Chapter

Global Burden of Cervical Cancer

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Abstract

Human papillomavirus (HPV) has caused infections and malignancies worldwide among which is cervical cancer. In 2004 WHO reported that cervical cancer was the most common cause of cancer deaths among women in developing countries. Globally, 570,000 cases per year in women are attributed to HPV, which is about 8.6% of all occurring cancers. Female mortality is estimated at 250,000 with 80% of incidence and mortality rates occurring in Latin America and Sub Saharan Africa (SSA). Cervical cancer demographic variation in 3rd world countries can be attributed to inadequate health care systems and screening process. As one of the most preventable cancers, early screening and vaccination have shown to limit the late stage of the disease. With present studies estimating worldwide incidence at 4.5% a year. The need for preventive measures to halt the progression of a global public health concern like cancer deaths in women cannot be overemphasized.

Keywords: HPV, Incidence, Mortality, Sub Saharan Africa, worldwide estimate, Global trends

1. Introduction

Cervical cancer is the most common cause of cancer deaths among women in developing countries [1]. Human papillomavirus (HPV) has caused severe infections globally including cervical cancer. HPV is responsible for malignancy and mortality in women across the world [2] and has claimed the lives of thousands of women. HPV infections have been estimated to reach 500,000 a year, with an estimated 80 percent being recorded in third world countries. Female mortality is recorded at 250,000 [2].

Research has shown that human papillomavirus causes cervical cancer in women. Early screening and treatment reduce this cancer rate significantly, preventing the formation of late-stage cancer.

The cervical cancer demographic includes mostly women who are of childbearing age. HPV predisposition is seen in the early onset of sexual intercourse, multiple sexual partners, HPV genome, women on oral contraceptive pills, immune-deficient individuals, or smoking lifestyle. Lack of adequate health care systems leading to inadequate screening has precipitated an increase in advanced cancer that is no longer controllable and difficult to treat. Half of the female population who are sexually active and are not immunized will come down with HPV during their adult lives [3].

HPV16 and 18 (high risk strains) have been found in almost all cases of cervical cancer. Women with HPV have no signs even after infection, so early detection

without screening is difficult, and so is the cancer progression. The time lag from the time of infection to the actual HPV disease or cervical cancer development takes a long time, approximately 10 to 20 years.

According to CDC guidelines, HPV vaccines should be administered to girls between 11 and 12 years old [4]. Although cervical cancer is one of the most preventable cancers, present studies estimate worldwide incidence at 4.5% [5] hence the need for preventive measures.

2. Global burden of the disease (incidence and mortality rates)

In 2012 cervical cancer was the fourth most commonly diagnosed cancer in women, with about 527,600 new cases worldwide and 265,700 estimated deaths which was about 7.5% of all cancer deaths in females. More than half were diagnosed in Central, South America and sub-Saharan Africa and with lowest rates in the Middle East, Northern America, Australia and New Zealand, China, and parts of Western Europe [6]. Present study estimates the worldwide incidence at 4.5% a year [5]. Cervical cancer is the second most commonly diagnosed cancer after breast cancer and the third leading cause of cancer death after breast and lung cancers with about 90% of cervical deaths in the world occurring in developing countries, with India alone accounting for about 25% of the total case [7].

The regions with the highest burden of cervical cancer are those not able to provide vaccines and essential screening methods due to inadequate health care system. About 570 000 women developed cervical cancer in 2018 and of that an estimated 311, 000 died from cervical cancer [8] with China and India contributing a large portion of the global burden.

3. Discussion

The Human papillomavirus belongs to the Papilloma viridae family; double-stranded circular DNA virus, protected by an icosahedral protein capsid which is none enveloped. Because there is no host genome integration of viral DNA, HPV types 6, 11,42, and 44 cause infection of lesser severity. Malignant HPV occurs when the P53 suppressor gene and retinoblastoma gene are inactivated due to the presence of oncoproteins E6 and E7. Several types (40, classified in the Alpha papillomavirus genus) are seen to infect mucosal tissue in the anogenital area and each has connections with cancer. Low grade cervical intra epithelial lesions (LSIL), condylomas, and respiratory papilloma are seen in low-grade HPV. The high-risk types can cause squamous and granular high-grade intra epithelial lesions and oropharyngeal cancer. The immune response is responsible for removing most of the HPV from the system. Types HPV16 and HPV18 have vaccines currently in use worldwide. HPV16 and 18 have cancerous lesions of the cervix in about 70% of all cases.

Women with HPV have no signs even after infection, so early detection without screening is difficult, and so is the cancer progression. It is crucial to find out the genomic types as the information can lead to knowledge of the spread, location and geographic areas of HPV infection. Different subsets of HPV16 and HPV18 have their specific geographic locations and specific ethnic groups that they predominate, whereas, in other types such as HPV 58, these parameters are not so exact. The time lag from the time of infection to the actual HPV disease or cervical cancer development takes a long time, approximately 10 to 20 years. It starts with transforming normal cells into precancerous cells and then into metastatic cancer cells

(dysplasia). This formation of koilocytosis in squamous cells also called a clear halo, is displayed by the cell containing a wrinkled, pyknotic nucleus. It is however, to determine the relationship between HPV and precancerous cervical lesions.

The area of metaplastic tissue between the squamous epithelium of the vagina and the glandular tissue of the cervix (susceptible to carcinogenesis) is the cervical transformation zone (CTZ). Cervical cancer is virtually impossible in the absence of sexually transmitted HPV infection [9] and the lack of intermediate progression to pre cancer [10]. HPV infection is the leading cause of cervical intra epithelial neoplasia [11]. Patients with persistent oncogenic HPV infection usually show cervical lesion progression from low to high grade and people with higher genomic copies [12, 13].

4. Conclusion

Cancer is the second leading cause of death in women, as reported by the Centers for disease control (CDC) and prevention in the United States of America. The need for preventive measures to stem cancer deaths in women cannot be overemphasized. Human papillomavirus causes cervical cancers in women, as seen in studies of many reviewed articles. HPV cancers are estimated to be about 100 types of HPV, with many of them being transmitted sexually. HPV is the most common among sexually transmitted diseases. The most carcinogenic forms are HPV types 16, 18, 31, and 35, among others. Other HPV types can cause cervical cancers and might be responsible for a sizeable portion of the cancers. The HPV 16 is the primary type indicated in 20% of HPV infections but which causes 40% of the high grade squamous intra epithelial lesion. HPV 18 is a close second and is implicated in the formation of adenocarcinomas [10].

According to CDC guidelines [4], HPV vaccines should be administered to girls between 11 and 12 years old. Three doses of the vaccine given within three months showed a high efficacy of preventing HPV disease when they become sexually active in later life. HPV is seen when there is an early onset of sexual intercourse and when individuals have multiple sexual partners. HPV screening should be instituted at 21 years of age with a Papanicolaou test (Pap smear test to check for cancer and pre cancers in women) every three years. Women over the age of 30 should be screened every five years, and women over 65 who are negative of previous screening should not necessarily be screened. Early screening has shown early detection and treatment of HPV and thereby reducing mortality in women. The justification for early screening is to offer low-cost accessible means of determining who in the population is likely to develop the disease and provide diagnostic testing and appropriate treatment.

The recommendation is to emphasize early detection of cancer or pre-cancerous cells, especially in vulnerable or very hard and remote communities. Studies showed that half of the women tested in remote locations are unaware of sexually transmitted infections or HPV. Some of those communities also have no HPV vaccine immunization programs [2]. There is a need to train health care workers to teach communities about sexually transmitted diseases. The importance of determining genomic copies and co-infections in HPV 16 and 18 is a better approach to predict the prognosis of HPV instead of relying solely on genotyping [12]. Women with more than one genotype infections were seen to have more cervical lesions. Public health aims to eradicate HPV cancers through effective screening detection programs and vaccinations across the population. Research should go beyond initial screening for HPV and include HPV genotyping to better manage precancerous treatment plans [14]. HPV eradication should be of primary focus.

Declaration

The author has no conflict of interest to declare.

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