

MÁSTER EN HEPATOLOGÍA



Asignatura: Cirrosis I

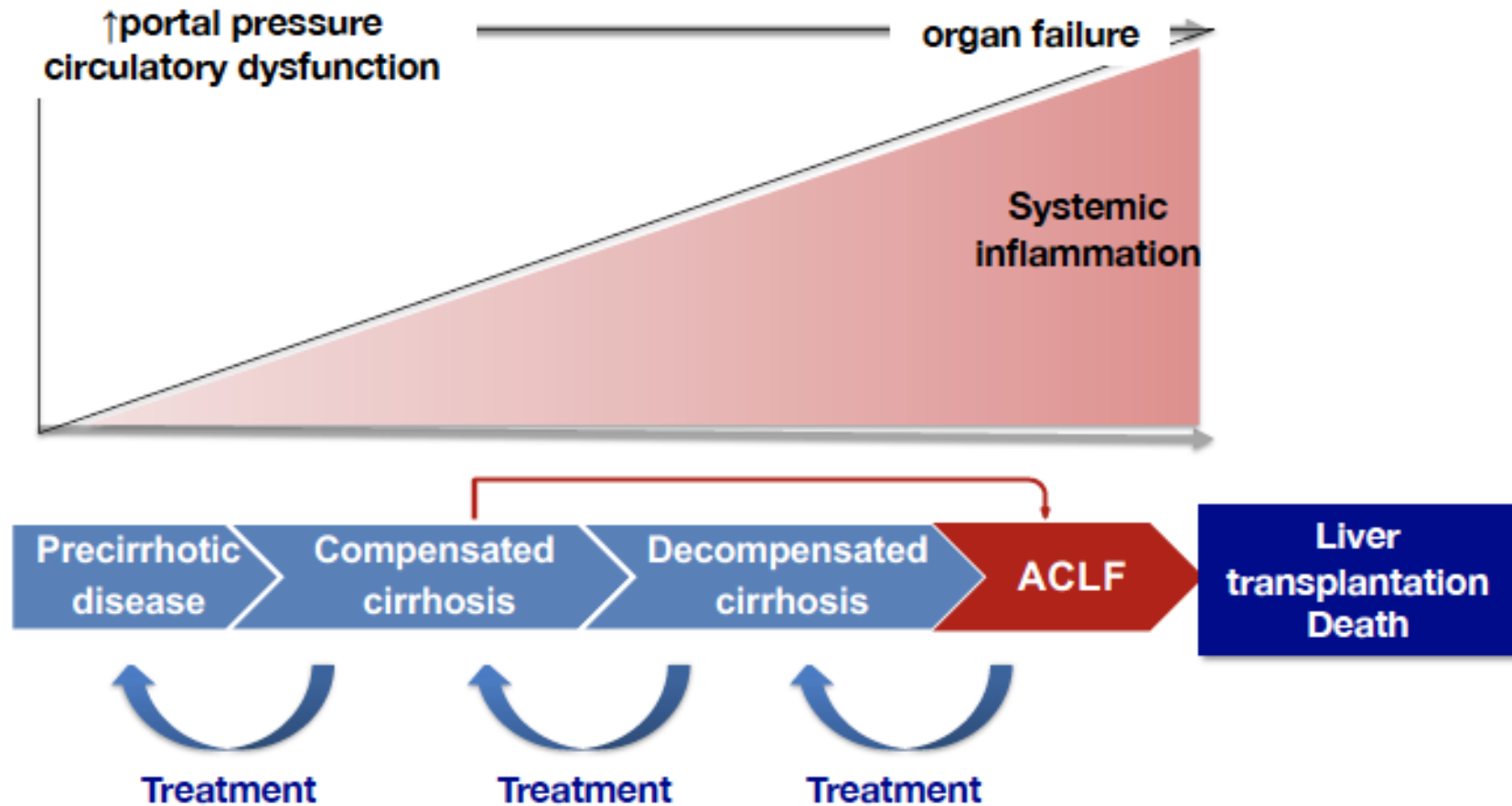
**“Historia natural de la cirrosis:
Cambios en los paradigmas”**

Agustín Albillos

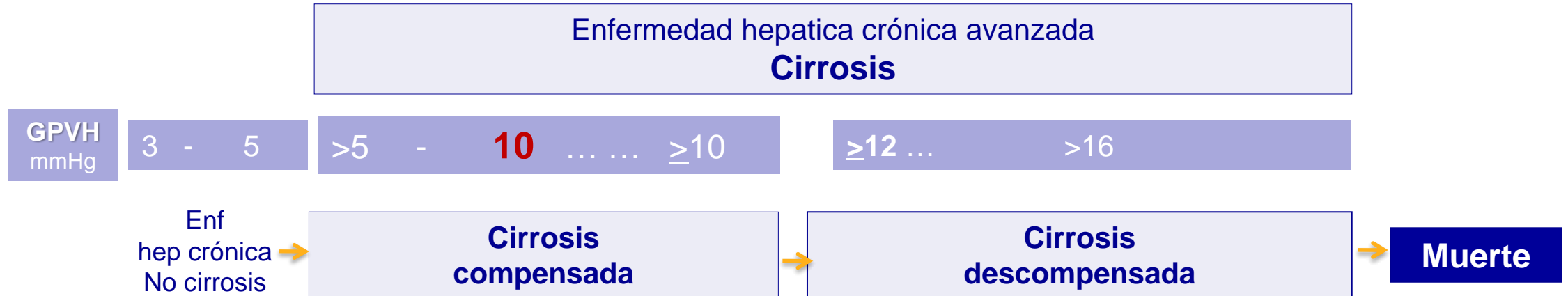
Hospital Universitario Ramón y Cajal, IRYCIS,
Universidad de Alcalá, CIBERehd, Madrid

“Cirrosis hepática: historia natural y estadios”

Natural history of cirrhosis: Changes in the paradigm

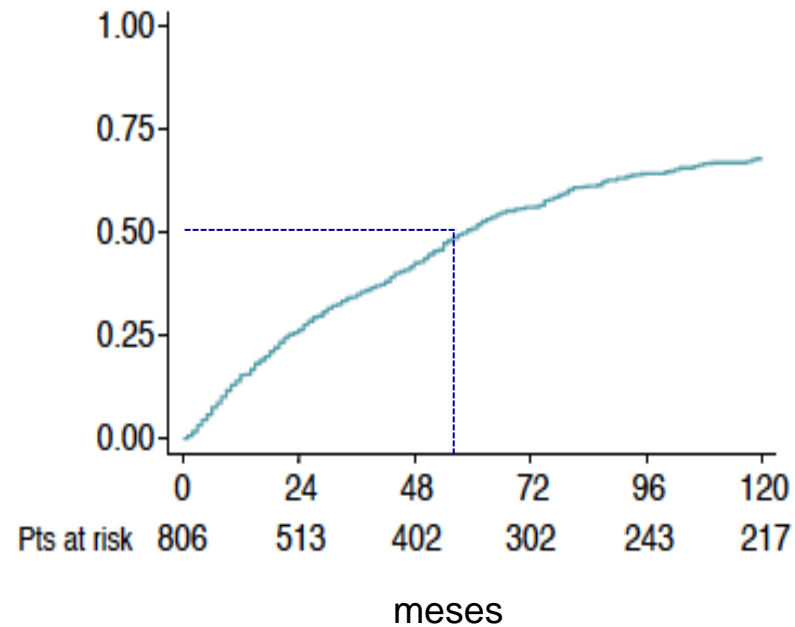


Estadios y subestadios de la cirrosis

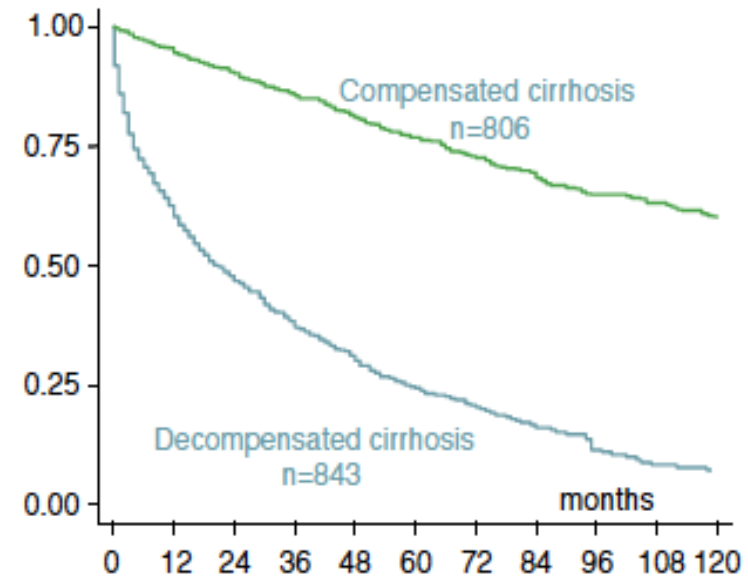


Cirrosis: transición desde el estadio compensado al descompensado

Tasa de descompensación

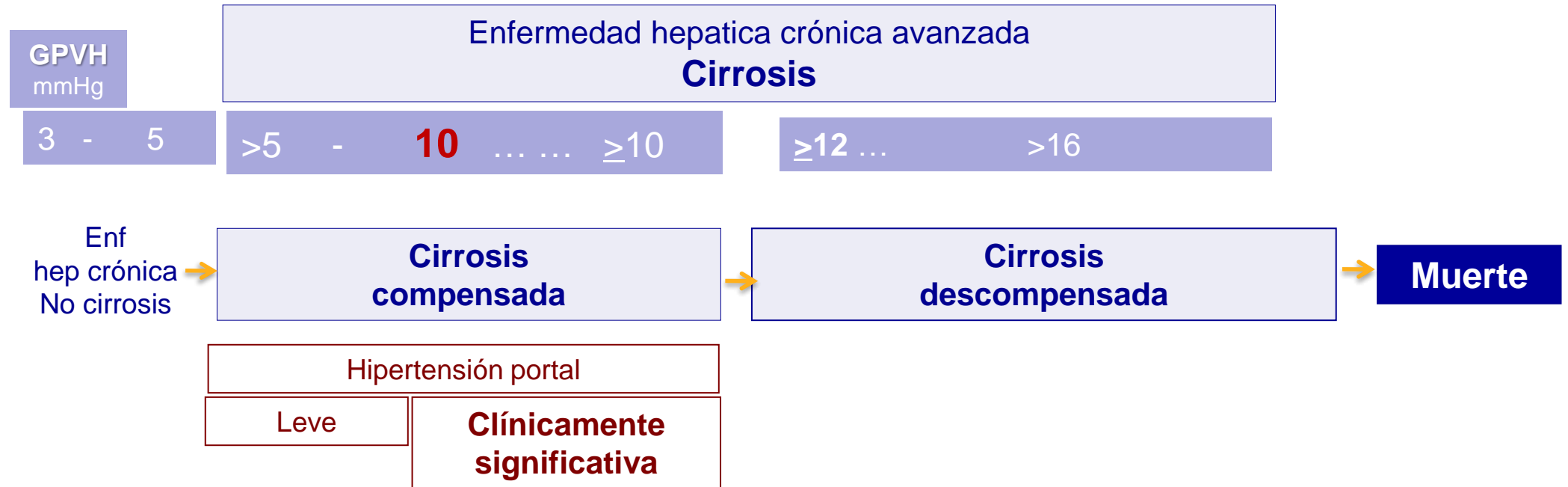


Supervivencia



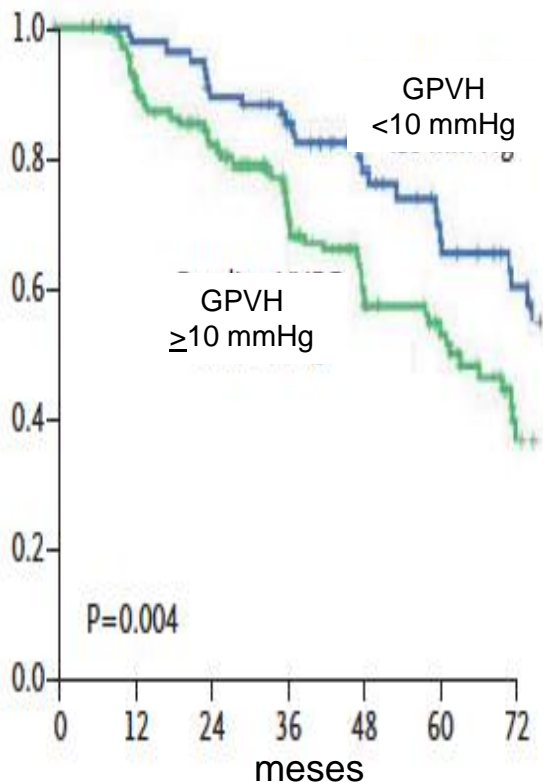
G D'Amico et al. J Hepatol 2005

Estadios y subestadios de la cirrosis



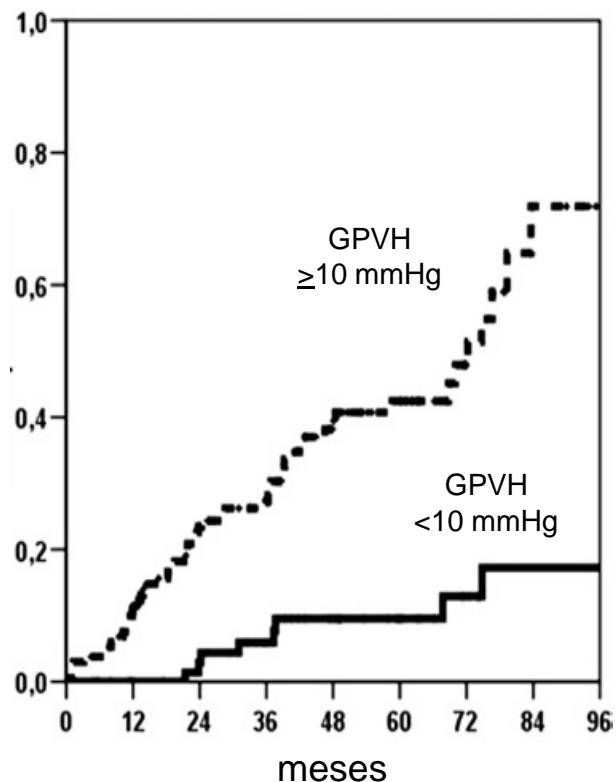
Cirrosis compensada: Relevancia de hipertensión portal clínicamente significativa (GPVH ≥ 10 mmHg, rigidez hepática >20 kPa)

Probabilidad de desarrollo de varices



RJ Groszmann et al.
NEJM 2005

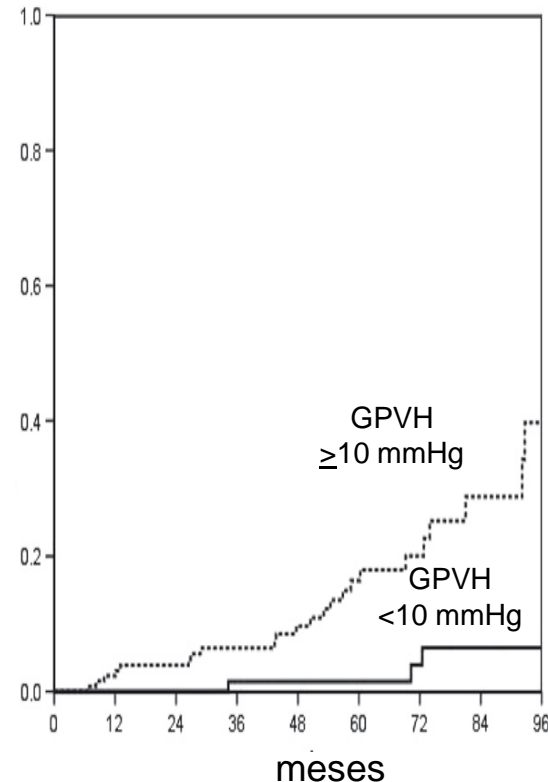
Probabilidad de descompensación clínica



Valor predictivo negativo 90% para **NO** desarrollo de descompensación

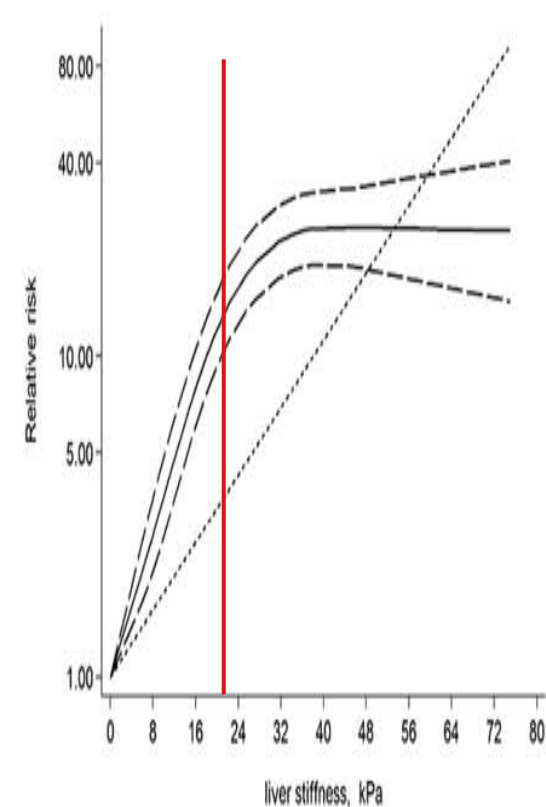
C Ripoll et al.
Gastroenterology 2007

Probabilidad de carcinoma hepatocelular



C Ripoll et al.
J Hepatol 2009

Riesgo relativo de muerte



J Wang et al.
Hepatol Comm 2018

Hipertensión portal clínicamente significativa (CSPH): aumento del gasto cardiaco y del flujo esplácnico (circulación hiperdinámica)

Mecanismo predominante
en hipertensión portal leve
(GPVH 5-10 mmHg)

Aumento
resistencia
intrahepática

HIPERTENSIÓN
PORTAL

Mecanismo
importante en
CSPH
(GPVH ≥ 10 mmHg)

Vasodilatación
esplácnica

Aumento flujo
esplácnico

↓ Volumen sanguíneo
efectivo

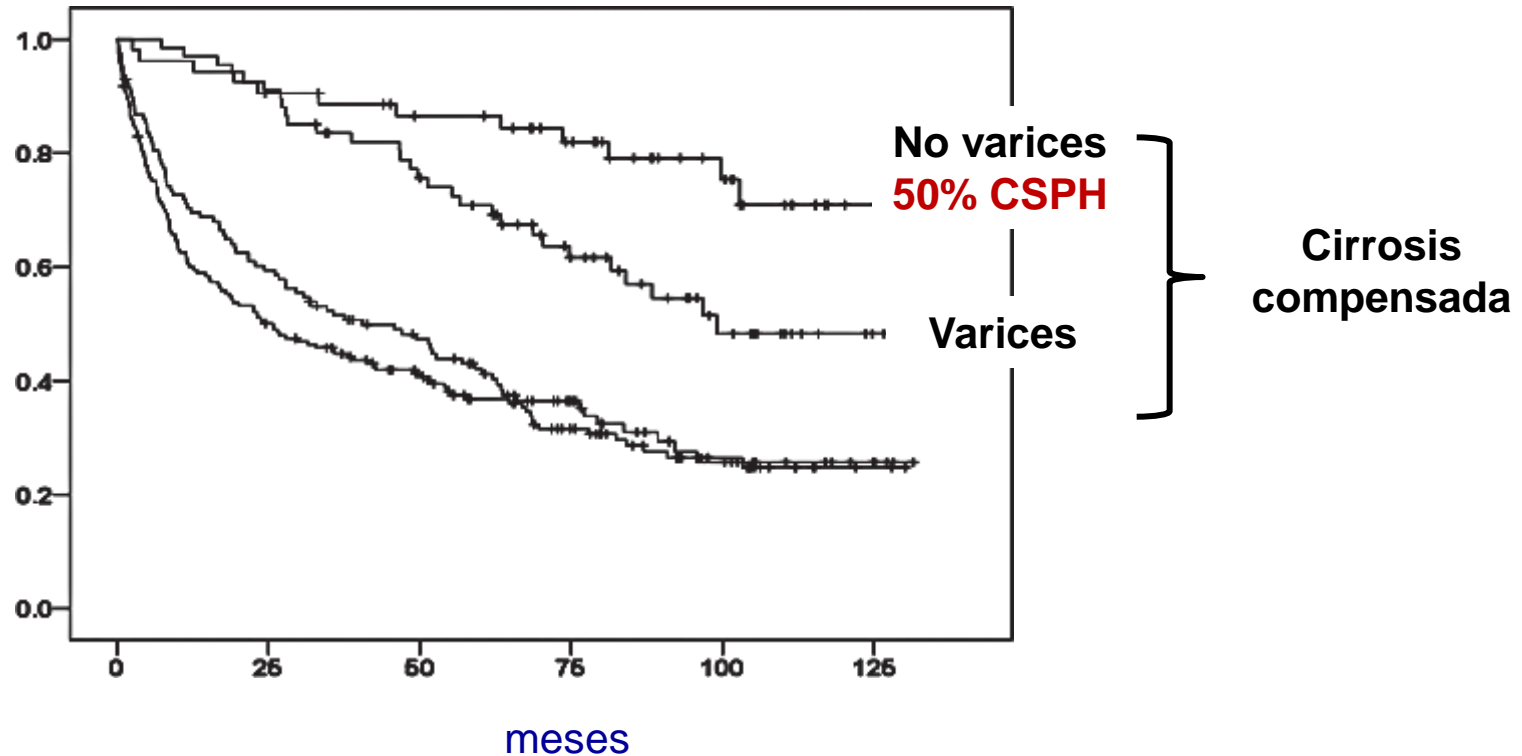
↑ Aumento
gasto cardiaco

Activación
sistemas neurohumorales

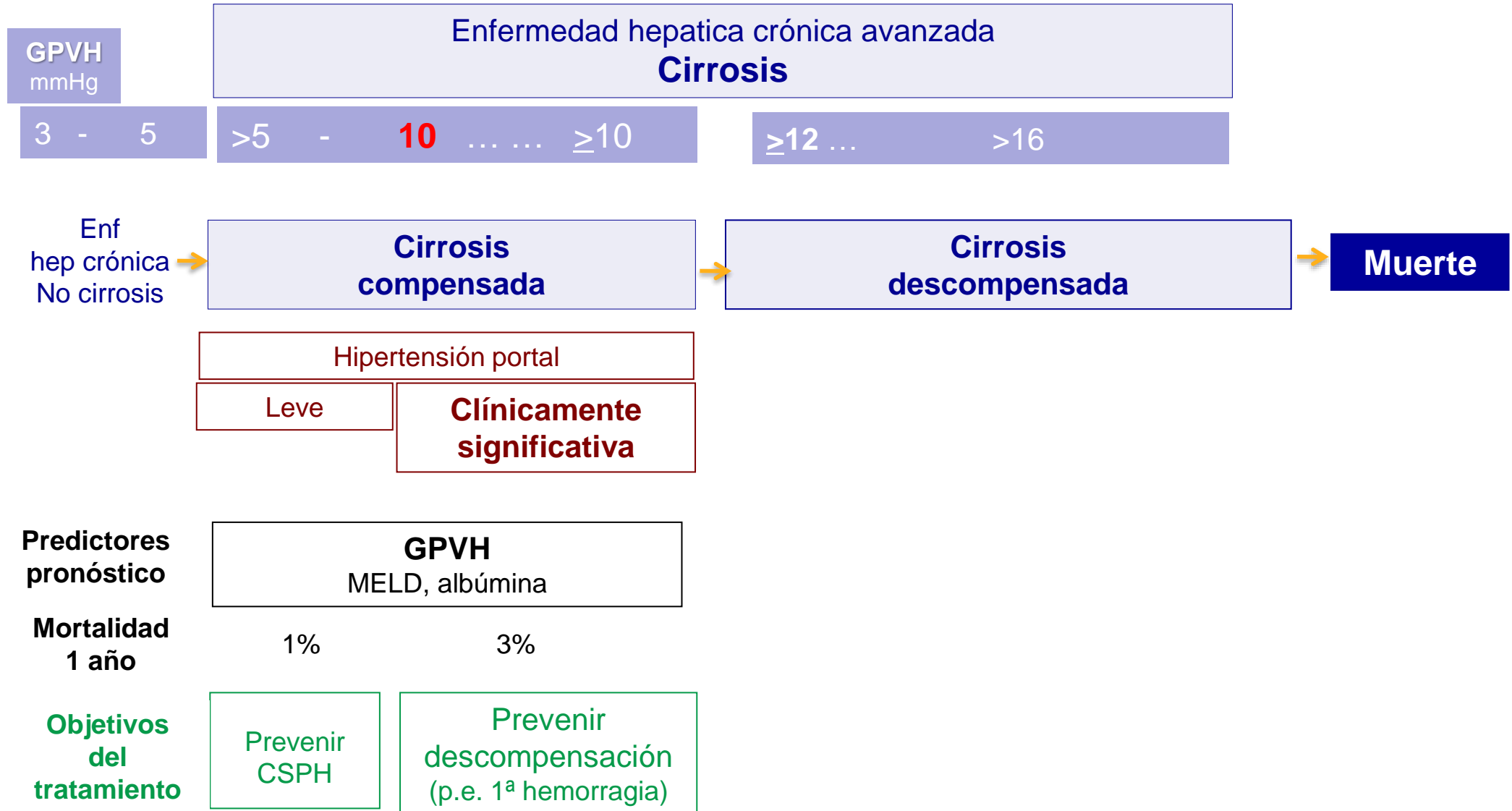
Retención renal de agua y sodio
Hipervolemia

Diferente supervivencia de la cirrosis compensada según la presencia de varices

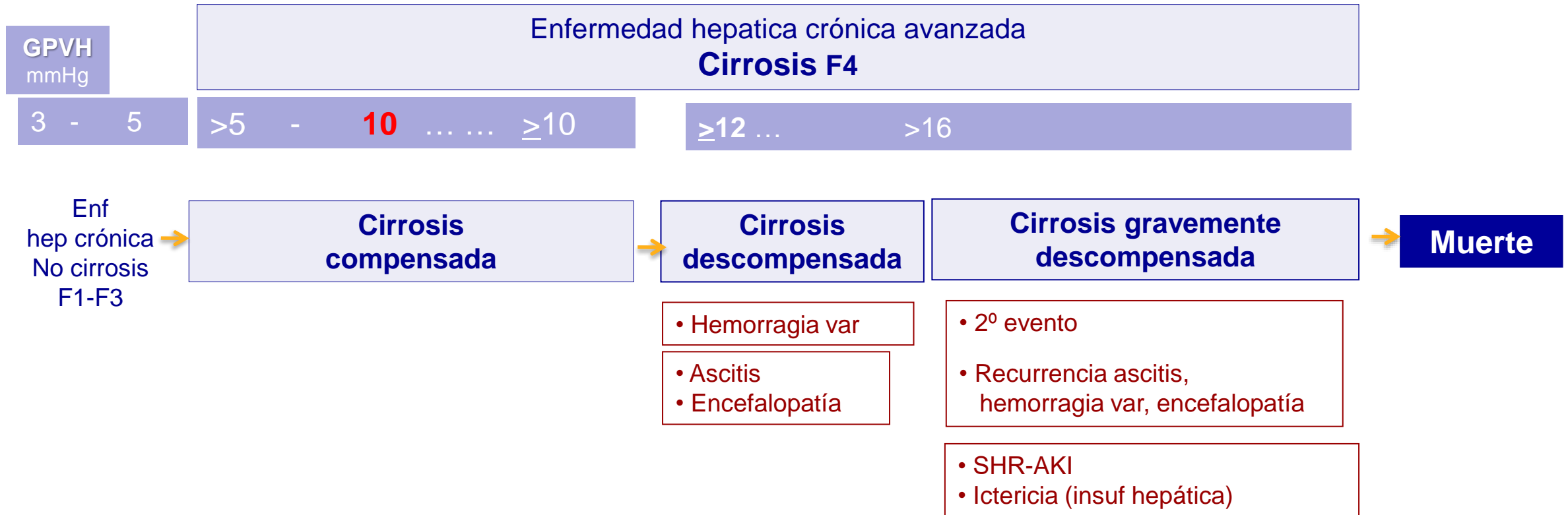
Supervivencia en cirrosis compensada



Estadios y subestadios de la cirrosis

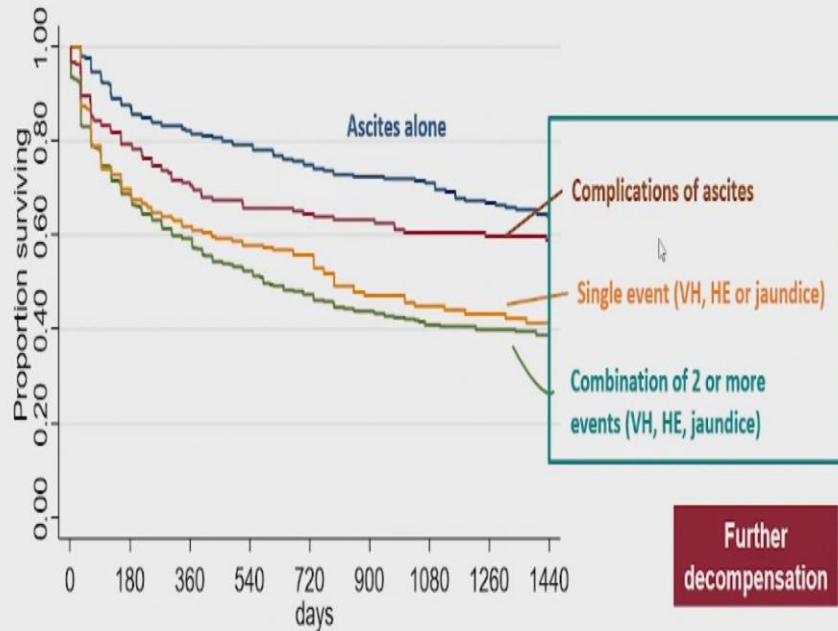


Estadios y subestadios de la cirrosis

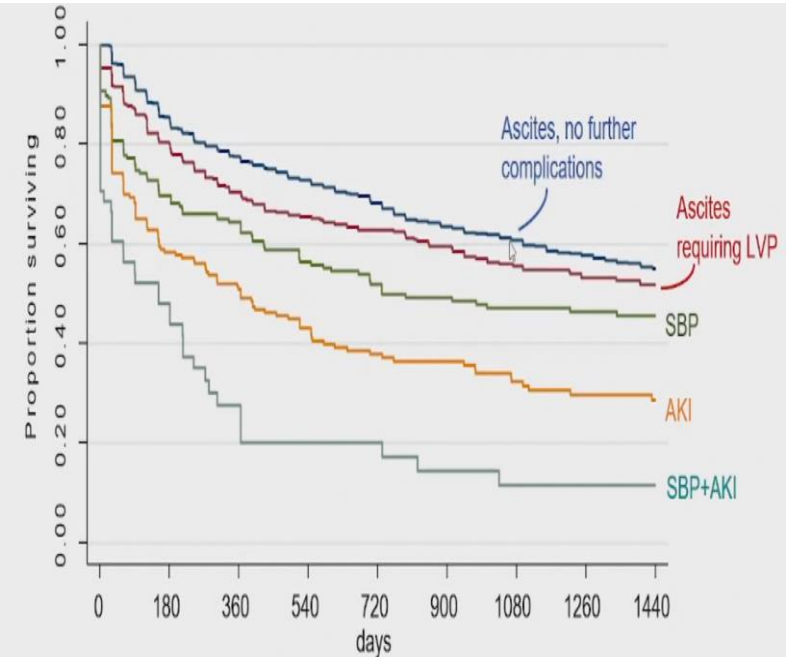


Cirrosis gravemente descompensada: Peor supervivencia de las complicaciones de la ascitis comparadas con ascitis no complicada

- 1,392 patients with ascites at inclusion or during follow-up.

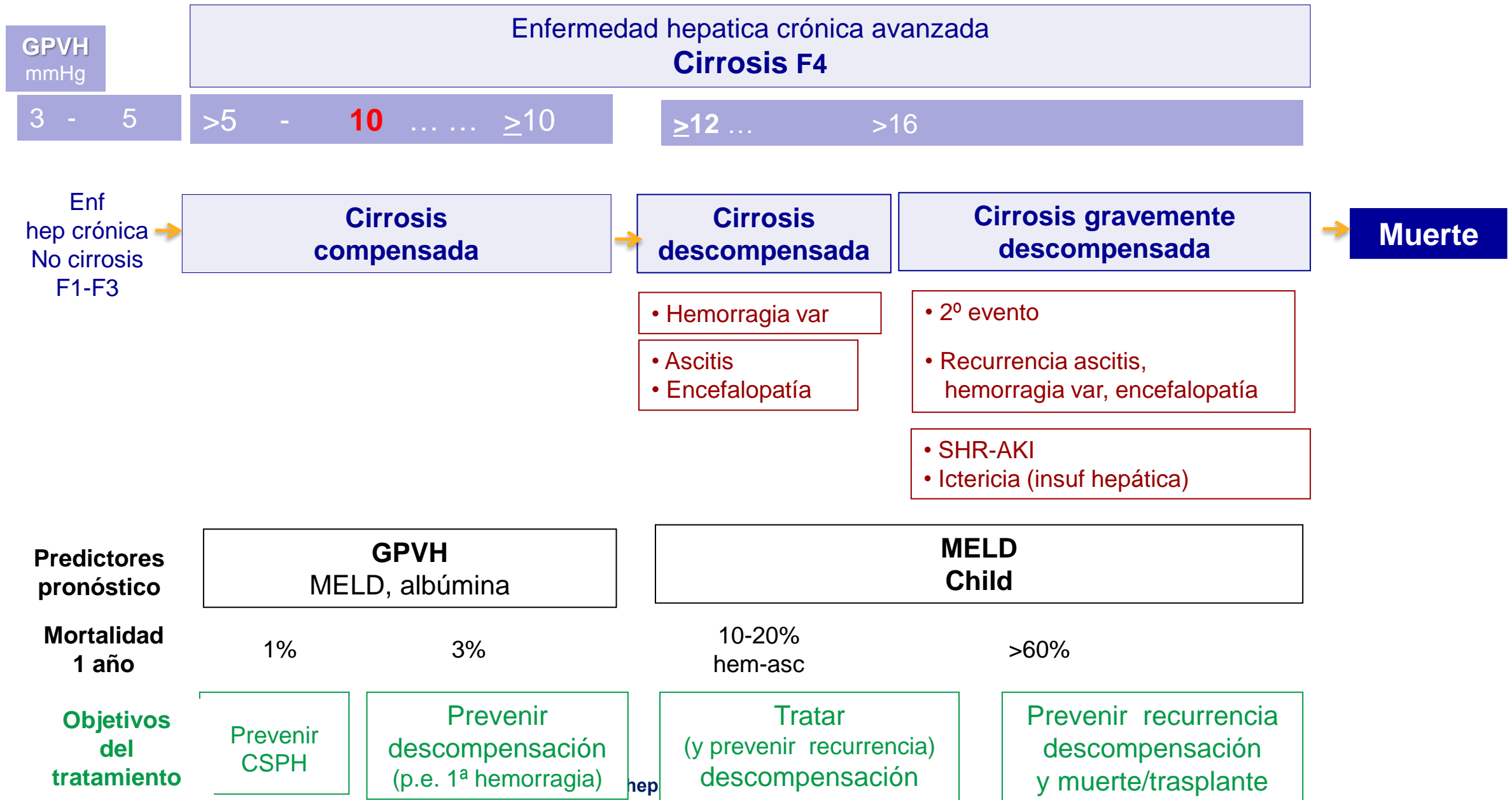


- 1775 patients with first ascites at inclusion or during follow-up



G D'Amico et al. Unpublished data

Estadios y subestadios de la cirrosis

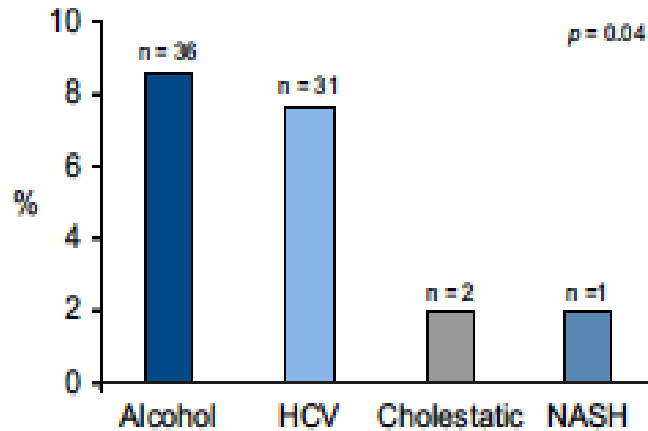


De-listing/re-compensation of patients with decompensated cirrhosis after treatment of etiology

8.6% with alcoholic cirrhosis delisted for improvement

Criteria for delisting: absence/easy control of complications
And improvement in liver function

Proportion of patients delisted according to etiology



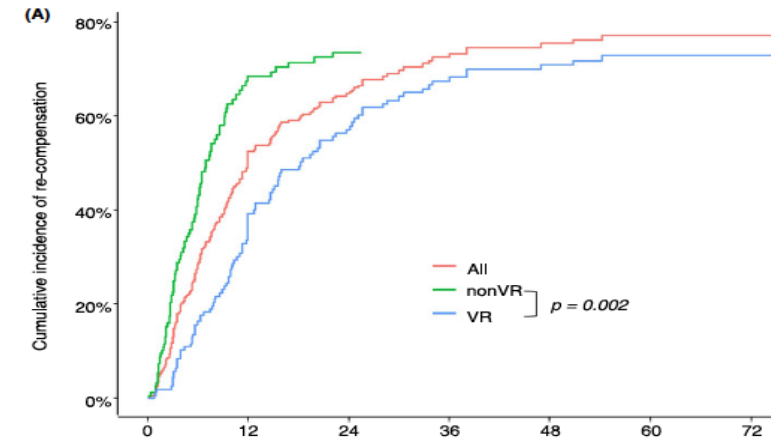
1,001 patients with decompensated cirrhosis
420 with alcoholic cirrhosis, abstinence >6 months

E Pose et al, JHEP 2021

62% with HBV cirrhosis delisted for improvement

Criteria for delisting: recovery to Child A5

Re-compensation by viral response to NUC

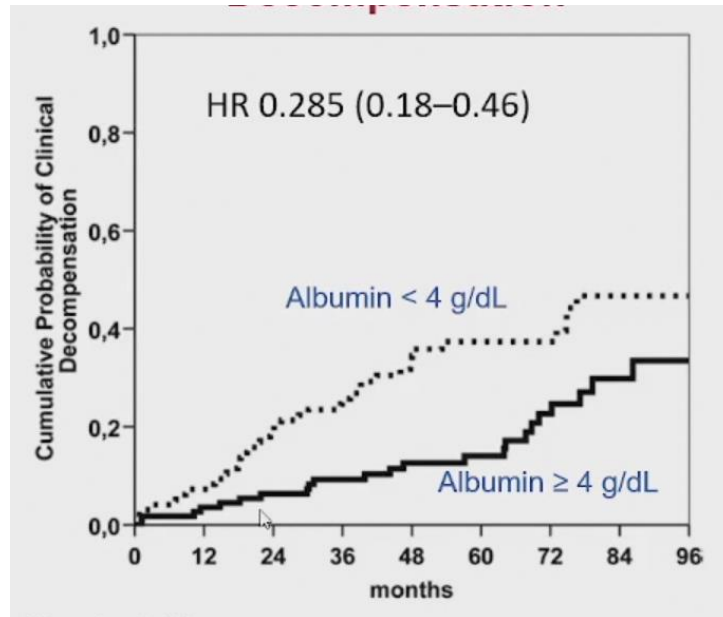


311 patients with HBV decompensated (Child B/C) cirrhosis
on NUC listed for liver transplantation

R Kim et al, APT 2021

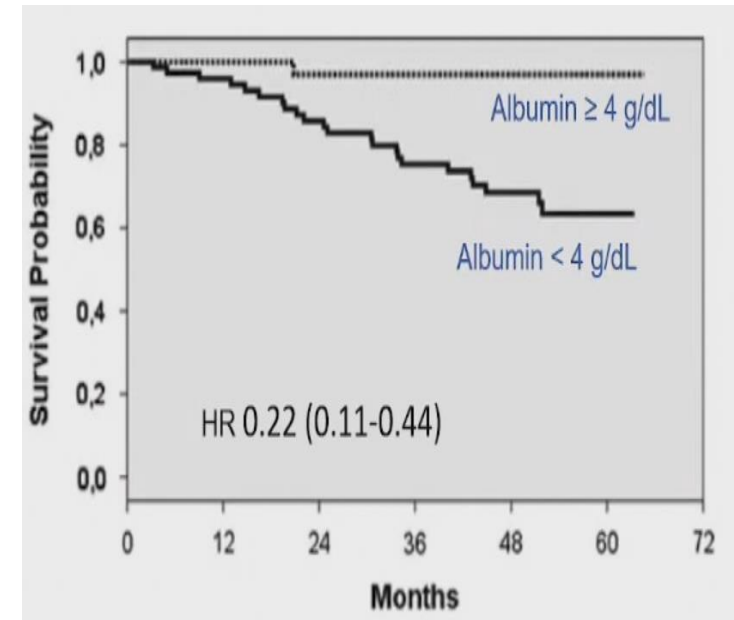
Serum albumin: strong predictor of decompensation in compensated cirrhosis

Decompensation



C Ripoll et al, Gastroenterology 2007

Survival



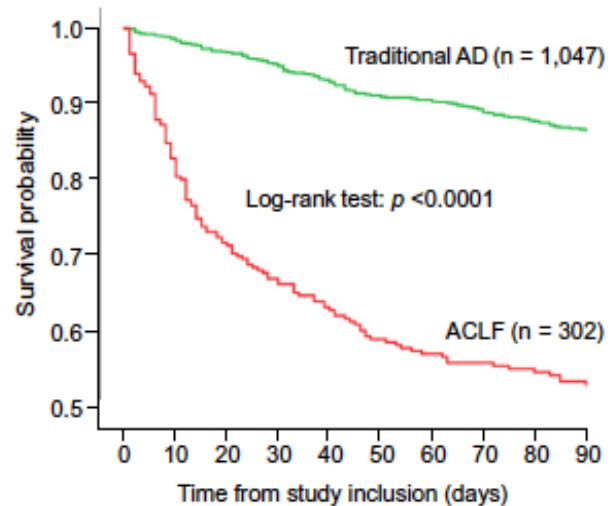
C Ripoll et al, JCG 2015

Acute-on-chronic liver failure (ACLF): The need of a new concept in acute hepatic decompensation

'Acute deterioration in liver function over a period of 2–4 weeks leading to severe deterioration in clinical status with a high SOFA/APACHE II score **with** jaundice and either hepatic encephalopathy or renal failure'

R Jalan, R Williams. Blood Purification 2002

Survival curves in patients with acute decompensation and ACLF (CANONIC study)



R Moreau et al. Gastroenterology 2013

Acute decompensation of cirrhosis:

- Ascites, encephalopathy, bleeding, encephalopathy, infection
- Worsening of survival (~2 yr)

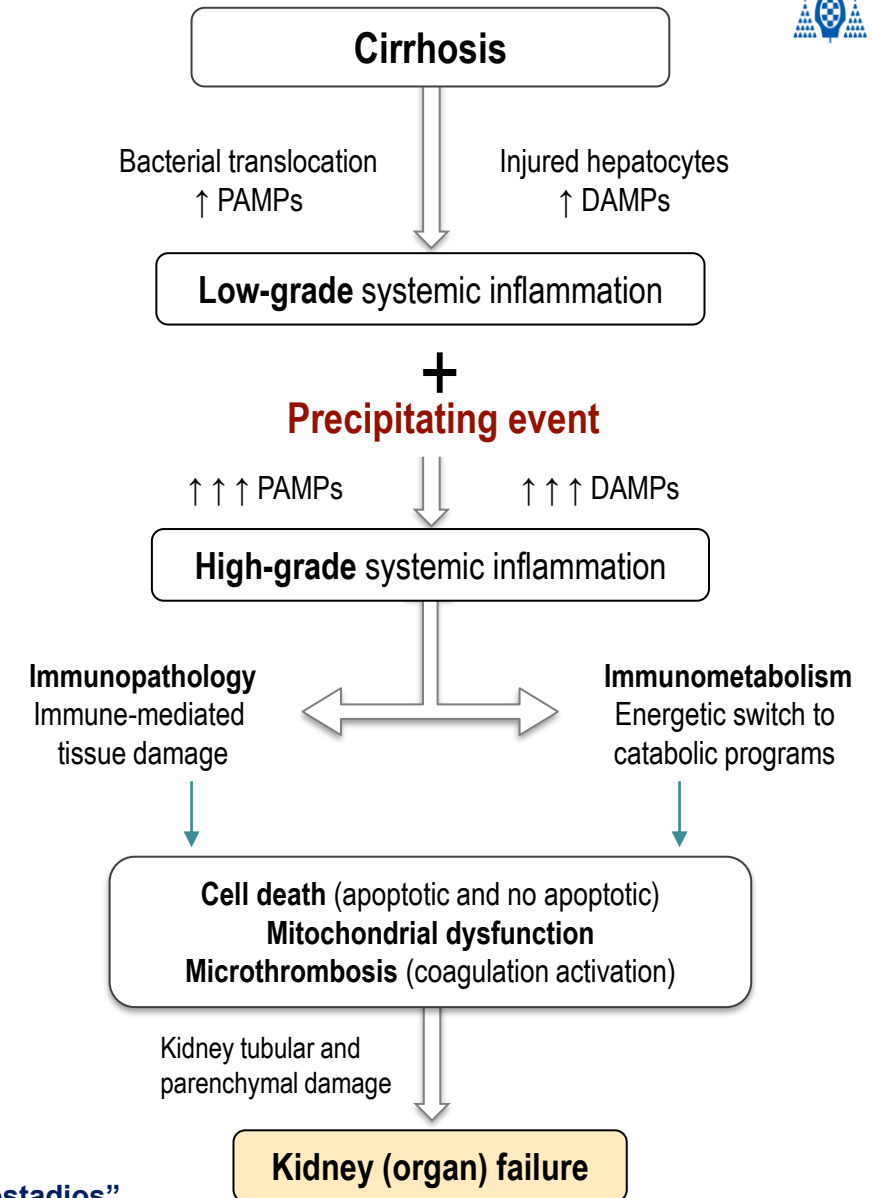
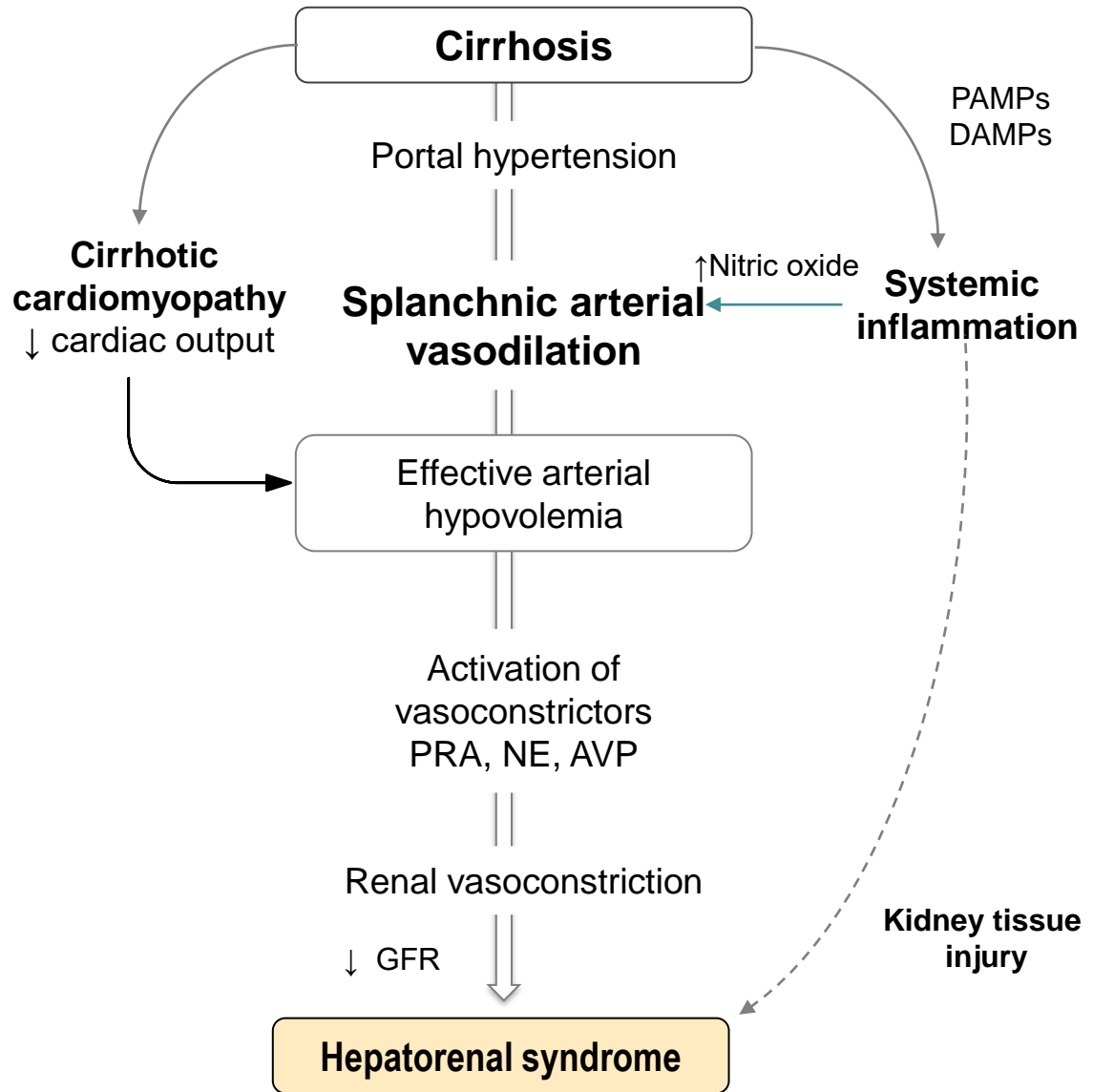
Acute decompensation of cirrhosis **AND**:

- **Organ failure** (hepatic and/or extrahepatic)
- **High short-term mortality**

Definitions

- Ascites and jaundice: **APASL**
- Organ failure: European (**EASL-CLIF**) and North America (**NACSLs**)

Portal hypertension, circulatory dysfunction and systemic inflammation as drivers of cirrhosis progression

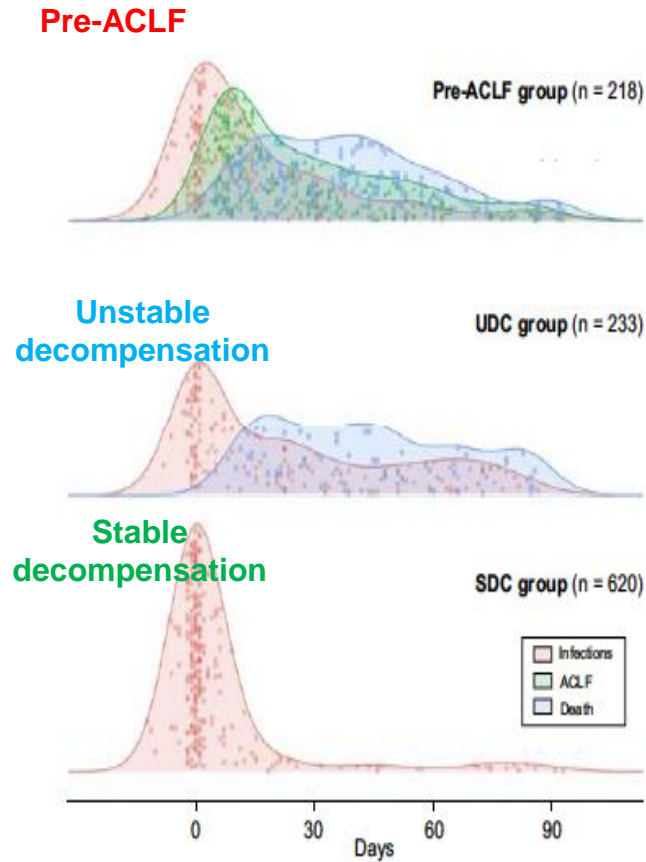


Acute decompensation in cirrhosis: three clinical courses

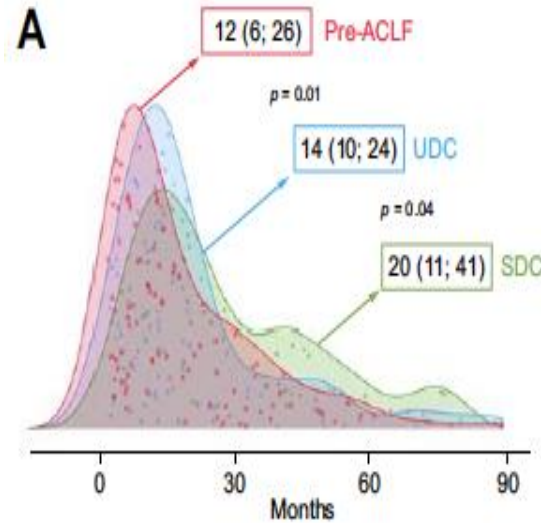
PREDICT study

1071 patients with cirrhosis with acute decompensation
Follow-up 3 months and 1 year after the event

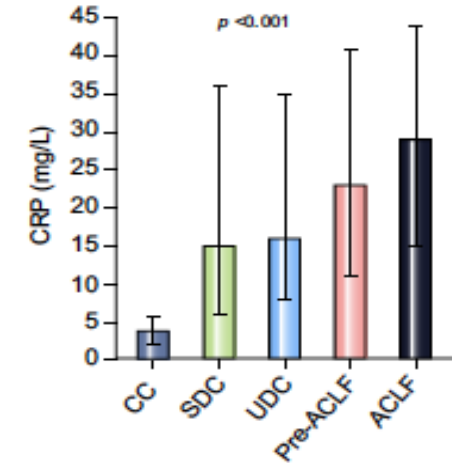
Density curves of events



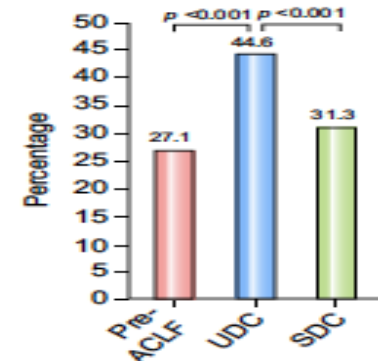
Density curves of LTx/death



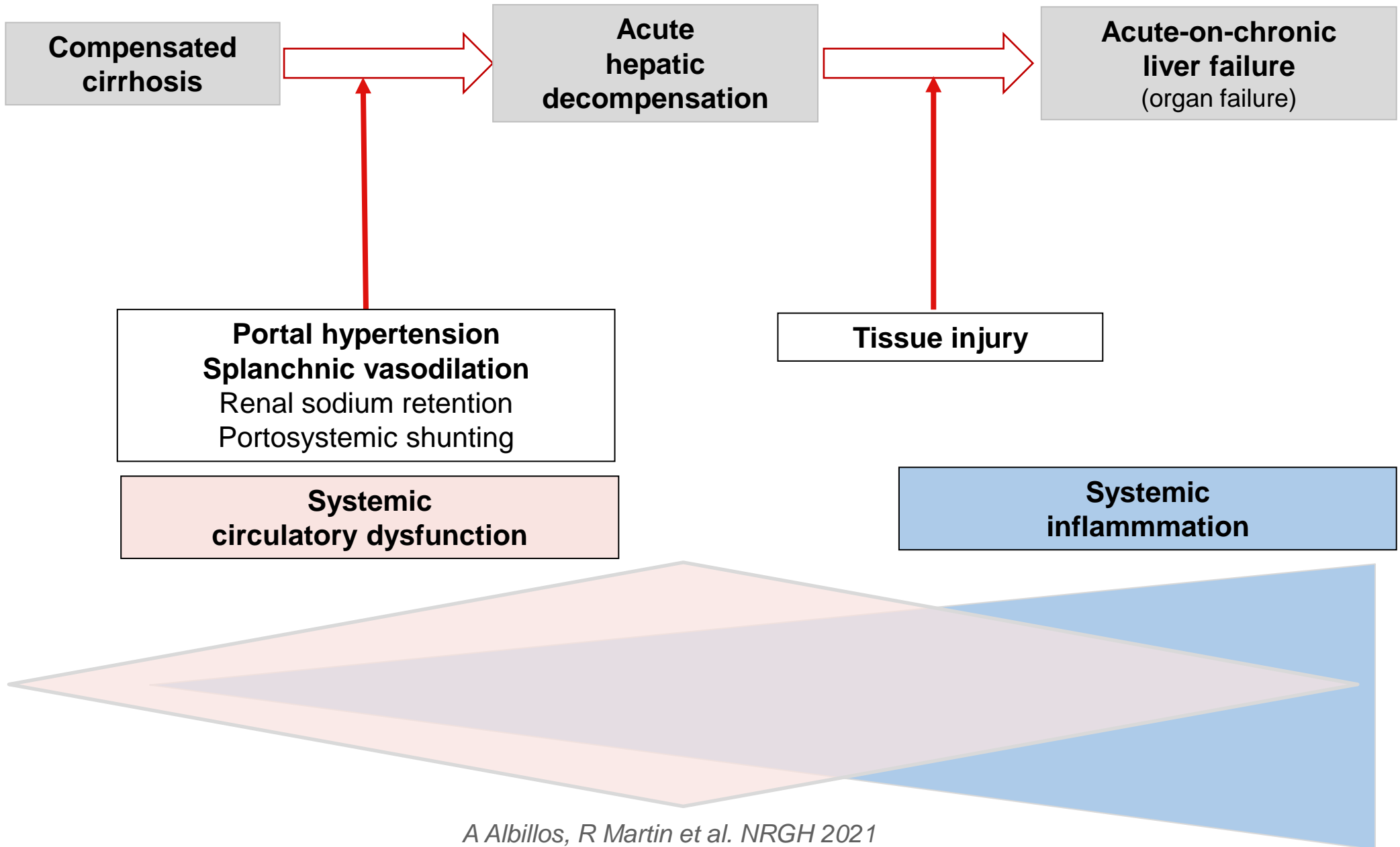
C-reactive protein



Surrogate of severe portal hypertension



Drivers of cirrhosis progression





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