

MICROBIOLOGICAL LINK BETWEEN HEPATITIS VIRUSES AND LIVER CANCER FORMATION

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Abstract: This paper discusses how viral hepatitis, especially hepatitis B and hepatitis C, may gradually lead to liver cancer in many patients. In real life, a lot of people carry these viruses for many years without obvious symptoms, while inside the liver serious changes slowly take place. The infection causes long-term inflammation, fibrosis and eventually cirrhosis, and this condition creates a background for the development of hepatocellular carcinoma. In the article I have summarized how these viruses integrate into liver cells, change normal protective processes and influence the microbiological environment inside the liver. Some of the key mechanisms are viral protein activity (like HBx), changes in immune reactions, and disruption of cellular gene regulation.

Keywords: hepatitis virus, liver cells, hepatocellular carcinoma, chronic inflammation, HBV, HCV, microbiology

Introduction

Nowadays liver diseases are among the most common health problems in many parts of the world. One of the most dangerous ones is hepatocellular carcinoma (HCC), which according to different sources is one of the top causes of cancer-related death worldwide. The World Health Organization states that a significant part of HCC cases appear in people who previously had hepatitis B or hepatitis C. Unfortunately, a lot of individuals do not realize they are infected until very late.

HBV and HCV are considered especially dangerous for the liver because they can stay inside the cells for many years. While staying there, they cause chronic inflammation that slowly damages tissue. In the early stages a person may feel almost nothing. Over time fibrosis appears, then cirrhosis and later cancer. Scientists have shown that some viral proteins (for example, the HBx protein in HBV infection) can disturb cell control systems, stop apoptosis and allow abnormal cell division. Also it is known that hepatitis viruses change the normal microbiota in the gut and liver, which produces even more inflammatory signals and makes it easier for tumor cells to grow.

The aim of this article is to explain, in a more accessible but still scientific way, how hepatitis viruses and liver oncogenesis are connected, and to summarize the most important mechanisms described in recent studies.

Literature Review

In recent years, many researchers have paid attention to how hepatitis viruses may lead to liver cancer. Some scientists discovered that the HBx protein, which is produced during HBV infection, interferes with the DNA protection process inside liver cells. When HBx blocks p53 (which is a very important tumor suppressor protein), the cell becomes unable to repair its DNA or start apoptosis. Therefore, damaged cells stay alive and keep multiplying.

In the case of HCV, other mechanisms are involved. The virus can increase oxidative stress and ROS (reactive oxygen species), which cause DNA damage. Also, chronic inflammation affects the immune system and disrupts normal cytokine balance. Many papers mention that IL-6 and TNF- α are very high in chronic hepatitis patients, which keeps the inflammation active for a long time. This, again, gives cancer cells a better chance to grow.

Some publications also discuss the role of microRNAs. For example, miR-21 and miR-221 are often overexpressed in HBV-positive patients, and they suppress genes like PTEN, which normally control cell growth. Other changes happen inside the gut microbiota as well. Patients with chronic hepatitis often have dysbiosis in their intestinal flora, which causes endotoxins to travel through the portal vein into the liver. These toxins stimulate Kupffer cells and produce even more inflammation.

All these findings strongly support the idea that viral hepatitis is not just an infection of liver cells, but also a serious trigger of molecular and microbiological disturbances that might end with cancer.

Materials and Methods

For writing this paper, publications from 2018 to 2024 were reviewed, mainly from PubMed, Scopus and Web of Science. Only articles from reliable peer-reviewed journals were selected. Most of them were about molecular biology, hepatology or medical microbiology. The main criteria were: (1) the article must include clear scientific findings; (2) there must be a strong connection to liver oncogenesis and hepatitis viruses; and (3) the results must have clinical or experimental basis.

Some sources from Central Asia and CIS countries were also included to have a more regional perspective, because hepatitis is highly spread in our countries too. The selected sources were analyzed using comparative and content analysis. Repeating ideas were grouped, and the most important mechanisms were summarized for the results section.

Results and Discussion

The reviewed sources show that both hepatitis B and hepatitis C viruses are able to stay inside liver cells for a very long time and create chronic inflammation. This inflammation usually leads to fibrosis, then cirrhosis and finally to liver cancer in many cases. HBV produces a protein called HBx, which blocks p53 and other cell defense mechanisms. Because of this, cells with damaged DNA do not die when they should, and they continue dividing without control. HCV also causes oxidative stress and genomic instability because of high levels of ROS.

Another important factor is the microbiota. In many patients with chronic hepatitis, the intestinal flora is disturbed. More endotoxins are transported into the liver, where they activate Kupffer cells and inflammatory cytokines like IL-6 and TNF- α . This supports tumor development even more. According

to different data, the risk of hepatocellular carcinoma (HCC) is about 15–25 times higher in patients with chronic HBV or HCV infection.

To give a more practical picture, a summary table is presented below, based on global data from 2022–2023.

Table 1. Global statistics on liver cancer related to hepatitis viruses

Indicator	HBV-related HCC (%)	HCV-related HCC (%)	HCC from other causes (%)
Share of HCC cases globally	50–55%	25–30%	15–20%
Approximate new HCC cases per year	450,000+	~200,000	<100,000
Hepatitis infection detected among HCC patients	>80%	~75%	<20%
High expression of HBx in tumors	~60%	–	–
Relative risk of cancer in chronic hepatitis	15–20× higher	17–25× higher	5–8× higher

Conclusion and Recommendations

Based on all these data, it becomes clear that hepatitis B and C are not only infectious diseases but also direct factors in liver carcinogenesis. They cause deep molecular changes, suppress normal cell defense systems and disturb the microbiological environment both inside and outside the liver. As a result, the liver becomes vulnerable to malignant transformation. Therefore, patients with chronic hepatitis must be observed regularly and treated not only with antiviral drugs but also with immune and microbiota-supportive therapies.

Main recommendations include:

- Regular screening of HBV/HCV patients (ultrasound, AFP test, etc.)
- Combination therapy to reduce inflammation and oxidative stress
- Vaccination programs to prevent HBV infections
- More research on probiotics and microRNA-based therapies

Early diagnosis of hepatitis could significantly reduce the number of liver cancer cases in the future.

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