Acute hepatitis C virus and cardiac surgeons

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The experience of having acquired hepatitis C virus (HCV) in the operating room as a practicing cardiovascular surgeon has led me to review the literature and to try and define the proper treatment and subsequently the social implications and options related to resuming practice as quickly as possible. Below is what I have gleaned from the literature and my own experience in the hope of providing recommendations for the surgical community.

Hepatitis C virus is a worldwide problem with an estimated 170 million people infected, including 1.4% of the US population. It can lead to cirrhosis and liver cancer in the minority (20%) of patients.¹ The prevalence is as high as 22% in Egypt and averages 3% in China. The incidence in the United States has been declining steadily, and approximately 20,000 cases are now thought to occur per year.² No vaccine is available. Seroconversion or illness with acute HCV in a practicing surgeon has legal, ethical, and personal considerations, as transmission from surgeon to a patient is well documented.

Hepatitis C virus was discovered and named in 1989. Before this it was known as "non A non B hepatitis." It is unrelated to hepatitis A or B virus. It is a single-stranded RNA virus that at 50 nm is smaller than the wavelength of light. It is 9400 base pairs long, and the coding regions for the 3 structural and 7 nonstructural viral proteins are known. The virus occurs in 6 major genotypes with more than 80 subtypes. The genotypes occur in specific geographic regions of the human population. The only host is the human hepatocyte, where viral replication rates can approach a trillion per day.³

Hepatitis C virus is transmitted by blood-borne routes, including transfusion, injection drug use, contaminated medical equipment, and tattoos, and less commonly by sexual contact, perinatally, or health care worker needle stick. In developed countries where blood is screened, the major transmission is seen in drug users and patients on dialysis. In the developing world, blood transfusion, the reuse of needles, and the unnecessary administration of medications by injection are contributory. For example, in Egypt the diseases of visceral leishmaniasis and schistosomiasis are treated by injection often with reused needles. Transmission in monogamous sexual couples is rare or nonexistent.⁴

Clinically, HCV is symptomatic in only 15% of cases; 85% of patients acquire the virus without knowledge that they have become infected. If symptoms occur, they are jaundice, indigestion, fatigue, myalgia, and right upper quadrant pain. Approximately 25% of patients will clear the virus spontaneously, but 75% go on to have a persistent viremia. Clearance of the virus either spontaneously or with treatment is considered a cure.⁵ The occurrence of acute jaundice and symptoms is associated with a vigorous immune response and a higher rate of clearance.⁶ The immune response is led by CD4 T cells and subsequently by CD8 killer T cells. A prolonged response by these cells correlates with clearance. However, no long-term immunity is produced and reinfection with HCV is possible. Other factors that may be associated with spontaneous clearance are genotype 3, female sex, a younger age, a low viral load, and Caucasian race. Chronic HCV infection leads to cirrhosis and liver cancer in the minority of patients.

Transmission to a health care worker by needle stick is a real phenomena and of increasing importance in the United States. The risk per stick from an infected patient seems to be approximately 1%.^{7,8} The lifetime risk of seroconversion to a cardiac surgeon over a 30-year career is as high as 5%.⁹ Transmission to patients by the operating surgeon has occurred in gynecology, orthopedics, dentistry, and cardiac surgery. Mathematic modeling with reasonable assumptions indicate that transmission would occur in 1 of 10,000 cases.¹⁰ This risk is lower than that from screened blood transfusion.

After exposure, seroconversion occurs at approximately 20 to 30 days, and symptoms, if they are to occur, occur at an average of 50 days. At 1 to 3 months, elevated alanine aminotransferase levels up to 20 times that of normal levels may be seen. Fulminant hepatic failure is rare. If antibodies against HCV are detected, it is mandatory to confirm the result with quantitative HCV polymerase chain reaction. These assays now are accurate to 2 copies of the RNA virus per milliliter of blood. The detection of HCV RNA without demonstrable antibodies suggests acute infection because viremia may precede antibody production. There are no specific recent guidelines for postexposure by needle stick that take into account the recent treatment literature. It now seems reasonable to test liver function studies (alanine aminotransferase), HCV antibody, and HCV RNA polymerase chain reaction at baseline and at 1, 2, 3, and 6 months. This would allow for the early institution of treatment if desired by the patient.

The goal of treatment is sustained virologic response. This is defined by the lack of detectable virus at 6 months after the end of treatment. This is compatible with cure because relapse rates are extremely low. For acute HCV, current

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treatment has evolved to pegylated interferon alpha-2b at a dose of 1.5 $\mu\text{g/kg}$. This is a subcutaneous injection given weekly for 24 weeks. Interferons are cytokine proteins produced by leukocytes in response to double-stranded RNA seen during viral replication. They are responsible for producing flu-like symptoms, and therefore the side effects are fever, myalgias, fatigue, insomnia, and neurologic symptoms such as depression. Some studies indicate an early responder with no detectable virus at 4 weeks need only be treated for 3 months. The addition of ribavirin as used for chronic HCV has not been shown to be additive for acute HCV.¹¹ The current recommendations for when to institute treatment are a balance between the known chance of spontaneous clearance and the recent indications in the literature that early therapy may increase clearance rates. In any case, treatment should be started within 3 months of symptoms.¹² Patients with genotype 1 may benefit from early treatment.¹³ A surgeon might choose immediate treatment to maximize his/her chances of sustained virologic response.

In pursuing medical, legal, malpractice, ethical, and clinical guidelines, there is little guidance available. It seems uniformly to make sense that a surgeon with an acute infection and a high viral load should not participate in operations. Also, if one achieves a sustained virologic response compatible with a cure, one would not limit one's practice. In the case of a surgeon who is persistently viremic after 6 months (ie, chronic HCV), the recommendations differ. In England, Ireland, and Australia,¹⁴ guidelines are in place that discourage or prohibit a surgeon from performing exposure-prone procedures. In the United States, the official recommendation of the Centers for Disease Control is that chronic HCV not limit a health care worker's ability to practice.¹⁵ The stance of the American College of Surgeons is that because of the extremely low incidence of surgeon-to-patient transmission, a surgeon not limit his/her practice and has no duty to disclose his/her status to the patient. The college, however, states a surgeon should know his/her own HCV status.¹⁶ It has been suggested that because of the high incidence of cardiac surgeon injury during closure with sternal wires, perhaps an assistant should close. Other than the above, there is no current call to test all surgeons and have their antibody status known, nor is it recommended to test all patients undergoing surgery.

In my own case, I choose not to try and keep any secrets, from partners, colleagues, or hospital staff. We informed each hospital administration of my status, and I voluntarily withdrew from the operating room. Regular meetings with the medical staff offices were undertaken, and they had access to my medical records. Consideration must be given to instituting discussions with malpractice carriers and disability insurers.

Recommendations from a knowledgeable hepatologist were taken into account to enable me to make rounds and see office patients. Personally, I chose immediate treatment with interferon alpha-2b, and I took ribavirin. At the present time, my most recent viral load is zero and I have resumed unrestricted practice.

References

1. Hoofnagle JH. Course and outcome of hepatitis C. *Hepatology*. 2002;36(5 Suppl. 1): S21-9. Review.
2. Centers for Disease Control and Prevention. *Surveillance for Acute Viral Hepatitis United States*. 56th ed. Atlanta, GA: Centers for Disease Control and Prevention; 2005.
3. Maheshwari A, Ray S, Thuluvath PJ. Acute hepatitis C. *Lancet*. 2008;372:321-32.
4. Vandelli C, Renzo F, Romano L, et al. Lack of evidence of sexual transmission of hepatitis C among monogamous couples: results of a 10 year prospective study. *Am J Gastroenterol*. 2004;99:855-9.
5. Weigand J, Jackel E, Cornberg M, et al. Long term follow up after successful interferon therapy of acute hepatitis C. *Hepatology*. 2004;40:98-107.
6. Santantino T, Medda E, Ferrari C, et al. Risk factors and outcome among a large patient cohort with community acquired hepatitis c in Italy. *Clin Infect Dis*. 2006; 43:1154-9.
7. Kubitschke A, Bader C, Tillman HL, et al. Injuries from needles contaminated with hepatitis C virus, how high is the risk of seroconversion really? *Der Internist (Berl)*. 2007;48:1165-72.
8. De Carli G, Puro V, Ippolito G. Studio Italiano Rischio Occupazionale da HIV Group. Risk of hepatitis C virus transmission following percutaneous exposure in healthcare workers. *Infection*. 2003;31:22-7.
9. Kuo Y, Fabiani J, Mohamed A, et al. Decreasing occupational risk related to blood-borne viruses in cardiovascular surgery in Paris, France. *Ann Thorac Surg*. 1999;68:2267-72.
10. Ross RS, Viazov S, Roggendorf M. Risk of hepatitis C transmission from infected medical staff to patients: model based calculations for surgical settings. *Arch Intern Med*. 2000;160:2313-6.
11. Calleri G, Cariti G, Gaiottino F, et al. A short course of pegylated interferon alpha in acute HCV hepatitis. *J Viral Hepat*. 2007;14:116-21.
12. Nomura H, Sou S, Tanimoto H, et al. Short term interferon alpha therapy for acute hepatitis C: a randomized controlled study. *Hepatology*. 2004;39:1213-9.
13. Watson K. Sharps injuries and hepatitis C risk. *Med J Aust*. 2004;181:366-7. Accessed July 21, 2008.
14. Available at: www.cdc.gov/hepatitis/HCV/HCVfaq.htm.
15. Available at: www.facs.org/fellows_info/statements/st_22_.html. Accessed April 2004.
16. Available at: www.facs.org/fellows_info/statements/st_22_.html. Accessed April 2004.