APPROACH TO THE PATIENT WITH DECOMPENSATED CHRONIC HEPATITIS B AND CIRRHOSIS CLINICAL CASE

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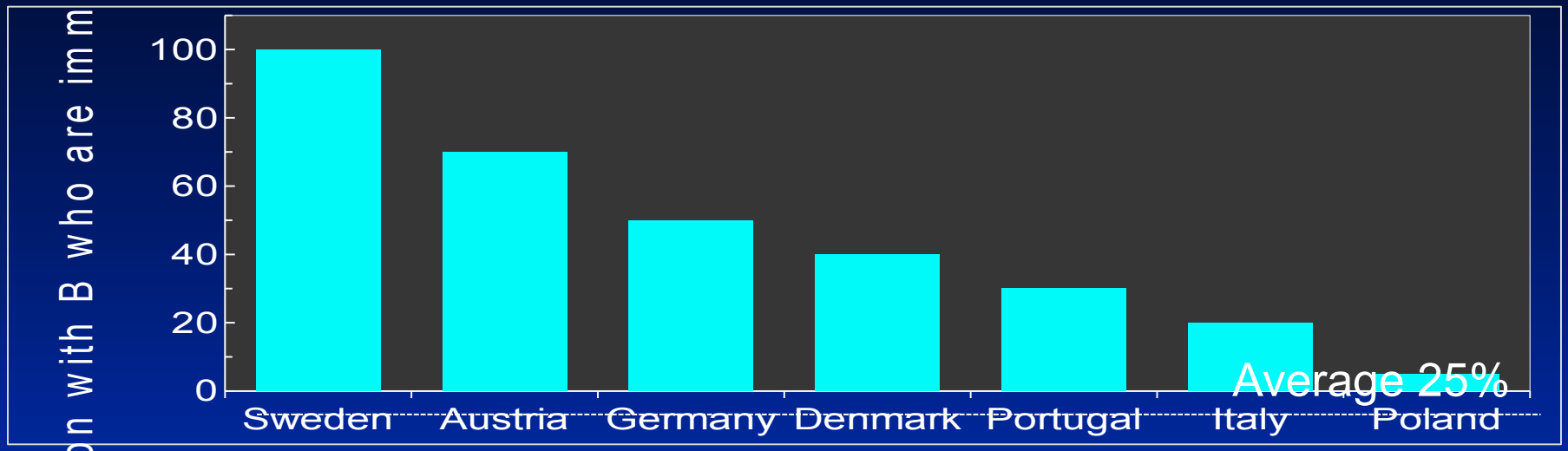
CHRONIC HBV EPIDEMIOLOGY

- Approximately 250 million persons worldwide
- Highest prevalence:
  - Asia, Sub-Sahran African, Eastern Europe
- A high percentage of patients in other countries are immigrants from these endemic areas:
  - USA 50% of persons are immigrants
  - EU 25% of persons are immigrants
- Immigrants with HBV often undiagnosed:
  - Lack of universal screening
  - Cultural/financial barriers limit access to health care

A Schweitzer et al. Lancet 2015; 386:1546-1555.
AA Ahmad et al. BMC Infectious Dis. 2018; 18:34.

CHRONIC HBV IN EU
IMPACT OF IMMIGRATION



Netherlands
Ireland
UK
Estonia
France
Spain
Croatia
Malta
Latvia
Iceland
Greece
Czech R
Bulgia
Romania
Slovakia

AA Ahmad et al
BMC Infectious Dis. 2018; 18:34.

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HB DECOMPENSATED CIRRHOSIS CASE

- 45 year old male
- Immigrated to the USA from Afghanistan
- Elevated serum liver enzymes 5 years later
- PMH: HTN, DM
- Serology:
  - HBsurface antigen positive. Titer 1,200 IU/ml
  - HBEantigen negative
  - Anti-HBE positive
  - HBV DNA 1,200 IU/ml
- Ultrasound: Echogenic consistent with steatosis
- Fibroscan: 15 kPa, CAP 285

CASE
LABORATORY DATA

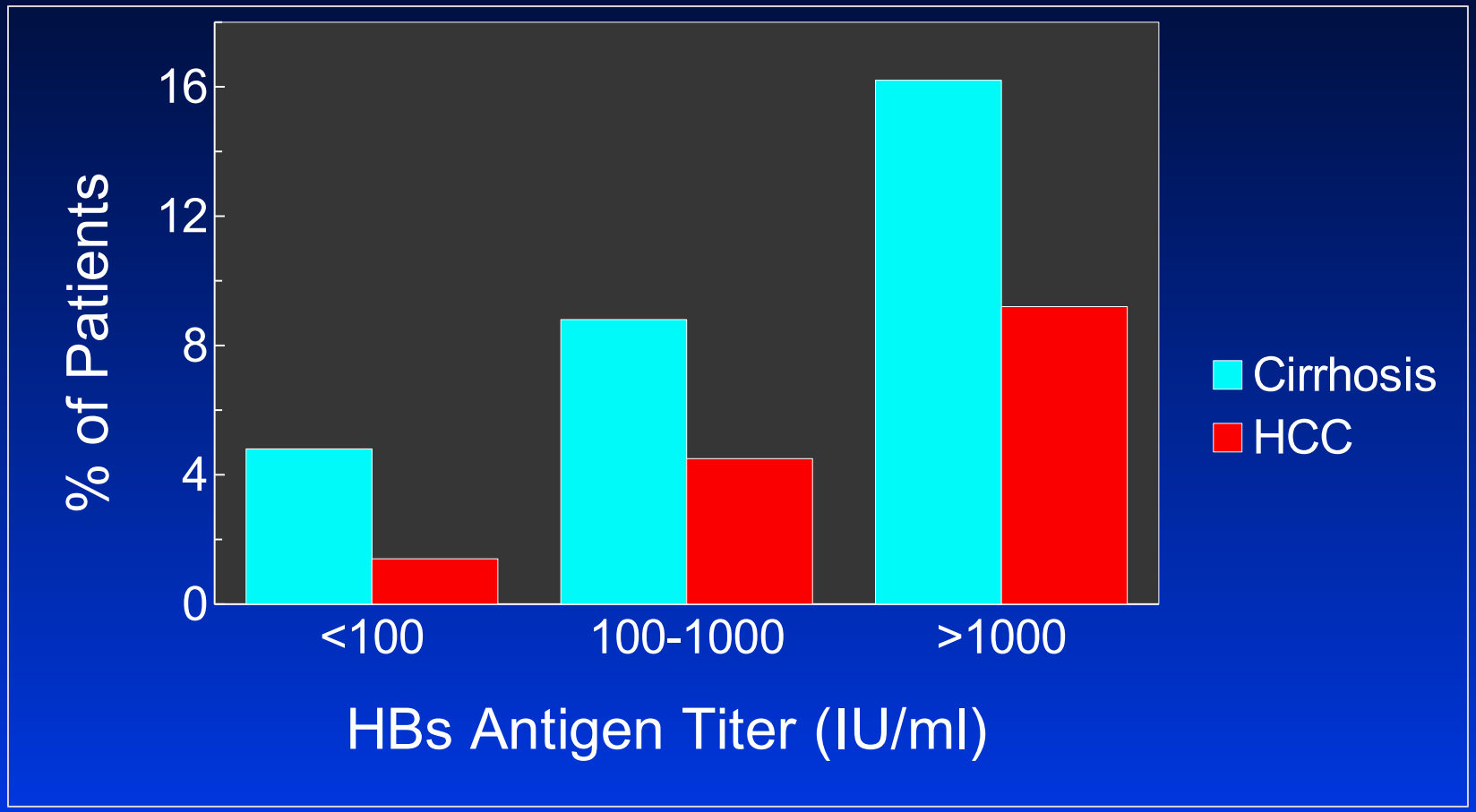
	Baseline
BMI (kg/m^2)	30.5
AST/ALT (IU/L)	85/71
Total bilirubin (mg/dl)	1.0
Albumin (gm/dl)	3.6
Sodium (mEq/L)	141
Creatinine (mg/dl)	0.9
Platelet Count	135,000

CTP Score: 5
MELD Score: 8

TREATMENT OF CHRONIC HBV CIRRHOSIS

- All patients with chronic HBV and cirrhosis should be treated
- Tenofovir disoproxil furmarate (TDF) 300 mg QD
- Tenofovir alafenamide (TAF) 25 mg QD
- Entecovir 0.5–1.0 mg QD
- Major reason for viral breakthrough or non-response is non-compliance
- Life long treatment required
- Goal of therapy is to prevent flair which could precipitate decompensation

HB SURFACE ANTIGEN TITER
RISK OF CIRRHOSIS AND HCC



J Liu et al.
Hepatology 2013; 57:441-450

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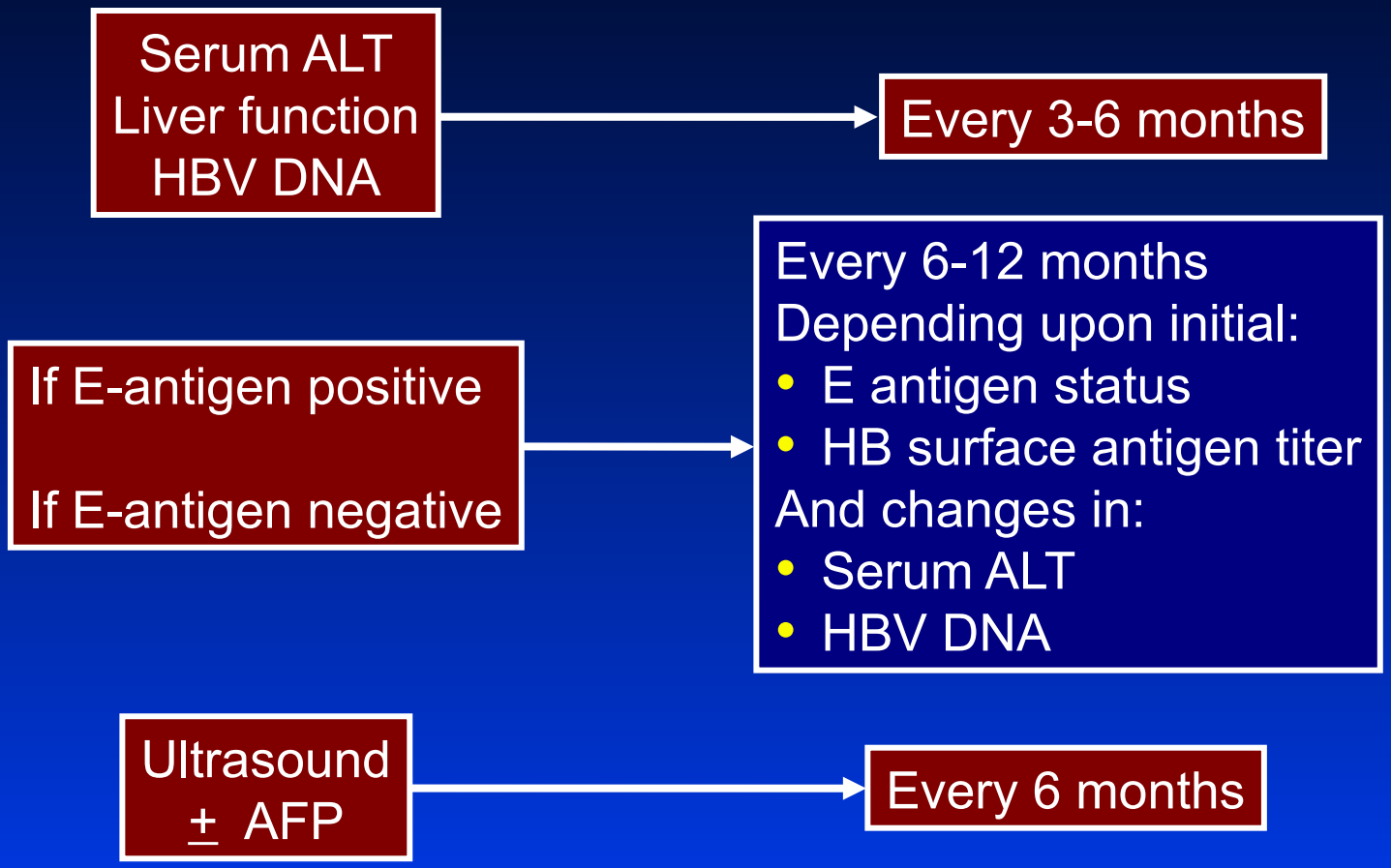
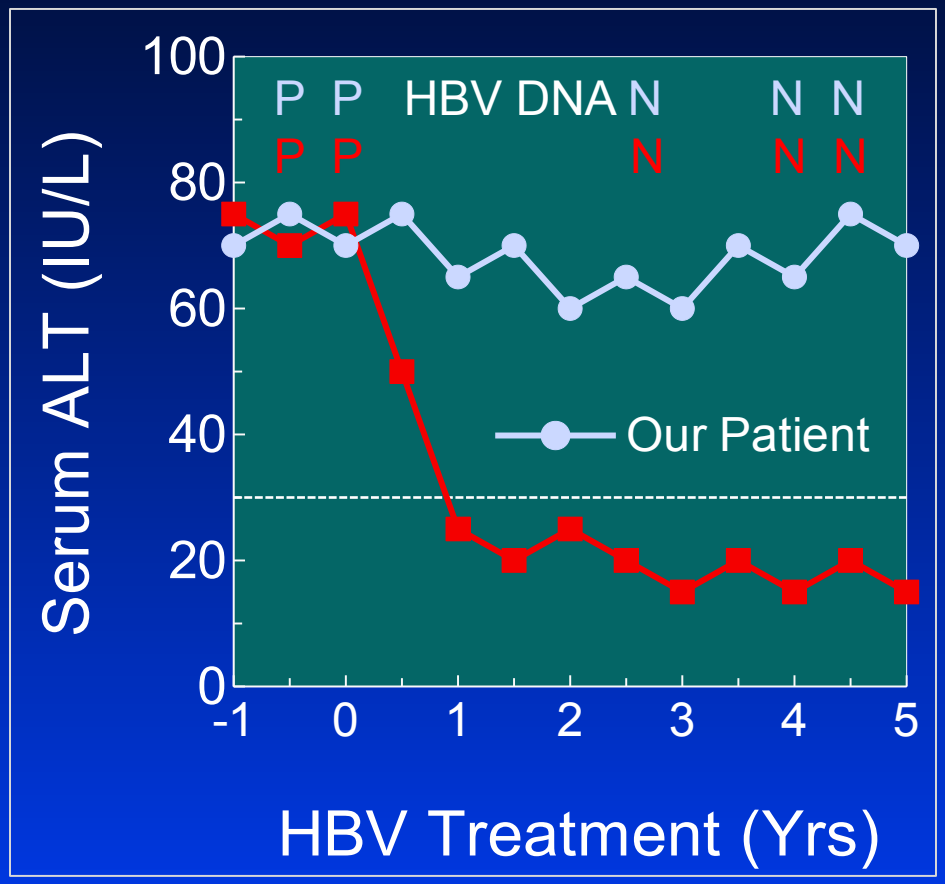
MONITORING TREATMENT OF HBV CIRRHOSIS



AS Lok, BJ McMahon
Hepatology 2009; 50:1-36.

TREATMENT OF CHRONIC HBV CASE



Lack of Biochemical Response:

- Persistent viremia
  - Viral resistance
  - Non-compliance
- Co-existent liver disease
  - NAFLD
  - Chronic HCV
  - Autoimmune hepatitis
  - ETOH

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HB DECOMPENSATED CIRRHOSIS CASE

- Chronic HBV with cirrhosis
- HBV DAA therapy initiated → UD in 3 months.
- Over the next 5 years:
  - HBV DNA remained undetectable
  - AST/ALT declined remained elevated
  - Gradual decline in PLT, ALB
  - Gradual rise in TBILI, INR, Screatinine
- At year 5:
  - Hepatic encephalopathy
  - Ascites
  - Spontaneous bacterial peritonitis

CASE
LABORATORY DATA

	Baseline	5 years
BMI (kg/m^2)	30.5	33.1
AST/ALT (IU/L)	85/71	91/75
Total bilirubin (mg/dl)	1.0	2.4
Albumin (gm/dl)	3.6	2.4
Sodium (mEq/L)	141	135
Creatinine (mg/dl)	0.9	1.9
Platelet Count	135,000	94,000

CTP Score: 5 → 10
MELD Score: 8 → 19

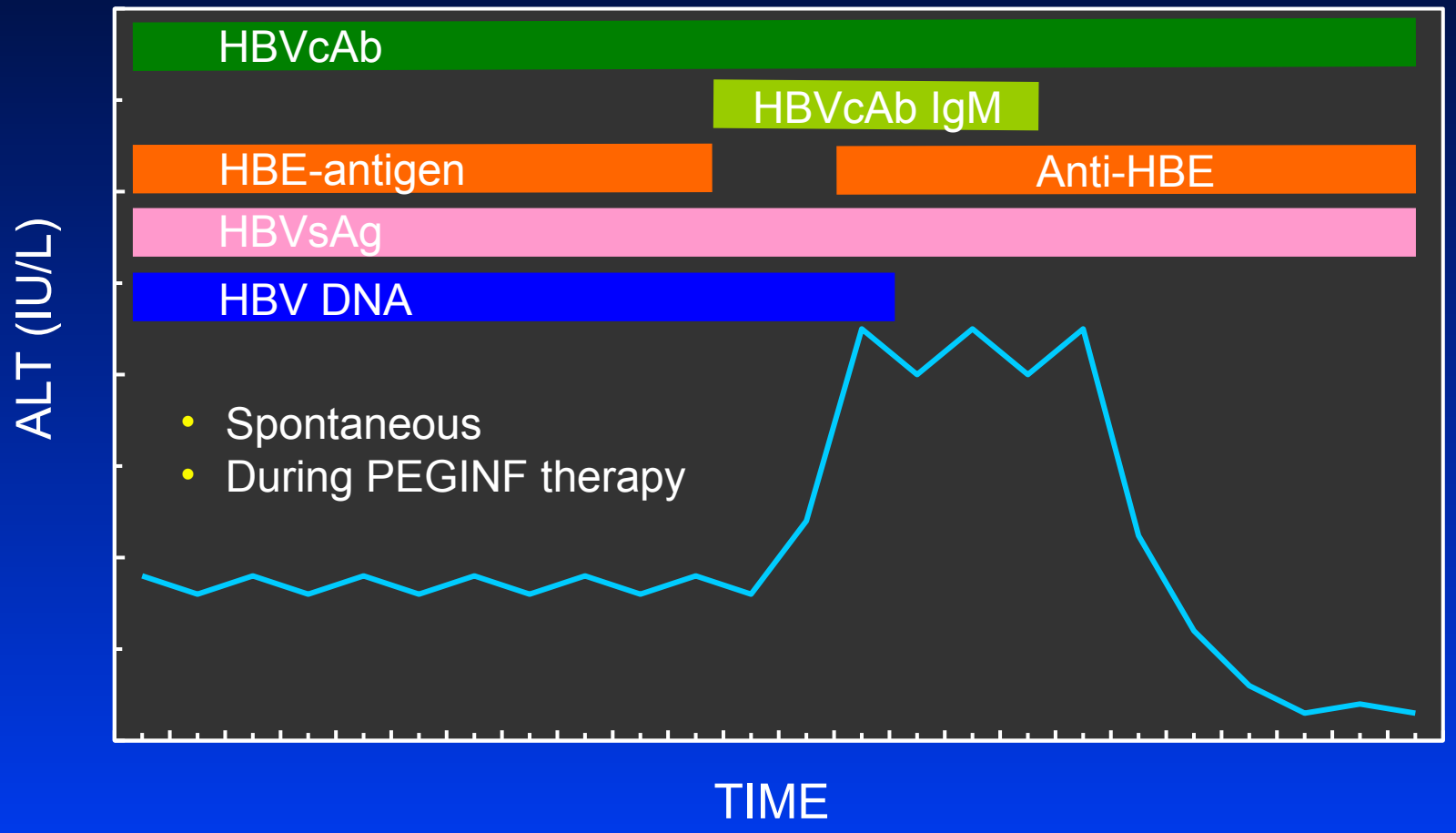
HB DECOMPENSATED CIRRHOSIS DUE TO HBV

HBV unknown HBV known but not being treated	Intervention

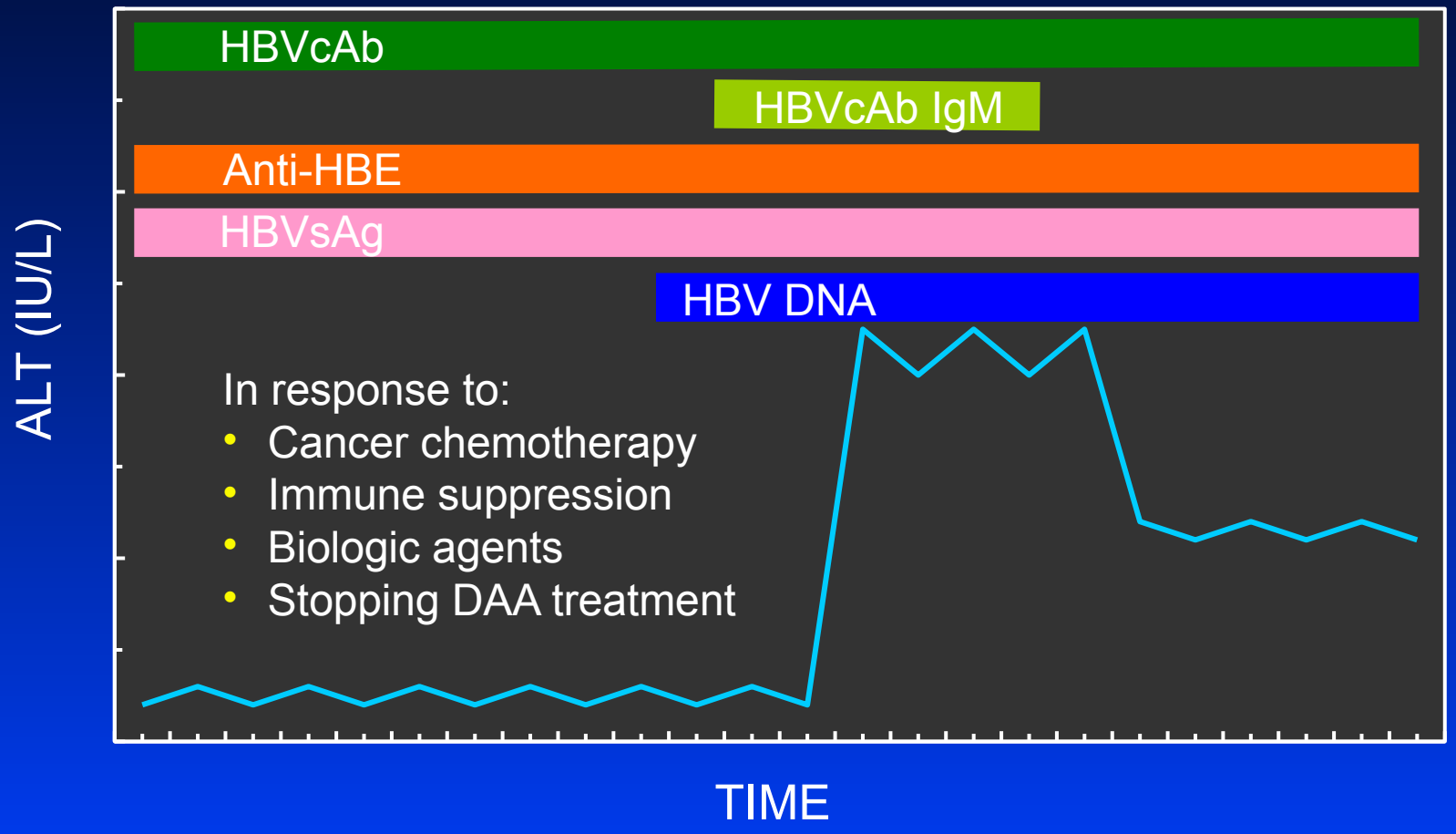
Chronic progression of active HBV
HBV flair as a result of:
• E-antigen seroconversion
• Treatment of HCV
• Cancer chemotherapy
• Treatment of immune disease
  ▪ High dose corticosteroids
  ▪ Calcineurin inhibitors
  ▪ Biologic agents
	Start or restart oral HBV treatment
	Stop inciting agent:
	• Chemotherapy
	• Immune suppressive agent
	• PEGINF
	Consider for transplant if liver function does not improve

HBV being treated • Flair during treatment with PEGINF • Stopping oral anti-viral therapy	

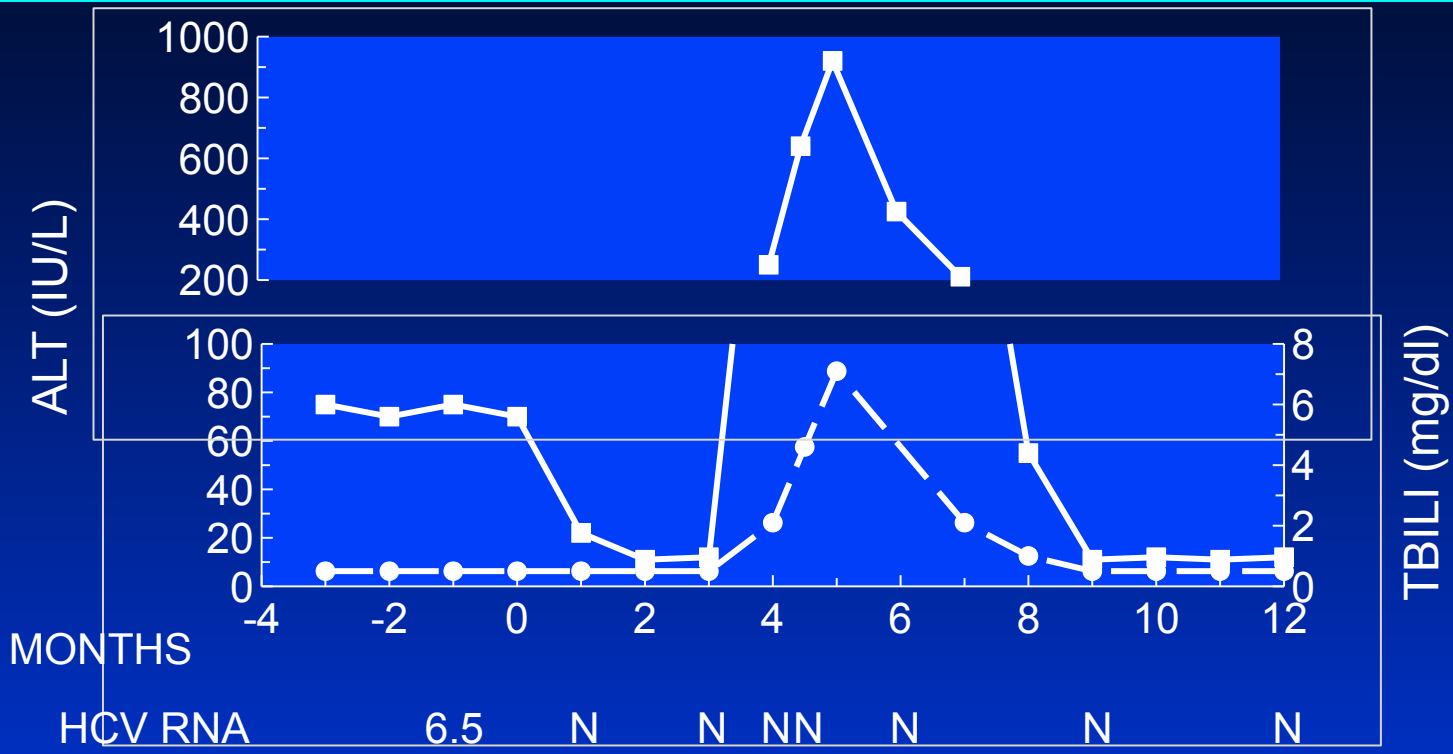
HBV FLAIR
E-ANTIGEN SEROCONVERSATION



HBV FLAIR
REACTIVATION OF INACTIVE HBV



HBV FLAIR
CHRONIC HCV TREATMENT



HCV RNA	6.5	N	N	NN	N	N	N
HBsAg	N			P	P	P	P
Anti-HBcore	P			P	P	P	P
Anti-HBsurface	N			N	N		
HBV DNA				5.4	4.1	2.9	1.7

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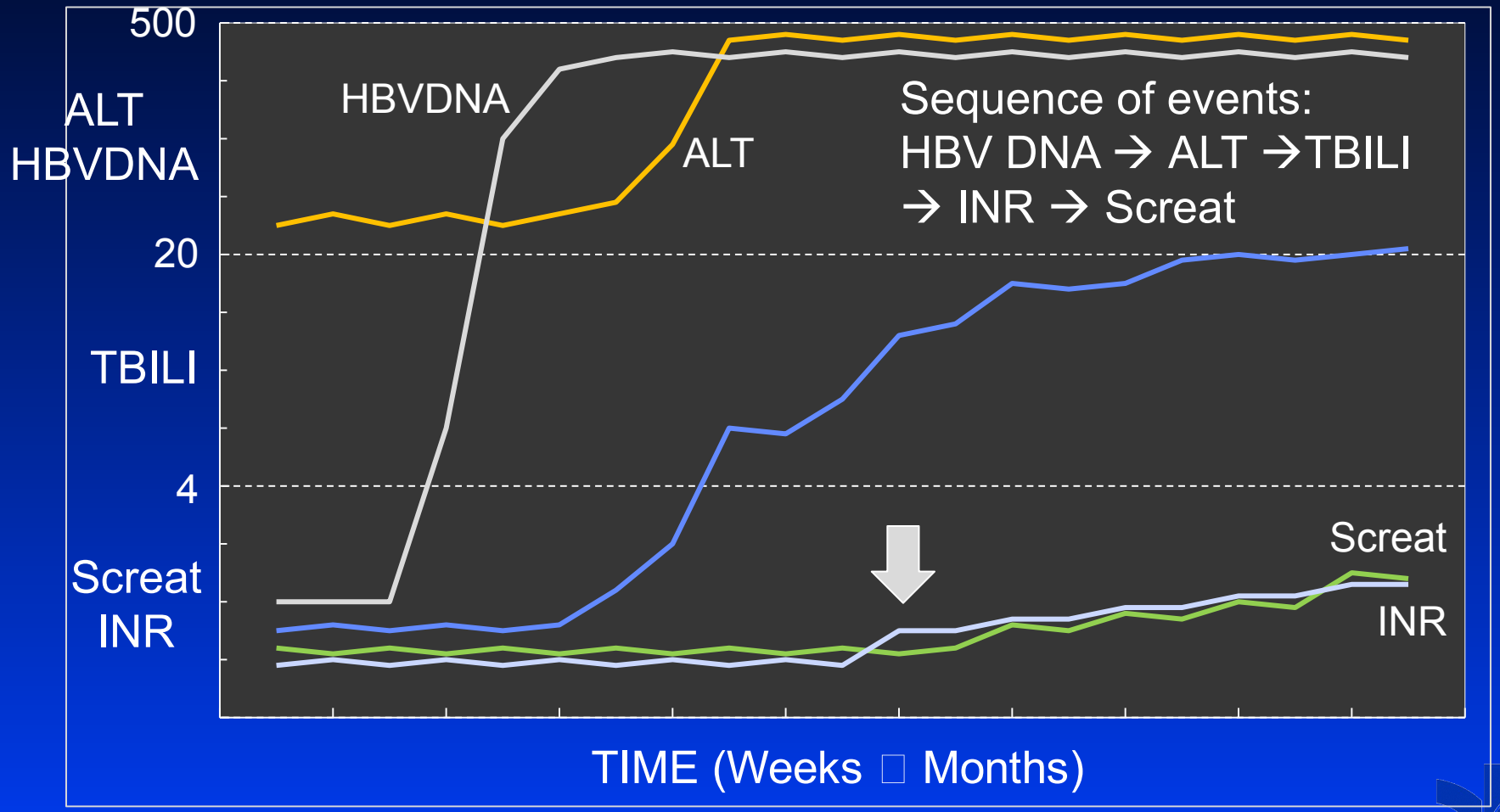
HB DECOMPENSATED CIRRHOSIS NOT DUE TO HBV

HBV inactive HBV suppressed on treatment	Intervention
Progression of another liver disease Acute alcoholic hepatitis Acute non-HBV hepatitis Drug induced liver injury • Cancer chemotherapy • Check-point inhibitors	Treat or continue treating HBV Treat the inciting agent Stop the inciting agent Consider for transplant if liver function does not improve

HB DECOMPENSATED CIRRHOSIS
CLINICAL COURSE



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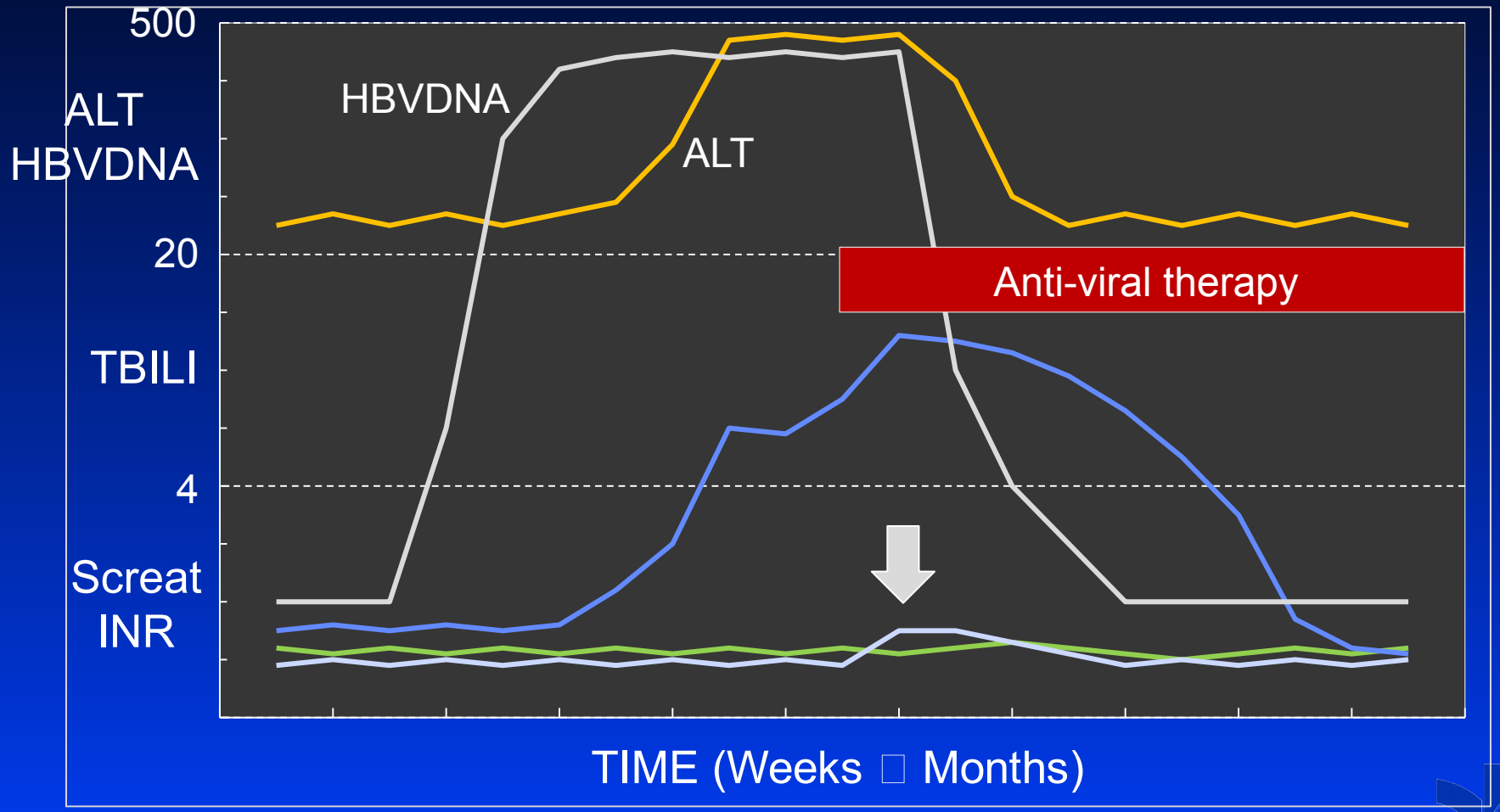
HB DECOMPENSATED CIRRHOSIS
IMPACT OF ANTI-VIRAL THERAPY



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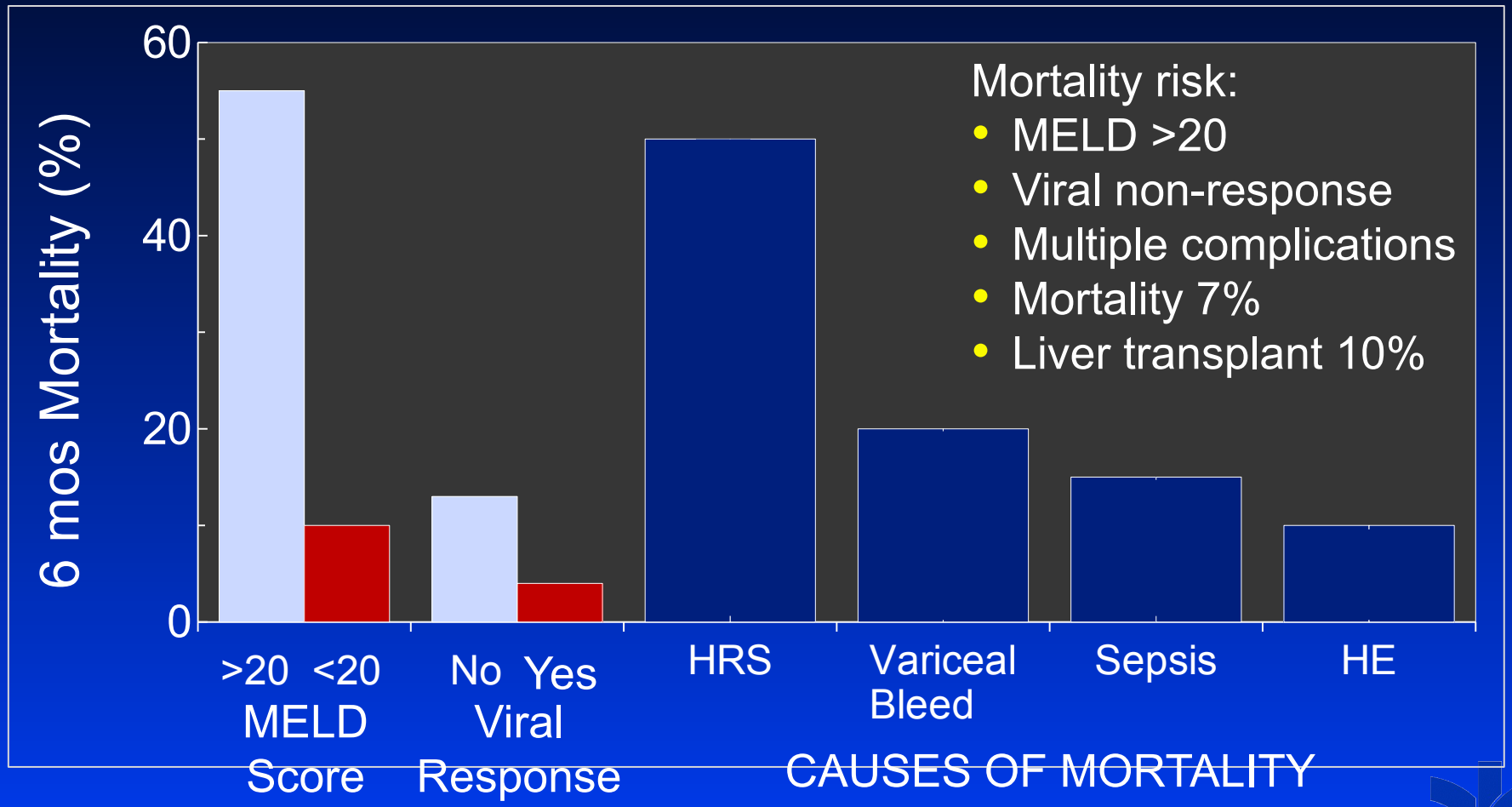
HB DECOMPENSATED CIRRHOSIS
FACTORS AFFECTING SURVIVAL



JW Jang et al.
Clin Gastroenterol Hepatol 2018; 16:1954-1963.

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HB DECOMPENSATED CIRRHOSIS CASE

- SBP treated with IV albumin, ABX.
- Liver ultrasound demonstrated new hypoechoic 2cm mass in right lobe
- Dynamic MRI confirmed HCC LIRADS-5
- HCC treated with embolic therapy
- Evaluated and listed for liver transplant
- Underwent liver transplant
  - HBIG given immediately prior to and after transplant
  - Anti-viral therapy continued after transplant
  - Liver explant demonstrated cirrhosis, steatosis and stained positive for HB surface antigen

HB DECOMPENSATED CIRRHOSIS SUMMARY

Many patients with B come from lands far away
If this is not found then the virus could prey
But if DNA if low and its inactive, as they say
A low S antigen, may just save the day

All patients with cirrhosis need to be treated
Entecovir, TDF or TAF, will not be defeated
Unless the patient stops taking the med
And the Hep B virus, awakens from bed

HB DECOMPENSATED CIRRHOSIS SUMMARY

Patients with B and cirrhosis can decompensate
The events that cause this can fill a big plate
Non-treated B can propagate
Treating with PEGINF can be the bait
Inactive B can reactivate
Liver injury from another cause may yield the same fate
And that is why we treat HBV, before its too late

HB DECOMPENSATED CIRRHOSIS SUMMARY

If flair and/or decompensation cause the BILI to soar
The key is, to watch the MELD score
And if the MELD remains less than 20
There is little to worry
And the patient is very likely, to make a recovery

However, if the MELD is too high
And complications of cirrhosis become a part of the pie
Then the patient is at risk to say goodbye
And we should consider giving liver transplant a try