ISSN 2218-4333 (online)

World Journal of *Clinical Oncology*

World J Clin Oncol 2024 September 24; 15(9): 1117-1255

Published by Baishideng Publishing Group Inc

World Journal of Clinical Oncology

Contents

Monthly Volume 15 Number 9 September 24, 2024

World Journal of Clinical Oncology

Monthly Volume 15 Number 9 September 24, 2024

Contents

CASE REPORT

LETTER TO THE EDITOR

Contents

NAME OF JOURNAL
World Journal of Clinical Oncology

ISSN
ISSN 2218-4333 (online)

LAUNCH DATE
November 10, 2010

FREQUENCY
Monthly

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PUBLICATION DATE
September 24, 2024

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World Journal of Clinical Oncology

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World J Clin Oncol 2024 September 24; 15(9): 1251-1255

DOI: 10.5306/wjco.v15.i9.1251

ISSN 2218-4333 (online)

LETTER TO THE EDITOR

Timing of antiviral therapy in patients with hepatitis B virus related hepatocellular carcinoma undergoing hepatectomy

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Specialty type: Gastroenterology and hepatology

Provenance and peer review: Invited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's classification

Scientific Quality: Grade A

Novelty: Grade B

Creativity or Innovation: Grade B

Scientific Significance: Grade B

P-Reviewer: Gómez-Aldana AJ

Received: April 23, 2024

Revised: August 8, 2024

Accepted: August 19, 2024

Published online: September 24, 2024

Processing time: 127 Days and 16.1 Hours

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Abstract

Globally, hepatocellular carcinoma (HCC) is among the most prevalent and deadly cancers. Hepatitis B virus (HBV) infection is an important etiology and disease progression factor for HCC. Hepatectomy is a widely accepted curative treatment for HCC, but the long-term survival rate is still unsatisfactory due to the high recurrence rate after resection. Preoperative or postoperative antiviral therapy plays an important role in improving the prognosis for HBV-related HCC patients who underwent hepatectomy. However, many patients miss out on the chance to receive long-term preoperative antiviral medication because their HBV and HCC infections are discovered concurrently, necessitating the start of remedial antiviral therapy in the perioperative phase. Therefore, it is of great value to know when antiviral therapy is more appropriate and whether perioperative rescue antiviral therapy can achieve the effect of preoperative long-term antiviral therapy.

Key Words: Hepatocellular carcinoma; Hepatitis B virus; Hepatectomy; Antiviral therapy; Hepatitis B virus-DNA; Hepatitis B virus-DNA

Core Tip: Hepatocellular carcinoma (HCC) is one of the most common and highly fatal malignancies worldwide and is usually associated with hepatitis B virus (HBV) infection. Hepatectomy is a widely accepted curative treatment for HCC, but the long-term survival rate is still unsatisfactory due to the high recurrence rate after resection. Preoperative or postoperative antiviral therapy plays an important role in improving the prognosis for HBV-related HCC patients who underwent hepatectomy. Therefore, we explored when antiviral therapy is more appropriate and whether perioperative rescue antiviral therapy can achieve the effect of preoperative long-term antiviral therapy in the paper.

Citation: Wan DL, Sun LQ. Timing of antiviral therapy in patients with hepatitis B virus related hepatocellular carcinoma undergoing hepatectomy. *World J Clin Oncol* 2024; 15(9): 1251-1255
URL: https://www.wjgnet.com/2218-4333/full/v15/i9/1251.htm
DOI: https://dx.doi.org/10.5306/wjco.v15.i9.1251

TO THE EDITOR

Hepatocellular carcinoma (HCC) ranks fourth in terms of cancer-related mortality and is the sixth most frequent cancer globally[1]. One of the main causes of HCC is a persistent hepatitis B virus (HBV) infection[2]. Currently, the most effective curative treatments for HCC are liver transplantation and curative surgical resection[3]. Even with these therapies, there is a significant chance that HCC may return; over 70% of patients do so within five years following surgery[4]. Antiviral therapies administered both before and after surgery are acknowledged to be essential for improving the prognosis and length of life for patients with hepatectomy-related HBV-associated HCC. It's still unclear when antiviral medication is more advantageous for patients and if perioperative rescue antiviral therapy can match the impact of preoperative long-term antiviral therapy.

HBV

HBV is a class I carcinogen and chronic HBV infection carries a lifetime risk of developing HCC with a 10%-25% probability. HBV-related HCC is the result of a multi-gene, multi-step interaction mechanism. The synergistic action of various cancer-promoting mechanisms accelerates the evolution of the disease from inflammation to tumorigenesis[5]. The pathogenesis of HBV-related hepatocellular carcinoma (HBV-related HCC) includes HBV gene integration, genomic instability, and activation of cancer-promoting signaling pathways caused by mutations. After HBV enters the host cell, part of the double-stranded DNA is transformed into a stable covalently closed circular DNA (cccDNA) in the nucleus of the host cell and retained in the nucleus of the infected host cell, which is used as a template for transcription of the intermediates of the four virus mRNA[6]. With the progress of the research on the pathogenesis of HBV-HCC, many new mechanisms, such as epigenetics, exosomes, autophagy, metabolic regulation, and immunosuppression, are also being explored. HBV induces immune suppressive cells, such as MDSCs, NK-reg, and T-reg cells, through an immunosuppressive cascade. Excessive immunosuppression could contribute to an HBV persistent infection and the progression of liver fibrosis and HCC[7]. Moreover, it has been reported that HBV variants increase the risk of HCC[8]. Therefore, HBV plays a crucial role in the development and progression of HCC.

ANTIVIRAL

Long-term antiviral therapy to reduce the risk of HCC is one of the cornerstones for the management of chronic HBV[9]. Several studies have confirmed that antiviral therapy can significantly delay the progression of liver fibrosis and reduce the incidence of HCC in patients with HBV[10]. The overall survival and relapse-free survival of the antiviral group were significantly better than those of the non-antiviral group. Therefore, antiviral treatment is crucial for improving the prognosis of HCC patients.

The measure of successful treatment (functional cure) is the persistence of undetectable HBsAg and HBV DNA in serum with or without anti-HBs after a limited course of treatment[11]. However, in the absence of cccDNA eradication, even the functional cure is achieved with antiviral therapy, HBV-infected patients are still at risk of developing HCC, especially those who have already developed cirrhosis[12]. Pegylated interferon-α can enhance cccDNA degradation and anti-HBV immune response, and also has moderate antiviral activity. Nucleos(t)ide analogs are safe and effective in inhibiting HBV replication but have no direct effect on cccDNA. Nucleos(t)ide analogs rarely clear HBsAg and require long-term treatment to prevent recurrence[13,14]. Multiple steps need to be met to achieve the goal of functional cure of HBV: Complete inhibition of HBV DNA replication, suppression of HBsAg production, and restoration of innate and HBV-specific immune responses. Therefore, new antiviral agents and immunomodulatory therapies are being developed, while combinations of several drug classes will also be necessary[15].

Some studies have suggested that partial hepatectomy for HBV-related HCC can lead to reactivation of HBV replication, especially in patients who do not receive antiviral therapy and may lead to liver dysfunction[16]. Therefore,

using antiviral therapy to reduce HBV viral load could lower the risk of HCC recurrence and improve postoperative patient survival. The timing of antiviral initiation is critical but remains controversial.

Preoperative long-term antiviral therapy for at least 24 weeks reduces preoperative HBV-DNA levels in patients, and low preoperative HBV-DNA levels are associated with a reduced incidence of early tumor recurrence and vascular invasion after hepatectomy in patients with HBV-related HCC[17]. Perioperative antiviral therapy significantly reduced the risk of HBV reactivation and improved postoperative liver function, recurrence-free survival (RFS), and overall survival (OS) in both HBsAg-positive and HBV DNA-negative or positive patients before hepatectomy[18-20]. Postoperative adjuvant antiviral therapy can improve liver inflammation and fibrosis, reduce recurrence rate, and improve survival rate in patients with HBV-related HCC after hepatectomy[21-23]. Therefore, patients with HBV-related HCC who need hepatectomy can benefit from antiviral therapy, whether it is given before, after, or during the perioperative period. In a large, multinational study of participants with HBV-related HCC who underwent hepatic resection with curative intent, researchers determined that antiviral treatment was associated with substantial improvements in OS for HBV-related HCC, especially when initiated before or within 6 months from HCC diagnosis. HBV antiviral treatment is only suppressive, and its efficacy is dependent on long-term patient adherence[24]. It has also been demonstrated that RFS and OS of patients who received NAs (ETV or TDF) before and after surgery were significantly better than those who received NAs (ETV or TDF) after surgery[25].

First-line antiviral drugs ETV, TDF, TAF, or TMF are recommended for patients with HBV-related liver cancer. Antiviral therapy should be initiated for HBsAg-positive HCC patients before hepatectomy regardless of their viral load levels or antiHBs status. If HBsAg-positive HCC patients do not receive antiviral therapy, viral reactivation may occur during the process of cancer treatment[26]. In most patients, oral administration of nucleoside antiviral drugs can achieve virus-negative conversion (viral load < 500 copies/mL) in 3-6 months. Long-term (life-long) antiviral therapy with NAs is recommended for patients with HBV-related liver cancer[27]. Patients with preoperative liver decompensation can be given symptomatic and supportive treatment such as liver protection based on antiviral therapy, and elective surgery can be performed after liver function is improved[26]. Patients with HBV-related HCC treated with nucleos(t)ide analogs (tenofovir or entecavir) had significantly improved long-term survival compared with patients treated with other antiviral agents. The efficacies of entecavir and tenofovir in terms of enhancing prognosis after curative treatment of hepatitis B virus-related hepatocellular carcinoma[28]. Whether there is a difference between two highly active antiviral agents (ETV and TDF) in the prognosis of patients with HBV-related HCC after curative resection is still controversial. Some studies have shown that patients treated with TDF have significantly lower postoperative recurrence or mortality rates than those treated with ETV[29,30]. However, some studies have found that there is no statistically significant difference in recurrence rate and mortality rate between ETV and TDF patients with HBV-related HCC after curative treatment[31,32].

Surgical treatment should be performed immediately after the diagnosis of resectable HCC. Perioperative anti-hepatitis B therapy not only can rapidly reduce the level of HBV DNA in a short time but also can effectively prevent the rebound of HBV DNA after operation[33]. The goal of antiviral therapy for HBV-related HCC is to maximize the inhibition of HBV DNA replication, prevent the progression of the disease, reduce liver damage caused by viral replication, improve liver function, and provide a good liver function foundation for HCC treatment based on HCC comprehensive treatment. Thereby significantly improving the prognosis of patients with HBV-related HCC and prolonging the overall survival of patients with HBV-related HCC.

CONCLUSION

HBsAg-positive HCC patients, regardless of HBV DNA level, are recommended to initiate antiviral therapy immediately, and for viral suppression, it is recommended to achieve undetectable levels of the virus as quickly as possible. For patients with HBV-related HCC, ideal preoperative long-term antiviral therapy helps patients to tolerate more extensive hepatectomy and have a better prognosis, but rescue antiviral therapy (reducing preoperative HBV-DNA level to< 4 Log10 copies of DNA/mL) also improved outcomes.

FOOTNOTES

Author contributions: Wan DL is the main author of the manuscript and has made substantial contributions to the reference acquisition and analysis, and manuscript preparation; Sun LQ is the guarantor of integrity of the entire manuscript final version approval; All authors read and approved the final manuscript.

Conflict-of-interest statement: All authors declare that they have no competing interests.

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S-Editor: Liu JH
L-Editor: A
P-Editor: Cai YX

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