Cervical Cancer: Open Access

Case Report Open Access

Precision Cervical Cancer Prevention and Treatment: New Ideas and Clinical Consequences

Saria Dbar*

Department of Urology and Pediatric Urology, Russia

Abstract

The third most frequent cancer in women globally is cervical cancer, and ideas and knowledge about its treatment and prevention are quickly advancing. Cervical cancer has been linked heavily to the human papillomavirus (HPV), despite the fact that the virus alone cannot bring about the disease. Since most infections are temporary and may be naturally cured by the host immune system, HPV-driven malignancy is actually a low chance event. Years must pass after a persistent HPV infection before cervical cancer develops. The foundation of this clinical intervention is to understand the carcinogenic pattern and appropriate targets during HPV host contact, and this extended time frame thus presents a perfect opportunity. In this review, we cover the main causes of HPV and cervical carcinogenesis, as well as new concepts and technologies that are being developed for cancer interventions. More urgently, we cover how these concepts and technologies might lead to clinical precision medicine, which could offer patients early diagnosis, early prevention, and early treatment.

Keywords: Cervical carcinogenesis, cervical screening, genome editing tools, HPV integration, NGS based HPV testing

Introduction

A cancer that starts in the cervix is called cervical cancer. It is caused by cells that have the capacity to invade or disseminate to different places of the body growing abnormally. Early on [1], there are frequently no signs visible. Later signs and symptoms could include abnormal vaginal bleeding, pelvic pain, or discomfort during sex. While bleeding after sex might not be a significant problem, it could also be a sign of cervical cancer [1-4].

More than 90% of cases are caused by human papillomavirus (HPV), yet the majority of women who have had HPV infections do not go on to develop cervical cancer. Nearly 50% of high grade cervical pre-cancers are caused by HPV 16 and 18 strains. Smoking, a weakened immune system, birth control pills, beginning sex at a young age, and having numerous sexual partners are other risk factors, albeit they are less significant. Cervical cancer risk is also influenced by genetic factors. Precancerous alterations usually lead to cervical cancer over the course of 10 to 20 years. Squamous cell carcinomas make up about 90% of instances of cervical cancer, adenocarcinomas make up 10%, and other kinds make up a tiny minority. Typically, a biopsy is performed after a cervical screening for diagnosis. The next step is to perform medical imaging to check for the spread of the cancer [5].

With over 529,800 new cases and 275,100 fatalities per year, cervical cancer is still the third most frequent malignancy in women overall. It's important to note that cervical cancer incidence rates differ significantly across industrialised and developing nations. Due to cancer screening programmes and HPV vaccination campaigns supported by sizable public expenditures, cervical cancer incidence rates and mortality rates have steadily decreased in well-developed nations [6].

Discussion

Cervical cancer is still one of the most common malignancies and the top cause of cancer deaths in women in less developed nations, where many women are frequently diagnosed when they are still raising families. For instance, cervical cancer incidence and mortality are trending dramatically upward in China, especially among young women. For Chinese women between the ages of 15 and 44, cervical cancer has risen to the second-most prevalent female cancer and the third-leading cause of cancer mortality. Cervical cancer is the second most common malignancy among women in India. In the second-most populous nation in the world, cervical cancer is thought to be the cause of 77,100 annual fatalities [6] or one-fourth of all cervical cancer deaths worldwide. It is important to note that cervical cancer incidences have been rising in underdeveloped nations in recent years as a result of the absence of reliable screening and prevention techniques.

Important variables that affect HPV persistence and cervical cancer development include: Host Susceptibilities to Cervical Cancer and Latent HPV Infections

Cervical cancer is a chronic, complex illness brought on by a confluence of genetic predispositions and environmental triggers, like many other types of malignancies. It has been found that HPV infections alone are not sufficient to cause cancer, despite being a significant environmental risk factor. The fact that 60% of HPV infections spontaneously recover within a year and 90% regress within two years supports this theory. This phenomenon leaves very few cases that may hold innate predisposition for developing to precancer or cancer. In order to gain a better knowledge of host-virus interactions and the overall aetiology of cervical carcinogenesis, it is crucial to make attempts to uncover inherited genetic risk factors. Warts, Hypogammaglobulinemia, Immunodeficiency [7], and Myelokathexis Syndrome (WHIM) and Hereditary No polyposis Colorectal Cancer (Lynch) Syndrome, two autosomal dominant genetic disorders characterised by extensive HPV infection and high risk of cervical

***Corresponding author:** Saria Dbar, Department of Urology and Pediatric Urology, Russia, E-mail: saradbar@yandex.ru

Received: 12-Jan-2023, Manuscript No. Ccoa-23-86896; **Editor assigned:** 30-Jan 2023, PreQc No. ccoa-23-86896; **Reviewed:** 06-Feb-2023, QC No. ccoa-23-86896; **Revised:** 13-Feb-2023, Manuscript No. ccoa-23-86896 (R); **Published:** 20-Feb-2023, DOI: 10.4172/2475-3173.1000144

Citation: Dbar S (2023) Precision Cervical Cancer Prevention and Treatment: New Ideas and Clinical Consequences. Cervical Cancer, 8: 144.

cancer, provide blatant evidence of genetic factors contributing to cervical carcinogenesis.

HPV integration

One important genetic stage in the development of cervical cancer is the integration of the HPV genome into the host chromosome. Numerous investigations have demonstrated that HPV integration typically entails fragmenting the viral E1 and E2 open reading frames [8], leading to the overexpression of oncogenes E6 and E7. Each of the cellular targets that E6 and E7 have helps to induce malignant transformation. For instance, E6 attaches to and breaks down the pro-apoptotic protein BAK and the tumour suppressor p53, enhancing the host cell's resistance to apoptosis and enabling viral DNA replication. On the other hand, E7 activates cyclin-dependent kinase 2 (CDK2)/cyclin A 58 and CDK2/cyclin E complex, reversing cell cycle arrest, and inhibits the tumour suppressor retinoblastoma 1 (RB1) to release E2F transcription factors [9].

DNA mutation of the host genome

The somatic mutations of the host genome during HPV-induced carcinogenesis have also been a crucial component in studying cervical carcinogenesis, in addition to HPV integrations into the human genome of the host genes. Analysis of DNA mutations is crucial for distinguishing between cancerous and healthy tissue as well as for determining the best course of treatment.

The most thorough genomic landscape research to date was released in Nature Journal, and it used NGS analysis to show both well-known and brand-new high frequency mutations. The scientists demonstrated that whereas PIK3CA (16%), ELF3 (13%), KRAS (8%), and CBFB (8%) were found in ACC 91, EP300 (16%), FBXW7 (15%), PIK3CA (14%), HLA-B (9%), and p53 (9%) were the most often occurring mutations in SCC. Notable findings include the discovery of driver mutations in the oncogenes HLA-B, EP300, and FBXW7 in cervical malignancies [10].

Conclusion

The precise prevention, detection, and treatment of cervical cancer are not only necessary but also now 194 due to the development of new concepts and technologies for cancer therapy. We will be able to forecast the prognosis of individuals with HPV infections at an earlier stage with the help of the molecular mechanism's elucidation that underlies HPV persistence and the associated cervical cancer. The future psychological and financial burdens of cervical screening programmes and HPV vaccination programmes could be significantly reduced by molecular classification based on HPV integration and genetic profiling, which enables clinicians to concentrate medical resources more on high-risk patients whose diseases are actually progressing.

References

1. Jane Erikson (2001) New Program Expanding Low-Income Benefits for Breast & Cervical Cancer Prevention Treatment. 23: 25.
2. Konstantinos Doufekas, Yaa Achampong (2022) Adeola Oration Prevention of Cervical Cancer 17-29.
3. Franco Borruto, Ciro Comparetto (2012) Treatment Follow-up, and Prevention of Papillomavirus Infection and Cervical Cancer 27-326.
4. Kenji Yoshida, Ryohei Sasaki, Hideki Nishimura, Daisuke Miyawaki, Kazuro Sugimur (2012) Cervical Cancer Treatment in Aging Women.
5. Oliver Sartor (2003) Clinical Prostate Cancer in Year Two: New and Promising Therapies for Treatment and Possible Prevention of Prostate Cancer 2: 71.
6. Emma J Davidson, Henry C Kitchener, Peter L Stern (2021) the Use of Vaccines in the Prevention and Treatment of Cervical Cancer 14: 193-200.
7. Symonds P (2022) Chemo radiation The New Gold Standard For Non-Surgical Treatment Of Cervical Cancer 14: 201-202.
8. Timothy R (2021) Rebbeck Abstract IA02: New concepts in precision prevention 13: IA02-IA02.
9. Ahmad Sanad 47 Performance of cone biopsy excision for treatment of cervical intraepithelial neoplasia.
10. Georgios Androutsopoulos, Raj Naik (2019) Standard and Novel Surgical Treatment in Cervical Cancer 165-189.