Detection of serum hepatitis B Virus antigen and hepatitis C virus antibody from prostate cancer patients in Japan

Ishiguro H 1,2*, Furuya K 2†, Izumi K 2†, Nagai T 1, Kawahara T 2,3, Kubota Y 1,2, Yao M 2 and Uemura H 2,3

1Photocatalyst Group, Kanagawa Academy of Science and Technology, 3-25-13, Tonomachi, Kawasaki-ku, Kawasaki, Kanagawa, Japan
2Department of Urology, Yokohama City University Graduate School of Medicine, 3-9, Fukuura, Kanazawa-ku, Yokohama, Kanagawa, Japan
3Departments of Urology and Renal Transportation, Yokohama City University Medical Center, Yokohama, Japan
†These authors contributed equally

Abstract

Objective: Hepatitis B virus (HBV) or hepatitis C virus (HCV) infection increases hepatocellular carcinogenesis. Some studies suggest that HBV and HCV can activate androgen signaling. We assessed the association between hepatitis virus infection and prostate cancer in the Japanese population.

Patients and methods: We retrospectively reviewed 212 patients who received needle biopsy of the prostate between 2013 and 2014. Hepatitis B surface antigen (HBsAg) and anti-HCV antibody in serum were evaluated by chemiluminescent immunoassay. Prostate cancer was detected in 182 (85.8%) patients.

Results: Serum HBsAg was positive in one (0.5%) prostate cancer patient and one (3.3%) control patient (p = 0.264). Serum anti-HCV antibody was positive in four (2.2%) prostate cancer patients and one (3.3%) control patient (p = 0.537).

Conclusions: Our results suggest that HBV and HCV infection is not associated with prostate cancer development and progression in Japanese patients.

Abbreviations: HBV: hepatitis B virus; HCV: hepatitis C virus; HBsAg: hepatitis B surface antigen; HCC: hepatocellular carcinoma; AR: androgen receptor; CLIA: chemiluminescent immune assay; PSA: prostate-specific antigen; HBx: hepatitis B virus X

Introduction

Bacterial or viral infections may be involved in prostate cancer development and progression [1,2]. In particular, patients with hepatitis C virus (HCV) infection have a higher prevalence and mortality of prostate cancer [3-5]. Hepatitis B virus (HBV) and HCV are well-known carcinogenic viruses in human hepatocellular carcinoma (HCC), and chronic infection increases HCC risk. In addition to these viruses, androgenic steroids might be responsible for HCC [6,7]. Some studies have shown that HBV/HCV infections promote androgen receptor (AR) signaling in HCC [8-14]. Prevalence of HBV-associated HCC is higher in men than women [15], supporting the association between AR signaling and HBV infection. AR signaling is essential for prostate cancer development and progression, therefore, its association with HBV/HCV infection might play an important role in prostate cancer development or progression. Here, we evaluated the association between HBV/HCV infection and prostate cancer in Japanese patients.

Patients and methods

Patients

A total of 212 biopsy specimens were obtained from Yokohama City University Hospital and related hospitals. Thirty specimens were diagnosed as non-cancer tissues and 182 as prostate cancer. Each patient gave informed consent for the use of clinical information, and the study was approved by Yokohama City University Ethical Committee.
In this study, we did not find any association between HBV and HCV infections and prostate cancer. In support of our results, other studies have also failed to show infection of HBV in prostate tissues [2,16]. The association between HBV and AR activation in liver tissues or cells is reported. Hepatitis B virus X (HBx) protein regulates AR expression [9]. HBx protein also regulates AR activity and androgen-dependent gene expression [10-12]. Therefore, HBV might have a role in prostate cancer progression in some cases. Furthermore, some studies suggest the association between HCV infection and prostate cancer. Krystyna et al. [3] reported the association between HCV and prostate cancer in African and African-American individuals. HCV-positive patients have higher cancer prevalence and there is a correlation between HCV infection and prostate cancer progression [4,5]. Kanda et al. [13] showed that HCV regulates androgen signaling through activation of signal transducer and activator of transcription 3. Thus, HCV also might be involved in prostate cancer development and progression through androgen signaling.

Although our study showed no significant associations between HBV and HCV and prostate cancer, it was limited by surveillance of clinical history. We did not distinguish patients with active infection from virus carriers. Therefore, further investigation is needed to clarify the importance of HBV and HCV infection in prostate cancer.

### Discussion

In this study, we did not find any association between HBV and HCV infections and prostate cancer. In support of our results, other studies have also failed to show infection of HBV in prostate tissues [2,16]. The association between HBV and AR activation in liver tissues or cells is reported. Hepatitis B virus X (HBx) protein regulates AR expression [9]. HBx protein also regulates AR activity and androgen-dependent gene expression [10-12]. Therefore, HBV might have a role in prostate cancer progression in some cases. Furthermore, some studies suggest the association between HCV infection and prostate cancer. Krystyna et al. [3] reported the association between HCV and prostate cancer in African and African–American individuals. HCV-positive patients have higher cancer prevalence and there is a correlation between HCV infection and prostate cancer progression [4,5]. Kanda et al. [13] showed that HCV regulates androgen signaling through activation of signal transducer and activator of transcription 3. Thus, HCV also might be involved in prostate cancer development and progression through androgen signaling.

Although our study showed no significant associations between HBV and HCV and prostate cancer, it was limited by surveillance of clinical history. We did not distinguish patients with active infection from virus carriers. Therefore, further investigation is needed to clarify the importance of HBV and HCV infection in prostate cancer.

### Conclusion

We investigated whether HBV and HCV infection is linked to prostate cancer progression. Although HBV and HCV infection could accelerate androgen signaling in HCC, virus infection was not prevalent in prostate cancer patients. Future work is needed to understand the
biological mechanisms of prostate cancer progression in patients with HBV or HCV infection.

Acknowledgements

This study was supported by the Regional Innovation Strategy Support Program of MEXT (The Ministry of Education, Culture, Sports, Science and Technology) and Japan Society for the Promotion of Science (JSPS) KAKENHI for Grant-in-Aid for Exploratory Research (Grant Number 26670709).

Conflicts of interest

The authors declare that there is no conflicts of interest.

References


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