



CHRONIC LIVER DISEASE AND SURGERY

**Registrar Teaching
Dr. Ronald Guevara**

LIVER

- The liver is vital for:
 - protein synthesis,
 - coagulation homeostasis,
 - glucose homeostasis,
 - bilirubin excretion,
 - drug metabolism, and
 - toxic removal, among other
- Liver has substantial functional reserve because of its dual blood supply: portal-venous (75%) and hepatic-arterial (25%).
- Clinical manifestations of liver damage occur only after considerable injury.



LIVER DYSFUNCTION – SIGNS AND SYMPTOMS

- **Spider nevi:** vascular lesions consisting of a central arteriole surrounded by many smaller vessels . From increased in estradiol.
- **Palmar erythema:** is a reddening of palms at the thenar and hypothenar eminences. From increased estrogen.
- **Gynecomastia:** From increased estradiol
- **Hypogonadism:** a decrease in sex hormones manifest as impotence, infertility, loss of sexual drive, and testicular atrophy
- Liver size can be enlarged, normal, or shrunken in patients with cirrhosis.
- **Ascites:** needs about 1500 ml to detect flank dullness.
- **Fetor hepaticus:** is a musty breath odor resulting from increased dimethyl sulfide.
- **Jaundice:** due to increased bilirubin (at least 2–3 mg/dL or 30 μ mol/L).



PORTAL HYPERTENSION

- Splenomegaly.
- Oesophageal varices.
- Caput medusa.
- Cruveilhier-Baumgarten murmur is a venous hum heard in the epigastric region



CAUSES OF LIVER DYSFUNCTION

- Globally, 57% of cirrhosis is attributable to either hepatitis B (30%) or hepatitis C (27%)
- Alcohol consumption accounts for about 20% of the cases.
- **Alcoholic liver disease (ALD)**: Alcoholic cirrhosis develops for 10–20% of individuals who drink heavily for a decade or more.
- **Non-alcoholic steatohepatitis (NASH)**: In NASH, fat builds up in the liver and eventually causes scar tissue. This type of hepatitis appears to be associated with obesity (40% of NASH patients) diabetes, protein malnutrition, coronary artery disease, and treatment with corticosteroid medications.
- **Chronic hepatitis C**: Infection with the [hepatitis C virus](#) causes inflammation of the liver and a variable grade of damage to the organ. Over several decades this inflammation and grade change can lead to cirrhosis.
- **Chronic hepatitis B**: The [hepatitis B virus](#) causes liver inflammation and injury that over several decades can lead to cirrhosis.
- **Primary biliary cirrhosis**: Damage of the bile ducts leading to secondary liver damage.



CAUSES OF LIVER DYSFUNCTION

- **Primary sclerosing cholangitis**: PSC is a progressive cholestatic disorder presenting with pruritus, steatorrhea, fat soluble vitamin deficiencies, and metabolic bone disease.
- **Autoimmune hepatitis**: This disease is caused by the immunologic damage to the liver causing inflammation and eventually scarring and cirrhosis
- **Hereditary haemochromatosis**: Usually presents with family history of cirrhosis, skin hyperpigmentation, diabetes mellitus, pseudogout, and/or cardiomyopathy, all due to signs of iron overload.
- **Wilson's disease**: Autosomal recessive disorder characterized by low serum ceruloplasmin and increased hepatic copper content on liver biopsy, and elevated 24-hour urine copper
- **Indian childhood cirrhosis**: a form of neonatal cholestasis characterised by deposition of copper in the liver.
- **Alpha 1-antitrypsin deficiency (A1AD)**: Autosomal recessive disorder of decreased levels of the enzyme alpha 1--antitrypsin.
- **Cardiac cirrhosis**: Due to chronic right sided heart failure which leads to liver congestion.
- **Galactosemia**
- **Glycogen storage disease type IV**
- **Cystic fibrosis**
- **Hepatotoxic drugs or toxins**



CIRRHOSIS

- Any patient with cirrhosis carries a risk of specific life-threatening complications such as variceal bleeding, sepsis, or hepatorenal syndrome.
- The general course of cirrhosis is characterized by longstanding phase of compensated cirrhosis, followed by occurrence of specific complications.
- 60% of develop decompensated cirrhosis 10 yrs after diagnosis.



OUTCOME OF CIRRHOSIS ACCORDING TO FOUR CONSECUTIVE CLINICAL STAGES

Medscape®

www.medscape.com

Clinical Status	Definition	Cumulative Probability of Death Per 1 Year	Cumulative Probability of Exiting this Status Per 1 Year
Compensated cirrhosis [†]			
• Stage 1	No varices, no ascites	1%	11%
• Stage 2	Varices, no ascites	3.4%	10%
Decompensated cirrhosis [†]			
• Stage 3	Ascites ± varices	20%	7.6%
• Stage 4	Bleeding ± ascites	57%	—

*From D'Amico G, Garcia-Tsao G, Pagliaro L. Natural history and prognostic indicators of survival in cirrhosis: a systematic review of 118 studies. *J Hepatol* 2006;44:217–231; and de Franchis R. Evolving consensus in portal hypertension: report of the Baveno IV consensus workshop on methodology of diagnosis and therapy in portal hypertension. *J Hepatol* 2005;43:167–176.⁹⁵

[†]Decompensated cirrhosis is defined by the presence of ascites, variceal bleeding, and/or encephalopathy.

Source: *Semin Liver Dis* © 2008 Thieme Medical Publishers



BUT...

- The course of cirrhosis is extremely variable from patient to patient due to several factors, including:
 - hepatic synthetic function,
 - the cause of cirrhosis,
 - the possibility of stopping or slowing the underlying damaging process to the liver, and
 - the occurrence of liver malignancy.



GRADING SYSTEMS

- **Child-Pugh:**
 - originally designed for predicting the outcome after surgery for portal hypertension (portocaval shunting and trans-section of the esophagus) in patients with cirrhosis
- **Model for End-Stage Liver Disease (MELD):**
 - originally designed for assessing the prognosis of cirrhotic patients undergoing transjugular intrahepatic portosystemic shunt (TIPS)



CHILD-PUGH SCORE

Parameter	Points assigned		
	1	2	3
Ascites	Absent	Slight	Moderate
Hepatic encephalopathy	None	Grade 1-2	Grade 3-4
Bilirubin micromol/L (mg/dL)	<34.2 (<2)	34.2-51.3 (2-3)	>51.3 (>3)
Albumin g/L (g/dL)	>35 (>3.5)	28-35 (2.8-3.5)	<28 (<2.8)
Prothrombin time			
Seconds over control	<4	4-6	>6
INR	<1.7	1.7-2.3	>2.3
CPT classification: Child A: score 5-6 (well compensated); Child B: score 7-9 (significant functional compromise); Child C: score 10-15 (decompensated)			



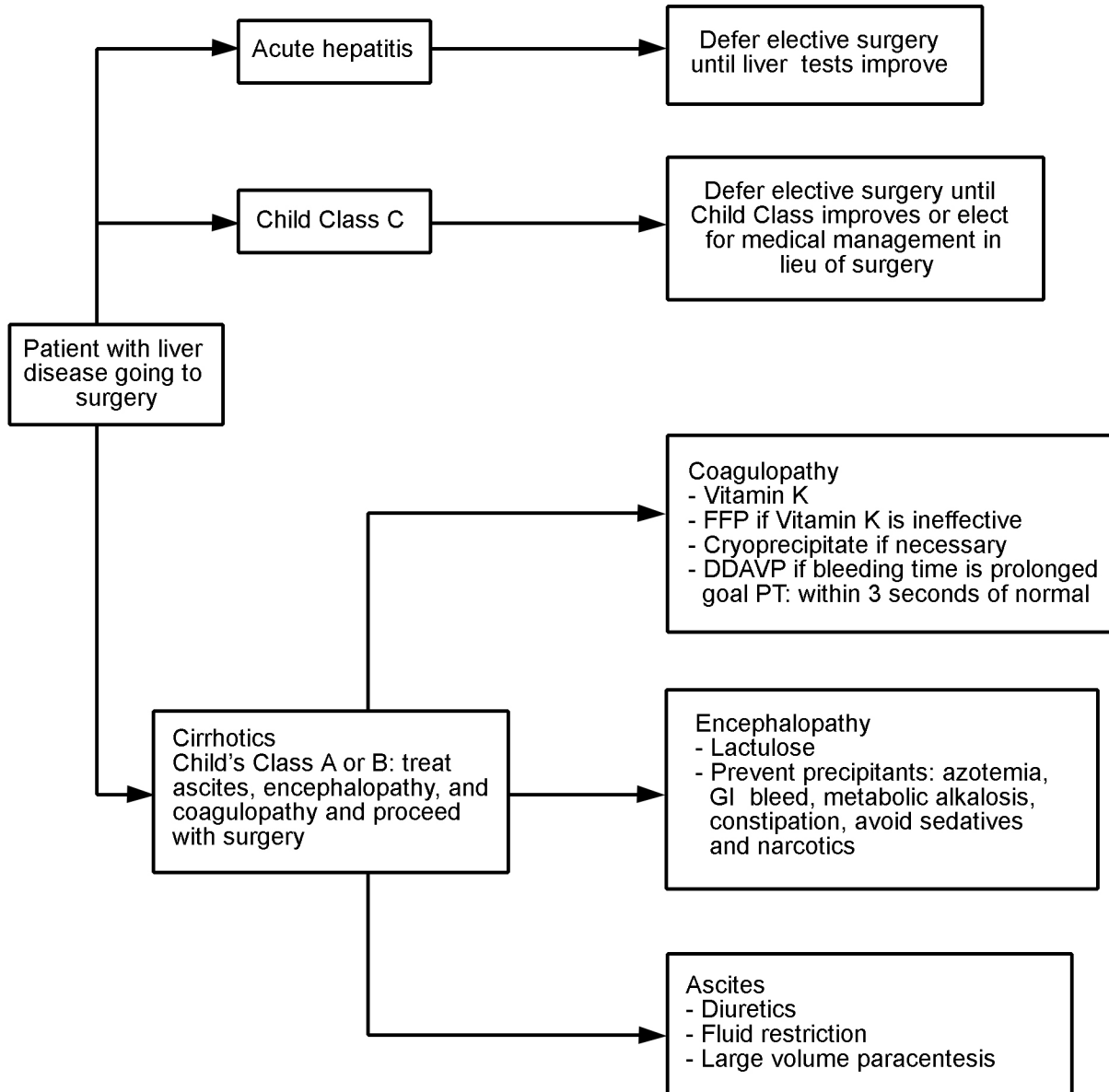
CHILD-PUGH AND SURGERY

- Class A disease are estimated to have a 10% mortality rate after abdominal surgery.
- For CTP class B that mortality rate increases to 30-31% and
- For CTP class C, the mortality rate is 76-82%.

- A 2010 study by Telem et al demonstrated lower mortality rates (CTP 2%, CTP B 12%, CTP C 12%) when surgeries were performed at an institution specializing in hepatology and liver transplantation.

- CTP scoring system has been challenged for its ambiguity and interobserver variability because it includes subjective parameters (eg, degree of ascites and encephalopathy).
- Additionally, all the factors are weighted equally. Patients within a given class are not homogenous but also not distinguished between, a feature for which it has also been criticized.^[10]





MELD

- Used since 2002 for organ allocation to patients listed for liver transplantation in the United States MELD score has also been adopted in several European countries as well as in South America.
- MELD score also proved to be a reliable marker of 1-year and 5-year survival across a broad spectrum of liver diseases including alcoholic cirrhosis and alcoholic hepatitis.
- In addition, MELD score has been shown to be a good prognostic marker in cases of
 - variceal bleeding,
 - spontaneous bacterial peritonitis, and
 - hepatorenal syndrome.
- A major limitation of MELD score is the need for computation, which makes it less friendly to use than Child-Pugh score at the bedside.



MELD SCORE AND ITS DERIVATIVES

Score	Components
MELD score*	$9.6 * \log_e (\text{creatinine mg/dL}) + 3.8 * \log_e (\text{bilirubin mg/dL}) + 11.2 * \log_e (\text{INR}) + 6.4$
MELD-sodium [†]	MELD + 1.59 * (135-Na [mEq/L])
MELD-XI	$5.11 * \log_e (\text{bilirubin mg/dL}) + 11.76 * \log_e (\text{creatinine mg/dL}) + 9.44$
Delta MELD	Difference between current MELD and the lowest MELD measure within 30 days prior to current MELD

*Values of creatinine, bilirubin, and INR below 1 are rounded to 1. Serum creatinine values above 4 mg/dL are rounded to 4. Patients on hemodialysis are given a creatinine value of 4 mg/dL. MELD score ranges from 6 to 40 points.

[†]Values of serum sodium below 120 mEq/L are rounded to 120. Values over 135 mEq/L are rounded to 135.

MELD, model for end-stage liver disease; INR, international normalized ratio.



MELD

- MELD score < 8 predicts good outcome after TIPS and a score >18 predicts poor outcome, with best outcomes seen in patients with scores < 14 .
- Avoidance of TIPS is generally recommended in patients with a MELD score >24 , unless the procedure is used as a measure of last resort to control active variceal bleeding.
- Since its implementation, the MELD score's use has been expanded to also predict the risk of mortality and morbidity after other procedures.
- A MELD score of at least 8 predicts an increased risk of postoperative complications, including death in patients undergoing cholecystectomy and cardiac surgery requiring cardiopulmonary bypass.



MELD

- MELD score has also been assessed for predicting non-transplant surgical mortality.
- There is approximately a 1% increase in mortality risk per MELD point below a score of 20.
- There is a 2% increase in mortality risk per MELD point over 20.
- Mortality is higher for intra-abdominal surgery (up to 25%) compared with other types of surgery.



REFERENCES

- <http://en.wikipedia.org/wiki/Cirrhosis>
- http://en.wikipedia.org/wiki/Child-Pugh_score
- <http://www.uptodate.com/contents/assessing-surgical-risk-in-patients-with-liver-disease>
- <http://emedicine.medscape.com/article/284667-overview#a1>
- <http://www.medscape.com/viewarticle/572659>

