Short Communication

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Viral Hepatitis

Ariana Koldas*

Editorial office, Pediatric Infectious Diseases: Open access, United Kingdom

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***Corresponding author:** Ariana Koldas

asianpediatrics@asiameets.com

Editorial office, Pediatric Infectious Diseases: Open access, United Kingdo.

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In the pediatric population, viral hepatitis is one of the most common causes of liver disease. Up to 10% of children at high risk in North America (such as the urban poor) have naturally acquired hepatitis A antibodies [1], and up to 10% of children at high risk in North America (such as the urban poor) have been infected with hepatitis B virus [2]. Hepatitis C is present in 0.2% to 0.4% of North American children aged 5 to 14 [3].

Hepatitis viruses A and B cause acute liver illness, which is commonly accompanied by jaundice and other symptoms. Hepatitis B and C cause chronic liver disease in children that seldom progresses quickly enough to cause morbidity; the main worry is the risk of cirrhosis and liver cancer. Fulminant hepatitis affects a small percentage of children who have been infected with the hepatitis A or B viruses. Delta hepatitis can induce fulminant hepatitis in children with hepatitis B in Europe and Asia, but it is uncommon in the United States. In the impoverished world, the hepatitis E virus causes pandemic sickness, although it is uncommon in the United States. Although the hepatitis G virus has been found in children in the United States, particularly those who have had blood transfusions, there is no evidence that it causes acute or chronic liver damage.

The three primary hepatitis viruses, A, B, and C, should be the emphasis of viral hepatitis research in North America. Epidemiology, pathogenesis, transmission, natural history, the function of the host immune response, and, in the case of hepatitis C, the creation of an effective vaccine should all be studied. Antiviral medicines that are both effective and safe for the hepatitis B and C viruses are desperately needed. Furthermore, new molecular virology techniques should be employed to study the function of unidentified viruses in fulminant hepatitis and inflammatory liver illnesses with viral origins, such as biliary atresia.

Hepatitis A Virus (HAV) used to cause mini-epidemics, especially in places with inadequate sanitation and high population density. It is unknown to what extent children act as a source of HAV for adults. Children's daycare centres appear to play a role in sporadic HAV infection and "minor outbreaks" of HAV. In children, the hepatitis A virus is a known cause of fulminant hepatitis. Since 1997, when a highly effective vaccine was introduced, one of the top research priorities has been to determine the vaccine's impact on the incidence, morbidity, and mortality of HAV infection in children in the post-vaccine era, as well as the cost-effectiveness of implementation strategies like universal early childhood vaccination. The vaccine has been suggested for children with any sort of liver illness [4], but the vaccine's efficacy in this population has not been studied.

Although the epidemiology, natural history, and transmission of Hepatitis B Virus (HBV) in children are well understood, data on the impact of the HBV vaccine on HBV infection rates and HBV-associated hepatocellular carcinoma are only now being reported. Because about 5% of children may not respond to the vaccine, establishing newer techniques to achieve the objective of 100% effectiveness will be a significant subject for future study. The American Academy of Pediatrics has recommended that this vaccine be given to all babies and adolescents in the United States [5]; nevertheless, anti-vaccine activists are a major roadblock to the program's effectiveness. Another issue is that the virus is still being brought into the United States through international adoptees.

Antigen presentation and subsequent T-cell-mediated hepatocytolysis are the pathogenesis of HBV in children with normal immunity. The pathophysiology of HBV in children with HIV-affected immunological defences, congenital immune weakness, or immunosuppressive drug administration following organ donation is poorly understood [6,7].

Hepatitis C Virus (HCV), which was first identified in 1989, is the most common cause of non-A and non-B hepatitis. Since then, understanding of this virus in relation to chronic infection in adults has skyrocketed. The epidemiology, mechanism of transmission, and natural history of the disease in children are all unknown. In children, the influence of quasi-species, mode of acquisition, genotype, disease duration, and cofactors such as liver iron level on the development of liver injury, for example, has not been

elucidated. The rate of maternal-fetal transfer varies between 0% and 36%, with an average of 5% to 6%.

HAV and HBV, adenovirus, CMV, and Epstein-Barr virus are all known viral causes of fulminant hepatitis in children. Hepatitis caused by metabolic, toxic, autoimmune, or drug-related causes make up a small percentage of cases. The majority of cases is of unknown origin and may be caused by viruses that have yet to be identified. Other viruses' roles in this situation could be investigated using molecular virology techniques like subtraction cloning. Timing liver transplantation, treating cerebral edema, and employing hepatic assist devices or liver cell transplantation as a bridge to transplant should all be explored as optimal therapeutic techniques.

Hepatitis B virus is the most common cause of hepatocellular carcinoma worldwide, and HCV is rapidly becoming a prominent cause of this cancer in adulthood. Despite the fact that HBV causes liver cancer in children, the role of HCV in this tumour in children needs to be explored. The capacity to detect molecular indicators of premalignant liver injury in children with HBV and HCV, as well as adults with the condition, may aid in preventing liver cancer, therefore defining the processes of hepatocarcinogenesis by HBV and HCV is critical. Such markers, as well as more sensitive methods of detecting tiny tumours, must be developed in order to improve liver cancer prevention and treatment techniques in both children and adults [8].

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