C A S E - B A S E D

Cirrhosis and Hepatitis C

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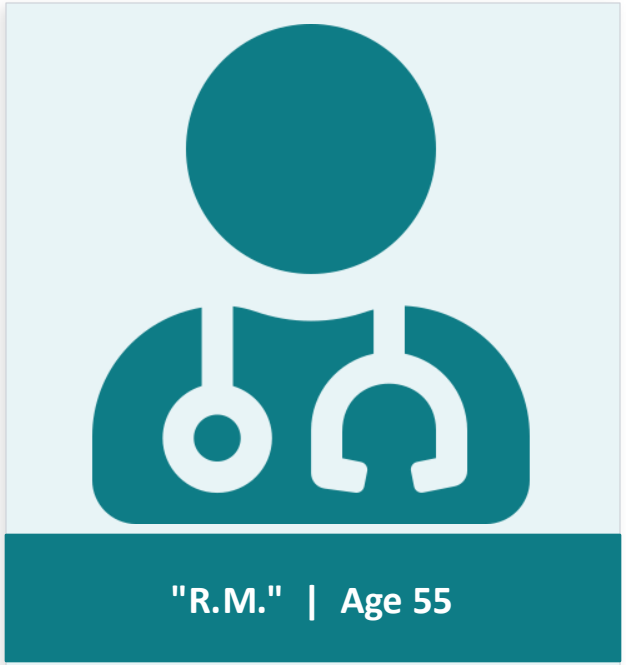
Learning Objectives

- Understand the evolution of HCV treatment — from interferon to pangenotypic DAAs, and define SVR
- Recognize compensated vs. decompensated cirrhosis and its clinical features
- Apply appropriate HCC surveillance strategies in cirrhotic patients — even after viral cure

Meet the Patient



Chief Complaint

Referred for evaluation of abnormal liver labs and imaging

Key History

HCV diagnosed ~25 years ago. Treated with interferon. Lost to follow-up.

Social History

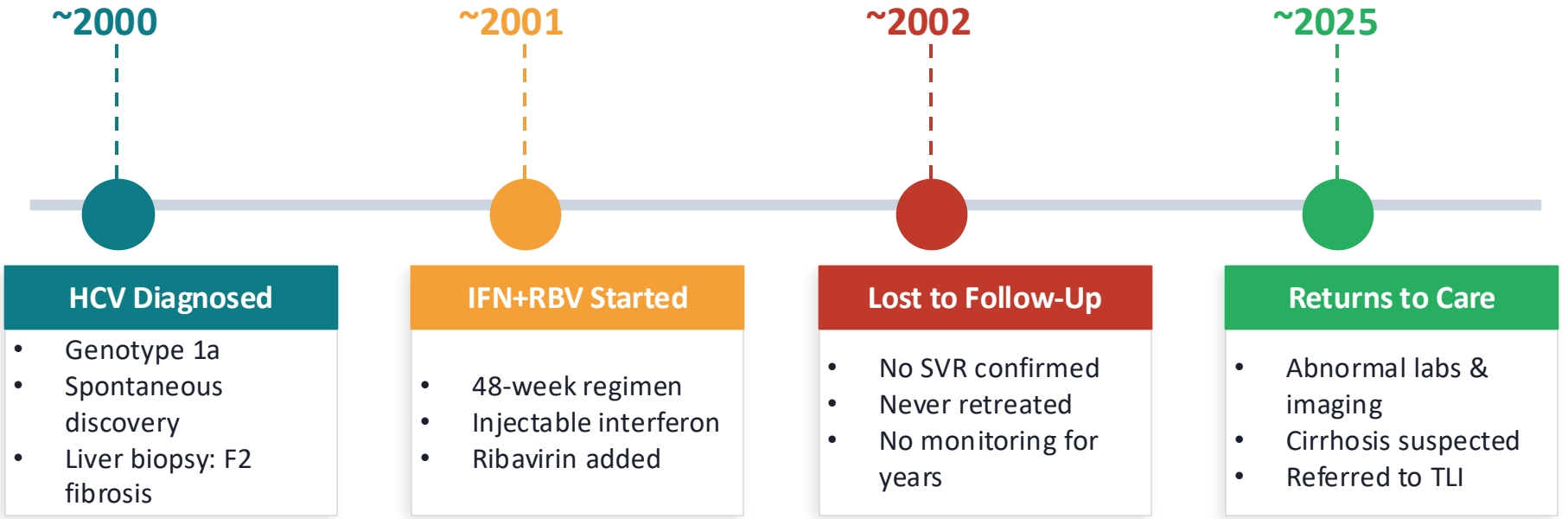
Judicially involved; in-and-out of care. Recently released from county jail.

Why Now?

Labs and imaging now suggestive of advanced liver disease

Past Medical History

Discussion: What could have been different with regular follow-up?



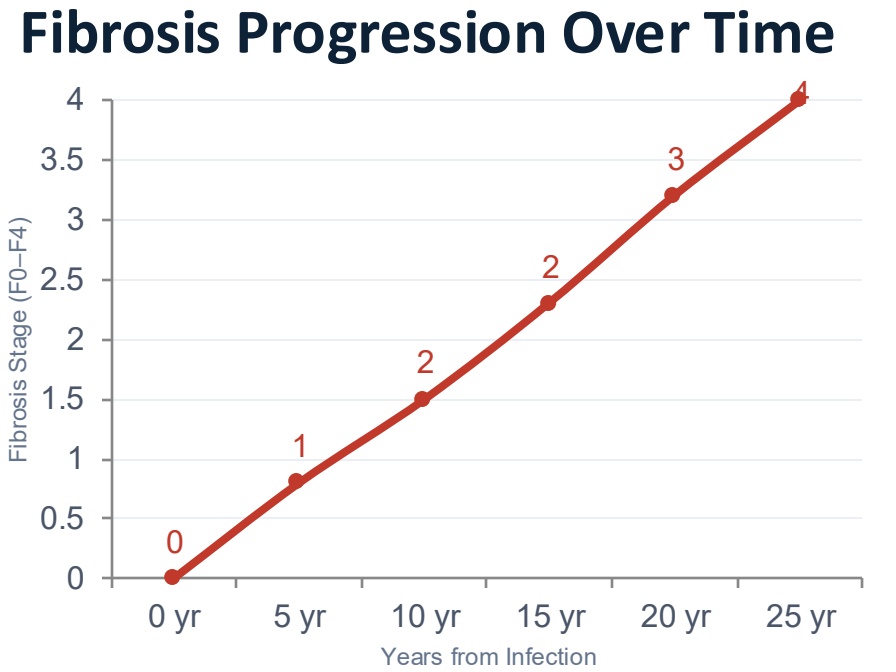
Natural History of HCV

Acute HCV
~80% become chronic

Chronic HCV
20–30% → cirrhosis

Cirrhosis (F4)
1–4%/yr → HCC

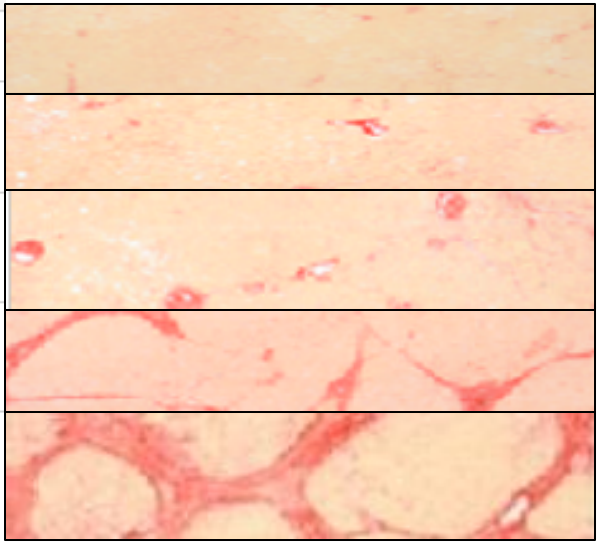
Decompensation
5%/yr decompensate



⚠ *Fibrosis can progress silently over 10–30 years with no symptoms*

Fibrosis Staging: METAVIR Score

Stage	Description	Histologic Features	Clinical Significance
F0	No fibrosis	Normal liver architecture	No liver disease
F1	Mild fibrosis	Portal fibrosis without septa	Low risk; primary care management
F2	Significant fibrosis	Portal fibrosis with few septa	Threshold for hepatology referral
F3	Advanced fibrosis	Numerous septa without cirrhosis	High risk of progression; requires specialist care
F4	Cirrhosis	Complete architectural distortion with regenerative nodules	Risk of decompensation, HCC, portal hypertension



Our patient started at F2 ~25 years ago.
With no follow-up or retreatment,
he silently progressed to F4 (cirrhosis).

Labs on Re-presentation

Lab Test	Value	Reference	Interpretation
Platelets	98 K/µL	150–400 K/µL	**Portal HTN**
AST	72 U/L	10–40 U/L	**↑ Hepatocellular**
ALT	58 U/L	7–56 U/L	**↑ Hepatocellular**
Total Bilirubin	1.4 mg/dL	0.1–1.2 mg/dL	**Borderline**
Albumin	3.4 g/dL	3.5–5.0 g/dL	**Low**
INR	1.2	0.9–1.1	**Mildly elevated**
HCV RNA	2.1×10^6 IU/mL	Undetectable	**Active viremia**

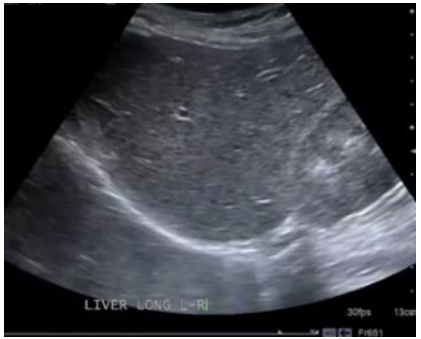
Clinical Pearl: Low platelets + elevated AST/ALT in a patient with known HCV history → think advanced fibrosis/cirrhosis until proven otherwise

Imaging Findings

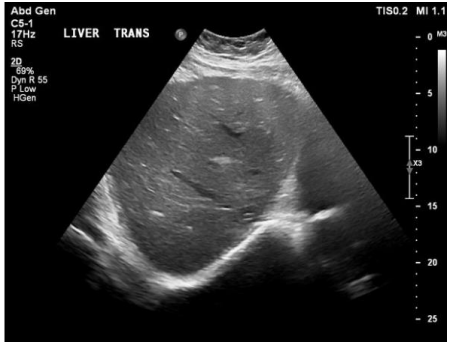
Ultrasound Report – Key Findings

Liver echogenicity:	Coarsened, heterogeneous echotexture
Surface contour:	Nodular liver surface — classic for cirrhosis
Liver size:	Mildly shrunken right lobe
Spleen*:	Splenomegaly (14.8 cm) — portal hypertension
Ascites:	None identified
Portal vein:	Slightly dilated at 13 mm — borderline PH
No focal lesions:	No discrete mass — baseline established

Normal Liver vs Cirrhotic Liver



Smooth surface
Uniform texture



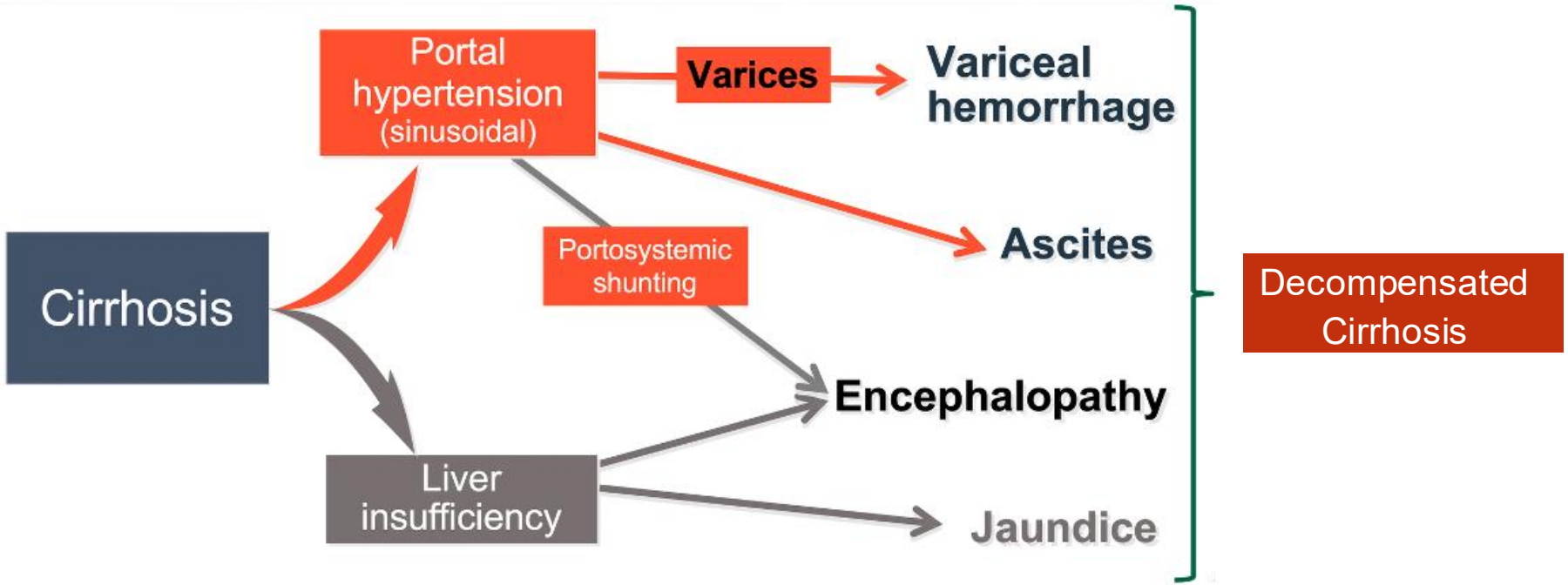
Nodular surface
Heterogeneous texture

Why This Ultrasound Matters

1. Establishes cirrhosis diagnosis without biopsy
2. No focal lesions — clean baseline for future HCC surveillance
3. No ascites yet — still compensated

*Important to get complete abdominal US instead of just RUQ which doesn't capture spleen.

Compensated vs Decompensated Cirrhosis



Child-Pugh Score

Parameter	1 Point	2 Points	3 Points	Our Patient
Bilirubin (mg/dL)	< 2	2–3	> 3	**1.4 → 1 pt**
Albumin (g/dL)	> 3.5	2.8–3.5	< 2.8	**3.6 → 1 pt**
INR	< 1.7	1.7–2.3	> 2.3	**1.2 → 1 pt**
Ascites	None	Controlled	Refractory	**None → 1 pt**
Encephalopathy	None	Grade I–II	Grade III–IV	**None → 1 pt**

1-Year Survival by Class

Class A (5–6 pts)
1-yr survival: 100%
Our patient: 5 pts

Class B (7–9 pts)
1-yr survival: 80%

Class C (10–15 pts)
1-yr survival: 45%

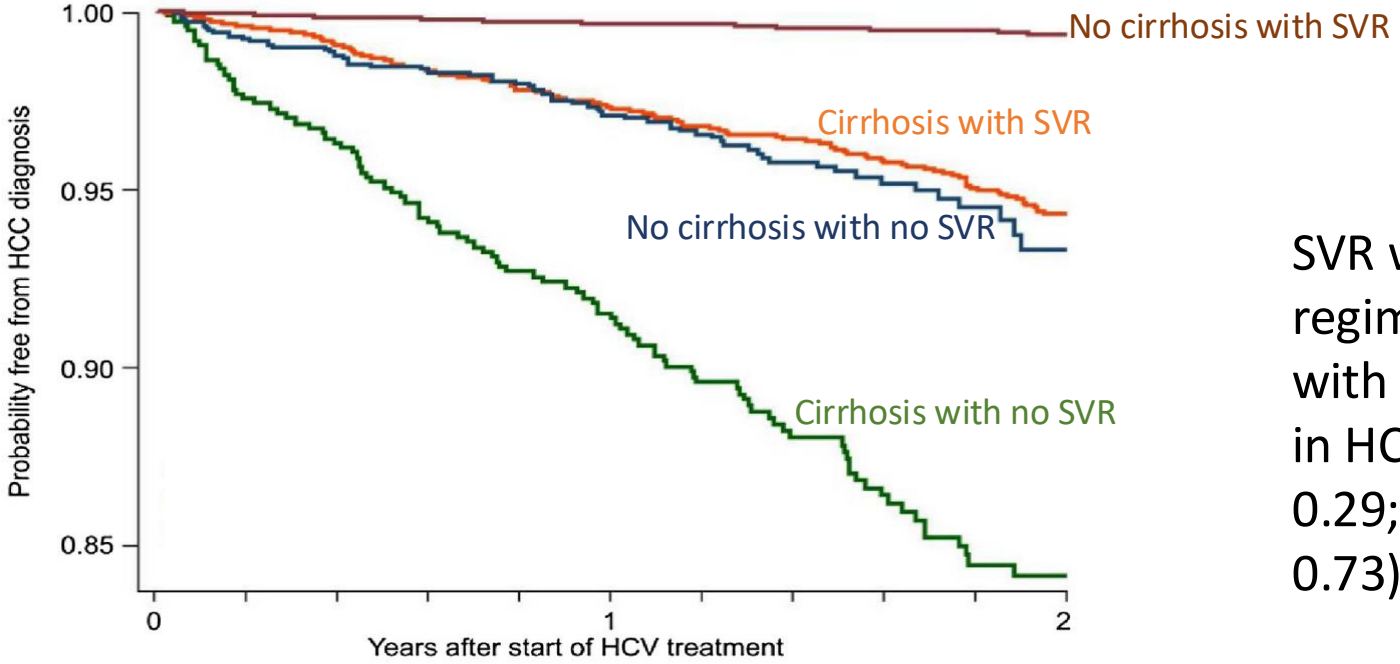
Clinical Assessment

Compensated HCV Cirrhosis (CTP-A)

Clinical	Laboratory	Imaging	HCV
• No encephalopathy • No ascites • Alert & oriented	• Low platelets (98K) • Elevated AST/ALT • INR 1.2	• Nodular liver surface • Splenomegaly • No focal lesion	• GT1a active viremia • Prior IFN non-response • No retreatment

HCV Eradication by DAAs Reduces the Risk of HCC

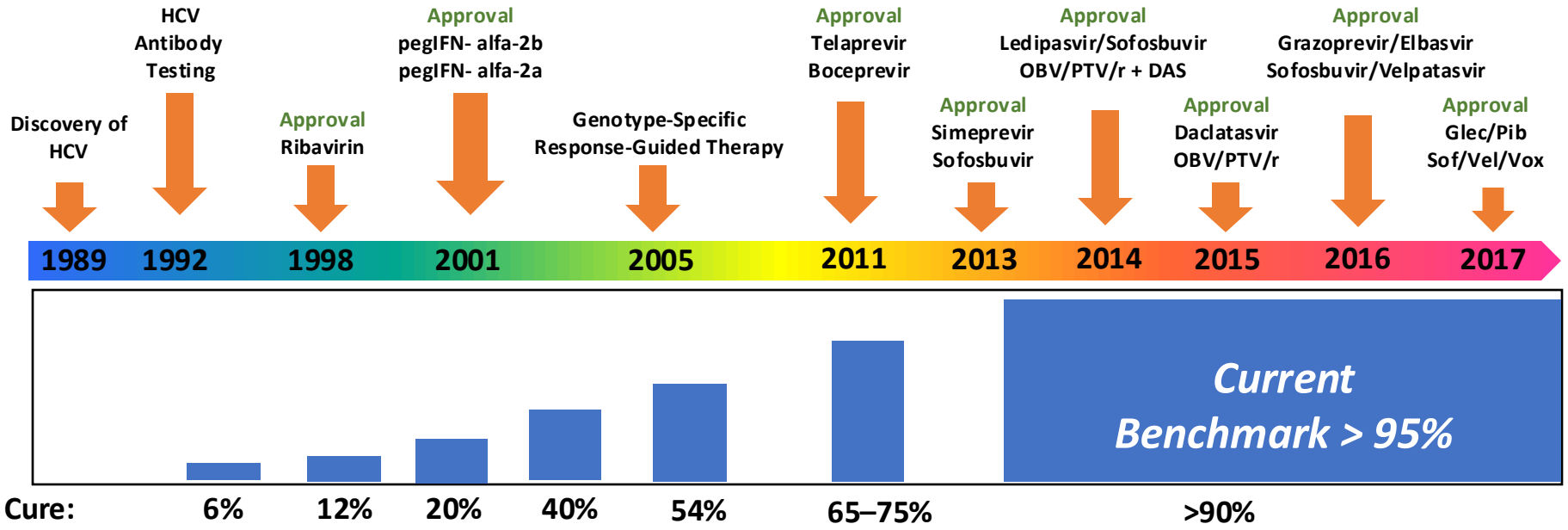
Retrospective VA cohort study: 1999-2015 (N = 62,354)
58% received IFN-only therapy; 35% received DAA-only therapy



SVR with DAA regimen associated with 71% reduction in HCC risk (AHR 0.29; 95% CI 0.32-0.73)

Ioannou, GN et al., *J Hep* 2017 Sep 5:S0168-8278(17)32273-0.

Discovery of Direct Acting Antivirals (DAAs) Revolutionized HCV Therapy



- Patients can be cured in 8-12 weeks
- Therapy with oral DAAs have very few adverse events
- Interferon and ribavirin rarely used

**Our patient started an 8-week DAA regimen.

What Defines HCV Cure?

SVR (Sustained Virologic Response):
Undetectable HCV RNA in the blood 12 weeks after completing antiviral treatment — the benchmark for virologic cure

Virus Eradicated

HCV RNA remains undetectable in >99% of SVR patients long-term

Liver Inflammation ↓

ALT/AST normalize. Ongoing hepatic inflammation resolves.

Fibrosis Can Regress

Early-stage fibrosis (F1–F3) may regress. Cirrhosis rarely reverses fully.

HCC Risk ↓ (Not Zero)

Risk drops ~70% after SVR but persists in cirrhotic patients

Mortality Benefit

SVR associated with 40–50% reduction in all-cause mortality

Quality of Life ↑

Fatigue, cognitive function, and overall QoL improve after cure

Our Patient Outcome — SVR Achieved

SVR12 CONFIRMED

HCV RNA: Undetectable | Lab confirmed February 12, 2026

Post-Treatment Labs

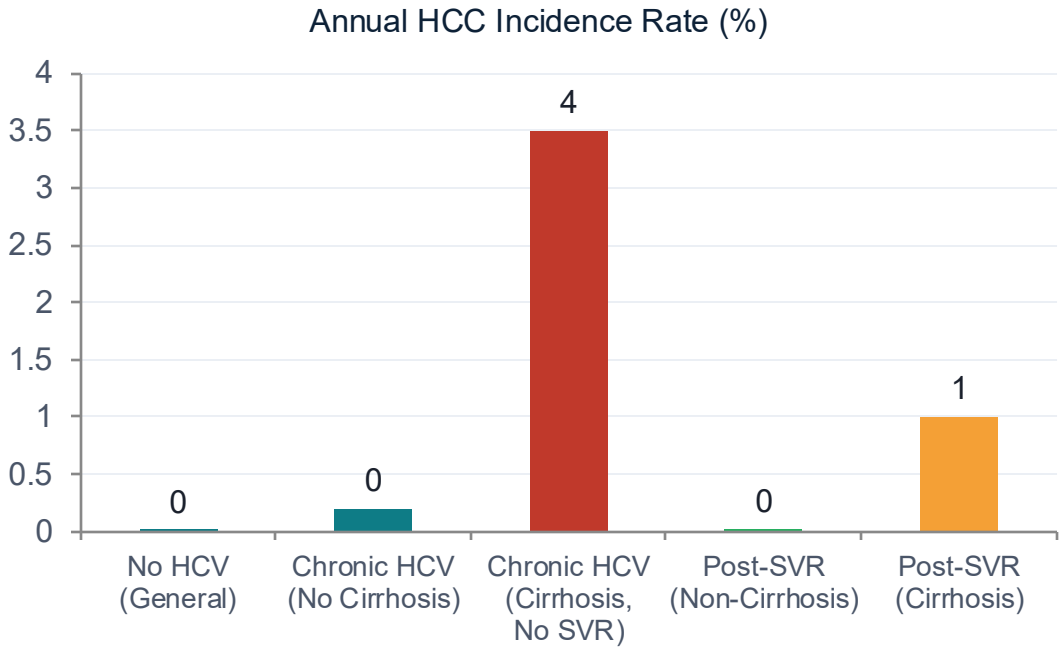
Test	Value	Change
AST	32 U/L	Normalized ↓
ALT	28 U/L	Normalized ↓
Total Bilirubin	1.0 mg/dL	Normalized ↓
Platelets	110 K/µL	Improving ↑
HCV RNA	Undetectable	Target achieved ✓

What Does This Mean for Your Patient?

- ✓ HCV is eradicated
- ✓ Liver inflammation resolved
- ✓ Lower risk of hepatic decompensation
- ✓ Reduced risk of liver-related mortality

- ⚠ Cirrhosis does NOT disappear with SVR
- ⚠ HCC risk persists — ongoing surveillance required

HCC Risk After SVR



Cirrhosis = Persistent Risk

Even after HCV cure, cirrhotic liver retains HCC risk. Risk is ~1% per year post-SVR in cirrhotics.

SVR Does Help

SVR reduces HCC risk by ~70% compared to untreated cirrhosis (3.5% → ~1% per year).

Surveillance Still Required

All cirrhotic patients — regardless of SVR status — require HCC surveillance per AASLD/EASL guidelines.

Kanwal et al., Gastroenterology 2017 | Persistent HCC risk post-SVR

The patient achieved SVR. Is he cured and done with liver care?

A. Yes! Virus gone = cured. Discharge from care.

B. No — cirrhosis persists; HCC screening every 6 months required

C. Needs repeat HCV treatment to be sure

D. Only needs follow-up if symptoms develop

Who Should Get Tested for Hepatitis C?

EVERY ADULT

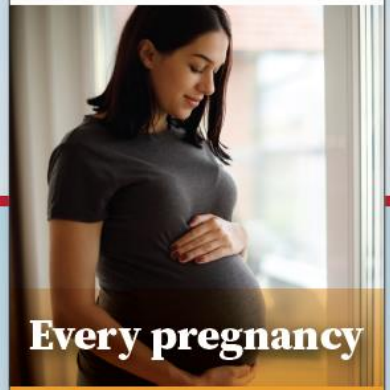
At least once

EVERYONE WITH RISK FACTORS

Regularly

- Persons who inject drugs and share needles, syringes, or other drug preparation equipment
- Persons receiving maintenance hemodialysis
- Persons with abnormal liver tests or liver disease (persistently abnormal ALT levels)
- Children born to mothers with HCV infection

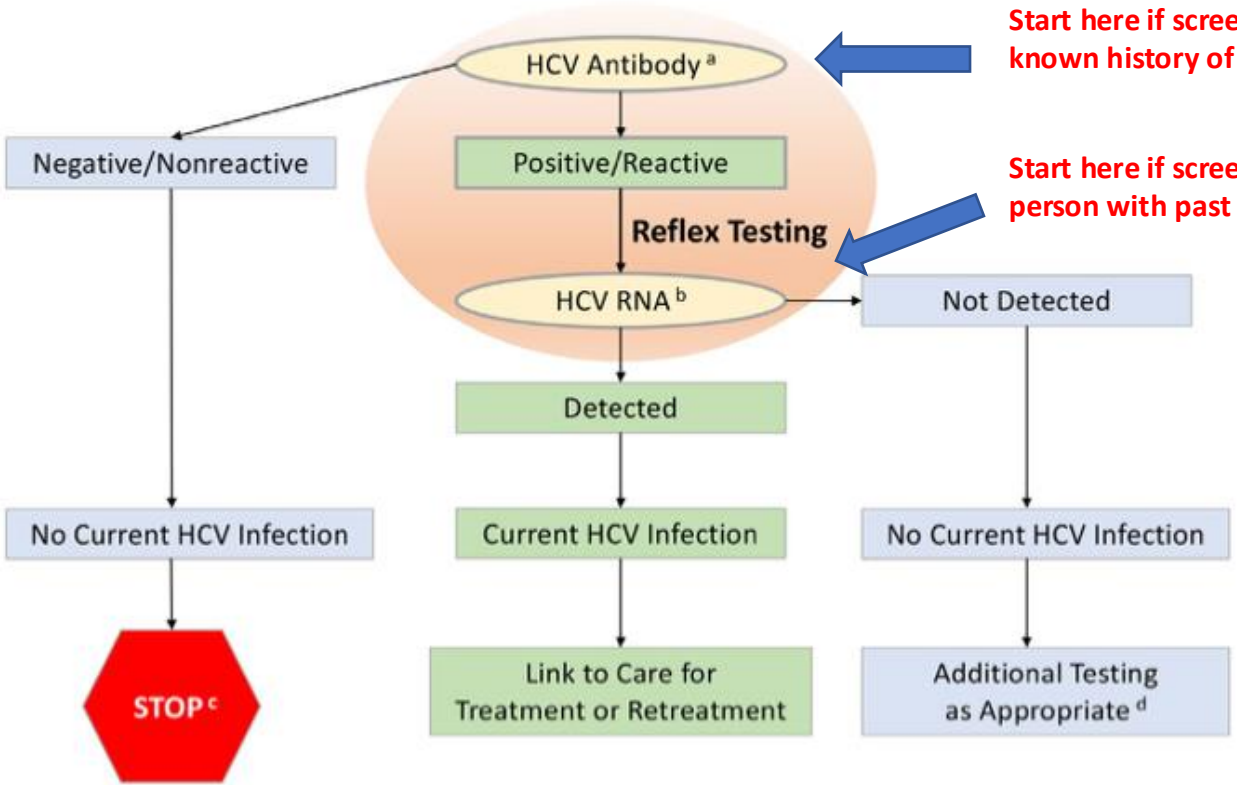
EVERY PREGNANT WOMAN



Every pregnancy

SOURCE: CDC Recommendations for Hepatitis C Screening, MMWR, April 2020

Recommended Testing Sequence for Identifying Current HCV Infection or Reinfection



AASLD-IDSA Hep C Guidance Panel, *Hepatology*, 71 (2), 2020

Ongoing Cirrhosis Management

HCC Surveillance

- Ultrasound ± AFP every 6 months
- Refer to hepatology if abnormal
- CT/MRI if indeterminate lesion

Variceal Screening

- EGD at cirrhosis diagnosis
- Repeat q2–3 yrs if no varices
- Non-selective β-blockers if varices present

Vaccinations

- Hepatitis A (if non-immune)
- Hepatitis B (if non-immune)
- Influenza, pneumococcal, COVID-19

Medications to Avoid

- NSAIDs → renal failure, GI bleed
- Opioids → precipitate HE
- Sedatives → precipitate HE

Nutrition & Lifestyle

- Avoid alcohol completely
- High protein diet (1.2–1.5 g/kg/day)
- Avoid prolonged fasting/protein restriction

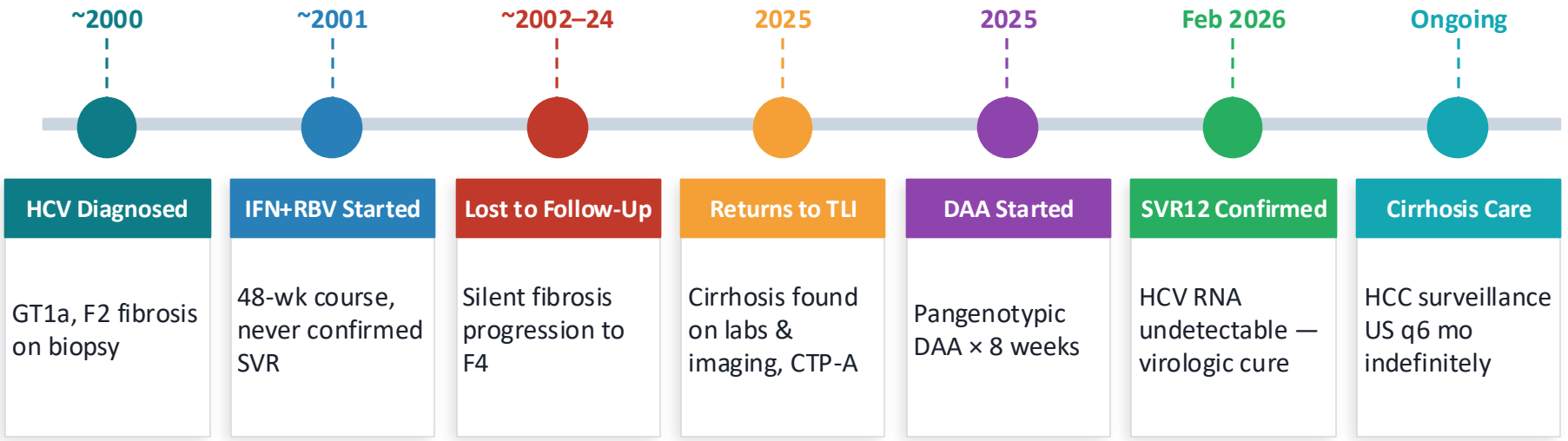
Lab Monitoring

- CBC, CMP, INR every 6 months
- MELD score trending
- AFP with surveillance ultrasound

**Repeat HCV RNA if high risk behaviors persist (reinfection)

Case Summary

Key Lesson: A 25-year journey from untreated GT1a HCV → compensated cirrhosis → DAA cure → lifelong surveillance.
SVR eliminates the virus. Cirrhosis — and its risks — remain.



What Everyone Should Remember

01 HCV can silently progress to cirrhosis over decades — even without symptoms.

02 Pangenotypic DAAs cure HCV in >95% of patients — short, oral, well-tolerated.

03 SVR = viral cure. It does NOT reverse cirrhosis.

04 Cirrhotic patients need ultrasound +/- AFP for HCC surveillance every 6 months — even after SVR.

Even when the virus is cured — the cirrhotic liver still needs surveillance. Screen every 6 months. 🎯