ORIGINAL RESEARCH PAPER

Volume-7 | Issue-8 | August-2018 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

HEPATITIS A VIRUS: A REVIEW

Veterinary Science

Dr. Sujata S. Bhave* Senior Research Fellow, Department of Veterinary Public Health, Bombay Veterinary College, Parel, Mumbai - 12. *Corresponding Author

ABSTRACT

Hepatitis A infection is present worldwide and primarily causing a public health concern in developing countries because of its persistent circulation in the environment. The improvement in sanitary conditions and rise in awareness about personal hygiene has resulted in the significant reduction of prevalence in HAV in developed countries in children and the infection has shifted towards adulthood. The virus is transmitted by fecal-oral route or by person-to-person contact or by ingestion of contaminated food or water leading to wide range of symptoms varying from asymptomatic infection to the fulminant hepatitis. Present guidelines recommend HAV vaccination for individuals who are at greater risk of exposure to HAV or with more risk of fulminant disease such as those with chronic hepatitis. The article reviews the epidemiological and clinical aspects of hepatitis A virus.

KEYWORDS

Hepatitis A virus, epidemiology, transmission, prevention

Introduction

Hepatitis A is a historic disease that has probably affected mankind since humans first initiated to live in groups large enough to sustain transmission of the causal agent, hepatitis A virus (HAV) (Lemon et al., 2017). A term, hepatitis A was not introduced until 1967 and is known to be a liver infection caused due to hepatitis A virus (HAV) who primarily replicates in the hepatocytes (Krugman et al., 1967). Globally, hepatitis A virus (HAV) is one of the major infectious causes of acute hepatitis (Lin et al., 2017). According to the WHO statistics, HAV lead to in 13.7 million illnesses and 28000 mortalities in 2010 (Havelaar et al., 2010). This review provides the current information about hepatitis A virus including the aspects such as virology, epidemiology, clinical illness and prevention.

Virology and physiochemical properties

Hepatitis A virus was previously categorized into genus Enterovirus within the family Picornaviridae. However, due to distinguishing characteristics of HAV such as stability, structural configuration, tissue tropism and genetic distance from other members of picornaviruses genera, now it is classified into a separate genus, Hepatovirus, which comprises only human HAV and other closely related mammalian viruses (Zell et al., 2016; Young and Son, 2009).

Hepatitis A virus was first recognized in 1973 by electron microscope and is one of the smallest and structurally simplest RNA animal viruses. The viral particle is non-lipid enveloped, thus showing resistance to ether, chloroform and alcohol. Morphologically, HAV is a single stranded, positive-sense RNA genome with approximately 7.5 kb in length. It is an isometric particle having a diameter of 27-32nm and entirely composed of 70% viral protein and 30% ribonucleic acid (Koff, 1998) and it appears as a sphere with no features under the electron microscope.

Similar to all enteric viruses, HAV is also acid stable and remains infectious below pH3. It retains its infectivity after refrigeration and freezing and is also resistant to heating at 60°C for 30 minutes. Nevertheless, it is readily inactivated by phenol, formaldehyde and ionizing radiation (Siegl et al., 1984).

Epidemiology

Annually, an estimated 1.5 million people are infected with HAV infection (WHO, 2012). However, this can be most probably an underestimated figure due to the asymptomatic infection of hepatitis A and the limitations regarding the collection of epidemiologic information on HAV (Lemon et al., 2017). The incidence of hepatitis A infection is correlated with the socio-economic conditions such as sanitation, density of housing, quality of water and income. Improvements in such conditions all over the world are resulting in the susceptibility shift of infection from early age to young ones and to even older adults.

Conventionally, the endemicity of HAV is categorized into low, intermediate, and high levels depending upon the hygienic and socio-economic status of each geographic area. In parts of Africa, Asia and Central and South America where enedmicity is very high, poor hygienic and sanitary conditions are observed. Infection is acquired in early childhood and majority of infections are asymptomatic. Even if the symptoms occur, they are mild and non-specific. Person to person and contaminated food and water are the main route of transmission in this area.

Developing countries and some parts of developed countries where sanitary and socio-economic conditions are upgraded are considered as areas with intermediate endemicity. Eastern and southern Europe and certain regions of Middle East are the examples where the drop in exposure to HAV in childhood has been observed. However, the rate of infection is higher in grown-up children, adolescents and young adults due to the rise in the level of circulating HAV through the food and waterborne transmission which leads to the outbreak.

Most of the population remains susceptible during the whole adulthood whenever the virus is introduced in the low and very low endemicity regions. However, as the opportunity for the exposure of virus is less, lower cases of hepatitis A infection are observed. In North America region, hepatitis A infection is seen mainly in the community as a whole, mostly affecting young adults and children from lower socio-economic conditions.

Transmission

Hepatitis A is a highly communicable disease, characteristically through the fecal-oral route either by person-to-person contact or by contaminated food and water, particularly shellfish, fruits and salad (Fiore, 2004).

The predominant route for the spread of virus is the transmission via person-to-person contact within the household. At present, outbreak in the community is described as the major source of HAV infection. School-going children, adolescents and young adults are at higher risk of acquiring this infection. Large sized families, lack of education, insufficient waste disposal system and socializing with other peers in the day care centre are associated with the HAV outbreaks. Children between 3-5 years are the main sources of the infection for others because they act as the major source of infection for others as they develop the asymptomatic or unidentified symptoms (Yong and Son, 2009).

Foodborne outbreaks of HAV are linked with raw food for example contaminated bivalve mollusks, salad or fruits, and cooked foods which are usually contaminated via water contaminated with feces in their growing area or during preparation via contact with surfaces contaminated with feces or infected food handlers (Jaykus, 2000). 50% of the total HAV foodborne reported cases are attributed to shellfish ingestion especially when they are often consumed as raw or undercooked (Cliver, 1985).

The effect of HAV transmitted by shellfish is significant. The first

outbreak of Hepatitis A involved 629 cases and was reported in 1955 in Sweden after the consumption of raw oysters. From then, many hepatitis A outbreaks associated with the shellfish consumption have been occurred globally. The magnitude of public hazard is represented by the classic example of the 1988 outbreak in Shanghai, China. The huge outbreak was the result of the consumption of contaminated uncooked raw shellfish by which over 3,00,000 people were affected (Halliday et al., 1991).

Clinical Illness

The average incubation period of HAV is 28 days (ranging from 15-50 days; Crowcroft et al., 2001). Maximum infectivity is seen two weeks before the onset of jaundice and quickly drops afterwards. Infants and children may excrete HAV in feces for longer duration than adults (WHO, 2005).

Symptoms of hepatitis A vary from mild to severe, and can include fever, fatigue, malaise, loss-of-appetite, nausea, diarrhoea, abdominal discomfort, anorexia, myalgia, arthralgia, headache, dark-coloured urine and jaundice. The disease is often asymptomatic in young children (Cuthbert 2001) and the severity of the infection rises with the age. The primary signs of jaundice are urine darkening and a lightening of stools. Signs such as yellowing of the eyes and enlargement of liver can also be seen. The patient may remain infectious for almost seven days after the initiation of the jaundice (Hunter 1998). Most of the adults who are infected show symptoms with acute cholestatic jaundice (Ledner et al., 1985).

The most severe form of the infection is fulminant hepatitis. The case-fatality rate is 80% (Hoofnagle et al. 1995), however, fulminant hepatitis is sporadic (less than 1% of cases overall). But the rates increase with the increasing age and where patients develop liver disease. Children also are at the risk of death. The typical age of the beginning of fulminant hepatic failure in children is observed to be six and a half years (Debray et al. 1997). The most severe complication of HAV infection is the hepatic insufficiency. It is often documented in adult patients. In majority of cases, the prognosis of hepatic insufficiency is rapidly favourable. Occasionally, hepatic insufficiency subsequently results into encephalopathy making emergency liver transplantation a necessary treatment. Relapsing of HAV is noted by increasing levels of serum enzyme, persistence of IgM anti-HAV antibodies and persistent fecal shedding of virus.

Prevention and Control

Hepatitis A is a vaccine-preventable disease. Protection against HAV infection is afforded by: i) Adequate sanitation and housing facilities, as well as personal hygiene; ii) pre- and post-exposure passive prophylaxis with immune globulin (IG), or iii) pre- or post-exposure active immunization with an HAV vaccine (Lemon et al., 2017). Vaccination provides immunity for 10 years. Furthermore, the virus can be inactivated by heating to 850C for 1 minute, Autoclaving (121°C for 20 min), ultraviolet radiation (1.1 W at a depth of 0.9 cm for 1 min), Formalin (8% for 1 min at 25°C), Iodine (3 mg/l for 5 min), Chlorine (free residual chlorine concentration of 2.0 to 2.5 mg/l for 15 min). Shellfish from contaminated areas should be heated to 90°C for 4 min or steamed for 90 sec (WHO, 2005)

References

1. Cliver DO (1985). "Vehicular transmission of hepatitis A." Pub. Health Review, Vol. 13, pp. 235-292.
2. Crowcroft NS, Walsh B, Davison KL, Gungabissoon U (2001). "Guidelines for the control of hepatitis A virus infection." Commun. Dis. Public Health, Vol. 4, pp. 213–227.
3. Cuthbert JA (2001). "Hepatitis A: old and new." Clin. Microbiol. Reviews, Vol. 14, pp. 38–58.
4. Debray D, Cullufi P, Devictor D, Fabre M, Bernard O (1997). "Liver failure in children with hepatitis A." Hepatology, Vol. 26, pp. 1018–1022.
5. Fiore AE (2004). "Hepatitis A transmitted by food." Clin. Infect. Dis., Vol. 38, pp. 705-715.
6. Halliday ML, Kang LY, Zhou TK, Hu MD, Pan QC, Fu TY, Huang YS, Hu SL (1991). "An epidemic of hepatitis A attributable to the ingestion of raw clams in Shanghai, China." J. Infect. Dis., Vol. 164, pp. 852-859.
7. Havelaar AH, Kirk MD, Torgerson PR, Gibb HJ, Hald T, Lake RJ, Praet N, Bellinger DC, de Silva NR, Gargouri N, Speybroeck N, Cawthorne A, Mathers C, Stein C, Angulo FJ, Devleesschauwer B (2015). "World Health Organization global estimates and regional comparisons of the burden of foodborne disease in 2010." PLoS Med, Vol. 12, pp. e1001923.
8. Hoofnagle JH, Carithers RL, Shapiro C, Ascher N (1995). "Fulminant hepatic failure: summary of a workshop." Hepatology, Vol. 21, pp. 240–252.
9. Hunter PR (1998.) "Waterborne Disease. Epidemiology and Ecology." John Wiley and Son Ltd, Chichester, UK.
10. Jaykus L (2000). "Enteric viruses as "emerging agents" of foodborne disease." Int. J. Agric. Food Res., Vol. 39, pp. 245-255.
11. Koff RS (1998). "Hepatitis A." Lancet, Vol. 351, pp. 1643-1649.
12. Krugman S, Giles JP, Hammond J. (1967). "Infectious hepatitis. Evidence for two distinctive clinical, epidemiological, and immunological types of infection." J. Am. Medical Assoc., Vol. 200, pp. 365-373.
13. Lemon SM, Ott JJ, Damme P, Shouval D (2018). "Type A viral hepatitis: A summary and update on the
14. molecular virology, epidemiology, pathogenesis and prevention." J Hepatology, Vol. 68, pp. 167–184.
15. Ledner WM, Lemon SM, Kirkpatrick JW, Redfield RR, Fields ML, Kelley PW (1985). "Frequency of illness associated with epidemic hepatitis A virus infections in adults." American J. Epidemiol., Vol. 122, pp. 226–233.
16. Lin KY, Chen GJ, Lee YL, Huang YC, Cheng A, Sun HY, Chang SY, Liu CE, Hung CC (2017). "Hepatitis A virus infection and hepatitis A vaccination in human immunodeficiency virus-positive patients: A review." World J. Gastroenterol., Vol. 23, pp. 3589-3606
17. Siegl G, Weitz M, Kronauer G (1984b). "Stability of hepatitis A virus." Intervirology, Vol. 22, pp. 218-226.
18. World Health Organization (WHO) (2005) Water Recreation and Disease. Plausibility of Associated Infections: Acute Effects, Sequelae and Mortality by Kathy Pond. Published by IWA Publishing, London, UK. ISBN: 1843390663
19. WHO (2012). "WHO Position paper on hepatitis A vaccines." Wkly. Epidemiol. Rec., Vol. 87, pp. 15.
20. Yong, H. T. and Son, R. Hepatitis A virus – a general overview. International Food Research Journal 16: 455-467 (2009)
21. Zell R DE, Gorbalenya AE, Hovi T, King AMQ, Knowles NJ, Lindberg AM, Pallansch MA, Palmenberg AC, Reuter G, Simmonds P, Skern T, Stanway G, Yamashita T (2016). "ICTV taxonomic proposal 2016.009aS.A.v2.Hepatovirus_ 8sp. Create 8 species in the genus Hepatovirus, family Picornaviridae, order Picornavirales.". https://talk.ictvonline.org/ICTV/ proposals/2016.009aS.A.v2.Hepatovirus_8sp.pdf.