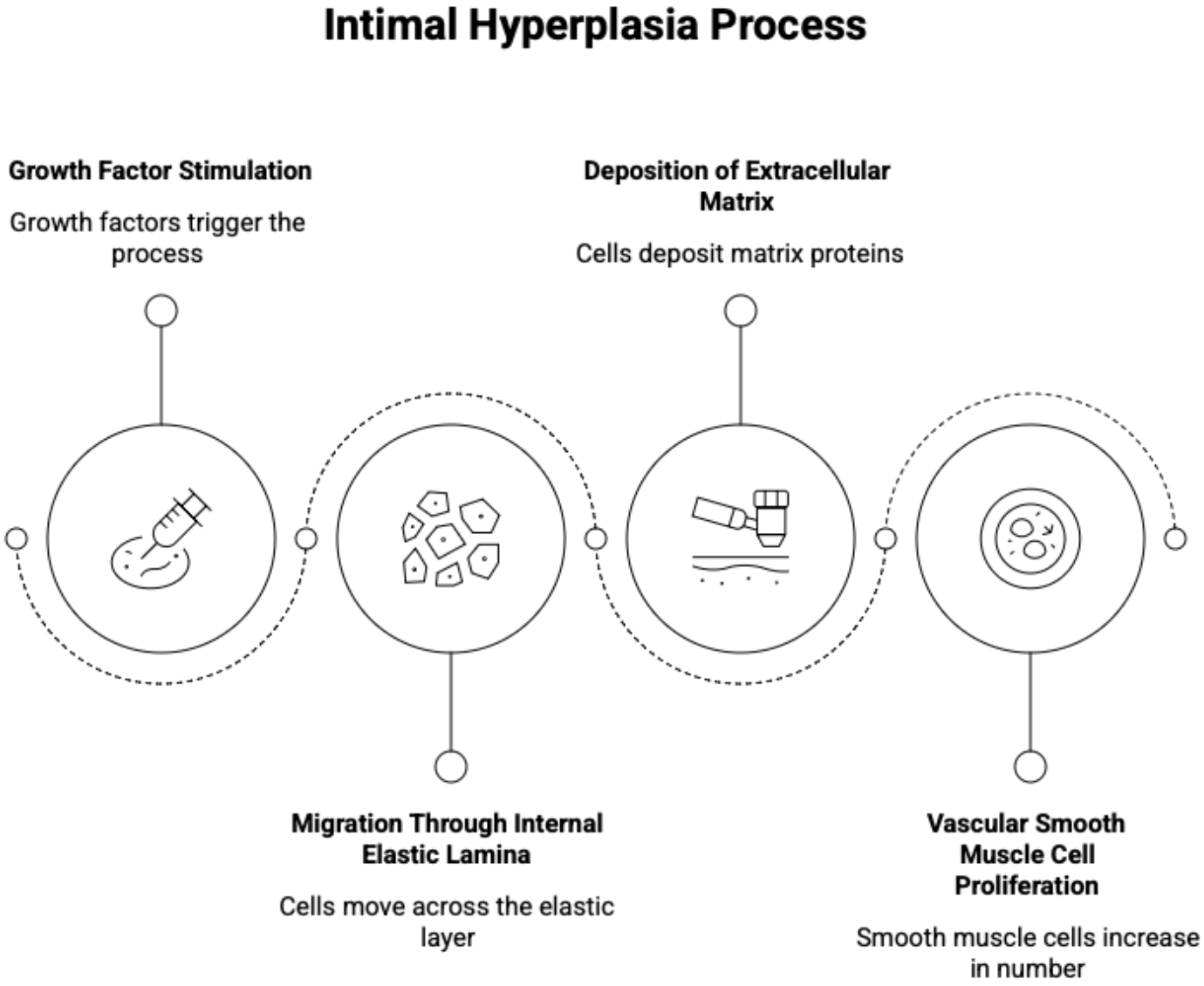


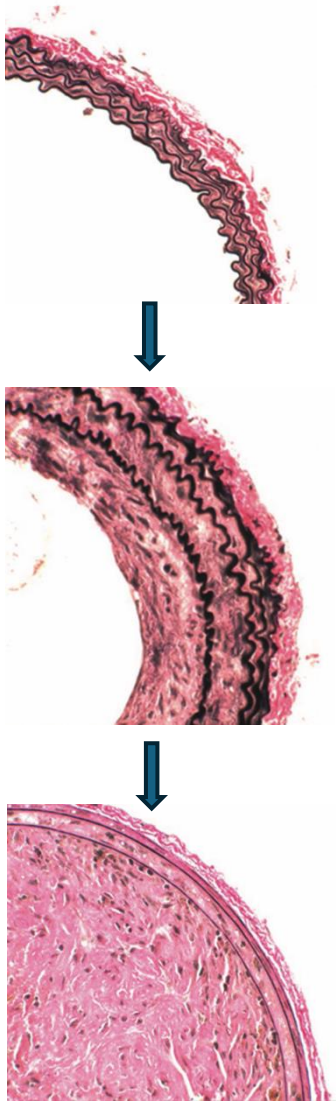
In cirrhosis The "hit"

- Markers of more severe liver disease
- Markers of increased inflammation
- Expression of low portal vein flow velocity
- Markers of portal hypertension

Endothelial lesion/ shear stress → **PVT**

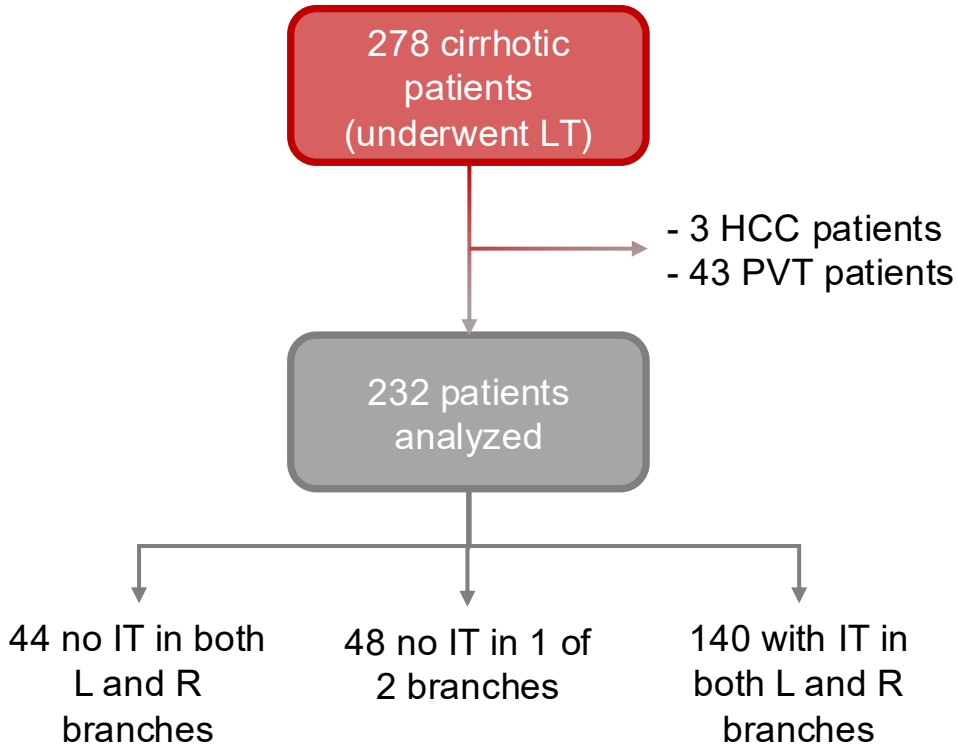
Pathophysiology of PVT in Cirrhosis – new concept

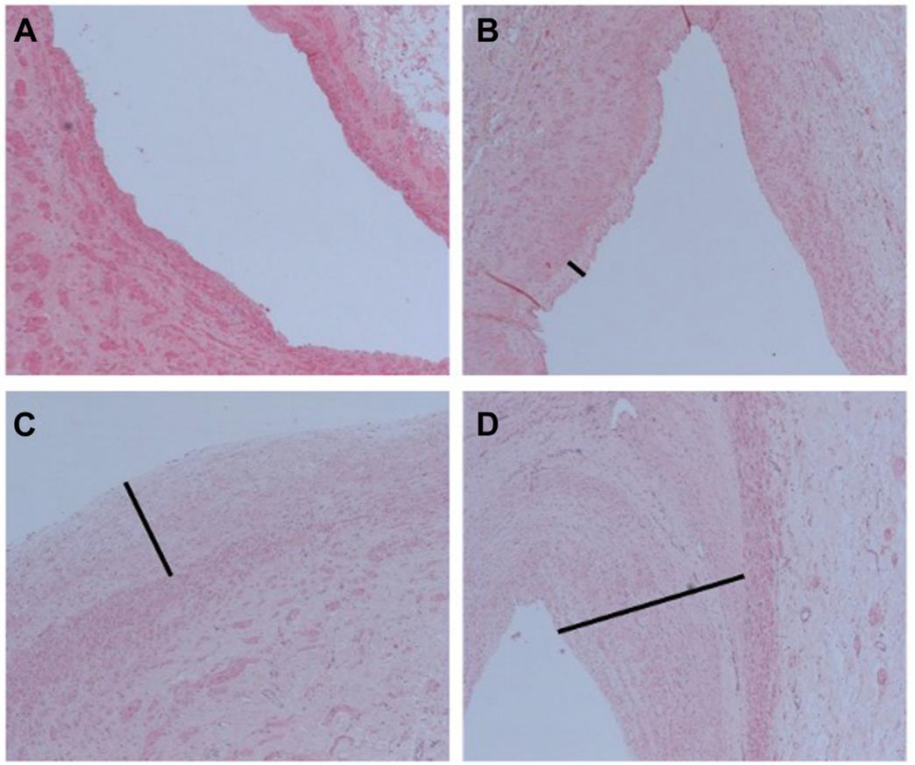




Mills B. et al, Perfusion 2012

Pathophysiology of PVT in Cirrhosis – new concept





Different intimal thickening IT:

A. No thickening
B. ±100 µm
C. ±500 µm
D. ±1000 µm

Median IT at the right hilum was 129 µm and at the left hilum was 112 µm.

Sub-analysis 43 PVT patients: at the right hilum was 364 µm and at the left hilum was 454 µm.

Intimal thickening (out of the context of PVT) was associated to:

- Obesity [1.34 (1.14-1.57)]
- History of grade 3-4 HE [1.28 (1.03-1.80)]
- History of ascites [1.19 (1.13-1.25)]
- History of variceal bleeding [1.34 (1.17-1.55)]

- Suggestion that portal vein IH is related to PH

- Notion that patients with PVT have more IH

Driever EG et al, J Thromb Haemost 2025

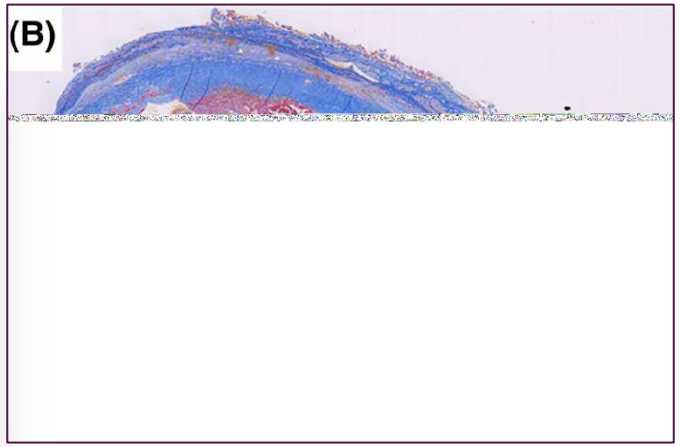
Pathophysiology of PVT in Cirrhosis – new concept

3 centres involved

- 16 PVT in LT (prospective) + 63 PVT (retrospective)

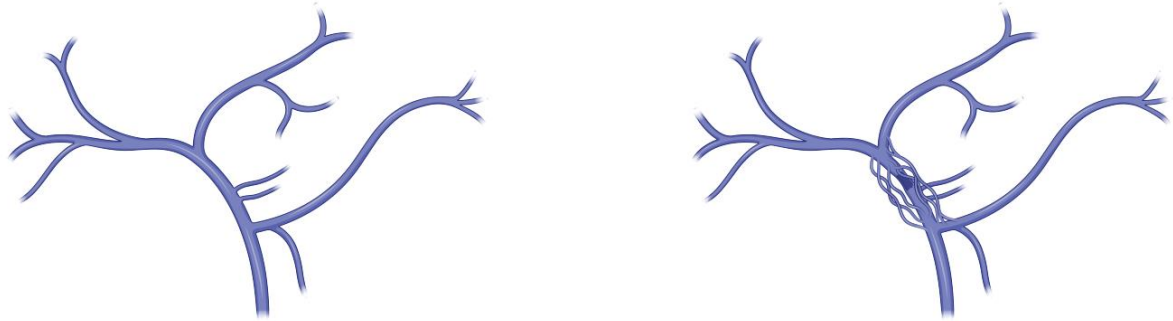
Consistent findings of IH in liver cirrhosis, also in portal vein wall at the liver hilum

- Increased thickness and fibrosis of tunica intima in all samples
- 5/16 (31%) with thickened wall >50% of lumen
- 9/16 (56%) with associated fibrin thrombus



- 48/63 (76%) with IH occupying >50% of lumen
- 21/63 (33%) with fibrin-rich thrombus
- Median IT=358 μm (cirrhosis non-PVT); 2406 μm (in PVT)

Pathophysiology of PVT in Cirrhosis – new concept



Normal PV

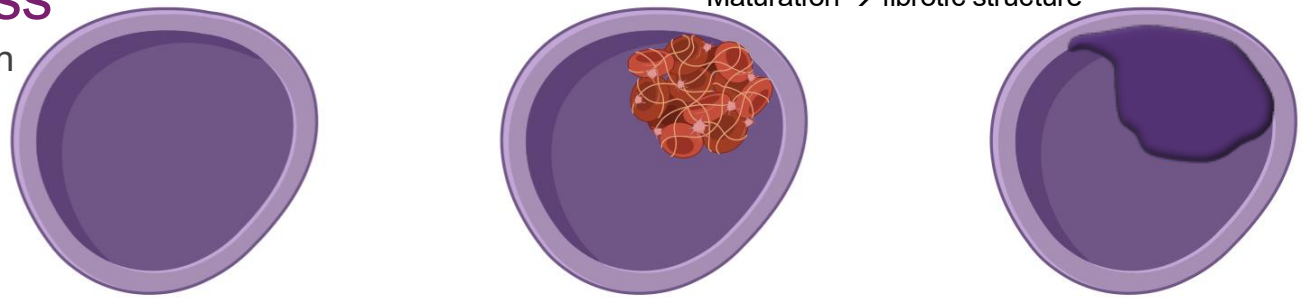
Initial fibrin-rich thrombus

Maturation → fibrotic structure

Reendothelization

Shear stress

Portal hypertension
Inflammation



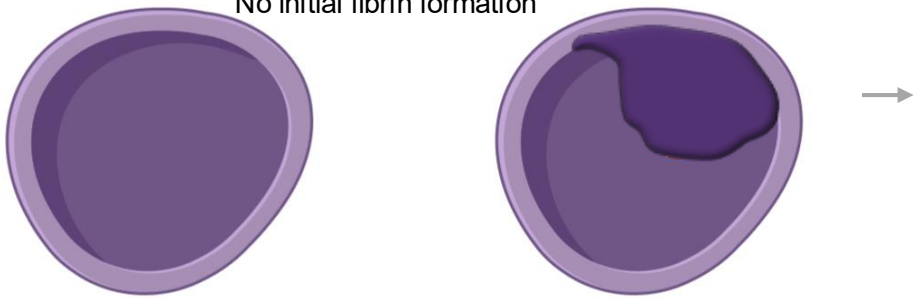
Normal PV

No initial fibrin formation

Intimal Hyperplasia

Endothelial cell stress

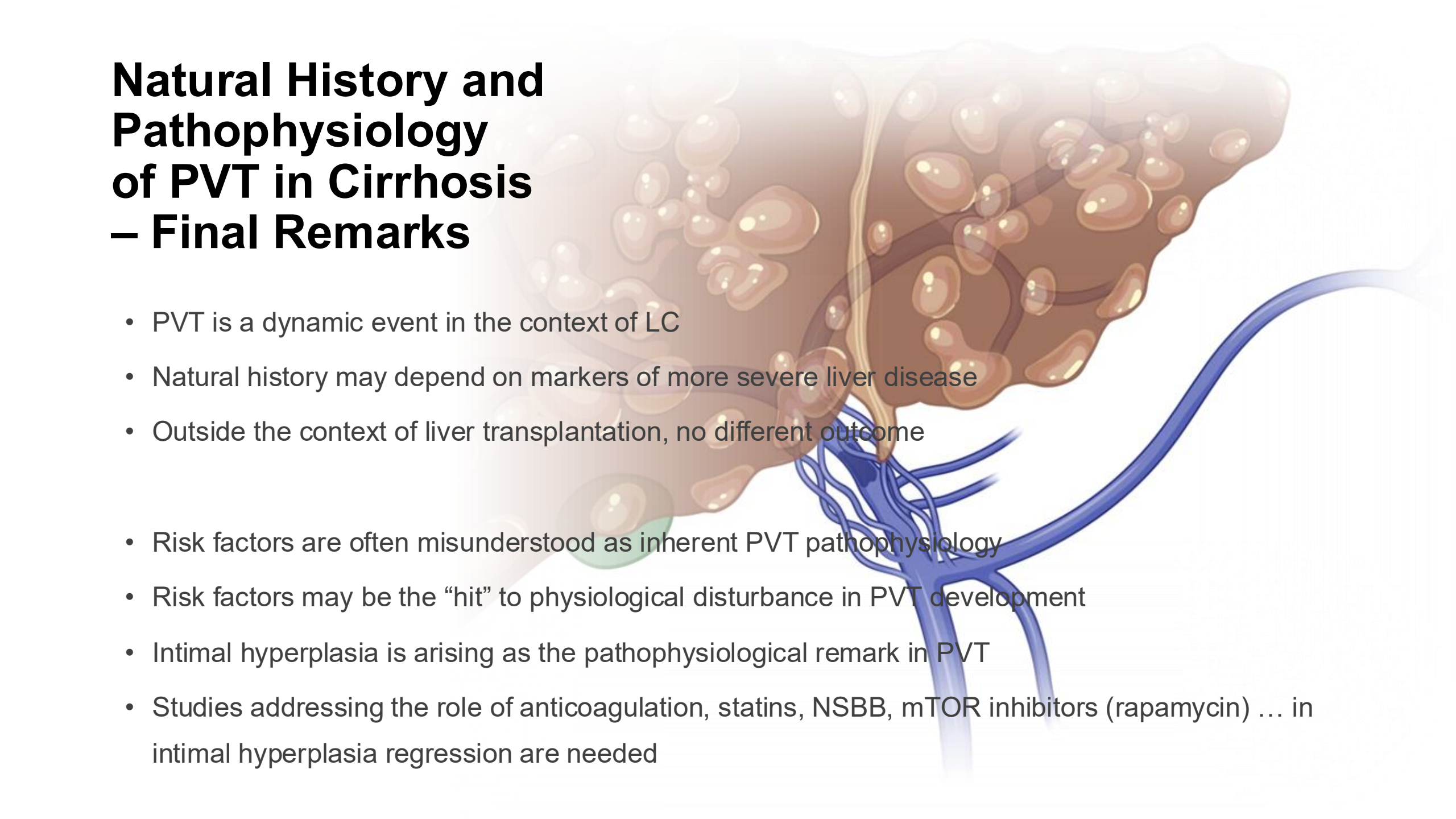
Thrombin



This process is known following all types of vascular reconstructive procedures (coronary artery bypass surgery, angioplasty, vascular stenting, endarterectomy, and vascular access grafting)

Driever EG et al, Hepatology 2022

Natural History and Pathophysiology of PVT in Cirrhosis – Final Remarks



- PVT is a dynamic event in the context of LC
- Natural history may depend on markers of more severe liver disease
- Outside the context of liver transplantation, no different outcome

- Risk factors are often misunderstood as inherent PVT pathophysiology
- Risk factors may be the “hit” to physiological disturbance in PVT development
- Intimal hyperplasia is arising as the pathophysiological remark in PVT
- Studies addressing the role of anticoagulation, statins, NSBB, mTOR inhibitors (rapamycin) … in intimal hyperplasia regression are needed