

CASE REPORT | LIVER

A Rare Case of Icteric Acute Hepatitis C Infection Acquired Through Intranasal Methamphetamine Use

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Abstract

Most patients with acute hepatitis C (HCV) infections are asymptomatic, while 15% present with jaundice. Intranasal drug use can uncommonly transmit HCV via contaminated instruments and nasal epithelial breakdown. Given a 15% prevalence of HCV infection in chronic methamphetamine users, recognition of potential transmission routes is important to target prevention and screening efforts in this population.

Introduction

Most patients with acute hepatitis C (HCV) infection are asymptomatic, while only about 15% present with jaundice. Although uncommon, acute icteric hepatitis is important to recognize because the accompanying intense immune response is associated with higher rates of spontaneous clearance of the infection. The most common route of HCV transmission is intravenous drug use, but up to 20–40% of individuals have unidentified routes of transmission.^{1,2} We present a rare case of acute icteric HCV infection acquired through intranasal methamphetamine use.

Case Report

A 38-year-old male presented with a 1-month history of generalized body aches, nausea, pruritus, and darkcolored urine. He drank a pint of bourbon daily. He reported intranasal methamphetamine use 6 weeks previously by sharing a straw with his mother who had known chronic HCV. He denied intravenous (IV) drug use, hepatotoxic medications, or herbal products. His vital signs were normal, and physical examination findings included icteric sclera and mild hepatomegaly. He was alert and oriented without asterixis. Laboratory data showed total bilirubin 6.2 mg/dL, direct bilirubin 5.4 mg/dL, aspartate aminotransaminase (AST) 1,735 U/L, alanine aminotransferase (ALT) 1,647 U/L, alkaline phosphatase 184 U/L, and INR 0.9. He had an undetectable acetaminophen level, normal serum ceruloplasmin 36.8 mg/dL, normal total IgG 703 mg/dL, and negative results for antinuclear antibody, anti-smooth muscle antibody, hepatitis A IgM, hepatitis B surface antigen, hepatitis B core IgM, EBV PCR, CMV PCR, and hepatitis C antibody (Ab). An abdominal ultrasound with Doppler was unremarkable, with no signs of vascular occlusion.

Repeat laboratory testing after transfer to our institution revealed positive HCV Ab and HCV RNA >6,000,000 IU/mL. His AST and ALT peaked at 2,698 U/L and 1,916 U/L, respectively. A liver biopsy (Figure 1) showed moderate to severe portal-based active hepatitis with moderate interface and lobular activity, consistent with acute HCV infection without fibrosis or steatohepatitis (<5%). Testing for the IL28B genotype returned as ho-

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Figure 1. Moderately active hepatitis without fibrosis or steatohepatitis; at 20x magnification.

mozygous C/C. His transaminases started to decline, and he was monitored for potential spontaneous viral clearance. Four weeks after discharge, his HCV RNA level was 461 IU/ mL, and 9 months after his initial presentation, his HCV RNA PCR (quantitative) and HCV TMA (qualitative) assays were both negative, indicating successful spontaneous clearance of the virus.

Discussion

We report what may be the first documented case of HCV transmission from intranasal methamphetamine use via a shared straw. Although the most common route of transmission of HCV is injection drug use, 20–40% of individuals have unidentified sources of infection.^{1,2} Potential transmission modalities include intranasal drug use, sharing of drug pipes, tattoos, or other instrumentation with unsterilized equipment.^{2–4} HCV is present in nasal secretions of infected individuals.² Active drug sniffing can increase nasal secretions and irritate the nasal epithelium, causing ulcerations and perforations.^{2–4} Instruments used for intranasal drug use can transfer secretions that contain the virus.^{2–4}

In their case–control study of risk factors for HCV transmission, Karmochkine et al found that intranasal cocaine use was independently associated with 4.5 increased odds of HCV transmission.¹ Prior studies have reported HCV transmission via intranasal cocaine and/or heroin use; however, we did not find any clearly documented cases of HCV transmission in the context of intranasal methamphetamine use.^{1–2,4} Our patient's lack of IV drug use and the temporal association of straw sharing with an HCV-infected individual support this route as the most likely source of his infection. Other causes of markedly elevated transaminases, including acute ischemic injury to the liver ("shock liver"), drug or toxin ingestion, other acute viral hepatitis, autoimmune hepatitis, and Budd-Chiari syndrome were ruled out. Our patient was actively consuming alcohol, but alcoholic hepatitis classically results in much more moderate transaminase elevations.⁵

His clinical presentation and work-up, including laboratory values (with noted HCV Ab seroconversion), imaging, and biopsy results, strongly support acute icteric HCV infection as the cause of his acute liver injury. Our patient presented with jaundice, which is a less common but paradoxically favorable sign because it represents an intense immune response that predicts a higher likelihood of spontaneous clearance of the virus.^{6,7} In addition, his favorable IL28B genotype has been shown to predict spontaneous clearance of acute HCV infection compared with the T/T or T/C genotypes.⁸ This case highlights the importance of polymerase chain reaction (PCR) testing for HCV as the diagnostic test of choice in an acute phase of the illness since seroconversion of HCV Ab takes 20–150 days.⁹

Conclusion

Although rare, HCV can be transmitted via contaminated instruments and nasal epithelial breakdown from intranasal drug use. Given a 15% prevalence of HCV infection in chronic methamphetamine users, recognition of potential transmission routes is important to target education, prevention, and screening efforts in this population.¹⁰

Disclosures

Author contributions: C. Chou wrote and proofread the manuscript, researched data, and is the article guarantor. KK Yimam, RT Frederick, and SL Swenson wrote and proofread the manuscript, and researched data. All authors drafted the manuscript and provided critical revision of the manuscript for important intellectual content.

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References

- 1. Karmochkine M, Carrat F, Dos Santos O, Cacoub P, Gaguin G. A casecontrol study of risk factors for hepatitis C infection in patients with unexplained routes of infection. *J Viral Hepat.* 2006;13(11):775–82.
- 2. Aaron S, McMahon JM, Milano D, et al. Intranasal transmission of hepatitis C virus: Virological and clinical evidence. *Clin Infect Dis.*

2008;47(7):931-4.

- Martinez A, Tala AH. No injection drug use: An under-appreciated risk factor for hepatitis C virus transmission. *Liver Int.* 2008;28(6):757–60.
- Scheinmann R, Hagen H, Lelutiu-Weinberger C,et al. Non-injection drug use and hepatitis C virus: A systematic review. *Drug Alcohol Depend*. 2007;89(1):1–12.
- Amini M, Runyon BA. Alcoholic hepatitis 2010: A clinician's guide to diagnosis and therapy. World J Gastroenterol. 2010;16(39):4905–12.
- 6. Maheshwari A, Ray S, Thuluvath PJ. Acute hepatitis C. *Lancet.* 2008;372:321–32.
- 7. Kamal SM. Acute hepatitis C: A systematic review. *Am J Gastroenterol.* 2008;103(5):1283–97.
- Spada E, Amoroso P, Taliani G, et al. Acute hepatitis Italian study group. Role of IL28B gene polymorphism and cell-mediated immunity in spontaneous resolution of acute hepatitis C. *Clin Infect Dis.* 2013;57(6):803–11.
- 9. Orland JR, Wright TL, Cooper S. Acute hepatitis C. *Hepatology*. 2001;33(2):321–7.
- 10. Gonzales R, Marinelli-Casey P, Shoptaw S, et al. Hepatitis C virus infection among methamphetamine-dependent individuals in outpatient treatment. *J Subst Abuse Treat*. 2006;31(2):195–202.

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