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Review Article

A Comprehensive Review of Etiological Factors and Preventive Strategies for Hepatocellular Carcinoma

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A B S T R A C T

Hepatocellular carcinoma (HCC), the most common primary malignancy of the liver, constitutes a significant global health challenge. It predominantly develops in the context of chronic liver disease and cirrhosis. This review outlines the principle etiological factors associated with HCC pathogenesis, including chronic infection with hepatitis B and C viruses, excessive alcohol consumption, metabolic syndrome, and exposure to environmental toxins such as aflatoxins. In addition, it examines evidence-based preventive strategies aimed at reducing HCC incidence and mortality. These include vaccination against hepatitis B, antiviral treatment for chronic hepatitis infections, lifestyle modifications targeting obesity and alcohol use, and implementation of surveillance programs in high-risk populations. A comprehensive understanding of these risk factors and preventive approaches is critical for effective disease control and improved patient outcomes.

Key Point: Hepatocellular Carcinoma, Aflatoxins, Hepatitis B and C Viruses, Non-Alcoholic Fatty Liver Disease.

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INTRODUCTION

Hepatocellular carcinoma (HCC) is the most common primary malignancy of the liver and a leading cause of cancer-related deaths worldwide. It ranks as the second most common cause of cancer-related mortality globally, with a rising incidence across many regions. The occurrence of HCC is closely tied to geographic and etiological factors, largely due to the uneven global distribution of risk factors such as chronic hepatitis B virus (HBV) and hepatitis C virus (HCV) infections, excessive alcohol consumption, and metabolic syndrome. Understanding these multifactorial causes is critical to developing effective prevention and control strategies. Data from the Global Burden of Disease (GBD) 2019 reveal that the age-standardized incidence rate (ASIR) for HCC was significantly higher in Asia (7.97 per 100,000; 95% CI: 7.15–8.93) and globally (6.51; 95% CI: 5.95–7.16) compared to India. Similarly, the age-standardized mortality rates (ASMRs) were also elevated in Asia (7.22; 95% CI: 6.50–9.94) and globally (5.95; 95% CI: 5.44– 6.44) relative to India. In India, the current incidence, prevalence, and mortality associated with HCC are higher in males. However, the annual rate of change is rising more rapidly in females, suggesting a shifting epidemiological trend. Approximately 90–95% of HCC cases result from the biological consequences of chronic HBV or HCV infections. Nevertheless, the rising global prevalence of metabolic risk factors—including metabolic syndrome, obesity, type 2 diabetes, and non-alcoholic fatty liver disease (NAFLD)—poses a growing threat. These conditions are expected to become the dominant causes of HCC worldwide shortly.

The highlights of the present review are summarized in the following lines. Although the current incidence (2.15% per 100,000), prevalence (2.27% per 100,000), and mortality (2.21% per 100,000) rates of HCC in India remain lower compared to the global data, the annual rates of change in

these parameters are higher in India. Among Indians, the present incidence, prevalence, and mortality related to HCC are higher in males, while the annual rate of change is higher in females. TheNortheastern states have higher incidence, prevalence, and mortality related to HCC, but the Western states of Gujarat, Maharashtra, Goa, and Kerala are emerging as newer hotspots with higher annual rates of change in incidence, prevalence, and mortality.

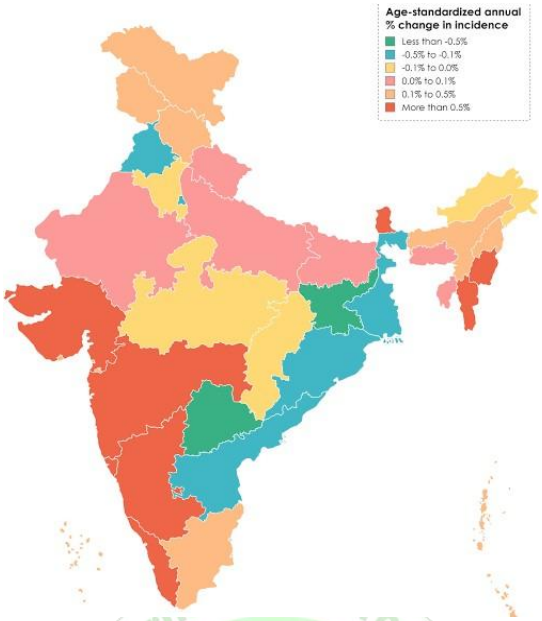
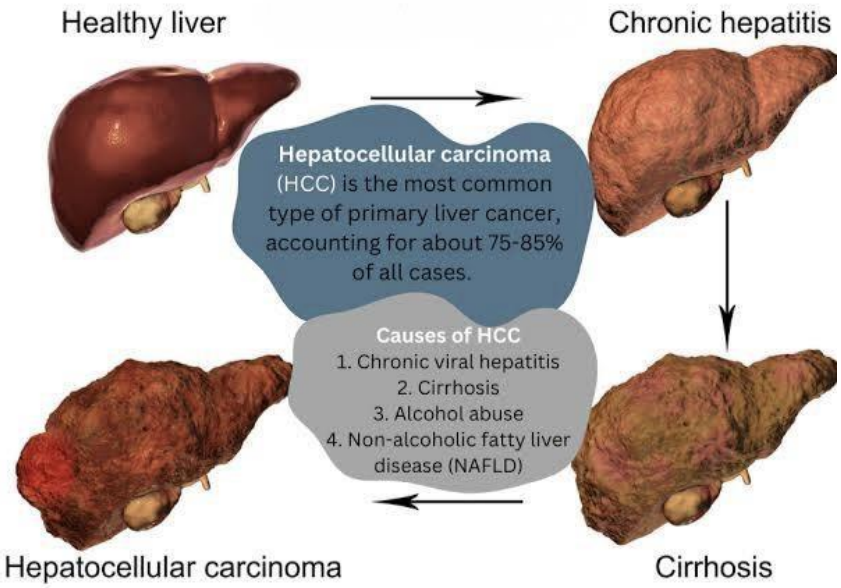


Figure: 1

CAUSES



1. Chronic hepatitis B- virus

Chronic HBV infection is well established as a risk factor for HCC development. In the United States, 25 per cent of patients with HCC are chronic carriers of HBV. Sixty to ninety per cent of patients with HBV-related HCC have cirrhosis, but cirrhosis development is not necessary for HCC development. HBV chronic infection raises HCC risk because of different factors. Genetic alteration in hepatocytes because of viral DNA; viral-induced chronic inflammation with high cellular proliferation and replication errors with low DNA restoration, producing premalignant cells; and HBV-mediated low activity of intrahepatic Natural Killer cells (NK), inducing low immunological surveillance. Gender is important in these patients because there is an association between high testosterone levels and HCC in early tumors.

2. Chronic Hepatitis C- Virus

Hepatitis C virus (HCV) infection accounts for approximately 30% of HCC cases, affecting more than 71 million people worldwide [4,8,19,95]. HCV is a hepatotropic, positive-strand RNA virus in the Flavivirus genus. HCV is divided into seven genotypes and different subtypes, and the distribution of genotypes varies among different

populations, with types 1, 2, and 3 dominating overall [19,96,97]. In contrast to HBV, HCV does not integrate its viral genes into the host cell genome and therefore does not cause direct mutations in host cell genes.

3. Aflatoxin

Aflatoxin is a toxin produced by Aspergillus flavus and A. Parasiticus, which grow in foods like peanuts. It causes alterations in the hepatocyte DNA (see genetic alterations). It is related to HCC in countries where infestation of crops and animal feed is common.18 Aflatoxin metabolism produces aflatoxin B1-8,9-epoxide, a toxic product that induces a G to T mutation of the p53 gene at codon 249 up-regulating insulin-like growth factor II that leads to a reduction of apoptosis and HCC formation.

4. Aristolochic Acid

Aristolochic acid (AA) is a potent nephrotoxin and mutagen found in plants in the genus Aristolochia and used in Chinese herbal medicine.59 Exposure to AA leads to T>A transversions in HCC and the unique molecular signature 22.60 An analysis of The Cancer Genome Atlas database signature 22 in 47% and 56% of the HCCs in China and Southeast Asia, respectively, far exceeding those of Europe (2%) and North America (5%).60 Since the early 2000s, the sales of AA-containing products have been banned in many countries.61 However, AA-containing products are being manufactured and sold worldwide under loose regulations and the AA signature 22 continues to be detected in new HCC cases.

5. Iron overload

Iron overload arises when iron homeostasis is disrupted. Excess iron stores can trigger reactive oxygen species production and lipid peroxidation, which damage cells and activate hepatic stellate cells, leading to accelerated liver fibrosis and hepatocarcinogenesis.African iron overload (AIO) is an acquired iron overload that is associated with HCC (OR, 3.1–10.6).AIO affects 10% of the people living in rural SSA, where iron-rich traditional beers are commonly consumed.However, most traditional beer drinkers (80%) do not develop AIO. Preliminary studies suggest that germline variants in iron regulatory genes might account for a potential hereditary component of AIO.An example is SLC40A1 Q248H, which has an allele frequency of 0.09 among people of SSA descent and is associated with the risk of AIO.

6. 6. Genetic and Congenital Abnormalities

7. Inbred strains of mice have shown genetic susceptibility to cirrhosis and liver cancer. However, in man, it has not been documented. Chinese and Alaskan inhabitants display familial clustering of HCC. The occurrence of HCC is rarely reported in congenital hepatic fibrosis, ataxia telangiectasia, familial polyposis coli, familial cholestatic cirrhosis, fetal alcohol syndrome, situs inversus, and neurofibromatosis. Hereditary tyrosinemia, an inborn error of metabolism, is associated with the maximum risk of liver carcinoma. Within a short period, these patients exhibited faster development of macro-nodular cirrhosis from micronodular cirrhosis, followed by dysplasia and finally HCC. Adenomas may be associated with type I glycogen storage disease but the occurrence of carcinoma is rare. Carcinogenic properties have been attributed to iron through free radical production. An autosomal recessive disorder, Wilson's disease, usually affects the male population and causes cirrhosis *via* copper buildup in the hepatic cells.

Deficiency of alpha-1-antitrypsin, a protease inhibitor, is related to jaundice and cirrhosis during infancy, as well as with pulmonary emphysema and cirrhosis in adults.

8. Anabolic steroids and androgen

Hepatocellular carcinoma (HCC) is the most common primary form of liver cancer. Sex hormones are involved in HCC development and progression. Estrogens and androgens play a role in the mechanisms of the development and progression of the different types of HCA. The main risk factors for developing HCA are oral contraceptives and AAS use, but they tend to develop with a higher incidence in patients with obesity, glycogenosis types 1 and 3, galactosemia, tyrosinemia, and polycystic-ovary syndrome.

9. Obesity

10. The prevalence of obesity has increased to epidemic proportions over the last three decades. Excess body mass is classified as overweight if the BMI is > 25 kg/m2 and < 30 kg/m2, or obese if the BMI is ≥ 30 kg/m2. In addition to the increase in an array of disease processes observed with being overweight or obese, both classifications of excess body mass are associated with a higher risk of developing all cancers, including liver cancer.

11. COMORBIDITIES AND ENVIRONMENTAL FACTORS

Alcoholic liver disease is the second most common risk factor for HCC in the USA, after hepatitis C. Women are more susceptible than men to liver injury from alcohol intake, and women are more likely than men to develop cirrhosis at equivalent alcohol intakes—owing to sex differences in alcohol metabolism. Coexisting viral hepatitis increases the effect of excessive alcohol intake on the risk of HCC.NASH is also emerging as a risk factor for HCC in many developed countries. Obesity, a major risk factor for NASH, has increased in the USA; contemporary survey data suggest that one-third of US adults are obese. Although the increased number of people with NASH (as a result of increases in the prevalence of both obesity and the metabolic syndrome) is believed to contribute to the rising incidence of HCC, few population-based data support this assumption, as the loss of liver steatosis with the development of cirrhosis makes it difficult to prove a history of NASH at the time of HCC diagnosis. Aflatoxin B is a mycotoxin that acts synergistically with HBV in the pathogenesis of HCC.50 Aflatoxin causes DNA mutations, particularly of the TP53 gene, that attenuate the tumor suppressor function of p53. This mycotoxin frequently contaminates food in regions with low medical resources, such as sub-Saharan Africa and eastern Asia. Efforts to eliminate aflatoxin B exposure are ongoing in regions with particularly high exposure to this mycotoxin, including China and West Africa.

SYMPTOMS

COMMON SYMPTOMS

- Unintentional weight loss
- Loss of appetite
- Upper abdominal pain(particularly on the right side)
- Feeling of fullness after a small meal
- Nausea and vomiting
- Fatigue or general weakness
- Hepatomegaly
- Splenomegaly
- Swelling or fluid buildup in the abdomen(ascites
- Jaundice
- Itching(pruritus)
- Dark coloured urine and pale stool

ADVANCED STAGE

- Bleeding or bruising easily.
- Confusion or altered mental status.
- Bone pain (if cancer has metastasized.

PATHOPHYSIOLOGY

The etiology of hepatocellular carcinoma. A variety of risk factors have been associated with the development of HCC, including hepatitis viruses, carcinogens, hereditary diseases, metabolic syndrome, and fatty liver disease. The mechanisms by which these etiological factors may induce hepatocarcinogenesis mainly include p53 inactivation, inflammation, oxidative stress, and telomere shortening leading to genomic instability and activation of multiple oncogenic signaling pathways.

Progression to HCC

Ongoing inflammation and injury cause:

1. **Initiating Factors:**
   - High Calorie Diet / NAFLD.
   - Leads to hyperglycemia and hyperlipidemia.
   - Causes insulin resistance.

   Results in dysregulated fatty acid (FA) metabolism and increased de novo lipogenesis Chronic.

   Alcohol Consumption/AFLD Ethanol is metabolized into acetaldehyde and then to acetate. This metabolic process contributes to liver damage via oxidative stress.

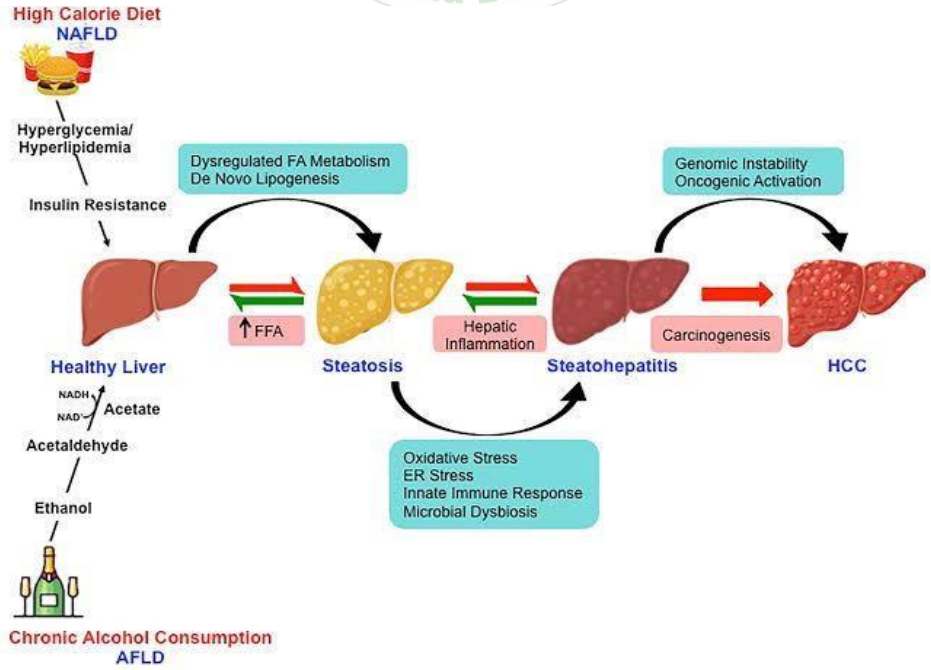
2. ***Steatosis (Fatty Liver)***

   Accumulation of free fatty acids (FFA) in the liver cell. Liver becomes infiltrated with fat, leading to hepatic steatosis.

3. ***Steatohepatitis***

   Persistent steatosis causes hepatic inflammation.

   Contributing mechanisms:
   - Oxidative stress.
   - Endoplasmic reticulum (ER) stress.
   - Innate immune response.
   - Microbial dysbiosis.
   - This stage is called non-alcoholic steatohepatitis (NASH) or alcoholic steatohepatitis.



Firuge: 2

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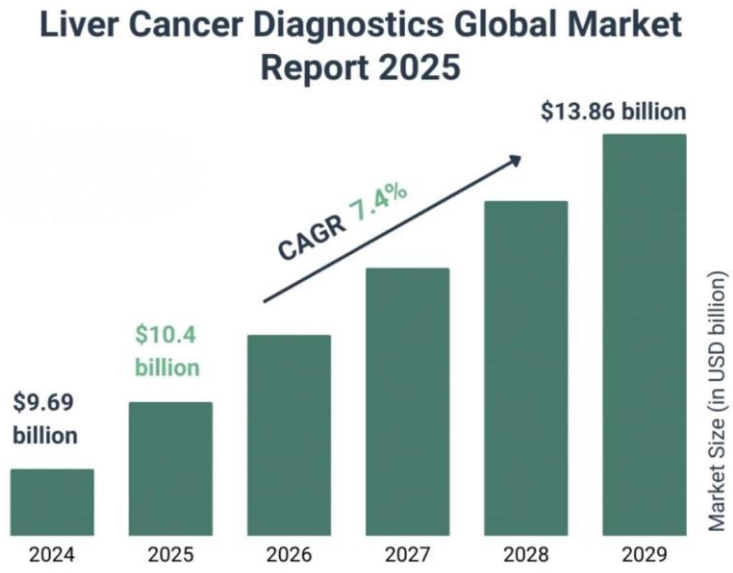
DIAGNOSIS



Figruge: 3

The liver cancer diagnostics market size has grown strongly in recent years. It will grow from $9.69 billion in 2024 to $10.4 billion in 2025 at a compound annual growth rate (CAGR) of 7.3%. The growth in the historic period can be attributed to biopsy techniques and histopathology, development of biomarker detection, evolution of screening programs, advances in genetic testing, and improvements in liver imaging modalities.

Serologic testing, diagnostic imaging, and histology can all aid in diagnosing HCC. Alphafetoprotein (AFP) has been the most commonly tested biomarker for HCC, despite limited sensitivity and specificity. Not only can AFP be elevated in a variety of conditions other than HCC, such as chronic liver disease, pregnancy, and other malignancies, but it can also be normal in a variant of HCC known as fibrolamellar carcinoma.

PREVENTION OF HCC

1. VISUALIZATION TECHNIQUES

With the increasing adoption of laparoscopic and robotic procedures, there has been a corresponding emergence and improvement in technologies aimed at enhancing visualization. Among these advancements, the combination of augmented reality navigation (ARN) and fluorescence imaging has proven to be a valuable tool set in assisting hepatectomy procedures. These techniques may improve outcomes and help decrease the rates of conversion to open operations. In addition to conventional sequences, diffusion-weighted imaging (DWI) enhances the ability to detect and characterize small tumors, and dynamic contrast-enhanced imaging identifies the vascular characteristics of HCC, similar to CT . Although MRI is generally more sensitive and specific for early HCC, its higher cost and limited availability in some regions remain challenges in routine screening programs.

2. HBV VACCINATION OF HCC

HBV vaccines were first licensed in the United States in 1981. Formerly, they were plasma-derived and composed of purified HBsAg. Nowadays, HBV vaccines are predominantly produced by recombinant DNA technology. In Worldwide more than 50% of HCC cases are attributed to chronic HBV infection. A recent systematic review and meta-analysis has revealed a strong correlation of HCC risk in patients with untreated HBV infection with the stage of liver disease and age. Preventing the complications of chronic HBV infection, the World Health Organization (WHO) recommends including hepatitis B vaccination in routine immunization services in all countries. Since this is well past the ages at which the risk of becoming a carrier is high these children effectively now have lifelong protection against hepatitis B-associated liver cancer. This protection has recently been established in Taiwan where the temporal association between the introduction of universal HBV vaccination and a decline in childhood liver cancer strongly suggests a direct causal effect. The vaccine is administered in a 3-dose series and has resulted in high immunogenicity and efficacy.

3. LIVER TRANSPLANTATION

Liver transplantation is the most definitive treatment option for HCC, as it removes not only the tumour but also the unhealthy liver that has limited functional capacity and a tendency to develop additional

metachronous HCCs within the cirrhotic tissue field prone to carcinogenesis. Of the extended criteria for liver transplantation, the University of California San Francisco criteria (a single nodule up to 6.5 cm, or up to three lesions, the largest of which is ≥4.5 cm, with the sum of the diameters ≥8 cm) have been the most widely accepted and have been shown to have excellent post-transplant outcomes136–138. A retrospective multicentre study of 187 consecutive HCC patients enrolled in the downstaging protocol at three liver transplant centres in California showed that liver transplantation was performed after successful downstaging in 58% of patients and their 5-year post-transplant survival was 80%.

4. ANTI-VIRAL THERAPY

DNA integration of hepatitis viruses alters the function of critical genes, promoting malignant transformation of virus-infected liver cells. Treatment of CHB infection aims to control viral replication and prevent the development of complications. There are currently seven drugs available for the treatment of CHB, five NAs and two interferon (IFN)-based therapies. Long-term treatment with NA is often required, and the decision to treat is based on the clinical assessment including the phase of CHB infection and the presence and extent of liver damage.

For example,

Entecavir and tenofovir are much more expensive than lamivudine and telbivudine in most middle and low-income countries. A cost-effectiveness analysis across the Asia-Pacific region has demonstrated that the use of telbivudine as the first-line agent and tenofovir for suboptimal on-treatment responders and drug resistance might be the most cost-effective approach in terms of 2-year HBV DNA suppression.

Data supporting the use of antiviral therapy in preventing the recurrence of HCC after initial anticancer approaches is even less available. Nevertheless, the weight of evidence suggests that treatment of HBV/HCV-related fibrosis will reduce the risk of developing HCC.

5. IMMUNOTHERAPY

Combining different immunotherapies or combining immunotherapies with conventional therapeutic approaches may provide synergistic effects and facilitate the development of personalized medicine. In this study, we provide an overview of the liver immune anatomy, the potential immune mechanisms of HCC, and current (pre)clinical developments in this field. Sorafenib, a receptor tyrosine kinase small-molecule inhibitor, is currently the gold standard and the only systemic therapy approved for the treatment of unresectable advanced HCC, prolonging survival from 4.2 to 6.5 months in the Asia-Pacific study and from 7.9 to 10.7 months in the SHARP study. Sorafenib is a targeted therapy drug used in the treatment of HCC. Researchers have developed nanoparticles that can encapsulate sorafenib and deliver it directly to the tumor site, improving drug efficacy and reducing side effects.

CONCLUSION

Hepatocellular carcinoma (HCC) remains a major global health concern. Many cases are preventable through early intervention, hepatitis B vaccination, lifestyle modifications, and routine surveillance in high-risk populations, underscoring the need for a comprehensive and integrated public health strategy. For patients who develop HCC, optimal care requires a multidisciplinary approach involving surgeons, medical and radiation oncologists, hepatologists, and interventional radiologists. Treatment planning must account for both tumor burden and the extent of liver dysfunction—a critical factor influencing prognosis. Advances in systemic and locoregional therapies have improved survival in patients with advanced disease and have expanded surgical options for those previously considered inoperable.

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