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Commentary

Transmission Mechanism on Hepatitis A Virus (HAV)

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ABOUT THE STUDY

Infection with hepatitis A virus (HAV) is a prevalent cause of acute viral hepatitis all over the world. The pathogenic mechanisms of hepatitis A are still unknown despite decades of investigation. Because HAV replication is non-pathogenic *in vitro*, it has long been assumed that virus-specific cytotoxic T lymphocytes are to blame for liver damage. However, evidence is mounting suggesting Natural Killer (NK) cells, Natural Killer T (NKT) cells, and even non-HAV-specific CD8+ T cells play a role in liver damage during HAV infection. In a mouse model of hepatitis A, intrinsic mortality of virus-infected hepatocytes has been linked to liver damage. Furthermore, hepatitis A severity has been linked to genetic polymorphisms in host factors such as T cell immunoglobulin-1 (TIM1) and IL-18 binding protein (IL-18BP).

The present state of knowledge about the mechanisms of hepatocellular damage in hepatitis A is summarized in this paper. Different mechanisms may be implicated depending on the circumstances, and they are not always mutually exclusive. A greater knowledge of these pathways might help with the diagnosis and treatment of HAV-related illnesses.

Infection with the Hepatitis A Virus (HAV) is a prevalent cause of acute viral hepatitis all over the world. HAV is spread through the feces-oral route and is most common in resource-poor nations, where outbreaks are generally caused by contaminated food or water. HAV infection is normally self-limiting, although it can cause serious illness and even death. Despite the availability of very efficient vaccinations, an estimated 47 million illnesses and 94,000 fatalities occurred in 2010. Although outbreaks in developed countries are infrequent, the most recent hepatitis A outbreak in the United States began in 2016, affecting 35 states and signaling the reemergence of the disease. There are 38,795 officially recorded cases as of April 16, 2021, resulting in 23,585 hospitalizations (61%) and 372 deaths.

HAV enters the bloodstream by a poorly understood pathway after fecal–oral transfer to acquire access to the liver, the target organ, for growth. The incubation period for acute HAV infection is long (4-6 weeks), during which considerable amounts of virus are released into the faeces. For several weeks before and after the beginning of clinical signs, the virus is found in the blood (viremia). Most patients develop HAV-specific IgM during the height of viral shedding, followed by IgG. Anti-HAV IgM appears in the serum at the same time as increased liver enzymes (Alanine Aminotransferase (ALT) and Aspartate Aminotransferase (AST)), a sign of liver injury.

In the acute and early convalescent phases, cytotoxic T cells are identified and play a key role in viral clearance. HAV is a Picornaviridae virus, which means it's a compact RNA virus. HAV multiplies slowly and does not appear to generate cytopathic effects in cell culture, unlike many other picornaviruses. This is consistent with the fact that acute HAV infection in people has a rather long incubation period. HAV usurps the cellular endosomal sorting complex to exit as membrane-cloaked, quasi-enveloped particles (eHAV), which provides a long-sought explanation for HAV's noncytolytic discharge and dissemination. Because eHAV particles are the sole virion type found in the patient's serum, they are most likely the virus's conduit to the liver.

Due to a lack of patient samples, studying HAV pathogensis in patients has been problematic. Non-Human Primates (NHPs) such as mamosets, owl monkeys, and chimps are vulnerable to HAV and induce disorders that are comparable to those seen in humans; hence they have been used to study the pathogenesis of HAV infection. Insights into the pathogenesis of HAV infection in humans have come from studies in these animal models. The mechanisms of hepatitis A-related liver damage are still unknown. While virus-specific CD8+ T cells have long been thought to be a significant source of HAV- induced liver damage, fresh research reveals that there are other factors at play.

Other immune cells that have been linked to liver injury include non-HAV-specific CD8+ T cells, NK cells, and NKT cells. In a mouse model of HAV infection, intrinsic apoptosis driven by MAVS signaling appears to be responsible for hepatitis. Hepatitis A severity can also be influenced by genetic variations in both the host and viral variables. These processes are not mutually exclusive, and they can cause liver damage in patients by acting alone or in combination. With ever-increasing knowledge and technological advancements, it's almost guaranteed that new mechanisms will be uncovered. Further understanding of these pathways of HAV-induced hepatic injury will throw fresh insight on the pathophysiology of hepatitis A and aid in the development of new therapeutics.

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