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Southeast Asian Journal of Health Professional

Journal homepage: https://www.sajhp.com/

Editorial

Overview of oncogenic virus and its role on cancer development

Tungki Pratama Umar[1,*]

[1] *Faculty of Medicine, Sriwijaya University, Palembang, Indonesia*

ARTICLE INFO

Article history:
Received 03-09-2022
Accepted 13-09-2022
Available online 20-10-2022

1. Brief Introduction into the Association of Viral Infection and Oncogenic Transformation

Viruses are responsible for promoting some malignancy processes, accounting for an estimated 10% of all cancer cases worldwide (about 1,400,000 cases). Several viruses can cause various types of cancer. These cancer-causing viruses can survive long enough to infect host cells. The presentation may take years and highly depends on host immunity, environmental factors, and host cellular mutations.[1]

Oncogenic viruses can be classified according to their genetic material, including DNA and RNA. Epstein-Barr virus (EBV), hepatitis B virus (HBV), human papillomaviruses (HPV), Kaposi's sarcoma-associated herpesvirus (KSHV)/human herpesvirus 8 (HHV-8), and Merkel cell polyomavirus (MCPyV) are all DNA viruses. Meanwhile, the RNA group includes hepatitis C virus (HCV) and human T-cell lymphotropic virus (HTLV-1). HPV is the most common etiological agent for virus-related cancer, accounting for approximately 636.000 cases in 2016.[2]

Several sociodemographic factors may play a role in the expansion of virus-related cancer. Individuals in the developing countries account for approximately 85% of the global burden of virus-induced cancers. Furthermore, sexual disparities may expose people to various types of cancer. For example, HPV is the most common cause of cancer in women (about 90% of cases), whereas HBV, HCV, and EBV are more common in men (approximately two-thirds of the cases).[2]

2. Mechanism of Virus-induced Tumorigenesis

Viral oncogenesis is associated with three distinct mechanisms, including the encoding of oncogenic viral proteins, the induction of chronic inflammation, the production of tissue injury, and the commencement of genomic instability (mutation) or different host cell alterations (such as cell death mechanism). These mechanisms can suppress immune system activity against viral infection (immune escape process). As a result, viruses can proliferate uncontrollably, generating significant amounts of reactive oxygen species that can drive the carcinogenesis mechanisms. Exposure to co-carcinogenic factors is usually required for malignant transformation (i.e., environmental and chemical factors). Viral carcinogenesis is a species-specific trait that can only cause cancer in humans and no other primates or mammals.[2,3]

3. Viral Detection

The Cancer Genome Atlas (TCGA) has provided a survey of viruses found in cancer patient's tissue. According to the study, HPV can be found in almost all cervical carcinomas and a small percentage of head and neck squamous cell

* *Corresponding author*.
E-mail address: tungkipratama@gmail.com (T. P. Umar).

https://doi.org/10.18231/j.sajhp.2022.013

carcinomas.[4] Meanwhile, HBV and HCV are found in a fraction of liver cancers, and EBV is found in some to gastric cancers.[5] These viruses can be detected using various methods, including whole-genome sequencing (WGS), whole exon sequencing (WXS) for DNA analysis, and RNA sequencing. According to one study, there are 34 viruses found in cancer cells. These viruses are classified into five families: Papillomaviridae, Polyomoviridae, Hepadnaviridae, Flaviviridae, and Herpesviridae, with detection rates ranging from 7.5 to 98.8%. Cancers with positive virus detection include urothelial (BLCA), cervical (CESC), colon (COAD), head and neck (HNSC), liver (LIHC), rectal (READ), and gastric (STAD). Precise detection can be helpful for gaining fundamental insights into carcinogenic pathways, emphasizing the importance of cancer risk assessment and prevention, and establishing targeted cancer therapy.[2,5]

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Author biography

Tungki Pratama Umar, Research Assistant at Department of Clinical Pathology, Faculty of Medicine, Sriwijaya University

Cite this article: Umar TP. Overview of oncogenic virus and its role on cancer development. *Southeast Asian J Health Prof* 2022;5(3):56-57.