Chapter 3: Hepatitis A

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I. Disease Description

Hepatitis A is caused by infection with hepatitis A virus (HAV), a non-enveloped RNA virus that is classified as a picornavirus. HAV was first identified by immune electron microscopy in 1973 and initially replicated in mammalian cell culture in 1979.[1,2] Humans are the only natural host, although several nonhuman primate species have been infected in laboratory settings.[3–6] Depending on conditions, HAV can be stable in the environment for months.[7] The virus is relatively stable at low pH levels and freezing to moderate temperatures, but can be inactivated by high temperature (185°F [85°C] or higher for one minute) or through disinfection of surfaces with a 1:100 dilution of sodium hypochlorite in water.[8,9]

Transmission and symptomology

Hepatitis A is typically acquired through fecal-oral transmission, either from direct person-to-person contact or consumption of contaminated food or water. HAV replicates in the liver, is excreted in bile, and is shed in the stool of infected people in high concentrations 2–3 weeks before and 1 week after onset of illness.[10] Peak infectivity occurs during the 2 weeks prior to onset of clinical signs and symptoms (jaundice or elevated liver enzymes). Most persons cease to be infectious 1 week after jaundice appears.[11] Although virus is present in serum of an infected person, its concentration is several orders of magnitude less than in feces. Infected children and infants may excrete virus longer than adults.[12,13]

The mean incubation period of hepatitis A is approximately 28 days (range 15–50 days). Symptomatic hepatitis A infection is clinically indistinguishable from other types of acute viral hepatitis, but is usually mild and self-limited. Fulminant hepatic failure occurs in less than 1% of cases.[14] The likelihood of symptomatic illness from HAV infection is directly related to age. In children younger than 6 years of age, most (70%) infections are asymptomatic.[15] In older children and adults, infection is usually symptomatic, with jaundice occurring in more than 70% of patients.[15,16] Clinical manifestations vary, but may include the abrupt onset of fever, malaise, anorexia, nausea, and abdominal discomfort, followed within a few days to a week by dark urine, pale stools, and jaundice.[15] Clinical illness usually resolves within 2–3 months (85% of cases), and complete recovery is seen within 6 months for nearly all cases.[17] However, up to 10% of persons with hepatitis A experience a biochemical and/or clinical relapse during the 6 months after acute illness.[18] Virus may be excreted in stool during a relapse.[19] Consequently, persons experiencing relapsing hepatitis A should be considered infectious. There is no specific treatment for hepatitis A. Disease is usually self-limiting and treatment and management of HAV infection are supportive. HAV infection does not result in chronic infection or chronic liver disease; however, HAV infection can complicate chronic liver disease.

II. Background

Prevalence

Population-based seroprevalence surveys play a critical role in the development of vaccination policies by supplementing data systems that monitor disease incidence, vaccination coverage, and vaccine adverse events. In the United States, seroprevalence is monitored by the National Health and Nutrition Examination Survey (NHANES), which is conducted by the CDC National Center for Health Statistics and obtains nationally representative data on the health and nutritional status of the non-institutionalized, civilian U.S. population. Before the availability of vaccine in 1995, seroprevalence of antibody to hepatitis A virus (anti-HAV) in the population solely reflected prior infection. Currently, seroprevalence can reflect immunity due to either previous infection or to vaccination. In US-born persons ≥2 years of age, the overall anti-HAV prevalence was 27.5% (95% confidence interval (CI) 25.8–29.2%) in 1999–2006

and increased to 31.2% (95% CI 29.5–33.0%) in 2007–2012.[20] However, in US-born persons ≥20 years of age, there was a significant decrease among adults in the overall age-adjusted prevalence of anti-HAV from 1999–2006 (29.5%) to 2007–2012 (24.2%).[20] These anti-HAV prevalence estimates suggest that a substantial proportion of the U.S. adult population remains susceptible to hepatitis A at ages when risk of morbidity and mortality from HAV infection is highest. Among US-born adults ≥20 years of age, older age, race/ethnicity other than non-Hispanic white, lower income, less education, and any health insurance coverage were significantly associated with anti-HAV positivity during 2007–2012.[20] Lower prevalence of immunity among adults increases the possibility of outbreaks among susceptible population clusters of adults at risk; examples of these occurrences are well documented in the European Union.[21] Additionally, a 2013 hepatitis A outbreak in the United States associated with frozen pomegranate arils imported from Turkey involved 165 identified case-patients; of these, 154 (93%) were adults 18 years of age or older.[22] Prevention of secondary transmission of HAV in the United States comes at an enormous public health cost, in large part due to the number of persons offered prophylaxis.[23]

Risk factors

The distribution of risk behaviors and exposures among hepatitis A cases has changed dramatically in the United States since implementation of hepatitis A vaccination. During 1983–1995, data from the Sentinel Counties Study identified international travel as a minor source of infection (4%) among hepatitis A cases.[24] In sites conducting enhanced hepatitis surveillance during 2005–2007, however, the most frequent potential source of infection (46%) among reported hepatitis A cases was travel outside the United States and Canada. These cases mostly reflected persons who traveled, but also included some persons who were exposed to a traveler without having traveled themselves.[25] Although data are limited, current risk factors are similar today. In 2014, only 7% of hepatitis A cases reported in the United States had an identified risk factor; the remaining 93% of cases either had no identified risk factors or were missing data on risk factors entirely.[26] Among cases with an identified risk factor, international travel continues to be the most frequently identified risk factor.[26] Other potential sources of infection identified among hepatitis A cases in 2014 included contact with a hepatitis A patient, being an employee or child in a day care center, injection drug use, being a man who has sex with men, or exposure during a common-source (food- or water-borne) outbreak. Food-borne hepatitis A outbreaks are of increasing concern globally.[27]

III. Importance of Rapid Identification

Rapid identification and prompt reporting of cases of hepatitis A are important because post-exposure prophylaxis, administered within 2 weeks after exposure, is highly efficacious and can prevent development of symptomatic illness in exposed persons and prevent further transmission to other persons. Contacts of infected persons are eligible to receive post-exposure prophylaxis within 2 weeks of exposure; the efficacy of hepatitis A post-exposure prophylaxis has not been established beyond this timeframe.

IV. Importance of Surveillance

The main goals of hepatitis A surveillance are to:

- detect and provide data to control outbreaks;
- identify contacts of case-patients who require post-exposure prophylaxis;
- characterize changes in the epidemiology of infected populations and risk factors; and
- guide vaccination policies and other prevention efforts.

Surveillance depends heavily on laboratory-initiated reporting of positive markers of hepatitis A. Persons with positive test results are investigated using traditional, notifiable diseases methods in most health departments in the United States. Hepatitis A case investigations can be labor intensive. As a result, providers should be discouraged from using IgM anti-HAV as a screening tool or as part of testing panels in workups of non-acute liver function abnormalities, since IgM screening of non-acute, abnormal liver function tests may result in a high percentage of false-positive IgM results.[28] Adherence to this practice will limit the need for health departments to conduct investigations of persons who are unlikely to have acute HAV infection.

Routine surveillance can supplement case notifications using seroprevalence surveys and administrative data. Additionally, electronic medical records, with coded clinical and laboratory criteria, hold promise as a means to improve completeness and timeliness of symptomatic hepatitis A infection identification.

V. Disease Reduction Goals

Healthy People 2020 disease reduction goals have been established for achieving the prevention of HAV transmission in the United States.[29]

IID–23: Reduce hepatitis A.

Baseline: 1.0 cases of hepatitis A virus per 100,000 population were reported in 2007.

Target: Reduce the rate of incident hepatitis A to 0.3 cases per 100,000 population.

VI. Case Definition

The following surveillance case definition for hepatitis A was adopted by the Council of State and Territorial Epidemiologists (CSTE) in 2012. Current and previous hepatitis A case definitions are available at: https://wwwn.cdc.gov/nndss/conditions/hepatitis-a-acute/case-definition/2012/

Clinical description

An acute illness with a discrete onset of any sign or symptom consistent with acute viral hepatitis (e.g., fever, headache, malaise, anorexia, nausea, vomiting, diarrhea, and abdominal pain), and either a) jaundice, or b) elevated serum alanine aminotransferase (ALT) or aspartate aminotransferase (AST) levels.

Laboratory criterion for diagnosis:

Immunoglobulin M (IgM) antibody to hepatitis A virus (anti-HAV) positive

Case classification

Confirmed:

- A case that meets the clinical case definition and is laboratory confirmed, OR
- A case that meets the clinical case definition and occurs in a person who has an epidemiologic link with a person who has laboratory-confirmed hepatitis A (i.e., household or sexual contact with an infected person during the 15–50 days before the onset of symptoms).

VII. Laboratory Testing

Specimen collection

Specimen collection and shipping are important steps in obtaining laboratory diagnosis or disease confirmation. Guidelines have been published for specimen collection and handling of microbiologic agents. Information is also available on using CDC laboratories as support for reference and disease surveillance; this includes:

- a central website (https://www.cdc.gov/laboratory/specimen-submission/index.html) for requesting lab testing;
- the form required for submitting specimens (https://www.cdc.gov/laboratory/specimen-submission/form.html) to CDC (See Appendix 23, Form # CDC 50.34};
- information on general requirements for shipment of etiologic agents (Appendix 24 https://www.cdc.gov/vaccines/pubs/surv-manual/appx/appendix24-etiologic-agent.pdf); and
- the CDC Infectious Diseases Laboratories Test Directory (https://www.cdc.gov/laboratory/specimen-submission/list.html), which provides an online test directory that contains not only a list of orderable tests for that institution, but also detailed information on appropriate specimen types, collection methods, specimen volume, and points of contact.

Serologic testing

Serologic testing is required to distinguish hepatitis A from other types of viral hepatitis, since clinical or epidemiologic features overlap. Virtually all patients with hepatitis A have detectable IgM anti-HAV. Acute HAV infection is confirmed during the acute or early convalescent phase of infection by the presence of IgM anti-HAV in serum. IgM generally becomes detectable 5–10 days before the onset of symptoms and can persist for up to 6 months. IgG anti-HAV appears in the convalescent phase of infection, remains present in serum for the lifetime of the person, and confers enduring protection against disease.

The antibody test for total anti-HAV measures both IgG anti-HAV and IgM anti-HAV. Persons who are total anti-HAV positive and IgM anti-HAV negative are considered immune, whether from past infection or vaccination history. Tests for IgG anti-HAV are also available.

CDC laboratory special studies

Molecular epidemiologic methods have been useful in understanding HAV transmission within networks of persons with similar risk factors. When applied in combination with conventional epidemiologic methods, HAV sequencing has also been useful in the investigation of outbreaks and determining transmission links. However, for routine surveillance purposes, detection of serologic markers—total anti-HAV or IgG anti-HAV and IgM anti-HAV—is sufficient actionable information.

Specimens collected as part of enhanced hepatitis A surveillance in the United States from 2007 through 2013 were sequenced; 472 (62.7%) of 753 available specimens were HAV RNA positive by PCR. Additional specimens from the 2013–2016 Food Safety Project A13FBM were sequenced: 98 (26.3%) of 373 available specimens were HAV RNA positive. HAV genotypes among these 570 case specimens were: IA (83.2%), IB (16.0%), and IIIA (0.9%) (CDC Division of Viral Hepatitis Laboratory, unpublished data).

Providers with questions about molecular virology methods should consult with their state health department and the CDC Division of Viral Hepatitis.

VIII. Reporting and Case Notification

Case reporting within a jurisdiction

Each state and territory has regulations and laws governing the reporting of diseases and conditions of public health importance.[30] These regulations and laws list the diseases that are to be reported, and describe those persons or institutions responsible for reporting, such as health-care providers, hospitals, laboratories, schools, daycare and childcare facilities, and other institutions. Detailed information on reportable conditions in each state is available through CSTE.[31] The Viral Hepatitis Case Report is included as Appendix 6 to serve as a guide for data collection during investigation of reported cases.

Case notification to CDC

Hepatitis A became nationally notifiable as a distinct entity in 1966. State health departments transmit hepatitis A case reports weekly to the National Notifiable Diseases Surveillance System (NNDSS) at CDC. This surveillance system monitors basic demographic information (excluding personal identifiers)—age, race/ethnicity, sex, date of onset, date of report, and county of residence of individual cases in addition to disease-specific information. At CDC, the Division of Viral Hepatitis uses the hepatitis A case reports submitted to NNDSS to develop and disseminate an annual report available at https://www.cdc.gov/hepatitis/statistics/index.htm

Incidence rates of hepatitis A disease were high in the 1960s and 1970s (>12 cases per 100,000 population). The last substantial peak in reported hepatitis A rates was in 1995. Since the introduction of effective vaccines in the United States in 1995, and due in large part to progressively expansive recommendations from the Advisory Committee on Immunization Practices (ACIP) issued between 1996 and 2006 (culminating in routine childhood vaccination nationwide), hepatitis A rates in the United States have declined by 98%.[26, 32] In 2014, the overall incidence rate was 0.4 cases per 100,000 population (1,239 cases).[26] In 2014, the hepatitis A incidence rate was highest for persons aged 20–29 years (0.55 cases per 100,000 population); the lowest age group incidence rate was among children aged 0–9 years (0.10 cases per 100,000 population).[26]

The CDC/CSTE hepatitis A surveillance case definition entails a combination of clinical and laboratory criteria. Thus, asymptomatic cases are not notifiable (see case definition above). To estimate all new infections, case reports are adjusted for the proportion of asymptomatic cases and surveillance underreporting. In 2014 there were an estimated 2,500 new hepatitis A infections.[26] Notifications for confirmed cases of acute hepatitis A should be sent to CDC using event code 10110 in the NNDSS.[33] Case notification should not be delayed because of incomplete information or lack of confirmation. The state in which the patient resides at the time of diagnosis should submit the case notification to CDC.

IX. Vaccination

Hepatitis A vaccines were first licensed in the United States in 1995 and 1996. Shortly thereafter, ACIP made recommendations for routine vaccination of children 2–18 years of age who lived in communities with the highest rates of infection and disease.[34] By 1999, there was epidemiologic evidence that the strategy had a limited impact on national disease incidence; thus, in 1999 ACIP recommended routine vaccination for children living in 11 (mostly western) states with average incidence rates that were at least twice the 1987–1997 national average (i.e., ≥20 cases per 100,000 population).[35] In an additional 6 states, where average incidence rates were greater than the national average but less than twice that value (i.e., 10–19 cases per 100,000 population), ACIP recommended consideration of routine vaccination of children.[35] In August 2005, the youngest age for which hepatitis A vaccine was licensed was lowered from 24 months to 12 months, and in May 2006 ACIP recommended routine hepatitis A vaccination of all children 12–23 months of age.[36] The expanded vaccination recommendations had a dramatic impact: the estimated annual average number of incident hepatitis A cases was 117,333 in the decade prior to hepatitis A vaccine licensure (1986–1995), and was 2,500 in 2014, representing a 98% decrease in incident hepatitis A cases during this timeframe.[26,30] The duration of protection from hepatitis A vaccine is estimated to be 40 years or longer.[37]

HAVRIX® is an inactivated vaccine that is available in 2 formulations: pediatric (720 ELISA units [EL.U.] per 0.5 mL dose) and adult (1,440 EL.U. per 1.0 mL dose; Table 1). Children 1 through 18 years of age should receive a single primary dose of the pediatric formulation followed by a booster dose 6 to 12 months later. Adults 19 years of age and older receive 1 dose of the adult formulation followed by a booster 6 to 12 months later. The vaccine should be administered intramuscularly into the deltoid muscle. A needle length appropriate for the vaccinee's age and size (minimum of 1 inch) should be used.

VAQTA® is an inactivated vaccine that is available in pediatric and adult formulations and quantified in units (U) of antigen (Table 2). Children 1 through 18 years of age should receive 1 dose of pediatric formulation (25 U per dose) with a booster dose 6 to 18 months later. Adults 19 years of age and older should receive 1 dose of adult formulation (50 U per dose) with a booster dose 6 to 18 months after the first dose. The vaccine should be administered intramuscularly into the deltoid muscle. A needle length appropriate for the vaccinee's age and size should be used (minimum of 1 inch).

The hepatitis A component of TWINRIX® (an inactivated vaccine) consists of 720 EL. U. of hepatitis A antigen in a 1.0 mL dose (Table 3). It is approved for vaccination of persons ≥18 years of age in 2 schedules: a 3-dose schedule (0, 1, and 6 months) and alternate 4-dose schedule (0, 7, and 21–30 days, followed by a dose at 12 months). The alternative 4-dose schedule can be used when vaccination with TWINRIX® or single-antigen HAV vaccine has been initiated and travel or other potential exposure is anticipated before the second dose is due. A person 19 years of age or older who receives 1 dose of TWINRIX® may complete the hepatitis A series with 2 doses of adult formulation single-antigen hepatitis A vaccine separated by at least 5 months. A person who receives 2 doses of TWINRIX® may complete the hepatitis A series with 1 dose of adult formulation single-antigen hepatitis A vaccine or TWINRIX® 5 months after the second dose. A person who begins the hepatitis A series with single-antigen hepatitis A vaccine may complete the series with 2 doses of TWINRIX® or 1 dose of adult formulation single-antigen hepatitis A vaccine. Individuals who are 18 years of age should follow the same schedule using the pediatric formulations of the single-antigen hepatitis A vaccine.

TWINRIX® should be administered by intramuscular injection in the deltoid muscle. Injections in the gluteus can result in a lower response. When given with other vaccines or IG they should be given with different syringes and in different injection sites.

The following are based on ACIP hepatitis A prevention recommendations from from 2006, 2009, and 2018.[36,38,39]

Vaccination of children

All children should receive hepatitis A vaccine at 1 year of age (i.e., 12–23 months). Vaccination should be completed according to the licensed schedules (Tables 1 and 2) and integrated into the routine childhood vaccination schedule. Children who are not vaccinated by 2 years of age can be vaccinated at subsequent visits. In 2015, among children 19–35 months of age, the national vaccination coverage estimate for at least 1 dose of HAV vaccine was 85.8%.[38]

Vaccination of persons at increased risk

Persons at increased risk for hepatitis A should be identified and vaccinated. Hepatitis A vaccine should be strongly considered for persons ≥6 months of age and older who are traveling to or working in countries where they would have a high or intermediate risk of hepatitis A virus infection. These areas include all areas of the world except Canada, Western Europe and Scandinavia, Japan, New Zealand, and Australia. The first dose of hepatitis A vaccine should be administered as soon as travel is considered. For healthy persons 40 years of age or younger, 1 dose of single antigen vaccine administered at any time before departure can provide adequate protection. Unvaccinated adults older than 40 years of age, immunocompromised persons, and persons with chronic liver disease planning to travel within 2 weeks or sooner should receive the first dose of vaccine and also can receive IG at the same visit with separate syringes and at different anatomic sites. Travelers who choose not to receive vaccine should receive a single dose of IG (0.1 mL/kg), which provides protection against HAV infection for up to 1 month.[40–41] For travel up to 2 months, a single dose of IG (0.2 mL/kg) is recommended.[41] Persons whose travel period is more than 2 months should be administered repeat doses of IG (0.2 mL/kg) every 2 months.[41] Hepatitis A vaccine should be administered to infants 6–11 months of age traveling internationally when protection against hepatitis A is recommended.[39]

Other groups that should be offered vaccine include men who have sex with men, persons who use injection and noninjected illicit drugs, contacts of newly arriving adoptees from countries with high or intermediate HAV endemicity, persons who have clotting factor disorders, persons with occupational risk of infection, and susceptible persons with chronic liver disease or who have received or are awaiting liver transplant. Persons with occupational risk include those who work with hepatitis A-infected primates or with hepatitis A virus in a laboratory setting. Persons with chronic liver disease are not at increased risk for HAV infection because of their liver disease alone. However, these persons, including those with chronic hepatitis C infection, are at increased risk for fulminant hepatitis A should they become infected. Susceptible persons who have chronic liver disease should be vaccinated and may be good candidates for vaccination with TWINRIX® to prevent both hepatitis A and B. Susceptible persons who either are awaiting or have received liver transplants should also be vaccinated.

Indications for pre-exposure prophylaxis hepatitis A vaccine

- Routine vaccination of children 12–23 months of age. (Children who have received 1 dose of hepatitis A vaccine before 24 months of age should receive a second dose 6–18 months after the first dose.)
- Any person 2 years of age and older seeking protection from hepatitis A virus (HAV) infection
- Previously unvaccinated persons who live in areas where vaccination programs target older children
- Persons traveling to or working in countries that have high or intermediate endemicity of infection
- Men who have sex with men
- Persons who use injection and non-injected illicit drugs
- Persons who work with HAV-infected primates or with HAV in a research laboratory
- Persons with clotting-factor disorders
- Persons with chronic liver disease, including from chronic hepatitis B or C virus infection
- Persons who anticipate close, personal contact (e.g., household or regular babysitting) with an international adoptee during the first 60 days after arrival in the United States from a country with high or intermediate endemicity. (The first dose should be administered as soon as the adoption is planned, ideally 2 or more weeks before the arrival of the adoptee.)

Post-exposure prophylaxis

In the absence of post-exposure prophylaxis, secondary attack rates of up to 50% have been reported in households, with higher rates of transmission occurring from infected young children than from infected adolescents and adults.[42] Attack rates among persons exposed to HAV-infected food handlers are generally lower.[43] Persons who have recently been exposed to HAV and who have not been vaccinated previously should be administered a single dose of single-antigen hepatitis A vaccine or IG (0.1 mL/kg) as soon as possible, within 2 weeks after exposure.[39, 41]

As of 2018, vaccine (either HAVRIX® or VAQTA®) is recommended as post-exposure prophylaxis in healthy persons ≥12 months of age because it induces active immunity, which provides enduring protection, has high acceptability and availability, and is easy to administer.[39]

IG is typically used for post-exposure prophylaxis of hepatitis A in susceptible persons who are children younger than 12 months of age, immunocompromised persons, persons with chronic liver disease, or persons for whom vaccine is contraindicated.[39] In addition to post-exposure prophylaxis hepatitis A vaccine, IG may be administered to persons aged >40 years depending on the provider's risk assessment, which includes consideration of age, immune status and underlying conditions, exposure type (risk of transmission), and availability of IG.[39]

Vaccination schedule

See additional details above in vaccination section.

In **Table 1** the dose of HAVRIX® is quantified in ELISA units (EL.U.). HAVRIX® is currently licensed in a 2-dose schedule of 720 EL.U. per dose (0.5 mL) for children and adolescents (12 months through 18 years of age), and 1440 EL.U. per dose (1.0 mL) for adults (19 years of age and older).

Table 1. Recommended doses of HAVRIX® (hepatitis A vaccine, inactivated)*

Group	Age	Dose (EL.U.)†	Volume	No. doses	Schedule§
Children and adolescents	12 months–18 years	720	0.5 mL	2	0, 6–12
Adults	≥19 years	1,440	1.0 mL	2	0, 6–12

* *GlaxoSmithKline*

† *Enzyme-linked immunosorbent assay units*

§ *Months; 0 months represents timing of the initial dose; subsequent number(s) represent months after the initial dose.*

In **Table 2** the dose of VAQTA® is quantified in units (U). The dose and schedule for children and adolescents (12 months through 18 years of age) is 25 U per dose in a two-dose schedule, and for adults (19 years of age and older), 50 U per dose in a two-dose schedule.

Table 2. Recommended doses of VAQTA® (hepatitis A vaccine, inactivated)*

Group	Age	Dose (U)†	Volume	No. doses	Schedule§
Children and adolescents	12 months–18 years	25	0.5 mL	2	0, 6–18
Adults	≥19 years	50	1.0 mL	2	6–18

* *Merck & Co., Inc.*

† *Units*

§ *Months; 0 months represents timing of the initial dose; subsequent number(s) represent months after the initial dose.*

In **Table 3** the dose of Twinrix is quantified in ELISA units (EL.U.) and micrograms. Each dose of Twinrix contains at least 720 EL.U. of inactivated hepatitis A virus and 20 µg of recombinant hepatitis B surface antigen (HBsAg) protein. There is a three dose schedule, given at 0, 1, and 6 months (the same schedule as that used for single-antigen hepatitis B vaccine), and a four dose schedule to accommodate travelers with short notice.

Table 3. Recommended doses of TWINRIX®*
(combined hepatitis A and B vaccine for adults ≥18 years of age only)

Group	Age	Dose†	Volume	No. doses	Schedule§
Adults	≥18 years	720 EL.U. and 20mcg of HBsAg	1.0 mL	3	0, 1m, 6m
Adults	≥18 years	720 EL.U. and 20mcg of HBsAg	1.0 mL	4	0, 7d, 21–30d, 12m

* *GlaxoSmithKline*
† *Enzyme-linked immunosorbent assay units*
§ *Months; 0 months represents timing of the initial dose; subsequent number(s) represent time (d=days, m=months) after the initial dose.*

X. Enhancing Surveillance

Provider education and case investigation

Providers should be educated about the importance of reporting all cases of hepatitis A to their respective health department. A common risk factor identified in persons with acute hepatitis A infection is contact with a previously identified case-patient. Aggressive case investigations of persons with acute disease provide the best opportunity for post-exposure prophylaxis of contacts and for reducing further transmission.

Surveillance and epidemiology staff should routinely investigate suspected cases of viral hepatitis. Basic information that should be routinely collected in the course of a hepatitis A case investigation is described below. Each jurisdiction may have their own protocols for conducting these investigations: CDC is available to provide support as needed (https://www.cdc.gov/hepatitis/contactus.htm).

Information to collect

A case report form that may be useful is available. Additional information may also be collected at the discretion of the local or state health department.

Basic information should include:

- Demographic information
- Clinical details, including
  - Date of onset of illness
  - Symptoms (i.e., jaundice, dark urine, pale stool, fever, headache, malaise, anorexia, nausea, vomiting, diarrhea, abdominal pain)
- Laboratory results
- Vaccination status
- Risk factors and occupation
- Contacts for investigation and prophylaxis

Streamlining reporting using electronic methods

Although many surveillance systems still rely on paper and pencil for data collection, use of data from sources such as electronic medical records, electronic case reporting,[44–50] and clinical laboratory information systems (LIMS) can significantly improve reporting speed, enhance data quality, and reduce workload.

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