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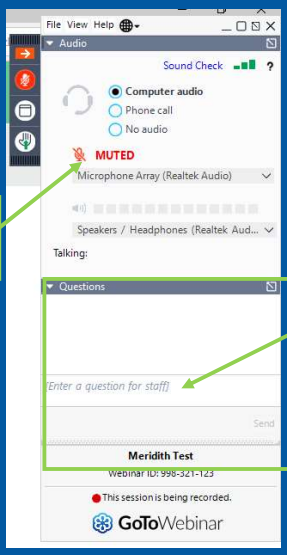
Participating in the Webinar

All attendees will be muted and will remain in Listen Only Mode.



Type your questions here so that the moderator can see them. Not all questions will be answered but we will get to as many as possible.

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How to Receive CME and MOC Points

LIVE VIRTUAL GRAND ROUNDS WEBINAR

ACG will send a link to a CME & MOC evaluation to all attendees on the live webinar.

ABIM Board Certified physicians need to complete their MOC activities by December 31, 2023 in order for the MOC points to count toward any MOC requirements that are due by the end of the year. No MOC credit may be awarded after March 1, 2024 for this activity.

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MOC QUESTION

If you plan to claim MOC Points for this activity, you will be asked to: Please list specific changes you will make in your practice as a result of the information you received from this activity.

Include specific strategies or changes that you plan to implement.
THESE ANSWERS WILL BE REVIEWED.

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ACG Virtual Grand Rounds

Join us for upcoming Virtual Grand Rounds!

Week 7 –Thursday, February 16, 2023
Post-COVID-19 Disorders of Gut-Brain Interaction/Functional Gastrointestinal Disorders
Faculty: Max L. Schmulson, MD
Moderator: Sarah K. McGill, MD, MSc, FACG
At Noon and 8pm Eastern

Week 8 – Thursday, February 23, 2023
3rd Space Endoscopy
Faculty: Gregory B. Haber, MD
At Noon and 8pm Eastern

Visit **gi.org/ACGVGR** to Register

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Disclosures

Ayse Aytaman, MD, FACG
Exact Sciences: Research Grant

Janice Jou, MD
Gilead: Research Grant

**All of the relevant financial relationships listed for these individuals have been mitigated*

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Liver Cancer Update and Review for the Gastroenterologist

Ayse Aytaman, MD, FACG
Chief of Gastroenterology and Hepatology
VA New York Harbor HCS Brooklyn
Clinical Professor of Medicine SUNY Downstate College of Medicine
Director VISN 2 Liver Team and Liver Tumor Board

VA Liver Cancer Team

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Educational Objectives

- Understand changes in epidemiology,
- Review available data on prevention
- Appreciate changes on surveillance considering challenging populations
- Go over the diagnostic challenges and role of imaging and liver biopsy
- Evaluate the staging of HCC
- Understand rapidly changing treatment landscape with an overview of curative and palliative options
- Review role of liver transplantation basics for HCC
- Role of palliative care, end of life care, frailty
- Impact of multidisciplinary teams, dedicated tumor boards and population health management

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Why is HCC so challenging?

HCC is unique among cancers:

- 1 patient, 2 diseases
  Cirrhosis leads to
  - multifocal liver cancer
  - high recurrence rates
- Cirrhosis complicates treatment and trial design
- HCC can be diagnosed by imaging alone
- HCC is the only solid organ malignancy for which transplant offers cure

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Epidemiology

Changing trends

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HCC: GLOBAL HEALTH CHALLENGE

- 3rd leading cause of cancer-related death worldwide
- Growing incidence worldwide.
- Estimates: >1 million affected individuals by 2025.
- Most common form of liver cancer (>90%).
- Most prominent risk factors for HCC:
  - HBV in most parts of Asia, Mongolia
  - HCV in Western Europe, North America, Japan
  - ETOH in Central and Eastern Europe
- Non-alcoholic steatohepatitis (NASH) is becoming the fastest growing etiology of HCC, particularly in the West.
- Mutational signatures have established aristolochic acid and tobacco as potential pathogenetic cofactors.

Llovet, J.M. *et al.* Hepatocellular carcinoma. *Nat Rev Dis Primers* **7**, 6 (2021).

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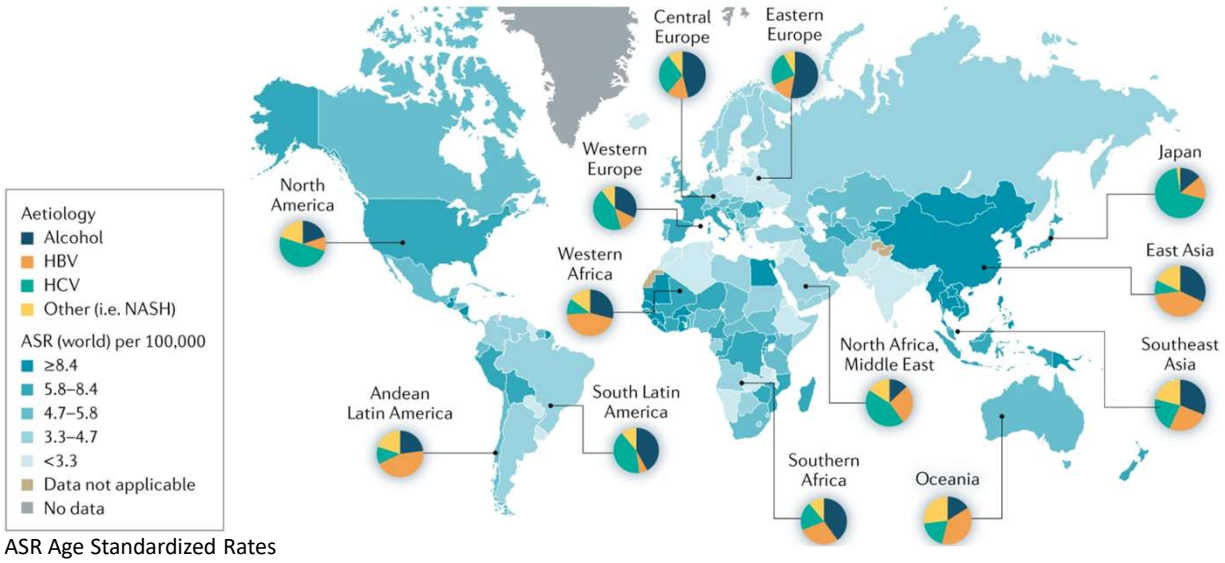
The incidence of HCC according to geographical area and etiology



ASR Age Standardized Rates

Llovet, J.M. *et al.* Hepatocellular carcinoma. *Nat Rev Dis Primers* **7**, 6 (2021).

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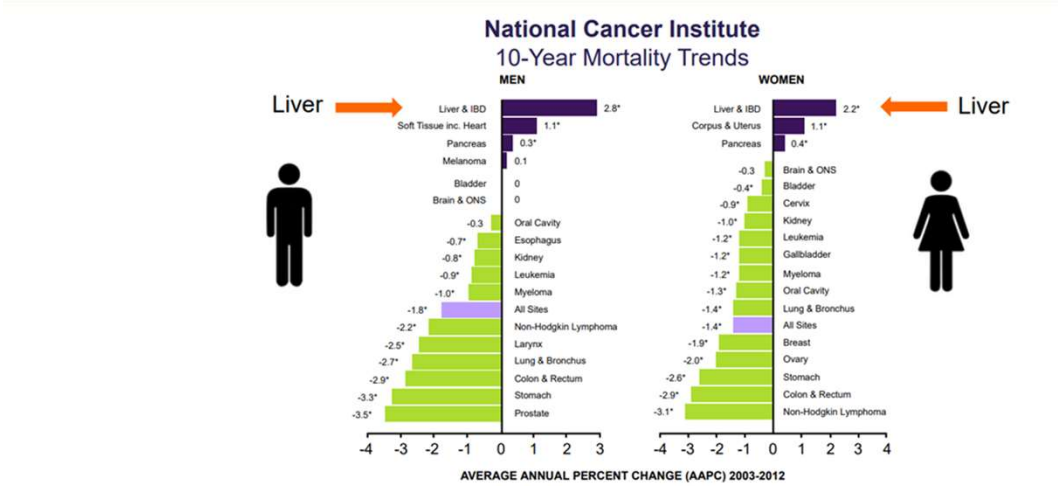
Fastest Rising Cause of Cancer-Related Death in the US



*AAPC is significantly different from zero ($p < .05$).
IBD = intrahepatic bile duct; ONS = other nervous system.
Ryerson et al. 2016.

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Changes in cirrhosis/HCC epidemiology: Next 5-10 years

1. HCV-related cirrhosis/HCC will decline dramatically:
   - Deaths in aging baby boomers with HCV
   - Eradication of HCV with DAAs in unprecedented numbers
2. Patients with cured-HCV and cirrhosis make up a large % of cirrhosis
   - What is their HCC risk and optimal screening?
3. NAFLD-cirrhosis/HCC will continue to increase slowly but steadily
   - Overall risk lower but population attributable fraction high
   - Epidemics of obesity and diabetes and ?other factors
4. Overall cirrhosis prevalence and HCC incidence may decline…transiently
5. Eventually, if NAFLD-cirrhosis predictions are true, overall cirrhosis prevalence and HCC incidence will start increasing again

Llovet, J.M. *et al.* Hepatocellular carcinoma. *Nat Rev Dis Primers* **7**, 6 (2021).

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Prevention Before Surveillance

Vaccinate/Eradicate/Control Viral Hepatitis
Alcohol cessation
Control of Metabolic Syndrome/Insulin Resistance
Healthy lifestyle with exercise

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Risk factors for HCC

RISK FACTORS FOR HCC

- Chronic hepatitis B
- Chronic hepatitis C
- Cirrhosis
  - Alcoholic
  - NASH
  - Autoimm
  - Primary s
  - Primary b
  - Wilson's d
  - Alpha-1 a
  - Hereditar
- Aflatoxins
- Microcystins
- Diabetes
- Smoking

AGE
STRONG MALE PREDOMINANCE
HISPANIC RACE
High incidence of PNPLA3 (NASH CIRRHOSIS)
SMOKING

PREVENTION

- Prevent infection with HBV and HCV
- and active
- Consider chemoprotection as appropriate

Forner A, Llovet JM, Bruix J. Hepatocellular carcinoma. Lancet 2012; 379:1245
Bruix J, Sherman M. AASLD Management of hepatocellular carcinoma: an update. Hepatology 2011; 53:1020

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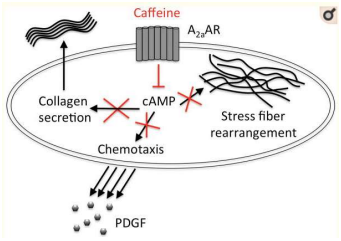
Coffee reduces risk for HCC: an updated meta-analysis.

2 or more cups a day associated with 45% reduction of HCC (0.38-0.53)

Finnish smokers' study:
RR of HCC per cup 0.82 (0.73-0.93)
CLD mortality 0.55 (0.48-0.63)

NHANES:
AST, ALT, AlkPhos & GGT all lower in coffee drinkers



Caffeine is an antagonist of the A_{2a} adenosinergic receptor expressed on activated hepatic stellate cells and other liver myofibroblasts:

- Reduction in liver metalloproteinase (MMP) secretion
- Anti-oxidant and anti-inflammatory

Improved enzymes,
Decreased risk of progression to cirrhosis
Lower mortality, lower rate of HCC
Improved responses to HCV Rx
Lower severity of steatohepatitis

Kennedy OJ et al. BMJ Open. 2017;7(5):e013739
Xiao Q, Sinha R, Graubard BI. NHANES 1999-2010. *Hepatology* Aug 13
Lai GY, Weinstein SJ, Albanes D, et al. *Br J Cancer* 2013 Sep 3;109(5):1344-51
Saab S, Mallam D, Cox GA. Liver Int. 2014 Apr; 34 (4): 495-504
Dranoff JA et al. Hepatology 2014 Aug:60(2): 464-467

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COFFEE EMERGES AS DOMINANT LIFESTYLE FACTOR IN POPULATION ATTRIBUTABLE RISK OF HEPATOCELLULAR CARCINOMA IN AN ETHNICALLY DIVERSE COHORT

Kali Zhou[1], Jennifer Dodge[1,2], Tiffany Lim[2], Norah Terrault[1], and V. Wendy Setiawan[2]

Keck School of Medicine of USC

		Prevalence	Overall (HR, 95% CI)	Prevalence	Lean	Prevalence	Overweight/Obese
	Never	27.1%	1.00	26.6%	1.00	27.2%	1.00
	Former	48.6%	1.64 (1.37, 1.97)	40.2%	2.08 (1.40, 3.09)	51.0%	1.55 (1.26, 1.89)
	Current	24.3%	2.98 (2.41, 3.69)	33.2%	4.46 (2.88, 6.89)	21.8%	2.62 (2.05, 3.36)
	p-value		<0.0001		<0.0001		<0.0001
	None	49.9%	1.00	49.7%	1.00	50.0%	1.00
	Moderate	27.0%	0.80 (0.67, 0.96)	27.2%	0.74 (0.51, 1.07)	26.9%	0.80 (0.65, 0.98)
	Heavy	23.1%	1.24 (1.01, 1.51)	23.1%	1.12 (0.73, 1.72)	23.1%	1.22 (0.98, 1.53)
	p-value		0.1652		0.7266		0.2755
	None	46.2%	1.00	50.3%	1.00	45.0%	1.00
	Low	30.5%	0.92 (0.78, 1.09)	32.5%	1.02 (0.72, 1.44)	30.0%	0.89 (0.73, 1.08)
	High	23.3%	0.87 (0.71, 1.05)	17.2%	0.67 (0.43, 1.04)	25.0%	0.91 (0.74, 1.13)
	p-value		0.1102		0.1021		0.3116
	Q1 (0-2)	21.4%	1.00	18.9%	1.00	22.1%	1.00
	Q2 (3)	18.5%	0.82 (0.65, 1.03)	17.8%	0.90 (0.55, 1.49)	18.7%	0.79 (0.61, 1.02)
	Q3 (4)	21.9%	0.86 (0.69, 1.08)	17.8%	0.81 (0.49, 1.34)	23.1%	0.86 (0.67, 1.10)
	Q4 (5)	21.0%	0.94 (0.75, 1.18)	21.9%	1.09 (0.67, 1.78)	20.7%	0.88 (0.68, 1.13)
	Q5 (6-9)	17.2%	0.69 (0.54, 0.88)	23.6%	0.93 (0.57, 1.52)	15.4%	0.60 (0.46, 0.80)
	p-value		0.0131		0.9176		0.0026
	None	28.0%	1.00	31.4%	1.00	27.1%	1.00
	1	51.7%	0.99 (0.84, 1.18)	54.4%	0.98 (0.80, 1.21)	50.9%	0.98 (0.80, 1.19)
	2-4	16.5%	0.72 (0.58, 0.91)	11.8%	0.70 (0.53, 0.92)	17.8%	0.78 (0.61, 1.00)
	≥ 4	3.8%	0.62 (0.42, 0.93)	2.4%	0.45 (0.26, 0.78)	4.2%	0.72 (0.47, 1.11)
	p-value		0.0006		0.0004		0.0219
	No	66.9%	1.00				
	Yes	33.1%	1.65 (1.41, 1.94)	--	--	--	--
	p-value		<0.0001				

AASLD 2021

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Prevention

METFORMIN

Galega officinalis: Goat's rue, French lilac, Italian fitch, professor weed

Metformin inhibits hepatocyte proliferation, induces cell cycle arrest at G0/G1 AMP-activated protein kinase

- 10 studies, 334K pts
- Metformin: OR HCC 0.50 (0.34-0.73)
- Sulfonylurea 1.62
- Insulin 2.6
- TZDs 0.54 (0.28-1.02).

Each year of metformin use:
7% reduction RR of HCC
in diabetic patients (OR=0.93, 0.91-0.94)

ASA

- NIH-AARP Diet and Health Study:
  RR of developing HCC = 0.59 (0.45-0.77)
- Reduces Fibrosis post transplant

STATINS

- Tsan: Clear dose response with HRs of
- 0.66 (29-90d), 0.41 (90-365d) and 0.34 (>365d)
- Meta-analysis of 10 studies with 1.6 million pts :
- OR of HCC with statins/no statins 0.63 (0.52-0.76)

Sahasrabuddhe VV, Graubard BI, et al. J Natl Cancer Inst 2012; 104: 1808
Poujol–Robert A, Conti F, et al. Clin Res Hepatol Gastroentrol 2014 Aug 14 (Epub)
Tsan YT, Lee CH, Wang JD, et al. J Clin Oncol 2012; 30: 623
Singh S, Singh PP, Singh AG, et al. Gastroenterology 2013; 144: 323

Singh Sm Singh PP, Singh AG, et al. Anti-diabetic medications and the risk of hepatocellular cancer: a systematic review and meta-analysis. *Br J Cancer.* 2013 Sep 3;109(5):1344-51.
Chen HP, Shieh JJ, Chang CC. Metformin decreases hepatocellular carcinoma risk in a dose-dependent manner: population-based and in vitro studies. *Gut.* 2013 Apr;62(4):606-15.
Miyoshi H, Kato K, Iwama H., et al. Effect of the anti-diabetic drug metformin in hepatocellular carcinoma in vitro and in vivo. Int J Oncol. 2013 Dec 30. Epub

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Effects of Metformin Exposure on Survival in a Large National Cohort of Patients With Diabetes and Cirrhosis

Table 3. Impact of Metformin and Other Medications on HCC, Decompensation, and MACE

HCC[a]	At-risk/events	HR	P
Exposure to metformin, time-updating	11,246/856	0.98 (0.81–1.18)	.86

Metformin use is associated independently
with reduced overall,
but not liver-related, mortality, hepatocellular carcinoma, or decompensation
after adjusting for concomitant statin and
ACE inhibitor/angiotensin-2–receptor blocker exposure.

HCC, hepatocellular carcinoma; MACE, major adverse cardiovascular events.
[a]Models were adjusted for age, sex, race/ethnicity, disease etiology, Child-Turcotte-Pugh stage, model for end-stage liver disease-sodium, platelet count, aspartate aminotransferase, alanine aminotransferase, academic affiliation of treatment site, socioeconomic indicator, and baseline history of CAD.
[b]Child-Turcotte-Pugh A patients only.
[c]Analysis includes only patients with no previous MACE events.

Kaplan DE, Taddei T et al. Gastroenterology 2019;156:1693–1706

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Effects of Hypercholesterolemia and Statin Exposure on Survival in a Large National Cohort of Patients With Cirrhosis:

VOCAL STUDY GROUP

		Statin naïve		
	Existing users	Non-initiator	New initiator	P value
Specific baseline comorbidities, %				
Hepatic decompensation	26.5	27.5	16.2	<.0001
Ascites	13.3	12.3	6.8	<.0001
Hepatic encephalopathy	2.7	3.6	1.9	<.0001
TIPSS	0.4	0.9	0.8	<.0001
Acute myocardial infarction	5.7	0.3	1.0	<.0001
Stroke	17.9	3.5	5.7	<.0001
MACEs	25.9	3.9	6.9	<.0001
Coronary artery disease	73.1	6.3	19.3	<.0001
Cardiac arrest	0.4	0.1	0.1	<.0001
Cardiac intervention (PCI or CABG)	3.1	0.1	0.3	<.0001
Congestive heart failure	33.2	6.2	10.7	<.0001
Atrial fibrillation	16.7	3.9	6.1	<.0001
Pulmonary embolus	1.5	0.5	0.6	<.0001
Prior myositis or myopathy	3.5	1.5	1.5	<.0001
Prior creatine phosphokinase >600 U/L	4.4	1.9	1.9	<.0001
Antiviral therapy during follow-up				
HCV DAA, n (% HCV-infected)	3499 (53.7)	10,706 (45.2)	2227 (59.4)	<.0001
HBV DAA, n (% HBV-infected)	221 (32.6)	486 (48.3)	111 (48.1)	<.0001
Event rates, per 100 person-years				
Death	12.7	14.1	7.2	<.0001
HCC	2.0	4.1	1.9	<.0001
Hepatic decompensation	8.5	14.5	7.3	<.0001
Ascites	4.7	7.9	3.6	<.0001
Hepatic encephalopathy	3.5	6.0	3.0	<.0001
TIPSS	0.7	0.9	0.4	<.0001
Acute myocardial infarction	1.1	0.3	1.3	<.0001
Stroke	1.9	1.4	2.8	<.0001
MACEs	3.3	1.8	4.5	<.0001
Coronary artery disease	3.3	1.7	5.6	<.0001
Cardiac arrest	0.3	0.3	0.2	.17
Cardiac intervention (PCI or CABG)	0.2	0.0	0.4	<.0001
Congestive heart failure	4.0	2.5	3.8	<.0001
Atrial fibrillation	2.1	1.3	1.9	<.0001
Pulmonary embolus	0.3	0.2	0.3	<.0001
Myositis or myopathy	0.5	0.5	0.5	.21
Creatine phosphokinase >600 U/L	0.7	0.7	0.8	.31

CABG, coronary artery bypass grafting; DAA, direct-acting antiviral; PCI, percutaneous coronary intervention; TIPSS, transjugular intrahepatic portosystemic shunt.

Comprehensive propensity matching

Kaplan DE, Aytaman A, Taddei T et al. Gastroenterology 2019;156:1693–1706

Exposure to statin therapy was associated with an

8.0%–8.7% annual decrease of mortality.

In well-compensated cirrhosis:

Lower risk of hepatic decompensation

Decreased liver cancer incidence

Patients with advanced cirrhosis (CTP C) did not derive a survival benefit.

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Prevention Key Take Aways

- Control of underlying liver disease
- Statins
- ASA
- Vitamin D
- Coffee
- Exercise
- Healthy diet
- Smoking cessation
- ? ACE/ARB
- ? Metformin (overall survival benefit)

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Screening

AFP+ SONO remains the current standard

AASLD, EASL, APASL, USPSTF

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HCC Surveillance

- All cirrhotics regardless of etiology
- HCV cirrhosis or HCV stage 3 fibrosis prior to HCV eradication
- HBV sAg positive patients from endemic areas:
  - Men over 40
  - Women over 50
  - Family history of HCC
  - Sub Saharan origin: starting in early 20ies

AASLD HCC Guidance 2018

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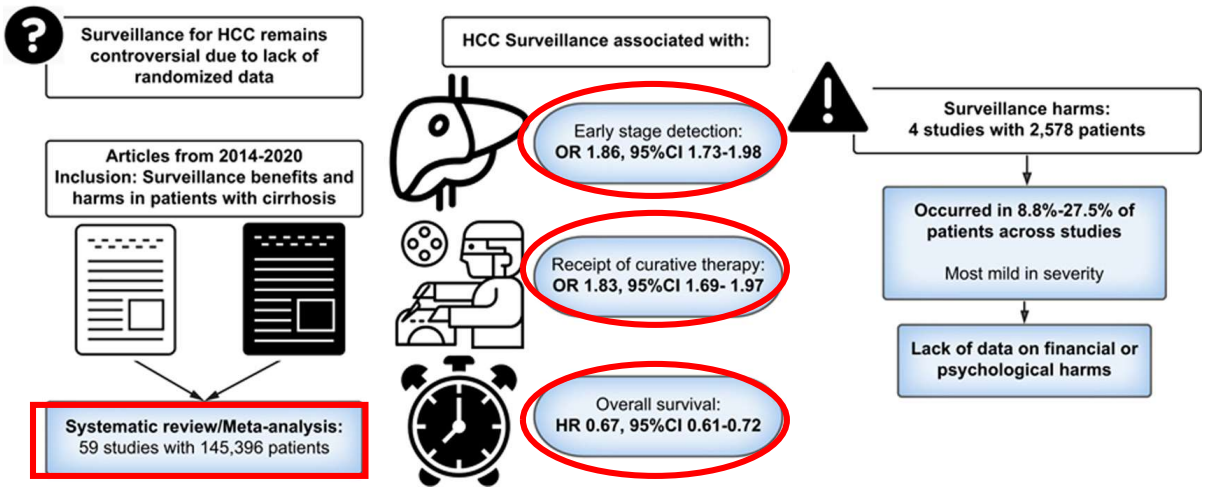
Liver cancer surveillance saves lives



Singal AG, et al. *J Hepatol*. 2022

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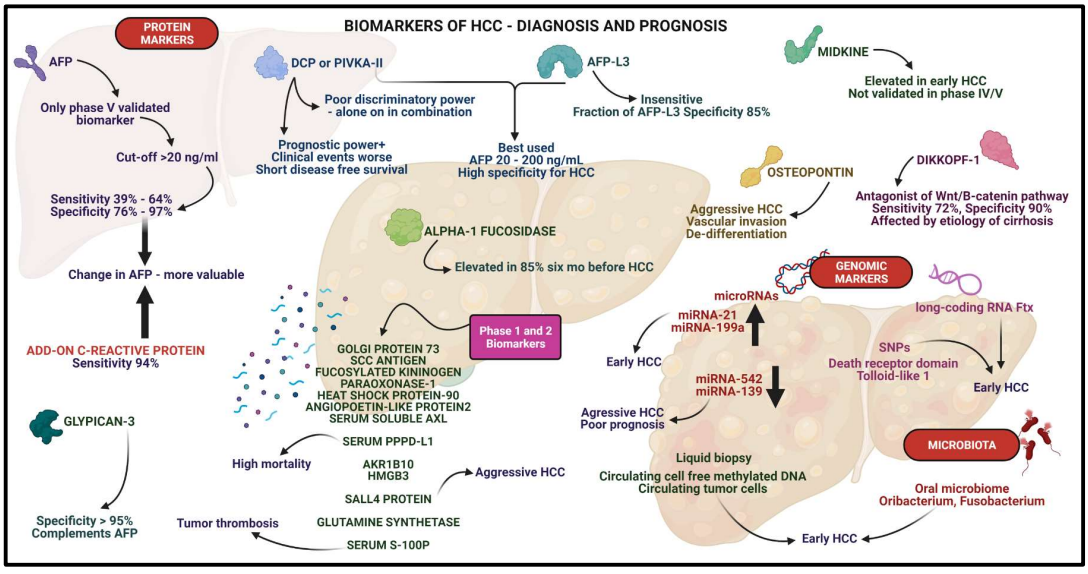
Protein-, genome- and microbiota biomarkers currently in use or potential future use in the surveillance and diagnosis of HCC



Philips C A et al. (November 05, 2021) Hepatocellular Carcinoma in 2021: An Exhaustive Update. Cureus 13(11): e19274.

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GALAD SCORE IMPROVES EARLY DETECTION OF HCC PRIOR TO THE DIAGNOSIS OF HCC: A PHASE 3 BIOMARKER VALIDATION STUDY

- *Objectives*:
  - To compare the performance of AFP and the GALAD score for the detection of HCC
    - **GALAD** = Gender, Age, AFP-L3, AFP, and Des-carboxy-prothrombin

Performance of AFP and GALAD at the time of HCC Diagnosis

	DCP	AFP-L3	AFP	GALAD

GALAD SCORE had the best performance characteristics for early detection of HCC in patients with cirrhosis Compared to AFP, AFP-L3, DCP alone

GALAD could prevent unnecessary MRI/CT in 54% of patients with cirrhosis.

82% of patients detected at very early or early stages of disease.

WE have data on Viral and non-viral HCC, FDA approved, might have a role in NASH surveillance where obesity decreases the efficacy of imaging

The Liver Meeting

Marrero JA, et al. #138

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HCC risk calculated by our web-based models for selected patients in "low-risk", "medium-risk" and "high-risk" categories

	LOW-RISK PATIENTS 5-year HCC risk <5% Annual HCC risk <1%				MEDIUM-RISK PATIENTS 5-year HCC risk 5% to 15% Annual HCC risk 1 to 3%				HIGH-RISK PATIENTS 5-year HCC risk >15% Annual HCC risk >3%			
Cirrhosis etiology	NAFLD	ALD	NAFLD	ALD	NAFLD	ALD	NAFLD	ALD	NAFLD	ALD	NAFLD	ALD
Age	61	67	62	62	66	60	62	66	63	62	63	62
Sex	F	M	M	M	F	M	M	M	M	M	M	M
Diabetes	No	No	Yes	No	Yes	Yes	No	Yes	Yes	Yes	Yes	Yes
BMI	31	28	31	28	35	23	35	24	37	29	37	29
Albumin	4.2	4.2	3.8	3.9	3.6	3.9	3.5	3.1	3.8	3.4	3.4	3.3
Serum AST	30	30	30	30	45	50	55	40	55	50	55	50
Serum ALT	35	30	35	30	20	20	20	20	25	20	25	20
Platelet Count	225	170	170	120	85	110	115	180	90	110	85	50
Estimated 5-year HCC Risk	0.40%	2.6%	3.5%	4.6%	6.41%	10.0%	12.8%	14.4%	15.5%	20.6%	21.3%	30.2%

Models available at: www.hccrisk.com
Ioannou J Hepatol. 2018 Nov;69(5):1088-1098

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At Risk

The magnitude of HCC risk varies depending on the underlying etiology:
Being highest in patients with active HCV cirrhosis,
Lower in cured HCV
Lowest in those with NAFLD cirrhosis.

Alcohol drinking and overweight were associated with an increased HCC risk irrespective of underlying etiology.

Ultrasound every six months ***with*** alpha-fetoprotein

Population Group	Incidence of HCC
Sufficient Risk to Warrant Surveillance	
Child-Pugh A-B cirrhosis, any etiology	
- Person from Africa at earlier age**	
- Family history of HCC	
- PAGE-B score ≥10#	
Insufficient Risk and in need of Risk Stratification Models/Biomarkers	
Hepatitis C and stage 3 fibrosis	< 0.2% per year
Noncirrhotic NAFLD	

Singal et al. 2022 AASLD HCC Guidance (in press)

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New Challenges for HCC Screening

Patients with cirrhosis and cured HCV:

- Risk is reduced after SVR, but residual risk persists
- How does it change over time after SVR?
- Cost effectiveness of screening?

Patients with NAFLD-cirrhosis:

- Nearly one-fourth of NAFLD-related HCC occurs in the absence of cirrhosis
- Annual HCC incidence of 0.008 per 100 person-years
  - Very large population of NAFLD-cirrhosis predicted in next 5-10 years
    Have lower HCC risk than HCV-cirrhosis → lower cost-effectiveness
    Older, more comorbidities → reduced treatment options
    Obesity: reduces the accuracy of ultrasound screening
- **Risk stratification tools to identify those at highest risk**
- **Surveillance on a case-by-case basis**
- **Developing better screening tests is critical**

Farhang Zangneh H et al. CGH 2019
Orci LA et al. CGH 2022

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Diagnosis and Staging Challenges

Severity of the underlying liver disease influences staging and management options.

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Liver Imaging Reporting and Data System (LI-RADS)

HCC is the only malignancy which can be diagnosed and treated by imaging only.

LI-RADs were developed by American College of Radiology 2011

LI-RADS: assign a relative probability for HCC

creating a standardized language

Now including CT, MRI with extracellular and hepatobiliary agents, ultrasound, contrast enhanced ultrasound, expansion under way for evaluation of benign liver lesions.

https://www.ajronline.org/doi/full/10.2214/AJR.20.24272

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When not to use LI-RADs

1) No risk factors for liver disease
2) Less than 18 years of age
3) Patients with cirrhosis due to congenital hepatic fibrosis
4) Cirrhosis due to vascular disorder (hereditary hemorrhagic telangiectasia, Budd-Chiari syndrome, chronic portal vein occlusion, cardiac congestion, or diffuse nodular regenerative hyperplasia)

LI-RADS should not be applied to path proven malignancies or path proven benign lesions

Patients with
chronic hepatitis C virus infection or
nonalcoholic steatohepatitis with advanced fibrosis
creates an important knowledge gap warranting further research

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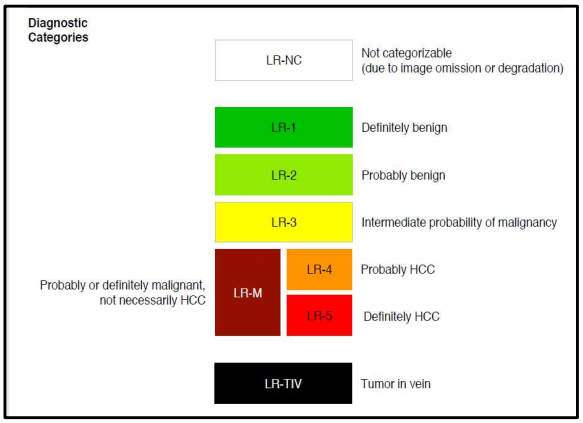
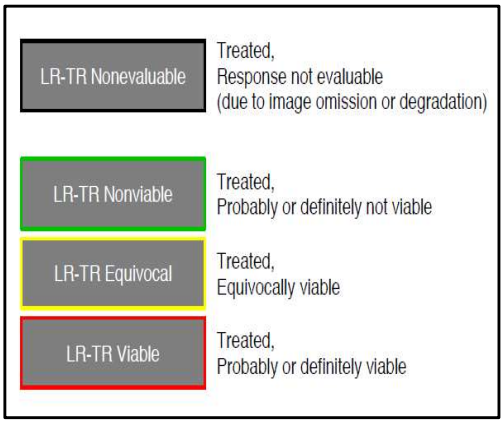
LI-RAD Diagnostic Categories





www.acr.org/-/media/ACR/Files/RADS/LIRADS/LI-RADS-2018-Core.pdf

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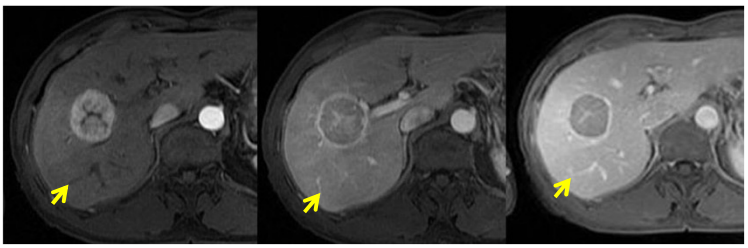
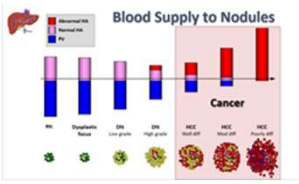
Criteria for HCC Diagnosis

- Dynamic imaging required for diagnosis



Arterial Phase Portal venous phase Equilibrium phase



Early arterial enhancement
Portal venous , delayed washout
Capsule formation

CT/MRI Diagnostic Table

Arterial phase hyperenhancement (APHE)		No APHE		APHE (not rim)		
Observation size (mm)		< 20	≥ 20	< 10	10-19	≥ 20
Count major features: • "Washout" (not peripheral) • Enhancing "capsule" • Threshold growth	None	LR-3	LR-3	LR-3	LR-3	LR-4
	One	LR-3	LR-4	LR-4	LR-4 / LR-5	LR-5
	≥ Two	LR-4	LR-4	LR-4	LR-5	LR-5

LR-4 / LR-5 Observations in this cell are categorized LR-4, except:
- LR-5g, if ≥ 50% diameter increase in < 6 months (equivalent to OPTN 5A-g)
- LR-5us, if "washout" and visibility at screening ultrasound (per AASLD HCC criteria)

https://sites.google.com/site/rlow53/HCC2.jpg
https://www.acr.org/Clinical-Resources/Reporting-and-Data-Systems/LI-RADS

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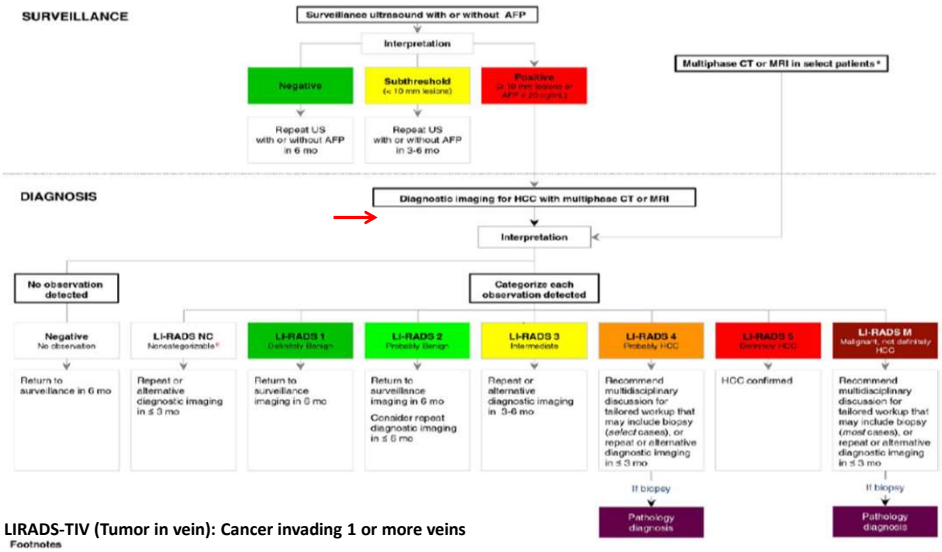
AASLD/LIRAD SURVEILLANCE AND DIAGNOSIS ALGORYTHM 2018



LIRADS-TIV (Tumor in vein): Cancer invading 1 or more veins
Footnotes

Marrero et al. Hepatology, Vol. 68, No. 2, 2018

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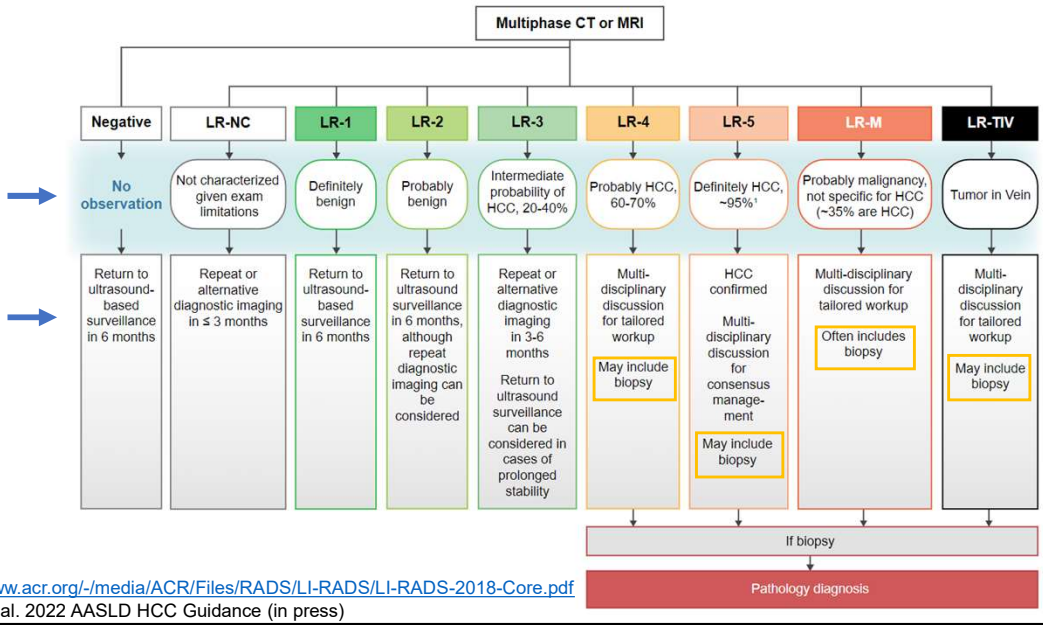
A More Liberal Approach to Biopsy



https://www.acr.org/-/media/ACR/Files/RADS/LI-RADS/LI-RADS-2018-Core.pdf
Singal et al. 2022 AASLD HCC Guidance (in press)

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Factors Determining Prognosis

- **Tumor stage**
  - - Size
  - - Number
  - - Location
  - - Vascular invasion
  - - Extrahepatic spread
- **Anatomy** is key to determining feasibility of resection
- **Liver function**
  - Principal driver of treatment decisions
  - Presence or absence of portal hypertension
- Overall patient **health/performance**
  - ECOG Performance score/Karnofsky score

Marrero JA et al. Hepatology, VOL. 68, NO. 2, 2018

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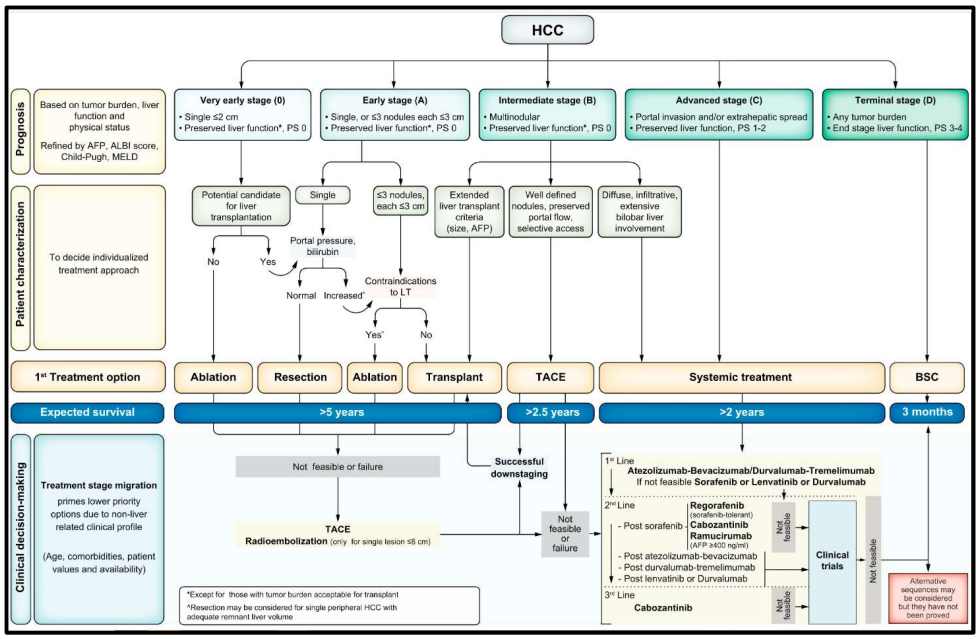
BCLC strategy for prognosis prediction and treatment recommendation:

The 2022 update



Reig M et al J of Hepatol Volume 76, Issue 3, March 2022, Pages 681-693

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Treatment basics

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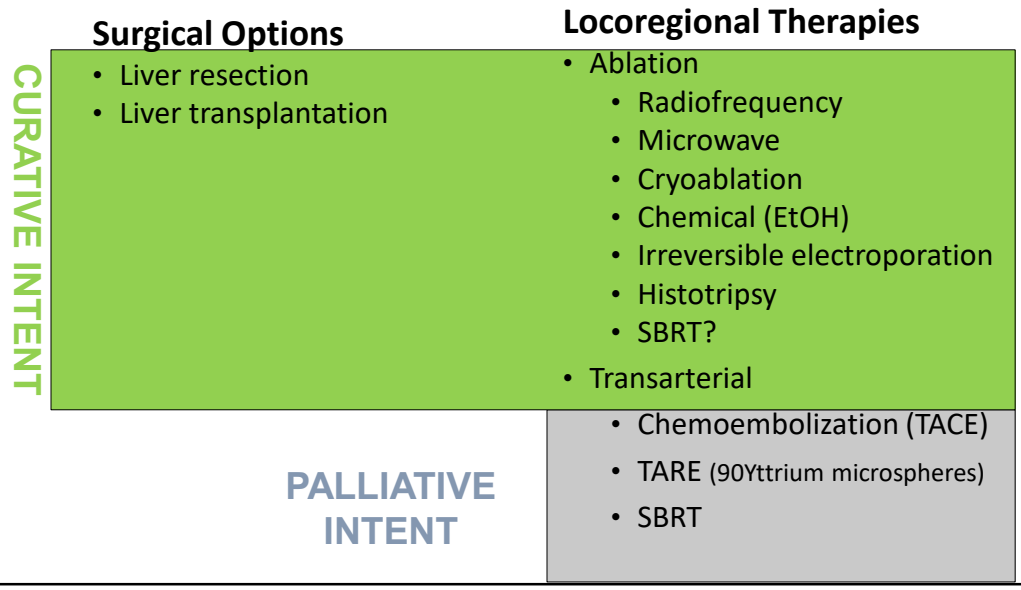
The treatment landscape (BCLC 0, A, B)



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AASLD HCC GUIDANCE 2018

- **Resection is the treatment of choice**

  For localized HCC occurring in the absence of cirrhosis or

  Resectable HCC and in the setting of cirrhosis with intact liver function and absence of CSPH.

- **Transplantation is the treatment of choice** for patients with early-stage HCC occurring in the setting of **CSPH and/or decompensated** cirrhosis.

  Access is limited by the extreme organ shortage.

  With many curative options transplant in well compensated cirrhotic should be reserved for recurrence and/or decompensation.

CSPH Clinically Significant Portal Hypertension

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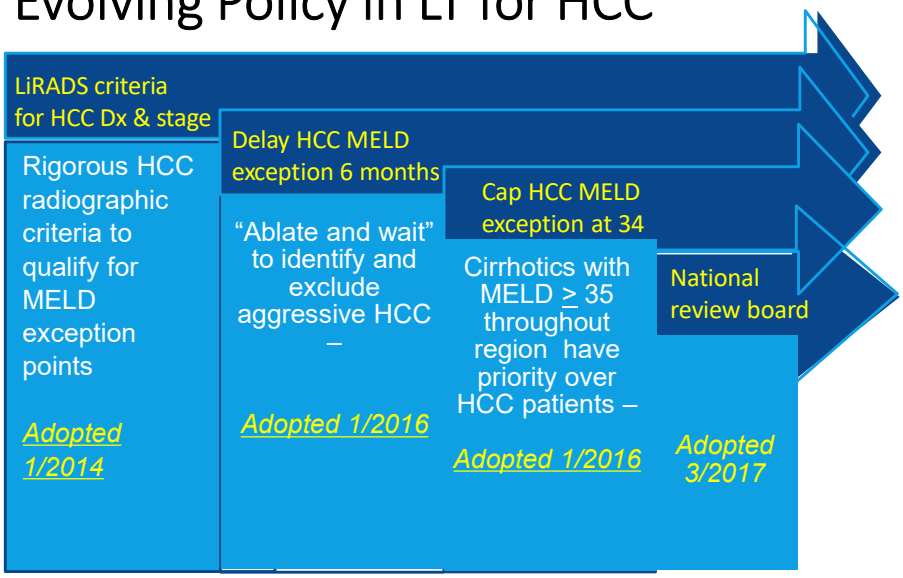
Evolving Policy in LT for HCC



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New Rules for MELD Exception Effective 1/21/2016

- *No MELD exception points are granted for 6 months from time of initial application for MELD exception*
- Must be within Milan criteria (1 lesion <5cm or 3 lesions or less each less then 3cm) at time of initial application
- Repeat imaging every 3 months *must demonstrate continued locoregional control*
- Exception score awarded:

TIME:	Initial	Points:	none (native MELD)
	3 months		none "
	6 months		28
	9 months		31
	12+ months		34 (maximum)

Share 35 does not apply to HCC's

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Final changes: 12/2017

- Candidates **within 150 mile circle** of the donor hospital

TRANSPLANT IS BECOMING A LESS VIABLE OPTION ESPECIALLY FOR PATIENTS WITH ADEQUATE LIVER FUNCTION

- For other MELD 15-31, organs will continue to be allocated within the donor service area according to MELD score
- Extended criteria livers from donation after cardiac death will continue to be allocated locally

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Variables to Consider in Decision Resection vs Ablation for Early HCC

- **Liver reserve**: CPT score, MELD score
  - CSPH (Clinically significant pHTN)
  - Plts (>100 or not)
  - Albumin >3.5
  - INR<1.5
  - Bilirubin
  - Ascites
- **Loc**
- **Number** of the lesions and lobes/segments involved
- Absence of metastasis and main PV and IVC **tumor thrombus**
- Absence/control of cardiopulmonary **comorbidities**
- **Available expertise** in each facility

Liver volume measurement
Right/left/total remnant volume
≥ 30% in non cirrhotic
≥ 50% in cirrhotic

Candidates for surgery:
5%-15% of HCC in Western countries but a higher percentage in Asia (HBV)

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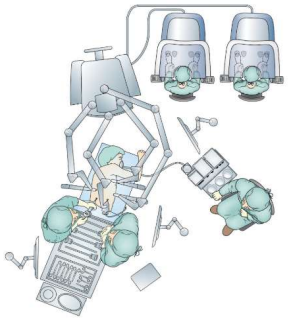
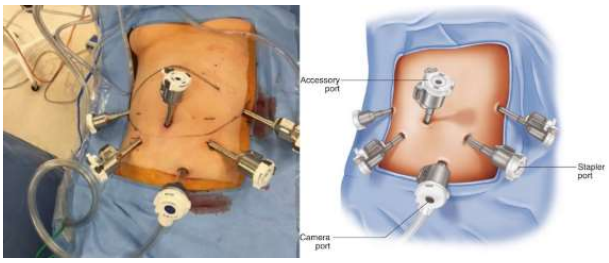
Liver Resection is Getting Less Invasive: Every Day We Are Pushing the Envelope



DaVinci Robotic Partial hepatectomy

New minimally invasive technics are now pushing the envelope



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Locoregional Therapy

Define upfront your goal with locoregional therapy:

1) Curative: Limited to very early tumors treated with RFA/MWA/?SBRT
2) **Bridge to liver transplantation (OLT)**
3) **Downstage to OLT/Resection**
4) **Palliative:** With goal to improve overall survival without the ability to cure

Talk upfront about systemic therapy: they're eventually going to be going down the road to systemic therapy if they can preserve their liver function.

The decision which liver-directed therapy to start with is dependent on

Patient and tumor characteristics

Heavily dependent upon the experience of the center

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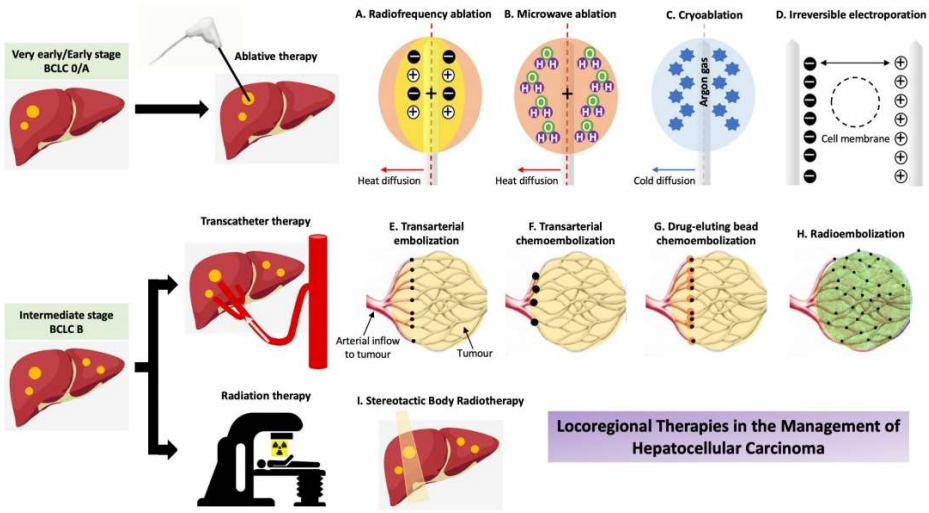
Locoregional Therapies for HCC



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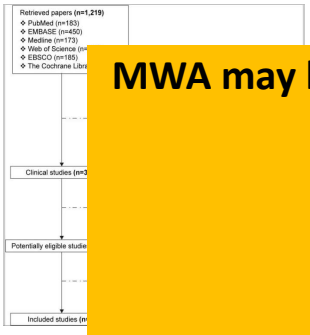
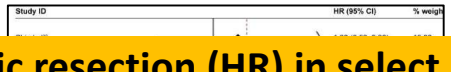
Comparison of microwave ablation and hepatic resection for HCC: A meta-analysis



9 Studies



MWA may be superior to hepatic resection (HR) in select cases:

As Effective as HR in terms of
Overall survival (OS),
Disease Free Survival (DFS),
Recurrence Rates,
Shorter Operation Time,
Less amount of blood loss in operation,
Fewer Complications

nd HRN

nd HRN
wave

Zhang M et al Onco Targets Ther 2017; 10: 4829–4839.

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Technology is Improving Considerably

Novel sono transducer
for ablation probe guidance

New-generation MWA may create a **more predictable ablation zone** and a **larger ablation volume in a shorter time period**.

Prof. Shuichiro Shiina Juntendo Univ Tokyo

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SBRT for HCC

- SBRT is an emerging treatment modality offering potentially curative local therapy for HCC.
- SBRT is applicable **across BCLC stages** (bridge to transplant, BCLC A,BCLC B, portal vein thrombosis) as an alternative treatment strategy to TACE/RFA, or in recurrent tumors as salvage therapy.
- The recent prospective and retrospective studies have shown the safety and efficacy of SBRT with 2-year local control ranging from 68-95%.
- Smaller randomized trials of external beam radiation therapy suggest high efficacy of radiation therapy compared to other treatments for patients with unresectable HCC, and phase III trials comparing SBRT with other modalities are ongoing.

Lewis S, Dawson L et al Review JHEP Reports 2022 vol. 4 j 100498

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SBRT for Small HCC
Photons versus Protons

Protons should be considered for dome and central tumors > 3 cm – maximal liver sparing,

potentially reduced radiation toxicity

Protons should also be considered for any tumor > 5 cm

if other ablative options fail to achieve adequate coverage

Gandhi, Pract Radiation Oncol, 2015

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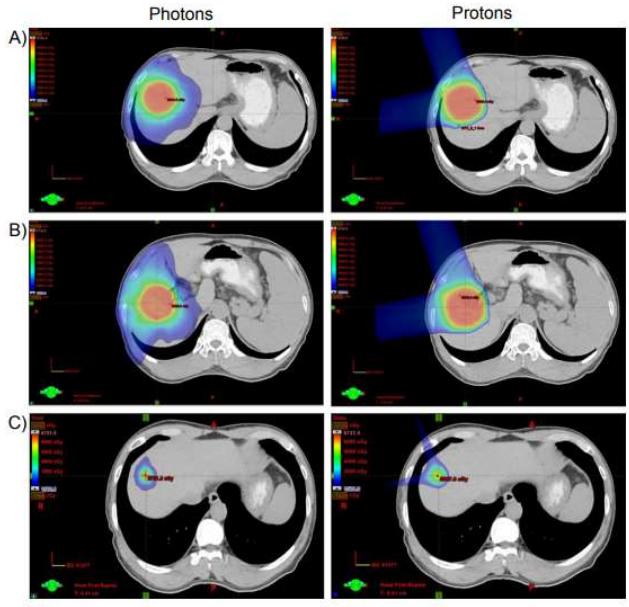
Location: Dome

Location: Central

Small Tumor:
No Difference



Gandhi, Pract Radiation Oncol, 2015

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SBRT vs RFA ?

Univ Michigan 2004 to 2012,

224 patients with inoperable, nonmetastatic HCC

RFA (n = 161) to 249 tumors or image-guided SBRT (n = 63) to 83 tumors

Expected overall survival of SBRT is nearly identical to RFA in HCCs smaller than 3 cm

SBRT may have an advantage for tumors 2 cm and larger

Wahl DR et al, Journal of Clinical Oncology 2016 Feb 10;34(5):452-9.

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TACE (Transarterial Chemoembolization) vs DEB (Drug eluding bead)-TACE

cTACE: Administration of an anticancer agent emulsified in ethiodized oil, followed by embolization of the tumor-feeding artery with gelatin sponge particles.

TACE with drug-eluting beads (DEB-TACE): Administration of spherical drug-eluting microspheres loaded with an anticancer agent.

Compared with conventional TACE,
drug-eluting bead TACE is a drug delivery embolization technique that permits fixed dosing
with the ability to provide sustained release of anticancer agents over a period of time.
Better tolerated but survival benefit in question…

C-TACE better 1-month and 3-month survival

Ikeda M et alJIVROSG-1302 PRESIDENT study Liver Cancer 2022;11:440–450

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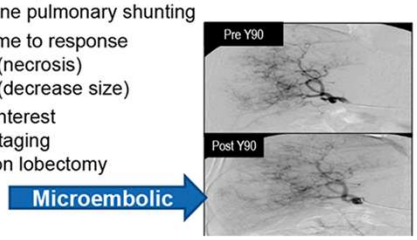
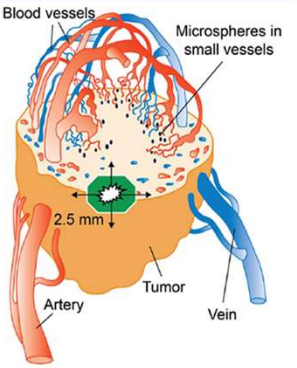
Radioembolization: Yttrium-90 microspheres

- Microspheres, 25-30 microns: preferentially trapped in the tumor capillary bed
- Staging angiogram performed 7-10 d prior to Y90
  - Identify variant anatomy and determine optimal catheter position
  - Determine pulmonary shunting
- Median time to response
  - 1.2 mo (necrosis)
  - 6.6 mo (decrease size)
- Areas of interest
  - Down-staging
  - Radiation lobectomy
  - **PVT**





Due to the fact that it is, **microembolic, seen on this angiogram pre- and post-, the hepatic arteries maintain** flow allowing for it to be **safely administered in patients with portal vein thrombosis.**
Emerging good data peritransplant impacting outcomes

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Potential Complications Y90

Complication	Prevention
Delivery of radiation to nontargets (eg, gastric ulcers, cholecystitis)	• Prophylactic coil embolization • Proper catheter placement • Avoid reflux of microspheres • ? Proton pump inhibitors
Post-radioembolization syndrome	No proven therapy
Hepatic dysfunction (radiation-induced liver disease)	• Patient selection • Selective or super-selective infusion • Avoid whole liver treatment
Biliary AE: necrosis, abscess, stricture	Avoid if compromised sphincter of Oddi
Radiation pneumonitis	Activity adjustment to decrease lung dose • Contraindicated if >30 Gy with 1 treatment OR >50 Gy cumulative

Riaz A et al J Vasc Interv Radiol 2009;20:1121-1130

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Systemic Treatments for HCC: From Dirt to Plenty

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Systemic Treatments for HCC

- **TYROSIN KINASE INHIBITORS :**
  Tumor growth suppression mediated by targeting serine/threonine kinases that are components of the Raf/MEK/ERK pathway (e.g., C-Raf, wild-type B-Raf, and mutant V600E B-Raf), a common downstream pathway of signals transduced via VEGFR, PDGFR, and EGFR;
  Angiogenesis suppression by targeting tyrosine kinases (e.g., VEGFR1, VEGFR2, VEGFR3, PDGFR-α/β, RET, and Fms-related tyrosine kinase 3
  - First Line: **Sorafenib, Lenvatinib**
  - Second Line: **Regorafenib, Cabozantinib**

- **IMMUNE CHECKPOINT INHIBITORS:**
  - ANTI-PD-1 antibodies: Nivolumab and Pembrolizumab,
  - Anti-PD-L1 antibodies: Avelumab, **Durvalumab,** and **Atezolizumab**
  - AntiCTLA-4 antibodies: Ipilimumab and **Tremelimumab**

- **MONOCLONAL ANTIBODIES:**
  - Anti VEGFR2: Ramucirumab
  - Anti VEGF-A: **Bevacizumab**

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Systemic Therapy

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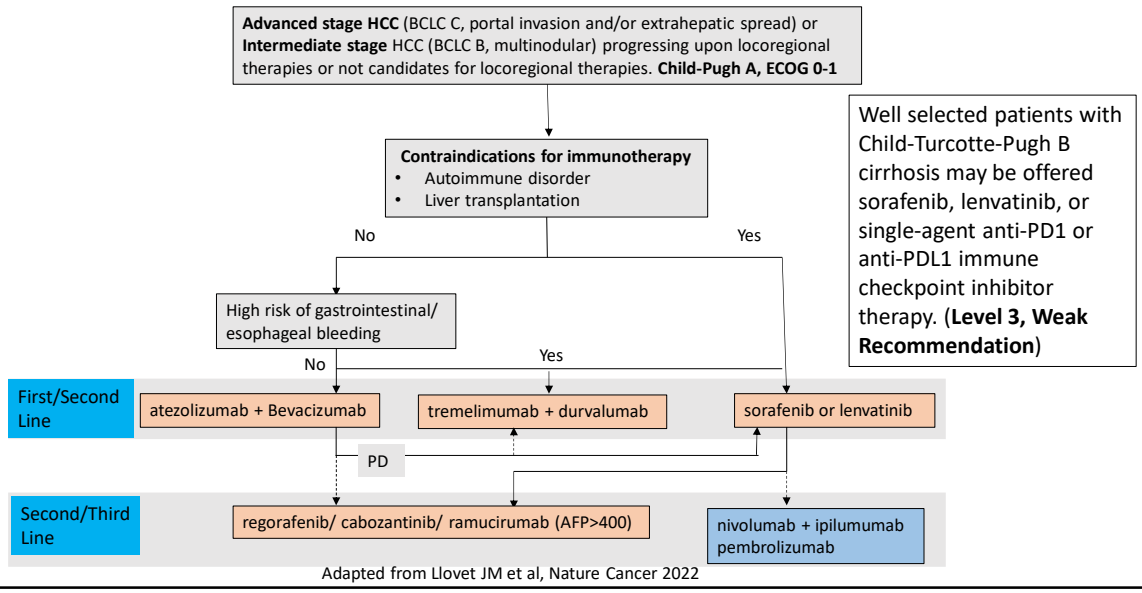
Systemic Therapy: Current Paradigm



Adapted from Llovet JM et al, Nature Cancer 2022

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Curative Treatments

Resection	Ablation	Transplant
▪ Noncirrhotics – Choice of therapy ▪ Cirrhotics – Reserved for CTP A – Avoid R hepatectomy ▪ Best for solitary HCC ▪ < 30% eligible	▪ Effective when < 3 cm ▪ Multiple modalities – Thermal – Chemical – *Stereotactic radiation* ▪ Minimally invasive	▪ Cures both ▪ MELD exception – Milan criteria – Downsizing ▪ Demand > supply
▪ **Survival** – 5 yrs: 70%	▪ **Survival** – 5 yrs: 40% to 50%	▪ **Survival** – 5 yrs: > 70%
▪ **Recurrence** – 5 yrs: 70%	▪ **Recurrence** – 5 yrs: 70%	▪ **Recurrence** – 5 yrs: 15%

Belghiti J, et al. HPB (Oxford). 2005;7:42-49. Bruix J, et al. Hepatology. 2011;53:1020-1022.
Feng Q, et al. J Cancer Res Clin Oncol. 2015;141:1-9. Sapisochin G, et al. at Rev Gastroenterol Hepatol. 2017;14:203-217.
Thuluvath PJ, et al. Liver Transpl. 2009;15:754-762.

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Biologically Aggressive HCC

- Features
  - Microvascular invasion
  - Satellite nodules
  - Diffuse infiltrating growth
  - Poorly differentiated
  - Mixed cholangiocarcinoma
  - Bad molecular signature
  - **FDG-PET scan positive**
  - High AFP and AFP-L3%
  - Rapid growth
- Associated with
  - Early metastasis
  - High risk of recurrence after resection or liver transplantation
  - Failure of local control with RFA/TACE
  - Poor prognosis

There is no consensus on how to incorporate biology into tumor staging

Kwee TC, et al. Eur J Nucl Med Mol Imaging. 2011;38:1158-1170. Trevisani F, et al. Carcinogenesis. 2008;29:1299-1305.
Saboo SS, et al. Cancer Imaging. 2011;11:37-41. Song BC, et al. J Clin Gastroenterol. 2002;35:398-402.

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Holistic Care of the Liver Cancer Patient

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Cachexia is Prevalent in Patients Following Hepatocellular Carcinoma Diagnosis and is Associated with Worse Survival

Nicole E. Rich, Samuel Phen, Nirali Desai, Sukul Mittal, George Cholankeril, Adam C. Yopp, Jorge A. Marrero, Puneeth Iyengar, Rodney E. Infante, Amit G. Singal

Division of Digestive and Liver Diseases, Department of Surgery, Department of Radiation Oncology and the Center for Human Nutrition, UT Southwestern Medical Center, Dallas TX

Baylor College of Medicine, Houston TX

BACKGROUND

- Cancer cachexia (CCX) is a clinical syndrome of wasting resulting in progressive functional impairment and poorer survival
- The impact of CCX has been described for other GI malignancies
- Data are limited on its prevalence and significance in HCC

AIMS

- Characterize the prevalence and outcomes of post-treatment CCX in HCC

METHODS

- We conducted a retrospective study of consecutive patients diagnosed with HCC between 2008 – 2018 at two U.S. health systems (UT Southwestern Medical Center and Parkland Hospital).
- All cases were adjudicated to ensure they met diagnostic criteria for HCC per AASLD guidelines.
  - All tumors staged according to the BCLC staging system
- Weights were manually recorded from the EMR at two time points:
  1) Time of HCC diagnosis
  2) 6 months post HCC diagnosis (+/- 1 month)
- **Cachexia was defined according to international consensus guidelines: >5% weight loss or >2% weight loss if BMI <20 kg/m²**
- **Exclusion criteria:** patients 1) without characteristic imaging (arterial enhancement with delayed washout) or histology confirming HCC diagnosis; 2) received HCC treatment at an outside facility prior to initial presentation at one of the study sites.
- ***Primary outcome*:** Overall survival (OS)
- ***Secondary outcomes:*** Objective response rate (ORR) to first HCC treatment
  - Treatment response assessed based on contrast-enhanced CT or MRI (typically 4-6 weeks post-treatment) and classified per mRECIST criteria.
- We estimated median overall survival (OS) from date of HCC diagnosis to last known date of follow-up, transplantation, or end of study period using Kaplan-Meier analysis; we compared survival between groups using log-rank test.
- We used Cox proportional hazard models and multivariable logistic regression models to identify factors associated with overall survival and response to HCC treatment.

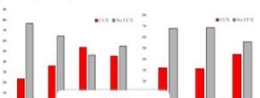
@Nicole_rich8

RESULTS

Table 1: Patient and tumor characteristics at HCC diagnosis, stratified by presence of post-treatment cachexia (n=928)

	CCX (n=304)	Non-CCX (n=624)	p value
Age, mean (SD)	61.1 (8.5)	60.7 (9.4)	
Male sex (%)	225 (74.0)	461 (73.9)	0.97
Race, ethnicity			0.009
White	101 (33.2)	223 (35.7)	
Black	111 (36.5)	175 (28.0)	
Hispanic	69 (22.7)	188 (30.1)	
Asian	21 (6.9)	27 (4.3)	
BMI category			0.27
<18.5	7 (2.3)	19 (3.1)	
18.5 – 24.9	81 (26.6)	198 (31.8)	
25 – 29.9	109 (35.9)	219 (35.2)	
>30.0	107 (35.2)	187 (30.0)	
Liver disease etiology			0.01
Hepatitis C	195 (64.1)	387 (62.0)	
NAFLD	31 (10.2)	95 (15.2)	
Alcohol-related	38 (12.5)	92 (14.7)	
Hepatitis B	29 (9.5)	30 (4.8)	
Child Pugh			0.12
A	179 (59.1)	377 (60.7)	
B	96 (31.7)	209 (33.7)	
C	28 (9.2)	35 (5.6)	
AFP			<0.001
<20 ng/mL	130 (42.9)	320 (51.3)	
20-200 ng/mL	74 (24.4)	174 (27.9)	
>200 ng/mL	99 (32.7)	130 (20.8)	
BCLC stage, n (%)			<0.001
0/A	120 (39.6)	390 (62.9)	
B	67 (21.1)	120 (19.4)	
C	87 (28.7)	75 (12.1)	
D	29 (9.6)	35 (5.7)	
Treatment, n (%)			<0.001
Surgical	50 (16.5)	105 (16.8)	
Ablation	25 (8.2)	90 (14.4)	
TACE/TARE/SIRT	113 (37.2)	309 (49.5)	
Systemic	74 (24.3)	54 (8.7)	
None/BSC	42 (13.8)	66 (10.6)	

Figure 1. Prevalence of post-treatment cachexia by a) BCLC stage and b) Child Pugh class



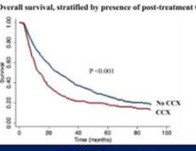
- **Patients w… …agnosis had worse median overall survival compared to those without cachexia (13.1 vs 26.5 months; p<0.001)**

Table 2: Correlates of overall survival (n=928)

	Univariate HR (95% CI)	Multivariable HR (95% CI)
Female sex	0.92 (0.77 – 1.09)	0.91 (0.76 – 1.09)
Age	0.99 (0.98 – 1.01)	1.00 (0.99 – 1.01)
Race, ethnicity		
White	Ref	Ref
Black	1.25 (1.04 – 1.50)	1.10 (0.90 – 1.35)
Hispanic	1.10 (0.91 – 1.33)	0.98 (0.80 – 1.21)
Liver disease etiology		
Viral	Ref	Ref
Non-viral	0.90 (0.76 – 1.06)	0.97 (0.80 – 1.17)
AFP (ng/mL)		
<20	Ref	Ref
20-200	1.31 (1.09 – 1.57)	1.23 (1.01 – 1.50)
>200	2.56 (2.14 – 3.06)	1.95 (1.59 – 2.39)
Child Pugh		
A	Ref	Ref
B/C	1.55 (1.32 – 1.82)	1.79 (1.50 – 2.12)
BCLC stage		
0/A	Ref	Ref
B	2.38 (1.96 – 2.89)	2.08 (1.68 – 2.57)
C	5.26 (4.31 – 6.42)	2.68 (2.03 – 3.53)
No cachexia	Ref	Ref
Cachexia	1.54 (1.32 – 1.78)	1.25 (1.05 – 1.48)

- On multivariable analysis, **cachexia at 6 months post HCC diagnosis was independently associated with lower odds of objective response per mRECIST (OR 0.52, 95% CI 0.28 – 0.97)** after adjusting for age, sex, race, cirrhosis etiology, Child Pugh class, tumor burden, AFP and HCC treatment modality

Figure 2. Overall survival, stratified by presence of post-treatment CCX



CONCLUSIONS

- Nearly 1 in 3 patients with HCC have cachexia post diagnosis, including many with early-stage tumors, compensated cirrhosis and good performance status
- Cachexia was an independent predictor of worse survival; multimodal interventions may improve outcomes

- **Nearly 1 in 3 patients with HCC have cachexia post diagnosis, including many with early-stage tumors, compensated cirrhosis and good performance status**
- **Cachexia was an independent predictor of worse survival; multimodal interventions may improve outcomes**

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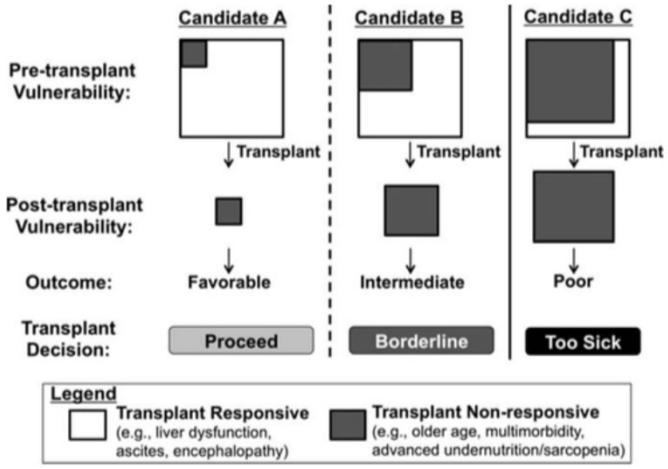
Frailty Measurement Tools



Liver Frailty Index:
Consists of 3 performance-based measures:
- **Grip strength,**
- **Chair stands, and**
- **Balance testing**

Improves the ability of MELDNa to predict mortality in cirrhotic patients awaiting liver transplantation.

The **Liver Frailty Index calculator** is available at: http://www.liverfrailtyindex.ucsf.edu/

Lai JC, et al. Hepatology. April 2017:1-35.

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Nutrition Pearls

Energy needs are high

Body mass index	Estimated daily recommended caloric intake per kg body weight
< 30 kg/m²	≥ 35 kcal
30-40 kg/m²	25-35 kcal
≥ 40 kg/m²	20-25 kcal

- Consult with a registered dietician to measure your resting energy expenditure
- Calculate your estimated resting energy expenditure using an on-line calculator (www.wellnesstoolbox.ca)

Eat enough : Strategies

Barriers	Strategies
"I get full fast"	• Eat often (every 3-4h) • High density foods (e.g., nuts, chia)
"Food tastes different"	• Food variety – buy one new food at the store every time
"Low salt diet makes food taste bland"	• Add flavor – garlic, lemon, chili flakes, turmeric • Relax salt restriction

At the right time : Late evening snack

Metabolic profile of a *patient with cirrhosis* after an **overnight fast** = Metabolic profile of a *healthy person* after **3 days starvation**

Lai JC et al. AASLD Practice Guidance on Malnutrition, Frailty&Sarcopenia in cirrhosis. Hepatology 2021

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Nutrition Pearls

Of the right stuff : Protein intake

Recommended protein intake: 1.2-1.5 grams/kg body weight/day

Body weight *(use ideal weight)*	Recommended daily protein intake
140 pounds (~60kg)	~75-95 grams
180 pounds (~80 kg)	~100-120 grams
220 pounds (100 kg)	120-150 grams
250 pounds (~110 kg)	130-165 grams

Strive to maintain recommended daily protein targets even if on caloric-restriction for weight loss.

Of the right stuff : Protein intake

GREAT SOURCES	
Chicken (3 ounces)	30 g
Greek yogurt (1 cup)	20 g
Tofu (1 cup)	20 g
Lentils, boiled (1 cup)	18 g
Edamame (1 cup)	17 g
Salmon (3 ounces)	17 g
Beans (1 cup)	15 g
Almonds/pistachios (1/2 cup)	15 g

GOOD SOURCES	
Peanut butter (2 T)	8 g
Quinoa (1 cup)	8 g
Milk (1 cup)	8 g
Penne (1 cup)	8 g
Egg	6 g
Brown rice (1 cup)	5 g
Sunflower seeds (1/4 c)	6 g
Potato (1 medium)	5 g
Chia seeds (1 T)	5 g

OTHER FOODS	
Avocado / guac	4 g
Broccoli (1 stalk)	4 g
Corn (1 ear)	4 g
Wheat bread (1 slice)	4 g
White rice (1 cup)	4 g
Ice cream (1 scoop)	3 g
Frozen yogurt (1 cup)	3 g
Orange (1 cup)	2 g
Banana	<2 g

Lai JC et al. AASLD Practice Guidance on Malnutrition, Frailty&Sarcopenia in cirrhosis. Hepatology 2021

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Multi-disciplinary, Multi-facility Liver Tumor Boards

Enhancing access, improving quality of care, aiding in complex decision making

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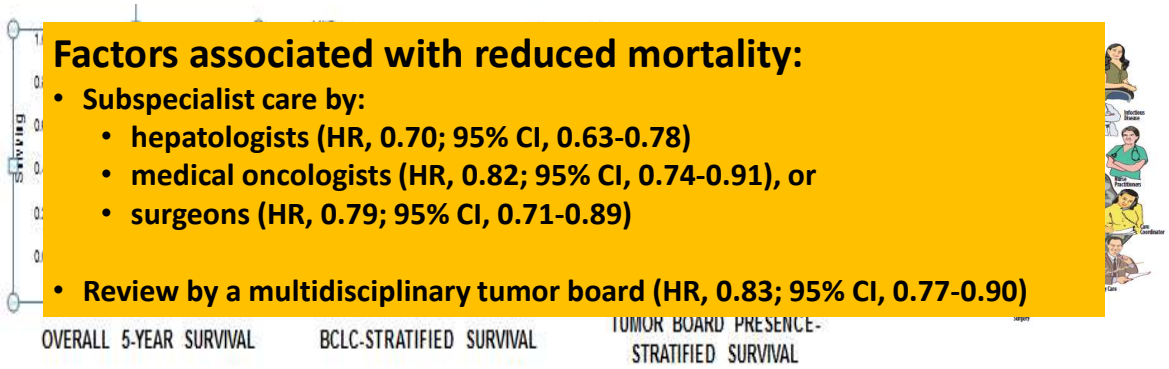
Multidisciplinary HCC Tumor Boards Impact Survival

VOCAL Study Group: National, retrospective cohort of patients diagnosed with HCC from January 1, 2008 through December 31, 2010 (n **3988**) and followed through December 31 2014 (**128 VA centers**)
Outcomes were receipt of active HCC therapy (liver transplantation, resection, local ablation, transarterial therapy, or sorafenib) and overall survival.



Factors associated with reduced mortality:

- **Subspecialist care by:**
  - **hepatologists (HR, 0.70; 95% CI, 0.63-0.78)**
  - **medical oncologists (HR, 0.82; 95% CI, 0.74-0.91), or**
  - **surgeons (HR, 0.79; 95% CI, 0.71-0.89)**
- **Review by a multidisciplinary tumor board (HR, 0.83; 95% CI, 0.77-0.90)**

M.Serper, T. Taddei, A. Aytaman, D. Kaplan: Gastroenterology 2017;152:1954–1964

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Our SCAN ECHO HCC Tumor Board Approach

Multidisciplinary multi facility discussion:

- Take into account CPT score/CSPH :
  - Below 7 , CPT A with no CSPH we have many options
  - Above 7 no resection or SBRT, but MWA, TAE, TACE, TARE with transplant consideration
- Take into account tumor size:
  - Greater than 3 cm surgery or SBRT better options
  - Smaller than 3 cm multiple options with similar efficacy
- Take into account tumor location:
  - Dome tough to biopsy or ablate, surgery and SBRT better options
  - Central location, close to major vessels MWA less desirable
- Take into account patient comorbidities/age
- Take into account local expertise and availabilities
- Take into account transplant candidacy
- Take into account patient goals and wishes

HCC with aggressive biology:
Poor differentiation,
Rapid doubling,
Vascular invasion
Presence of satellites
High AFP
High neutrophil/lymphocyte ratio
Circulating tumor cells
Diffusion restriction on MRI
PET positivity

CPT Child Pugh Turcot
CSPH Clinically Significant Portal HTN
SBRT Stereotactic Body Radiation Therapy

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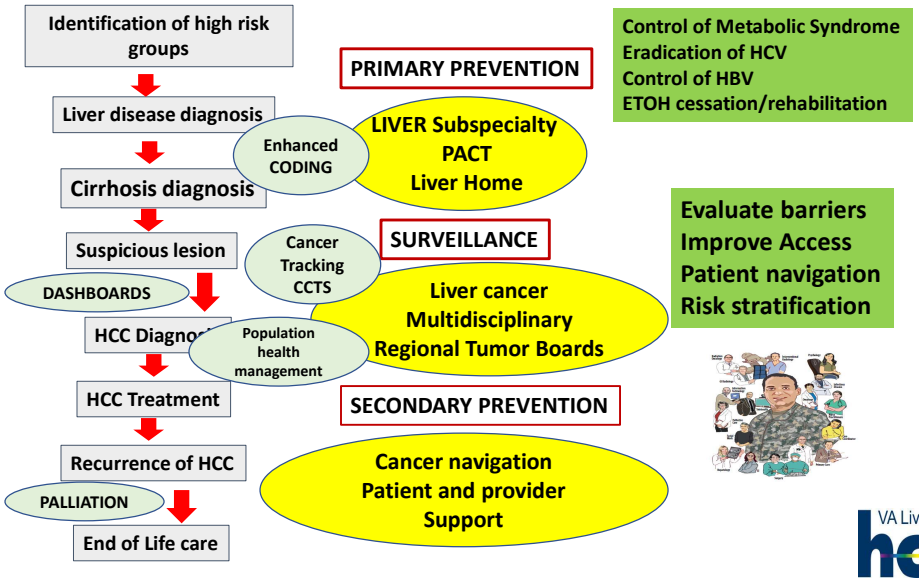
THE CONTINUUM OF CARE
THE PATIENT IS AT THE CENTER



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TAKE HOME POINTS

- Epidemiology is changing with eradication of hepatitis C and emergence of NAFLD/NASH with obesity epidemic.
- Screening needs to be refined with changing risk allocations and epidemiology.
- Imaging quality of contrast enhanced MRI and CT is crucial in diagnosis and management and standardized communication between radiologists and providers area crucial: LIRAD integration nationally.
- Resection, ablation via MWA, RFA, SBRT are curative options for small lesions in addition to surgical resection.
- Liver transplantation cures the cancer and underlying disease, limited by access. Refer timely.

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TAKE HOME POINTS

- Multiple targeted therapies are available and in development.
- Combination regimens used timely and sequentially can improve overall survival. Do not delay transition to systemic therapy.
- Evaluation of nutritional status, sarcopenia, frailty with associated interventions are key to survival with good quality of life.
- Recognition of HCC with aggressive biology may avoid futile therapies.
- Palliative Care should be an integral part of management starting at time of diagnosis

A multidisciplinary approach is the mainstay for complex decision-making

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Questions?

Ayse Aytaman, MD, FACG

Janice Jou, MD

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ACG Hepatology Circle
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