Hepatitis A Transmitted by Food

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Hepatitis A is caused by hepatitis A virus (HAV). Transmission occurs by the fecal-oral route, either by direct contact with an HAV-infected person or by ingestion of HAV-contaminated food or water. Foodborne or waterborne hepatitis A outbreaks are relatively uncommon in the United States. However, food handlers with hepatitis A are frequently identified, and evaluation of the need for immunoprophylaxis and implementation of control measures are a considerable burden on public health resources. In addition, HAV-contaminated food may be the source of hepatitis A for an unknown proportion of persons whose source of infection is not identified.

FEATURES OF HEPATITIS A

Hepatitis A virus (HAV) is classified as a picornavirus. Primates are the only natural host [1]. There is only 1 HAV serotype, and immunity after infection is lifelong [2]. After ingestion, uptake in the gastrointestinal tract, and subsequent replication in the liver, HAV is excreted in bile, and high concentrations are found in stool specimens. Transmission occurs by the fecal-oral route, either by direct contact with an HAV-infected person or by ingestion of HAV-contaminated food or water. The median incubation period (i.e., time from exposure to onset of symptoms) is 28 days (range, 15–50 days) [3]. Peak infectivity occurs during the 2-week period that precedes the onset of jaundice and declines during the week after onset. In persons without jaundice, peak infectivity likely occurs as serum alanine aminotransferase [ALT] concentrations increase. Viremia can be detected before the ALT concentration increases, and HAV RNA levels often remain detectable even after the ALT level has normalized and symptoms have resolved [4].

Asymptomatic or anicteric HAV infection without the clinical signs and symptoms of hepatitis A is common in children, and <10% of children aged <6 years with HAV infection have jaundice [5]. Clinical manifestations of symptomatic HAV infection vary from mild, anicteric illness to fulminant hepatitis. Among young adults with HAV infection, 76%–97% have symptoms,

and 40%–70% are jaundiced [6]. Children and occasionally young adults can also have inapparent infection, in which symptoms and elevation of ALT levels are absent but seroconversion occurs [7].

Hepatitis A begins with symptoms such as fever, anorexia, nausea, vomiting, diarrhea, myalgia, and malaise. Jaundice, dark-colored urine, or light-colored stools might be present at onset or might follow constitutional symptoms within a few days. Physical findings can include abdominal tenderness, hepatomegaly, or splenomegaly [8]. For most persons, hepatitis A lasts for several weeks. Relapsing symptoms, accompanied by renewed elevation of serum aminotransferase levels, occur in 10% of cases, and relapses might continue for as long as 6 months [9]. The overall case-fatality rate is 0.3%, but it is 1.8% among persons aged ≥50 years. Persons with underlying chronic liver disease have an increased risk of death [10].

Serologic testing is necessary to distinguish hepatitis A from other forms of viral hepatitis. The serologic marker of acute HAV infection, IgM antibody to HAV (IgM anti-HAV), is detectable 5–10 days before the onset of symptoms and usually decreases to undetectable concentrations within 6 months after recovery [10]. However, 13.5% of patients with acute hepatitis A had detectable IgM levels >200 days after illness in one study [11]. The sensitivity and specificity of commercially available IgM anti-HAV tests is >95%, and these tests reliably distinguish hepatitis A from other forms of hepatitis [10]. However, interpretation of a positive IgM anti-HAV test result is problematic for persons with no symptoms or laboratory evidence of acute hepatitis and no epidemiologic link to other cases. Total anti-HAV (IgG plus IgM) is detectable during illness, remains detectable indefinitely, and is a reliable indicator of immunity

to HAV infection. Total anti-HAV can be used to determine susceptibility before giving immunoprophylaxis but is not otherwise useful to clinicians.

No specific treatment for hepatitis A has been shown to be effective. Medications that are metabolized by the liver, including non–prescription medications (such as acetaminophen), should be used with caution or avoided during acute viral hepatitis. Hepatitis A rarely causes fulminant hepatitis, but hospitalization and evaluation for liver transplantation is necessary for patients with signs of liver failure, such as hepatic encephalopathy or coagulopathy.

EPIDEMIOLOGY

HAV is primarily transmitted by the fecal-oral route, either by person-to-person contact or by ingestion of contaminated food or water. Transmission also occurs after exposure to HAV-contaminated blood or blood products, but not by exposure to saliva or urine. Asymptomatic and nonjaundiced HAV-infected persons, especially children, are an important source of HAV transmission [12].

The incidence of hepatitis A in the United States varies in a cyclical pattern, with large increases approximately every 10 years, followed by decreases to less than the previous baseline incidence (figure 1). Incidence rates in the western and southwestern United States have been consistently higher than in other regions of the United States. From 1980 through 2001, an average of 25,000 cases each year were reported to the Centers for Disease Control and Prevention (CDC), but when corrected for underreporting and asymptomatic infections, an estimated average of 263,000 HAV infections occurred per year [13]. On the basis of surveillance data, children aged 5–14 years historically have the highest incidence of hepatitis A [14], although the incidence of HAV infection is probably highest among those <5 years old [15]. Approximately one-third of the United States population has been previously infected with HAV, with higher seroprevalence with increasing age and among persons with lower household incomes or of Hispanic ethnicity [10]. Since 1999, the hepatitis A incidence has decreased to historic lows in the United States [16] (CDC, unpublished data).

Risk factors for infection among reported cases are shown in figure 2 [14]. Personal contact (usually among household contacts or sexual partners) is the most important reported risk factor. Relatively few reported cases (2%–3% per year) are identified through routine surveillance as part of common source outbreaks of disease transmitted by food or water. However, some hepatitis A transmission attributed to personal contact or other risk factors is likely to have been foodborne, occurring when an HAV-infected person contaminated food eaten by others. The proportion of sporadic cases that might be from foodborne sources is unknown but could be considerable; ∼50% of reported patients with hepatitis A do not have an identified source of infection [10, 14].

In developing countries, HAV transmission often is unrecognized, because most residents acquire HAV infection during early childhood. It is interesting to note that, as hygiene improves, the mean age of infected persons increases and the clinical manifestations of hepatitis A are more often recognized, leading to an increase in the hepatitis A incidence (i.e., symptomatic HAV infection), even as the incidence of HAV infection (which is commonly asymptomatic or does not cause jaundice in young children) may be decreasing [17]. Foodborne outbreaks of infection are uncommon in developing countries because of high levels of immunity in the resident population, but foodborne transmission to nonimmune travelers might be an important source of travel-associated hepatitis A.

CHARACTERISTICS OF FOODBORNE TRANSMISSION

HAV contamination of a food product can occur at any point during cultivation, harvesting, processing, distribution, or preparation. Recognizing foodborne transmission using routine surveillance data may be difficult because (1) case patients may have difficulty recalling food histories during the 2–6 weeks before illness, (2) cases may accrue gradually or not be reported, (3) a food item may be focally contaminated, (4) some exposed persons have unrecognized HAV infection, (5) some exposed persons have preexisting immunity (from a previous infection or previous vaccination), (6) persons who acquire infection through contaminated food are not recognized amid an ongoing high incidence in the community, and (7) cases are geographically scattered over several public health jurisdictions.
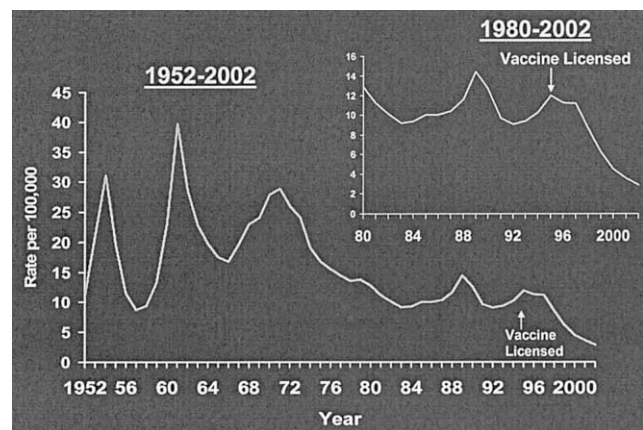

Figure 1. Incidence of hepatitis A, United States, 1952–2002. Data are from the National Notifiable Disease Surveillance System of the Centers for Disease Control and Prevention [16] (Centers for Disease Control and Prevention, unpublished data). Data for 2002 are provisional.
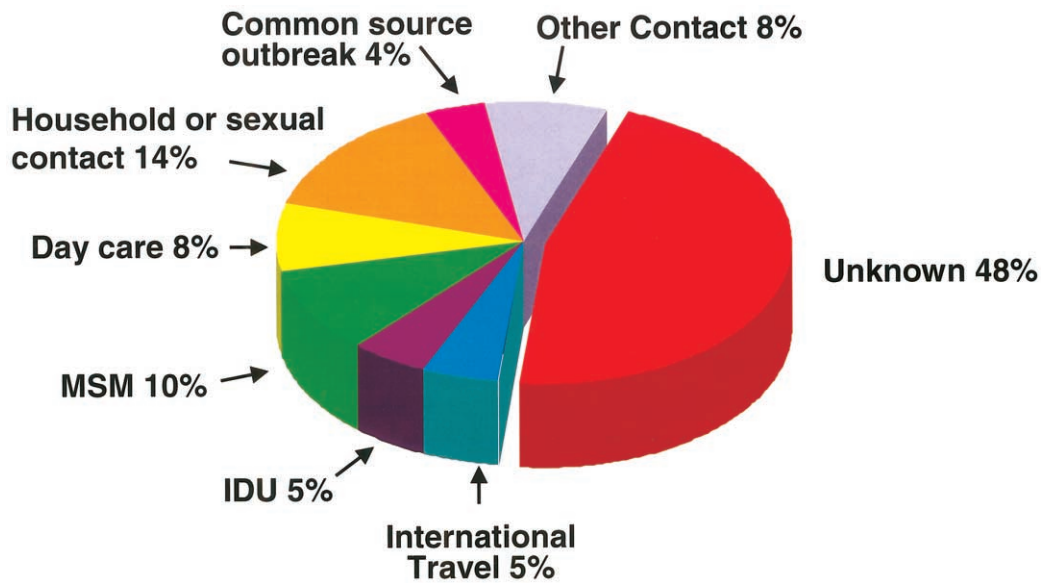
Figure 2. Reported risk factors among persons with hepatitis A, United States, 1990–2000. Data are from the Viral Hepatitis Surveillance Program, Centers for Disease Control and Prevention [14]. IDU, injection drug use; MSM, men who have sex with men.

Transmission due to contamination of food at the point of sale or service. The source of most reported foodborne hepatitis A outbreaks has been HAV-infected food handlers present at the point of sale (such as in a restaurant) or who prepare food for social events (such as a wedding). A single HAV-infected food handler can transmit HAV to dozens or even hundreds of persons and cause a substantial economic burden to public health [18, 19]. The societal cost of a single foodborne outbreak of hepatitis A in Denver involving 43 cases was estimated to be more than $800,000, with >90% of these costs borne by the public health department and attributed to immunoglobulin administration [18]. Table 1 lists selected food handler-associated outbreaks. Common themes of these outbreaks include (1) the presence of an HAV-infected food handler who worked while potentially infectious (2 weeks before to 1 week after symptom onset) and had contact with uncooked food or food after it had been cooked, (2) secondary cases among other food handlers who ate food contaminated by the index case, and (3) relatively low attack rates among exposed patrons.

Food handlers are not at higher risk of hepatitis A because of their occupation. However, food handlers may belong to demographic groups, such as young persons and persons with lower socioeconomic status, who have a higher incidence of hepatitis A than does the rest of the population [10]. Median hourly wages for food service workers are lower than the overall median hourly wage [33], and nearly 2 of 3 food counter attendants are aged 16–19 years [34]. Among 38,881 adults with hepatitis A reported to the CDC during the period of 1992–2000 who had occupational data reported, 8% were identified as food handlers, including 13% of the 3292 persons aged 16–19 years (CDC, unpublished data). The number of patients who were food handlers reflects the number of persons employed in the industry; there were 6.5 million food and beverage serving jobs in 2000 [33], and the industry is the largest private employer in the United States [35].

Most food handlers with hepatitis A do not transmit HAV to consumers or restaurant patrons, as determined on the basis of surveillance data, but many hundreds of restaurant workers have hepatitis A every year. Evaluating HAV-infected food handlers is a common task for many public health departments, and assessing the need for postexposure immunoprophylaxis and implementing control measures consumes considerable time and resources at the state and local health department level. In a retrospective analysis of HAV-infected food handler investigations conducted during 1992–2000 in Seattle/King County, Washington, and the state of Massachusetts, 230 HAV-infected food handlers were identified. Of these, 140 (59%) had worked during a time when they were potentially infectious, but only 12 (7%) were evaluated as representing an infection risk to those who ate food they had prepared; an average of 377 doses of immunoglobulin were dispensed by public health personnel in each episode. Coworkers of the infected food handlers were given immunoglobulin in 121 investigations (51%; CDC, unpublished data).

Transmission due to contamination of food during growing, harvesting, processing, or distribution. Hepatitis A outbreaks have been also associated with consumption of fresh produce contaminated with HAV during cultivation, harvesting, processing, or distribution (table 2). Outbreaks involving

Table 1. Characteristics of selected published foodborne hepatitis A outbreaks in the United States associated with an infected food handler.

Reference	Year	Location	No. of infected persons	Contaminated food	Symptoms while working	Reason Ig not given[a]	Coworkers infected	Identified hepatitis A risk factor of food handler
20	1968	Bakery	61	Pastry icing	Vomiting, dark urine	Unknown	None	Not reported
21	1974	Cafeteria	133	Salad, fresh fruit	Diarrhea, vomiting	Food handling not initially reported	"Several"	Personal contact with another case patient
22	1973	Cafeteria	44	Sandwiches	None	Index case patient did not seek medical care	4	Household contact with another case patient
23	1974	Restaurant	107	Sandwiches	Malaise, vomiting	Diagnosis delayed	3	Household contact with another case patient
24	1975	Cafeteria	22	Multiple foods	Fatigue, nausea, vomiting	Unknown	5	Unknown
24	1975	Restaurant	33	Salads	None	Unknown	13	Unknown
25	1979	Cafeteria	30	Sandwiches	Back pain, "kidney infection"	Index case patient did not seek medical care	None	Not reported
26	1981	Cafeteria	37	Sandwiches	Jaundice, lethargy	Not reported to health department	None	Not reported
27	1986	Restaurant	103	Salad	Vomiting, dark urine	Index case patient did not seek medical care	5	MSM
28	1988	Restaurant	54	Multiple foods	Diarrhea	Unknown	None	Injection drug user
29	1990	Restaurant	110	Salads	Not reported	Food handling not initially reported	4	Not reported
29	1991	2 Restaurants	228	Sandwiches	None	Assessed as low risk	None	Not reported
29	1992	Restaurant	11	Sandwiches	Nausea, vomiting, diarrhea	Delayed diagnosis	None	Not reported
18	1992	Caterer	43	Multiple foods	None	Assessed as low risk	9	Not reported
30	1994	Caterer	91	Multiple foods	Gastroenteritis	Assessed as low risk	None	Not reported
31	1994	Bakery	64	Glazed baked goods	Diarrhea	Index case patient did not seek medical care	9	Not reported
32	2001	Restaurant	43	Sandwiches	None; food handler had a colostomy	Assessed as low risk	None	Not reported

NOTE. IG, immunoglobulin; MSM, man who has sex with men.

[a] To exposed persons within 2 weeks of last exposure.

Table 2. Characteristics of selected published foodborne hepatitis A outbreaks associated with produce contaminated during growing, harvesting, or processing.

Reference	Year	No. of infected persons	Implicated food	No. and location of sites	Source of implicated food
36	1983	24	Frozen raspberries	Aberdeen, United Kingdom	United Kingdom
37	1988	202	Iceberg lettuce	3 Restaurants in Kentucky	Unknown, probably Mexico
38	1990	35	Frozen strawberries	2 Schools in Georgia and Montana	California
39	1997	262	Frozen strawberries	5 States	Mexico
40	1998	43	Green onions	1 Restaurant in Ohio	Mexico or California
41	2000	31	Green onions or tomatoes	2 Restaurants in Kentucky and Florida	Green onions: Mexico or California; tomatoes: unknown
42	2003	>700	Green onions	4 States	Mexico

a food item that was contaminated before distribution are particularly challenging to identify and might be widely distributed geographically. For example, HAV-contaminated frozen strawberries were implicated as the source of an outbreak involving at least 262 persons in 5 states [39]. Low attack rates are common, probably because contamination is only found in a small portion of the distributed food. Additional cases might be prevented by rapid epidemiologic identification of the contaminated item, traceback, and product recall.

Experimental contamination studies suggest that the physical characteristics of some produce items might facilitate transmission. Lettuce, carrots, and fennel were immersed in HAV-contaminated water for 20 min followed by refrigerated storage; infectious HAV was recovered from lettuce for 9 days after immersion, but it was recovered from carrots and fennel for only 4–7 days. Washing reduced but did not eliminate detectable HAV [43]. No investigation to date has determined the point in cultivation, harvesting, or processing at which contamination occurs. Produce might be contaminated by the hands of HAV-infected workers or children in the field, by contact with HAV-contaminated water during irrigation or rinsing after picking, or during the processing steps leading to packaging. Removal of stems by workers in the field during picking might be a potential mechanism for strawberry contamination [38]. Green onions require extensive handling during harvesting and preparation for packing and receive no further processing until they reach the restaurant or the consumer's home, where they are often served raw or partially cooked. Recent large outbreaks associated with imported green onions that were contaminated before arrival in restaurants indicate a need for a better understanding of how contamination of fresh produce occurs and why certain produce items (e.g., strawberries and green onions) seem particularly prone to contamination [42].

HAV-contaminated shellfish have been the source of food-borne outbreaks of hepatitis A, including several outbreaks involving many thousands of cases (table 3). Although reports of shellfish-related hepatitis A outbreaks continue to occur in some other countries, none have been reported recently in the United States. Factors contributing to contamination in shellfish-related outbreaks may include inappropriate or illegal shellfish harvesting near known sources of sewage, inappropriate discharge of sewage from fishing boats or oil platforms near shellfish beds, and use of fecally contaminated water to immerse harvested live shellfish. Identification of HAV in shellfish taken from approved areas in the United States has also been reported.

Transmission due to exposure to contaminated water. Waterborne outbreaks of hepatitis A are unusual in developed countries. Water treatment processes and dilution within municipal water systems are apparently sufficient to render HAV noninfectious, although no studies have demonstrated which specific treatment processes are the most effective. Outbreaks of hepatitis A among persons who use small private or community wells or swimming pools have been reported, and contamination by adjacent septic systems has been implicated as the source of contamination [49–53]. Although the potential for hepatitis A outbreaks after flooding-related sewage contamination of potable water sources is recognized, no such incidents have been reported in the United States in several decades.

DETERMINING THE BURDEN OF FOODBORNE HEPATITIS A IN THE UNITED STATES

Approximately 50% of persons with hepatitis A in the United States do not have an identified risk factor. Molecular epidemiologic techniques hold promise for identifying unsuspected links between patients with foodborne hepatitis A. RNA sequences from serum specimens obtained from HAV-infected persons can be amplified even after clinical recovery [4]. By comparing viral sequences, previously unrecognized links be-

Table 3. Characteristics of selected foodborne hepatitis A outbreaks associated with shellfish.

Reference	Year	No. of infected persons	Implicated food	Location of cases	Source of implicated food	Suspected cause of contamination
[44]	1973	278	Oysters	Texas, Georgia	Louisiana	Untreated sewage from oil platforms and fishing boats
[45]	1981	132	Cockles	19 Boroughs in the United Kingdom	United Kingdom	Sewage discharged near shellfish beds
[46]	1988	61	Oysters	Alabama, Georgia, Florida, Tennessee, Hawaii	Florida	Harvest from unapproved oyster beds near sewage treatment plant
[47]	1988	292,301	Raw clams	Shanghai, China	Qi-Dong County, China	Untreated sewage discharged near shellfish beds
[19]	1996	5889	Mussels and clams	Puglia	Italy	Unknown
[48]	1997	444	Oysters	New South Wales, Australia	Wallis Lake, Australia	Untreated sewage
[49]	1999	184	Coquina clams	Spain	Peru	Unknown

tween cases can be inferred. In a 1997 outbreak among Michigan and Maine schoolchildren that was linked to strawberries, others with hepatitis A who had eaten strawberries from the same processor were identified in Wisconsin, Arizona, and Louisiana; viral sequences from all of these cases were identical to each other and were different from viral sequences obtained from non–outbreak-related cases [39]. Determining whether hepatitis A acquired from contaminated food or water is an important contributor to the burden of hepatitis A in the United States will require more widespread application of molecular epidemiologic techniques, as well as obtaining more-detailed exposure histories during case investigations.

PREVENTION OF HEPATITIS A

Preexposure prophylaxis. Hepatitis A is the only common vaccine-preventable foodborne disease in the United States. Hepatitis A vaccine is an inactivated preparation of a cell-culture adapted virus and was licensed in 1995 for persons aged ≥2 years. More than 95% of adults and children have seroconversion after a single dose of hepatitis A vaccine, and long-term protection is provided by a second (booster) dose given ≥6 months later. Protective concentrations of anti-HAV are measurable in 54%–62% of persons by 2 weeks and in ≥90% by 4 weeks after receipt of a single dose of vaccine. The vaccine's efficacy is 94%–100%, and protection is likely to last for ≥20 years after vaccination; booster doses after the primary 2-dose series are not currently recommended [10]. Recent vaccination may confuse interpretation of diagnostic test results for hepatitis A, because IgM anti-HAV can be detected in some persons shortly after vaccination [55]. However, when tested 1 month after vaccination, <1% of vaccinated persons had detectable IgM anti-HAV [56].

Hepatitis A vaccination is recommended for people at higher risk for hepatitis A, including men who have sex with men and illicit drug users (regardless of whether they inject the drugs or not). Because recent travel to countries where HAV infection is endemic is a commonly identified risk factor among patients in the United States, persons planning travel to developing countries for any reason, frequency, or duration who can receive the first dose of vaccine at least 2–4 weeks before departure should also be vaccinated [10, 57]. Persons with chronic liver disease are at risk for more severe hepatitis A and should receive vaccination also. Routine childhood vaccination is recommended in states and communities with a consistently high incidence of hepatitis A [10].

Routine vaccination of all food handlers is not recommended, because their profession does not put them at higher risk for infection. However, local regulations mandating proof of vaccination for food handlers or offering tax credits for food service operators who provide hepatitis A vaccine to employees have been implemented in some areas. One economic analysis concluded that routine vaccination of all food handlers would not be economical from a societal or restaurant owner's perspective. Costs in the economic model were driven by the turnover rate of employees and the small percentage of hepatitis A cases that are attributable to infected food service workers [58]. Another analysis concluded that vaccination of 100,000 food handlers in the 10 states with the highest incidence of hepatitis A would cost $13,969 per year-of-life saved [59].

Employers concerned about reducing the risk of hepatitis A among employees should focus on providing hepatitis A vaccination for those persons who have risk factors for infection, including men who have sex with men, illicit drug users, and persons who plan to travel to developing countries [10, 57]. Food handlers aged <19 years who live in a state or community where routine childhood vaccination is recommended have both an indication for vaccine [10] and a potential source for reimbursement for vaccination and for some administrative costs (i.e., the Vaccine for Children Fund).

Immunoglobulin provides short-term (1–2-month) protection from hepatitis A. Immunoglobulin is a sterile preparation of concentrated antibodies (immunoglobulins) made from pooled human plasma processed in a way that inactivates viruses. The intramuscular preparation (0.02 mL/kg) is often used in persons planning to travel within 2–4 weeks and who require immediate protection or for those with contraindications for vaccination [10, 57]. Immunoglobulin is also recommended for travelers aged <2 years, for whom the vaccine is not licensed. Although children of this age usually have mild infection, they commonly serve as a source of infection for contacts, and they occasionally have severe illness themselves.

Postexposure prophylaxis. Postexposure prophylaxis with immunoglobulin is >85% effective in preventing hepatitis A if administered within 2 weeks after exposure to HAV, but the efficacy is highest when administered early in the incubation period [60]. There are several specific circumstances in which the use of postexposure prophylaxis is indicated, including use for nonimmune persons who have had (1) household or sexual contact with an HAV-infected person during a time when the HAV-infected person was likely to be infectious (i.e., 2 weeks before to 1 week after onset of illness), and (2) whose last contact was within the previous 2 weeks. Postexposure prophylaxis consists of a single intramuscular dose of immunoglobulin (0.02 mL/kg) [10]. Persons who received a dose of hepatitis A vaccine ≥1 month previously or who have a history of laboratory-confirmed HAV infection should be considered immune and do not require immunoglobulin. Immunoglobulin is not necessary for persons whose only exposure to a person with hepatitis A occurred >1 week after the onset of jaundice. Food service workers with hepatitis A can expose other food service workers, and immunoglobulin should be given to all other food service workers in the same establishment who do not have proof of previous vaccination or HAV infection.

CDC guidelines recommend that postexposure prophylaxis also be considered for persons who consume food prepared by an infected food handler if (1) the food handler had contact with food that was not cooked after contact, (2) the food handler had diarrhea or poor hygienic practices during the time when he or she was likely to be infectious, and (3) patrons can be identified and treated within 2 weeks after their last exposure [10, 61]. An algorithm for determining whether immunoprophylaxis is needed has been published (figure 3) [61]. Although this algorithm is a useful framework for assessing the risk of transmission from an infected food handler, postexposure prophylaxis decisions are still largely based on retrospective hygiene assessments and other subjective information obtained during the case interview, as well as on the judgment and experience of public health officials. Interviews should include detailed, open-ended questions about job duties, work dates, clinical symptoms, and hygiene during the period of infectivity. Interviews with supervisors and coworkers and an inspection of restrooms and food preparation areas are also recommended. Opportunities for postexposure prophylaxis are often missed, either because the infected food handler did not receive a diagnosis of HAV infection until after transmission to patrons had occurred, the food handler with hepatitis A was not reported to the local public health authorities, or reported food handling practices incorrectly indicated that the risk of transmission to patrons was low. Postexposure prophylaxis should not be administered to exposed persons after cases have begun to occur, because the 2-week period during which immunoglobulin is effective will have passed, unless other infected food handlers with later onsets have been identified.

Hepatitis A vaccine has also been used for postexposure prophylaxis [62]. However, the effectiveness of postexposure prophylaxis using hepatitis A vaccine has not been directly compared with immunoglobulin in a controlled clinical trial, and immunoglobulin remains the recommended choice for postexposure prophylaxis in the United States [10]. Hepatitis A vaccine can be given at the same time (but in a different anatomic site) as immunoglobulin, and exposed persons who have an indication for vaccination should receive both [10].

HYGIENE PRACTICES

The minimum infectious dose required for HAV infection in humans is unknown. In primate studies, HAV can remain infectious after 1 month on environmental surfaces at ambient temperatures [63], and it is more resistant than poliovirus (another picornavirus) to degradation over time while on environmental surfaces [64]. Heating foods to 85°C (>185°F) for 1 min or disinfection with a 1:100 dilution of household bleach in water or cleaning solutions containing quaternary ammonium and/or HCl (including concentrations found in many toilet cleaners) is effective in inactivating HAV. HAV is resistant to disinfection by some organic solvents and by a pH as low as 3 [65].

No specific food handler hygiene practice has been shown to reduce the likelihood of transmission. Experimental deposition of fecally suspended HAV onto hands indicates that infectious HAV remains present for ≥4 h after application [66]. In experimental settings, water rinsing alone reduces the amount of HAV that is transferred to lettuce by 10- to 100-fold [67].

Hygiene training for food handlers should include practical advice about the techniques of hand washing and education about the need to seek medical attention for postexposure prophylaxis after contact with a person with hepatitis A. Reducing bare hand contact with foods that are not subsequently cooked is also a reasonable preventative measure. Employers should
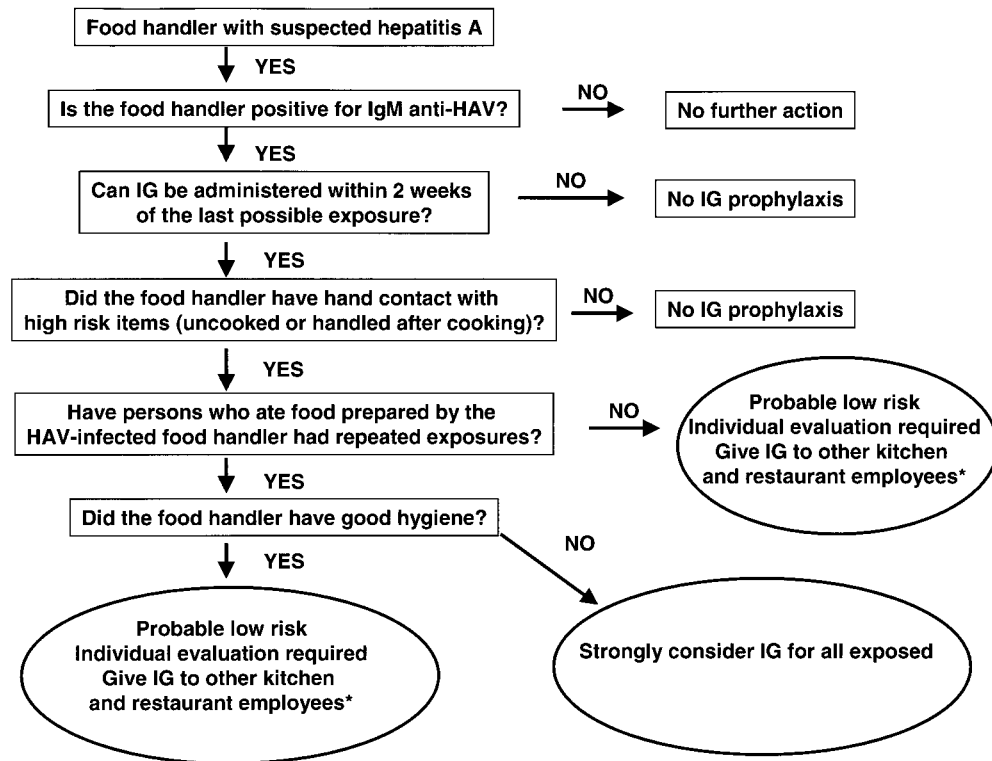
Figure 3. Algorithm for determining need for immunoprophylaxis after exposure to food prepared by a food handler with hepatitis A. Consider hepatitis A vaccine in addition to immunoglobulin (IG) for those with other risk factors for hepatitis A. Hygiene assessments are subjective; a visit to the food handling area and interviews with the infected food handler, coworkers, and supervisors are often helpful. Factors to consider include the food handler's self-assessment, assessments obtained from supervisors or coworkers, whether the food handler had bowel movements (especially diarrhea) while at work, presence of medical conditions that might make hygiene more difficult to maintain, glove use, availability of functioning hand washing facilities, hygiene training, and previous assessments of sanitation practices in the facility that employs the infected food handler. Adapted from [61]. anti-HAV, antibody to hepatitis A virus.

provide access to hand washing stations and encourage ill food handlers to seek medical attention and to stay out of the workplace. Exclusion from duties that involve contact with food for at least 1–2 weeks after the onset of jaundice or until symptoms resolve is reasonable. Asymptomatic food handlers who are IgM anti-HAV positive are sometimes identified during investigations and measurements of ALT levels, in combination with likely dates of exposure, might be used to estimate whether the food handler has had recent infection and is potentially still capable of transmission. However, the validity of this approach is unknown.

Providing sanitary facilities for field workers and discouraging the presence of children in areas where food is harvested reduces the potential for contamination of food during harvesting or processing. Chlorinated water or water from a source not likely to be contaminated by sewage should be used for rinsing produce or ice used for packing.

DISINFECTION OF POTENTIALLY CONTAMINATED FOODS

Development of disinfection procedures for produce or shellfish has been hampered by the technical difficulties involved with detection of infectious HAV in food. Cell culture assays can indicate the presence of infectious HAV, but they are expensive and require several days to perform. Wild-type virus is not easily detectable, because it usually is not cytopathic. RT-PCR protocols can detect viral particles more rapidly but cannot readily distinguish infectious virus from noninfectious HAV RNA, and the variety of PCR inhibitors present in foods requires the development of food-specific protocols. Specific methods to detect enteric viruses, such as HAV, are necessary, because water and shellfish with low coliform counts (commonly used as a measure of fecal contamination) have been shown to contain viable HAV [68], and outbreaks of hepatitis A associated with shellfish harvested from waters where fecal

coliform counts were within accepted limits have been reported [69]. Despite these challenges, methods are being developed to detect HAV on some types of produce [43, 70], in shellfish [68, 71], and in water [54].

The effectiveness of various disinfection methods in reducing HAV contamination of fresh fruits and vegetables is an area of active investigation. Preliminary results indicate that disinfection modalities that are potentially applicable to produce, including chlorinated water [72], hydrostatic pressure [73], and heat [74], are effective in reducing or eliminating HAV infectivity; however, adapting these techniques for use on commercially distributed produce will require further refinement.

Other than thorough cooking, no reliable disinfection method for shellfish exists. Shellfish are typically cooked until they open, which may occur at temperatures as low as 70°C [75]. Steaming or boiling shellfish still in the shell for <2 min may not fully inactivate HAV [76]. Shellfish have HAV concentrations as much as 100-fold that of surrounding water [77], and HAV has been detected in clams, mussels, and oysters harvested from areas linked to hepatitis A outbreaks [43, 47]. Depuration (placing harvested live shellfish in clean water to promote purging of gastrointestinal contents) for up to 1 week reduces but does not eliminate HAV that has been taken up by shellfish [77]. If HAV-contaminated water is used during depuration, it may even introduce HAV into previously uncontaminated shellfish.

Reducing HAV contamination of foods should be possible using approaches, such as Hazard Analysis and Critical Control Point (HACCP) systems, similar to those recommended for reducing contamination by other foodborne pathogens [78]. US Food and Drug Administration (FDA) guidance on improving food safety can be found on the FDA Web site (http://www.foodsafety.gov/~dms/fs-toc.html#specific) [79]. Defining specific critical points for hepatitis A contamination will require a better understanding of how and when contamination occurs. The efficacy of various chemicals or washing processes in disinfecting fresh fruits, vegetables, and shellfish will have to be considered in the context of the need to preserve the marketability and quality (e.g., consistency, taste, and odor) of products.

CONCLUSION

Reducing foodborne transmission of hepatitis A can be achieved by improving food production and food handler hygiene and providing preexposure prophylaxis to persons at risk for infection. Food handlers acquire HAV infection from others within their communities, and reducing foodborne transmission of HAV will ultimately be achieved through routine vaccination of persons at risk for HAV infection within these communities.

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