curred, ELISA reactivity had decreased, SIA reactivity against HCV core protein had disappeared, and SIA reactivity against NS3 and NS4 proteins had decreased (table 1). Evolution of the HCV status showed that the nurse was recovering from her infection.

In the case report by Morand et al. [1], complete seroconversion never occurred, despite weak ELISA reactivity at week 5. A short period (1 month) of viral replication was associated with a high ALT level. In our experience, seroconversion occurred and was followed by seroreversion that included a 2–3-month replication period and a transiently moderate elevation of the ALT level. These 2 cases outline the fact that absence of seroconversion or rapid seroreversion is strongly related to quick virus eradication, either spontaneously or with treatment [1–3, 5]; a recent report by Jaeckel et al. [4] supports this finding. Further studies of T cell response in such treated patients might help us improve our understanding of the mechanisms of early virus elimination. In our experience, follow-up with SIA is a useful tool for prognosis. Finally, the sustained response obtained with IFN-α therapy in our patient, despite the persistence of virus replication at 1 month of treatment and a genotype 1a virus, outlines the different virological response profile of primary and chronic infection with HCV. All parameters—virological, biochemical, and immunological—are of importance when making decisions regarding adaptation of postexposure treatment (i.e., type, dose, and duration of treatment).

In conclusion, acute hepatitis C is often associated with the absence of seroconversion or seroreversion, as demonstrated by the case reports presented here and elsewhere [1, 3, 4]. This may lead to underestimation of the global prevalence of hepatitis C and, moreover, the chronic evolution of hepatitis C may be overestimated.

References


Reprints or correspondence: Dr. Sophie Alain, Laboratoire de Virologie, CHU Dupuytren, 2 avenue Martin Luther King, 87042 Limoges cedex, France (sophie.alain@unilim.fr).

Clinical Infectious Diseases 2002;34:717–9 © 2002 by the Infectious Diseases Society of America. All rights reserved. 1058-4838/2002/3405-0028$03.00

Campylobacter Species: Don’t Put All Your Eggs in One Chicken

Sir—We read with interest the recent article “Campylobacter jejuni Infections: Update on Emerging Issues and Trends” by Allos [1], which raised the profile of this most important yet frustratingly elusive pathogen. However, we believe that a number of statements are not supported by the references cited by the author. First, it is stated that “the reported incidence of Campylobacter infection among homosexual men is almost 40 times greater than in the general population” (p. 1204), citing a study by Sorvillo et al. [2]. In fact, this study from Los Angeles compared the incidence of reported gastroenteritis due to Campylobacter species in the general population with the incidence among patients with AIDS, who were not exclusively homosexual men. The figure quoted by the author is likely to be an overestimation, because persons with AIDS would be expected to present to the health services (and, hence, be reported) more than would the general population.

It is also stated that, “depending upon the population studied, as many as 50% of persons who are infected during outbreaks are asymptomatic” (p. 1202). The study cited by the author [3] reported a ratio of illness to infection for Campylobacter species of 1:2 among Mexican children <5 years of age who were followed up for 1 year, but there is no indication in that study that the cases were related to an outbreak of infection. Indeed, the ratio of illness to infection depends upon the prevalence of immunity and is likely to be influenced by underlying endemicity and the degree of previous exposure, regardless of whether infection is acquired sporadically or as part of an outbreak. This was supported in the study by the positive relationship between age and proportion of asymptomatic infections.

Most importantly, the author states, “In studies in many parts of the United States, Europe, and Australia, 50%–70% of all Campylobacter infections have been attributed to consumption of chicken” (p. 1203). One of the studies cited is that by Adak et al. [4], which was performed in England in 1990. The results of this study appear to have been misinterpreted. The study did not report population-attributable fractions for the exposures identified, and, contrary to what was implied, the study demonstrated an inverse relationship between handling or consumption of chicken prepared in the home and risk of gastroenteritis due to Campylobacter species (OR, 0.44; 95% CI, 0.24–0.79). Among the risk factors...
identified, however, were occupational exposure to raw meat, presence of a household pet with diarrhea, and ingestion of untreated water from lakes, rivers, and streams. More recent studies have shown that consumption of commercially-prepared chicken (but not chicken that is prepared in the home) is a risk factor [5-7]. Moreover, although the consumption of chicken accounts for a minority of cases, most infections remain unexplained by recognized risk factors [8]. Thus, the role of chicken consumption as a route of transmission of Campylobacter species to humans remains unclear, and while it is plausible that cross-contamination of other foods occurs after the handling of raw poultry, this has not been demonstrated convincingly in case-control studies. Twenty years of emphasizing the importance of poultry in Campylobacter transmission has yielded little success in terms of its control and prevention. There may well be much more to Campylobacter infection than chickens. We need to keep an open mind.

Clarence C. Tam, Sarah J. O’Brien, Goutam K. Adak, and Iain A. Gillespie
Gastrointestinal Diseases Division, Communicable Disease Surveillance Centre, London, United Kingdom

References


Reprints or correspondence: Clarence C. Tam, Gastrointestinal Diseases Division, PHLs Communicable Disease Surveillance Centre, 61 Colindale Ave., London NW9 5EQ, United Kingdom.

Clinical Infectious Diseases 2002;34:719-20 © 2002 by the Infectious Diseases Society of America. All rights reserved. 1058-4838/2002/3405-0029$03.00

Reply

Sir—Dr. Tam and colleagues raise 3 interesting points about the epidemiology of Campylobacter infections [1]. First, they point out that Campylobacter infections that occur in a group of persons with AIDS who are largely homosexual men are more likely to be reported than are such infections in healthier persons [2]. I agree with Tam et al. [1] that these studies may overestimate the excess risk. Furthermore, as stated in my paper, the most recent analyses [3] suggest that the rate of Campylobacter infection in HIV-negative homosexual men is no higher than the rate in heterosexual men of a similar age.

Second, my colleagues dispute the statement that, in some studies, up to 50% of persons infected with Campylobacter species are asymptomatic. They point out that such high rates of asymptomatic infection are typical only among young children residing in areas of endemicity. Tam and colleagues are quite correct both in their statement and in their interpretation of the cited references. High rates of asymptomatic Campylobacter infections detected during outbreaks in Western countries, although reported [4-6], are distinctly unusual. Most persons infected with Campylobacter species, especially in industrialized nations, are likely to exhibit symptoms.

Finally, Tam and colleagues argue against the importance of poultry that is not eaten in restaurants as a leading source of Campylobacter infection in humans. My review of the relevant literature leads me to disagree. The following facts bear repeating:

1. US Department of Agriculture statistics indicate that ~90% of broiler chicken carcasses are contaminated with Campylobacter species [7]. Although other studies have shown lower rates of contamination, depending on the season and geographic location, in virtually all investigations, the contamination rate is ≈50%.

2. The quantity of Campylobacter organisms on the surface of a fresh chicken carcass is estimated to be 10⁸ to 10⁹ per chicken [8].

3. The infective dose of Campylobacter species is not known precisely, but as few as 500 organisms can produce illness in some healthy persons [9].

4. The importance of cross-contamination of food by Campylobacter species is not known precisely, but as few as 500 organisms can produce illness in some healthy persons [9].

5. Although numerous case-control studies of Campylobacter-infected persons, including the studies cited by the authors, have documented a variety of potential sources, when one reviews the literature in its totality, the evidence is rather overwhelming that poultry consumption and preparation is implicated in most infections in humans [3].

6. The emergence of antibiotic resistance among Campylobacter isolates recovered from humans has directly followed and mirrored such resistance in Campylobacter isolates recovered from poultry flocks [11]. Additional molecular subtyping of strains recovered from chickens and humans has confirmed the association between Campylobacter con-