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Book review

Human Papillomavirus (HPV)-Associated Oropharyngeal Cancer, D. Miller, M. Stack (Eds.), 1st ed. Springer, Switzerland (2015). 314 pp., $149.00, ISBN: 978-3-319-21100-8

The book deliberates, in thirteen chapters, the epidemiology of human papillomavirus (HPV)-associated oropharyngeal squamous cell carcinoma (OPSCC), its molecular pathogenesis and clinical parameters as well as the oncogenesis and treatment of HPV in OPSCC.

Chapter 1 discusses in details the biology of HPV, with emphasis on the E5-7 proteins, possible routes of infection, prevalence of cross-infection, pathogenesis in OPSCC and possible ways of preventing HPV-OPSCC. It also describes the molecular features of HPV-driven OPSCC in the cell lines UPCI: SCC090 and UPCI: SCC152. Immunohistochemical validity of $p16^{INK4a}$ (CDKN2A) is accentuated especially for detecting the ethnic varieties between Caucasian and African-American OPSCC patients. Gardasil® and Cervarix® vaccinations are briefly viewed and recommended. Educating the patients about the risk of getting HPV infection during copulation, cunnilingus and rimming was stressed. Several sections of chapter 1 are repeated several times in the next chapters. Interestingly, chapter 2 reiterates the transmissibility of HPV in HPV-driven OPSCC which is attributed, first, to unsafe sexual behavior, as underpinned in previous American and few multicenter studies. Moreover, other predisposing factors of developing HPV-driven OPSCC are discussed. This includes smoking of marijuana, excessive intake of some micronutrients and loss of oral 'alveolar' bone. Liaising HPV-driven OPSCC between tobacco and alcohol use is questioned. Chapter 3 repeats several introduced section in the first two chapters and coins a new designation: oral and pharyngeal cancers. However, it contrasts studies which report a white-population striking predilection to studies which deny any racial preferences. Later, chapter 3 discusses some determinants of OPSCC disparity.

Chapter 4 views the significance of E5-7 oncogenes and possible pathways of HPV oncogene-induced malignant transformation. The blamed cellular immortalization pathways are correlated to the causative E6 and E7 oncogenes. The immune system evasion pathways are connected to E5 and E2 oncogenes. Chapter 4 discusses other tethering factors which could promote any of the above mentioned oncogenes. This includes driving mutagenesis by corrupting DNA repair and chromosomal instability. Chapter 5 tackles, in details, the replication of HPV at the microstructural level. The viral and host factors which govern the process of replications are also deliberated.

Chapter 6 views the MicroRNAs which are associated with HPV-positive OPSCC. Tabulating all the identified micro-RNAs and their chromosomal locations, the upregulation of miR-20b (@Xq26.2) as well as of miR-106b and miR-93 (@7q22.1) are discussed and transcribed. Moreover, miR-9, especially miR-9-1 in the CROC-1 locus, and its relation to tonsils and to limiting cancer progression are explored. Chapter 6 concludes with negotiating potentials, rationale of and challenges against MicroRNA as biomarkers in serology and salivary secretions.

Highlighting the role of E6 and E7 in disrupting the p53/Rb tumor suppressor network, Chapter 7 focuses light on the possibility of targeting the PI3 K-mTOR signaling circuitry in HPV-associated oral malignancies as novel precision molecular therapies. Alterations in PI3 KCA, PTEN, STK11/LKB1, AKT1, TSC1, TSC2 RICTOR, and MTOR are considered the most strategic targets. Chapter 8 repeats the major findings of chapter 7; reflecting on the Ras/Raf/MEK/ERK, Mitogen-activated protein kinase 1, NOTCH signaling pathways in explaining molecular oncogenesis associated with HPV-positive cervical squamous cell carcinoma (CSSC) and in briefly relating to nasopharyngeal carcinomas.

Chapter 9 introduces some basic knowledge about the clinical presentation of OPSCC in terms of epidemiology, clinical parameters, staging, characteristic features and the available diagnostic modalities. It also refers briefly to the *'would-be'* potential features of HPV-positive OPSCC. Pursuing this clinical view further, Chapter 10 contrasts CSSC to OPSCC with reference to transmission routes, epidemiology, preventive measures and treatment modalities. Chapter 11 views the surgical perspectives in the management of oropharyngeal cancer with emphasis on the transoral robotic surgery.

Chapter 12 tabulates the underpinned studies of postoperative radiation, used for HPV-positive compared to HPV-negative OPSCCs, after primarily surgical treatment of treating HPV-positive versus HPV-negative OPSCCs. Improved outcome is higher in HPV + OPSCCs. Interestingly, characteristics of high-risk HPV-positive OPSCC patients are tabulated and partially discussed. Ongoing clinical trials of treatment de-intensification for HPV + OPC- which include alternatives to Cisplatin given concurrently with radiation, reduction of radiation dose, Induction chemotherapy with response-adapted radiation as well as minimally invasive surgery and radiation- are overviewed. Chapter 13 repeats several findings of chapter 4 about the immune modulation in the carcinogenesis and investigational immunotherapies of HPV-associated OPSCC.

Unlike Vancouver style sheet, using APA style sheet in citing the reference connives against smooth readability. The overall cited references are 1288 of which 90 references are repeated in several chapters. The most cite authors are W.H. Westra, K. Munger, L. A. Laimins, P. M. Howley, P. F. Lambert, A.K. Chaturvedi, G. D'Souza, C. Fakhry, M. Pawlita and S. Duensing; creating a 12–18% exact-to-near repetition. The most cited journals, however, are "Cancer Research" and "Proceedings of the National Academy of Sciences".

All in All, Springer's HPV-Associated Oropharyngeal Cancer (2015) is much recommended than Springer's HPV and Head and Neck Cancer (2015) and Springer's HPV Infection in Head and Neck Cancer (2016).

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