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Review Article

Hepatocellular carcinoma in the context of chronic hepatitis C infection: pathogenesis, diagnosis and therapeutic advances

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ABSTRACT

Hepatocellular carcinoma is one of the most formidable cancers worldwide, and chronic infection with hepatitis C virus (HCV) plays a crucial role in its development. Even though the advent of direct-acting antivirals has greatly improved the rate of viral eradication, the lingering effects of long-term HCV—characterized by ongoing inflammation, advancing fibrosis, and intricate genetic alterations—continue to drive the formation of cancer. Based on my own clinical observations as well as recent scientific findings, this chapter delves deeply into the process by which HCC arises in an HCV-affected liver. I describe the sequential molecular events set off by persistent infection, assess modern imaging and diagnostic techniques (including both tissue-based assessments and liquid biopsy approaches), and review a range of treatment strategies from surgical removal to cutting-edge systemic therapies. In addition, I discuss future possibilities, particularly the potential benefits of precision medicine and the integration of multi-omics data. This review is designed to serve as both a practical resource for healthcare professionals and a catalyst for further research.

Keywords: Hepatocellular carcinoma, Hepatitis C, Direct-acting antivirals, Cirrhosis, Molecular pathogenesis, Advanced imaging, Targeted therapy, Immunotherapy, Precision medicine

INTRODUCTION

Hepatocellular carcinoma (HCC) is the most common primary liver cancer accounting for 80% of liver cancers and is a leading cause of cancer mortality worldwide.[1] A well-recognized etiological factor for HCC is chronic hepatitis C virus (HCV) infection especially in patients with high grade fibrosis or cirrhosis, despite the advent of direct-acting antivirals (DAAs) that has led to a paradigm shift in the management of HCV.[2,3] The mechanism of oncogenesis in HCV-infected livers is complex and multifactorial which may involve direct viral effects, chronic inflammatory responses, and alterations at a genetic level that promotes formation of tumor.[4]

This literature provides a detailed review of HCC in the background of chronic HCV infection. We begin by highlighting the epidemiology and burden of HCV-associated HCC, followed by description of the intricate molecular pathways and cellular mechanisms involved hepatocarcinogenesis induced by HCV. Further focus on risk stratification, diagnostic modalities, current management strategies, and recent advances that are reshaping the therapeutic landscape. At the end future directions in research and clinical practice are proposed to address unresolved and unsolved challenges in this rapidly evolving field.

EPIDEMIOLOGY AND CLINICAL BURDEN

HCC ranks among the top causes of cancer-related deaths globally, with its incidence rising particularly in regions with high prevalence of HCV.[5] Chronic HCV infection is estimated to account for 20–30% of HCC cases in developed countries and even higher percentages in endemic areas.[6] Although the widespread use of DAAs

has led to improved viral clearance rates, the long latency between HCV infection and the development of HCC means that many patients remain at risk even after achieving a sustained virological response (SVR).[7]

In populations with chronic HCV, the annual incidence of HCC ranges from 1% to 4% among cirrhotic patients.[8] Importantly, coexisting factors can synergize with HCV to further increase the risk of carcinogenesis like alcohol abuse, metabolic syndrome, and coinfection with other hepatotropic viruses.[9] Delay in diagnosis owing to the often-asymptomatic nature of early HCC along with limited therapeutic options at advanced stages leads to an increase in the overall clinical burden.[10] Hence, effective risk stratification and detecting the disease early in its course, remains cornerstone to improving outcomes in this patient cohort.

PATHOGENESIS AND MOLECULAR MECHANISMS

The process of hepatocarcinogenesis in the context of HCV is complex, involving both direct and indirect mechanisms that culminate in malignant transformation.

CHRONIC INFLAMMATION AND FIBROGENESIS

Chronic HCV infection is characterized by a persistent inflammatory milieu that leads to repeated cycles of hepatocyte injury, regeneration, and fibrosis. The inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), play key roles in activating hepatic stellate cells, which deposit extracellular matrix proteins and drive fibrosis.[11] Over time, the fibrotic microenvironment fosters genetic instability and epigenetic modifications that contribute to neoplastic transformation.[12]

DIRECT VIRAL ONCOGENIC EFFECTS

HCV is an RNA virus that does not integrate into the host genome. However, several HCV proteins (e.g., core, NS3, and NS5A) have been implicated in perturbing cellular signalling pathways. The HCV core protein, for example, can modulate transcription factors and interfere with apoptosis, thereby promoting cell survival and proliferation.[13] In addition, NS5A has been shown to interact with various intracellular proteins, altering cell cycle regulation and enhancing oncogenic potential.[14]

OXIDATIVE STRESS AND DNA DAMAGE

The chronic inflammatory state in HCV infection leads to the generation of reactive oxygen species (ROS), which in turn induce DNA damage and lipid peroxidation. Oxidative stress is recognized as a critical factor that exacerbates genetic alterations in hepatocytes, paving the way for the accumulation of oncogenic mutations.[15] Moreover, the interplay between oxidative stress and inflammation further accelerates fibrogenesis and cellular transformation.[16]

EPIGENETIC AND GENETIC ALTERATIONS

Emerging research has highlighted the importance of epigenetic changes, including DNA methylation and histone modifications, in HCV-associated HCC.[17] Aberrant methylation patterns may silence tumor suppressor genes and activate oncogenes, thereby facilitating malignant progression. In addition, recent genomic studies have identified recurrent mutations in key pathways, such as the Wnt/β-catenin, p53, and telomerase reverse transcriptase (TERT) promoter mutations, which are closely associated with HCC pathogenesis.[18,19]

RISK FACTORS AND NATURAL HISTORY

Host and environmental factors

The development of HCC in HCV-infected individuals is influenced by a combination of host, viral, and environmental factors. Key host factors include older age, male gender, and genetic predisposition.[20] In addition, comorbidities such as diabetes mellitus, obesity, and alcohol abuse significantly amplify the risk of HCC.[21] Environmental exposures, including aflatoxin exposure in certain regions, further contribute to hepatocarcinogenesis.[22]

Natural history of HCV-induced hepatocarcinogenesis

The progression from chronic HCV infection to HCC is typically a multi-stage process that begins with chronic inflammation, followed by progressive fibrosis and cirrhosis. While cirrhosis is the most significant risk factor, HCC may develop even in the absence of cirrhosis, although less commonly.[23] The latency period from initial HCV infection to the development of HCC can span decades, underscoring the need for long-term surveillance even after viral clearance.[24]

SYNERGISTIC INTERACTIONS WITH OTHER HEPATIC INSULTS

Coinfections with hepatitis B virus (HBV) or human immunodeficiency virus (HIV), as well as lifestyle factors such as alcohol consumption, have been shown to have a synergistic effect in promoting liver injury and carcinogenesis in HCV patients.[25] The cumulative effect of these factors complicates the risk assessment and necessitates a personalized approach to patient management.

DIAGNOSTIC STRATEGIES AND SCREENING

Imaging modalities

Early detection of HCC in HCV-infected patients is critical for improving survival. Ultrasound is the primary

imaging modality recommended for routine screening in patients with cirrhosis due to its non-invasive nature, accessibility, and cost-effectiveness.[26] However, ultrasound may have limitations in sensitivity, particularly in obese patients or those with nodular cirrhotic livers. Consequently, computed tomography (CT) and magnetic resonance imaging (MRI) have emerged as valuable tools for further characterization and staging of lesions.[27] Advanced MRI techniques, including diffusion-weighted imaging (DWI) and contrast-enhanced studies, have improved the detection of small HCC nodules.[28]

Serum biomarkers

Alpha-fetoprotein (AFP) remains the most widely used serum biomarker for HCC screening, despite its limited sensitivity and specificity.[29] Recent advances have led to the identification of novel biomarkers—such as des-gamma-carboxy prothrombin (DCP) and lectin-bound AFP (AFP-L3)—which, when combined with AFP, improve diagnostic accuracy.[30] Emerging studies suggest that multi-marker panels, incorporating genetic and epigenetic markers, may offer even greater predictive value for early HCC detection.[31]

SURVEILLANCE GUIDELINES

International guidelines advocate regular surveillance for HCC in high-risk HCV patients, particularly those with established cirrhosis.[32] The recommended interval for imaging, typically every six months, is based on balancing the benefits of early detection with the costs and risks associated with over-screening.[33] Adherence to these guidelines has been shown to improve early detection rates and overall survival.[34]

MANAGEMENT STRATEGIES

The management of HCC in the setting of chronic HCV infection requires a multidisciplinary approach. Treatment modalities are dictated by the stage of the disease, liver function, and overall patient performance status.

SURGICAL RESECTION AND LIVER TRANSPLANTATION

For patients with early-stage HCC and preserved liver function, surgical resection remains the treatment of choice.[35] Resection offers the potential for cure; however, recurrence rates remain high, particularly in the context of underlying cirrhosis. Liver transplantation is considered the definitive treatment for patients with early HCC and decompensated cirrhosis, as it addresses both the tumor and the diseased liver.[36] Recent refinements in selection criteria and perioperative management have led to improved outcomes and lower recurrence rates following transplantation.[37]

Locoregional therapies

Locoregional treatments, including radiofrequency ablation (RFA), Transarterial chemoembolization (TACE), and selective internal radiation therapy (SIRT), have become integral to the management of intermediate-stage HCC.[38] RFA is particularly effective for small lesions, while TACE is widely used in patients with multifocal disease or those who are not candidates for surgery.[39] Advances in imaging guidance and delivery techniques have enhanced the precision and efficacy of these treatments.[40]

Systemic therapies

For advanced HCC, systemic therapy has evolved rapidly over the past decade. Sorafenib, a multi-kinase inhibitor, was the first agent approved for advanced HCC and has been shown to modestly improve survival.[41] More recently, other tyrosine kinase inhibitors such as lenvatinib and regorafenib have expanded the therapeutic armamentarium.[42] The advent of immunotherapy has further revolutionized the treatment landscape. Immune checkpoint inhibitors—targeting programmed cell death protein 1 (PD-1) and its ligand (PD-L1)—have demonstrated promising activity in clinical trials and are now being integrated into standard care for advanced HCC.[43]

Integration with HCV management

The interplay between HCV treatment and HCC management is complex. While DAAs have significantly improved HCV cure rates, their impact on HCC incidence is nuanced. Some studies suggest a reduction in HCC risk post-SVR, whereas others report cases of "occult" HCC emerging despite viral clearance.[44] This underscores the importance of continued surveillance and a coordinated treatment strategy that addresses both the viral and oncologic aspects of the disease.

RECENT ADVANCES AND FUTURE DIRECTIONS

Advances in immunotherapy and targeted agents

Recent years have witnessed a paradigm shift with the integration of immunotherapy in HCC treatment. Combination regimens involving checkpoint inhibitors—such as the anti-PD-1 antibody nivolumab—with anti-angiogenic agents have demonstrated synergistic effects and improved survival outcomes.[45] In parallel, genomic profiling has enabled the identification of novel molecular targets. Targeted agents aimed at specific pathways, such as the Wnt/β-catenin signalling cascade, hold promise for personalized therapy in HCV-related HCC.[46]

Precision medicine and biomarker development

The advent of next-generation sequencing and other high-throughput techniques has accelerated the discovery of biomarkers predictive of therapeutic response and prognosis. Liquid biopsy technologies—based on circulating tumor DNA (ctDNA) and exosomal markers—are emerging as non-invasive tools for early detection, monitoring treatment response, and identifying mechanisms of resistance.[47] Such precision medicine approaches are expected to tailor therapy to individual molecular profiles, thereby enhancing efficacy and minimizing toxicity.[48]

Novel surgical and interventional techniques

Innovations in surgical techniques, including laparoscopic and robotic-assisted resections, have improved the safety and feasibility of curative interventions in HCC.[49] In the interventional radiology domain, advances in embolic materials and drug-eluting beads have refined TACE procedures, increasing tumor necrosis while sparing healthy liver tissue.[50] These developments have contributed to enhanced outcomes in both surgical and non-surgical settings.

Emerging strategies in chemoprevention and adjuvant therapy

Recent research has focused on identifying chemopreventive strategies to reduce HCC incidence in high-risk HCV patients. Agents such as metformin, statins, and certain anti-inflammatory drugs are under investigation for their potential to modulate the oncogenic microenvironment.[51] In addition, adjuvant therapies post-resection or ablation—aimed at eradicating micro metastatic disease—are being evaluated in clinical trials to reduce recurrence rates.[52]

CHALLENGES AND UNANSWERED QUESTIONS

Despite significant advances, several challenges remain. The mechanisms underlying HCC recurrence, even after curative interventions, are not fully understood.[53] The heterogeneous nature of HCV-induced HCC, compounded by variable host factors, necessitates further research into robust predictive models and individualized treatment algorithms.[54] Moreover, long-term follow-up studies are required to clarify the impact of DAA-induced SVR on HCC recurrence and overall survival.[55] Collaborative efforts across centres and disciplines will be essential to address these critical gaps.

CONCLUSION

Hepatocellular carcinoma in the background of chronic hepatitis C infection represents an incredible challenge for a Hepatologist, reflecting the complex interactions between viral factors, chronic inflammation, and most importantly the host genetics. A thorough overview of the epidemiology, pathogenesis, course of HCV-associated HCC, along with a comprehensive overview of current diagnostic strategies with available treatment modalities is highlighted through this chapter. The management strategy has undergone a paradigm shift with the dynamic evolution of immunotherapy, targeted treatments, and precision medicine.

Detecting the disease early with effective risk stratification remains the cornerstone of successful treatment and for improving the outcomes for the HCC patients. Our approach to deal with this complex pathology effectively, would depend on seamless integration of the unceasing advancements in molecular diagnostics and interventional techniques. Integration of eradication of virus, vigilant surveillance, and tailored oncologic therapy as a part of multidisciplinary strategy, will be pivotal in reducing the worldwide burden of HCC associated with Chronic HCV.

Incorporating the principles of scientific research and discoveries into clinical practice through innovation and active collaboration is key to win this battle against HCV-induced HCC. Research should aim to address the existing challenges and clinicians and researchers should come forward to achieve better prognoses and improve the quality of life for patients afflicted by this devastating condition.

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