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Hepatitis E

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Introduction

Hepatitis E is a single-stranded RNA virus. Hepatitis E infection was recognised as a new human disease in 1980 in India [1]. The virus was characterised in 1991 and was accorded its own genus - *Hepevirus* and its own family - Hepeviridae [2].

The virus is transmitted via the faecal-oral route but is also thought to be a zoonosis with domestic swine and other animals as possible reservoirs for human infection [2]. Five genotypes have been identified, four of which cause human disease. Genotypes 3 and 4 also cause infection in swine [3], and genotype 5 is thought to be of avian origin [2].

Hepatitis E occurs in many areas of the world, including the United Kingdom (UK) [3].

Epidemiology

Global epidemiology

Hepatitis E is endemic in regions of the world where sanitation is poor, and is one of the leading causes of hepatitis in adults in North Africa, Asia and the Middle East [4]. Hepatitis E often occurs in epidemics particularly in South Asia, Mexico and Africa. The largest outbreak of hepatitis E was reported in Kanpur city in Uttar Pradesh in 1991 where over 79,000 clinical cases were reported [5]. This outbreak was attributed to faecal contamination of drinking water supplied from the river Ganges. Nepal, Pakistan, Bangladesh, and Myanmar have also reported outbreaks up to 1999 [6]. In 2004 and 2005, outbreaks involving over 100 deaths were reported in Goz Amer and Goz Beida, Sudanese refugee camps in eastern Chad [7, 8]. In Uganda in 2009, nearly 10,000 cases with 155 deaths were reported [9].

Sporadic cases and small outbreaks have also been reported in high-income countries including France, Germany, and the Netherlands [4]. Some cases have been linked to contact with pigs and the consumption of pork meat. In England, a recent study determined that the overall seroprevalence of hepatitis E was 13% [3].

Hepatitis E in travellers from England, Wales, and Northern Ireland

Surveillance on travel-related cases of hepatitis E is difficult due to incomplete reporting. For example, in 2009, 180 cases of hepatitis E were reported, of these only 22 had a travel history recorded; 18 of these reported recent travel [10]. Between 2004 and 2009, a travel history was recorded for 256 of the 1,294 cases reported. Where a destination was known, the majority had travelled to India (95), Bangladesh (69) or Pakistan (26) [10]. Indigenous transmission could be responsible for many of these cases [11].

In 2008, hepatitis E was confirmed in four passengers returning to the United Kingdom from a world cruise [12]. An epidemiologic investigation found that of the 789 passengers who provided a blood sample, 33 had IgM levels consistent with recent acute infection. They were most likely exposed to the infection from ingesting seafood on board the ship.

Risk for travellers

All travellers to hepatitis E endemic regions are at risk of infection. Those at higher risk include travellers visiting friends and relatives and those visiting areas of poor sanitation [13]. Outbreaks are more common following heavy rain and flooding which results in contamination of water supplies with untreated sewage.

Transmission

The primary source of infection with hepatitis E virus appears to be from faecally contaminated water. Food-borne transmission also occurs through contaminated wild boar meat, undercooked pigs liver and shellfish [12, 14-16]. Direct person-to-person transmission is uncommon but can occur; one case of nosocomial transmission has been reported [12, 17]. Hepatitis E infection has recently been associated with blood transfusion [18] and organ transplantation [19].

Signs and Symptoms

As with the other hepatitis viruses, hepatitis E infection can cause sub-clinical illness, acute, symptomatic hepatitis with jaundice, and fulminant hepatitis. Hepatitis E has been described in organ transplant patients [19].

Following an incubation period of two to nine weeks (mean six weeks) signs and symptoms of hepatitis E infection include jaundice, fatigue, fever, loss of appetite and abdominal pain. The majority of hepatitis E infections are self-limited followed by complete recovery; sub-clinical illness is most common in children. Pregnant women are at the greatest risk of serious illness.

The overall mortality rate ranges from 1-4%, however pregnancy can be associated with fulminant hepatitis and a mortality rate of 20% in the third trimester [13].

Diagnosis of hepatitis E infection is usually made by detection of IgM anti hepatitis E virus in blood.

Treatment

There is no specific treatment for hepatitis E infection, but rather supportive intervention.

Prevention

As most infections are contracted through the faecal-oral route, the risk of acquiring hepatitis E can be reduced by taking [food and water hygiene](#) precautions and by ensuring good personal hygiene.

In particular, pregnant women should be advised about the importance of strict food, water and personal hygiene practices.

Although several studies are currently in progress, there is no vaccine available to protect against hepatitis E.

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Link

[Health Protection Agency Hepatitis E](#)