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OPINION ARTICLE

Nuclear Factor-Kappa B p65 Signaling in Renal Cell Carcinoma

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Description

Few researches have examined the role of viruses in the development of Renal Cell Carcinoma (RCC). This study looked at potential relationships between Clinicopathological characteristics, cellular biomarkers such p53, p16INK4a, Ki-67, and nuclear factor-kappa B (NF-B), and Epstein - Barr virus (EBV) infection in RCC tumours.

Over 90% of renal neoplasms are caused by Renal Cell Carcinoma (RCC), one of the top 10 most prevalent cancers worldwide. A diverse category of malignancies known as RCCs develop from renal tubular epithelial cells. These tumours can be linked to hereditary disorders, have a variety of clinical presentations, and are frequently asymptomatic. Most cases of RCC are clear cell, papillary, and chromophobe RCCs. Recent developments in genetics and molecular biology of the renal cell have linked the condition to a number of inherited and non-hereditary risk factors, although they do not fully account for all occurrences of RCC. At least 15% of all human malignancies, including Epstein-Barr virus, may be caused by oncogenic viruses, according to mounting data (EBV).

EBV, also known as human herpes virus 4, belongs to the subfamily Gammaherpesvirinae of the genus Lymphocryptovirus, which after a primary infection induces lifelong latent infections in memory B cells. A primary lytic infection in the oropharynx, which may be asymptomatic or present as infectious mononucleosis, may result in an infection with this pervasive double-stranded DNA virus in around 90% of the general population. EBV was found in the proximal tubule cells of human kidney tissue samples taken from patients with chronic interstitial nephritis as well as in the renal biopsies of people who had glomerular mesangial damage. Additionally, transgenic mice have been shown to develop renal tumours when the EBV Nuclear Antigen 2 (EBNA2) is expressed in renal tubule cells. Additionally, mounting evidence points to a role for EBV in the aetiology of RCC. Together, these data reveal the carcinogenic potential of EBV in the renal tissue as well as the likelihood that renal tissue serves as an EBV reservoir. However, it is not yet clear how RCCs and EBV infection might be related.

Another tumour suppressor molecule connected to the Retinoblastoma protein (Rb) pathway is p16INK4a. It is generally known that p16INK4a helps to control cell cycle progression by halting the S phase. Nuclear protein Ki-67 can be used as a marker for cell proliferation. Higher levels of Ki-67 expression are linked to a worse prognosis and have been shown to correlate with the grade of ccRCC tumours. We looked into the frequency of EBV infection in RCC tumours in this study. In addition, the expression of each biomarker was assessed in all RCC tumour specimens and their matching peritumoral tissues due to the significance of p53, p16INK4a, Ki-67, and NF-B in viral oncogenesis. Additionally, for the first time, the relationship between EBV infection, demographic details, and cellular biomarker expression was examined.

There is little information on the connection between viral infections and RCC, despite mounting evidence that viral infections have an impact on the development of cancer. We did this work to look into the potential role of EBV in RCCs given that renal tissue is most likely a reservoir for EBV and this virus has a carcinogenic potential. We looked at 122 RCC cases, 96 samples of normal kidney tissue, and 19 tissue samples from patients who had renal damage. Our findings point to a connection between RCC and EBV infection. In two

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other earlier studies, the prevalence of EBV infection in RCC tumours was estimated to be 15.6% and 29.6%. Our findings and those of the others indicate that EBV infection is widespread in RCCs. Furthermore, despite a recent study finding a link between EBV infection and tumour grade in RCC patients, we were unable to replicate that finding. This might have been because only 27 RCC patients were included in the study described, and two RCC cases had incomplete information regarding the tumour grades. In conclusion, the novel findings of this study suggest that EBV participates in RCC pathogenesis by activating the NF-B p65 signalling pathway, which speeds up tumour growth.