

SVR and reinfection risk: What the data reveals?

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Introduction

The advent of direct-acting antivirals (DAAs) has revolutionized the treatment of hepatitis C virus (HCV), offering cure rates exceeding 95%. The benchmark for cure is Sustained Virologic Response (SVR)—defined as undetectable HCV RNA 12 to 24 weeks after completing therapy. While SVR is a major milestone, it does not confer immunity. Reinfection remains a concern, particularly among high-risk populations. This article explores what current data reveals about SVR durability and reinfection risk, and what it means for global HCV elimination efforts. SVR is the gold standard for HCV treatment success. Achieving SVR significantly reduces the risk of liver-related complications, including cirrhosis and hepatocellular carcinoma [1, 2].

It also improves overall survival and quality of life. However, SVR does not eliminate the possibility of reinfection, especially in individuals with ongoing exposure risks such as injection drug use or unprotected sex. Relapse refers to the return of the same HCV strain after treatment. Reinfection involves a new infection with a different HCV strain post-SVR. Distinguishing between the two requires genotypic analysis. Most post-SVR viremia cases are reinfections rather than relapses [3, 4].

The HERO study followed 415 people who inject drugs (PWID) post-SVR and found a reinfection rate of 11.4 per 100 person-years. A Veterans Health Administration study identified 1129 cases of repeat viremia post-SVR, with 25.5% confirmed reinfections and 37.3% presumed late relapses. A Swedish NSP cohort reported a reinfection rate of 9.3 per 100 person-years, with younger age and homelessness as significant risk factors [5, 6].

These findings underscore that reinfection is not rare in high-risk groups and must be addressed in elimination strategies. Several factors elevate reinfection risk: Injection drug use: The most significant driver of reinfection. Unstable housing: Associated with limited access to healthcare and harm reduction. Incarceration: Interrupts treatment continuity and increases exposure risk. Lack of opioid agonist therapy (OAT): Reduces adherence and increases risky behaviors [7, 8].

The median time from SVR to reinfection is approximately 504 days, with reinfection rates peaking within the first two years post-treatment. This highlights the need for ongoing surveillance and support beyond the treatment window. Unlike some viral infections, HCV does not confer lasting immunity. Patients who achieve SVR still retain anti-HCV antibodies, but these do not protect against reinfection. Reinfection can occur with different genotypes, and in some cases, may be more severe than the initial infection [9, 10].

Conclusion

SVR is a transformative milestone in HCV care, but it's not the end of the story. Reinfection remains a real and measurable risk, especially among vulnerable populations. The data reveals that reinfection is common, often preventable, and must be integrated into elimination strategies. By combining clinical vigilance with social support, we can ensure that SVR leads not just to cure—but to lasting health.

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