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Editorial: Hepatitis A

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Hepatitis A virus (HAV) is a non-enveloped RNA virus that causes acute inflammation of the liver. Infections in children are often asymptomatic, whereas adults commonly present with jaundice. Although the case fatality rate is generally low, it tends to be higher among individuals with pre-existing liver disease or advanced age. Transmission primarily occurs via the fecal–oral route, through the consumption of contaminated food and water.

Globally, approximately 1.4 million cases of Hepatitis A are reported annually [1]. The resurgence of cases in certain regions is likely attributable to suboptimal vaccine coverage, resulting in reduced population immunity. While the incidence in the United Kingdom has declined, outbreaks have been observed among gay, bisexual, and other men who have sex with men [2]. The infection remains prevalent in low- and middle-income countries, largely due to inadequate sanitation and poor hygienic practices [3]. Following ingestion of contaminated food or water, the virus travels to the liver, where it replicates. Within approximately two weeks of infection, HAV becomes detectable in both blood and feces. Transmission may also occur through intravenous drug use, sexual contact and rarely, blood transfusion. Populations at increased risk include men who have sex with men, international travelers to endemic regions, people who use illicit drugs, individuals with intellectual disabilities and laboratory personnel handling HAV [3]. Those with chronic liver disease, alcohol-related liver injury, or co-infection with HIV, Hepatitis B or Hepatitis C are more likely to develop severe disease.

The average incubation period is around 28 days [3]. Common clinical manifestations include fever, malaise, vomiting and abdominal discomfort. As the disease progresses, patients may develop jaundice, dark urine, diarrhea, arthralgia and hepatomegaly [4]. Infectivity is highest before the onset of jaundice. Although Hepatitis A infection is typically self-limiting, complications such as acute liver failure, pancreatitis, cholecystitis and acute renal failure may occur. In pregnant women, infection has been associated with premature rupture of membranes and preterm labor. Diagnosis is primarily clinical and confirmed through laboratory testing. Liver function tests typically reveal elevated transaminases, alkaline phosphatase and bilirubin levels. IgM anti-HAV antibodies are detectable approximately five days after infection, peak at around one month and may persist for up to six months [4]. IgG antibodies appear during the convalescent phase and confer lifelong immunity. There is no specific antiviral therapy for Hepatitis A; treatment is supportive, focusing on adequate hydration and rest. In cases of fulminant hepatic failure, liver transplantation may be necessary. Vaccination with inactivated HAV vaccines remains the most effective preventive measure. Monovalent vaccines, such as Havrix and Avaxim, are available, while combination vaccines such as *Twinrix and Ambirix* (HAV and HBV) and *ViATIM* (HAV and typhoid) may offer broader protection . *ViATIM* and monovalent vaccines are administered in two doses, six months apart, whereas *Twinrix* requires three doses, with the second given one month after the first and the third at six months. Individuals at high risk should receive pre-exposure prophylaxis. Vaccine-induced immunity is estimated to last at least 25 years [5]. Post-exposure immunization with HAV vaccine, with or without human normal immunoglobulin, is recommended for close contacts within 14 days of exposure. Preventive measures also include maintaining safe drinking water, practising proper hand hygiene and engaging in safe sexual behaviors.

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