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Barriers to achieving SVR: What clinicians need to know?

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Introduction

Sustained Virologic Response (SVR) is the gold standard for successful treatment of chronic Hepatitis C Virus (HCV) infection. Defined as the absence of detectable HCV RNA in the blood 12 weeks or more after completing antiviral therapy, SVR is synonymous with a virologic cure for most patients. With the advent of direct-acting antivirals (DAAs), SVR rates have soared above 95% in clinical trials. Yet, real-world data reveal persistent barriers that prevent many patients from achieving SVR. For clinicians, understanding these obstacles is essential to bridging the gap between therapeutic potential and actual outcomes [1, 2].

Despite the efficacy of DAAs, certain biological factors can hinder SVR: Genotype 3 and the presence of NS5A RASs are associated with lower SVR rates. Patients with cirrhosis or hepatocellular carcinoma (HCC) may have reduced treatment response due to impaired liver function and altered drug metabolism. HIV/HCV co-infected individuals often face complex treatment regimens and drug-drug interactions. Patient characteristics and behaviors play a pivotal role in SVR outcomes: Missed doses or premature discontinuation of therapy can compromise treatment efficacy. Active drug use may lead to poor adherence and reinfection risks [3, 4].

Depression, anxiety, and cognitive impairments can interfere with treatment engagement. Misunderstanding of treatment protocols and disease implications can reduce compliance. Structural and systemic issues often impede access to care: Many individuals remain unaware of their HCV status due to inadequate screening programs. In underserved areas, lack of hepatologists or infectious disease specialists delays treatment initiation. Poor coordination between primary care, specialty clinics, and pharmacies can disrupt the treatment continuum [5, 6].

Economic constraints and policy decisions significantly affect SVR rates: Although prices have declined, treatment remains unaffordable for many without insurance or government support. Prior authorization requirements, sobriety mandates, and fibrosis staging criteria can delay or deny access. Variability in treatment protocols across regions leads to inequitable care delivery. Accurate diagnosis and follow-up are critical for achieving SVR: In low-resource settings, lack of HCV RNA testing hampers treatment eligibility and SVR confirmation. Patients may not return for post-treatment testing, leaving SVR status unknown [7, 8].

Poor record-keeping and lack of integrated electronic health records hinder longitudinal monitoring. To overcome these barriers, clinicians can adopt several proactive measures: Use pan-genotypic DAAs with minimal side effects and short durations. Collaborate with primary care, mental health, and addiction services. Use culturally sensitive materials and visual aids to improve understanding. Support initiatives that expand access to DAAs and remove restrictive insurance criteria [9, 10].

Conclusion

Achieving SVR is not just a pharmacological triumph—it's a multifaceted challenge that requires clinical vigilance, systemic reform, and patient-centered care. By recognizing and addressing the barriers outlined above, clinicians can play a transformative role in eliminating HCV and improving long-term health outcomes.

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