

# Cervical Cancer and Role of Screening and Prevention in India

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**Abstract-** Developing countries account for more than a quarter of the global burden of cervical cancer. It is the main factor in female cancer mortality. India lacks a robust government-sponsored screening program me despite alarmingly high figures. This study evaluated the role of cervical cancer screening and prevention in India and reviewed the performance characteristics of available cervical cancer screening tools and prevention methods in order to provide evidence-based recommendations for the use of the most practically useful screening test to be used in field settings with limited resources.<sup>1,6</sup>

**Keywords:** Cervical cancer, HPV, Screening, Prevention.

## I. INTRODUCTION

The cervix, which connects the uterus and the vagina, is the site of the malignancy known as cervical cancer. It results from aberrant cell growth that has the potential to infiltrate or spread throughout the body. Human papilloma virus (HPV), a pathogen that causes sexually transmitted infections, is the main culprit. Consequently, cervical cancer can be prevented by efficient HPV infection prevention measures.<sup>[4][5]</sup>

Cancer of the cervix is one of the most prevalent cancers worldwide. It accounts for 23.3% of all cancer fatalities in India, making it one of the leading causes of death for females. More than three-quarters of these patients have advanced disease, which makes the chances of long-term survival and recovery dismal.<sup>[2]</sup> Pap smear examinations can be used to identify cervical cancer in its early stages. When compared to India, where rates range from 2.6 percent to 6.9 percent among women in communities, developed countries have a range of 68 percent to 84 percent for the percentage of women who receive Pap smear testing.<sup>[3]</sup> However, there are noticeable differences in the distribution of cancer locations across the globe. India alone accounts for one-fourth of the worldwide burden, making rising nations like India more affected by cervical cancer than industrialized ones. It is one of the leading causes of cancer mortality in women between the ages of 30 and 69, accounting for 17% of all cancer deaths. Cervical cancer is estimated to affect 1 in 53 Indian women over their lifetime, compared to 1 in 100 in more developed nations.<sup>[6]</sup>

## II. History of Cervical Cancer

The Greek physician Pericles Hippocrates first wrote about cervical cancer approximately 400 B.C. At the time, it was thought to be incurable. Approximately 2000 years later, a pioneering Italian surgeon's work allowed for the recognition of the pathogenic process. Dr. Rigoni Stern discovered that nuns had a low prevalence of cervical cancer in the middle of the 19th century (Rigoni-stern, 1842). All of these research points to a connection between sexual activity and cervical cancer pathogenesis. As a result, cervical cancer was thought to be very contagious. German scientists Zur Hausen published their findings on transmitting agents in 1976 after finding human papilloma virus DNA in warts and ca cervix. The structure and sequence of HPV were further discovered by Zur Hausen, Gissmann, and their colleagues in 1985. The milestone in the disease's cure came with the subsequent discovery of the HPV vaccination.<sup>[9]</sup>

The latest recent figures show, there are an estimated 466,000 new occurrences of cervical cancer among women each year in the world, with the great majority of these instances occurring in poor nations. The disease is the most common cause of cervical cancer-related mortality in women, accounting for around 80% of the 231,000 women who die from the condition each year in developing countries.

## III. Pathology

Cells that divide uncontrolled and invade neighboring tissues are what cause cancer. DNA alterations are the root cause of cancer. Most DNA alterations that cause cancer take place in regions of DNA known as genes. Genetic alterations are another name for these modifications.<sup>[8]</sup>

A non-keratinizing, stratified squamous epithelium covers the outside of a normal cervix; it is continuous with the squamous epithelium covering the vagina below and borders the mucus-secreting columnar epithelium lining the endocervical canal and its accompanying crypts above. Although this relationship is not continuous, the intersection between the two epithelia often corresponds with the exterior os. Changes in the cervix's size and shape occur throughout puberty, pregnancy, and in certain steroid contraceptive users, which causes the squamo- columnar junction to be performed on the anatomic ectocervix. Squamous cell carcinoma and adenocarcinoma are the two principal histologic abnormalities that make up the bulk of cervical cancer cases. SCC, which is believed to develop from the cervix's transformation zone, accounts for the vast majority of occurrences of cervical cancer (>70%). Squamo-columnar junction, where the squamous and columnar cells of the cervix meet, is the location of the transformation

zone, which with advancing age migrates from the exocervix to the distal endocervical canal. Adenocarcinoma, the second form of cervical cancer, arises from the endocervical cells that produce mucus and makes up around 18% of cervical carcinomas. Adenocarcinoma (4%) and other carcinomas (5%), or malignancies, make up the balance of cervical carcinomas (1.5%). The primary precancerous lesion is known by the designation of cervical intraepithelial neoplasm, or CIN. It can be classified into three categories: CIN1 denotes mild dysplasia, CIN2 denotes moderate dysplasia, and CIN3 includes severe hyperplasia, carcinoma in situ, and invasive carcinomas. The Bethesda method aims to make cytological diagnosis simpler. This approach classifies lesions into two categories: low-grade squamous intraepithelial lesions and high-grade squamous intraepithelial lesions (HGSIL). LSIL stands for low-grade squamous intraepithelial lesions. When cervical cancer is discovered in its late stages, the prognosis is quite bad and treatment is exceedingly difficult.<sup>[10],[11],[12]</sup>

#### IV. Risk Factors

Most cervical cancers are caused by HPV. The median age at which cervical cancer manifests itself is 47 years. Even if skin-to-skin contact with a region of the body that is infected by HPV can spread the infection without sexual contact. The duration of an HPV infection in young women is 8 to 13 months.<sup>[9]</sup>

#### V. Other Risk Factor

1. **Dietary habits:** A diet deficient in fruits and vegetables, as well as being overweight or obese relative to one's ideal weight, increases the risk of cervical cancer.
2. **Sexual activity:** The most common way that HPV is spread is through sexual contact. very early initiation of sexual activity, risky partners. Having several partners and without using condoms
3. **Family history:** Cancer is genetically passed from the mother to her baby or to the sister, who has developed the disease 2-3 times.  
**Smoking:** Smoking raises the risk of squamous cell cancer by sensitizing the body to chemical changes that might cause cancer and by lowering immune function.
4. **Multiple pregnancies:** Pregnant women who have three or more pregnancies have hormonal shifts and weakened immune systems.
5. **Diethylstilbestol (DES):** DES raises the incidence of cervix adenocarcinoma, particularly in women whose mothers used DES while they were expecting.
6. **Oral contraceptives:** According to certain research studies, using oral contraceptives for birth control may increase your chance of developing cervical cancer.

#### VI. Causes

1. The sexually transmitted human papilloma virus is the primary cause of cervical cancer (HPV).
2. Genital warts are brought on by the same virus known as HPV. There are over 100 distinct HPV strains. Only certain strains can lead to cervical cancer; the two most prevalent ones are HPV-16 and HPV-18. being infected with an HPV strain that causes cancer.

In both men and women, HPV can result in other malignancies. among them are:

1. Vulvar cancer
2. Uterine cancer
3. Prostate cancer
4. Anal cancer
5. Colon cancer
6. Tongue cancer

#### VII. HPV

Most sexually active women and men will get the Human papilloma virus (HPV), which infects the reproductive tract most frequently, at some point in their lives. Additionally, some people could contract the same infection again. Humans and males are most susceptible to infection right after starting to have sexual activity. Although penetrative intercourse does not require skin-to-skin contact for transmission, HPV is sexually transmitted. The most prevalent method of transmission is genital touch. Certain HPV strains have a tiny percentage of virus that can linger and develop into cervical cancer. A part of ca of the breast, anus, vulva, vagina, penis and oropharynx are also brought on by infection with certain HPV strains; these cancers can be prevented using primary preventive methods that are similarly effective against cervical cancer.

#### VIII. HPV Vaccine

In India there are two types of HPV Vaccines are available. Both of them are licensed in globally. The prevention of HPV-related illnesses is presently supported by the availability and marketing of two different prophylactic vaccinations, a quadrivalent vaccine that offers protection against Human Papilloma Virus 6, 11, 16, and 18, and a bivalent vaccine that offers protection against HPV 16 and 18. The bivalent vaccination Cervarix (Glaxo Smith Klein, Belgium) and the quadrivalent vaccine Gardasil (Merck, USA) received their licenses was in 2006. From 9 years of age to 45 years of age, people receive the 14 HPV vaccination. Most persons over the age of 26 won't benefit from receiving an HPV vaccine. Children under the age of 15 must receive two doses of the HPV vaccination.

When received before the start of sexual activity or the first exposure to HPV infection, HPV vaccinations are the most effective. From the age of nine years old forward, both vaccines must be given as a 0.5 ml IM in the deltoid area. Girls 9–14 years old who

had two doses of vaccine (at 0 and 6 months) compared favorably to women between 15–25 years of age who received the recommended three doses.<sup>15</sup>

If second vaccination dosage is given sooner than six months following the first dose, then the third dose is necessary. Alternatively, the vaccine can be given using a 3 dose schedule for those under the age of 14 (0.5 ml at 0, 2, and 6 months), and For people who are older than 14, it must be provided on a 3-dose schedule, with a minimum gap of one month between doses 1 and 2 and a minimum gap of three months between doses 2 and 3. Girls over 15 are encouraged to have the vaccine in three doses, whereas girls between the ages of 9 and 14 are advised to receive the bivalent HPV vaccine in two doses (0.5 ml at 0 and 6 months) (0.5 ml at 0, 1 and 6 months). If the second vaccination dose is administered before the fifth month has passed since the first dose, regardless of age, the third dose must be administered. 14 Both immunizations should not be frozen and should be stored at 2 to 8°C. High antibody titers have been observed in both the quadrivalent immunization and the bivalent vaccine with 100% seropositivity for at least 8.4 years and 8 years, respectively. <sup>[16],[17]</sup>

## IX. Symptoms

1. During the menstrual cycle, there may be blood spots or little bleeding.
2. Abnormally prolonged and heavy menstrual bleeding.
3. Bleeding following douching, sex, or a pelvic exam.
4. A rise in vaginal release
5. When having a sexual encounter, there is pain.
6. Blood loss with menopause

## X. Prevention And Control of Cervical Cancer

Around the world, several cervical cancer preventive and control approaches have been created and put into practice. The prevalence of the illness has significantly decreased as a result of these treatments, which include early identification and treatment of precancerous lesions. Cervical cytology (Pap smear), visual acetic acid testing of the cervix, and HPV, testing of the DNA are a few methods for checking for precancerous lesions. Every one of these approaches has unique benefits, drawbacks, and demands on the health system that nations should take into account when designing screening programs. It is quite possible to gradually reduce the prevalence of cervical cancer by immunizing adolescents and women before their first sexual experience and, consequently, before they are exposed to HPV infection. The prevention of the illness depends on raising women's awareness of the dangers of ca cervical and the benefits of screening programs.

## XI. Conclusion

The enormous disease burden of uterine cervix cancer, which is fully avoidable, is unjustified. In less developed regions of the world, especially India, concentrated prevention and control measures can considerably lower the number of cases. Giving up smoking, delaying the commencement of sexual activity until beyond the age of 18, limiting the number of sexual partners, and using condoms are some fundamental prevention measures for cervical cancer. HPV vaccination of the eligible population and early detection and treatment of cervical pre-cancers utilizing a single-visit "screen-and-treat" technique appear promise for low-middle-income countries, particularly for women living in rural and remote places.

## REFERENCES:

1. Globocan Fact Sheets. International Cancer Research. World Health Organization. Available from: <http://www.globocan.iarc.fr/factsheet.asp#WOMEN>.
2. Guidelines for cervical cancer screening. Government of India and WHO Collaborative Program. Available from: [http://www.whoindia.org/LinkFiles/Cancer\\_resource\\_Guidelines\\_for\\_CCSP.pdf](http://www.whoindia.org/LinkFiles/Cancer_resource_Guidelines_for_CCSP.pdf). [Last accessed on 2012 Jan 23].
3. Swan J, Breen N, Coates RJ, Rimer BK, Lee NC. Progress in cancer screening practices in the United States. Results from the 2000 national health interview survey. *Cancer* 2003; 97:1528-40.
4. World Health Organization (WHO). Comprehensive Cervical Cancer Control. A guide to essential practice. Geneva: WHO; 2006.
5. Underwood SM, Ramsay-Johnson E, Dean A, Russ J, Ivalis R. Expanding the scope of nursing research in low resource and middle resource countries, regions, and states focused on cervical cancer prevention, early detection, and control. *J Natl Black Nurses Assoc JNBNA*. 2009;20(2):42.
6. Saurabh Bobdey, Jignasa Sathwara, Aanchal Jain, and Ganesh Balasubramaniam Burden of cervical cancer and role of screening in India, *Indian J Med Paediatr Oncol*. 2016 Oct-Dec; 37(4): 278–285.doi: 10.4103/0971-5851.195751.
7. Rakhi Kunkule, Ruchita Pakale, Swati Jadhav, Review on Cervical Cancer, SDNCRES's Mahalaxmi Institute of Pharmacy, Raigaon, Satara 415020, M.S., India. <http://e-currentscience.com/journal/e/CTPPC>
8. [https://screening.iarc.fr/doc/RH\\_natural\\_history\\_of\\_cc\\_fs.pdf](https://screening.iarc.fr/doc/RH_natural_history_of_cc_fs.pdf)
9. Sanjay, S. P., Kumar, M. A., & Soman, K. P. (2015). AMRITA\_CEN-NLP@ FIRE 2015: CRF Based Named Entity Extractor For Twitter Microposts. In FIRE Workshops (pp. 96- 99). Greenwald, P. (2002). *Cancer chemoprevention*. BMJ, 324(7339), 714-718.
10. Arends MJ. How do cancer cells die? Apoptosis and its role in neoplastic progression. In: Leake R, Gore M, Ward RH. *The biology of gynecological cancer*, London, RCOG Press, 1995, 73–91. 38.
11. Buckley CH. Oncogene expression in gynecological cancer. *The biology of gynecological cancer*, RCOG Press 1995:21–33. 39.

12. Griffin NR, Wells M. Pre-malignant and malignant disease (adenocarcinoma) of the endocervix. Obstetrical and gynecological pathology. 4th ed. Edinburgh: Churchill Livingstone, 1995:323–43
13. WHO: Weekly Epidemiological Record. Geneva, 2014, No. 43, vol 89, pp 465-492. <http://www.who.int/wer/2014/wer8943/en/>
14. Romanowski B, Schwarz TF, Ferguson LM, Ferguson M, Peters K, Dionne M, Schulze K, Ramjattan B, Hillemanns P, Behre U, Suryakiran P, Thomas F, Struyf F: Immune response to the HPV-16/18 AS04-adjuvanted vaccine administered as a 2-dose or 3-dose schedule up to 4 years after vaccination: results from a randomized study. Hum Vaccin Immunother 2014;10:1155-1165.
15. Romanowski B: Long term protection against cervical infection with the human papillomavirus: review of currently available vaccines. Hum Vaccin 2011;7:161-169.
16. Ferris D, Samakoses R, Block SL, Lazcano-Ponce E, Restrepo JA, Reisinger KS, Mehlsen J, Chatterjee A, Iversen OE, Sings HL, Shou Q, Sausser TA, Saah A: Long-term study of a quadrivalent human papilloma virus vaccine. Pediatrics 2014;134:e657-e665.